A Study of Left Ventricular Function in Coronary Patients Before and After Physical Training

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SUMMARY  Fifteen subjects recovering from a myocardial infarction or suffering from angina were given a maximum effort test on a bicycle ergometer and hemodynamic and angiographic investigations before and after a period of physical training. The training program consisted of three sessions of 60 to 75 min each week for two months. Maximum effort tests showed that physical capacity had increased by 17% ($P < 0.02$) and that for the same amount of effort the heart rate had decreased by 13% and the blood pressure by 7% ($P < 0.01$).

ALTHOUGH THE BENEFICIAL EFFECTS OF PHYSICAL TRAINING in subjects suffering from coronary insufficiency have been demonstrated, its exact mechanism is not well understood. Hypotheses that training encourages development of the collateral circulation or increases myocardial contractility have not been confirmed. The few control coronary arteriograms which have been done have shown no appreciable changes in the coronary network. No direct comparative study of ventricular function and myocardial contractility in coronary patients before and after training has been carried out.

Training in coronary patients can be expected to lead to an improvement similar to that in healthy subjects when training for sport, but the high level of exercise required raises the question of whether this can adversely affect a diseased myocardium.

The object of the present study is to compare hemodynamic and angiographic studies before and after training in coronary patients suffering from varying degrees of myocardial damage.

Methodology and Material

Fifteen patients suffering from coronary insufficiency were selected to start a program of physical training. All of them had been studied hemodynamically and by coronary and left ventricular angiograms within one week before the first training session.

The 15 subjects were either convalescent from a myocardial infarction (12 cases of which 9 had residual anginal pain) or suffering from angina without evidence of previous infarction (3 cases). All patients were male, the mean age was 50 years (range, 40 and 65 years). The coronary lesions as seen at coronary angiography were in all cases severe, with stenosis at least equal to or more than 70% in two of the three main coronary arteries. In the 15 of the 15 subjects the lesions were more severe and involved all three main coronary arteries. Two patients had had a single coronary bypass, one of which had become obstructed. Eight patients had marked left ventricular akinesia or dyskinesia.

Hemodynamic and angiographic investigations showed no significant changes after training in the left ventricular end-diastolic pressure, ventricular volume fraction, VCF, percentage of shortening and segmental contractility, in the total group, in those patients whose contractility was considerably impaired, or in those who had large dyskinetic areas or widespread akinesia.

It is concluded that training had no direct influence on the myocardium, either beneficial or detrimental.

The physical program followed the pattern commonly practiced in our center. In short, a two month period of training composed of three weekly sessions of 60 to 75 min during which a variety of exercises (walking, running, cycling, rowing and gymnastics) were carried out. The total effort during these sessions was considerable both in intensity and in duration. During the exercises, the heart rate remained at about 80% of its maximum. As usual the subjects underwent a maximal effort test on a bicycle ergometer before and after the period of training. This test was done within eight days prior to the start of the training program and then within the same period after its completion. The stress test was done one or two days before the hemodynamic study. In two patients who had undergone coronary bypass, training was started two months after the operation, and the hemodynamic and exercise tests were done as with the other cases — a few days before the first training session and then again two months later after the last. During these control exercise tests, the following parameters were studied: degree of maximal effort (W max) in Watts; heart rate (HR); systolic blood pressure (BP) in mm Hg; product of the heart rate and systolic blood pressure at the same level of effort in both (before and after the two months training period) tests. Comparison of these parameters was made at the same level of effort in both tests, i.e., before and after the two months training period. This level of effort was usually the maximal effort value of the pretraining test.

The initial hemodynamic and angiography study included the measurement of left ventricular pressures, coronary angiography, and left ventricular angiography in the right anterior oblique position. The post-training study was confined to pressure measurements and to left ventricular angiography. The heart rate in these two investigations was comparable to within 10 beats/min except in four cases where atrial pacing by means of a catheter introduced via the femoral vein was used so that the heart rate could be kept at the same rate as that seen in the first study.

