Intensity-dependent tolerance to exercise after attaining $\dot{V}O_2\text{max}$ in humans

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The tolerable duration of high-intensity, constant-load cycle ergometry is a hyperbolic function of power, with an asymptote termed critical power (CP) and a curvature constant ($W^\ast$) with units of work. It has been suggested that continued exercise after exhaustion may only be performed below CP, where predominantly aerobic energy transfer can occur and $W^\ast$ can be partially replenished. To test this hypothesis, six volunteers each performed cycle-ergometer exercise with breath-by-breath determination of ventilatory and pulmonary gas exchange variables. Initially, four exercise tests to exhaustion were made: I) a ramp-incremental and 2) three high-intensity constant-load bouts at different work rates, to estimate lactate ($\dot{L}_\text{L}$) and CP thresholds, $W^\ast$, and maximum oxygen uptake ($\dot{V}O_2\text{max}$). Subsequently, subjects cycled to the limit of tolerance (for $\sim360$ s) on three occasions, each followed by a work rate reduction to I) $110\%$ CP, 2) $90\%$ CP, and 3) $80\%$ $\dot{L}_\text{L}$, for a 20-min target. W$\ast$ averaged $20.9 \pm 2.35$ kJ or $246 \pm 30$ J/kg. After initial fatigue, $110\%$ CP was tolerated for only $30 \pm 12$ s. Each subject completed 20 min at 80% $\dot{L}_\text{L}$, but only two sustained 20 min at 90% CP; the remaining four subjects fatigued at $577 \pm 306$ s, with oxygen consumption at $89 \pm 8\% \dot{V}O_2\text{max}$. The results support the suggestion that replenishing $W^\ast$ after fatigue necessitates a sub-CP work rate. The variation in subjects’ responses during 90% CP was unexpected but consistent with mechanisms such as reduced CP consequence to prior high-intensity exercise, variation in lactate handling, and/or regional depletion of energy substrates, e.g., muscle glycogen.

Maximal oxygen uptake; fatigue; gas exchange dynamics

The tolerable duration of high-intensity, constant-load cycling, running, or swimming has been shown to be a hyperbolic function of the power (or velocity) of the exercise (9, 11, 12, 18, 22, 27). This power-duration relationship has an asymptote on the power axis termed the critical power (CP; or fatigue threshold). Although this relationship is difficult to determine at the extremes of muscular exercise, such as short (e.g., $<1-2$ min) all-out efforts greater than $\sim135\%$ of the work rate (WR) associated with maximal oxygen consumption ($\dot{V}O_2\text{max}$) (10) or for durations of $\geq60$ min (5), for durations between these limits, the hyperbolic relationship appears to provide a good characterization of the physiological response. The curvature constant of the hyperbola ($W^\ast$) is mathematically equivalent to a constant amount of work, i.e., the product of WR (above CP) and time (6, 19, 22), and has been suggested to be notionally equivalent to an energy store consisting of oxygen, high energy phosphates, and a source related to anaerobic glycolysis (17, 19). However, it may also relate to the buildup of fatigue-inducing metabolites or conditions to a critical level, such as low intramuscular pH and/or high intramuscular $P_i$ concentration ([P$\text{i}$]).

The CP threshold has also been shown to be a close correlate of the highest metabolic rate that is associated with pulmonary oxygen uptake ($\dot{V}O_2$), and acid-base status and blood lactate concentration ([La]) being maintained at a constant level (10, 22). As such, CP has been suggested to characterize the transition between, what Whipp (30) has termed, the heavy- and very heavy-intensity exercise domains. Above CP, in the very heavy-intensity domain, $\dot{V}O_2$ and [La] increase inexorably, terminating at fatigue with $\dot{V}O_2$ attaining its maximum (e.g., Refs. 21, 22) and depletion of (or attainment of the critical metabolite accumulation determining) $W^\ast$. Therefore, Fukuba and Whipp (6) have hypothesized that continued exercise after exhaustion (i.e., with $W^\ast$ depleted) would only be possible if the power output were reduced to a level below CP, where predominantly aerobic energy transfer can occur, i.e., permitting $W^\ast$ to be partially restored or replenished. Conversely, it is suggested that if at the point of fatigue (where fatigue is used in the sense of a level of fatigue

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that limits exercise tolerance) the power output were reduced to a level greater than CP, then continued exercise would not be possible because repletion of W would not occur in a domain where obligatory increases in VO₂, [La], and [Pi] were evident. We were interested in testing this hypothesis by determining whether, after induction of limiting fatigue with an initial intense exercise bout, it was possible to continue to exercise at a reduced WR if that rate was either above or below CP.

