Dynamic Changes of Performance Fatigability and Muscular O_2 Saturation in a 4-km Cycling Time Trial

RAFAEL A. AZEVEDO[1,2], FABIO MILIONI[1,3], JUAN M. MURIAS[1], ROMULO BERTUZZI[2], and GUILLAUME Y. MILLET[1,4,5]

[1]Faculty of Kinesiology, University of Calgary, CANADA; [2]Endurance Performance Research Group (GEDAE-USP), School of Physical Education and Sport (GEDAE-USP), University of São Paulo, São Paulo, BRAZIL; [3]Faculty of Sciences, Department of Physical Education, UNESP–Universidade Estadual Paulista, Bauru, São Paulo, BRAZIL; [4]Laboratoire Interuniversitaire de Biologie de la Motricité, Université de Lyon, UJM Saint-Etienne, Saint-Etienne, FRANCE; and [5]Institut Universitaire de France (IUF), Paris, FRANCE

ABSTRACT

AZEVEDO, R. A., F. MILIONI, J. M. MURIAS, R. BERTUZZI, and G. Y. MILLET. Dynamic Changes of Performance Fatigability and Muscular O_2 Saturation in a 4-km Cycling Time Trial. *Med. Sci. Sports Exerc.*, Vol. 53, No. 3, pp. 613–623, 2021. Exercise intensity variations throughout a cycling time trial (TT) might be influenced by subject's functional state. **Purpose:** The current study characterized the performance fatigability etiology, immediately after exercise cessation, and its relation to the dynamic changes in muscle O_2 saturation (SmO_2) at different TT phases. **Methods:** Twelve males performed three separated TT of different distances, in a crossover counterbalanced design, until the end of the fast-start (FS, 827 ± 135 m), even-pace (EP, 3590 ± 66 m), or end-spurt (ES, 4000 m) TT phases. Performance fatigability was characterized by using isometric maximal voluntary contractions (IMVC), whereas the maximal voluntary activation (VA) and contractile function of knee extensors (e.g., peak torque of potentiated twitches [TwPt]) were evaluated using electrically evoked contractions performed before and immediately after each exercise bouts. SmO_2, power output (PO), and EMG were also recorded. **Results:** Immediately after the FS phase, there were lower values for IMVC (−23%), VA (−8%), and TwPt (−43%) (all $P < 0.001$), but no further changes were measured after EP (IMVC, −28%; VA, −8%; TwPt, −38%). After the ES phase, IMVC (−34%) and TwPt (−59%) further decreased compared with the previous phases ($P < 0.05$). There were lower SmO_2 and higher EMG/PO values during FS and ES compared with EP phase. **Conclusion:** FS and EP phases had similar performance fatigability etiology, but ES showed further impairments in contractile function. This later finding might be due to the abrupt changes in SmO_2 and EMG/PO because of the high exercise intensity during the ES, which elicited maximal decline in contractile function at the finish line. **Key Words:** CENTRAL FATIGUE, EXERCISE PERFORMANCE, NIRS, OXYGEN AVAILABILITY, PERIPHERAL FATIGUE

Exercise performance in cycling events is determined by the interconnection between physiological and psychological capacities, which ultimately interfere on the fatigue etiology throughout the exercise and at the finish line (1). Fatigue can be defined as a disabling symptom in which the interaction between the performance and the perceived fatigabilities is the main determinant (2). Perceived fatigability refers to changes in the psychological state of the performer, such as the rating of perceived exertion (RPE) throughout the self-paced task (2). Performance fatigability is identified by a decline in performance measurements (e.g., torque, power, and/or speed) for a given task (2) and is subsequently categorized between impairments in maximal voluntary activation (VA) and contractile function, which can be distinguished by physiological changes central and distal to the neuromuscular junction, respectively (2). Although the decrease in VA is commonly represented by changes in corticospinal mechanisms related to the descending neural motor drive to the exercised musculature (2), the contractile function impairments result from altered intramuscular mechanisms caused by the accumulation of metabolites (e.g., inorganic phosphate and hydrogen ions) (3).

It is noteworthy that most of the current understanding of fatigue etiology in cycling bouts is based on constant-load exercises and does not take into account the self-selection of exercise intensity, which occurs in real-world tasks such as a cycling time trial (TT) and is directly related to the fatigue etiology (1,4). Exercise intensity (e.g., power output [PO]) variations throughout a 4-km cycling TT can be divided in distinct phases, and this pattern may have direct consequences in the performance fatigability etiology (4). Specifically, the first part of the trial (first ~600 m) was characterized by a high

Address for correspondence: Rafael A. Azevedo, M.Sc., Faculty of Kinesiology, University of Calgary, KNB 434, 2500 University Drive NW, Calgary, AB, Canada T2N 1N4; E-mail: dalmeidazevedo@gmail.com.
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PO, which was reduced and maintained stable until ~3600 m, when PO was increased again during a final sprint, also known as end spurt (ES) (5). Those variations in PO may result in the exercise being performed within different intensity domains throughout the TT, as the first and last parts were performed within the severe domain (i.e., beyond the PO associated with the respiratory compensation point [RCP]). Indeed, exercise performed beyond the heavy domain has been characterized by distinct intramuscular metabolite accumulation and performance fatigability etiology (6). It has been demonstrated that both VA and contractile function are reduced within the first part of the TT, with no further reductions observed after the middle and ES phases (4). However, the performance fatigability measurements in the abovementioned study (4) had 1 min time delay from the exercise cessation, which could have underestimated both VA and contractile function end-exercise level (7,8). Indeed, different time delays for performance fatigability measurements after similar TT distance/duration resulted in contrasting results for both variables and led to conflicting conclusions (9,10). Previous findings with small muscle mass (i.e., unilateral knee extension) and self-paced exercise have shown that the exercise intensity variations, specifically the ES phase, have considerable influence on contractile function results when the measurements are performed immediately after the TT (11).

Concomitantly to the muscle specific changes, the exercise variation throughout a self-paced exercise seems to be an interconnection of several systemic and local factors (1). For example, in the abovementioned recent study (4), it is interesting to note that although the performance fatigability did not change after the first part of the TT, the perceived fatigability was constantly increasing until the finish line, which might suggest that other factors might be taken into account for exercise intensity variations. Among the main variables that directly influence exercise intensity variations during a cycling TT, the O_2 availability in the exercising muscles has been shown to be of extreme relevance (12). In line with this assumption, previous findings (13) have shown acute changes in a 5-km cycling TT performance related to systemic O_2 availability (i.e., hypoxia, normoxia, and hyperoxia), yet postexercise VA and contractile function changes were similar. Thus, those findings suggest that O_2 availability influences the rate of contractile function impairment and TT performance (12). More recent studies using noninvasive near-infrared spectroscopy (NIRS)–derived measurements of microvasculature O_2 saturation have shown that decreased local O_2 availability accelerated the occurrence of impairments in contractile function and had a direct effect on exercise performance (12). Although it would be reasonable to speculate that local musculature O_2 saturation is regulated to maintain contractile function and intramuscular accumulation of metabolites within tolerable concentrations throughout a cycling TT (14), most of the findings related to cycling performance and muscle O_2 saturation (SmO_2) have been either based on constant-load exercises until task failure (15–17) or did not measured SmO_2 within the same TT (13).

