Influence of Knowledge of Sprint Number on Pacing during Repeated-Sprint Exercise

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ABSTRACT

BILLAUT, F., D. J. BISHOP, S. SCHAERZ, and T. D. NOAKES. Influence of Knowledge of Sprint Number on Pacing during Repeated-Sprint Exercise. Med. Sci. Sports Exerc., Vol. 43, No. 4, pp. 665–672, 2011. Purpose: The anticipation of exercise-induced stress influences performance during continuous exercise. However, not all exercise is continuous. This study explores the influence of prior knowledge of sprint number on mechanical work, surface EMG, and RPE during repeated-sprint exercise (RSE). Methods: Fourteen athletes performed three RSE in random order. In one trial, subjects were informed that they would perform ten 6-s cycle sprints (with 24 s of rest) and then completed 10 sprints (control trial, CL). In a second trial, subjects were told to perform five sprints, but after the fifth sprint, they were asked to perform an additional five sprints (deception trial, DC). In a third trial, subjects were not told how many sprints they would be performing but were stopped after 10 sprints (unknown trial, UN). Data were recorded for every sprint. Results: Both the initial sprint work and work accumulated during the first five sprints were greater (6.5%, P < 0.05) in the CL than in the DC and UN trials. Furthermore, the work accumulated during the ten sprints was lower (4.0%, P < 0.05) in the UN trial than in the two other trials. The EMG was greater (P < 0.05) in the DC than in the CL and UN trials during the initial sprint (8.8%) and during the first five sprints (9.1%). The sprint-induced decrease in EMG and work occurred earlier in the UN trial compared with the CL and DC trials. The RPE profile was similar in all trials. Conclusions: Results demonstrate that pacing occurs during short repeated-sprint efforts in anticipation of the number of sprints that are included in the trial. Key Words: REPEATED-SPRINT ABILITY, DECEPTION, ANTICIPATION, MUSCLE RECRUITMENT

Competitive pacing strategies involve the conscious and/or subconscious variation of workload over the duration of an exercise to limit premature fatigue and, thereby, increase the likelihood of winning an event (1,11,14). It has been argued that this strategy would be established at the beginning of the exercise, based on pre-exercise expectations of task duration and intensity as well as physiological (e.g., muscle glycogen levels, arterial oxygen saturation) and psychological (e.g., motivation level, presence of competitors) inputs (19,24,25,27). The mass of skeletal muscle recruited during exercise would subsequently be regulated to adjust mechanical output and to maintain performance (19,24). To date, most studies devoted to examining pacing patterns during exercise have focused on continuous exercise and have demonstrated that pacing strategy is a major determinant of performance during continuous exercise lasting >30 s (2,12,16,21,24). However, not all exercise is continuous. Many sport disciplines such as team and court sports require very short (≤6 s) bouts of all-out intensity (>300% intensity at which VO2max is achieved) interspersed with incomplete (≤30 s) recovery during an extended period (23). To date, there has been little research investigating the potential presence of pacing during such tasks.

Exercise physiologists agree that the determinants of performance (i.e., power output and/or speed) during repeated-sprint exercise (RSE) include peripheral factors such as substrate depletion, metabolite accumulation, purine nucleotide loss, and ionic disturbances (for review, see Billaut and Bishop (6) and Spencer et al. (23)). The modulation of motor unit activity has also been invoked as a determinant of performance during RSE (5,7,18,22). Therefore, owing to the high level of physiological stress imposed on the body during RSE, it is possible that athletes also consciously or subconsciously adopt a pacing strategy during these activities, with alterations in skeletal muscle recruitment occurring throughout the exercise, to minimize metabolic disturbances and mechanical constraints occurring in the muscles and the joints of active limbs. In addition to these all-out sprints, RSE is typically interspersed with short and often incomplete recovery intervals, which may exacerbate the metabolic disturbances of the task, and in turn, result in negative “emotional signals” and a deterioration in exercise self-confidence (i.e., one’s perceived ability to succeed in the task). Taken
together, these psychophysiological changes could lead to the adoption of a pacing strategy to delay the occurrence of fatigue and/or intolerable discomfort. A recent report of pacing strategy during high-intensity sprints has demonstrated that the level of eccentric muscle loading (i.e., mechanical constraint) influences the pacing pattern during ten 60-m running sprints separated by 4 min of rest (4). However, to our knowledge, no study has yet evaluated the presence of pacing during RSE where the number of sprints to be performed is known before performance. This is of crucial interest because the most important parameter establishing the pacing strategy is probably the knowledge of the end point of exercise (16,19,24). In the context of RSE, knowledge of the end point of exercise refers to the number of all-out sprints to be performed with a given rest period between sprints.

