Effect of Active Recovery on Intracellular pH Following Muscle Contraction, A $^{31}$P-MRS Study

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The effect of active recovery after intense muscular exercise was examined by $^{31}$P-MRS. Seven healthy males participated in this study, and their right wrist flexor muscles were examined. Each subject flexed the right wrist at 60% of the maximum voluntary contraction (MVC) until the intracellular pH in the wrist flexor muscle decreased to approximately 6.4. This was followed either by active recovery (AR) which consisted of 5, 10, 20, 30 or 40%MVC exercise, and by passive recovery (PR) during a 10-min-recovery period. The intracellular pH (pHi) recovered faster during AR at each condition than during PR. Besides, from the 2nd to the 5th min of the recovery period, the pHi was significantly higher during AR than during PR. The quadratic regression curve of pHi recovery during the 10-min-recovery-period against the %MVC was obtained. The optimal contraction intensity determined from this curve was 8.7% MVC for a 10-min-recovery-period. The optimal levels were determined for another recovery duration within 10 min, and the level decreased with the prolonged recovery duration. These data suggest that mild exercise is an effective maneuver to promote the recovery from intracellular metabolic acidosis, and that the intensity of the recovery exercise should be determined according to the cooling down duration or the resting interval before the subsequent exercise performance. (Ann. Physiol. Anthropol. 12(3): 173-179, 1993)

Key words: Active Recovery, $^{31}$P-MRS, Intracellular pH

Intense muscular exercise results in the production of lactate. Accumulation of lactate causes metabolic acidosis which presumably is one of the important factors in the development of muscle fatigue (Kirkendall, 1990). Our previous $^{31}$P magnetic resonance spectroscopy ($^{31}$P-MRS) study on human muscle indicated that muscle fatigue was deeply related to the metabolic acidosis (Iwanaga et al., 1991). It is, therefore, critical for the recovery from fatigue to eliminate rapidly the blood lactate and to restore the intracellular pH.

Jervell (1928) showed that moderate muscular exercise in the recovery period (active recovery) allowed faster lactate removal compared with resting conditions (passive recovery). This observation was verified by several other investigators. The optimal exercise intensity for active recovery has been well studied. Davies (1970) indicated that recovery exercise at the intensity ranging between 35 and 45% of the maximum $O_2$ uptake ($V_{O_{2\max}}$) was effective in promoting blood lactate disappearance. Belcastro and Bonen (1975) also reported that the optimal level of exercise for active recovery was 32% of $V_{O_{2\max}}$. However, the effects of the active recovery on the intracellular pH are uncertain. The optimal exercise intensity required for the intracel-
ular pH recovery also remains undetermined. $^{31}$P-MRS has recently been used for the noninvasive and repeated measurement of the ATP, inorganic phosphate, phosphocreatine level as well as the intracellular pH (Kent-Braun et al., 1990; Sairyo et al., 1991; Taylor et al., 1983). The purpose of this study was to investigate the effect of active recovery on the intracellular pH and to examine the optimal exercise intensity for the recovery in the intracellular pH by using the $^{31}$P-MRS.

**METHODS**

Seven healthy Japanese male volunteers participated in this study. Following an explanation of the procedures to be used, a written informed consent was obtained from all participants. The right wrist flexor muscles were examined. Pertinent data describing the subjects are presented in Table 1.