Therefore, the aim of the present study was (i) to characterize the performance fatigability etiology after specific phases of the TT with minimal time delay between exercise cessation and VA and contractile function measurements and (ii) to analyze the relationship among SmO_2, exercise intensity variations, and performance fatigability etiology throughout the TT. We hypothesized that (i) performance fatigability etiology would display a similar time course as that observed in our previous study (4), but with an exacerbated amplitude because of the minimal time delay between exercise cessation and the performance fatigability etiology measurements, and (ii) SmO_2 responses would have an inverse profile compared with exercise intensity and that it would be linked to the contractile function impairments throughout the TT.

METHODS

Participants

Twelve healthy young males (age = 28 ± 5 yr, body mass = 70.9 ± 7.2 kg, height = 173.5 ± 5.9 cm) participated in the study. The participants were classified as untrained cyclists in accordance with their $\dot{V}O_{2peak}$ and peak PO (see Results section) (18). Participants were informed of the experimental protocol and all associated risks before giving their written informed consent. The study was conducted according to the Declaration of Helsinki and was approved by the University of Calgary Conjoint Health Research Ethics Board (REB17-0891).

Experimental Design

Participants visited the laboratory on five occasions, which were separated by 48–72 h. All procedures were conducted in an environmentally controlled laboratory (i.e., temperature, ~21°C; relative humidity, ~36%) at a similar time of the day (±1 h) to avoid possible influence of circadian rhythms in TT performance (19). In the first session, participants underwent anthropometric measurements (i.e., body mass and height) and a maximal incremental test to task failure on a customized electromagnetically braked recumbent cycle ergometer (8), which was used in all following cycling tests. After the incremental test, and after 15 min of passive rest, the participants were familiarized with the performance fatigability assessments and the TT. The second session was composed of a TT standardized to the PO variations (i.e., pacing profile) in the remaining sessions and performance fatigability assessments (see below) before and immediately after the TT. The TT performance was used to create a software avatar for each individual's TT pacing strategy used in the experimental trials (see details in TT section). From the third to the fifth sessions, the participants performed three separate TT in a crossover counterbalanced design, which was ended at different phases of the TT, such as the end of the fast-start (FS) phase, the even-pace (EP) phase, and the ES phase. Identical pacing was maintained across all trials using the avatar provided by the software. Performance fatigability parameters were assessed

before (PRE) and immediately after (POST) each exercise session, whereas gas exchange and ventilatory responses, HR, EMG activity, and RPE were recorded during the TT. All tests were performed 2 h after the participants had their last meal, which was requested to be the same before each testing session. Participants were instructed to refrain from any exhaustive or unaccustomed exercise as well as to maintain the same food and liquid intake 24 h before the experimental sessions. A schematic representation from the study design and measurements is presented in Figure 1.

Measurements

Maximal incremental test. An incremental test to task failure was conducted to evaluate key cardiorespiratory parameters. The test began with 4 min of cycling at 20 W and then PO increased by 25 W steps every minute, and task failure was defined as the inability to maintain a minimum pedal cadence of 80 rpm for at least consecutive 5 s despite strong verbal encouragement. Breath-by-breath pulmonary gas exchange and ventilation were continuously measured using a metabolic cart (Quark, CPET; COSMED, Rome, Italy). Before each test, the gas analyzer was calibrated according to the recommendations of the manufacturer. HR was measured by an HR monitor (Garmin International, Schaffhausen, Switzerland) connected to the metabolic cart.

TT. In the second session, after a 5-min warm-up at 100 W and 90 rpm, participants performed the TT and performance fatigability assessment before and immediately after the exercise. The TT was performed within the instructions to complete the trial as fast as possible. The participants were allowed to vary the PO and select their preferred pedal frequency throughout the task. In the next three sessions, the participants were asked to cycle the TT following a preprogrammed avatar on the cyclosimulator software (Velotron 3D, Computrainer™), using the same setup and equipment as in the preliminary test. The avatar was programmed to reproduce the same PO profile adopted from the TT performed during the second visit. The participants cycled following the avatar until they completed the FS (827 ± 135 m), EP (3590 ± 66 m), or ES (4000 m) phases, and they were blinded from the distance they would cycle for during each experimental session. These three distinct phases were individually identified by a mathematical method for pacing analysis, which has been demonstrated to be highly reliable (5) and previously used in a similar study design (4). Briefly, the pacing phases identification is based on the mean PO maintained by subjects from 25% until 75% of the trial (i.e., EP phase) plus 2 SD to identify the boundaries among the other two phases. Although the FS phase is determined from the start until the point where the PO is lower than the set boundaries, the ES phase is determined at the point where the PO rises above the boundaries during the last 75% of the trial. PO during the TT was recorded at 1 Hz.

Cycle ergometer. The recumbent cycle ergometer with electromagnetically braked Velotron system (Racemate Inc., Seattle, WA) has been shown to be reliable and similar to conventional chair for isometric torque (8), where the pedals can be locked instantly in a fixed position. When the pedals were locked in position, the cranks were parallel to the ground. Both the height and the force-aft position of the cycle ergometer seat were adjusted, such that subjects were able to cycle comfortably, and when the pedals were locked in a fixed position, hip and knee angle were 100° and 90°, respectively. Participants were also secured at the hip and chest by noncompliant straps.

Torque recording. Voluntary and electrically evoked torques were recorded using a wireless PowerForce pedal

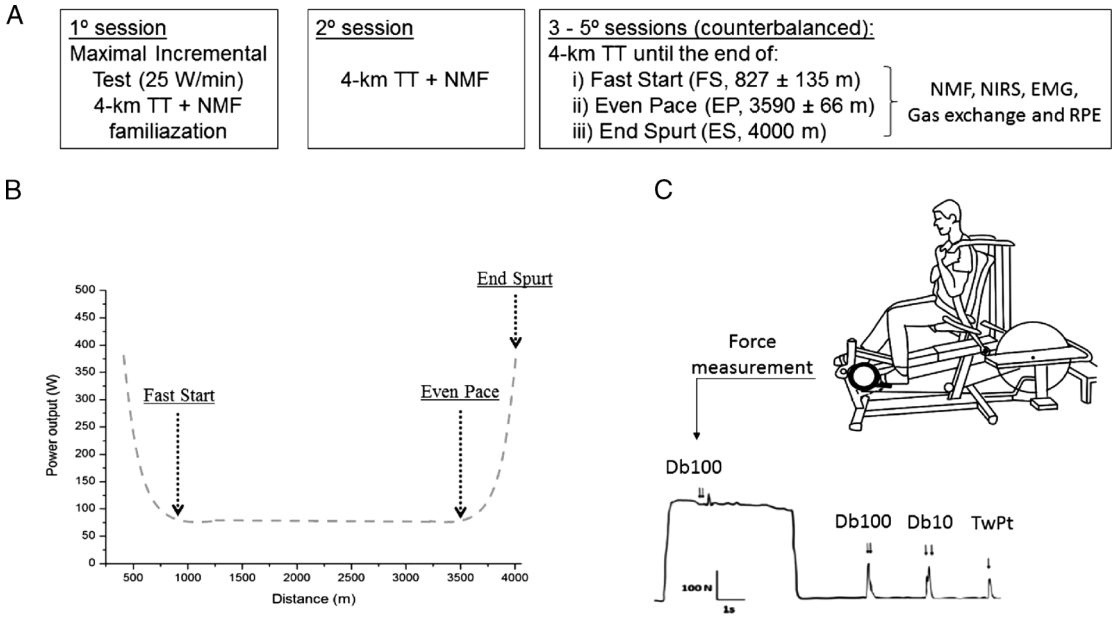

FIGURE 1—Schematic representation from the study design and measurements. A, Overall study design with each session's characteristics. B, Schematic representation from experimental sessions details. C, Equipment setup for cycling and performance fatigability measurements and the stimulation's protocol. TT, 4-km cycling TT; NMF, neuromuscular fatigue assessment.

