CARDIOVASCULAR AND SERUM LIPID CHANGES OF POST-MYOCARDIAL INFARCTION PATIENTS INDUCED BY EXERCISE REHABILITATION PROGRAMS

by

Jack Edward Taunton
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NAME: Jack Edward Taunton

DEGREE: Master of Science (Kinesiology)

TITLE OF THESIS: Cardiovascular and serum lipid changes of post-myocardial infarction patients induced by exercise rehabilitation programs.

Department Chairman Dr. E. W. Banister

Chairman of Graduate Studies Dr. W. D. Ross

Dr. E. W. Banister
Senior Supervisor

Dr. W. G. Trapp
External Examiner
Associate Professor, Department of Surgery
University of British Columbia

Dr. L. M. Lawton
External Examiner
General Practitioner, Health Services
Simon Fraser University

Date Approved: 7 April 1971.

ABSTRACT

This study has compared the effectiveness of three types of exercise programs in the recovery of physical work capacity of fourteen post myocardial infarction patients (age 30-54 years) who were an average of five months (range three to seven months) post-infarction. The three types of exercise programs included two different types of bicycle ergometry programs, one consisting of interval training and the other of continuous effort, both of which were done five days per week for thirty minutes. The third program type was a calisthenic-walk-jog program carried out for three days per week for fifty minutes. The two bicycle ergometry programs were controlled by continuous ECG monitoring of the patients throughout exercise consisting of an initial five minute warm-up period, thirty minute exercise and a five minute recovery period whereas the calisthenic-walk-jog program was controlled by manual "heart rate monitoring" of the patients and if warranted by a telemetric recording of exercise heart rates. The training rate for continuous ergometry was set at 50 per cent of the estimated current maximal physical working capacity (PWC_{190} est) of the patient carried on for a one-half hour period. Interval training consisted of three series of work consisting of four minutes at each of 40 per cent of the PWC_{190} est and 60 per cent of the PWC_{190} est separated by a rest period of two minutes unloaded cycling. The training in the walk-jog program was carried out at a heart rate of 135-145 b/min during the jogging periods. This dropped below 100 b/min in the walking phases.

Evaluation tests were made on each patient, serially, every two weeks over a period of twenty-four weeks and on the basis of these tests each

patient's exercise therapy was upgraded accordingly. The initial status of each patient was established by response to three separate evaluation tests. During the re-evaluation tests, measurements were made at rest, during a controlled five minute warm-up (no load 50 rpm) and during the course of three progressively increasing six minute work sessions separated by five minute free-pedalling pauses. The parameters measured at rest included pulmonary function (tidal volume, respiratory rate, vital capacity and FEV_1), weight, serum lipids, blood pressure, hemotology (RBC, WBC, hematocrit, mean cell volume and hemoglobin) and lactate and acid-base balance. During the controlled warm-up period cardiac output, stroke volume, $\dot{V}O_2$ and $\dot{V}CO_2$ and acid-base balance were measured. During the three exercise sessions $\dot{V}O_2$, $\dot{V}CO_2$ and PWC_{170} and PWC_{190} were measured. Oxygen uptake and carbon dioxide elimination were measured continuously by rapid response meters. Cardiac output, stroke volume, blood pressure, lactate and acid-base balance were measured at the end of the third exercise session. In all conditions cardiac output was measured by a CO_2 rebreathing technique. Derived data consisted of myocardial oxygen demand, ventilation equivalent and oxygen pulse.

It was established that while all forms of training produced significant improvement, continuous ergometry was more effective than either of the other methods. The calisthenic-walk-jog program was least effective. Significant improvement was observed in work capacity (PWC_{170} and PWC_{190} est), decreased cholesterol levels, with no concomitant weight change, increased vital capacity and FEV_1, decreased resting heart rate, diastolic blood pressure, myocardial oxygen demand and lactate levels, decreased ventilation, heart rate, systolic blood pressure and myocardial oxygen demand, $\dot{V}O_2$, $\dot{V}CO_2$, R and ventilatory equivalent for standard work rates, increased oxygen pulse

for standard work rates, decreased cardiac output in submaximal work and lower levels of metabolic acidosis for equivalent work. As a result of the training, freedom from arrhythmias, angina and dependence on drug therapy was also observed. An overall well controlled therapeutic scheme for progression of the post-infarction patient from the Intensive Care Unit back to physical vigor is discussed.

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CHAPTER I

INTRODUCTION

This thesis will analyse serum lipid, cardiovascular and related changes observed in post myocardial infarct patients participating in three extensive exercise programs. Exercise therapy is often advised in the rehabilitation of coronary patients, although it has not been established what are, respectively: the optimal time of commencement of exercise after the initial hospitalization, the intensity, the amount or type of exercise needed, and the extent of the physiological changes which occur in the rehabilitative period. In the initial stages of recuperation the coronary heart disease patient will possess a low maximal oxygen uptake and a poor tolerance of effort, as might well be expected considering the dominant role of the heart in the oxygen transport system. The following remarks of Varnauskas (1967), supported by Karvonen at the Helsinki Symposium on Physical Activity and the Heart (1966), are pertinent:

> "It is unclear what type of circulatory adjustment is to be expected in patients with cardiac disorders. Will physical training cause an increase in cardiac output and/or stroke volume or be accompanied by more effective redistribution of blood flow in individuals with coronary disease. Cardiovascular centers should be stimulated and supported in studies of training effects on small groups of healthy individuals and in patients with cardiac disorders."

The rehabilitation of coronary patients is virtually a world wide concern as coronary heart disease (CHD) has now reached epidemic proportions. In North America, especially, figures on the incidence of CHD are staggering. The rehabilitation of these patients is relatively new.

Incidence, Mortality and Cost

In 1969 the National Heart Institute (NHI) of the United States illustrated the magnitude of the problem. Data compiled between 1960 and 1962 by the National Health Survey (NHI) indicated that 14,621,000 Americans, age 18-79, have definite heart disease and an additional 12,979,000 have "suspect heart disease". Thus, approximately 25 per cent of American adults have either definite or suspect heart disease. Armstrong (1965) reported that the prevalence of cardiovascular disease in Canada was about 2.5 million in a total population of 20 million.

Cardiovascular disease, in the United States accounted for 54 per cent, or 1,002,000, of all deaths in 1967. Atherosclerotic heart disease accounted for 57 per cent (573,153) of these deaths, strokes 20 per cent (202,184) and hypertension 6 per cent (61,126). Two hundred and fifty thousand, nine hundred and seventy-seven (250,977) premature cardiac deaths occurred during the productive years under 65 (see Figure 1). Haskell (1968) reported that the annual incidence of "heart attacks" in the United States was currently about 700,000, and over 573,000 coronary patients die each year.

The 1969 Canada Yearbook lists the most recently available mortality figures in Canada, among which arteriosclerotic and degenerative heart disease are the leading causes of death since 1931 (see graph Figure 2). In 1966 they accounted for 48,290 deaths out of a total number of deaths of 149,863 (32.2 per cent). Deaths caused by all forms of heart disease accounted for 35.3 per cent of all deaths. This is a rate of 241.3 per 100,000 population. Cardiovascular diseases killed 11,654 men aged forty-five to sixty-four years in 1966 which is a rate of 653 per 100,000 population.

The British Columbia Department of Health Services and Hospital

FIGURE 1. U. S. Coronary heart disease rate per 100,000 deaths - all men under 65 years.

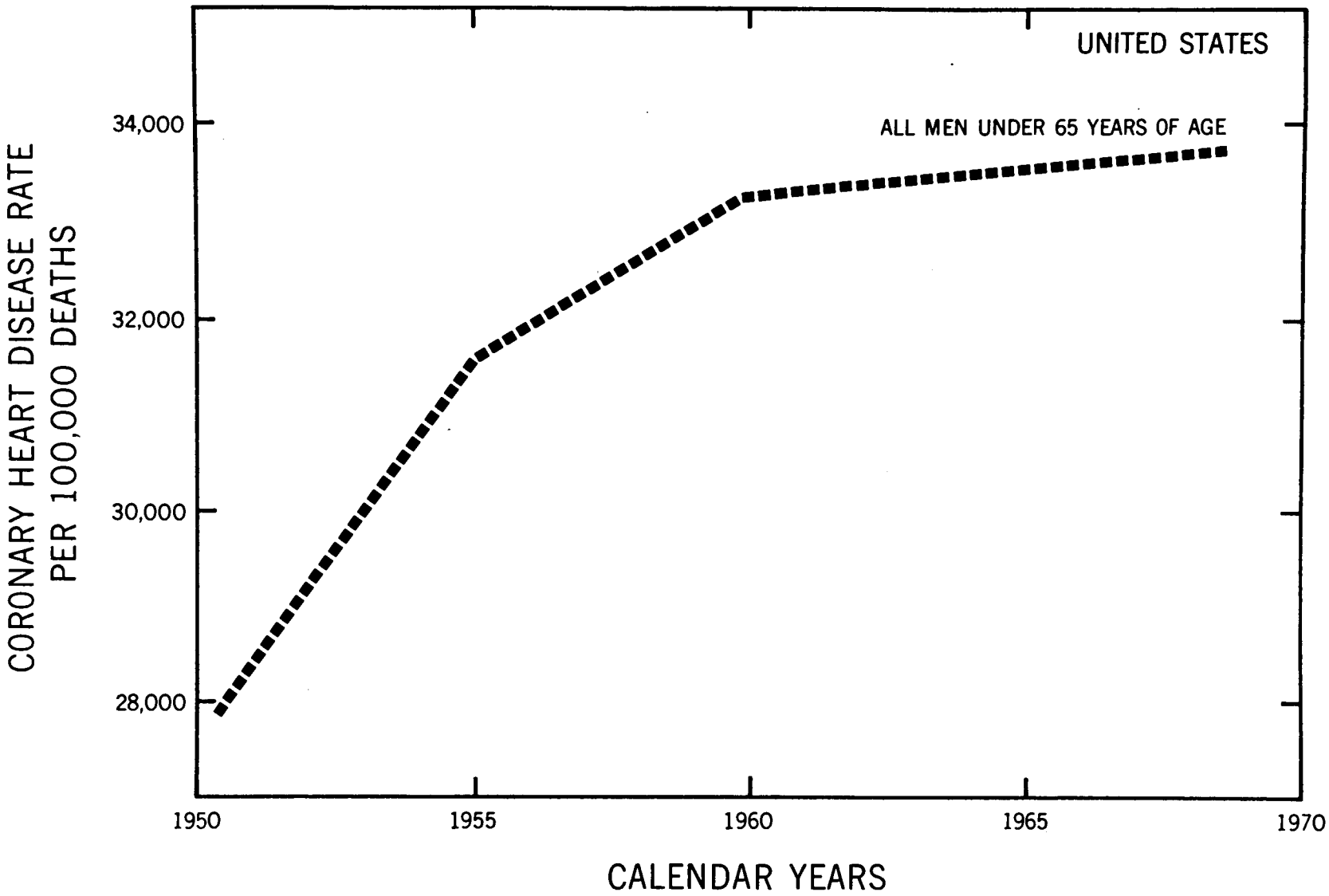
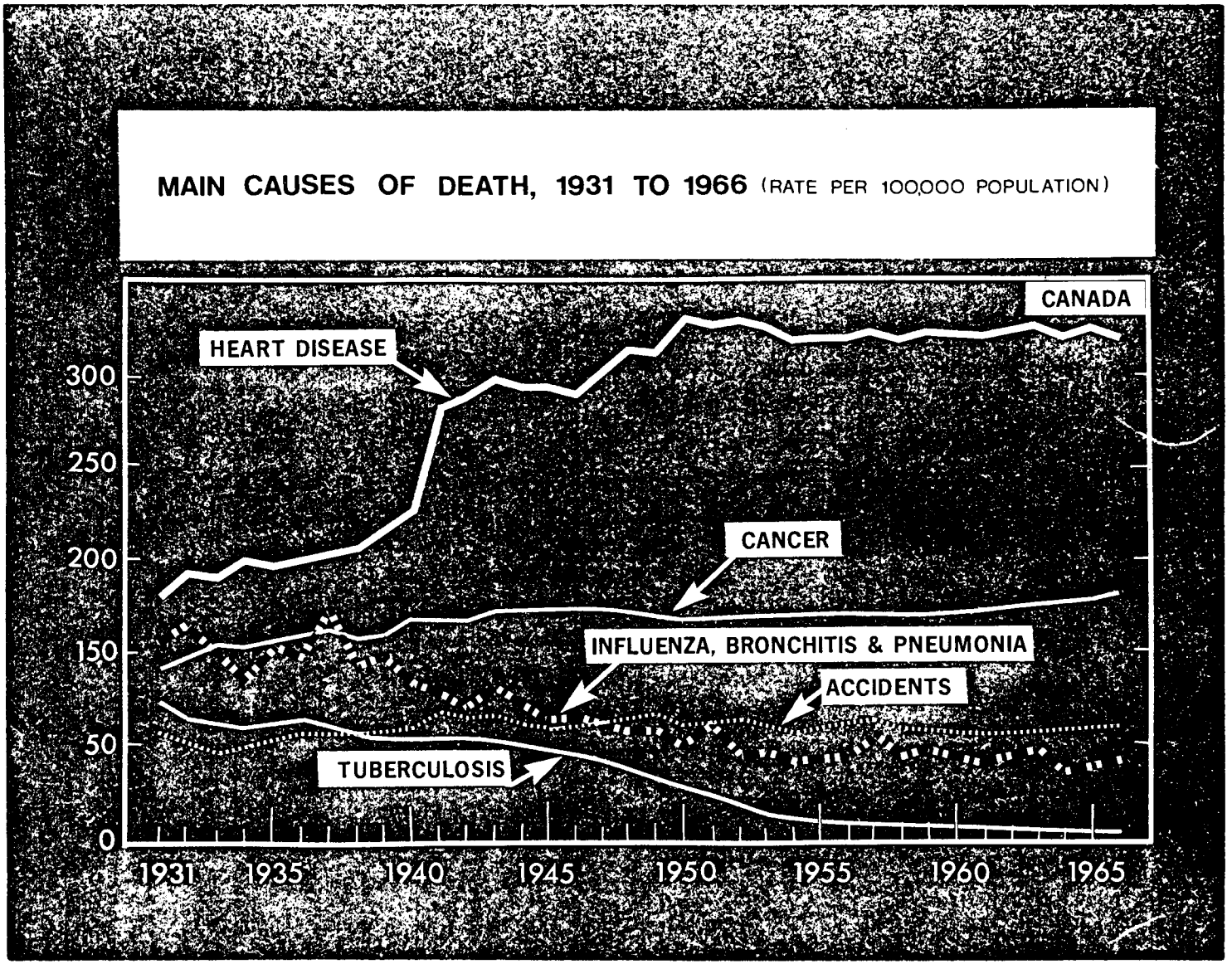

UNITED STATES
ALL MEN UNDER 65 YEARS OF AGE
CORONARY HEART DISEASE RATE
PER 100,000 DEATHS
34,000
32,000
30,000
28,000
1950
1955
1960
1965
1970
CALENDAR YEARS

FIGURE 2. Main causes of death, 1931-1966, Canada.


MAIN CAUSES OF DEATH, 1931 TO 1966 (RATE PER 100,000 POPULATION)
CANADA
HEART DISEASE
CANCER
INFLUENZA, BRONCHITIS & PNEUMONIA
ACCIDENTS
TUBERCULOSIS
300
250
200
150
50
0
1931
1935
1940
1945
1950
1955
1960
1965

Insurance (1968) list diseases of the heart as the major cause of death in B.C. from 1957 to 1966. In 1957 they accounted for 4,913 deaths (37.3 per cent) and in 1966 accounted for 5,927 deaths (37.6 per cent). In the male productive years, forty to forty-nine, disease of the heart caused mortality at a rate of 498.8 per 100,000 population.

The 1969 report of the NHI, using data compiled in earlier years, stated that the annual economic toll exacted by the cardiovascular diseases was estimated to exceed 25 billion dollars. Klarman (1964) estimated the total cost to be 30.7 billion dollars. The total economic cost may be divided into direct and indirect costs. The direct costs, totalling 3.07 billion dollars, included expenditures for personal services and supplies -- hospital care, services of physicians and provision of drugs (2.58 billion dollars) and non-personal items such as research, training, public health services and insurance schemes (0.49 billion dollars). The NHI report stated that indirect costs totalled 22.9 billion dollars, whereas, Klarman (1964) estimated this latter expenditure to be closer to 27.6 billion dollars. The indirect costs included losses of production due to illness, disability, premature death and grief. The total Klarman cost of 30.7 billion dollars divided among a population of 200 million amounted to an annual charge of 154 dollars per capita or about 300 dollars per wage earner. Over a working life of 50 years cardiovascular diseases cost each American worker 15,000 dollars (Shephard, 1969).

Shephard (1969), using estimates of Armstrong (1965), calculated the total annual cost to the Canadian economy at 1.7 billion dollars. This is about 86 dollars per capita or 170 dollars per wage earner.

Helander (1970) reported that in Sweden the loss to the gross national production due to cardiovascular and cerebrovascular diseases, resulting in

90 day absence from work, exceeded 125 million dollars in the year of 1970.

The long term prognosis of patients, comprising the NHI report, who had survived a myocardial infarction is presently much less favorable than that of the general population. The survey indicated that the average annual mortality rate for patients surviving a first myocardial infarction by at least two months ranged from twelve times the normal rate in the 30-39 age group to 2.5 times the normal rate in the 60-69 age group. The cumulative five year mortality rate among recovered myocardial infarction patients is 30 per cent compared to only 7 per cent for normal subjects. It is against this very high mortality rate that it is hoped exercise therapy might prove effective.

Use of Exercise Therapy

Wyndham (1967) stated that, before 1957, the cardiac patient was essentially someone whom the medical practitioner saw in bed. And he (the practitioner) was not particularly interested in the rehabilitation of this type of patient. A revolution in medical thinking was started at the Wisconsin Conference on Work and the Heart, 1959. Various studies resulted from this conference, however, to date, satisfactory exercise rehabilitation programs have not been created and only superficial results have been obtained from the few published studies.

Although Naughton et al. (1966), among others, presented evidence supporting the idea that superior recovery from myocardial infarction occurs with physical conditioning programs many physicians still restrict, indefinitely, the physical activity of their post infarction patients. In fact, Shanoff, as recently as 1965, stated that "on this continent (North America) physical training and rehabilitation have not yet found acceptance

as integral parts of the management of the coronary patient as it has in Europe." The case for the protective role of physical activity rests on retrospective studies by Morris et al. (1953) and Fox and Skinner (1964) which demonstrated the different incidence of coronary disease and myocardial infarction between sedentary workers (bus drivers, clerks, white-collar workers) and active workers (bus conductor, switchmen, blue-collar workers). Astrand (1967) stated that in those studies which have been reported, the risk of a coronary heart attack is two to three times greater for the inactive than for the active individual and the chance of surviving the first heart attack is also two to three times better in those who are active.

Appropriate Exercise Training for Cardiacs

Roskamm (1967) recently discussed the optimum patterns of exercise for adults in terms of the type of training. He concluded that training in weight lifting and gymnastics had no significant training on the circulatory system, and, for this reason these exercises should not be used either in the prevention or rehabilitation of CHD. Lind (1970) investigated the cardiovascular responses to isometric exercises and concluded that all forms of static exercises are undesirable in patients with coronary artery disease or with borderline hemodynamic function. There is now general agreement (Enselberg, 1970) that endurance-type activities (walking, running, jogging and cycling) are superior to strength-type activities (isometrics and weight-lifting) in rehabilitation programs. Enselberg suggested that wherever feasible exercise testing should be done repeatedly during the succeeding stages of training. "Leg exercises carried out in the erect position are the best for this purpose, hence the popularity of steps, bicycle ergometers and treadmills .... Bicycle ergometers and treadmills are in use not only

for exercise testing, but also for providing easily quantitated training methods." M. Bekes (1968) of the Hungarian Institute of Cardiology, Budapest, at the European Society of Cardiology Symposium, supported the use of the bicycle ergometer in the following statement:

> "As far as vocational rehabilitation of cardiac patients is concerned, ergometry proves to be of great help to objectivate work capacity as well as the influence of training programs. This is why the State Hospital of Balatonfured, a large cardiological rehabilitation center, is now introducing bicycle ergometry on a big scale in connection with already well established swimming programs .... standardization of techniques is absolutely necessary."

The intensity of training done during a rehabilitation session should be based upon physiological responses to exercises during initial and subsequent tests. Denolin, Messin and Degre (1967), among others, extensively studied the problem of testing the work capacity of cardiac patients. They were looking for a useful clinical test that was simple, safe, reproducible and had a sound physiological basis. They selected a test based on the publications of Sjostrand (1947) and Wahlund (1948), that yielded the physical working capacity at a heart rate of 170 (PWC_{170}). The PWC_{170} meets both clinical and physiological criteria. The use of heart rate as the criterion of circulatory adaptation during work may appear to be an oversimplification, however, Denolin, Messin and Degre (1967) state, "heart rate represents the most sensitive parameter of cardiovascular nonadaptation to exercise, whatever the cause of the nonadaptation may be, such as insufficient cardiac output, or circulatory and/or metabolic disturbances in the working muscles."

The intensity of training is usually selected to achieve a heart rate between 135 and 150 beats/min, if this can safely be achieved. Roskamm (1967) has shown that a sustained training heart rate of 150 can significantly increase work performance and cardiovascular parameters in healthy middle-

age males. The critical level to yield significant improvements approximately corresponded to resting heart rate plus 60 per cent of the difference between resting and maximal heart rate or as suggested above about 140 beats/min in the age group 35 to 50. Wyndham (1967) stated that cardiac patients should be rehabilitated at about 40-50 per cent of maximum working capacity. This level has been successful in the cardiac training programs to date (Enselberg, 1970). The optimum duration of each session, type of training (interval or continuous) and frequency per week, of training regimes have only been explored in healthy individuals (Shephard, 1968b), not determined for cardiac patients. The safety feature of course is of the utmost importance when training cardiac patients. Generally, it appears that exercise five times per week is best, three times per week is adequate while twice a week is an absolute minimum for the average healthy individual (Kasch and Boyer, 1969; Roskamm, 1967). Less than twice weekly can often be deleterious and dangerous. Shephard (1968a) stated that five thirty minute sessions per week should be recommended for the treatment of cardiac or sedentary patients to develop fitness and three sessions per week for maintenance. Roskamm (1967), among others, found significant changes with half an hour of training five times per week. Some training effect was found by Hollman (1965) in ten persons who trained by stationary running for only ten minutes per day for three months. In further studies by Roskamm on the type of training, he concluded that training by means of an

> "uninterrupted work load is most effective in decreasing the heart rate at rest and in the low and middle intensity exercise encountered in daily life. However, interval training is equally effective or even more effective in improving the maximum working performance .... As a practical conclusion, we recommend that persons with vaso-regulatory disturbances train with uninterrupted work loads. Persons with normal circulation and a low resting heart rate can also do interval training."

Commencement of Exercise Therapy

A critical problem to be solved, before engaging in an exercise program for cardiac patients, is when to start. Enselberg (1970) stated that after discharge from the hospital the patient should be kept at a low level of activity with very slow progression for at least six weeks from the onset "since it is usually felt that this is the minimum time for significant healing of the infarct." Shephard (1968a), on the other hand, advised that six months following an acute episode "is probably a good time to start upon a serious programme of rehabilitation; if exercise is undertaken earlier, repair of the infarct may be incomplete, and if it is further delayed, the enthusiasm for exercise generated by the "critical incident" may be lost." Wyndham (1967) suggested that physical and occupational rehabilitation of uncomplicated cases should begin within three months of the acute episode. Full work, within the assessed physical working capacity of the patient, should be achieved within six months. This time schedule appears to be the current practice for establishing the starting date of rehabilitation. It is generally felt that if one delays work too long, physical rehabilitation is more difficult and psychological difficulties may result, such as the individual regarding himself as a chronic invalid.

Safety of Testing and Training of Cardiac Patients

Another distinct problem confronting any group initiating an exercise program is the incidence of myocardial infarctions attributable to exercise testing or training of coronary patients. However, contrary to many earlier expectations the testing and rehabilitation of cardiac patients has proven remarkably safe. Bruce, Hornsten and Blackmon (1968) reported that myocardial infarction was an extremely rare complication of maximal exercise testing

when appropriate precautions were taken. These included selection to avoid patients with recent active myocardial or pulmonary lesions, and professional monitoring during the testing. They found with such selection and supervision, safety was assured in over 2,000 cardiac patients in Seattle and in over 2,000 men in Taiwan. In this report the authors reported one case of acute infarction following exercise testing in an apparently healthy person. In this individual there was no evidence of infarction after his exhausting effort but symptoms occurred immediately on exposure to hot water while he was taking a shower a few minutes later. Ellestad et al. (1969) recently reported their experience with maximal treadmill stress testing with normal and cardiac patients with ages ranging from 7 to 83 years. No deaths occurred in their experience of 4,028 tests. Ventricular asystole and ventricular fibrillation were not seen. Transient ventricular tachycardia, lasting less than twenty seconds and reverting spontaneously occurred nine times. To the author's knowledge no deaths or serious conditions have arisen from submaximal cardiac rehabilitation studies reported in the literature. Varnauskas (1967) reported that in submaximally testing 25,000 patients with cardiac, or suspected cardiac disease in a twelve year period, Professor Carlsten (Head, Clinical Physiological Department, University of Goteborg) encountered myocardial infarction only once and ventricular tachycardia twice.

Objectives

Recently, a European cardiologist, Frick (1969), discussed the problem of rehabilitation studies and appealed to other investigators interested in the scientific evaluation of the effects of exercise on patients with coronary disease to investigate the "mechanisms" which have brought about increased efficiency. He considered it "established" that moderate physical

conditioning improves the efficiency of a heart crippled by impaired circulation. This study was initiated to investigate some of the "mechanisms" leading to the improvement.

Specifically, the purpose of this study was to evaluate first, physiologically, the pattern of cardiovascular recovery of post cardiac infarct patients engaged in exercise programs. Secondly, to compare the results of three variations of exercise training methods to determine that which is most effective. Thirdly, to establish what are the specific variables in which the improvement is most easily seen.

The cardiovascular measures investigated were carbon dioxide output and oxygen uptake, blood pressure, heart rate and cardiac output (using the bloodless carbon dioxide rebreathing technique of Jones et al. (1967)).

The standard pulmonary function tests of vital capacity and forced expiratory volume were also performed. Associated with these variables are acid-base balance changes, lactates, hematological variables (RBC, WBC, MCV, hematocrit and Hb), serum cholesterol and triglycerides. Derived measures included respiratory exchange ratio (R), stroke volume, ventilatory equivalent, oxygen pulse, and estimated myocardial oxygen demand. In a few patients coronary angiograms were to be performed initially and after the program.

CHAPTER II

REVIEW OF LITERATURE

Rehabilitation of Cardiac Patients

Changes in cardiovascular function and work capacity

The important problem of preventing and rehabilitating coronary heart disease (CHD) and myocardial infarction, although recognized, is not solved. After an extensive review of the literature of current population activity levels and incidence of CHD, Fox and Skinner (1964) concluded that "it is an attractive hypothesis that physical activity can contribute to the prevention or modification of CHD and conceivably of all the athero-thrombotic diseases, especially since these diseases are the major health hazard of the Western World". The work by Fox and Skinner is a result of what Wyndham (1967) calls a "revolution in medical thinking on the subject of rehabilitation of the cardiac patient" which took place in the decade following 1957. The revolution started at the Wisconsin Conference on Work and the Heart. In 1964 a very important publication in this area was the World Health Organization Technical Report No. 270 entitled Rehabilitation of Patients with Cardiovascular Diseases. The aims of rehabilitation as stated in the 1964 WHO report are as follows: "The aim of rehabilitation is not only to restore the patient with cardiovascular disease to his previous capacity, but to develop his physical and mental functions to the optimum".

The beneficial effects of exercise have been well substantiated by reports on the training of normal fit and unfit children and adults (Ekblom, 1969 and Skinner et al., 1967). The World Health Organization Technical Report No. 436 (1969) presents a comprehensive discussion of

exercise training effects and optimum physical performance capacity in middle aged adults. However, as Hellerstein (1968) states "physical training has not as yet found acceptance in this country (United States) as an integral part of the management of the patient with myocardial infarct". One of the earliest reports of such an undertaking was in 1964 by Naughton and Blake. In this investigation five male, healed post myocardial infarction patients trained for a period of three to five months. The training program was daily participation in simple yet highly competitive ball games. In all instances training led to a lower pulse rate, blood pressure, minute ventilation and serum cholesterol concentration at rest. Greater cardio-respiratory efficiency at comparable levels of energy demands occurred as well as an increased work capacity. All of the changes recorded were similar to those known to occur with training in normal healthy individuals. Similar findings were reported by Holloszy et al. (1964). Naughton et al. (1966) showed an improvement in the work capacity of twelve post coronary patients who volunteered for an eight month physical conditioning program. The subjects engaged in an hour-long medically supervised exercise session three times a week, consisting of competitive game calisthenics and noncompetitive jogging. Each subject gradually increased his level of energy expenditure. As in their earlier study, they reported significant training effects in the exercising cardiacs as reflected by a significant decrease in the resting and exercise levels of systolic and diastolic blood pressure following the physical conditioning. The resting and exercising pulse rate was significantly lower after the training program. Only two of the twelve completed the initial eighteen minute treadmill walk, however, after the program, eleven of the twelve completed the test. Actual work capacities and oxygen uptakes were not measured.

Barry et al. (1966) used a bicycle ergometer to train six patients with healed myocardial infarction. The training period lasted from six to sixty-one weeks depending on the subject and in all cases started at least twelve weeks after the occurrence of the infarction. Physical work capacity was increased and the work electrocardiogram was improved in five of the six subjects, however, marked variations in the cardiovascular response to exercise was noted throughout the program. In some cases, significant improvement occurred only after thirteen months of training. However, much of the training was done at home and consistent improvement was noted during the last six weeks of the program when they trained under supervision in the hospital.

Hellerstein et al. (1967a, 1967b, 1968) reported the cardiorespiratory changes during training of 50 and later 100 coronary patients. The WL_{150} (work load necessary to reach a heart rate of 150 beats per minute) increased from 586 to 639 kpm/min and the heart rate-systolic blood pressure product (the systolic tension time index (STTI)) which has been shown to be a good estimation of myocardial oxygen uptake (Katz and Feinberg, 1958; Sarnoff et al., 1958) showed a significant decrease. Predicted maximal oxygen uptake rose significantly and the exercise systolic blood pressure decreased. Many patients (63 per cent) had arrhythmias present in the exercise electrocardiograph before training, but not after training. The cardiac patients were followed for an average of 2.7 years and during this period the mortality rate was found to be lower for the exercise patients. The mortality rate of comparable coronary subjects treated in the traditional manner ranged from 4.5 to 6.0 per hundred patient years. In this study, the rate was lowered for the physically conditioned patients to 1.95 per hundred patient years. Heller (1967) reported the results of a "physician-approved

conditioning program for patients following a myocardial infarction ... carried on for three years without specific measurements of cardiac function". Twenty-two patients were participating three to five times weekly and "all show definite clinical improvement" (decreased resting blood pressure and increased work capacity). Rechnitzer et al. (1965, 1967b) reported the effects of a 24 week exercise program (two days per week) on eight patients with previous myocardial infarction. The lapsed time post-infarction varied from six months to four years. The program resulted in a significant reduction of body fat, increase in "muscular endurance" and subjective improvement in mood. Muscular endurance was measured by the performance of a five minute endurance test that consisted of a battery of six individual test items (push-ups, sit-ups, chest raise, double leg raise, side leg raise and sitting tucks). No significant changes occurred in the pulmonary function tests. In all studies so far reported, only initial and post-training tests have been done, also, only heart rate, blood pressure and some work loads were measured. Predicted maximal oxygen uptake was used only by Hellerstein (1968). The relative effectiveness of the training programs has not been measured.

Varnauskas et al. (1966) reported a more detailed study in which they evaluated the hemodynamics and metabolic changes at rest and during exercise after four to six weeks of physical training in nine patients with coronary disease. Coronary angiography was done one year after training in five patients. Exercise tests were done initially and at the end of the program. Each individual was "expected" to participate three times per week in a thirty minute program consisting of calisthenics, a run-walk sequence and recreational games. At rest, heart rate, blood pressure, oxygen consumption and cardiac output were measured and blood samples for blood-gas analysis

were taken. The same measurements were repeated after five and twenty-five minutes of exercise (bicycle ergometry) and also during the recovery period. The ECG was monitored continuously.

Training consisted of a thirty minute bicycle ride. A basal work load was chosen for each person and this increased for an exercise period of one to three minutes. Five minute rest intervals were given between work loads. Using the suggestions of previous investigators, all training was done under the supervision of a doctor.

The results reported were as follows. Work capacity, assessed clinically, increased in all patients. In response to standard exercise, heart rate "showed a small decrease" after training in the majority of the patients. There were only small changes in the ventilation and no consistent changes in the oxygen uptake were noted. The cardiac output, as determined by the dye-dilution method, was significantly lower after the training than before, in all conditions of rest and exercise. Respiratory quotients were lower after training than before in all phases of rest and exercise. The arterio-venous oxygen difference increased with the decreasing cardiac output. Stroke volume was significantly lower. Blood pressure, in rest and exercise, was slightly lower after training. Blood lactates were lower at rest and after exercise at the end of the training period. The hemoglobin values were slightly and consistently higher after the training than before. They concluded that the decrease of cardiac output for equivalent submaximal work was the most outstanding hemodynamic effect of physical training. The mechanism suggested for this decrease was a more effective redistribution of blood flow from different organ systems and non-exercising muscles to the capillary bed of the exercising muscles. The postulated reason for the small changes was in part due to the short training period of only four to six

weeks.

Frick and Katila (1968) studied the hemodynamic consequences of physical training, three periods weekly for one to two months, after myocardial infarction. The training was assessed in seven patients by cardiac catheterization at rest and during exercise. During the weeks of training the exercise tolerance of all subjects gradually increased. The exercise consisted of supine pedalling at two loads from 350 to 600 kpm/min. The training was followed by reduction in exercise heart rate and tension time index as determined from brachial artery pressures. Stroke volume during exercise was increased. No significant change occurred in the heart volume. Left ventricular function was improved and a significant increase in the rate of rise of right ventricular pressure occurred. The authors suggested these results indicated myocardial hypertrophy, which through reduced compliance accounted for increased ventricular filling pressures. Arterial lactate was reduced during exercise without any change in the arterio-venous oxygen difference. Increased coronary blood flow was suggested as another positive hemodynamic change. Small and not significant changes occurred in vital capacity and forced expiratory volume.

A recent report by Kasch and Boyer (1969) investigated the changes of maximal aerobic power and associated parameters in coronary patients during a six month training regimen. Eleven subjects, a minimum of one year post-infarction, participated in an interval training program of forty-five to sixty minutes four days per week. Training was done at a heart rate of approximately seventy percent of maximum. Three bicycle ergometer tests, twenty-seven minutes in duration, were performed, initially, and after three monthly and six monthly intervals. Maximal oxygen uptake, blood pressure, heart rate and blood lactates were measured. Myocardial oxygen consumption

estimated by the product of heart rate and systolic blood pressure was also measured.

As a result of the training, the maximal oxygen uptake increased from 19.9 ml/kg/min to 27.5 ml/kg/min at three months and to 30.6 ml/kg/min at six months. Consistent improvement was also observed by a decrease in resting and exercising heart rates and blood pressures. The mean maximal working capacity increased by 52 per cent. A mean decrease was seen in the index of myocardial oxygen utilization per unit of work which the authors suggested might be a sign that the myocardium had become more efficient.

Clausen et al. (1969) reported the effects of a short (four to six weeks; five days per week) bicycle ergometry program on the cardiovascular and respiratory response to exercise in nine patients with coronary artery disease. Clinical improvement and increased working capacity (average increase from 522 kpm/min to 696 kpm/min) were observed in all patients. The average cardiac output during rest or exercise was unchanged, however, the authors pointed out that it was especially interesting that there was a significant decrease in the cardiac output for a given oxygen uptake in two patients. As mentioned earlier, Varnauskas et al. (1966) found a significant decrease in cardiac output during exercise, after training, in six patients with coronary artery disease. Clausen et al. (1969) also observed a significant reduction in exercise heart rates and brachial artery mean blood pressure. The stroke volume increased and the myocardial work, assessed by the tension-time index, decreased during exercise at a given work load. It was suggested that at submaximal work loads a reduction of the blood flow to the working muscles occurs after training. Exercise ventilation was also significantly lower with oxygen uptake remaining unchanged. Exercise lactate values were significantly lower after training. In the resting state the

reduction in the arterial blood pressure was the only significant change.

Tobis and Zohman (1970) have recently reported a follow-up study of cardiac patients on a rehabilitation service that had been in practice for four years. In this period, 57 patients were treated, 42 men and 15 women. Forty-seven of the group suffered acute myocardial infarction and the others had cardiac surgery (3), pacemaker implantation (4) and coronary insufficiency (3). Of the total group, eight patients (14 per cent) have died since the beginning of the program. Only one (1.8 per cent) died on the service and the others (12.3 per cent) died withing 1.5 years of infarct. This total number of deaths represented only two additional deaths since the first six which occurred during the initial 18 months of the project. During the same period in the same hospital, approximately 23 per cent of the patients who were admitted with myocardial infarction died in the hospital.

Of the 49 survivors, 31 returned to their previous work or were retrained for other types of employment. Eighteen of the 49 did not return to work but 10 of these were already over the retirement age of 65 years. Two of the remaining who were medically capable of re-employment, however, could not obtain placement.

In 1967, the same authors reported on the physiological improvement of an additional 18 patients with angina pectoris (Zohman and Tobis, 1967). The patients had trained over a six week period at exercise levels just below that which consistently produced angina. The exercise resulted in a decrease in the frequency of angina, a lessened need for nitroglycerin and increased work capacity. There was also an increased ability to exercise to higher levels of performance in six to eighteen patients and electrocardiographic changes indicated improvement in eight. When ECG improvement occurred there was also a tendancy for oxygen debt to increase and lactate/pyruvate ratios

to increase. No change was seen in the pulmonary function tests, vital capacity and expiratory flow measurements, as a result of training. There was also little change in arterial blood gases or carbon monoxide diffusing capacity of the lung. Ventilatory equivalent (liters of ventilation per 100 cc of oxygen consumed) was unchanged.

Gottheiner (1968) reported on a unique cardiac rehabilitation program that had been operational in Tel Aviv, Israel since 1955. This program utilized strenuous outdoor sports activities. After an initial tolerance test the patients participated in sports that were characterized by rhythmicity and endurance in locomotion over increasing distances and increasing speed, such as brisk walking, running, cycling, swimming and rowing. Of 1103 patients with ischemic heart disease, (548 had sustained myocardial infarction and 555 had coronary insufficiency) observed over five years, only 49 died. Nine of these deaths occurred from non-cardiac causes. The mortality rate over five years was therefore only 3.6 per cent compared to 12 per cent in a comparable series of physically inactive post infarction patients in Israel.

Changes in coronary arteries and circulation

It is quite apparent from the above discussion that exercise therapy can, to a considerable degree, improve cardio-respiratory function and work capacity. The key to the improvement might be, as many have suggested, the direct influence of the exercise stress on the structure or function of the coronary vessels or myocardium.

Animal studies

Eckstein (1957) originally reported that moderate and severe arterial narrowing results in extensive collateral development proportional to the

degree of narrowing and exercise leads to even greater anastomoses. The results also showed that while mild arterial narrowing fails to initiate collateral arterialization by itself, the addition of exercise promotes an effective collateral circulation. Using these results, the author suggested that "the judicious use of early and continued physical exercise may reduce the clinical manifestations of coronary disease" by the increased growth of coronary arteries and improved nourishment of a segment of myocardium.

Thorner (1935) had reported earlier that new capillaries were formed in the hearts of two dogs that exercised daily over several months. This early finding has now been substantiated in guinea pigs by Petren and Sylven (1937) and in rats by Tittel et al. (1966) and Leon and Bloor (1968). Tomanek (1970) has shown that exercise increased the myocardial capillary bed in young, adult and old rats. The latter author has also shown that while aging is associated with a decrease in the number of ventricular capillaries, chronic exercise provides some mechanism which enhances the extent of the capillary bed. Tepperman and Pearlman (1961), Stevenson et al. (1964) and Leon and Bloor (1968) have also demonstrated that exercise is effective in increasing the size of the coronary tree. The evidence that exercise induces collateral capillary proliferation is encouraging for the post-myocardial infarction patient.

Human studies

Kattus (1968) presented evidence that myocardial revascularization may be achieved in cardiac patients by means of an exercise training program. Coronary angiograms were performed before and after a year of exercise management on three cardiac patients. These were only three of nineteen patients who Kattus had documented improved exercise tolerance. In all cases reported, it appears that the angiogram taken after the exercise

program disclosed that the local stenotic area had remained the same. However, a distinct improvement in collateral circulation was clearly evident. In one case, widening of local stenotic arterioschlerotic lesions in the coronary arteries was observed and the post stenotic dilation was less striking. In another, improved cardiac function brought about by the exercise program had been associated with improved collateral circulation circumventing an obstruction in the right coronary artery. Fox and Haskell (1967) reported that similar results have been found by Hornsten.

Changes in Serum Lipids

A high serum lipid level in the blood stream is associated with a high risk of coronary heart disease (NHI, 1969). It is generally assumed that factors capable of lowering serum lipid levels will have an inhibiting effect on atherogenesis and therefore be beneficial. Since exercise has been shown to improve the functioning of the cardiac patient, investigations of the effects of exercise programs on serum lipids have been undertaken. However, conflicting evidence appears in the literature on the effect of exercise on the reduction of serum lipids.

Malinov, Perley and McLaughlin (1968a, 1968b, 1969, 1970) have shown that in adult male and female rats and in adult female squirrel monkeys the oxidation of cholesterol increases with muscular contraction. Oxidation of the side chain of cholesterol occurs in the synthesis of bile acids which are formed in the process of cholesterol elimination from the body. Their findings suggest that the liver and adrenals are mainly responsible for splitting the side chain of cholesterol during rest and during muscular contraction.

Naughton and Blake (1964) reported in five post-myocardial infarction

patients that training of three to five months led to lower serum cholesterol concentrations at rest. Naughton and McCoy (1966) obtained similar reductions in serum cholesterol. Fox and Haskell (1967) criticized these and other studies (Golding, 1961; Romanova and Babarin, 1961) where exercise appeared to have a beneficial influence on serum cholesterol because there was little or no information on diet and some subjects had significant reduction in weight. In well-controlled studies, such as those conducted by Goode, Firstbrook and Shephard (1966), Varnauskas et al. (1966), Skinner et al. (1967) and Pollock et al. (1969), where increased physical activity was not accompanied by a decrease in body weight, there was no reduction in cholesterol levels. Skinner et al. (1967) observed that when body weight decreased markedly there was a reduction in serum cholesterol concentration. In general, changes in serum cholesterol level were usually associated with changes in diet and/or body weight.

In contrast to the conflicting evidence about serum cholesterol, it has been demonstrated in numerous studies that exercise can reduce the increased serum triglyceride level of cardiac patients and patients suffering from hyperlipemia (Hoffman et al., 1967; Skinner et al., 1967; Pollock et al., 1969). The decrease in the fasting serum triglycerides observed in the exercise study of Skinner et al. (1967) was an acute short-term effect that persisted for at least 44 hours. This effect also appeared to be cumulative when a regular endurance exercise program was performed.

Exercise Testing and Evaluation

Shephard (1968a) discussed the essential parameters to be measured in exercise tests and which now appear to be universally accepted. He indicates some additions and modifications, and the methodology to be employed for exercise testing of cardiac patients. These include oxygen

consumption, cardiac output, blood pressure, heart rate and the exercise electrocardiogram. The main features of interest in the exercise ECG is depression of the ST segment which is standardized in mv. Shephard considered deflections greater than 0.1 mv to be of clinical significance and he stated that the test should be stopped if depression exceeds 2mm (0.2mv).

Submaximal testing is recommended (Varnauskas, 1967) by most,.however, Ellestad et al. (1969) and Bruce and Hornsten (1969) routinely use maximal stress tests for evaluating cardiac patients. The choice of the bicycle ergometer, treadmill or step test is still controversial. However, for safety, ease of obtaining test data and reproducibility, with cardiac patients, the ergometer is often used (WHO report, 1968; Bekes, 1968).

In respect to the appropriate type of testing the use of the PWC_{170} test by Denolin, Messin and Degre (1967) for cardiac patients has already been discussed. They found that the PWC_{170} was simple, safe and reproducible and had a sound physiological basis. Wyndham (1967) criticized this test because it often underestimated work capacity and he also felt a bicycle ergometer test can be misleading in a population not accustomed to cycling. Hellerstein uses the PWC_{150} while others employ various "physical fitness indexes" (Gottheimer, 1968; Bruce et al., 1959; Rechnitzer et al., 1965, 1967b). Wyndham (1967) reviews the current methods of evaluating work capacity used in cardiac rehabilitation studies and in summary, he states a clear need for the standardization in the evaluation of cardiac patients in order to reach a physiological criterion that the patient has reached the limit of physical effort for endurance work. He suggested that, initially, heart rates, pulmonary ventilation and blood concentration of lactic and pyruvic acids should be plotted against oxygen consumptions for submaximal

tests and from these graphs the following be determined:

(a) the maximal oxygen uptake (the oxygen consumption equivalent to the maximal heart rate of 190 on a straight line fitted to $HR/\dot{V}O_2$)

(b) the level of oxygen uptake at which anaerobic metabolism occurs (where lactic and pyruvic acid in blood increase, usually heart rate 120)

(c) the level of oxygen uptake at which pulmonary ventilation increases significantly (in some cases the criterion for judging the maximum capacity should be based upon ventilation and not heart rate reaction).

On the basis of these values training schedules can be constructed so that the patient works at approximately forty to fifty per cent of maximal oxygen uptake.

Cardiovascular Measures

In the field of exercise physiology, the measurement of oxygen uptake and carbon dioxide output, by the open circuit technique is universal. This is not the case for the evaluation of cardiac output although Varnauskas et al. (1966) have shown it to be a valuable parameter for assessing improvement of coronary patients. Many investigators have found studies of cardiac output in man during rest and exercise are hampered by the difficulty involved in the measurement procedures. Magel and Anderson (1968) summarized the problem: "the dye-dilution technique and the direct Fick procedure require intravascular catheterization and blood sampling which limit their use to hospital laboratories". In long term cardiac rehabilitation studies, although the measurement of cardiac output is desirable, even in a hospital repeated catheterization and blood sampling is not a favourable proposition.

Indirect techniques of determining cardiac output, using carbon dioxide values in the Fick equation, were developed and used early in this century (Bock et al., 1928; Christiansen et al., 1914; Loewy and von Schrotter, 1903). All indirect methods were later replaced by the direct Fick and dye-dilution methods which had only limited use due to reasons indicated above. The

advent of instantaneous gas analyzers has renewed the interest in, and use of, the indirect carbon dioxide rebreathing method for estimating cardiac output.

The bloodless techniques used to measure cardiac output are in general less accurate but have much greater applicability. The simplest of these procedures is the carbon dioxide method introduced by Defares (1956, 1958), modified by Jernerus, Lundin and Thomson (1963). This method utilized the Fick procedure, estimating mixed venous PCO_2 from the exponential increase of carbon dioxide in a lung-bag system during rebreathing. Measured values for carbon dioxide output and $PaCO_2$ are substituted into the Fick equation. Cardiac output for the carbon dioxide method is thus calculated from

$$\dot{Q} = \dot{V}CO_2/(C\bar{v}_{CO_2} - Ca_{CO_2})$$

where $\dot{Q}$ = cardiac output in litres per minute

$\dot{V}CO_2$ = carbon dioxide output in litres per minute

$C\bar{v}_{CO_2}$ = ml of carbon dioxide per litre mixed venous blood

Ca_{CO_2} = ml of carbon dioxide per litre arterial blood.

This method has recently been employed in a number of experimental studies (Klausen, 1965; Jones, 1967; Jones et al., 1967, 1969; Lundin and Thomson, 1965) during rest and exercise and has been reported to be both reproducible and valid (Ferguson et al., 1968; Klausen, 1965; Muisam et al., 1968). Clausen, Larsen and Trap-Jensen (1970) report this method to be reproducible and reliable during exercise with cardiac patients in the absence of pulmonary disease. Faulty estimates were found in some cases and these were attributed to older age and/or pulmonary disorders. Magel and Anderson (1968) found this method to be both accurate and reproducible in steady state exercise situations.

The derived parameter, myocardial oxygen consumption, is related, as

Kasch and Boyer (1969) suggested, to the tension-time index (TTI). The TTI is the product of the heart rate and systolic blood pressure (Kemp and Ellestad, 1967 and Sarnoff et al., 1958). Kasch and Boyer (1969) gave data which, as discussed earlier, showed a mean decrease in this index of myocardial oxygen utilization per unit of work performed and suggested that this may be a sign that the myocardium has become more efficient. This confirmed the results of Hellerstein (1968), Frick and Katila (1968) and Clausen and Trap-Jensen (1970) following exercise therapy in cardiac disease.

CHAPTER III

METHODS

Subjects

Fourteen adult males averaging 47 years of age (range 30-45 years) and who were an average of five months (range three to seven months) post-myocardial infarct comprised the subjects of this study. Table 1 gives further details on each individual. They had been matched as closely as possible on their clinical history, age, height, weight and response to an initial treadmill exercise tolerance test with twelve lead ECG, given by the cardiologist. There was 100 per cent retention of all subjects in the program.

Training Methods

Three training methods were employed in this study, two of which were performed on the von Döbelin (AB Cyclefabriken, Monark, Sweden) bicycle ergometer and the third consisted of running and calisthenics. Variations in training on the bicycle ergometer were:

(a) Continuous effort in bicycling which was performed at a moderate resistance of 50 per cent of maximum work (as determined by the PWC_{190}). This training was for thirty minutes at each session five days per week. The ECG was monitored continuously each training session for each subject. All training started with a five minute warm-up and terminated after a five minute recovery period of unloaded slow cycling.

(b) Cyclic effort in bicycling which consisted of alternate high effort and low effort interspersed with rest pauses. High effort was 60 per cent of the maximum work capacity (determined by the PWC_{190}). Low effort was 40

TABLE 1

SUBJECT AND GROUP INFORMATION

Subject	Occupation	Age	Height (cm)	Weight (kgm)	New York Heart Association Functional Classification	Diagnosis*	Months Since MI	Training Group	Initial Type of Training
M.B.	Truck Driver	50	172.0	72.3	2	Inferior MI	3	1	Cyclic
L.M.	Stock Broker	38	180.6	86.0	2	Posterior MI	6	1	Cyclic
N.S.	Tug Boat Worker	54	172.0	78.3	2	Inferior MI	6	1	Cyclic
W.S.	Warehouseman	43	179.7	69.6	2	Inferior MI	4	1	Cyclic
M.W.	Caterer	56	174.3	54.4	3	Diagnosed CI	4	1	Cyclic
Mean		48.2	175.7	72.1			4.6		
±SD		±7.56	±4.16	±11.7			±1.30		
I.B.	Prison Guard	45	176.5	84.6	2	Posterior MI	4	2	Continuous
D.B.	Bus Driver	34	184.6	80.3	2	Anterior MI	7	2	Continuous
F.C.	Type Setter	51	172.0	72.5	2	Anterior MI	5	2	Continuous
H.C.	Officeworker	55	169.8	73.5	2	Posterior MI	6	2	Continuous
H.G.	Customs Investigator	50	178.5	74.3	2	Inferior MI	6	2	Continuous
Mean		47.0	176.3	77.0			5.6		
±SD		±8.09	±5.80	±5.21			±1.14		
J.D.	Officeworker	45	185.3	90.6	3	Anterior MI	3	3	Calisthenics-Jogging
W.F.	Draftsman	50	170.7	71.9	2	Anterior MI	7	3	Calisthenics-Jogging
E.K.	Minister	48	176.0	88.1	2	Inferior MI	6	3	Calisthenics-Jogging
B.Q.	Manager	41	175.0	74.5	2	Inferior MI	7	3	Calisthenics-Jogging
Mean		46.0	176.8	81.3			5.8		
±SD		±3.92	±6.15	±9.44			±1.89		
Total Group Mean		47	176.2	76.5			5.3		
SD		±6.49	±4.96	±9.26			±1.44		

* MI Myocardial Infarction
CI Coronary Insufficiency

per cent of the PWC_{190}. Each exercise period was four minutes in length while each rest interval was two minutes and consisted of unloaded pedalling. Three high effort-low effort series were completed each session. Training always began with five minutes of unloaded cycling and ended similarly. The ECG was monitored continuously and the total time of each exercise session was forty-five minutes.

(c) The calisthenics-jogging program was conducted three days per week. The program consists of thirty-five minutes of gradual warm-up stretching and flexibility exercises followed by twenty minutes of a run-walk series. The intensity of exercising and jogging was controlled by heart rate monitoring. Duncan, Ross and Banister (1968) describe the program. Controlled brisk walking for thirty minutes was done on the two days (Tuesday and Thursday) of the week between the exercise classes. In all groups attendance averaged 92 per cent, with an inter-individual range of 85 to 100 per cent.

Training Groups and Experimental Design

Four subjects participated in the exercise-walk-jog program. Five subjects trained initially on the continuous method and five initially on the cyclic effort program. Ergometry training was conducted in the physiotherapy departments of two hospitals or in the university laboratory. It was deemed neither feasible nor morally justifiable to use a control unexercised group.

The experiment ran twenty-six weeks with an alternating design. The first two weeks were used to establish each subject's initial exercise tolerance. Three initial tests were made and then the training programs were begun. After ten weeks of training the continuous effort group

switched to cyclic training and the cyclic to continuous. A further ten weeks of training was completed. The calisthenic-run group continued for the full twenty weeks on their program with no change. The final four weeks of training was done by all three groups using the most successful of the three methods which was the continuous effort on the bicycle. Group 1 refers to the group that started with cyclic training, Group 2 started with continuous training and Group 3 refers to those in the calisthenics-jogging program.

Exercise Tolerance Tests

The exercise tolerance of all fourteen men was initially established by three submaximal ergometric test sessions. From the results of the three initial tests the PWC_{190} was calculated as the "true" maximal physical working capacity. Training work rates were then based on this maximum, initially they were, in all cases, a little lower than sixty per cent of the PWC_{190} determined. The method outlined by Wyndham (1967) was followed in the test procedures. Submaximal tests were run to the limits of the patients' subjective tolerance, angina, a heart rate limit of 170 beats per minute or electrocardiographic changes (ST segment depression of 2 mm and/or arrhythmias). The tests began with a six minute warm-up (fifty revolutions per minute, no load), followed by three progressively increasing work rates, each of six minutes in duration, with a five minute pause between the three loads. Usually the first work rate was 300 kpm/min, however, in some cases it was 150 kpm/min. As training progressed, the first work rate was increased so that a heart rate of 110 ± 10 was achieved. The following two work rates were adjusted accordingly. Continuous ECG monitoring was performed and a carbon dioxide rebreathing method used to estimate mixed

venous carbon dioxide tension ($P_{\bar{v}CO_2}$) was administered between the warm-up and first load. Blood pressure was determined in the initial period also. Continuous recordings of oxygen and carbon dioxide were obtained during all the work intervals. Final measurements of $P_{\bar{v}CO_2}$ and blood pressure were made at the highest submaximal work rate.

These exercise tolerance tests were done, as described above, every two weeks and serial data was collected. All exercise tolerance tests were administered in an environmental chamber with controlled humidity (below eighty per cent) and temperature (15 to 24° C). The exact conditions were set to each subject's comfort and best performance. The use of environmental control was found to be especially good in the hot summer weather. A D. C. defibrillator and cardiac resuscitation equipment, including oxygen and appropriate drugs were immediately available during all training and testing periods. The university hospital service were alerted at all testing times.

The bicycle ergometers used during training and testing were calibrated frequently. The rate of pedalling was controlled by a metronome at fifty rpm on all occasions.

Gas Administration and Respiratory Gas Analysis

The subjects breathed room air through a Collins Triple-J, low resistance valve. Exhaled gas was passed through a Parkinson-Cowan, high-precision, low-resistance gas meter from which flow rates were recorded. From the gas meter the exhaled gas was passed into a Wedge spirometer which recorded both flow (liters) and flow rates (liters per second) breath by breath.

A small bore connection on the Collins Triple-J valve permitted

inspired and expired air to be continuously drawn for breath by breath analysis for carbon dioxide concentration (Godart Capnograph) and oxygen concentration (Westinghouse Analyzer). Both rapid response analyzers were connected to separate Phillips Flatbed Recorders on which a full scale deflection corresponded to a ten per cent change. Calibration of each instrument was performed before and after each test with a series of different tank gases. Zero points were determined by 100 per cent nitrogen. Calibration gases were analyzed frequently by the micro-Scholander technique.

