Research Article
VO₂ Kinetics during Moderate Effort in Muscles of Different Masses and Training Level

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Purpose. To examine the relative importance of central or peripheral factors in the on-transient VO₂ response dynamics to exercise with “trained” and relatively “untrained” muscles. Methods. Seven professional road cyclists and seven elite kayak paddlers volunteered to participate in this study. Each completed two bouts of constant-load “square-wave” rest-to-exercise transition cycling and arm-cranking exercise at a power output 50–60% of the mode-specific VO₂peak presented in a randomized order. Results. In the cyclists, the mean response time (MRT) as well as the phase II VO₂ time constant (τ₂) was significantly slower in the untrained compared with the trained muscles. The opposite was the case in the kayakers. With respect to the relatively untrained muscle groups, while both demonstrated faster VO₂ kinetics than normal (moderately fit) subjects, the kayakers evidenced faster VO₂ kinetics than the cyclists. This suggests that there is a greater stabilizing-counterforce involvement of the legs in the task of kayaking than of the arms for cycling. Conclusions. The results of the present study provide no support for the “transfer” of a training effect onto the VO₂ on-transient response for moderate exercise, but rather support earlier reports demonstrating that peripheral effects may be important in dictating this kinetics.

1. Introduction

The time course of the pulmonary oxygen uptake (VO₂) response to constant-load exercise of moderate intensity can be characterized by two transient phases. In Phase I, the initial, usually rapid, increase in VO₂ is mediated by an increase in cardiac output, or more properly pulmonary blood flow, whilst the gas contents of the mixed-venous blood perfusing the lungs remain similar to those at rest. Phase II transition is triggered by the gas contents of the blood perfusing the lungs being altered by the influence of active muscle metabolism; it therefore represents the blood transport delay between the active muscles and the lungs. During Phase II VO₂ reflects the decreasing mixed venous O₂ content supplementing the continuing increase in pulmonary blood flow. This is characterized by a monoexponential rise in VO₂ up to the asymptotic or steady-state level (Phase III), the time course of which closely reflects that of the increased muscle oxygen consumption [1, 2]. However, if the work rate is appreciably above the individual’s lactate threshold (LT), VO₂ may not reach a steady state, associated with a continued slower rise in VO₂ (slow component; VO₂slow) of a delayed onset. Opinions are divided over whether VO₂ kinetics is limited by the rate of O₂ delivery to the working muscle [3] or by peripheral factors such as oxidative enzyme activity within the muscle mitochondria or the rate at which carbohydrates are processed into the mitochondria at the pyruvate-acetyl CoA site, that is, rate limitation of O₂ utilization by working muscles despite adequate O₂ delivery [4, 5].

It has been repeatedly shown that single-legged training (relatively small muscle mass) causes significant local (peripheral) changes (such as concentration of high energy phosphate compounds, ratio of ATP to ADP, and inorganic phosphate), with only minor alterations of the cardiovascular (central) system [6]. It has also been acknowledged [6] that,
in order to induce contralateral training modifications (cross-training), larger muscle mass that produces both peripheral and central adaptations should be involved. Indeed several reports (e.g., [7]) have demonstrated that arm training did not produce significant alterations in heart rate, stroke volume, or peripheral blood flow, either at rest or during exercise performed with the nontrained muscles (legs). After leg training; however, the increase in the centrally mediated variables was approximately the same in the trained (legs) and the nontrained muscles (arms) [6, 8]. It is indeed widely accepted that there is more of a transfer effect on the central hemodynamic after training with large muscle groups, compared with training with small muscle groups. It is, therefore, suggested that arm muscles have a greater potential for local (peripheral) rather than centrally mediated improvement in function and that central circulatory changes occur in proportion to the muscle mass used during the training. In most of the studies that have examined the transfer of training phenomenon, the conclusions have been based on changes occurring in the maximal aerobic power (VO_{2max}) and its determinates. However, in recent years, the on-transient VO_{2} kinetics during exercise has been considered as a valid indicator of the integrated cardiovascular, respiratory, and muscular systems’ response to meet the increased metabolic demand of the exercise [8, 9]. VO_{2} kinetics has also been shown to be faster in relatively fit individuals and to be speeded by training, both in normal subjects and in those with cardiovascular and/or pulmonary disease [10, 11]. However, there are only a limited number of studies where VO_{2} kinetics has been applied to trained athletes [12, 13].

The metabolic and physiological responses to arm cranking differ markedly from those of leg cycling (see [14, 15] for reviews). At the same absolute power output, arm exercise results in higher rates of VO_{2}, carbon dioxide output (VCO_{2}), ventilation (V_e), heart rate (HR), and greater increases in core temperature (T_{core}), plasma epinephrine, and blood lactate, than does leg exercise [14, 15]. In untrained individuals VO_{2peak} during arm cranking is approximately 60–70% of their leg-cycling VO_{2peak} [15]. When the physiological and metabolic responses to arm exercise are expressed as a percentage of the mode-specific VO_{2peak}, the differences between arm and leg exercise become less pronounced [15, 16]. The above-mentioned differences coupled with established records indicating that arm cranking results in an increased recruitment of type II muscle fibers [15, 17] and that type II muscle fibers have significantly lower metabolic efficiency than type I fibers [1] explains at least partially, why mechanical efficiency is lower in arm cranking than in leg cycling [14, 15, 17].

