THE RATE OF PERIPHERAL CIRCULATORY ADJUSTMENT AT THE ONSET OF EXERCISE

Atsuko Kagaya

Abstract

The rate of peripheral circulatory adjustment at the onset of exercise was studied in six active women aged 20–22 yrs. Five bouts of exercise with different durations of 15, 30, 60, 120 and 180 s were performed on a level treadmill at an intensity of 70% V̇O₂max. Calf blood flow was measured with a mercury-in-rubber strain gauge plethysmograph immediately after cessation of each bout of exercise. Heart rate and V̇O₂ were measured during running.

Calf blood flow after 15-s exercise increased to 25.98 ± 3.51 ml/100 ml/min (mean ± SD), which corresponded to 70% of the mean calf blood flow (32.80 ml/100 ml/min) immediately after 180-s exercise. The relative increases in calf blood flow after 15- and 30-s exercise were significantly higher than those of heart rate. The V̇O₂ after exercise of identical duration showed a smaller percentage increase in comparison with heart rate.

The increase of calf blood flow or heart rate was fitted best by a monoexponential equation, Yt=C-ae⁻kt, where Yt is the response at time t(s) expressed as a percentage of the value at 180-s exercise, C is 100 in the present study, and k is a rate constant. The rate constant k in the equations ranged from 0.025 to 0.179 for blood flow and 0.025 to 0.036 for the heart rate. The calculated half-times (t₁/₂) for the increase in blood flow averaged 12.85 s, ranging from 5.6 to 20.0 s. This was significantly (p<0.05) shorter than HRt₁/₂, which averaged 21.7 s.

The present study therefore showed that the adjustment of the peripheral circulatory system at the commencement of treadmill running at an intensity of 70% V̇O₂max preceded the central circulatory adjustment.

key words: Rate of adjustment, Calf blood flow, V̇O₂ kinetics.

I. Introduction

Many investigators have studied the kinetics of V̇O₂ at the commencement of exercise. The rate of change in aerobic energy release has been shown to depend on the work intensity, prior exercise state, muscle groups involved in the exercise, age, state of training, and aerobic capacity of the subjects. Among the physiological factors associated with the rate of increase in oxygen uptake, many studies have indicated a faster rise in cardiac output (Q) or heart rate. The rate of adjustment of the oxygen-transporting system has been suggested to be important as a limiting factor of V̇O₂ kinetics at the onset of exercise. Sady reported that in children the adjustment of the heart rate was faster than that of V̇O₂ and that the rapid increase in heart rate contributed to the faster rise of V̇O₂. In contrast, several authors have suggested that the rate of increase in V̇O₂ at the start of exercise is limited by the utilization of oxygen at the muscle, which is reflected in the rate of oxygen extraction from the blood. Recently Hughson and colleagues discussed these two conflicting mechanisms for limiting the rate of V̇O₂ increase at the start of exercise, and suggested that oxygen transport limits V̇O₂ kinetics. Before determining which is responsible for the limitation of V̇O₂ kinetics, the capacity...
of oxygen transport to the muscle or the ability of the muscle to utilize oxygen, the rate of peripheral adjustment at the onset of exercise should be examined, because the amount of oxygen which can be utilized at the working muscle will be determined by the peripheral distribution of cardiac output. However, in previous studies on the rate of circulatory adjustment, the rate of cardiac output or heart rate increase has been determined as an index of oxygen-transporting ability and very few studies have included the rate of peripheral circulatory adjustment upon commencement of exercise22, 28).

The purpose of the present study, therefore, was to compare the relative rates of peripheral and central circulatory adjustment at the onset of treadmill running at an intensity of 70% \( \dot{V}O_2 \) max.

II. Subjects and Methods

A. Subjects

Six female physical education students participated as subjects in the present study. Their mean (±SD) age, height and body weight were 21.3 ± 0.98 yrs., 160.2 ± 5.89 cm and 57.7 ± 7.48 kg, respectively. \( \dot{V}O_2 \) max of the present subjects determined on an inclined treadmill (3°) was 43.5 (±3.62) ml/kg/min on average.

B. Experimental Procedure and Methods

To determine the HR-\( \dot{V}O_2 \) relation, subjects performed a 12-min, progressive run on a level treadmill at three different speeds for 4 minutes each. On a different day, \( \dot{V}O_2 \) max was determined on an inclined treadmill.