Problems of Breath by Breath Gas Analysis

Technical difficulties arose when oxygen uptake ($\dot{V}O_2$) and carbon dioxide output ($\dot{V}CO_2$) measurements were attempted using the rapid response analyzers. In the conventional method the expired gas either passes through a mixing chamber or into a Douglas bag and the expired gas is collected over a minute or several minutes. The expired gas is then analyzed. Breath by breath analysis of $\dot{V}O_2$ and $\dot{V}CO_2$ is a difficult task. Auchincloss, Gilbert and Baule (1968) described the problems they encountered in their attempt. In this present study, as described above, the line leaving the mouthpiece was positioned so that both inspired and expired gas concentrations could be obtained. In order to compute $\dot{V}O_2$ continuously expired ventilation ($\dot{V}_E$) would have to be measured each breath, however, using the present equipment this was a very difficult task because the Wedge Spirometer had a limited capacity and the bellows would have to be recycled every few breaths. If a venturi meter such as Wigertz (1969) described was placed in the expired line this difficulty might be overcome, however, this was not the case. As a result, expired ventilation was measured each minute on the Parkinson Cowan Ventilation meter and tidal volumes were measured throughout the

experiment using the Wedge Spirometer.

The response times and frequency response of the two analyzers was adequate for a breath by breath analysis at submaximal work rates. The main problem was to determine the mean expiratory gas concentration which occurs somewhere between the inspired and expired or end-tidal point. In a normal respiratory cycle the mean alveolar air can be obtained by sampling the normal expiration volume about half way through the expiratory cycle (DuBois, 1952). This method is adequate during complete rest however during exercise when the tidal volume becomes large and the lungs are rapidly cleared with high flow rates (8-12 l/sec) this sampling method is not accurate and the mean alveolar air point would occur closer to the end-tidal expiratory point than half way through the cycle. To overcome this difficulty, expired gas samples were collected from a mixing chamber placed in line proximal to the Parkinson-Cowan meter. These samples were collected in the middle thirty seconds of each minute in fifty cc oiled glass syringes and were analyzed. The amplitude of the signal was then used as the point of mean mixed-expired gas for the breath by breath analysis which was then averaged for each minute. Corrections in ventilation were made for the amount drawn by the analyzers (total two liters/min). Oxygen consumption ($\dot{V}O_2$) and carbon dioxide consumption ($\dot{V}CO_2$) were derived by a computer program from the analysis of inspired and expired gas and of minute ventilation.

Cardiac Output

Cardiac output was determined at rest and after exercise by a carbon dioxide rebreathing method to obtain mixed venous carbon dioxide tension ($P_{\bar{v}CO_2}$) as introduced by Defares (1958, 1961) and modified by Jernerus,

Lundin and Thomson (1963). This method has been shown to be accurate especially during steady state exercise such as in the submaximal tests used in this study (Magel and Anderson, 1968). The procedure requires breath by breath analysis of the carbon dioxide content of a five liter rubber bag from which the subject rebreathes. The bag is initially filled with approximately ten per cent carbon dioxide (balance oxygen) to a volume less than twice the subject's tidal volume. Gas analysis will be performed on a rapid response carbon dioxide analyzer (Godart Capnograph; response time 0.08 seconds). When the carbon dioxide content of the bag is analyzed breath by breath, a plateau representing the equilibrium level (between the carbon dioxide partial pressure in the bag and the carbon dioxide tension of pulmonary mixed venous blood) will be reached after a few breaths. At rest, the plateau is easily reached however it is more difficult to assess during exercise and therefore the graphical breath by breath method used by Magel and Anderson (1968) was employed.

The Pa_{CO_2} was obtained in two ways. First, it was measured at rest and during exercise from the alveolar Pa_{CO_2} measured from end-tidal gas by the end-normal expiration method of Comroe (1950). Magel and Anderson (1968) found that alveolar P_{CO_2}, as measured by the end-normal expiratory methods was lower (three mm Hg) than P_{CO_2} of arterial blood and therefore all recorded end-tidal P_{CO_2} values were corrected by adding three mm Hg to each. These corrected values compared well with other reported values for P_{CO_2} of arterial blood. Lambertsen and Benjamin (1959) suggested that the lower value of alveolar P_{CO_2} with the end-normal expiratory method results from contamination with air from the respiratory dead space. The Pa_{CO_2} was also obtained from the Siggaard-Andersen Nomogram employing the Astrup method.

During both rest and exercise arterialized blood was drawn from the ear lobe which had been warmed with a hot compress. Blood samples were collected in heparinized fifty to seventy-five µl capillaries while the rebreathing was taking place. The pH was measured immediately by the Astrup method (Radiometer, Copenhagen, Model AMEI). By measuring the pH on two additional capillary samples taken at the same time as the post exercise samples and equilibrated at known P_{CO_2} (twenty-eight and fifty-five mm Hg respectively) Pa_{CO_2} standard bicarbonate (SB) and base excess (BE) were calculated according to Siggaard-Andersen (1964). The apparatus was calibrated with precision buffer at two points in the pH range (6.840 and 7.389) to 0.001 pH units. Desai et al. (1967) and Docrat and Kenny (1965) have shown the close similarity of arterialized capillary blood and actual arterial blood. Hemoglobin concentrations were measured by a hemoglobin meter.

Calculation of the cardiac output used the Fick equation applied to carbon dioxide transport. Arterial and mixed venous carbon dioxide content were obtained from the determined (Pa_{CO_2}) and mixed ($P_{\bar{v}CO_2}$) carbon dioxide tensions together with the subject's hemoglobin concentration and the carbon dioxide dissociation curves presented by Rahn and Fenn (1955). Cardiac output was determined from the relation:

$$CO = \frac{VCO_2}{C_{\bar{v}CO_2} - Ca_{CO_2}} .$$

Heart Rate

Throughout the warm-up phase, the three exercise phases and the recovery phase the heart rates were taken. A bipolar transthoracic ECG lead (modified Lead 1) tracing was taken with the RA lead at the sternal angle on the

manubrium of the sternum and the LA lead at the fifth intercostal space one inch lateral and one inch down from the left nipple. Tracings were done on an Overseas ECG Monitor. During exercise testing and training periods a sequence of ten beats was recorded at the end of each minute and the heart rate was immediately recorded. Recovery recordings were taken as in exercise for a period of six minutes. Heart rate, by telemetry, was also recorded during the calisthenics-run program for those subjects who needed monitoring.

Blood Pressure

Blood pressure measurements were taken pre and post exercise using the standard ausculatory method. The product of systolic blood pressure and heart rate was used to measure the relative myocardial oxygen demand or tension time index (Tension Time Index = Systolic Blood Pressure X Heart Rate). This measure has been described by Hellerstein (1968) and Kasch and Boyer (1969).

Pulmonary Function

A 13.5 liter Collins Respirometer was used to measure the standard pulmonary function parameters, tidal volume (TV), vital capacity (VC), respiratory rate and forced expiratory volume during one second (FEV_1). The methods are described by Collins (1965).

Blood Chemistry

Subjects were instructed to have only a light meal two hours previous to the test and blood samples for analysis were taken during the test session as indicated below.

Lactate

Venous blood samples were taken before and after exercise from the median cubital vein. Five cc samples were immediately deproteinized with five cc of trichloracetic acid (ten per cent TCA in 0.5 N HCl), the supernatant was separated by centrifugation and stored in ice until analyzed. The serum lactate concentrations were obtained using the spectrophotometric Boehringer Mannheim enzymatic method (Bergmeyer, 1962).

Cholesterol and triglycerides

Another ten cc venous blood samples was taken for the determination of serum cholesterol and triglyceride levels. The usual precedure of sampling for cholesterol and triglycerides after a twelve hour fast was not used. It was felt that the present method reflected the actual level of serum lipid the subject possessed during the majority of the day not an artificial fasting value. The serum was separated by centrifugation and stored in ice until analyzed. The serum triglyceride levels were determined using the spectrophotometric Boehringer Mannheim enzymatic method (Eggstein, 1966). The cholesterol levels were obtained by the colorimetric Boehringer Mannheim method (Watson, 1960).

Hematological Parameters

Red blood cells (RBC), white blood cells (WBC), hematocrit (Hct) and mean cell volume (MCV) levels were determined, pre and post exercise from a portion of the whole venous blood sample taken for the blood lipid determinations. A Coulter Counter (Model FN) with a Mean Cell Volume Computer was used (Coulter Electronics, 1968). Calibration of the instrument was performed using standard serum supplied by Coulter Electronics (4C Coulter Cell Control).

Analysis of Data

Since serial measurements were taken, analysis of the data was made by:

(1) Comparing rates of cardiovascular improvement of the three training groups. Individual and group assessments were made.

(2) Comparing changes in the measured variables in the alternating periods using the special types of training.

All parameters which were measured at rest during the controlled warm-up period, during exercise and sequentially over the several phases of training were investigated by a split-plot two-factor design analysis of variance (between groups and within groups--training times). The computer program for this analysis is shown in the appendix. If the over all F ratio was significant, Scheffe's (1959) S method was used to make all possible comparisons among means to test for significant differences. An analysis of variance could not be done on all the exercise values because each subject progressed at his own rate and common work rates were not performed within each group. However, for the changes in physical working capacity (PWC_{170} and PWC_{190}) an analysis of variance was performed. Tables and graphs were constructed to show the changes in all variables during the twenty-four week training period.

Note: 1kpm/min = 1 kgm/min

where

1 kp is the force acting on the mass of 1 kg at normal acceleration of gravity.

CHAPTER IV

RESULTS

A computer program, shown in the Appendix 1 was written to calculate the various cardio-respiratory parameters. A typical printout for one of the subjects (HG) is shown in Figure 3. A plot program (Appendix 2) was also written to plot heart rate, ventilation, oxygen uptake and carbon dioxide elimination for a specific work rate throughout the training period. The change of each parameter was also recorded throughout this time. Figure 4 shows a typical set of graphs for one subject (DB).

From individual computer printouts and plots, tables or results were composed (Tables 4-14) and graphical representation of the variations of all the parameters as training progressed are shown in Figures 6a,b to 19a,b. Throughout all tables weeks ten, twenty and twenty-four refer to ten, twenty and twenty-fourth weeks of training, respectively. However, due to illness and holidays the tenth, twentieth and twenty-fourth weeks do not always correspond to weeks ten, twenty and twenty-four on the graphs but to the values obtained at the end of that training phase. For groups 1 and 2, week ten is the end of the first type of training, week twenty is the end of the second type of training and week twenty-four is the end of training. For Group 3 week ten is week ten, week twenty is the end of walk-jog training and week twenty-four is the end of training.

The F-ratios and significant differences for all parameters between the separate periods as determined by analysis of variance and Scheffe (1959) S method, are presented in Table 3. This table shows significant positive changes with training in terms of increased physical work capacity, decreased

FIGURE 3. Print out of typical subject cardiovascular parameters.

RESULTS OF CARDIOVASCULAR AND RESPIRATORY DATA

TAKEN FROM CHARLES GODFREY TENTH TEST 930 1110 228 231 328 371 JULY 8TH 1970

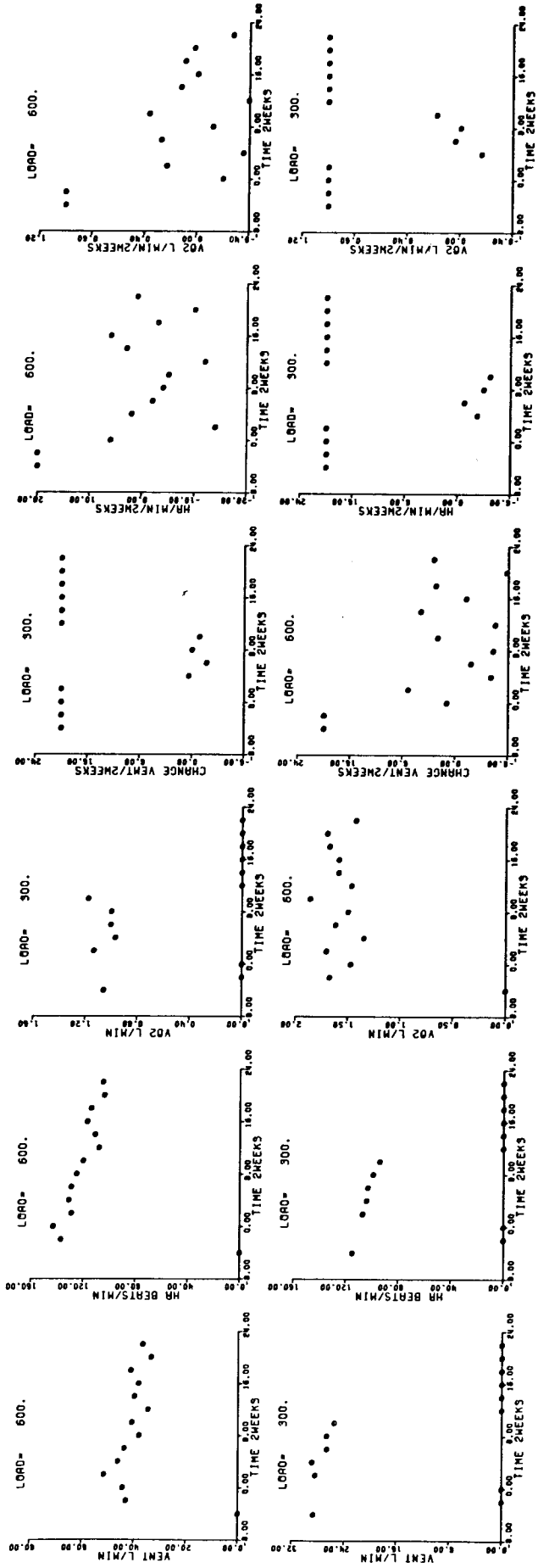
LOAD1= 450
LOAD2= 600
LOAD3= 750

VEATPS(L/MIN)	VESTPD(L/MIN	VCO2(L/MIN)	VO2(L/MIN)	R	TRUE O2	O2 PULSE(L/BEAT)	VENT EQUIV	HR
		WARM-UP						
18.7	15.6	0.446	0.558	0.799	3.581	.0066	0.28	85
18.0	15.0	0.429	0.618	0.694	4.123	.0075	0.24	82
		LOAD 1						
20.0	16.7	0.552	0.818	0.675	4.908	.0072	0.20	113
37.0	30.8	1.114	1.432	0.778	4.647	.0125	0.22	115
42.7	35.4	1.305	1.572	0.830	4.446	.0138	0.22	114
42.4	35.1	1.269	1.504	0.844	4.285	.0127	0.23	118
42.0	34.8	1.283	1.546	0.830	4.446	.0131	0.22	118
42.0	34.8	1.257	1.490	0.844	4.285	.0122	0.23	122
		LOAD 2						
38.0	31.7	1.097	1.541	0.712	4.868	.0135	0.21	114
36.4	30.3	1.096	1.409	0.778	4.647	.0115	0.22	122
46.4	38.7	1.426	1.788	0.798	4.627	.0138	0.22	130
50.0	41.6	1.349	1.977	0.682	4.747	.0150	0.21	132
52.2	43.5	1.572	1.942	0.809	4.466	.0149	0.22	130
47.8	39.6	1.461	1.760	0.830	4.446	.0133	0.22	132
		LOAD 3						
32.0	26.7	0.944	1.340	0.704	5.028	.0118	0.20	114
54.8	45.6	1.616	2.213	0.730	4.848	.0163	0.21	136
65.0	54.1	1.917	2.233	0.858	4.125	.0156	0.24	143
69.4	57.8	2.003	2.291	0.874	3.964	.0158	0.25	145
68.6	57.1	1.937	2.276	0.851	3.984	.0154	0.25	148
75.0	62.5	2.070	2.501	0.828	4.004	.0167	0.25	150

VENT1	VENT2	VENT3	HR1	HR2	HR3	VO2 1	VO2 2	VO2 3
-0.9	4.7	20.0	-9.0	-8.0	20.0	0.013	0.452	1.000

CARDIAC OUTPUT(L/MIN)	SYSTOLIC	DIASTOLIC	MYOCARDIAL DEMAND	ARTERIAL DISTENSIBILITY	HR
		WARM-UP			
4.88	106	54	8692	.001103	82
		LAST MINUTE			
13.99	154	80	23100	.002305	150

FIGURE 4. Computer graphs for submaximal cardiovascular parameters.


LOAD= 600.
LOAD= 300.
TIME 2WEEKS
VO2 L/MIN/2WEEKS
HR/MIN/2WEEKS
CHANGE VENT/2WEEKS
VO2 L/MIN
HR BEATS/MIN
VENT L/MIN

A053RU17

resting diastolic blood pressure, decreased warm-up myocardial oxygen demand, decreased cholesterol, increased vital capacity and FEV_1 and finally decreased resting lactate and base excess levels. The computer program for the analysis of variance is presented in the Appendix 3. Figure 5 shows a typical computer printout for one of the parameters studied (PWC_{170}). The group means for PWC_{170} and the serum lipids during the separate periods of training are graphically presented in Figures 20 and 21. The largest changes in PWC_{170} occurred during the periods of continuous ergometry training. The smallest changes occurred during the calisthenics-walk-jog program. Exercise training decreased the mean serum cholesterol level in all groups however did not change the serum triglyceride levels.

Each subject's progress during the twenty-four weeks is shown in individual graphs. Due to the large inter-individual differences in terms of their initial response to exercise and their rate of improvement throughout the period of training each subject will be discussed separately. An indication of the reliability and reproducibility of the respiratory gas exchange measurements is shown in Table 2 by the warm-up values for $\dot{V}O_2$ and $\dot{V}CO_2$ for all subjects. In some subjects it must be noted that a slight training effect during the twenty-four week period is apparent.

Red blood cell count, hematocrit, mean cell volume and hemoglobin were routinely measured. However, no changes were seen and this data is not included.

The carbon dioxide rebreathing method for determining cardiac output was both reproducible (see warm-up values) and safe with post infarction patients. One subject only, (EK) did have difficulty in the early period of training which disappeared later.

TABLE 2

REPRODUCIBILITY OF WARM-UP VALUES OF VENTILATION, OXYGEN UPTAKE AND CARBON DIOXIDE ELIMINATION FOR 14 SUBJECTS DURING A 24 WEEK TRAINING PROGRAM. THE VALUE "n" REFERS TO NUMBER OF INDIVIDUAL TESTS

Group	Subject	n	$\dot{V}_E$ ± SD (l/min STPD)	$\dot{V}CO_2$ ± SD (l/min STPD)	$\dot{V}O_2$ ± SD (l/min STPD)
1	M.B.	14	19.7±2.1	.543±.044	.567±.051
	L.M.	15	17.3±2.1	.488±.046	.612±.055
	N.S.	15	14.3±2.5	.445±.070	.503±.061
	W.S.	14	13.4±1.2	.420±.034	.528±.048
	M.W.	15	15.5±3.1	.427±.049	.547±.069
2	I.B.	14	16.6±2.8	.483±.079	.576±.043
	D.B.	15	14.4±2.3	.434±.046	.540±.043
	F.C.	14	15.3±2.2	.442±.042	.566±.047
	H.C.	15	14.8±2.2	.464±.047	.561±.048
	H.G.	15	16.1±0.9	.491±.043	.614±.050
3	J.D.	14	25.1±2.3	.520±.046	.642±.042
	W.F.	15	10.1±1.0	.397±.029	.526±.026
	E.K.	14	10.8±1.2	.378±.034	.496±.046
	B.Q.	14	24.4±4.1	.616±.066	.629±.070

TABLE 3

RESULTS OF THE SPLIT-PLOT TWO-FACTOR, DESIGN ANALYSIS OF VARIANCE WHERE FACTOR A IS BETWEEN GROUPS, FACTOR B IS WITHIN GROUPS (VALUES DURING INITIAL, 10 WEEKS, 20 WEEKS AND 24 WEEKS) AND FACTOR AB IS THE INTERACTION. SIGNIFICANT DIFFERENCES BETWEEN MEANS WERE DETERMINED BY THE SCHEFFE METHOD

Parameter	Factor	F-ratio	Significant Differences
PWC_{170}	A	.29	Grp 1 i, ii, iv, v **
	B	96.88 **	2 i, iii, iv, v **
	AB	6.19 **	3 i, iv, v **
			Interaction between Grp 1 & 2 due to alternating design of the training.
PWC_{190}	A	.64	Grp 1 i, ii, iv, v **
	B	78.77 **	2 i, iii, iv, v **
	AB	5.72 **	3 i, iv, v **
			Interaction between Grp. 1 & 2 due to alternating design of the training.
Blood Pressure Systolic-rest	A	.57	
	B	2.10	vi
	AB	.48	
Blood Pressure Diastolic-rest	A	1.90	Grp 1 i, iv *
	B	6.73 **	2 i, iv, v **
	AB	.73	3 vi
Cardiac Output -warm up	A	10.17 **	Significant difference (α=.01) between
	B	2.58	groups occurred at 10 and 20 weeks
	AB	.61	between Grp 2 & 3 - Grp 3 sign. larger.
Stroke Volume -warm up	A	5.19 *	Significant difference (α=.05) between
	B	1.96	groups occurred at 10 and 20 weeks
	AB	.70	between Grp 2 & 3 - Grp 3 sign. larger.
Myocardial Oxygen Demand -warm up	A	1.71	Grp 1 vi
	B	3.35 *	2 i, iv, v *
	AB	.33	3 vi

a. * Significance at α=.05 ** Significance at α=.01

A	.05	F = 3.98	A	.01	F = 7.21
B & AB	.05	F = 2.92	B & AB	.01	F = 4.51

b.
- i Significant difference between Initial and 10 weeks
- ii Significant difference between 10 weeks and 20 weeks
- iii Significant difference between 20 weeks and 24 weeks
- iv Significant difference between Initial and 20 weeks
- v Significant difference between Initial and 24 weeks
- vi No significant differences

Parameter	Factor	F-ratio	Significant Differences
Cholesterol	A	.22	Grp 1 v **
	B	9.26 **	2 iv, v **
	AB	.57	3 v **
Triglyceride	A	2.69	
	B	2.54	vi
	AB	.20	
Vital Capacity	A	1.55	Grp 1 i, v **
	B	9.64 **	2 i, v **
	AB	1.22	3 i, v **
FEV_1	A	2.02	Grp 1 v *
	B	3.39 *	2 i, v *
	AB	.53	3 vi
Lactate - rest	A	1.34	Grp 1 iii, v **
	B	5.28 **	2 v *
	AB	.97	3 vi
Base Excess	A	.38	Grp 1 i, iv, v **
	B	4.82 **	2 i, iv, v
	AB	.88	3 i, iii, v

FIGURE 5. Print out of PWC_{170} for analysis of variance.

PWC170

FACTOR A ISA=GROUPS

FACTOR B ISB=TIME

CELL MEANS

758.2000	950.0000	1157.0000	1172.0000
725.0000	1024.0000	1020.0000	1195.0000
822.5000	950.0000	1007.5000	1047.5000

CELL STANDARD DEVIATIONS

134.5760	102.7112	119.0899	124.9800
87.3041	114.4203	137.3841	107.9333
71.9375	107.3934	134.5052	153.9221

SUMMARY OF SPLIT-PLOT,UNWEIGHTED-MEANS ANALYSIS

SOURCE	SUM SQUARES	DEGREES FREEDOM	MEAN SQUARES	F-RATIOS
A	26150.77	2	13075.38	0.2909
SUBJ.W.A	494384.00	11	44944.00	
B	1055921.00	3	351973.63	96.8832
AB	135013.81	6	22502.30	6.1939
B X SUBJ W.A	119888.00	33	3632.97	

Subject Progress Reports

Group 1: Training: Cyclic Ergometry (10 weeks), Continuous Ergometry (10 weeks), Continuous Ergometry Continued (4 weeks); 5 times per week, 30 minutes per day.

Subject MB Age 50 years Height 172 cm Weight 72.3 kg (initial) 74.0 kg (final) Started training 3 months post infarction

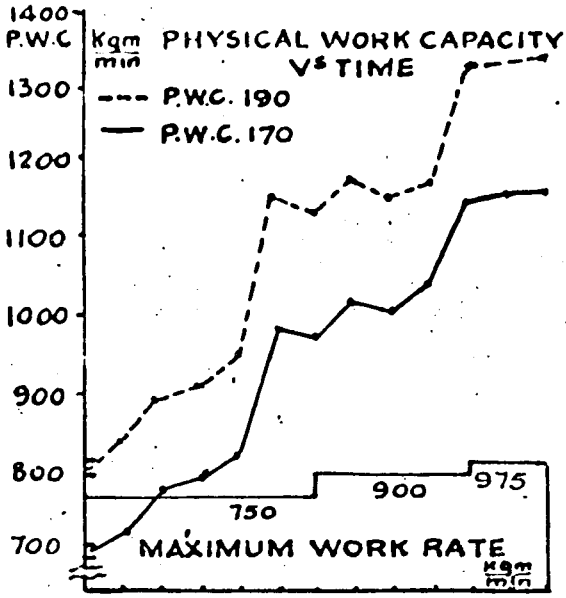
Physical Work Capacity - Figure 6a

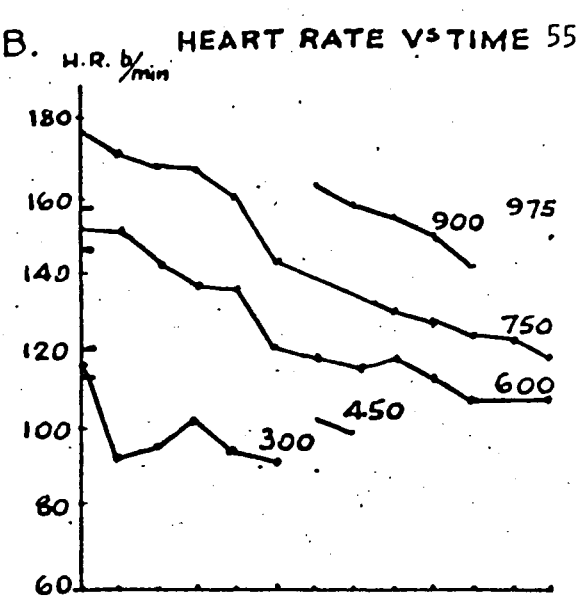
This subject had an initial physical work capacity of 700 kpm/min at a heart rate of 170 (PWC_{170}) and 810 kpm/min at a heart rate of 190 (PWC_{190}). During cyclic training his PWC (170 and 190) increased gradually for the first eight weeks after which a large increase occurred towards the end of cyclic training when he attained a PWC_{170} of 990 kpm/min and a PWC_{190} of 1160 kpm/min. His PWC_{170} increased 44.4 per cent and his PWC_{190} 43.2 per cent during this period. His PWC during subsequent continuous training increased slowly for eight weeks and another large increase occurred at the end of the tenth week of training. By the end of continuous training his PWC_{170} had increased a total of 65 per cent above initial levels and his PWC_{190} increased 64.2 per cent. He completed the twenty-four weeks of training, the last four weeks of which were with continuous ergometry, with a PWC_{170} of 1170, a total increase of 67.2 per cent and a PWC_{190} of 1340 representing a total increase of 65.5 per cent.

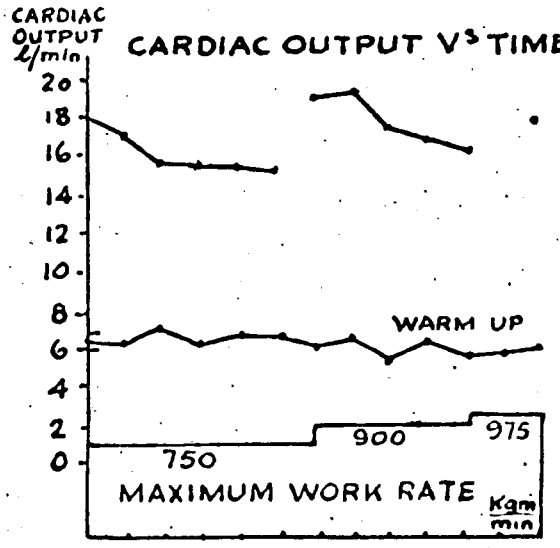
Heart Rate - Figure 6a

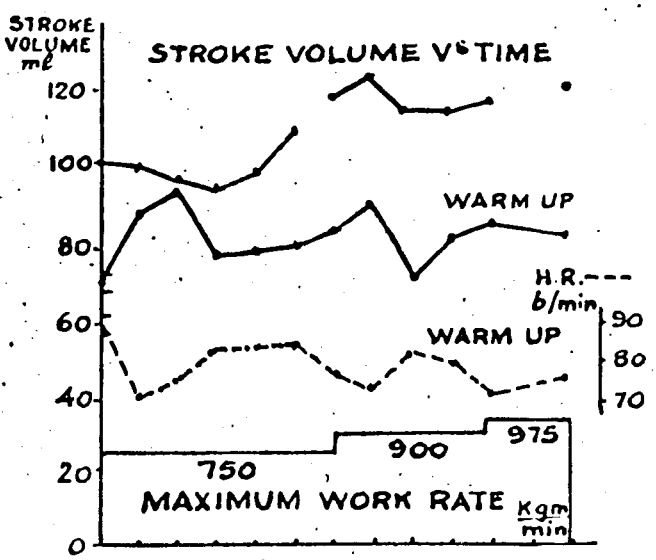
Accompanying the increase in work capacity was a large decrement in heart rate for a specific submaximal work rate (e.g. at 750 kpm/min from 176 to 119 b/min). The heart rates shown in the second graph are the sixth minute exercise values. At the beginning of training he completed three six

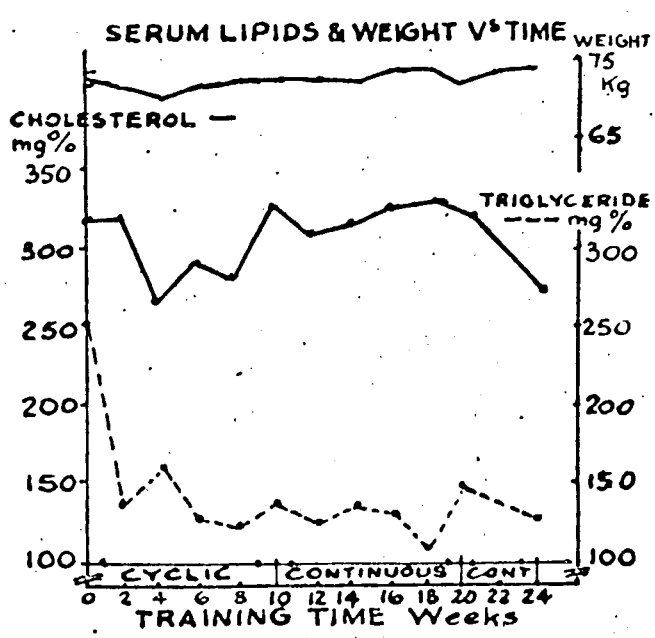
FIGURE 6a. Subject MB. Graphs of physical work capacity, heart rate, cardiac output, stroke volume, lung function, body weight and serum lipids for the 24 week rehabilitation period.

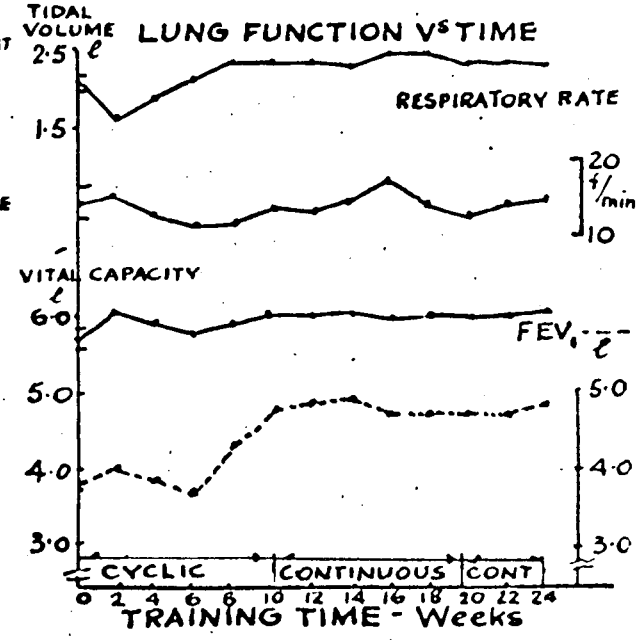

P.W.C
Kgm/min
PHYSICAL WORK CAPACITY Vs TIME
--- P.W.C. 190
— P.W.C. 170
1400
1300
1200
1100
1000
900
800
700
750
900
975
MAXIMUM WORK RATE Kgm/min


H.R. b/min
HEART RATE Vs TIME
180
160
140
120
100
80
60
900
975
750
600
450
300


CARDIAC OUTPUT l/min
CARDIAC OUTPUT Vs TIME
20
18
16
14
12
10
8
6
4
2
0
WARM UP
750
900
975
MAXIMUM WORK RATE Kgm/min


STROKE VOLUME ml
STROKE VOLUME Vs TIME
120
100
80
60
40
20
0
WARM UP
H.R.--- b/min
90
80
70
WARM UP
750
900
975
MAXIMUM WORK RATE Kgm/min


SERUM LIPIDS & WEIGHT Vs TIME
WEIGHT
75 Kg
65
CHOLESTEROL — mg%
TRIGLYCERIDE --- mg%
350
300
250
200
150
100
300
250
200
150
100
CYCLIC
CONTINUOUS
CONT
0 2 4 6 8 10 12 14 16 18 20 22 24
TRAINING TIME Weeks


TIDAL VOLUME l
LUNG FUNCTION Vs TIME
2·5
1·5
RESPIRATORY RATE
20
f/min
10
VITAL CAPACITY l
6·0
5·0
4·0
3·0
FEV1 --- l
5·0
4·0
3·0
CYCLIC
CONTINUOUS
CONT
0 2 4 6 8 10 12 14 16 18 20 22 24
TRAINING TIME - Weeks

minute periods of exercise at work rates of 300 kpm/min, 600 kpm/min and 750 kpm/min, respectively. These were the testing loads used throughout cyclic training. The testing loads for continuous training were raised to 600, 750 and 900 kpm/min. The last test was at three work rates of 600, 750 and 975 kpm/min. His heart rate at the end of the 975 kpm/min test (150) was the same as that of the 600 kpm/min test in his initial ride twenty-four weeks before. During cyclic training a continuous reduction in heart rate was observed in the two lower work rates of the test session (300 and 600) however the heart rate for 750 kpm/min decreased only slightly until the sixth week; a large decrement (17 beats) was observed by the end of the tenth week. During continuous training a steady reduction in the heart rate for standard work rates was observed. The warm-up heart rate showed a small but consistent decrease during the twenty-four weeks.

The initial treadmill exercise tolerance test given by a cardiologist showed characteristic ST segment depression in the lateral precardial leads indicative of ischemic heart disease. By the fourteenth week of training the ST segment depression had disappeared in the training and testing electrocardiogram.

Cardiac output and stroke volume - Figure 6a

Figure 6a shows the changes of cardiac output and stroke volume during warm-up and at the end of the highest work rate of the test session. Warm-up cardiac output remained very stable during the twenty-four weeks. The mean of the initial tests was 6.12 l/min, at ten weeks it was 6.46 l/min, at twenty weeks it decreased slightly to 5.85 l/min and after the last month of continuous it was 5.94 l/min. Mean warm-up stroke volume during the initial tests was 65.4 ml, at the end of cyclic training it increased to 77.9 ml and,

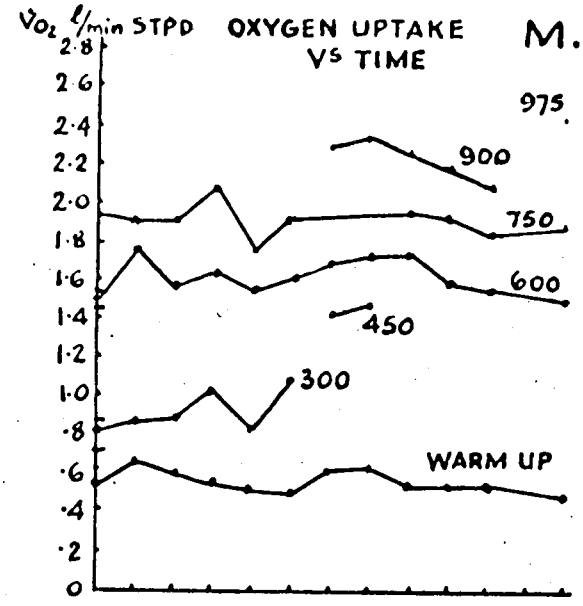
except for the fourteenth and sixteenth tests it remained at 80 ml. The exercise-cardiac output showed an initial decrease then remained constant to the end of cyclic training. Exercise-cardiac output during continuous training steadily decreased from 19.12 l/min for a work rate of 900 kpm/min to 16.3 l/min. The final value for 975 kpm/min was 17.99 l/min, a value similar to the first test at 750 kpm/min during the initial tests. During the period of training stroke volume decreased slightly up to the eighth week (from 103 ml to 98 ml) then increased to 108 ml by the end of cyclic training. At the higher load during continuous training stroke volume remained quite constant at 115 ml. During the final test at 975 kpm/min, although the cardiac output was the same as in the initial tests at 750 kpm/min, the stroke volume was higher (120.5 ml compared to 103 ml).

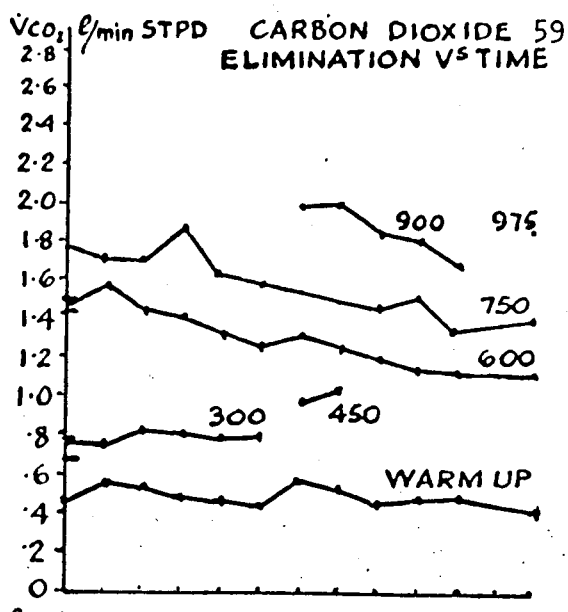
Respiratory gas exchange - Figure 6b

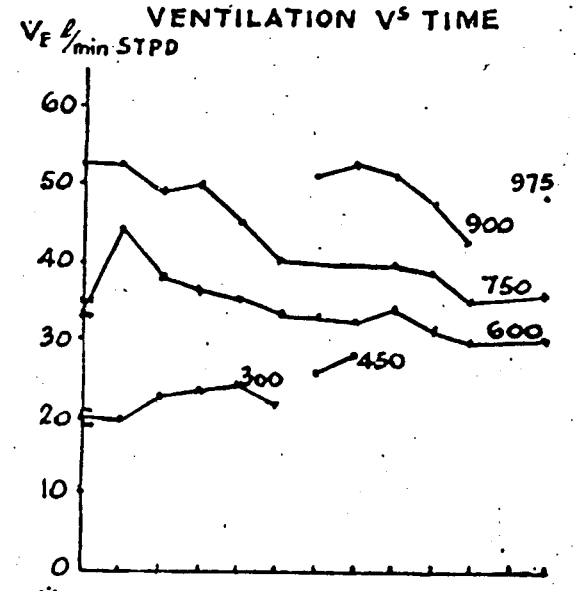
Graphical changes in $\dot{V}O_2$, $\dot{V}CO_2$ and $\dot{V}_E$ during the submaximal tests are shown in Figure 6b. These values are appropriate to the highest work rate of the test session. A marked reduction in ventilation occurred in work rates above 450 kpm/min throughout the training period. Oxygen uptake remained steady during the cyclic training period and then slowly decreased during continuous training especially for 900 kpm/min of work. Carbon dioxide elimination steadily decreased throughout training for all work rates above 450 kpm/min. Warm-up values for $\dot{V}OC_2$ and $\dot{V}O_2$ remained quite constant throughout the training. The respiratory exchange ratio remained close to the initial values throughout most of the cyclic training period however a steady reduction in the R values was seen during continuous training.

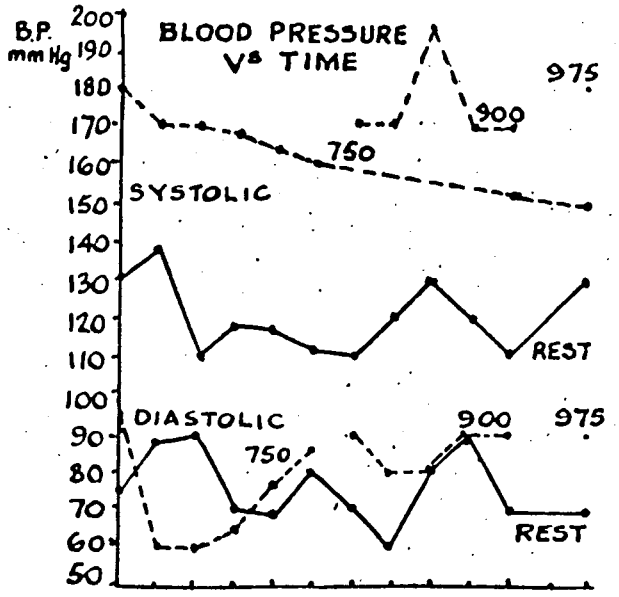
FIGURE 6b. Subject MB. Graphs of respiratory gas exchange, blood pressure and base excess for the 24 week rehabilitation period.

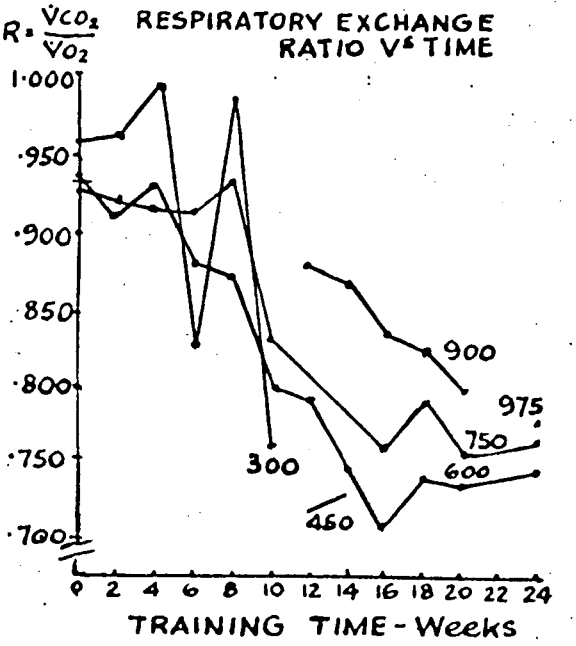
M.B.

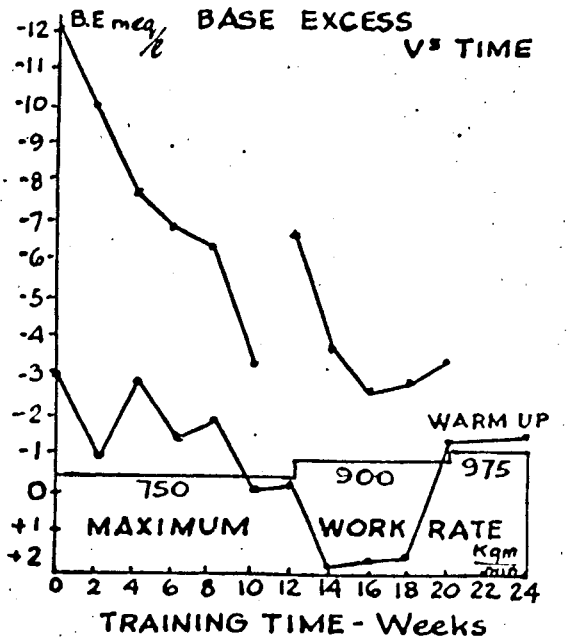

V̇O2 l/min STPD OXYGEN UPTAKE Vs TIME
975
900
750
600
450
300
WARM UP


V̇CO2 l/min STPD CARBON DIOXIDE ELIMINATION Vs TIME
900
975
750
600
300
450
WARM UP


V̇E l/min STPD VENTILATION Vs TIME
975
900
750
600
450
300


B.P. mm Hg BLOOD PRESSURE Vs TIME
975
900
750
SYSTOLIC
REST
DIASTOLIC
900
975
750
REST


R = V̇CO2 / V̇O2 RESPIRATORY EXCHANGE RATIO Vs TIME
900
975
750
600
300
450
TRAINING TIME - Weeks


B.E meq/l BASE EXCESS Vs TIME
WARM UP
750
900
975
MAXIMUM WORK RATE Kgm/min
TRAINING TIME - Weeks

Exercise oxygen pulse and ventilation equivalent - Table 10

Although not shown graphically the exercise values for oxygen pulse (l/beat) increased for all test work rates while the ventilation equivalent (V_E l/min BTPS/VO_2 l/min STPD) decreased. The following changes occurred in oxygen pulse at the designated work rates as a result of training: 0.0066 → 0.0126 l/beat at 300 kpm/min; 0.0090 → 0.0145 l/beat at 600 kpm/min and 0.0100 → 0.0160 l/beat at 750 kpm/min. The changes in ventilatory equivalent were as follows: 26 → 19 at 300 kpm/min; 26 → 19 at 600 kpm/min and 30 → 19 at 750 kpm/min.

Base excess and lactate - Figure 6b and Tables 7 and 12

Exercise metabolic acidosis observed from base excess (BE) values and lactate (La) accumulation shows marked reduction throughout both types of training. This reduction was most obvious at higher work rates (900 and 975 kpm/min) during the continuous training period. The following reductions were observed: BE, -12.0 → 3.0 meq/l, and La, 90.3 → 24.5 mg %, 750 kpm/min; BE, -6.8 → -3.5 meq/l, and La 35.3 → 21.0 mg %, 900 kpm/min.

Blood pressure - Figure 6b

Resting systolic blood pressure decreased gradually up to the twelfth week of training, and remained at this level until week twenty when an increase to the initial level (130 mm Hg) occurred. The resting diastolic blood pressure remained relatively constant throughout training. Exercise systolic blood pressure at 750 kpm/min decreased from 180 mmHg to 150 mmHg during the training period. The blood pressure at 900 kpm/min did not change with training. Exercise-diastolic pressure at 750 kpm/min showed an initial decrease but returned, over the next eight weeks, to the initial value

(90 mmHg). The diastolic pressure at 900 kpm/min remained constant.

Myocardial oxygen demand - Table 14

Warm-up myocardial oxygen demand showed steady reduction until week twenty (10929 → 7700 mmHg X HR) however it then increased by the end of week twenty-four to 9768 mmHg X HR. Training reduced not only the resting values but also the exercise values of this variable. During the first ten weeks of training the values for a 750 kpm/min work rate reduced from 24640 to 22880 mmHg X HR. A small reduction was also seen during the following ten weeks when the highest testing work rate was 900 kpm/min (24600 → 23970 mmHg X HR).

Lung Function - Figure 6a

Tidal volume showed little change after a slight initial increase. The respiratory rate remained constant throughout the period. The largest changes were observed in FEV_1, the air volume expired in one second, which increased by the end of the sixth week from an initial level of 3.9 l to 4.8 l. It remained constant thereafter until the end of the training. Vital capacity showed a small increase during the whole training period (5.68 l → 6.03 l).

Body weight and serum lipids - Figure 6a

Body weight remained stable during the twenty-four weeks. Cholesterol values showed an early decrease however they then steadily increased to the initial level where they remained throughout the twenty weeks and then decreased during the final month of continuous training. Serum triglycerides showed a large early decrease and then remained stable throughout the twenty-four weeks. No obvious differences occurred, attributable to the type of training practiced.

Subject LM Age 38 years Height 180.6 cm Weight 86.0 kg (initial) 85.7 (final) Started training six months post infarction

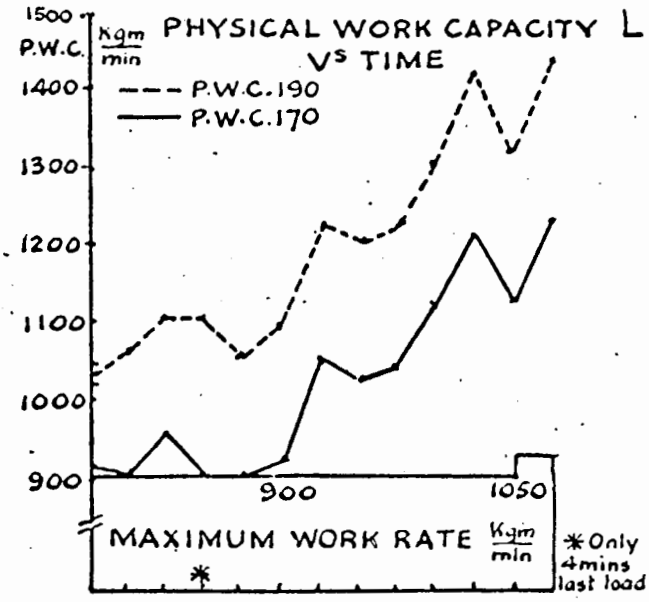
Physical work capacity - Figure 7a

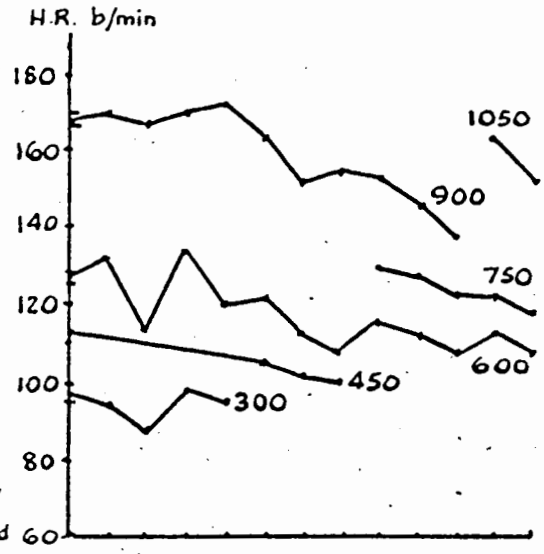
The mean initial average PWC_{170} of this subject was 907 kpm/min and his PWC_{190} was 1037 kpm/min. Ten weeks of cyclic training made very little change in his PWC_{170} (907 → 938 kpm/min) and only a small increase was noted in his PWC_{190} (1037 → 1107 kpm/min). Ten weeks of continuous training that followed produced a dramatic increase in both his PWC_{170} (34 per cent - 907 → 1215 kpm/min) and PWC_{190} (37.4 per cent - 1037 → 1415 kpm/min). The final month of continuous training increased his PWC_{170} (1230 kpm/min) by a total of 35.6 per cent and increased his PWC_{190} (1430 kpm/min) by a total of 38.8 per cent. An increased sense of well being and ability to work were quite marked in this subject. His early fear of physical exertion also disappeared.

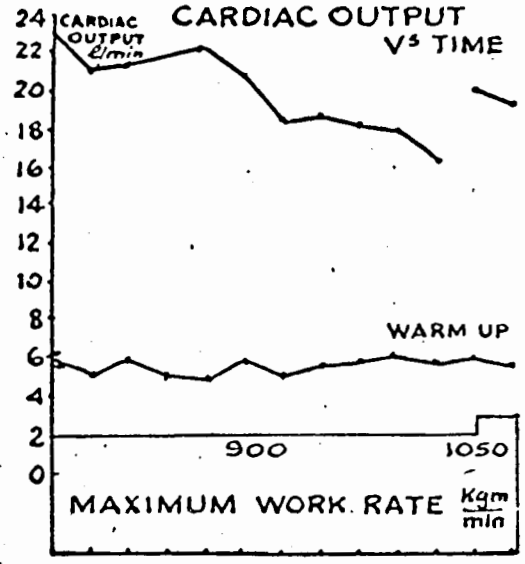
Heart rate - Figure 7a

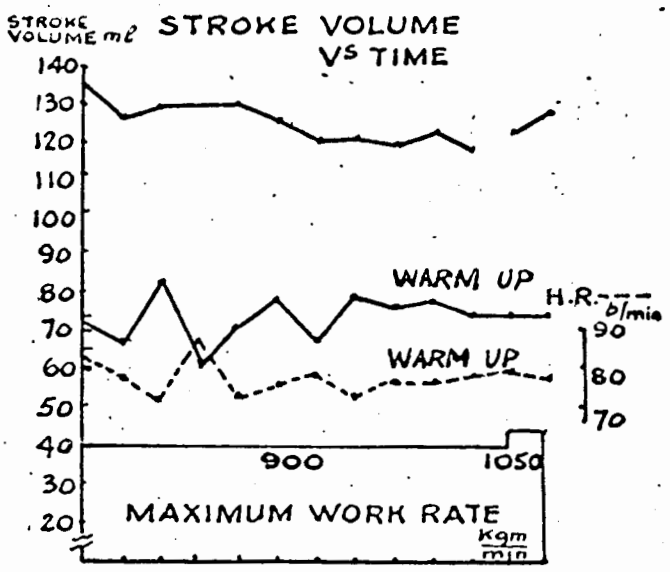
Cyclic training failed to lower the heart rate response to exercise, however, once he was changed to continuous training a large decrease was seen in this parameter expecially at 900 kpm/min (170 b/min → 138 b/min). By the middle of the ten week period of continuous training the subject's three test work rates were increased from 300, 600 and 900 to 600, 750 and 900 kpm/min, respectively. During the last month of continuous training (weeks twenty to twenty-four) the maximum task was increased to 1050 kpm/min with final exercise heart rates of 164 and 153 b/min, respectively, as compared to 170 b/min during the evaluation tests at 900 kpm/min in weeks zero to ten. Warm-up heart rate, as seen in Figure 7a, was slightly decreased (82 b/min → 78 b/min).

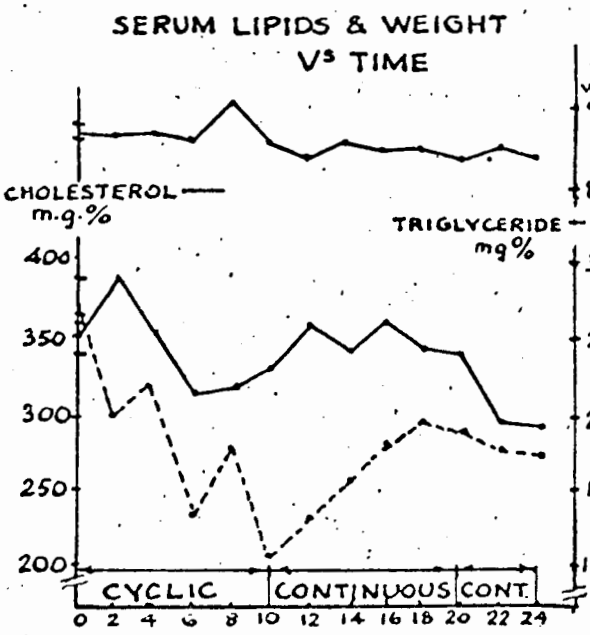
FIGURE 7a. Subject LM. Graphs of physical work capacity, heart rate, cardiac output, stroke volume, lung function, body weight and serum lipids for the 24 week rehabilitation period.