There is evidence that arm cranking results in slower Phase II VO_{2} kinetics than leg cycling at similar absolute [8] and relative (to mode-specific maximal load) power outputs [17, 18]. Furthermore, arm muscles have been shown to have a lower capillary-to-muscle fiber ratio, reduced total capillary cross-sectional area and may induce intramuscular pressures during exercise that exceed blood perfusion pressure, when compared with leg muscles [14, 15]. It is also well documented that the proportion of type II muscle fibers is significantly higher in the muscles of the upper body compared to those of the lower body [19]. The reduced relative perfusion of arm muscle fibers combined with findings that type II muscle fibers have slower VO_{2} kinetics than type I fibers could result in slower active muscle oxygen consumption kinetics, thereby slowing Phase II VO_{2} kinetics. Several other studies have already compared the on- and off-transient VO_{2} responses of arms and legs [8, 17]. Our present study, however, addresses the issue of the relative importance of central or peripheral factors in the on-transient VO_{2} kinetics to exercise with “trained” and “untrained” muscles in elite competitive athletes specializing in sport disciplines that require intensive and long-term training, predominantly with their arm muscles (kayakers) or leg muscles (cyclists).

2. Material and Methods

Seven professional road cyclists and seven elite flat water kayak paddlers volunteered to participate in this study during the maintenance phase of their normal training, after giving their written informed consent. All procedures were conducted in accordance with ethical standards of the Institutional Committee of the Italian National Olympic Committee and with the Helsinki Declaration of 1975.

Table 1 lists their physical characteristics. Each had trained and competed extensively at national and international levels for 5 to 10 years. Their training regimen included largely intensive and long-term aerobic activities, predominantly with their arm (kayakers) or leg (cyclists) muscles. Subjects came to the laboratory on four occasions to perform arm- and leg-cranking exercise studies. Each test was scheduled at a similar time of day in order to minimize the effect of diurnal biological fluctuation.

2.1. Measurement of Arm-Cranking and Leg-Cycling Peak Oxygen Uptake (VO_{2peak}) and Gas Exchange Threshold (GET).

During the first two visits to the laboratory each subject performed two incremental exercise tests to the limit of tolerance in order to determine the arm-cranking and leg-cycling GET and VO_{2peak}. For the lower limbs, all athletes were tested on a cycle ergometer (Ergoline, Germany). For the upper limbs, the cyclists used a standard arm-cranking ergometer (Technogym, Top.XT, Italy); the kayakers used an arm paddling ergometer (Technogym, K-Race, Italy), mimicking the actual arm movement for which the kayakers’ arm muscles were trained. The subjects were seated upright such that the crank axis of the ergometer was aligned with the glenohumeral joint. The height of the seat was adjusted to allow for a slight bend in the elbow when the crank handles were at their greatest distance from the subject. Additionally, the legs were not braced and the feet were placed on a footrest, mimicking the actual position in the kayak. The subjects were encouraged to use only their arms and shoulders to perform the exercise, whereas the use of lower back and legs was discouraged. For the leg-cycling test, the seat height was adjusted such that the legs were in slight flexion (170°) at the nadir of the down stroke, while the handlebars were set
Table 1: Physical characteristics of subjects by group.

|          | Age       | Height      | Weight     |
|----------|-----------|-------------|------------|
| Cyclists | 24.0 ± 3.7| 175.0 ± 6.7 | 65.9 ± 5.9 |
| Kayakers | 22.0 ± 2.8| 180.6 ± 5.5 | 79.7 ± 7.9 |

Bold letters denote significant difference between groups (P < 0.05).

according to the individual preferences. Handle bar arm-pull was discouraged during leg cycling. During all tests the crank/cycle cadence was strictly kept between 90 and 100 strokes/revolutions per minute (SPM/RPM), respectively (typical rhythm for both activities), despite the fact that the ergometers provided speed-independent power. All three ergometers were calibrated prior to the beginning of the study using a dynamic calibration rig (Cerini, Italy).

After a 15-minute standardized warm-up, consisting of either pedaling or cranking at 60 RPM or SPM at a work rate of 100 and 50 W, respectively, and following a 5-min. rest, the subject then commenced the leg-cycling or arm-cranking task. The power output was increased progressively every minute from an initial work rate for the cyclists of 100 and 50 W for the legs and arms, respectively, and 75 and 100 W for the kayakers in order to bring the subject to the limit of tolerance in 8–12 minutes. This was achieved by increasing the power output in increments of 25 watts-min⁻¹ for leg cycling and 20 watts min⁻¹ for arm cranking until the subject was unable to keep the pedaling (or stroke) rate above 50 per minute.

During both incremental exercise tests, HR, VO₂, VCO₂, and V̇e were measured breath by breath via standard open-circuit spirometric techniques using computerized metabolic cart (Quark b2, Cosmed, Italy). Daily calibration of volume (with 3-L syringe) and pretest calibration of carbon dioxide and oxygen gas analyzers (with precision gas mixers) were carried out. Heart rate was continuously monitored by means of a telemetric system (Polar, Electro, Finland).

