Randomized 4-Week Exercise Program in Patients With Impaired Left Ventricular Function

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**Background.** This study was designed to determine the controlled effects of a short-term exercise rehabilitation program on patients with moderate-to-severe left ventricular dysfunction after a recent myocardial infarction.

**Methods and Results.** Thirty-nine male patients 51±8 years old with a large anterior myocardial infarction less than 10 weeks old were recruited for the study. The patients were randomly assigned to either one of two training or control groups on the basis of their resting ejection fraction: training, less than 30%; control, less than 30%; training, 31–50%; or control, 31–50%. Patients were evaluated for filling pressures, radionuclide ventriculography, heart volume, echocardiography, and work capacity. Patients who underwent training participated in an intensive 4-week in-hospital exercise program, whereas the control patients were restricted to a minimal activity program. Results indicated that there were no significant improvements in resting, submaximal, and maximal hemodynamic measurements as a result of the program. Mean work capacity and peak oxygen consumption improved significantly in the less-than-30% training group but was accompanied by a significant increase in mean pulmonary wedge pressure. Resting ejection fraction improved markedly in both less-than-30% training and control patients, but ejection fraction measures were not associated with work capacity. Training did not cause further deterioration in ventricular function.

**Conclusions.** It was concluded that in the present study, exercise training had little or no effect on hemodynamic measurements and that the training effects achieved in patients with left ventricular dysfunction are most likely due to corrected impaired vasodilation, not necessarily to cardiac function. The importance of using a control group in this type of study and the wide interindividual variations in training responses are emphasized. *(Circulation 1991;84:1561–1567)*

In recent years, some studies, but not all, have indicated that patients with stable chronic heart failure attributable to ventricular systolic dysfunction are able to improve their physical work capacity as a result of an exercise program despite the limitations imposed by their condition. The improvements in exercise tolerance in these patients were shown to be primarily associated with peripheral adaptations and to a lesser extent with central hemodynamics. A general conclusion of these studies was that patients with depressed left ventricular function and well-healed infarcts can safely participate in exercise rehabilitation without further deterioration in ventricular function. The major limitations of these studies, however, were that the investigators did not use a matched control group, and it was not always possible to maintain an identical and unchanging drug regimen in the patients throughout the course of the program. Also, in many of the earlier studies, hemodynamic measurements were limited to the basal state.

At Klinik Roderbirken, a specialized cardiac rehabilitation center in the Federal Republic of Ger-

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many, approximately 3,500 cardiac patients are referred annually for a 4-week in-hospital program consisting of diagnostic evaluation, medication therapy, and physical rehabilitation. This provides a unique opportunity to evaluate the response of patients with severe left ventricular dysfunction to an intensive short-term exercise program immediately
after the acute stage of their infarct and, in particular, to determine if ventricular function is influenced by training at this early phase of rehabilitation.

The present study was designed to determine the controlled effects of a short-term in-hospital exercise rehabilitation program on patients with moderate-to-severe left ventricular dysfunction after a recent anterior myocardial infarction.

**Methods**

**Patients**

Thirty-nine male patients (mean age, 50.8±8.4 years) with a first (large) anterior myocardial infarction less than 10 weeks old (mean infarction age, 45.6±10.5 days), no previous history of left ventricular dysfunction, and an ejection fraction (EF) of less than 50% as measured by radionuclide ventriculography (RNVA) agreed to participate in the study. Voluntary consent was obtained from all patients in accordance with the policies of Klinik Roderbirken pertaining to human experimentation. The patients had been referred to Klinik Roderbirken for a 6-week period of in-hospital diagnostic evaluation and rehabilitation. The patients were provided with a diet compatible with their clinical status. Two patients were of New York Heart Association (NYHA) functional class I, 17 were functional class II, and 17 were functional class III. Excluded from the study were patients with poor exercise tolerance (working capacity, less than 1 W/kg body wt), heart rhythm disturbances greater than Lown classification IVa, valve dysfunction, peripheral claudication, or orthopedic difficulties that prevented them from participating in an exercise program. All patients had coronary heart disease and were considered to be clinically stable. Patients were randomly assigned to one of two training groups or to one of two control groups as follows: training group 1 (n=10), patients with an EF of less than 30% (TRG <30%); control group 1 (n=8), patients with an EF of less than 30% (CON <30%); training group 2 (n=11), patients with an EF of 31-50% (TRG ≥30%); or control group 2 (n=10), patients with an EF of 31-50% (CON ≥30%). Three patients in the TRG <30% group eventually developed complications and were removed from the study.

