Physiological Responses to Cycling for 60 Minutes at Maximal Lactate Steady State

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Catalog Data

Key words: Rate of perceived exertion, heart rate, oxygen consumption, blood lactate, cycling

Mots clés: Taux de perception de l’effort, fréquence cardiaque, consommation d’oxygène, lactaté, cyclisme

Abstract/Résumé

Changes in physiological variables during a 60-min continuous test at maximal lactate steady state (MLSS) were studied using highly conditioned cyclists (1 female and 9 males, aged 28.3 ± 8.1 years). To determine power at MLSS, we tested at 8-min increments and interpolated the power corresponding to a blood lactate value of 4 mmol/L. During the subsequent 60-min exercise at MLSS, we observed a sequential increase of physiological parameters, in contrast to stable blood lactate. Heart rate drifted upward from beginning to end of exercise. This became statistically significant after 30 min. From 10–60 min of exercise, a change of +12.6 ± 3.2 bpm was noted. Significant drift was seen after 30 min for the respiratory exchange ratio, after 40 min for the rate of perceived exertion using the Borg scale, and after 50 min for % VO₂max/kg and minute ventilation. This slow component of VO₂ max may be the result of higher recruitment of type II fibers.

L’objectif de cette étude était d’évaluer les variations physiologiques lors d’un exercice continu de 60 min à une intensité d’exercice correspondant à l’état stable maximal du lactate (MLSS). Des cyclistes en bonne condition physique (1 femme et 9 hommes; 28.3 ± 8,1 ans) ont participé à l’étude. Afin de déterminer la charge de pédalage correspondant à...
MLSS, nous avons utilisé un test progressif avec des incréments de 8 min et interpolé la puissance correspondant à une concentration plasmatique de lactate de 4 mmol/L. Pendant 60 min d'exercice à la puissance correspondant à MLSS, nous avons observé une augmentation séquentielle des paramètres physiologiques alors que la concentration sanguine de lactate était stable. De 10 à 60 min d'exercice, une augmentation de la fréquence cardiaque de $+12.59 \pm 3.24$ bpm a été mesurée devenant significativement différente à 30 min. Cette dérive des variables physiologiques a aussi été significative à 30 min pour le quotient d'échange respiratoire, à 40 min pour la perception de l'effort selon l'échelle de Borg, et 50 min pour le % $\dot{V}O_2\text{max}$/kg et la ventilation-min. La phase lente du $\dot{V}O_2\text{max}$ est probablement le fait d'un recrutement plus important des fibres musculaires de type II.

**Introduction**

Maximal lactate steady state (MLSS) corresponds to the highest power or speed that can be maintained while blood lactate remains stable for the duration of exercise (Stegmann et al., 1981). It can be a useful tool for exercise prescription in athletes (Sjödin and Svedenhag, 1985; Weltman et al., 1992). Lactate levels maintained at such a power or speed can vary from 2.2 to 7.4 mmol/L (Beneke and von Duvillard, 1996; Billat et al., 1994; Stegmann and Kindermann, 1982; Urhausen et al., 1993).

Physiological responses during prolonged exercise have been studied abundantly. However, little is known about physiological responses during a prolonged exercise at MLSS. Such data have been collected only for quite short periods of time (15–30 min), and only a few variables were measured at a time. However, training sessions at MLSS can often last 60 min or more (Billat, 1996). The purpose of this study was to document the changes in physiological variables, including heart rate (HR), $\dot{V}O_2$/kg, minute ventilation, respiratory exchange ratio (RER), rate of perceived exertion (RPE), and blood lactate, during a 60-min continuous exercise performed at MLSS.

**Methods**

One female and 9 male, well-trained cyclists, aged 28.3 ± 8.1 years, participated in the study. The experiments took place at the end of their competitive season, between mid-September and mid-December. The time elapsed between their first and last sessions in the laboratory was no more than 3 weeks. The subjects were asked to avoid consumption of food for 4 hr, and alcohol and caffeine for 12 hr, before each test. They were also instructed to avoid heavy exercise 24 hr before each test. Their participation was approved by our institutional ethics committee, and all subjects gave a written, informed consent before entering the study.