Peak oxygen uptake (VO₂max) was defined as the highest average VO₂ during a 30-second period of the last 90 seconds of the test. The criteria for the noninvasive determination of the gas exchange threshold (GET) were as follows. (1) The modified V-slope method [20], in which V̇CO₂ is plotted against VO₂. The GET was defined as the last value prior to the departure of V̇CO₂ versus VO₂ slope from linearity. (2) A systematic increase in the ventilatory equivalent for O₂ (V̇e/VO₂) without an increase in the ventilatory equivalent for CO₂ (V̇e/V̇CO₂), when plotted against exercise time [20]. The GET was determined by inspection in a blinded manner by two investigators. A third investigator was consulted to adjudicate between the two when the two investigators did not agree on threshold placement.

2.2. Oxygen Kinetics during Moderate-Intensity Constant-Load Exercise. On the two following visits, each subject performed one arm-cranking and one leg-cycling 4-5-minute constant-load test, in a randomized order. Following 10 minutes at rest, the work rate, equal to that which elicited 50% of VO₂max during the incremental test, was applied instantaneously (from absolute rest) without prior warning given to the subject. During both tests the crank/cycle cadence was strictly kept between 90 and 100 SPM and RPM, respectively. Gas exchange was measured breath-by-breath using the same apparatus as for the incremental tests. Heart rate was monitored and recorded continuously during all constant-load exercise tests by means of a telemetric system (Polar, Electro, Finland).

2.3. Calculation of Oxygen Uptake Kinetics. Individual responses during the rest-to-exercise transitions were linearly interpolated to give 1-s values. For each subject and each exercise protocol, data were time-aligned to the start of exercise, superimposed, and averaged to reduce the breath-to-breath noise and enhance the underlying physiological response characteristics. The baseline VO₂ (VO₂_b0) was defined as the average VO₂ measured during the last two minutes before the start of exercise (rest period). The VO₂ mean response time (MRT) was fitted by combining the first and the second exponential terms. The MRT was then used to indicate the overall rate of change of the VO₂ toward its new steady state. The MRT for a single-term exponential model is equivalent to τ + T_D and therefore provides response information including not only the time constant (τ) but also the time delay (T_D). At the MRT, this response has attained 63% of its final value. To estimate the phase II time constant for the VO₂ kinetics (τ₂) we used a nonlinear least-squares monoexponential fit to the data as previously described [17, 21]. However, in order to maximize the amount of transient data available for the characterization (an important determinant of the goodness of fit [22]) we chose to discard the first 15 sec rather than the more common 20 sec—reasoning that the more rapid cardiac output kinetics in our fit subjects would reduce the limb-to-lung transit time and hence the duration of Phase I.

VO₂ kinetics tends to be slower at higher work rates even when the work rates are not associated with a sustained increase in blood lactate [23]. Therefore, in order to facilitate comparison across subjects exercising at different absolute work rates, the relative gain of the response (G = A/work rate) and the exercise specific relative oxygen deficit were computed using the following equation:

\[ \frac{O_2}{W} = G(\frac{VO_2}{W}) \times MRT \text{ (sec)} = mL/min/W. \]  

(1)

2.4. Data Analysis. Group data are reported as means and standard deviation. A two-way ANOVA with repeated measures for training status (trained or nontrained) and for muscle group (upper or lower extremity) (independent variables) was used to determine differences and relationships in and among the various dependent (O₂ kinetics parameters) and independent parameters between arm cranking and leg cycling and between the trained and nontrained muscles. Tukey’s post hoc test was utilized to determine where significant differences existed. Statistical significance was accepted at P < 0.05.
Table 2: Peak and sub-maximal responses to arm and leg exercise by each group (mean ± SD).

|                     | Kayakers | Arm | Cyclists | Arm |
|---------------------|----------|-----|----------|-----|
| Peak work rate (W)  | 298 ± 42cd | 279 ± 20cd | 390 ± 52bd | 157 ± 17abc |
| Peak VO2 (mL/min)   | 4268± 656cd | 4087 ± 499cd | 4921 ± 380bd | 3147 ± 436d abc |
| Peak HR (b/min)     | 183 ± 3  | 183 ± 8  | 192 ± 11d | 178 ± 11c |
| VO2ss (mL/min)      | 2015 ± 368cd | 1943 ± 404cd | 2952 ± 195abd | 1501 ± 3844bc |
| Work ratea (W)      | 108 ± 31cd | 106 ± 15d | 170 ± 16bd | 69 ± 12abc |
| Work ratea (% VO2peak)** | 39 ± 3 | 40 ± 4 | 44 ± 4 | 43 ± 3 |
| VO2ss (% VO2peak)** | 47 ± 6c | 47 ± 6c | 58 ± 4abd | 50 ± 8c |
| VO2 at GET (L/min)  | 2.84 ± 0.39b | 3.31 ± 0.25cd | 3.86 ± 0.33babc | 2.50 ± 0.33babc |
| VO2ss/VO2 at GET (%)| 72.3 ± 21bd | 62.5 ± 15abc | 74.1 ± 11bd | 61.6 ± 17abc |
| HRss (% VO2peak)**  | 66 ± 7  | 63 ± 6  | 67 ± 5  | 62 ± 6  |

Like letters denote significant difference (P < 0.05).
**Values are percentages of their respective peak values.

3. Results

3.1. Peak and Related Values. Table 2 presents data obtained during and at the end of all incremental and submaximal constant-load tests performed with the trained and untrained-muscle groups.

3.1.1. Cyclists. As expected for cyclists, all peak mechanical and cardiovascular-related responses (central) were significantly higher in the trained (legs) compared with the nontrained (arms) muscles. The exception was the peak respiratory exchange ratio values (RERS), which were similar in the two muscle groups (1.16 ± 0.05 versus 1.16 ± 0.06), and of a magnitude, which was consistent with maximal effort in both. The arms-to-legs ratio of VO2peak in this group was 64% (Table 2).

