Effect of Muscle Strength Training and Muscle Endurance Training on Muscle Deoxygenation Level and Endurance Performance

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Abstract. [Purpose] The purpose of this study was to compare the effects of muscle strength training and muscle endurance training on muscle deoxygenation level and endurance performance. [Subjects and Methods] Nineteen healthy young men were randomly assigned to a muscle strength training (STR: n = 6) group, muscle endurance training (END: n = 6) group, or a control (CON: n = 7) group. The training intensity for STR was 60°/sec × 10 repetitions × 5 sets/day and that for END was 240°/sec × 50% fatigue repetitions × 2 sets/day, 3 days/week, for 6 weeks. All subjects performed cardiopulmonary exercise testing (CPX) to measure maximum oxygen uptake, exercise time and muscle deoxygenation level of vastus lateralis, and underwent muscle strength and muscle endurance measurements pre- and post-training. [Results] In the STR group, muscle strength tended to increase, while muscle endurance significantly increased in the END group. Muscle deoxygenation level was significantly increased in both training groups. Maximum oxygen uptake did not change; however, in the END group alone, exercise time was significantly prolonged. [Conclusion] These results suggest that muscle endurance training is more effective at increasing endurance performance than muscle strength training.

Key words: Muscle endurance training, Muscle deoxygenation level, Exercise time

INTRODUCTION

It has been shown that muscle oxidative capacity, measured by skeletal muscle mitochondrion content and oxidative enzyme activity, is decreased with hypoactivity owing to cardiac disease, respiratory disorder, and ageing1–3). As a consequence, endurance performance, e.g. maximum oxygen uptake (peak VO₂) and exercise time, declines.

On the other hand, it has been reported that exercise training increases skeletal muscle mitochondrion content and oxidative enzyme activity4), increasing oxygen extraction from arterioles and capillaries in muscle. Consequently, endurance performance improves with an increase in aerobic adenosine triphosphate production and a decrease in lactic acid production in muscle and blood during exercise5). However, it is currently unclear which training is the most effective at increasing oxygen extraction.

Recently, near-infrared spectroscopy (NIRS) has been used to evaluate oxygen extraction of skeletal muscle. NIRS can monitor the tissue oxygenation of the region of interest, non-invasively and continuously, through different absorptions of near-infrared light in the wavelength region of 700–1000 nm by oxygenated hemoglobin/myoglobin (oxy-Hb/Mb) and deoxygenated hemoglobin/myoglobin (deoxy-Hb/Mb)6–7). Since this technique was introduced by Jobsis in 19778), who monitored the changes in the absorption of light in cats and humans, it has been widely applied in various clinical situations and experiments, which have confirmed its validity and reliability9,10) . In 1992, Chance et al. monitored the skeletal muscle oxygenation during exercise11). Many researchers have used this technique to evaluate the oxygenation in brain and muscle during dynamic exercise, such as incremental and constant work rate exercise. However, there is limited evidence for the effects of exercise training on muscle oxygenation responses during exercise6), and furthermore, to our knowledge, there is no evidence based on comparisons of the effects among two or more different training groups.

In this study, we hypothesized that muscle endurance training could improve muscle deoxygenation level and endurance performance, but muscle strength training could not. The purpose of this study was to compare the effects of muscle strength training (STR) and muscle endurance
training (END) using muscle deoxygenation level, measured by NIRS, and endurance performance (peak VO₂ and exercise time).

SUBJECTS AND METHODS

Subjects

Nineteen young men (age = 22.9 ± 2.3 yr) who gave their written informed consent participated in this study. All subjects were healthy with no known orthopedic or cardiorespiratory disease. Participants were randomly assigned to a muscle strength training (STR: n = 6) group, a muscle endurance training (END: n = 6) group, or a control group (CON: n = 7). Their physical characteristics and fitness habits are shown in Table 1. All subjects answered our questionnaires about activity in daily life and were instructed to continue with their activities as usual until their training was completed. Subjects were also given instructions to report to us when they performed other physical exercise. This study was approved by Kanazawa University Ethics Committee.