Within 2 weeks after measurements of the HR-\( \dot{V}O_2 \) relation and \( \dot{V}O_2 \) max, the subjects ran on a level treadmill at an intensity of 70% \( \dot{V}O_2 \) max. Five bouts of running with durations of 15, 30, 60, 120 and 180 s were performed. Following 5-min resting measurement in the sitting position on a treadmill, the subjects ran for 15 sec. A specially devised chair enabled subjects to sit over the treadmill belt, so that they could start exercise immediately after resting measurement. The other 4 runs were performed successively. At least 5 min of rest followed each exercise period, so that calf blood flow and heart rate returned to the pre-exercise resting levels before the runs of different duration.

Expired air was collected in a Douglas bag for the last 30 s of each running except for 15-s running, when collection was done for 15 s. A sample of expired air was analyzed for \( O_2 \) and \( CO_2 \) concentrations by an electrical expired gas analyzer (SANEI C. O. 1H 21 A) calibrated with Scholander's micro-gas analyzer. ECG was recorded continuously for heart rate determination.

Calf blood flow was measured within 5 s after cessation of exercise in a sitting position on the treadmill by means of a mercury-in-rubber strain gauge plethysmograph.

C. Calculation of the Rate of Adjustment

To determine the rate of cardio-respiratory adjustment to exercise, data were fitted by non-linear least squares regression to a single component, exponential equation; \( Y_t = C - ae^{-kt} \), where \( Y_t \) is the value at time \( t(s) \), \( C \) is a steady state value and \( k \) is a rate constant8, 28, 33). In the present study, \( Y_t \) is the relative increase expressed as a percentage of increase in 180-s exercise (C=100). The \( t \frac{1}{2} \) for the heart rate (HRt \( \frac{1}{2} \)) and blood flow (BFt \( \frac{1}{2} \)) was calculated as the time for the responses to achieve one-half of the difference between the resting and peak values for 180-s exercise.
III. Results

The mean values of calf blood flow at rest and immediately after running for 15, 30, 60, 120, or 180 s are presented in Table 1 and Fig. 1. Calf blood flow increased to 25.98±3.51 ml/100 ml/min immediately after 15-s running, and reached 32.80 ml/100 ml/min after 180-s running. Significant differences were found between the means of blood flow at rest and after 15-s exercise (p<0.001), and after 15-s and 180-s exercise (p<0.05). There were no significant differences among the mean values of blood flow after 30-, 60-, 120- and 180-s exercise. HR counted from the R-R interval at a time identical to that of blood flow measurement averaged 148.4 beats/min at the end of the 180-s exercise (Table 1). Mean \( \dot{V}_O_2 \) was 28.1 ml/kg/min for the last 30 s of 180-sec exercise.

The relative increase of each parameter at a given time was calculated as a percentage of the peak value (for \( \dot{V}_O_2 \) and HR) or immediate post-exercise value (for blood flow) for 180-s exercise. After 15-s exercise, calf blood flow

<table>
<thead>
<tr>
<th>Exercise time</th>
<th>( \dot{V}_O_2 ) (ml/kg/min)</th>
<th>Heart Rate (beats/min)</th>
<th>Calf Blood Flow (ml/100 ml/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rest</td>
<td>3.10±0.33</td>
<td>64.78±11.52</td>
<td>1.20±0.36</td>
</tr>
<tr>
<td>15 sec</td>
<td>9.73±3.72</td>
<td>100.33±7.69</td>
<td>25.98±3.51</td>
</tr>
<tr>
<td>30</td>
<td>11.93±0.92</td>
<td>106.23±7.89</td>
<td>31.00±10.37</td>
</tr>
<tr>
<td>60</td>
<td>22.90±2.30</td>
<td>130.87±3.16</td>
<td>34.49±6.86</td>
</tr>
<tr>
<td>120</td>
<td>27.55±3.05</td>
<td>143.97±7.22</td>
<td>35.00±12.81</td>
</tr>
<tr>
<td>180</td>
<td>28.10±3.82</td>
<td>148.43±7.32</td>
<td>32.80±5.92</td>
</tr>
</tbody>
</table>

Mean±SD (N=6)

![Fig. 1. Calf blood flow at rest and immediately after running for 15, 30, 60, 120 and 180 s (Mean±SD, N=6).](image-url)
increased to 70.1% of the immediate post-exercise flow after 180-s running, while the relative increase in HR at the corresponding time was 42.5%. This difference in the ratio of increase between blood flow and HR at 15 s was statistically significant (p<0.01). The difference at 30 s was also significant (p<0.01). The relative increase for \( \dot{V}O_2 \) for 30-s exercise was only 35.7%, which was significantly lower (p<0.05) than that shown by heart rate (49.4%). The difference for 60-s exercise was also significant.