3.1.2. Kayakers. For these athletes the results were considerably different, demonstrating similar peak responses (mechanical and physiological) in both the trained and the nontrained muscles (Table 2). The arms to legs ratio of VO2peak in this group was 96%.

Also presented in Table 2 are values representing relative physiologic stress and strain during the constant submaximal load exercise challenges. Work rates, VO2, and HR achieved during the steady-state phase of the respective submaximal constant-load exercise, in percent of their respective peak values, as well as the ratio between the measured VO2 during the constant-load exercise challenges, and the respective VO2 of the muscle group-specific GETs, were not significantly different within and between groups (Table 2).

3.2. VO2 Kinetics. Figures 1–4 compare the groups’ mean Phase II of the VO2 response kinetics (excluding Phase I in each response), during the transition from rest to constant moderate exercise level between and within groups and muscles, along with the best exponential fit to each mean response. Visual inspection of these plots reveals that phase II of the VO2 response rose in biphasic fashion toward phase III (the exercise steady state levels). It seems that the relative load selected for this study (50–60% mode-specific VO2peak) was not only physiologically similar (in relative terms) (see Table 2), but also sufficiently low for both the lower and upper body musculatures for an attainment of a steady-state VO2 (after 2-3 min) without a development of VO2slow component, in the trained and untrained muscles alike.

A more quantitative assessment of the relative speed of VO2 response as a function of muscle group and training status is presented in Table 3 (means ± SD).

3.2.1. Within-Group Comparisons

Trained versus Untrained Muscles

(1) Cyclists. Onset of lower and upper extremity exercise at 50 to 60% of the mode-specific VO2max was associated with significantly higher amplitude-related values (VO2ss and A), and with faster overall (MRT) and Phase II response time (t2) in the trained muscles (legs) compared with the nontrained muscles (arms) (Figure 1 and Table 3).

It should be pointed out that when normalized for work rate attained by each muscle group (170 versus 79 W for the legs and arms, resp.), the “relative” rise (amplitude) in VO2 per unit load (G) was similar in the two muscle groups. In contrast, relative oxygen deficit (O2D/W) remained significantly larger in the untrained (arm) muscles compared with the trained (leg) muscles (594.5 versus 370.3 mL/min/W) (Table 3).

(2) Kayakers. Although arm training (kayakers) did not bring about superiority in any of the upper body musculature VO2 amplitude- or response-related parameters at the onset of a below threshold square-wave exercise, such training promoted the VO2 response-related parameters to a level approaching that of their legs (Table 3).
Table 3: Parameters of oxygen uptake response during moderate exercise as a function of exercise modality (muscle group involved) (mean ± SD).

| Variable                | Legs<sup>a</sup> | Arms<sup>b</sup> | Legs<sup>c</sup> | Cyclists |
|-------------------------|------------------|------------------|------------------|-----------|
| VO<sub>2bs</sub> (L/min) | 0.46 ± 0.04      | 0.46 ± 0.09      | 0.41 ± 0.07      | 0.40 ± 0.10 |
| VO<sub>2ss</sub> (L/min) | 1.99 ± 0.34<sup>cd</sup> | 1.94 ± 0.40<sup>cd</sup> | 2.85 ± 0.33<sup>bd</sup> | 1.50 ± 0.38<sup>ac</sup> |
| A (L/min)               | 1.54 ± 0.34<sup>cd</sup> | 1.45 ± 0.36<sup>cd</sup> | 2.45 ± 0.38<sup>bd</sup> | 1.03 ± 0.26<sup>abc</sup> |
| G (mL O<sub>2</sub>/min/W) | 14.35 ± 1.11    | 13.6 ± 2.21      | 14.38 ± 0.84     | 14.5 ± 3.83 |
| τ<sub>2</sub> (sec)     | 16.50 ± 2.6<sup>bd</sup> | 18.81 ± 3.7<sup>ac</sup> | 14.90 ± 2.7<sup>bd</sup> | 19.70 ± 3.6<sup>ac</sup> |
| MRT (sec)               | 22.62 ± 3.1<sup>cd</sup> | 26.73 ± 8.9<sup>ad</sup> | 25.76 ± 4.9<sup>bd</sup> | 40.90 ± 5.6<sup>abc</sup> |
| O<sub>2</sub>D (mL)     | 34.6 ± 11.9<sup>c</sup> | 39.4 ± 11.5<sup>c</sup> | 61.2 ± 17.4<sup>abd</sup> | 40.7 ± 15.3<sup>c</sup> |
| Relative O<sub>2</sub>D (mL O<sub>2</sub>/min/W) | 323.2 ± 97<sup>d</sup> | 363.2 ± 92<sup>d</sup> | 370.3 ± 90<sup>d</sup> | 594.5 ± 155<sup>abc</sup> |

Like letters denote significant difference (P < 0.05).

VO<sub>2bs</sub>: average value over the two min of resting baseline; VO<sub>2ss</sub>: rate of oxygen uptake at steady state level; A: the asymptotic amplitude for the exponential term; τ<sub>2</sub>: time constant of primary phase; MRT: mean VO<sub>2</sub> response time; O<sub>2</sub>D: calculated oxygen deficit; G: relative (to work rate) gain of the VO<sub>2</sub> response; Relative O<sub>2</sub>D: O<sub>2</sub> deficit normalized to work rate.

