Exercise Training After Anterior Q Wave Myocardial Infarction: Importance of Regional Left Ventricular Function and Topography

BODH I. JUGDUTI, MBCHB, FACC, BOODAN L. MICHOROWSKI, MD, C. TI SS A KAPPA G O D A, MBBS, FACC

Edmonton, Alberta, Canada

To determine whether the extent of left ventricular dysfunction and the degree of shape distortion can predict outcome in survivors of moderate-sized anterior Q wave myocardial infarction who are undergoing exercise training, these variables were measured by two-dimensional echocardiography before and after 12 weeks of a low level exercise training program starting 15 weeks after infarction in 13 patients (7 in group 1 and 6 in group 2) and 12 weeks apart in 24 matched control patients without training. By the end of training, the functional class score had increased in group 2 (from 2.25 to 2.67, p < 0.005) but had not changed in group 1. Further discrimination of groups 1 and 2 was provided by an initial asynnergy (akinesia or dyskinesia, or both) <18% or ≥18%. Compared with group 1, group 2 had greater initial asynnergy (32 versus 6%, p < 0.001), expansion index (asynergy/normal wall thickness: 1.8 versus 1.6, p < 0.025) and peak shape distortion index (12.2 versus 10.0 mm, p < 0.005) but lower ejection fraction (43 versus 59%, p < 0.05) and thinning ratio (asynergy/normal wall thickness: 0.61 versus 0.74, p < 0.05).

These variables did not change with training in group 1. However, in group 2, training caused significant increase in asynnergy (from 32 to 40%, p < 0.05), expansion index (from 1.8 to 2.0, p < 0.01) and peak shape distortion (from 12.2 to 20.9 mm, p < 0.05) associated with a decrease in thinning ratio (from 0.61 to 0.51, p < 0.001) and ejection fraction (from 43 to 30%, p < 0.005). Initial values for these variables were similar for corresponding control groups but did not change over the 12 weeks. Thus, patients with ≥18% left ventricular asynnergy on the initial echocardiogram showed more shape distortion, expansion and thinning before exercise training and developed further functional and topographic deterioration with training.

(Cardiac rehabilitation after acute myocardial infarction using exercise training programs has been suggested in the post-discharge phase to shorten the duration of convalescence and improve physical performance, the goal being an earlier return to work (1). Survivors of anterior transmural or Q wave infarction are at increased risk of complications (2) such as aneurysm formation, congestive heart failure, reinfarction and sudden death. Attempts have been made to separate low from high risk patients (3) using coronary arteriography and clinical variables before discharge from the hospital, electrocardiographic (ECG) Holter monitoring, low grade exercise testing, radionuclide wall motion studies, thallium-201 perfusion studies and electrophysiologic testing (3–8). It is recognized that prognosis after infarction depends on infarct size (reflected in the extent of left ventricular dysfunction), residual left ventricular function, presence of jeopardized myocardium and ventricular arrhythmias (3) as well as alteration in left ventricular topography (9). When prescribing exercise programs, it is not conventional to seriously consider the extent of ventricular dysfunction, residual ventricular function, altered geometry or the interval since the acute infarct. Because healing of an infarct can take several weeks to months depending on infarct size (10,11), even low levels of exercise might be injurious in patients with an incompletely healed Q wave infarct.)
The aim of this study was therefore to determine the
effect of a standard low level exercise program on left
ventricular function and topography in a group of survivors
of anterior Q wave acute myocardial infarction of moderate
size who were referred for cardiac rehabilitation 15 weeks
after the acute event; serial two-dimensional echocardiog-
raphy was used, a noninvasive tool that has been utilized
previously for such studies (12–16). The results of echocar-
diographic and exercise tests were analyzed in blinded
fashion to determine whether a two-dimensional echocardi-
ogram obtained before entry into the exercise program might
predict outcome and identify a subgroup likely to benefit.