Pressures were measured by means of an external manometer type Statham P 23 DB. The ventricular volumes were calculated from angiography of the left ventricle at 48 frames/second. Opacification of the ventricle was carried out through an 8F pigtail catheter with an injection of 40 to 50 ml of 26% meglumine diatrizoate at 12 ml/second. The end-diastolic and the end-systolic volumes were calculated by a method derived from Chapman's method which is an
The diastole.

of

intersection

systole

two axes at right angles to the longitudinal axis and parallel to the equatorial axis were drawn on the tracings of the ventricular cavities in both systole and diastole. The three axes labeled A, B and C from the base to apex separated the main longitudinal axis into three equal segments allowing segmental contractility to be studied.

The following parameters were measured or calculated: 1) left ventricular systolic pressure (LVSP) and end-diastolic pressure (LVEDP) in mm Hg; 2) left ventricular end-diastolic volume (LVEDV) and end-systolic volume (LVESV) in ml/m²; 3) ejection fraction (EF) in % according to the formula:

\[
\text{LVEDV} - \text{LVESV} \times 100
\]

4) mean shortening velocity of circumferential fibers (VCF) at the level of the transverse axis A, B (equatorial axis) and C, expressed in circumferences per second (circ/sec) calculated by Karliner method; 5) percentage of shortening (% short) of the same axis A, B, and C were calculated.

A statistical comparison was made before and after training, using the paired Student's t-test.

Results

Data obtained during the maximum effort tests on the bicycle ergometer and from the hemodynamic studies, each performed before and after training, were compared.

After the two months training period, all 15 patients considered that they had greatly improved. This improvement was not only subjective but it was verified by a comparison of the exercise tests before and after training. The results of these tests are given in table 1 with individual and mean values.

The values of the hemodynamic parameters obtained in the 15 subjects before and after physical training are given with their individual and mean values and standard deviation in table 2. None of the differences in the measures was statistically significant.

If the eight subjects whose ventricular function was most affected (ejection fraction < 45%) are looked at separately, an improvement in the control exercise tests done before and after training is still apparent, though definitely less than in the case of the other subjects, but the hemodynamic and angiographic findings remained unchanged as for the whole group (table 3).

Discussion

Physical training had a favorable effect on all 15 patients. They felt much better and their subjective improvement was confirmed by a comparison of the results of the exercise tests done before and after. After training there was an increase in maximum effort and a decrease in the product HR × BP indicating less work by the heart for a comparable amount of effort, and thus an improvement in the circulation. The favorable results obtained in this group of 15 subjects are comparable to those obtained in a much larger series of patients after physical training in our center.

The comparison of the hemodynamic and angiographic data before and after training showed that there are no significant changes in the parameters studied. The ventricular volume was not increased and the ejection fraction was unchanged. In fact these parameters remained perfectly stable.

There was no change in segmental contractility as evaluated by the percentage of ventricular shortening and by VCF measured at three positions of the left ventricle. The pressures, especially the end-diastolic, showed considerable variations in both directions. It may be noted that all five patients who had left ventricular end-diastolic pressures above 15 mm Hg had these pressures lowered after training.

This could result from an improvement of left ventricular compliance and indicate some improvement of performance. However, it is recognized that pressure readings cannot be exactly reproduced in repeated studies, unlike those of angiography, and this was evident in the present study.

There was no change, either, in the subjects who had shown areas of akinesia and widespread dyskinesia at the first angiography.