METHODS

Exercise tests. Six healthy male subjects (age 22 ± 2.7 yr; height 183 ± 1.6 cm; body mass 85 ± 5.2 kg; Table I) from the Medical School population volunteered to take part in the study after providing informed consent, as approved by the Local Research Ethics Committee (in accordance with the Declaration of Helsinki). After familiarization with the laboratory and procedures, each subject performed 7 exercise tests on an electromagnetically braked cycle ergometer (Excalibur Sport, Lode, Groningen, The Netherlands); each test was performed on a different day. Subjects initially performed an incremental ramp test (25 W/min, incremented at a rate of 5 W every 12 s) to the limit of tolerance. This allowed the VO₂ max and the lactate threshold (θ_L) to be estimated independently by two experimenters using standard noninvasive pulmonary gas exchange criteria (3, 32).

Exercise tests were then performed to exhaustion at three different high-intensity constant-load WRs, chosen to span a range of fatigue times of ~4–10 min. The subjects were required to maintain a pedal cadence between 60 and 80 rpm for all constant-load tests. The times to exhaustion were measured to the nearest second with a stopwatch and used to estimate each subject’s CP by using a three-point linear regression and extrapolation of the time^-1 vs. WR relationship. The slope of this relationship provides the W', and its WR intercept corresponds to CP (e.g., Refs. 9, 22). All tests were preceded by (3–4 min) and followed by (6 min) a 20-W control period. All constant-load tests were assigned in a randomized sequence.

Finally, each subject completed three subsequent tests consisting of an initial exhaustive WR (WR exh; targeted from the power-duration relationship to exhaust the subject in ~6 min), followed by, at the point of fatigue (when subjects were no longer able to maintain a pedal cadence of at least 60 rpm), an abrupt reduction in the WR to each of three intensities (for a 20-min target period; 1) 110% CP (very heavy), 2) 90% CP (heavy), and 3) 80% θ_L (moderate). Each subject performed the experiments requiring the recovery intensities of very heavy, heavy, and moderate in a randomized sequence and separated by at least 48 h. If the subject was unable to maintain the recovery exercise for the required target of 20 min, the fatigue time was recorded and the WR was reduced to 20 W for the remainder of the 20 min.

Equipment. The subjects breathed through a mouthpiece connected to a low-dead space (90 ml), low-resistance (<1.5 cmH₂O at 3 l/s) turbine volume transducer (Interface, Irvine, CA) for the measurement of inspiratory and expiratory volume. Respired gas was continuously sampled (at 1 ml/s) from the mouthpiece and analyzed by mass spectrometer (QP5000, Morgan Medical, Gillingham, UK) for the concentrations of oxygen, carbon dioxide, and nitrogen. Before each experiment, calibration was made from two precision-analyzed gas mixtures chosen to span the range of respired gas concentrations; this calibration was verified at the end of each experiment. The time delay between the gas concentrations and the volume signals was measured by passing a bolus of known gas mixture through the system by using a low dead-space solenoid valve (2). The electrical signal outputs were digitally converted every 20 ms and sampled by computer for the calculation of VO₂, carbon dioxide output (VCO₂), and minute ventilation (Ve) by using the algorithms of Beaver et al. (1, 2).

Analyses. Editing of data was performed from VO₂ in the time domain to exclude occasional aberrant breaths caused by swallowing, coughing, sighing, etc., which were considered not to reflect the underlying response, i.e., values greater than three standard deviations from the local mean were omitted (15). All breath-by-breath VO₂ responses were interpolated on a second-by-second basis and time averaged by using discrete 10-s time bins to produce a standard weighted response.