Methods

Study patients. As part of ongoing studies of changes in
left ventricular topography and function after acute myocar-
dial infarction using serial tomographic imaging with two-
dimensional echocardiography between 1979 and 1981, con-
secutive patients admitted to the coronary care unit with a
first anterior transmural or Q wave acute myocardial infarc-
tion were studied prospectively. Consecutive survivors who
were referred for exercise training 15 weeks (range 6 to 32)
after the acute infarct were entered into this study if the
following criteria were met: 1) a history of anterior Q wave
acute myocardial infarction ≥6 weeks previously, supported
by a typical history of chest pain, evolutionary electrocar-
diographic (ECG) changes and new pathologic Q waves
>0.03 s in duration in at least two adjacent precordial leads
and leads I and aVL, a typical pattern of elevated serum
cardiac enzymes and persistent left ventricular asynergy on
two-dimensional echocardiography, 2) no contraindications
to exercise training, and 3) New York Heart Association
functional class I and II.

There were 22 such patients: 21 men and 1 woman with a
mean age of 47 years (range 29 to 61). The control patients
were selected from survivors of a first anterior Q wave
infarction during the same period who were not referred for
but fulfilled the criteria for exercise training and matched the
exercised patients in interval since infarction (mean 15
weeks, range 6 to 32). The 24 control patients comprised 19
men and 5 women with a mean age of 58 years (range 39 to
77) who did not undergo exercise training. Written informed
consent was obtained in each case.

Exercise program and two-dimensional echocardiograms.
Each patient had a complete echocardiographic study at rest
on entry followed by a standard upright exercise stress test
on a bicycle ergometer. The load was initially set at 200
kp-m/min (33 W) and increased every 3 min by 100 kp-m/min
(16 W). The ECG was monitored continuously from lead V4
and blood pressure (by sphygmomanometer) was recorded
every 3 min. The end point was at least one of the following:
1) 85% predicted maximal heart rate; 2) symptoms (chest
pain, dyspnea, fatigue, dizziness); 3) ST segment depression
of ≥0.1 mV at 80 ms after the QRS complex; 4) significant
ventricular arrhythmias; 5) >5 mm Hg decrease in blood
pressure or failure of blood pressure to increase over three
consecutive work loads.

After the initial evaluation, these patients underwent 12
weeks of training on the Canadian Air Force Exercise SBX
Program (17). Briefly, this program requires 11 min of
unsupervised daily training. The exercise time was split
equally between calisthenics and a stationary run; the fre-
quency of each exercise was increased as training pro-
gressed to keep within the set time. Initially, patients re-
ported to the laboratory three times each week for 2 weeks
and the exercises were performed with ECG monitoring.
During the first week, the patients performed the first level of
exercise during the laboratory visits only. During the second
week, they performed the second level of exercise both at
home and during laboratory visits. Thereafter, they reported
to the laboratory every week when a new level of exercise
was performed with ECG monitoring.

After completion of training, echocardiograms at rest
were repeated and followed by an exercise stress test. In the
control patients echocardiograms at rest were obtained
initially and again 12 weeks later. All patients were followed
for at least 12 months for complications, cardiovascular
events and functional class as indicated by questionnaire.
Medications were not manipulated. All patients in the exer-
ccise protocol underwent exercise thallium-201 scintigraphy
before and after training and radionuclide angiography (tech-
netium-99m) at rest before training, but only nine agreed to
coronary and biplane left ventricular angiography before
training.

Analysis of two-dimensional echocardiograms (Fig. 1). All
patients had complete echocardiograms with adequate visu-
alization of the four cardiac chambers and ventricular walls
for detailed analysis of left ventricular asynergy (18–20),
 volumes (13,18–20) and topographic variables (14,15,21,22)
including the degree of regional shape distortion (16,21).
Standard views, including parasternal long-axis, four short-
axis (at mitral, chordal, midpapillary and low papillary
levels), apical four and two chamber and subxiphoid, were
videotaped systematically. The positions of the patients and
diagram were noted for use in serial studies (14,15,18–21).
Images were videotaped for review in real time, slow motion
and single frame format. Coded recordings (tape and log
numbers) were analyzed separately in three steps by two of
us (B.L.J., B.L.M.). Differences were resolved by consen-
sus. Interobserver and intraobserver errors in measurements
were small (<1% in marking asynergy, segment length and
wall thickness; <5% in areas of outlines).

