Effect of Low-Intensity Leg Exercise on Ventilatory Threshold during Incremental Arm Exercise

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Introduction

It is known that lactate, the level of which is increased during exercise, disappears more rapidly during low-intensity exercise than during passive recovery (Belcastro and Bonen, 1975; Stamford et al., 1981). This is because lactate can be used as an energy source during low-intensity exercise. If lactate can be used in such a way, it is supposed that the lactate that is produced during high-intensity exercise would disappear more rapidly during simultaneous low-intensity exercise. Therefore, in this study, we had subjects perform both incremental arm exercise and low-intensity steady-state leg exercise at the same time. This protocol was adopted for the following reason. During incremental exercise, if lactate elimination is affected by simultaneous leg exercise, it is supposed that the lactate threshold (LT), the point at which lactate production exceeds elimination, would also be affected. However, because arm exercise was performed, it was difficult to obtain blood samples from the fingertip. Therefore, we used respiratory gas kinetics to examine the effect of low-intensity leg exercise on ventilatory threshold (VT) during incremental arm exercise.

Methods

Eight healthy male subjects with a mean age of 20 – 1.7 years, a mean weight of 64.7 – 5.4 kg, and a mean height of 174 – 4.8 cm participated in this study. Three experiments were performed: 1) incremental leg exercise (LE), 2) incremental arm exercise (AE), and 3) incremental arm exercise together with low-intensity steady-state leg exercise (combined exercise: CE). During LE, the subject performed leg cycling in an upright position with his arms at his sides using a cycle ergometer (Ergometer 232C: Combi). First, the subjects remained in a resting state on the ergometer for 5 min. Then, after 4 min of zero-load cycling, the work load was increased by 20 W per minute until exhaustion. During AE, the center of the ergometer (Ergometer 232CXL: Combi) shaft was positioned at the level of the subject’s shoulder, and the subject performed arm cranking in an upright position. The incremental load exercise was the same as that during LE. The protocol for CE was the same as that for AE except that the subjects performed simultaneous steady-state leg exercise. Steady-state leg exercise was performed at 30% of the peak oxygen uptake (VO₂ peak) recorded during LE until the arm exercise finished. All exercises were performed at 60 rpm. The subject performed each exercise once a day.

Respiratory gas exchange data were collected throughout the 5-min rest period, throughout the exercise test and for 10 min after the exercise by the breath-by-breath method using a computerized system (Aeromonitor AE-208S: Minato). Then, the average oxygen uptake (VO₂) and carbon dioxide output (VCO₂) for each 20-sec interval was calculated.

We plotted VCO₂ against VO₂, and took the ventilatory threshold (VT) to be the point at which the VCO₂ began to increase rapidly (V-slope method; Beaver et al., 1986).

We used a paired student’s t-test to compare the variables of the two groups. A value of p<0.05 was regarded as significant. The strength of the relationships between dependent and independent variables were expressed by pearson’s correlation coefficient.

Results

Table 1 summarizes the average values and standard deviations of work rate and VO₂ at the VT, work rate at exhaustion (peak work rate), peak oxygen uptake (VO₂ peak), and the slope of the VCO₂-VO₂ relationship obtained below and above VT during all exercise modes. Significant differences (p<0.01) were observed between the slope below VT and the slope above VT during all exercise modes. During CE, VO₂ at the VT was higher than that during AE because of VO₂ on steady state-leg exercise. Peak work rate during CE tended to be lower than that during AE. However, for 6 out of the 8 subjects, the work rate at the VT during CE was higher than that during AE. For the two remaining subjects, the values were slightly

Table 1: Summary of the Average Values and Standard Deviations of Work Rate and VO₂ at the VT, Work Rate at Exhaustion, and the Slope of the VCO₂-VO₂ Relationship.

<table>
<thead>
<tr>
<th>Exercise Mode</th>
<th>Work Rate at VT (W)</th>
<th>VO₂ at VT (mL/kg/min)</th>
<th>Work Rate at Exhaustion (W)</th>
<th>VO₂ peak (mL/kg/min)</th>
<th>VCO₂-Slope Below VT</th>
<th>VCO₂-Slope Above VT</th>
</tr>
</thead>
<tbody>
<tr>
<td>LE</td>
<td>20.5</td>
<td>40.2</td>
<td>400</td>
<td>80.0</td>
<td>0.04</td>
<td>0.02</td>
</tr>
<tr>
<td>AE</td>
<td>20.0</td>
<td>40.0</td>
<td>400</td>
<td>80.0</td>
<td>0.04</td>
<td>0.02</td>
</tr>
<tr>
<td>CE</td>
<td>20.5</td>
<td>40.5</td>
<td>390</td>
<td>78.0</td>
<td>0.05</td>
<td>0.03</td>
</tr>
</tbody>
</table>
lower during CE. Therefore, no significant difference was observed in the work rate at the VT between AE and CE. However, when we observed the relationship between the difference in work rate at the VT between AE and CE (Δwork rate) and the ratio of VO₂ at the VT to VO₂ peak during LE (%VO₂ peak-Leg), a significant positive correlation (r=0.78, p<0.05) was observed between Δwork rate and %VO₂ peak-Leg (Fig. 1).

**Discussion**

Lactate that is generated in active muscles is diffused into the blood, and then taken into liver, inactive muscles or into active muscles performing low-intensity exercise. Throughout this process, lactate is removed from muscle engaged in high-intensity exercise. It is thought that the blood lactate concentration is determined by the rate of systemic lactate production and elimination (Stanley et al., 1985). Since production exceeds elimination, the blood lactate concentration rises during incremental exercise. In this study, we had subjects perform CE under the assumption that if lactate elimination is affected by low-intensity steady-state leg exercise, the VT would rise.

In this study, we set the intensity of the steady-state leg exercise at 30%VO₂ peak recorded during LE. As a result, a rise in the VT was not observed in all subjects. However, it seemed that the higher the VT level was during LE, the larger was the rise in the VT during CE. The VT during LE was determined by the rate of systemic lactate production and elimination; the higher the rate of elimination is, the higher the VT can be. Therefore, it is possible that lactate generated during arm exercise can be removed at a higher rate in the subjects who have a higher VT level during LE. On the other hand, it is supposed that since the capacity of the legs for lactate elimination was low, no rise in the VT was observed in subjects whose VT level during LE was 30–40%VO₂ peak.

It is thought that subjects in whom lactate generation is inhibited also have a higher VT level. Therefore, the rise in the VT level during CE does not necessarily demonstrate that low-intensity leg exercise enhanced lactate elimination. Further study is required to clarify this process.

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