3.2.2. Between-Groups Comparisons

**Trained Muscles (Leg (Cyclists) versus Arm (Kayakers) Muscles).** As expected, and partially due to the differences in muscle mass and consequently in work rate, VO<sub>2ss</sub> and A, were significantly higher in the trained lower limbs than in the trained upper limbs (Figure 3 and Table 3). Similarly, the Phase II time constant (τ<sub>2</sub>), was faster in the trained lower than the trained upper limbs. Nevertheless, the overall VO<sub>2</sub> transient during the square wave exercise (MRT) and the relative O<sub>2</sub>D did not differ significantly between the large and the relatively small trained-muscle groups (Table 3).

**Nontrained Muscles (Leg (Kayakers) versus Arm (Cyclists) Muscles).** Except for VO<sub>2bs</sub> and G, all other VO<sub>2</sub> response kinetic parameters (A, MRT, and τ<sub>2</sub>) were higher (or faster) in the untrained lower limbs compared with the untrained upper limbs (Table 3). Similarly, relative O<sub>2</sub>D showed significantly smaller volume when exercising with the legs compared with arm exercise (323.2 versus 594.5 mL O<sub>2</sub>/min/W, resp.).

**Trained versus Nontrained Muscles**

(1) **Lower Limbs.** Whereas load- or muscle mass-associated variables (VO<sub>2ss</sub> and A) were significantly higher in the trained legs compared with the nontrained legs, variables related to the rate of VO<sub>2</sub> response during moderate constant load exercise (τ<sub>2</sub> and MRT) showed no significant differences between the trained and the untrained legs (Table 3). Similar trend was also evident in the load-normalized rise in oxygen uptake (G) and oxygen deficit (O<sub>2</sub>D) being statistically similar in the trained and the nontrained leg muscles (Table 3).

(2) **Upper Body.** Except for the τ<sub>2</sub> (statistically similar in the trained and untrained upper body muscles), and unlike in the lower limbs, the trained arms demonstrated significantly higher (A, VO<sub>2ss</sub>) and faster (MRT) O<sub>2</sub> kinetics-related values than the untrained arms (Figure 4 and Table 3). Relative O<sub>2</sub>D showed significantly smaller volume in the trained muscles.
Hence, despite differences in the absolute VO₂, the relative intensity of each square-wave transition was successfully matched across exercise modes: the %ΔVO₂, the % of the mode-specific peak power output, and the % of mode-specific of maximal heart rate were not significantly different (Table 2). Therefore, our study allows a comparison of the fundamental components of the VO₂ kinetics between cycling and arm cranking within the same intensity domain.

Unsurprisingly, the VO₂ response to moderate exercise in the cyclists revealed different patterns for the trained (legs) and the untrained (arms) muscles (Figure 2; Table 3). The higher absolute work rate in the trained-muscle group naturally resulted in a higher steady-state amplitude of the VO₂ response than for the untrained-muscle group. However, the phase II time constant (a functional correlate of the muscle VO₂ time constant) [2, 9, 24] as well as the VO₂ mean response time (MRS) reflecting, in addition, the utilization of oxygen from the oxygen stores [26] was significantly faster in the trained than the untrained muscles. Consequently, the oxygen deficit per unit power output (O₂D/D) was significantly larger in the tests with the untrained muscles than with the trained muscles (Table 3).

While the VO₂ kinetics for the cyclists’ arms (r₂ = 19.7 sec; MRT = 40.9 sec) were slower than those of their leg (r₂ = 14.9 sec; MRT = 25.8 sec), they were appreciably faster than those previously reported for arm-cranking exercise in normal untrained males (60–80 s) [8, 18]. In fact, the VO₂ time constant for the cyclists’ arms was similar to, and sometimes even faster than, those previously reported for normal nontrained legs (30–40 s) [8, 27]. This suggests that the muscles used by the cyclists for the arm-cranking exercise ought not be considered “untrained,” that is, reflecting the additional compensatory component arising from dynamic stabilization of the body during cycling and even periods of active “pulling” on the handlebars. Support for the above contention comes from studies by Baker et al. [28, 29] who reported that power generated during sprint cycling was significantly higher in a protocol that allowed for the gripping of the handle bars than in another protocol without the gripping of handle bars. Their results demonstrated that the arms and the upper body were involved in stabilizing the entire body so that the lower limbs could exert forces downwards onto the cycle pedals to generate the mechanical power and that the contribution of muscle groups not directly involved during sprint cycling toward power generation cannot be discounted.

Alternatively, of course, a “transfer effect” on the VO₂ kinetics may have been contributory, resulting from an increased amount of blood and, therefore, oxygen, available to the cyclists’ arms as a result of a “central” training effect consequent to the leg training. However, for this to be contributory, the VO₂ kinetics would need to be limited by oxygen delivery at this work intensity. However, there is no convincing evidence that increases in maximum VO₂ and, hence, maximum cardiac output induced by training alter the steady-state cardiac output response to a moderate-intensity work rate, at least for leg exercise (i.e., [26]). Furthermore, the VO₂ time constant for moderate exercise has been demonstrated not to be speeded by: experimentally-induced
increases in muscle blood flow [4], beginning the exercise when blood flow has remained high following a prior bout of higher intensity exercise [30], and even by increased inspired O₂ fractions [31]. Hence, any improvement in central indices of cardiovascular function is unlikely to be contributory.

