The Effect of Moderate Physical Exercise on the Plasma Lipoprotein Subfractions of Male Survivors of Myocardial Infarction

Fiona C. Ballantyne, Ph.D., Robert S. Clark, M.R.C.P., Harrison S. Simpson, B.Sc., and David Ballantyne, M.D., F.R.C.P.

SUMMARY The effect of regular, moderate exercise on the lipoprotein subfractions of male survivors of myocardial infarction was studied. Nineteen men were randomly allocated to an incremental exercise program and 23 to a control group. Both groups were studied for 6 months.

No change occurred in any lipoprotein class in the control group. In the trained group, total triglyceride and low-density lipoprotein (LDL) cholesterol concentrations decreased significantly (0.01 > p > 0.001 and 0.05 > p > 0.01, respectively) and high-density lipoprotein (HDL) cholesterol and apolipoprotein A-1 rose (both p < 0.001). The concentration of the HDL₂ subfraction increased with training (0.01 > p > 0.001) and HDL₄ did not change. No relationship was found between changes in lipoproteins and treadmill exercise test performance. Thus, in survivors of myocardial infarction, exercise may alter plasma lipoprotein values beneficially.

REGULAR, VIGOROUS EXERCISE may reduce the risk of developing coronary heart disease.¹⁻³ Training may have a protective influence by altering the levels of known risk factors, such as plasma lipids. Males and females who exercise regularly have significantly lower fasting concentrations of total triglyceride and cholesterol in very low and low-density lipoproteins (VLDL and LDL), but higher cholesterol concentrations in high-density lipoproteins (HDL) than age- and sex-matched sedentary control subjects.⁴⁻⁶ In longitudinal studies of normal subjects,⁷⁻¹⁰ exercise leads to lower plasma concentrations of triglyceride and VLDL and LDL cholesterol, whereas HDL cholesterol increases. The increase in HDL on exercise may be due mainly to an increase in the less dense HDL₂ subfraction.¹ⁱ⁻¹²

In two reports¹³,¹⁴ of the effect of exercise on plasma lipoproteins in patients with overt coronary heart disease, moderate exercise was found to increase HDL cholesterol. Erkelens et al.¹³ did not report LDL cholesterol measurements. Streja and Mymin¹⁴ did not estimate LDL cholesterol, but they did calculate an approximate value from the concentrations of total lipids and of HDL cholesterol. We have assessed the effect of a 6-month physical training program on the plasma lipoprotein distribution of male survivors of myocardial infarction and compared the results with a control group of survivors who did not take regular exercise. The major lipoprotein classes were analyzed and the compositions of HDL₂ and HDL₄, isolated by rate-zonal ultracentrifugation,¹⁵⁻¹⁶ were determined.

Materials and Methods

Subjects

Subjects were selected from admissions to a coronary care unit. All males younger than age 65 years with a definite acute myocardial infarction, as defined by a typical history, typical electrocardiographic changes and a diagnostic rise in cardiac enzymes, were considered. Survivors were asked to return for review 3 months later, when they were assessed for entry into the trial. Lipid and protein metabolism, which show changes in response to myocardial infarction, can be assumed to have returned to preinfarction levels before 3 months.¹⁷ Exclusion criteria were angina pectoris, cardiac failure, cardiac dysrhythmias, chronic respiratory disease, obesity, physical disability and elevated serum concentrations of creatinine, bilirubin, alkaline phosphatase, aspartate aminotransferase and alanine aminotransferase.

Protocol

The subjects were randomly allocated either to a training group (19 men) or to a control group (23 men). Both groups of subjects were reviewed by medical staff and by dieticians at the beginning of the study and at 1, 2, 4 and 6 months. The ages, weights and heights of the groups were closely matched (table 1). They had similar initial food and alcohol intake levels, and these did not change during the study. Only three trained and two control subjects smoked, although others had smoked before their myocardial infarctions. Five trained and six control subjects took diuretics and one trained subject and four control subjects took β blockers. Both of these groups of drugs have been reported to affect lipoproteins,¹⁸⁻²⁰ but only a minority of subjects received them and since no change was made in therapy during the period of study, we assumed that their influence could be ignored.