First step. Endocardial and epicardial left ventricular
outlines were traced on plastic overlays from ECG-gated
images frozen at end-diastole and end-systole, and modified
on multiple playbacks. Special attention was given to ana-
tomic landmarks (papillary muscle markings, right and left ventricular junctions) and shape.

**Second step.** Markings of the extent of left ventricular asynergy, defined as akinesia (no systolic inward motion and thickening) or dyskinesia (systolic outward motion and thinning), or both, were made on each left ventricular endocardial diastolic outline by careful visual assessment of wall motion and thickening on repeated real-time video playbacks, as reported from this laboratory (18-21). Care was taken to ensure that asynergic segments did not show systolic inward endocardial motion and thickening by comparing wall thicknesses in systole and diastole (light pen system) and comparing aligned diastolic and systolic outlines. The endocardial diastolic outlines and asynergic segments were then digitized (HP 9874A and 9835A) for computing the circumferential and angular extents of asynergy in each short-axis section as percent, as well as the total extent of left ventricular asynergy as percent surface area of the endocardial shell, using the apical four chamber and four serial short-axis data (Fig. 1C) as described previously (18-21). Because regional shape distortion was present in the asynergic zones on diastolic outlines in patients with anterior Q wave infarction, the "ideal" asynergic segment corresponding to the angular extent of "actual" asynergy was used to compute total asynergy and avoid overestimation (16). Global ejection fraction was calculated from left ventricular end-diastolic and end-systolic volumes that were computed from outlines of two long-axis views (apical four and two chamber) and four short-axis views using the modified Simpson's rule (13,18-20).

**Third step.** Topographic measurements were made on end-diastolic outlines of short-axis images at the mid left ventricular or papillary level (Fig. 1A and B). Expansion index was computed as the ratio of the asynergy-containing endocardial segment length to that of the nonasynergic-containing endocardial segment length (14,15,20,21). Thinning ratio was computed as the ratio of the average thickness of the asynergic zone to the average thickness of the nonasynergic zone (14,20,21). Regional shape distortion was characterized by two previously described indexes (16,21).
after isolating the distortion by computing the ideal asynergic segment (Fig. 1B): 1) peak of the angular distribution of distortion (P_k), and 2) area of the distortion (A_d).

Statistics. All data were coded and analyzed at the end of the study in blinded fashion. Results are expressed as mean values ± SD. The significance of difference within a group was assessed by analysis of variance (ANOVA) and between groups by multivariate ANOVA. Differences were assumed significant for a p value < 0.05.

Results

Initial patient data (Table 1). Pertinent initial patient data for the four groups are summarized in Table 1. Thirteen of the 22 patients referred for exercise training completed the protocol. By the end of training, the functional class score (Fig. 2) was unchanged in seven patients (group 1) but had increased in six patients (group 2). To define an objective echocardiographic index for separating the two groups, multivariate analysis of variance was done on echocardiographic variables. Initial left ventricular asynergy ranked as the best discriminator (F value 65.84, well above the critical F_{0.05 (1,11)} ratio of 4.84), followed by ejection fraction (F value 28.51) and peak distortion (F value 14.15). Fisher's discriminant function (23) for the cutoff value between the groups yielded a value of 18.15% initial asynergy, which was 2.25 SD from the means for these two groups. An initial ejection fraction of 52.4% was 1.49 SD from the means for these groups. The exercise and control patients were therefore grouped on the basis of whether the total asynergy on the initial echocardiogram was > or < 18%; thus, asynergy was < 18% for exercise group 1 and control group 3, and > 18% for exercise group 2 and control group 4.