The medication profile of the patients is shown in Table 1. Medication was not altered during the course of the exercise program, with the exception of two patients who received an increase in diuretic therapy.

**Study Protocol**

After admission to Klinik Roderbirken, the electrocardiographic records of the patients were first evaluated for indication of a large anterior wall myocardial infarct (day 1). Patients with such an indication were then referred for echocardiography examination, chest radiograph for determination of heart volume, and a detailed medical history to confirm their status (day 2). Patients submitted to a supine exercise stress test with a bicycle ergometer (day 3) and to an RNVA examination (day 4). Day 5 was reserved for right heart catheterization (Swan-Ganz). Patients who met all retention criteria and gave informed consent were then randomly assigned by the throwing of a die to either a training or a control group. Three patients with known Lown IVa rhythm disturbances were successfully treated with antiarrhythmic medication and permitted to enter the study.

**Equipment and Procedure**

Chest roentgenograms were administered while patients were in the anteroposterior standing and lying-prone positions. Heart volume was measured by the method of Musshoff and Reinindell.17 Echocardiography was administered using a Hewlett-Packard Cardio Imaging System (model 77010 AC, Ultrasound Echocardiography System, Hewlett-Packard, Andover, Mass.). Measurements were recorded by one-dimensional echocardiography of the aortic and mitral valves and of the left ventricle in the parasternal view with the patient lying on the left side supine and by two-dimensional echocardiography in parasternal long-axis and apical four- and two-chamber views.

The exercise stress test was performed while the patient was in the supine position with a Siemens bicycle ergometer with continuous electrocardiographic monitoring. The initial work load was set at 40 W and increased by 40 W until the patient could no longer sustain the effort. Each load lasted 4 minutes. The end points of exercise were exhaustion, dyspnea, mean pulmonary artery pressure (PAP) of more than 45 mm Hg, or maximal age-predicted heart rate.

RNVA was performed in patients at rest in the supine position and during maximal exercise. The method of Borer et al18 was used to measure global ejection fraction. A conventional gamma camera and data processing system were used for recording, storing, and processing the depiction of the heart and were triggered by the electrocardiograph. The con-

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**Table 1. Medication Profile of Training and Control Groups**

<table>
<thead>
<tr>
<th>Medication</th>
<th>TRG &lt;30% (n=7)</th>
<th>CON &lt;30% (n=8)</th>
<th>TRG ≥30% (n=11)</th>
<th>CON ≥30% (n=10)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Digitalis</td>
<td>7</td>
<td>6</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>Diuretics</td>
<td>6</td>
<td>7</td>
<td>8</td>
<td>4</td>
</tr>
<tr>
<td>Angiotensin converting enzyme inhibitors</td>
<td>2</td>
<td>2</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Nitrites or molsidomin</td>
<td>6</td>
<td>8</td>
<td>11</td>
<td>9</td>
</tr>
<tr>
<td>Calcium channel blockers</td>
<td>2</td>
<td>4</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>ß-Blockers</td>
<td>3</td>
<td>3</td>
<td>8</td>
<td>5</td>
</tr>
<tr>
<td>Antiarrhythmics</td>
<td>2</td>
<td>3</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Combination therapy</td>
<td>7</td>
<td>8</td>
<td>10</td>
<td>9</td>
</tr>
</tbody>
</table>

