Is the $\dot{V}O_2$ slow component dependent on progressive recruitment of fast-twitch fibers in trained runners?

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Borrani, F., R. Candau, G. Y. Millet, S. Perrey, J. Fuchslocher, and J. D. Rouillon. Is the $\dot{V}O_2$ slow component dependent on progressive recruitment of fast-twitch fibers in trained runners?. J Appl Physiol 90: 2212–2220, 2001.—The goal of this study was to use spectral analysis of EMG data to test the hypothesis that the $O_2$ uptake ($\dot{V}O_2$) slow component is due to a recruitment of fast fibers. Thirteen runners carried out a treadmill test with a constant speed, corresponding to 95% of the velocity associated with maximal $\dot{V}O_2$. The $\dot{V}O_2$ response was fit with the classical model including three exponential functions. Electrical activity of six lower limb muscles (vastus lateralis, soleus, and gastrocnemius of both sides) was measured using electromyogram surface electrodes. Mean power frequency (MPF) was used to study the kinetics of the electromyogram discharge frequency. Three main results were observed: 1) a common pattern of the MPF kinetics in the six muscles studied was noted; 2) MPF decreased in the first part of the exercise, followed by an increase for all the muscles studied, but only the vastus lateralis, and gastrocnemius muscles of both sides increased significantly ($P < 0.05$); and 3) the beginning of the MPF increase of the four muscles mentioned above corresponded with the beginning of the slow component. Our results suggest a progression in the average frequency of the motor unit discharge toward the high frequencies, which coheres with the hypothesis of the progressive recruitment of fast-twitch fibers during the $\dot{V}O_2$ slow component. However, this interpretation must be taken with caution because MPF is the result of a balance between several phenomena.

$\dot{V}O_2$ kinetics; mean power frequency; electromyogram; power spectrum; $O_2$ uptake

AT THE ONSET OF EXERCISE with constant power output performed below the work rate that elicits the onset of blood lactate accumulation (OBLA), $O_2$ uptake ($\dot{V}O_2$) increases exponentially, after the initial cardiodynamic response, toward a steady state (62). Healthy subjects reach the stable state after ~2 to 3 min. During high-intensity, constant-load cycling, the initial rapid rise in $\dot{V}O_2$ is followed by a slower increase in $\dot{V}O_2$ exceeding the value extrapolated from the $\dot{V}O_2$ work rate relationship established at sub-OBLA work rates. The slower $\dot{V}O_2$ rise continues for several minutes and may reach maximal $\dot{V}O_2$ ($\dot{V}O_{2\text{max}}$) when exhaustion occurs (59, 62). The increase in $\dot{V}O_2$ during high-intensity exercise at a constant load is known as the slow component of $\dot{V}O_2$ (19, 49, 51, 62).

The mechanisms underlying the $\dot{V}O_2$ slow component are not yet completely understood. Among the physiological factors suspected to cause the slow component, lactate accumulation initially attracted the most attention (5, 7, 47, 56). In fact, it has been shown that the amplitude of the slow component and the rise in blood lactate were strongly correlated during stationary cycling (51). However, it is now generally accepted that lactate is not a cause of the $\dot{V}O_2$ slow component but coincides with its appearance (47). Epinephrine was also proposed as an explanation of the slow component because its infusion increases basal metabolism (53), but because the administration of epinephrine has not shown an effect on $\dot{V}O_2$ kinetics during exercise, epinephrine cannot be regarded as a factor causing the $\dot{V}O_2$ slow component (25, 64). A strong correlation between plasma potassium and $\dot{V}O_2$ suggested that it may have an effect on the slow component (26, 66). However, the concentration of potassium increased until the third minute of exercise and then remained stable despite the appearance of the slow component. Thus it seems improbable that potassium plays a significant role in the slow component (48). Altered availability or use of energy substrates has been hypothesized to influence the $\dot{V}O_2$ slow component, but their influence is questionable or weak. Contradictory results of the studies on this topic do not allow a clear interpretation (26). On the basis of data showing that an increase in mitochondria temperature decreased the coupling of oxidative phosphorylation (P/O ratio), it has been suggested that the increase in body temper-
ature could explain $\sim 25\%$ of the slow component (29, 60, 63). However, this attractive hypothesis was unconfirmed by experiments. In fact, Koga et al. (34) did not find a significant rise in the slow component of VO$_2$ after having increased muscle temperature by the use of hot water-perfused pants. It has been suggested that the VO$_2$ that corresponds to the VO$_2$-increased work of the respiratory muscles due to increased breathing contributes to the VO$_2$ slow component (19, 64), and it has been found that it could explain up to $25\%$ of the slow component during exercise of 95% VO$_2$ max (16). However, a hypoxia (12% O$_2$) experiment reported contradictory results. Compared with normoxia, the subjects increased their ventilation as much as 40 l/min although the VO$_2$ slow component was not affected (22). The authors argued that the VO$_2$ of the contracting muscles (or other tissues such as renal or splanchnic) was reduced under the hypoxic condition, and other metabolic processes must have increased to compensate because whole body VO$_2$ was unchanged.