force analysis system (Model PF 1.0.0; Radlabor GmbH, Freiburg, Germany) located between the pedal and the crank and previously validated (8). The range of force measurements is from −500 to +1500 N, and the measuring accuracy error is <2.35%. The torque produced by the knee extensors (KE) was sampled at 500 Hz and analog-to-digitally converted using Imago Record (version 8.50, Radlabor GmbH) software. Torque was provided in real-time visual feedback by converting the PowerForce signal to analog signal and exported via data acquisition card (NI PCI-6229; National Instruments, Austin, TX) and connector block (BNC-2111, National Instruments) to Powerlab system (16/35; ADInstruments, Bella Vista, Australia). The data recorded using Imago Record were converted to a text file and analyzed offline using the Labchart 8 software (ADInstruments).

Femoral nerve electrical stimulation. Transcutaneous electrically evoked contractions of the KE were induced using a high-voltage stimulator (DS71; Digitimer, Welwyn Garden City, Hertfordshire, UK) to deliver a monophasic rectangular pulse (1-ms duration) to the femoral nerve. The femoral nerve was stimulated via a monopolar 10-mm diameter cathode electrode (Meditrace 100; Covidien, Mansfield, MA) placed on the inguinal triangle and 50 × 90 mm rectangular anode electrode (Durastick Plus; DJO Global, Vista, CA) in the gluteal fold. For the optimum stimulation intensity, single stimulus was delivered incrementally (starting at 10 mA and increment of 10 mA every 30 s) until plateau in twitch torque and maximal M-wave amplitudes were reached. Supramaximal stimulation at 130% of the intensity to elicit maximal twitch and M-wave amplitudes (peak-to-peak area) was delivered to confirm supramaximal intensity (178 ± 27 mA). Electrical stimulation protocol consisted in high-frequency (100 Hz, Db100) and low-frequency (10 Hz, Db10) paired stimulus and single stimuli (peak torque of potentiated twitches [TwPt]). During the 5-s isometric maximal voluntary contraction (IMVC), a superimposed high-frequency (i.e., 100 Hz) twitch was applied when the subjects produced maximal torque, and once the muscle were relaxed, electrically evoked torques were elicited by Db100, Db10, and TwPt interspaced by 2 s (11). The participants performed a standardized warm-up consisting of five 5-s contractions of the knee extensors, interspersed by 30-s rest periods, at intensities corresponding to 50%, 60%, 70%, and 80% of the maximum subjective torque, followed by one IMVC. Thereafter, participants performed three 5-s IMVC, interspersed by 30-s rest period.

Electromyography. EMG activity of the right KE (vastus lateralis [VL] and rectus femoris [RF]) was recorded via self-adhesive Ag-AgCl surface electrodes (Meditrace 100, Covidien) in a bipolar configuration with a 30-mm interelectrode distance and the reference on the patella. To prevent movement artifact, the electrode wires were taped to the skin using adhesive tape. A low impedance (<5 kΩ) between electrodes was obtained by shaving and gently abrading the skin and then cleaning it with isopropyl alcohol. EMG signals were analog-to-digitally converted at a sampling rate of 2000 Hz by PowerLab system (16/35, ADInstruments) and octal bioamplifier (ML 138, ADInstruments) with a band-pass filter (5–500 Hz) and analyzed offline using the Labchart 8 software (ADInstruments).

Local muscle oxygenation. Local oxygenation of the VL muscle was obtained via wireless NIRS Moxy monitor device (Moxy Muslce Oxygen Sensor, Hutchinson, MN). Briefly, the Moxy device functions by sequentially sending light waves (630–850 nm) from four light emitting diodes into the tissue beneath it and recording the amount of returned scattered light at two detectors positioned 12.5 and 25 mm from the light source. The penetration depth of the light received at each detector is half the distance between the light is processed by an algorithm, which combines a tissue light propagation model and the Beer–Lambert law to determine the amount of light absorbed at wavelengths pertaining the oxygenated and deoxygenated hemoglobin (Hb). The system allows for the estimation of changes in total Hb (t[Hb]) and SmO_2 in the region of NIRS interrogation. The device was placed over the belly of VL muscle (i.e., midway from the greater trochanter and border of the patella), covered by a dark rubber shield using double side tape and wrapped with an elastic bandage. Changes in light intensities were recorded continuously at 2 Hz by the Peripedal software (Napoleon, IN) and analyzed offline.

Blood lactate concentration. Blood lactate ([Lac]) was measured using a portable [Lac] analyzer (Lactate Scout; SensLab GmbH, Leipzig, Germany) 1 min after exercise cessation. Briefly, after wiping the finger with an alcohol swab and performing a pin prick, a 2-μL capillary sample of whole blood was collected and immediately analyzed for determination of [Lac].

Data Analysis

Pulmonary gas exchange and ventilatory data were cleaned by removing data points laying ±3 SD from the local mean, and then linearly interpolated to 1-s intervals using Origin software (Origin Lab, Northampton). $\dot{V}O_2$ data were individually analyzed as previously described (20). $\dot{V}O_{2peak}$ was defined as the highest $\dot{V}O_2$ computed from a 20-s rolling average. The gas exchange threshold (GET) and the RCP were determined by two independent investigators. GET was identified as the first rise in the $\dot{V}_E/\dot{V}O_2$ without a concomitant rise in the $\dot{V}_E/\dot{V}CO_2$, and the first increase in the end-tidal oxygen tension ($PETO_2$) (mm Hg) with stable end-tidal carbon dioxide tension ($PETCO_2$) over the $\dot{V}O_2$ response. RCP was identified as the first point of a nonlinear increase in the $\dot{V}_E/\dot{V}CO_2$, a constant increase in the $\dot{V}_E/\dot{V}O_2$, and the first decrease in the $PETCO_2$ over the $\dot{V}O_2$ response. The PO associated with RCP was calculated after accounting for the mean response time during the ramp incremental exercise and used in an equation (i.e., equation 2) to also account for loss of mechanical efficiency by applying an extra correction, which enables better translation from $\dot{V}O_2$ ramp tests values to different exercise modes (21). The PO associated with the RCP was used to account for the time spent above RCP throughout the TT tests as a surrogate of the boundary between heavy and severe exercise domains.