TRG, training (<30% or ≥30% ejection fraction); CON, control (<30% or ≥30% ejection fraction).
tractions of the heart were shown by multiple-gated acquisition of the heart rate scintigram. A representative heart cycle was displayed on the monitor, and definition of the left ventricular wall in end diastole was defined using a light pen. The posttraining RNVA was measured at a work load equal to the heart rate achieved during the first testing session. Detailed methodology for calculation of the various parameters has been reported previously.8

Filling pressures (PAP and pulmonary wedge pressure [PWP]) were obtained from the Swan-Ganz floating catheter technique using a 5F catheter with a Statham P23 Db strain gauge.19,20 The measurements of cardiac output and oxygen consumption were derived from measurements taken during right heart catheterization.20

All examinations were repeated after 4 weeks of training using the same procedures. Patients were auscultated daily.

The 4-week daily (Monday through Friday) schedule for training patients consisted of the following activities.

Morning sessions. Activities included prebreakfast jogging (modified triangular run for 5 minutes performed twice three times weekly), calisthenics (30 minutes), relaxation training (20 minutes), and monitored ergometer cycling (15 minutes).

Afternoon sessions. Patients walked on paths graded to the capacity of the patient (30–60 minutes).

The intensities of training for the jogging and ergometer training sessions were maintained at 70–80% of the maximal heart rate achieved during the exercise stress test. Training was monitored daily by heart rate and blood pressure recordings before and after each training session. Ergometer cycling was further monitored by continuous electrocardiography. In addition, each patient received electrocardiographic Holter monitoring as well as ambulatory blood pressure monitoring once a week.

A chest radiograph during the midpoint of the program was readministered to all patients to check for clinical symptoms of silent congestion (observed in only one patient).

Statistical Analyses

Data analyses were performed on a Macintosh SE/30 computer using the STATVIEW 512+ program (BrainPower Inc., Calabasas, Calif.). Analysis of variance with repeated measurements and paired t tests were used to determine whether significant differences (p<0.05) in the variables existed between the experimental and control subjects as a result of the training program.

Results

Data collection was carried out over a period of 12 months. Of the 21 training patients, 18 completed the training program. The data of the three patients in the TRG <30% group who had to abandon the training program because of serious clinical complications were excluded from the data analyses. In two control patients, it was not possible to perform right heart catheterization after the training program because of difficulties in inserting the catheter. One TRG <30% patient refused to submit himself to the posttraining Swan-Ganz and RNVA examinations.

Age and physical characteristics of the subjects are shown in Table 2. There were no significant differences among groups with respect to age, age of infarct, heart volume, height, weight, and body mass index. Both training groups showed a noticeable but nonsignificant mean weight loss after the study: 4.2 kg for the TRG <30% group and 4.3 kg for the TRG ≥30% group.

There were no statistical differences in any of the resting measurements (Table 3). Mean maximal work increased significantly (17%, 75 to 88 W) in the TRG <30% group, whereas it decreased 9% (79 to 72 W) in the CON <30% group (Table 3). The TRG <30% group improved significantly in peak oxygen consumption (by 22%), whereas the CON <30% group decreased 8%. There were no significant changes with respect to maximal work and peak oxygen consumption in the TRG and CON ≥30% groups.

There were no significant changes as a result of the program in maximal heart rate, systolic blood pressure, rate-pressure product, and cardiac output. However, mean maximal PWP after training increased significantly in the TRG <30% patients. Correlational analyses (data not shown) indicated that maximal work rate before the training program was significantly associated with maximal cardiac output (0.85, p<0.01), peak oxygen consumption (0.85, p<0.01), and resting PWP (0.54, p<0.01). The correlation coefficients for all subjects between the changes in work capacity and the changes in resting PWP (0.07) and maximal PWP (−0.25) were nonsignificant.
### Table 3. Selected Right Heart Catheterization (Swan-Ganz) Measurements Before and After Training Program