Methods

All subjects performed incremental exercise using an upright electromagnetically braked cycle ergometer (Rehcor 500P, Lode B.V., Netherlands) to determine muscle deoxygenation level, peak VO₂, and exercise time before and after training. The height of the saddle was set to flex the knee joint of each subject at 40° at the bottom dead center of the pedal. After a warm-up exercise of 3 min at 10 W, incremental exercise began, which increased progressively by 20 W every minute. Subjects were instructed to maintain a speed of 60 revolutions per minute (rpm) during the exercise. The incremental exercise was terminated when subjects could not maintain 50 rpm. Exercise time was determined from the start to the finish of the incremental exercise. Heart rate was monitored continuously with an electrocardiogram (DynaScope DS-2202, Fukuda Denshi, Japan) during the exercise. Blood pressure was also measured using a mercury sphygmomanometer at 1 min intervals.

Expired gas analysis (AE-300S, Minato Medical Science Co., Ltd., Japan) was used to measure oxygen uptake (VO₂) and carbon dioxide output (VCO₂) during incremental exercise. This equipment can continuously monitor VO₂ and VCO₂ using a breath-by-breath method under the control of a microcomputer, which integrated a gas analyzer (O₂ and CO₂ concentration meter) and a respiratory flow meter. Gas meter calibration was performed using standardized gas of known concentration before each test. The collected VO₂ data was averaged every 8 sec and peak VO₂ was defined as the average VO₂ value during the last 30 sec of the test. All subjects were instructed not to take caffeine, alcohol, or to smoke for one day before the test. In addition, they were prohibited from eating and drinking (only water was permitted) for 2 hours before the test on the test day.

Muscle deoxygenation level was measured using a NIRS device (OM-220, Shimadzu Co., Japan). This device is composed of a main computer and a probe, and the probe has a light source and two detectors. The light source emits near-infrared light at two different wavelengths (760 nm and 830 nm) in turn, which penetrates the biological tissue with scattering and absorption and then reaches the detectors. This device can evaluate the hemoglobin concentration in biological tissue by a spatially resolved method based on diffusion theory. The distances between the emitter and two detectors are 2.5 cm and 4.0 cm, respectively. It has been shown that the depth of penetration of the light can be calculated by the distances between emitter and detector. In this device, the depth is considered to be 20–30 mm.

The probe was placed on the VL muscle of the non-dominant leg, which is the agonist in cycling, 5 finger-breadths proximally from patella and on the outside of the thigh. To prevent misalignment of the probe during the exercise and irruption of light from outside, the probe was fixed on the site of interest using black packing tape and Velcro.

Generally, a spectral photometer can quantify the concentration of materials on the basis of the Beer-Lambert Law when a specimen is transparent and dilute, and the path length and extinction coefficient of the materials are known. However, it is impossible to measure the path length of the light in biological tissue because it is a scattering media. Therefore, it is difficult to evaluate the absolute value of tissue oxygenation. In this study, therefore, muscle deoxygenation level was normalized by the femoral artery occlusion method before incremental exercise. A tourniquet was twisted around the proximal femur vertically, then each subject was subjected to pressure at 300 mmHg for about 7 min until their changes in oxy-Hb/Mb and deoxy-Hb/Mb (Δoxy-Hb/Mb and Δdeoxy-Hb/Mb, respectively) became constant. Because deoxy-Hb/Mb can be regarded as being essentially insensitive to change in blood volume during occlusion and exercise, we employed this to indicate muscle deoxygenation level.

Muscle deoxygenation level was calculated by the following equation:

Muscle deoxygenation level (%) = ΔEx/ΔOc (see Fig. 1),

where ΔEx is the change in Δdeoxy-Hb/Mb during incremental exercise from start to maximum value and ΔOc is the change in Δdeoxy-Hb/Mb during occlusion from start to maximum value. The signal was recorded at a sampling frequency of 1 Hz and averaged every 10 sec.

