The Maximal Lactate Steady State in Elite Endurance Athletes

A. R. HOOGEVEEN, J. HOOGSTEN, and G. SCHEP

St. Joseph Hospital Veldhoven, Postbox 7777, 5500 MB Veldhoven, The Netherlands

Summary: The upper limit of blood lactate resulting in a lactate steady state during prolonged exercise is called the maximal lactate steady state (MLSS). The purpose of this study was to investigate the lactate response to steady-state exercise during a field test in elite endurance athletes. Plasma lactate levels were assessed in 13 elite triathletes and 13 elite cyclists (mean $\pm$ SD; age 23.7 ± 5.1 yr; HT 180.2 ± 6.3 cm; WT 70.3 ± 5.9 kg; $\dot{V}O_{2\max}$ 68 ± 3.7 ml/min/kg) during a 40 km-long time trial on a bicycle (4 km course $\times$ 10 laps). The steady state was demonstrated by monitoring the heart rate and timing every course

run. The lactate levels were expected to correspond to MLSS. The mean level of lactate during the time trial was 7.4 ± 2.5 mmol/l. Five athletes maintained plasma lactate levels which exceeded 10 mmol/l or more for almost 1 h. The large value of individual variability was conspicuous (range 3.2–12.2 mmol/l). These values exceeded all previous reported levels for MLSS from other investigators. Our observations are important in sport medical practice since the different lactate responses to exercise are used as parameters in training management. [Japanese Journal of Physiology, 47, 481–485, 1997]

Key words: lactate, steady state, endurance exercise, MLSS.

The upper limit of blood lactate resulting in a lactate steady state during constant workload is called the maximal lactate steady state (MLSS). The determination of MLSS requires the performance of several constant workloads on different days [1]. MLSS is expected to correspond to the anaerobic threshold (AT) which reflects the transition of aerobic to anaerobic conditions [2, 3]. An athlete is assumed to sustain exercise intensities of around AT and MLSS for a relatively long period of time (20 min up to 1.5 h). The lactate level during steady-state exercise is assumed to be within defined values. Some investigators proposed a fixed lactate concentration of 4 mmol/l [2]. In an attempt to define AT, some investigators described an individual anaerobic threshold (IAT) [1, 4] and a lactate threshold (LT) [5, 6]. The average lactate concentration at AT, LT or IAT in these studies varies from 1.7 ± 0.3 [4] to 4.2 ± 0.8 mmol/l [1]. It is remarkable that only very few investigators have compared these laboratory data with the values assessed during a practical situation in a field test [7, 8]. However, these studies concerned moderately trained subjects. In ad-

dition, lactate in these studies was assessed only post-exercise, and steady state was not controlled. Therefore, in this study, we determined the lactate steady-state level in elite endurance athletes during a field test. The main question in this study was whether the plasma lactate concentration during steady-state exercise in elite endurance athletes corresponded with the limited levels of lactate observed by numerous investigators in laboratory settings.

Thirteen elite triathletes and 13 elite cyclists (mean $\pm$ SD; age 23.7 ± 5.1 yr; HT 180.2 ± 6.3 cm; WT 70.3 ± 5.9 kg) volunteered to participate in this experiment after giving written informed consent. The mean $\dot{V}O_{2\max}$, determined 2 weeks before the time trial during incremental exercise (20 W/min) on a cycle ergometer using oxymetry, was 68 ± 3.7 ml/min/kg. The 26 subjects were free from serious diseases and took no drugs or medication during the course of the study. On the test days, no one showed any sign of disease or infection. The 26 subjects had trained regularly for at least 4 years (10 ± 3 yr). The mean training duration was 14 ± 4.2 h a week. The percentage of body fat, es-

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Correspondence should be addressed to: A. R. Hoogeveen, St. Joseph Hospital, Department of Sports Medicine, Postbox 7777, 5500 MB, Veldhoven, The Netherlands. Tel: +31-402588210/402531007, Fax: +31-402588212, E-mail: A.Hoogeveen-Bos@inter.NL.net

timated using the measurements of four skinfolds according to Durnin and Womersley [9] was 8.1 ± 3.3 .

After a period of 4 weeks following the laboratory tests, the subjects were instructed to ride an individual time trial on a flat circuit of 4.05 km which had to be covered ten times. On the day of the triathletes contest, the temperature was 14–17°C, humidity was 70%, there was no wind and the sky was clear. On the day of the cyclists contest, the temperature was 15–18°C, humidity was 40%, there was no wind and the sky was clear. The subjects had refrained from intensive exercise for the previous 24 h, and they were in-

structed to eat a carbohydrate-rich meal before the day of the time trial. The subjects started at 1-min intervals. Heart rate was measured every 5 s with a heart rate monitor (Polar, Electro OY, Finland). Steady state was demonstrated by the heart rate and the timing of every course run.