Among control patients, 11 were in group 3 and 13 in group 4. There was no difference in the initial data for the combined exercise groups 1 and 2 compared with control groups 3 and 4. Initially, group 2 differed from group 1 not only in having more asynergy (32.3 versus 5.5%, p < 0.001) and a higher functional score (2.3 versus 1.9, p < 0.05) but also a higher peak creatine kinase level (2,305 versus 747 IU/
EXERCISE TRAINING AFTER MYOCARDIAL INFARCTION

EXERCISE TRAINING

Figure 2. Effect of exercise training (ET) on functional class in exercise groups 1 and 2. NYHA = New York Heart Association; Echo #1 and #2 refer to two-dimensional echocardiographic evaluations in control groups 3 and 4.

Filter 2. Effect of exercise staining (ET) on functional class in exercise groups 1 and 2. NYHA = New York Heart Association; Echo #1 and #2 refer to two-dimensional echocardiographic evaluations in control groups 3 and 4.

Table 2. Exercise Stress Test Data Pre- and Postexercise Training in Groups 1 and 2

<table>
<thead>
<tr>
<th>Group</th>
<th>Pre</th>
<th>Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR at rest (beats/min)</td>
<td>71 ± 11</td>
<td>70 ± 8</td>
</tr>
<tr>
<td>Mean BP at rest (mm Hg)</td>
<td>98 ± 13</td>
<td>96 ± 7</td>
</tr>
<tr>
<td>Maximal HR on exercise test (beats/min)</td>
<td>115 ± 11</td>
<td>125 ± 22</td>
</tr>
<tr>
<td>Maximal mean BP on exercise test (mm Hg)</td>
<td>123 ± 19*</td>
<td>116 ± 8</td>
</tr>
<tr>
<td>RPP at rest (mm Hg x beats/min x 10^6)</td>
<td>8.9 ± 2.0</td>
<td>8.9 ± 1.5</td>
</tr>
<tr>
<td>RPP at peak exercise (mm Hg x beats/min x 10^6)</td>
<td>19.6 ± 4.0*</td>
<td>20.8 ± 4.9</td>
</tr>
<tr>
<td>Δ RPP at peak exercise (mm Hg x beats/min x 10^6)</td>
<td>—</td>
<td>1.3 ± 1.7</td>
</tr>
<tr>
<td>Total work during exercise (W)</td>
<td>738 ± 882</td>
<td>725 ± 398</td>
</tr>
<tr>
<td>Δ total work during exercise (W)</td>
<td>—</td>
<td>350 ± 372</td>
</tr>
</tbody>
</table>

*p < 0.05, *p < 0.01, statistical significance comparing pre-exercise data between groups 1 and 2; t* < 0.05, statistical significance comparing pre- and posttraining data within groups. BP = blood pressure; HR = heart rate; RPP = heart blood rate-pressure product.
Figure 3. Effect of exercise training on left ventricular asynergy (panel A) and ejection fraction (panel B). Pre = before; post = after; other abbreviations as in Figure 2. Paired studies are indicated by lines joining individual points. For both panels: *p < 0.001, comparing group 2 with group 1 pre-training; **p < 0.001, comparing group 2 with group 1 post-training; *p < 0.001, comparing Echo #1 in group 3 with group 4; tp < 0.001, comparing Echo #2 in group 3 with group 4.

The initial peak regional shape distortion index \( P_k \) was greater in group 2 than group 1 (1.22 versus 0.10 cm, \( p < 0.005 \)) and in group 4 than group 3 (1.21 versus 0.08 cm, \( p < 0.001 \)) (Fig. 5). Between the two evaluations, \( P_k \) did not change in group 1, 3 or 4 but increased in group 2 (1.22 versus 2.09 cm, \( p < 0.05 \)). Similar changes were found in the other topographic indexes, including an increase in end-diastolic area, and volume after training in group 2.

Coronary arteriographic and radionuclide data. Coronary arteriography before training in nine patients on the exercise protocol (four in group 1, five in group 2) revealed left anterior descending coronary artery occlusion in all; three vessel disease was present in six of these patients (four in group 1, two in group 2) and two vessel disease in the other three patients (all in group 2). Exercise thallium-201 scintigrams before and after training, in all patients on the exercise protocol showed a persistent anteroseptal defect consistent with scar, and no significant difference in the size of the defect or extent of redistribution between the studies. Ejection fraction at rest, on radionuclide angiography before training, correlated closely with that on initial echocardiography \( (y = 1.1x - 6.5, \text{SEE} = 2.8, r = 0.97, p < 0.001) \), in agreement with our previous findings (20).