Knowledge of the end point of exercise has been demonstrated to be a major factor in the allocation of physiological and psychological resources during exercise (24,25). Expectation of task duration and anticipation of the end point influence subjective ratings of fatigue, RPE, and muscle activation. Indeed, a typical finding in the literature is that pacing strategy is altered in association with changes in the EMG activity (14,16,25), which strongly suggests that altered skeletal muscle activation levels are, in part, responsible for the altered exercise performance. Therefore, analyzing this neurophysiological coupling during RSE could advance our knowledge of the mechanisms responsible for the selection of a pacing strategy during repeated, short-sprint efforts. Furthermore, Noble and Robertson (20) have proposed the “Perceptual-Cognitive Reference Filter” (which was recently developed into the “Anticipatory-RPE model” (26)), in which the brain continuously compares the conscious RPE generated from the sum of the signals elicited from varied physiological systems with a RPE “template” based on experience. The subjective effort sensation has been hypothesized to contribute to the regulation and cessation of exercise performance because of its potentially strong biological link with exercise-induced physiological changes (20,26,28). Thus, the main notion of these models is that exertional perceptions mediate pacing strategy, and as a result, RPE is often scaled proportionally to the remaining exercise time/distance of a continuous exercise. Such data are not yet available during RSE where the sprint-induced metabolic changes may influence RPE, muscle activation, and the pacing strategy.

Accordingly, the current study was designed to determine, through deception, the influence of an unknown and an unexpected increase in sprint number on the mechanical output profile, EMG activity, and RPE during RSE. Our hypotheses were as follows: 1) the anticipation of performing fewer sprints would lead to higher skeletal muscle recruitment (as evidenced by changes in EMG activity), greater mechanical output, and an increased perception of exertion; and 2) not knowing the number of sprints to be performed would result in a more “economical” pacing strategy (i.e., reduced EMG, work, and RPE). Such changes would provide evidence that

preexercise expectations of the end point of exercise also affect the pacing strategy adopted during RSE.

**METHODS**

**Subjects**

Fourteen physically fit women from the university’s track-and-field team took part in this study (21.1 ± 4.5 yr, 166.2 ± 4.3 cm, 60.4 ± 3.1 kg, 14.2% ± 2.5% body fat). All subjects were healthy and with no known neurological or cardiovascular diseases. They trained on a regular basis (current training volume = 11.2 ± 3.2 h·wk⁻¹) and had at least 4 yr of competition experience. These subjects were chosen because they were accustomed to high-intensity exercise and were familiar with laboratory testing. The study was conducted with the ethical approval of the Human Subject Research Committee of the University of Lethbridge; before the trials, all subjects were informed of the nature of the investigation, after which they gave written informed consent.

**Experimental Design**

Subjects visited the laboratory four times. During the first visit, anthropometric measurements (stature, body mass, and body fat percentage) and a familiarization session of the main trial (10 × 6-s sprints with 24 s of recovery between sprints to become familiar with the equipment and procedures until fully confident of producing an all-out effort from a stationary start) were performed. Within 1 wk of the familiarization session, subjects performed three sprint trials (4 d apart), in a random order, on a stationary cycle ergometer with resistance set at 0.095 kg·kg⁻¹ body mass (22). In one trial, subjects were informed that they would perform ten 6-s cycling sprints (with 24-s recovery between every sprint) and then completed ten sprints (control trial, CL). In a second trial, subjects were told that they would only perform five 6-s cycling sprints, but after performing the fifth sprint, they were asked to perform another five sprints, so that the total number of sprints was 10 (deception trial, DC). In this trial, the maximal allowed period of recovery (24 s) was maintained between the fifth and the sixth sprints. In a third trial, subjects were not told how many sprints they would be performing but were stopped after 10 sprints (unknown trial, UN). Therefore, the total number of sprints was the same for all three trials, but subjects were informed differently about the expected sprint number before the start of each trial. The same investigators were present during all trials, and the same level of encouragement was given to subjects in every trial. Subjects performed the three trials at the same time of day so that effects of circadian rhythm on physiological and psychological functions were negated, and at least 4 d of rest were allowed between each trial. Subjects were asked to consume no food or beverages (other than water) 2 h before the test. They were instructed to refrain from any form of intense physical exercise for the 2 d before the
test. The laboratory was air-conditioned, and the temperature was kept constant at 19°C–21°C.

