Rationale of Exercise Therapy in Patients with Angina Pectoris with Normal and Impaired Ventricular Function*

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The implementation of comprehensive coronary care (CCC), including multiple measures involving physical, psychologic, and pharmacologic procedures, together with a new concept of pharmacologic and surgical revascularization, have caused a remarkable improvement in the treatment of coronary patients with angina pectoris. The role of physical training per se is one measure of CCC and one must observe the fact that a single factor cannot be expected to influence a multifactorial disease. The irrefutable evidence that shows that physical training has an effect on longevity is lacking. On the other hand, a number of important physiologic and psychologic benefits have been found to be accompanied by prolonged exercise therapy in selected patients with angina pectoris suffering from coronary artery disease. The definitive evidence scientifically documented which would prove the importance of exercise therapy also as a secondary preventive measure is lacking because the randomized trials were relatively of a short duration, and they have included a rather small number of patients. The reasons for reaching any conclusive outcome were the number of unavoidable biases involved in long-term prospective studies, including high drop-out and drop-in rates, patient compliance, and contaminations. There are many studies that were not randomized, but controlled, with a follow-up of more than 15 years, which have shown lower mortality rates in the intervention group, but certainly they cannot be accepted as more than anecdotal experience. There is sufficient material in the literature concerning the hemodynamic and physiologic effects of a physical training program.

Physiologic Benefits of a Physical Training Program

It is generally accepted that physical training improves circulatory conditions and diminishes cardiac work. As a response to effective physical training, heart rate and rate-pressure product for given work levels decrease. The proper training effect includes the following: (1) decrease of heart rate, systolic blood pressure, the rate-pressure product, an increase of stroke volume, overall physical work performance, oxygen pulse, and in some instances, the rise of the angina pectoris threshold heart rate and threshold rate-pressure product in patients with angina pectoris.

The discussion as to whether physical exercise should be initiated in coronary patients with stable angina pectoris may make the impression of being superfluous. Thus, a discussion has been held during the years many times and there is no doubt to date that the benefit of exercise therapy in this group of patients has been well-established. It can be stated that patients after myocardial infarction with or without impaired ventricular function can reach a physiologic training effect after 16 weeks of an extensive training program. Despite the rather great number of well-designed studies that can be found in the literature, some cardiologists are still having doubts about the basic necessity of the initiation of such a program. It must be repeatedly underlined that physical exercise is only one measure that should be a part of a comprehensive coronary care (CCC) program, which includes multiple measures involving physical as well as psychologic and pharmacologic procedures together and this is very important with a new concept of pharmacologic and surgical revascularization. It is not to be expected that the single use of physical training per se can influence a multifactorial disease. There is, in my opinion, no irrefutable evidence that shows that physical training has an effect on longevity. The studies that have been multicenter randomized trials and have demonstrated a beneficial effect in the intervention group in comparison to a randomized control are not very many. On the other hand, a number of important

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short-term training. Interestingly, it was found that the coronary patient has a much better tolerance for physical exertion than believed. Certainly, in assessing the physiologic response of training, one must differentiate between the various types of training. The effect of physical training is dependent on sufficient intensity, duration, and frequency, in order to produce measurable effects and enhanced overall performance. We have found that the cardiocirculatory response in arm training is especially beneficial in patients with angina pectoris. In our experience, physical training in coronary patients should be supervised and implemented only according to possible benefits.

**Psychologic Benefits of Physical Training**

Evidence was given that physical training improves the emotional stability of an individual. It has been found that there is no correlation between training intensity, i.e., physiologic improvement and the psychologic effect. In coronary patients, fear, anxiety, and frustration decrease as a consequence of training. Furthermore, it enhances the return to work of the patient; his self-esteem and his general motivation to life also improve. It is our experience that in patients with angina pectoris undergoing prolonged physical training programs, there is a significant drop in absence at work and they are less dependent on drugs.

**Different Training Intensities**

To assess the importance of the training intensity and a possible placebo effect, we have studied the effect of different training programs in 33 patients after myocardial infarction with angina pectoris. Fifteen of these 33 patients underwent a low-intensity program based on calisthenics while the remaining 18 patients were switched after 40 weeks of calisthenics training to a high-intensity ergometric training. The patients were divided into 2 groups according to the severity of pain during stress testing and daily activities: (1) those with severe pain started intensive (90% of pain threshold heart rate), prolonged (continuous 30 min) ergometric training, and (2) patients with lesser complaints who continued with the calisthenics program. The results of the latter group, after 18 months of training, did not reveal a significant change in submaximal heart rate (HR), systolic blood pressure (SBP), O₂ pulse, double or triple product. However, in 20% of the patients, a higher pain threshold HR was tolerated and the higher double product reached before onset of anginal pain. Ergometric training caused a significant change in all the circulatory parameters mentioned above. In addition, 4 patients increased their pain threshold of both HR and double product. Twenty percent of the patients increased their maximal HR and double product, regardless of exercise intensity. The lowering of the systolic blood pressure x heart rate product (RPP) and the decrease of the triple product (RPP x LVET) is related to appearance of bradycardia and a decrease in systemic arterial pressure; the latter findings seem to be an important factor when evaluating the mechanical work of the heart after training (Table 1).