It is not surprising then that the origin of the slow component has been attributed to the muscles concerned with exercise (48). It has been suggested that the VO$_2$ slow component is due to the progressive recruitment of fast-twitch fibers to compensate for the deficiency of slow-twitch fibers (26, 47). In fact, efficiency of fast-twitch fibers is lower than the efficiency of slow-twitch fibers (36, 55, 63). This phenomenon was also observed in situ in humans. At a given VO$_2$, the subjects with a high percentage of slow-twitch fibers produced a higher mechanical power than their counterparts with a lower percentage of slow-twitch fibers (20). On the basis of amplitude changes in an integrated electromyogram (EMG) during the slow component of VO$_2$, it has been suggested that fast-twitch fibers are gradually recruited during exhausting exercises (52). It is well known that, for progressive exercises, small motor units (i.e., composed of slow-twitch fibers) are first recruited at a submaximal level of force. At high intensity, metabolic modifications, such as a reduction in muscular pH, inorganic phosphate, and potassium accumulation, are associated with an alteration in the excitation-contraction coupling (for a review, see Ref. 1). As a result, other fibers must be recruited to sustain the needed muscular work. As a result of the recruitment law, these progressively recruited fibers are likely to include fast-twitch fibers (31, 52). A previous investigation (6) has shown that the amplitude of the VO$_2$ slow component was correlated with the percentage of fast-twitch fiber of the vastus lateralis muscle. On the basis of the increase in integrated EMG observed during a constant-load exercise (52), Shinohara and Moritani argued that fast-twitch fibers are progressively recruited. Nevertheless, to the best of our knowledge, no direct relationship between the VO$_2$ slow component and the progressive recruitment of fast-twitch fibers has been shown. Consequently, the purpose of this study was to test, through spectral analysis of the EMG signal, the assumption that the slow component of VO$_2$ is partially induced by a progressive recruitment of fast-twitch fibers.

**MATERIAL AND METHODS**

**Subjects**

Thirteen regional-level competitive runners agreed to take part in the study. The local ethics committee approved the experiment, and the subjects gave their written consent.

**Experimental Design**

Two running tests separated by 72 h were completed. The first test consisted of progressive exercise on a treadmill (Adal race, Tecmachine, Andérézieux-Bouthéon, France). A warm-up at a speed of 12 km/h (3.3 m/s) for 5 min was first carried out. The test then started at 14 km/h (3.9 m/s), and the speed was increased by 1 km/h every 2 min according to the method described by Léger and Bouché (41).

The second test consisted of a run at constant speed corresponding to 95% of VO$_2$ max until exhaustion after a warm-up of 10 min at a velocity of 3.3 m/s. During the test, the breath-by-breath gases were measured on a computerized system (CPX, Medical Graphics, St. Paul, MN). This system uses an infrared sensor and a zirconium oxide electrode for measuring fractional concentration of CO$_2$ and O$_2$. A pneumotachograph was used to measure expired gas volume. Immediately before each exercise, known-composition gases and a 3-liter Rudolph syringe were used for calibration of the gas analyzers and the pneumotachograph.