**EXPERIMENTAL PROCEDURES**

*First laboratory session.* During their first session, various anthropometric measurements were taken: body mass, height, and percentage of body fat (Jackson and Pollock, 1977). All cycling measurements were performed using each subject's own bicycle mounted on a Computrainer ergometer (Racermate,
Computrainer Professional 6001, Seattle, WA). This system has been validated previously (Cane et al., 1996). Pedalling rate was chosen by each subject during their warm-up before VO₂ max determination and kept constant during every subsequent test session. An average of 88.5 ± 1.37 rpm was maintained by our subjects. VO₂ max was measured with a protocol involving 3-min increments, each of 40 W, starting at 80 W. VO₂ max criteria were (a) attainment of predicted HR, (b) a RER greater than 1.1, and (c) a plateau indicated by a change of <150 ml/min between 2 stages. During VO₂ max determination and continuous exercise tests, expired gas was analyzed every 30 s with a Vacumed gas analyzer (Vacumed, Ventura, CA) calibrated before each test throughout the continuous exercise while subjects were connected to the metabolic cart.

The values of physiological variables at specific times (5, 10, 20, 30, 40, 50, and 60 min) were an average of the preceding 2 min. HR was measured continuously with a Polar belt transmitter coupled to a receiver connected to the data acquisition system. A wrist receiver was used simultaneously as a back-up (Polar Vantage XL, Finland). Throughout the continuous exercise, the RPE was measured 1 min before the physiological variables. Blood lactate was quantitated from a fingertip capillary sample with a portable analyzer (Accusport, Boehringer-Mannheim, Mannheim, Germany) during the last 30 s of the increment or 30 s before the time of measurement (5, 10, 20, 30, 40, 50, and 60 min) in the case of continuous testing. The finger used for sampling was wiped before each measurement. Laboratory temperature was kept constant at 21°C with air conditioning. The subjects had to drink 500 ml of water in the laboratory during the 30 min preceding the continuous exercise tests. A fan provided convection during exercise to decrease thermal stress.

Second laboratory session. During their second session, subjects had to perform a progressive maximal exercise with 8-min increments until exhaustion for the determination of power at MLSS. The increments were performed respectively at every 30 W, beginning at 100 W. To test the reliability of this procedure, we had all subjects complete the test twice within 3 weeks.

After the progressive maximal exercise test during the second session, we calculated the power for the continuous test potentially corresponding to MLSS. The calculation was based on an inverse linear interpolation to 4 mmol/L blood lactate, using the increments immediately higher and lower than 4 mmol/L. Despite the fact that MLSS can range between 2 and 7 mmol/L of lactate according to the individual, 4 mmol/L corresponds to the highest statistical likelihood of finding MLSS (Heck et al., 1985). The next laboratory sessions were planned according to the algorithm found in Figure 1.

When the load corresponding to MLSS was found, subjects performed a constant load test. They had to maintain this constant load for 60 min and were stopped if necessary. Blood lactate was measured at 5, 10, 20, 30, 40, 50, and 60 min without interruption of the cycling. HR and other physiological measurements (RER, min ventilation, VO₂/kg) from the metabolic analyzer were recorded continuously. MLSS was considered to be attained when blood lactate variations were lower than ± 0.75 mmol/L from 10 to 60 min of continuous exercise.

Third and fourth laboratory sessions. For subjects with a decreased or constant blood lactate during the first attempt, a second constant load exercise was
performed with +5% of the estimated MLSS power used in the third session. Subjects who failed the constant load exercise performed the second attempt at −5% of estimated MLSS power. The same physiological measurements were taken as during the third session. For some subjects it was necessary to perform a last continuous test with adjustments according to the variation of lactate level during the previous continuous test.

