A 30-Min Rest Protocol Does Not Affect *W′*, Critical Power, and Systemic Response

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ABSTRACT

TRISKA, C., J. HOPKER, B. WESSNER, A. REIF, H. TSCHAN, and B. KARSTEN. A 30-Min Rest Protocol Does Not Affect *W′*, Critical Power, and Systemic Response. *Med. Sci. Sports Exerc.*, Vol. 53, No. 2, pp. 404–412, 2021. **Purpose:** This study aimed to assess and compare the systemic response of oxygen uptake kinetics and muscle deoxygenation between a 30-min rest protocol and a multivisit protocol on the parameters of the power–duration relationship (i.e., critical power [CP] and *W′*). **Methods:** Nine endurance-trained triathletes reported to the laboratory on five occasions: a preliminary graded exercise test and a familiarization, a 30-min single-visit protocol (time trials of 10, 5, and 2 min in that order interspersed with 30 min rest), and a multivisit protocol (time trials of 10, 5, and 2 min in randomized order interspersed by >24 h rest). Heart rate (HR) was recorded continuously, respiratory gases were measured breath by breath, and deoxygenation was recorded at 10 Hz using near-infrared spectroscopy (NIRS) during all tests. Blood lactate (BLa^-) concentration was measured before all time trials. Maximal HR (HR_{max}), oxygen uptake ($\dot{V}O_2$) during the first 2 min ($\dot{V}O_{2onset}$), mean response time, end-exercise $\dot{V}O_2$ ($\dot{V}O_{2peak}$), $\dot{V}O_2$ amplitude (ampl$\dot{V}O_2$), O_2 deficit, NIRS τ, amplitude (amplNIRS), and time delay were assessed. To compare the two protocols and to assess the differences in *W′* and CP, a paired sample *t*-test was used as well as a two-way ANOVA to assess the differences between trials and/or protocols, including trial–protocol interactions. **Results:** No significant differences, and trivial effect sizes, were found for *W′* and CP between protocols ($P = 0.106–0.114$, $d < 0.01–0.08$). Furthermore, no significant differences between protocols were found for all parameters, except for [BLa^-]. Significant differences between trials were found for $\dot{V}O_{2ampl}$, $\dot{V}O_{2onset}$, NIRS τ, amplNIRS, [BLa^-], and HR_{max}. **Conclusion:** Results suggest that *W′* and CP can be determined using the 30-min rest protocol without confounding effects of previous severe exercise compared with the multivisit protocol. **Key Words:** ENDURANCE, POWER–DURATION RELATIONSHIP, SINGLE-VISIT PROTOCOL, PRIMING EFFECT

Determining the parameters of the power–duration relationship (i.e., critical power [CP] and its related maximum work above CP [*W′*]) traditionally has required multiple constant-power trials until volitional exhaustion (TTE), interspersed by 24–48 h recovery (1). During the last decade, several research approaches consequently focused on (a) making the test protocol more time efficient (2–5) and (b) enhancing the ecological validity of the exhaustive trials (6,7). For example, compared with the multiday protocol, using a single-visit protocol in cycling and running produced valid results for CP, but not for *W′* when using a 30-min and 60-min interexhaustive trial recovery period (2,4,6). Furthermore, compared with the traditional TTE approach, time trial (TT) has been suggested to determine *W′* and CP with notably lower standard errors (6). Importantly, TT has been shown to produce reliable results for *W′* and CP (7) while also providing a higher level of ecological validity (8). Another attempt to shorten the testing protocol was the 3-min all-out test (5). This approach has been suggested to be valid (9) and sensitive to training adaptations (10); however, its validity has been questioned particularly in elite athletes (11–14).

To date, a continuously debated question is whether *W′* determined using a single-visit protocol is affected by previous severe exercise (2–4,15). Previous research has shown this not to be the case for CP (2,4,6); however, this demonstration is still outstanding for *W′*. In fact, research comparing *W′* values between conditions (laboratory vs field) or protocol (TTE vs TT and/or a shortened recovery protocol) has consistently provided low levels of agreement and high variability for *W′* (e.g., 3,4,6,16,17). Only by matching work or time between protocols recent studies (18,19) were able to demonstrate consistent values of *W′* and its equivalence in running, *D′*.

There is a limited number of studies including measures of oxygen uptake ($\dot{V}O_2$) kinetics and muscle deoxygenation (HHb) kinetics when assessing different *W′*/CP testing modalities (6,18).

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However, these works did not consider different resting protocols. Importantly, using the promising 30-min intertrial recovery protocol, research that considers systemic responses, such as $\dot{V}O_2$ kinetics, HHb kinetics, or blood lactate (BLa^-) concentration, is currently lacking. It has been speculated that either an incomplete reconstitution of W' or a confounding systemic response negatively affects performance of subsequent maximum effort trials or, alternatively, that a "priming" effect (i.e., faster $\dot{V}O_2$ on-kinetics or faster HHb kinetics and therefore, a lesser breakdown of anaerobic sources) of a previous maximum severe intensity exercise might create a performance-enhancing effect (for reviews, see 20,21). A "priming" effect after an intense exercise bout >CP has been shown to increase performance and to alter $\dot{V}O_2$ kinetics in a subsequent trial (e.g., 22–28). This effect (i.e., faster $\dot{V}O_2$ on-kinetics) was also present after a severe intensity bout and 20 min of "unloaded" cycling, but this previous trial did not alter deoxygenation kinetics (22). Similarly, it was indicated that the effect of an elevated $\dot{V}O_2$ amplitude is persistent for a minimum of 45 min (25). However, results for "priming" and parameters of HHb do not indicate overall faster muscle deoxygenation kinetics (29–31). For example, no effect was demonstrated for CP, but W' was increased after "priming" using a 10-min rest period between a "priming" bout and a predictive TTE trial when compared with "nonprimed" condition (24).