Before warming up, and during and immediately after exercise, 1 ml of blood was taken from the ante-cubital vein with a needle to determine lactate concentration (Fig. 1). Subjects were stopped after completing five laps and detained for 150 s while a blood sample was obtained. Every sample was obtained

Fig. 1. The speed and heart rate during the time trial for each subject. a, 13 triathletes; b, the 13 cyclists. The speed is the mean speed over each course run of 4 km for each individual. The heart rates are the mean of overlapping 5 s and scanned by a computer from the original polar hardcopy. The black arrows indicate the moment of sampling.

within 30 s. The collection tubes for lactate contained potassium fluoride. Plasma lactate concentration was determined using a Cobas enzymatic test kit (Boehringer, Mannheim, Germany). All blood samples were kept in melting ice, and were centrifuged (3,000 rpm, $1,590\times g$, 20°C) and analyzed within 1 h. The mean heart rate was determined for the last 20 min of the first and second half of the time trial. The subjects were instructed not to sprint before the sample was taken. To be sure that the subject had not sprinted before sampling, the mean heart rate was also computed for the last 2 min just before sampling. The mean lactate levels and mean heart rates in the first part of the time trial were compared with those in the second part using a paired *t*-test. Pearson's Product Moment Correlation was used to calculate correlation coefficients. The level of significance was set at 0.05.

The mean speed was 41.4 ± 1.0 km/h in both parts of the time trial. According to the average speed of the course run times of each individual, all subjects performed at a nearly constant exercise intensity (Fig. 1). The mean resting lactate level was 1.4 ± 0.4 mmol/l and the mean lactate level of the two samples during the time trial was 7.4 ± 2.5 mmol/l (Fig. 2). The mean lactate concentration in the first part was (20 km) 7.7 ± 2.6 mmol/l, and in the second part (40 km) 7.2 ± 2.5 mmol/l. The difference of 0.5 mmol/l between the two lactate values during the time trial was not statistically significant, and they showed a significant correlation ($r=0.89$, $p<0.00001$).

The heart rates of each individual during the time trial are shown in Fig. 1. The mean heart rate during the first and second part showed no significant difference (173.7 ± 7.1 and 174.3 ± 8.1 , respectively). The mean heart rate in the last 2 min before the two blood samples showed no significant difference, nor was there a difference between the previous periods (173.4 ± 7.6 and 174.2 ± 7.4 , respectively).

There was no significant difference between cyclists and triathletes considering height, weight, percentage of body fat, or speeds during the time trial. Considering the lactate levels at rest and during the time trial, there was no significant difference between triathletes and cyclists. The heart rates during the time trial did not show significant differences between the 2 groups. We observed no correlation between anthropometrics, speed, resting levels of lactate, or lactate levels during the time trial.

The most important observation in this study was that the mean plasma lactate level during steady-state exercise was extremely high. The large level of individual variability is conspicuous (range 3.2–12.2 mmol/l). Three triathletes and two cyclists main-

Fig. 2. The resting levels and the mean of the two levels of lactate during the time trial.

tained plasma levels which exceeded 10 mmol/l or more for almost 1 h. These values exceeded all previous lactate levels at AT, LT, IAT and MLSS observed in a laboratory [1, 4, 8, 11–14]. Since it is known that there are differences in the lactate concentration of whole blood and plasma, this may have influenced the comparison with other studies [15]. However, in the studies we compared, lactate was assessed in plasma [11–14] as well as whole blood [4, 8, 10]. In addition, it would be theoretically possible that some subjects did not reach their maximal steady state during the time trial for psychological reasons. This possibility makes our data even more remarkable.

The time trial in the current study is considered to be a steady-state exercise since the course run time and heart rate remained constant during the whole time trial. Since all subjects were experienced cyclists to ride a time trial at a maximal steady-state intensity, the measured lactate values during the time trial should approximate MLSS.

Only a few investigators have described lactate levels assessed during a field test. Beneke *et al.* [7] reported blood lactate levels of 4–6.5 mmol/l in well-trained speed skaters. Pages *et al.* [8] described lactate values of 4.95 ± 0.5 mmol/l in seven triathletes during a triathlon race, which is considerably lower than our observations. However, their triathletes were only moderately trained and the duration of the total triathlon had an average of 2.50 h, so the lactate values cannot be considered maximal steady-state values.