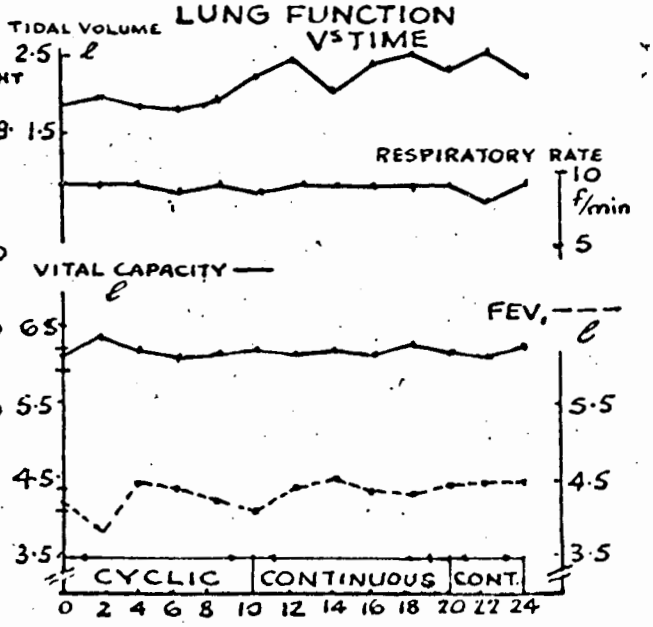

PHYSICAL WORK CAPACITY L.M.
VS TIME
P.W.C. Kgm/min
1500
1400
1300
1200
1100
1000
900
--- P.W.C.190
—— P.W.C.170
900
1050
MAXIMUM WORK RATE Kgm/min
*Only 4mins last load


HEART RATE VS TIME
H.R. b/min
180
160
140
120
100
80
60
1050
900
750
600
450
300


CARDIAC OUTPUT
VS TIME
CARDIAC OUTPUT ℓ/min
24
22
20
18
16
14
12
10
8
6
4
2
0
WARM UP
900
1050
MAXIMUM WORK RATE Kgm/min


STROKE VOLUME
VS TIME
STROKE VOLUME mℓ
140
130
120
110
100
90
80
70
60
50
40
30
20
WARM UP
H.R. b/min
WARM UP
90
80
70
900
1050
MAXIMUM WORK RATE Kgm/min


SERUM LIPIDS & WEIGHT
VS TIME
CHOLESTEROL m.g.%
TRIGLYCERIDE mg%
WEIGHT Kg.
400
350
300
250
200
300
250
200
150
100
90
80
CYCLIC
CONTINUOUS
CONT.
0 2 4 6 8 10 12 14 16 18 20 22 24
TRAINING TIME - Weeks


LUNG FUNCTION
VS TIME
TIDAL VOLUME ℓ
2·5
1·5
RESPIRATORY RATE
10
f/min
5
VITAL CAPACITY ℓ
FEV, ℓ
6·5
5·5
4·5
3·5
5·5
4·5
3·5
CYCLIC
CONTINUOUS
CONT.
0 2 4 6 8 10 12 14 16 18 20 22 24
TRAINING TIME - Weeks

Cardiac output and stroke volume - Figure 7a

A consistent change in warm-up cardiac output did not occur over the twenty-four week period of rehabilitation. However, a large decrease in exercise cardiac output was seen (23.00 l/min → 16.30 l/min; 900 kpm/min) over this period. The reduction was more obvious from weeks ten to twenty once continuous training began. A small change (23.00 l/min → 20.50 l/min) was seen during the cyclic training period. Warm-up stroke volume was slightly increased (70 → 75 ml) however exercise-stroke volume was decreased (135 ml → 118 ml; 900 kpm/min). Again, the major part of the decrease occurred during the continuous type of training. As may be observed in the graph of heart rate and cardiac output, decreases occurred during cyclic training only during the last two weeks.

Respiratory gas exchange - Figure 7b

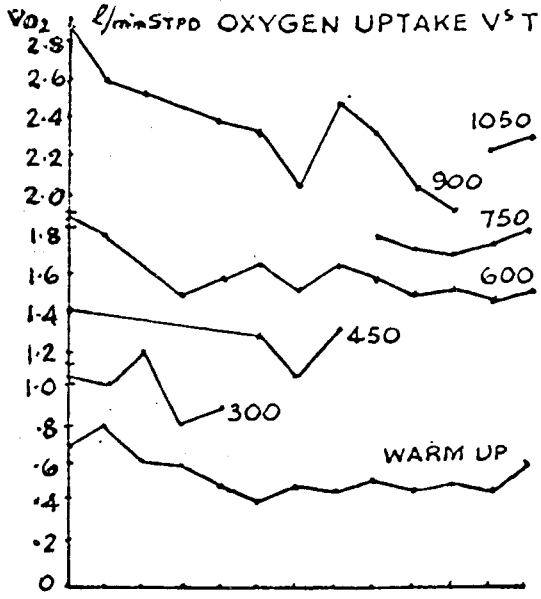
A large decrease in ventilation was observed at the more strenuous work rates, especially from week ten to twenty-four. At lower work rates (300, 450 and 600 kpm/min) a small decrement in ventilation occurred, however the ventilation at 750 kpm/min increased slightly. Oxygen uptake and carbon dioxide elimination decreased for all work loads except 750 kpm/min; the largest changes were observed at 900 kpm/min. Carbon dioxide elimination appeared unchanged from weeks two to eight at all work rates, however, $\dot{V}O_2$ decreased throughout the twenty-four week period. The respiratory exchange ratio (R) increased from the initial tests up to the eighth week and decreased considerably thereafter.

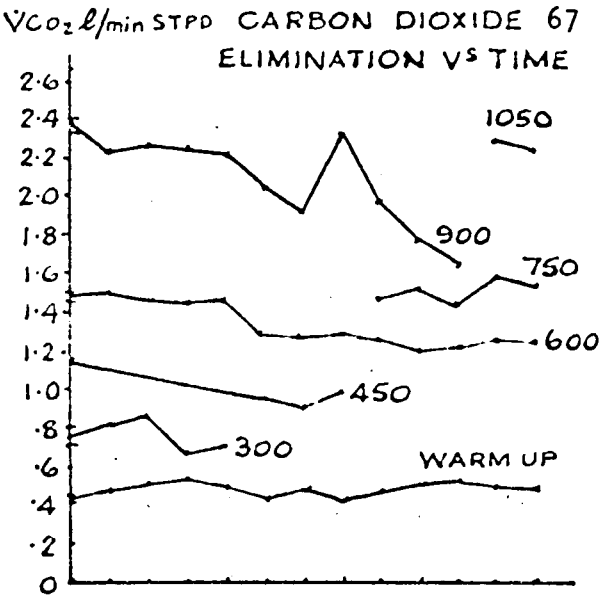
Exercise-oxygen pulse and ventilation equivalent - Table 10

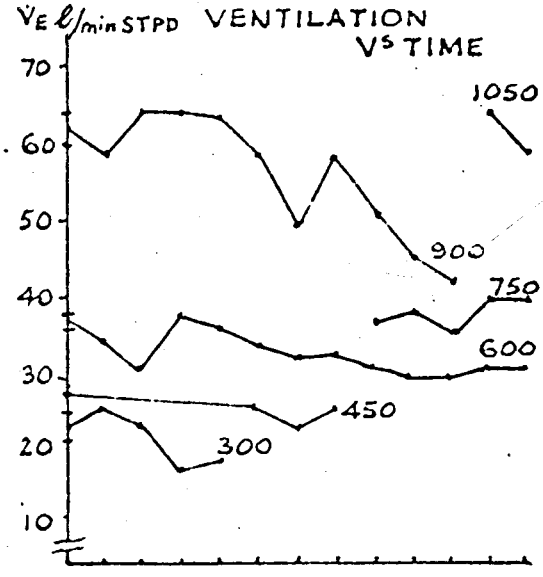
The ventilation equivalent did not change with training. The value for

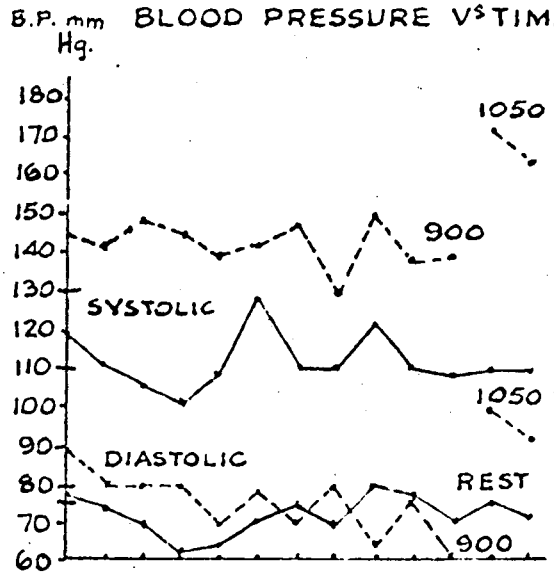
FIGURE 7b. Subject LM. Graphs of respiratory gas exchange, blood pressure and base excess for the 24 week rehabilitation period.

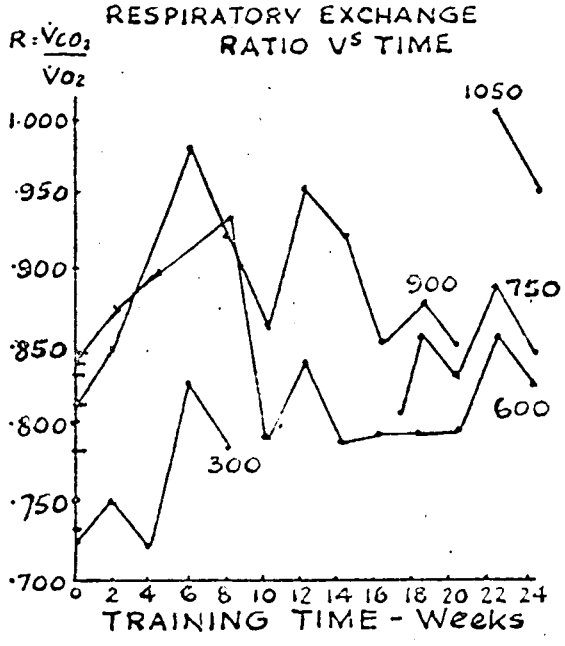
L.M.

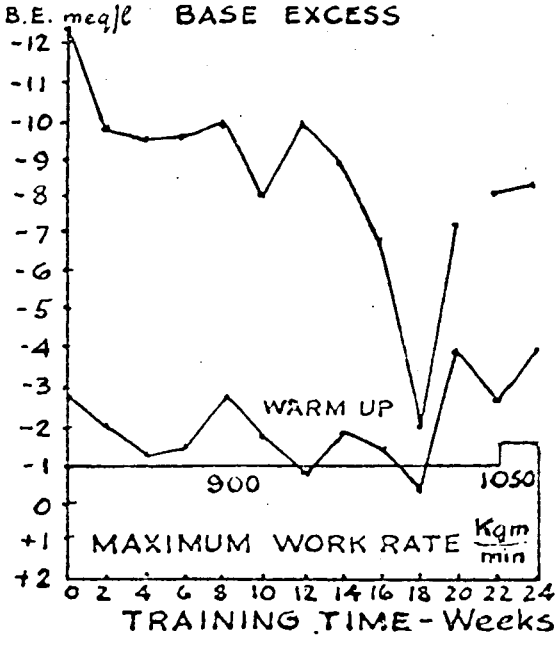

$\dot{V}O_2$ l/min STPD OXYGEN UPTAKE Vs TIME
2·8
2·6
2·4
2·2
2·0
1·8
1·6
1·4
1·2
1·0
·8
·6
·4
·2
0
1050
900
750
600
450
300
WARM UP


$\dot{V}CO_2$ l/min STPD CARBON DIOXIDE ELIMINATION Vs TIME
2·6
2·4
2·2
2·0
1·8
1·6
1·4
1·2
1·0
·8
·6
·4
·2
0
1050
900
750
600
450
300
WARM UP


$\dot{V}E$ l/min STPD VENTILATION Vs TIME
70
60
50
40
30
20
10
1050
900
750
600
450
300


B.P. mm Hg. BLOOD PRESSURE Vs TIME
180
170
160
150
140
130
120
110
100
90
80
70
60
1050
900
SYSTOLIC
1050
DIASTOLIC
REST
900


R: $\frac{\dot{V}CO_2}{\dot{V}O_2}$ RESPIRATORY EXCHANGE RATIO Vs TIME
1·000
·950
·900
·850
·800
·750
·700
1050
900
750
600
300
0 2 4 6 8 10 12 14 16 18 20 22 24
TRAINING TIME - Weeks


B.E. meq/l BASE EXCESS
-12
-11
-10
-9
-8
-7
-6
-5
-4
-3
-2
-1
0
+1
+2
WARM UP
900
1050
MAXIMUM WORK RATE Kgm/min
0 2 4 6 8 10 12 14 16 18 20 22 24
TRAINING TIME - Weeks

all work rates from 300 to 750 kpm/min was 20 throughout the training and 23 and 25 respectively for 900 and 1050 kpm/min. Oxygen pulse remained unchanged with training at 300 kpm/min. A small increase was observed at work rates of 450, 600 and 750 kpm/min (0.0136 → 0.0164 l/beat at 750 kpm/min). A small decrease occurred during the tests at 900 kpm/min (0.0152 → 0.0140 l/beat).

Base excess and lactate - Figure 7b and Table 7 and 12

Exercise-metabolic acidosis decreased immediately after cyclic training started and then remained constant. A further decrease did not occur until the type of training was changed to continuous exercise. For the final tests at 1050 kpm/min the base excess and lactate values (-8.0 meq/l and 15 mg %, respectively) were lower even compared to values earlier in training at a lower work rate (900 kpm/min, BE -12.7 meq/l and La 40 mg %). The same measurements during warm-up decreased up to the eighteenth week and then increased during the final few weeks.

Blood pressure - Figure 7b

Resting systolic and diastolic blood pressure decreased up to the eighth week and then increased to the initial values. A significant decrease in exercise-diastolic pressure was observed throughout the twenty-four week period. Exercise systolic pressure showed a small decrease during the training period.

Myocardial oxygen demand - Table 14

The warm-up myocardial oxygen demand first increased and then returned to the initial value (8400 mmHg X HR). There was no change in exercise-myocardial oxygen demand at the beginning of training however a large decrease

occurred after the twelfth week of training and remained at this lower level (22950 → 19320 mmHg X HR, 900 kpm/min and 28208 → 25092 mmHg X HR, 1050 kpm/min).

Lung function - Figure 7a

Vital capacity and the respiratory rate remained unchanged while FEV_1 increased early in training and then remained unchanged. Resting tidal volume remained unchanged up to the eighth week and then increased and remained at this level.

Body weight and serum lipids - Figure 7a

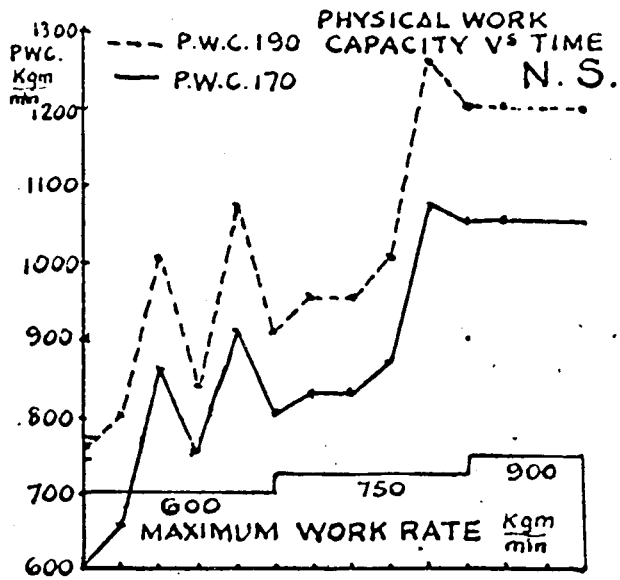
Body weight gradually decreased from 86.7 kg to 84.7 kg over the twenty-four week period. Cholesterol decreased up to the eighth week (350 mg % → 317 mg %) then increased during the next eight weeks to the initial level and steadily decreased again from the sixteenth week to a low of 296 mg % at the end of the program. Serum triglycerides decreased greatly up to the tenth week (263 mg % → 108 mg %) then steadily increased again up to the eighteenth week (199 mg %) and decreased over the final six weeks of continuous training to a level of 177 mg %.

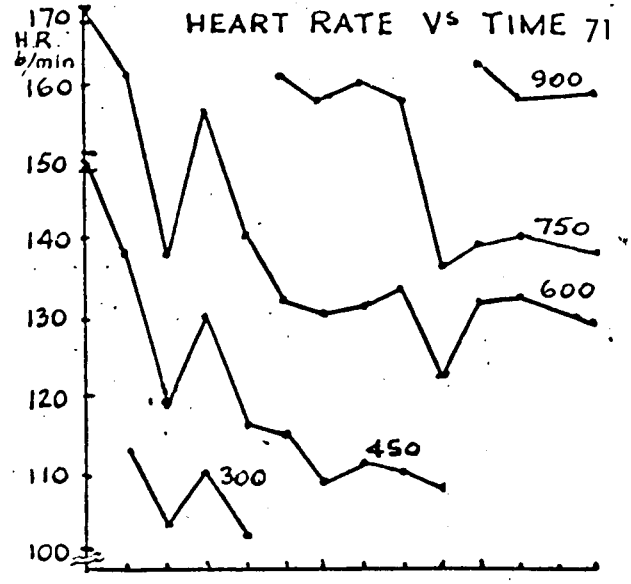
Subject NS Age 45 years Height 172.0 cm Weight 78.3 kg (initial) 87.8 kg (final) Started training six months post infarction

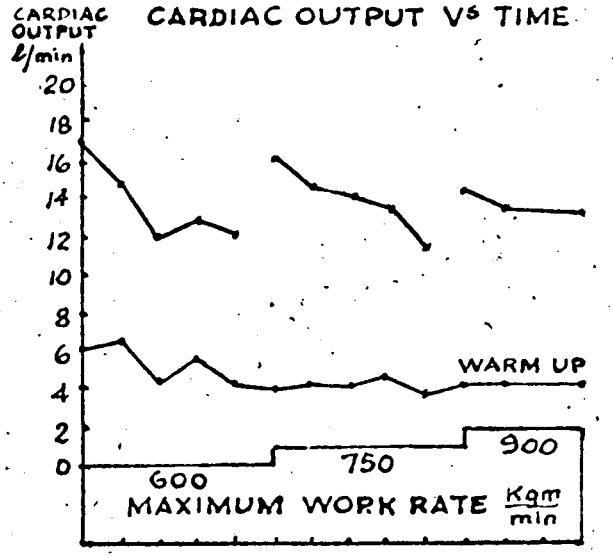
Physical work capacity - Figure 8a

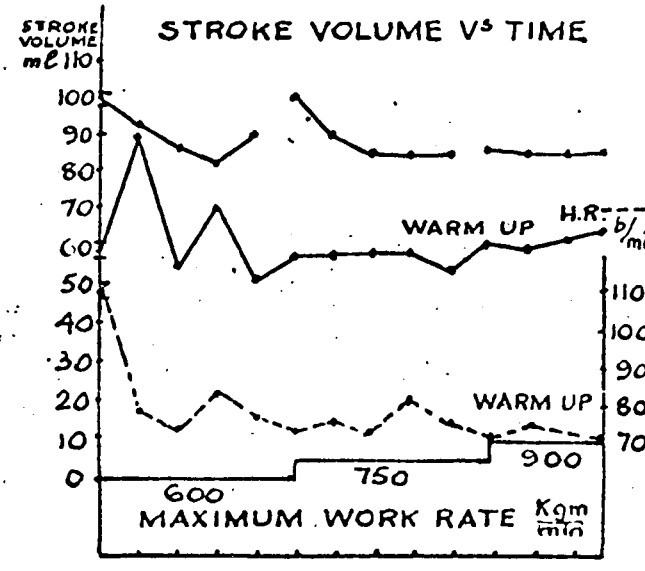
At the end of cyclic training this subject's PWC_{170} had increased by 38.4 per cent (600 → 830 kpm/min) and his PWC_{190} had increased by 26.6 per cent (750 → 950 kpm/min). During this period of twelve weeks his response to exercise was eratic. This was in part due to the fact that he started

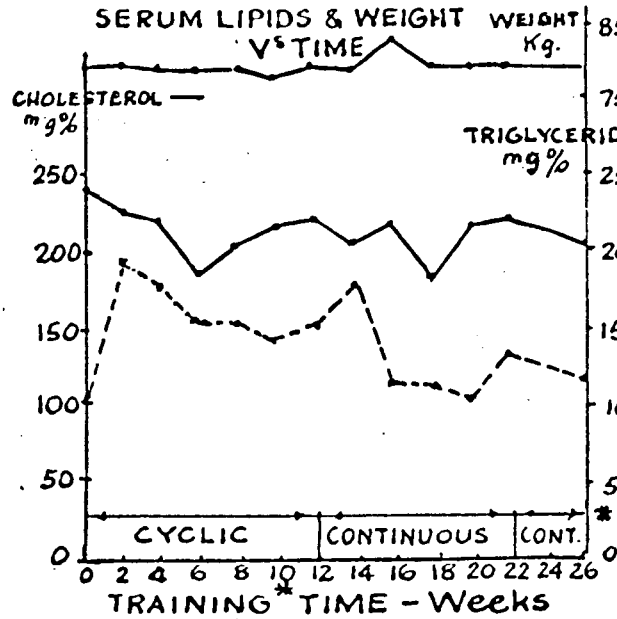
FIGURE 8a. Subject NS. Graphs of physical work capacity, heart rate, cardiac output, stroke volume, lung function, body weight and serum lipids for the 24 week rehabilitation period.

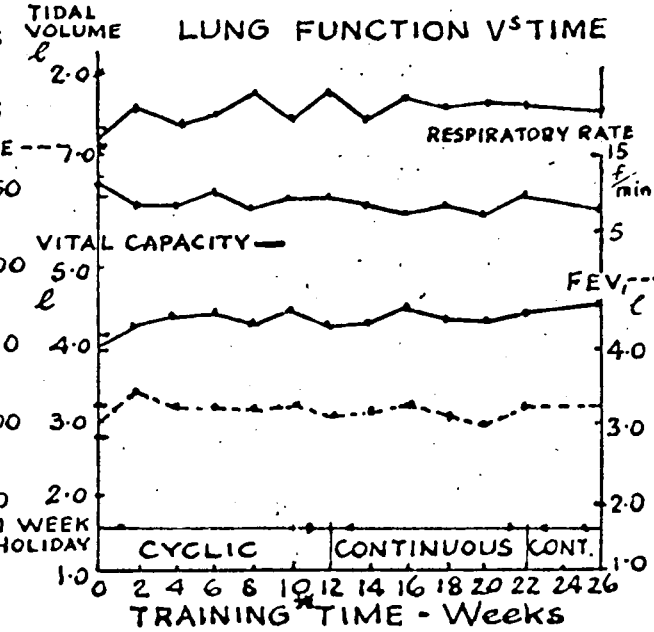

PHYSICAL WORK CAPACITY Vs TIME
N.S.
P.W.C. 190
P.W.C. 170
PWC. Kgm/min
1300
1200
1100
1000
900
800
700
600
600
750
900
MAXIMUM WORK RATE Kgm/min


HEART RATE Vs TIME
H.R. b/min
170
160
150
140
130
120
110
100
900
750
600
450
300


CARDIAC OUTPUT Vs TIME
CARDIAC OUTPUT l/min
20
18
16
14
12
10
8
6
4
2
0
WARM UP
600
750
900
MAXIMUM WORK RATE Kgm/min


STROKE VOLUME Vs TIME
STROKE VOLUME ml
110
100
90
80
70
60
50
40
30
20
10
0
WARM UP
H.R. b/min
WARM UP
600
750
900
MAXIMUM WORK RATE Kgm/min


SERUM LIPIDS & WEIGHT Vs TIME
WEIGHT Kg.
CHOLESTEROL mg%
TRIGLYCERIDE mg%
250
200
150
100
50
0
85
75
250
200
150
100
50
0
CYCLIC
CONTINUOUS
CONT.
0 2 4 6 8 10 12 14 16 18 20 22 24 26
TRAINING * TIME - Weeks


LUNG FUNCTION Vs TIME
TIDAL VOLUME l
2·0
1·0
RESPIRATORY RATE f/min
15
5
VITAL CAPACITY
FEV1
5·0
4·0
3·0
2·0
1·0
l
CYCLIC
CONTINUOUS
CONT.
* 1 WEEK HOLIDAY
0 2 4 6 8 10 12 14 16 18 20 22 24 26
TRAINING * TIME - Weeks

work again at a particularly physically strenuous job. A total increase of 75 per cent above his initial PWC_{170} (1050 kpm/min) was observed after ten weeks of continuous training. His PWC_{190} (1200 kpm/min) increased by 60 per cent above the initial value. No further change in either his PWC_{170} nor PWC_{190} occurred after the final month of continuous training although he could now successfully complete the test session at a maximum work rate of 900 kpm/min compared to the earlier work rate of 750 kpm/min.

Heart rate - Figure 8a

Figure 8a shows the large decrease in heart rate for all work rates throughout twenty weeks. The final month of continuous training resulted in no further decrease in heart rate. During the cyclic training period the three test work rates were 300, 450 and 600 kpm/min, however, once continuous training was begun he could, for the same heart rate response, accomplish work rates of 450, 600 and 750 kpm/min, respectively and later 900 kpm/min. After an initial immediate decrease in warm-up heart rate after two weeks of training, a gradual decrease took place to a low value of 70 b/min (initial average warm-up was 106 b/min). Resting values showed a similar response.

Cardiac output and stroke volume - Figure 8a

Figure 8a shows the changes of cardiac output and stroke volume during the training period. After the second week of training a considerable reduction occurred in warm-up cardiac output (6.76 l/min → 4.32 l/min average) and stroke volume (90 ml → 59 ml average). No further decrease occurred after the tenth week. During both cyclic and continuous ergometry training exercise cardiac output decreased. During cyclic training the cardiac output at 600 kpm/min decreased up to the fourth week (16.93 → 11.99 l/min) then

increased slightly to 13.13 l/min and 12.65 l/min during the next four weeks. The cardiac output when measured first at 750 kpm/min was 16.00 l/min and after six weeks of continuous training this was reduced to 11.57 l/min. A small reduction in cardiac output at the highest work rate occurred during the final month of continuous training (14.50 l/min → 13.66 l/min at 900 kpm/min). Exercise-stroke volume at 600 kpm/min, during cyclic training, decreased up to the sixth week (100 ml → 83 ml) then increased 89.7 ml). Stroke volume for 750 kpm/min of work decreased during continuous training (100 ml → 85 ml). No stroke volume change occurred over the last six weeks at 900 kpm/min (86.5 ml).

Respiratory gas exchange - Figure 8b

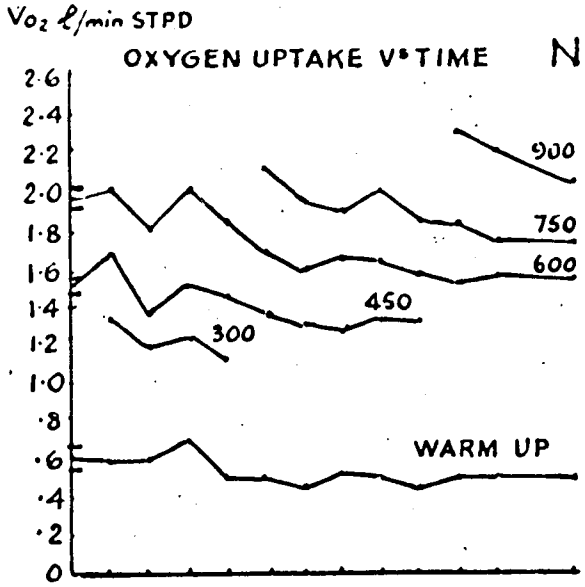
Ventilation during all testing work rates decreased. A large reduction occurred during the first six weeks for the 300, 450 and 600 kpm/min in this parameter. After this point a small but steady reduction continued to occur up to week twenty. A similar early large reduction in ventilation occurred for work rates of 750 kpm/min and 900 kpm/min when these were introduced during the test session as the subject became fitter. The same pattern of reduction was observed for $\dot{V}O_2$ and $\dot{V}CO_2$. After week twenty and values for both $\dot{V}O_2$ and $\dot{V}CO_2$ remained constant. Warm-up values for $\dot{V}O_2$ and $\dot{V}CO_2$ gradually decreased throughout training. Values for R decreased steadily up to week eight and then increased gradually up to week sixteen although this increase did not attain the initial value, after this a further reduction occurred up to week twenty.

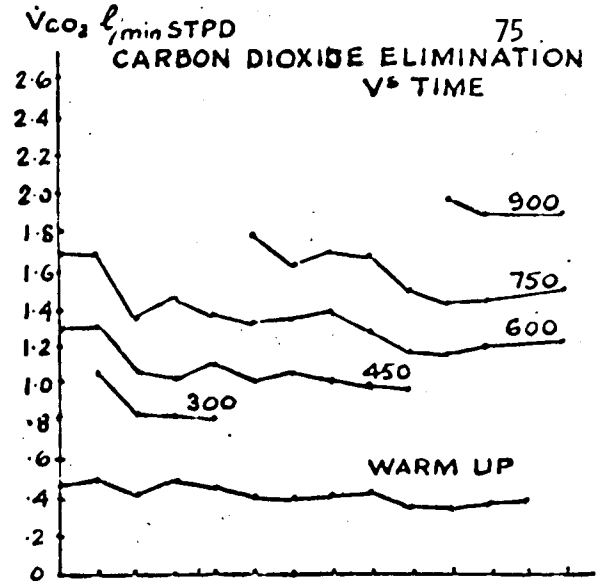
Exercise oxygen pulse and ventilation equivalent - Table 10

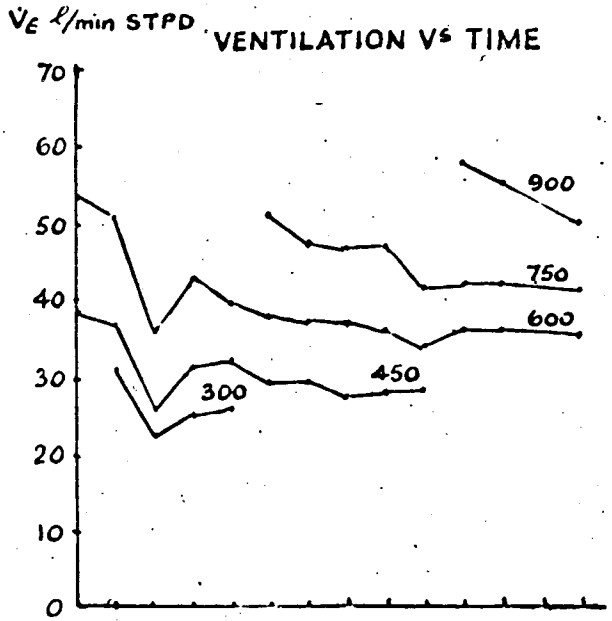
Oxygen pulse remained constant for a low work rate of 300 kpm/min

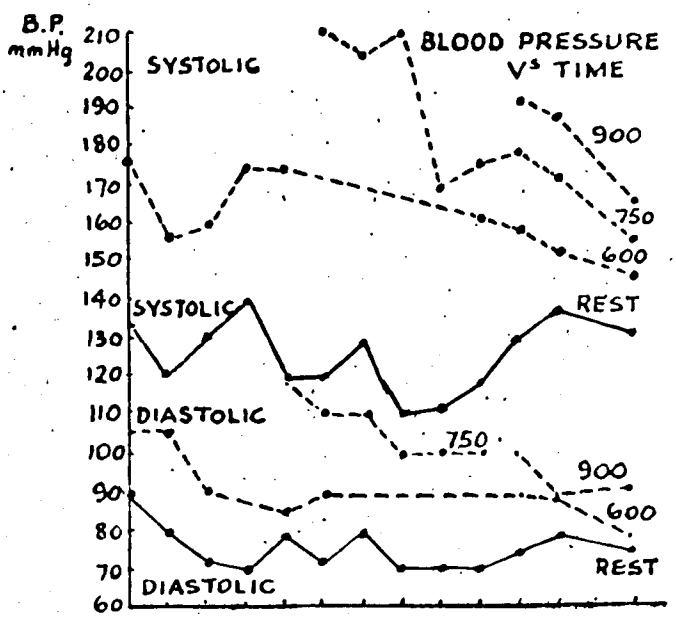
FIGURE 8b. Subject NS. Graphs of respiratory gas exchange, blood pressure and base excess for the 24 week rehabilitation period.

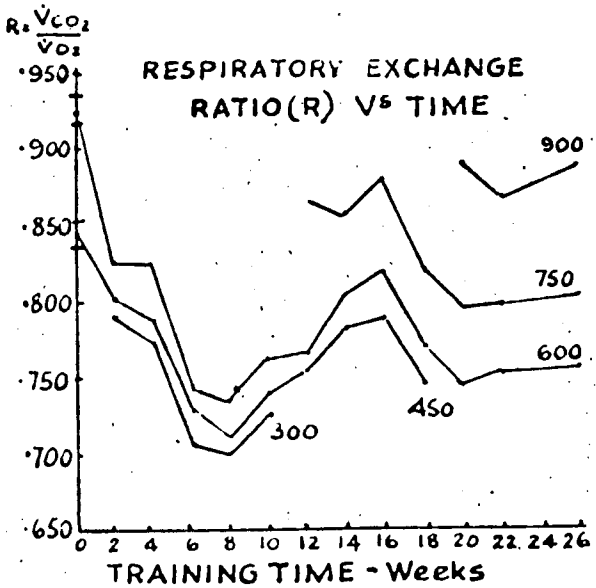
N. S.

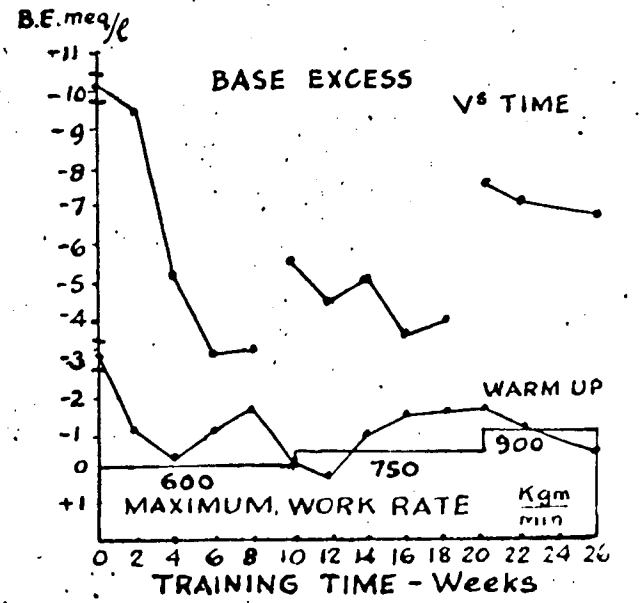

$\dot{V}_{O_2}$ ℓ/min STPD
OXYGEN UPTAKE Vs TIME
2·6
2·4
2·2
2·0
1·8
1·6
1·4
1·2
1·0
·8
·6
·4
·2
0
900
750
600
450
300
WARM UP


$\dot{V}_{CO_2}$ ℓ/min STPD
CARBON DIOXIDE ELIMINATION
Vs TIME
2·6
2·4
2·2
2·0
1·8
1·6
1·4
1·2
1·0
·8
·6
·4
·2
0
900
750
600
450
300
WARM UP


$\dot{V}_E$ ℓ/min STPD
VENTILATION Vs TIME
70
60
50
40
30
20
10
0
900
750
600
450
300


B.P. mmHg
BLOOD PRESSURE
Vs TIME
SYSTOLIC
DIASTOLIC
210
200
190
180
170
160
150
140
130
120
110
100
90
80
70
60
900
750
600
SYSTOLIC
REST
DIASTOLIC
750
900
600
REST


R = $\frac{\dot{V}_{CO_2}}{\dot{V}_{O_2}}$
RESPIRATORY EXCHANGE
RATIO(R) Vs TIME
·950
·900
·850
·800
·750
·700
·650
900
750
600
450
300
0 2 4 6 8 10 12 14 16 18 20 22 24 26
TRAINING TIME - Weeks


B.E. meq/ℓ
BASE EXCESS
Vs TIME
+11
-10
-9
-8
-7
-6
-5
-4
-3
-2
-1
0
+1
WARM UP
900
600
750
MAXIMUM WORK RATE
Kgm/min
0 2 4 6 8 10 12 14 16 18 20 22 24 26
TRAINING TIME - Weeks

however it increased for the rates of 450 and 600 kpm/min (0.0111 → 0.0130 l/beat, 450 kpm/min and 0.0118 → 0.0133 l/beat, 600 kpm/min). No change was observed with training over the tests at 750 kpm/min (average 0.0133 l/beat). Both exercise-ventilation and oxygen uptake decreased together in the same proportions resulting in no change in the ventilatory equivalent. The following mean values for the ventilatory equivalent at the various work rates were observed: 22± 0.5 at 300 kpm/min; 21± 1 at 450 kpm/min; 22± 1 at 600 kpm/min; 23± 0.25 at 750 kpm/min and 25± 0 at 900 kpm/min.

Base excess and lactate - Figure 8b and Table 7 and 12

Warm-up base excess and lactate values after training were lower than the initial values. A large reduction in exercise values was also observed. The final values for these parameters at 750 kpm/min (BE -3.7 meq/l, La 10.0 mg %) were similar to those originally measured at 600 kpm/min (BE -3.1 meq/l, La 27.0 mq %). Only a small reduction occurred at 900 kpm/min during the last six weeks of training.

Blood pressure - Figure 8b

Resting diastolic blood pressure decreased (90 mmHg → 75 mmHg) during training. Resting-systolic pressure, although variable, decreased up to week fourteen (135 mmHg → 110 mmHg). However, it then increased to the initial level. Exercise-diastolic and systolic pressure decreased for all work rates although the reduction of systolic pressure was not apparent until week fourteen. Diastolic pressure gradually reduced from the onset of training.

Myocardial oxygen demand - Table 14

The warm-up or resting value for myocardial oxygen demand generally decreased throughout the training period (13965 → 9782 mmHg X HR). The lowest

values occurred at week fourteen (8030 mmHg X HR) and from here on an increase was observed. All exercise values decreased with training (29568 → 24534 at 600 kpm/min; 33600 → 24150 mmHg X HR at 750 kpm/min; 29704 → 26726 at 900 kpm/min).

Lung Function - Figure 8a

Resting respiratory rate decreased slightly while resting tidal volume increased. An increase was seen in both vital capacity (4.00 → 4.50 l) and FEV_1 (3.00 → 3.20 l).

Body weight and serum lipids - Figure 8a

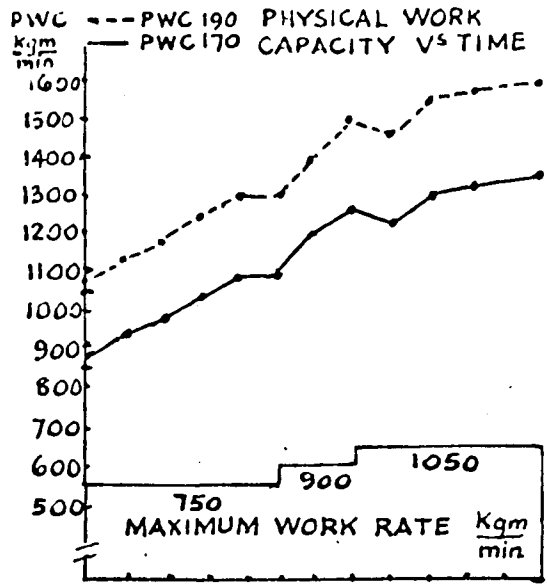
Cholesterol reduced (240 mg % → 205 mg %) while body weight remained constant throughout the training period. Serum triglycerides showed an early increase (103 mg % → 194 mg % and from this point a steady decrease occurred back to the initial value).

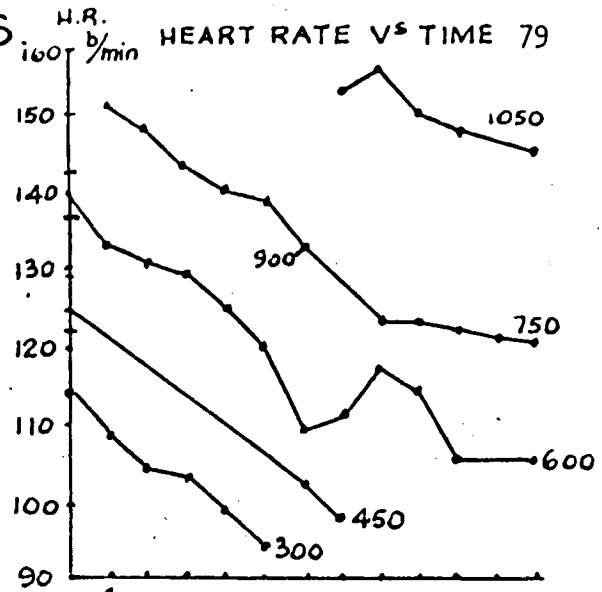
Subject WS Age 43 Height 179.7 cm Weight 69.6 kg (initial)
70.5 kg (final) Started training four months post infarction

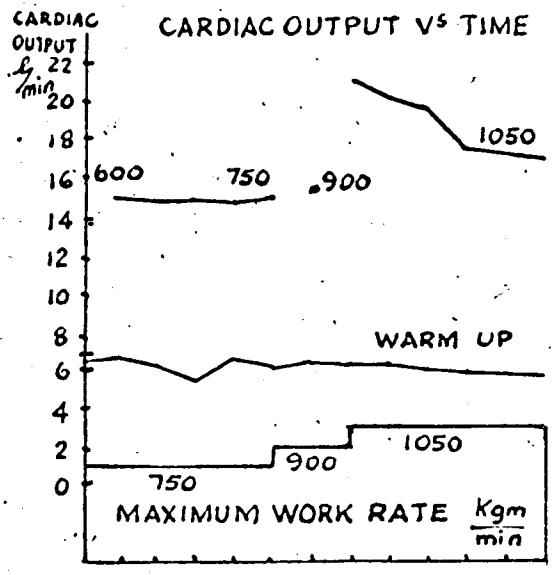
Physical work capacity - Figure 9a

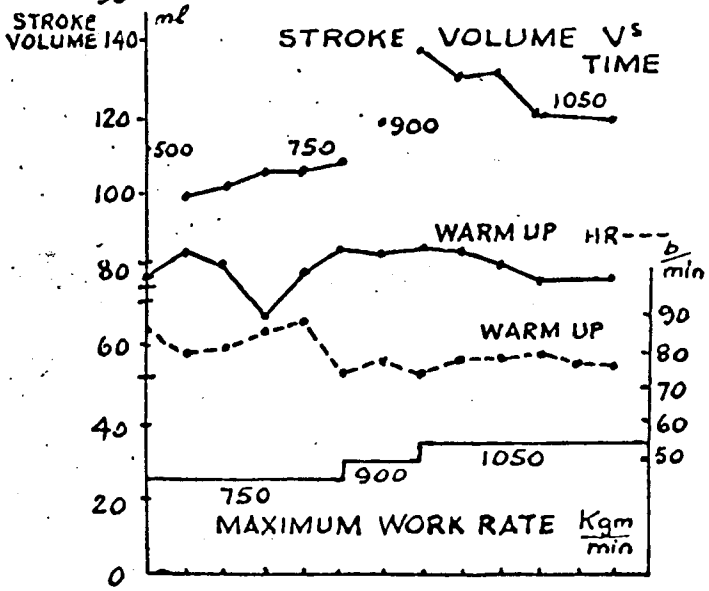
Figure 9a shows the subject's initial average PWC_{170} was 987 kpm/min and his PWC_{190} was 1097 kpm/min. Ten weeks of the cyclic ergometry increased his PWC_{170} by 22.6 per cent (1100 kpm/min) and his PWC_{190} by 17.5 per cent (1330 kpm/min). By the end of another ten weeks, after continuous training, his PWC_{170} had increased 48.9 per cent (1325 kpm/min) above the initial value and his PWC_{190} by 44.9 per cent (1565 kpm/min). The final month of continuous training produced further small increases in the PWC_{170} (1350 kpm/min) and PWC_{190} (1600 kpm/min).

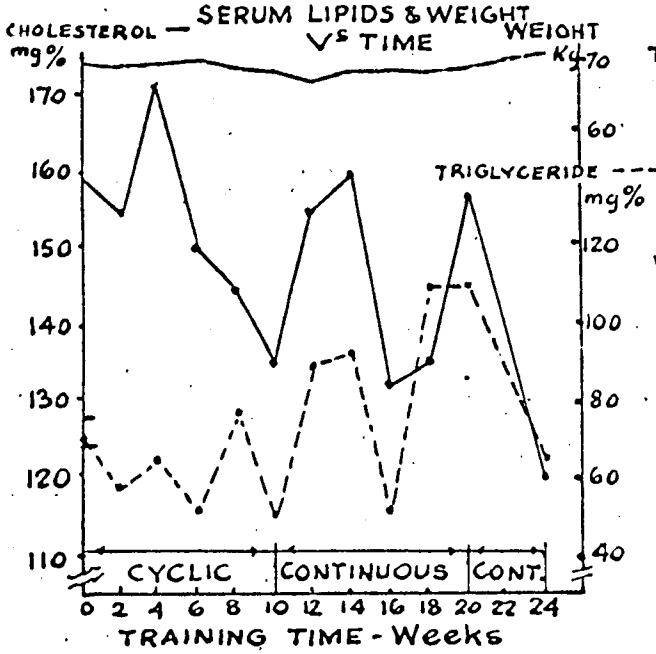
FIGURE 9a. Subject WS. Graphs of physical work capacity, heart rate, cardiac output, stroke volume, lung function, body weight and serum lipids for the 24 week rehabilitation period.

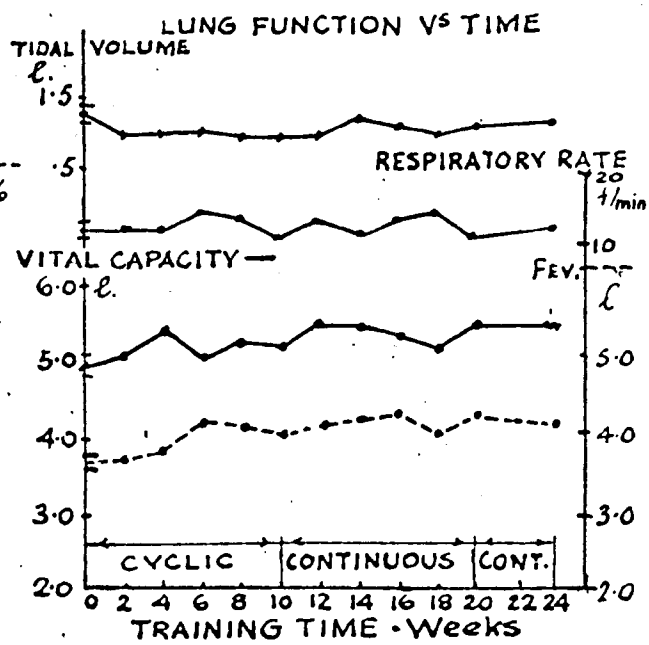

PWC ---PWC 190 PHYSICAL WORK
PWC 170 CAPACITY Vs TIME
Kgm/min
1600
1500
1400
1300
1200
1100
1000
900
800
700
600
500
750
900
1050
MAXIMUM WORK RATE Kgm/min


H.R. b/min
HEART RATE Vs TIME
160
150
140
130
120
110
100
90
1050
900
750
600
450
300


CARDIAC OUTPUT Vs TIME
CARDIAC OUTPUT l/min
22
20
18
16
14
12
10
8
6
4
2
0
600
750
900
1050
WARM UP
750
900
1050
MAXIMUM WORK RATE Kgm/min


STROKE VOLUME ml
STROKE VOLUME Vs TIME
140
120
100
80
60
40
20
0
500
750
900
1050
WARM UP HR---
b/min
90
80
70
60
50
WARM UP
750
900
1050
MAXIMUM WORK RATE Kgm/min


CHOLESTEROL mg%
SERUM LIPIDS & WEIGHT Vs TIME
WEIGHT Kg
170
160
150
140
130
120
110
70
60
TRIGLYCERIDE ---
mg%
120
100
80
60
40
CYCLIC
CONTINUOUS
CONT.
0 2 4 6 8 10 12 14 16 18 20 22 24
TRAINING TIME - Weeks


LUNG FUNCTION Vs TIME
TIDAL VOLUME
l.
1·5
·5
RESPIRATORY RATE
20
10
f/min
VITAL CAPACITY —
FEV. ---
6·0 l.
5·0
4·0
3·0
2·0
l
5·0
4·0
3·0
2·0
CYCLIC
CONTINUOUS
CONT.
0 2 4 6 8 10 12 14 16 18 20 22 24
TRAINING TIME - Weeks

Heart rate - Figure 9a

Training resulted in large reductions in the heart rate for equivalent work rates. During cyclic training the three work rates were 300, 600 and 750 kpm/min, however, once continuous training was begun this subject was able, for the same heart rate response, to accomplish work rates of 600, 750 and 1050 kpm/min, respectively. The final tests at 1050 kpm/min ended with a heart rate similar to that at the end of the initial 600 kpm/min test. Both warm-up and resting heart rates were also reduced through the training program.

This subject trained and was tested in the early parts of training with a number of multi-focal premature beats during exercise and recovery. However, by the middle of continuous training these had disappeared. He was also able to work throughout the day without experiencing angina which was present at the beginning of his training. Associated with this disappearance of angina was an increased sense of well being. At the sixteenth week test this subject was taken off medication for his ventricular instability.

Cardiac output and stroke volume - Figure 9a

Figure 9a shows a decrease in warm-up-cardiac output (6.59 l/min → 5.91 l/min) as a result of twenty-four weeks of training although no change was evident in warm-up-stroke volume. The cardiac output for the 750 kpm/min did not change during ten weeks of cyclic training. A large decrease was noted however in the cardiac output at 1050 kpm/min after continuous training (21.00 l/min → 16.39 l/min). The exercise-stroke volume after cyclic training increased at 750 kpm/min (99.5 ml → 108.5 ml), however, during the later portion of the program the stroke volume decreased considerably at 1050 kpm/min (137.1 ml → 118.5 ml).

Respiratory gas exchange - Figure 9b

Figure 9b shows a large reduction in ventilation during all work rates, especially at the higher loads 750 kpm/min (63 l/min → 46 l/min) and 1050 kpm/min (89 l/min → 65 l/min). Both $\dot{V}O_2$ and $\dot{V}CO_2$ decreased during the training period with the largest reductions occurring at the more strenuous work rates. Warm-up values for $\dot{V}O_2$ and $\dot{V}CO_2$ did not change appreciably. At the more strenuous work rates (600 and 750 kpm/min) the R values remained constant for a period of ten weeks (to the end of cyclic training) and then showed a large decrease. Only a small decrease in R value occurred during the ten week period when the maximum work rate was 1050 kpm/min. The R value for the 300 kpm/min load increased to week six and then remained constant at this increased level.

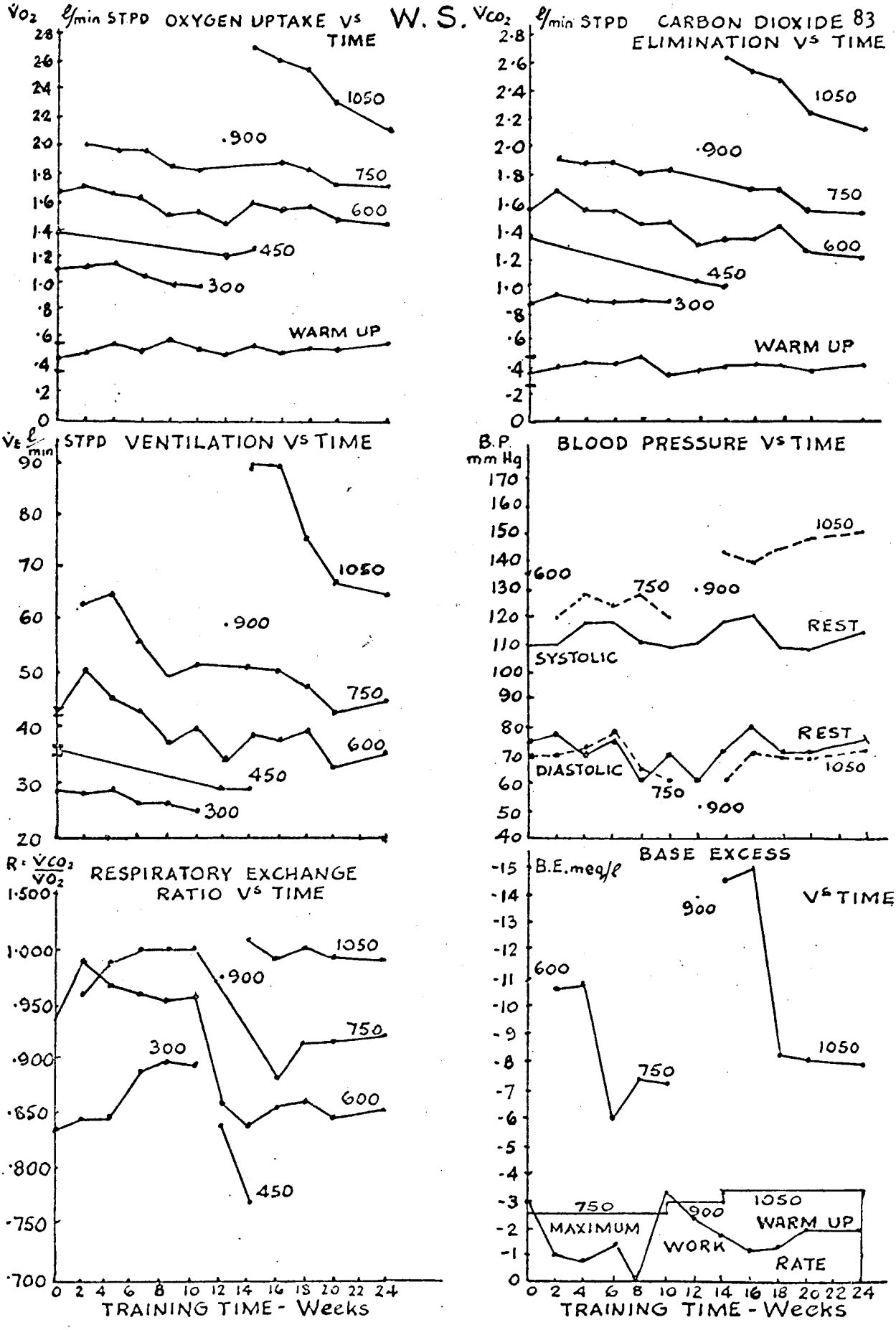
Exercise oxygen pulse and ventilation equivalent - Table 10

Oxygen pulse remained constant for a low work rate of 300 kpm/min however it increased for the rates of 450 and 600 kpm/min (0.0111 → 0.0130 l/beat, 450 kpm/min and 0.0118 → 0.0133 l/beat, 600 kpm/min). No change was observed with training over the tests at 750 kpm/min (average 0.0133 l/beat). Both exercise-ventilation and oxygen uptake decreased together in the same proportions resulting in no change in the ventilatory equivalent. The following mean values for the ventilatory equivalent at the various work rates were observed: 22± 0.5 at 300 kpm/min; 21± 1 at 450 kpm/min; 22± 1 at 600 kpm/min; 23± 0.25 at 750 kpm/min and 25± 0 at 900 kpm/min.

Base excess and lactate - Figure 9b and Table 7 and 12

Warm-up and resting values of base excess and lactate (BE -3.5 → -2.0 meq/l; La 8.8 → 7.0 mg %) decreased as did exercise values. The decrease in

FIGURE 9b. Subject WS. Graphs of respiratory gas exchange, blood pressure and base excess for the 24 week rehabilitation period.


W. S.
V̇O2 l/min STPD OXYGEN UPTAKE Vs TIME
V̇CO2 l/min STPD CARBON DIOXIDE ELIMINATION Vs TIME
V̇E l/min STPD VENTILATION Vs TIME
B.P. mm Hg BLOOD PRESSURE Vs TIME
R = V̇CO2/V̇O2 RESPIRATORY EXCHANGE RATIO Vs TIME
BASE EXCESS B.E. meq/l Vs TIME
1050
900
750
600
450
300
WARM UP
REST
SYSTOLIC
DIASTOLIC
MAXIMUM WORK RATE
TRAINING TIME - Weeks

exercise value occurred after four weeks at the particular load. Thereafter the level of metabolic acidosis showed only a small decrease with further training. The final exercise values for metabolic acidosis at 1050 kpm/min (BE -8.0 meq/l, La 19.0 mg %) were lower than those at 750 kpm/min ten weeks earlier (BE -10.7 meq/l, La 66.4 mg %).

Blood pressure - Figure 9b

Resting diastolic and systolic blood pressure were not consistently changed during the twenty-four week period. Exercise values also remained constant and in fact a small increase was evident in the 1050 kpm/min sessions.

Myocardial oxygen demand - Table 14

During the twenty-four week period of training a small increase occurred (7500 → 8300 mmHg X HR) in the warm-up-myocardial oxygen demand. An increase was observed up to week eight (7500 → 9460 mmHg X HR); a decrease followed this up to week twenty-four (9460 → 8300 mmHg X HR). Exercise values showed a decrease for 750 kpm/min (18120 → 15290 mmHg X HR) however a small increase occurred at 1050 kpm/min (21420 → 21750 mmHg X HR).

Lung function - Figure 9a

Resting tidal volume and respiratory rate did not show any change during the training period. A small increase was noted in vital capacity (4.91 l → 5.36 l) and in FEV_1 (3.62 l → 4.20 l).