Interestingly, it has been demonstrated that W' nearly fully reconstitutes after about 20 min (28). The same researchers also showed that $\dot{V}O_2$ returns to baseline levels notably faster (after approximately 5 min) with [BLa^-] demonstrating the slowest off-kinetics of approximately 1 h (28). After 30 min passive rest, it is suggested that W' is fully reconstituted and $\dot{V}O_2$ has returned to baseline levels; however, BLa^- removal is only about 75% (28). Although elevated [BLa^-] concentrations <5 $mmol \cdot L^{-1}$ are associated with an increase in performance in subsequent performance trials, concentrations >5 $mmol \cdot L^{-1}$ have demonstrated a contrasting effect of performance deterioration (27). Therefore, it is currently unclear how these factors (BLa^-, $\dot{V}O_2$, and HHb) affect the determination of W' and CP when using a 30-min intertrial recovery protocol. As a consequence, the aim of the present study was to compare and assess the effect on physiological and performance parameters between two resting protocols (i.e., single-visit vs multivisit protocol). Based on the above, we hypothesized primed oxygen kinetics, elevated [BLa^-], and faster HHb but nonsignificant differences and a high agreement for W' and CP between the protocols.

METHODS

Participants. Nine male endurance-trained triathletes (mean ± SD; age = 27.7 ± 4.3 yr, body mass = 75.6 ± 5.6 kg, body height = 1.81 ± 0.04 m, $\dot{V}O_{2peak}$ = 60.0 ± 6.5 $mL \cdot kg^{-1} \cdot min^{-1}$, maximal minute power = 377 ± 35 W) volunteered to participate in this study. The number of required participants was determined using *a priori* statistical power analysis for previously determined reliability parameter estimates, with the smallest worthwhile effects assumed to be 9 W for CP and 1230 J for W' (7), an alpha level of $P < 0.05$, and a statistical power of >80%. Before the start and after being informed about all the benefits and risks of this study, participants had to fill in a health questionnaire and had to give written informed consent. All procedures were conducted in accordance with the Declaration of Helsinki and were approved by the host institution's local ethical committee (no. 00307).

Experimental design. Participants had to report to the laboratory on five occasions, separated by at least 72 h. For all tests, participants used their personal racing or TT bikes, which were mounted to a Cyclus2 ergometer (RBM electronics GmbH, Leipzig, Germany). The first visit was a graded exercise test (GXT), which was followed by participants performing one session of test familiarization (7). W' and CP were determined during visits 2 to 5, and they were performed in a randomized order: visit 2, a single-visit protocol using TT of 10, 5, and 2 min in this order interspersed by 30 min passive rest; visits 3–5 (multivisit protocol), a single TT of 10, 5, or 2 min. Tests were performed at the same time of the day (±1.5 h) in an air-conditioned controlled laboratory, where temperature and relative humidity were between 19°C and 22°C and 40% to 55%, respectively. Participants were advised to avoid strenuous exercise and alcohol intake in the 24 h before testing and to arrive at the laboratory in a fully hydrated and carbohydrate-rich state. Participants were also instructed to refrain from food and caffeine intake 3 h before testing. During all tests, participants were allowed to drink water *ad libidum*.

GXT and determination of $\dot{V}O_{2peak}$. Before the GXT, and in accordance with the recommendations of the manufacturer, the flow volume of a breath-by-breath portable gas analyzer (MetaMax 3B-R2; Cortex Medical GmbH, Leipzig, Germany) was calibrated using a 3-L syringe, and the analyzer was calibrated using gases of known concentration (15% O_2 and 5% CO_2, Cortex Medical GmbH). After a 3-min unloaded baseline cycling period, work rate was set to 100 W, and it consequently increased every minute by 20 W. During the GXT, $\dot{V}O_2$ was measured continuously, and the highest rolling 30-s average was considered as $\dot{V}O_{2peak}$ whereby maximal minute power was considered as the highest work rate before volitional exhaustion.

Time trials for the determination of W' and CP. Using TT for the determination of W' and CP has been demonstrated to be valid (7) and reliable (6). Moreover, compared with TTE and when considering the SEE, TT is suggested to provide a more accurate estimate of W' and CP (6,7). All maximal TT efforts were preceded by 3 min unloaded cycling, and participants were able to adopt their preferred pacing strategy and cadence throughout. Participants were advised to produce the highest mean power output (PO) during each trial and to fully "empty the tank" at the end of each TT. Participants were blinded for PO and cadence; however, they were not blinded for remaining time. This strategy was implemented by participants being able to change gear ratio using virtual gear changers mounted on the bicycle handlebars to mimic real-life TT. To

avoid reconstitution of W', PO during each TT was continuously required to exceed CP. After finishing all trials, it was assessed whether PO during each TT was continuously >CP, if not testing would have been repeated. This was not required for any participant. The PO of each TT was measured at 2 Hz and was subsequently computed into 10-s intervals.

$\dot{V}O_2$ and heart rate measurement. During all trials, gas exchange was measured breath by breath. Heart rate (HR) was measured continuously using an HR sensor (H7; Polar Electro Oy, Kempele, Finland) that was connected via Bluetooth™ to the gas analyzer.

Raw $\dot{V}O_2$ data were exported and analyzed for outliers. Data points lying more than 3 SD from the subsequent five breaths were removed and excluded from further analysis. Following this, filtered data were interpolated to 1-s averages using an Excel® spreadsheet. A least square nonlinear regression was used to model $\dot{V}O_2$ on-kinetics. According to recent work, the fitting window was set from the onset of exercise to 120 s (i.e., the minimum time across all trials) (6). A biexponential function to determine $\dot{V}O_2$ on-kinetics was not feasible because only a single trial in each condition was conducted, and consequently a monoexponential function was used:

$$\dot{V}O_2\,(t) = \dot{V}O_{2baseline} + A\left(1 - e^{-t/MRT}\right) \quad [1]$$

where $\dot{V}O_2(t)$ is $\dot{V}O_2$ at any time point ($L \cdot min^{-1}$), $\dot{V}O_{2baseline}$ represents the $\dot{V}O_2$ baseline ($L \cdot min^{-1}$), A is the amplitude of the response ($L \cdot min^{-1}$), t is any time point (s), and MRT is the mean response time (s).