Follow-up (Table 4). The functional score at a mean of 6 months follow-up remained lower in group 2 than in group 4 (2.67 versus 2.23, \( p < 0.05 \)). The echocardiographic data at 6 months did not differ from those at the second evaluation. The trends persisted up to the final follow-up at 40 months during optimal therapy, but there was one late death in exercise group 2. Of the nine patients who did not complete training, four would have belonged to group 1 and five to group 2; their initial data were similar to those of patients who completed training. On follow-up, events included: reinfarction in two, coronary bypass surgery in three, left ventricular thrombus and stroke in one, aneurysmectomy in one, coronary angioplasty in one, abdominal surgery in one.

Discussion

Left ventricular function before exercise training. There are two major findings in this study. First, two-dimensional echocardiography can be used to noninvasively assess global left ventricular function as well as the extent of regional left ventricular dysfunction and topographic abnormality before exercise training in survivors of acute myocardial infarction. Thus, echocardiographic imaging of the patients with a
recent anterior Q wave myocardial infarction who fulfilled criteria for low level exercise training indicated that those with ≈18% regional left ventricular asynergy (an index of contractile dysfunction) also had a lower global ejection fraction at rest (an index of left ventricular performance), more topographic abnormalities such as higher expansion index, lower thinning ratio, higher regional shape distortion indexes, as well as a poorer functional class score compared with those with <18% asynergy. In addition, these differences persisted in control patients (who did not undergo exercise training) at subsequent evaluations after 12 weeks, 6 months and 40 months.

**Effect of exercise training.** The second major finding is that the patients with ≈18% left ventricular asynergy on the initial two-dimensional echocardiogram were prone to further topographic and functional deterioration with exercise training. Thus, after exercise training, these patients had a lower global ejection fraction, greater expansion index, lower thinning ratio, larger regional shape distortion indexes and poorer functional class score. These changes were persistent at 6 and 40 months. In contrast, patients with <18% asynergy on the initial two-dimensional echocardiogram did not develop further functional or topographic deterioration with exercise training. It is important to note that the standard variables measured at exercise stress testing performed before and after training, including "total work", did not identify the patients prone to functional deterioration or distinguish those with 18% left ventricular asynergy. On the other hand, the cutoff value of 18% initial asynergy clearly separated the means for the groups by 1.25 SD.

**Changes in topography during infarct healing.** Pathophysiologically, healing after acute myocardial infarction in humans takes place over 6 weeks to 6 months depending on...
infarct size (10). It involves a sequence of acute inflammation in the first week, chronic inflammation in the second week and collagen deposition from the third week onward (11). The healing process can be classified into early and late phases depending on whether significant collagen deposition has occurred (24). In the early phase, before collagen deposition, when the infarct is soft and inflammation is active, the infarct segment is prone to expansion with stretching, thinning and dilation that contribute to impaired function and early morbidity (9). In the late phase after collagen deposition, there is remodeling of the scar with compaction, further thinning and increased distensibility (25) resulting in aneurysmal bulging and cardiac dilation.

Data from this laboratory (26) indicate that three major mechanical factors contribute to infarct expansion in the early phase: 1) intracavitary distending forces such as preload and afterload, 2) mural stretching forces such as the contractile pull of adjacent normal myocardium, and 3) the frequency of application of these distending and stretching forces represented by heart rate. These forces most likely also contribute to distension of the infarct zone in the late phase of healing. Early and late remodeling of the infarcted left ventricle has been demonstrated in rats (27), dogs (22,24) and humans (14,15,21,26,28).

**Timing of exercise training in relation to infarct healing.** Ideally, timing of exercise training after infarction should

![Figure 4. Effect of exercise training on the ventricular expansion index (panel A) and thinning ratio (panel B).](image)

For both panels: *p < 0.05, comparing group 2 with group 1 pretraining; **p < 0.025, comparing group 2 with group 1 posttraining; $t_p < 0.01$, comparing Echo #1 in group 3 with group 4; $t_p < 0.01$, comparing Echo #2 in group 3 with group 4.