Body weight and serum lipids - Figure 9a

Figure 9a shows that while his body weight remained constant, the serum cholesterol level decreased over the twenty-four week period (159 mg % → 120 mg %). Although this was not a steady reduction, these levels were

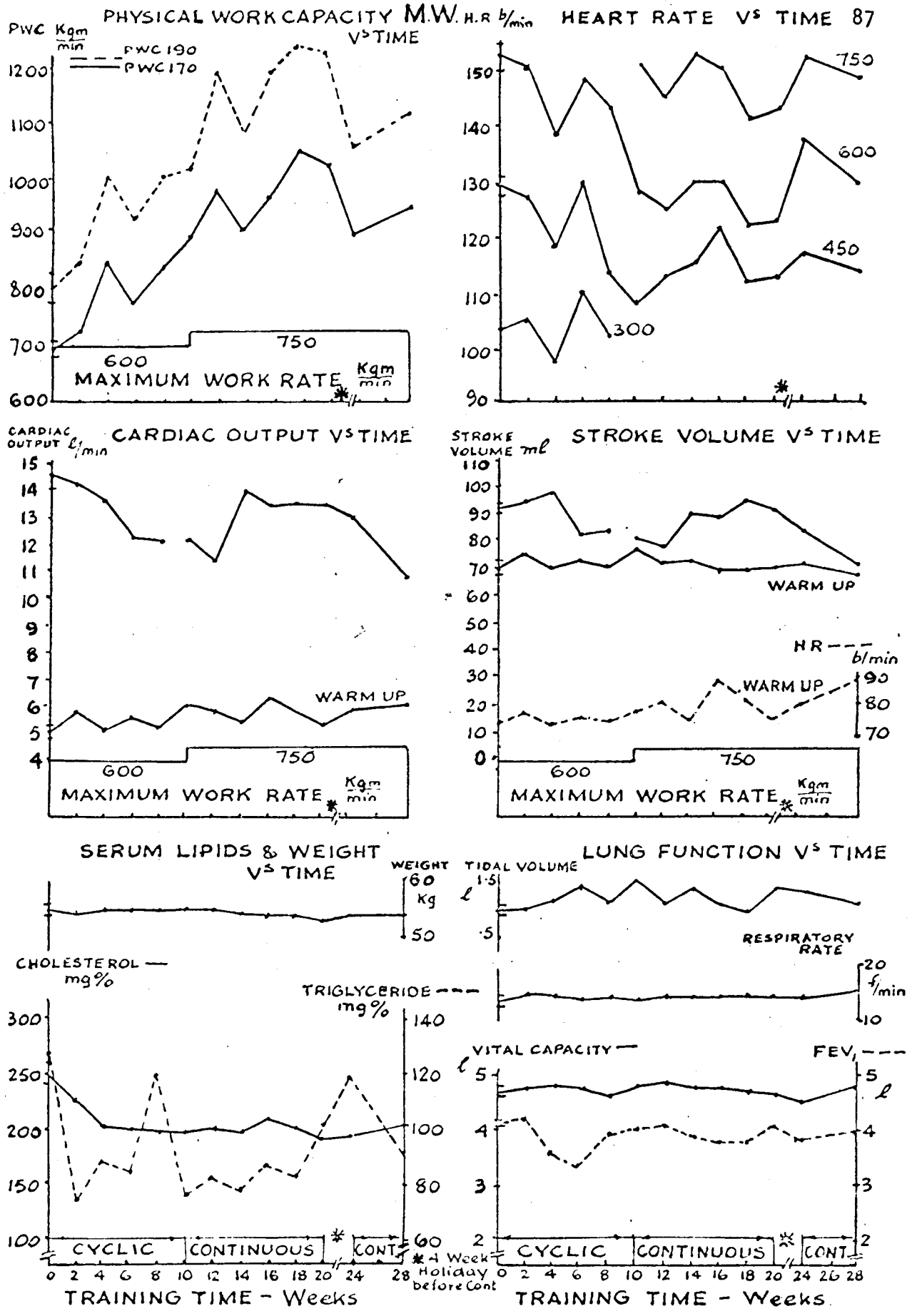
gradually reduced despite a low initial serum cholesterol level. The mean initial value for serum triglycerides was very low (71 mg %) and no discernible change occurred during the training period. Values ranged from 50 mg % to 110 mg % throughout this period with a final value of 64 mg %.

Subject MW Age 56 Height 174.3 cm Weight 54.4 kg (initial) 54.0 kg (final) Strated training four months post hospitalization. (This subject did not have a myocardial infarction however was hospitalized for coronary insufficiency)

Physical work capacity - Figure 10a

This subject was physically very weak and had considerable trouble completing the more strenuous work rates. After the ten weeks of cyclic ergometry training his PWC_{170} had increased 28.4 per cent (693 → 890 kpm/min) and his PWC_{190} had increased 26.3 per cent (802 → 1010 kpm/min). The change to continuous riding for thirty minutes was very difficult for him,however, after a period of four weeks he could complete it quite easily. By the end of the ten weeks of continuous training his PWC_{170} (1040 kpm/min) had increased a total of 50.1 per cent above the initial value and his PWC_{190} (1230 kpm/min) had increased by a total of 53.8 per cent. This subject was physically capable of work, however, he could not find employment. This was a concern to him and in fact he took an unsuccessful month's leave in an attempt to find employment. After this month's leave his work capacity had decreased to the level achieved after only ten weeks of training. The month of continuous ergometry did however increase his PWC to some extent. He has now, after another month of training, reached the twenty week level (not shown on the graph).

FIGURE 10a. Subject MW. Graphs of physical work capacity, heart rate, cardiac output, stroke volume, lung function, body weight and serum lipids for the 24 week rehabilitation period.


PHYSICAL WORK CAPACITY M.W. V^S TIME
PWC Kgm/min
PWC 190
PWC 170
1200
1100
1000
900
800
700
600
600
750
MAXIMUM WORK RATE Kgm/min
H.R b/min HEART RATE V^S TIME
150
140
130
120
110
100
90
750
600
450
300
CARDIAC OUTPUT V^S TIME
CARDIAC OUTPUT l/min
15
14
13
12
11
10
9
8
7
6
5
4
WARM UP
600
750
MAXIMUM WORK RATE Kgm/min
STROKE VOLUME V^S TIME
STROKE VOLUME ml
110
100
90
80
70
60
50
40
30
20
10
0
WARM UP
HR b/min
WARM UP
90
80
70
600
750
MAXIMUM WORK RATE Kgm/min
SERUM LIPIDS & WEIGHT V^S TIME
WEIGHT
60 Kg
50
CHOLESTEROL mg%
TRIGLYCERIDE mg%
300
250
200
150
100
140
120
100
80
60
CYCLIC
CONTINUOUS
CONT
0 2 4 6 8 10 12 14 16 18 20 24 28
TRAINING TIME - Weeks
* 4 Week Holiday before Cont
LUNG FUNCTION V^S TIME
TIDAL VOLUME
l 1·5
·5
RESPIRATORY RATE
20
f/min
10
VITAL CAPACITY
FEV1
l
5
4
3
2
CYCLIC
CONTINUOUS
CONT
0 2 4 6 8 10 12 14 16 18 20 24 26 28
TRAINING TIME - Weeks

Heart rate - Figure 10a

Figure 10a shows the uneven reduction of heart rate up to week twenty. The increase in heart rate for equivalent work that followed this period resulted from the four week break in training. The month of continuous training, that followed, again decreased in the heart rate at each testing work rate. During the ten week period of cyclic ergometry training the testing work rates were 300, 450 and 600 kpm/min. After continuous training the heart rate at these same work rates was not reached until they were now 450, 600 and 750 kpm/min, respectively. Warm-up heart rate was not changed.

This subject experienced periods of angina, usually in the morning or while walking, before the onset of the program and during the first twelve weeks of training, however, after this time the angina had disappeared. This was naturally of great benefit to him psychologically. During his hospitalization, four months prior to training, a coronary angiogram revealed "sparse central branches" and his coronary arteries showed some atherosclerotic changes.

Cardiac output and stroke volume - Figure 10a

Warm-up-cardiac output ranged from 5.10 l/min to 6.30 l/min during the twenty weeks of training. No consistent change was apparent. The mean initial value was 5.10 l/min and at week twenty it was 5.42 l/min. The last value at week twenty-four was 6.20 l/min. Warm-up-stroke volume remained constant (initial 70 ml → final 69 ml) and ranged between 69.0 ml and 73.5 ml. During cyclic training the cardiac output at 600 kpm/min decreased (14.26 l/min → 12.01 l/min). At 750 kpm/min the cardiac output increased (11.40 l/min → 13.97 l/min) then decreased slightly (13.97 l/min → 13.06 l/min) until the last month of continuous bicycle ergometry when a large decrease

was observed (13.06 l/min → 10.81 l/min) to a level below the first values for 750 kpm/min. Exercise-stroke volume did not increase significantly above the warm-up values. The stroke volume at 600 kpm/min decreased during the first eight week period (93.2 ml → 84 ml). The stroke volume at 750 kpm/min first showed a decrease then progressively increased up to week eighteen (78.7 ml → 96.1 ml). From this point a decrease occurred to below the first values obtained at this load (96.1 ml → 72.2 ml).

Respiratory gas exchange - Figure 10b

At all test work rates a decrease in ventilation took place until the subject's four week absence after week twenty. Exercise oxygen uptake values for 750 kpm/min decreased initially (2.40 l → 2.26 l) and then remained steady at 2.2 l. A small decrease occurred in $\dot{V}O_2$ values at both 450 and 600 kpm/min. Exercise-$\dot{V}CO_2$ values decreased at all work rates (about 0.2 l for all loads) during the period of training. Warm-up values for $\dot{V}O_2$ increased slightly over the initial values while $\dot{V}CO_2$ remained constant. The R values for the higher work rates of 600 and 750 kpm/min showed a decrease up to week twenty whereas no change was seen in R at the lower rates.

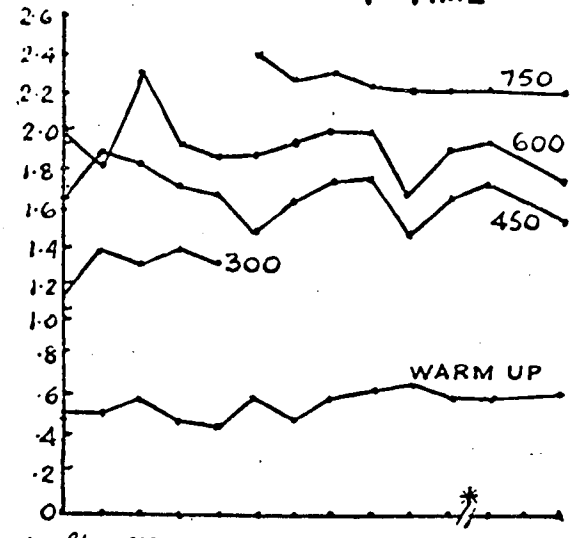
Exercise oxygen pulse and ventilation equivalent - Table 10

Neither of these parameters changed with training in tests at 300 and 450 kpm/min. A small increase in oxygen pulse after training occurred at the higher work rates of 600 and 750 kpm/min (0.0130 → 0.0166 l/beat at 600 kpm/min and 0.0151 → 0.0172 l/beat at 750 kpm/min). Ventilation equivalent decreased during these tests from 30 to 24 at 600 kpm/min and from 34 to 27 at 750 kpm/min. After four weeks absence from training an increased ventilation equivalent and decreased oxygen pulse was observed.

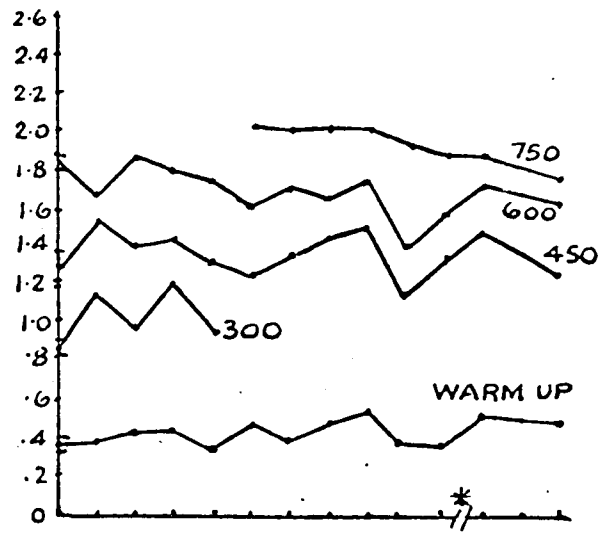
FIGURE 10b. Subject MW. Graphs of respiratory gas exchange, blood pressure and base excess for the 24 week rehabilitation period.

M.W.

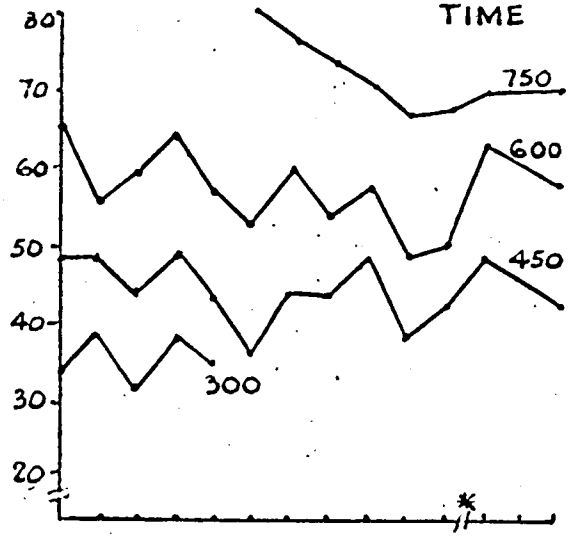
$\dot{V}O_2$ ℓ/min STPD. OXYGEN UPTAKE V^s TIME



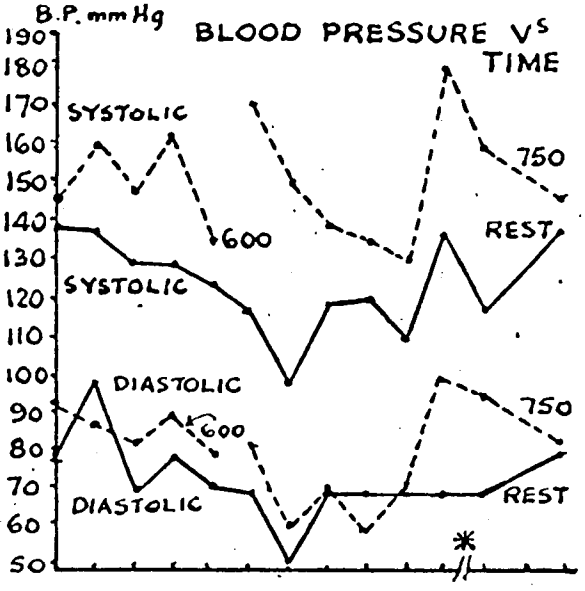
$\dot{V}CO_2$ ℓ/min STPD. CARBON DIOXIDE ELIMINATION V^s TIME



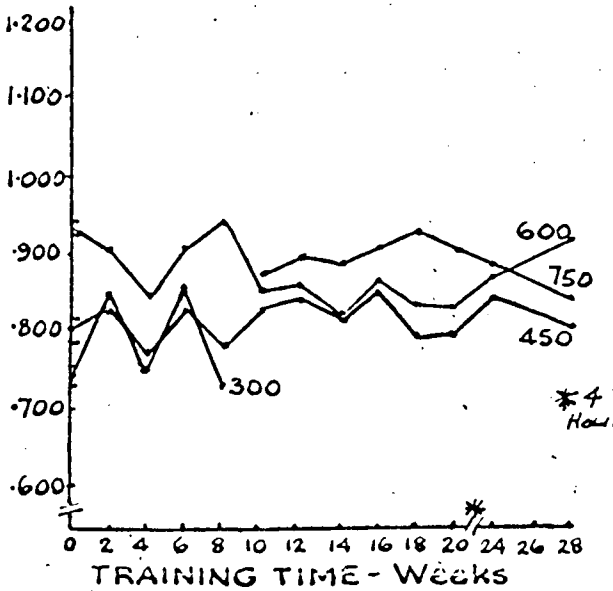
$\dot{V}E$ ℓ/min STPD VENTILATION V^s TIME



B.P. mm Hg BLOOD PRESSURE V^s TIME

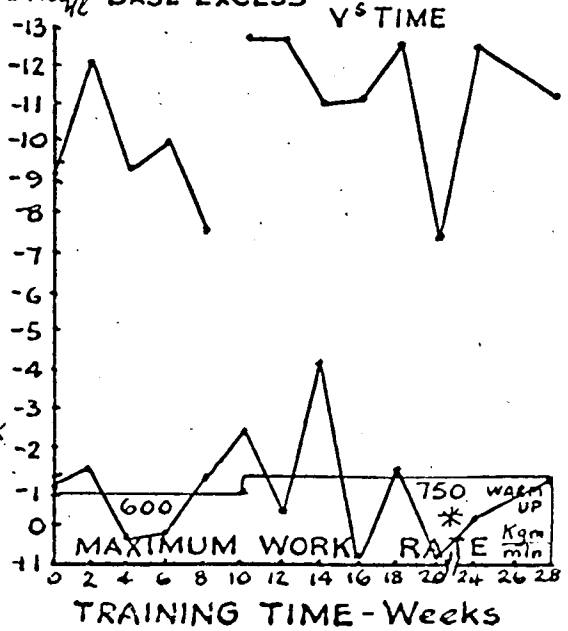


$R = \frac{\dot{V}CO_2}{\dot{V}O_2}$ RESPIRATORY EXCHANGE RATIO V^s TIME



TRAINING TIME - Weeks

BE meq/ℓ BASE EXCESS V^s TIME



* 4 WEEK HOLIDAY

TRAINING TIME - Weeks

Base excess and lactate - Figure 10b and Table 7 and 12

No consistent changes occurred in resting or warm-up levels in these parameters however training resulted in lower exercise levels of metabolic acidosis. The largest decrease occurred at the end of continuous training (BE -12.5 → 7.6 meq/l, at 750 kpm/min). The level increased after the four week absence from training. However, training again decreased the acidosis (BE -12.5 → -11.2 meq/l at 750 kpm/min). During cyclic training a smaller reduction in the post-exercise acidosis level was observed (BE -9.1 → -7.8 meq/l, 600 kpm/min).

Blood pressure - Figure 10b

Resting levels of blood pressure, both systolic and diastolic decreased during the twenty week training period. Exercise-systolic values decreased during training. The most continuous decrease occurred during continuous training. A small reduction in exercise-diastolic pressure was also observed.

Myocardial oxygen demand - Table 14

The myocardial oxygen demand or tension-time index during warm-up decreased up to week eighteen (10220 → 9000 mmHg X HR) and then increased again to the initial value. During the eight weeks that 600 kpm/min was the maximum work rate the myocardial oxygen demand decreased (22500 → 19350 mmHg X HR). The value for 750 kpm/min decreased up to week eighteen (25800 → 18330 mmHg X HR). The additional month of continuous training after four weeks of absence produced another decrease (24480 → 22050 mmHg X HR, 750 kpm/min).

Lung function - Figure 10a

This subject did not show any change in any of the lung function

parameters shown in figure 10a.

Body weight and serum lipids - Figure 10a

Body weight remained unchanged although serum cholesterol decreased up to the fourth week of training. It then remained at this level throughout the remaining twenty weeks (250 mg % → 177 mg %). A small increase occurred in this parameter as a result of four weeks absence from training. Serum triglyceride levels were "normal" when he started training (126 mg %) and a decrease to 80 mg % occurred by the end of continuous training. After four weeks absence from training the triglyceride level increased once more to the initial level and decreased again with the resumption of training.

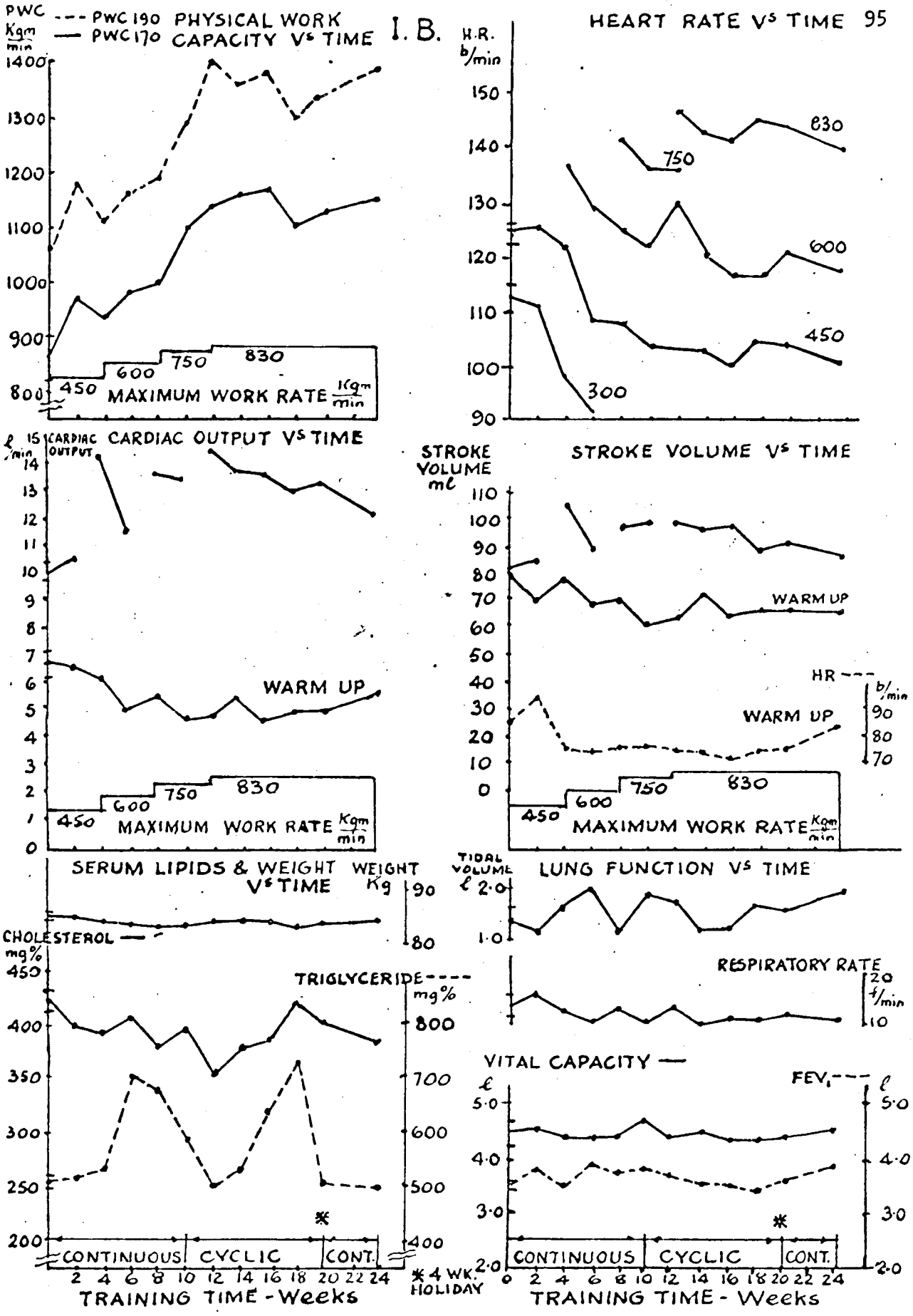
Group 2: Training: Continuous Ergometry (ten weeks), Cyclic Ergometry (ten weeks), Continuous Ergometry (four weeks); five times per week, thirty minutes per day.

Subject IB Age 45 years Height 176.5 cm Weight 84.6 kg (initial) 83.5 kg (final) Started training four months post infarction

Physical work capacity - Figure 11a

This subject had a mean initial PWC_{170} of 860 kpm/min and a PWC_{190} of 1055 kpm/min. After the ten weeks of continuous bicycle ergometry training his PWC_{170} increased to 1100 kpm/min (27.9 per cent above initial value and his PWC_{190} increased to 1290 kpm/min (22.3 per cent). The change to the cyclic training resulted in only a marginal increase in his work capacity. The rate of improvement declined and in fact, his work capacity decreased at the eighteenth week. By week twenty his PWC_{170} had increased a total of 30.8 per cent above the initial value (425 kpm/min) and his PWC_{190} had increased 27.0 per cent (1340 kpm/min). After a four week absence from

FIGURE 11a. Subject IB. Graphs of physical work capacity, heart rate, cardiac output, stroke volume, lung function, body weight and serum lipids for the 24 week rehabilitation period.


PWC --- PWC 190 PHYSICAL WORK
Kgm/min — PWC 170 CAPACITY Vs TIME
I. B.
MAXIMUM WORK RATE Kgm/min
450
600
750
830
HEART RATE Vs TIME
H.R. b/min
CARDIAC OUTPUT Vs TIME
CARDIAC OUTPUT
WARM UP
STROKE VOLUME Vs TIME
STROKE VOLUME ml
HR --- b/min
SERUM LIPIDS & WEIGHT Vs TIME
WEIGHT Kg
CHOLESTEROL — mg%
TRIGLYCERIDE ---- mg%
LUNG FUNCTION Vs TIME
TIDAL VOLUME ℓ
RESPIRATORY RATE f/min
VITAL CAPACITY — ℓ
FEV1 --- ℓ
CONTINUOUS
CYCLIC
CONT.
* 4 WK. HOLIDAY
TRAINING TIME - Weeks

training a change back to continuous training increased his PWC_{170} to 1150 kpm/min (total increase above initial 33.7 per cent) and his PWC_{190} to 1360 kpm/min (29 per cent).

Heart rate - Figure 11a

Three exercise tests at 300, 450 and 600 kpm/min, later conducted at 450, 600 and 750 kpm/min, after continuous training, indicated a comparatively favorable response to the latter type of training by a decrease of heart rate observed in all the subjects of Group 1. During cyclic training this rate of reduction in heart rate was substantially decreased and in fact an increase was observed in this parameter after week sixteen. During this period the subject could successfully complete a final six minutes of exercise at 830 kpm/min compared to 750 kpm/min previously. There was no decrement in the heart rate at this level of exercise. However, the final month of continuous training lowered the heart rate response to exercise at this work rate. Warm-up heart rate, shown in Figure 11a, showed a steady decline (85 → 77 b/min).

Cardiac output and stroke volume - Figure 11a

Warm-up cardiac output measured during continuous training (ten weeks) decreased greatly (6.71 → 4.70 l/min). No further decrease occurred after this. After the four week absence from training and following a months continuous training a small increase was observed (5.00 → 5.62 l/min). The stroke volume, during the warm-up phase of testing, decreased up to week ten (79.0 → 61.1 ml). A small increase occurred after ten weeks further training by bicycle ergometry (61.1 → 66.0 ml). Exercise-cardiac output and stroke volume values showed a decrease throughout the training

program. The largest increase in cardiac output at the 830 kpm/min occurred after the final month of training by continuous training (13.30 l/min → 12.16 l/min).

Respiratory gas exchange - Figure 11b

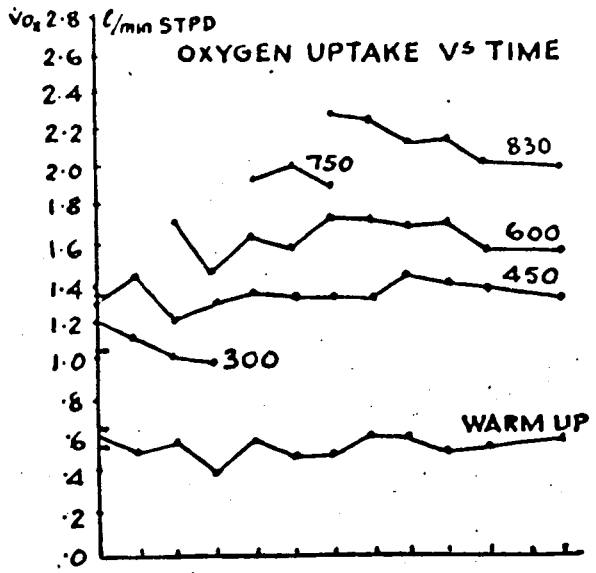
Exercise-ventilation showed an early large decrease for all work rates (300, 450 and 600 kpm/min) and then showed a slight decrease up to the middle of cyclic training when an increase occurred. The final month of continuous training resulted in a large decrease in ventilation for all work rates (450, 600 and 830 kpm/min). Exercise values for $\dot{V}O_2$ and $\dot{V}CO_2$ decreased again following the pattern shown with the ventilation. Carbon dioxide elimination for any particular work rate showed the largest decrease (approximately 0.25 l for any load). Values for the respiratory exchange ratio decreased up to week fourteen and then increased until the start of the final month of continuous training when a decrease occurred.

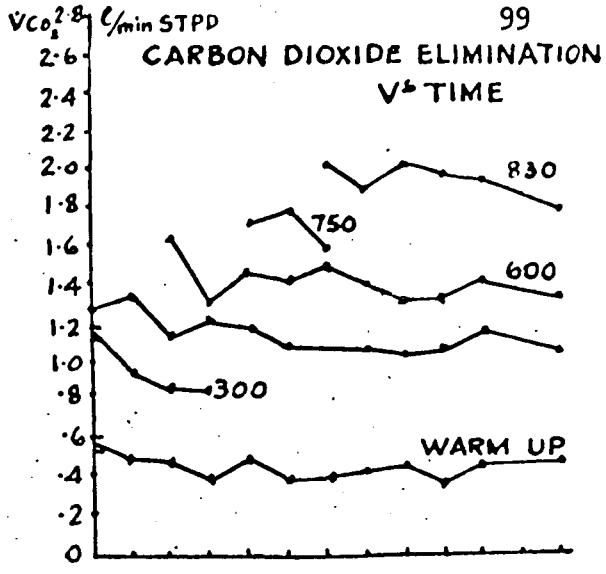
Exercise oxygen pulse and ventilation equivalent - Table 10

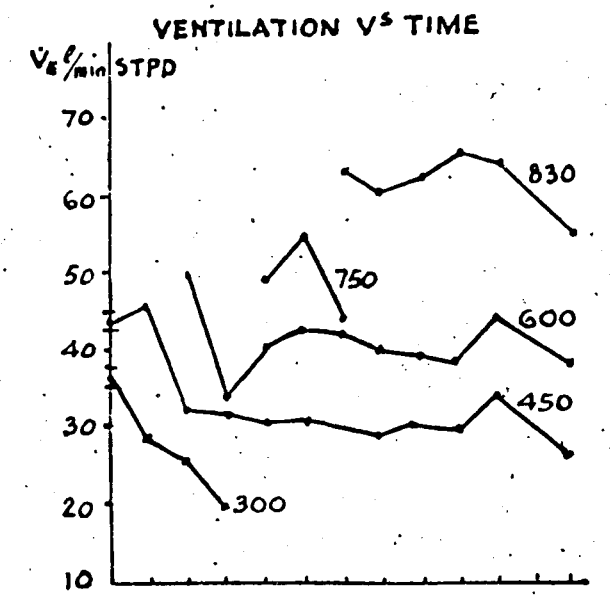
During tests at 300 kpm/min the oxygen pulse remained unchanged (0.0103 l/beat) whereas the ventilation equivalent decreased considerably (33 → 23). Oxygen pulse increased with training during work rates of 450 and 600 kpm/min (0.0106 → 0.0133 l/beat at 450 kpm/min and 0.0113 → 0.0149 l/beat at 600 kpm/min). Ventilation equivalent decreased considerably at 450 kpm/min (35 → 21) but was unchanged at 600 kpm/min. At the highest work rate of 830 kpm/min the oxygen pulse gradually decreased (0.0160 → 0.0145 l/beat) and ventilation equivalent slightly increased (27 → 30). After not training for a period, the O_2 pulse increased to 0.0162 l/beat, the level of the initial test, and the ventilation equivalent decreased to 25. Thus, absence from training in

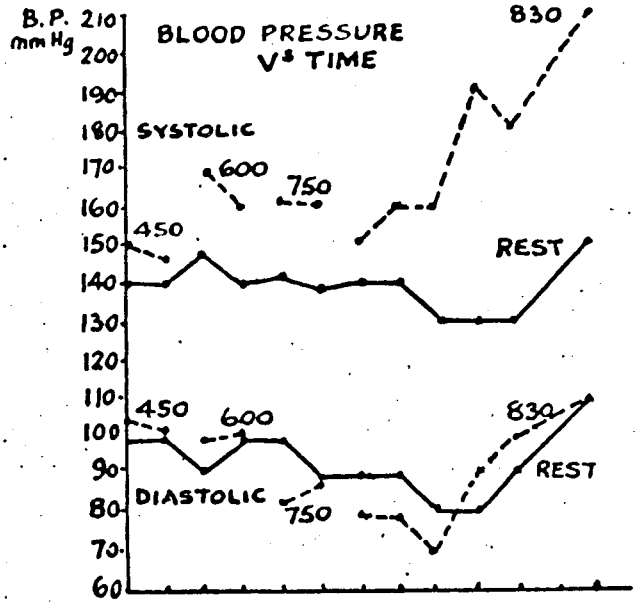
FIGURE 11b. Subject 1B. Graphs of respiratory gas exchange, blood pressure and base excess for the 24 week rehabilitation period.

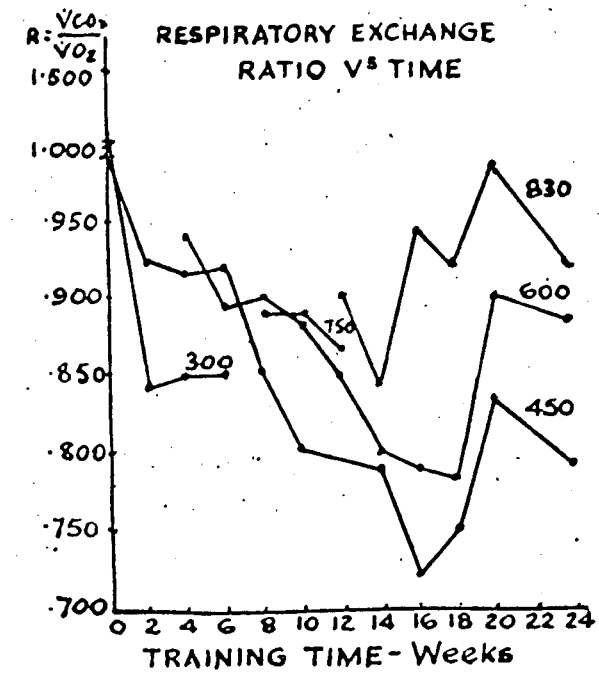
I.B.

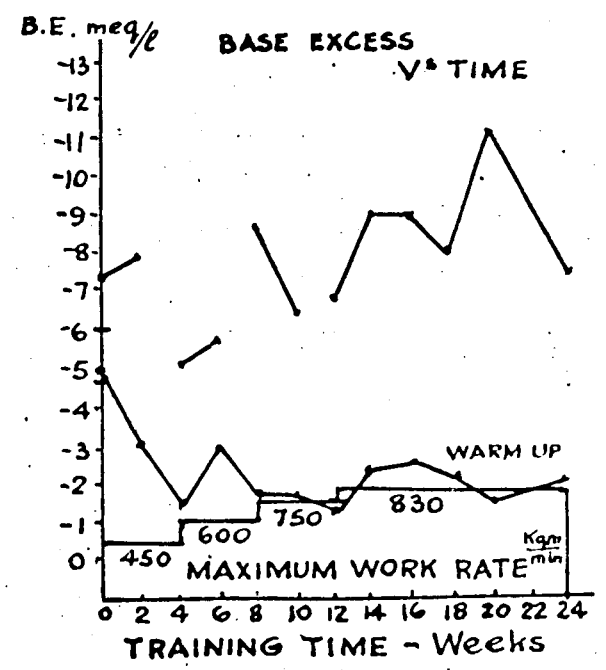

$\dot{V}O_2$ 2·8 ℓ/min STPD
OXYGEN UPTAKE Vs TIME
2·6
2·4
2·2
2·0
1·8
1·6
1·4
1·2
1·0
·8
·6
·4
·2
·0
830
750
600
450
300
WARM UP


$\dot{V}CO_2$ 2·8 ℓ/min STPD
CARBON DIOXIDE ELIMINATION
Vs TIME
2·6
2·4
2·2
2·0
1·8
1·6
1·4
1·2
1·0
·8
·6
·4
·2
0
830
750
600
300
WARM UP


VENTILATION Vs TIME
$\dot{V}_E$ ℓ/min STPD
70
60
50
40
30
20
10
830
750
600
450
300


B.P. mm Hg
BLOOD PRESSURE
Vs TIME
210
200
190
180
170
160
150
140
130
120
110
100
90
80
70
60
SYSTOLIC
830
600
750
450
REST
450
600
830
DIASTOLIC
750
REST


R: $\frac{\dot{V}CO_2}{\dot{V}O_2}$
RESPIRATORY EXCHANGE
RATIO Vs TIME
1·500
1·000
·950
·900
·850
·800
·750
·700
830
600
450
300
750
0 2 4 6 8 10 12 14 16 18 20 22 24
TRAINING TIME - Weeks


B.E. meq/ℓ
BASE EXCESS
Vs TIME
-13
-12
-11
-10
-9
-8
-7
-6
-5
-4
-3
-2
-1
0
WARM UP
830
750
600
450
Kgm/min
MAXIMUM WORK RATE
0 2 4 6 8 10 12 14 16 18 20 22 24
TRAINING TIME - Weeks

this subject resulted in less oxygen extracted per heart beat and increased ventilation per unit of oxygen taken up at all work rates.

Base excess and lactate - Figure 11b and Tables 7 and 12

Warm-up base excess and lactate values decreased with training (BE -5.0 → -2.0 meq/l, La 11.6 → 5.0 mg %) as compared to the initial tests. Exercise values did not change until the last weeks of the continuous training method. An increase occurred during ten weeks of the cyclic training and a large decrease occurred during the last month of the continuous training.

Blood pressure - Figure 11b

Resting systolic pressure remained constant (140 mmHg) up to week fourteen and then decreased (130 mmHg). After four weeks absence from training the systolic pressure had increased to 160 mmHg. Resting diastolic pressure gradually decreased up to week eighteen (100 mmHg → 80 mmHg), however, this increased to 130 mmHg after absence from training. Exercise values for both diastolic and systolic blood pressure remained constant or showed a small decrease during the period of continuous training, however, systolic blood pressure increased during cyclic training. During the final month of continuous training a slight further increase in blood pressure occurred.

Myocardial oxygen demand - Table 14

Warm-up values for myocardial oxygen demand decreased up to the twentieth week (11900 → 9424 mmHg X HR). After not training for a period this value increased to 11840 mmHg X HR and increased again after the final month of continuous training (12750 mmHg X HR). Exercise values first remained constant and then decreased during the ten weeks period of continuous training (19775 → 21250 mmHg X HR at 450 kpm/min; 23120 → 20640 mmHg X HR at 600 kpm/min

and 22842 → 21120 mmHg X HR at 750 kpm/min). An increase occurred throughout the period of cyclic training (22050 → 25920 mmHg X HR, 830 kpm/min). After no training this increased to 27354 mmHg X HR (830 kpm/min) and a further increase occurred during the final month of training (30240 mmHg X HR).

Lung function - Figure 11a

Resting tidal volume increased during the last part of his training while the resting respiratory rate decreased slightly. Vital capacity did not change (4.65 l) however his FEV_1 showed an increase (3.5 l → 3.93 l).

Body weight and serum lipids - Figure 11a

Body weight showed a small decrease (84.6 kg → 83.5 kg). The serum cholesterol values were initially very high (420 mg %) and after twelve weeks of training this had decreased to 351 mg %, however, after another six weeks of cyclic training this had increased to the initial level (420 mg %). During the final month of training this value decreased again (385 mg %). Serum triglyceride levels did not change over the twenty-four week period (500 mg %); small increases occurred during this time.

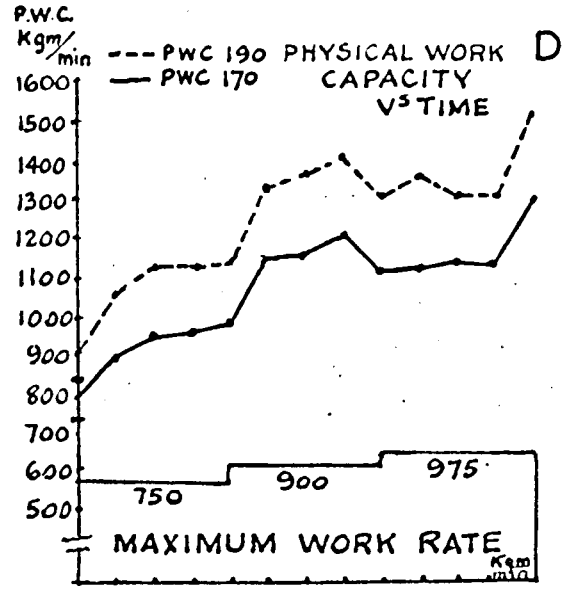
Subject DB Age 34 Height 184.6 cm Weight 80.3 kg (initial) 68.7 kg (final) Started training seven months post infarction

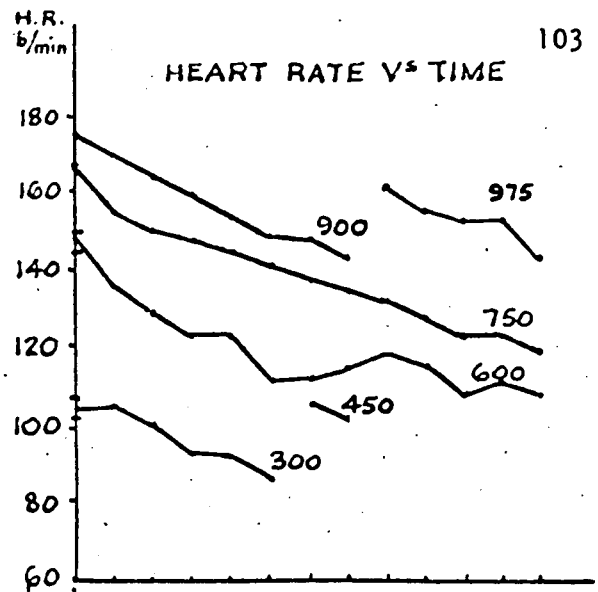
Physical work capacity - Figure 12a

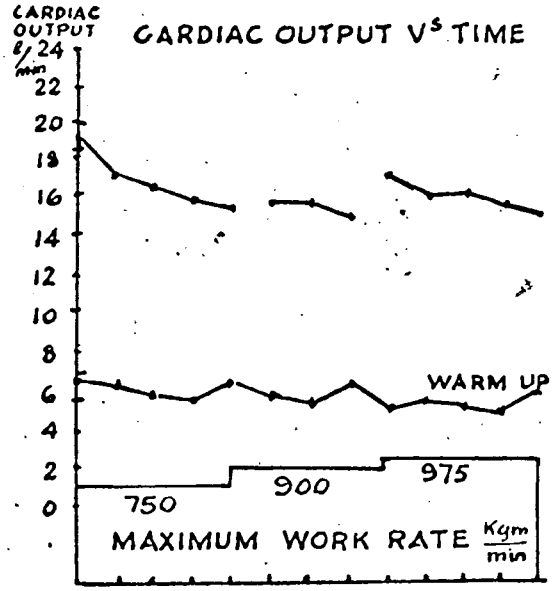
The mean initial PWC_{170} was 760 kpm/min and the PWC_{190} was 900 kpm/min. After ten weeks of continuous training his PWC_{170} increased 50 per cent (1140 kpm/min) and his PWC_{190} increased 46.7 per cent (1320 kpm/min). The period of cyclic training slightly decreased both his PWC_{170} to 1125 kpm/min and his PWC_{190} to 1300 kpm/min. During the final month of continuous training

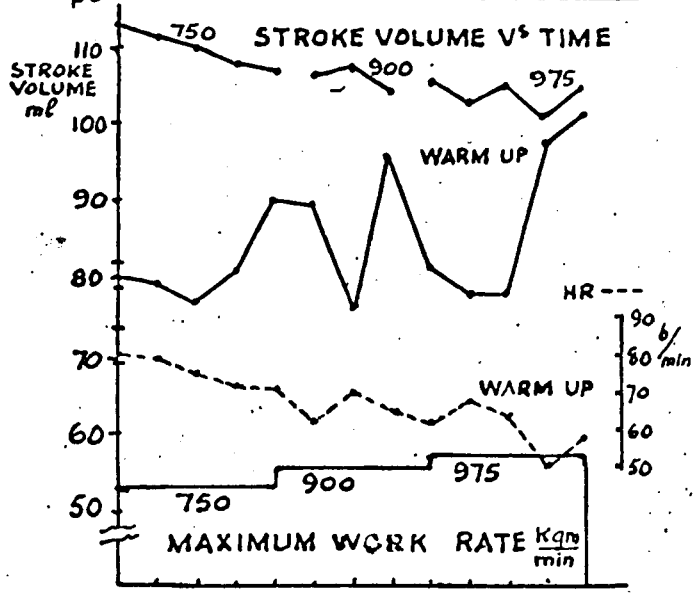
FIGURE 12a. Subject DB. Graphs of physical work capacity, heart rate, cardiac output, stroke volume, lung function, body weight and serum lipids for the 24 week rehabilitation period.

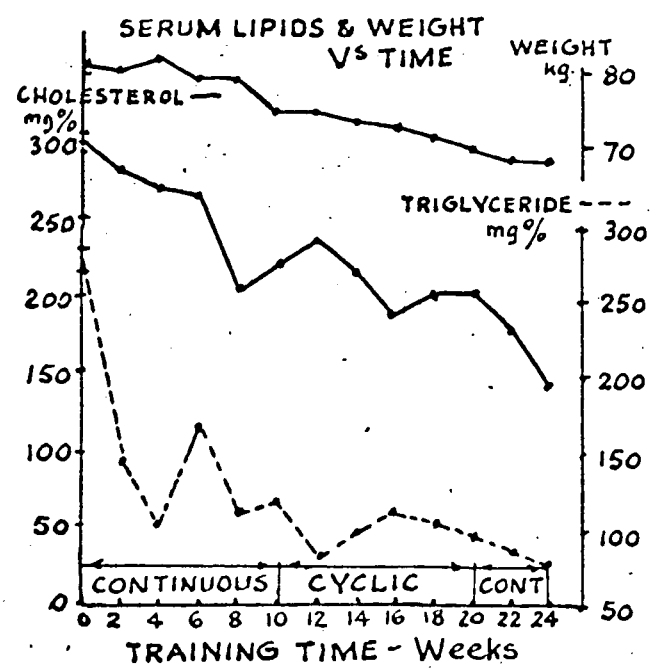
D.B.

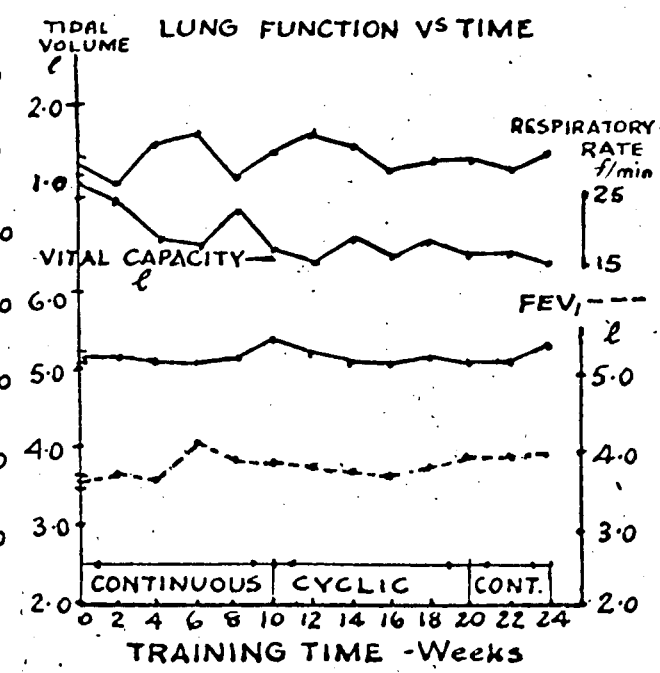

P.W.C.
Kgm/min
--- PWC 190
— PWC 170
PHYSICAL WORK CAPACITY Vs TIME
1600
1500
1400
1300
1200
1100
1000
900
800
700
600
500
750
900
975
MAXIMUM WORK RATE Kgm/min


H.R.
b/min
HEART RATE Vs TIME
180
160
140
120
100
80
60
975
900
750
600
450
300


CARDIAC OUTPUT
l/24 min
CARDIAC OUTPUT Vs TIME
22
20
18
16
14
12
10
8
6
4
2
0
WARM UP
750
900
975
MAXIMUM WORK RATE Kgm/min


750
STROKE VOLUME Vs TIME
900
975
STROKE VOLUME ml
110
100
90
80
70
60
50
WARM UP
HR ---
90
80
70
60
50
b/min
WARM UP
750
900
975
MAXIMUM WORK RATE Kgm/min


SERUM LIPIDS & WEIGHT Vs TIME
WEIGHT kg.
80
70
CHOLESTEROL mg%
300
250
200
150
100
50
0
TRIGLYCERIDE --- mg%
300
250
200
150
100
50
CONTINUOUS
CYCLIC
CONT
0 2 4 6 8 10 12 14 16 18 20 22 24
TRAINING TIME - Weeks


TIDAL VOLUME l
LUNG FUNCTION Vs TIME
2·0
1·0
RESPIRATORY RATE f/min
25
15
VITAL CAPACITY — l
FEV1 --- l
6·0
5·0
4·0
3·0
2·0
5·0
4·0
3·0
2·0
CONTINUOUS
CYCLIC
CONT.
0 2 4 6 8 10 12 14 16 18 20 22 24
TRAINING TIME -Weeks

a total increase above the initial PWC_{170} (1290 kpm/min) value of 69.8 per cent and 67.8 per cent above the initial PWC_{190} value (1510 kpm/min) was observed.

Heart rate - Figure 12a

The decrease of the test heart rate for all work loads is shown in Figure 12a. The rate of decrease is larger in the first ten weeks of training. During cyclic training only a small improvement was noted and in fact the heart rate increased at 600 kpm/min. During the final month of continuous training an improvement occurred especially in the heart rate response at the higher work rate of 975 kpm/min. The test work rates during the first ten weeks (continuous training) were 300, 600 and 750 kpm/min and later 450, 600 and 900 kpm/min. During the period of cyclic training these increased to 600, 750 and 975 kpm/min, respectively. Warm-up and resting heart rate decreased considerably over the twenty-four week period (warm-up 80 → 50 b/min).

Prior to the start of training this subject had coronary angiograms taken which revealed a thrombosis of one of the secondary branches of the left coronary artery. Post training angiograms were to be performed on this subject, however, the hospital board would not permit them to be done because of the possible risk. His increased work capacity and heart rate response plus his weight loss has had a pronounced effect on his outlook on life.

Cardiac output and stroke volume - Figure 12a

Warm-up-cardiac output decreased up to week twelve (6.44 → 5.23 l/min) and then remained at this level. Warm-up-stroke volume remained steady until week six, then increased together with decreasing heart rate (80.5 → 101 ml, week twenty-four). Exercise-cardiac output decreased throughout the twenty-

four week period with the largest change occurring during the periods of the continuous type of training (19.25 → 15.51 l/min at 750 kpm/min; 15.85 → 14.95 l/min at 900 kpm/min and 17.04 l/min at 975 kpm/min). At the end of the program the exercise-cardiac output for the 975 kpm/min load was lower than the 750 kpm/min load after eight weeks of training. Exercise-stroke volume showed a small decrease at all loads, again the most consistent changes occurred during the continuous period although the loads were not as strenuous (750 as compared with 900 kpm/min).

Respiratory gas exchange - Figure 12b

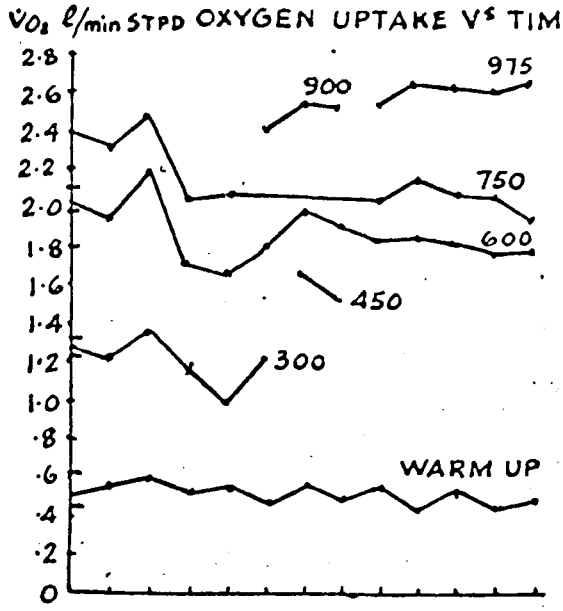
Ventilation decreased for all work rates throughout the training period. The largest decrements occurred early in training (weeks zero to ten). Exercise values for $\dot{V}O_2$ and $\dot{V}CO_2$ during these tests decreased up to week sixteen and remained constant thereafter until the last month of the continuous training when a further small decrease occurred at 600 and 750 kpm/min. The overall decrease in $\dot{V}CO_2$ at 600 and 750 kpm/min was 0.45 l/min. Associated with these changes in $\dot{V}O_2$ and $\dot{V}CO_2$ was a decrease in the exercise-R values until week eighteen when an increase occurred. At every work rate the R value again decreased during the final month of continuous training.

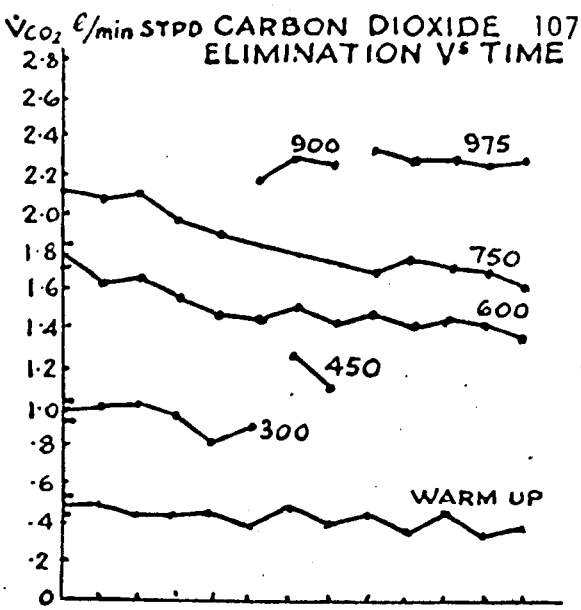
Exercise oxygen pulse and ventilation equivalent - Table 10

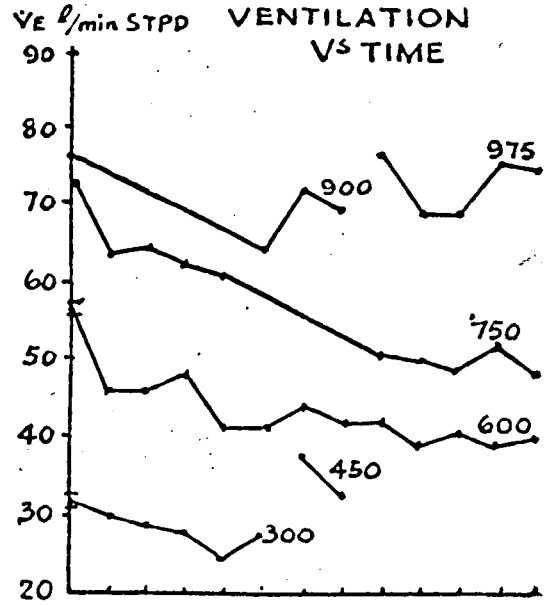
Pulmonary ventilation, oxygen uptake and heart rate, all decreased for a standard work rate with training. The ratios of oxygen pulse and ventilation equivalent show how ventilation and heart rate relate to oxygen uptake. For this subject, training resulted in a considerable increase in oxygen pulse at all work rates used in his physiological evaluation. Initial and final values for oxygen pulse at specific work rates were: 0.0121 → 0.0164 l/beat, at 300

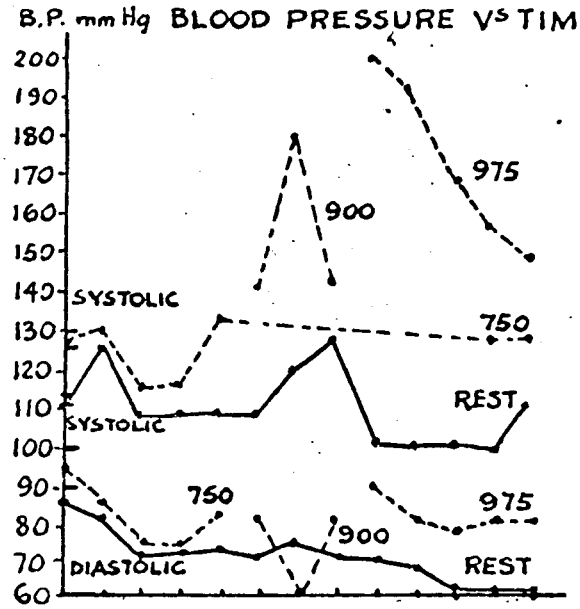
FIGURE 12b. Subject DB. Graphs of respiratory gas exchange, blood pressure and base excess for the 24 week rehabilitation period.