The following parameters were analyzed: 1) baseline $\dot{V}O_2$, which was considered as the last minute of baseline cycling; 2) $\dot{V}O_{2peak}$, measured as the highest rolling 30-s average during each TT; 3) $\dot{V}O_2$ during the first 2 min of each trial; 4) MRT, considered as an indicator of the rapidity of $\dot{V}O_2$ rise; 5) $\dot{V}O_2$ amplitude; and 6) O_2 deficit at 2 min, calculated as the product of MRT and $\dot{V}O_2$.

Near-infrared spectroscopy measurement. Deoxygenated hemo- and myoglobin was detected using a portable near-infrared spectroscopy (NIRS) device (PortaMon; Artinis Medical Systems, Elst, Netherlands) during all TT at 10 Hz. Optodes were placed on the belly of the right vastus lateralis muscle on a line between the greater trochanter of the femur and the lateral epicondyle, 10 cm proximal to the knee joint (32). Δ[HHb] raw data from the second optode were exported at 1-s interval, then normalized, and subsequently analyzed using a monoexponential model including a time delay (TD) (23). The fitting window was set from the first data point, which was 1 SD above the baseline mean to the 60-s data point (22). The following equation was used to calculate for NIRS derived parameters:

$$\Delta[HHb](t) = \Delta[HHb]_{baseline} + A\left(1 - e^{-(t-TD)/\tau}\right) \quad [2]$$

where $\Delta[HHb](t)$ is the difference in deoxygenation at any time point (%), $\Delta[HHb]_{baseline}$ is the baseline (%), A is the amplitude of the response (%), t is any time point (s), TD is the time delay (s), and τ is the time constant (s).

BLa^- analysis. Blood samples (20 μL) to determine [La] were collected from the hyperemic earlobe directly before the start of each TT. Samples were immediately diluted in 1000 μL glucose solution and subsequently analyzed using an automated lactate analyzer (Biosen S_Line; EKF-diagnostic GmbH, Barleben, Germany).

Calculation of W' and CP. Two linear (LIN and INV) and one nonlinear model (HYP) were used to determine W' and CP. Although all three models are mathematically equivalent, these equations result in different parameter estimates. The following equations for LIN, INV, and HYP and in that order were used to calculate for W' and CP:

$$\text{work} = \text{CP} \times t + W' \quad [3]$$

$$\text{PO} = W'/t + \text{CP} \quad [4]$$

$$t = W'/(\text{PO} - \text{CP}) \quad [5]$$

In addition, SEE values in relative and absolute numbers were calculated for both parameter estimates, and the model that provided the smallest combined %SEE for both parameter estimates (i.e., sum of %SEE of W' and CP) was used for further analysis. For all participants. equation 4 (INV) resulted in the lowest combined %SEE, and consequently these values were used for further analyses.

Data analysis and statistics. After assessing data for normality using the Shapiro–Wilk test, a two-way repeated-measures ANOVA with trial (i.e., 10, 5, and 2 min) and protocol (i.e., single- and multivisit protocol) as model factors was used to analyze systemic response (i.e., $\dot{V}O_2$, ΔHHb, [BLa^-], and HR). Systemic responses between the TT and the GXT were assessed using a one-way repeated-measures ANOVA. Bonferroni *post hoc* procedures were used to follow-up significant main effects. Paired samples *t*-tests were used to assess differences in power data, CP, and W'. Pearson product moment correlation was used to indicate the strength of the relation between the protocols, and additionally, 95% limits of agreement (LoA) were applied to assess the validity of the single-visit estimates of W' and CP (33). Effect sizes are reported as partial eta squared (η_p^2) to provide an estimate of effect sizes from the ANOVA (small $\eta_p^2 = 0.01$, moderate $\eta_p^2 = 0.10$, large $\eta_p^2 = 0.25$). Effect sizes for the *t*-tests were calculated using the Cohen's d calculated as the quotient of mean differences and variance (small $d = 0.2$, moderate $d = 0.5$, large $d = 0.8$). Typical error, intraclass correlation coefficient (ICC), and coefficient of variation (CoV%) were calculated using a spreadsheet (34). Statistical significance was accepted as $P < 0.05$, and data are reported as mean ± SD. GraphPad Prism (version 6.00 for Mac; GraphPad Software, La Jolla CA, www.graphpad.com) was used to conduct all statistical analyses.

RESULTS

All data were normally distributed. Descriptive data (mean ± SD) are presented in Tables 1 and 2. Results of the ANOVA are

TABLE 1. Descriptive data of CP, *W′*, and TT.

	Single-Visit Protocol	Multivisit Protocol	Typical Error	CoV%	ICC	*P*	Effect Size
CP (W)	299 ± 30	304 ± 32	5 (3–10)^{T}	1.7 (1.1–3.2)	0.98 (0.92–1.00)	0.106	0.08^{T}
W′ (kJ)	15.1 ± 3.0	16.1 ± 3.4	1.2 (0.8–2.2)^{S}	7.1 (4.8–14.1)	0.90 (0.64–0.98)	0.114	0.00^{T}
10-min TT (W)	321 ± 32	326 ± 34	4 (3–9)^{T}	1.4 (0.9–2.7)	0.99 (0.95–1.00)	0.038	0.13^{T}
5-min TT (W)	354 ± 36	362 ± 36	3 (2–7)^{T}	0.9 (0.6–1.8)	0.99 (0.97–1.00)	0.001	0.35^{S}
2-min TT (W)	424 ± 46	436 ± 48	8 (5–15)^{T}	1.7 (1.2–3.3)	0.98 (0.92–1.00)	0.008	0.11^{T}

Data are presented as mean ± SD. Typical error, CoV%, and ICC are presented with 95% CI.
^{T}Trivial effect size; ^{S}Small effect size.
CP, critical power; *W′*, maximum work above CP.

presented in Table 3. Table 4 presents the typical error, CoV%, and ICC of the TT. The $\dot{V}O_2$ response of a representative participants is shown in Figure 1A–C, and the pacing profiles of the TT (mean ± SD) are depicted in Figure 1D–F.