Knee extensor strength and endurance of the non-dominant leg were measured using an isokinetic dynamometer (Cybex Norm, Cybex International Inc., USA) 3–7 days after CPX. Subjects were seated and securely strapped at the pelvis, trunk, and thigh. The axis of the dynamometer was aligned at the pivot of the knee joint. First, muscle strength measurement was performed for 3 trials at an angular velocity of 60°/s. Ten minutes later, muscle endurance measurement was performed for 60 trials at 240°/s.

Peak torque obtained from muscle strength measurement was divided by body weight. Muscle endurance was determined by the endurance ratio, which was computed by dividing the total work from the second half of the
repetitions performed by the total work of the first half, then multiplying the result by 100. Fifty percent of fatigue repetitions was also computed and employed on muscle endurance training (see below); this was the number of repetitions performed before 50% of maximum work in the first 3 repetitions was reached.

Subjects were supervised and given verbal encouragement by investigators during both measurements. After the training period, the same measurements were performed by the same investigators.

Training was initiated 7 days after the pre-training measurements and consisted of either 18 sessions of STR or END 3 days/week, for 6 weeks, with each training session separated by 1–2 days of rest. Subjects were prohibited from performing training for 3 consecutive days and training sessions were spaced by more than 3 days. Training was performed using Cybex Norm. Subjects were supervised and given encouragement by investigators during training sessions.

Each STR session consisted of 5 sets of 10 repetitions of

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Table 1. Physical characteristics of subjects pre-training

<table>
<thead>
<tr>
<th></th>
<th>CON (n = 7)</th>
<th>STR (n = 6)</th>
<th>END (n = 6)</th>
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<tbody>
<tr>
<td>Age (years)</td>
<td>21.7 ± 1.9</td>
<td>23.0 ± 2.0</td>
<td>24.2 ± 2.6</td>
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<tr>
<td>Height (m)</td>
<td>1.70 ± 0.07</td>
<td>1.76 ± 0.05</td>
<td>1.76 ± 0.04</td>
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<td>Body weight (kg)</td>
<td>59.3 ± 9.8</td>
<td>64.9 ± 9.3</td>
<td>64.4 ± 9.8</td>
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<tr>
<td>Body mass index (kg/m^3)</td>
<td>20.3 ± 1.8</td>
<td>21.0 ± 2.1</td>
<td>20.7 ± 2.9</td>
</tr>
<tr>
<td>Body fat percentage (%)</td>
<td>15.8 ± 2.6</td>
<td>17.5 ± 3.6</td>
<td>18.0 ± 5.3</td>
</tr>
<tr>
<td>Muscle strength (N•m/kg)</td>
<td>2.8 ± 0.4</td>
<td>2.7 ± 0.7</td>
<td>3.0 ± 0.3</td>
</tr>
<tr>
<td>Endurance ratio (%)</td>
<td>53.5 ± 6.1</td>
<td>51.8 ± 6.7</td>
<td>50.2 ± 6.2</td>
</tr>
<tr>
<td>Deoxygenation level (%)</td>
<td>47.5 ± 25.8</td>
<td>38.2 ± 19.2</td>
<td>47.7 ± 18.2</td>
</tr>
<tr>
<td>Peak VO₂ (ml/min/kg)</td>
<td>39.1 ± 8.2</td>
<td>36.8 ± 6.1</td>
<td>36.1 ± 5.6</td>
</tr>
<tr>
<td>Exercise time (sec)</td>
<td>581.1 ± 160.6</td>
<td>584.0 ± 74.9</td>
<td>591.5 ± 54.6</td>
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Fitness habit

<table>
<thead>
<tr>
<th>CON</th>
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<tr>
<td>No</td>
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<td>5</td>
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<tr>
<td>Yes</td>
<td>2</td>
<td>1</td>
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Values are expressed as mean ± SD. Differences in age (F = 2.05, p = 0.16), height (F = 2.41, p = 0.12), body weight (F = 0.67, p = 0.53), body mass index (F = 0.14, p = 0.87), body fat percentage (F = 0.52, p = 0.60), muscle strength (F = 0.39, p = 0.68), endurance ratio (F = 0.45, p = 0.65), muscle deoxygenation level (F = 0.38, p = 0.69), peak VO₂ (F = 0.34, p = 0.72), and exercise time (F = 0.02, p = 0.99) among the groups were examined by one-way ANOVA. Fitness habit was analyzed by the chi-square test. CON: control group, STR: muscle strength training group, END: muscle endurance training group.

