

CARDIAC REHABILITATION OF PATIENTS WITH CORONARY HEART DISEASE:  
A CRITICAL EVALUATION OF ITS BENEFICIAL EFFECTS

MEDICAL GRAND ROUNDS

The University of Texas Health Science Center at Dallas

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## I. INTRODUCTION

### A. Definition of Cardiac Rehabilitation

1. Hellerstein, H.K., Ford, A.B.: Rehabilitation of the cardiac patient. JAMA 164:225, 1957.
2. Stoeckel, K.G.: The principles of conducting exercise programs. In: Exercise Testing and Exercise Training in Coronary Heart Disease, edited by J. Naughton, H.K. Hellerstein. New York: Academic Press, 1973, p. 299.
3. Kellermann, J.J., Denolin, H. (eds.): Critical Evaluation of Cardiac Rehabilitation. Basel: S. Karger, 1977.
4. Denolin, H.: Rehabilitation of the coronary patient. In: Advances in Cardiology, Vol. 24: Cardiac Rehabilitation, edited by K. Konig, H. Denolin. Basel: S. Karger, 1978, p. 1.
5. Fox, S.M. III: Heart disease and rehabilitation: scope of the problem. In: Heart Disease and Rehabilitation, edited by M.L. Pollock, D.H. Schmidt. Boston: Houghton Mifflin, 1979, p. 3.
6. Hellerstein, H.K.: Cardiac rehabilitation: a retrospective view. Ibid, p. 509.
7. Shephard, R.J.: Cardiac rehabilitation in prospect. Ibid, p. 521.
8. Froelicher, V.F., and Pollock, M.L.: Cardiac rehabilitation: a new focus. J Cardiac Rehab 1:11, 1981.
9. Beljan, J.R. (for the Council): Physician-supervised exercise programs in rehabilitation of patients with coronary heart disease. (Council on Scientific Affairs, American Medical Association). JAMA 245:1463, 1981.
10. Wenger, N.K.: Rehabilitation of the coronary patient: scope of the problem and responsibility of the primary care physician. Cardiovasc Rev & Reports 2:1249, 1981.

In the early 1950's, a patient who suffered an acute myocardial infarction faced a minimum of six weeks of absolute bed rest and possibly a lifetime of invalidism. During the past 30 years the outlook for such a patient has changed considerably. Patients with coronary heart disease now commonly function in the most demanding of positions. Recent examples include Dwight Eisenhower and Lyndon Johnson of the United States, Menachem Begin of Israel, and Anwar Sadat of Egypt.

Cardiac rehabilitation in coronary heart disease has been defined as the process by which a patient is restored to an optimal physiological, psychological, and sociological status. If possible, the process should also decrease the risks of recurrent cardiac events and increase the life span.

During the past ten years cardiac rehabilitation has been slowly accepted as beneficial medical practice by many primary care physicians. The reason for this acceptance is that many patients with symptomatic coronary heart disease including angina pectoris, post-myocardial infarction, and post-coronary artery bypass surgery can be returned to active, satisfying and productive lives.

Knowledge concerning cardiac rehabilitation is disseminated by means of books, a new journal, an international congress, and special conferences and workshops. Also, there are cardiac rehabilitation programs at many of our major medical centers, and Dallas has had such programs for several years.

#### B. Components of Cardiac Rehabilitation

9. Loc. cit.
10. Loc. cit.
11. Erb, B.D., Fletcher, G.F., Sheffield, T.L.: Standards for cardiovascular exercise treatment program (AHA Committee Report). Circulation 59:1084A, 1979.

Cardiac rehabilitation consists of three components and these are shown in Figure 1.

Figure 1

## COMPONENTS OF CARDIAC REHABILITATION

- I. The Medical Component
- II. The Educational Component
- III. The Exercise Component

The medical component is under the direction of a physician who is in charge of the program. He is medically responsible for the patient, supervises his program, and makes recommendations to the personal physician for appropriate medical therapy.

The educational component is directed toward modifying coronary artery disease risk factors and changing the attitude concerning the patient's disease and occupational status. The emphasis on risk factor modification includes cessation of smoking, modification of diet, and control of high blood pressure.

The exercise component is directed toward early ambulation in the hospital and a prescribed program of dynamic exercise to increase cardiovascular physiological performance.



It must be emphasized that cardiac rehabilitation is not synonymous with dynamic exercise training and must include the medical and the educational components which are equally important. However, it is the exercise program that serves as the main focus for the program and makes it more than a simple counselling service. Also, the dynamic exercise training program causes cardiovascular physiological adaptations and may facilitate changes in coronary heart disease risk factors.

C. Phases of Cardiac Rehabilitation

10. Loc. cit.

11. Loc. cit.

Cardiac rehabilitation consists of three phases and these are shown in Figure 2.

Figure 2

## PHASES OF CARDIAC REHABILITATION

- I. The Inpatient Program
- II. The Outpatient Therapeutic Program
- III. The Outpatient Maintenance Program

The inpatient program is important for early ambulation and for increased physical activity to prevent deconditioning while the patient is still in the hospital. Also the educational component is started in this phase of the program.

The outpatient therapeutic program follows the inpatient program and is a prescribed medically supervised exercise program. It consists of three sessions per week for the purposes of delivering the desired goals of behavioral and vocational counselling and dynamic exercise training. In most cases this phase lasts from three to six months.

The outpatient maintenance program encourages the patient to maintain the level of physical training achieved in the therapeutic program. Also continued reinforcement must be given for coronary artery disease risk factor modification.

It is not the purpose of these Grand Rounds to be a "How-To Session" on Cardiac Rehabilitation. The information for the establishment of such programs is completely described in the books on cardiac rehabilitation that are in the reference list and can be obtained from numerous workshops which are continually being held over the country. It is the purpose of this Grand Rounds to present a critical evaluation of the beneficial effects of cardiac rehabilitation for patients with coronary heart disease.

## II. EVALUATION OF CARDIAC REHABILITATION

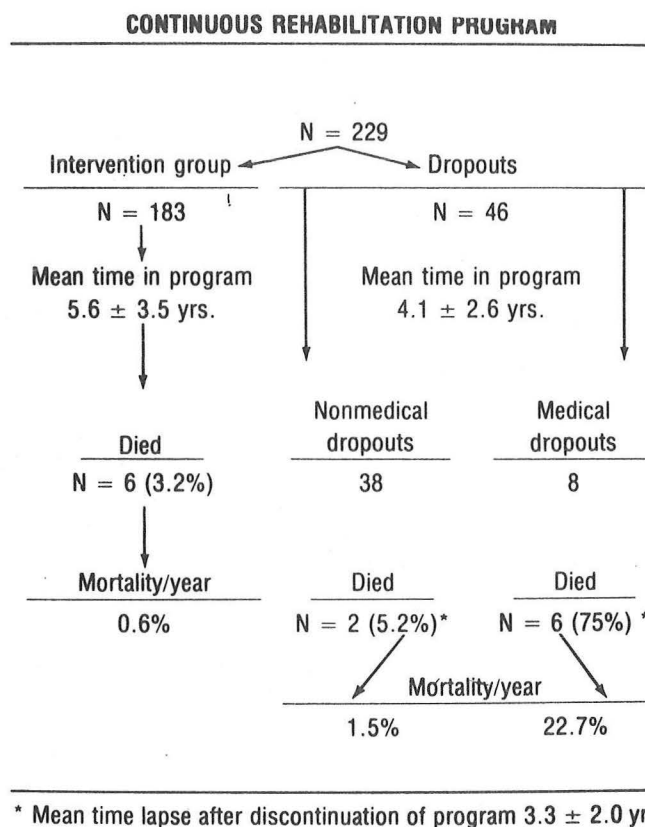
### A. Effects on Cardiovascular Mortality and Morbidity

12. Gottheiner, V.: Long-range strenuous sports training for cardiac reconditioning and rehabilitation. *Am J Cardiol* 22:426, 1968.
13. Bruce, R.A.: The benefits of physical training for patients with coronary heart disease. In: Controversy in Internal Medicine II, edited by F.J. Ingelfinger, R.V. Ebert, M. Finland, et al. Philadelphia: Saunders, 1974, p. 145.
14. Hirsch, E.Z., Hellerstein, H.K., Macleod, C.A.: Physical training and coronary heart disease. In: Exercise and the Heart, edited by R.E. Morse. Springfield: Charles C. Thomas, 1972.
15. Kellermann, J.J.: Rehabilitation of patients with coronary heart disease. *Prog Cardiovasc Dis* 17:303, 1975.
16. Kellermann, J.J.: Cardiac rehabilitation: reminiscences, international variations, experiences. *J Cardiac Rehab* 1:43, 1981.
17. Wilhelmsen, L., Sanne, H., Elmfeldt, D.: A controlled trial of physical training after myocardial infarction. *Prev Med* 4:491, 1975.
18. Rechnitzer, P.A., Sangal, S.A., Cunningham, D.A., Andrew, G., Buck, C., Jones, N.L., Kavanagh, T., Parker, J.O., Shephard, R.J., Yuhasz, M.S.: A controlled prospective study of the effect of endurance training on the recurrence rate of myocardial infarction; a description of the experimental design. *Am J Epidemiol* 102:358, 1975.
19. Naughton, J.: The national exercise and heart disease project. *Cardiology* 63:352, 1978.
20. Shaw, L.W. (for the Project Staff): The national exercise and heart disease project: effects of a prescribed supervised exercise program on mortality and cardiovascular morbidity in patients after a myocardial infarction. *Am J Cardiol* 48:39, 1981.
21. Kallio, V., Hamalainen, H., Hakkila, J., and Lauuvila, O.J.: Reduction in sudden death by a multifunctional intervention programme after acute myocardial infarction. *Lancet* 2:1091, 1979.
22. Naughton, J.: The national exercise and heart disease project. In: Physical Conditioning and Cardiovascular Rehabilitation, edited by L.S. Cohen, M.B. Mock, I. Ringqvist. New York: John Wiley & Sons, 1981, p. 247.
23. Kallio, V.: Evaluation of earlier studies: Europe. *Ibid*, p. 257.
24. Shephard, R.J.: Evaluation of earlier studies: Canada. *Ibid*, p. 271.
25. Froelicher, V.F., Brown, P.: Exercise and coronary heart disease. *J Cardiac Rehab* 1:277, 1981.

26. Froelicher, V.: Exercise and health. Amer J Med 70:987, 1981.
27. Blackburn, H.: Personal Communication.
28. Naughton, J.: Physical activity for myocardial infarction patients. Cardiovasc Rev & Reports 3:237, 1982.

Numerous uncontrolled studies over the years have demonstrated that selected post-myocardial infarction patients who are placed in a rehabilitation program have very low mortality rates. One such study was reported by Kellerman and his group and is shown in Figure 3.

Figure 3



Non-medical drop-outs from the program were considered to be the "control" group. The mortality rate in the intervention group was 0.6% and was 1.5% in the "control" group. Since the patients were not randomized at the beginning of the study, it is quite possible that the patients with the more severe disease dropped out of the program. No conclusions can be drawn from such uncontrolled studies.

Four randomized controlled trials concerning cardiac rehabilitation and its effects on cardiovascular mortality and morbidity have been reported. The first is the study by Wilhelmsen et al. conducted in Goteborg (Fig. 4).