EMG activity was obtained from the vastus lateralis, gastrocnemius lateralis, and soleus muscles of both lower limbs. The bipolar surface electrodes (Biochip, Grenoble, France) had a constant intrapair distance of 12 mm and included a differential amplifier (impedance = 2 GΩ, filter 6–600 Hz). The motor points were located with an electrostimulator (Compex, Ecballens, Switzerland). The surface of the skin was prepared by removing the hair and rubbing it with abrasive paper, then washing it with acetone. The electrodes were fixed longitudinally over the muscle belly. An electrolytic gel was applied between the skin and the surface of the electrodes to improve conductivity. The neutrals of the electrodes were placed on the front and median part of the tibia. An analog-digital board (12 bits, National Instrumentation, LPM16, Paris, France) was used to acquire the data, and a personal computer managed the system. Acquisition was continuous throughout the test at a sample rate of 1,000 Hz. The signal was temporarily cut out with respect to each stride. Because the treadmill was equipped with piezoelectric sensors able to measure the force exerted during foot contact in three dimensions, the definition of each stride was performed using the vertical force signal.

**Data Analysis**

VO$_2$ kinetics. Several models have been proposed to describe the VO$_2$ kinetics. For primary analysis, we used the exponential model (8)

$$
\dot{V}O_2(t) = \dot{V}O_2b + A_1(1 - e^{-[(t - t_{d1})/U_1]}) + A_2(1 - e^{-[(t - t_{d2})/U_2]})
$$

where $t_{d1}$ and $t_{d2}$ are the time constants, and $U_1$ and $U_2$ are the asymptotic amplitudes for the second and third exponential, respec-
tively; $\tau_1$ and $\tau_2$ are the time constants of each exponential; and $t_d1$ and $t_d2$ represent the time delays of each equation. Because the focus of our study was the slow component of VO$_2$ kinetics, and the primary component phase is not distorted by any early cardiodynamic influence (45, 61), the initial component was not modeled in this study. As a consequence, the first 20 s were removed from analysis to ensure that the early initial component did not influence the result (61).

The amplitude of slow component was assigned the value ($A_2$)

$$A_2 = A_2(1 - e^{-([t_e - t_d2]/\tau_2)})$$

where $t_e$ is the time at end of exercise.

As pointed out by Linnarsson (42), when the second exponential component has a time constant that is substantially longer than the duration of the data collection, it is indistinguishable from a linear “drift.” If this case appears, the linear model proposed by Paterson and Whipp and colleagues (45, 61) would be used.

The parameters of the model were determined with an iterative procedure by minimizing the sum of the mean squares of the differences between the estimated VO$_2$ based on the model and the measured VO$_2$. Values of the measured VO$_2$ that were greater than three standard deviations from the VO$_2$ of the model were considered outliers and were removed. These outlier values were assumed to be due to abnormal breaths during exercise, such as shallow breathing or breath holding. These values represented <1% of the total data collected. Iterations continued until successive repetitions reduced both the sum of residuals by $<10^{-8}$ and the correlation coefficient of the relationship between residuals and time by $<10^{-6}$. The bootstrap method was used to test the confidence interval of the model parameters. This method estimates the potential error in the determination of model parameters using repeated samples from the original data set.

EMG. The rough signal was divided into temporal segments corresponding to the strides. For each muscle, the segments were determined between two successive impacts of the opposing foot on the treadmill, detected with the vertical force signal of the treadmill. Each signal was filtered (band-pass filter 20–500 Hz, Butterworth 5th degree). To clear the truncation error, the signal was multiplied by the Hanning function (Hanning’s window). The spectrum was analyzed using the MPF. The MPF was calculated using a fast Fourier transformation of 1,024 points, applying the power spectral density to the results of the fast Fourier transformation, and then by calculating MPF from the power spectral density to the results of the fast Fourier transformation.

Table 1 presents individual results of the incremental tests. The mean value for VO$_{2\text{max}}$ was 64.6 ± 4.6 ml·min$^{-1}$·kg$^{-1}$, and the average speed corresponding to this VO$_{2\text{max}}$ was 5.36 ± 0.28 m/s, indicating that the subjects were well-trained runners.