On the initial treadmill exercise test,¹¹ the trained group had a longer time to maximum exercise than the control group (table 1), but the rate-pressure product
TABLE 1. Characteristics of the Trained and Control Groups at the Start and End of the Study

<table>
<thead>
<tr>
<th>Group</th>
<th>Time (months)</th>
<th>Age (years)</th>
<th>Weight (kg)</th>
<th>Height (cm)</th>
<th>Quotet index (kg/cm² × 10⁴)</th>
<th>Time to max exercise (min)</th>
<th>Rate-pressure product (beats × mm Hg/min × 10⁴)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trained (n = 19)</td>
<td>0</td>
<td>51.0 ± 5.2</td>
<td>71.7 ± 6.0</td>
<td>175 ± 6.0</td>
<td>1.94 ± 0.24</td>
<td>8.45 ± 1.06</td>
<td>21.1 ± 3.0</td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>—</td>
<td>71.7 ± 8.0</td>
<td>—</td>
<td>1.95 ± 0.25</td>
<td>11.60 ± 3.02</td>
<td>24.0 ± 5.1</td>
</tr>
<tr>
<td>Significance*</td>
<td>NS</td>
<td>NS</td>
<td>p &lt; 0.001</td>
<td>NS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control (n = 23)</td>
<td>0</td>
<td>54.3 ± 6.2</td>
<td>73.5 ± 7.2</td>
<td>174 ± 6.0</td>
<td>2.00 ± 0.10</td>
<td>6.23 ± 2.43</td>
<td>20.0 ± 4.9</td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>—</td>
<td>74.0 ± 7.2</td>
<td>—</td>
<td>1.99 ± 0.10</td>
<td>7.51 ± 2.33</td>
<td>19.1 ± 3.6</td>
</tr>
<tr>
<td>Significance*</td>
<td>NS</td>
<td>NS</td>
<td>0.01 &gt; p &gt; 0.001</td>
<td>NS</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Values are mean ± SD.
*Comparison of results at 0 and 6 months within group by paired sample t test.
†Comparison of results at 0 months between groups by two-sample t test. 0.02 > p > 0.01.

was not significantly different. By the end of the study, both groups had increased their time to maximum exercise, but the change was greater in the trained subjects. Neither group showed any change in the rate-pressure product.

The subjects fasted overnight before each visit and blood was removed for analysis of VLDL (with and without chylomicrons), LDL and HDL, at 0, 1, 2, 4 and 6 months. At 0 and 6 months, additional blood was removed for separation of HDL₃ and HDL₄ subfractions as described below. Both the trained and control groups performed exercise electrocardiography at these times.

Training Schedule

The Canadian Air Force 5BX plan is an incremental exercise program. In our recent study of healthy volunteers, we found that this was an acceptable exercise program that could be easily followed. It also produced the best results in terms of fitness. Since the patients had definite coronary heart disease the interval between each increment was lengthened to 10 days.

The training group received instruction in the exercise from physiotherapists. They were assessed by the physiotherapists at monthly intervals for the first 4 months and then at 6 months to monitor progress. The control group did not receive prescribed exercise.

Exercise Electrocardiography

Exercise electrocardiography was performed using the Bruce protocol. During the exercise test, the ECG (modified V₆) and blood pressure were monitored.

Lipoproteins

For separation of VLDL, LDL and HDL, 20 ml of blood was taken after an overnight fast, and the serum was separated by low-speed centrifugation. For preparation of subclasses of HDL, an additional 50 ml of blood was taken.

VLDL, LDL and HDL were separated from serum by preparative ultracentrifugation/heparin-MnCl₂ precipitation. The cholesterol concentrations of serum and lipoprotein fractions and the triglyceride concentrations of plasma and VLDL fractions were estimated as previously described. Each patient's sera were stored at −20°C until completion of the study for estimation of apolipoprotein A-1 (apo A-1) by immunonephelometry.

HDL subfractions were prepared by rate-zonal ultracentrifugation. The subfractions were concentrated, dialyzed and stored at −20°C until completion of the 6-month study. The total protein, cholesterol, triglyceride and phospholipid contents of each subfraction were estimated and apo A-1 in the subfraction was analyzed by immunonephelometry.

Statistical Methods

Results that fell within normal distributions are expressed as mean ± SD. Results for total triglyceride, VLDL triglyceride and VLDL cholesterol, HDL₄ and the ratio of HDL₂ to HDL₃ had distributions that were positively skewed. They are expressed as median and semiquartile range. These distributions became normal after logarithmic transformation of the data, and parametric statistical analyses could then be applied.