Figure 4

### GOTEBORG EXERCISE TRIAL\*

EVENT	E		C		P
	N=158		N=157		
	#	%	#	%	
TOTAL DEATHS	28	18	35	22	0.40
SUDDEN CD	6		3		
NONFATAL MI	25	16	28	18	NS

**\*MEN AND WOMEN, AGED 55-57, RANDOMIZED AT ATTACK, 4 YEAR RATES.**

**PREVENTIVE MEDICINE, 4:1975.**

Patients were randomized at the time of their acute myocardial infarction into a control and an exercise group. They were followed for approximately four years. The number of cases randomized (158 in the exercise group and 157 in the control group) was inadequate. The difference in total and sudden coronary deaths and in non-fatal myocardial infarction was not statistically significant. No conclusions can be drawn concerning either positive or negative effects of exercise due to the number of patients.

The Ontario Exercise Heart Study asked the question whether high-intensity conditioning exercise (HIE) would produce a significant difference in recurrence rates of cardiovascular event as compared to low intensity exercise (LIE) insufficient to produce a conditioning effect (Fig. 5).

Figure 5

## ONTARIO EXERCISE HEART STUDY 1981\*

<u>EVENT</u>	<u>HIE</u> N=379		<u>LIE</u> N=354	
	#	%	#	%
RE-MI	36	9.5	26	7.3

**\*MEN, LATE CONVALESCENT, 4 YEAR RATES.**

In Figure 5, 379 individuals were randomized into the high-intensity group and 354 into the low-intensity group. The high intensity exercise group had a 9.5% reinfarction rate within four years compared to only a 7.3% in the low intensity group. Again, due to inadequate sample size, no conclusions of a positive or a negative effect in this study could be made.

The National Exercise Heart Disease Project (NEHDP) coordinated efforts among five university centers. These were the University of Alabama, Emory University, George Washington University, Case Western Reserve University, and Lankenau Hospital. Some of the results of that study are shown in Figure 6.

Figure 6

**NEHDP 1981\***

<b>EVENTS</b>	<b>E N=323</b>		<b>C N=328</b>	
	<b>#</b>	<b>%</b>	<b>#</b>	<b>%</b>
<b>ALL DEATHS</b>	<b>15</b>	<b>4.6</b>	<b>24</b>	<b>7.3</b>
<b>RECURRENT MI</b>	<b>29</b>	<b>9.0</b>	<b>31</b>	<b>9.5</b>
<b>SCD</b>	<b>8</b>		<b>6</b>	
<b>TOTAL CVD DEATHS</b>	<b>14</b>	<b>4.3</b>	<b>20</b>	<b>6.1</b>

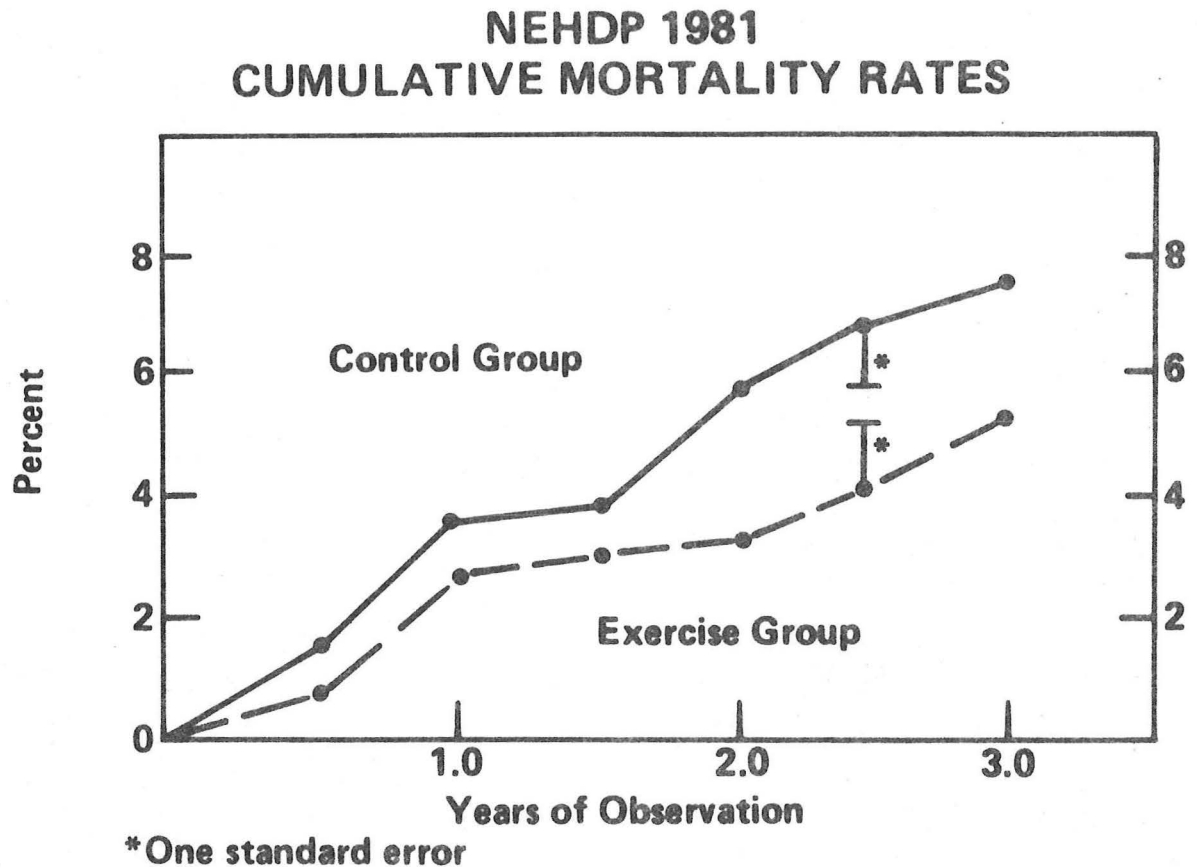
**\*MEN, AGES 30-64, LATE CONVALESCENT, 3 YEAR DEATH RATES.**

**AMERICAN JOURNAL OF CARDIOLOGY, 1981.**

There were 323 patients randomized into the exercise rehabilitation regimen and 328 patients into the control group. Follow-up was for approximately 3 years. Figure 6 indicates 4.6% deaths in the exercise group compared to 7.3% in the control group. However, there was no difference in total cardiovascular deaths (4.3% vs. 6.1%), recurrent myocardial infarctions (9% vs. 9.5%), and sudden cardiac death (8 patients vs. 6 patients). All cardiovascular mortality and morbidity were in a favorable direction for the exercise groups, but again the numbers were inadequate. For the more objective forms of coronary heart disease, i.e., recurrent myocardial infarction and coronary deaths, there was no difference.

However, it should be noted that the survival trends offer some possibility that an adequate sized trial would have shown an effect (Fig. 7).

Figure 7



The cumulative mortality rates for progressive time periods for the control and exercise group are not significantly different ( $p=.05$ ) at any time or collectively. However, if the 37 percent benefit from exercise reported in this study had been obtained in a comparable study of 1,400 patients, the results would have been "statistically significant".

Finally, the study by Kallio et al. in Finland is of importance. They reported data on a comprehensive cardiac rehabilitation program which included multiple risk factors intervention during a three year follow-up. The limitations of the study were the small number of patients and the fact that it was not designed to rate the independent effects of dynamic exercise training. It was the purpose of the program to reduce simultaneously smoking, blood pressure, and blood lipids, as well as to enhance cardiovascular fitness with dynamic exercise training (Fig. 8).

Figure 8

### FINNISH MULTIPLE RISK FACTOR TRIAL POST-INFARCTION\*

EVENTS	E		C		P
	N=188		N=187		
	#	%	#	%	
ALL DEATHS	42	21.8	56	29.9	0.10
SCD	11	5.8	27	14.4	0.01
TOTAL CHD DEATHS	35	18.6	55	29.4	0.02
NON FATAL MI	34	18.1	21	11.2	0.10
NEW Q/QS	49		45		

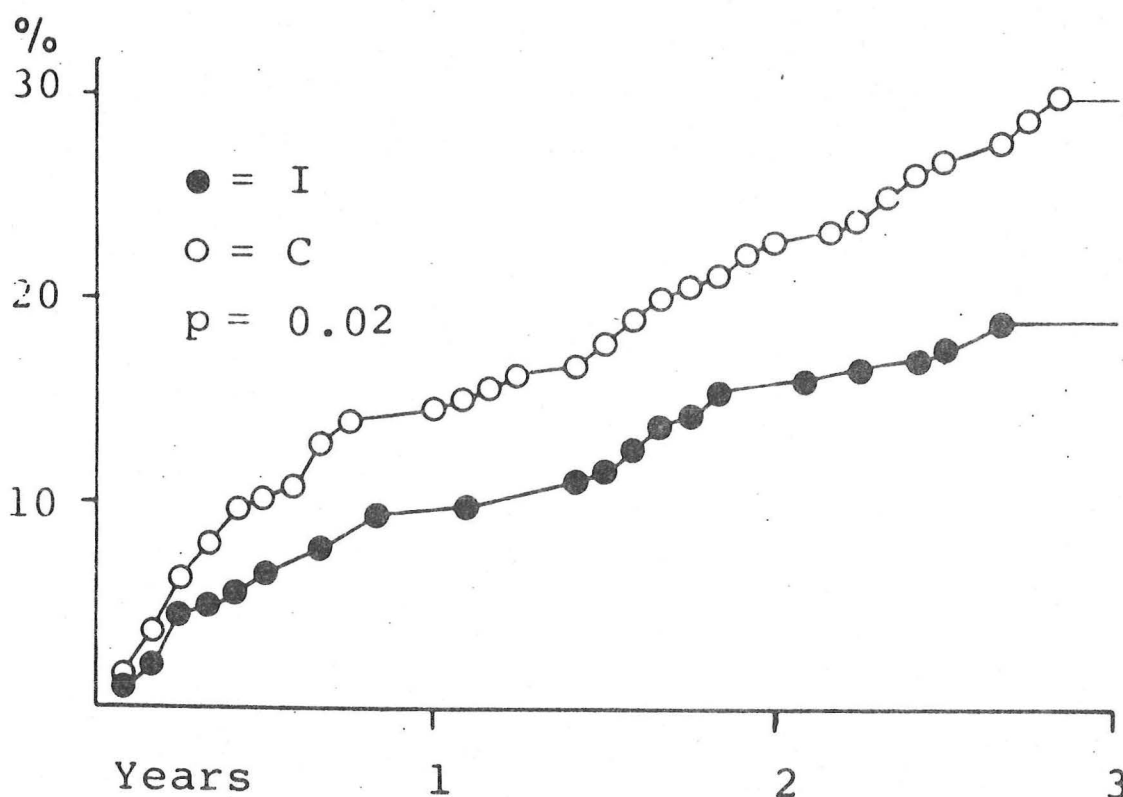
\*MEN AND WOMEN UNDER AGE 65, RANDOMIZED AT ATTACK, 3  
YEAR RATES.  
KALLIO, LANCET, 1979.

The difference in sudden coronary deaths and total coronary deaths are significantly lower in the "exercise" group. More objective manifestations, i.e., non-fatal myocardial infarction and new development of significant Q or QS wave patterns on the electrocardiogram, were not different between the groups.



The cumulative percentage of deaths from coronary heart disease in the intervention (I) and the control (C) groups are shown in Figure 9.