Table 2 shows kinetic parameters for the exponential curve fitting of the individual VO$_2$ responses. The primary component had an amplitude ($A_1$) of 48.5 ± 6 ml·min$^{-1}$·kg$^{-1}$ (CV = 1.7%) with a time delay ($t_d1$) of 4.9 ± 5.6 s (CV = 105%) and a time constant ($\tau_1$) of 17.2 ± 5.8 s (CV = 20.4%). For all subjects, a slow component was observed. The average value ($A_2$) was 6.9 ± 2.2 ml·min$^{-1}$·kg$^{-1}$ (CV = 11.4%). The time delay for the slow component ($t_d2$) was 119 ± 25 s (CV = 10.7%), and the time constant ($\tau_2$) was 84 ± 46 s (CV = 15.6%). The time constant $\tau_2$ was substantially shorter by 10.220.32.247 on October 7, 2016 http://jap.physiology.org/ Downloaded from
(~2.7 time in the worst case) than the exercise duration, and the sum of residuals was lower than that obtained with the linear model. Thus the double mono-exponential model was used for each case (42).

The coefficient of determination ($R^2$) obtained between actual $\dot{V}O_2$ and modeled responses was 0.77 ± 0.19. Figure 1A shows an example of breath-by-breath $\dot{V}O_2$ associated with the $\dot{V}O_2$ model. Figure 1B represents the distribution of the residual errors. The sum of residuals ($sr = 10.22\pm 0.32$) and the coefficient of correlation ($r = 10.22$) clearly indicate that the breath-by-breath “noise” was independent of time, distributed randomly around zero, and similar for the whole group.

Typical EMG signals of the six muscles studied and the vertical force signal of the treadmill are shown in Fig. 2. Generally, the activation of the vastus lateralis and soleus muscle started before the contact of the foot on the treadmill showing a clear phase of preactivation (see right line in Fig. 2). The beginning of EMG activity for the gastrocnemius muscle and impact of the foot on the treadmill were simultaneous.

A common pattern of MPF changes over time was observed among the six muscles studied (Fig. 3). MPF decreased during the primary component. This drop was significant for the right and left gastrocnemius, the left soleus, and the right vastus lateralis muscles ($P < 0.05$, $P < 0.001$, $P < 0.001$, and $P < 0.05$, respectively). During the slow component period, MPF increased significantly ($P < 0.05$), except for the right and left soleus muscles ($P = 0.47$ and $P = 0.19$, respectively). The onset of the increase in MPF values for the vastus lateralis and the gastrocnemius lateralis muscles were found to be concurrent with the beginning of the slow component, whereas the minimal MPF values were delayed (~13%) for the soleus muscles. For all muscles, the difference in time delay between the onset of the slow component of $\dot{V}O_2$ and the lowest point of the MPF response failed to be significant.

No significant difference was found in the evolution of MPF when comparing the left side to the right. The relative amplitude of the MPF was significantly correlated to the relative amplitude of the slow component of $\dot{V}O_2$ for the left gastrocnemius lateralis muscle and the right soleus muscle ($R = 0.70$, $P < 0.05$ and $R = 0.75$, $P < 0.05$, respectively). For all other muscles studied, this relationship was never significant.

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<th>$A_2$ (ml·min$^{-1}$·kg$^{-1}$)</th>
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Mean ± SD 48.5 ± 6.0 4.9 ± 5.7 17.2 ± 5.8 6.8 ± 2.3 120.5 ± 26.4 84.3 ± 46.6 553 ± 88 0.87 ± 0.12

CV Mean 1.7% 105.2% 20.4% 11.4% 10.7% 15.6%

$A_1$ and $A_2$, amplitude terms for $\dot{V}O_2$; $\tau_1$ and $\tau_2$, time constants of each component; $t_d1$ and $t_d2$, time delays to onset of each component; $t_e$, time to exhaustion; $r$, coefficient of correlation of the model; CV, coefficient of variation estimated by the bootstrap method.

Fig. 1. Best fit of $\dot{O}_2$ uptake ($\dot{V}O_2$) in the square-wave exercise (A) and residual sum of squares (B). Note that the breath-by-breath “noise” of the individual exercise transition was independent of time, indicating that the residuals were distributed randomly around zero. This result suggests that no further improvement of the fit could be obtained without using a more complex model.
The two most important findings of this study are that both VO\textsubscript{2} and MPF of the extensor muscles increased from the end of the second minute to exhaustion during running at 95% of VO\textsubscript{2\text{max}} and that the start of these increases were concurrent.