To determine whether the trained and control groups were well-matched, their initial lipoprotein concentrations were compared by two-tailed t test. The control group established the inherent variability of plasma lipoproteins during the period of study; for example, did the clinical interest influence the subjects to modify their lifestyle? The lipoprotein-lipid and apolipoprotein concentrations of the control group at 0, 1, 2, 4 and 6 months were assessed by analysis of variance of linear regression to determine if there was any trend in these results during the period of study. The compositions of the HDL subfractions of the control group after 6 months were compared with their initial values by paired sample t test. After these
questions had been satisfactorily resolved, we could establish whether training per se exerted an effect on the lipoproteins. The techniques of analysis of variance of linear regression and paired-sample *t* test were used to analyze data available at serial times and those determined only at the start and end of the 6-month exercise program. For lipoproteins that did change on training, linear regression analysis was performed either on the results or on their logarithms to decide whether there were any relationships between changes in the lipoprotein classes and also between changes in lipoproteins and in treadmill exercise test performance.

### Results

#### Total Lipids, Apolipoproteins and Major Lipoprotein Classes

The initial lipid and apolipoprotein concentrations of the control and trained subjects were well matched (table 2). No significant differences were found between the two groups. The concentrations in the control group did not change during the study.

In the trained group, total triglyceride concentration decreased; triglyceride or cholesterol in VLDL, the major transporter of triglyceride, did not change significantly. The LDL cholesterol concentration also decreased. The HDL cholesterol concentration increased, as did apo A-1, the major protein component of HDL. There was no significant correlation between these changes in lipoproteins.

#### Subfractions HDL₂ and HDL₃

The control and trained patients had similar initial HDL subfraction concentrations (table 3). The concentrations in the control group did not change. In the trained group, the HDL₂ subfraction increased significantly on training, but the HDL₃ subfraction did not. The rise in the HDL₂ subfraction correlated with the logarithm of the fall in total triglyceride concentration (*r* = 0.63; 0.05 > *p* > 0.01). The concentrations of the individual lipids and apolipoproteins in the lipoprotein subfractions are listed in table 4. As with total subfraction concentrations, significant changes occurred only in the trained group.

### Relationship Between Changes in Lipoproteins and in Physical Fitness

Linear regression analysis was used to assess whether there was significant relationship between the changes in total triglyceride, in LDL and HDL cholesterol, and in the HDL₃ subfraction and changes in performance on the treadmill test; no relationship was found.

### Discussion

The difference in initial treadmill test performance may be because the group that was to undergo training had been instructed in the possible advantages of an exercise program and was probably more highly motivated. The rate-pressure product was similar in both groups. After the study, both groups increased their time to maximum exercise, with the change being more significant in the trained group. The changes at 9 months after infarction compared with 3 months may at least partly reflect the increased familiarization of the subjects with the test and perhaps also their greater physical confidence. The rate-pressure product did not change in either group. This is similar to previous findings. The problems that apply in relating treadmill exercise test performance to physical activity have been reviewed.

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**Table 2. Lipid and Apolipoprotein Concentrations of the Trained and Control Groups During the 6 Months of Study**