Figure 9



The survival trends clearly demonstrate the beneficial effect in the "exercise group". However, the significant results of this study of a secondary prevention program to lower multiple risk factors cannot be interpreted as due to dynamic exercise training. The study shows that a rational, prudent approach to secondary prevention by reducing risk factors along with dynamic exercise training and good medical care reduces cardiac deaths in patients with coronary heart disease. Such a strategy would also provide the most appropriate control for studies of surgical therapy for coronary heart disease.

B. Effects on Risk Factors for Coronary Heart Disease

15. Loc. cit.
16. Loc. cit.
29. McGee, D.: The probability of developing certain cardiovascular diseases in eight years at specified values of some characteristics. In: The Framingham Study. Section 28. DHEW Publication No. (NIH) 74-618. Washington, DC: US Government Printing Office, 1973.
30. Sorlie, P.: Cardiovascular disease and death following myocardial infarction and angina pectoris: the Framingham study, 20-year follow-up. Section 32. DHEW Publication No. (NIH) 77-1247. Washington, DC: US Government Printing Office, 1977, p. 1.
31. Kannel, W.B., McGee, D., Gordon, T.: A general cardiovascular risk profile: the Framingham study. *Am J Cardiol* 38:46, 1976.
32. Gordon, T., Sorlie, P., Kannel, W.B.: Coronary heart disease, atherothrombotic brain infarction, intermittent claudication--a multivariate analysis of some factors related to their incidence: Framingham study, 16-year follow-up. Section 27. DHEW Publication No. (NIH) 1740-0320. Washington, DC: US Government Printing Office, 1971.
33. Gordon, T., Castelli, W.P., Hjortland, M.C., Kannel, W.B., Dawber, T.R.: High density lipoprotein as a protective factor against coronary heart disease: the Framingham study. *Am J Med* 62:707, 1977.
34. Blackburn, H.: The potential for preventing reinfarction. In: Rehabilitation of the Coronary Patient, edited by N.K. Wenger, H.K. Hellerstein. New York: John Wiley & Sons, 1978, p. 67.
35. Kannel, W.B.: Cardiovascular disease: a multifactorial problem (insights from the Framingham Study). In: Heart Disease and Rehabilitation, edited by M.L. Pollock, D.H. Schmidt. Boston: Houghton Mifflin, 1979, p. 15.
36. Kannel, W.B.: Prospects for risk factor modification to reduce risk of reinfarction and premature death. *J Cardiac Rehab* 1:63, 1981.
37. Wallace, A.G.: Personal Communication.
38. Freis, E.D.: VA Cooperative Study. *JAMA* 213:1143, 1970.
39. Hjermann, I., VelveByre, K., Holme, I., Leren, P.: Effect of diet and smoking intervention on the incidence of coronary heart disease. Report from the Oslo Study Group of a randomized trial in healthy men. *Lancet* 3259:1303, 1981.
40. Castelli, W.P.: Personal Communication.

In epidemiological studies three primary risk factors have been identified as being very important in the development of coronary heart disease. These are an elevated serum cholesterol, smoking, and elevated blood pressure. Physical inactivity has also been incriminated, but the data is much less convincing. Recently it has been reported from the Framingham Study that an increased ratio of HDL cholesterol to total cholesterol appeared to be protective against the development of coronary heart disease. However, the interpretation of this finding is controversial, and the mechanism to explain the protective effect of HDL cholesterol has not been found experimentally.

Several studies have reported the effects of a Cardiac Rehabilitation program on coronary heart disease risk factors. The data from the study by Kellerman are shown in Figure 10.

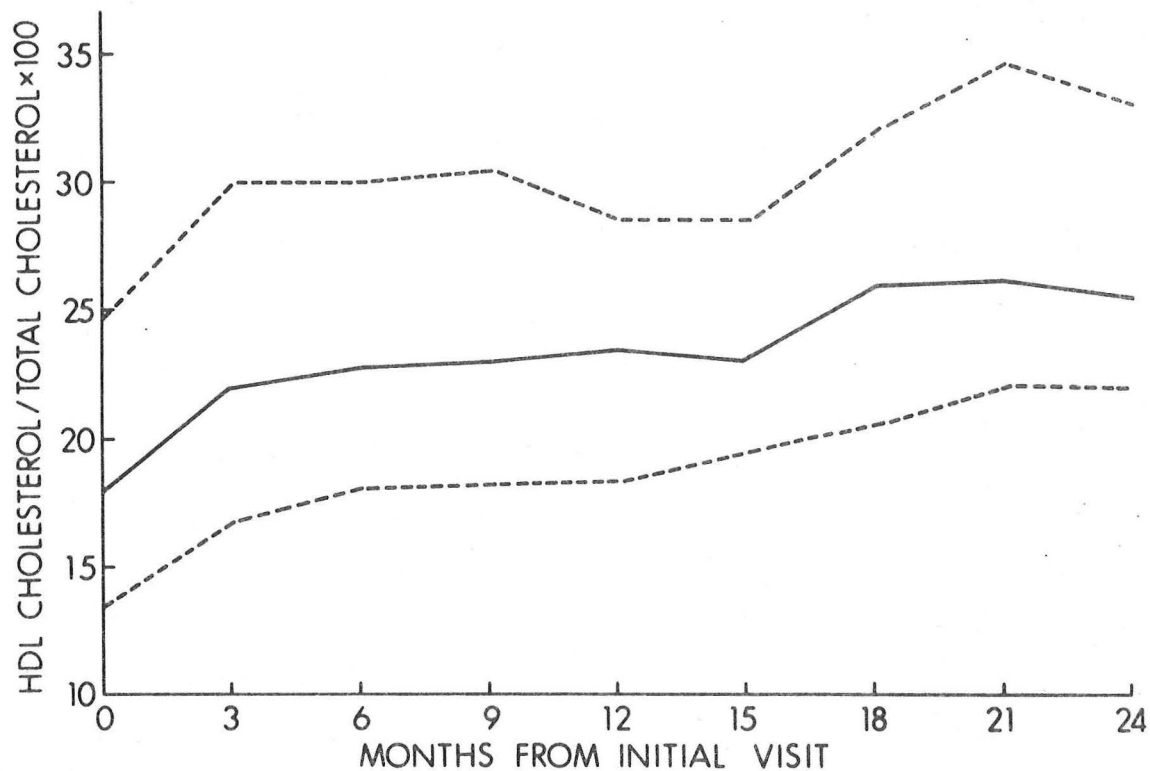
Figure 10

RISK FACTORS BEFORE AND AFTER A CONTINUOUS REHABILITATION PROGRAM			
N = 183			
Cholesterol	I	239.5 $\pm$ 53.0	$P < 0.001$
	II	217.7 $\pm$ 51.5	
<hr/>			
Systolic blood pressure	I	133.2 $\pm$ 19.4	$P < 0.001$
	II	124.5 $\pm$ 16.8	
<hr/>			
Diastolic blood pressure	I	87.3 $\pm$ 11.0	$P < 0.001$
	II	78.9 $\pm$ 8.8	
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Smoking	Nonsmokers		21.4%
	Smokers		10.9%
	Stopped smoking		66.5%
<hr/>			
Mean cigarettes per day		Mean years of smoking	
27.6		28.1	

Cholesterol and systolic and diastolic blood pressures were higher before (I) entering the program than after (II) a mean follow-up of 5.6 years. There was also a marked reduction in the number of smokers (77.4% vs. 10.9%).

Similar type changes have been found in risk factor modification in the Duke University Preventive Approach to Cardiology (DUPAC) program of Wallace et al. The effects on the ratio of HDL cholesterol to total cholesterol is shown in Figure 11.

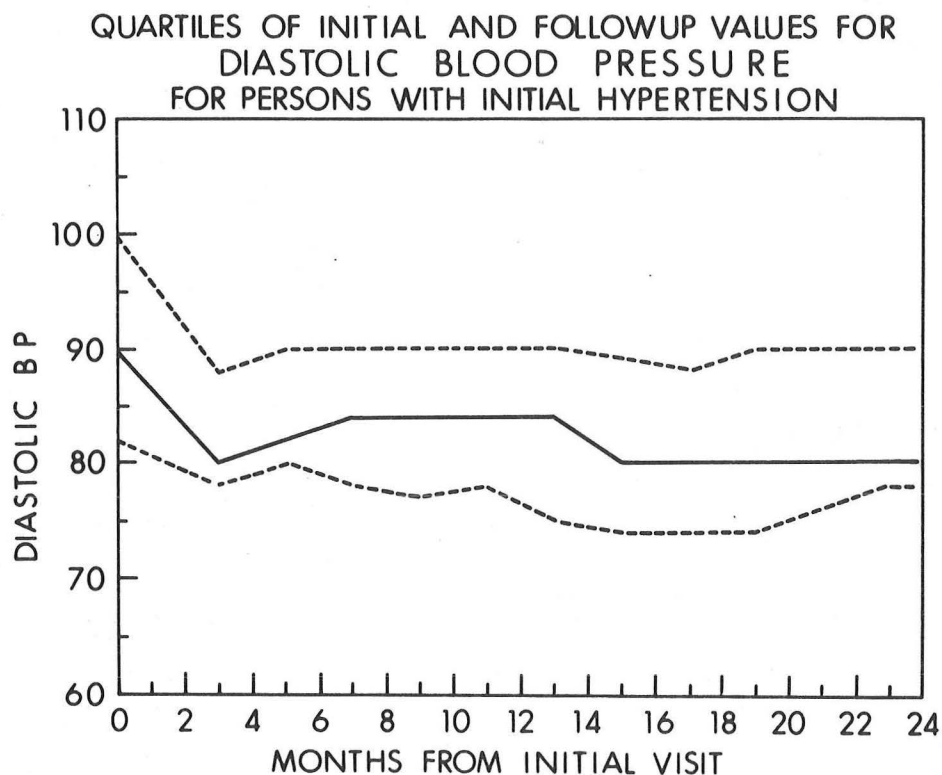
Figure 11



At the start of the program the ratio was 18 and after two years the ratio was 25. As stated earlier, the meaning of this change is controversial.

In the DUPAC study there was also a reduction in diastolic blood pressure in those who were initially hypertensive as is shown in Figure 12.