**Limits of the Methods**

One could argue that the number of transitions between rest and exercise is not sufficient to assess accurately the parameters of the model. In the studies focusing on the primary phase of VO\textsubscript{2} kinetics, the transition was repeated several times (generally 2–4 times) to collect a sufficient number of points during this short phase to decrease the breath-by-breath noise using an averaging procedure. However, recent studies focusing on the slow component (9, 10, 39, 40, 44) used only a single transition, because enough measurements were obtained to fit a monoexponential function (>400 points during the slow component phase in our case). The common practice in modeling is to collect a number of measurement points greater than 10 times the number of degrees of freedom of the model plus 10 points. In our case, the minimal number of points is 70 (6 × 10 + 10). The number of breaths collected in the present study is clearly greater than the minimal number required. The estimated coefficient of variation, especially those of the two critical parameters (i.e., t\textsubscript{d2} and A\textsubscript{2}), were relatively small (~10%) suggesting an accurate determination of the critical parameters even if a single transition was performed. Moreover, it is not possible to exclude the fact that the breath-by-breath variability may have biological significance, although Lamarra et al. (38) suggested stochastic properties of the breath-by-breath noise. In the present study, the lack of relationship between the residuals and the time supports the view of these authors.

In the present study designed to test the fiber type hypothesis, no measurement from moderate exercise has been achieved. Thus our results could not demonstrate that the slow component of VO\textsubscript{2} is excessive with respect to the values predicted from moderate exercise. However, the prediction of energy demand corresponding to high-intensity exercise requires a number of assumptions, and there are inherent limitations. It is necessary to assume that it is possible to predict the energy demand of high-intensity exercise on the basis of linear extrapolation from submaximal work rates. In addition, it was necessary to assume that the energy demands of these high-intensity work rates remained constant throughout the period over which we evaluated the kinetics (32). The energy demand for exercise at work rates above the OBLA cannot be accurately predicted because it is essential to sum to total aerobic plus anaerobic energy contributions (4). Finally, a major obstacle in the extrapolation of energy requirement during heavy exercise is the unknown contribution of slow-twitch muscle fibers. As work rate increases, the fast-twitch fibers are gradually recruited (28). Taking into account these limitations in the assessment of energy demand for high-intensity exercise, it seems...
difficult to evaluate accurately the energy demand from moderate exercise. Nevertheless, the fact that the slow component corresponds to excessive \( V\dot{O}_2 \) seems widely accepted for cycling exercise (5, 8, 26, 29, 60, 61, 64). This could be more problematic with treadmill exercise for two reasons. First, the power output does not necessarily increase linearly with the speed of progression. Second, the quantification of the power output in running is complex and is still the object of controversies. Therefore it seems difficult to verify whether the slow component amplitude represents \( O_2 \) excess or not.

**Slow Component: Comparison With the Known Facts**

Two models are generally used in the literature to describe the phenomenon of the slow increase in \( V\dot{O}_2 \) as a function of time: the exponential model (8) and the linear model (2, 45). As pointed out by Linnarsson (42), if this second exponential component has a time constant (\( \tau_2 \)) that is substantially longer than the duration of the data collection (\( t_d \)), it is indistinguishable from a linear “drift,” and the linear model must be used. Because this was not the case in our study (see Table 2), application of a linear model would be inappropriate. Moreover, the random distribution of residuals about zero suggests that no further improvement of the fit can be obtained. As explained by Casaburi et al. (18), the advantage of this exponential equation is that if the underlying response is truly monoexponential, the amplitude of the \( V\dot{O}_2 \) slow component (\( A_2 \)) will converge to zero (7). Furthermore, if the second component is linear (rather than exponential) over the time observed, the amplitude (\( A_2 \)) and the time constant of the second component (\( \tau_2 \)) will converge to unphysiologically high values. However, the derivative of the second term (\( A_2/\tau_2 \)) would be identical to the best fitting slope of a linear function to the data (7, 18). It seems that the time constant of response (\( \tau_2 \)) tends to be longer with increasing work rate (18).

The relatively small coefficients of variation found for \( t_d \) (10.7% ± 4.9) lend support to the robustness of the estimated onset of the \( V\dot{O}_2 \) slow component. Moreover, the \( t_d \) observed in the present study is in agreement with the values reported previously (6, 7, 45).