**Figure 5. Effect of exercise training on the peak regional shape distortion index (Pp).** *p < 0.005, comparing group 2 with group 1 pretraining; $t_p < 0.025$, comparing group 2 with group 1 posttraining; $t_p < 0.001$, comparing Echo #1 in group 3 with group 4; $t_p < 0.001$, comparing Echo #2 in group 3 with group 4.

---

**Table 4. Pertinent Follow-up Patient Data at 6 Months**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Exercise</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Group 1</td>
<td>Group 2</td>
</tr>
<tr>
<td>Functional score</td>
<td>2.1 ± 0.2</td>
<td>2.7 ± 0.3*</td>
</tr>
<tr>
<td>Coronary artery bypass graft</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Reinfarction</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Deaths</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

*p < 0.005, statistical significance comparing group 1 versus 2 or group 3 versus 4.
consider the healing status of the infarct. Although human autopsy data indicate that small infarcts typically heal by 6 weeks, incomplete healing with persistent foci of soft necrotic tissue has been demonstrated in large transmural infarcts at 6 months (10,11). Thus, it is possible that the hemodynamic stress of exercise, such as increased heart rate, systolic blood pressure, and contractility of normal myocardium might precipitate further stretch of the scar with chamber dilation and aneurysmal bulging in patients with an incompletely healed, large transmural infarct. The problem is further complicated by the finding that some therapies used after infarction can adversely modify healing and topography (22,29).

Despite the trend to earlier exercise testing and training after infarction, there is no proven objective method for assessing the status of healing during life. In the rat myocardial infarction model in which healing is completed by 3 weeks (27), forced exercise during the first 24 h after coronary artery occlusion produced scar thinning (30) whereas exercise begun 1 week after coronary occlusion had a similar but less dramatic effect (31).

The results of this study indicate that some patients with a moderately large transmural infarct (Q waves, creatine kinase infarct size 34 ± 14 gEq [range 20 to 62], ≥18% asynergy [range 26 to 42%]) have more marked left ventricular topographic abnormalities (peak shape distortion [P<sub>k</sub>] > 1.0 cm) and dysfunction at 15 weeks (range 6 to 32) after infarction and show further functional and topographic deterioration with low level exercise training. It is possible that these patients had incomplete healing and were more susceptible to topographic changes during training.

Echocardiographic assessment of function and topography before exercise. Tomographic imaging with two-dimensional echocardiography is a valuable tool for the noninvasive assessment of postinfarction changes in left ventricular regional and global function (12,18-20), topography (14-16,21,25,26,28) and regional shape distortion (16,21). The relation between regional left ventricular dysfunction and infarct size (12,18,19,32) and the effect of therapy on infarct size (18) and topography (21,22) have been documented using this tool. However, it has not been widely applied to evaluate patients undergoing exercise training. In this study, careful systematic recordings in multiple views were done by trained technicians and these were adequate for the analysis of function and topography in all cases. The results suggest that the extent of regional ventricular dysfunction can be useful in risk stratification.

The degree of regional shape distortion might also be useful. Thus, the mean initial peak shape distortion index (P<sub>k</sub>) exceeded 10 mm in patients with ≥18% asynergy and nearly doubled after exercise training (12.2 versus 20.9 mm, p < 0.03). This was associated with concordant changes in other topographic indexes suggesting stretching, thinning and bulging of the scar. In a previous study (33), a P<sub>k</sub> value ≥10 mm at 2 days after anterior Q wave infarction correlated with the development of infarct expansion and associated with higher mortality and morbidity by 10 days. In this study, however, the initial P<sub>k</sub> in the group with ≥18% asynergy varied over a wide range (5.4 to 21.6 mm) as did the other topographic indexes so that separation of the groups on the basis of initial P<sub>k</sub> or other topographic indexes was less marked than with initial asynergy. This might be explained by the fact that the initial evaluation was done 6 to 32 weeks after acute infarction so that the infarcts were at different stages of healing. Marked changes in topographic indexes have been documented at these different stages of healing (14-16,22,24-28). The cutoff value for initial P<sub>k</sub> in this study was 2.8 mm, which was 1.65 SD from the means of the two groups, significantly greater (p < 0.005) than the mean in group 1 and less (p < 0.01) than the mean in group 2.

