Short-Term Physical Training Improves Vasodilatory Capacity in Cardiac Patients

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SUMMARY

There have been no previous studies that clearly demonstrate the effects of training on the relation between exercise capacity and vasodilatory capacity in skeletal muscle. This study was performed to clarify the effects of short-term, moderate-intensity physical training on exercise tolerance and vasodilatory capacity in cardiac patients.

We studied 21 patients after acute myocardial infarction, coronary artery bypass grafting, or valve replacement. Each patient performed symptom-limited incremental exercise tests before and after a 2-week training program of moderate-intensity exercise. A cycle ergometer was used for both the training and exercise tests. Blood pressure measurement and respiratory gas analysis were continuously performed during the tests. Cardiac output was also measured using a dye-dilution method at rest and every 2 minutes during incremental exercise. Reactive hyperemic calf blood flow was measured at rest.

After the training program, the subjects attained a significant decrease in systemic vessel resistance and significant increases in oxygen uptake and cardiac output at peak exercise. Changes in reactive hyperemic calf blood flow were significantly correlated with the changes in cardiac output, systemic vascular resistance, and the kinetics of oxygen uptake during warm-up exercise.

By improving the peripheral vasodilatory capacity in these patients, short-term, moderate-intensity physical training was found to improve the cardiovascular adaptation not only at peak exercise, but also during the onset of exercise. (Jpn Heart J 2002; 43: 13-24)

Key words: Physical training, Vasodilatory capacity, Incremental exercise test

The importance of physical training in cardiac patients has been widely recognized.1-9) Though physical training was applied only to patients with ischemic heart disease up until the 1980s,10) it is now used for a variety of cardiac patients, including those who have undergone cardiac surgery.11-13) Foster, et al reported that exercise capacity was increased by physical training in patients who had undergone coronary artery bypass graft surgery (CABG).11) Several other investi-
gators have discussed the significance of physical training even in patients with chronic heart failure.\textsuperscript{1,2,5,7,8} It has also been reported that physical training improves exercise capacity more than conventional medical therapy alone.\textsuperscript{14}

Exercise capacity is mainly determined by central factors such as cardiac reserve and lung function, and peripheral factors including vasodilatory capacity and skeletal muscle function.\textsuperscript{15} The decreased exercise capacity in cardiac patients is partly attributable to these peripheral factors. If the physical training could improve the impaired blood flow to the skeletal muscle in patients with cardiovascular disease as noted by Sinoway,\textit{et al} in normal subjects,\textsuperscript{16} the improvement in peripheral circulation would increase their exercise capacity.\textsuperscript{7} There have been no previous studies that clearly demonstrate the effects of short-term physical training on the relation between exercise capacity and vasodilator capacity in the skeletal muscle in patients, although Hambrecht,\textit{et al} have reported on the effect of long-term physical training.\textsuperscript{17}

This study was designed to clarify the effects of short-term, moderate-intensity physical training on exercise and vasodilatory capacity in patients after cardiac surgery and after acute myocardial infarction (AMI).

**METHODS**

**Study patients:** The study was performed between 1997 and 1999 at the Cardiovascular Institute Hospital in Tokyo, Japan. The subjects included 10 patients after valve replacement (VR), 6 after AMI, and 5 after CABG. The physical and clinical characteristics of the patients are shown in Table I. Patients who could not perform exercise testing due to physical limitations were excluded. None of the patients were athletes, and all of them were sedentary before the study period. At the initiation of the study, every patient was clinically stable and in sinus rhythm. Three patients were taking an angiotensin converting enzyme inhibitor, and 1 patient was prescribed a β-blocker. All AMI and CABG patients were prescribed nitrates, and all VR patients were prescribed diuretics. Medications were not altered during the training period. Post-AMI patients underwent successful percutaneous coronary intervention before starting physical training. The protocol was approved by the Institutional Ethics Committee of the Cardiovascular Institute. The nature and purpose of the study and risks involved were explained, and written informed consent was obtained from all of the patients prior to their enrollment.