The fact that there were no changes in the ventricular function parameters suggests that training has no direct effect upon the heart, and that its beneficial effect is in the peripheral vascular system. Previous hemodynamic studies carried out before and after physical rehabilitation have usually assessed cardiac function indirectly by measurement of the cardiac output, the pulmonary and systolic pressures and the arterio-venous oxygen difference. If they have shown that training brings about a clear fall in heart rate, a moderate decrease in the blood pressure, a decrease in the cardiac output and an increase in the arterio-venous O₂ difference. These vascular changes, which are due at least in part to an increase in vagal tone and decrease in sympathetic tone, lead to a better distribution of blood in the viscera and peripheral muscles. There is a decrease in the blood flow through the muscles during exercise, an increase in their capacity to extract oxygen and an improvement in their cellular metabolism at the mitochondrial level.

Several experimental studies have shown that physical training in animals increases the vascularity of the myocardium and the activities of enzymes and enhances the cardiac performance. On the other hand, it has been demonstrated that there was no increase in the intrinsic contractile state of the myocardium in hearts of physically trained animals. However, extrapolation of these experimental data to humans would be speculative. In one study using echocardiography, it has been suggested that exercise training in normal subjects could improve cardiac performance.

The present study shows that training has no effect on the myocardium of patients with coronary disease, a finding which contradicts Hellerstein's hypothesis that training in man can lead to an increase in myocardial contractility.

Since angiography before and after training showed no lessening of coronary artery narrowing, physical training can be said to have no direct effect on the heart. If the present work shows that no improvement in left ventricular function in coronary patients results from physical training, it is also of interest to see that no harm...
was done to the heart in patients whose coronary arteries were severely affected and ventricular function impaired. Even in the eight subjects with gross impairment of ventricular function, there was no change in the state of the heart, especially no aggravation. This is important for the clinician since he needs assurance when he selects patients for a training program that these subjects are likely to benefit from training. In general, those with signs of frank ventricular failure and those with a large aneurysm are routinely excluded. Even when the clinical state and the hemodynamic findings differ, a clinical improvement may be brought about by changes in the peripheral circulation although the ventricular myocardium remains unimproved.

Until recently it was assumed that physical activity is dangerous in coronary patients. Experience in the rehabilitation units has shown, however, that effort tolerance is much greater than would have been expected under this assumption. Our findings that the myocardial functions did not change — and deficiencies were not exacerbated — further confirms the high tolerance of coronary disease patients for this type of physical training.

The methodology deserves comment. The number of patients in the study was not very large considering the variations in coronary lesions and in the initial myocardial lesions. Since the hemodynamic studies had to be repeated at the end of training, the control hemodynamics were deliberately done only in the basal state so as to limit the duration of the study, as adding a stress technique would have increased the number of procedures and lengthened the duration of the investigation. However, since the physical training involves dynamic stress on the circulatory system, provocative techniques such as exercise or pacing might have uncovered changes in ventricular function. Finally, the period of training (3 sessions a week for 2 months) may have

Table 1. Results of the Maximal Exercise Tests Before and After Physical Training

<table>
<thead>
<tr>
<th>Pt</th>
<th>W max. (Watts)</th>
<th>HR at equal W</th>
<th>BP (mm Hg) at equal W</th>
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<td>B</td>
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<tr>
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W = before training; A = after training; W max = degree of maximal effort; HR = heart rate; BP = blood pressure; sd = standard deviation.

Table 2. Results of the Hemodynamic and Angiographic Study Before and After Physical Training

<table>
<thead>
<tr>
<th>Pt</th>
<th>LVSP (mm Hg)</th>
<th>LVPD (mm Hg)</th>
<th>EDVI (ml/m²)</th>
<th>EF %</th>
<th>% Shortening</th>
<th>VCF Axis A</th>
<th>Axis B</th>
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Abbreviations: B = before training; A = after training; LVSP = left ventricular systolic pressure; LVPD = left ventricular diastolic pressure; EDVI = end-diastolic volume index; EF = ejection fraction; VCF = mean shortening velocity of circumferential fibers.
been too short to have any effect on myocardial performance. Nevertheless this training was always sufficient to produce a definitive improvement in the clinical parameters studied during the comparative exercise tests.

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