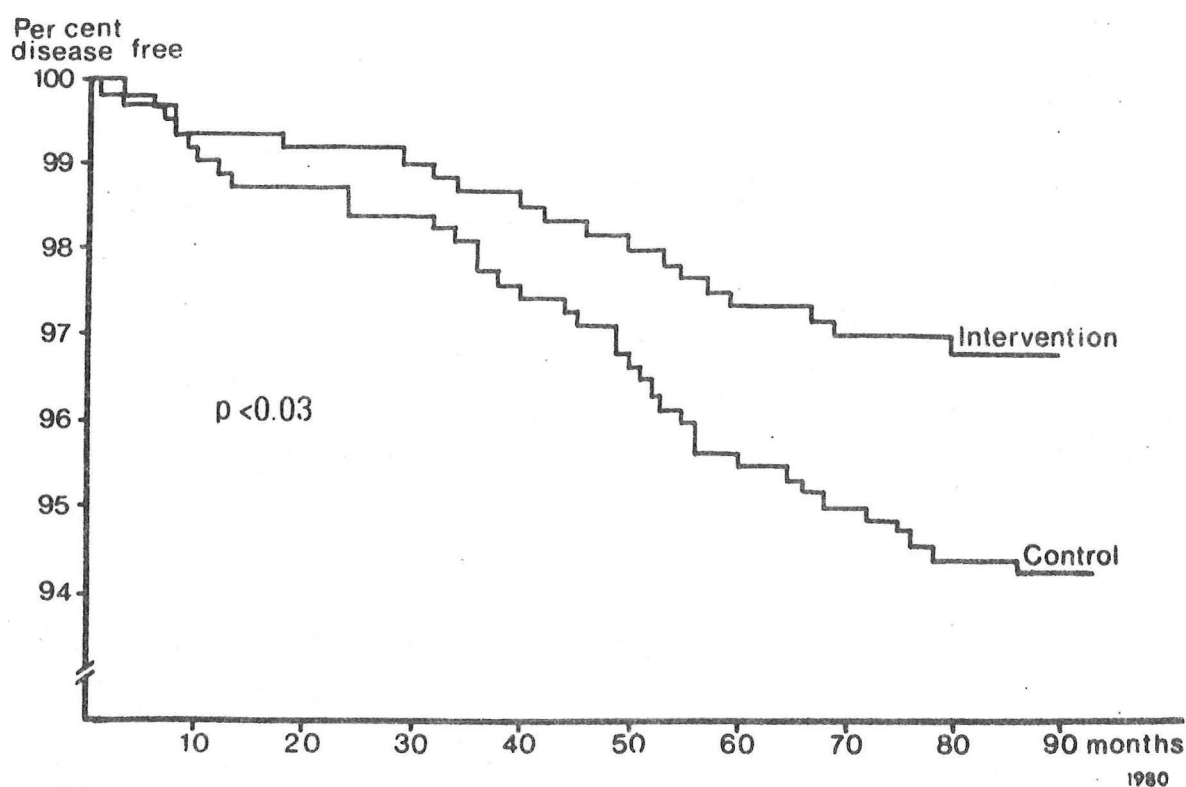
Figure 12



In this group the mean diastolic pressure was 90 mmHg upon entering into the program and 80 mmHg after two years of cardiac rehabilitation.

It does seem clear that a comprehensive cardiac rehabilitation program for patients with coronary heart disease can change the primary risk factors in a favorable direction. However, there is still some debate concerning whether such risk factor modification can alter the clinical course of coronary heart disease. The study of Freis conducted in our VA Hospital indicates that blood pressure control decreases the morbidity and mortality in coronary heart disease. Also, a recently published primary prevention study from Oslo indicates that decreased smoking and decreased levels of cholesterol reduce the incidence of coronary heart disease. Results of this study are shown in Figure 13.

Figure 13



The study was conducted on healthy, middle-aged (40-49 years old) men at high risk for coronary heart disease. They were normotensive but had elevated levels of serum cholesterol and all smoked. The men in the intervention group were given informative advice concerning diet and smoking habits, and this was frequently reinforced. At the conclusion of the study, serum cholesterol concentrations were 13% lower in the intervention group, and smoking was 45% lower. At the end of the observation period, the incidence of myocardial infarction (fatal and non-fatal) and sudden death was 47% lower in the intervention group.

### C. Cardiovascular Physiological Effects

41. Clausen, J.P., Larsen, O.A., Trap-Jensen, J.: Physical training in the management of coronary artery disease. *Circulation* 40:143, 1969.
42. Kasch, F.W., Boyer, J.L.: Changes in maximum work capacity resulting from six months' training in patients with ischemic heart disease. *Med & Sci Sports* 1:156, 1969.
43. Clausen, J.P., Trap-Jensen, J.: Effects of training on the distribution of cardiac output in patients with coronary artery disease. *Circulation* 42:611, 1970.
44. Detry, J.R., Rousseau, M., Vandenbroucke, G., Kusumi, F., Brasseur, L.A., Bruce, R.A.: Increased arteriovenous oxygen difference after physical training in coronary heart disease. *Circulation* 44:109, 1971.
45. Ehsani, A.A., Heath, G.W., Hagberg, J.M., Sobel, B.E., Holloszy, J.O.: Effects of 12 months of intense exercise training on ischemic ST-segment depression in patients with coronary artery disease. *Circulation* 64:1116, 1981.
46. Ehsani, A.A., Biello, D.R., Bloomfield, S.A., Holloszy, J.O.: Exercise training improves intrinsic left ventricular performance in ischemic heart disease. *Clin Res*, in press, 1982 (abst.).
47. Kitamuro, K., Jorgensen, C.R., Gobel, F.L., Taylor, H.L., Wang, Y.: Hemodynamic correlates of myocardial oxygen consumption during upright exercise. *J Appl Physiol* 32:516, 1972.
48. Gobel, F.L., Nordstrom, L.A., Nelson, R.R., Jorgensen, C.R., Wang, Y.: The rate-pressure product as an index of myocardial oxygen consumption during exercise in patients with angina pectoris. *Circulation* 57:549, 1978.
49. Holmberg, S., Wieslaw, S., Varnauskas, E.: Coronary circulation during heavy exercise in control subjects and patients with coronary heart disease. *Acta Med Scand* 190:465, 1971.
50. Sonnenblick, E.H., Ross, J., Jr., Braunwald, E.: Oxygen consumption of the heart; newer concepts of its multifactorial determination. *Am J Cardiol* 22:328, 1968.
51. Mitchell, J.H., Hefner, L.L., Monroe, R.G.: Performance of the left ventricle. *Am J Med* 53:481, 1972.
52. Trap-Jensen, J., Clausen, J.P.: Effect of training on the relation of heart rate and blood pressure to the onset of pain in effort angina pectoris. In: Coronary Heart Disease and Physical Fitness, edited by O.A. Larsen, R.O. Malmberg. Baltimore: University Park Press, 1971.

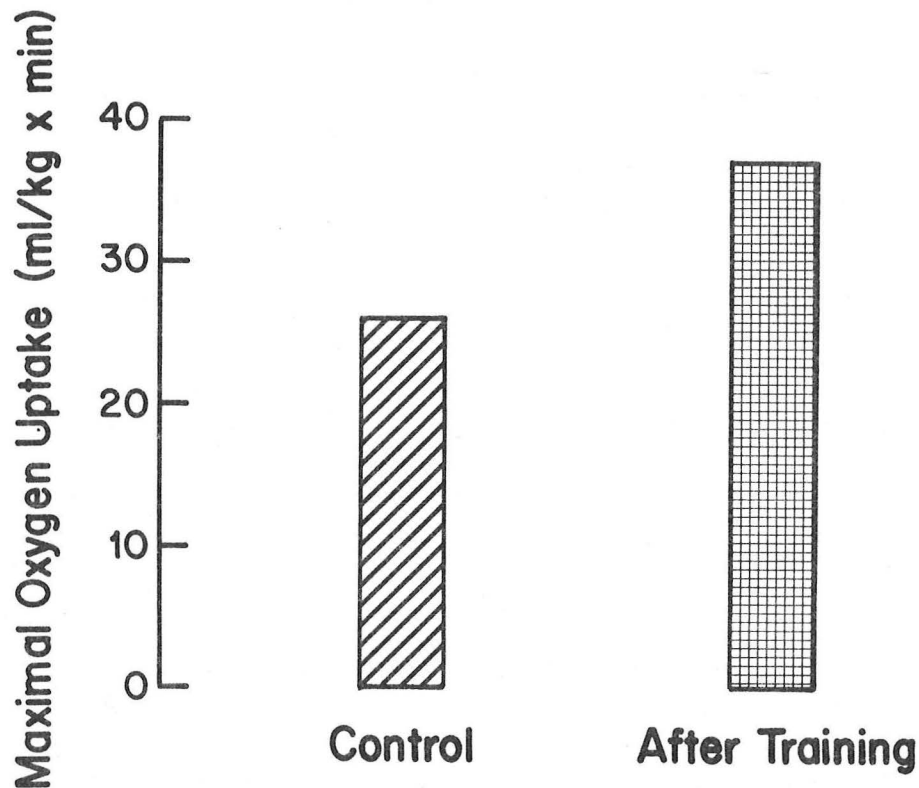
53. Redwood, D.R., Rosing, D.R., Epstein, S.E.: Circulatory and symptomatic effects of physical training in patients with coronary artery disease and angina pectoris. *N Engl J Med* 286:959, 1972.
54. Mitchell, J.H.: Exercise training in the treatment of coronary heart disease. *Adv Intern Med* 20:249, 1975.
55. Dehn, M.M., Pansegrau, D.G., Mitchell, J.H.: Exercise training after acute myocardial infarction. In: Exercise and the Heart, edited by N.K. Wenger. *Cardiovasc Clinics* 9:117, 1978.
56. Saltin, B.: Central circulation after physical conditioning in young and middle-aged men. In: Coronary Heart Disease and Physical Fitness, edited by O.A. Larsen, R.O. Malmberg. Copenhagen: Munksgaard, 1971, p. 21.
57. Haskell, W.L.: Mechanisms by which physical activity may enhance the clinical status of cardiac patients. In: Heart Disease and Rehabilitation, edited by M.L. Pollock, D.H. Schmidt. Boston: Houghton Mifflin, 1979, p. 276.
58. Raffo, J.A., Luksic, I.Y., Kappagoda, C.T., Mary, D.A.S.G., Whitaker, W., Linden, R.J.: Effects of physical training on myocardial ischaemia in patients with coronary artery disease. *Brit Heart J* 43:262, 1980.
59. Hindman, M.D., Wallace, A.G.: Radionuclide exercise studies. In: Physical Conditioning and Cardiovascular Rehabilitation, edited by L.S. Cohen, M.B. Mock, I. Ringqvist. New York: John Wiley & Sons, 1981, p. 33.
60. McManus, B.M., Waller, B.F., Graboyes, T.B., Mitchell, J.H., Siegel, R.J., Miller, H.S., Jr., Froelicher, V.F., Roberts, W.C.: Exercise and sudden death: physiologic and morphologic features and implications for management and prevention. Part I. *Curr Prob Cardiol* 6(9):1, 1981.
61. Hellerstein, H.K., Hornsten, T.R., Goldbarg, A., Burlando, A.G., Friedman, E.H., Hirsch, E.Z., Maris, S.: The influence of active conditioning upon subjects with coronary artery disease: cardiorepiratory changes during training in 67 patients. *Canad Med Assn J* 96:758, 1967.
62. Kattus, A.A., Grollman, J.: Patterns of coronary collateral circulation in angina pectoris: relation to exercise training. In: Changing Concepts in Cardiovascular Disease, edited by H. Russek, B. Zahman. Baltimore: Williams & Wilkins, 1972, p. 352.
63. Ferguson, R.J., Petitclerc, R., Choquette, G., Chaniotis, L., Gauthier, P., Hout, R., Allard, C., Jankowski, L., Campeau, L.: Effect of physical training on treadmill exercise capacity, collateral circulation and progression of coronary disease. *Am J Cardiol* 34:764, 1974.