Several authors have evaluated lactate response to steady-state exercise in laboratories. For 8 elite cyclists, Jenkins and Quigley [10] observed a mean lactate concentration of 8.9 ± 1.6 mmol/l on a cycle ergometer during 30 min of exercise. However, for 6 subjects, the

workload used in this experiment was decreased during the test when the initial workload could not be maintained. For this reason, these lactate values cannot be interpreted as steady-state values, but the observations were an indication that endurance-trained cyclists can tolerate high blood lactate levels for at least 30 min. Coyle *et al.* [5] observed lactate values of 7.2 ± 0.7 mmol/l in 15 elite cyclists during a laboratory test with a constant workload. However, the lactate values of Coyle *et al.* [5] are not quite comparable with our data since the standard deviation is significantly lower than ours. Furthermore, the initial workload in Coyle's study was altered during this test.

This study was a field test study. When we compare laboratory observations from other investigators with the field test in this study, we have to consider that environmental factors like temperature and humidity may influence the comparison [16].

The results of this study are not easily explained. According to many authors, lactate is seen as a dead-end waste product of metabolism. The resulting acidosis is viewed as the major factor which causes fatigue and exhaustion during exercise [2, 3]. However, several studies have indicated that lactate is not a waste dead-end metabolite during exercise and recovery [17, 18]. In contrast, the results of these studies strongly suggest that lactate plays an important role in metabolism during exercise [19, 20]. Lactate which is produced in areas with high glycogenolytic and glycolytic activity (type IIb fibres) is oxidized in areas with high oxidative activity (type I and IIa fibres). Brooks called this phenomenon the lactate shuttle [18]. Lactate reaches the oxidative areas by diffusion [21] or by circulation. The lactate levels during steady-state exercise are the result of the production and elimination of lactate [22], so these levels do not reflect the net turnover of lactate. A second function of lactate is maintaining levels of blood glucose as a precursor of liver gluconeogenesis [22]. The glycogen synthesis in the liver is lowered during exercise by decreased perfusion. In addition, during exercise, glucagon levels increase and insulin levels decrease, resulting in an inhibiting effect on glycogen synthesis in the liver [23].

The role of lactate as an acid is still a controversial issue. Acidosis is assumed to inhibit ATP regeneration [16, 24]. There have been indications that acidosis inhibits the enzyme phosphofructokinase, which is involved in glycolysis [24]. In addition, another consequence of acidosis is a shift of the oxyhaemoglobin dissociation curve to the right, allowing O_2 to unload oxygen from haemoglobine [25]. Furthermore, the decreasing pH results in a vasodilatation of the vascular

bed in the contracting muscle [25]. As a result, both phenomena allow an increasing amount of oxygen to the tissues and better endurance performance. Summarizing, it may be favourable to have high levels of lactate in combination with a high buffer capacity. The physiological explanation for the accumulation of lactate during exercise remains to be elucidated [2]. To explain this phenomenon, local hypoxia, increasing recruitment of type-II fibres and a shift in the pyruvate-lactate balance has been described [2].

This study proved that endurance-trained athletes can maintain extremely high levels of lactate during steady-state endurance exercise. Our data suggest an individual lactate tolerance with a great individual variation. Of course, high levels of lactate during steady-state exercise do not prove that lactate plays a positive role, but the high values of plasma lactate concentration in this study certainly do not support a negative role. We hypothesized that high levels of lactate during exercise may be necessary to provide a concentration gradient for some tissues to maintain the higher metabolic demand (for example, the heart and contracting muscles). This hypothesis may be supported by recent studies in which measurements of lactate concentrations may be very useful to indicate early overtraining [26, 27]: low submaximum and maximum lactate levels are believed to be indicators of overtraining, which is associated with decreased performance.

In our opinion, the results of this study are in contradiction with those of previous investigators and the different definitions of lactate response to exercise. According to these definitions and observations, the lactate concentration during steady-state exercise is expected to be within defined levels (see Introduction). Therefore, it must be concluded that these definitions may not be valid for a practical situation. This may be caused by the fact that, in most studies, the different definitions were based upon empiricism and different scientific backgrounds. Furthermore, most studies were not evaluated for a practical situation or involved moderately trained subjects.

Summarizing, we hypothesized that lactate may not be a waste product but a major metabolite during steady-state exercise. It is possible, and may be favourable or even necessary for an athlete to have high levels of lactate during steady-state exercise. This view is very important in sport medical practice since lactate response has been accepted as a measure of endurance capacity [6, 28]. Our data strongly suggest that the practical usefulness of these parameters is less significant than has been assumed.

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