<table>
<thead>
<tr>
<th>Variable</th>
<th>TRG &lt;30% (n=7) Before</th>
<th>TRG &lt;30% (n=8) After</th>
<th>TRG ≥30% (n=11) Before</th>
<th>TRG ≥30% (n=10) After</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rest HR (beats/min)</td>
<td>72±10</td>
<td>72±10</td>
<td>72±10</td>
<td>72±10</td>
</tr>
<tr>
<td>Rest SBP (mm Hg)</td>
<td>122±21</td>
<td>122±21</td>
<td>122±21</td>
<td>122±21</td>
</tr>
<tr>
<td>Rest Cardiac output (l/min)</td>
<td>10±5.6</td>
<td>10±5.6</td>
<td>10±5.6</td>
<td>10±5.6</td>
</tr>
<tr>
<td>Rest PWP (mm Hg)</td>
<td>9.4±2.2</td>
<td>9.4±2.2</td>
<td>9.4±2.2</td>
<td>9.4±2.2</td>
</tr>
<tr>
<td>RPP*</td>
<td>107±30</td>
<td>107±30</td>
<td>107±30</td>
<td>107±30</td>
</tr>
</tbody>
</table>

Maximal work load

<table>
<thead>
<tr>
<th>Variable</th>
<th>Maximal work HR (beats/min)</th>
<th>Maximal HR (beats/min)</th>
<th>Maximal SBP (mm Hg)</th>
<th>Maximal Cardiac output (l/min)</th>
<th>Maximal PWP (mm Hg)</th>
<th>Maximal Peak VO_2 (ml/min)</th>
<th>Maximal RPP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before</td>
<td>114±19</td>
<td>114±19</td>
<td>114±19</td>
<td>10±2</td>
<td>24±15</td>
<td>104±37</td>
<td>204±40</td>
</tr>
<tr>
<td>After</td>
<td>112±20</td>
<td>112±20</td>
<td>112±20</td>
<td>9±1</td>
<td>24±15</td>
<td>104±37</td>
<td>204±40</td>
</tr>
</tbody>
</table>

Comparative work load

<table>
<thead>
<tr>
<th>Variable</th>
<th>HR (beats/min)</th>
<th>SBP (mm Hg)</th>
<th>Cardiac output (l/min)</th>
<th>PWP (mm Hg)</th>
<th>VO_2 (ml/min)</th>
<th>RPP*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before</td>
<td>114±19</td>
<td>114±19</td>
<td>10±2</td>
<td>24±15</td>
<td>104±37</td>
<td>204±40</td>
</tr>
<tr>
<td>After</td>
<td>112±20</td>
<td>112±20</td>
<td>9±1</td>
<td>24±15</td>
<td>104±37</td>
<td>204±40</td>
</tr>
</tbody>
</table>

There were no significant differences before and after the training program in variables recorded at a comparable submaximal work load (Table 3). Cardiac output remained stable in all groups. However, heart rate decreased slightly in both training groups, and systolic blood pressure decreased by 10 mm Hg in the TRG ≥30% group. All groups showed an increase (nonsignificant) in PWP at a comparable submaximal work load after the program, except for the TRG <30% group, in which it did not change. Rate-pressure product also decreased appreciably in both TRG groups, although statistical significance was not reached.

RNVNA measurements before and after the training program are shown in Table 4. Resting EF increased slightly (nonsignificant) in both TRG and CON <30% patients. There was a marked but nonsignificant reduction in Δ EF (EF at exercise minus EF at rest) in the CON <30% group. Both TRG and CON ≥30% groups showed moderate but nonsignificant increases in resting EF after the program, whereas no significant differences were observed in exercise EF or Δ EF. Work capacity after the program showed correlations of 0.24 (nonsignificant) with EF at rest and 0.38 with maximal EF (p<0.03). There were no associations between the changes observed before and after the program in work capacity and EF at rest (r=0.08) and maximal EF (r=0.13). There also were no significant associations between the magnitude of the changes in heart rate at submaximal work load and the magnitude of changes in resting EF (r=0.14) and maximal EF (r=0.23).