Performance fatigability was characterized as the change in IMVC postexercise in relation to baseline (i.e., before any

cycling exercise) (9,22). The exercise-induced effects on maximal central drive were measured as the change in VA (23,24), and contractile function impairments were measured from Db100 and TwPt as well as low-frequency fatigue (Db10:100) (11). Maximal voluntary torque was calculated as the highest IMVC from two (or three) IMVC at baseline, and the IMVC performed ~2 s postcycling. A third IMVC at baseline was performed if the peak torque variation before the superimposed twitch between the first two IMVC was greater than 5%. The electrically evoked torque from the doublets and single pulse was determined as the peak torque of each stimulation on the relaxed muscle. The M-wave peak-to-peak amplitude in response to single stimuli was measured as the absolute difference between the maximum and the minimum points of the biphasic M-wave.

VA was assessed by a superimposed paired-pulse technique as per the method described by Strojnik and Komi (41) shown in equation 1:

$$\mathrm{VA}\ (\%) = 100 - D\left(\mathrm{IMVC}_{\mathrm{Db100}}/\mathrm{IMVC}_{\mathrm{peak}}\right)/\mathrm{Db100} \times 100 \quad [1]$$

where $\mathrm{IMVC}_{\mathrm{Db100}}$ is the voluntary torque when superimposed Db100 was delivered, $\mathrm{IMVC}_{\mathrm{peak}}$ is the highest torque during the IMVC before the superimposed Db100, D is the difference between the torque level at the time of $\mathrm{IMVC}_{\mathrm{Db100}}$ and the maximum torque during superimposed Db100, and Db100 is the electrically evoked torque on relaxed muscle 2 s after IMVC. Practice, visual feedback of performance, and standardized verbal encouragement were adopted in the present study to ensure the maximum effort of participants during the IMVC and performance fatigability parameters assessments. Furthermore, participants were familiarized with the torque measurements and electrically evoked twitch responses on two different days (i.e., first and second visits).

The root mean square (RMS) of the EMG signal was calculated during the best baseline IMVC and for each burst during the TT. During cycling, the period of activation of each burst onset and offset was determined as the period where the signal exceeded a threshold of 15% of the maximum activity of that muscle during the trial for at least 100 ms (4). The muscle recruitment for each 400 m was represented by the mean RMS of all bursts within a given 400-m interval. These parameters were selected based on the signal-to-noise relationship of the EMG data and were confirmed to be accurate by careful visual inspection of the periods of muscle activation, where the misclassification of burst onset and offset was manually corrected. The manual correction followed the same criteria of 15% of the maximum activity for at least 100 ms. The 500 ms of peak torque before the superimposed twitch was used to calculate RMS during the highest baseline IMVC and was used to normalize the RMS during the TT. These analyses were performed using a specific algorithm developed in Matlab (version R2015a; MathWorks, Natick, MA).

Perceived fatigability was characterized by the changes in the RPE throughout the TT in every condition. The instructions for RPE scale (25) were standardized in accordance with procedures previously described (26). Briefly, subjects were asked to rate their conscious sensation of how hard, heavy, or strenuous the physical task was. For example, a rating of 9 in the scale corresponded to "very light" exercise (e.g., walking slowly at a self-selected own pace for several minutes). A rating of 17 in the scale corresponded to a "very hard" and strenuous exercise (e.g., a healthy individual must strongly push themselves to continue, as the exercise feels very heavy).

NIRS-derived signal data were analyzed by examining $\mathrm{SmO_2}$ changes throughout the TT. Given the uncertainty of the optical path length in the VL during exercise, NIRS data were represented by delta change from pre-TT ($\%\mathrm{SmO_2}$) for each participant and experimental trial.

During all TT bouts, the variables acquired during the exercise, such as PO, gas exchange ($\dot{V}O_2$ and $\dot{V}_E$), HR, EMG (VL and RF muscles), and $\%\mathrm{SmO_2}$ were averaged in separated 400-m bin. RPE was also noted at the end of each 400 m.

Statistical Analysis

Normal distribution of the data was confirmed by Shapiro–Wilk's test, and results were reported as means ± SD. A two-way repeated-measures ANOVA (distance–condition) was used to evaluate if PO, gas exchange ($\dot{V}O_2$ and $\dot{V}_E$), HR, EMG, RPE, and $\%\mathrm{SmO_2}$ were similar among the three experimental trials. In addition, for the ES condition (i.e., the whole TT), a one-way repeated-measures ANOVA was used to analyze the changes in PO, physiological variables, and performance fatigability parameter delta change at each distance (i.e., 400 m intervals) compared with baseline measurement. Finally, a two-way ANOVA (PRE–POST × FS, EP or ES) was used to evaluate the performance fatigability parameter differences among conditions and possible differences between the TT familiarization and the ES sessions. When F values were significant, Tukey *post hoc* tests were used to determine where differences existed. Partial eta squared $\left(\eta_p^2\right)$ (27) for ANOVA comparisons was computed, assuming small (<0.02), medium (0.02–0.26), and large (>0.26) effect sizes (28). Pearson product moment correlation was calculated to quantify the relationship between PO, EMG, $\%\mathrm{SmO_2}$, and performance fatigability variables (IMVC, VA, and TwPt) throughout the TT phases. The significance level was set at $P \leq 0.05$. All statistical analyses were performed using a statistical software package (version 10.0; Statistica, Tulsa, OK).

RESULTS

Incremental test peak values and submaximal thresholds. Peak values achieved at the end of the incremental test were as follows: PO (281 ± 55 W), $\dot{V}O_2$ (3.37 ± 0.48 L·min^{-1}), $\dot{V}_E$ (159 ± 8 L·min^{-1}), and HR (182 ± 8 bpm). The following PO and $\dot{V}O_2$ values were associated with GET (123 ± 34 W and 1.85 ± 0.37 L·min^{-1}) and RCP (169 ± 28 W and 2.63 ± 0.46 L·min^{-1}).

Exercise performance, physiological responses, and perceived fatigability throughout the TT. The total

time to complete the TT was 430 ± 39 s, with an average time to complete the FS, EP, and ES of 91 ± 19 s, 297 ± 34 s, and 42 ± 9 s, respectively. PO distribution, $\%SmO_2$, muscle recruitment, and RPE during each experimental session and the profile of PO performed in the severe domain (% above the RCP) are shown in Figure 2. PO at each point of analysis throughout the TT was consistent among experimental sessions ($P > 0.05$) and highly correlated ($r = 0.99$, $P < 0.001$) (Fig. 2A). PO was greater at the 400- and 4000-m splits compared with the rest of the trial ($P < 0.001$, $\eta_p^2 = 0.430$).

The $\%SmO_2$ changed ($P < 0.001$, $\eta_p^2 = 0.430$) throughout the TT, with the 400-, 800-, and 4000-m splits displaying greater changes in percent values compared with the rest of the trial ($P < 0.001$) (Fig. 2B). In addition, the baseline SmO_2 values (i.e., 5-min warm-up at 100 W and 90 rpm) were not different among the FS (70% ± 5%), EP (70% ± 5%), and ES (73% ± 7%) sessions ($P > 0.05$).