<table>
<thead>
<tr>
<th>SUBJECT</th>
<th>AGE (yrs)</th>
<th>HEIGHT (cm)</th>
<th>WEIGHT (kg)</th>
<th>COFA (cm)</th>
<th>MVC (Nm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>26</td>
<td>163.5</td>
<td>62.0</td>
<td>26.0</td>
<td>27.7</td>
</tr>
<tr>
<td>B</td>
<td>32</td>
<td>171.0</td>
<td>62.8</td>
<td>26.8</td>
<td>29.9</td>
</tr>
<tr>
<td>C</td>
<td>25</td>
<td>167.0</td>
<td>57.7</td>
<td>26.2</td>
<td>29.9</td>
</tr>
<tr>
<td>D</td>
<td>30</td>
<td>173.5</td>
<td>84.5</td>
<td>30.4</td>
<td>33.7</td>
</tr>
<tr>
<td>E</td>
<td>33</td>
<td>174.0</td>
<td>62.0</td>
<td>26.0</td>
<td>25.4</td>
</tr>
<tr>
<td>F</td>
<td>33</td>
<td>171.2</td>
<td>60.3</td>
<td>25.0</td>
<td>24.7</td>
</tr>
<tr>
<td>G</td>
<td>33</td>
<td>177.3</td>
<td>75.2</td>
<td>29.4</td>
<td>40.5</td>
</tr>
</tbody>
</table>

Each subject placed his right forearm on a surface coil in a superconducting magnet. Exercise consisted of wrist flexion over an arc against a handle connected to an isokinetic ergometer (Cybex II) (Kent-Braun et al., 1990). The subjects had to control the level of power output by watching the force trace displayed on an oscilloscope. Prior to each experiment, each subject was instructed to exert the maximum voluntary effort in wrist flexion to measure the maximum voluntary contraction (MVC). The highest force of 5 contractions was defined the MVC strength. After a 2-min-rest, each subject flexed his wrist in the isokinetic manner (30 degrees per sec) every 2 sec at 60%MVC (exercise period) until the intracellular pH decreased to approximately 6.4. This was followed by a 10-min-recovery period, during which the subject either rested for passive recovery (PR) or continued wrist flexion exercises at a power level of 5, 10, 20, 30 or 40% MVC for active recovery (AR).

The magnetic resonance spectrometer used in this study had a 28cm bore, 1.9 Tesla superconducting magnet (BEM 250/80, Otuka Electronics). Phosphorus signals were collected with an optimal pulse width (55 μsec) every 2 sec. Data consisted of 30 scans taken at rest and during exercise as well as the recovery period. To evaluate energy metabolism, we calculated the phosphocreatine (PCr) to inorganic phosphate (Pi) ratio (PCr/Pi) using the areas of these peaks in the spectra. The chemical shift (δppm) between Pi and PCr was used to determine the intracellular pH (pHi) by the following equation (Taylor et al., 1983):

$$pHi = 6.75 + log([\delta - 3.27]/(5.69 - \delta))$$

Statistical analysis was made using multiple comparisons of Fisher's least significant differences (Snedecor and Cochran, 1968). A p<0.05 was taken as the significant.

**RESULTS**

Fig. 1 shows the changes in pHi during AR at the 6 recovery conditions. The pHi began to recover immediately after starting AR at 20, 30 and 40% MVC, whereas that in AR at 5 and 10%MVC and in PR decreased still further 1 min after exercise and increased thereafter. The pHi recovered more rapidly during AR at each condition than during PR. The most rapid restoration of pHi occurred from the 2nd to the 5th min during AR at each condition, which were significantly higher than those during PR (p<0.05) (Table 2).

The PCr/Pi changes during AR and PR are shown in Fig. 2. Each value of the PCr/Pi decreased to the lowest level of approximately 0.1 at the end of the exercise period at 60%MVC, and then gradually increased during recovery period, recovery rate of
Fig. 1 Changes in intracellular pH during the recovery period.
ex.: exercise period; AR: active recovery;
PR: passive recovery.
Each point represents the mean value and the bars show the standard error.

Fig. 2 Changes in the PCR/Pi during the recovery period.
PCR: phosphocreatine; Pi: inorganic phosphate.
Each point represents the mean value and the bars show the standard error.
Table 2  Statistical analysis by Multiple comparisons of Fisher's least significant difference of the differences in intracellular pH under any of the conditions for AR and that in PR.
* shows a significant difference (p<0.05) compared with PR.
ex, exercise period; ns, not significant.