Few data are available on the amplitude of the slow component for trained runners. For an intensity of exercise similar to the present study, Candau et al. (16) found an amplitude of 216 ml/min. A recent study (17) reported an amplitude of 301 ml/min during a running exercise at similar intensity as in the present study, whereas a higher amplitude (700 ml/min) was reported in experiment by Sloniger et al. (54) in which the intensity was 99% of \( V\dot{O}_{2\text{max}} \). However, Billat et al. (12) found no significant rise in \( V\dot{O}_2 \) (−0.9 ± 2.1 ml·min\(^{-1} \cdot \text{kg}^{-1} \)) between the third minute and the end of a test performed at 90% of \( V\dot{O}_{2\text{max}} \). In the study of Carter et al. (17), the amplitude of the slow component was slightly lower in running than in cycling exercise. Thus, to the best of our knowledge, no satisfactory explanation has been proposed in the literature which would support a lack of slow component in running exercise observed in the study of Billat et al. (12).

**Kinetics of \( V\dot{O}_2 \) and EMG Signal**

**Primary component of \( V\dot{O}_2 \)** As previously described in the literature, a decrease in MPF was noted during the primary phase of \( O_2 \) kinetics. Three main phenomena can explain this result, namely 1) muscle wisdom, 2) changes in muscle fiber conduction velocity, and 3) synchronization of the slow motor units.

Muscle wisdom refers to the decline in the rate at which motor unit action potentials are discharged. This decrease is not thought to be associated with a modification of the action potential generation and propagation but is more likely connected to an adaptation that matches the neural activity to the changing conditions in the muscle. For instance, it has been reported that a declining frequency of electrical stimulation induced a lower increase in strength over a 60-s interval when compared with a constant frequency of stimulation (14). Fatigue shifts leftward the stimulus frequency-force relationship showed (13). This change is usually interpreted as an increase in time course of the twitch, such that the activation rate decline induces a decrease in MPF without change in force. In fact, because the time of relaxation increases in fatiguing conditions, the discharge rate does not need to be as high as in normal conditions when it induces tetanus. It was hypothesized that peripheral feedback reflex from either type III/IV mechanoreceptors, muscle spindles, or type Ib tendon organs (24) could lead to changes in stimulus frequency such that the decrease in force with fatigue is minimized.

Nevertheless, other experiments on both motor unit (57) and whole muscle (13) have found that the stimulus frequency-force relationship does not always shift to the left with fatigue. Powers and Binder (50) have suggested that the type of motor unit can indirectly influence the kinetics of MPF. According to these authors, the frequency-force relationship of fast-twitch fatigue resistant motor units shifted to the left immediately after a stimulation period and to the right after about 30 min of recovery, whereas this relationship shifted only to the right in fast-twitch fatigable motor units. Because the measurement of electrical activity of the entire muscle is influenced by the weighted average of the motor unit response, Thomas et al. (57) suggested that the direction of shift depends on the balance between time course of fatigability and potentiation for each unit. Also, Garland et al. (27) suggest that muscle wisdom is not applicable for submaximal contractions because they find a decrease in discharge rate even in the absence of any slowing of muscle relaxation time. Thus, although muscle wisdom may be an appropriate expression for isometric contractions, it is more questionable for cyclic activity like running.

Changes in muscle fiber conduction velocity are a second factor that could potentially influence the shift of MPF toward low frequency with fatigue. The MPF is directly related to the muscle fiber conduction velocity,
and changes in muscle fiber conduction velocity produced by changes in electrolytes or metabolites have direct implications on the MPF value. Bouissou et al. (15) have found a correlation between muscle lactate concentration and MPF during supramaximal dynamic exercise, suggesting that metabolite accumulation can influence MPF. However, Mills and Edwards (43) have observed that patients with myophosphorylase deficiency also present a power spectral shift to the left with fatigue without any modification of muscle fiber conduction velocity and lactate or H⁺ accumulation. To explain this apparent contradiction, loss of K⁺ in the intracellular space has been suggested because ionic change affects membrane potential, which changes the muscle cell membrane excitability. It can be speculated that the resulting alteration of the action potential propagation has an effect on the decrease of MPF (33). This hypothesis is supported by the study of Poole et al. (48), who found that the potassium rise takes place mainly during the first 3 min of exercise.

The third factor that could explain the shift in MPF toward the left is the synchronization of the slow motor units. In fact, Lago and Jones (37), as well as Kranz et al. (35), have shown that a rise in synchronization can alter the EMG spectrum. Synchronization increases the relative power in low frequency, and this could decrease MPF.