**Ergometer**

A modified, friction-loaded, cycle ergometer (Monark Ergometric 874 E, Varberg, Sweden), interfaced with a microcomputer, was used to collect the data and to calculate the power generated on each flywheel revolution and the work performed during each sprint. An optical sensor measured the velocity of the flywheel at a rate of 128 pulses per flywheel revolution. The instantaneous power generated during the sprints was corrected for the changes in kinetic energy of the flywheel (15), and results were averaged during 0.5-s intervals. Work done was then totaled for the sprint, which was also expressed relative to time to calculate power. By taking into account the work done in accelerating the flywheel during the initial seconds of the sprint, peak power was always reached before peak speed. Peak power output (W) and mechanical work (kJ) were calculated for each sprint. The total work and percentage decrement over the repeated sprints for work ($W_{\text{dec}}$) and power ($P_{\text{dec}}$) were also calculated (8).

The handlebars and seat were adjusted to the subjects’ characteristics, and feet were secured to the pedals using toe clips. Visual feedback of power output and pedaling rate were available to the subjects during every sprint. Starting pedal position was initiated with the dominant leg and the crank arm located 90° forward to the vertical axis. Subjects remained seated during all sprints. They were instructed to cycle as fast as possible from the start and were strongly encouraged throughout each sprint to promote a maximal effort. We acknowledge that it is difficult to “standardize” encouragement, but we used extra care to ensure that encouragement was uniform across every subject and every trial. The same experimenters were present during every trial.

**Perceptual and Physiological Responses to Exercise**

**RPE.** As an index of overall feeling of subjective perceived exertion, the RPE was assessed with the Borg 15-point category scale (10). The scale was anchored so that 6 represented the resting state and 20 corresponded to maximal exertion. Subjects were thoroughly instructed in the use of the RPE scale before performing every trial. The scale was located in full view of the subjects throughout testing, and subjects pointed to the perceptual ratings that best reflected their conscious effort sensations. RPE readings were taken at rest and immediately after every sprint.

**Surface EMG acquisition and analysis.** The EMG signals of four muscles (vastus lateralis, rectus femoris, biceps femoris, and gastrocnemius medialis) were recorded from the dominant lower limb via surface electrodes (DE-2.1 single differential electrodes; DelSys, Inc., Boston, MA). Recording electrodes were fixed longitudinally (aligned parallel to the underlying muscle fiber direction) over the muscle belly. The reference electrode was fixed over an electrically neutral site (epicondyle of femur). Electrode site preparation was thoroughly performed before the beginning of every test (skin impedance < 2 kΩ), and electrode location was marked with a waterproof felt-tip pen to ensure reliable electrode replacement in subsequent testing sessions. To ensure low levels of movement artifact, electrode cables were fastened to the subjects’ limb with medical adhesive tape and wrapped in elastic bandage. The raw EMG signal was preamplified and filtered (bandwidth frequency = 20–450 Hz, common mode rejection ratio = 92 dB, gain = 1000). A 50-Hz line filter was applied to the EMG data to prevent interference from electrical sources. The filtered EMG signal was sampled at 2 kHz (Bagnoli EMG System, DelSys, Inc.). During postprocessing, the onset and offset of activation of all EMG bursts in every sprint were detected using a constant electrical threshold of 0.2 mV (5). Each EMG burst was visibly inspected to verify the timing identified by the computer.

Muscle activity was considered as the integrated EMG (iEMG) of the signal between the onset and offset of activation of every burst. Instead of using the iEMG results of individual muscles, the EMG integrals of every muscle were added together, and a new sum–iEMG parameter was used to represent the general behavior of muscle electrical activity during sprints (see Billaut and Smith (7) for details). Sum–iEMG is reported as raw data (V/s) and as a percent of the initial sprint value.