<table>
<thead>
<tr>
<th>Group</th>
<th>Time (months)</th>
<th>Total cholesterol (mg/dl)</th>
<th>Total triglyceride (mg/dl)</th>
<th>Total Apo A-I (mg/dl)</th>
<th>VLDL triglyceride (mg/dl)</th>
<th>VLDL cholesterol (mg/dl)</th>
<th>LDL cholesterol (mg/dl)</th>
<th>HDL cholesterol (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trained</td>
<td>0</td>
<td>253 ± 32</td>
<td>159 (60)</td>
<td>122 ± 26</td>
<td>96 (40)</td>
<td>25 (12)</td>
<td>176 ± 32</td>
<td>49 ± 9</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>248 ± 33</td>
<td>157 (71)</td>
<td>125 ± 25</td>
<td>97 (66)</td>
<td>26 (15)</td>
<td>168 ± 36</td>
<td>52 ± 10</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>247 ± 33</td>
<td>151 (58)</td>
<td>125 ± 25</td>
<td>87 (52)</td>
<td>25 (17)</td>
<td>167 ± 37</td>
<td>53 ± 10</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>242 ± 32</td>
<td>140 (58)</td>
<td>128 ± 25</td>
<td>97 (44)</td>
<td>24 (15)</td>
<td>165 ± 34</td>
<td>54 ± 11</td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>246 ± 31</td>
<td>124 (47)</td>
<td>137 ± 26</td>
<td>71 (47)</td>
<td>23 (14)</td>
<td>163 ± 37</td>
<td>56 ± 13</td>
</tr>
<tr>
<td>Control</td>
<td>0</td>
<td>259 ± 46</td>
<td>177 (44)</td>
<td>122 ± 30</td>
<td>113 (34)</td>
<td>23 (11)</td>
<td>178 ± 31</td>
<td>51 ± 9</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>255 ± 43</td>
<td>184 (38)</td>
<td>119 ± 29</td>
<td>137 (31)</td>
<td>29 (13)</td>
<td>174 ± 36</td>
<td>50 ± 9</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>264 ± 45</td>
<td>189 (44)</td>
<td>119 ± 27</td>
<td>126 (47)</td>
<td>32 (6)</td>
<td>179 ± 43</td>
<td>50 ± 10</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>259 ± 45</td>
<td>186 (38)</td>
<td>130 ± 29</td>
<td>128 (43)</td>
<td>31 (6)</td>
<td>183 ± 39</td>
<td>50 ± 8</td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>261 ± 43</td>
<td>168 (35)</td>
<td>127 ± 29</td>
<td>113 (49)</td>
<td>24 (12)</td>
<td>180 ± 40</td>
<td>51 ± 8</td>
</tr>
</tbody>
</table>

Lipids are from 19 trained and 23 control subjects; apo A-I is from 18 trained and 16 control subjects. Results for total cholesterol and apo A-I, LDL and HDL cholesterol are mean ± sd. Results for total and VLDL triglyceride and VLDL cholesterol are median (semiquartile range) because they did not have normal distributions. These data were converted to logarithms, which normalized their distributions, before being subjected to statistical analysis.

* *Analysis of variance of linear regression.*

Abbreviations: VLDL = very low density lipoprotein cholesterol; LDL = low-density lipoprotein cholesterol; HDL = high-density lipoprotein cholesterol.
TABLE 3. Concentrations of Lipoprotein Subfractions of the Trained and Control Groups at the Beginning and End of the 6-month Study

<table>
<thead>
<tr>
<th>Group</th>
<th>Time (months)</th>
<th>HDL$_2$ (mg/100 ml) median (semiquartile range)</th>
<th>HDL$_3$ (mg/100 ml) median (mean ± sd)</th>
<th>HDL$_2$:HDL$_3$ median (semiquartile range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trained (n = 14)</td>
<td>0</td>
<td>9.1 (9.1)</td>
<td>139 ± 28 (0.069)</td>
<td>0.062</td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>17.2 (9.1)</td>
<td>135 ± 31 (0.134)</td>
<td></td>
</tr>
<tr>
<td>Significance*</td>
<td></td>
<td>0.01 &gt; p &gt; 0.001</td>
<td>NS</td>
<td>0.01 &gt; p &gt; 0.001</td>
</tr>
<tr>
<td>Control (n = 18)</td>
<td>0</td>
<td>10.2 (4.4)</td>
<td>146 ± 30 (0.036)</td>
<td>0.067</td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>8.6 (3.1)</td>
<td>145 ± 28 (0.017)</td>
<td></td>
</tr>
<tr>
<td>Significance*</td>
<td></td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

Results for HDL$_2$ and HDL$_2$:HDL$_3$ were converted to logarithms, which normalized their distributions, before being subjected to statistical analysis.
*Paired-sample t test.
Abbreviations: HDL = high-density lipoprotein.

After the program, the trained subjects showed increased HDL cholesterol and apo A-1 and decreased total triglyceride and LDL cholesterol concentrations. The rise in HDL could be ascribed to an increase in the HDL$_2$ subfraction. No significant change was found in any lipoprotein fraction in the control group; therefore, the lipoprotein changes found in the trained group cannot be ascribed to the effect of the clinical interest, or to the natural progression after myocardial infarction or to any variation in performance of the lipoprotein assays. The findings are in agreement with the findings from studies on normal subjects$^4$-$^10$ that physical activity affects the major lipoprotein classes in a characteristic and possibly beneficial manner. Although the total triglyceride concentration fell in the trained group, no change occurred in the triglyceride and cholesterol concentrations or in the ratio of the two lipids in VLDL, the main endogenous transporter of triglyceride. The decrease in total triglyceride is therefore probably due to a decrease in triglyceride in the LDL class, since triglyceride in the HDL class increased.