Exercise training after myocardial infarction. Most studies suggest that exercise training improves work capacity (34). The same external work is achieved at a lower rate-pressure product with lower myocardial oxygen consumption (35) and alleviation of angina (34). However, a large randomized trial (36) failed to show reduction in mortality or reinfarction rate with exercise training. Furthermore, Cobb et al. (37) found that improved exercise performance was not accompanied by improved ventricular function (radionuclide angiography) at rest or on exercise in 11 patients. In patients with impaired ventricular function (ejection fraction ≤40%), Lee et al. (38) also found that increased work capacity was not associated with improved ventricular function (angiographic). In both these studies (37,38), end-diastolic volume at rest was not different after training and changes in topography were not measured. In our study, total work was similar in patients whose indexes of infarct size, ventricular function and topography were significantly different. It is possible that the slightly larger left ventricular dimension and volume in group 2 permitted greater stroke output to be achieved on exercise by virtue of the Starling effect. The lack of decrease in heart rate at rest with the 5BX program might be related to the low level of exercise and medications (for example, nine patients were being treated with beta-adrenergic blocking agents and two with calcium blocking agents [Table I]). During training, there was no evidence of ongoing ischemia or necrosis, and thallium scintigraphic defects did not differ before and after training.

Also in this study, there was no discrete sudden event that marked functional deterioration or necessitated withdrawal from the program in group 2. However, there was clear echocardiographic evidence of infarct expansion in that group; this was supported by greater stretching and thinning of the infarct zone (increased expansion index, anterior segment length and regional shape distortion indexes; decreased thinning ratio) and chamber dilation (greater area of regional distortion, internal dimension and volume). It is
possible that this "late" expansion is a less dramatic event than the "acute" expansion that occurs "early" after infarction (9,14,15,21,26,33) before marked collagen deposition when the infarct is soft. Several factors might have contributed to the expansion in group 2: 1) increased wall stress as a result of the larger volume, 2) further increase in wall stress due to increased afterload during exercise, and 3) the increased contractile pull of the nonasymmetric segment during exercise. However, "contractile function" of the normal segment at rest, measured by the percent change in inward systolic motion and percent systolic thickening on two-dimensional echocardiography, assisted analysis of images, did not change significantly in group 1 or 2 (p > 0.1). Recognizing that asynchrony on echocardiography overestimates infarct size (32), an initial asynchrony of 18% as a cutoff for those exhibiting infarct expansion in this study is not unreasonable because the critical infarct size for acute expansion in animal studies ranges from 11 to 17% of left ventricular mass (25,26).

Conclusions. Two-dimensional echocardiography might be useful in selecting those patients with poor left ventricular function who are likely to respond adversely to exercise training after acute myocardial infarction. The extent of left ventricular dysynchrony on an initial echocardiogram might predict the response of patients with a first anterior Q wave infarct to exercise training. Patients with >18% left ventricular asynchrony on the initial study had significant topographic abnormalities with marked regional shape distortion and showed further functional and topographic deterioration with low level exercise training 15 weeks (range 6 to 37) after infarction. The results support the view that exercise training might be injurious in patients with an extensive transmural infarct that has not healed completely. Equally important, echocardiographic measurements identified those patients in whom low level exercise training did not have an adverse effect on left ventricular function and topography. These preliminary results should be confirmed in larger studies.

We thank Cheryl Trudell, RTN, for technical assistance. Michael Maidens, MSc, for aid with statistical and Christine Scott and Catherine Jugdutt for secretarial assistance.

References


33. Jagduti BL. Can the degree of regional shape distortion on an early two-dimensional echocardiogram after myocardial infarction identify patients prone to infarct expansion? (abstr). J Am Coll Cardiol 1986;7:148A.