64. Conner, J.F., LaCamera, F., Jr., Swanick, E.J., Oldham, M.J., Holzaepfel, D.W., Lyczkowskyj, O.: Effects of exercise on coronary collateralization -- angiographic studies of six patients in a supervised exercise program. *Med Sci Sports* 8:145, 1976.
65. Froelicher, V.F., Sebrechts, C., Streitwieser, D., Battler, A., McKirnan, M.D., Ashburn, W.: Rest and exercise electrocardiograms and radionuclides in patients presenting for cardiac rehabilitation. *Clin Cardiol* 4:59, 1981.
66. Jensen, D., Atwood, J.E., Froelicher, V., McKirnan, M.D., Battler, A., Ashburn, W., Ross, J., Jr.: Improvement in ventricular function during exercise studied with radionuclide ventriculography after cardiac rehabilitation. *Amer J Cardiol* 46:770, 1980.
67. Froelicher, V., Jensen, D., Atwood, J.E., McKirnan, M.D., Gerber, K., Slutsky, R., Battler, A., Ashburn, W., Ross, J., Jr.: Cardiac rehabilitation: evidence for improvement in myocardial perfusion and function. *Arch Phys Med Rehab* 61:517, 1980.
68. Atwood, E., Jensen, D., Froelicher, V., Gerber, K., Witztum, K., Slutsky, R., Ashburn, W.: Radionuclide perfusion images before and after cardiac rehabilitation. *Aviat Space Environ Med* 51:892, 1980.
69. Froelicher, V.F.: Personal Communication.
70. Eckstein, R.W.: Effects of exercise and coronary artery narrowing on coronary collateral circulation. *Circ Res* 5:230, 1957.
71. Wyatt, H.L., Mitchell, J.: Influences of physical conditioning and deconditioning on coronary vasculature of dogs. *J Appl Physiol* 45: 619, 1978.
72. McElroy, C.L., Gissen, S.A., Fishbein, M.C.: Exercise-induced reduction in myocardial infarct size after coronary occlusion in the rat. *Circulation* 57:958, 1978.
73. Scheel, K.W., Ingram, L.A., Wildon, J.L.: Effects of exercise on the coronary and collateral vasculature of beagles with and without coronary occlusion. *Circ Res* 48:523, 1981.

Previously reported studies have demonstrated that dynamic exercise training can increase maximal oxygen uptake in patients with coronary heart disease. The training studies have usually lasted for less than six months and have found approximately a 20% or less increase in maximal oxygen uptake. Two recent studies have reported the results of training studies of a longer duration in patients with coronary heart disease. Ehsani et al. have recently reported the change in maximal oxygen uptake that occurs in coronary heart disease patients after a heavy dynamic exercise training program for 12 months (Fig. 14).

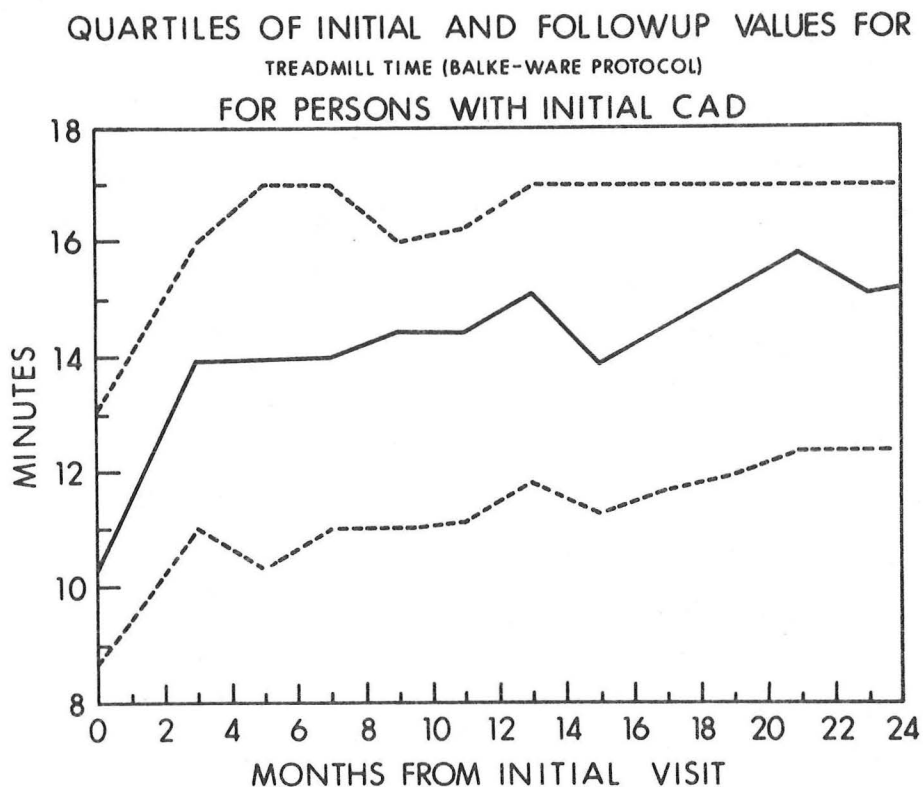
Figure 14



Maximal oxygen uptake was 26 ml/kg x min in the control study and increased to 35 ml/kg x min after 12 months of intensive exercise training. This represents a 35% increase in aerobic capacity and was significant at a p value <0.001.

Wallace et al. in the DUPAC program have studied the change in maximal exercise performance that occurred over a two-year training program in their coronary heart disease patients (Fig. 15).

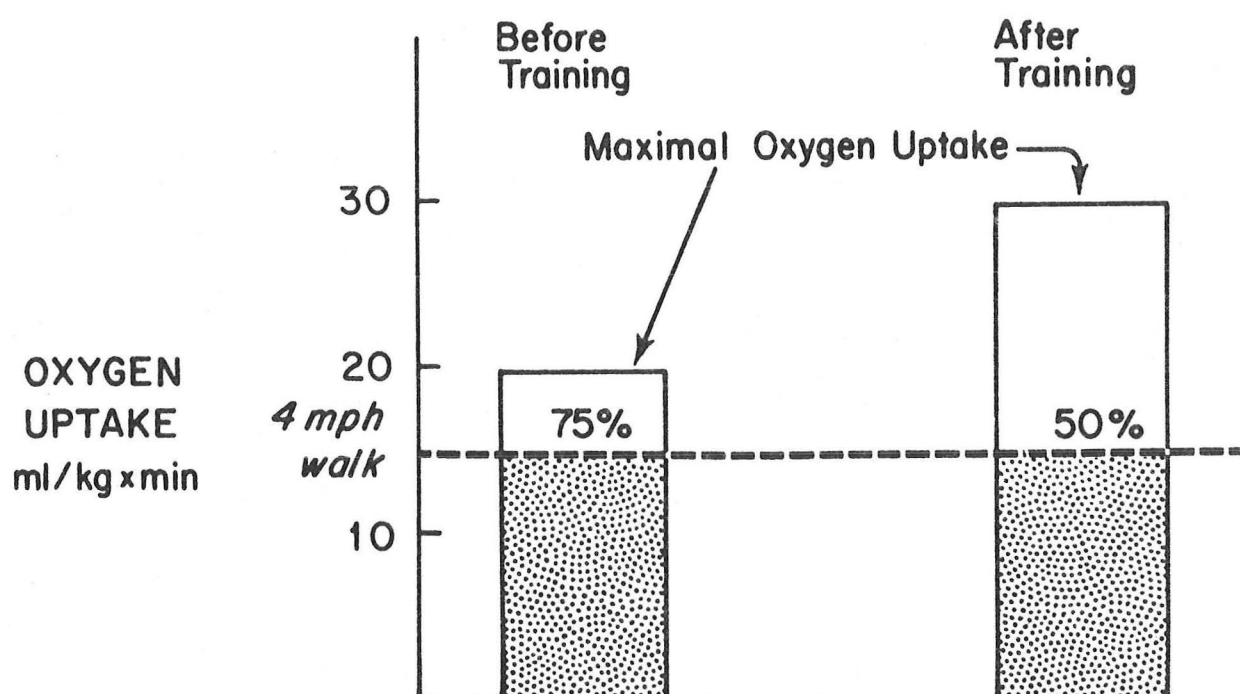
Figure 15



The maximal exercise testing was performed on a treadmill using the Balke-Ware protocol. The initial or control maximal time on the treadmill was 10 minutes which gives an estimated maximal oxygen uptake of 28 ml/kg x min. After two years of training, the maximal time on the treadmill was 15 minutes which gives an estimated maximal oxygen uptake of 36 ml/kg x min. This represents a 29% increase in aerobic capacity. It should be noted, however, that the major portion of the training effect occurred during the first three months of the program.

It is clear from these studies that maximal oxygen uptake of patients with coronary heart disease can be markedly increased by exercise training. Since patients will probably not be performing at such high workloads, this change in itself may not be of much clinical importance. However, before training the oxygen consumption at a given submaximal workload will be a higher percent of the patients' maximal oxygen uptake than it will be at the same workload after training. This may be the most important aspect of dynamic exercise training for coronary heart disease patients, and what this means is shown in Figure 16.

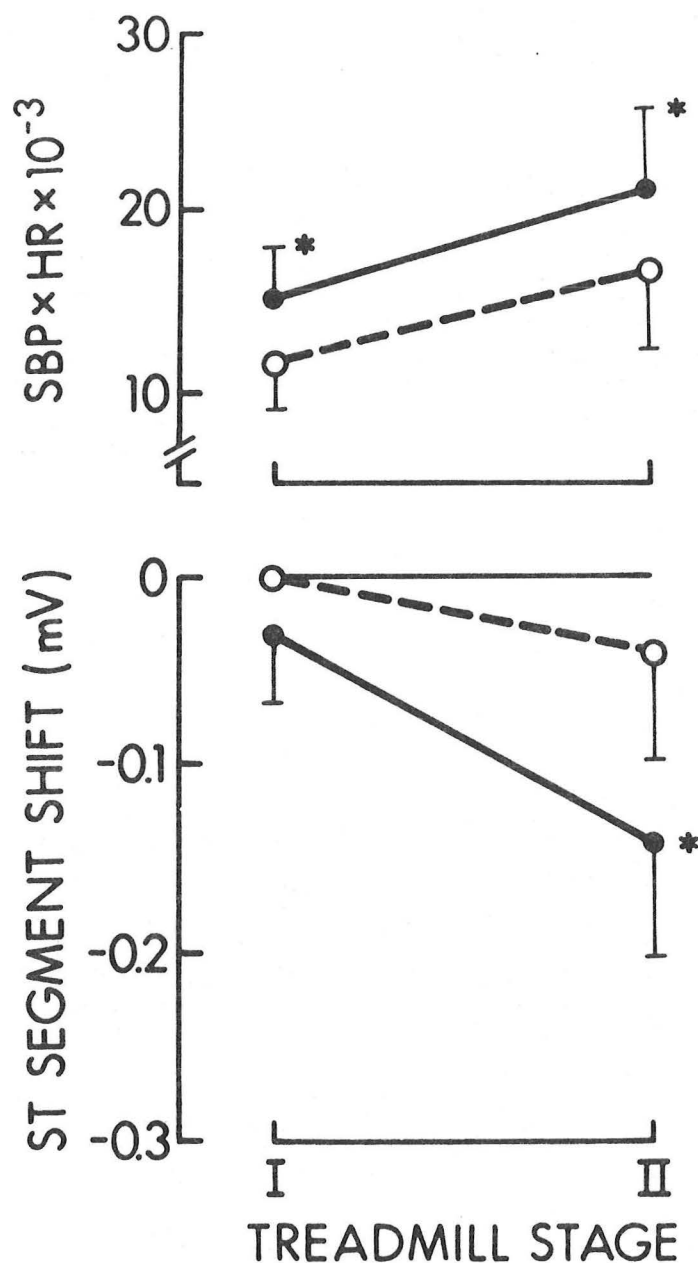
Figure 16



Maximal oxygen uptake is increased from 20 ml/kg x min to 30 ml/kg x min in a patient with coronary heart disease by dynamic exercise training. Before training walking at 4 mph which costs an oxygen uptake of 15 ml/kg x min represents 75% of the patient's maximal oxygen uptake. After training this same oxygen cost of walking represents only 50% of the maximal oxygen uptake. Since the heart rate and blood pressure response to a given workload is proportional to the relative oxygen cost, a reduction in heart rate and blood pressure at a given constant submaximal workload and total body oxygen consumption would occur with an increase in myocardial oxygen uptake. Also the level of activity of the sympathetic nervous system during exercise is related to the relative and not to the absolute workload.