The upper extremity training is based on cycling at 90% of the subjective maximal work capacity. All the patients were trained twice weekly in a gymnasium supplied with resuscitation equipment. The reason for choosing arm ergometric training derived from earlier findings that for a given submaximal work load, heart rate, RPP, minute ventilation, and oxygen consumption were higher when compared with leg work. The most interesting finding was that the angina decreased after 3 months of arm training. In

**Table 1—Circulatory Measurements in Patients with Angina Pectoris before and after 4 Months of Intensive Ergometric Training Programs**

<table>
<thead>
<tr>
<th>Load and Time</th>
<th>O₂ Pulse, ml O₂/Beat</th>
<th>HR, Beats/min</th>
<th>RPP/100</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before</td>
<td>After</td>
<td>p</td>
</tr>
<tr>
<td>Rest 5 min at 55% of pain threshold heart rate</td>
<td>4.2 ± 1.4</td>
<td>4.4 ± 1.04</td>
<td>NS</td>
</tr>
<tr>
<td>10 min at 90% of pain threshold heart rate</td>
<td>7.7 ± 1.8</td>
<td>8.7 ± 1.5</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>25 min at 90% of pain threshold heart rate</td>
<td>9.8 ± 1.5</td>
<td>9.9 ± 1.8</td>
<td>NS</td>
</tr>
</tbody>
</table>

*HR = heart rate; O₂P = oxygen pulse; RPP = rate-pressure product; NS = not significant.

**Table 2—Comparison of Arm vs Leg Work Capacity (Watts), Angina Pectoris Threshold Heart Rate (ATHR), and Threshold Rate Pressure Product (RPP) in 19 Patients with Angina Pectoris before (T1) and after (T2/T3) Training**

<table>
<thead>
<tr>
<th>T1</th>
<th>Watts</th>
<th>ATHR</th>
<th>p Value</th>
<th>RPP/100</th>
<th>Angina</th>
<th>No Angina</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arm</td>
<td>55 ± 7</td>
<td>130 ± 12</td>
<td>&lt;0.01</td>
<td>200 ± 30</td>
<td>3</td>
<td>16</td>
</tr>
<tr>
<td>Leg</td>
<td>80 ± 15</td>
<td>117 ± 10</td>
<td></td>
<td>195 ± 21</td>
<td>19</td>
<td>—</td>
</tr>
<tr>
<td>T2</td>
<td>Arm</td>
<td>55 ± 8</td>
<td>130 ± 10</td>
<td>&lt;0.01</td>
<td>200 ± 25</td>
<td>3</td>
</tr>
<tr>
<td>Leg</td>
<td>90 ± 10</td>
<td>117 ± 8</td>
<td></td>
<td>181 ± 15</td>
<td>16</td>
<td>3</td>
</tr>
<tr>
<td>T3</td>
<td>Arm</td>
<td>73 ± 15</td>
<td>135 ± 15</td>
<td>&lt;0.01</td>
<td>195 ± 20</td>
<td>1</td>
</tr>
<tr>
<td>Leg</td>
<td>90 ± 14</td>
<td>124 ± 12</td>
<td></td>
<td>207 ± 32</td>
<td>5</td>
<td>14</td>
</tr>
</tbody>
</table>

*T1 = before training; T2 = after 8 weeks of training; T3 = after 12 weeks of training.
addition, the peak heart rate was significantly higher during arm cycling when compared with leg cycling training. Table 2 shows the comparison of arm vs leg ergometry before (T1) and after (T2) leg and arm (T3) training of 16 weeks. The most striking finding was the decrease in angina complaints during arm exercise when compared with leg training. All 19 patients complained of angina pectoris before the program started. Eighteen of 19 had no chest pain during arm exercise at T1, while 14 of these were free of pain at T3. Threshold rate-pressure product was comparable at arm and leg exercise while threshold heart rate during upper extremity training was higher (Table 2).

**ANGINA PECTORIS THRESHOLD HEART RATE**

A comparative study prepared at our laboratory showed that in a 5-year follow-up, the mortality in angina pectoris patients with angina threshold heart rate (ATHR) of less than 120 beats/min was 25.3% in an untrained control group, as against 9.3% in a trained intervention group. In the group of patients with ATHR above 120 beats/min, there was again a significant difference in the 5-year mortality—8.3% in the control group and 2.5% in the intervention group. The control group visited our institute once or twice per year for follow-up only, while the trained patients attended our CCC program twice weekly.