**Measurements:** Resting left ventricular ejection fraction was assessed in patients with valve replacement by radionuclide angiography using \textsuperscript{99mTc}-human serum albumin with the first-pass technique. In all of the other patients it was assessed by left ventriculography using the area-length method.
Calf blood flow in the right leg was determined at rest using mercury-in-silastic strain gauge venous occlusion plethysmography (EC 5R plethysmograph, Hokanson, Bellevue, WA, USA). Before measurements, patients rested in a supine position for at least 10 minutes with the right leg slightly elevated. A cuff around the thigh was connected to a rapid cuff inflator and a cuff inflator air source (E-20 and AG-101, respectively, Hokanson). Plethysmography recordings were obtained immediately after inflation of the thigh cuff to 40 mmHg. Two consecutive measurements were performed at rest in both calves, and the mean value was used for the subsequent analysis. Reactive hyperemic calf blood flow was measured immediately after the measurement of resting calf blood flow. The cuff was inflated to 40 mmHg above systolic blood pressure and then maintained for 5 minutes. Two flow measurements were obtained immediately after release of the cuff, and the averaged value was determined as reactive hyperemic calf blood flow. Values on leg blood flow are expressed in milliliter blood flow per 100 mL tissue per minute (mL/100 mL·1/min⁻¹).

Exercise test: A symptom-limited incremental exercise test was performed 1 week after the onset of AMI, VR, or CABG, and then repeated after 2 weeks of physical training. An upright, electromagnetically braked cycle ergometer (CPE-2000, Med Graphics, USA) was used for the exercise testing. After a 4-minute rest on the ergometer, exercise started with a 4-minute warm-up period at 20 watts and 50 rpm, and then the work rate was increased by 1 watt every 6 seconds (ramp pattern). Patients were monitored continuously with a 12-lead electrocardiogram (ML-5000, Fukuda Denshi, Tokyo) throughout the exercise tests. Cuff blood pressures were obtained every minute with an automatic indirect manometer (STBP-760, Colin Denshi, Aichi, Japan). Cardiac output was measured at rest, at the 20-watt warm-up, and every two minutes during the incremental exercise. The measurements were taken by a dye dilution method using indocyanine green dye and an ear photoelectric transducer. The output was analyzed by a cardiac output computer (DDG-2001, Nihon Kohden, Tokyo). Systemic vascular resistance was calculated by dividing the mean blood pressure by cardiac output, and expressed in dynes·cm⁻⁵/sec.

Oxygen uptake (VO₂), carbon dioxide output (VCO₂), and the rate of respiratory airflow were measured during the test using an aeromonitor (AE280S, Minato Medical Science, Osaka, Japan). This system consists of a hot-wire flow meter and oxygen and carbon dioxide gas analyzers (zirconium element-based oxygen analyzer and infrared carbon dioxide analyzer). Gas was sampled at a rate of 220 mL/min⁻¹ with a suction pump and passed through a filter and gas analyzers. VO₂ was calculated in each patient at rest, at the 20-watt of warm-up, and at peak exercise. VO₂ at the anaerobic threshold (AT) was determined as the breakpoint in the VCO₂-VO₂ plot (V-slope method). Peak VO₂ was determined.
as the highest VO₂ attained over a 10 second period during incremental exercise. The slope of the increase in VO₂ to the increase in work rate (ΔVO₂/ΔWR) was calculated by linear regression from one minute after the start of the incremental exercise until AT. The time constant of VO₂ was determined by fitting a single exponential function to the response at the onset of 20-watt exercise, with resting VO₂ as the baseline, using least square nonlinear regression analysis.\(^{18}\)

**Training protocol:** All patients were instructed to perform 30 minutes of physical training twice a day, 5 days a week, for 2 consecutive weeks using a cycle ergometer in the hospital. Physical training was started the day following the first exercise testing. The intensity of physical training was determined on the basis of a preliminary incremental exercise test. Appropriate training intensity was determined for each patient at the heart rate of his or her AT (Table I).

**Statistical analysis:** All data are expressed as the mean±SD. Comparisons of parameters among groups were made using analysis of variance (ANOVA). Parameters before and after 2 weeks of training were compared using Student's paired t-test. A p value <0.05 was considered significant.