Last laboratory test. Subjects with a decreased or constant blood lactate in the second continuous test (fourth session) were submitted to a last test at +2.5%, or +7.5% of estimated MLSS power for those who were underestimated at their first continuous test. For those who were overestimated and failed to complete the second trial, the next continuous test was performed at −2.5% or −7.5% of estimated MLSS power.

Statistics. Data are presented as mean values ± standard deviations (X ± SD). Mean differences were determined by analysis of variance for repeated measurements and post hoc Scheffé analysis with P < .05. To verify the test-retest capacity of the incremental test or relationships between variables, we calculated Pearson’s correlation coefficient. For all statistical analyses, differences were considered significant when P < .05.

Results

The characteristics of the subjects (age, height, weight, percentage of fat, $\dot{V}O_{2\max}$, power at MLSS, and peak work rate) are listed in Table 1.
Table 1 Anthropometric and Physiological Characteristics of Subjects

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Age</th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
<th>% fat</th>
<th>$VO_{2\text{max}}$ (ml/kg/min)</th>
<th>MLSS</th>
<th>Power (watts)</th>
<th>Peak work rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>39</td>
<td>163</td>
<td>69.9</td>
<td>12.7</td>
<td>62.5</td>
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<td>2</td>
<td>22</td>
<td>186.5</td>
<td>78.8</td>
<td>5.7</td>
<td>67.6</td>
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<td>340</td>
<td>460</td>
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<td>4.4</td>
<td>64.7</td>
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<td>4</td>
<td>31</td>
<td>171.5</td>
<td>70.7</td>
<td>9.2</td>
<td>61.4</td>
<td></td>
<td>300</td>
<td>380</td>
</tr>
<tr>
<td>5</td>
<td>46</td>
<td>171.3</td>
<td>77.5</td>
<td>12.8</td>
<td>52.5</td>
<td></td>
<td>220</td>
<td>340</td>
</tr>
<tr>
<td>6</td>
<td>25</td>
<td>168.5</td>
<td>71.6</td>
<td>7.4</td>
<td>59.0</td>
<td></td>
<td>270</td>
<td>340</td>
</tr>
<tr>
<td>7</td>
<td>23</td>
<td>178</td>
<td>66.6</td>
<td>3.9</td>
<td>60.9</td>
<td></td>
<td>290</td>
<td>380</td>
</tr>
<tr>
<td>8*</td>
<td>25</td>
<td>163</td>
<td>61.4</td>
<td>4.4</td>
<td>70.5</td>
<td>MD</td>
<td>320</td>
<td>380</td>
</tr>
<tr>
<td>9</td>
<td>21</td>
<td>171.5</td>
<td>65.5</td>
<td>6.4</td>
<td>72.3</td>
<td></td>
<td>320</td>
<td>460</td>
</tr>
<tr>
<td>10 (W)</td>
<td>26</td>
<td>162.5</td>
<td>53.4</td>
<td></td>
<td>55.6</td>
<td>MD</td>
<td>190</td>
<td>260</td>
</tr>
<tr>
<td>Mean</td>
<td>28.3</td>
<td>170.8</td>
<td>68.7</td>
<td>7.4</td>
<td>62.7</td>
<td></td>
<td>276.7</td>
<td>376</td>
</tr>
<tr>
<td>SD</td>
<td>8.1</td>
<td>7.43</td>
<td>7.5</td>
<td>3.4</td>
<td>6.3</td>
<td></td>
<td>49.0</td>
<td>61</td>
</tr>
</tbody>
</table>

*Subject present only for the incremental tests. Did not show up to the continuous tests. MD: missing data; W: woman.