### Table 4. Selected Radionuclide Ventriculography Measurements Before and After Training Program

<table>
<thead>
<tr>
<th>Variable</th>
<th>TRG &lt;30% (n=7) Before</th>
<th>TRG &lt;30% (n=8) After</th>
<th>TRG ≥30% (n=11) Before</th>
<th>TRG ≥30% (n=10) After</th>
</tr>
</thead>
<tbody>
<tr>
<td>Resting EF (%)</td>
<td>23.9±3.5</td>
<td>25.3±4.4</td>
<td>39.5±5.7</td>
<td>40.0±7.4</td>
</tr>
<tr>
<td>Exercise EF (%)</td>
<td>21.6±8.3</td>
<td>23.6±9.6</td>
<td>37.2±5.9</td>
<td>39.7±6.5</td>
</tr>
<tr>
<td>Δ EF</td>
<td>-2.3±5.2</td>
<td>-2.6±5.6</td>
<td>-2.3±2.4</td>
<td>-1.3±5.0</td>
</tr>
</tbody>
</table>

TRG, training (<30% or ≥30% ejection fraction); CON, control (<30% or ≥30% EF). Values are given as mean±SD.
Echocardiographic parameters measured before and after the program are shown in Table 5. There were no significant differences in these parameters among groups of subjects before the study, and they were not significantly influenced after training. Work capacity at the completion of the program showed an inverse relation with end-diastolic distance (−0.38, p<0.03) and E-septum distance (−0.50, p<0.003).

The individual changes that occurred in maximal work rate after the program were compared with the changes in resting and maximal PWP and EF. Notably, there was much interindividual variation among these variables. For example, in the five patients in the TRG <30% group who showed an improvement in maximal work rate, resting PWP decreased in three patients and remained unchanged in two; exercise PWP was higher in four and unchanged in one; resting EF improved in three patients, decreased in one, and remained unchanged in one; and exercise EF was higher in four and lower in one.

In the TRG ≥30% group, five of the 11 subjects had improved exercise tolerance after the program. Of these, three decreased in resting PWP, whereas one showed an increase and the other remained unchanged; maximal exercise PWP increased in three and decreased in two. Four of these training subjects improved in resting EF and one decreased; exercise EF increased in four and decreased in one.

Several complications occurred during the study. In the TRG <30% group, three patients had to be withdrawn from the program: two, after 16 and 17 days of training, respectively, because of left heart decompensation as diagnosed by pulmonary rales, dyspnea, hypotension, and observed weight gain. Of these, one suffered acute left heart failure with low output syndrome and hypotension, and the other showed slowly progressive left heart decompensation with pulmonary rales, NYHA functional class III and IV symptoms, and cardiac dilatation on radiography. The other subject was removed early in the program after developing ventricular tachycardia that required defibrillation. The data of these three patients were not included in the analyses. One additional patient in the TRG ≥30% group developed border line decompensation with cardiac dilatation with unspecified symptoms (hypotension, bradycardia, general weakness). He was given a higher dose of diuretic and was able to continue training without further complications.

All patients who developed complications during the exercise program exhibited the following characteristics before the onset of the program: more than 50 years old, resting PWP of more than 15 mm Hg, maximal exercise PWP of more than 30 mm Hg, EF at exercise of less than 20%, E-septum distance (echocardiogram) of more than 1.2 cm, and manifestation of frequent arrhythmias during Holter monitoring (Lown grade III).

There were no indications of complication or decompensation in the control group.