When the relative increase above the resting level was plotted as a function of time, an exponential rise was shown for calf blood flow as well as heart rate. A typical rise in calf blood flow is illustrated in Fig. 2 for one subject, T. S., and is compared with the change in heart rate. Values of both calf blood flow and heart rate were fitted best by a monoexponential equation, \( Y_t = C - ae^{-kt} \). The rate constant \( k \) in the equation ranged from 0.025 to 0.179 for the blood flow and from 0.025 to 0.036 for the heart rate, respectively. The calculated \( t_{1/2} \) for blood flow (\( BFt_{1/2} \)) ranged from 5.6 s to 20.0 s,

<table>
<thead>
<tr>
<th>Subj</th>
<th>( BFt_{1/2} ) (sec)</th>
<th>( HRt_{1/2} ) (sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>K, H</td>
<td>5.6</td>
<td>21.3</td>
</tr>
<tr>
<td>Y, H</td>
<td>12.3</td>
<td>24.6</td>
</tr>
<tr>
<td>T, T</td>
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<td>T, S</td>
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<tr>
<td>N, M</td>
<td>20.0</td>
<td>25.9</td>
</tr>
<tr>
<td>S, T</td>
<td>16.2</td>
<td>13.9</td>
</tr>
<tr>
<td>Mean</td>
<td>12.85</td>
<td>21.65</td>
</tr>
<tr>
<td>SD</td>
<td>4.97</td>
<td>4.35</td>
</tr>
</tbody>
</table>

Fig. 3. The rate of adjustment for calf blood flow (\( BFt_{1/2} \)) and heart rate (\( HRt_{1/2} \)) (mean±SD),
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and averaged 12.85 (SD = 4.97) s (Table 2). As shown in Fig. 3, the mean BFt 1/2 was significantly shorter than the HRt 1/2 (p < 0.05), which averaged 21.7 s with a range of 13.9 to 25.9 s. No significant correlation coefficient was obtained between the half-times for these two parameters.

IV. Discussion

The magnitude of the increase in blood flow during exercise changes with the intensity of muscle contraction. During walking or running, calf blood flow increases linearly in relation to the running/walking speed or %Vo2max, and levels off at higher speeds. In the present study, running at an intensity of 70% Vo2max caused an increase in blood flow to 32.80 ml/100 ml/min. In comparison with previous findings, this result seems reasonable.

The purpose of the present study was to compare the rate of peripheral and central circulatory adjustment upon commencement of running at 70% Vo2max. The half-time calculated as an index of the rate of adjustment averaged 12.9 s for calf blood flow. This was significantly shorter than the t 1/2 for heart rate. This result confirms that the adjustment occurring in the peripheral circulatory system is faster than the central one and is in agreement with the findings of Kagaya and Hata and Koch, who investigated these parameters in children, and with the results of Pendergast et al., who reported a rapid adjustment of muscle blood supply upon commencement of arm or leg exercise. However, the time required for the peripheral adjustment in the present study was a little slower than that reported by Pendergast et al. (1980). They reported that adjustment of muscle blood flow was complete within 6 to 18 s in fit subjects. The most likely explanations for this discrepancy would include differences in the physical characteristics of the subjects, the type and intensity of exercise used in the experiments, and the position of the subjects during measurement of blood flow. In the present study, blood flow was measured in an upright position after running, whereas in the study by Pendergast et al., it was measured in a supine position during arm or leg exercise.

Since an increase in heart rate affects the Qt 1/2, a slower adjustment of the heart rate indicates a slower increase in cardiac output. Therefore, the present study showed that the blood supply to the working muscle was peripherally adjusted within a short time after the start of exercise, and that the blood flow requirement of the muscle was met before the central circulatory system had adjusted to the given exercise intensity.