### Table 1. Physical Characteristics. Cardiac Diseases, Resting Left Ventricular Ejection Fraction, Oxygen Uptake, and Heart Rate during Incremental Exercise

<table>
<thead>
<tr>
<th>Valvular disease</th>
<th>Patient Age (years)</th>
<th>Sex</th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
<th>Valvular lesion</th>
<th>Infarct site</th>
<th>Angiographic findings</th>
<th>Ejection fraction (%)</th>
<th>VO₂ (mL/min/kg)</th>
<th>Peak VO₂ (mL/min/kg)</th>
<th>Peak HR (beat/min)</th>
<th>Training HR (beat/min)</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>LAD</td>
<td>Lcx</td>
<td>RCA</td>
<td>LMT</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute myocardial infarction</td>
<td>11 64 M 168.0 63.5 inf</td>
<td>100</td>
<td>90</td>
<td>99</td>
<td>56</td>
<td>14.5</td>
<td>16.3</td>
<td>130</td>
<td>119</td>
<td></td>
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<tr>
<td></td>
<td>12 71 M 153.0 49.0 A-S</td>
<td>100</td>
<td>90</td>
<td>99</td>
<td>90</td>
<td>41</td>
<td>11.9</td>
<td>15.2</td>
<td>120</td>
<td>115</td>
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<tr>
<td></td>
<td>13 66 M 161.8 53.2 inf</td>
<td>75</td>
<td>90</td>
<td>99</td>
<td>48</td>
<td>11.9</td>
<td>15.2</td>
<td>120</td>
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<tr>
<td></td>
<td>14 78 M 161.8 56.0 A-S</td>
<td>99</td>
<td>75</td>
<td>75</td>
<td>68</td>
<td>13.4</td>
<td>17.5</td>
<td>126</td>
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<td>15 55 M 160.0 59.0 inf</td>
<td>100</td>
<td>75</td>
<td>50</td>
<td>14.3</td>
<td>17.3</td>
<td>144</td>
<td>125</td>
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<tr>
<td></td>
<td>16 61 M 165.5 62.4 inf</td>
<td>75</td>
<td>100</td>
<td>76</td>
<td>12.1</td>
<td>17.3</td>
<td>114</td>
<td>90</td>
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<tr>
<td>Mean</td>
<td>65.8 161.7 57.2</td>
<td>60.1</td>
<td>2.1</td>
<td>16.8</td>
<td>126</td>
<td>105.2</td>
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<tr>
<td>SD</td>
<td>8.0 5.2</td>
<td>13.4</td>
<td>1.2</td>
<td>0.9</td>
<td>18.5</td>
<td>20.2</td>
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<tr>
<td>Coronary artery graft</td>
<td>17 66 M 162.0 48.2</td>
<td>75</td>
<td>62</td>
<td>10.5</td>
<td>15.7</td>
<td>126</td>
<td>110</td>
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<td></td>
<td>18 49 M 164.0 52.0</td>
<td>75</td>
<td>60</td>
<td>8.9</td>
<td>16.5</td>
<td>120</td>
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<td></td>
<td>19 50 M 168.8 66.0</td>
<td>75</td>
<td>68</td>
<td>11.4</td>
<td>14.8</td>
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<td>100</td>
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<tr>
<td></td>
<td>20 73 F 143.0 47.0 A-S</td>
<td>90</td>
<td>75</td>
<td>99</td>
<td>50</td>
<td>12.0</td>
<td>17.4</td>
<td>118</td>
<td>100</td>
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<tr>
<td></td>
<td>21 67 M 160.0 54.0</td>
<td>100</td>
<td>90</td>
<td>66</td>
<td>10.8</td>
<td>14.2</td>
<td>108</td>
<td>100</td>
<td></td>
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<tr>
<td>Mean</td>
<td>61.0 159.6 53.4</td>
<td>61.7</td>
<td>10.7</td>
<td>15.8</td>
<td>116.0</td>
<td>99.0</td>
<td></td>
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</tr>
<tr>
<td>SD</td>
<td>7.0 9.8</td>
<td>7.6</td>
<td>1.2</td>
<td>1.2</td>
<td>7.9</td>
<td>8.9</td>
<td></td>
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</tbody>
</table>

M=male; F=female; AS=aortic valve stenosis; MR=mitral valve regurgitation; MS=mitral valve stenosis; TR=tricuspid valve regurgitation; MR=mitral valve stenosis and regurgitation; ASR=Aortic valve stenosis and regurgitation; Inf=inferior; A-S=anteroseptal; LAD=Left anterior descending coronary artery; Lcx=left circumflex coronary artery, RCA=right coronary artery; LMT=left main trunk, AT=anaerobic threshold; HR=Heart rate; VO₂=oxygent uptake. Significant coronary stenosis was defined as ≥ 75% reduction in luminal diameter of a coronary vessel.
RESULTS

All of the patients safely completed 2 weeks of physical training. In the incremental exercise tests prior to physical training, the end point was leg fatigue in 17 patients and shortness of breath in the other 4 patients. In the tests after physical training, it was leg fatigue in 15 patients and shortness of breath in the other 6. Significant ST depression was demonstrated during exercise testing both before and after physical training in 2 patients (1 with CABG and 1 with VR).