### Table 5. Selected Echocardiographic Measurements Before and After Training Program

<table>
<thead>
<tr>
<th>Variable</th>
<th>TRG &lt;30% (n=7) Before</th>
<th>TRG &lt;30% (n=8) Before</th>
<th>TRG ≥30% (n=11) Before</th>
<th>CON ≥30% (n=10) Before</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>After</td>
<td>After</td>
<td>After</td>
<td>After</td>
</tr>
<tr>
<td>EDD (cm)</td>
<td>0.56±0.04</td>
<td>0.55±0.06</td>
<td>0.57±0.08</td>
<td>0.54±0.09</td>
</tr>
<tr>
<td>E-S distance (cm)</td>
<td>0.97±0.30</td>
<td>0.83±0.35</td>
<td>0.99±0.44</td>
<td>0.81±0.36</td>
</tr>
<tr>
<td>FS (%)</td>
<td>25.7±4.9</td>
<td>30.3±4.8</td>
<td>26.9±11.6</td>
<td>27.3±9.1</td>
</tr>
<tr>
<td>mVcf (diameter/sec)</td>
<td>0.97±0.23</td>
<td>1.03±0.19</td>
<td>1.20±0.47</td>
<td>0.94±0.23</td>
</tr>
<tr>
<td>LVET (seconds)</td>
<td>0.272±0.042</td>
<td>0.299±0.010</td>
<td>0.264±0.046</td>
<td>0.284±0.023</td>
</tr>
</tbody>
</table>

TRG, training (<30% or ≥30% ejection fraction); CON, control (<30% or ≥30% ejection fraction); EDD, end-diastolic distance; E-S distance, E-septum distance; FS, fractional shortening; mVcf, mean velocity of circumferential shortening; LVET, left ventricular ejection time.

Values are given as mean±SD.

Discussion

The results of the present study indicate that patients who have severe left ventricular dysfunction (EF, less than 30%) after a recent large anterior myocardial infarct and engage in a short-term exercise program of moderate intensity, such as that offered at Klinik Roderbirken, can increase their exercise tolerance over that of control patients who are limited to a low-level intensity program. In the present study, the TRG <30% patients achieved significant improvements in work capacity and peak oxygen consumption, whereas the CON <30% patients showed a slight reduction during the program. However, resting EF and left ventricular ejection time improved in both TRG and CON <30% groups. This suggests that the training effect observed in the TRG <30% group was probably the result of improved oxygen delivery, most likely through a corrected impaired vaso dilution and not necessarily resulting from cardiac function. Furthermore, individual recovery mechanisms, such as spontaneous improvement, may be occurring that are not related to the exercise rehabilitation program.

As was shown by Sullivan et al.,7 improvement in exercise performance in patients with chronic heart failure resulting from left ventricular dysfunction after participating in an exercise rehabilitation program is primarily the result of an increase in peak blood flow to active skeletal muscles and a more efficient peripheral oxygen extraction, leading to reduced myocardial oxygen demand. It is well known that improved capillary supply and increased oxida-
tive enzymes are important factors of dynamic exercise performance.\textsuperscript{21,22}

In the present study, central hemodynamics, whether at rest or exercise, appears to be poorly related to improvements in work capacity. These results are consistent with those of Cobb et al.,\textsuperscript{4} Tubau et al.,\textsuperscript{13} Verani et al.,\textsuperscript{11} and Tavazzi et al.\textsuperscript{6} This would indicate that improvements in absolute and relative exercise tolerance and reduced ischemic symptoms after an exercise program appear to be due in large part to a reduction in myocardial oxygen demand brought about by improved nutritive flow, improved capillary supply, and increased oxidative enzymes. Exercise training did not lead to resting volume overload or left ventricular hypertrophy as assessed by end-diastolic diameter. As such, it appears that patients with depressed ventricular function can safely participate in a carefully supervised exercise program as provided in the present study without further deterioration in ventricular function.