Among the physiological factors which affect Vo2 kinetics at the start of exercise, a number of investigations have demonstrated the importance of circulatory adjustment as an index of oxygen-transporting ability. In contrast, Cerretelli et al. and Pendergast et al. indicated that oxygen utilization by the working muscle would limit Vo2 kinetics at the commencement of exercise. In discussing these two conflicting mechanisms, Hughson and Morrissey concluded that the ability to transport oxygen to the working muscle would limit the increase in Vo2. Their argument was based on their own findings that reduction of the increase in heart rate by β-blockade produced slower Vo2 kinetics, and faster Vo2 kinetics with the prior circulatory occlusion of non-working leg muscles. However, the hypothesis that Vo2 kinetics are controlled by the ability to transport oxygen to the working muscle has been supported by observations of central circulatory adjustment, and only a few
investigators have attended direct measurement of peripheral circulatory adjustment. In the present study, peripheral blood flow was measured to determine whether any time difference existed between the rate of increase in central circulation and the rate of increase in the peripheral distribution of blood to the working muscle. The results obtained indicated that the working muscle was able to receive an adequate blood supply at the start of exercise before cardiac output had increased to an adequate degree. Although there is some question about rapid vasodilatation at the onset of exercise\textsuperscript{15}, Corcondilas et al.\textsuperscript{5} have already demonstrated that a brief forearm contraction of 0.3 s caused vasodilatation, in both healthy and sympathectomized subjects. They suggested that this rapid vasodilatation was due to release of local metabolites. Such rapid vasodilatation could possibly be caused by constriction of non-working vascular beds\textsuperscript{1,9,33}. This hypothesis is apparently supported by the findings of Iwamoto et al.\textsuperscript{19}, who found that commencement of voluntary muscular exercise was accompanied by an immediate increase in blood pressure. If immediate vasodilatation occurs in the working muscle without any vasoconstriction in other organs before an increase in $Q$, then blood pressure should decrease.

The results of the present study suggest that the working muscle had sufficient blood supply while $\dot{V}O_2$ was still increasing. Therefore, the delayed rate of oxygen extraction could be one of the factors limiting $\dot{V}O_2$ kinetics at the start of exercise. There is further indirect evidence in the literature that oxygen extraction at the start of exercise is delayed. Raynaud et al.\textsuperscript{30} reported that oxygen delivery to the tissues ($\dot{Q}$, $CaO_2$) increased abruptly while $Cvo_2$ decreased slowly. A recent study by Henriksson et al.\textsuperscript{111} indicated that muscle NADH increased maximally in the first few seconds of muscle contraction at two-thirds of MVC. This increase would reflect a reduced mitochondrial redox state at the start of exercise, or muscle hypoxia\textsuperscript{22}. From these findings presented above, it seems probable that the rate of oxygen extraction, or oxygen utilization at the working muscle is one of the rate-limiting factors for $\dot{V}O_2$ kinetics at the onset of exercise. The rate of oxygen utilization would be influenced by capillary density, myoglobin concentration, and enzymatic oxidative potential\textsuperscript{12,29}. The cellular level of ADP, which triggers the rise in oxidative phosphorylation in mitochondria, is also important\textsuperscript{29}.

In the present study, $\dot{V}O_2$ was measured at the mouth, and was compared with peripheral circulation in the calf muscles. The muscle groups involved in running are not limited to the calf muscles. Therefore, the circulation to other muscles which influence the total body oxygen intake ($\dot{V}O_2$) should be investigated to explain the time difference between peripheral circulatory adjustment and $\dot{V}O_2$ increase.

In relation to individual differences in $\dot{V}O_2t \frac{1}{2}$, Hagberg et al.\textsuperscript{10} found that $\dot{V}O_2$ adaptation at the onset of exercise was rapid in highly trained athletes. The aerobic capacity of studied subjects\textsuperscript{7,15,27,29,34}, anaerobic threshold\textsuperscript{27} and maximal peripheral blood flow immediately following a combination of arterial occlusion and toe-raising exercise\textsuperscript{27} were significantly related to $\dot{V}O_2$ kinetics upon commencement of exercise. A significant relation has been found between $\dot{V}O_2max$ and $\dot{V}O_2t \frac{1}{2}$, and suggested that $\dot{V}O_2$ kinetics are influenced, at least partly, by genetics\textsuperscript{29}. However, no relation has yet been established between the rate of peripheral circulatory adjustment and $\dot{V}O_2t \frac{1}{2}$. Since the present study was not designed to determine the relation between
BFt $\frac{1}{2}$ and $\dot{V}O_2t\frac{1}{2}$, the role of peripheral circulatory adjustment in determining $\dot{V}O_2t\frac{1}{2}$ remains to be investigated.

In conclusion, the peripheral circulatory adjustment in exercising muscle was found to precede the central circulatory adjustment at the onset of running at an intensity of 70% $\dot{V}O_2$ max.

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