**Effects of physical training on resting cardiac function and calf blood flow:** Both heart rate and systolic blood pressure were significantly decreased after 2 weeks of physical training (Table II). Stroke volume was increased from 42.9±7.6 to 46.7±9.2 mL ($p<$0.05). Reactive hyperemic calf blood flow was significantly increased from 14.9±4.0 to 17.7±4.5 mL/min/100 mL tissue ($p<$0.001) (Figure 1).

**Effects of physical training on cardiac function during 20-watt warm-up exercise:**

Table III shows cardiac function during 20-watt warm-up exercise before and after the 2 weeks of physical training. Heart rate and systolic blood pressure during the 20-watt exercise were decreased significantly, while diastolic blood pressure did not change markedly. VO$_2$ during the warm-up exercise was decreased from 9.4±1.9 to 8.5±1.7 mL/min/kg ($p<$0.001). The time constant of VO$_2$ was shortened significantly from 59.4±17.9 to 44.8±16.8 seconds ($p<$0.001). Cardiac output and stroke volume were significantly increased by physical training. Systemic vascular resistance decreased remarkably from 1507.3±404.1 to 1317.9±319.4 dynes⋅cm$^5$/sec ($p<$0.001) (Table III).

**Effects of physical training on exercise capacity:** AT was increased significantly from 10.9±2.1 to 11.7±1.7 mL/min/kg ($p<$0.05) (Table IV). The peak work rate

| Table II. Resting Hemodynamic Data before and after 2 Weeks of Physical Training in All Patients |
|-----------------------------------|------------------|-----------------|--------|
| Heart rate (beat/min)            | 89.1±12.0        | 83.4±11.5       | 0.001  |
| Systolic blood pressure (mmHg)   | 127.7±17.0       | 119.2±14.6      | 0.010  |
| Diastolic blood pressure (mmHg)  | 73.6±10.1        | 70.6±9.2        | NS     |
| Oxygen uptake (mL/min/kg)        | 4.3±0.7          | 4.1±0.4         | NS     |
| Cardiac output (L/min)           | 3.7±0.6          | 3.8±0.6         | NS     |
| Stroke volume (mL)               | 42.9±7.6         | 46.7±9.2        | 0.031  |
| Systemic vascular resistance (dynes⋅cm$^5$/sec) | 1978.3±357.0 | 1845.6±307.7   | NS     |
| Calf blood flow (mL/min/100 mL tissue) | Rest         | 2.0±0.8         | NS     |
|                                  | Hyperemic       | 14.9±4.0        | <0.001 |

Values are expressed as the mean ± SD. $P$ value was determined by the paired $t$-test.
Figure 1. Effects of physical training on rest and reactive hyperemic calf blood flow. This figure shows a significant increase in the hyperemic calf blood flow (CBF) after physical training. *$p<0.001$ for the hyperemic calf blood flow (paired $t$-test).

Table III. Cardiovascular Variables during 20-watt Warm-up Exercise before and after 2 Weeks of Physical Training

<table>
<thead>
<tr>
<th></th>
<th>Before</th>
<th>After</th>
<th>$P$ value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate (beat/min)</td>
<td>103.1±13.6</td>
<td>94.2±13.5</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>154.5±32.6</td>
<td>142.9±24.7</td>
<td>0.006</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>78.8±12.5</td>
<td>75.7±10.3</td>
<td>NS</td>
</tr>
<tr>
<td>Oxygen uptake (mL/min/kg)</td>
<td>9.4±1.9</td>
<td>8.5±1.7</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Cardiac output (L/min)</td>
<td>5.8±1.5</td>
<td>6.2±1.3</td>
<td>0.026</td>
</tr>
<tr>
<td>Stroke volume (mL)</td>
<td>56.8±14.9</td>
<td>66.3±15.4</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Systemic vessel resistance (dynes $\cdot$ cm$^5$/sec)</td>
<td>1507.3±404.1</td>
<td>1317.9±319.4</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Time constant of $VO_2$ (sec)</td>
<td>59.4±17.9</td>
<td>44.8±16.8</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

$VO_2$=oxygen uptake. Values are expressed as the mean ± SD. $P$ value was determined by the paired $t$-test.