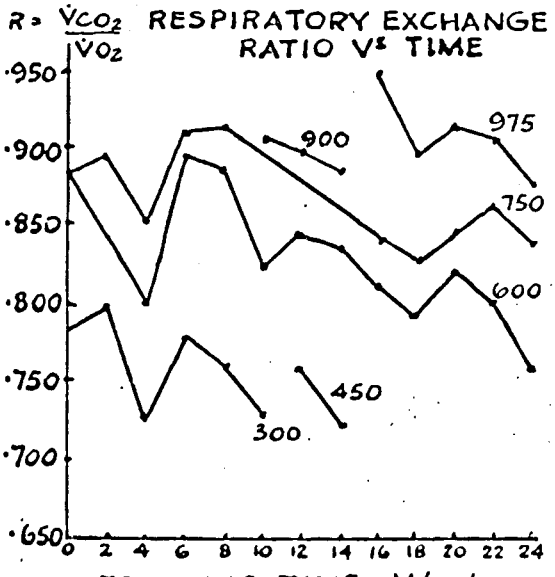
D.B.

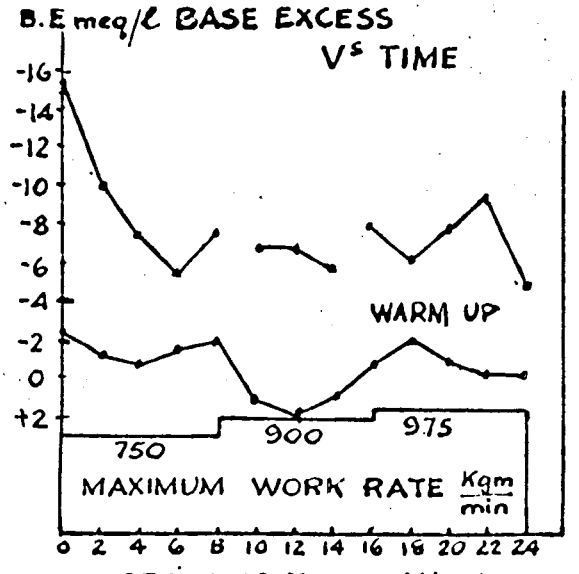

$\dot{V}O_2$ ℓ/min STPD OXYGEN UPTAKE Vs TIME
2·8
2·6
2·4
2·2
2·0
1·8
1·6
1·4
1·2
1·0
·8
·6
·4
·2
0
975
900
750
600
450
300
WARM UP


$\dot{V}CO_2$ ℓ/min STPD CARBON DIOXIDE ELIMINATION Vs TIME
2·8
2·6
2·4
2·2
2·0
1·8
1·6
1·4
1·2
1·0
·8
·6
·4
·2
0
900
975
750
600
450
300
WARM UP


$\dot{V}E$ ℓ/min STPD VENTILATION Vs TIME
90
80
70
60
50
40
30
20
975
900
750
600
450
300


B.P. mm Hg BLOOD PRESSURE Vs TIME
200
190
180
170
160
150
140
130
120
110
100
90
80
70
60
975
900
SYSTOLIC
750
REST
SYSTOLIC
750
975
900
DIASTOLIC
REST


R = $\frac{\dot{V}CO_2}{\dot{V}O_2}$ RESPIRATORY EXCHANGE RATIO Vs TIME
·950
·900
·850
·800
·750
·700
·650
975
900
750
600
450
300
0 2 4 6 8 10 12 14 16 18 20 22 24
TRAINING TIME - Weeks


B.E meq/ℓ BASE EXCESS Vs TIME
-16
-14
-12
-10
-8
-6
-4
-2
0
+2
WARM UP
750
900
975
MAXIMUM WORK RATE Kgm/min
0 2 4 6 8 10 12 14 16 18 20 22 24
TRAINING TIME - Weeks

kpm/min; 0.0138 → 0.0169 l/beat at 600 kpm/min; 0.0152 → 0.0166 l/beat at 750 kpm/min; 0.0145 → 0.0180 l/beat at 900 kpm/min and 0.0158 → 0.0187 l/beat at 975 kpm/min. An increase in pulmonary ventilation efficiency was thus indicated by gradually decreasing values for ventilation for equivalent work. The reduction was most apparent at lower rates (25 → 21 at 300 kpm/min and 29 → 22 at 600 kpm/min).

Base excess and lactate - Figure 12b and Tables 7 and 12

Resting and warm-up values of base excess (-2.5 → -1.5 meq/l) and lactate (12.7 → 7.0 mg %) decreased up to week twelve, then increased up to week eighteen (BE -2.0 meq/l, La 9.0 mg %) and decreased once again by the end of the program (BE -0.3 meq/l, La 3.0 mg %). Exercise values for the various work rates decreased up to week eighteen, increased during the next four weeks, then decreased again during the last two weeks of continuous training. The levels of metabolic acidosis at the 900 (final lactate 42.3 mg %) and 975 kpm/min (final lactate 15.0 mg %) work rates were comparable to those previously found at 750 kpm/min.

Blood pressure - Figure 12b

Resting diastolic blood pressure decreased considerably throughout the program (86 → 60 mmHg). The systolic pressure showed a small decrease overall (117 → 100 mmHg) although an increase did occur on three occasions. Exercise-diastolic pressure decreased at 750 and 975 kpm/min. The exercise-systolic pressure remained quite constant at 750 and 900 kpm/min, however, at 975 kpm/min a large decrease (200 → 148 mmHg) occurred.

Myocardial oxygen demand - Table 14

The mean initial warm-up value for myocardial oxygen demand was 8779 mmHg

X HR and by the tenth week this had decreased to 6696 mmHg X HR. An increase occurred at weeks twelve and fourteen (8300 mmHg X HR), however, it returned to a value of 6200 mmHg X HR for the remainder of the training period. Exercise values at the 750 and 900 kpm/min decreased slightly (20815 → 19140 mmHg X HR at 750 kpm/min and 20720 → 20306 mmHg X HR at 900 kpm/min). Values at 975 kpm/min were reduced (32200 → 21164 mmHg X HR).

Lung function - Figure 12a

The respiratory rate decreased significantly during twenty-four weeks of training (25 → 15 b/min). Resting-tidal volume remained quite constant as did vital capacity (5.29 → 5.40 l). An increase occurred in the values for FEV_1 (3.68 → 4.01 l).

Body weight and serum lipids - Figure 12a and Tables 7 and 12

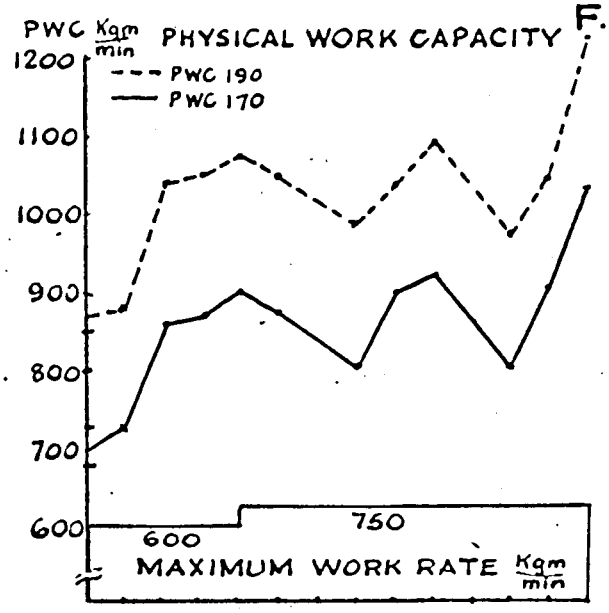
The weight of this subject decreased greatly (80.3 → 68.7 kg). Serum cholesterol also showed a concomitant large decrease (302 → 145 mg %) as did serum triglyceride levels (275 → 89 mg %). This subject showed the largest decreases in body weight and serum lipid of all the subjects.

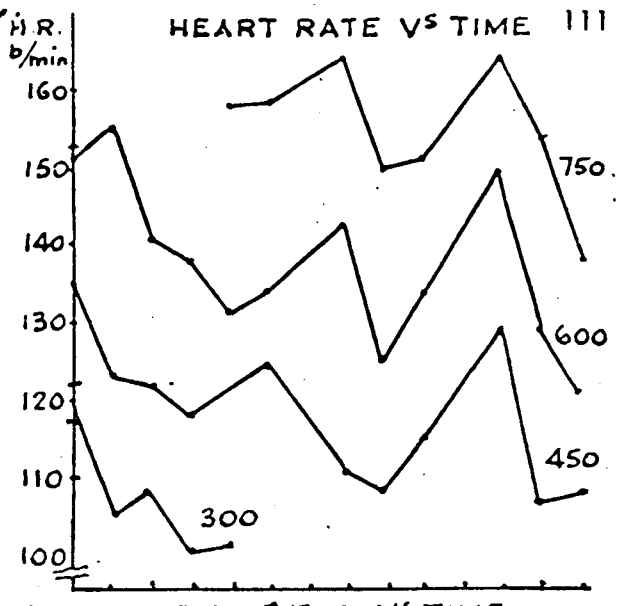
Subject FC Age 51 years Height 172.0 cm Weight 72.5 kg (initial) 68.6 kg (final) Started training five months post infarction

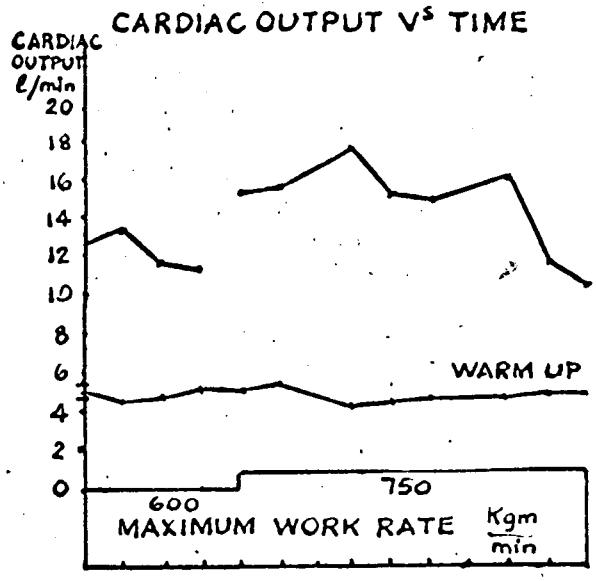
Physical work capacity - Figure 13a

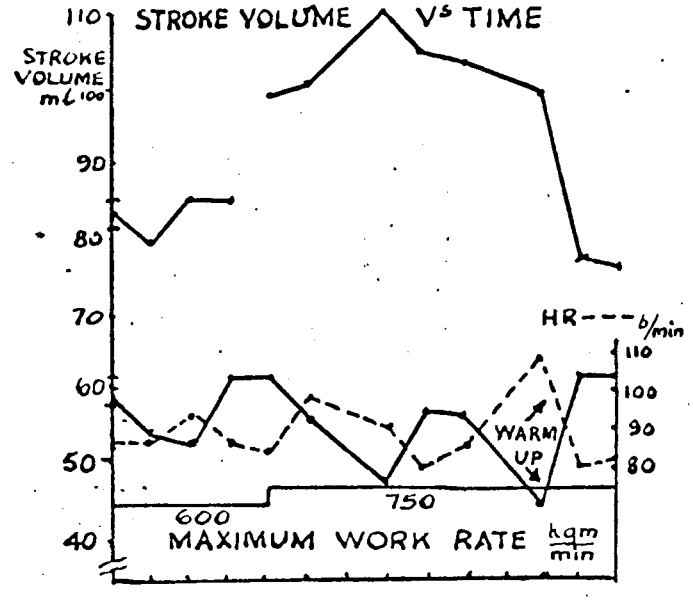
Based on the three initial evaluation tests this subject had a mean PWC_{170} of 698 kpm/min and a PWC_{190} of 867 kpm/min. After ten weeks of continuous training his PWC_{170} increased 26.1 per cent (880 kpm/min) and his PWC_{190} increased 21.1 per cent (1050 kpm/min) above the initial levels. The original tests were at work rates of 300, 450 and 600 kpm/min. During

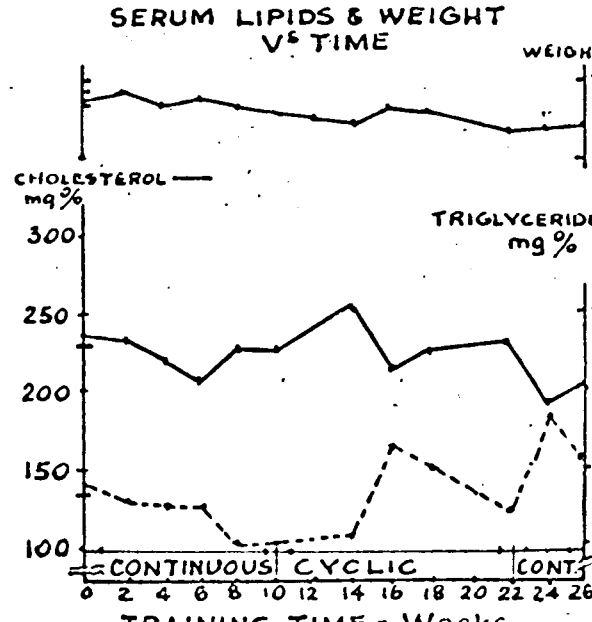
FIGURE 13a. Subject FC. Graphs of physical work capacity, heart rate, cardiac output, stroke volume, lung function, body weight and serum lipids for the 24 week rehabilitation period.

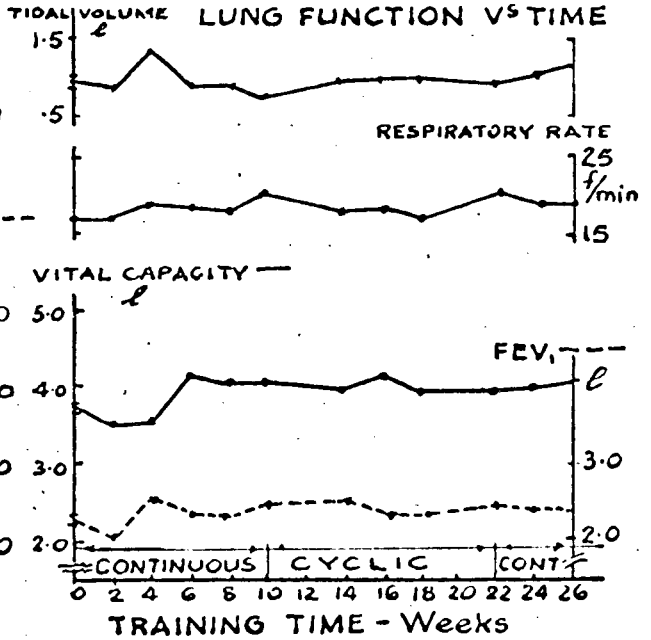

PWC Kgm/min
PHYSICAL WORK CAPACITY
PWC 190
PWC 170
1200
1100
1000
900
800
700
600
600
750
MAXIMUM WORK RATE Kgm/min


H.R. b/min
HEART RATE Vs TIME
160
150
140
130
120
110
100
750
600
450
300


CARDIAC OUTPUT Vs TIME
CARDIAC OUTPUT ℓ/min
20
18
16
14
12
10
8
6
4
2
0
WARM UP
600
750
MAXIMUM WORK RATE Kgm/min


STROKE VOLUME Vs TIME
STROKE VOLUME mℓ/100
110
90
80
70
60
50
40
HR --- b/min
110
100
90
80
WARM UP
600
750
MAXIMUM WORK RATE kgm/min


SERUM LIPIDS & WEIGHT Vs TIME
WEIGHT
75
Kg
65
CHOLESTEROL mg%
TRIGLYCERIDE mg%
300
250
200
150
100
CONTINUOUS
CYCLIC
CONT.
0 2 4 6 8 10 12 14 16 18 20 22 24 26
TRAINING TIME - Weeks


TIDAL VOLUME ℓ
LUNG FUNCTION Vs TIME
1·5
·5
RESPIRATORY RATE
25
f/min
15
VITAL CAPACITY ℓ
FEV1 ---
5·0
4·0
3·0
2·0
CONTINUOUS
CYCLIC
CONT.
0 2 4 6 8 10 12 14 16 18 20 22 24 26
TRAINING TIME - Weeks

cyclic training his PWC_{170} (925 kpm/min) and PWC_{190} (1100 kpm/min) were maintained for a month but they then decreased, the PWC_{170} to 800 kpm/min and the PWC_{190} to 980 kpm/min. These latter evaluation tests were based on test work rates of 450, 600 and 750 kpm/min. During the final month of continuous training his PWC_{170} increased to 1040 kpm/min (a total increase of 48.6 per cent above mean initial level) and his PWC_{190} again increased to 1230 kpm/min (a total increase of 41.4 per cent above the initial mean level).

Heart rate - Figure 13a

This subject developed severe angina in his initial evaluation tests at a heart rate of 150 b/min and the tests were terminated there. After eight weeks of training no further angina occurred during training or evaluation tests.

Figure 13a shows the heart rate decrements for standard work rates up to week sixteen. During the last six weeks of cyclic training the heart rate increased at all work rates until training was changed to the continuous method, when once more the heart rate again decreased. Warm-up rate remained constant at an average of 88 b/min (range 80-109 b/min).

Cardiac output and stroke volume - Figure 13a

Warm-up cardiac output did not change consistently during the twenty-four week exercise period (initial 5.03 l/min, final 5.06 l/min). Stroke volume during warm-up varied between 50 and 60 ml however no general trend was apparent. During six weeks continuous training the cardiac output for 600 kpm/min decreased from 13.07 to 11.70 l/min. The cardiac output at 750 kpm/min did not change during the final four weeks of continuous training or the ten

weeks of cyclic training. However, once training was changed back to the continuous ergometry for a month a large decrease occurred (16.30 → 10.64 l/min, 750 kpm/min). Exercise-stroke volume remained constant for a standard work rate until the last month of the continuous training when a large decrease occurred (100 → 76 ml at 750 kpm/min).

Respiratory gas exchange - Figure 13b

Ventilation decreased for equivalent work rates during ten weeks and then remained quite constant. Exercise values for $\dot{V}O_2$ and $\dot{V}CO_2$ decreased steadily throughout the training period. The largest decrease occurred in $\dot{V}CO_2$ (mean decrease 0.36 l/min). The exercise-R values increased at the second week, however, a decrease followed up to week eight. The R value remained constant for the 750 kpm/min tests but an increase occurred for the two lower work rates for six weeks after which a large decrease occurred for all work rates.

Exercise oxygen pulse and ventilation equivalent - Table 10

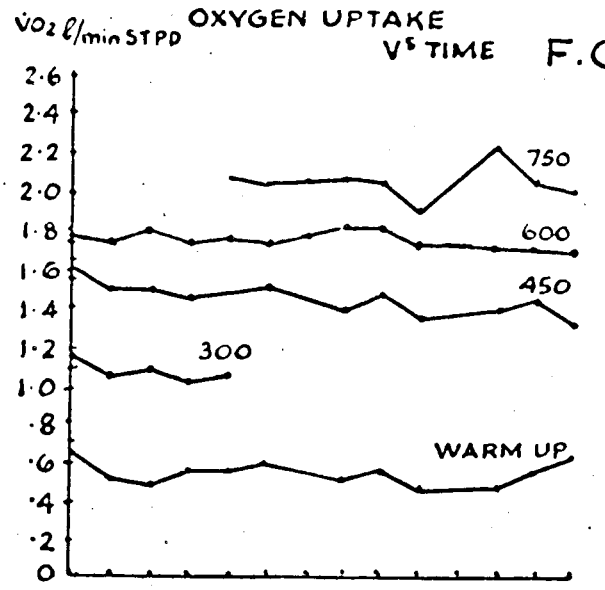
Training did not appear to change the exercise values for oxygen pulse or ventilation equivalent. During the period of cyclic training the oxygen pulse values were quite variable due to a variable heart rate response but a decrease was noted in it for all work rates between weeks sixteen to twenty-two. The final month of continuous training increased these values to the values before the start of cyclic training. However, training did decrease pulmonary ventilation, oxygen uptake and carbon dioxide elimination for a standard work rate.

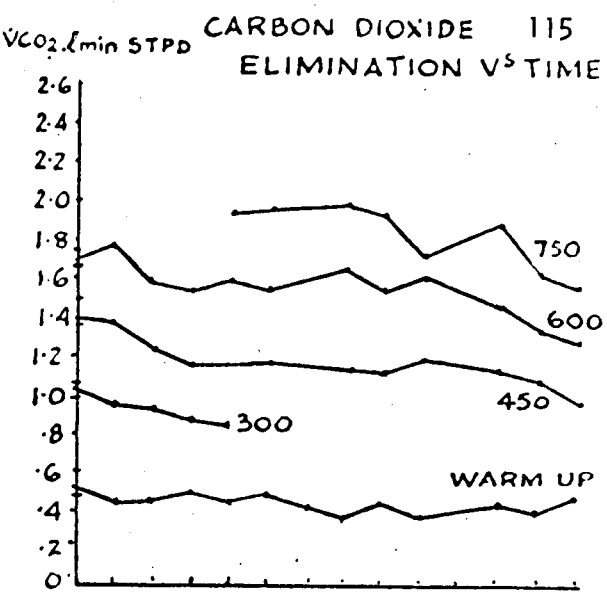
Base excess and lactate - Figure 13b and Tables 7 and 12

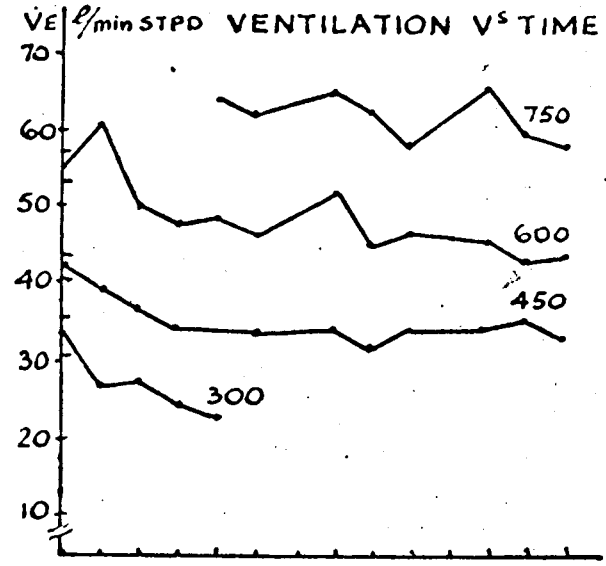
Warm-up and resting base excess (BE) and lactate (La) values showed a

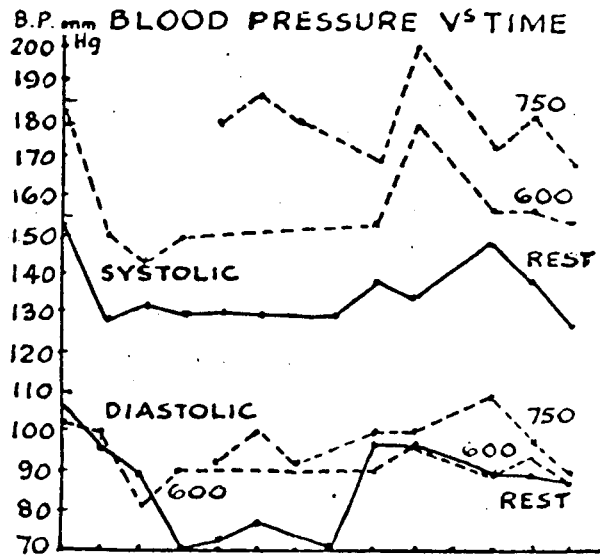
FIGURE 13b. Subject FC. Graphs of respiratory gas exchange, blood pressure and base excess for the 24 week rehabilitation period.

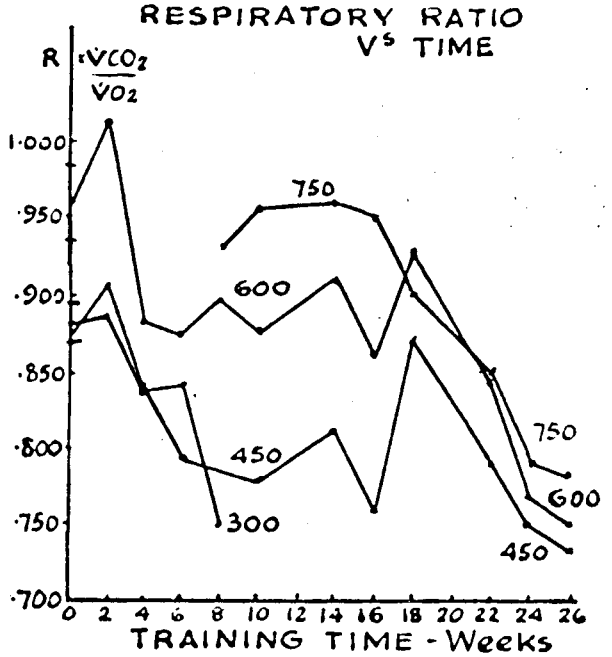
F.C.

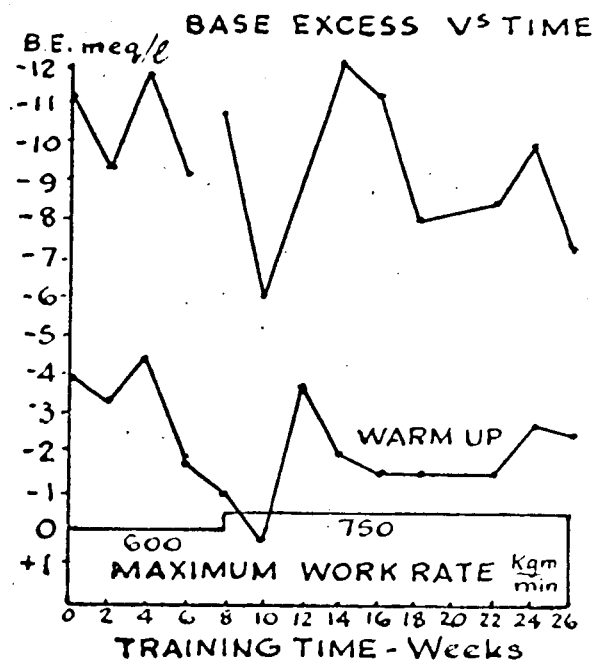

V̇O2 ℓ/min STPD
OXYGEN UPTAKE
Vs TIME
2·6
2·4
2·2
2·0
1·8
1·6
1·4
1·2
1·0
·8
·6
·4
·2
0
750
600
450
300
WARM UP


V̇CO2 ℓmin STPD
CARBON DIOXIDE
ELIMINATION Vs TIME
2·6
2·4
2·2
2·0
1·8
1·6
1·4
1·2
1·0
·8
·6
·4
·2
0
750
600
450
300
WARM UP


V̇E ℓ/min STPD
VENTILATION Vs TIME
70
60
50
40
30
20
10
750
600
450
300


B.P. mm Hg
BLOOD PRESSURE Vs TIME
200
190
180
170
160
150
140
130
120
110
100
90
80
70
750
600
SYSTOLIC
REST
DIASTOLIC
750
600
600
REST


RESPIRATORY RATIO
Vs TIME
R V̇CO2 / V̇O2
1·000
·950
·900
·850
·800
·750
·700
750
600
450
300
750
600
450
0 2 4 6 8 10 12 14 16 18 20 22 24 26
TRAINING TIME - Weeks


B.E. meq/ℓ
BASE EXCESS Vs TIME
-12
-11
-10
-9
-8
-7
-6
-5
-4
-3
-2
-1
0
+1
WARM UP
600
750
MAXIMUM WORK RATE Kgm/min
0 2 4 6 8 10 12 14 16 18 20 22 24 26
TRAINING TIME - Weeks

decrease from the initial values (BE -4.0 → -2.5 meq/l, La 10.4 → 6.0 mg %). A progressively lower exercise metabolic acidosis was observed. The lowest test levels were achieved after continuous training.

Blood pressure - Figure 13b

Resting-systolic blood pressure decreased during the training program. During the ten weeks of continuous training it had decreased from 150 mmHg to 130 mmHg, however, it had increased to the initial level once again by the end of cyclic training. Once continuous training was again employed, however, the resting-systolic blood pressure was decreased to 128 mmHg. Resting-diastolic pressure followed a similar pattern, initially a decrease from 106 mmHg to 70 mmHg, followed by an increase to 98 mmHg and finally a decrease to 88 mmHg.

Exercise-systolic blood pressure showed a decrease with continuous training which was maintained at this level, except for an increase at week eighteen. Exercise-diastolic pressure increased slightly during the period of cyclic training and decreased during the continuous training.

Myocardial oxygen demand - Table 14

Warm-up myocardial oxygen demand decreased from an initial value of 12700 mmHg X HR to 10496 mmHg X HR at the end of all training. During the first six weeks of training the myocardial oxygen demand at a work rate of 600 kpm/min decreased from 27300 to 20700 mmHg X HR. The value at 750 kpm/min did not change until the training was changed in the last month to the continuous sytle (29000-23800 mmHg X HR).

Lung function - Figure 13a

No change occurred in resting-tidal volume and respiratory rate during

the twenty-four week training period. Both vital capacity and FEV_1 values increased in the early part of the training and then remained at this level throughout (3.77-4.06 l, vital capacity and 2.30-2.50 l, FEV_1).

Body weight and serum lipids - Figure 13a

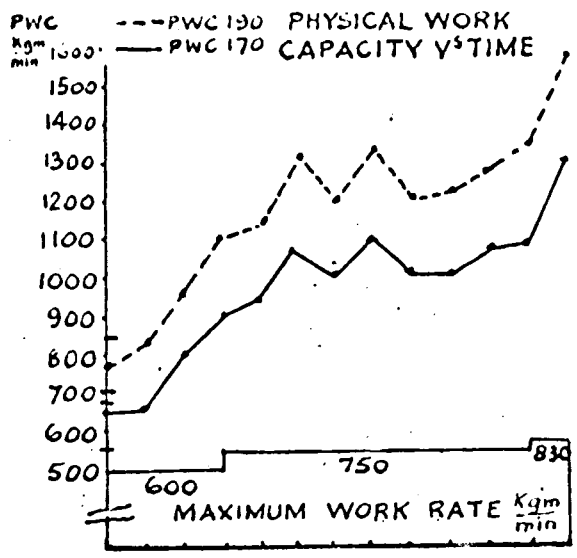
Body weight decreased from 72.6 kg to 68.6 kg during the training period. Serum cholesterol decreased from 235 mg % to 207 mg % (largest decrease occurred in the last month of continuous training) and serum triglyceride levels decreased in the first ten weeks from 140 mg % to 109 mg % but increased during the period of cyclic training to 121 mg %. The final month of continuous training failed to decrease serum triglyceride again (final value in fact was 151 mg %).

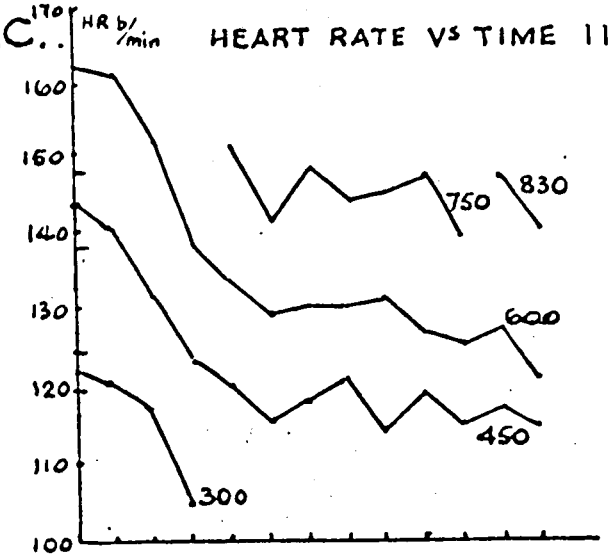
Subject HC Age 55 Height 169.8 cm Weight 73.5 kg (initial) 72.8 kg (final) Started training five months post infarction

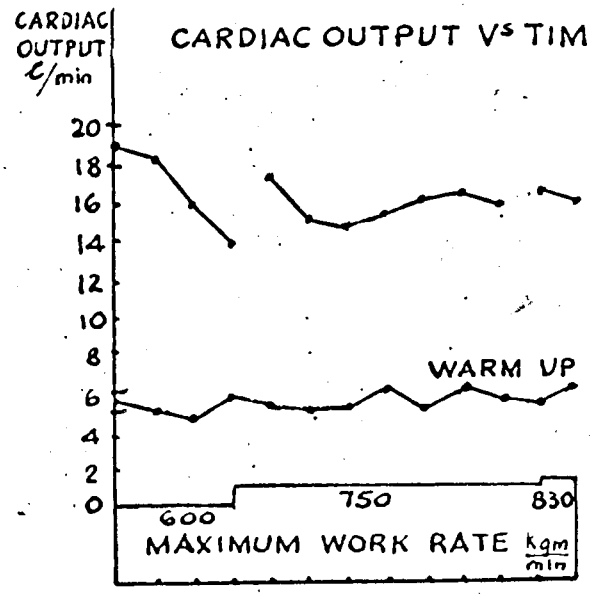
Physical work capacity - Figure 14a

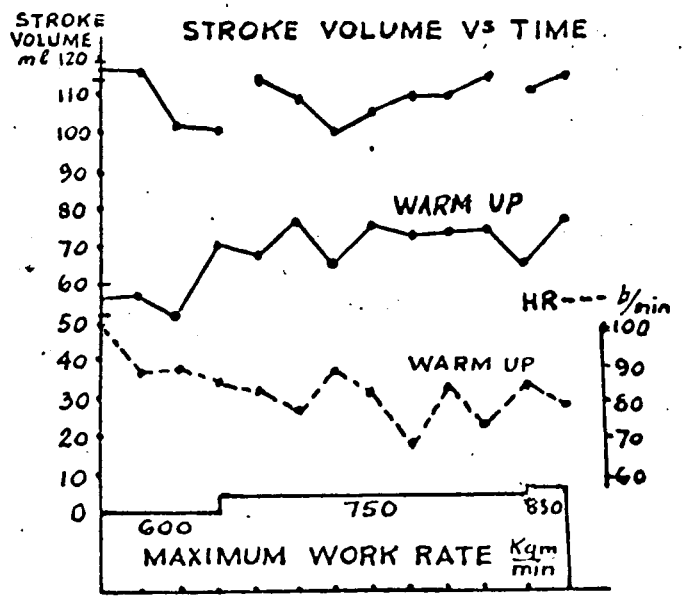
This subject increased his PWC_{170} from an initial value of 647 kpm/min to 1075 kpm/min (66.1 per cent) and his PWC_{190} from 750 kpm/min to 1300 kpm/min (73.3 per cent) after ten weeks continuous bicycle ergometry training. No further increase occurred after the ten weeks of cyclic bicycle training. The three work rates used in the evaluation of the latter were the same as during the tests of continuous training which was restarted after cyclic training finished. The return to continuous training during the last month of the program resulted in a large increase in both PWC_{170} and PWC_{190}. The subject's PWC_{170} increased to 1300 kpm/min a total increase above initial of 102 per cent) and his PWC_{190} increased to 1570 kpm/min (a total increase

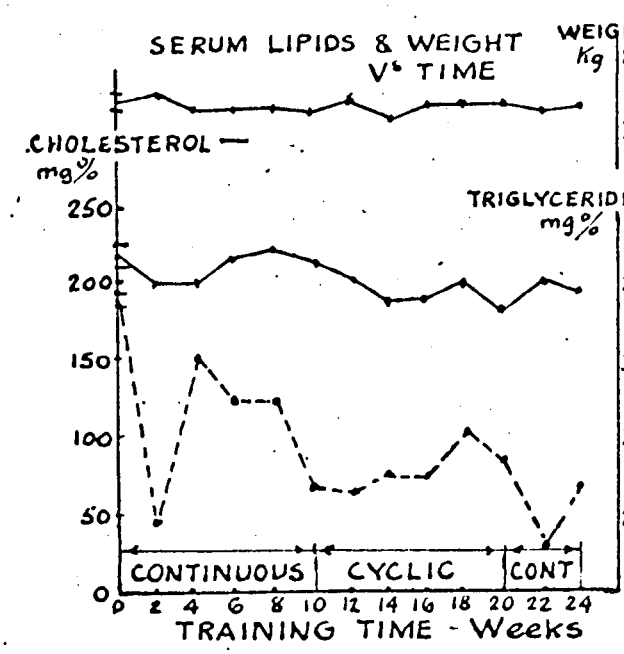
FIGURE 14a. Subject HC. Graphs of physical work capacity, heart rate, cardiac output, stroke volume, lung function, body weight and serum lipids for the 24 week rehabilitation period.

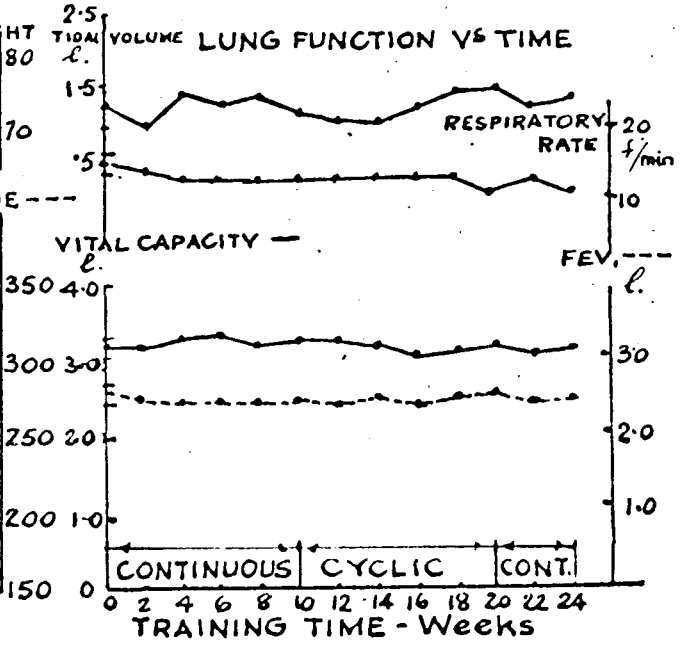

PWC
Kgm/min
---PWC 190
——PWC 170
PHYSICAL WORK CAPACITY Vs TIME
1600
1500
1400
1300
1200
1100
1000
900
800
700
600
500
600
750
830
MAXIMUM WORK RATE Kgm/min


HR b/min
HEART RATE Vs TIME
170
160
150
140
130
120
110
100
750
830
600
450
300


CARDIAC OUTPUT
l/min
CARDIAC OUTPUT Vs TIME
20
18
16
14
12
10
8
6
4
2
0
WARM UP
600
750
830
MAXIMUM WORK RATE Kgm/min


STROKE VOLUME ml
STROKE VOLUME Vs TIME
120
110
100
90
80
70
60
50
40
30
20
10
0
WARM UP
HR--- b/min
100
90
80
70
60
WARM UP
600
750
830
MAXIMUM WORK RATE Kgm/min


SERUM LIPIDS & WEIGHT Vs TIME
WEIGHT Kg
80
70
CHOLESTEROL —
mg%
250
200
150
100
50
0
TRIGLYCERIDE ---
mg%
350
300
250
200
150
CONTINUOUS
CYCLIC
CONT
0 2 4 6 8 10 12 14 16 18 20 22 24
TRAINING TIME - Weeks


2·5
TION VOLUME l.
LUNG FUNCTION Vs TIME
1·5
·5
RESPIRATORY RATE
20
f/min
10
VITAL CAPACITY —
l.
FEV, ---
l.
4·0
3·0
2·0
1·0
3·0
2·0
1·0
CONTINUOUS
CYCLIC
CONT.
0 2 4 6 8 10 12 14 16 18 20 22 24
TRAINING TIME - Weeks

of 109.3 per cent).

Heart rate - Figure 14a

Figure 14a shows the large decrement in heart rate for all work rates until week ten (end of continuous training) after which no further decrease occurred. The final month of continuous training however resulted in a further reduction. During this time the subject completed a test at 830 kpm/min with a heart rate similar to that which occurred 450 kpm/min in the initial tests. Warm-up heart rate steadily decreased, up to week ten (99 → 76 b/min) and then remained quite constant at this level.

Cardiac output and stroke volume - Figure 14a

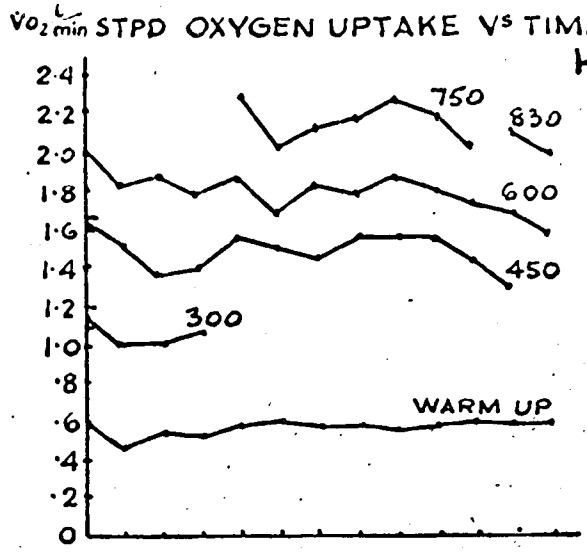
Warm-up-cardiac output did not change with training and warm-up-stroke volume increased (57 → 77 ml) with the decreasing heart rate. The increase of warm-up-stroke volume stopped at week ten. During the first twelve weeks of rehabilitation the exercise-cardiac output at a standard work rate decreased considerably (18.80 → 13.80 l/min at 600 kpm/min; 17.24 → 14.36 l/min at 750 kpm/min). During the remaining time of cyclic training exercise-cardiac output increased slightly (14.36 → 16.00 l/min at 750 kpm/min). A decrease in this parameter occurred during the final month of continuous training. Exercise-stroke volume, decreased up to week twelve (116 → 99 ml at 600 kpm/min; 113 → 98 ml at 750 kpm/min) and then increased (98 → 113 ml, 750 kpm/min) up to week twenty.

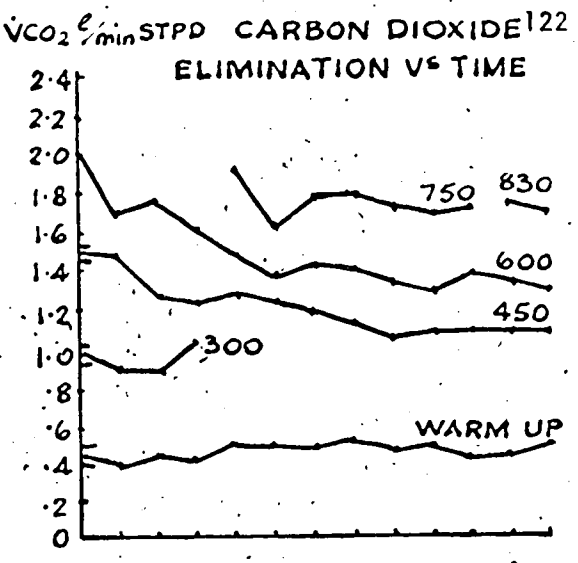
Respiratory gas exchange - Figure 14b

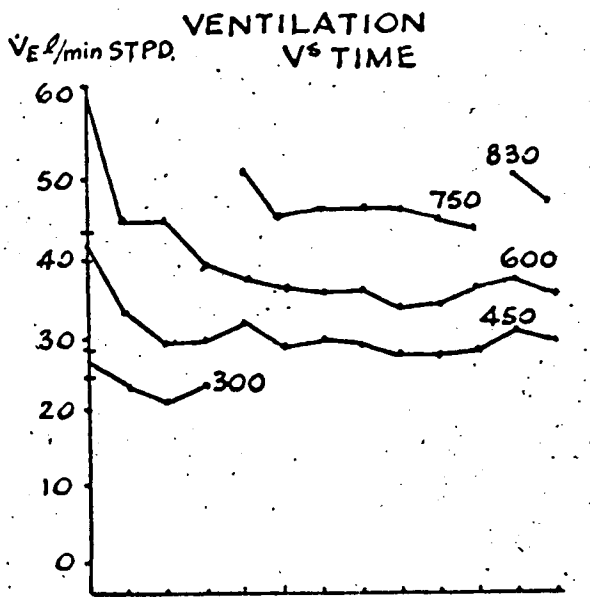
Exercise-ventilation during initial evaluation decreased considerably for all work rates up to week ten and then remained constant until week twenty-two after which a small decrease occurred. For a standard work rate,

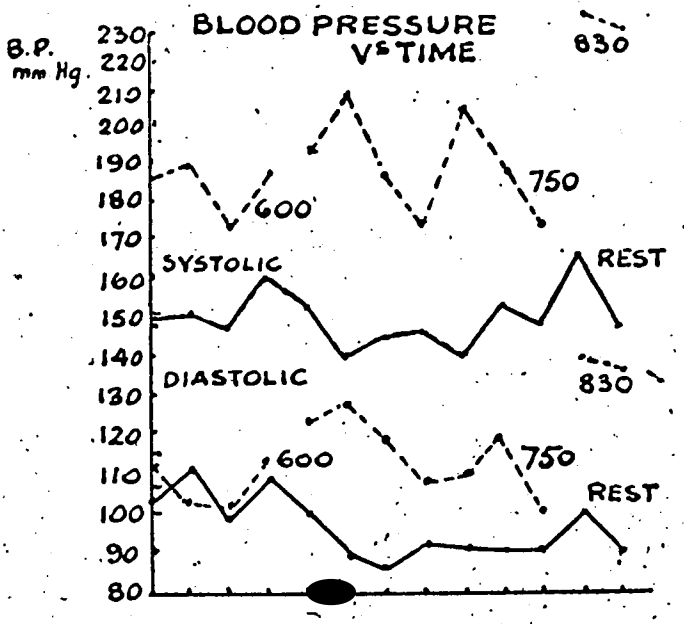
FIGURE 14b. Subject HC. Graphs of respiratory gas exchange, blood pressure and base excess for the 24 week rehabilitation period.

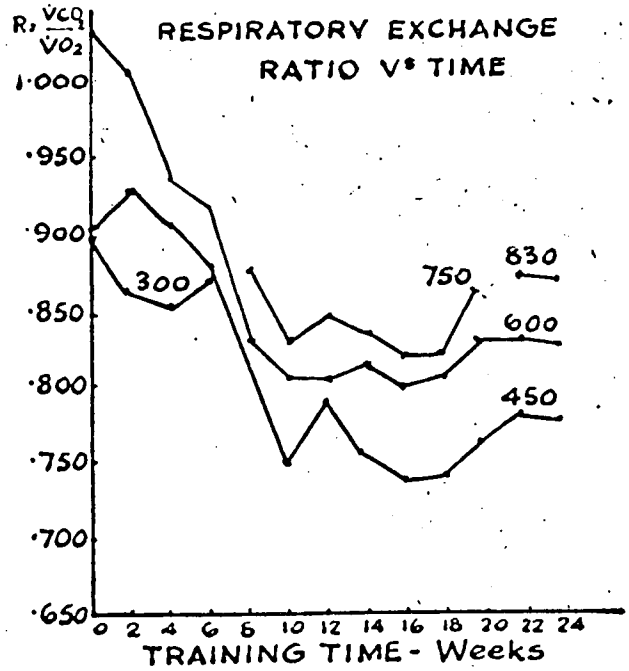
H.C.

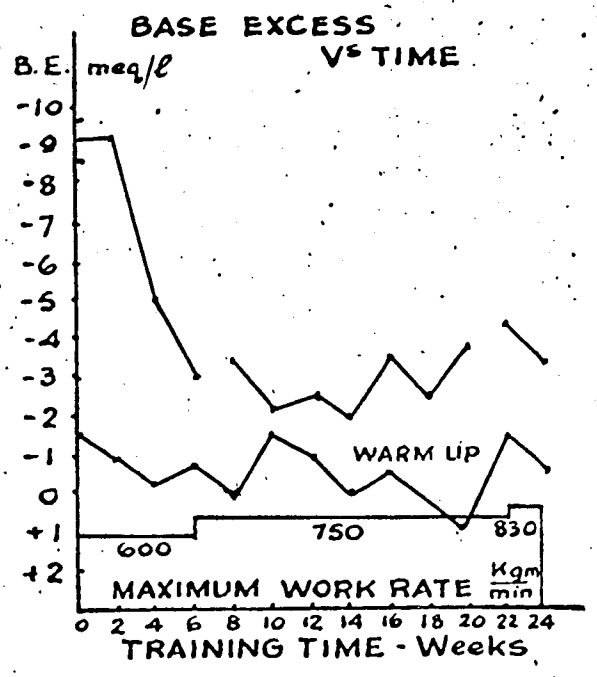

$\dot{V}O_2$ L/min STPD OXYGEN UPTAKE Vs TIME
2·4
2·2
2·0
1·8
1·6
1·4
1·2
1·0
·8
·6
·4
·2
0
750
830
600
450
300
WARM UP


$\dot{V}CO_2$ ℓ/min STPD CARBON DIOXIDE ELIMINATION Vs TIME
2·4
2·2
2·0
1·8
1·6
1·4
1·2
1·0
·8
·6
·4
·2
0
750
830
600
450
300
WARM UP


$\dot{V}_E$ ℓ/min STPD. VENTILATION Vs TIME
60
50
40
30
20
10
0
830
750
600
450
300


B.P. mm Hg. BLOOD PRESSURE Vs TIME
230
220
210
200
190
180
170
160
150
140
130
120
110
100
90
80
830
600
750
SYSTOLIC
REST
DIASTOLIC
830
600
750
REST


R, $\frac{\dot{V}CO_2}{\dot{V}O_2}$ RESPIRATORY EXCHANGE RATIO Vs TIME
1·000
·950
·900
·850
·800
·750
·700
·650
300
750
830
600
450
0 2 4 6 8 10 12 14 16 18 20 22 24
TRAINING TIME - Weeks


B.E. meq/ℓ BASE EXCESS Vs TIME
-10
-9
-8
-7
-6
-5
-4
-3
-2
-1
0
+1
+2
WARM UP
600
750
830
MAXIMUM WORK RATE Kgm/min
0 2 4 6 8 10 12 14 16 18 20 22 24
TRAINING TIME - Weeks

exercise values for $\dot{V}O_2$ and $\dot{V}CO_2$ decreased up to week ten, remained constant in cyclic training and decreased again with the change of training back to the continuous method at week twenty. Associated with these changes were decreased R values for all work rates. The rate of decrease of this variable was reduced from week ten and in fact, in the later stages of cyclic training the R values increased.

Exercise-oxygen pulse and ventilation equivalent - Table 10

Training did not significantly change the exercise values for oxygen pulse or ventilation equivalent for the low work rate of 300 kpm/min and the highest rate of 750 kpm/min. At the intermediate work rates of 450 and 600 kpm/min values for oxygen pulse increased (0.0100 → 0.0132 l/beat at 450 kpm/min; 0.0125 → 0.0138 l/beat at 600 kpm/min) while those for ventilation equivalent decreased (26 → 18 at 450 kpm/min; 30-21 at 600 kpm/min). By the eighth week of training these changes had occurred and were maintained throughout the remainder of the program.

Base excess and lactate - Figure 14b and Tables 7 and 12

The general finding in warm-up and resting conditions was a decreased level of acidosis (BE -1.5 → 1.0 meq/l, La 10.4 → 9.0 mg %). Exercise-metabolic acidosis decreased during both periods of continuous ergometry (BE -3.5 → 2.2 meq/l, La 21.2 → 18.9 mg % at 750 kpm/min; BE -4.4 → -3.3 meq/l, La 25.0 → 18.0 mq % at 830 kpm/min). A small increase occurred during weeks ten to twenty.

Blood pressure - Figure 14b

Resting-systolic blood pressure decreased (150 → 140 mmHg) up to week sixteen when an increase to the initial value took place. The highest value

was at week twenty-two (170 mmHg). Resting-diastolic pressure decreased up to week twelve (104 → 90 mmHg) and then remained constant at this level. Exercise-systolic pressure was not changed with training while exercise-diastolic pressure decreased. The increased exercise blood pressure at weeks twenty-two and twenty-four (830 kpm/min) resulted when medication was terminated.

Myocardial oxygen demand - Table 14

Warm-up values for myocardial oxygen demand decreased (15016 → 11400 mmHg X HR). During the rehabilitation program exercise values decreased (29140 → 26790 mmHg X HR at 600 kpm/min; 29682 → 23630 at 750 kpm/min).

Lung function - Figure 14a

Twenty-four weeks of rehabilitation did not change any of the lung function parameters.

Body weight and serum lipids - Figure 14a

Body weight did not change during the twenty-four weeks whereas serum cholesterol decreased slightly (220 → 195 mg %). A large change occurred in the levels of serum triglycerides (335 → 217 mg %). The largest decrease occurred during the period of continuous training (335 → 240 mg %).

Subject HG Age 50 years Height 178.5 cm Weight 74.3 kg (initial) 79.0 kg (final) Started training six months post infarction

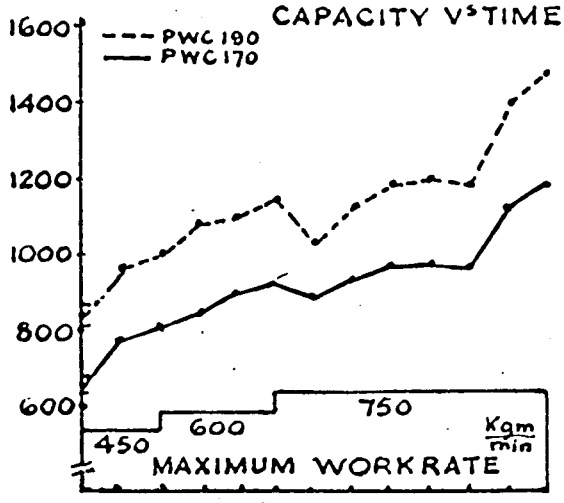
Physical work capacity - Figure 15a

This subject had an initial PWC_{170} of 660 kpm/min and a PWC_{190} of 840 kpm/min. After ten weeks of continuous bicycle ergometry training his PWC_{170} increased 40.2 per cent (925 kpm/min) and his PWC_{190} increased 35.8 per cent

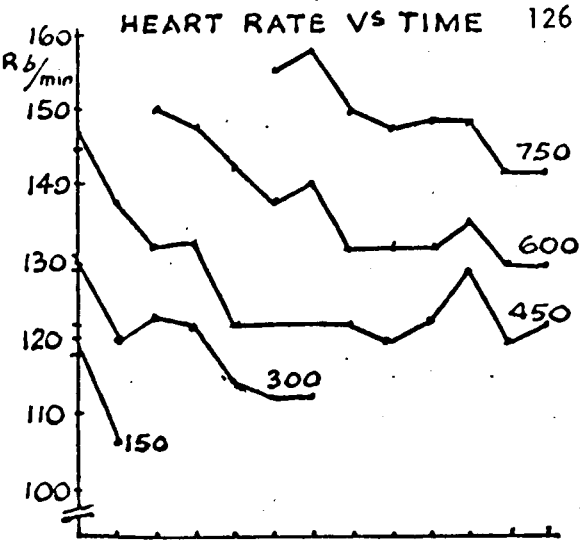
FIGURE 15a. Subject HG. Graphs of physical work capacity, heart rate, cardiac output, stroke volume, lung function, body weight and serum lipids for the 24 week rehabilitation period.

H.G.