GXT. Mean PO, HR_{max}, and $\dot{V}O_{2peak}$ during the GXT were 377 ± 35 W, 184 ± 15 bpm, and 60.0 ± 6.5 mL·kg^{-1}·min^{-1}.

Time trials. The mean PO values were 321 ± 32 and 326 ± 34 W for the 10-min TT, 354 ± 36 and 362 ± 36 W for the 5-min TT, and 424 ± 46 and 436 ± 48 W for the 2-min TT for the single- and multivisit protocol, respectively. PO values of the single TT were significantly different between the protocols demonstrating a trivial to small effect sizes (10-min TT: $P = 0.038$, $d = 0.13$, 95% CI = 0 to 10 W; 5-min TT: $P = 0.001$, $d = 0.35$, 95% CI = 5 to 12 W; 2-min TT: $P = 0.008$, $d = 0.11$, 95% CI = 4 to 21 W), but TT values were significantly correlated (10-min TT: $r = 0.984$; 5-min TT: $r = 0.991$; 2-min TT: $r = 0.975$; all at $P < 0.001$). During all TT, $\dot{V}O_{2max}$ obtained from the GXT was not significantly different from that of the TT ($F_{6,42} = 1.42$, $P = 0.230$, $\eta_p^2 = 0.17$). HR_{max}, however, was significantly different from that derived during the GXT in both 2-min TT ($P < 0.001$, $d = 0.17$, 95% CI = −12 to −1 bpm, and $P = 0.009$, $d = 0.23$, 95% CI = −14 to −3 bpm, for the single-visit and multivisit, respectively).

Parameters of the power–duration relationship. No significant differences and trivial effect sizes were found between the protocols for the parameters of the power–duration relationship ($t_8 = 1.78$, $P = 0.114$, $d < 0.01$, 95% CI = −1 to 10 W, and $t_8 = 1.82$, $P = 0.106$, $d = 0.08$, 95% CI = −0.3 to 2.2 kJ, for *W′* and CP, respectively) (Table 1). *W′* and CP were significantly correlated between the protocols ($r = 0.878$, $P = 0.002$ and $r = 0.976$, $P < 0.001$, for *W′* and CP, respectively) (Fig. 2A and B), and the mean bias and 95% LoA were −1.0 ± 1.7 J (95% LoA = −4.2 to 2.3 kJ) for *W′* and −4 ± 7 W (95% LoA = −19 to 10 W) for CP (Fig. 2B and C). The absolute SEE for CP was 6 ± 4 and 8 ± 6 W ($P = 0.158$, $d = 0.12$, 95% CI = −1 to 6 W) and for *W′* was 1.1 ± 0.7 and 1.5 ± 1.2 kJ ($P = 0.159$, $d < 0.01$, 95% CI = −0.2 to 1.1 kJ) for the single- and the multivisit, respectively. The relative SEE for CP was 1.9% ± 1.3% and 2.7% ± 2.1% ($P = 0.176$, $d = 0.34$, 95% CI = −0.4% to 1.9%) and for *W′* was 7.8 ± 5.9 and 10.0 ± 8.2 kJ ($P = 0.303$, $d = 0.06$, 95% CI = −2.4% to 6.8%) for the single- and the multivisit, respectively. The R^2 values of the power–duration relationships were 0.992 ± 0.011 and 0.984 ± 0.019 for the single- and the multivisit protocol, respectively.

$\dot{V}O_2$ kinetics ($n = 8$). One set of data was lost due to equipment failure; therefore, only eight data sets were analyzed. *Post hoc* procedures for trials revealed significant differences between 10 min, and all other trial durations, between 5- and 2-min durations for $\dot{V}O_2$ amplitude (all at $P < 0.001$), and across trial durations for O_2 consumed during the first 2 min ($P < 0.001$ for 10 vs 2 min and 5 vs 2 min, and $P = 0.006$ for 10 vs 5 min).

Muscle deoxygenation ($n = 8$). One set of data was lost due to equipment failure; therefore, only eight data sets were analyzed. *Post hoc* procedures for trials revealed significant differences between 10 min and all other trial durations for NIRS τ ($P = 0.003$ and $P < 0.001$ for 10 vs 5 min and 10 vs 2 min, respectively), and between 2 min and all other trial durations for NIRS primary amplitude ($P = 0.003$ and $P < 0.001$ for 2 vs 5 min and 2 vs 10 min, respectively.

TABLE 2. Physiological measures for the single-visit protocol and the multivisit protocol.

