Predischarge Early Exercise Therapy in Patients with Acute Myocardial Infarction on the Basis of Anaerobic Threshold (AT)

KOICHI TANIGUCHI, M.D.¹, HARUKI ITOH, M.D.², TAKASHI YAJIMA, M.D.³
MAYUMI DOI, M.D.³, AKIHIRO NIWA, M.D.³
AND FUMIAKI MARUMO, M.D.¹

The aim of this study was to assess the effectiveness of an early predischarge exercise therapy, started 2 weeks following acute myocardial infarction (AMI), to working functional capacity. Seventeen AMI patients (10 males, 7 females, mean age 62 ± 11 years) were examined in this study. Six, serious clinical symptoms and complications, were excluded, while the remaining 11 patients completed, the whole exercise therapy protocol (2 weeks). Patients performed exercise performed for 20—30 min twice daily at a target heart rate (90% level of heart rate at AT) on the basis of anaerobic threshold (AT) determined using treadmill ramp exercise with our protocol. Heart rates (HR) at rest, warming up and AT point decreased significantly (p<0.05) after exercise therapy, although peak HR remained unchanged. O₂ pulse at the AT point and endpoint, after exercise therapy, improved significantly (p<0.05), when compared to that before therapy. Moreover, AT and peak VO₂ improved remarkably (p<0.05), as did exercise time to the AT point and endpoint after exercise therapy (p<0.05), when compared to that before therapy. These results indicate that the predischarge early exercise therapy begun 2 weeks after AMI will be effective and beneficial in improving working capacity with improvement of physical deconditioning. Additionally, it is necessary that patients with severe clinical symptoms and complications be excluded.

RECENT hospital rehabilitation programs recommend a short-term bed rest, early commencement of physical exercise and early discharge! Early initiation of cardiac rehabilitation and subsequent physical training after acute myocardial infarction² is essential to promote recovery of cardiac and system function and to improve physical³; psychological⁴ and socialprofessional capacities⁵. Several studies have reported improved physical working capacity, decreased anxiety, augmented self-confidence and an increased number of patients returning to work sooner. On the other hand, there is the possibility of serious complications such as fatal arrhythmia, ventricular aneurysm, cardiac rupture and heart failure, with physical activity during the early stage of myocardial infarction as it might cause hemodynamic overload in diseased hearts. Therefore, it is extremely important to commence cautiously with a safe and useful rehabilitation therapy in the early phase after acute myocardial infarction.

Anaerobic threshold (AT) is defined as the level of exercise oxygen consumption above which aerobic energy production is

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1. Second Department of Internal Medicine, Tokyo Medical Dental University, Japan
2. Department of Internal Medicine, Chiba Shakai-Hoken Hospital, Japan
3. Department of Internal Medicine, Musashino Red-Cross Hospital, Japan
Mailing address: Koichi Taniguchi M.D., The Second Department of Internal Medicine, Tokyo Medical Dental University, Yushima, Bunkyo-ku, Tokyo 113, Japan

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supplemented by anaerobic metabolisms leading to increased lactate production. Accordingly, physical exercise less than the level of AT is consistent with aerobic exercise with balanced oxygen demand-supply relationship. In the present study we introduced AT exercise protocol and assessed the effect of exercise therapy on the basis of AT in patients with acute myocardial infarction.

METHODS

1. Patients
The study included seventeen patients (mean age 60 ± 11 years, range 40 to 77 years), whose body weights ranged from 52 to 74 kg (mean 58 ± 9 kg). Patients with severe complications were excluded. Ten patients suffered an anterior wall infarction and the remaining 7 an inferior one. Of the 17 patients whose cardiac index ranged from 2.0 to 3.4 l/min/m² (mean 2.8 ± 0.4 l/min/m²) and pulmonary arterial wedge pressure 4 to 15 mmHg (mean 7 ± 4 mg), 6 had ventricular premature beats (VPC), 2 ventricular aneurysm and 1 AV block. Eight patients had a one vessel disease, 6 had two-vessel disease and the remaining 3 had three-vessel disease. All patients were able to walk safely along a flat surface of more than 200m, 2 weeks after acute myocardial infarction.

2. Rehabilitation protocol
The exercise protocol consisted of preliminary exercise test, main exercise test and
exercise therapy. The schedule of exercise rehabilitation is shown in Fig. 1. In the exercise therapy, work load was defined weekly by the result of the main exercise test on the basis of AT.

1) Preliminary exercise test: Patients were asked to perform a few days before the main exercise test to alleviate anxiety and familiarize him with the exercise testing treadmill exercise (work rate: 2 km/h, grade: 0%, exercise time: 3–5 min) monitoring blood pressure and heart rate (HR) and also determining respiratory gas analysis.

2) Main exercise test: The main exercise test consisted of a treadmill ramp exercise test to measure AT and one-stage steady treadmill exercise test to determine a work rate level for exercise therapy.