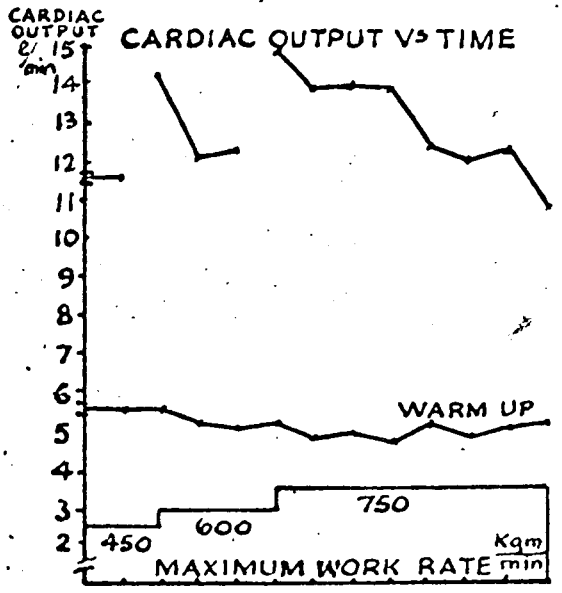
PWC $\frac{Kgm}{min}$ PHYSICAL WORK CAPACITY Vs TIME



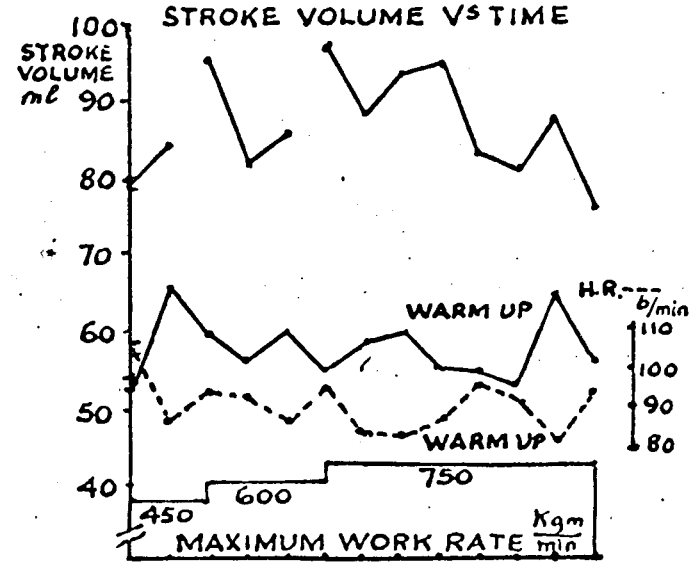
HEART RATE Vs TIME



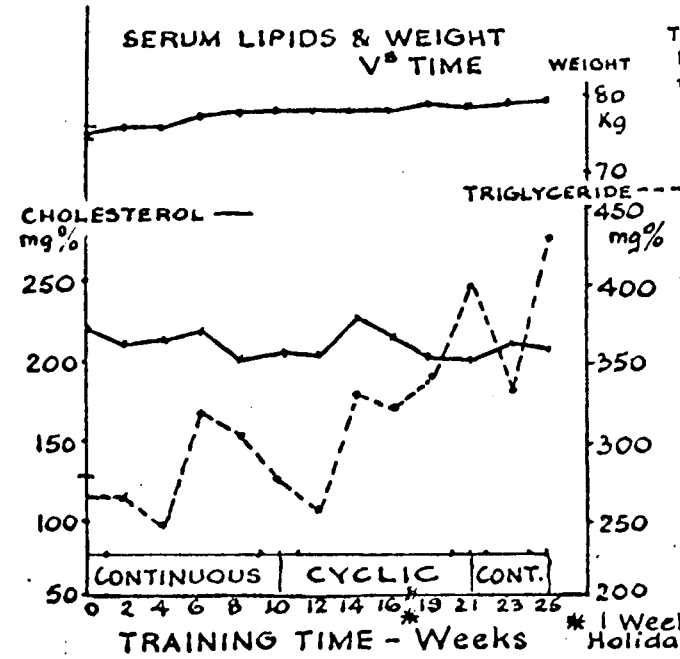
CARDIAC OUTPUT Vs TIME



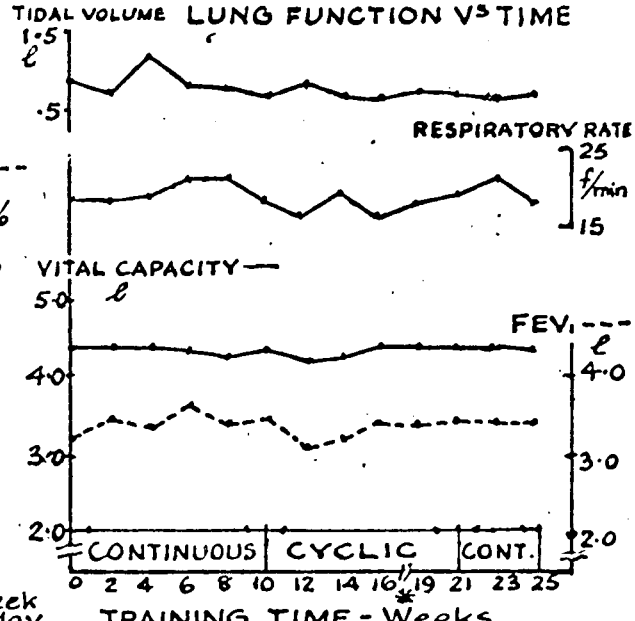
STROKE VOLUME Vs TIME



SERUM LIPIDS & WEIGHT Vs TIME



LUNG FUNCTION Vs TIME



(1140 kpm/min). The cyclic bicycle ergometry method yielded only a small gain. His PWC_{170} increased to 975 kpm/min (total increase of 47.8 per cent above initial) and his PWC_{190} increased to 1200 kpm/min (total increase of 42.8 per cent above initial). The final period of continuous training increased his PWC_{170} to 1180 kpm/min (total increase above initial of 78.8 per cent) and his PWC_{190} to 1475 kpm/min (total increase above initial of 75.6 per cent).

Heart rate - Figure 15a

Figure 15a shows heart rate values for standard work rates throughout the rehabilitation program. During the first ten weeks of training (continuous effort) at all work rates, the heart rate was much lower. The obvious decrease in heart rate ended abruptly at the end of continuous training for all work rates and remained constant at this level until the last month of training, at which time continuous training was once again instituted. Test work rates during the first ten weeks were first 300, 450 and 600 kpm/min changing to 300, 600 and 750 kpm/min. The tests throughout the rest of the program were conducted at work rates of 450, 600 and 750 kpm/min. Warm-up-heart rate steadily decreased during the program except for three tests when small increases occurred (106 → 85 b/min).

Cardiac output and stroke volume - Figure 15a

Warm-up-cardiac output showed a small decrease during the training program (5.66 → 5.10 l/min). The stroke volume during the controlled warm-up period ranged from 52.8 ml to 65.8 ml with an average of 57.4 ml; no apparent change occurred. Exercise-cardiac output decreased steadily throughout the program (11.73 → 11.59 l/min, at 450 kpm/min; 14.25 → 12.20 l/min, at 600 kpm/min; 15.00 → 10.39 l/min at 750 kpm/min). The decrease

was evident during both types of training. Exercise-stroke volume remained constant during the tests at 450 kpm/min (average 80.4 ml), however, further training resulted in a decreased stroke volume at both 600 kpm/min and 750 kpm/min (95.0 → 85.0 ml at 600 kpm/min; 96.8 → 73.2 ml at 750 kpm/min).

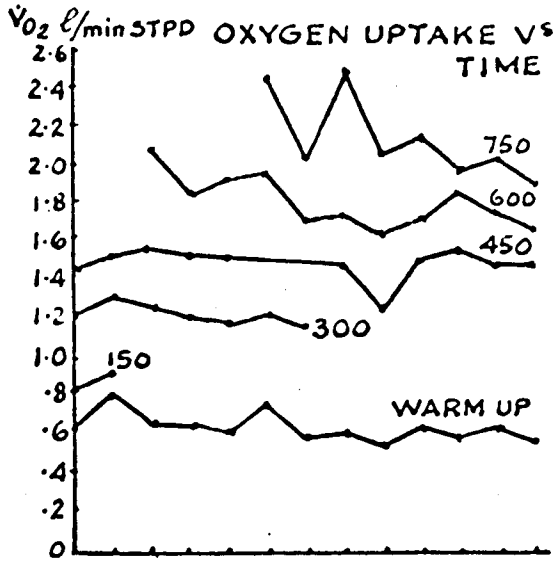
Respiratory gas exchange - Figure 15b

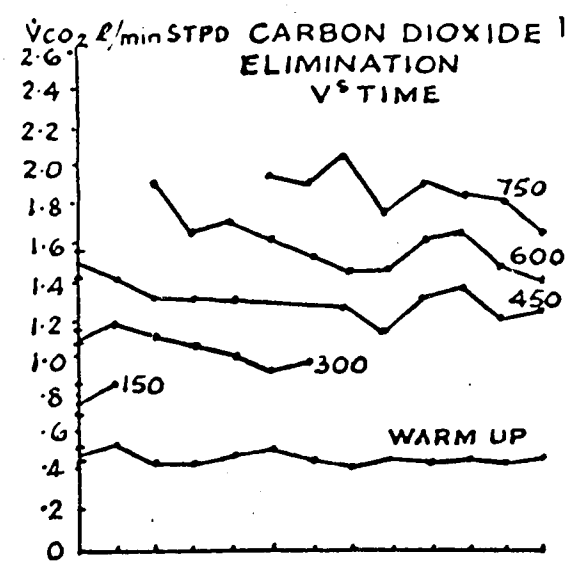
Exercise-ventilation for a given work rate progressively decreased up to week sixteen, increased marginally until continuous training was readopted after which another decrease occurred. Oxygen uptake decreased for more strenuous work rates (600 and 750 kpm/min) and remained unchanged for lower work rates of 150, 300 and 450 kpm/min. Exercise values for $\dot{V}CO_2$ decreased for all work rates. The pattern of $\dot{V}CO_2$ decrease parallelled that of ventilation (i.e. a decrease up to week sixteen, a small increase from week sixteen to twenty-one followed by another decrease). The exercise R values decreased up to week ten, then increased up to week fourteen and showed another consistent decrease from week twenty-one to twenty-five.

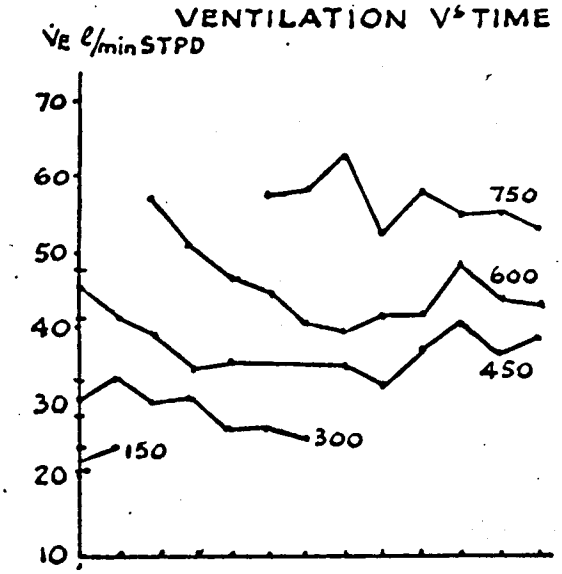
Exercise oxygen pulse and ventilation equivalent - Table 10

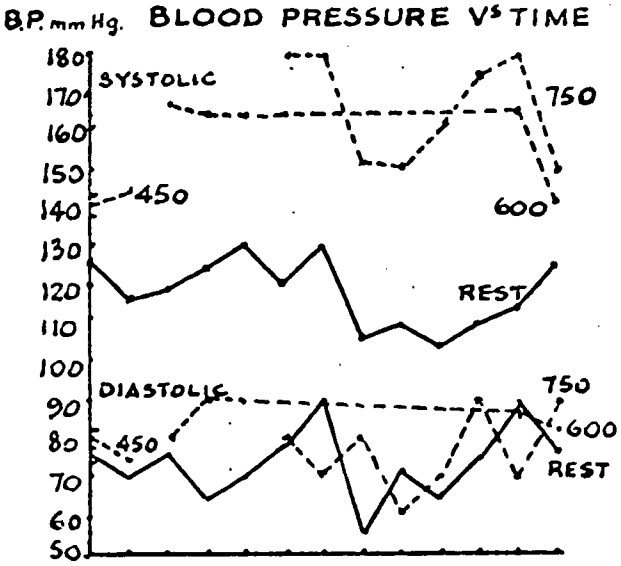
During tests at 300 kpm/min oxygen pulse increased (0.0091 → 0.0105 l/beat) and ventilation equivalent decreased (26 → 22). A similar change was seen at 450 kpm/min (0.0093 → 0.0123 l/beat and 35 → 25). The lowest values were recorded at the end of week ten (22), they increased slightly with the change to cyclic training. At a work rate of 600 kpm/min the oxygen pulse increased up to week ten (0.0126 → 0.0143 l/beat), however, this then decreased to 0.0130 l/beat by the end of the program. At this same work rate, ventilation equivalent decreased throughout the period of continuous training (27 → 22), however it increased again with increasing

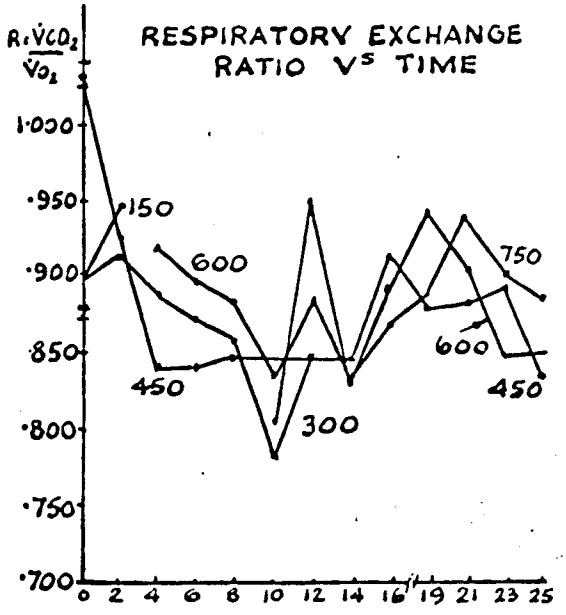
FIGURE 15b. Subject HG. Graphs of respiratory gas exchange, blood pressure and base excess for the 24 week rehabilitation period.

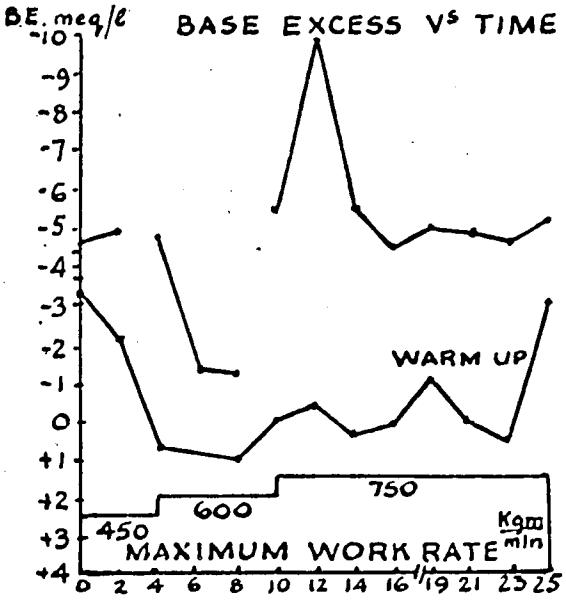

$\dot{V}_{O_2}$ ℓ/min STPD
OXYGEN UPTAKE Vs TIME
2·6
2·4
2·2
2·0
1·8
1·6
1·4
1·2
1·0
·8
·6
·4
·2
0
750
600
450
300
150
WARM UP


$\dot{V}_{CO_2}$ ℓ/min STPD
CARBON DIOXIDE ELIMINATION Vs TIME
2·6
2·4
2·2
2·0
1·8
1·6
1·4
1·2
1·0
·8
·6
·4
·2
0
750
600
450
300
150
WARM UP


VENTILATION Vs TIME
$\dot{V}_E$ ℓ/min STPD
70
60
50
40
30
20
10
750
600
450
300
150


B.P. mm Hg.
BLOOD PRESSURE Vs TIME
180
170
160
150
140
130
120
110
100
90
80
70
60
50
SYSTOLIC
750
450
600
REST
DIASTOLIC
750
450
600
REST


$R = \frac{\dot{V}CO_2}{\dot{V}O_2}$
RESPIRATORY EXCHANGE RATIO Vs TIME
1·000
·950
·900
·850
·800
·750
·700
150
600
750
450
300
600
450
0 2 4 6 8 10 12 14 16 19 21 23 25
TRAINING TIME - Weeks


B.E. meq/ℓ
BASE EXCESS Vs TIME
-10
-9
-8
-7
-6
-5
-4
-3
-2
-1
0
+1
+2
+3
+4
WARM UP
750
600
450
Kgm/min
MAXIMUM WORK RATE
0 2 4 6 8 10 12 14 16 19 21 23 25
TRAINING TIME - Weeks

ventilation up to week twenty-one (22 → 26). A similar pattern was seen at 750 kpm/min (i.e. with the change from continuous to cyclic training oxygen pulse decreased and ventilation equivalent increased).

Base excess and lactate - Figure 15b and Tables 7 and 12

Warm-up values for base excess (BE) and lactate (La) (BE -3.3 → -3.0 meq/l, La 9.4 → 8.0 mg %) decreased. No change occurred when the maximum test work rate was 450 kpm/min (week zero to two) however a decrease was noted for 600 kpm/min tests (BE -4.7 → -1.2 meq/l, La 18.0 → 8.0 mg %). The level of metabolic acidosis did not change during the period when the maximum work rate was 750 kpm/min (week ten to twenty-five).

Blood pressure - Figure 15b

Resting blood pressure, both diastolic and systolic, were quite variable and no overall change occurred. Exercise-systolic and diastolic pressure did not change during the test periods with 450 and 600 kpm/min as the maximum work rates. Exercise-systolic pressure with 750 kpm/min as the maximum work rate decreased up to week sixteen (continuous training) then increased up to week twenty-three (cyclic training) and decreased during the last weeks of continuous training.

Myocardial oxygen demand - Table 14

Warm-up-myocardial oxygen demand decreased up to week twelve and then remained at this level (13440 → 11000 mmHg X HR). Exercise values decreased during the training program (20930 → 19872 mmHg X HR at 450 kpm/min; 24900 → 23452 mmHg X HR at 600 kpm/min; 27900 → 21300 mmHg X HR at 750 kpm/min). During cyclic training from week sixteen to week twenty-one an increase did occur (to 26075 mmHg X HR, week twenty-one) but it decreased again with

continuous training.

Lung function - Figure 15a

Vital capacity, resting respiratory rate and tidal volume remained unchanged by the training program. The values for FEV_1 showed a very small increase (3.27 → 3.45 l).

Body weight and serum lipids - Figure 15a

Body weight showed a small consistent increase from the initial test (74.5 → 79.0 kg). Even with the increase in body weight, serum cholesterol decreased with training (213 → 200 mg %). Serum triglyceride progressively increased (261 → 439 mg %).

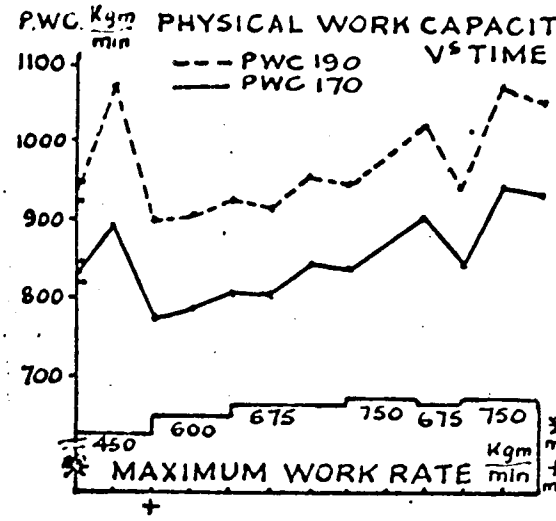
Group 3: Training: Calisthenic-walk-jog program (twenty weeks), continuous ergometry (four weeks), three times per week for fifty minutes with walking on the two days between the walk-jog days. Subjects WF and EK started training four weeks before being given initial tests.

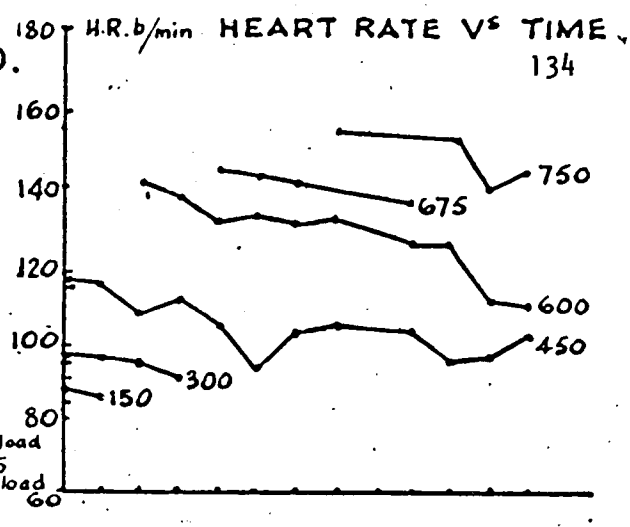
Subject JD Age 45 years Height 185.3 cm Weight 90.6 kg (initial) 93.1 kg (final) Started training three months post infarction

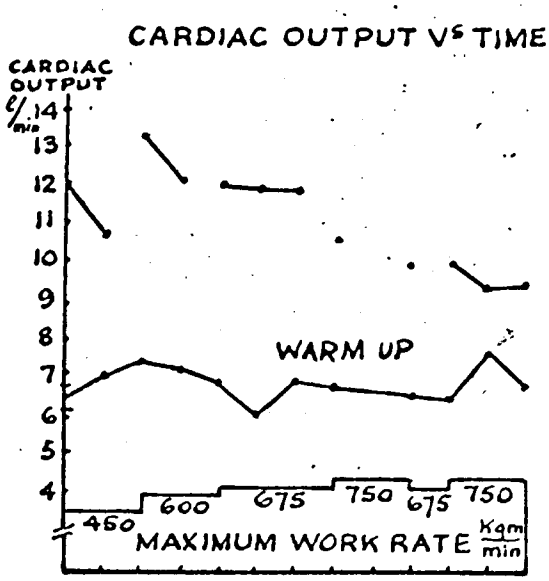
Physical work capacity - Figure 16a

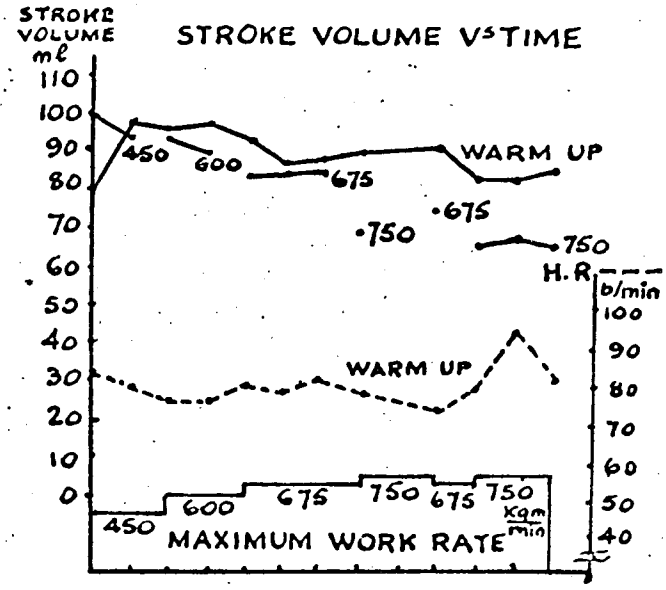
This subject was very weak, nervous and overweight when he started the training. During three initial evaluation tests he developed severe angina at a heart rate of 115 b/min and the exercise was terminated. Due to the fact that the physical work capacity was determined on such low heart rates the values for the initial and second week tests are questionable. For this reason an initial value for his PWC_{170} was taken to be 750 kpm/min and 880 kpm/min as his PWC_{190}. After ten weeks of training his PWC_{170} had increased to 810 kpm/min (8 per cent increase) and his PWC_{190} to 920 kpm/min (4.55 per

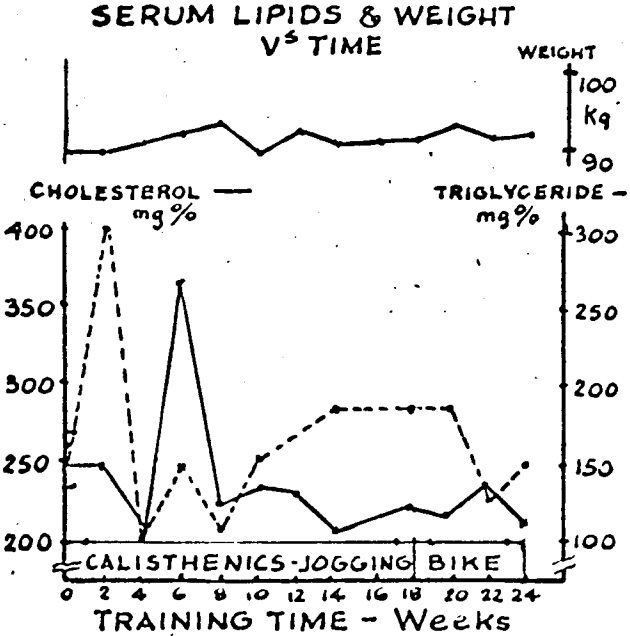
FIGURE 16a. Subject JD. Graphs of physical work capacity, heart rate, cardiac output, stroke volume, lung function, body weight and serum lipids for the 24 week rehabilitation period.

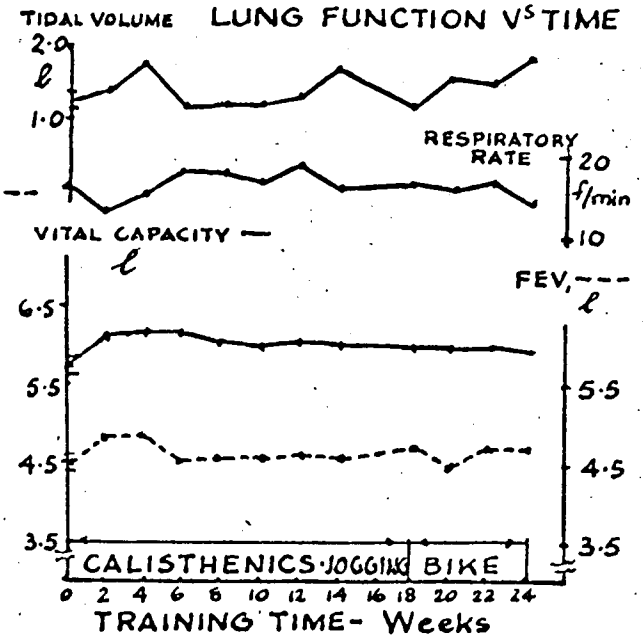

P.W.C. Kgm/min
PHYSICAL WORK CAPACITY Vs TIME
J.D.
--- PWC 190
—— PWC 170
1100
1000
900
800
700
450
600
675
750
675
750
MAXIMUM WORK RATE Kgm/min
* Only 4 mins last load
+ Only 5 mins last load


H.R. b/min
HEART RATE Vs TIME
180
160
140
120
100
80
60
750
675
600
450
300
150


CARDIAC OUTPUT Vs TIME
CARDIAC OUTPUT ℓ/min
14
13
12
11
10
9
8
7
6
5
4
WARM UP
450
600
675
750
675
750
MAXIMUM WORK RATE Kgm/min


STROKE VOLUME Vs TIME
STROKE VOLUME ml
110
100
90
80
70
60
50
40
30
20
10
0
450
600
675
WARM UP
675
750
750
H.R. b/min
100
90
80
70
60
50
40
WARM UP
450
600
675
750
675
750
MAXIMUM WORK RATE Kgm/min


SERUM LIPIDS & WEIGHT Vs TIME
WEIGHT
100
Kg
90
CHOLESTEROL mg%
TRIGLYCERIDE mg%
400
350
300
250
200
300
250
200
150
100
CALISTHENICS-JOGGING
BIKE
0 2 4 6 8 10 12 14 16 18 20 22 24
TRAINING TIME - Weeks


TIDAL VOLUME
LUNG FUNCTION Vs TIME
2·0
ℓ
1·0
RESPIRATORY RATE
20
f/min
10
VITAL CAPACITY ℓ
FEV, ℓ
6·5
5·5
4·5
3·5
5·5
4·5
3·5
CALISTHENICS-JOGGING
BIKE
0 2 4 6 8 10 12 14 16 18 20 22 24
TRAINING TIME- Weeks

cent increase). After twenty weeks of the calisthenics-walk-jog program his PWC_{170} increased to 910 kpm/min (total increase of 21.4 per cent) and his PWC_{190} increased to 1030 kpm/min (total increase of 17.2 per cent). During the final six weeks continuous bicycle ergometry was the training method. This training was also done three times per week with walking on the two intermediate days. At the end of the twenty-four weeks his PWC_{170} had increased to 950 kpm/min (26.6 per cent above the initial level) and his PWC_{190} had increased to 1070 kpm/min (21.6 per cent above the initial level).

Up to the fourth week of training the subject could not exercise at a heart rate greater than 115 b/min without angina. Between the fourth and twelfth week this maximum rate, without angina, increased to 140 b/min; at this time the highest work rate of the test session was 675 kpm/min. Continuous training on a bicycle ergometer increased his maximum test work rate to 750 kpm/min without angina. After six weeks of training his doctor felt that he had improved to a point that he could return to work. This, plus the ability to exercise without angina, gave him increased confidence and allowed him to relax and enjoy his leisure time.

Heart rate - Figure 16a

Figure 16a shows heart rate changes at standard work rates. These decreased for all work rates. Warm-up heart rate did not change during the twenty-four weeks. The important improvement with this subject was his ability to perform at higher evaluation work rates (750 kpm/min) without angina or ST segment depression. He would not take medication for this ischemic heart condition. During the later stages of training he could cycle at a heart rate of 150-155 b/min for a period of twenty minutes. On

many bicycle training days, however, he did continue to develop angina at a heart rate of 140 b/min after seven to ten minutes of exercise. If the work rate was reduced to free pedalling for three to five minutes he could invariably complete the final twenty minutes at a heart rate of 150 b/min with no further angina. The severity was reduced and time of onset of the angina was also alleviated if extended warm-up was done. The most successful warm-up consisted of five minutes free pedalling at 50 rpm followed by an additional five minutes at 300 kpm/min. On many occasions in the later stages of training slight angina would develop, however, he could continue pedalling through this period. This appeared to be similar to the "walk-through" phenomena discussed by Kattus (1968).

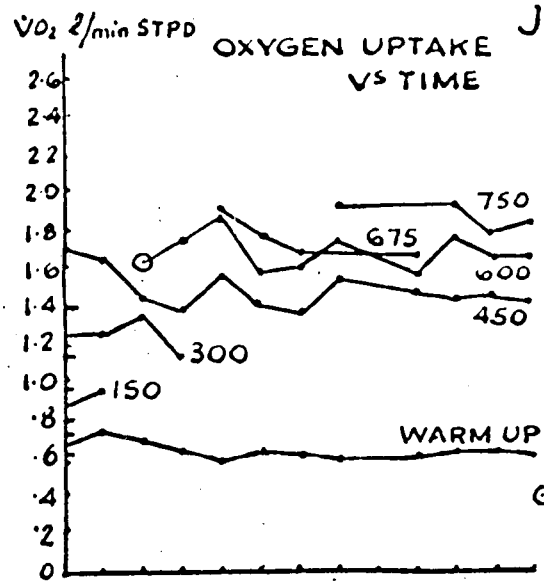
Cardiac output and stroke volume - Figure 16a

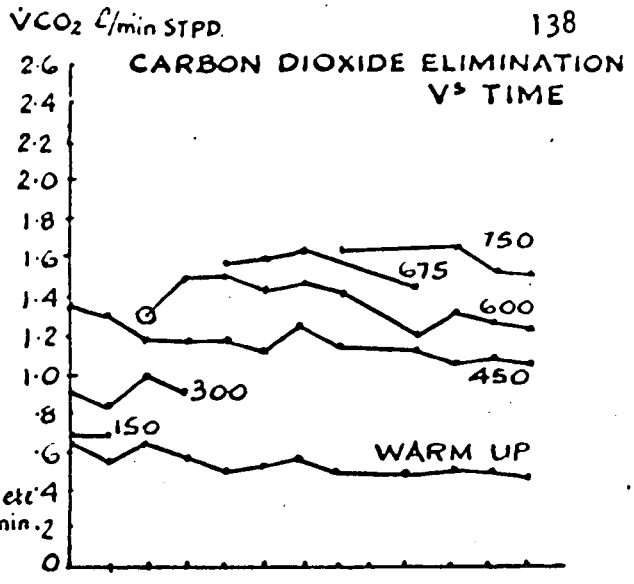
Warm-up-cardiac output did not change as a result of the rehabilitation program. Warm-up-stroke volume decreased after the second week test (97.6 → 85.4 ml). Exercise-cardiac output decreased with training. A considerable decrease was seen in the last six weeks at 750 kpm/min compared to the earlier tests at lower work rates. The final cardiac output at 750 kpm/min was 9.55 l/min whereas the values for the first test at 675 kpm/min was 12.10 l/min, 13.40 l/min at 600 kpm/min and 11.95 l/min at 450 kpm/min. Exercise-stroke volume was below the warm-up values and a decrease was observed with training at all work rates.

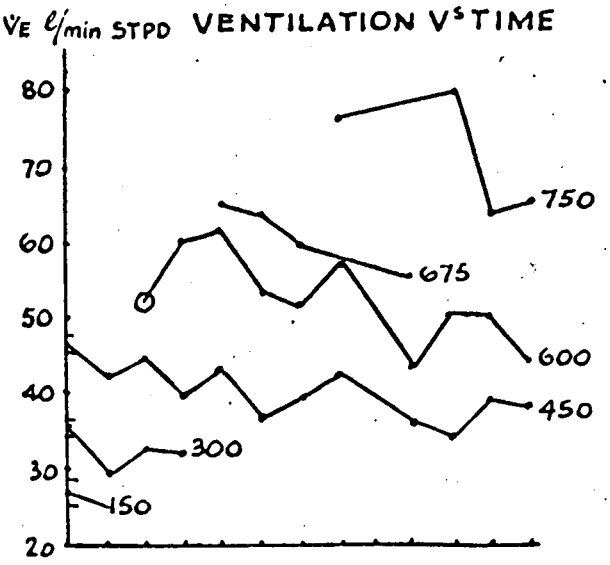
Respiratory gas exchange - Figure 16b

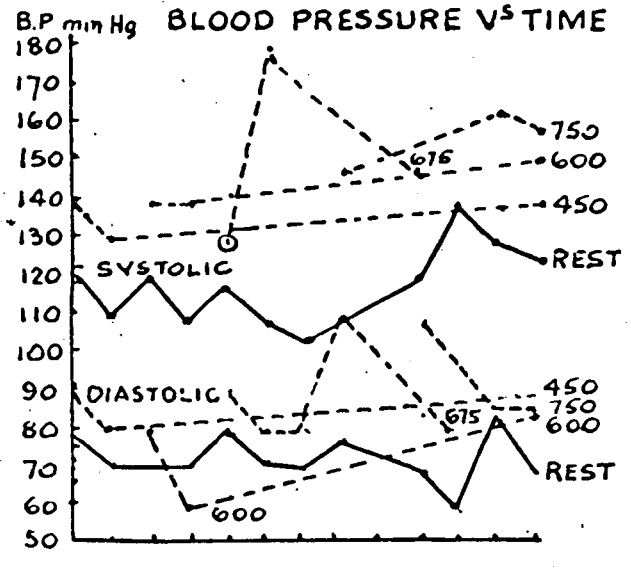
Exercise-ventilation decreased with training. Exercise $\dot{V}O_2$ and $\dot{V}CO_2$ also decreased with the training. Exercise R values initially increased up to week ten and then decreased during the following weeks of training.

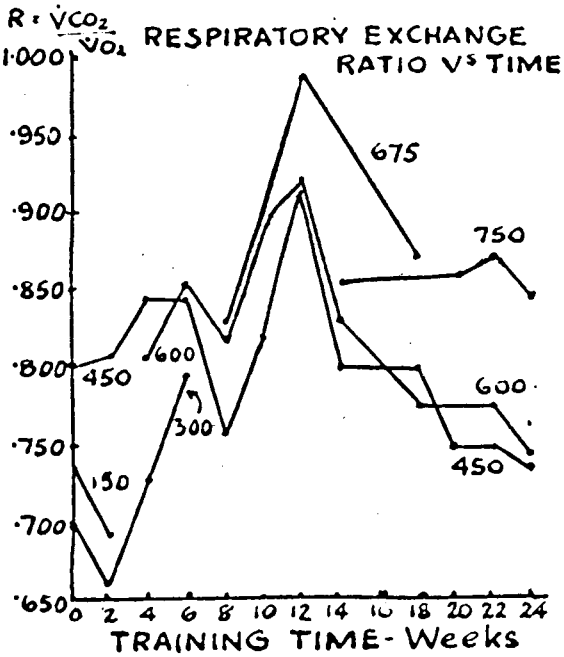
FIGURE 16b. Subject JD. Graphs of respiratory gas exchange, blood pressure and base excess for the 24 week rehabilitation period.

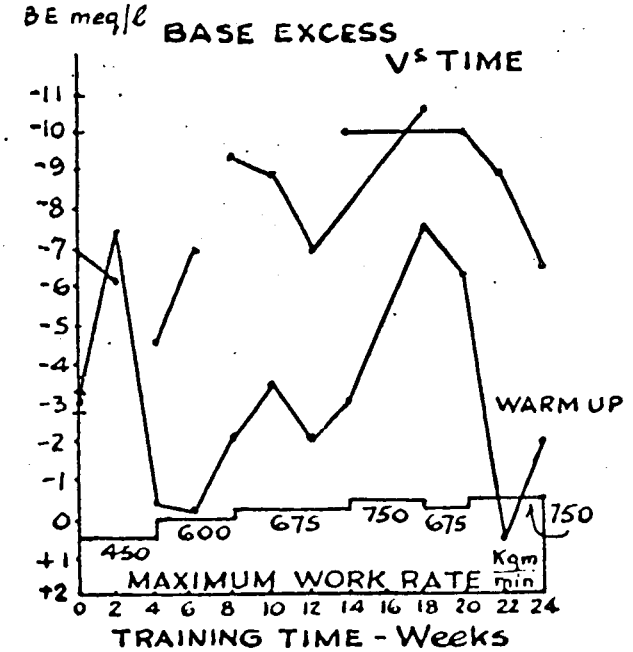

V̇O₂ ℓ/min STPD
OXYGEN UPTAKE V^S TIME
2·6
2·4
2·2
2·0
1·8
1·6
1·4
1·2
1·0
·8
·6
·4
·2
0
750
675
600
450
300
150
WARM UP


V̇CO₂ ℓ/min STPD
CARBON DIOXIDE ELIMINATION V^S TIME
2·6
2·4
2·2
2·0
1·8
1·6
1·4
1·2
1·0
·8
·6
·4
·2
0
750
675
600
450
300
150
WARM UP
⊙ Incomplete test · 5min.


V̇E ℓ/min STPD VENTILATION V^S TIME
80
70
60
50
40
30
20
750
675
600
450
300
150


B.P mm Hg BLOOD PRESSURE V^S TIME
180
170
160
150
140
130
120
110
100
90
80
70
60
50
SYSTOLIC
DIASTOLIC
750
675
600
450
REST


R = V̇CO₂ / V̇O₂ RESPIRATORY EXCHANGE RATIO V^S TIME
1·000
·950
·900
·850
·800
·750
·700
·650
675
750
600
450
300
150
0 2 4 6 8 10 12 14 16 18 20 22 24
TRAINING TIME - Weeks


BE meq/ℓ BASE EXCESS V^S TIME
-11
-10
-9
-8
-7
-6
-5
-4
-3
-2
-1
0
+1
+2
WARM UP
450
600
675
750
675
750
Kgm/min
MAXIMUM WORK RATE
0 2 4 6 8 10 12 14 16 18 20 22 24
TRAINING TIME - Weeks

Exercise oxygen pulse and ventilation equivalent - Table 10

At lower test work rates of 300 and 450 kpm/min, training did not appear to change consistently, values for oxygen pulse (0.0129 l/beat at 300 kpm/min and 0.0145 l/beat at 450 kpm/min). The ventilation equivalent for both of these work rates averaged 27 and this was not significantly changed with training. A small increase in oxygen pulse occurred with the calisthenics-walk-jog program at the 600 kpm/min work rate (0.0115 → 0.0124 l/beat) and a larger increase occurred during continuous bicycle ergometry training (0.0124 → 0.0152 l/beat). Training decreased the ventilation equivalent at this work rate (33 → 27). No change occurred in oxygen pulse at 750 kpm/min, however, a small change in ventilation equivalent was observed (40 → 36).

Base excess and lactate - Figure 16b and Tables 7 and 12

The warm-up base excess (BE) and lactate (La) was variable and exhibited no net change. Exercise-metabolic acidosis decreased (BE -10.0 → -6.5 meq/l, La 33.0 → 17.0 mg % at 750 kpm/min) from week twenty onwards. There was a variable response prior to this time.

Blood pressure - Figure 16b

The resting-systolic blood pressure decreased up to week twelve (122 → 104 mmHg) then returned to the initial level. The diastolic blood pressure (resting) remained constant. Exercise values of systolic pressure remained constant at lower work rates (450 kpm/min), decreased at 675 kpm/min sessions and remained unchanged for the 750 kpm/min tests. Diastolic pressure decreased for the 750 kpm/min tests but remained constant for all other work rates.

Myocardial oxygen demand - Table 14

Warm-up values of myocardial oxygen demand decreased up to week eighteen (1000 → 8315 mmHg X HR) and then returned to the initial values. The exercise values decreased for the 450 kpm/min test (17464 → 14820 mmHg X HR) and the 675 kpm/min test (25454 → 19720 mmHg X HR) however no change was seen during the ten weeks when the maximum work rate was 750 kpm/min at the test session.

Lung function - Figure 16a

Resting respiratory rate did not change however resting tidal volume increased (1.30 → 1.83 l). Both vital capacity (5.84 → 6.26 l) and FEV_1 (4.52 → 4.75 l) showed a small increase with the training.

Body weight and serum lipids - Figure 16a

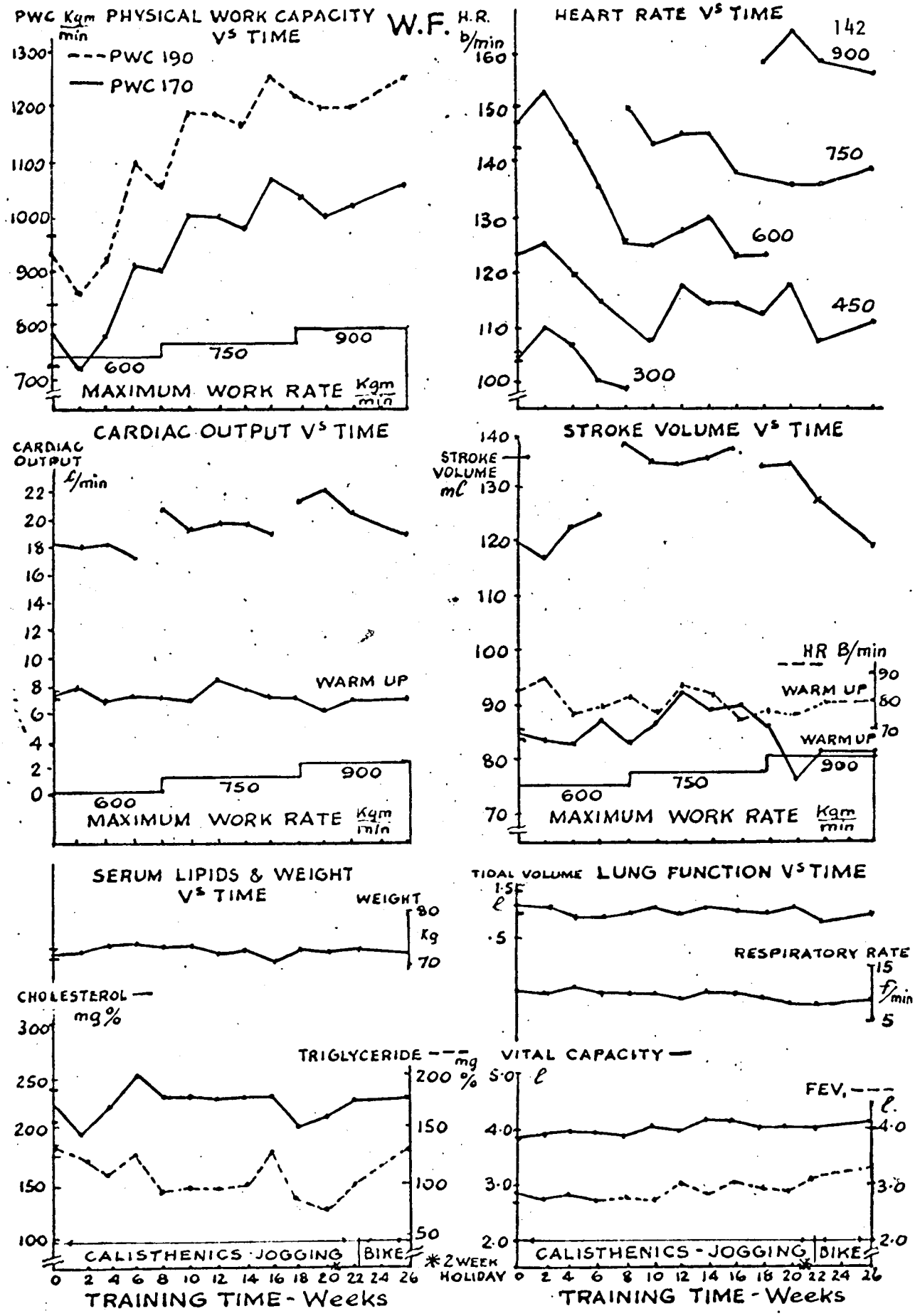
The training did not decrease his body weight and in fact a small increase occurred (90.6 → 93.1 kg). Serum cholesterol decreased (250 → 210 mg %) despite the small weight increase. The serum triglyceride level remained unchanged overall (150 mg %) after a transitory large initial increase at the second test session.

Subject WF Age 50 years Height 170.7 cm Weight 71.9 kg (initial) 72.0 kg (final) Started training seven months post infarction -- four weeks before initial tests.

Physical work capacity - Figure 17a

This subject had a mean initial PWC_{170} of 785 kpm/min and a PWC_{190} of 960 kpm/min. After ten weeks of the calisthenics-walk-jog program his PWC_{170} increased to (27 per cent) 1000 kpm/min and his PWC_{190} increased to (28.0 per cent) 1190 kpm/min. After a further ten weeks of this training

FIGURE 17a. Subject WF. Graphs of physical work capacity, heart rate, cardiac output, stroke volume, lung function, body weight and serum lipids for the 24 week rehabilitation period.


PWC Kgm/min PHYSICAL WORK CAPACITY Vs TIME
W.F.
---PWC 190
—— PWC 170
1300
1200
1100
1000
900
800
700
600
750
900
MAXIMUM WORK RATE Kgm/min
H.R. b/min
HEART RATE Vs TIME
160
150
140
130
120
110
100
142
900
750
600
450
300
CARDIAC OUTPUT Vs TIME
CARDIAC OUTPUT ℓ/min
22
20
18
16
14
12
10
8
6
4
2
0
WARM UP
600
750
900
MAXIMUM WORK RATE Kgm/min
STROKE VOLUME Vs TIME
STROKE VOLUME mℓ
140
130
120
110
100
90
80
70
HR B/min
WARM UP
90
80
70
WARM UP
600
750
900
MAXIMUM WORK RATE Kgm/min
SERUM LIPIDS & WEIGHT Vs TIME
WEIGHT
80
Kg
70
CHOLESTEROL —
mg%
300
250
200
150
100
TRIGLYCERIDE - - -
mg %
200
150
100
50
CALISTHENICS
JOGGING
BIKE
0 2 4 6 8 10 12 14 16 18 20 22 24 26
TRAINING TIME - Weeks
TIDAL VOLUME
LUNG FUNCTION Vs TIME
1·5ℓ
·5
RESPIRATORY RATE
15
f/min
5
VITAL CAPACITY —
5·0 ℓ
4·0
3·0
2·0
FEV1 - - -
ℓ.
4·0
3·0
2·0
CALISTHENICS
JOGGING
BIKE
*2 WEEK HOLIDAY
0 2 4 6 8 10 12 14 16 18 20 22 24 26
TRAINING TIME - Weeks

he had not increased his PWC_{170} and his PWC_{190} only increased to 1200 kpm/min (total increase of 29.0 per cent). A further month of bicycle ergometry training increased his PWC_{170} to 1060 kpm/min (total increase of 35.1 per cent) and his PWC_{190} to 1260 kpm/min (total increase of 35.5 per cent). Both his mood and his nervousness improved as training progressed.

Heart rate - Figure 17a

Figure 17a shows the heart rate decrement at a standard work rate during rehabilitation. During the first eight weeks the test sessions were 300, 450 and 600 kpm/min. These tests were increased to 450, 600 and 750 kpm/min during weeks 6 to 18 after which they were 450, 750 and 900 kpm/min. A heart rate decrement occurred in all work rates. However, the greatest rate of reduction occurred up to week 8 and after this a small but steady decrease took place until the end of the program. Warm-up-heart rate showed a decrease during the program (88 → 77 b/min).

After 20 weeks of training his physician felt that he had improved to the point that the use of a cardiotonic agent (digoxin) was no longer necessary.

Cardiac output and stroke volume - Figure 17a

Both warm-up-stroke volume (84.4 → 80.0 ml) and cardiac output (7.13 → 6.40 l/min) showed an overall decrease. A small increase occurred up to week 12 after which a decrease occurred. During tests at 600 kpm/min the stroke volume increased slightly (120 → 125 ml). Since the heart rate decreased concomitantly only a small change in cardiac output took place (18.00 → 17.00 l/min). During the later period when the maximum work rate was 750 kpm/min stroke volume remained constant and because the heart rate continued to decrease the cardiac output decreased from 20.63 l/min to

18.89 l/min. The stroke volume during the last month of training showed a large decrease at 900 kpm/min (134 → 119 ml) as did the exercise-cardiac output (22.00 → 18.59 l/min).

Respiratory gas exchange - Figure 17b

Exercise-ventilation showed only a small decrease at any particular work rate. Values for $\dot{V}O_2$ and $\dot{V}CO_2$ decreased for less strenuous work at 300 and 450 kpm/min throughout training, however, after an early initial decrease at higher work rates no further change was seen.

The respiratory exchange ratio during the tests was quite variable during the early period of training however the general trend was a decrease for the lower work rates (300 and 450 kpm/min). In the later stages of training no change occurred for the more strenuous tests (i. e. 750 and 900 kpm/min).

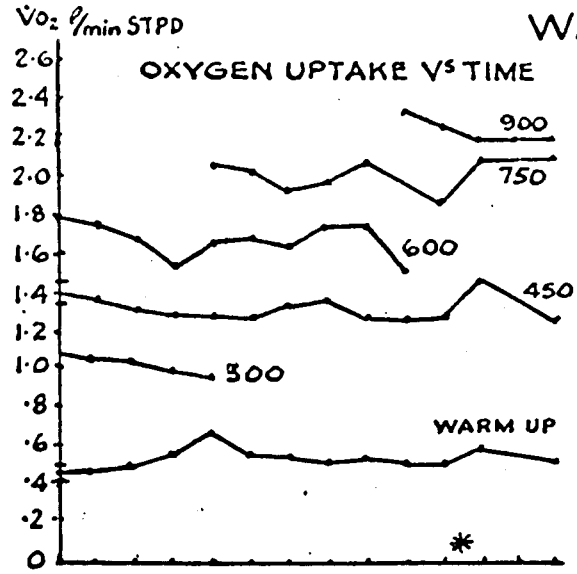
Exercise oxygen pulse and ventilation equivalent - Table 10

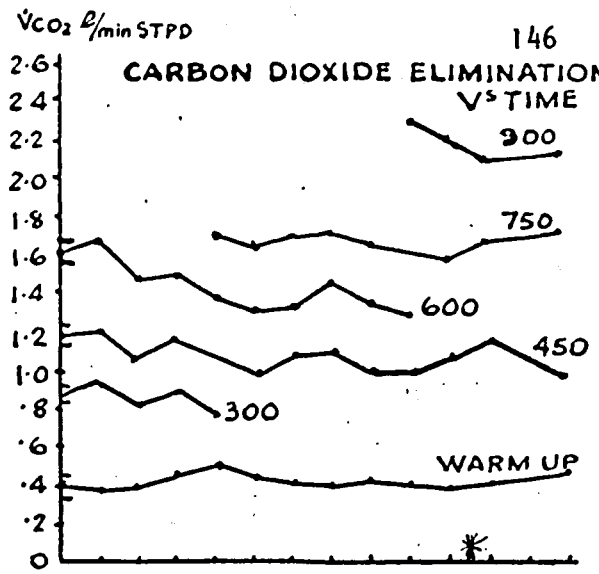
Values for ventilation equivalent remained unchanged throughout training of whatever type. The values for oxygen pulse remained unchanged for all work rates except 750 kpm/min. At this work rate an increase occurred after training (0.0140 → 0.0152 l/beat).

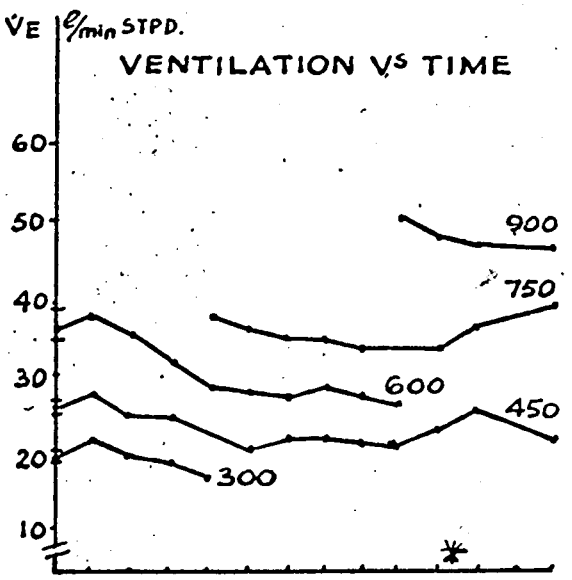
Base excess and lactate - Figure 17b and Tables 7 and 12

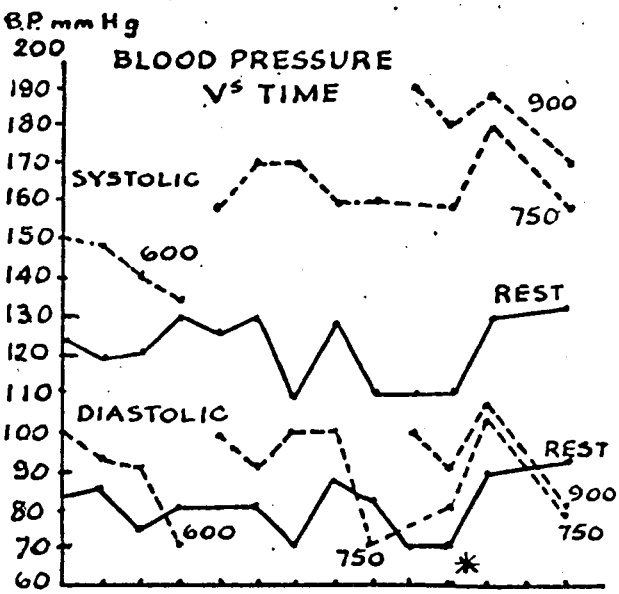
Warm-up and resting values for base excess (BE) and lactate (La) decreased (BE -3.0 → -1.2 meq/l, La 9.4 → 5.0 mg%). Exercise metabolic acidosis decreased during the first six weeks when 600 kpm/min was the maximum work rate (BE -8.7 → -2.0 meq/l, La 27.0 → 14.1 mg%). No change occurred when the rate was increased later to 750 kpm/min and the level of metabolic acidosis was considerably lower than observed at lower work rates during the initial tests (BE -4.4 → -4.1 meq/l, La 24.0 → 23.0 mg% at 750

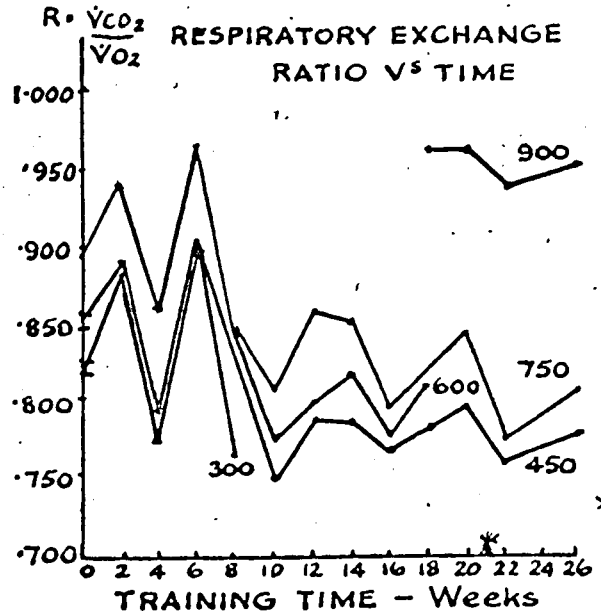
FIGURE 17b. Subject WF. Graphs of respiratory gas exchange, blood pressure and base excess for the 24 week rehabilitation period.