Times to fatigue were compared by using ANOVA and post hoc Neuman-Keuls tests. Differences between the six square-wave exhaustive exercise bouts were considered significant if P < 0.05. The dispersion about the mean is expressed as ± standard deviation.

RESULTS

Incremental ramp exercise. VO₂ increased linearly throughout the incremental ramp test (after a delay due to the VO₂ time constant), up to VO₂ max (averaging 4.5 ± 0.3 l/min). The slope of the VO₂ response as a function of WR (or gain) averaged 12.3 ± 0.6 ml·min⁻¹·W⁻¹, falling within the expected normal limits for cycle ergometer exercise (28). θ_L averaged 2.7 ± 0.3 l/min, which corresponded to 60.0 ± 4.6% of VO₂ max (Table I).

Constant-load tests to estimate CP. In the subsequent exhaustive constant-load tests, VO₂ max was not significantly different from that obtained during the incremental ramp test (4.5 ± 0.4 l/min). As expected, the time to fatigue was inversely correlated (P < 0.05)

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age, yr</th>
<th>Height, cm</th>
<th>Weight, kg</th>
<th>VO₂ max, l/min</th>
<th>θ_L, l/min</th>
<th>θ_L % VO₂ max</th>
<th>CP, % Δ*</th>
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</thead>
<tbody>
<tr>
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<td>182</td>
<td>85</td>
<td>5.1</td>
<td>2.9</td>
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<td>56.7</td>
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<tr>
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<td>27</td>
<td>183</td>
<td>95</td>
<td>4.2</td>
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<td>57.1</td>
<td>45.8</td>
</tr>
<tr>
<td>3</td>
<td>20</td>
<td>180</td>
<td>82</td>
<td>4.4</td>
<td>2.5</td>
<td>56.8</td>
<td>44.0</td>
</tr>
<tr>
<td>4</td>
<td>22</td>
<td>185</td>
<td>81</td>
<td>4.6</td>
<td>3.1</td>
<td>67.4</td>
<td>37.0</td>
</tr>
<tr>
<td>5</td>
<td>24</td>
<td>183</td>
<td>82</td>
<td>4.2</td>
<td>2.7</td>
<td>64.3</td>
<td>31.7</td>
</tr>
<tr>
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<td>21</td>
<td>183</td>
<td>85</td>
<td>4.5</td>
<td>2.6</td>
<td>57.8</td>
<td>35.9</td>
</tr>
<tr>
<td>Mean</td>
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<td>85</td>
<td>4.5</td>
<td>2.7</td>
<td>60.0</td>
<td>41.8</td>
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<tr>
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<td>1.6</td>
<td>5.2</td>
<td>0.3</td>
<td>0.3</td>
<td>4.6</td>
<td>9.0</td>
</tr>
</tbody>
</table>

VO₂ max, maximal oxygen uptake; θ_L, lactate threshold; CP, critical power. Δ* is defined as the difference between the work rate at θ_L and VO₂ max (see Ref. 21 for further discussion).

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to power output in all subjects. An example from subject 1 is shown in Fig. 1A. With the use of the time to fatigue at each of these WRs, a linear regression and extrapolation of the power vs. time\(^{-1}\) relationship was used to calculate CP (the abscissa intercept) and W\(^{\prime}\) (the slope of the linear fit) for each subject (an example from subject 1 is shown in Fig. 1B). The 95% confidence limits of the fit (dotted lines in Fig. 1B) were also calculated, and the fit was deemed acceptable if the confidence of CP estimation was within \(\pm 5\%\), which was the case in all but one instance. In this case, a fourth constant-load test was performed at another WR to improve the accuracy of CP estimation. The CP threshold occurred at \(246 \pm 24\) W, which was equivalent to \(41.8 \pm 9.0\%\) of the change in WR range (defined as the difference between the WRs at \(\theta_L\) and \(V_{O2\text{max}}\) taken from the ramp incremental test, c.f., Özyener et al. (21)) (Table 1). The W\(^{\prime}\) value averaged \(20.9 \pm 2.35\) kJ or \(246 \pm 30\) J/kg for the six subjects.