Table IV. Parameters of Exercise Capacity before and after 2 Weeks of Physical Training

<table>
<thead>
<tr>
<th></th>
<th>Before</th>
<th>After</th>
<th>$P$ value</th>
</tr>
</thead>
<tbody>
<tr>
<td>AT (mL/min/kg)</td>
<td>10.9±2.1</td>
<td>11.7±1.7</td>
<td>0.019</td>
</tr>
<tr>
<td>Peak exercise</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Work rate (watts)</td>
<td>58.6±14.0</td>
<td>73.8±14.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Heart rate (beat/min)</td>
<td>123.8±17.0</td>
<td>132.3±18.3</td>
<td>0.001</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>179.3±33.9</td>
<td>179.4±31.4</td>
<td>NS</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>86.0±13.9</td>
<td>87.1±13.5</td>
<td>NS</td>
</tr>
<tr>
<td>Oxygen uptake (mL/min/kg)</td>
<td>15.4±2.2</td>
<td>17.5±2.1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Cardiac output (L/min)</td>
<td>9.2±2.1</td>
<td>11.1±2.3</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Stroke volume (mL)</td>
<td>74.9±16.3</td>
<td>85.2±19.8</td>
<td>0.002</td>
</tr>
<tr>
<td>Systemic vessel resistance (dynes $\cdot$ cm$^5$/sec)</td>
<td>1052.5±248.1</td>
<td>885.8±256.9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>$\Delta VO_2/\Delta WR$ (mL/min/W)</td>
<td>7.5±2.4</td>
<td>9.3±1.5</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

$AT$=anaerobic threshold; $VO_2$=oxygen uptake; $WR$=work rate; $W$=watt. Values are expressed as the mean ± SD. $P$ value was determined by the paired $t$-test.
and peak heart rate were both significantly increased by physical training ($p<0.01$). The peak VO$_2$ and peak cardiac output were also increased by training. The peak stroke volume increased from $74.9 \pm 16.3$ to $85.2 \pm 19.8$ mL ($p<0.001$), and the peak systemic vascular resistance decreased from $1052.5 \pm 248.1$ to $885.8 \pm 256.9$ dynes•cm$^5$/sec ($p<0.001$). $\Delta$VO$_2$/WR was increased significantly by physical training from $7.5 \pm 2.4$ to $9.3 \pm 1.5$ mL/min/W ($p<0.001$).

**Relation between reactive hyperemic calf blood flow and exercise parameters:**

There was a significant negative correlation between the change in reactive hyperemic calf blood flow ($\Delta$CBF) and the change in the time constant during the 2 weeks of physical training ($r=-0.68$, $p<0.001$) (Figure 2). However, $\Delta$CBF was not significantly correlated with the change in AT, peak VO$_2$, or $\Delta$VO$_2$/WR. There was a significant positive correlation between $\Delta$CBF and the change in warm-up cardiac output ($r=0.60$, $p<0.01$), but there was no correlation between $\Delta$CBF and the change in rest and peak cardiac output (Figure 3). There was a significant negative correlation between $\Delta$CBF and the change of warm-up systemic

![Figure 2](#)

**Figure 2.** Relationship between the change in the time constant of oxygen uptake (VO$_2$) and the change in reactive hyperemic calf blood flow (CBF) during 2 weeks of physical training. The change in value was defined as a delta ($\Delta$) value. There was a significant negative correlation between the $\Delta$time constant of VO$_2$ and $\Delta$CBF ($r=-0.68$, $p<0.001$).
vascular resistance \( r = -0.73, \ p < 0.001 \), but there was no correlation between \( \Delta \text{CBF} \) and the change in rest and peak systemic vascular resistance (Figure 4).

**Figure 3.** Relationship between the change in reactive hyperemic calf blood flow (CBF) and the change in cardiac output (CO) by the physical training. There was a significant positive correlation between \( \Delta \text{CBF} \) and the change in warm-up cardiac output \( (r = 0.60, \ p < 0.01) \).