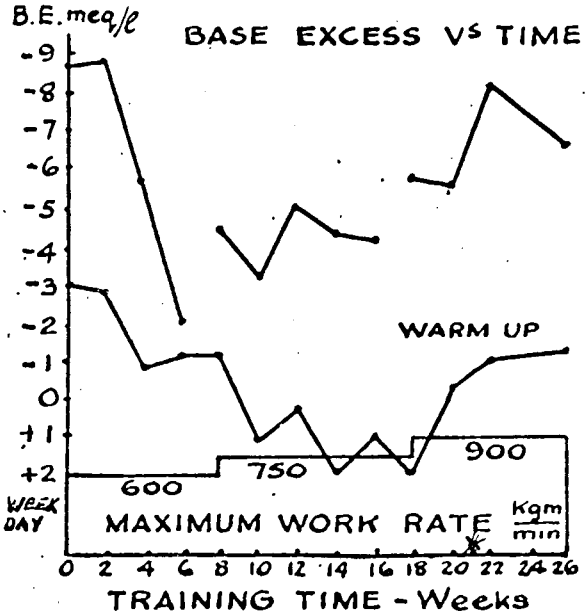

V̇O2 ℓ/min STPD
OXYGEN UPTAKE Vs TIME
2·6
2·4
2·2
2·0
1·8
1·6
1·4
1·2
1·0
·8
·6
·4
·2
0
900
750
600
450
300
WARM UP
*


V̇CO2 ℓ/min STPD
CARBON DIOXIDE ELIMINATION Vs TIME
2·6
2·4
2·2
2·0
1·8
1·6
1·4
1·2
1·0
·8
·6
·4
·2
0
900
750
600
450
300
WARM UP
*


V̇E ℓ/min STPD.
VENTILATION Vs TIME
60
50
40
30
20
10
900
750
600
450
300
*


B.P. mm Hg
BLOOD PRESSURE Vs TIME
200
190
180
170
160
150
140
130
120
110
100
90
80
70
60
SYSTOLIC
DIASTOLIC
900
750
600
REST
REST
900
750
600
750
*


R = V̇CO2 / V̇O2
RESPIRATORY EXCHANGE RATIO Vs TIME
1·000
·950
·900
·850
·800
·750
·700
900
750
600
450
300
0 2 4 6 8 10 12 14 16 18 20 22 24 26
TRAINING TIME – Weeks


B.E. meq/ℓ
BASE EXCESS Vs TIME
-9
-8
-7
-6
-5
-4
-3
-2
-1
0
+1
+2
WARM UP
900
750
600
* 2 WEEK HOLIDAY
MAXIMUM WORK RATE Kgm/min
0 2 4 6 8 10 12 14 16 18 20 22 24 26
TRAINING TIME - Weeks

kpm/min). The values at 900 kpm/min increased after two weeks of holiday but the lactate level was decreased during the next month of continuous bicycle ergometry (La 39.0 → 19.0 mg% at 900 kpm/min).

Blood pressure - Figure 17b

Resting systolic and diastolic values decreased with training until his holiday after which they returned to their initial level. Exercise values also decreased with training (150:100 → 134:70 at 600 kpm/min) and again an increase took place after his two week holiday. The final month of continuous bicycle ergometry was successful in decreasing blood pressure (188:108 → 170:80 at 900 kpm/min).

Myocardial oxygen demand - Table 14

The final warm-up values did not differ from the initial (10600 mmHg X HR) however a decrease was observed at weeks 14 to 20 (8500 mmHg X HR). Exercise values for the tension time index or myocardial oxygen demand decreased with training (22500 → 18224 mmHg X HR at 600 kpm/min; 23700 → 22080 mmHg X HR at 750 kpm/min and 30020 → 26860 mmHg X HR at 900 kpm/min).

Lung function - Figure 17a

No change occurred in the tidal volume or respiratory rate, however, small increases in vital capacity (3.89 → 4.12 l) and FEV_1 (2.87 → 3.30 l) were seen with the training.

Body weight and serum lipids - Figure 17a

Body weight, serum cholesterol and serum triglyceride levels all remained unchanged within the 24 weeks of training.

Subject EK Age 48 years Height 176.0 cm Weight 88.1 kg (initial) 86.6 kg (final) Started training six months post infarction -- four weeks before initial test.

Physical work capacity - Figure 18a

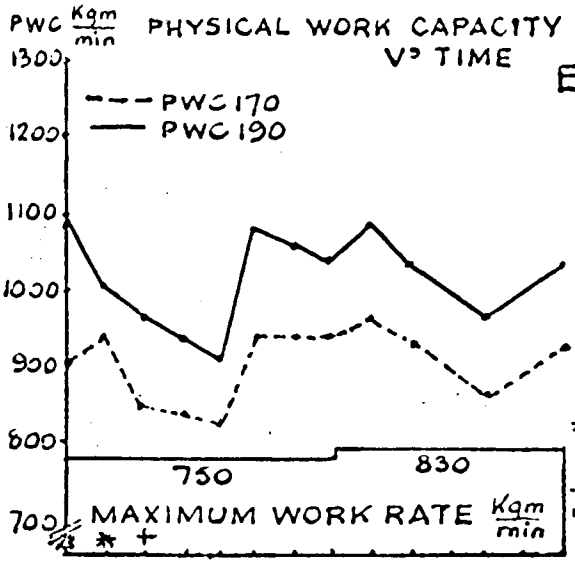
This subject had very weak legs and could not complete the six minute test at 750 kpm/min during his three initial tests and the first two serial tests which followed. At week four he completed four minutes compared to three minutes previous to this. During these tests, besides the problem with leg pain, he also complained of "tightness in the chest". For these reasons the tests were terminated early and the first two points on the graph are estimates only. His initial PWC_{170} was taken to be 840 kpm/min and his PWC_{190}, 960 kpm/min. After ten weeks of testing his PWC_{170} increased to 930 kpm/min (10.70 per cent increase) and his PWC_{190} increased to 1070 kpm/min (11.5 per cent increase). No further increase took place in his work capacity and at the end of the calisthenics-walk-jog program his PWC_{170} was 920 kpm/min and his PWC_{190} was 1025 kpm/min. Two weeks absence from training occurred before he started bicycle ergometry training. At this time his PWC_{170} had decreased to 860 kpm/min and his PWC_{190} to 950 kpm/min. After one month of continuous training his PWC_{170} increased again to 910 kpm/min and his PWC_{190} to 1020 kpm/min. Since week 14 he had been able to complete the full six minutes of work at a rate of 830 kpm/min with no leg pain or tightness in his chest.

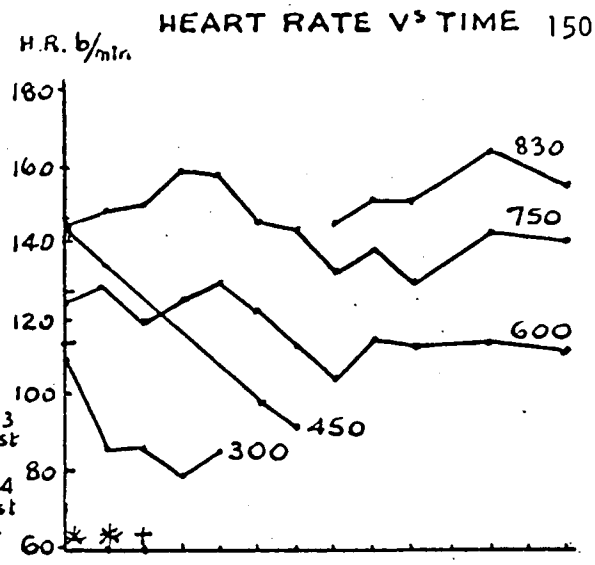
Heart rate - Figure 18a

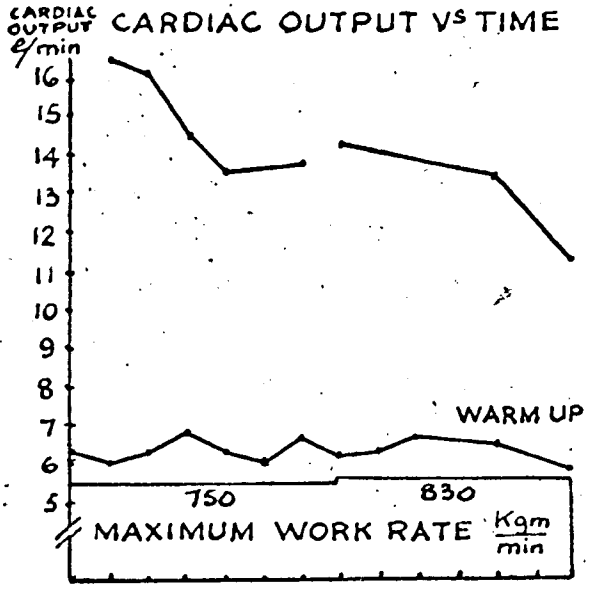
Heart rate values for a particular work rate decreased (except at 830 kpm/min) after the eighth week of testing and continued decreasing to week 18. After two weeks absence the heart rate for equivalent work increased. The increased heart rate for standard work was then followed by another decrease

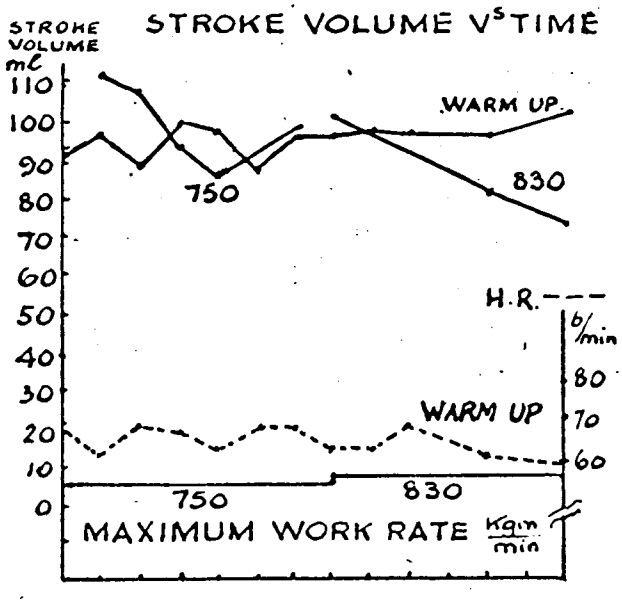
FIGURE 18a. Subject EK. Graphs of physical work capacity, heart rate, cardiac output, stroke volume, lung function, body weight and serum lipids for the 24 week rehabilitation period.

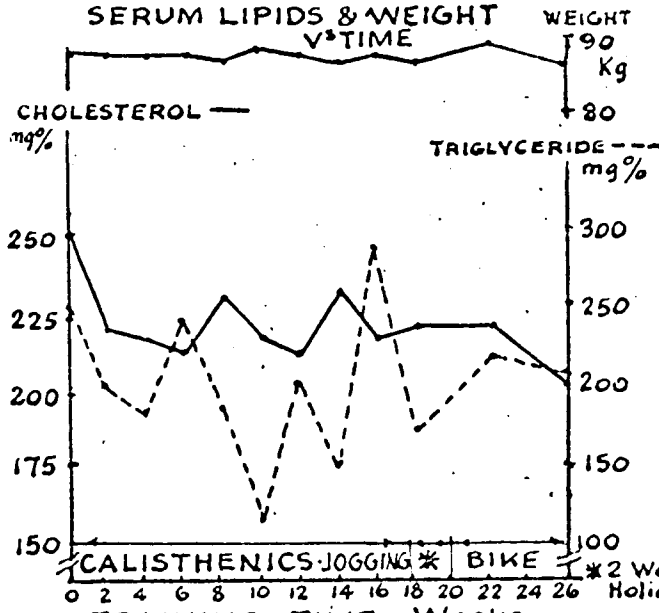
E.K.

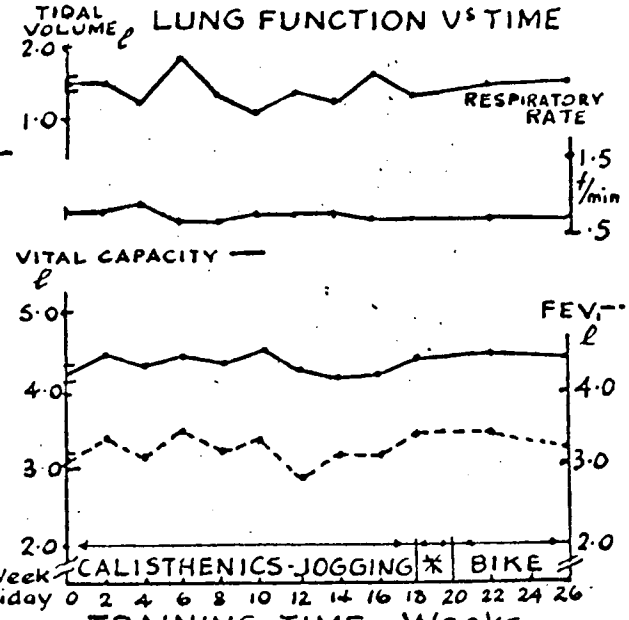

PWC Kgm/min
PHYSICAL WORK CAPACITY Vs TIME
PWC 170
PWC 190
1300
1200
1100
1000
900
800
700
750
830
MAXIMUM WORK RATE Kgm/min
* Only 3 min. last load.
+ Only 4 min. last load.


H.R. b/min
HEART RATE Vs TIME
180
160
140
120
100
80
60
830
750
600
450
300


CARDIAC OUTPUT l/min
CARDIAC OUTPUT Vs TIME
16
15
14
13
12
11
10
9
8
7
6
5
WARM UP
750
830
MAXIMUM WORK RATE Kgm/min


STROKE VOLUME mℓ
STROKE VOLUME Vs TIME
110
100
90
80
70
60
50
40
30
20
10
0
WARM UP
750
830
H.R. - - -
b/min
80
70
60
WARM UP
750
830
MAXIMUM WORK RATE Kgm/min


SERUM LIPIDS & WEIGHT Vs TIME
WEIGHT
90
Kg
80
CHOLESTEROL —
mg%
TRIGLYCERIDE - - -
mg%
250
225
200
175
150
300
250
200
150
100
CALISTHENICS
JOGGING
*
BIKE
*2 Week Holiday
0 2 4 6 8 10 12 14 16 18 20 22 24 26
TRAINING TIME - Weeks


TIDAL VOLUME ℓ
LUNG FUNCTION Vs TIME
2·0
1·0
RESPIRATORY RATE
1·5
1/min
·5
VITAL CAPACITY —
ℓ
FEV1 - -
ℓ
5·0
4·0
3·0
2·0
4·0
3·0
2·0
CALISTHENICS
JOGGING
*
BIKE
0 2 4 6 8 10 12 14 16 18 20 22 24 26
TRAINING TIME - Weeks

by the end of continuous bicycle ergometry training. The largest decrease of heart rate occurred at the last month of the lower work rates of 300 and 450 kpm/min. Training did not decrease exercise-heart rate values at 830 kpm/min. Warm-up-heart rate varied between 65 and 70 b/min until after test 18 after which it progressively decreased to 60 b/min.

Cardiac output and stroke volume - Figure 18a

This subject had difficulty with the carbon dioxide rebreathing used for the determination of cardiac output. Although a resting plateau could be obtained it was difficult or impossible to obtain an exercise value in some tests.

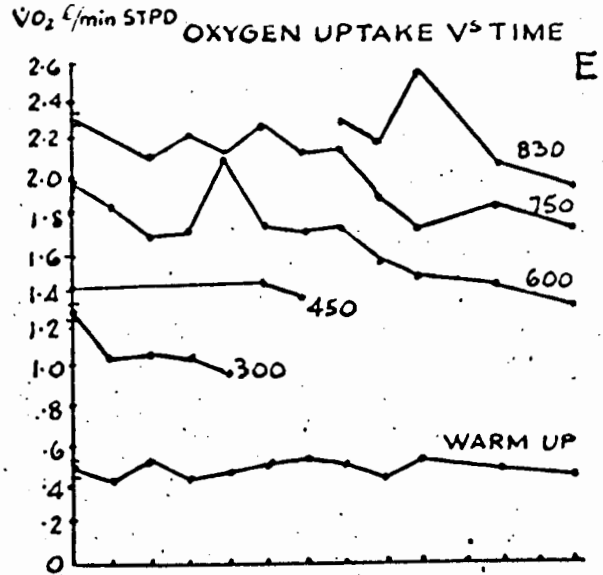
Warm-up stroke volume increased with training (91.7 → 101.0 ml). Warm-up cardiac output did not show consistent changes with training (range 5.95 → 6.86 l/min). Exercise did not increase stroke volume above the warm-up level. For a standard work rate stroke volume decreased with training (111 → 92 ml at 750 kpm/min and 101 → 73 ml at 830 kpm/min). Mirroring the decreased stroke volume and heart rate, cardiac output decreased considerably with training (16.50 → 13.85 l/min at 750 kpm/min and 14.70 → 11.34 l/min at 830 kpm/min). The largest rate of decrease occurred during the period of bicycle ergometry training.

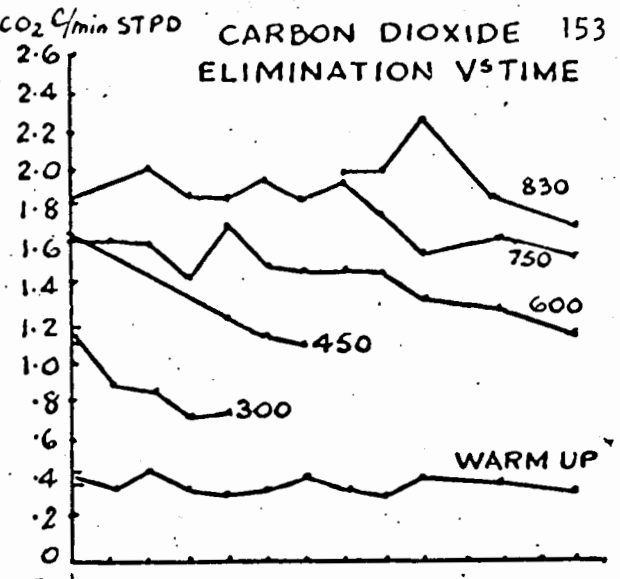
Respiratory gas exchange - Figure 18b

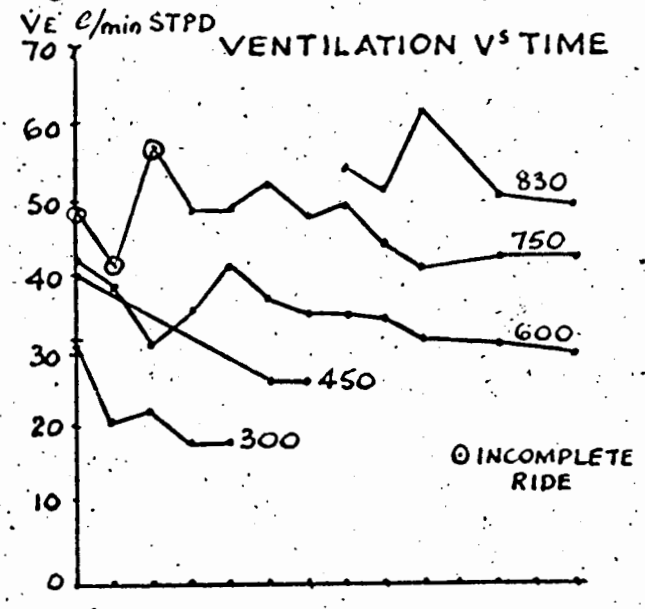
Exercise ventilation for standard work rates decreased with training. The largest decreases occurred with the lower work rates. Training also resulted in consistent decreases in exercise values for $\dot{V}O_2$ and $\dot{V}CO_2$. The relationship and degree of these decreases can be seen in Figure 18b showing the respiratory exchange ratios for a particular work rate during the training. The R values showed large decreases up to the test at week

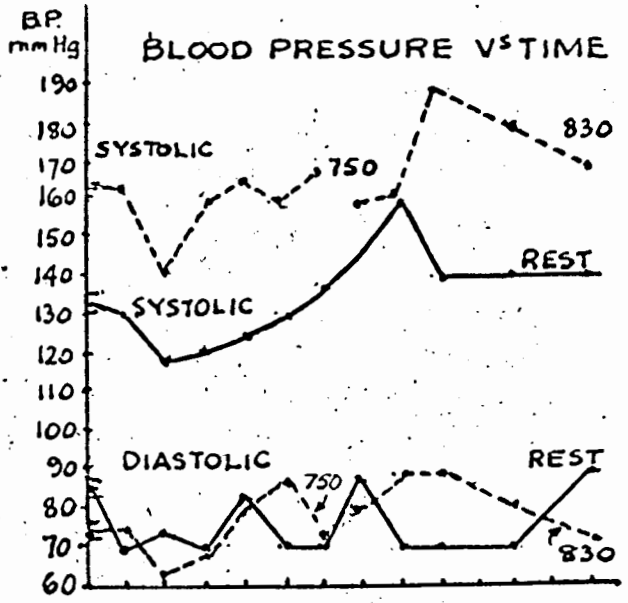
FIGURE 18b. Subject EK. Graphs of respiratory gas exchange, blood pressure and base excess for the 24 week rehabilitation period.

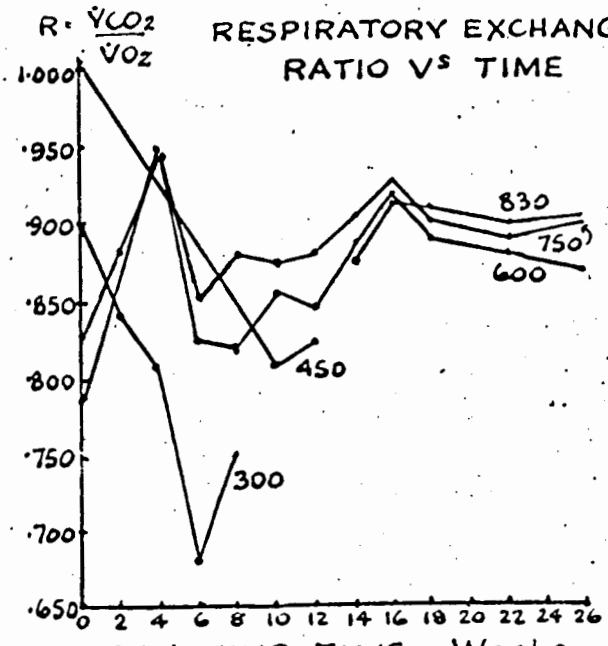
E.K.

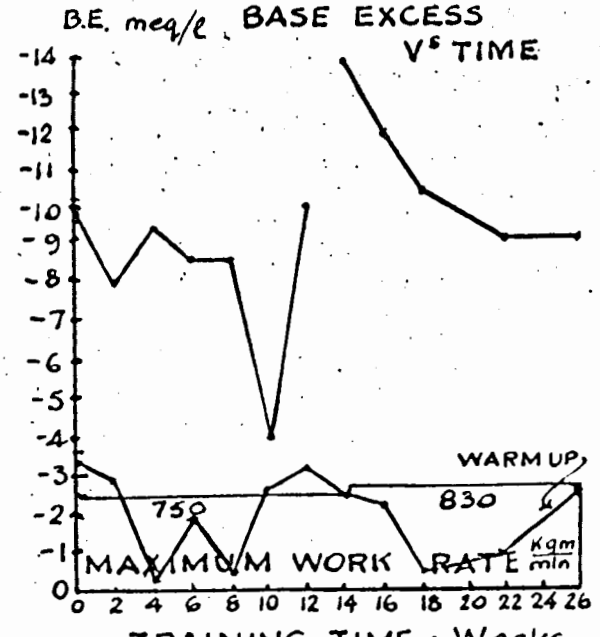

V̇O₂ ℓ/min STPD
OXYGEN UPTAKE Vs TIME
2·6
2·4
2·2
2·0
1·8
1·6
1·4
1·2
1·0
·8
·6
·4
·2
0
830
750
600
450
300
WARM UP


V̇CO₂ ℓ/min STPD
CARBON DIOXIDE ELIMINATION Vs TIME
2·6
2·4
2·2
2·0
1·8
1·6
1·4
1·2
1·0
·8
·6
·4
·2
0
830
750
600
450
300
WARM UP


V̇E ℓ/min STPD
VENTILATION Vs TIME
70
60
50
40
30
20
10
0
830
750
600
450
300
⊙ INCOMPLETE RIDE


B.P. mm Hg
BLOOD PRESSURE Vs TIME
190
180
170
160
150
140
130
120
110
100
90
80
70
60
SYSTOLIC
830
750
REST
SYSTOLIC
DIASTOLIC
750
REST
830


R = V̇CO₂ / V̇O₂
RESPIRATORY EXCHANGE RATIO Vs TIME
1·000
·950
·900
·850
·800
·750
·700
·650
0 2 4 6 8 10 12 14 16 18 20 22 24 26
830
750
600
450
300
TRAINING TIME - Weeks


B.E. meq/ℓ
BASE EXCESS Vs TIME
-14
-13
-12
-11
-10
-9
-8
-7
-6
-5
-4
-3
-2
-1
0
0 2 4 6 8 10 12 14 16 18 20 22 24 26
WARM UP
750
830
MAXIMUM WORK RATE Kgm/min
TRAINING TIME - Weeks

ten, they then increased steadily to week sixteen and during the period of continuous ergometry training the R values showed a small decrease.

Exercise oxygen pulse and ventilation equivalent - Table 10

Exercise training decreased the values of ventilation equivalent in the lower work rates of 300 and 450 kpm/min (24 → 19 at 300 kpm/min and 29 → 19 at 450 kpm/min) however no change was observed in the higher test work rates (22 at 600 kpm/min; 23 at 750 kpm/min and 24 at 830 kpm/min). Values for oxygen pulse were variable, mirroring the rather variable heart rate changes. The general pattern for most work rates was an increase in value of oxygen pulse up to week 14 and after this point a decrease occurred. Figure 18a shows the subject's decreased heart rate up to week 14 and the slight increase thereafter. The following changes are from week 14 to the final values: 0.0161 → 0.0120 l/beat at 600 kpm/min; 0.0163 → 0.0123 l/beat at 750 kpm/min and 0.0158 → 0.0123 l/beat at 830 kpm/min.

Base excess and lactate - Figure 18b and Tables 7 and 12

No change was seen in the warm-up values of base excess (BE) or lactate (La). The exercise values remained unchanged until week 16 after which a steady decrease in the level of metabolic acidosis for a standard work rate occurred until the end of training (BE -13.8 → -9.2 meq/l, La 74.0 → 25.0 mg% at 830 kpm/min).

Blood pressure - Figure 18b

Rest-systolic blood pressure decreased up to week four (133 → 120 mmHg). This initial decrease was followed by a progressive increase up to week 16 (120 → 160 mmHg) and then decreased and was maintained at 140 mmHg. Except for three tests the resting-diastolic blood pressure decreased with training

from 90 to 70 mmHg. Exercise-systolic and diastolic pressures were unchanged except for an increase after his absence from training. A decrease followed establishment of continuous training.

Myocardial oxygen demand - Table 14

The warm-up-myocardial oxygen demand decreased from 9480 mmHg X HR to 8190 mmHg X HR at week eight, then increased steadily to 10400 mmHg X HR at week 16 and finally decreased at the end to 8400 mmHg X HR. Exercise values remained unchanged for the 750 kpm/min tests (24000 mmHg X HR), however, they increased for the 830 kpm/min from 24150 to 28800 mmHg X HR (weeks 18 and 22) and decreased to 26350 mmHg X HR at the end of the program.

Lung function - Figure 18a

No significant changes occurred in the four lung function parameters studied.

Body weight and serum lipids - Figure 18a

Body weight remained unchanged with the training however serum cholesterol decreased (250 → 200 mg%). The decrease occurred during weeks 0-2 and 22-26 (ergometry training) but no change occurred between these times. Serum triglyceride levels showed considerable variability however the general trend was a decrease (initial 260 mg% → final 202 mg%). A decrease occurred up to week 10 (161 mg%) followed by an increase up to week 16 and then the final decrease.

Subject BQ Age 41 years Height 175.0 cm Weight 74.5 kg (initial) 73.1 kg (final) Started training seven months post infarction.

Physical work capacity - Figure 19a

The subject's mean initial PWC_{170} was 915 kpm/min and the PWC_{190} was 1060 kpm/min. By the tenth week of the calisthenics-walk-jog program he had increased his PWC_{170} to 1060 kpm/min (15.85 per cent) and his PWC_{190} to 1210 kpm/min (14.25 per cent). At the end of this form of training his PWC_{170} was 1200 kpm/min (total increase of 31.2 per cent) and his PWC_{190} was 1380 kpm/min (total increase of 30.2 per cent). The final values after the period of continuous ergometry training were 1260 kpm/min for PWC_{170} (total increase of 37.8 per cent) and 1430 kpm/min for PWC_{190} (total increase of 35.0 per cent). His physical work capacity had reached a plateau at the end of the calisthenic-walk-jog program but a month of continuous ergometry produced an increase.

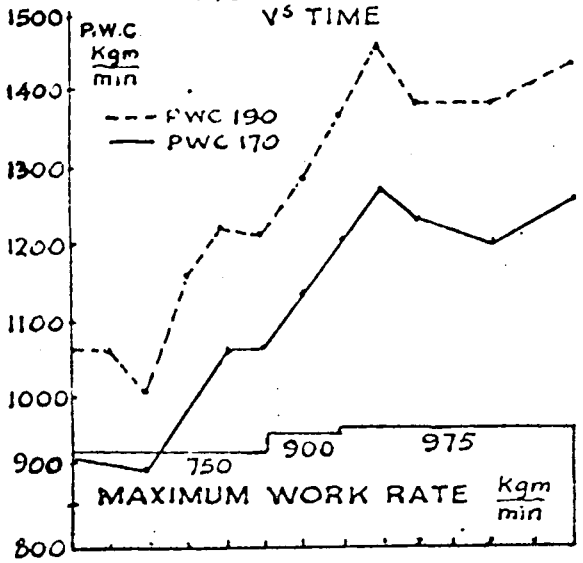
Heart rate - Figure 19a

At work rates up to 750 kpm/min a large decrement in heart rate occurred with training. After a decrease up to week 16 at the more strenuous work rates of 900 and 975 kpm/min, a gradual increase occurred up to week 22, after which a decrease followed with the introduction of continuous ergometry training.

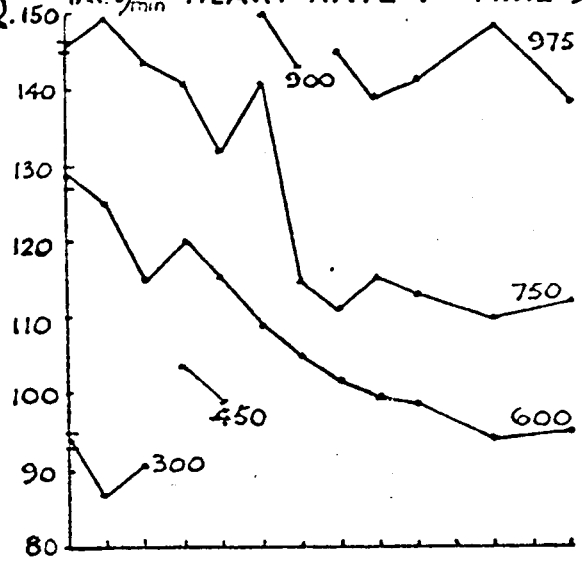
This subject had been placed on quinidine sulphate medication for the treatment of cardiac arrhythmias. As his training progressed the quantity of this medication was gradually removed until week seven when he was tested with no medication. During the recovery period, after the first evaluation work rate, a series of premature beats were present in his ECG. The evaluation test was stopped and he was placed back on moderate medication. He continued training under telemetry monitoring and no further arrhythmias appeared. At week 22 the medication was again removed, however, arrhythmias were again present during training so he completed the 24 weeks with a reduction but not

FIGURE 19a. Subject BQ. Graphs of physical work capacity, heart rate, cardiac output, stroke volume, lung function, body weight and serum lipids for the 24 week rehabilitation period.

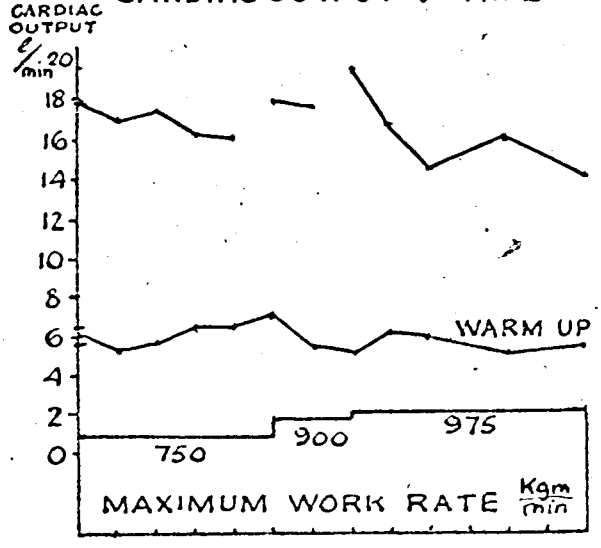
PHYSICAL WORK CAPACITY Vs TIME



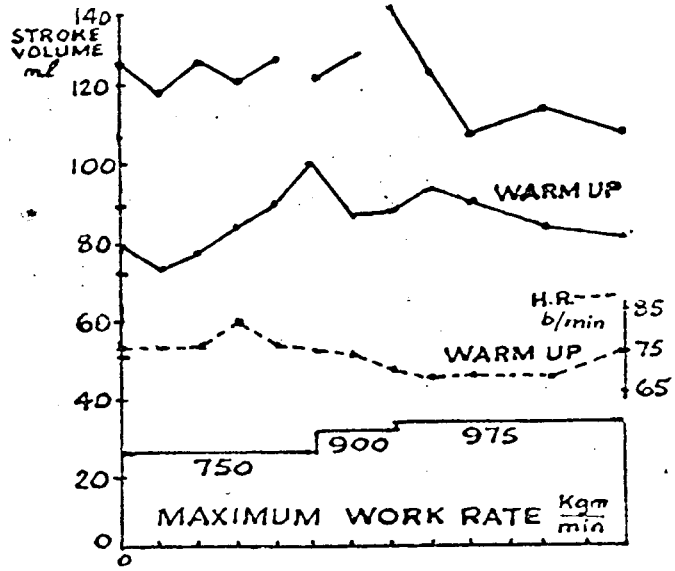
HEART RATE Vs TIME



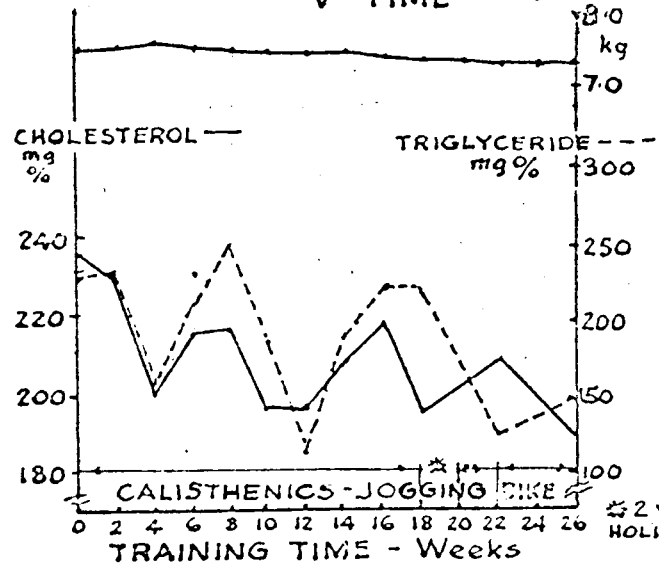
CARDIAC OUTPUT Vs TIME



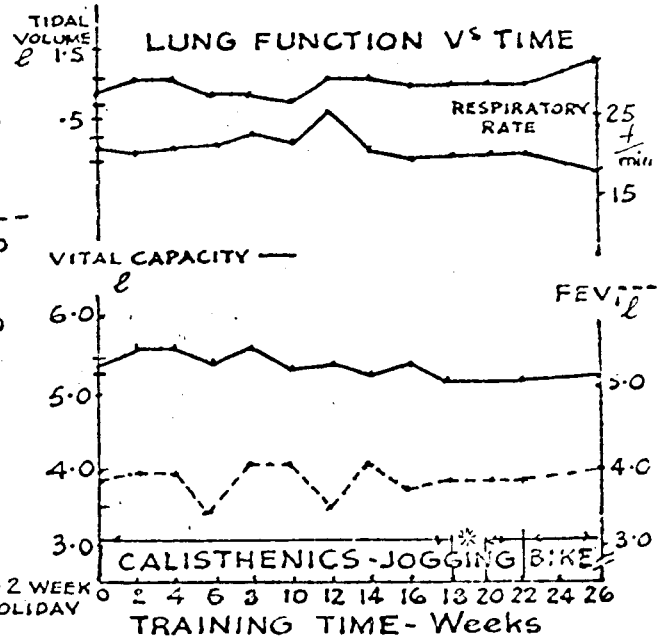
STROKE VOLUME Vs TIME



SERUM LIPIDS & WEIGHT Vs TIME



LUNG FUNCTION Vs TIME



total removal of medication.

Cardiac output and stroke volume - Figure 19a

The final result of training did not change either warm-up-stroke volume or cardiac output, however, an increase did occur in both, up to week ten. Following this, a decrease occurred to the initial values. Exercise-stroke volume remained unchanged up to week 14 and then showed a decrease (140 → 106 ml at 975 kpm/min). Cardiac output during the tests at 750 kpm/min (18.49 → 17.01 l/min) and 975 kpm/min (20.28 → 14.56 l/min) decreased.

Respiratory gas exchange - Figure 19b

During the training period exercise-ventilation decreased at lower work rates. However, it did not change for tests at 900 and 975 kpm/min. The oxygen uptake and carbon dioxide elimination, after a period of time, decreased for all work rates. As the R values suggest, the largest decreases occurred within the $\dot{V}O_2$ value because the R values for all loads increased after the fourth week.

Exercise oxygen pulse and ventilation equivalent - Table 10

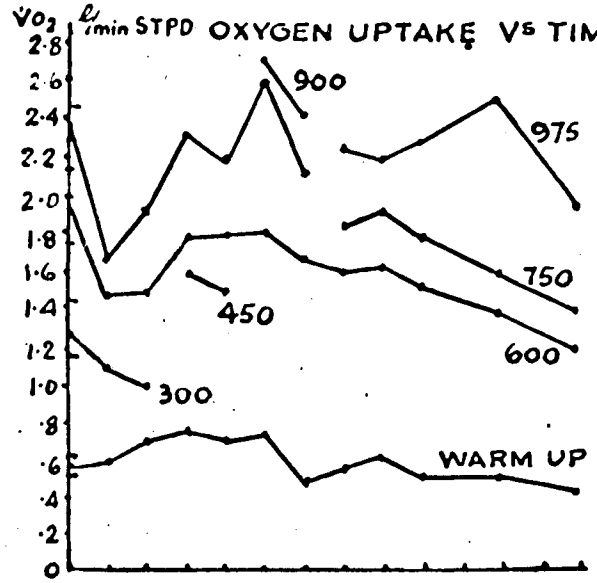
Although small changes were observed over the 24 week period the overall effect was that in this subject exercise training had no significant effect on the ratios of oxygen pulse and ventilation equivalent for any test work rate. It was noted that at the lowest work rate of 300 kpm/min, which was used for tests from the initial to the fourth week of training, the values for ventilation equivalent were high (32) compared to 600 kpm/min (average 25) or 750 kpm/min (average 26).

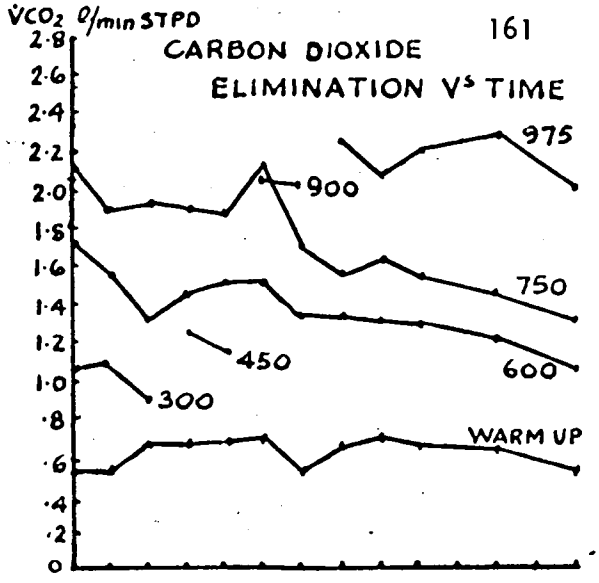
Base excess and lactate - Figure 19b and Tables 7 and 12

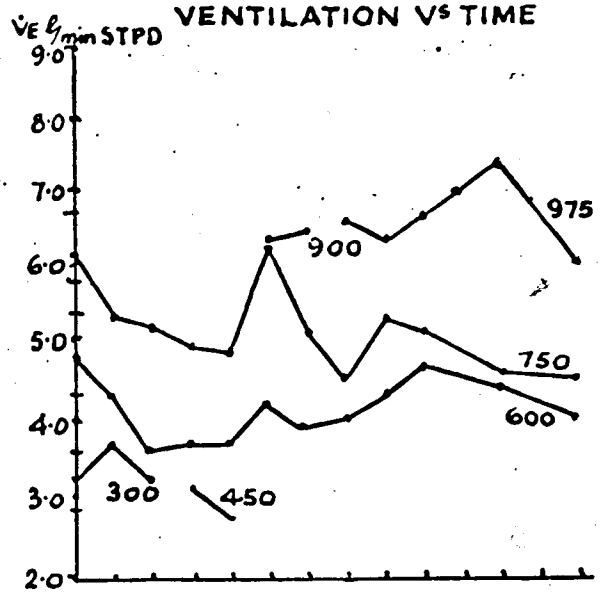
No change occurred with warm-up values for base excess (BE) and lactate

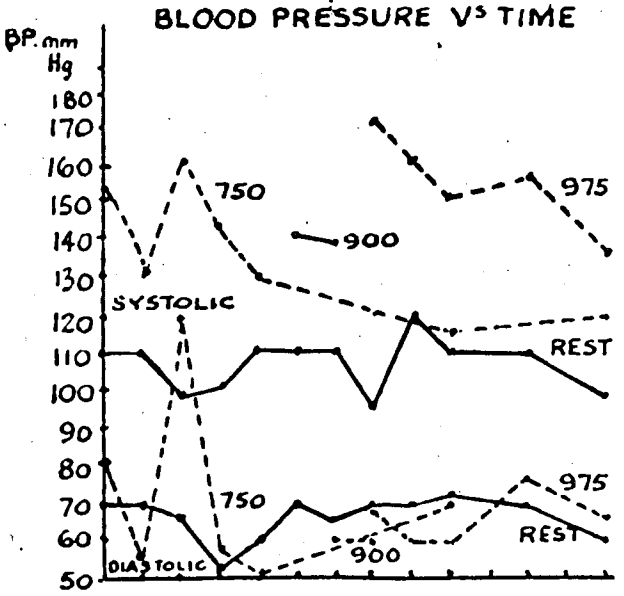
FIGURE 19b. Subject BQ. Graphs of respiratory gas exchange, blood pressure and base excess for the 24 week rehabilitation period.

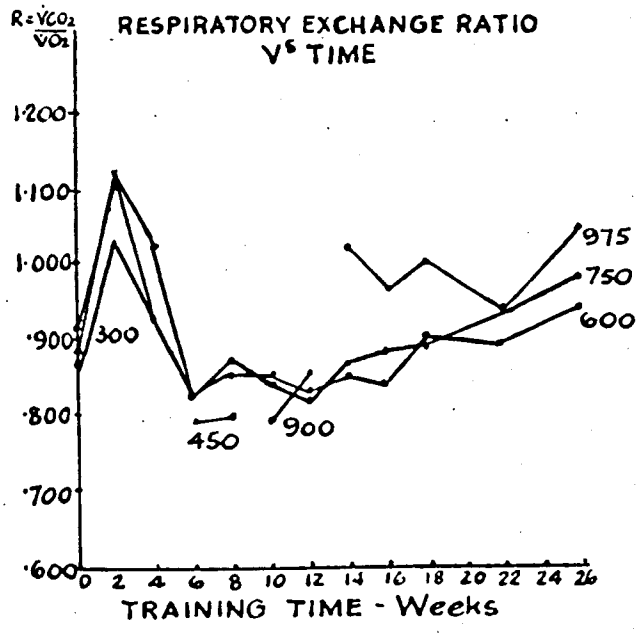
B.Q.

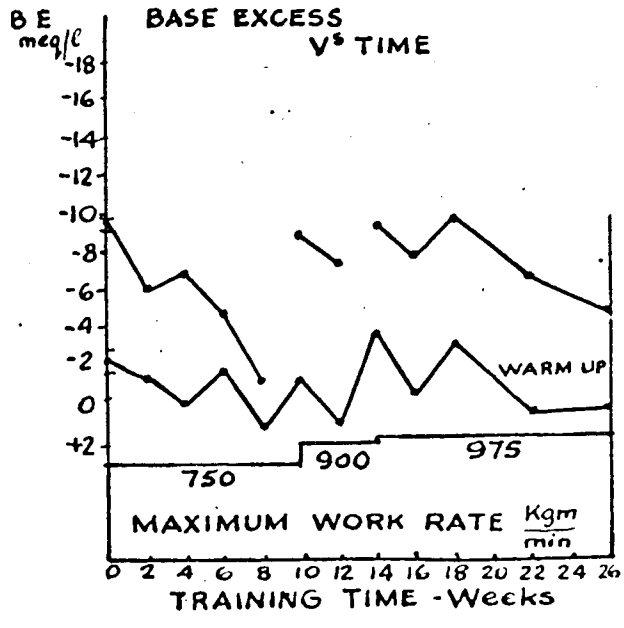

$\dot{V}O_2$ ℓ/min STPD OXYGEN UPTAKE Vs TIME
2·8
2·6
2·4
2·2
2·0
1·8
1·6
1·4
1·2
1·0
·8
·6
·4
·2
0
900
975
750
450
600
300
WARM UP


$\dot{V}CO_2$ ℓ/min STPD
CARBON DIOXIDE ELIMINATION Vs TIME
2·8
2·6
2·4
2·2
2·0
1·8
1·6
1·4
1·2
1·0
·8
·6
·4
·2
0
975
900
750
450
600
300
WARM UP


$\dot{V}E$ ℓ/min STPD
VENTILATION Vs TIME
9·0
8·0
7·0
6·0
5·0
4·0
3·0
2·0
975
900
750
600
300
450


BP. mm Hg
BLOOD PRESSURE Vs TIME
180
170
160
150
140
130
120
110
100
90
80
70
60
50
750
975
900
SYSTOLIC
REST
750
975
900
REST
DIASTOLIC


$R = \frac{\dot{V}CO_2}{\dot{V}O_2}$
RESPIRATORY EXCHANGE RATIO Vs TIME
1·200
1·100
1·000
·900
·800
·700
·600
975
750
600
300
450
900
0 2 4 6 8 10 12 14 16 18 20 22 24 26
TRAINING TIME - Weeks


B E meq/ℓ
BASE EXCESS Vs TIME
-18
-16
-14
-12
-10
-8
-6
-4
-2
0
+2
WARM UP
750
900
975
MAXIMUM WORK RATE Kgm/min
0 2 4 6 8 10 12 14 16 18 20 22 24 26
TRAINING TIME - Weeks

(La), however, exercise values were decreased with training at all work rates (BE -9.8 → -1.3 meq/l and La 48.0 → 15.9 mg% at 750 kpm/min; BE -8.8 → -7.7 meq/l and La 34.0 → 35.0 mg% at 900 kpm/min and BE -9.5 → -4.9 meq/l and La 37.0 → 16.0 mg% at 975 kpm/min).

Blood pressure - Figure 19b

Both the resting diastolic and systolic blood pressure remained unchanged. Exercise-systolic values showed a large decrease (158 → 120 mmHg at 750 kpm/min and 170 → 136 mmHg at 975 kpm/min) while exercise-diastolic values remained largely unchanged.

Myocardial oxygen demand - Table 14

Warm-up-myocardial oxygen demand showed a small but consistent decrease with training (8360 → 7350 mmHg X HR). The exercise values, after a period of time, decreased with training for each load (22400 → 17160 mmHg X HR at 750 kpm/min; 21000 → 19734 mmHg X HR at 900 kpm/min and 24650 → 18800 mmHg X HR at 975 kpm/min).

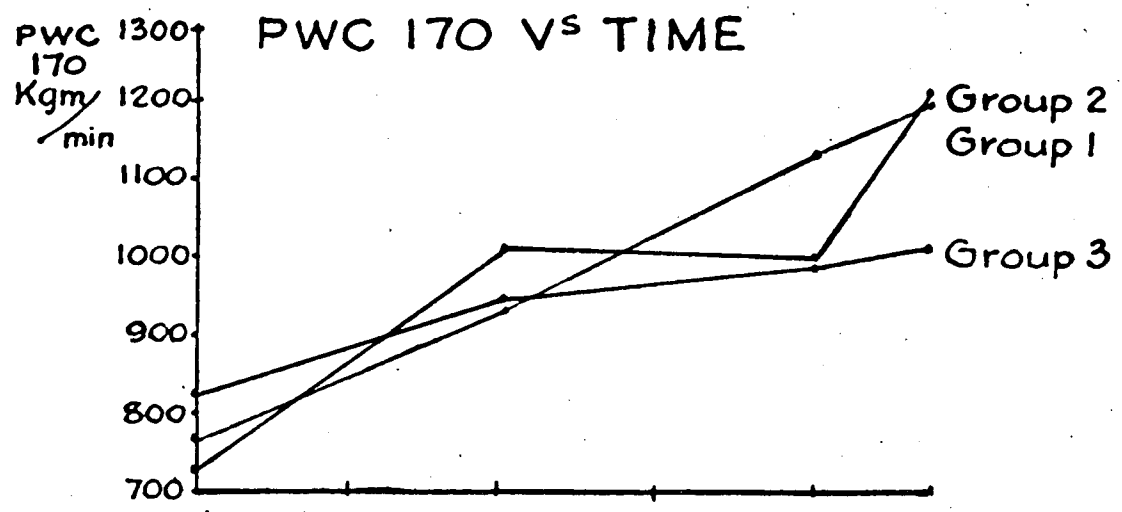
Lung function - Figure 19a

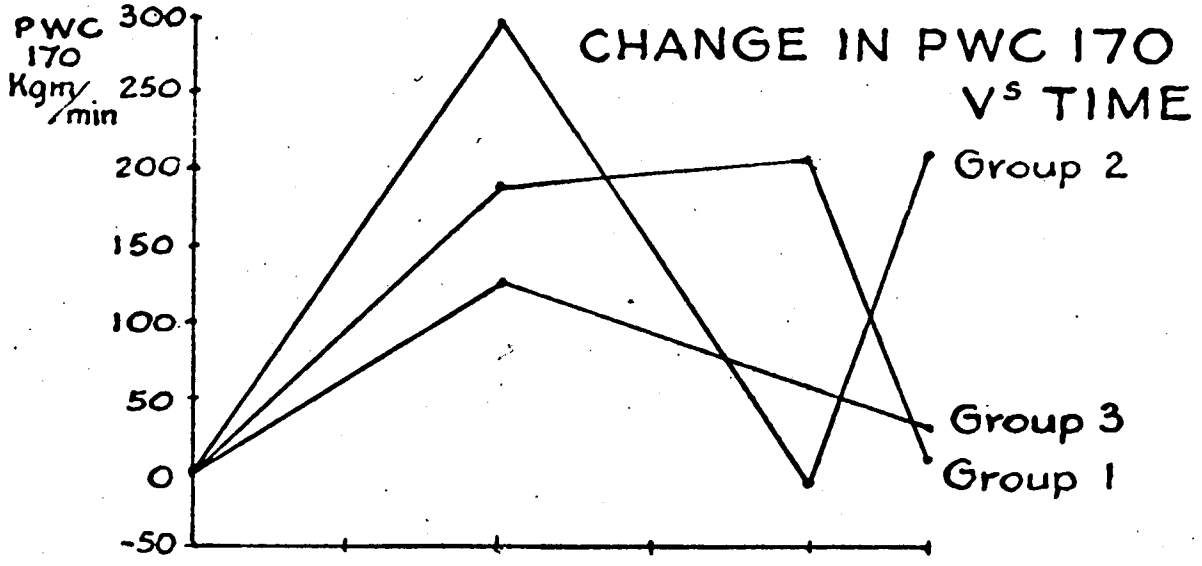
Training did not change the vital capacity, FEV_1 or the resting tidal volume. The resting respiratory rate decreased (23 → 18 breaths/min) with training.

Body weight and serum lipids - Figure 19a

Body weight remained constant throughout the 24 week training period but serum cholesterol decreased overall (235 → 190 mg%). Serum triglyceride levels decreased (221 → 147 mg%) with training although this was not a consistent decrease but fluctuated during the first 12 weeks of training.

FIGURE 20. Graphs showing the mean, the mean change and the mean per cent change above the initial value of physical work capacity (PWC_{170}) for the three groups during the 24 week rehabilitation period.


PWC 170 Kgm/min
1300
1200
1100
1000
900
800
700
PWC 170 Vs TIME
Group 2
Group 1
Group 3


PWC 170 Kgm/min
300
250
200
150
100
50
0
-50
CHANGE IN PWC 170 Vs TIME
Group 2
Group 3
Group 1

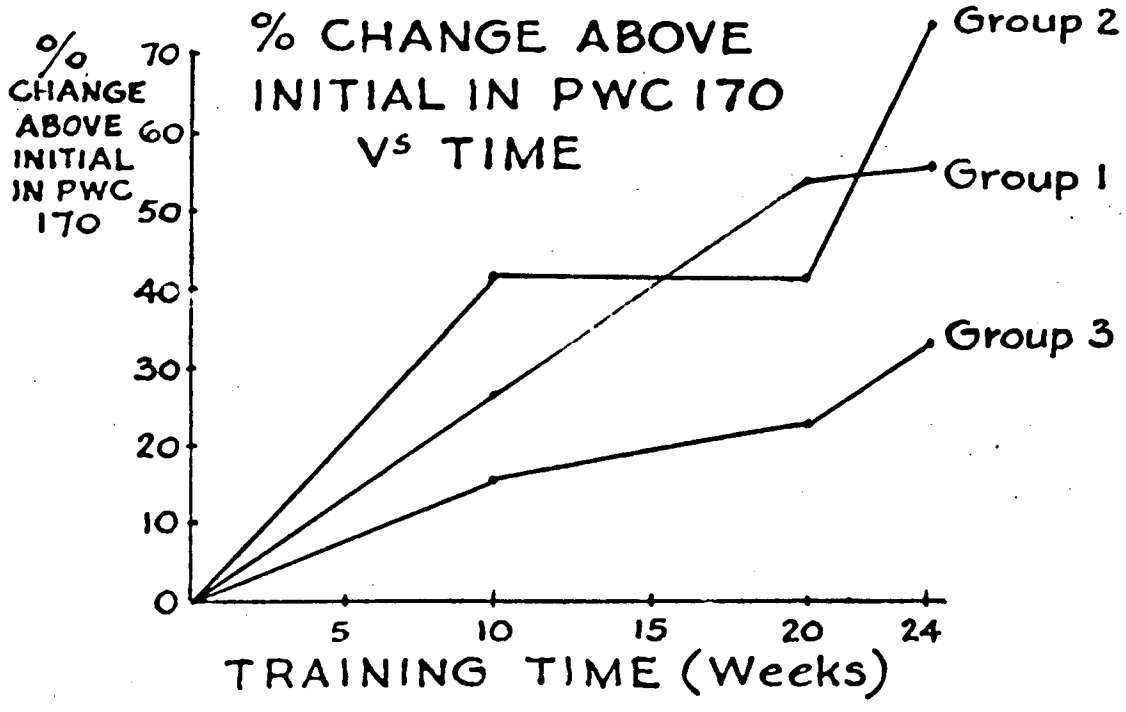
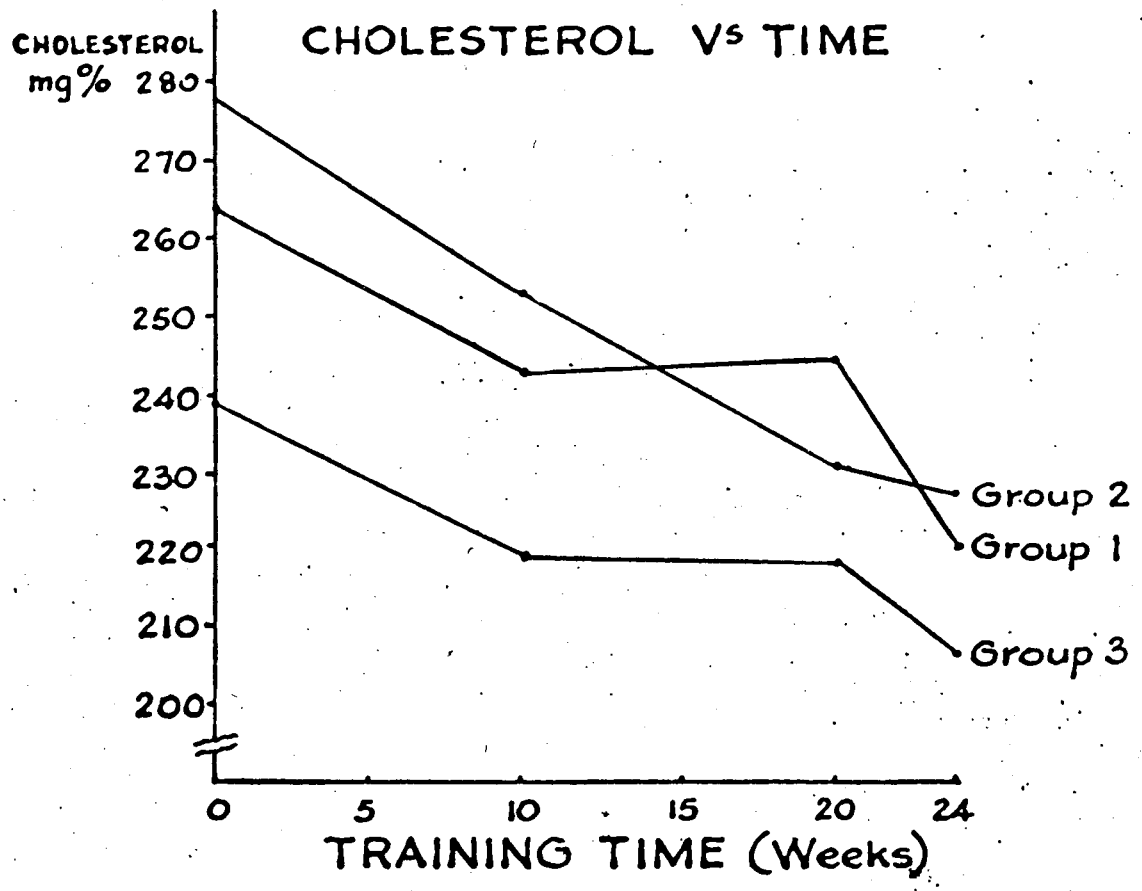

% CHANGE ABOVE INITIAL IN PWC 170
70
60
50
40
30
20
10
0
% CHANGE ABOVE INITIAL IN PWC 170 Vs TIME
Group 2
Group 1
Group 3
5
10
15
20
24
TRAINING TIME (Weeks)

FIGURE 21. Graph showing the mean cholesterol and triglyceride levels for the three groups during the 24 week rehabilitation period.