Intensity-dependent recovery tests. WR\(_{\text{exh}}\), predicted from the power-duration relationship, to fatigue the subject in \(\sim 6\) min averaged \(305 \pm 22\) W (Table 2). Subjects completed an average of \(348 \pm 40\) s at WR\(_{\text{exh}}\) before fatigue (there being no significant difference between each of the 3 bouts). The subsequent WRs for the three experimental conditions (110 and 90% CP, and 80% \(\theta_L\)) are given in Table 2. When the WR was reduced to 110% CP (very heavy-intensity domain; a reduction in WR averaging \(34 \pm 7\) W), exercise was sustained, on average, for only \(30 \pm 12\) s beyond the point of initial fatigue (i.e., after exhaustion at WR\(_{\text{exh}}\); Table 2). Even though WR was reduced by an average of \(11 \pm 2\%\), subjects were still unable to sustain a significant duration of exercise. This was calculated to be equivalent to only \(3.5 \pm 1.7\%\) of W\(^{\prime}\) (on the basis of the WR above CP multiplied by the recovery duration expressed as a percentage of W\(^{\prime}\)). Examples of this response are shown in Fig. 2, A and B.

In contrast, when WR was dropped to 80% \(\theta_L\) (the moderate-intensity domain; a reduction in WR averaging \(170 \pm 22\) W), all subjects completed the 20-min target period, as expected (Table 2; Fig. 2A). This corresponded to a \(56 \pm 4\%\) reduction in WR.

Interestingly, however, only two of six subjects completed the target 20 min at the 90% CP WR (the heavy-intensity exercise domain; a reduction in WR averaging \(83 \pm 5\) W) with the remaining four subjects fatiguing before the 20-min target. The exercise duration at 90% CP was highly variable and averaged \(785 \pm 400\) s (range: \(188\)–\(1,200\) s; Table 2). The drop in WR from WR\(_{\text{exh}}\) averaged \(27 \pm 2\%\). Two examples of the \(V_{O2}\) response are shown in Fig. 2, A and B. Figure 2A provides an example of a subject who could not sustain the 20-min target exercise period, and Fig. 2B provides an example of a subject who was able to sustain the 20 min of exercise after initial fatigue at WR\(_{\text{exh}}\). Figure 3 illustrates the times to fatigue for each subject (after the initial fatiguing WR\(_{\text{exh}}\) bout) at the three different exercise intensities.

The four subjects who were unable to sustain exercise for the 20-min target period all fatigued at a value of \(V_{O2}\) less than the maximum (e.g., subject 1 in Fig. 2A). In all cases, standard respiratory measurements revealed that metabolic demands were falling at the time of fatigue. The responses of \(V_{O2}, V_{CO2}\), and \(V_{E}\) were falling in all cases. In these four subjects, \(V_{O2}\) at fatigue averaged \(4.1 \pm 0.4\) l/min, or \(89 \pm 8\%\) of their \(V_{O2\text{max}}\), \(V_{CO2}\) averaged \(3.9 \pm 0.3\) l/min, or \(73 \pm 5\%\) of \(V_{CO2\text{max}}\) (of the WR\(_{\text{exh}}\) constant load), and \(V_{E}\) averaged \(113 \pm 9\) l/min, or \(77 \pm 10\%\) of \(V_{O2\text{max}}\) (of the WR\(_{\text{exh}}\) constant load). Figure 4, A and B, shows examples of \(V_{CO2}\) and \(V_{E}\) in subjects 1 and 2, respectively (subject 1 fatigued before the 20-min target; subject 2 achieved the 20-min target). Heart rate, however, was observed to have a secondary rise during the 90% CP recovery exercise bout, causing the four subjects who fatigued in recovery to attain a heart rate of \(100 \pm 3\%\) of their maximum heart rate (HR\(_{\text{max}}\)), compared with the two subjects who achieved the 20-min target who attained...
96 ± 2% of HRmax (2 individual examples are shown in Fig. 4, A and B).

ANOVA and post hoc (Neuman-Keuls) tests revealed that, although there were no differences between exercise durations at WRexh on the three tests, there was a significant difference between the exercise duration during the recovery from a fatiguing bout (WR exh) within each of the moderate, heavy, and very-heavy exercise intensity domains (P < 0.05). Figure 5 shows a summary of the group mean responses. The mean power duration curve and the CP and \( \hat{\theta}_L \) thresholds are shown, and the arrows depict the mean durations in recovery after depletion of W during WRexh.