**ESTIMATION OF MLSS**

The test-retest reliability of the progressive maximal protocol with 8-min increments was demonstrated by a correlation coefficient of $r = 0.96$, $P < .001$ ($n = 10$) between 2 series of lactate levels during 2 different incremental tests. When we considered only the test-retests performed within 14 days, the coefficient ratio increased to $r = 0.98$, $P < .001$ ($n = 7$). In 6 out of 9 subjects, the first power estimate was real power at MLSS. Subjects who had stable blood lactate at the first attempt ($n = 6$) were submitted to a second continuous exercise at +5% of the predicted wattage to confirm that they were at their real MLSS. In 3 subjects, the power estimate was lower than MLSS. Among these 3 subjects, 1 failed to complete 60 min, and 2 had increased blood lactate. The three subjects were then submitted to a continuous protocol at −5% of the predicted wattage.

After 3 continuous sessions, MLSS was finally determined for all subjects at the nearest 10 W. At the real MLSS, the average blood lactate level showed no significant variation between 5 and 60 min of the continuous test (Table 2). The resulting average blood lactate at MLSS was $4.48 \pm 0.88$ mmol/L with a range of 3.5 to 6.4 mmol/L from 10 to 60 min of continuous exercise ($n = 9$). Average power at the real MLSS was not statistically different from the first MLSS estimate ($276.67 \pm 49.0$ vs. $281.7 \pm 49.9$ W, $n = 9$).
PHYSIOLOGICAL VARIATIONS AT MLSS

Of course, the variation in blood lactate was minimal at MLSS, from 10 to 60 min of exercise (Table 2). However, some other variables increased linearly and significantly with exercise duration. HR drifted upward from the beginning until the end of exercise. This upward drift became significant after 30 min, and from 10 to 60 min of exercise a variation of $+12.59 \pm 3.24$ bpm was observed (Table 2).

The RER decreased significantly from 0.998 $\pm$ 0.01 to 0.956 $\pm$ 0.01, being statistically significant from the 30th min (Table 2). Drifts were also significant after 40 min for the RPE on the Borg scale and at 50 min for % VO$_2$/kg as well as minute ventilation (Table 2).

Discussion

The main purpose of our study was to evaluate changes in physiological parameters during 60 min of exercise at MLSS. Our first task was then to determine cycling power corresponding to the MLSS. For all subjects, we were able to measure power at MLSS after a maximum of three laboratory sessions.

The average lactate level at MLSS was not much different in our study (4.48 mmol/L) than in other studies on cyclists (5.4 mmol/L for Beneke and von Duvillard, 1996; Swensen et al., 1999; and 4.2 mmol/L for Beneke et al., 1996). Our results also suggest that it would be unrealistic to rely only on the 4 mmol/L blood lactate level as a universal criterion for exercise prescription at MLSS. We do not advocate the use of our 4.48 mmol/L as a universal criterion either. Indeed, the average blood lactate level that we measured at MLSS was made up of individual values ranging from 3.5 to 6.4 mmol/L. Moreover, MLSS is an individual variable.

The subjects' cycling experience, the use of their own bicycle, and particularly the lengthening of exercise increments probably all contributed to the optimisation of MLSS determination by progressive maximal exercise during the second session. The rationale for lengthening increments to 8 min was first suggested by Foxdal and colleagues (1994; 1996). The 8-min increment seems optimal for lactate diffusion from the intramuscular compartment to plasma since lactate diffusion is partly limited by the capacity of its transporters (Juel et al., 1994). When the increment is shorter than 5 min, lactate may fail to equilibrate, and its measurement in blood may not yield a value as close to the intramuscular lactate level during this particular increment. This may explain the postexercise increase of blood lactate in protocols using increments shorter than 5 min (McLellan and Jacobs, 1993).