Thus, one of the important aspects of the increase in maximal oxygen uptake that occurs with dynamic exercise training in patients with coronary heart disease is the cardiovascular effect at the same submaximal workload. Myocardial oxygen consumption has been shown to parallel closely the double product and other derived indices. It has been shown that the various indices of myocardial oxygen demand (double product, triple product) are less at a constant submaximal workload. Recently, Ehsani has reported the effects of dynamic exercise training on the double product and the extent of ST segment depression before training (●—●) and after training (○--○) at two constant submaximal workloads (Fig. 17).

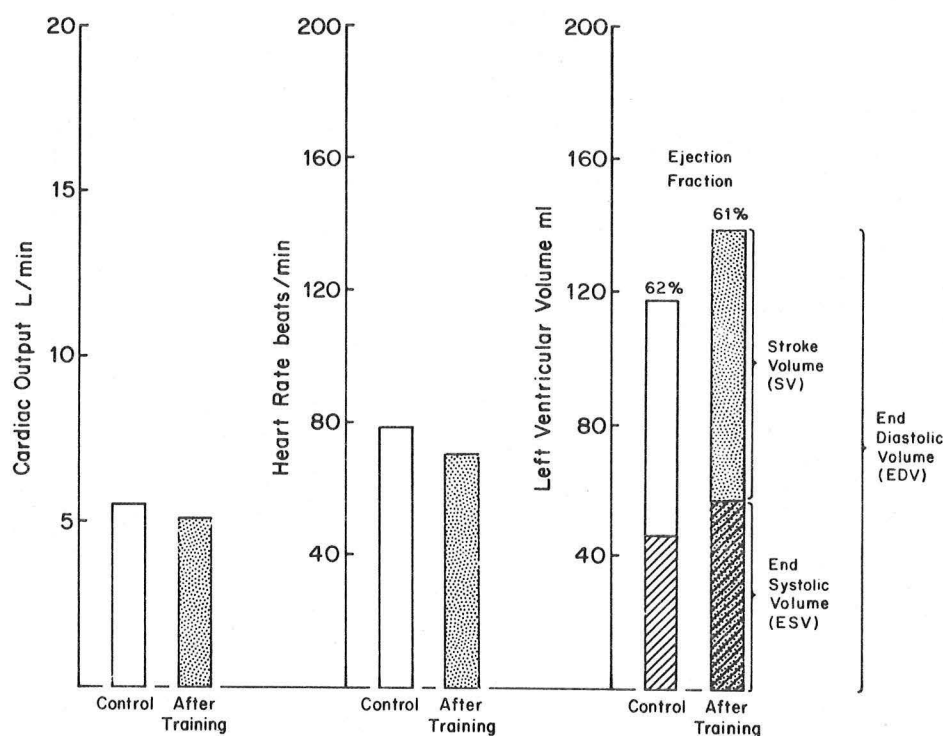
Figure 17



The studies were performed on a treadmill utilizing the Bruce protocol. After training the double product was significantly lower at the same submaximal workload because of a slower heart rate and a lower systolic blood pressure. As a consequence the extent of ST segment depression was also less at the same submaximal workload after training.

Recently, Wallace and his group in the DUPAC program have studied patients with coronary heart disease using nuclear cardiology techniques before and after a dynamic exercise training program. The resting studies before and after the training program are shown in Figure 18.

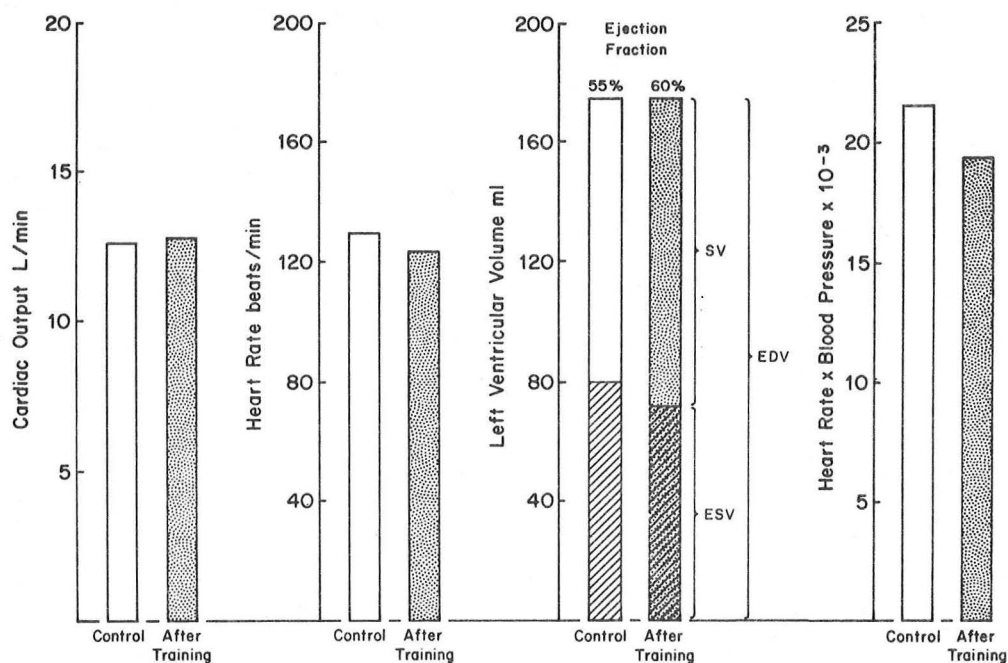
Figure 18



After training there was no change in resting cardiac output but it was achieved with a lower heart rate and higher stroke volume. In addition, the end-diastolic volume had increased and the ejection fraction remained the same at rest.

At the same workload before and after training, the cardiac output also reached the same level (Fig. 19).

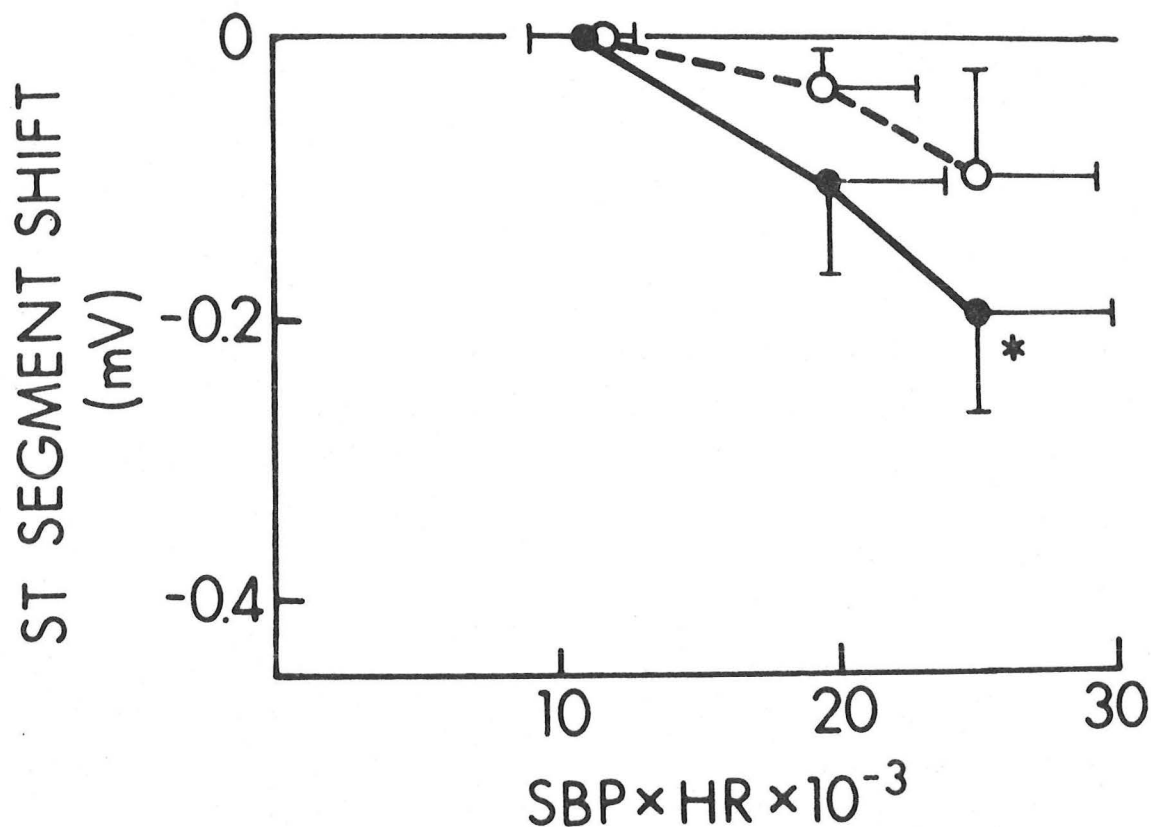
Figure 19



At the same cardiac output, the heart rate was slower and the stroke volume was higher. In addition, the end-systolic volume was decreased and the ejection fraction was higher. Also, there was no change in the end-diastolic volume and a decrease in the double product at the same workload. Thus, when the patient was exercising at the same workload and presumably total body oxygen consumption, the left ventricle did not demand as much oxygen as indicated by the lower double product at the same end-diastolic volume.

Eshani et al. also examined the relationship between the extent of ischemic ST segment depression and the double product at various workloads during the progressive exercise test, and this data is shown in Figure 20.