Similar to the PO variations, muscle recruitment for VL and RF, as measured by the RMS signal, was higher at 400- and 4000-m splits compared with the other parts of the trial ($P < 0.001$, $\eta_p^2 = 0.507$ and 0.362) (Fig. 2C and D). There were no significant differences for the RMS signal of either muscle between the three experimental sessions ($P > 0.05$).

The PO ($P < 0.001$, $\eta_p^2 = 0.561$) at the 400- and 4000-m splits were performed in a greater percentage beyond the PO associated with the RCP in comparison with the other parts of the trial ($P < 0.001$). There were no differences among the three experimental sessions ($P > 0.05$). The time spent above and below the PO associated with RCP varied throughout the TT ($P < 0.001$, $\eta_p^2 = 0.484$). The percentage of time spent above the PO associated with RCP was higher during the FS (99% ± 1%) and the ES (95% ± 12%) when compared with EP (81% ± 24%) ($P < 0.001$).

In relation to the perceived fatigability, the RPE increased throughout the TT ($P < 0.001$, $\eta_p^2 = 0.796$), where there was a consistent increase until the 2400-m split and a greater value at the 4000-m split compared with the other parts of the trial ($P < 0.05$) (Fig. 2E).

Performance fatigability parameters. The comparison between familiarization and ES conditions for performance fatigability variables only showed a main effect of time for IMVC ($\eta_p^2 = 0.823$), VA ($\eta_p^2 = 0.611$), Db100 ($\eta_p^2 = 0.895$), Db10:100 ($\eta_p^2 = 0.891$), and TwPt ($\eta_p^2 = 0.890$) (all $P < 0.001$) but no main effect of condition nor interaction ($P > 0.05$). Absolute values for performance fatigability parameters measured before and after FS, EP, and ES are reported in Table 1. IMVC decreased after the TT and was different among phases ($P = 0.026$, $\eta_p^2 = 0.281$), where ES phase showed lower values than FS ($P < 0.001$). VA also showed a decrease after the TT ($P < 0.001$, $\eta_p^2 = 0.775$), but it was not significantly different among phases ($P > 0.05$).

Regarding the contractile function parameters, TwPt decreased after the TT, and it was different among phases ($P = 0.014$, $\eta_p^2 = 0.320$), where ES had lower values when compared with FS ($P = 0.012$) and EP ($P = 0.003$). Similarly, Db100 ($P < 0.048$, $\eta_p^2 = 0.240$) and Db10:100 ($P = 0.020$, $\eta_p^2 = 0.296$)

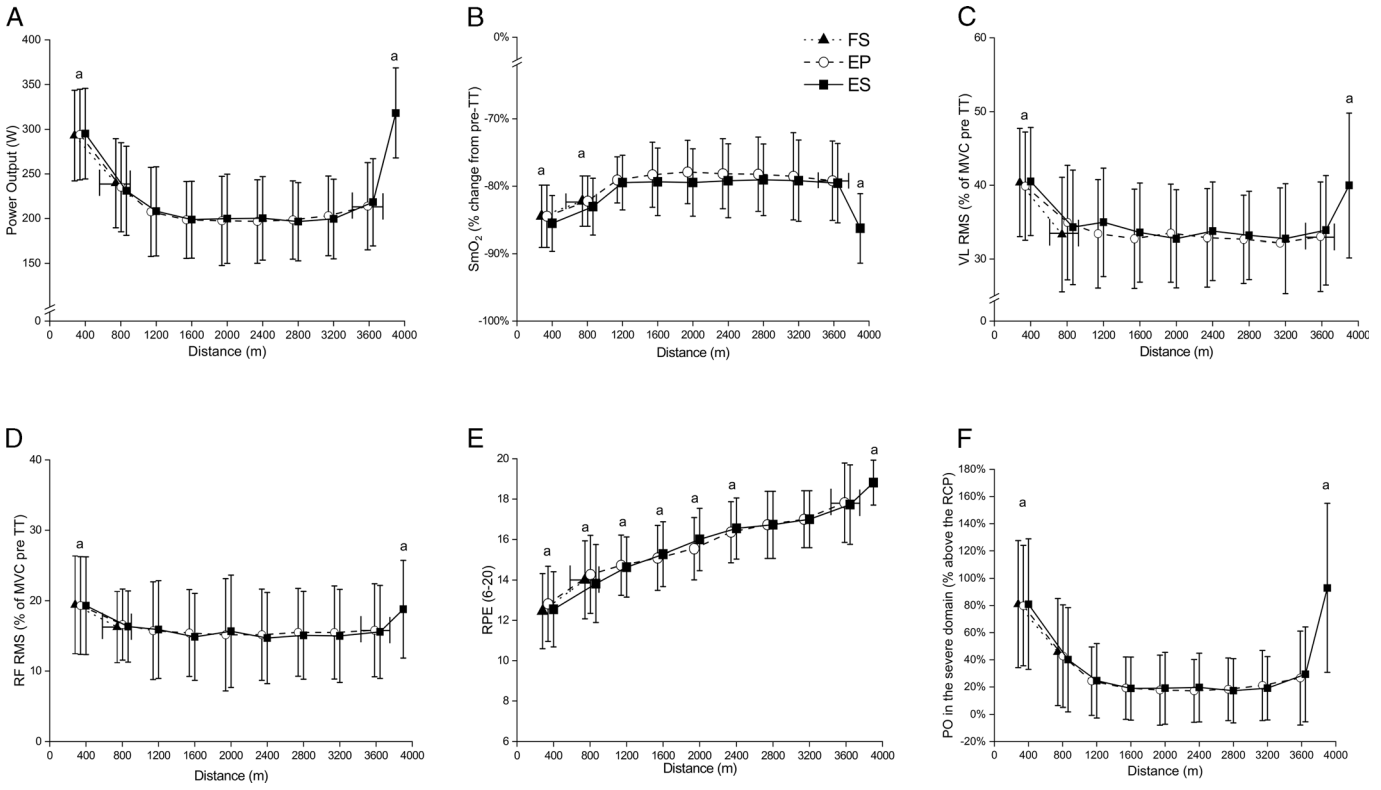

FIGURE 2—Time course of PO (A), SmO_2 (B), EMG of VL (C) and VM (D), RPE (E), and the relative PO performed in relation to the PO associated with the RCP (F) throughout the cycling time trial. SmO_2, represented as percentage from pre-TT; RMS: root mean square of the VL and RF represented as percentage of the maximal voluntary contraction measured at pre-TT. Data are presented as mean ± SD. [a]Main effect of distance ($P < 0.05$).

TABLE 1. Absolute values for neuromuscular variables from pre- to postexercise after three phases of a cycling TT.