<table>
<thead>
<tr>
<th></th>
<th>rest</th>
<th>ex</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10 (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>5%MVC</td>
<td>ns</td>
<td>ns</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>ns</td>
</tr>
<tr>
<td>10%MVC</td>
<td>ns</td>
<td>ns</td>
<td>ns</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>ns</td>
</tr>
<tr>
<td>20%MVC</td>
<td>ns</td>
<td>ns</td>
<td>ns</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>ns</td>
</tr>
<tr>
<td>30%MVC</td>
<td>ns</td>
<td>ns</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>ns</td>
</tr>
<tr>
<td>40%MVC</td>
<td>ns</td>
<td>ns</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>*</td>
<td>ns</td>
</tr>
</tbody>
</table>

Fig. 3  Relationship between the relative power output during AR and the recovery rate of intracellular pH (ΔpH) during the recovery period.

which was faster during passive recovery than during active recovery.

Fig. 3 shows the relationship between power output (%MVC) during AR and recovery in the pH (ΔpH) at the 10th min of recovery period. There was a significant relationship in the quadratic regression curve between %MVC and ΔpH (p<0.01). From this regression equation, the peak ΔpH was predicted to
Table 3  Regression equations and optimal contraction levels expressed as %MVC for each duration of AR lasting 1 to 10 minutes.
DUR.: duration of recovery time;
OPT LEVEL: optimal contraction level;

<table>
<thead>
<tr>
<th>DUR. (min)</th>
<th>regression equation</th>
<th>OPT. (%MVC)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>$Y = -2.50 \times 10^{-4}X^2 +1.28 \times 10^{-2}X-8.99 \times 10^2$  $(r=-0.52, p&lt;0.01)$</td>
<td>25.7</td>
</tr>
<tr>
<td>2</td>
<td>$Y = -3.84 \times 10^{-4}X^2 +1.89 \times 10^{-2}X-7.02 \times 10^2$  $(r=-0.61, p&lt;0.01)$</td>
<td>24.6</td>
</tr>
<tr>
<td>3</td>
<td>$Y = -5.52 \times 10^{-4}X^2 +2.54 \times 10^{-2}X+6.72 \times 10^2$  $(r=-0.63, p&lt;0.01)$</td>
<td>23.2</td>
</tr>
<tr>
<td>4</td>
<td>$Y = -6.68 \times 10^{-4}X^2 +2.73 \times 10^{-2}X+0.14$  $(r=-0.58, p&lt;0.01)$</td>
<td>20.5</td>
</tr>
<tr>
<td>5</td>
<td>$Y = -6.47 \times 10^{-4}X^2 +2.56 \times 10^{-2}X+0.20$  $(r=-0.58, p&lt;0.01)$</td>
<td>19.8</td>
</tr>
<tr>
<td>6</td>
<td>$Y = -4.90 \times 10^{-4}X^2 +1.82 \times 10^{-2}X+0.31$  $(r=-0.49, p&lt;0.01)$</td>
<td>18.6</td>
</tr>
<tr>
<td>7</td>
<td>$Y = -4.75 \times 10^{-4}X^2 +1.62 \times 10^{-2}X+0.38$  $(r=-0.52, p&lt;0.01)$</td>
<td>17.0</td>
</tr>
<tr>
<td>8</td>
<td>$Y = -3.43 \times 10^{-4}X^2 +8.93 \times 10^{-2}X+0.47$  $(r=-0.52, p&lt;0.01)$</td>
<td>13.0</td>
</tr>
<tr>
<td>9</td>
<td>$Y = -2.71 \times 10^{-4}X^2 +6.49 \times 10^{-3}X+0.51$  $(r=-0.48, p&lt;0.01)$</td>
<td>12.0</td>
</tr>
<tr>
<td>10</td>
<td>$Y = -2.34 \times 10^{-4}X^2 +4.05 \times 10^{-3}X+0.55$  $(r=-0.52, p&lt;0.01)$</td>
<td>8.6</td>
</tr>
</tbody>
</table>

Fig. 4  Optimal contraction levels expressed as %MVC for AR according to the duration of AR.