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Fig.1. A typical example of the kinetics of Δdeoxy-Hb/Mb of vastus lateralis (VL) muscle during occlusion and incremental exercise. W-up: warm up. Muscle deoxygenation level was calculated by dividing the changes in Δdeoxy-Hb/Mb during incremental exercise by changes during occlusion.
knee extension at an angular velocity of 60°/s. Each set was separated by 1 min recovery time.

Each END session consisted of 2 sets of 50% fatigue repetitions of knee extension at 240°/s, separated by 1 min recovery time. Subjects performed muscle endurance measurement every 2 weeks during the training period to readjust the number of repetitions.

Subjects of the CON group were instructed to continue their normal daily living for 6 weeks.

Statistical analysis was performed using SPSS for Windows 11.0 J (SPSS Inc., 1989–2001). A repeated two-way ANOVA was used to analyze the interactions of muscle strength, muscle endurance, muscle deoxygenation level, peak VO2, and exercise time between training group and training period. When an interaction was not found, differences in each parameter between pre- and post-training were compared by the paired t-test. Differences between the STR and END groups in terms of change ratio of each parameter were compared by the unpaired t-test. A p value less than 5% was considered statistically significant.

RESULTS

Subjects’ physical characteristics and each parameter of pre-training are shown in Table 1. There was no significant difference in the physical characteristics and each parameter among the 3 groups. These results suggest that subjects of each group had similar physical function and endurance performance. All subjects continued with their normal daily living activities during the training period. The numbers of training sessions were 15.7 ± 2.6 sessions (mean ± SD, 13–17 sessions) in the STR group and 16.3 ± 2.6 in the END group (14–18 sessions). No significant difference between the training groups was found in the number of training sessions (p = 0.50, unpaired t-test).

Table 2 shows the results of two-way ANOVA on muscle deoxygenation level, peak VO2, and exercise time. A main effect of time was found for muscle deoxygenation level and exercise time. However, there was no interaction between group and time for any of the parameters. No effect was found on peak VO2.

Table 3 shows changes in parameters at pre- and post-training in each group.

In the STR group, muscle strength tended to increase (p = 0.09) after 6 weeks of STR training but muscle endurance did not, while muscle endurance significantly increased after 6 weeks of END training but muscle strength did not. Muscle deoxygenation level was significantly increased in both training groups. Maximum oxygen uptake did not change in either training group; however, in the END group alone, exercise time was significantly prolonged.

There were no significant changes in any parameter of the CON group.

DISCUSSION

In this study, we compared the effects of muscle strength training (STR) and muscle endurance training (END) on muscle deoxygenation level measured by NIRS, and endurance performance (i.e. peak VO2 and exercise time). The major results of the present study are as follows: 1) according to the training specificity, STR tended to increase muscle strength (p = 0.09) but did not change muscle endurance, while END significantly increased muscle...
endurance but did not change muscle strength; 2) muscle
deoxygenation level during incremental exercise was
significantly increased by STR and END, and there was no
difference between the two training groups in its change
ratio; 3) maximum oxygen uptake did not change in the STR
and END groups, but exercise time was significantly
prolonged in the END group.

In the present study, STR tended to increase muscle
strength (p = 0.09), but muscle endurance did not change;
END significantly increased muscle endurance but muscle
strength did not change. Thus, we can say that both training
methods employed in this study had an effect on skeletal
muscle.