		FS	EP	ES
IMVC (N)	Pre	337 ± 101	325 ± 77	326 ± 91
	Post	258 ± 82*	231 ± 53*	208 ± 40*,**
VA (%)	Pre	97 ± 4	97 ± 2	97 ± 3
	Post	89 ± 8*	89 ± 8*	87 ± 8*
TwPt (N)	Pre	131 ± 20	128 ± 18	128 ± 22
	Post	74 ± 29*	77 ± 16*	51 ± 22*,**,***
Db100 (N)	Pre	177 ± 30	172 ± 34	170 ± 39
	Post	121 ± 34*	124 ± 29*	101 ± 29*,**,***
Db10:100	Pre	1.00 ± 0.07	1.02 ± 0.13	1.04 ± 0.11
	Post	0.76 ± 0.11*	0.76 ± 0.10*	0.67 ± 0.13*,**,***
VL M-wave	Pre	11.9 ± 4.6	12.3 ± 4.3	12.4 ± 4.1
PPA (mV)	Post	11.6 ± 4.9	13.0 ± 4.6	12.6 ± 4.5
	% Change	5 ± 19	−5 ± 10	−1 ± 13
RF M-wave	Pre	5.4 ± 2.4	5.5 ± 1.8	5.4 ± 2.1
PPA (mV)	Post	5.4 ± 1.9	5.8 ± 2.1	5.6 ± 2.4
	% Change	−0 ± 14	−4 ± 10	−1 ± 21

Data are presented as mean ± SD.
*Statistically lower than pre ($P < 0.05$).
**Statistically lower than FS condition ($P < 0.05$).
***Statistically lower than EP condition ($P < 0.05$).
TwPt, single twitch electrically evoked torque (1 Hz); Db100, high-frequency (100 Hz) doublet electrically evoked torque; Db10, low-frequency (10 Hz) doublet electrically evoked torque; Db10:100, ratio between Db10 and Db100; PPA, peak-to-peak amplitude. % Change, percentage change from prevalues.

decreased significantly after the TT and were different among phases, where the ES phase had lower values than FS and EP for Db100 ($P = 0.002$ and 0.036) and Db10:100 ($P = 0.042$ and 0.043).

The delta changes (Δ) from PRE for performance fatigability variables are also shown in Figure 3. The change in ΔIMVC was different among conditions ($P = 0.011$, $\eta_p^2 = 0.331$), where ES had a greater decrease when compared with FS phase ($P = 0.008$) (Fig. 3A). ΔVA parameter did not change among phases ($P = 0.496$) (Fig. 3B). ΔTwPt was different among conditions ($P = 0.007$, $\eta_p^2 = 0.361$), where ES showed greater decrease compared with FS ($P = 0.045$) and EP ($P = 0.007$) (Fig. 3C). ΔDb100 ($P = 0.023$, $\eta_p^2 = 0.288$) was also different among phases, where ES had greater decrease compared with EP ($P = 0.020$) (Fig. 3D). In addition, ΔDb10:100 ($P = 0.011$, $\eta_p^2 = 0.335$) was different among conditions, where it showed greater decrease after ES compared with FS ($P = 0.003$) and EP ($P = 0.023$) (Fig. 3E). The M-wave amplitude for VL and RF muscles during performance fatigability assessments did not change either after the trial or among conditions ($P > 0.050$).

Cardiorespiratory variables and blood [Lac] concentration. Cardiorespiratory variables and blood [Lac] are shown in Figure 4. There was a distance effect for $\dot{V}O_2$ ($P < 0.001$, $\eta_p^2 = 0.493$), $\dot{V}_E$ ($P < 0.001$, $\eta_p^2 = 0.574$), and HR ($P < 0.001$, $\eta_p^2 = 0.779$). *Post hoc* analysis revealed lower $\dot{V}O_2$ at the 400-, 800-, and 1200-m splits compared with the other parts of the trial ($P < 0.05$). $\dot{V}_E$ had lower values for the 400-m split compared with the other parts of the trial ($P < 0.05$). HR consistently increased until the 3200-m split compared with the other evaluation points throughout the TT ($P < 0.05$). [Lac] was different among conditions ($P = 0.017$, $\eta_p^2 = 0.308$), where FS had lower values than ES ($P = 0.013$). $\dot{V}O_2$, $\dot{V}_E$, HR, and metabolic PO at respective evaluation points throughout the TT were not different between the three experimental sessions ($P > 0.05$). In addition, the end ES values for $\dot{V}O_2$ ($P = 0.271$), $\dot{V}_E$ ($P = 0.143$), HR ($P = 0.944$), and PO ($P = 0.138$) were not significantly different from peak values achieved in the incremental test.

Correlations from PO, EMG, %SmO_2, and performance fatigability variables. PO and EMG were negatively correlated with %SmO_2 changes throughout the TT ($r = -0.55$ and $P < 0.001$; and $r = -0.36$ and $P = 0.02$, respectively). Also, PO and EMG were positively correlated ($r = 0.48$, $P = 0.003$). There was a negative correlation between PO and ΔTwPt for FS ($r = -0.70$, $P = 0.001$) and ES ($r = -0.63$, $P = 0.001$) phases. EP did not show any correlation with the selected variables (all $P > 0.05$).

DISCUSSION

The main findings from this study were that (i) the performance fatigability etiology immediately after the exercise was characterized by decreased VA and contractile function after the FS phase and was stable after EP phase, whereas the ES phase showed further contractile function impairments, including low-frequency fatigue, and (ii) SmO_2 showed an inverse pattern compared with PO and EMG, with an exacerbated decrease in SmO_2 during the FS and ES, where exercise intensity was greater than EP phase. Taken together, the present results are in accordance with our initial hypothesis that time delay from exercise cessation is of paramount importance for performance fatigability assessments, as shown by the different conclusion in the present paper compared with our previous one (4) regarding fatigue induced by ES. In addition, based on the inverse profiles from the SmO_2 and the PO and EMG variables, the present findings highlight the integration of performance fatigability etiology, local muscular changes, and self-controlled exercise intensity variations throughout a TT.

Performance fatigability responses throughout the TT and time delay effect. The exercise intensity variation during a self-paced task, defined as pacing, is intended to achieve the best performance and avoid premature task failure (1). Indeed, during self-paced cycling exercise of middle duration (i.e., ~6 min), such as in the present study, the pacing has been often characterized by a higher PO and muscle activation during the FS and ES phases when compared with the EP phase (4). It has recently been shown that performance fatigability characterization varies throughout the TT, where the PO adopted during the FS and ES phases were followed by decreased values from VA and contractile function parameters (4). It is noteworthy that previous findings related to performance fatigability and self-paced exercises might be limited by the delay from exercise cessation and performance fatigability assessments, where VA and contractile function show recovery within the first minute and thereafter measurements might be underestimated (7,8). This limitation was circumvented in the present study by the cycle ergometer used (8), which allows immediately performance fatigability assessments. The