**PERCEPTUAL AND PHYSIOLOGIC RESPONSES TO TRAINING**

Ben-Ari and Kellermann examined, at our laboratory, the physiologic and perceptual (RPE) responses to very low and moderate intensity training programs. Perceptual response was obtained using Borg's scale consisting of grades from 6 to 20, arranged as follows: 7 = very very light; 9 = very light; 11 to 19 = very hard. Four months of low intensity training showed significant (p < 0.01 to 0.08) decrease in HR, SBE, and RPE at similar work loads. Additional 4 months of moderate intensity resulted in further decrease in HR and RPE. Thus, in a well-balanced, controlled program, psychologic factors play a major role, rather than physiologic changes, in the first part of cardiac rehabilitation involving mostly low-intensity programs.

**EXERCISE THERAPY IN IMPAIRED VENTRICULAR FUNCTION**

Data available to date point to a wide range of conclusions concerning the effect of physical training in patients with impaired ventricular function with and without angina pectoris. While some authors speculate that ventricular function is improved after training, others found unchanged left ventricular dimensions and concluded that training has no direct influence on the myocardium, either beneficial or detrimental. It seems that controlled well-dosed physical training applied in coronary patients with mildly to moderately impaired ventricular function has no deteriorating effect on the myocardium. While there is doubt as to whether exercise has a beneficial influence on myocardial perfusion, ie, increase of coronary blood flow, one can, in our experience, safely recommend individually adapted supervised exercise training in selected patients with coronary artery disease. Most authors found unchanged ejection fraction, left ventricular end-diastolic pressure, and volume after training. It must be emphasized that methods to measure distinctive changes in left ventricular dimensions and especially of regional function still remain somewhat unsatisfactory.

On reviewing the literature, we have quoted 8 studies in which physical training was used in small groups of patients with coronary artery disease (CAD) in whom the ejection fractions were low. The aim of all these studies was to examine the effect of physical training on left ventricular performance. In all studies, a positive training effect was obtained. The duration of training varied from 2 months to 43 months, and this was also the case in patients with quite severely impaired left ventricular function. The recently published article by Carrol and co-workers on systolic function during exercise in patients with CAD seems to be of special interest. The authors found that systolic function in CAD is determined by acute and chronic alterations in regional function. During exercise, there is an interplay between regional dysfunction from ischemia or infarction and regional hyperfunction of nonischemic myocardium that determines global performance.

**DISCUSSION**

Physical training in selected patients with angina pectoris can be accepted as a therapeutic modality with the primary aim to improve the functional capacity and exercise performance of these patients. It has been demonstrated repeatedly that training not only improves physical work performance, but some data are available that myocardial oxygen delivery may be improved. The effect of training in these patients depends on the reduction of myocardial oxygen requirement (MVO2) at a given work intensity, suggested by a decrease of heart rate and RPE. Moreover, we have found that in just over 20% of our patients, a higher angina pectoris heart rate threshold and RPE have been obtained after training.

The possible mechanisms of these improvements are not quite clear. The following possibilities should be considered: an increased maximal VO2, enhanced oxygen supply; increased oxygen extraction; acceleration of collateral vessels; and decrease of myocardial ischemic episodes by influencing metabolic balance.

For most of the aforementioned interpretations, there exists no sufficient scientific proof, nor can definitive evidence be provided for other determinants of MVO2, such as left ventricular end-diastolic volume and left wall thickness, which are beneficially affected directly by physical training. However, there is some possibility that contractility may be improved as a consequence of long-term exercise programs.

On the other hand, there is little doubt about the beneficial psychologic effect and enhanced quality of life pattern as a consequence of training. This has been observed in all of our studies, whether or not a positive physiologic training effect was obtained. As demonstrated in one of our studies, there are significant changes in perceptual responses after training. We found that in our patients, pain perception was decreased, ie, the subjective feeling of pain was not only diminished but pain was often neglected.

In summary, it can be stated that the implementation of exercise therapy in angina pectoris patients, regardless of their ventricular function, can be recommended provided that absolute contraindications are strictly observed. The
importance of exercise training may become once again a modality of therapy worthwhile to critically reexamine in the light of new observations concerning the pathophysiology of myocardial ischemia. The reversion of abnormal metabolic and hormonal responses concerning the functional balance of the entolothelial system may in the not too distant future result in a reassessment of our therapeutic approach utilized presently in coronary patients suffering from angina pectoris. Nuclear studies have shown that at least in selected patients, physical training may improve left ventricular systolic function. However, the results of other studies concerning left ventricular performance and myocardial perfusion are equivocal.

Important data are accumulating in CAD patients with angina pectoris and impaired ventricular function. The results of our study with a follow-up of 4 years revealed that long-term exercise programs in this latter group cause no further deterioration of ventricular function.

Exercise therapy applied in selected patients with angina pectoris can be considered a therapeutic modality causing improvement in the quality of life and functional performance of these patients.

REFERENCES


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