	Singe-Visit Protocol			Multivisit Protocol		
	10-Min TT	5-Min TT	2-Min TT	10-Min TT	5-Min TT	2-Min TT
Baseline $\dot{V}O_2$ (L·min^{-1})	1.79 ± 0.37	1.75 ± 0.46	1.78 ± 0.41	1.78 ± 0.36	1.79 ± 0.39	1.80 ± 0.42
VO_{2peak} (L·min^{-1})	4.55 ± 0.37	4.71 ± 0.45	4.58 ± 0.45	4.60 ± 0.44	4.63 ± 0.42	4.57 ± 0.58
O_2 consumed during the first 2 min (L)	6.91 ± 0.74	7.52 ± 0.80	8.14 ± 0.78	6.95 ± 0.59	7.21 ± 0.90	8.04 ± 1.07
%VO_{2peak}	101 ± 5	104 ± 3	101 ± 3	102 ± 6	102 ± 4	101 ± 7
MRT (s)	14 ± 6	15 ± 4	14 ± 3	16 ± 8	15 ± 4	15 ± 4
$\dot{V}O_2$ amplitude (L·min^{-1})	3.80 ± 0.49	4.15 ± 0.54	4.29 ± 0.36	3.84 ± 0.57	4.04 ± 0.49	4.37 ± 0.63
O_2 deficit at 2 min (L)	0.45 ± 0.23	0.53 ± 0.19	0.48 ± 0.09	0.55 ± 0.39	0.52 ± 0.18	0.56 ± 0.20
NIRS τ (s)	16 ± 8	11 ± 4	8 ± 3	15 ± 7	10 ± 4	10 ± 2
NIRS primary ampl (%)	58.7 ± 11.3	71.3 ± 12.7	82.1 ± 4.6	61.4 ± 14.3	64.6 ± 14.8	78.8 ± 8.6
NIRS TD (s)	3 ± 3	2 ± 1	1 ± 1	2 ± 1	2 ± 1	2 ± 1
[BLa^-] pre (mmol·L^{-1})	1.3 ± 0.4	4.4 ± 1.3	5.1 ± 1.4	1.2 ± 0.3	1.2 ± 0.2*	1.1 ± 0.2*
HR_{max} (bpm)	185 ± 13	183 ± 12	177 ± 13	183 ± 14	181 ± 14	175 ± 13

Data are presented as mean ± SD.
*Significantly different at $P < 0.05$ from the corresponding trial using Bonferroni *post hoc* tests.
$\dot{V}O_{2peak}$, peak oxygen uptake; ampl, amplitude of the response.

TABLE 3. Results of the two-way repeated-measures ANOVA and the effects sizes presented as partial eta squared.

	Main Effect of Trial			Main Effect of Protocol			Trial–Protocol Interaction		
	F	*P*	η_p^2	*F*	*P*	η_p^2	*F*	*P*	η_p^2
Baseline $\dot{V}O_2$	0.2	0.857	0.04^T	0.3	0.626	0.00^T	0.3	0.712	0.05^T
$\dot{V}O_{2peak}$	2.0	0.174	0.40^M	0.1	0.792	0.02^T	1.9	0.192	0.21^S
MRT	0.1	0.890	0.08^T	3.7	0.096	0.30^S	1.6	0.235	0.19^T
$\dot{V}O_2$ amplitude	64.5	<0.001	0.80^L	0.0	0.926	0.00^T	1.1	0.374	0.13^T
O_2 deficit at 2 min	0.1	0.889	0.06^T	2.3	0.172	0.24^S	1.7	0.227	0.19^T
O_2 consumed during the first 2 min	52.4	<0.001	0.91^L	1.2	0.314	0.14^T	1.5	0.257	0.18^T
NIRS τ	15.9	<0.001	0.62^M	0.0	0.961	0.00^T	0.4	0.668	0.06^T
NIRS TD	2.7	0.105	0.25^S	0.0	0.910	0.00^T	0.8	0.491	0.10^T
NIRS primary amplitude	24.2	<0.001	0.85^L	1.6	0.241	0.11^T	2.2	0.150	0.24^S
[BLa^-] pre	33.9	<0.001	0.82^L	67.9	<0.001	0.91^L	35.2	<0.001	0.83^L
HR_{max}	44.2	<0.001	0.86^L	2.5	0.151	0.38^S	0.3	0.779	0.03^T

[T]Trivial effect size; [S]Small effect size; [M]Moderate effect size; [L]Large effect size.

BLa^- concentration and HR. *Post hoc* procedures for trial revealed significant differences between 10 min and all other trials for [BLa^-] (both at $P < 0.001$) and between 2 min and all other trials for HR_{max} (both at $P < 0.002$). Moreover, *post hoc* procedures for single- vs multivisit protocols revealed significant differences for [BLa^-] between the 5-min and the 2-min trials (both at $P < 0.001$).

DISCUSSION

This study aimed to assess the effects of a 30-min passive rest on *W′* and CP compared with a >24-h rest using TT in a cohort of well-trained triathletes. Our results are in contrast to earlier studies assessing different resting protocols for the determination of the parameters of the power–duration relationship (2,4). Different to earlier work, this study applied ecologically valid and highly reliable TT under controlled laboratory conditions. The high reproducibility and low variation of these TT are thought to be the reason for a successful translation of multivisit protocols into single-visit protocols.

In a cohort of trained triathletes using TT durations between 2 and 10 min, our results demonstrate that a shorter rest of not more than 30 min does not affect estimates of *W′* and CP on a statistically significant level. Consequently, researchers and coaches can make an informed decision on the interchangeability between the single-visit and the multivisit protocols. Similar to *W′* and CP, variables of $\dot{V}O_2$ as well as HHb were not significantly different between protocols. Unsurprisingly, when applying the 30-min rest period, [BLa^-] did not return to baseline levels at the onset of a subsequent trial ($P < 0.001$). Although elevated BLa^- levels are suggested to alter subsequent performance (35,36), there was no such effect evident in the current study.