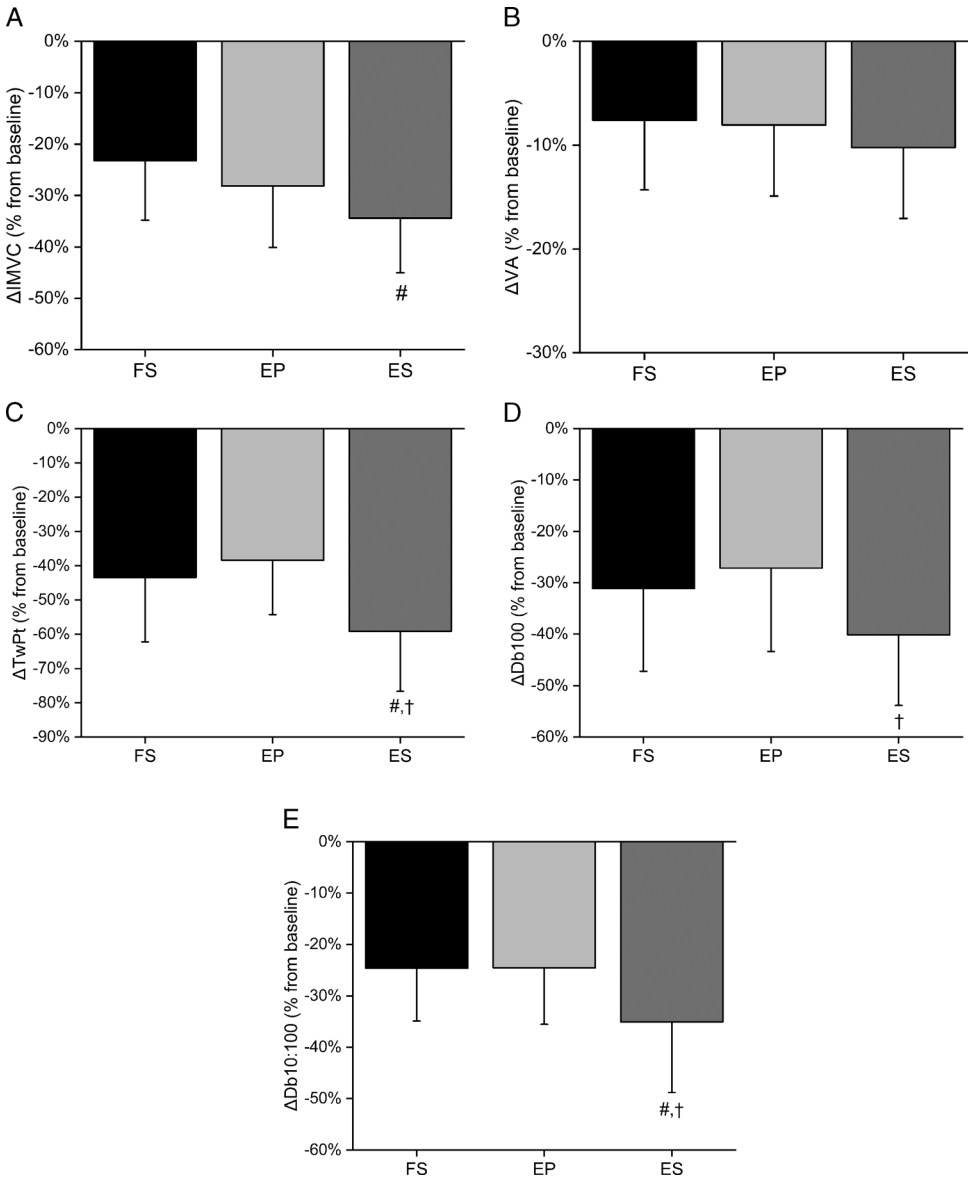

FIGURE 3—Neuromuscular variables changes from baseline for each condition. IMVC (A); VA (B); TwPt, single twitch electrically evoked torque (1 Hz) (C); Db100, high-frequency (100 Hz) doublet electrically evoked torque (D); Db10:100, ratio between Db10 and Db100 (E); % Change, percentage change from prevalues. Data are presented as mean ± SD. #Statistically lower than FS condition ($P < 0.05$). †Statistically lower than EP condition ($P < 0.05$).

present findings are in line with previous study (4) regarding the early decrease in VA, more specifically after the FS phase, and stable values throughout the remaining phases (i.e., EP and ES). In addition, the present findings demonstrate valuable insights regarding the contractile function impairments with minimal time delay from exercise cessation. Indeed, in addition to larger changes from pre- to postphases in the present study compared with our previous study (4), the present finding showed an exacerbated decrease in contractile function after the ES phase when compared with the other phases (i.e., FS and EP), whereas the contractile function (e.g., the TwPt delta change from baseline until the end of each phase: FS, −23% ± 8%; EP, −23% ± 13%; ES, −26% ± 12%) was not significantly different among the phases. It is noteworthy that the characterization of VA changes and contractile function impairments after cycling exercises has long been underestimated because of the time delay between exercise cessation and measurements (8,29). For example, early evidence had suggested that there was no VA change after a 5-km cycling TT, which had ~4 min time delay (30), and more recent evidence consistently showed an exacerbated decrease on VA after similar TT distance/duration (e.g., 4- and 5-km cycling TT) (3,9,31,32). Regarding the contractile function responses, the present findings are in line with self-paced exercises, which used single limb (e.g., knee extension) of different duration and showed that the final sprint (i.e., ES) could result in exacerbated contractile function impairment, when the measurements were performed with minimal time delay (11). In fact, recent findings from a 5-km cycling TT have also shown exacerbated decline in contractile function

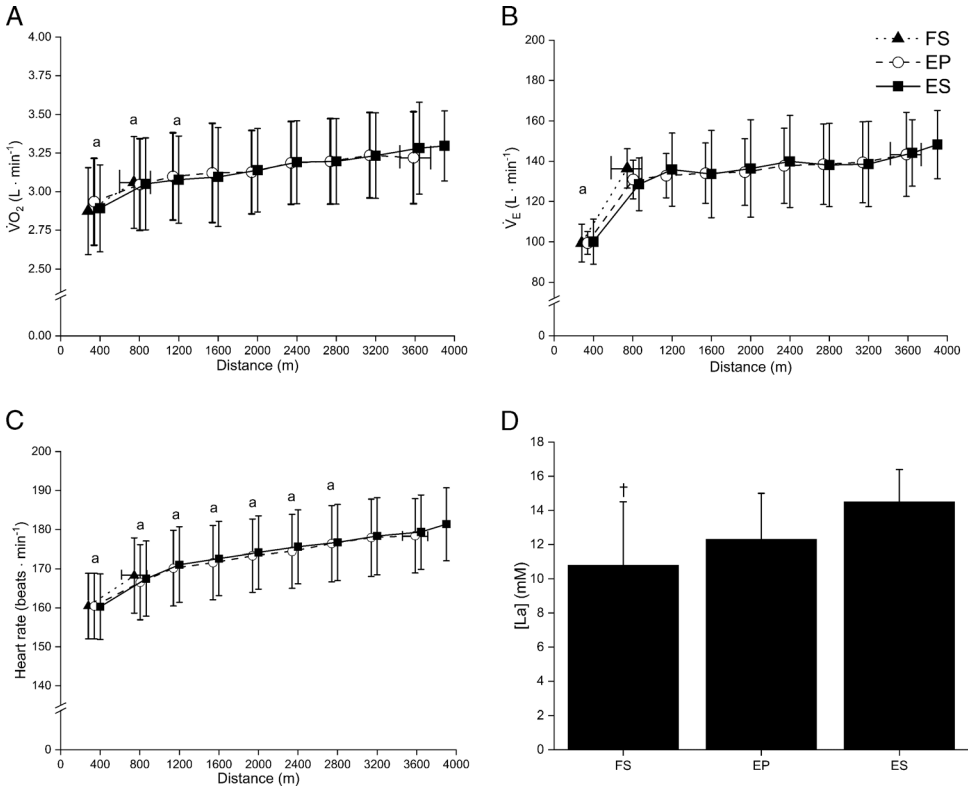

FIGURE 4—The time course of oxygen uptake (A), minute ventilation (B), HR (C), blood [Lac] accumulation (D), measured during the experimental trials. $\dot{V}O_2$, oxygen uptake; $\dot{V}_E$, minute ventilation. Data are presented as mean ± SD. [a]Main effect of distance ($P < 0.05$). †Statistically lower than ES condition ($P < 0.05$).