Figure 20

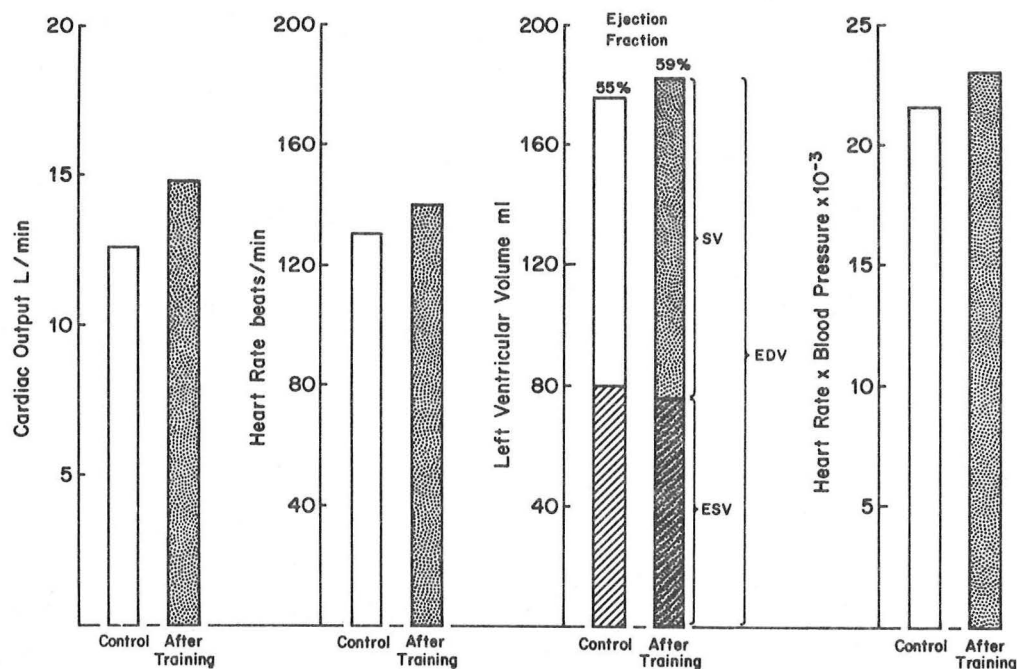


The ST segment displacement at the same double product was less after dynamic exercise training. This suggests an improvement in myocardial blood flow. However, without left ventricular volume measurements it is difficult to make this interpretation.



Wallace and his group also demonstrated that after the patients were trained, they could achieve a higher workload and a higher cardiac output (Fig. 21).

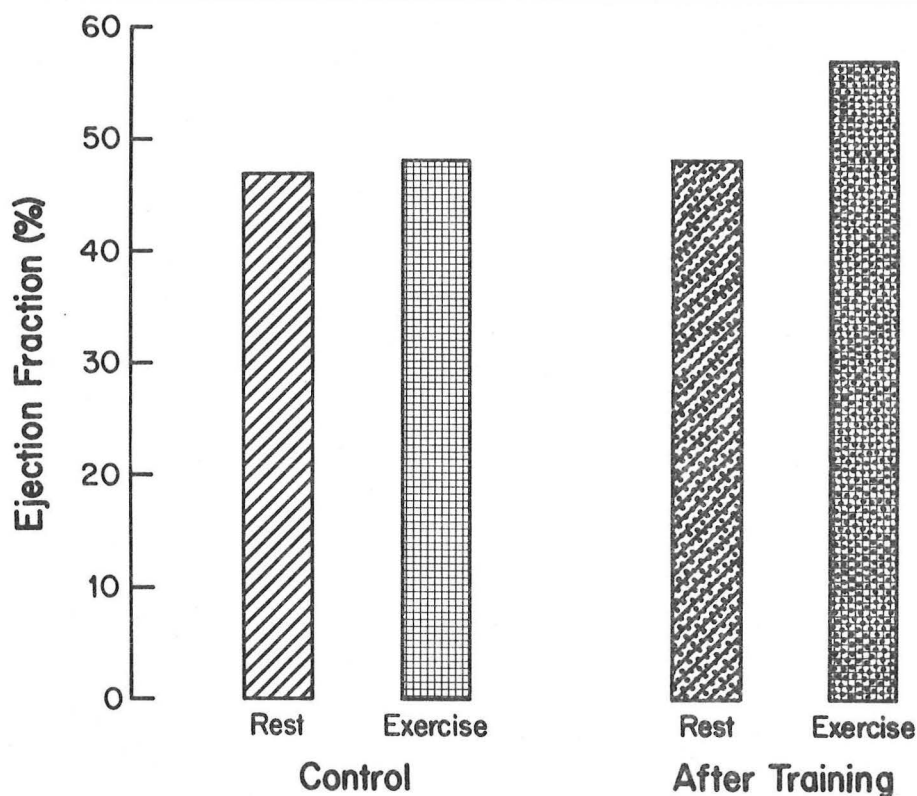
Figure 21



The increased maximal cardiac output was due both to an increased heart rate and to a higher stroke volume after training. This finding is contrary to the work of Detry et al. who found that after training the increased maximal oxygen uptake was entirely due to an increased widening of the arteriovenous oxygen difference. Also after dynamic exercise training the patients achieved a higher double product from a larger end-diastolic volume. At this increased level of myocardial oxygen demand, there was no change in left ventricular performance as indicated by the ejection fraction. This finding could be interpreted as showing that myocardial oxygen supply had been improved by the exercise training program.

Ehsani et al. have also studied the ejection fraction before and after their intensive training program for one year in patients with coronary heart disease (Fig. 22).

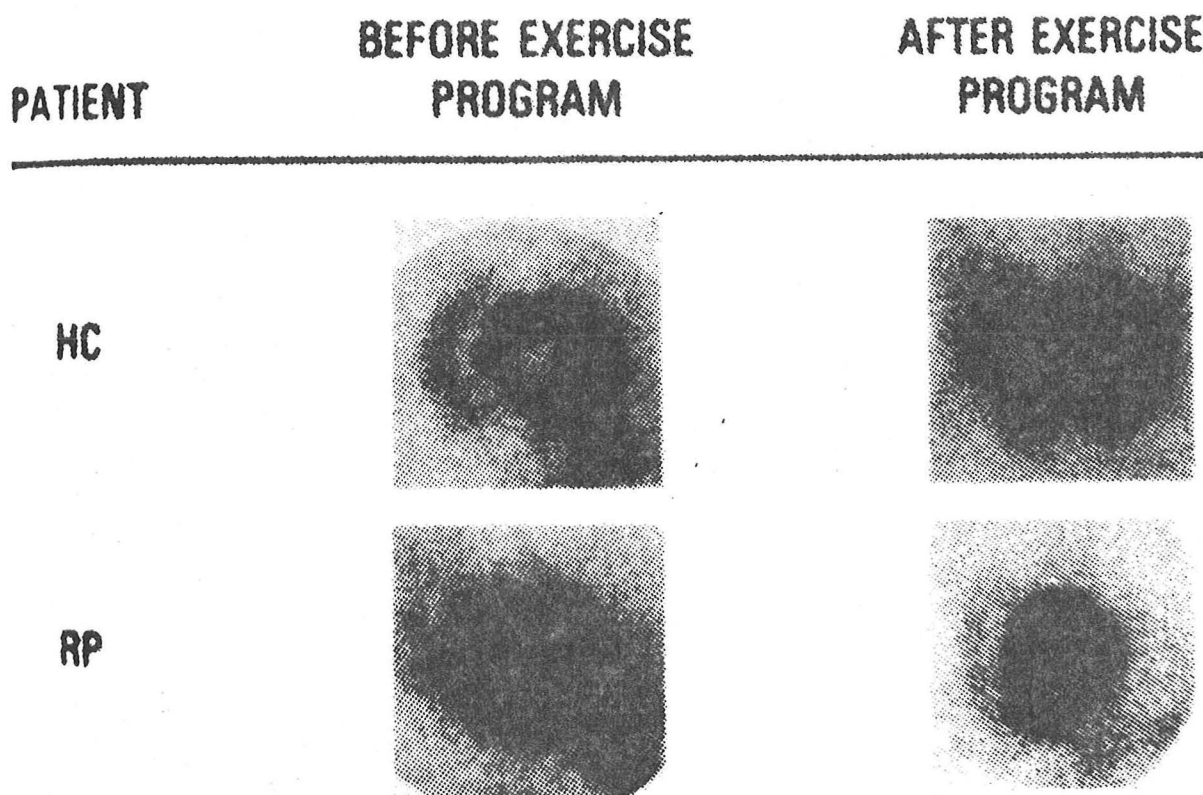
Figure 22



Before training the ejection fraction at rest was 47% and did not increase with exercise. After training the resting ejection fraction was the same at rest as before training but increased with exercise. In addition, the increased ejection fraction was from a higher double product. This study could also be interpreted as showing an improvement in myocardial oxygen supply.

No training studies have demonstrated by coronary arteriography an anatomic improvement in coronary atherosclerosis or an increase in coronary collaterals. However, it is possible that such changes are too subtle to be seen by the present methods used in man. Froelicher and his group have been conducting dynamic exercise training studies in their cardiac rehabilitation program in San Diego. Changes in left ventricular performance are being correlated with changes in exercise Thallium-201 scans. The scans of two patients before and after the exercise training program are shown in Figure 23.

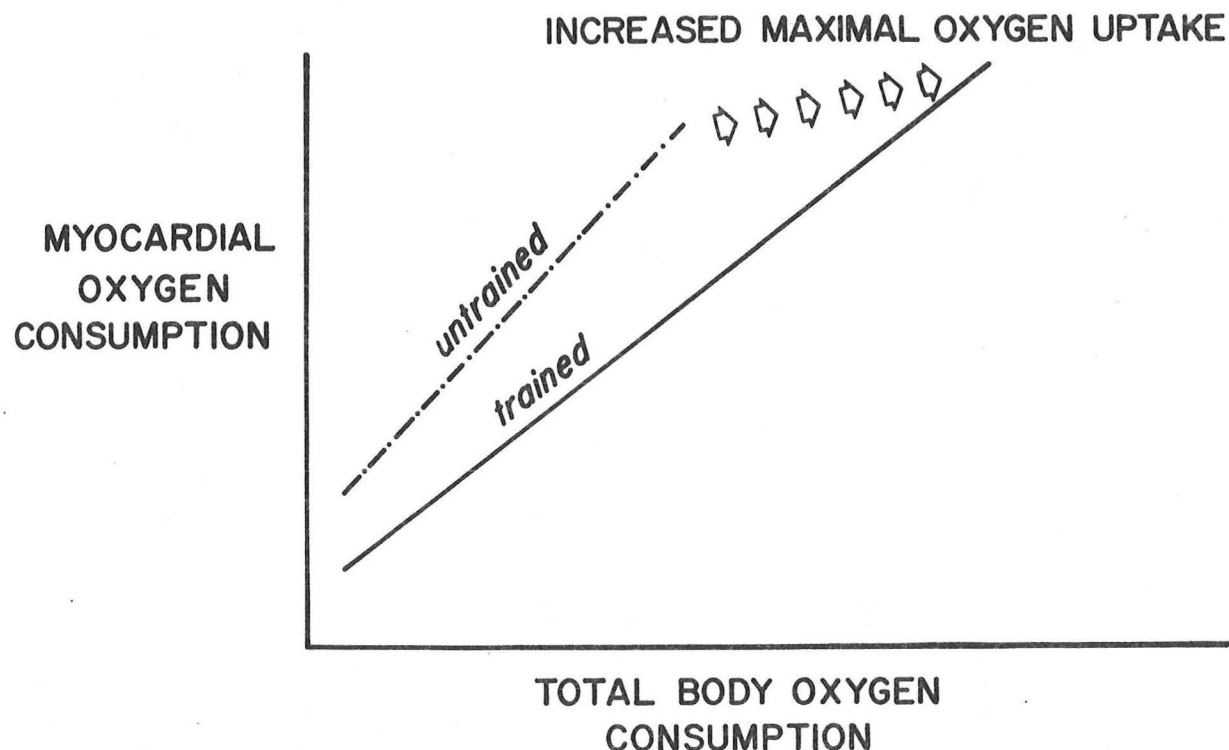
Figure 23



Patient HC was able to reach a higher maximal double product and had an "improved" Thallium scan after exercise training. Patient RP could not reach as high a double product and had a "worsened" Thallium scan. These studies are quite preliminary, and further quantitation of the Thallium-201 scan method is needed. As nuclear methods for studying myocardial blood flow become more quantitated, it is hoped that an answer will be obtained as to whether dynamic exercise training in patients with coronary heart disease can truly increase myocardial oxygen supply during heavy exercise. Several studies in animals have shown an improvement in coronary vascularity and flow after a dynamic exercise training program.