Time trials and the parameters of the power–duration relationship. Corresponding mean TT PO values across resting protocols were highly correlated, but significantly different between protocols, with effect sizes merely of a trivial to small order. Moreover, the difference between the protocol corresponding TT PO values was <3%, which is clearly within day-to-day variation for TT (34). In our cohort of well-trained and familiarized triathletes, the typical error for all TT between the protocols is interpreted as trivial (3–8 W), the CoV% was low (<2%), and the ICC values were high and close to 1 (ICC $r = 0.98–0.99$) (Table 1). Our results (i.e., typical error, CoV%, and ICC) for the laboratory-based TT are in accordance with previous literature assessing TT performance between 4 and 40 km under laboratory and field conditions (37–40). The pacing profiles of the TT indicate significant differences in particular at the onset of exercise (Fig. 1D–F). When applying the single-visit protocol, our participants adopted a slower start compared with the multivisit protocol, and PO was significantly lower in the middle portion of the 10-min and 5-min TT. This might be due to teleoanticipation, which is described as subconscious fatigue avoidance during the single-visit protocol (41). Despite these statistically significant differences in PO of the TT, *W′* and CP estimates were not statistically affected, and high levels of agreement and significant correlations were evident between protocols. Moreover, the typical error of CP is interpreted as trivial, CoV% is <2%, and ICC is $r > 0.90$. Our findings for CP are therefore consistent with recent works in cycling and running (2,4,7). However, in contrast to previous studies, *W′* was not significantly different ($P > 0.05$), but was significantly correlated ($r = 0.878$) between protocols, suggesting it could be used interchangeably. Moreover, the mean bias (1.0 kJ) and the 95% LoA of *W′* (±3.2 kJ) were acceptable between protocols. A further analysis revealed that the typical error and intraindividual variation of *W′* was notably smaller between protocols (1.5 kJ and 10.7%, respectively) compared with previous research, which has reported errors >2.5 kJ and 19%–40% between different testing modalities (i.e., TTE vs TT and single- vs multivisit protocol) (2–4,16,17). Indeed, it has been suggested that a higher variation in *W′* when using TTE trials resulted in meaningful differences between a single- and a multivisit protocol (4). Furthermore, several previous research studies have indicated that the use of TT results in notably lower variations of *W′*, especially when the prediction trials are of the same work or duration (6,7,18,19,42). Compared with earlier work, it can therefore be suggested that the application of TT and its related lower SEE compared with TTE resulted in different responses of *W′*. Consequently, in a cohort of well-trained athletes, applying TT between 2 and 10 min provided reproducible values for *W′* and CP across different testing protocols.

TABLE 4. Typical error, CoV%, and ICC of the 10-min, 5-min, and 2-min TT between protocols.

	10-Min TT			5-Min TT			2-Min TT		
	Typical Error	CoV%	ICC	Typical Error	CoV%	ICC	Typical Error	CoV%	ICC
Baseline $\dot{V}O_2$ ($L \cdot min^{-1}$)	0.1 (0.1 to 0.2)^S	4.9 (3.2 to 10.3)	0.96 (0.83 to 0.99)	0.1 (0.1 to 0.2)^S	5.9 (3.8 to 12.3)	0.97 (0.87 to 0.99)	0.1 (0.1 to 0.2)^S	6.1 (4.0 to 12.8)	0.95 (0.78 to 0.99)
$\dot{V}O_{2peak}$ ($L \cdot min^{-1}$)	0.1 (0.1 to 0.2)^S	2.2 (1.5 to 4.6)	0.96 (0.81 to 0.99)	0.1 (0.1 to 0.2)^S	2.1 (1.4 to 4.4)	0.97 (0.84 to 0.99)	0.2 (0.1 to 0.4)^S	4.2 (2.8 to 8.7)	0.91 (0.61 to 0.98)
MRT (s)	2.8 (1.8 to 5.7)^S	15.7 (10.1 to 34.6)	0.90 (0.57 to 0.98)	1.7 (1.1 to 3.5)^S	13.1 (8.5 to 28.5)	0.85 (0.43 to 0.97)	2.2 (1.5 to 4.5)^M	15.6 (10.0 to 34.2)	0.72 (0.11 to 0.94)
$\dot{V}O_2$ amplitude ($L \cdot min^{-1}$)	0.2 (0.1 to 0.3)^S	4.0 (2.7 to 8.4)	0.95 (0.77 to 0.99)	0.1 (0.1 to 0.2)^T	2.0 (1.3 to 4.1)	0.98 (0.92 to 1.00)	0.3 (0.2 to 0.6)^M	7.2 (4.7 to 15.2)	0.70 (0.07 to 0.93)
O_2 deficit at 2 min (L)	0.1 (0.1 to 0.3)^S	17.6 (11.3 to 39.1)	0.88 (0.52 to 0.98)	0.1 (0.0 to 0.1)^S	13.3 (8.6 to 28.9)	0.92 (0.67 to 0.98)	0.1 (0.1 to 0.3)^L	22.1 (14.1 to 50.1)	0.40 (–0.36 to 0.84)
O_2 consumed during the first 2 min (L)	0.4 (0.3 to 0.6)^M	6.0 (3.9 to 12.5)	0.73 (0.13 to 0.94)	0.2 (0.1 to 0.4)^S	2.6 (1.7 to 5.3)	0.97 (0.88 to 0.99)	0.4 (0.2 to 0.7)^S	4.6 (3.0 to 9.6)	0.90 (0.57 to 0.98)
NIRS τ (s)	7.3 (4.9 to 14.5)^L	47.7 (29.4 to 121.1)	0.06 (–0.63 to 0.70)	3.7 (2.4 to 7.5)^L	40.7 (25.3 to 100.4)	0.16 (–0.57 to 0.75)	1.8 (1.2 to 3.6)^M	23.8 (15.1 to 54.3)	0.52 (–0.23 to 0.88)
NIRS TD (s)	1.8 (1.2 to 3.7)^L	90.9 (53.3 to 272.7)	0.23 (–0.52 to 0.78)	0.8 (0.5 to 1.6)^L	153.3 (84.9 to 563.1)	0.55 (–0.18 to 0.89)	0.6 (0.4 to 1.2)^M	50.5 (31.0 to 129.7)	0.61 (–0.10 to 0.91)
NIRS primary amplitude (%)	6.4 (4.2 to 13.1)^S	12.7 (8.2 to 27.5)	0.82 (0.33 to 0.96)	7.8 (5.2 to 15.9)^M	12.7 (8.2 to 27.5)	0.75 (0.17 to 0.95)	5.3 (3.5 to 10.7)^M	6.9 (4.5 to 14.6)	0.48 (–0.27 to 0.87)
[BLa$^-$] pre (mmol·L^{-1})	0.3 (0.2 to 0.7)	29.3 (18.5 to 68.7)	–0.24 (–0.78 to 0.50)	0.9 (0.6 to 1.7)^L	19.9 (12.7 to 44.6)	0.17 (–0.56 to 0.75)	1.1 (0.7 to 2.2)	28.9 (18.3 to 67.6)	–0.09 (–0.71 to 0.61)
HR_{max} (bpm)	3 (2 to 6)^S	1.8 (1.2 to 3.5)	0.96 (0.82 to 0.99)	5 (3 to 9)^S	2.7 (1.8 to 5.2)	0.89 (0.60 to 0.98)	1 (1 to 3)^T	0.8 (0.5 to 1.4)	0.99 (0.97 to 1.00)

Data are presented with 95% CI.
^{T}Trivial effect size; ^{S}Small effect size; ^{M}Moderate effect size; ^{L}Large effect size.