**Statistical Analysis**

Analyses were performed using Statistica 5.5 for Windows (Statistica; Statsoft, Inc., Tulsa, OK). Tests for homogeneity of variances (Levene test) were performed to ensure the normality of the population for every dependent variable. With the assumption of normality confirmed, one-way ANOVA with repeated measures for trial were used to compare the following variables between trials: first sprint power, work and sum–iEMG, cumulated work and cumulated sum–iEMG over the first five sprints (1–5) and during the entire RSE, as well as $P_{\text{dec}}$ and $W_{\text{dec}}$. Two-way repeated-measures ANOVA (trial × sprint) were used to compare the following dependent variables between trials across repetitions: power, work, sum–iEMG, and RPE. Tukey HSD *post hoc* analyses were used to locate differences among pairs of means when ANOVA revealed significant F-ratio for main or interactive effects. The level of significance was set at 0.05. Data are reported as mean ± SD.

**RESULTS**

**Mechanical data.** Power and work values recorded for every sprint are displayed in Figure 1. During the first sprint of the series, power and work were significantly higher in the DC trial (20.5 ± 0.6 W kg$^{-1}$ and 83.3 ± 7.5 J kg$^{-1}$, respectively) than both the CL (19.7 ± 0.9 W kg$^{-1}$ and 78.6 ± 8.2 J kg$^{-1}$, respectively) and UN trials (19.5 ± 0.5 W kg$^{-1}$ and 77.9 ± 6.8 J kg$^{-1}$, respectively). During the first five
sprints (sprints 1–5), the cumulated work was also significantly greater in the DC (396.3 ± 23.1 J·kg⁻¹) than in the CL (376.4 ± 20.2 J·kg⁻¹) and UN trials (365.3 ± 19.8 J·kg⁻¹; Fig. 2A). When the 10 sprints were considered, the main effect of the trial was significant for power, with the DC trial exhibiting higher mean values (18.5 ± 1.4 W·kg⁻¹) than both the CL (18.0 ± 1.2 W·kg⁻¹) and UN trials (17.8 ± 0.9 W·kg⁻¹). No significant difference was noted between the CL and UN trials for the above parameters. On the basis of work values, we observed that the work cumulated during the 10 sprints was significantly lower in the UN (682.6 ± 21.5 J·kg⁻¹) compared with both the CL (702.2 ± 25.3 J·kg⁻¹) and DC trials (707.8 ± 28.9 J·kg⁻¹; Fig. 2B). No significant difference was noted between the CL and DC trials. Furthermore, an interaction effect (trial × sprint) was noted for both power and work. The post hoc results were as follows: in the CL and DC trials, power decreased significantly from sprint 4 (5.1% and 4.9%, respectively) compared with the first sprint of the series. In the UN trial, however, the power decrement occurred earlier, from sprint 3 (5.2%). Similarly, work decreased significantly from sprint 4 in the CL (6.4%) and DC (6.7%) trials and from sprint 3 (6.8%) in the UN trial. $P_{dec}$ did not vary between trials (range = 8.4%–10.7%), whereas $W_{dec}$ was greater in the DC (14.9%) than in both the CL and UN trials (10.6% and 11.7%, respectively).

**EMG activity.** The changes in sum–iEMG are presented in Figure 1C. During the first sprint of the series, sum–iEMG was significantly higher in the DC trial (0.34 ± 0.04 V·s) than in both the CL (0.32 ± 0.03 V·s) and UN trials (0.31 ± 0.02 V·s). The cumulative value of sum–iEMG during the first five sprints (sprints 1–5) was also significantly greater in the DC (1.65 ± 0.45 V·s) than in the CL (1.53 ± 0.37 V·s) and UN trials (1.48 ± 0.36 V·s; Fig. 3A). However, there was no significant difference in this variable between the trials when the 10 sprints were considered (Fig. 3B). No significant difference was noted between the CL and UN trials for the above parameters. A significant interaction effect (trial × sprint) was also observed, with post hoc results detailed as follows: sum–iEMG decreased

![Figure 1](http://www.acsm-msse.org)

**FIGURE 1**—Peak power, mechanical work, and sum–iEMG for each sprint in the three trials. Main effect of trial for initial sprint performance: $P < 0.05$. *significant difference from both CL and UN trials. Main effect of sprint for power, work, and sum–iEMG: $P < 0.05$. Main effect of trial for power only: $P < 0.05$. Interaction effect for power, work, and sum–iEMG: $P < 0.05$. †significant difference with sprint 1 for the CL and DC trials; §significant difference with sprint 1 for the UN trial.