Occur at 8.7%MVC, which was expressed as the optimal contraction level for recovery exercise when the recovery period was set at 10 min. Similarly, there was a significant relationship between %MVC and ΔpH at any time of the recovery period (Table 3), and the optimal %MVC level decreased with the prolonged recovery time (Fig. 4); for example, the most rapid recovery in intracellular pH with a 1 min recovery exercise appeared to be at the 25.7%MVC level, that with a 2 min recovery exercise at the 24.6%MVC level and so on.

**DISCUSSION**

In the present study, the effect of active recovery on the intracellular pH and high-energy phosphates following intense muscle contractions was evaluated by $^{31}$P-MRS. Active recovery was sufficient to elicit the recovery from the metabolic acidosis following intense muscle contraction, the optimal level of which was varied by the active recovery duration. Sahlin (1986) demonstrated that a decrease in the muscular intracellular pH during exercise was closely related to the lactate production due to glycolysis. Therefore, lactate disappearance in the muscle may produce the recovery of the decreased intracellular pH. The intracellular pH recovery during AR, therefore, might be provided by promoting the biochemical lactate removal and/or by increasing the efflux of lactate from the intracellular space into the extracellular space. Gluconeogenesis and oxidation in the tricarboxylic cycle highly determine the biochemical lactate removal (Rontoyannis, 1990). Oxidation within the
skeletal muscle is considered to play a main role in the metabolic pathway for lactate removal (Hatta, 1990; Rontoyannis, 1990). Tesch and Wright (1983) demonstrated that the rate of lactate disappearance from the muscle was influenced by its capillary density. Minotti et al. (1990) measured forearm blood flow during the submaximum exercise by using venous occlusion plethysmography, and showed the increased blood flow with the reinforced exercise intensity. Based on the above view of the intracellular pH recovery, active recovery may be capable of increasing the forearm blood flow, resulting in enhancing the oxygen supply to the muscle tissue and further activate the aerobic metabolic pathway, thereby accelerate the oxidation of the lactate in exercising muscles.

Previous investigations on blood lactate concentration have demonstrated that an optimal recovery exercise intensity allows rapid lactate removal (Belcastro and Bonen, 1975; Bonen and Belcastro, 1976; Davies and Belcastro, 1970; McLellan and Skinner, 1982). Davies (1970) indicated that recovery exercise at the intensity ranging between 35 and 45% of \( V_{O_{2}}\max \) was effective in promoting blood lactate disappearance. Belcastro and Bonen (1976) also reported that the optimal level of the active recovery was 32% of \( V_{O_{2}}\max \). However, the resting interval before the subsequent exercise was not postulated in their reports. In our study, the optimal intensity for the intracellular pH recovery was found to be in relation to the cooling down duration, being 25.7% MVC for a 1-min period interval, 24.6% MVC for a 2-min, and 8.7% MVC for a 10-min. In addition, these results were relevant to determine the optimal intensity for the resting interval before subsequent performance. Weltman et al. (1977) revealed the enhanced subsequent exercise performance following active recovery. Therefore, if the recovery exercise intensity is programmed in relation to the resting interval, the active recovery greatly reinforces the following performance for which recovery exercise intensity should be adjusted according to the resting interval.

Weltman et al. (1977) also suggested that although active recovery enhanced lactate removal, it was not sufficient to explain the efficacy of active recovery for the subsequent performance. In the present study, although active recovery enhanced the intracellular pH recovery, that was not found to have good effect on the recovery of high-energy phosphate which was evaluated by changes in PCr/Pi ratio. These results indicated that the optimal intensity for recovery in intracellular pH might not be always optimal intensity for the subsequent performance.

In general, athletes are likely to perform recovery exercise as a decremental manner. The optimal exercise intensity obtained with this \( ^{31}P \)-MRS study decreased with the prolongation of the recovery period, suggesting that decremental power output might be crucial for the intracellular pH recovery.

In conclusion, active recovery promotes recovery in intracellular pH after intense muscle exercise, for which the optimal intensity are determined in relation to the duration of either cooling down or resting interval before the subsequent exercise performance.

**REFERENCES**


Table 1 Pertinent data describing the subjects.

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