Limited studies have reported the effect of training on
local muscle oxygenation measured by NIRS 17–20) .
Furthermore, most of these studies used oxy-Hb/Mb to
evaluate the local muscle oxygenation during exercise 17–18)
or the recovery time after occlusion 19). In this study, we used
deoxy-Hb/Mb to evaluate muscle deoxygenation level
because this signal is regarded as being essentially
insensitive to change in blood volume 15). McKay et al. 20)
also used change in deoxygenated hemoglobin/myoglobin
(Δ[HHb]) to evaluate local muscle oxygenation of VL
muscle for a similar reason. They examined the change in
time constants for pulmonary oxygen uptake (τVop2) and
muscle oxygenation (τΔ[HHb]) during moderate constant
loading exercise after 8 sessions of high-intensity interval
training (8–12 × 1 min intervals at 120% maximal oxygen
uptake separated by 1 min of rest) or 8 sessions of low-
intensity endurance training (90–120 min at 65% maximal
oxygen uptake) performed by 12 subjects. They observed a
significant reduction in τVop2 in both training groups, but
τΔ[HHb] did not change in either groups. In the present
study, we observed a significant increase in muscle
deoxygenation level. One of the reasons why we observed
an improvement in muscle deoxygenation level is that we
conducted about twice the number of training sessions as
McKay et al. (16.0 ± 1.6; mean ± SD vs. 8 training sessions,
respectively). In addition, subjects in our training groups
performed training focused on the quadriceps femoris
muscle using Cybex. Thus, we assume that the training
effect on VL muscle in this study was larger than that in the
experiment of McKay et al. using a cycle ergometer. Thus,
we consider that training performed by the STR and END
groups in this study increased muscle deoxygenation level.

Neary et al. 18) hypothesized that muscle deoxygenation
observed in their subjects, who demonstrated significant
ddeoxygenation, was related to a number of peripheral
factors including a increased capillarization, mitochondrion
density, and activity of oxidative enzymes, all of which
likely augmented the arterial - venous oxygen content
difference as a result of training. As for the adaptation of
mitochondria to whole endurance training (i.e. cycling or
running), Holloszy 21) reported that whole endurance
training improved the capacities of pyruvate oxidation and
oxidative enzymes, such as cytochrome c oxidase, which
doubled in soleus muscle of rats, and also that the run time
to exhaustion was prolonged (29 ± 3 min vs. 186 ± 18 min,
control rats vs. trained rats, respectively). Burrelle and
Hochachka 4 also reported that citrate synthase and
cytochrome c oxidase were significantly activated by 4
weeks of treadmill running. However, there is little evidence
of the adaptation of mitochondrion content and oxidative
enzyme activation to muscle endurance training (i.e.
focused on the local muscle).

We hypothesized that only END improved muscle
deoxygenation level because it has been reported that
resistance training usually reduces mitochondrion content
and oxidative enzyme capacity 22,23). However, both STR
and END resulted in significant and similar improvement in

<p>| Table 3. The results of each parameter pre- and post-training |
|---------------------------------|-----------------|-----------------|
| | Muscle strength (N•m/kg) | Endurance ratio (%) |</p>
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<tbody>
<tr>
<td>CON</td>
<td>2.8 ± 0.4</td>
<td>2.9 ± 0.4</td>
<td>53.5 ± 6.1</td>
<td>51.7 ± 7.9</td>
</tr>
<tr>
<td>STR</td>
<td>2.7 ± 0.7</td>
<td>3.1 ± 0.6</td>
<td>51.8 ± 6.7</td>
<td>51.6 ± 4.2</td>
</tr>
<tr>
<td>END</td>
<td>3.0 ± 0.3</td>
<td>3.0 ± 0.2</td>
<td>50.2 ± 6.2</td>
<td>57.1 ± 8.1**</td>
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</tbody>
</table>

<p>| | Muscle deoxygenation level (%) | Peak VO₂ (ml/min/kg) |</p>
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<tbody>
<tr>
<td>CON</td>
<td>47.5 ± 25.8</td>
<td>51.8 ± 22.2</td>
<td>39.1 ± 8.2</td>
<td>39.3 ± 9.2</td>
</tr>
<tr>
<td>STR</td>
<td>38.2 ± 19.2</td>
<td>53.2 ± 18.3*</td>
<td>36.8 ± 6.1</td>
<td>38.2 ± 4.3</td>
</tr>
<tr>
<td>END</td>
<td>47.7 ± 18.2</td>
<td>64.9 ± 17.9*</td>
<td>36.1 ± 5.6</td>
<td>40.1 ± 9.0</td>
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<p>| | Exercise time (sec) |</p>
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<tbody>
<tr>
<td>CON</td>
<td>581.1 ± 160.6</td>
<td>589.3 ± 122.2</td>
</tr>
<tr>
<td>STR</td>
<td>584.0 ± 74.9</td>
<td>626.3 ± 58.8</td>
</tr>
<tr>
<td>END</td>
<td>591.5 ± 54.6</td>
<td>641.5 ± 71.3*</td>
</tr>
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</table>