The VO₂ response to moderate exercise in the kayakers, in contrast, revealed similar patterns (amplitude, kinetics, and O₂D) for the arm and the leg exercise, both in absolute and relative terms (Figure 2; Table 3). These results suggest either that the long-term intensive training with the relative small muscle mass of the arms did not cause any appreciable cross-training effect on the VO₂ kinetics and/or that there is a significant leg contribution to kayaking [32]. The fact that the values for the VO₂ time constant (16.5 sec) and MRT (22.6 sec) for leg exercise in the kayakers are appreciably faster than for normal untrained cycle ergometry [2, 8] suggests that the latter explanation is more likely.

Our finding that the VO₂ kinetics for the kayakers’ arms are not significantly different from those of the cyclists’ arms, despite the “central” capacity (as reflected by the leg VO₂peak) being appreciably lower in the kayakers, suggests that the VO₂ kinetics at the onset of subthreshold square-wave exercise depend primarily on peripheral factors (muscle mass, distribution of muscle fiber type, number of mitochondria, activity levels of oxidative enzymes, and possibly muscle vascularization) and not on central factors (cardiac output, pulmonary ventilation, etc.). These results are in line with previous reports suggesting that on-transient VO₂ kinetics for moderate square-wave exercise is mainly reflective of and dictated by peripheral (local) rather than central attributes [4, 5, 33].

While the phase II time constant (reflective of the kinetics of muscle oxygen utilization, that is, [1, 24]) for the exercise involving the trained-muscle groups were both fast relative to normal subjects, the value in the cyclists (14.9 sec) was even (and significantly) faster than that for the kayakers (18.8 sec). However, the mean ratio of leg to arm r₂ in our highly trained subjects was 81 ± 5%, being appreciably higher than the respective ratio of 50–60% observed in healthy untrained subjects [8, 17, 27]. That is, while both muscle groups were evidently “highly trained,” the cyclists were trained for longer-duration exercise (i.e., hours); the kayaker’s training program, preparing for all-out races lasting 2–7 minutes, included both aerobic- and anaerobic-type activities (including resistance training) [32]. Furthermore, the energy demand for the kayakers, during both competitions and training, was frequently in excess of VO₂peak, which was not the case for the cyclists. These factors may have contributed to even greater improvements in aerobic enzymatic function and capillarity—thought to be important contributors to VO₂ kinetics [5, 24].

With respect to the relatively untrained-muscle groups, the kayakers evidenced faster VO₂ kinetics both with respect to r₂ and MRT, than the cyclists (Table 3). This suggests that, while both muscle groups may be considered to be relatively trained [29, 32] with respect to normal moderately fit subjects, it seems that there is a greater stabilizing-counterforce involvement of the legs in the task of kayaking [32] than of the arms for cycling.

Another interesting and, possibly, surprising finding of the present study was the similarity of the VO₂ response kinetics for the trained (cyclists) and untrained (kayakers) leg muscles. This finding is not only surprising, but also contrary to several previous reports demonstrating a significant speeding of these kinetics following endurance training [13, 34]. One possible explanation for this unexpected outcome is that the speeding of the VO₂ kinetics with endurance training does not increase pari passu with the increase of VO₂peak, but rather effectively attains a plateau. We speculate that this “asymptotic” level of the phase II time constant is relatively “easy” to reach since even the relatively mild “whole body” training (including leg muscles) of our kayakers group appeared sufficient for their leg VO₂ kinetics to attain this assumed critical limiting level. This suggestion is further supported by the similar MRT in the trained and untrained leg muscles, despite significant differences in the respective VO₂peak values (73.2 versus 53.4 mL/kg/min). Furthermore, when comparing the ratio of r₂ to the leg muscles to that of the arm muscles (r₂ legs/r₂ arms) between the two elite athlete groups, it becomes evident that the kayakers arm muscles’ “machinery” (as determined by the r₂) is significantly closer to that of their leg muscles (88.3 versus 73.4% in the kayakers and cyclists, resp.). It is clear that such proximity is not due to relatively slow legs’ r₂ in the kayakers, as the latter is as fast in the kayakers as it is in the cyclists (see Table 3). Such high ratio implies that (a) arm training, as used by our kayakers, provides stronger stimulus for improving the VO₂ response kinetics during constant and moderate exercise task, and (b) for achieving high level in kayak competition, one needs to promote his arm muscle functioning to a level very close to that of his legs. It should be pointed out that the difference in the MRT legs-to-arms ratio between the two groups was even greater (86.7 versus 63.2% for the kayakers and cyclists, resp.).

**Limitations of Study Design.** Probably, the most powerful way to test the hypothesis of this study would be to conduct a “classic” training study (pre- versus posttraining comparisons). However, legalistically and logistically, such “classic” approach will not allow a long and intensive training regimen, such as that pursued by our subjects. Further, the design of this study does not allow to exclude the possibility of genetic predisposition influence on the observed results and hence on the final conclusions of the study.

Also, in the present study we used differing testing modes of arm exercise between groups. The logic for using different exercise modalities to test and compare arm exercise between cyclists and kayakers was intended to allow subjects in each group to perform exercise (in their respective trained muscles) similar to those they were most accustomed to and trained for.