The lack of an association between changes in EF at either rest or exercise, and exercise tolerance confirms the statement of Shabetal\textsuperscript{23} that the index of ventricular function is of little value in assessing the result of treatment in individual patients. Furthermore, the index appears unrelated to the patient’s ability to exercise and consume oxygen and consequently bears no relation to symptoms of heart failure. In our patients, contrary to the observations of Williams et al.,\textsuperscript{10} we did not observe a significant association between the magnitude of the changes in heart rate at a comparable submaximal work load and changes in EF at rest or exercise with training.

The improvement in exercise tolerance observed in the TRG <30\% group was, however, accompanied by a higher maximal PWP. This observation was also reported by Buchwalsky\textsuperscript{3} but not discussed. This increase in PWP to exercise could suggest that these patients were able to achieve a higher level of exercise intensity at the cost of some overloading of functional capacity (increased pressure and dilation) of the left ventricle. The consequences of developing an increase in exercise tolerance by physical conditioning with ventricles that are essentially unchanged require further study.

The results of the present study also attest to the wide variation in individual responses that are likely to occur as a result of an exercise rehabilitation program in cardiac patients. The obvious implication is that mean results should always be interpreted with circumspection. For example, at the onset of the program, resting PWP was found to be pathological (12 mm Hg or more) in 11 patients: six in the training groups and five in the control groups. After the program, four of the six experimental patients improved to normal, one worsened, and one remained pathological. Of the five control patients, two improved to normal values, two worsened, and one did not repeat the test.

With respect to exercise PWP, nine experimental and 10 control patients demonstrated a pathological response to exercise before the program. Normal PWP in response to supine cycling at any intensity should not exceed 20 mm Hg in patients 40 years old or younger or 25 mm Hg in older patients. After the program, one of the three patients in TRG <30\% group remained pathological and two worsened. One patient who was initially normal became pathological. In the CON <30\% group, one remained pathological, two worsened, one did not repeat the test, and one patient who was initially normal became pathological. In the TRG ≥30\% group, two of the six patients initially pathological worsened, one became normal, and three showed some improvement but remained within the pathological range; in this group, three patients who were initially normal became pathological. In the CON ≥30\% group, one of the six patients initially pathological remained pathological, one worsened, one improved to normal, two improved but remained pathological and one did not repeat the test. Three patients who were initially normal became pathological.

There also were wide individual variations in resting EF after the program. In the TRG <30\% group, one patient worsened, four improved, one remained unchanged, and one did not repeat the test; in the CON <30\% group, two worsened, and six improved. In the TRG ≥30\% group, four worsened, and seven improved. With respect to exercise EF, one patient worsened, five improved, and one did not repeat the test in the TRG <30\% group. In the CON <30\% group, maximal EF was lower in four patients and improved in four. In the TRG ≥30\% group, four worsened, and seven improved; in the CON ≥30\% group, five worsened, and five improved.

The complications that can occur over a relatively brief training period in patients with very low EFs whose infarcts are relatively new (mean age, 6 weeks), remain difficult to predict. Various authors\textsuperscript{5,6,24} have reported that 8–10 weeks are usually required for the scar tissue to heal completely and that training before this time could disturb the healing process and thus delay improvements in resting ventricular function. Our observations indicate that most patients with 6-week-old infarcts are able to tolerate the type of program provided at Klinik Roderbirken. Except for the three patients in the TRG <30\% group, whose resting EFs were less than 20\% and PWP were more than 15 mm Hg (of which two were found to have resectable aneurysms), there were no complications that precluded participation. However, patients with EFs of less than 30\% should be carefully screened on the basis of the criteria outlined in “Results” and exercised under close clinical supervision. With a view to minimizing complications, daily pulmonary auscultation for rales and Shortness of breath as well as questioning the patient for signs of nycturia are recommended. Particular consideration should be given to indications of psychological depression and sensitivity as these conditions appear to precipitate complications.\textsuperscript{25} The issue of predicting outcome in
cardiac rehabilitation programs has also been discussed recently by Myers and Froelicher.  

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References


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