![Figure 2](http://www.acsm-msse.org)

**FIGURE 2**—Cumulative value of mechanical work calculated during the first five sprints (sprints 1–5; A) and during the entire protocol (sprints 1–10; B) in the three trials. *Significant difference from the CL and UN trials. †Significant difference from the CL and DC trials.
significantly from sprint 3 in the UN trial (10.4%) and from sprint 5 in the CL (10.3%) and DC trials (6.2%; Fig. 1C).

**RPE.** The RPE (all trials compounded) progressed from the baseline value of 7 to 19 after sprint 10 (Fig. 4). No significant main effect of the trial or interaction effect (trial × sprint) was observed for RPE.

**DISCUSSION**

Studies of pacing strategy have relied significantly on the measurement of EMG activity to examine the neural drive subjects allocate to a task (14,16,25). In addition, changes in EMG activity (muscle activation) have been shown to be tightly related to mechanical output during RSE (7,18) and, therefore, may be used to indicate the presence of a centrally mediated global pacing strategy. In this study, we examined for the first time the influence of knowledge of the end point of exercise on selected physiological responses and pacing during short repeated sprints separated by short (i.e., ≤30 s) recovery intervals in athletes. The major findings were that the anticipation of performing fewer sprints (i.e., 5 instead of 10) resulted in enhanced muscle recruitment (as evidenced by higher EMG activity in the lower limb), which was associated, in turn, with a higher mechanical output during the first five sprints. On the other hand, when subjects were not informed of the actual sprint number, they recruited less muscle and, hence, exhibited a lower mechanical output profile, despite similar levels of encouragement and similar perceived exertion.

**Initial sprint performance.** When athletes were deceived so as to believe that they only had to perform five sprints (i.e., the DC trial), they recruited more skeletal muscle fibers (as indicated by greater sum–iEMG) and produced higher power and work than in the two other conditions (Figs. 1 and 3) despite similar strong encouragement. This suggests that, in the DC trial, there was a greater subconscious neural drive (probably because of a perceived fewer number of sprints) from the beginning of the exercise, which led to greater muscle recruitment and thus greater mechanical output. This greater mechanical output would have demanded a greater supply of energy, predominantly from the anaerobic energy stores (13). Although we were unable to perform muscle biopsies in the current study, our finding suggests that factors other than purely the metabolic reserves and the maximum rate at which such reserves can be depleted contribute to the mechanical output of the first repetition of a series of sprints. In particular, these results suggest that the control of the initial sprint performance was, at least in part, regulated in anticipation of the exercise. This anticipation would have been based on the knowledge of the number of sprints to be performed, which may be considered in the current study as the end point of exercise (19, 24, 25).

**First half of the RSE.** An additional novel finding of the current study was that the differences in neuromuscular and mechanical responses between trials during the initial sprint remained evident during the first five sprints (Figs. 1–3). Indeed, the cumulated EMG activity and mechanical work were significantly higher in the DC trial compared with both the CL and UN trials during the first half of the series. This demonstrates that, when subjects expect to perform fewer sprints, they consciously or subconsciously recruit more muscle to produce a higher performance. This also indicates the selection of a pacing strategy, which is established before exercise and which depends on the knowledge of the required

**FIGURE 3—Cumulative value of sum–iEMG calculated during the first five sprints (sprints 1–5; A) and during the entire protocol (sprints 1–10; B) in the three trials. *Significant difference from the CL and UN trials.**

**FIGURE 4—RPE at baseline (rest) and during the sprints in the three trials. Main effect of sprint: \( P < 0.05 \). *Significant difference from baseline. Main effect of trial: NS. Interaction effect: NS.**
sprint number. Indeed, it is likely that the variation in the pacing pattern was due to the lack of, or inappropriate, knowledge of the exercise and that the pacing pattern would have been set by selecting the work rate according to the end point of exercise (19,24,25). A likely explanation for the higher mechanical output in the DC trial is therefore psychological. Subjects believed that the DC trial required only five sprints, and such knowledge motivated them to exert further effort compared with the CL trial, which required five more sprints.