Notwithstanding the above-mentioned limitations, the results of the present study provide no support for the “transfer” of a training effect onto the VO₂ on-transient response for moderate exercise, but rather support earlier reports demonstrating that peripheral (local) and not central (hemodynamic) effects may be important in dictating these kinetics. As a consequence, we suggest that predominantly
local and/or specific training is required to speed the muscle O_{2} consumption response to moderate exercise. This consequently reduces the associated oxygen deficit and hence the reliance on stored energy resources, predominantly phosphocreatine and O_{2}, and anaerobic lactate production.

Finally and in line with the above-mentioned limitations, further effort should be attempted to validate the study’s findings.

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B. Whipp, who passed away in 2011, was a pioneer and a highly prolific and respected researcher at the forefront of physiology in general and exercise physiology in particular. His work benefited countless scientists, physicians, coaches, and students. On top of all that he was a gifted educator, a unique lecturer, and above all, a warm, humble, and sincere human being. The authors are highly fortunate to have worked under his supervision and as his colleagues throughout various stages of his distinguished career and are indebted to the invaluable guidance, mentorship, and inspiration he bestowed upon them. Whipp was profoundly involved in writing this paper but did not live to see it published. His judgment and perspectives of future works will be greatly missed by many. The authors wish to dedicate this study to his legacy and cherished memory. This study was greatly missed by many. The authors wish to dedicate this study to his legacy and cherished memory. This study was financed, in part, by the Italian National Olympic Committee (CONI). Many grateful thanks to the athletes for their participation in the study.

References

[1] B. J. Whipp and S. A. Ward, "Physiological determinants of pulmonary gas exchange kinetics during exercise," Medicine and Science in Sports and Exercise, vol. 22, no. 1, pp. 62–71, 1990.
[2] B. Grassi, D. C. Poole, R. S. Richardson, D. R. Knight, B. K. Erickson, and P. D. Wagner, "Muscle O_{2} uptake kinetics in humans: implications for metabolic control," Journal of Applied Physiology, vol. 80, no. 3, pp. 988–998, 1996.
[3] R. L. Hughson, J. K. Shoemaker, M. E. Tschakovsky, and J. M. Kowalchuk, "Dependence of muscle V\textsubscript{O}2 on blood flow dynamics at onset of forearm exercise," Journal of Applied Physiology, vol. 81, no. 4, pp. 1619–1626, 1996.
[4] B. Grassi, L. B. Gladden, M. Samaja, C. M. Stary, and M. C. Hogan, "Faster adjustment of O\textsubscript{2} delivery does not affect V\textsubscript{O}2 on-kinetics in isolated in situ canine muscle," Journal of Applied Physiology, vol. 85, no. 4, pp. 1394–1403, 1998.
[5] C. A. Kindig, P. McDonough, H. H. Erickson, and D. C. Poole, "Nitric oxide synthase inhibition speeds oxygen uptake kinetics in horses during moderate domain running," Respiratory Physiology and Neurobiology, vol. 132, no. 2, pp. 169–178, 2002.
[6] B. Saltin, K. Nazar, and D. L. Costill, "The nature of the training response; peripheral and central adaptations to one legged exercise," Acta Physiologica Scandinavica, vol. 96, no. 3, pp. 289–305, 1976.
[7] J. P. Clausen, J. Trap-Jensen, and N. A. Lassen, "The effects of training on the heart rate during arm and leg exercise," Scandinavian Journal of Clinical and Laboratory Investigation, vol. 26, no. 3, pp. 295–301, 1970.
[8] P. Cerretelli, D. Shindell, and D. P. Pendergast, "Oxygen uptake transients at the onset and offset of arm and leg work," Respiration Physiology, vol. 30, no. 1-2, pp. 81–97, 1977.
[9] H. B. Rossiter, S. A. Ward, V. L. Doyle, F. A. Howe, J. R. Griffiths, and B. J. Whipp, "Inferences from pulmonary O_{2} uptake with respect to intramuscular [phosphocreatine] kinetics during moderate exercise in humans," Journal of Physiology, vol. 518, no. 3, pp. 921–932, 1999.
[10] J. M. Hagberg, R. C. Hickson, A. A. Ehsani, and J. O. Holloszy, "Faster adjustment to and recovery from submaximal exercise in the trained state," Journal of Applied Physiology Respiratory Environmental and Exercise Physiology, vol. 48, no. 2, pp. 218–224, 1980.
[11] S. M. Phillips, H. J. Green, M. J. MacDonald, and R. L. Hughson, "Progressive effect of endurance training on V\textsubscript{O}2 kinetics at the onset of submaximal exercise," Journal of Applied Physiology, vol. 79, no. 6, pp. 1914–1920, 1995.
[12] R. T. Casaburi and B. Soll, "Dynamics of ventilation and gas exchange in athletes trained for upper body exercise," Sports Medicine, Training and Rehabilitation, vol. 3, pp. 251–260, 1992.
[13] A. M. Edwards, N. V. Challis, J. H. Chapman, D. B. Claxton, and M. L. Fysh, "V\textsubscript{O}2 kinetics determined by PRBS techniques differentiate elite endurance runners from elite sprinters," International Journal of Sports Medicine, vol. 20, no. 1, pp. 1–6, 1999.
[14] D. R. Pendergast, "Cardiovascular, respiratory, and metabolic responses to upper body exercise," Medicine and Science in Sports and Exercise, vol. 21, no. 5, pp. S121–S125, 1989.
[15] M. N. Sawka, "Physiology of upper body exercise," Exercise and Sport Sciences Reviews, vol. 14, supplement, pp. 175–211, 1986.
[16] C. T. M. Davies, J. Few, K. G. Foster, and A. J. Sargeant, "Plasma catecholamine concentration during dynamic exercise involving different muscle groups," European Journal of Applied Physiology and Occupational Physiology, vol. 32, no. 3, pp. 195–206, 1974.
[17] K. Koppo, J. Bouckaert, and A. M. Jones, "Oxygen uptake kinetics during high-intensity arm and leg exercise," Respiratory Physiology and Neurobiology, vol. 133, no. 3, pp. 241–250, 2002.
[18] S. Koga, T. Shiojiri, M. Shibasaki, Y. Fukuba, Y. Fukuoka, and N. Kondo, "Kinetics of oxygen uptake and cardiac output at onset of arm exercise," Respiration Physiology, vol. 103, no. 2, pp. 195–202, 1996.
[19] M. A. Johnson, J. Polgar, D. Weightman, and D. Appleton, "Data on the distribution of fibre types in thirty-six human muscles. An autopsy study," Journal of the Neurological Sciences, vol. 18, no. 1, pp. 111–129, 1973.
[20] K. Wasserman, B. J. Whipp, S. N. Koyal, and W. L. Beaver, "Anaerobic threshold and respiratory gas exchange during exercise," Journal of Applied Physiology, vol. 35, no. 2, pp. 236–243, 1973.
[21] B. J. Whipp, S. A. Ward, and N. Lamarra, "Parameters of ventilatory and gas exchange dynamics during exercise," Journal of Applied Physiology Respiratory Environmental and Exercise Physiology, vol. 52, no. 6, pp. 1506–1513, 1982.
[22] N. Lamarra, B. J. Whipp, S. A. Ward, and K. Wasserman, "Effect of interbreath fluctuations on characterizing exercise gas exchange kinetics," Journal of Applied Physiology, vol. 62, no. 5, pp. 2003–2012, 1987.
[23] F. Özyener, H. B. Rossiter, S. A. Ward, and B. J. Whipp, "Influence of exercise intensity on the on- and off-transient kinetics of pulmonary oxygen uptake in humans," Journal of Physiology, vol. 533, no. 3, pp. 891–902, 2001.
[24] M. Burnley, J. H. Doust, D. Ball, and A. M. Jones, “Effects of prior heavy exercise on VO₂ kinetics during heavy exercise are related to changes in muscle activity,” Journal of Applied Physiology, vol. 93, no. 1, pp. 167–174, 2002.