A second reason for the relatively good determination capacity of progressive maximal exercise during the second session was the use of cycling power at 4 mmol/L as the criterion for linear interpolation. Despite the dispersion of individual blood lactate values at MLSS between 3.5 and 6.4 mmol/L in our own study (average 4.48 $\pm$ 0.88 mmol/L), 4 mmol/L is a central point around which most individual values are scattered. Similar dispersions were measured at MLSS on a cycle ergometer (2 to 7 mmol/L; Stegmann and Kindermann, 1982; Stegmann et al., 1981) and during treadmill running (3.0 to 5.5 mmol/L; Heck et al., 1985). We determined MLSS at 71.3% VO$_2$max/kg, between the 65% VO$_2$max of Stegmann and Kindermann (1982) and the 75% VO$_2$max of Chassain (1986).
Table 2  Physiological Variations During the 60-Minute Exercise at MLSS

<table>
<thead>
<tr>
<th>Minutes</th>
<th>5</th>
<th>10</th>
<th>20</th>
<th>30</th>
<th>40</th>
<th>50</th>
<th>60</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lactate (mmol/L)</td>
<td>4.27 ± 0.25</td>
<td>4.79 ± 0.42</td>
<td>4.59 ± 0.39</td>
<td>4.37 ± 0.21</td>
<td>4.61 ± 0.49</td>
<td>4.21 ± 0.36</td>
<td>4.27 ± 0.37</td>
</tr>
<tr>
<td>% VO₂_max/kg</td>
<td>73.1 ± 2.0</td>
<td>74.6 ± 1.9</td>
<td>75.6 ± 2.0</td>
<td>76.1 ± 1.8</td>
<td>76.9 ± 1.6</td>
<td>78.2 ± 1.5*</td>
<td>79.3 ± 1.4*</td>
</tr>
<tr>
<td>Heart rate (bpm)</td>
<td>152.8 ± 5.3</td>
<td>157.4 ± 4.4</td>
<td>160.2 ± 13.1</td>
<td>163.1 ± 4.2*</td>
<td>165.8 ± 4.2*</td>
<td>168.7 ± 4.0*</td>
<td>170.0 ± 3.8*</td>
</tr>
<tr>
<td>VE³ (1/min)</td>
<td>75.1 ± 3.6</td>
<td>80.7 ± 4.0</td>
<td>81.9 ± 4.2</td>
<td>83.6 ± 4.4</td>
<td>85.3 ± 4.1</td>
<td>87.8 ± 4.4*</td>
<td>90.6 ± 4.8*</td>
</tr>
<tr>
<td>Borg scale (RPE)</td>
<td>12.5 ± 0.8</td>
<td>13.2 ± 0.7</td>
<td>13.9 ± 0.7</td>
<td>14.5 ± 0.6</td>
<td>15.1 ± .5*</td>
<td>16.0 ± .7*</td>
<td>16.5 ± .7*</td>
</tr>
<tr>
<td>RER</td>
<td>0.998 ± 0.01</td>
<td>0.987 ± 0.01</td>
<td>0.972 ± 0.01</td>
<td>0.965 ± 0.01*</td>
<td>0.960 ± .01*</td>
<td>0.958 ± .01*</td>
<td>0.956 ± .01*</td>
</tr>
</tbody>
</table>

*Significantly different from 10 minutes value (P < 0.05).
³Minute-ventilation.
Furthermore, our results suggest that it is not necessary to perform the whole stepwise exercise test to estimate MLSS. Just by knowing which workloads sit immediately over and under the point of 4 mmol/L would be sufficient. The test can be stopped as soon as the associated power is determined.

VARIATION OF PHYSIOLOGICAL PARAMETERS

Increased HR during the 60-min exercise at MLSS was quite significant (+12.59 ± 3.24 bpm). Two other studies reported augmented HR during cycling exercise, also at MLSS, but over 30 min (Snyder et al., 1994; Urhausen et al., 1993). In the first study, it was an increase of 9 bpm between 10 and 30 min of exercise (Snyder et al., 1994), and in the second study, 5 bpm between 15 and 30 min (Urhausen et al., 1993). Only 1 study reported stable HR over a 40-min cycling trial at MLSS (Swensen et al., 1999).