It is interesting that, despite consisting of a similar total exercise duration, it has previously been reported that a pacing strategy is not apparent during 30 s of maximal continuous sprinting (2,30). This suggests that the mechanical output profiles during such tasks might not only be the immediate consequence of peripheral, metabolic perturbations, as previously thought (23), but may also be influenced by the neural drive. In fact, the presence of a neural component to pacing has already been recognized as a factor influencing mechanical output during supramaximal exercise. For example, the observed similarity in pacing patterns during Wingate tests of varied durations led to the hypothesis that the pacing strategy during such exercise is regulated in anticipation and is independent on the total work to be performed (2). While exploring the self-chosen pacing strategies during ten 60-m running sprints separated by 4 min of rest on level, uphill or downhill surfaces, Baron et al. (4) demonstrated that the level of mechanical stress imposed on muscles, originating from feedback from musculoskeletal mechanoreceptors, also influences the pacing strategy. Therefore, in addition to the exercise duration and the level of eccentric muscle loading, our results add to the current understanding by revealing that the knowledge of the sprint number (acting as the end point of exercise in the current conditions) also modulates muscle recruitment and performance during supramaximal exercise. Moreover, the time ratio between recovery and exercise phases—that is, the recovery pattern—can also be considered as a key component of the pacing strategy because mechanical output is altered during continuous (30) but not intermittent (i.e., the current DC trial) efforts lasting 30 s.

Second half of the RSE. While the first half of the RSE exhibited the highest mechanical output, both power and work fell quickly between the fifth and the sixth sprints in the DC trial, after the deception was revealed, and subjects were asked to perform an additional five sprints. Thereafter, the mechanical scores remained low, and there was no significant difference between trials, although we noted a tendency for lower work in sprints 6–10 in the DC trial (−5%, $P > 0.05$). This raises an interesting question about the underlying purpose of the pacing strategy observed in the current study. If, as has been previously suggested, the conscious or subconscious regulation of the initial mechanical output is designed to reduce premature muscle fatigue, we would have expected performance of the last five sprints to be lower in the DC trial. In contrast, the adoption of a pacing strategy in the CL trial did not seem to limit fatigue or to “protect” performance. In fact, we failed to observe a significant difference in work between trials during sprints 6–10. It is possible that our protocol was too short (i.e., did not require the subjects to perform enough sprints) for the pacing strategy to become truly efficient and to attenuate further development of peripheral, locomotor muscle fatigue in the final repetitions. Therefore, our results indicate that, although prior knowledge of the sprint number influenced performance during early sprints, it did not significantly affect the rate of fatigue development in subsequent sprint repetitions. It may thus be hypothesized that this pacing strategy was not the result of an “in-task” conscious adjustment but rather the result of a central control planned in advance of the task and based on the knowledge of the end point of exercise. That being said, the total sprinting duration in the current study was 60 s ($10 \times 6$-s sprints), which is well beyond the 30- and 36-s sprint used in the studies by Wittkink et al. (30) and Ansley et al. (2), in which performance was significantly downregulated. The major difference between these studies was that the current study used an intermittent protocol that allowed subjects to recover (even partially) between sprints. This suggests that the consequences of pacing for performance are different during continuous versus intermittent supramaximal exercise.

Entire RSE. We also observed that $W_{dec}$ was greater in the DC trial compared with that in the CL trial. Although it is tempting to attribute this greater decrement to the subjects being deceived as to the final sprint number, some methodological considerations should be discussed. Changes in mechanical output during RSE are critically dependent on peripheral metabolic perturbations in the active tissues (6). Indeed, several studies of repeated-sprint ability have demonstrated the influence of the initial mechanical output on the performance decrement over subsequent sprints (8,9,13,18). However, a closer analysis of the current data showed that none of the observed effects of deception during the first sprint persisted during the last sprints (Fig. 1). Therefore, in contrast to a pacing hypothesis, it seems that the greater $W_{dec}$ in the DC trial can largely be attributed to a consequence of the higher initial work.

Although the initial sprint performance may have influenced some of our results, it did not explain the profile of muscle recruitment and mechanical output in every trial. In fact, when subjects were not aware of the sprint number (i.e., the UN trial), the decline in power and work occurred earlier during the series (Fig. 1), and the work cumulated during the 10 sprints was significantly lower than that in the CL trial (Fig. 2B), without any significant difference in the initial sprint work. This strategy is in good agreement with the well-accepted hypothesis that, when a task of unknown duration is performed, subjects will be more economical in their use of physiological resources, presumably to maintain a reserve in anticipation of a longer exercise bout and greater physiological demand (19,24–26). Our EMG data (Fig. 1C) support this hypothesis because muscle
PACING IN INTERMITTENT SPRINTS

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