$\dot{V}O_2$ and muscle deoxygenation. $\dot{V}O_{2peak}$ was attained at the end of all exercise bouts with no significant differences between trials and protocols. Baseline $\dot{V}O_2$ and MRT were also not significantly different between testing protocols and trials, although participants adopted a self-selected pacing strategy. Therefore, MRT indicates no "priming" effect after a 30-min rest period, and that the oxidative contribution at the onset of exercise and the O_2 deficit were not significantly different between protocols and trials. As a consequence, it can be neglected that there was a "priming" or O_2 deficit effect potentially influencing TT performance (43). The large CoV% in the O_2 deficit might be attributed to the differences in PO at the onset of the TT (Fig. 1D–F). Moreover, the fast start during the onset of the TT similarly sped-up $\dot{V}O_2$ and ATP turnover across trials and protocols (6). Nevertheless, our data suggest that a fast start influenced the $\dot{V}O_2$ amplitude as well as the $\dot{V}O_2$ response within the first 2 min between trials but not between protocols, which further supports no "priming" effect from repeated bouts in the single-visit protocol. These increases in $\dot{V}O_2$ amplitude between trials could be attributed to a higher initial PO during the shorter trials (i.e., 5 and 2 min). It was shown that previous severe "priming" exercise with a 10-min rest period between trials increased the $\dot{V}O_2$ amplitude and elevated end-exercise $\dot{V}O_2$ (24). W' and CP were, however, not significantly affected after the severe "priming" exercise bout. Most studies assessing the effect of "priming" focused on shorter rest periods such as 2 to 15 min (e.g., 23,24,26–28). Using previous severe exercise or heavy exercise, two studies used longer rest periods than 20 min to investigate $\dot{V}O_2$ kinetics (22,25). In contrast to the present work, significant differences in $\dot{V}O_2$ kinetics and performance were found. Although it is suggested that previous severe exercise elevates muscle perfusion and O_2 availability for subsequent trials for at least 45 min (25), our data do not support this notion as data of HHb as well as $\dot{V}O_2$ demonstrate nonsignificant differences between resting protocols. Although previous work stated that no "priming" effect exists after 60 min passive rest, the results of the present study suggest that as short as 30 min of passive rest is sufficient to minimize "priming" effects (6). Previous severe exercise had no beneficial or hindering effect on HHb in the present study, which is in accordance with $\dot{V}O_2$ data. Despite the suggestion of muscle perfusion and O_2 availability to be improved after a priming exercise (22,36), this was not confirmed by the present results.

Research work that previously assessed the effect of "priming" on muscle deoxygenation has predominantly been conducted in the heavy exercise intensity domain, with rest periods between 6 and 20 min (29–31). Results are ambiguous, with a study demonstrating a nonobligatory feature of a "priming" response (i.e., elevated baseline and slower MRT after "priming") (29), another study not finding any changes in HHb (30), and a third study demonstrating just an elevated NIRS amplitude (31). The present study, however, suggests that a fast start (i.e., higher PO within the first minutes) does alter τ and the primary amplitude, which interestingly was not shown in $\dot{V}O_2$ MRT. This might be due to the fact that

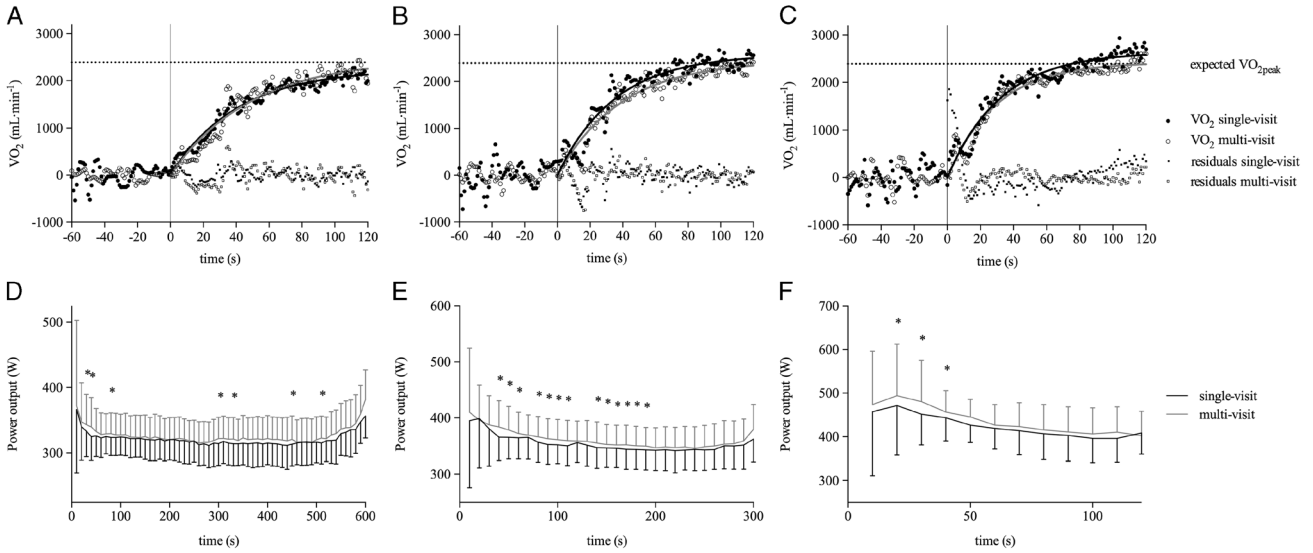

FIGURE 1—Baseline-corrected $\dot{V}O_2$ response and residuals of a representative subject during the first 120 s of the 10-min TT (A), 5-min TT (B), and 2-min TT (C). The horizontal dotted line indicates $\dot{V}O_{2peak}$ obtained from the GXT. Panels D–F represent the 10-s PO intervals of each TT. The error bars represent SD. *Significantly different at $P < 0.05$.