**DISCUSSION**

Despite an abrupt reduction in the WR at the point of fatigue, a significant amount of exercise could not be

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**Table 2. Work rates and exercise duration at 110% CP, 90% CP, and 80% \( \hat{\theta}_L \) after an exhaustive all-out constant-load bout**

<table>
<thead>
<tr>
<th>Subject</th>
<th>WRexh, W</th>
<th>110% CP, W</th>
<th>90% CP, W</th>
<th>80% ( \hat{\theta}_L ), W</th>
<th>110% CP, s</th>
<th>90% CP, s</th>
<th>80% ( \hat{\theta}_L ), s</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>350</td>
<td>323</td>
<td>265</td>
<td>140</td>
<td>12</td>
<td>685</td>
<td>1,200</td>
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<tr>
<td>2</td>
<td>300</td>
<td>261</td>
<td>213</td>
<td>120</td>
<td>23</td>
<td>1,200</td>
<td>1,200</td>
</tr>
<tr>
<td>3</td>
<td>295</td>
<td>266</td>
<td>218</td>
<td>130</td>
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<td>188</td>
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<tr>
<td>4</td>
<td>295</td>
<td>265</td>
<td>217</td>
<td>145</td>
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<td>518</td>
<td>1,200</td>
</tr>
<tr>
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<td>255</td>
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<td>208</td>
<td>135</td>
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<tr>
<td>Mean</td>
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<td>271*</td>
<td>222**</td>
<td>135*</td>
<td>30†</td>
<td>785†</td>
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<td>22</td>
<td>9</td>
<td>12</td>
<td>400</td>
<td>0</td>
</tr>
</tbody>
</table>

WRexh, exhaustive work rate. *Significant difference (P < 0.05) between each of the other work rates. †Significant difference (P < 0.05) between each of the other recovery durations.

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Fig. 2. \( \dot{V}O_2 \) responses of 2 subjects, each exercising to the limit of tolerance at exhaustive work rate (WRexh), followed by a reduction in work rate to 80% of lactate threshold (\( \hat{\theta}_L \); ●), 90% CP (○), and 110% CP (□). *Indication of fatigue. The graphs represent subjects who either did not (A) or did (B) achieve the 20-min target recovery period at 90% CP.
maintained if this WR was above a subject’s CP. The results are, therefore, in accordance with the suggestions of Fukuba and Whipp (6) and have broad implications for the demarcation of the domains of exercise intensity in humans. First, they suggest that the physiological underpinnings of the concept of CP are valid and remain so even during and after a fatiguing bout of exercise, i.e., once W is depleted, exercise cannot continue unless the WR is reduced into a domain where “wholly aerobic” energy transfer may allow exercise to be sustained. The very heavy-intensity domain has been demonstrated to result in a continuously increasing metabolic rate when WR is abruptly increased (e.g., Refs. 21, 22). Second, therefore, the results of the present study demonstrate that this intensity domain is associated with a progressively increasing drive to metabolism that is independent of the prior energy state of the muscle, i.e., despite a reduction in WR, subjects were unable to meet the energy demands of the task. As such, it appears that functional recovery within the very heavy-intensity domain [with the use of the definition of Whipp (30)] is not possible; that is, $\dot{V}O_2$ was not able to fall, and it is inferred, therefore, that [La] would also not reduce (c.f., Ref. 22), and hence pH would be likely to continue to fall, high-energy phosphate stores, e.g., phosphocreatine concentration ([PCr]), would continue to decrease.

Fig. 3. Times achieved for each subject during recovery exercise at each of 80% $\theta_i$ (moderate), 90% CP (heavy), and 110% CP (very heavy).

Fig. 4. Responses of carbon dioxide output ($\dot{V}CO_2$; top), minute ventilation ($V_e$; middle), and heart rate (bottom) in 2 subjects during the 90% CP exercise bout. Both subjects exercised to the limit of tolerance at WRexh. bpm, Beats/min. A: subject who did not achieve the 20-min target recovery period at 90% CP. B: subject who did achieve the 20-min target.
(24), and [P_i] [a potent inductor of fatigue (29)] would continue to rise.