Values are expressed as mean ± SD. CON: control group, STR: muscle strength training group, END: muscle endurance training group. **: p<0.01, *: p<0.05
During incremental exercise, muscle deoxygenation level. Recently, Tang et al. showed that mitochondrial enzymes, such as citrate synthase, were activated by resistance training. Thus, their report indicates that resistance training can improve the skeletal muscle oxidative capacity. We did not evaluate the skeletal muscle oxidative capacity in the present study, but it is possible that our training protocol also increased mitochondrial content and oxidative enzyme capacity. Accordingly, muscle deoxygenation level would have increased with enhanced oxygen extraction by skeletal muscle.

Many studies have reported that whole endurance training improves peak VO\textsubscript{2}. Because of the training adaptations to oxygen delivery capacity, such as structural adaptation (i.e. capillary density) and functional adaptation (i.e. vascular dilation), and oxygen extraction (i.e. arterial-venous oxygen content difference). The results for resistance training are conflicting, showing that it can increase peak VO\textsubscript{2} and that it cannot. The present study demonstrated similarly increasing muscle deoxygenation levels in the STR group and the END group. However, peak VO\textsubscript{2} did not change, contrary to our hypothesis. Several studies have reported that the adaptations of mitochondria and capillaries to exercise training appear to be more pronounced in the regions and muscle fibers recruited during training. Subjects in this study trained unilateral femoral muscles. Therefore, we consider that the adaptations of mitochondria to STR and END in this study were limited in the femoral region. Thus, the training provided in this study appears to have been insufficient to induce a significant increase in peak VO\textsubscript{2} as seen in whole endurance training.

Although exercise time did not change in the STR group, it was significantly prolonged in the END group. Jobrias et al. examined the cellular energetic and structural adaptations of elderly VL muscle to exercise training. Their experiment resulted in a rise in muscle oxidative capacity with both endurance training and resistance training (57% and 31%, respectively) and a decline in glycolytic ATP supply (56%) with endurance training. Their results describe the dissociation of our results; i.e. only END induces a decline in glycolytic ATP production (i.e. rise in aerobic ATP production), delaying the accumulation of lactic acid during exercise, and prolonging exercise time during incremental exercise.

Generally, peak VO\textsubscript{2} is considered the index of maximal exercise capacity. The results of this study suggest that muscle endurance training is more effective at extending exercise time than muscle strength training. Thus, muscle endurance training may increase submaximal exercise capacity safely because it imposes less strain on the cardiopulmonary system than muscle strength training. It may be possible to use these results to benefit patients with cardiorespiratory disease and the elderly, so further investigations are needed.

In conclusion, we compared the effects of muscle strength training and muscle endurance training on muscle deoxygenation level by NIRS and endurance performance (peak VO\textsubscript{2} and exercise time). Muscle deoxygenation level was increased in the STR group and the END group and the change ratios in these groups did not differ. These training regimes appeared insufficient to induce a significant increase in peak VO\textsubscript{2}; however, in the END group, exercise time during incremental exercise was significantly prolonged. Thus, it was indicated that muscle endurance training is more effective at increasing endurance performance than muscle strength training.

**ACKNOWLEDGEMENT**

We are grateful to T. Nakagawa, M. Hosoi, M. Yokogawa, A. Ootsubo, T. Kusudo, N. Hashimoto, as well as to all subjects who were involved in this study.

**REFERENCES**

20. McKay BR, Patterson DH, Kowalchuk JK: Effects of short-term high-