local muscle deoxygenation reacts more "sensitively" to a change in PO than pulmonary $\dot{V}O_2$. A high-intensity "priming" exercise with 30-min passive rest has, however, no effects on τ and the primary amplitude of muscle deoxygenation.

BLa^- concentration. Unsurprisingly, after about a 30-min passive rest, [BLa^-] was significant higher at the onset of a subsequent TT under the single-visit condition (~4–5 mmol·L^{-1}). Similar to results of $\dot{V}O_2$ kinetics and HHb, elevated [BLa^-] had no confounding effect on subsequent performance. After different modes of ~4 min "priming" exercise, performance did not significantly change despite [BLa^-] levels reaching between 3 and 8 mmol·L^{-1} (44). This is consistent with other works who found no increased TT performance and no altered $\dot{V}O_2$ kinetics after "priming" in the heavy domain with [BLa^-] <4 mmol·L^{-1} (45). Similar concentrations were also evident in the present work, supporting the notion that a notably higher level of acidemia is required to speed up $\dot{V}O_2$ kinetics and to result in a subsequent performance improvement. Our results are in contrast to previous work that suggested that [Bla^-] levels of <5 mmol·L^{-1} are associated with an increase in subsequent performance (27). However, as previous exercise in the present study was rather

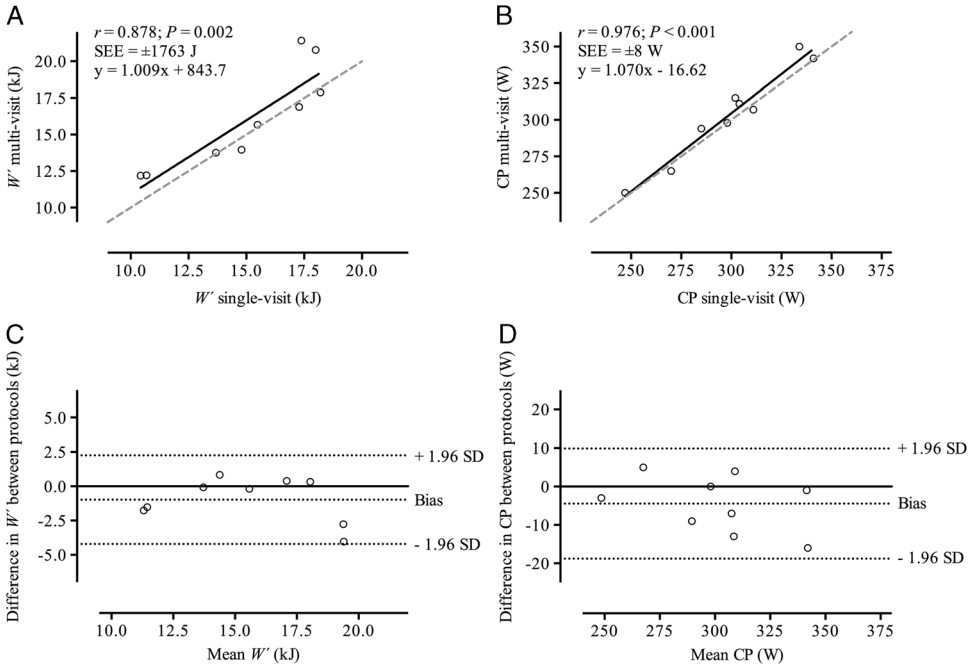

FIGURE 2—Relationship of CP (A) and *W′* (B) between the different rest protocols. The *gray dotted line* represents the line of identity and the *solid black line* represents the linear regression. Bland–Altman plots of the differences between the rest protocols of CP (C) and *W′* (D).

long (10 and/or 5 min) and as the intensity was located within the severe intensity exercise domain, any "priming" effect might have been balanced by residual decrements in performance. It is currently unclear if this is the case only in well-trained individuals or if this holds true across all fitness levels. Because of the lag in the rise of HR, this parameter demonstrated to be significantly different in the 2-min trials from all other trial durations, despite $\dot{V}O_{2peak}$ having been attained.

Limitations of the current study. This study merely compared two different rest protocols and performance trials at, above, and/or below CP were not included. Therefore, it is unclear if our CP values truly reflect a maximal metabolic steady state. Moreover, *W′* was not validated in a performance trial >CP. Furthermore, the expected statistical power of >80% was not achieved, and a *post hoc* analysis revealed a statistical power of 74%.

CONCLUSIONS

The results of the present study demonstrate no significant differences in *W′* and CP parameter estimates between the 30-min rest period and the >24-h rest period testing protocols, eliminating the need for using a time-disruptive multivisit protocol. Our results further suggest that *W′* and CP can be determined accurately (i.e., with a low SEE) within less than 90 min with no confounding effects compared with the multivisit protocol in well-trained athletes using TT durations between 2 and 10 min.

The results of the study are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation. No funding was received for this study. The authors have no conflicts of interest to disclose in relation to this study. The results of the present study do not constitute endorsement by the American College of Sports Medicine. Parts of this manuscript were accepted to be presented at the 2020 American College of Sports Medicine conference.

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