In the very heavy-intensity domain the functional volume of work that is described by W’ is progressively depleted until exhaustion, and, as the present results suggest, that recovery or repletion of W’ is not possible at any level above CP. It is, therefore, suggested that for exercise to continue at a supra-CP WR a proportion of W’ must remain. If W’ were depleted during an exercise bout then a reduction in the WR to a level below CP is required for W’ be replenished. In a practical sense [similar to Fukuba and Whipp (6)], this suggests that an athlete who performs with such an ill-considered pace strategy that W’ is depleted before the end of the race would have to not only reduce their WR to continue but reduce it to a sub-CP level.

The determinants of W’ are presently poorly understood but have been suggested by Knuttgen (14), Monod and Sherrer (18), and Moritani et al. (19), and in review by Hill (9) to be related to the anaerobic work capacity or maximum oxygen deficit (9, 16). These reflect energy stores, such as PCr (and ATP), stored oxygen, and stores of muscle glycogen related to anaerobic glycolysis (17). Because of its relationship with the oxygen deficit, however, W’ may also reflect an inverse function of this, such as the attainment of maximum rates of aerobic function (attainment of VO_2_max), or even a buildup of fatigue-inducing metabolites such as H^+ and Pi. What is interesting about the CP concept is that it predicts that fatigue will ensue as soon as W’ is depleted, an event that occurs only above CP; these data add that W’ may also not be replenished in this intensity domain despite reducing the WR, i.e., the flux of W’ is unidirectional above CP.

W’ in the present study averaged 20.90 ± 2.35 kJ or 246 ± 30 J/kg, consistent with values reported by Hill and Smith (9, 11) in humans. The amount of work actually performed by the subjects in the very heavy-intensity domain after initial fatigue corresponds to only a small proportion of this value (3.5 ± 1.7%). However, the ~30-s tolerance duration at the supra-CP intensity may actually reflect a tolerance time of, or close to, zero were the subject (and W’) actually exhausted by the fatiguing ride. That is, the duration of the recovery period was determined from the point at which the subject could no longer maintain a pedal cadence of >60 rpm, at which point the WR was dropped and the subject was instructed to again increase the pedal cadence; this recovery then continued until the subject could again no longer maintain 60 rpm. It was, however, necessary to allow the subjects some time at the new, reduced WR to determine whether they could actually sustain the required exercise (during which time they received verbal encouragement from the experimenters and the pedal cadence was in some cases continually falling throughout). This time amounted to ~10 s. Thus we had to allow the subjects sufficient W’ reserve to be able to bring the cycling cadence back up to the target level. In a few subjects, it appears we did not judge this to a sufficiently fine tolerance. This seems more likely than W’, having been completely depleted at end exercise and being immediately repleted by 3–4% as the WR reduced. These findings are summarized in Fig. 5.

It was not surprising that all the subjects were able to continue exercise in the moderate-intensity domain (below the \( \theta_i \)). This necessitates a greatly reduced rate of ATP provision and, therefore, VO_2 requirement. This domain does not require a significant proportion of energy derived from anaerobic mechanisms and, therefore, allows for the clearance of fatigue-inducing substances.