No correlation was found between the decrease in total triglyceride and that in LDL cholesterol. A recent population survey revealed a quasiparabolic relationship between VLDL triglyceride and LDL cholesterol, which probably reflects the known metabolic relationships between the two lipoprotein

TABLE 4. Composition of Lipoprotein Subfractions of the Trained and Control Groups at the Beginning and End of the 6-month Study

<table>
<thead>
<tr>
<th>Group</th>
<th>Time (months)</th>
<th>Cholesterol (mg/dl)</th>
<th>Triglyceride (mg/dl)</th>
<th>Phospholipid (mg/dl)</th>
<th>Total protein (mg/dl)</th>
<th>Apo A-1 (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trained (n = 14)</td>
<td>0</td>
<td>1.6 (1.6)</td>
<td>0.6 (0.5)</td>
<td>2.5 (1.6)</td>
<td>4.5 (2.5)</td>
<td>1.9 (1.0)</td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>3.4 (1.5)†</td>
<td>1.2 (0.7)*</td>
<td>4.3 (1.8)†</td>
<td>8.3 (2.9)†</td>
<td>3.4 (1.9)†</td>
</tr>
<tr>
<td>HDL$_2$</td>
<td>0</td>
<td>23.5 ± 7.5</td>
<td>5.9 ± 3.1</td>
<td>29.0 ± 9.9</td>
<td>83.6 ± 16.3</td>
<td>35.0 ± 12.2</td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>23.6 ± 10.4</td>
<td>5.9 ± 2.1</td>
<td>27.6 ± 6.2</td>
<td>80.2 ± 18.9</td>
<td>33.5 ± 13.1</td>
</tr>
<tr>
<td>Control (n = 18)</td>
<td>0</td>
<td>2.2 (0.7)</td>
<td>1.0 (1.0)</td>
<td>2.5 (1.5)</td>
<td>4.6 (1.9)</td>
<td>1.9 (1.1)</td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>1.8 (0.8)</td>
<td>0.8 (0.4)</td>
<td>2.0 (0.8)</td>
<td>4.1 (1.4)</td>
<td>1.7 (0.8)</td>
</tr>
<tr>
<td>HDL$_2$</td>
<td>0</td>
<td>24.0 ± 5.1</td>
<td>5.7 ± 3.2</td>
<td>33.8 ± 8.0</td>
<td>82.0 ± 17.0</td>
<td>38.9 ± 9.2</td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>24.2 ± 5.4</td>
<td>6.0 ± 3.5</td>
<td>33.3 ± 8.6</td>
<td>80.9 ± 14.6</td>
<td>36.0 ± 9.1</td>
</tr>
</tbody>
</table>

Results for HDL$_2$ are median and semiquartile range. These data were converted to logarithms, which normalized their distribution before being subjected to statistical analysis. Results for HDL$_3$ are mean ± sd.
Comparison of results at 0 and 6 months by paired sample t test:
*0.02 > p > 0.01;
†0.01 > p > 0.001.
Abbreviation: HDL = high-density lipoprotein.
classes. No such correlation was found in the present study, probably because of the much smaller number of subjects and the narrower range of VLDL-triglyceride concentrations.

The total HDL₂ concentration and its individual components all increased on exercise, with no alteration in percent composition. This suggests that the total number of HDL₂ particles in circulation had increased. The concentration of HDL₁ showed no consistent change. HDL₄ and HDL₅ are metabolically interrelated. HDL₅ particles acquire lipids and apoproteins from the catabolism of triglyceride-rich lipoproteins under the action of lipoprotein lipase, and are then converted to the larger, more lipid-rich particles, HDL₄. The correlation between the increase in HDL₄ and the fall in total triglyceride on training is consistent with this metabolic relationship. As has been reviewed in recent publications physical exercise leads to an increase in muscle and adipose tissue lipoprotein lipase.

Our data and those from other studies indicate that changes in lipoproteins, particularly an increase in HDL, can be achieved with moderate exercise. This is important in the management of patients with proved coronary heart disease who cannot perform strenuous exercise. However, we found no direct relationship between exercise performance on a treadmill and changes in the major lipoprotein classes or in the HDL₄ subclass. This is consistent with the findings of a recent epidemiologic study of the relationship between physical activity and HDL cholesterol.