(i) Treadmill ramp exercise test: AT was determined by respiratory gas analysis during exercise in each patient in order to decide a work load for exercise therapy. 90% of HR at the AT point was defined as the target HR. Exercise testing employed a treadmill ramp exercise with our own protocol \( V_O_2 = 0.067V^2 + 0.289VG + 7.37 \), \( V \): speed of treadmill, \( G \): grade of treadmill slope). It is said that \( V_O_2 \) during incremental treadmill exercise is proportionate to the grade of treadmill slope and a square of treadmill speed? Our ramp exercise protocol employed workload increments of treadmill speed and grades of slope at every stage (30 sec) after min in each patient, according to the above-mentioned equation. Warming-up was performed at the speed of 1 km/h (Fig. 2).

(ii) One-stage exercise test: In each patient a workload sufficient to maintain a target HR (90% level of heart rate at AT point) was studied using one-stage treadmill exercise testing 2–3 h after the treadmill ramp exercise. Treadmill speed was deter-
Fig. 4. Left: Improvement in AT and peak $\dot{V}O_2$ after exercise therapy (1W and 2W). Right: Improvement in $O_2$ pulse at AT point and endpoint (peak) after therapy.

### TABLE 1 $\dot{V}O_2$ AND $O_2$ PULSE AT AT POINT AND ENDPOINT (MAXIMUM) AFTER EXERCISE THERAPY COMPARED WITH THOSE BEFORE THERAPY: HEART RATE AT REST AND WARMING-UP AFTER THERAPY DECREASED SIGNIFICANTLY COMPARED WITH THOSE BEFORE THERAPY

<table>
<thead>
<tr>
<th>Period of exercise therapy</th>
<th>Rest</th>
<th>Warming-up</th>
<th>AT point</th>
<th>Peak point</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate (beats/min)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>before</td>
<td>88±4.7</td>
<td>98±5.0</td>
<td>108±4.0</td>
<td>122±3.5</td>
</tr>
<tr>
<td>1 week</td>
<td>81±4.4*</td>
<td>87±4.4*</td>
<td>99±4.4*</td>
<td>124±4.1</td>
</tr>
<tr>
<td>2 weeks</td>
<td>79±4.1*</td>
<td>86±4.1*</td>
<td>101±3.5*</td>
<td>128±4.7</td>
</tr>
<tr>
<td>$\dot{V}O_2$ (ml/beats/kg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>before</td>
<td>4.0±0.2</td>
<td>6.4±0.4</td>
<td>12.0±0.5</td>
<td>15.8±1.0</td>
</tr>
<tr>
<td>1 week</td>
<td>4.2±0.2</td>
<td>6.6±0.5</td>
<td>13.5±0.7</td>
<td>18.6±1.5*</td>
</tr>
<tr>
<td>2 weeks</td>
<td>4.4±0.2</td>
<td>6.8±0.6</td>
<td>14.0±0.7*</td>
<td>19.3±1.6*</td>
</tr>
<tr>
<td>$O_2$ pulse (ml/min/beats)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>before</td>
<td>2.9±0.2</td>
<td>4.2±0.4</td>
<td>6.5±0.8</td>
<td>7.5±0.8</td>
</tr>
<tr>
<td>1 week</td>
<td>2.9±0.3</td>
<td>4.7±0.5</td>
<td>8.5±0.9*</td>
<td>9.3±1.0*</td>
</tr>
<tr>
<td>2 weeks</td>
<td>2.9±0.3</td>
<td>4.9±0.6</td>
<td>8.6±0.9*</td>
<td>9.3±1.1*</td>
</tr>
</tbody>
</table>

before vs 1W, 2W (*p<0.05) (n=11m±SE)

AT: anaerobic threshold, $O_2$ pulse: $\dot{V}O_2$/heart rate, peak: endpoint of exercise (maximum workload), before: before exercise therapy, 1 week: 1 week after therapy, 2 weeks: 2 weeks after therapy

mined according to the workload, maintaining the target heart rate in each patient. All patients used a pulse meter during treadmill exercise.

(iii) Exercise therapy protocol: Exercise therapy started with a 20—30 min walking exercise on a flat surface at the target HR. It was repeated twice a day for one week. Thereafter, the treadmill ramp exercise and mentioned before. Blood pressure, HR and respiratory gas analysis including AT were determined, a new target heart rate was decided for exercise testing. Exercise therapy was then continued for one more week, and the same cardiopulmonary parameters were measured. This completed the predischARGE exercise therapy. After the patients were discharged from the hospital, they underwent a post-discharge exercise therapy at a new target HR as determined in the last ex-

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RESULTS

Of the 17 initial patients, 6 discontinued the exercise therapy due to complications such as angina pectoris with ST depression, pain and unsteadiness of legs, cerebral embolism and drug hepatitis. The remaining 11 patients continued the exercise therapy until they were discharged from the hospital.