**Figure 4.** Relationship between the change in reactive hyperemic calf blood flow (CBF) and the change in systemic vascular resistance (SVR) by the physical training. There was a significant negative correlation between \( \Delta \text{CBF} \) and the change in warm-up SVR \( (r = -0.73, \ p < 0.001) \).
DISCUSSION

Main findings: In the present study, we found that short-term physical training at the AT level safely and significantly increased exercise capacity in patients after AMI or after cardiac surgery. We also newly demonstrated that the shortening of VO$_2$ kinetics at the beginning of exercise, a change that reflects improved cardiovascular adaptation,$^{19}$ is closely related to the improvement in peripheral vasodilatory capacity in patients who perform short-term physical training.

Intensity of physical training: The intensity of physical training has been a matter of debate, especially in patients with heart failure. In its guideline, the World Health Organization recommends a regular and moderate exercise regimen based on each patient's exercise capacity.$^{20}$ In our study, we determined the intensity of physical training according to the AT level for each patient.$^{21,22}$ Koike, et al demonstrated that left ventricular ejection fraction, a parameter that initially increases during incremental exercise, started to decrease at the AT level in cardiac patients.$^{23}$ This phenomenon indicates that exercise-induced deterioration in left ventricular function occurs at work rates above AT in these patients. With work rates below or equal to AT, patients can sustain exercise for a prolonged period since exercise at this level does not seem to accompany increases in plasma lactate and catecholamine concentrations or the development of an acid-base imbalance.

Effects of physical training on cardiovascular function: Inappropriate distribution of blood flow to exercising muscles has been implicated as an important mechanism of exercise limitation in cardiac patients.$^{2,24-32}$ Decreased muscular power as well as metabolic and endocrine disorders are also known as factors of exercise limitation. $^{9,33-37}$ In 1968, Zelis, et al reported a decreased vasodilatory capacity in cardiac patients.$^{23}$ Since then, a number of investigations have been made on this matter. Hattori, et al reported that reactive hyperemic calf blood flow is decreased remarkably in cardiac patients.$^{38}$ The present study is the first to report on the relation between an improvement in vasodilatory capacity and the shortening of VO$_2$ kinetics during the onset of exercise after short-term physical training in cardiac patients.

There are several reports on the improvement of vasodilatory capacity and endothelial function by long-term physical training in cardiac patients.$^{1,5,8,39}$ Although endothelial function was not evaluated in the present study, improvement in vasodilatory capacity after physical training may be related to a change in endothelial function and/or nitric oxide production.$^{8,39,40}$ Sullivan, et al noted that long-term physical training significantly increases the blood flow and arteriovenous oxygen difference in exercising muscles in patients with congestive heart failure.$^{1}$ In the present study, both VO$_2$ and cardiac output at peak exercise
were significantly increased by short-term, low intensity physical training. Fur-
thermore, the change in vasodilatory capacity was significantly correlated with
the changes in three parameters at warm-up exercise, namely cardiac output, sys-
temic vessel resistance, and the time constant of VO$_2$. These results indicate that
the improvement of peripheral blood flow by physical training facilitated muscle
O$_2$ uptake at the beginning of exercise. The improved exercise capacity could
probably be imputed to the decreased systemic vascular resistance together with
the improved cardiac output response reflected by the shorter time constant of
VO$_2$. The volume of skeletal muscle, sectional area of capillaries, and volume of
mitochondria were probably increased by physical training, but they were not
measured in the present study.

**Study limitations:** Although vasodilatory capacity was measured at rest using
strain-gauge plethysmography, we did not evaluate blood flow to the exercising
muscles during exercise. The effects of physical training on exercise tolerance
and vasodilator capacity in the skeletal muscle might depend, at least in part, on
the etiology of cardiac disease and/or history of cardiac surgery. However, there
were too few subjects in the present study to compare the effects of physical train-
ing among the 3 groups of patients with different etiologies. We could not find a
significant correlation between the improvement in vasodilatory capacity and that
in peak exercise capacity. These limitations of the present study should be
addressed in a future study.

**CONCLUSION**

Two-week physical training at the AT level safely increased exercise capac-
ity in patients after AMI or after cardiac surgery. The shortening of VO$_2$ kinetics
by physical training was closely related to the improvement in peripheral
vasodilatory capacity, reflecting improved cardiovascular adaptation at the begin-
ning of exercise. Short-term, moderate-intensity physical training was found to
improve the cardiovascular adaptation not only at peak exercise, but also during
the onset of exercise, by improving peripheral vasodilatory capacity in these
patients.

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REFERENCES