(i.e., Db10), IMVC, and VA because of the ES when compared with the performance fatigability at the 4-km time point with a 10-s delay from exercise cessation (33). Taken together, the present results give valuable insights into the understanding of performance fatigability etiology throughout a self-paced exercise, once the assessments were performed with minimal time delay.

Exercise intensity variations, SmO_2, and performance fatigability interconnection. Although previous studies have suggested that exercise intensity variation would be due to acute muscular metabolic changes, which would be reflected in decreased SmO_2 and contractile function impairments (12,13), there is a lack of empirical data to support this assumption. The findings from the present study revealed that SmO_2 showed an inverse profile compared with PO and muscle recruitment throughout the TT, and that there was a negative correlation among PO, EMG and $\%SmO_2$ variables throughout the TT. Thus, the present study reinforces the possible interconnection between the metabolic changes within the muscle and the CNS commonly shown in constant-load exercises (15) and further expanded these findings to self-paced tasks (13).

Our results from SmO_2, PO, and performance fatigability etiology throughout the TT also suggest an interconnection among these variables and self-regulation of exercise intensity. It was shown that the decrease in SmO_2 and the contractile function impairments were exacerbated during FS and ES phases, but both variables did not change during the EP phase. It must be highlighted that decreased SmO_2 during an exercise bout could be interpreted as greater reliance on O_2 extraction and augmented metabolic demand within the interrogated muscle area (34). In fact, our results are in line with this assumption, where the exercise intensities performed during the FS and the ES phases were mostly within the severe domain (Fig. 2F) and had greater PO and muscle recruitment when compared with EP phase. In line with this observation, although the PO in the FS and ES phases were correlated with the ΔTwPt ($r = -0.70$ and -0.63, respectively), this correlation was not evidenced for EP phase in the present study. These findings would represent greater reliance on local O_2 extraction and use of anaerobic resources to match the metabolic demand and resynthesize ATP (6). As a result, the acute increase on metabolic demand would accelerate intramuscular metabolite accumulation and contractile function impairments (35), possibly increasing the feedback from afferent muscle fibers (i.e., type III and type IV fibers) to the CNS and modulating the motor neural drive to the active musculature (12,13,15).

Although the link between contractile function and muscle activation throughout a TT is not fully understood and still under debate (36), previous evidence suggests that O_2 availability might be a key variable related to the intramuscular metabolite accumulation and afferent feedback to CNS (12). Thus, it would be plausible to suggest that the early decrease on SmO_2 during the FS phase would represent a rapid increase in intramuscular metabolite accumulation and contractile function impairments, activating the afferent feedback to the CNS and lowering the PO/muscle activation to a tolerable

level during the EP. It is interesting to note that the high PO adopted during the ES phase resulted in abrupt decrease in muscular O_2 saturation and greater contractile function impairment without changes in VA when compared with the other phases. Therefore, other physiological variables might also be taken into account by the CNS to determine the descending motor drive to the active musculature and exercise intensity variations throughout the TT.

Perceived fatigability responses throughout the TT. It is widely accepted that exercise intensity variations throughout a self-paced TT is regulated by the perceived exertion (i.e., RPE), which is characterized by a linear increase until the finish line (1,37). Indeed, previous studies have shown that regardless the TT duration, the rate of increase in the RPE is similar and directly influences the exercise intensity adopted throughout the task (37). It is suggested that several feedback and feedforward information, from internal and external sources, are taken into account for the RPE changes (38). For example, reduced systemic and muscular O_2 saturation results in impaired TT performance but did not change the end-exercise RPE, contractile function, and VA responses when compared with control condition (13). In addition, it has been suggested that cardiopulmonary feedback might also contribute to the RPE response during exercise, where greater muscle mass involved on the task would result in augmented cardiopulmonary demand and inhibitory feedback to CNS, thus diminishing contractile function impairments at exhaustion (36).

In this context, an integrative perspective could be used to explain the self-paced exercise intensity changes from a physiological perspective. Given that the exercise intensity is driven by perceived exertion in self-paced bouts, the high intensity adopted in FS phase followed by a constant decrease until its stabilization during the EP would be a result of muscular and systemic physiological changes. At the muscular level, the high-intensity exercise adopted, within the severe intensity domain, resulted in exacerbated decrease of SmO_2 and probably eliciting faster rate of metabolite accumulation and contractile function impairments (12). Thereafter, the exercise intensity was probably regulated based on a slower intramuscular metabolite accumulation and systemic physiological changes, in which the PO performed during the EP phase was closer to the PO associated with the RCP threshold (Fig. 2F), to maintain the perceived exertion within a tolerable and constant increase until beginning of the ES phase. In line with this assumption, the muscular O_2 saturation and the performance fatigability etiology were characterized by stable values throughout the EP phase (i.e., Fig. 2B and Table 1), whereas the RPE response continuously increased until the 2800-m split (Fig. 2E). Finally, the high exercise intensity during the ES phase was probably a result of conscious decision based on feedback from physiological variables and feedforward information within the CNS, eliciting the highest perceived exertion at the finish line. In line with this, the muscle activation, SmO_2, contractile function, and exercise intensity all had drastically changed in a short period when compared with the other phases.

Limitations. Before any conclusion can be addressed, some limitations of the present study design must be acknowledged. First, exercise intensity variation during a self-paced exercise can be influenced by voluntary decision of the participants, rather than solely driven by physiological measurements (39). For example, external clues, such as riding against an opponent (40), might directly influence the pacing profile and end-exercise perceived and performance fatigability etiology. Thus, the assumptions based solely internal physiological measurements must be taken with caution in self-paced exercises. Second, the present study only investigated a relatively short distance and duration TT (i.e., 4 km, ~7 min). It has been shown that depending on the TT duration, the end-exercise performance fatigability etiology will be affected (11). Third, the present study setup was performed on a recumbent cycle ergometer rather than conventional upright position, which might interfere on exercise performance, as stated elsewhere (8). However, this interference seems to not affect performance fatigability because our measurements were of similar magnitude as reported by previous findings in conventional upright cycling 4-km TT (4,9,33). In addition, the recumbent ergometer has been shown to be highly reliable and considerable faster to assess end-exercise performance fatigability compared with conventional setup (8). Finally, these results might be restricted to healthy young male individuals. It is likely that some potential sex- and age-related characteristics could influence the present results.

CONCLUSION

The present findings showed that there is an early decrease in VA and contractile function variables after the FS that is maintained throughout the EP phase. When the performance fatigability etiology is measured with minimal time delay from exercise cessation, there is a further decrease on contraction function variables immediately after the ES that can be explained by an increased exercise intensity and decreased SmO_2 in the last 400 m. This is likely because perceived fatigability is not the regulating variable anymore when approaching the finish line.

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