1. Heart rate

The slope of incremental heart rate curves during exercise after therapy (one week, two weeks) were much steeper than that before therapy (Fig. 3). Namely at both one and two weeks after exercise therapy, heart rates decreased significantly (p<0.05) compared to before therapy, at rest (88±47 to 81±4.4 and 78±4.1 beats/min), warming-up (98±5.0 to 87±4.4 and 86±4.1 beats/main) and at the AT point (108±1.0 to 99±4.4 and 101±3.5 beats/min). However, heart rates at the endpoint showed no significant increase with exercise therapy (122±3.5 to 124±4.1 and 128±4.7 beats/min).

2. VO₂, AT, peak VO₂ and O₂ pulse

The slope of VO₂ response curves during ramp exercise testing after exercise therapy (one week, two weeks) were much steeper than before therapy at the AT point to endpoint (peak) as shown in Fig. 4 and Table I. On the other hand, the inclination of O₂ pulse profile from rest to AT point during exercise after therapy was much steeper than before therapy, although there was no difference in O₂ profile from AT point to endpoint (peak) before and after therapy. AT at 1 and 2 weeks after exercise therapy increased significantly (p<0.05) compared with that before therapy (12.0±0.5 to 13.5±0.7 and 14.0±0.7 ml/min/kg). In the same way, peak VO₂ increased significantly in comparison with that before therapy (15.8±1.0 to 18.6±1.5 and 19.3±1.6 ml/min/kg) and there was a significant improvement (p<0.05) in O₂ pulse at the AT point (6.5±0.8 to 8.5±0.9 and 8.6 ml/beats/kg) and endpoint (7.5±0.8 to 9.3±1.0 and 9.3±1.0 ml/beats/kg).

3. Exercise times to AT point and endpoint (peak)

Figure 5 shows exercise times to the AT point and endpoint before and after exercise therapy. Exercise time to the endpoint (peak exercise) after exercise therapy (1
week and 2 weeks) increased significantly (p<0.05), as compared with that before therapy (7.9±0.4 to 9.9±0.6 and 10.3±0.6min). Moreover, there was a significant improvement (p<0.05) in exercise time to the AT point (5.6±0.3 to 6.6±0.4 and 7.1±0.4).

DISCUSSION

Early commencement of exercise rehabilitation following acute myocardial infarction (AMI) promotes physical, psychological and social recovery from a disordered condition, and also improves working functional capacity. Overload on the infarcted heart, however, may occasionally induce serious complications such as ventricular aneurysm, fatal arrhythmias, heart failure and cardiac rupture. Maximum oxygen uptake (peak VO₂), exercise time during symptom-limited exercise and maximum work rate have generally been considered reliable indices of exercise working capacity. In exercise rehabilitation, however, a safe and appropriate workload based on a balanced oxygen demand-supply relationship should be given to patients with AMI in the early stage. Patients with AMI can easily continue exercise rehabilitation for a long time without an occurrence of metabolic acidosis, when they undergo physical exercise less than the AT level¹⁰,¹¹. To determine AT, a treadmill ramp exercise test was applied to AMI patients without complications using our protocol, in which VO₂ is propionate to work load.

Of the 17 patients, 6 with AMI in the early stage discontinued within 1 week due to complications. In the remaining patients, the predischARGE early exercise therapy (20—30 minute-walking) was started from the 15th day following AMI and performed twice daily at a target HR defined as 90% of HR at the AT point. As previously described¹²–¹³, HR at rest, warming up, and AT point except endpoint (peak) decreased significantly after the predischARGE early exercise rehabilitation in patients with AMI, although not in the 4 patients who discontinued the exercise therapy. On the other hand, O₂ pulses at AT point and endpoint (peak) increased significantly after the predischARGE early exercise therapy, whereas those at rest and warming-up remained unchanged. Moreover, the predischARGE early exercise rehabilitation according to work load less than AT improved significantly working functional capacity such as AT and peak VO₂, but this improvement was not observed in patients who discontinued the exercise therapy. Additionally, in the present study a remarkable improvement in exercise tolerance as shown by exercise time to AT and endpoint, using symptom-limited incremental exercise, was also observed in patients who underwent the exercise therapy. This improvement, however, was not noted in patients who gave up the rehabilitation due to complications. Fioretti et al reported that working capacity improved after rehabilitation, but their exercise rehabilitation began one month after hospital discharge. There are no reports on the effect of predischARGE early exercise rehabilitation begun from the end of 2 weeks after AMI and underwent at a target HR defined as 90% level of HR at the AT point.

In summary, predischARGE early exercise rehabilitation improved working capacity, such as O₂ pulse, AT and peak VO₂, in AMI patients with improved physical deconditioning. However, AMI patients, who have exercise induced angina, ST segment depression, and severe arrhythmias, should be excuded.

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