The effect of training on the relation between myocardial oxygen consumption and total body oxygen consumption is summarized in Figure 24.

Figure 24



After a training program the curve relating myocardial oxygen consumption to total body oxygen consumption moves to the right. Thus, the myocardial oxygen demand after training is reduced for any given total body oxygen demand. This can explain why the patient who has angina at a given workload before training does not have angina at the same workload after training. His coronary blood flow may not be improved but rather his heart requires less oxygen at the same exercise load. The portrayal of the curve going higher after training as well as rightward was done deliberately, although there has been no proof that total myocardial oxygen supply can be improved in normal individuals or in patients. However, it is difficult to explain otherwise the finding that a patient can achieve a higher double product from an even higher end-diastolic volume before there is a discrepancy between oxygen supply and demand.

D. Psychological Effects

7. Loc. cit.
10. Loc. cit.
74. McPherson, B.D., Paivio, A., Yuhasz, M.S., Rechnitzer, P.A., Pickard, H.A., Lefcoe, N.M.: Psychological effects of an exercise program for post-infarct and normal adult men. *J Sports Med* 7:95, 1967.
75. Bruhn, J.G.: Obtaining and interpreting psychosocial data in studies of coronary heart disease. In: Exercise Testing and Exercise Training in Coronary Heart Disease, edited by J.P. Naughton, H.K. Hellerstein. New York: Academic Press, 1973, p. 263.
76. Fisher, S.: Unmet needs in psychological evaluation of intervention programs. Ibid., p. 289.
77. Kavanagh, T., Shephard, R.J., Tuck, J.A., Qureshi, S.: Depression following myocardial infarction: the effects of distance running. *Annals N.Y. Academy Sciences* 301:1029, 1977.
78. Hackett, T.P., Cassem, N.H.: Psychological factors related to exercise. In: Exercise and the Heart, edited by N.K. Wenger. *Cardiovas Clinics* 9:223, 1978.
79. Hackett, T.P., Cassem, N.H.: Psychologic aspects of rehabilitation after myocardial infarction. In: Rehabilitation of the Coronary Patient, edited by N.K. Wenger, H.K. Hellerstein. New York: John Wiley & Sons, 1978, p. 243.
80. Acker, J.E., Jr.: Psychological aspects of cardiac rehabilitation. In: Advances in Cardiology, Vol. 24: Cardiac Rehabilitation, edited by K. Konig, H. Denolin. Basel: S. Karger, 1978, p. 116.
81. Hackett, T.P.: The use of groups in the rehabilitation of the post-coronary patient. In: Advances in Cardiology, Vol. 24: Cardiac Rehabilitation, edited by K. Konig, H. Denolin. Basel: S. Karger, 1978, p. 127.
82. Carr, D.B., Bullen, B.A., Skrinar, G.S., Arnold, M.A., Rosenblatt, M., Beitins, I.Z., Martin, J.B., McArthur, J.W.: Physical conditioning facilitates the exercise-induced secretion of beta-endorphin and beta-lipotropin in women. *N Engl J Med* 305:560, 1981.
83. Stern, M.J., Cleary, P.: National exercise and heart disease project. Psychosocial changes observed during a low-level exercise program. *Arch Intern Med* 141:1463, 1981.
84. Hackett, T.P.: Personal Communication.

The two principal psychological problems that affect patients with coronary heart disease are depression and anxiety. These conditions can be extremely debilitating and need to be corrected.

Hackett states that depression should be diagnosed in a patient after acute myocardial infarction who has five or more of the following indications:

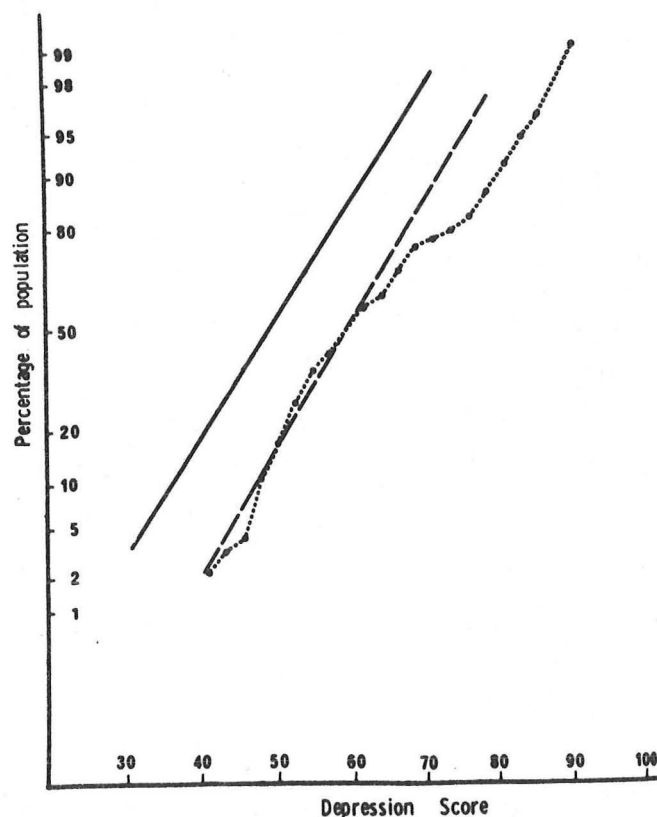
#### INDICATIONS OF DEPRESSION

1. Sleep Disturbance (esp. early morning awakening)
2. Changes in Appetite (resulting in marked weight loss or weight gain)
3. Marked Fatigue (esp. in morning)
4. Vegetation (sits all the time)
5. Loss of Interest in Activities (including sex)
6. Decreased Ability to Concentrate
7. Feelings of Guilt
8. Recurrent Thoughts of Death and Suicide
9. Irritability
10. Anxiety
11. Somatic Symptoms (esp. headache, palpitations, vague chest pain)
12. Poor Memory

Using this criteria, Hackett finds that almost 100% of patients are depressed for three to five months after an acute myocardial infarction, and about 15% remain depressed after that time.

Kavanagh et al. have also studied depression in patients after a myocardial infarction, and their data are shown in Figure 25.

Figure 25



The data are plotted to demonstrate normality in the distribution of D scores (Depression) on the Minnesota Multiphasic Personality Inventory (MMPI). The plot of D scores for the healthy population is shown by the solid line. The estimate for a non-depressed post-infarction patient population is shown by the broken line. The plot for the post-myocardial patients is shown by the dotted line. This graph suggests that the entire post-infarction population may have been somewhat depressed.

Drug therapy has not proven to be very effective treatment for depression in patients after a myocardial infarction. The best results have been obtained with dynamic exercise training and with group therapy. A comprehensive cardiac rehabilitation program provides both of these interventions.

The majority of patients with coronary heart disease do not seem to be very concerned over their longevity. Their principal concerns seem to be the quality of their remaining years and the way they feel about their present state. Patients have reported that they "feel better" after dynamic exercise training. Unfortunately, changes of feeling are hard to quantitate by formal psychometric tests. Such studies, however, have suggested an improvement in depression and possibly in anxiety. "Feeling better" could be due to the

recently reported increased levels of plasma endorphins that are found after acute dynamic exercise and after acute dynamic exercise training.

The psychological effects of cardiac rehabilitation in patients with coronary heart disease may be the most important clinical benefit. This improvement in mood ("feeling better") may be in itself a sufficient justification for patients to take part in such a program.

#### E. Sociological Effects

10. Loc. cit.

85. Croog, S.H.: Social aspects of rehabilitation after myocardial infarction: a selective review. In: Rehabilitation of the Coronary Patient, edited by N.K. Wenger, H.K. Hellerstein. New York: John Wiley & Sons, 1978, p. 255.
86. Gentry, W.D.: Psychosocial concerns and benefits in cardiac rehabilitation. In: Heart Disease and Rehabilitation, edited by M.L. Pollock, D.H. Schmidt. Boston: Houghton Mifflin, 1979, p. 690.
87. Oberman, A.: Endpoint considerations. In: Physical Conditioning and Cardiovascular Rehabilitation, edited by L.S. Cohen, M.B. Mock, I. Ringqvist. New York: John Wiley & Sons, 1981, p. 289.
88. Hellerstein, H.K., Friedman, E.H.: Sexual activity and the post-coronary patient. Arch Intern Med 125:987, 1970.
89. Skinner, J.S.: Sexual relations and the cardiac patient. In: Heart Disease and Rehabilitation, edited by M.L. Pollock, D.H. Schmidt. Boston: Houghton Mifflin, 1979, p. 587.
90. Wenger, N.K., Hurst, J.W.: Coronary bypass surgery as a rehabilitative procedure. Cardiac Rehabilitation 11:1, 1980.

A critical evaluation of the sociological benefits of cardiac rehabilitation in patients with coronary heart disease is extremely difficult. Endpoints of sociological beneficial effects would include an evaluation of the rate and extent of return to pre-illness lifestyle and to work. Multiple, unmeasurable factors are complexly interrelated to determine the outcome in these two areas.

It seems likely that the educational component and the cardiovascular physiological effects of exercise training would facilitate the return to normal sexual activity. The occurrence of a lower double product at the same submaximal workload would seem to be of benefit during intercourse.

The resumption of gainful employment is dependent on multiple factors. These factors have recently been described in a study which evaluated coronary bypass surgery as a rehabilitative procedure. It would seem that symptomatic and functional improvement would be of importance; however, they are both non-predictive factors. Predictive factors which appear to impede the return to work include increased age, decreased education and level of social class,



depression, availability of disability compensation, pension benefits, and employer liability. With so many uncontrolled factors involved, it is impossible to determine the effect of cardiac rehabilitation on the return to work.

### III. CONCLUSIONS

A comprehensive program of cardiac rehabilitation in patients with coronary heart disease should include risk factor modification, dynamic exercise training, and appropriate psychological and sociological support. Studies of the effect of risk factor modifications suggest that such therapy can reduce mortality and morbidity in patients with coronary heart disease. However, control studies of the effect of exercise training, per se, have not demonstrated a statistically significant decrease in these endpoints of the progression of coronary heart disease.

Dynamic exercise training may potentiate the improvement in risk factors and in addition cause beneficial cardiovascular physiological effects. These include an increase in maximal exercise performance (maximal oxygen uptake), a decrease in heart rate at rest, and a decrease in heart rate and systolic blood pressure (double product) at a constant submaximal workload. Thus there is a decreased myocardial oxygen demand at a given level of total body oxygen consumption and this is of clinical benefit to the patient. In addition the demonstration of the achievement of a higher oxygen demand during exercise with no change or an improvement in left ventricular function suggests an increase in myocardial oxygen supply. More definitive studies are needed to determine if myocardial oxygen supply can be increased in patients with coronary heart disease.

Additional beneficial effects of cardiac rehabilitation include the patients "feeling better" and an improvement in depression and anxiety. Since patients "feel better" and have a better quality of life, cardiac rehabilitation should be considered as one of the treatments for coronary heart disease which is complementary to drug therapy and to coronary artery bypass surgery.