Although there is no definitive metabolic evidence as to why a high HDL and a low LDL concentration protects against atherosclerosis, possible mechanisms have been suggested. Plasma LDL is the precursor of lipid and protein components deposited in the arterial wall in atherosclerosis. A low plasma LDL concentration should therefore be advantageous. A possible benefit of a high HDL level follows from the role of HDL as a carrier of free cholesterol removed from tissues, with esterification of this cholesterol under the action of lecithin-cholesterol acyltransferase. Another possible mechanism, which does not preclude the first, is inhibition by HDL or a subtraction of HDL, of the cellular uptake of LDL, so that HDL can increase the rate of cholesterol efflux from, and decrease the cholesterol influx into cells.

Epidemiologic studies suggest that a high HDL and a low LDL concentration may protect against coronary heart disease. There is also evidence that regular exercise may be of value in the primary and secondary prevention of the disease. We have shown that exercise increases HDL, with its main effect on the HDL₂ subclass, and that exercise decreases LDL.

Acknowledgment
We thank the physiotherapists who supervised the exercise program, the physiologic measurement technicians who assisted with the exercise electrocardiography, and the dieticians who took the dietary histories. We are grateful to Dr. J. Shepherd for advice on isolation of lipoprotein subfractions and for preparation of antiserum to apo A-1. E.A. Strevans provided helpful suggestions in the statistical analysis of the data.

References
Myocardial Infarct Extension: Incidence and Relationship to Survival

JOHN T. BAKER, M.D., DEAN A. BRAMLET, M.D., ROBERT M. LESTER, M.D., DAVID G. HARRISON, M.D., CHARLES R. ROE, M.D., AND FREDERICK R. COBB, M.D.

SUMMARY  Myocardial infarct extension, defined as reelevation or reappearance of creatine phosphokinase-MB (CK-MB) 48 hours after the onset of symptoms, was evaluated prospectively in 56 consecutive patients with acute myocardial infarction. Myocardial infarct extension occurred in eight patients (14%). The sensitivity, specificity and predictive accuracy in the diagnosis of myocardial infarct extension were 63%, 85% and 42%, respectively, for recurrent chest pain requiring morphine; 50%, 65% and 19% for recurrent ST-segment elevation on routine 12-lead ECGs; and 88%, 63% and 28% for reelevation of total CK. Three of the eight episodes of extension were clinically silent. Four of eight patients (50%) with extension died, compared with one of 46 patients (2%) without extension (p = 0.0009). CK-MB persisted for 72 hours or longer in 16 patients and identified seven of eight patients who subsequently had infarct extension.

We conclude that myocardial infarct extension is an infrequent complication of acute myocardial infarction and is associated with a very high mortality rate. Persistence of CK-MB for 72 hours or more identifies a subgroup of patients at high risk for subsequent infarct extension and death.

The frequency of myocardial infarct extension, its clinical manifestations, and its relationship to morbidity and mortality are poorly defined. The reported frequency of myocardial infarct extension ranges from 9–86%.1, 2 In general, studies that define myocardial infarct extension on the basis of myocardial-specific enzyme changes report a lower incidence of extension1, 3 than studies that define it by electrocardiographic changes.2, 4 The classic clinical hallmark of myocardial infarct extension are recurrent chest pain or a sudden deterioration in functional class. However, myocardial infarct extension may be a silent complication of acute myocardial infarction.2, 4 The relationship between myocardial infarct extension and the acute prognosis of patients with myocardial infarction has not been clearly defined. Studies that reveal a high frequency of myocardial infarct extension show an associated low hospital mortality,2, 4 and studies that reveal a low frequency of myocardial infarct extension show an associated high hospital mortality.1, 3 The value of many reports on myocardial infarct extension is limited by the small number of patients studied or by failure to examine myocardial infarct extension prospectively.2, 4, 7

The purpose of the present study was to determine prospectively the frequency, clinical manifestations and relationship to hospital mortality of myocardial infarct extension more than 48 hours after the onset of symptoms in a consecutive group of patients with acute myocardial infarction.
The effect of moderate physical exercise on the plasma lipoprotein subfractions of male survivors of myocardial infarction.
F C Ballantyne, R S Clark, H S Simpson and D Ballantyne

Circulation. 1982;65:913-918
doi: 10.1161/01.CIR.65.5.913

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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