Slow component of VO₂. In this study, the MPF of the muscles studied increased during the period corresponding to the slow component of VO₂, i.e., with fatigue. Although it is not statistically significant, it is of interest to note that the time delay for the onset of the MPF increase in the soleus appeared greater than for the other muscles (see Fig. 3). The soleus is known for having a high percentage of slow-twitch fibers. We speculate that this might be due to a higher possibility of turnover in slow-twitch motor units (24) for this muscle, because the pool of this type of fibers is higher.

Taking the factors discussed above into consideration, another physiological phenomenon is probably involved. We suggest that this phenomenon could be the progressive recruitment of fast-twitch fibers. It is interesting to note that the beginning of the slow component corresponded to the beginning of the increase in MPF for the vastus lateralis and gastrocnemius lateralis muscles. The two muscles with a major composition of slow-twitch fibers did not display a significant increase. Moreover, the significant correlation observed between the amplitude of the slow component and the rise in MPF for two of the six muscles studied reinforced the link between the two phenomena.

Progressive recruitment of new motor units agrees favorably with the study of Vøllestad et al. (58). A sequential depletion of glycogen in type IIA, IIB, and IIB fibers was observed during constant-load submaximal exercise (58). In a fatigued state, the slow-twitch motor units recruited at the beginning of the exercise seem to be unable to sustain work.

This hypothesis of sequential fiber recruitment is also indirectly corroborated by the study of Enoka et al. (23). Some low-threshold motor units active during a ramp-and-hold task in nonfatigued conditions were not active in fatigued conditions, although the task was identical to the one performed before the fatigue exercise (23). Fatigue-related studies show that accumulation of metabolites or ions (hydrogen ions, inorganic phosphate, magnesium, potassium) may impair 1) the Ca²⁺ release from the sarcoplasmic reticulum, 2) troponin sensitivity to Ca²⁺, and consequently 3) the contraction force of the cross-bridge attachments. Finally, the increase in activity of the motor units toward higher frequencies could be interpreted as a reflection of fast-twitch fiber recruitment. The strong relationship reported by Wretling et al. (65) between MPF and muscle fiber composition during dynamic exercise, the positive and significant relationship between the changes in integrated EMG and amplitude of the slow component previously reported by Shinohara and Moritani (52) and the pattern of the group response in the present study (Fig. 3) lend some support for the tested hypothesis.

Nevertheless, the increase in activity of the motor units toward the high frequencies is not necessarily due to the recruitment of fast-twitch fibers but may be also the consequence of 1) an increase in motor neuron discharge rate of the slow-twitch fibers and/or 2) a rise in temperature. The first suggestion, to the best of our knowledge, has not been described in the literature. Concerning the second proposition, there are some controversies. Holewijn and Heus (30) reported a lack of change in MPF between a reference condition and warming (40°C water) on arm muscle function, whereas Petrofsky and Lind (46) showed a positive correlation between MPF and intramuscular exercising muscle temperature during brief isometric contractions. The temperature increase may affect MPF under specific conditions through a modification of muscle fiber conduction velocity (11). It is well known that thermoregulation processes increase during exercise and that heat muscle production is quite constant during constant-power exercise. Therefore, the increase in muscle temperature is more marked at the beginning of exercise than during the slow component phase (3). The fact that MPF increased mainly in the second part of the exercise suggests that the increase in muscle temperature cannot explain the entire increase observed for the EMG signal. The lack of major effect of temperature and muscle fiber conduction velocity on MPF must be further confirmed by direct measurements.

To summarize, the MPF profile is probably the result of a balance between negative and positive effects in action potential shape and the motor unit discharge rate. On the basis of this aspect, one could not exclude the possibility that the fiber type recruitment is initiated before the minimum of the MPF profile. The fiber type recruitment could occur sooner and could be masked by a more prominent declining phase.

In conclusion, an interesting concomitance was observed in this study between the beginning of the slow component of VO₂ and the beginning of the increase in MPF. Even if an increase in motor neuron discharge
rate of the slow-twitch together with an increase in muscle temperature and a rise in muscle fiber velocity conduction cannot be completely ruled out to explain rise of MPF, the present study gives some support to the hypothesis that low-efficiency type II fibers are progressively recruited during short-term fatigue. This partially clarifies the existence of the slow component of VO₂.

REFERENCES