[25] M. C. Hogan, “Fall in intracellular Po₂ at the onset of contractions in Xenopus single skeletal muscle fibers,” Journal of Applied Physiology, vol. 90, no. 5, pp. 1871–1876, 2001.

[26] P. Cerretelli and P. E. di Prampero, “Gas exchange in exercise,” in Handbook of Physiology, Section 3. The Respiratory System, A. P. Fishman, L. G. Sarhi, J. M. Tenney, and S. R. Geiger, Eds., pp. 297–339, American Physiology Society, Bethesda, Md, USA, 1987.

[27] P. D. Chilibeck, D. H. Paterson, W. D. F. Smith, and D. A. Cunningham, “Cardiorespiratory kinetics during exercises of different muscle groups and mass in old and young,” Journal of Applied Physiology, vol. 81, no. 3, pp. 1388–1394, 1996.

[28] J. Baker, J. Gal, B. Davies, D. Bailey, and R. Morgan, “Power output of legs during high intensity cycle ergometry: influence of hand grip,” Journal of Science and Medicine in Sport, vol. 4, no. 1, pp. 10–18, 2001.

[29] J. Baker, E. Brown, G. Hill, G. Phillips, R. Williams, and B. Davies, “Handgrip contribution to lactate production and leg power during high-intensity exercise,” Medicine and Science in Sports and Exercise, vol. 34, no. 6, pp. 1037–1040, 2002.

[30] A. Gerbino, S. A. Ward, and B. J. Whipp, “Effects of prior exercise on pulmonary gas-exchange kinetics during high-intensity exercise in humans,” Journal of Applied Physiology, vol. 80, no. 1, pp. 99–107, 1996.

[31] M. J. MacDonald, M. A. Tarnopolsky, and R. L. Hughson, “Effect of hyperoxia and hypoxia on leg blood flow and pulmonary and leg oxygen uptake at the onset of kicking exercise,” Canadian Journal of Physiology and Pharmacology, vol. 78, no. 1, pp. 67–74, 2000.

[32] V. B. Issurin, Biomechanics of Canoeing and Kayaking, FIS Publication, Moscow, Russia, 1986.

[33] T. J. Barstow, A. M. Jones, P. H. Nguyen, and R. Casaburi, “Influence of muscle fiber type and pedal frequency on oxygen uptake kinetics of heavy exercise,” Journal of Applied Physiology, vol. 81, no. 4, pp. 1642–1650, 1996.

[34] J. M. Hagberg, S. D. McCole, M. D. Brown et al., “ACE insertion/deletion polymorphism and submaximal exercise hemodynamics in postmenopausal women,” Journal of Applied Physiology, vol. 92, no. 3, pp. 1083–1088, 2002.