Such an upward drift of HR has been observed previously as one of the components of “cardiovascular drifting” during exercise at a fixed workload not necessarily corresponding to MLSS (Ekelund, 1967; Trudeau et al., 1997). During continuous exercise, the HR drift has been associated with exercise-induced dehydration and blood pooling in the lower extremities and the skin, resulting in lower stroke volume (Rowell, 1993). However, Hamilton and colleagues (1991) observed that dehydration and thermoregulation may not be the only causes of higher HR during continuous exercise.

The acceleration of HR could also partially reflect an increased central command subsequent to a decrease in muscle efficiency associated with fatigue (Woledge, 1998). Indeed, lower muscle efficiency is reflected by the elevation of VO$_2$/kg observed during continuous exercise, also associated with the higher demand of ventilation reflected by higher minute ventilation. McLellan and Cheung (1992) also reported increased VO$_2$/kg during exercise at MLSS. During a 20-min session at MLSS, the percentage of VO$_2$ max sustained 5 min after the start of exercise was 71% and reached 76% VO$_2$ max after 20 min (McLellan and Cheung, 1992). During a 15-min test, they observed a rise from 60.4% to 72% VO$_2$ max (McLellan et al., 1992). We measured a change in percentage of VO$_2$ max sustained from 71.3 to 79.3% VO$_2$ max, which seems similar to what was reported by McLellan and Cheung (1992), given their exercise duration, which was shorter. However, observations by McLellan and us may seem somewhat lower than the averaged 80.2% VO$_2$ max measured by Swensen and colleagues (1999) over a 30-min MLSS cycling session. In the study by Swensen and colleagues (1999), it is unfortunately impossible to verify a potential drift of data since only average data over the 30-min trial are available. The upward drift of VO$_2$ during continuous exercise at a fixed workload has also been termed the slow component of O$_2$ uptake (Whipp and Jackman, 1994), an appellation that appears to be preferred by some authors (Gaesser and Poole, 1996).

From studies on muscle oxygenation during heavy exercise, it seems accepted that most of the increased VO$_2$ during the slow component can be ascribed to a higher VO$_2$ in active muscles (Poole et al., 1991). This increase of oxygen consumption at the same workload could reflect a lower economy of muscle exercise potentially caused by (a) higher muscle temperature as exercise duration is
lengthened and (b) alterations of muscle metabolism. Elevated muscle temperature as a mechanism of \( \text{VO}_2 \) drifting has been suggested by the demonstration of higher oxygen drift during downhill running (30 min) compared with level running at the same relative intensity (Westerlind et al., 1992). It was then proposed that the higher increase of \( \text{VO}_2 \), in the former was due to higher muscle temperature. However, external warming of quadriceps during cycling did not potentiate the slow component of \( \text{VO}_2 \) (Koga et al., 1997). Thus, whether increases in muscle temperature can be considered a potential mechanism of \( \text{VO}_2 \) upward drifting remains to be elucidated.

Higher relative oxygen consumption could also be the result of altered muscle metabolism. This higher \( \text{VO}_2 \) is associated with lowering of RER \( (r = -0.66, P < 0.05) \), indicating higher relative lipid oxidation at the end of exercise than at the beginning, despite the same absolute workload. Higher lipid oxidation produces higher oxygen demand to generate the same quantity of ATP. A similar decrease of RER has been measured during prolonged exercise at MLSS (Beneke et al., 1996) or at other workloads (Powers et al., 1980). The hypothesis of progressive recruitment of type II fibers as exercise continues might account for the upward drift in \( \text{VO}_2 \) (Barstow et al., 1996). Animal studies showed that type II fibers are less efficient, consuming more oxygen than type I fibers at the same tension level in the cat (Kushmerick et al., 1992), rat, and rabbit (Reggiani et al., 2000). Whether a similar mechanism might account for the added \( \text{VO}_2 \) during long duration exercise remains to be resolved but has been supported by the findings of Barstow and colleagues (1996) reporting a negative correlation between the percentage of type I fibers and the slow component of \( \text{VO}_2 \). In support to this hypothesis for humans, studies performed on skinned human muscle fibers showed that fast myosin heavy chains (MHC), in higher proportion in type II fibers, are associated with higher tension cost than fibers mostly composed of slow MHC (Reggiani et al., 2000).