What was more surprising, however, was that four of six subjects were unable to maintain exercise in the heavy-intensity domain for the required 20-min target; the overall average was ~13 min. The hypothesis, based on the models of Fukuba and Whipp (6), suggested that subjects would be able to sustain exercise in this intensity domain after initial fatigue. Although we cannot rule out the possibility that the wide variability observed between the recovery duration tolerated at this intensity may, at least in part, be due to day-to-day variability in performance, we believe this to be unlikely on the basis of both the magnitude of the differences and the different response profiles in those who attained the target recovery time and those who did not. Although each recovery bout was not repeated, the initial WR_exh was undertaken on three occasions that yielded an average coefficient of variation for constant load performance of 10%. This value is far lower than the ~50% coefficient of variation for the duration of the 90% CP recovery bout. The mechanism of this fatigue is unclear. The four subjects fatigued at a VO_2 averaging 89 ± 8% of their maximum but a heart rate of 100 ± 3% of their HR_max, compared with the two subjects who achieved the target at 86 ± 4% VO_2_max and 96 ± 2% of HR_max (2 individual examples are shown in Figs. 2 and 4). It is possible, therefore, that
the heart rate in the four subjects who fatigued reflects a condition, presumably (although not absolutely certainly) in the contracting muscles, that is not tolerable. This is despite a reduction in metabolic demand and oxygen pulse inferred from the VO\textsubscript{2} and heart rate responses (e.g., Fig. 4A). The falling oxygen pulse is consistent with stroke volume and/or the arteriovenous oxygen content difference being lower than that required for still-elevated metabolic demands. Further work is required to elucidate the mechanism(s) of this novel feature. We assume that the four subjects who could not recover sufficiently to meet the 20-min target would have been successful had the WR been lower in the domain.

W’ has previously been demonstrated to be dependent (at least in part) on the glycogen storage status of the subject (17). Thus exercise designed to deplete W’ will also reduce muscle glycogen content in the contracting units, but the degree to which this occurred in our subjects is unknown. It is well established that during WRs that induce fatigue in ~10 min, the average muscle glycogen content remains high at the point of exhaustion (e.g., Ref. 25). At WR ranges that demand between ~60 and 85% VO\textsubscript{2}\text{max} (on the basis of the linear WR-VO\textsubscript{2} relationship from the ramp incremental test), muscle glycogen is almost completely depleted at fatigue (e.g., Ref. 4). Newsholme et al. (20), for example, have suggested that muscle glycogen limitation might even limit maximum performance at durations of <30 min by using plausible estimates of muscle glycogen storage and utilization rates (13). Our subjects exercised for a total of ~19 min on average, of which 6 min was at a WR that resulted in 100% of VO\textsubscript{2}\text{max} and an additional 13 min where VO\textsubscript{2} declined, but only to a value of ~88% VO\textsubscript{2}\text{max}. It is, therefore, possible that fatigue ensued in some subjects during the 90% CP bout, because of the differences in muscle glycogen availability, but not in others. Furthermore, this degradation may be exacerbated in different muscle fiber types (26) and, therefore, may be dependent on an individual’s fiber-type profile and recruitment pattern during the exercise. Another possible mechanism for the differences observed is on the basis of lactate transport in the different individuals; this too, however, may be exacerbated by a different fiber-type profile (e.g., Ref. 8). Because the fiber-type characteristics of these subjects were not known, we hypothesized that parameters of aerobic fitness, such as VO\textsubscript{2}\text{max} or \(\theta_t\), which are related to fiber-type proportion, or, conversely, anaerobic parameters, such as W’, might provide a basis on which to distinguish between subjects. We were, however, unable to find any correlation between the tolerable duration of recovery at the 90% CP WR and these parameters, but, of course, the subject number is small.

At the extremes, subject 5 exercised for a total of ~21 min before fatiguing (i.e., 365 s at WR\text{exh} and 916 s at 90% CP), whereas subject 3 managed only ~7 min (294 s at WR\text{exh} and 118 s at 90% CP), which would appear to be too short a duration to deplete muscle glycogen stores [by using the assumptions of Newsholme et al. (20)]. Another explanation, therefore, is that high-intensity exercise itself altered CP, CP has previously been shown to be dependent on state of training (7, 23) and on the inspired oxygen fraction, being increased in hyperoxia (31) and decreased in hypoxia (19, 31). In contrast, glycogen depletion affected W’ only and not the value of CP (17). It is possible that if the fatiguing exercise itself (WR\text{exh}) affected the value of CP, then the recovery exercise may have been at a level >90% CP. We believe that this is unlikely, at least to within ±10% of CP because exercise was not well tolerated at 110% CP but was better tolerated at 90% CP. It is not possible, however, to establish CP immediately after fatigue by using presently available techniques.

In conclusion, therefore, our findings demonstrate that functional recovery after fatiguing exercise is highly intensity dependent, and this has implications for the optimization of training strategies for athletic performance.

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**DISCLOSURES**

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**REFERENCES**