The continuous increase of minute ventilation we measured over the 60-min MLSS exercise is consistent with other observations made during shorter cycling bouts, also at MLSS. McLellan and colleagues (1991) and McLellan and Cheung (1992) measured an increased minute-ventilation over 20 and 30 min in two different experiments (+11.6 and +20 l/min, respectively). The magnitude of our variation in ventilation minute seems lower than for the McLellan group observations. If it is associated with the slightly lower level of fitness of their subjects (\( \text{VO}_2 \text{max/kg} = 57.6 \) and 54.4 ml/kg/min vs. 62.7 ml/kg/min in our subjects) is not clear. One hypothesis to explain the higher minute ventilation and the lower RER as exercise duration increases is the muscle glycogen depletion typical of such an intense exercise (>70% \( \text{VO}_2 \text{max} \)). Heigenhauser and colleagues (1983) hypothesized that during exercise, minute ventilation is higher with low glycogen stores than with normal stores.

The RPE value we measured was similar over the 60-min period to that reported by Swensen and colleagues (1999) over 40 min at MLSS (14.5 vs. 15, respectively). However, we measured a significant increase of this value from 12.5 to 16.5 between 5 and 60 min. A close association between RPE and blood lactate has been suggested in other studies using incremental tests (Steed et al., 1994; Weltman, 1995). However, the continuous increase of RPE we noted during exercise at MLSS, despite constant blood lactate, does not seem compatible with the
observation of a relationship between RPE and lactate measured during incremental tests. Instead, we found a significant correlation ($r = 0.90$, $P < .05$; $N = 9$) between HR and the Borg scale during exercise at MLSS, HR being an index of central command (Rowell, 1993). A progressively increasing rate of perceived exertion could also reflect a higher central command.

Another observation is the sequential change in physiological parameters we measured during MLSS; that is, some parameters increased statistically before other parameters. The order of appearance of statistical difference was as follows: HR and RER at 30 min, RPE at 40 min, $\overline{VO_{2}}$/kg and minute ventilation at 50 min. To determine if this observation has a physiological mechanism deserves further study.

In conclusion, we noted an increase of physiological parameters over a 60-min exercise at MLSS. Despite stable blood lactate, we measured elevation of HR, RPE, $\overline{VO_{2}}$/kg, and min ventilation. The rise in $\overline{VO_{2}}$/kg can be attributed most probably to a change in muscle fiber recruitment involving more type II fibers, and to a lower proportion due to higher ventilation work, as shown by the increase in minute ventilation (Gaesser and Poole, 1996). However, augmented $\overline{VO_{2}}$/kg cannot be ascribed to variations of lactate since plasma lactate did not rise during continuous exercise at MLSS. The potential causality or relationship between increasing plasma lactate and the slow component (Barstow et al., 1996; Ryan et al., 1988) has been challenged recently (Gaesser and Poole, 1996).

Such a variation in physiological measurements may have some influence on exercise prescription. It is more convenient for an athlete to adjust exercise intensity at MLSS with other physiological variables than with blood lactate. As an example, a device measuring wattage would be the best tool to adjust exercise intensity for sessions at MLSS in cyclists since it will take into account other factors like wind and rolling resistances, as well as grade. However, due to cost factors, HR is used more often as the physiological variable for adjustment of exercise intensity. Our results suggest that in some instances, the HR drift must be taken into account to avoid the reduction of workload that would be associated when maintaining HR at the same level. In some situations like racing or tempo training, it would not be advisable to rely only on HR to adjust exercise intensity. However, in other situations like cardiac rehabilitation, it is preferable to rely on HR to monitor exercise intensity, since it is a component of the rate pressure product, an index of cardiac oxygen consumption.

References


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