CHOLESTEROL Vs TIME
CHOLESTEROL
mg%
280
270
260
250
240
230
220
210
200
Group 2
Group 1
Group 3
0
5
10
15
20
24
TRAINING TIME (Weeks)

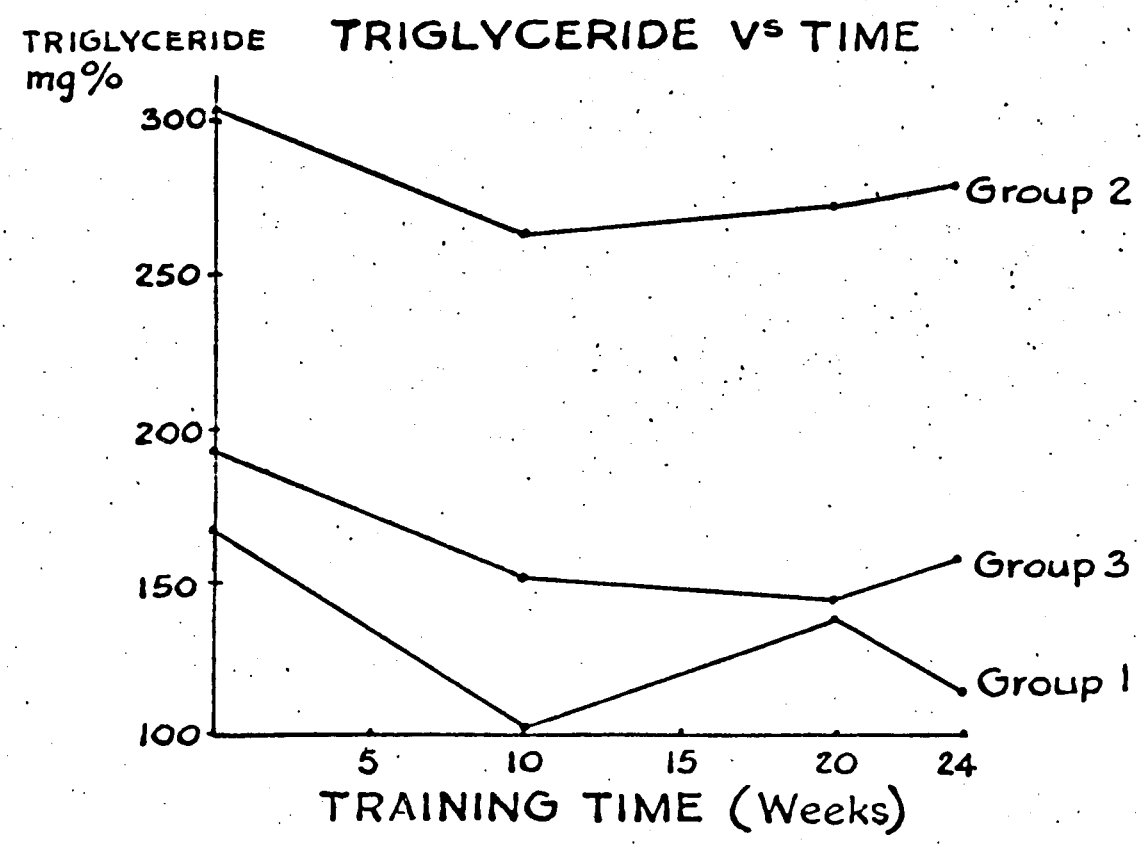

TRIGLYCERIDE Vs TIME
TRIGLYCERIDE
mg%
300
250
200
150
100
Group 2
Group 3
Group 1
5
10
15
20
24
TRAINING TIME (Weeks)

TABLE 4

CHANGES IN PHYSICAL WORK CAPACITY OF 14 SUBJECTS DURING A 24 WEEK TRAINING PROGRAM. THE INITIAL VALUES ARE MEANS OF THREE TESTS

		PWC_{170} (kpm/min)						
Group	Subject	Initial	10 wk	Δ	20 wk	Δ	24 wk	Δ
1	M.B.	700	990	+290	1155	+165	1170	+ 15
	L.M.	907	940	+ 33	1215	+275	1230	+ 15
	N.S.	600	830	+230	1050	+220	1050	0
	W.S.	897	1100	+203	1325	+225	1350	+ 25
	M.W.	693	890	+197	1040	+150	950*	- 80*
Mean		758	950	+192	1157	+207	1200	+ 14
±SD		±132	±103	± 96	±119	± 50	±125	± 8
2	I.B.	860	1100	+240	1125	+ 25	1150*	+ 25*
	D.B.	760	1140	+380	1125	- 15	1290	+165
	F.C.	698	880	+182	800	- 75	1040	+240
	H.C.	647	1075	+428	1075	0	1300	+225
	H.G.	660	925	+265	975	+ 50	1200	+225
Mean		725	1024	+299	1020	- 4	1203	+214
SD		± 87	±114	±107	±137		±120	± 33
3	J.D.	750	810	+ 60	910	+100	950	+ 40
	W.F.	785	1000	+215	1000	0	1060	+ 60
	E.K.	840	930	+ 90	920	- 10	910	- 10
	B.Q.	915	1060	+145	1200	+140	1260	+ 60
Mean		822	950	+128	1008	+ 58	1045	+ 38
±SD		± 72	±107	± 68	±135		±157	

* This value is not included in group averages due to 4 week absence from training.

PWC_{190} (kpm/min)						
Initial	10 wk	Δ	20 wk	Δ	24 wk	Δ
810	1160	+350	1330	+170	1340	+ 10
1037	1100	+ 63	1415	+315	1430	+ 15
750	950	+200	1200	+250	1190	- 10
1097	1330	+233	1565	+235	1600	+ 35
802	1010	+208	1230	+220	1120*	-110*
899	1110	+211	1348	+238	1390	+ 13
±156	±147	±102	±148	± 53	±172	± 5
1055	1290	+235	1340	+ 50	1360*	+ 20*
900	1320	+420	1300	- 20	1510	+210
867	1050	+183	980	- 70	1230	+255
750	1300	+550	1280	- 20	1570	+290
840	1140	+300	1200	+ 60	1475	+275
882	1220	+338	1220	0	1446	+258
±111	±119	±148	±144		±149	± 35
880	920	+ 40	1030	+ 80	1070	+ 40
930	1190	+260	1200	+ 10	1260	+ 60
960	1070	+110	1025	- 45	1020	- 5
1060	1210	+150	1380	+170	1430	+ 50
958	1098	+140	1159	+ 54	1195	+ 36
± 76	±134	± 92	±168		±188	

TABLE 5

SERUM LIPID CHANGES OF 14 SUBJECTS DURING A 24 WEEK TRAINING PROGRAM. THE INITIAL VALUES ARE MEANS OF THREE TESTS

		Cholesterol (mg%)						
Group	Subject	Initial	10 wk	Δ	20 wk	Δ	24 wk	Δ
1	M.B.	320	328	+ 8	326	- 2	273	-53
	L.M.	350	332	-18	344	+12	296	-48
	N.S.	240	225	-15	220	- 5	205	-15
	W.S.	159	135	-24	157	+22	120	-37
	M.W.	250	193	-57	177	-16	211	+34
Mean		263.8	242.6	-21.2	244.8	+ 2.2	221	-23.8
±SD		±74.7	±86.1		±85.7		±68.7	
2	I.B.	420	396	-24	333	-63	385	+52
	D.B.	302	225	-77	207	-18	145	-62
	F.C.	235	225	-10	230	+ 5	207	-23
	H.C.	220	215	- 5	185	-30	195	+10
	H.G.	213	205	- 8	200	- 5	207	+ 7
Mean		278.0	253.2	- 4.8	231.0	-22.2	227.8	- 3.2
±SD		±86.9	±80.3		±59.3		±91.5	
3	J.D.	250	234	-16	215	-19	210	- 5
	W.F.	221	230	+ 9	226	- 4	227	+ 1
	E.K.	250	215	-35	223	+ 8	200	-23
	B.Q.	235	196	-39	210	+14	190	-20
Mean		239.0	218.8	-20.2	218.5	-0.3	206.7	-11.8
±SD		±13.9	±17.2		± 7.3		±15.8	

Triglyceride (mg%)						
Initial	10 wk	Δ	20 wk	Δ	24 wk	Δ
270	130	-140	145	+ 15	127	- 18
274	108	-166	192	+ 84	177	- 15
103	150	+ 47	135	- 15	115	- 20
71	50	- 21	110	+ 60	64	- 46
126	75	- 51	104	+ 29	89	- 15
168.8 ± 96.2	102.6 ± 40.5	- 66.2	137.2 ± 35.0	+ 34.6	114.4 ± 42.6	- 22.8
510	582	+ 72	510	- 72	499	- 11
275	121	-154	100	- 21	89	- 11
140	109	- 31	121	+ 12	151	+ 30
335	240	- 95	235	- 5	217	- 18
261	272	+ 11	397	+125	439	+ 42
304.2 ±135.1	264.8 ±191.2	- 39.4	272.6 ±177.5	+ 7.8	279.0 ±180.5	+ 6.4
157	153	- 4	186	+ 33	150	- 36
136	106	- 30	99	- 7	130	+ 31
260	161	- 99	170	+ 9	202	+ 32
221	184	- 37	125	- 59	147	+ 22
193.5 ± 57.2	151.0 ± 32.8	- 42.5	145 ± 40.1	- 6.0	157.3 ± 31.1	+ 12.3

TABLE 6

WARM-UP CARDIAC OUTPUT AND STROKE VOLUME VALUES DURING A 24 WEEK TRAINING PERIOD. THE INITIAL VALUES ARE MEANS OF THREE TESTS

		Cardiac Output (l/min)						
Group	Subject	Initial	10 wk	Δ	20 wk	Δ	24 wk	Δ
1	M.B.	6.12	6.46	+0.34	5.85	-0.61	5.94	+0.09
	L.M.	5.81	5.99	+0.12	5.84	-0.15	5.85	+0.01
	N.S.	6.00	4.42	-1.58	4.46	+0.04	4.77	+0.31
	W.S.	6.26	6.23	-0.03	5.96	-0.27	5.91	-0.05
	M.W.	5.10	6.01	+0.91	5.42	-0.59	6.20	+0.78
Mean		5.86	5.82	-0.06	5.51	-0.31	5.73	+0.22
±SD		±0.45	±0.81		±0.62		±0.56	
2	I.B.	6.71	4.70	-2.01	5.00	+0.30	5.62	+0.62
	D.B.	6.44	5.56	-0.55	5.00	-0.56	5.77	+0.77
	F.C.	5.03	5.51	+0.11	4.48	-1.03	5.06	+0.58
	H.C.	5.55	5.80	+0.25	5.49	-0.31	6.02	+0.53
	H.G.	5.66	5.20	-0.46	4.78	-0.42	5.20	+0.42
Mean		5.88	5.35	-0.53	4.95	-0.40	5.53	+0.58
±SD		±0.69	±0.42		±0.37		±0.40	±0.13
3	J.D.	6.52	6.17	-0.35	6.68	+0.51	6.91	+0.23
	W.F.	7.13	6.57	-0.56	6.39	-0.18	6.45	+0.06
	E.K.	6.38	6.01	-0.37	6.73	+0.72	5.95	-0.78
	B.Q.	6.32	7.65	+1.32	5.55	-2.10	5.99	+0.44
Mean		6.59	6.60	+0.01	6.34	-0.26	6.33	-0.01
±SD		±0.37	±0.74		±0.55		±0.45	

Stroke Volume (ml)						
Initial	10 wk	Δ	20 wk	Δ	24 wk	Δ
65.4	77.9	+12.5	83.6	+ 5.7	80.2	- 3.4
71.8	78.8	+ 7.0	75.0	- 3.8	75.0	0
60.0	58.3	- 1.7	60.3	+ 2.0	65.5	+ 5.2
78.2	85.4	+ 7.2	76.5	- 8.9	77.8	+ 1.3
70.0	77.2	+ 7.2	71.4	- 5.8	69.0	+18.6
69.1 ± 6.8	75.5 ±10.2	+ 6.4	73.4 ± 8.5	- 2.1	73.5 ± 6.1	+ 0.1
79.0	61.1	-17.9	65.8	+ 4.7	66.0	+ 0.2
80.5	89.7	+ 9.2	78.0	-11.7	101.2	+23.2
59.0	56.2	- 2.8	45.0	-11.2	61.8	+16.8
56.1	76.4	+20.3	74.2	- 2.2	77.0	- 2.8
52.8	54.7	+ 1.9	52.6	- 2.1	55.5	+ 2.9
65.5 ±13.2	67.6 ±15.0	+ 2.1	63.1 ±14.1	- 4.5	72.2 ±18.0	+ 9.1
78.6	86.9	+ 8.3	91.5	+ 4.6	85.4	- 6.1
84.4	86.5	+ 2.1	80.9	- 5.6	80.6	- 0.3
91.7	87.0	- 4.7	96.0	+ 9.0	100.0	+ 4.0
78.0	100.0	+22.0	82.0	-18.0	80.0	- 2.0
83.2 ± 6.4	90.1 ± 6.6	+ 6.9	87.6 ± 7.3	- 2.5	86.5 ± 9.3	- 1.1

TABLE 7

WARM-UP BASE EXCESS AND RESTING LACTATE VALUES OF 14 SUBJECTS DURING A 24 WEEK TRAINING PROGRAM. THE INITIAL VALUES ARE MEANS OF THREE TESTS

		Base Excess (meq/l)						
Group	Subject	Initial	10 wk	Δ	20 wk	Δ	24 wk	Δ
1	M.B.	-3.0	-0.2	+2.8	-1.4	-1.2	-1.7	-0.3
	L.M.	-2.8	-1.8	+1.0	-4.0	-1.2	-4.0	0
	N.S.	-3.0	+0.2	+3.2	-1.0	-1.2	-0.5	+0.5
	W.S.	-3.5	-3.3	+0.2	-2.0	+1.3	-2.0	0
	M.W.	-1.0	-2.5	-1.5	+0.3	+2.8	-1.3	-1.6
Mean		-2.6	-1.5	+1.1	-1.6	+0.1	-1.9	-0.3
±SD		±0.9	±1.5		±1.6		±1.3	
2	I.B.	-5.0	-1.6	+3.4	-1.6	0	-2.0	-0.4
	D.B.	-2.5	+1.0	+3.5	-1.0	-2.0	-0.3	+0.7
	F.C.	-4.0	+0.4	+4.4	-1.5	-1.9	-2.5	-1.0
	H.C.	-1.5	-1.5	0	+1.0	+2.5	-0.5	-1.5
	H.G.	-3.3	0	+3.3	+0.2	+0.2	-3.0	-3.2
Mean		-3.6	-0.3	+2.9	-0.6	-0.2	-1.7	-1.1
±SD		±1.3	±1.2		±1.1		±1.2	
3	J.D.	-3.0	-3.5	-0.5	-7.5	-4.0	-2.0	+5.5
	W.F.	-3.0	+1.0	+4.0	-1.0	-2.0	-1.2	-0.2
	E.K.	-3.3	-2.6	+0.7	-0.8	+1.8	-2.5	+1.7
	B.Q.	-2.4	-1.3	-1.1	+0.5	+1.8	-0.1	-0.6
Mean		-2.9	-1.6	+0.7	-2.2	-0.6	-1.4	+1.6
±SD		±0.4	±1.9		±3.6		±1.0	

Lactate (mg%)						
Initial	10 wk	Δ	20 wk	Δ	24 wk	Δ
11.8	18.9	+ 7.2	12.9	- 6.0	5.0	- 7.9
11.8	5.0	- 6.8	6.0	+ 1.0	5.0	- 1.0
17.8	22.3	+ 4.5	14.0	- 8.3	12.0	- 2.0
8.8	9.0	+ 0.2	13.0	+ 4.0	7.0	- 6.0
11.2	14.0	+ 2.8	16.0	+ 2.0	6.0	-10.0
12.3 ± 3.3	13.8 ± 7.0	+ 1.6	12.4 ± 3.8	+ 0.9	7.0 ± 2.9	- 5.4
11.6	11.0	- 0.6	8.0	- 3.0	5.0	- 3.0
12.7	19.8	+ 7.1	11.0	- 8.8	3.0	- 8.0
10.4	7.1	- 3.3	5.0	- 2.1	6.0	+ 1.0
10.4	12.9	- 2.5	9.0	- 3.9	11.0	+ 2.0
9.4	13.5	+ 4.1	11.0	- 2.5	8.0	- 3.0
10.9 ± 1.3	12.9 ± 4.6	+ 1.0	8.8 ± 2.5	- 4.1	6.6 ± 3.0	- 2.2
6.6	6.0	- 0.6	10.0	+ 4.0	13.0	+ 3.0
9.4	13.0	+ 3.6	8.0	- 5.0	5.0	- 3.0
8.2	6.4	- 1.8	11.8	+ 5.4	10.0	- 1.8
8.3	14.1	+ 5.8	6.0	- 8.1	5.0	- 1.0
8.1 ± 1.1	9.9 ± 4.3	+ 1.8	9.0 ± 2.5	- 0.9	8.3 ± 3.9	- 0.7

TABLE 8

LUNG FUNCTION CHANGES OF 14 SUBJECTS DURING A 24 WEEK TRAINING PROGRAM. THE INITIAL VALUES ARE MEANS OF THREE TESTS

		Vital Capacity (l BTPS)						
Group	Subject	Initial	10 wk	Δ	20 wk	Δ	24 wk	Δ
1	M.B.	5.68	6.00	+0.32	5.84	-0.16	6.03	+0.19
	L.M.	6.10	6.15	+0.05	6.15	0	6.08	-0.07
	N.S.	4.02	4.19	+0.17	4.12	-0.07	4.50	+0.38
	W.S.	4.91	5.19	+0.28	5.34	+0.15	5.36	+0.02
	M.W.	4.68	4.74	+0.06	4.53	-0.21	4.74	+0.21
Mean		5.08	5.25	+0.17	5.20	-0.05	5.34	+0.14
±SD		±0.82	±0.83		±0.86		±0.75	
2	I.B.	4.49	4.74	+0.25	4.17	-0.57	4.65	+0.48
	D.B.	5.29	5.50	+0.21	5.28	-0.22	5.40	+0.12
	F.C.	3.77	4.06	+0.29	3.94	-0.12	4.05	+0.11
	H.C.	3.19	3.28	+0.09	3.12	-0.16	3.06	-0.06
	H.G.	4.40	4.37	-0.03	4.24	-0.13	4.40	+0.16
Mean		4.23	4.39	+0.16	4.15	-0.24	4.31	+0.16
±SD		±0.79	±0.82		±0.77		±0.86	
3	J.D.	5.84	6.16	+0.32	6.05	-0.11	6.26	+0.21
	W.F.	3.89	4.08	+0.19	4.01	-0.07	4.12	+0.11
	E.K.	4.25	4.46	+0.21	4.39	-0.07	4.38	-0.01
	B.Q.	5.18	5.25	+0.07	5.12	-0.13	5.17	+0.05
Mean		4.79	4.99	+0.20	4.89	-0.10	4.98	+0.09
±SD		±0.89	±0.92		±0.90		±0.96	

FEV_1 (1 BTPS)						
Initial	10 wk	Δ	20 wk	Δ	24 wk	Δ
3.46	4.73	+1.27	4.69	-0.04	4.82	+0.13
4.27	4.02	-0.25	4.40	+0.38	4.45	+0.05
3.03	3.03	0	3.00	-0.03	3.20	+0.20
3.62	3.42	-0.20	4.26	+0.84	4.14	-0.12
4.08	3.98	-0.10	4.04	+0.06	3.84	-0.20
3.69	3.84	+0.15	4.08	+0.24	4.09	+0.01
±0.49	±0.65		±0.65		±0.62	
2.68	3.76	+1.08	3.29	-0.47	3.93	+0.64
3.68	3.90	+0.22	3.90	0	4.01	+0.11
2.30	2.50	+0.20	2.46	-0.04	2.38	-0.08
2.58	2.45	-0.13	2.50	+0.05	2.48	-0.02
3.27	3.46	+0.19	3.46	0	3.42	-0.04
2.90	3.21	+0.31	3.12	-0.09	3.24	+0.12
±0.56	±0.69		±0.63		±0.78	
4.52	4.64	+0.12	4.51	-0.13	4.75	+0.24
2.87	2.69	-0.18	3.16	+0.47	3.30	+0.14
3.12	3.34	+0.22	3.42	+0.08	3.21	-0.21
3.80	4.00	+0.20	3.40	-0.60	4.00	+0.60
3.58	3.67	+0.09	3.62	-0.05	3.82	+0.20
±0.74	±0.84		±0.60		±0.72	

TABLE 9

EXERCISE VALUES FOR VENTILATION, OXYGEN UPTAKE AND CARBON DIOXIDE ELIMINATION DURING LAST MINUTE OF EXERCISE OF EVALUATION TESTS THROUGHOUT A 24 WEEK TRAINING PROGRAM. THE INITIAL VALUES ARE MEANS OF THREE TESTS. THE WORK RATE IS GIVEN IN PARENTHESES

		Oxygen Uptake (l/min STPD)					
Group	Subject	Initial		10 wk		Δ	
1	M.B.	1.920 (750)		1.933 (750)		+0.013 (750)	
	L.M.	2.881 (900)		2.375 (900)		-0.506 (900)	
	N.S.	1.952 (600)	1.840 (600)		2.122 (750)	-0.112 (600)	
	W.S.	1.676 (600)	2.002 (750)		1.810 (750)	-0.192 (750)	
	M.W.	1.975 (600)	1.870 (600)		2.408 (750)	-0.105 (750)	
2	I.B.	1.296 (450)	1.455 (450)	2.006 (750)	2.018 (750)	+0.159 (450)	+0.012 (750)
	D.B.	2.400 (750)	2.085 (750)		2.413 (900)	-0.315 (750)	
	F.C.	1.775 (600)	1.750 (600)	2.082 (750)	2.072 (750)	-0.025 (600)	-0.010 (750)
	H.C.	2.020 (600)	1.769 (600)	2.238 (750)	2.014 (750)	-0.251 (600)	-0.224 (750)
	H.G.	1.417 (450)	2.096 (600)	1.947 (600)	2.465 (750)	-0.149 (600)	
3	J.D.	1.693 (450)	1.623 (450)	1.850 (675)	1.773 (675)	-0.350 (450)	-0.423 (675)
	W.F.	1.790 (600)	1.533 (600)	2.056 (750)	2.023 (750)	-0.257 (600)	-0.033 (750)
	E.K.	2.319 (750)		1.980 (750)		-0.339 (750)	
	B.Q.	2.339 (750)	2.118 (750)		2.647 (900)	-0.221 (750)	

Oxygen Uptake (continued)

10 wk	→	20 wk	Δ		20 wk	→	24 wk	Δ
2.307		2.166	-0.141			2.500		-
(900)		(900)	(900)			(975)		
	1.942		-0.433		2.237		2.343	+0.106
	(900)		(900)		(1050)		(1050)	(1050)
1.842		2.300	-0.280			2.042		-0.258
(750)		(900)	(750)			(900)		(900)
2.030	2.692	2.300	-0.392			2.182		-0.118
(900)	(1050)	(1050)	(1050)			(1050)		(1050)
	2.082		-0.326			2.079		-0.003
	(750)		(750)			(750)		(750)
2.360		2.005	-0.355			1.959		-0.046
(830)		(830)	(830)			(830)		(830)
2.540	2.546	2.649	+0.127	+0.103		2.669		+0.020
(900)	(975)	(975)	(900)	(975)		(975)		(975)
	2.236		+0.164			2.200		-0.036
	(750)		(750)			(750)		(750)
	2.004		-0.010		2.045		2.000	-0.045
	(750)		(750)		(830)		(830)	(830)
	1.991		-0.474			1.900		-0.091
	(750)		(750)			(750)		(750)
	1.651		-0.122		1.920		1.833	-0.087
	(675)		(675)		(750)		(750)	(750)
2.072	2.337	2.194	+0.049	-0.143		2.197		+0.003
(750)	(900)	(900)	(750)	(900)		(900)		(900)
2.137	2.289	2.059	+0.157	-0.230		1.911		-0.148
(750)	(830)	(830)	(750)	(830)		(830)		(830)
2.381	2.199	2.462	-0.266	-0.263		1.921		-0.541
(900)	(975)	(975)	(900)	(975)		(975)		(975)

TABLE 9 (continued)

Carbon Dioxide Elimination (l/min STPD)									
Initial		10 wk		Δ		10 wk →		20 wk	
1.780 (750)		1.603 (750)		-0.177 (750)		2.027 (900)		1.727 (900)	
2.406 (900)		2.050 (900)		-0.356 (900)			1.644 (900)		
1.667 (600)	1.392 (600)		1.792 (750)	-0.275 (600)		1.457 (750)		1.980 (900)	
1.560 (600)	1.904 (750)		1.849 (750)	-0.055 (750)		1.988 (900)	2.648 (1050)	2.275 (1050)	
1.855 (600)	1.765 (600)		2.113 (750)	-0.090 (600)			1.875 (750)		
1.277 (450)	1.343 (450)	1.717 (750)	1.789 (750)	+0.066 (450)		+0.072 (750)	2.008 (830)		1.968 (830)
2.100 (750)	1.911 (750)		2.152 (900)		-0.189 (750)		2.261 (900)	2.352 (975)	2.330 (975)
1.702 (600)	1.527 (600)	1.932 (750)	1.960 (750)	-0.175 (600)		+0.028 (750)		1.618 (750)	
2.020 (600)	1.604 (600)	1.914 (750)	1.629 (750)	-0.416 (600)		-0.285 (750)		1.743 (750)	
1.491 (450)	1.926 (600)	1.717 (600)	1.981 (750)		-0.209 (600)			1.870 (750)	
1.343 (450)	1.306 (450)	1.573 (675)	1.607 (675)	-0.037 (450)		+0.034 (675)		1.440 (675)	
1.601 (600)	1.493 (600)	1.712 (750)	1.629 (750)	-0.108 (600)		-0.083 (750)	1.623 (750)	2.253 (900)	2.063 (900)
1.819 (750)		2.263 (750)			+0.444 (750)		1.884 (750)	1.998 (830)	1.855 (830)
2.118 (750)	1.851 (750)		2.052 (900)		-0.267 (750)		2.030 (900)	2.251 (975)	2.298 (975)

Carbon Dioxide Elimination (continued)					
	Δ		20 wk →	24 wk	Δ
	-0.300		1.907		-
	(900)		(975)		
	-0.406	2.291		2.239	-0.052
	(900)	(1050)		(1050)	(1050)
	-0.335		1.912		-0.068
	(750)		(900)		(900)
	-0.373		2.164		-0.111
	(1050)		(1050)		(1050)
	-0.238		1.763		-0.112
	(750)		(750)		(750)
	-0.040		1.800		-0.168
	(830)		(830)		(830)
+0.109		-0.022	2.344		+0.014
(900)		(975)	(975)		(975)
	-0.342		1.575		-0.043
	(750)		(750)		(750)
	+0.114	1.782		1.762	-0.020
	(750)	(830)		(830)	(830)
	-0.111		1.684		-0.186
	(750)		(750)		(750)
	-0.167	1.653		1.547	-0.106
	(675)	(750)		(750)	(750)
-0.006		-0.190	2.101		+0.038
(750)		(900)	(900)		(900)
-0.379		-0.143	1.723		-0.132
(750)		(830)	(830)		(830)
-0.022		+0.047	2.009		-0.289
(900)		(975)	(975)		(975)

TABLE 9 (continued)

Ventilation (l/min STPD)									
Initial		10 wk		Δ		10 wk →		20 wk	
53.0 (750)		40.2 (750)		-12.8 (750)		51.1 (900)		42.8 (900)	
61.2 (900)		58.1 (900)		- 3.1 (900)			41.8 (900)		
53.0 (600)	39.3 (600)		51.4 (750)	-13.7 (600)		41.8 (750)		55.4 (900)	
43.7 (600)	62.8 (750)		52.6 (750)	-10.2 (750)		59.3 (900)	88.3 (1050)	67.3 (1050)	
65.6 (600)	57.2 (600)		81.3 (750)	- 8.4 (600)			70.6 (750)		
44.1 (450)	45.7 (450)	48.8 (750)	54.9 (750)	- 1.6 (450)		+ 6.1 (750)	63.9 (830)		64.4 (830)
72.4 (750)	61.8 (750)		64.1 (900)		-10.6 (750)		70.2 (900)	77.1 (975)	69.3 (975)
53.1 (600)	47.6 (600)	63.2 (750)	62.0 (750)	- 5.5 (600)		- 1.2 (750)		65.3 (750)	
61.9 (600)	38.8 (600)	50.9 (750)	44.4 (750)	-23.1 (600)		- 6.5 (750)		43.3 (750)	
45.5 (450)	57.3 (600)	46.9 (600)	57.7 (750)		-10.4 (600)			54.4 (750)	
46.6 (450)	42.6 (450)	65.0 (675)	63.4 (675)	- 4.0 (450)		- 1.6 (675)		55.1 (675)	
36.3 (600)	32.9 (600)	39.2 (750)	37.0 (750)	- 3.4 (600)		- 2.2 (750)	34.9 (750)	51.4 (900)	47.2 (900)
48.2 (750)		52.2 (750)			+ 4.0 (750)		47.6 (750)	54.1 (830)	50.1 (830)
61.2 (750)	49.1 (750)		64.1 (900)		-12.1 (750)		65.6 (900)	66.1 (975)	73.8 (975)

Ventilation (continued)					
Δ			20 wk →	24 wk	Δ
	- 8.3 (900)		48.4 (975)		-
	-16.3 (900)		63.5 (1050)	57.8 (1050)	- 5.7 (1050)
	- 9.6 (750)		50.0 (900)		- 5.4 (900)
	-21.0 (1050)		64.9 (1050)		- 2.4 (1050)
	-10.7 (750)		71.4 (750)		+ 0.8 (750)
	- 0.5 (830)		55.6 (830)		- 8.8 (830)
+ 6.1 (900)		- 7.8 (975)	77.3 (975)		+ 8.0 (975)
	+ 3.3 (750)		58.1 (750)		- 7.2 (750)
	- 1.1 (750)		49.6 (830)	47.0 (830)	- 2.6 (830)
	- 3.3 (750)		52.7 (750)		- 1.7 (750)
	- 8.3 (675)		79.8 (750)	65.2 (750)	-14.6 (750)
- 2.1 (750)		- 4.2 (900)	48.1 (900)		+ 0.9 (900)
- 4.6 (750)		- 4.0 (830)	49.8 (830)		- 0.2 (830)
+ 1.5 (900)		+ 7.7 (975)	60.9 (975)		-12.9 (975)

TABLE 10

EXERCISE VALUES FOR HEART RATE, OXYGEN PULSE AND VENTILATION EQUIVALENT DURING A 24 WEEK TRAINING PROGRAM. THE EXERCISE VALUES ARE FOR LAST MINUTE OF THIRD EVALUATION WORK RATE. THE WORK RATES ARE GIVEN IN PARENTHESES

Exercise Heart Rate (b/min)								
Group	Subject	Initial		10 wk		Δ		
1	M.B.	173 (750)		143 (750)		-30 (750)		
	L.M.	167 (900)		166 (900)		- 1 (900)		
	N.S.	170 (600)	141 (600)		161 (750)	-29 (600)		
	W.S.	140 (600)	151 (750)		138 (750)	-13 (750)		
	M.W.	153 (600)	143 (600)		151 (750)	-10 (600)		
2	I.B.	124 (450)	125 (450)	141 (750)	136 (750)	+ 1 (450)		- 5 (750)
	D.B.	167 (750)	145 (750)		148 (900)		-22 (750)	
	F.C.	151 (600)	138 (600)	158 (750)	158 (750)	-13 (600)		0 (750)
	H.C.	161 (600)	138 (600)	151 (750)	141 (750)	-23 (600)		-10 (750)
	H.G.	147 (450)	150 (600)	143 (600)	155 (750)		- 7 (600)	
3	J.D.	116 (450)	114 (450)	145 (675)	143 (675)	- 2 (450)		- 2 (450)
	W.F.	147 (600)	136 (600)	150 (750)	143 (750)	-11 (600)		- 7 (750)
	E.K.	145 (750)		145 (750)			0 (750)	
	B.Q.	146 (750)	132 (750)		150 (900)		-14 (750)	

Exercise Heart Rate (continued)

10 wk	→	20 wk	Δ		20 wk	→	24 wk	Δ
164 (900)		141 (900)	-23 (900)			149 (975)		-
	138 (900)		-28 (900)		164 (1050)		153 (1050)	-11 (1050)
136 (750)		157 (750)	-25 (750)			158 (900)		+ 1 (900)
132 (900)	153 (1050)	148 (1050)	- 5 (1050)			145 (1050)		- 3 (1050)
	143 (750)		- 8 (750)			149 (750)		+ 6 (750)
147 (830)		144 (830)	- 3 (830)			141 (830)		- 3 (830)
143 (900)	161 (975)	153 (975)	- 5 (900)	- 8 (975)		143 (975)		-10 (975)
	164 (750)		+ 6 (750)			140 (750)		-24 (750)
	139 (750)		- 2 (750)		147 (830)		140 (830)	- 7 (830)
	149 (750)		- 6 (750)			142 (750)		- 7 (750)
	136 (675)		- 7 (675)		152 (750)		145 (750)	- 7 (750)
138 (750)	158 (900)	158 (900)	- 5 (750)	0 (900)		155 (900)		- 3 (900)
143 (750)	145 (830)	164 (830)	- 2 (750)	+19 (830)		155 (830)		- 9 (830)
143 (900)	145 (975)	148 (975)	- 7 (900)	+ 3 (975)		138 (975)		-10 (975)

TABLE 10 (continued)

Exercise Oxygen Pulse (l/beat)									
Initial		10 wk		Δ		10 wk →		20 wk	
.0087		.0136		+.0049		.0142		.0154	
(750)		(750)		(750)		(900)		(900)	
.0159		.0145		-.0014			.0141		
(900)		(900)		(900)			(900)		
.0116	.0130		.0133	+.0014		.0129		.0140	
(600)	(600)		(750)	(600)		(750)		(900)	
.0113	.0133		.0144	+.0011		.0151	.0176	.0155	
(600)	(750)		(750)	(750)		(900)	(1050)	(1050)	
.0144	.0131		.0161	-.0013			.0172		
(600)	(600)		(750)	(750)			(750)		
.0103	.0130	.0142	.0168	+.0027	+.0026	.0160		.0139	
(450)	(450)	(750)	(750)	(450)	(750)	(830)		(830)	
.0160	.0144		.0163	-.0016		.0178	.0158	.0152	
(750)	(750)		(900)	(750)		(900)	(975)	(975)	
.0127	.0140	.0144	.0144	+.0013	0		.0136		
(600)	(600)	(750)	(750)	(600)	(750)		(750)		
.0125	.0128	.0149	.0143	+.0003	-.0006		.0145		
(600)	(600)	(750)	(750)	(600)	(750)		(750)		
.0093	.0126	.0136	.0159	+.0010			.0144		
(450)	(600)	(600)	(750)	(600)			(750)		
.0143	.0142	.0133	.0124	-.0001	-.0009		.0141		
(450)	(450)	(675)	(675)	(450)	(675)		(675)		
.0111	.0113	.0137	.0140	+.0002	+.0003	.0150	.0149	.0145	
(600)	(600)	(750)	(750)	(600)	(750)	(750)	(900)	(900)	
.0141		.0156		+.0015		.0149	.0158	.0126	
(750)		(750)		(750)		(750)	(830)	(830)	
.0156	.0160		.0176	+.0004		.0167	.0152	.0160	
(750)	(750)		(900)	(750)		(900)	(975)	(975)	

Exercise Oxygen Pulse (continued)					
Δ		20 wk	→	24 wk	Δ
+.0012			.0178		-
(900)			(975)		
-.0004		.0136		.0153	+.0017
(900)		(1050)		(1050)	(1050)
-.0004			.0130		-.0010
(750)			(900)		(900)
-.0021			.0150		-.0005
(1050)			(1050)		(1050)
+.0011			.0139		-.0033
(750)			(750)		(750)
-.0021			.0162		+.0023
(830)			(830)		(830)
+.0015	-.0006		.0187		+.0035
(900)	(975)		(975)		(975)
-.0008			.0144		+.0008
(750)			(750)		(750)
+.0002		.0139		.0136	-.0003
(750)		(830)		(830)	(830)
-.0015			.0134		-.0010
(750)			(750)		(750)
+.0017		.0129		.0126	-.0003
(675)		(750)		(750)	(750)
+.0010	-.0004		.0141		-.0004
(750)	(900)		(900)		(900)
-.0007	-.0032		.0123		-.0003
(750)	(830)		(830)		(830)
-.0009	+.0008		.0155		-.0005
(900)	(975)		(975)		(975)

TABLE 10 (continued)

Ventilation Equivalent									
Initial		10 wk		Δ		10 wk →		20 wk	
31 (750)		21 (750)		-10 (750)		22 (900)		20 (900)	
21 (900)		24 (900)		- 3 (900)			22 (900)		
28 (600)	21 (600)		24 (750)	- 7 (600)		23 (750)		25 (900)	
28 (600)	31 (750)		28 (750)	- 3 (750)		30 (900)	33 (1050)	29 (1050)	
30 (600)	31 (600)		34 (750)	+ 1 (600)			28 (750)		
34 (450)	28 (450)	24 (750)	25 (750)	- 6 (450)		+ 1 (750)	27 (830)		32 (830)
27 (750)	29 (750)		27 (900)		+ 2 (750)		27 (900)	30 (975)	30 (975)
28 (600)	27 (600)	28 (750)	27 (750)	- 1 (600)		- 1 (750)		29 (750)	
30 (600)	22 (600)	23 (750)	22 (750)	- 8 (600)		- 1 (750)		22 (750)	
35 (450)	27 (600)	24 (600)	23 (750)		- 3 (600)			27 (750)	
27 (450)	26 (450)	34 (675)	36 (675)	- 1 (450)		+ 2 (675)		33 (675)	
22 (600)	19 (600)	19 (750)	18 (750)	- 2 (600)		- 1 (750)	17 (750)	20 (900)	20 (900)
24 (750)		23 (750)			- 1 (750)		22 (750)	24 (830)	24 (830)
26 (750)	23 (750)		25 (900)		- 3 (750)		28 (900)	30 (975)	30 (975)

Ventilation Equivalent (continued)					
Δ		20 wk	→ 24 wk		Δ
	- 2 (900)		18 (975)		-
	- 2 (900)	28 (1050)		25 (1050)	- 3 (1050)
	- 1 (750)		25 (900)		0 (900)
	- 4 (1050)		30 (1050)		+ 1 (1050)
	- 6 (750)		34 (750)		+ 6 (750)
	+ 5 (830)		25 (830)		- 7 (830)
0 (900)		0 (975)	29 (975)		- 1 (975)
	+ 2 (750)		29 (750)		0 (750)
	0 (750)	24 (830)		25 (830)	+ 1 (830)
	+ 4 (750)		27 (750)		0 (750)
	- 3 (675)	41 (750)		36 (750)	- 5 (750)
- 1 (750)		0 (900)	22 (900)		+ 2 (900)
- 1 (750)		0 (830)	26 (830)		+ 2 (830)
+ 3 (900)		0 (975)	30 (975)		0 (975)

TABLE 11

EXERCISE VALUES FOR CARDIAC OUTPUT AND STROKE VOLUME DURING A 24 WEEK TRAINING PROGRAM. THE EXERCISE VALUES ARE FOR LAST MINUTE OF THIRD EVALUATION WORK RATE. THE WORK RATE IS GIVEN IN PARENTHESES

		Exercise Cardiac Output (l/min)						
Group	Subject	Initial		10 wk		Δ		
1	M.B.	18.20 (750)		15.45 (750)		-2.75 (750)		
	L.M.	23.00 (900)		20.50 (900)		-2.50 (900)		
	N.S.	16.93 (600)	12.65 (600)		16.00 (750)	-4.28 (600)		
	W.S.	16.25 (600)	15.03 (750)		15.10 (750)	+0.07 (750)		
	M.W.	14.26 (600)	12.01 (600)		12.29 (750)	-2.25 (600)		
2	I.B.	9.91 (450)	10.50 (450)	13.62 (750)	13.55 (750)	+0.59 (450)		-0.07 (750)
	D.B.	19.25 (750)	15.51 (750)		15.85 (900)		-3.74 (750)	
	F.C.	13.07 (600)	11.70 (600)	15.58 (750)	15.80 (750)	-1.37 (600)		+0.22 (750)
	H.C.	18.81 (600)	13.85 (600)	17.24 (750)	15.18 (750)	-4.96 (600)		-2.06 (750)
	H.G.	11.65 (450)	14.25 (600)	12.25 (600)	15.00 (750)		-2.00 (600)	
3	J.D.	11.95 (450)	10.70 (450)	12.10 (675)	12.00 (675)	-1.25 (450)		-0.10 (675)
	W.F.	18.00 (600)	17.00 (600)	20.63 (750)	19.00 (750)	-1.00 (600)		-1.63 (750)
	E.K.	-	16.50 (750)		13.63 (750)		-2.87 (750)	
	B.Q.	18.49 (750)	16.80 (750)		18.33 (900)		-1.69 (750)	

Exercise Cardiac Output (continued)

10 wk	→	20 wk		Δ		20 wk	→	24 wk	Δ
19.12		16.30		-2.82			17.99		-
(900)		(900)		(900)			(975)		
	16.27			-4.23		20.08		19.60	-0.48
	(900)			(900)		(1050)		(1050)	(1050)
11.57		14.40		-4.43			13.66		-0.74
(750)		(900)		(750)			(900)		(900)
15.55	21.00	17.75		-3.25			16.39		-1.36
(900)	(1050)	(1050)		(1050)			(1050)		(1050)
	13.49			+1.20			10.81		-2.68
	(750)			(750)			(750)		(750)
14.55		13.30		-1.25			9.99		-3.31
(830)		(830)		(830)			(830)		(830)
14.95	17.04	16.05	-0.90		-0.99		14.21		-1.93
(900)	(975)	(975)	(900)		(975)		(975)		(975)
	16.30			+0.50			10.64		-5.66
	(750)			(750)			(750)		(750)
	15.71			+0.53		16.20		15.96	-0.24
	(750)			(750)		(830)		(830)	(830)
	12.00			-3.00			10.39		-1.61
	(750)			(750)			(750)		(750)
	10.14			-1.86		10.08		9.55	-0.53
	(675)			(675)		(750)		(750)	(750)
18.99	21.05	20.03	-0.01		-1.02		18.59		-1.44
(750)	(900)	(900)	(750)		(900)		(900)		(900)
13.85	14.20	13.25	+0.22		-0.95		11.34		-1.91
(750)	(830)	(830)	(750)		(830)		(830)		(830)
18.12	20.28	16.65	-0.20		-3.63		14.56		-2.09
(900)	(975)	(975)	(900)		(975)		(975)		(975)

TABLE 11 (continued)

Exercise Stroke Volume (ml)									
Initial		10 wk		Δ		10 wk →		20 wk	
103.1		108.0		+ 4.9		116.5		115.7	
(750)		(750)		(750)		(900)		(900)	
135.0		125.0		-10.0			118.0		
(900)		(900)		(900)			(900)		
100.0	89.7		100.0	-10.3		85.0		87.2	
(600)	(600)		(750)	(600)		(750)		(900)	
112.5	99.5		108.5	+ 9.0		118.0	137.1	120.0	
(600)	(750)		(750)	(750)		(900)	(1050)	(1050)	
93.2	84.1		82.0	- 9.1			93.0		
(600)	(600)		(750)	(600)			(750)		
80.6	84.0	104.0	99.5	+ 3.4		- 4.5	99.0		92.4
(450)	(450)	(750)	(750)	(450)		(750)	(830)		(830)
113.5	107.0		107.0		- 6.5		104.5	106.0	105.0
(750)	(750)		(900)		(750)		(900)	(975)	(975)
83.1	85.0	98.6	100.0	+ 1.9		+ 1.4		99.3	
(600)	(600)	(750)	(750)	(600)		(750)		(750)	
116.5	100.3	112.8	107.7	-16.2		- 5.1		113.0	
(600)	(600)	(750)	(750)	(600)		(750)		(750)	
78.7	95.0	85.6	96.8		- 9.4			80.5	
(450)	(600)	(600)	(750)		(600)			(750)	
98.7	93.9	83.4	83.9	- 4.8		+ 0.4		74.6	
(450)	(450)	(675)	(675)	(450)		(675)		(675)	
120.0	125.0	138.0	134.8	+ 5.0		- 3.2	136.8	133.5	127.0
(600)	(600)	(750)	(750)	(600)		(750)	(750)	(900)	(900)
-	111.0		86.5		-24.5		97.0	101.0	80.8
	(750)		(750)		(750)		(750)	(830)	(830)
126.6	127.3		122.0		+ 0.7		126.7	140.0	112.5
(750)	(750)		(900)		(750)		(900)	(975)	(975)

Exercise Stroke Volume (continued)					
Δ		20 wk	→	24 wk	Δ
	+ 0.8		120.5		-
	(900)		(975)		
	- 7.0	122.0		128.0	+ 6.0
	(900)	(1050)		(1050)	(1050)
	-15.0		86.2		- 1.0
	(750)		(900)		(900)
	-17.1		118.5		- 1.5
	(1050)		(1050)		(1050)
	+11.0		72.2		-20.8
	(750)		(750)		(750)
	- 6.6		86.9		- 5.5
	(830)		(830)		(830)
- 2.5		- 1.0	105.0		0
(900)		(975)	(975)		(975)
	- 0.7		76.1		-23.2
	(750)		(750)		(750)
	+ 5.3	110.2		114.0	+ 3.8
	(750)	(830)		(830)	(830)
	-16.3		73.2		- 7.3
	(750)		(750)		(750)
	- 9.3	66.3		65.9	- 0.4
	(675)	(750)		(750)	(750)
+ 2.0		- 6.5	119.0		- 8.0
(750)		(900)	(900)		(900)
+10.5		-20.2	73.3		- 7.5
(750)		(830)	(830)		(830)
+ 4.7		-27.5	106.0		- 6.5
(900)		(975)	(975)		(975)

TABLE 12

EXERCISE VALUES FOR BASE EXCESS AND LACTATE DURING A 24 WEEK TRAINING PROGRAM. THE EXERCISE VALUES ARE FOR LAST MINUTE OF THIRD EVALUATION WORK RATE. THE WORK RATES ARE GIVEN IN PARENTHESES

Exercise Base Excess (meq/l)							
Group	Subject	Initial		10 wk		Δ	
1	M.B.	-12.0 (750)		- 3.0 (750)		+ 9.0 (750)	
	L.M.	-12.7 (900)		- 8.0 (900)		+ 4.7 (900)	
	N.S.	-10.0 (600)	- 3.1 (600)		- 5.5 (750)	+ 6.9 (600)	
	W.S.	-10.7 (600)	-10.7 (750)		- 7.3 (750)	+ 3.4 (750)	
	M.W.	- 9.2 (600)	- 7.8 (600)		-12.7 (750)	+ 1.4 (600)	
2	I.B.	- 7.8 (450)	- 8.0 (450)	- 8.7 (750)	- 6.5 (750)	- 0.2 (450)	+ 2.2 (750)
	D.B.	-15.5 (750)	- 7.8 (750)		- 6.8 (900)	+ 7.7 (750)	
	F.C.	-11.5 (600)	- 9.1 (600)	-10.8 (750)	- 5.7 (750)	+ 2.4 (600)	+ 5.1 (750)
	H.C.	- 9.4 (600)	- 3.0 (600)	- 3.5 (750)	- 2.2 (750)	+ 6.4 (600)	+ 1.3 (750)
	H.G.	- 4.6 (450)	- 4.7 (600)	- 1.2 (600)	- 5.3 (750)	+ 3.5 (600)	
3	J.D.	- 7.0 (450)	- 7.5 (450)	- 9.4 (675)	- 9.0 (675)	- 0.5 (450)	+ 0.4 (675)
	W.F.	- 8.7 (600)	- 2.0 (600)	- 4.4 (750)	- 3.1 (750)	+ 6.7 (600)	+ 1.3 (750)
	E.K.	-10.0 (750)		- 3.9 (750)		+ 6.1 (750)	
	B.Q.	- 9.8 (750)	- 1.3 (750)		- 8.8 (900)	+ 8.7 (750)	

Exercise Base Excess (continued)

10 wk	→	20 wk		Δ		20 wk	→	24 wk	Δ
- 6.8 (900)		- 3.5 (900)		+ 3.3 (900)			- 5.7 (975)		-
	- 7.2 (900)			+ 0.8 (900)		- 8.0 (1050)		- 8.2 (1050)	- 0.2 (1050)
- 3.7 (750)		- 7.3 (900)		+ 1.8 (750)			- 6.7 (900)		+ 0.6 (900)
-14.0 (900)	-14.5 (1050)	- 8.2 (1050)		+ 6.3 (1050)			- 8.0 (1050)		+ 0.2 (1050)
	- 7.6 (750)			+ 5.1 (750)			-11.2 (750)		- 3.6 (750)
- 6.8 (830)		-11.1 (830)		- 4.3 (830)			- 7.5 (830)		+ 3.6 (830)
- 5.8 (900)	- 8.0 (975)	- 7.9 (975)	+ 1.0 (900)		+ 0.1 (975)		- 4.8 (975)		+ 3.1 (975)
	- 8.5 (750)			- 2.8 (750)			- 7.2 (750)		+ 1.3 (750)
	- 3.8 (750)			- 1.6 (750)		- 4.4 (830)		- 3.3 (830)	+ 1.1 (830)
	- 4.8 (750)			+ 0.5 (750)			- 5.2 (750)		- 0.4 (750)
	-10.6 (675)			- 1.6 (675)		-10.0 (750)		- 6.5 (750)	+ 3.5 (750)
- 4.1 (750)	- 5.7 (900)	- 5.5 (900)	- 1.0 (750)		+ 0.2 (900)		- 6.5 (900)		- 1.0 (900)
-10.1 (750)	-13.8 (830)	- 9.2 (830)	- 6.2 (750)		+ 4.6 (830)		- 9.2 (830)		0 (830)
- 7.7 (900)	- 9.5 (975)	- 6.5 (975)	+ 1.1 (900)		+ 3.0 (975)		- 4.9 (975)		+ 1.6 (975)

TABLE 12 (continued)

Lactate (mg%)									
Initial		10 wk		Δ			10 wk	→	20 wk
90.3		24.5		-65.8			35.3		21.0
(750)		(750)		(750)			(900)		(900)
40.0		17.0		-23.0				11.0	
(900)		(900)		(900)				(900)	
-	27.0		23.0	-			10.0		16.0
(600)	(600)		(750)	(600)			(750)		(900)
40.7	66.4		21.0	-45.4			120.0	126.9	20.0
(600)	(750)		(750)	(750)			(900)	(1050)	(1050)
58.8	31.5		84.0	-27.3				-	
(600)	(600)		(750)	(600)				(750)	
23.5	24.0	25.7	18.9	+ 0.5		- 6.8	23.0		31.0
(450)	(450)	(750)	(750)	(450)		(750)	(830)		(830)
61.1	46.0		48.0		-15.1		42.3	99.9	36.0
(750)	(750)		(900)		(750)		(900)	(975)	(975)
47.0	38.2	112.8	-	- 8.8		-		37.0	
(600)	(600)	(750)	(750)	(600)		(750)		(750)	
42.3	17.2	21.2	18.9	-25.1		- 2.3		22.0	
(600)	(600)	(750)	(750)	(600)		(750)		(750)	
21.2	18.0	8.0	21.2		-10.0			18.0	
(450)	(600)	(600)	(750)		(600)			(750)	
13.4	11.8	59.0	58.8	- 1.6		- 0.2		33.0	
(450)	(450)	(675)	(675)	(450)		(675)		(675)	
27.0	14.1	24.0	23.0	-12.9		- 1.0	-	-	39.0
(600)	(600)	(750)	(750)	(600)		(750)	(750)	(900)	(900)
47.0		24.0			-23.0		39.0	74.0	30.0
(750)		(750)			(750)		(750)	(830)	(830)
48.0	15.9		34.0		-32.1		35.0	37.0	28.0
(750)	(750)		(900)		(750)		(900)	(975)	(975)

Lactate (continued)					
Δ		20 wk	→	24 wk	Δ
-14.3			29.0		-
(900)			(975)		
- 6.0		15.0		15.0	0
(900)		(1050)		(1050)	(1050)
-13.0			15.0		- 1.0
(750)			(900)		(900)
-106.9			19.0		- 1.0
(1050)			(1050)		(1050)
-			24.0		-
(750)			(750)		(750)
+ 8.0			18.0		-13.0
(830)			(830)		(830)
- 5.7	-63.9		15.0		-35.0
(900)	(975)		(975)		(975)
-			25.0		-12.0
(750)			(750)		(750)
+ 3.1		25.0		18.0	- 7.0
(750)		(830)		(830)	(830)
- 3.2			17.0		- 1.0
(750)			(750)		(750)
-25.8		33.0		17.0	-16.0
(675)		(750)		(750)	(750)
-			19.0		-20.0
(900)			(900)		(900)
+15.0	-44.0		25.0		- 5.0
(750)	(830)		(830)		(830)
-	- 9.0		16.0		-12.0
(900)	(975)		(975)		(975)

TABLE 13

RESTING AND EXERCISE BLOOD PRESSURE VALUES OF 14 SUBJECTS DURING A 24 WEEK TRAINING PROGRAM. THE EXERCISE VALUES ARE FOR LAST MINUTE OF EXERCISE OF THIRD EVALUATION WORK RATE. THE INITIAL VALUES ARE MEANS OF THREE TESTS. THE WORK RATES ARE GIVEN IN PARENTHESES

Resting Blood Pressure - Systolic/Diastolic (mmHg)								
Group	Subject	Initial	10 wk	Δ	20 wk	Δ	24 wk	Δ
1	M.B.	130/ 75	112/ 80	-18/+ 5	110/ 70	- 2/-10	130/ 70	+20/ 0
	L.M.	117/ 78	128/ 70	+11/- 8	108/ 70	-20/ 0	110/ 72	+ 2/+ 2
	N.S.	134/ 92	120/ 72	-14/-20	136/ 79	+16/+ 7	134/ 78	- 2/- 1
	W.S.	110/ 75	108/ 70	- 2/- 5	108/ 70	0 / 0	114/ 76	+ 6/+ 6
	M.W.	140/ 80	118/ 70	-22/-10	138/ 70	+20/ 0	140/ 80	+ 2/+10
Mean ±SD		126/ 80 ±12/ 7	117/ 72 ± 8/ 4		120/ 72 ±16/ 4		126/ 75 ±13/ 4	
2	I.B.	140/100	140/ 90	0 /-10	130/ 90	+10/ 0	150/100	+20/+10
	D.B.	110/ 86	108/ 68	- 2/-18	98/ 60	-10/- 8	110/ 60	+12/ 0
	F.C.	150/106	130/ 76	-20/-30	150/ 90	+20/+14	128/ 88	-22/- 2
	H.C.	150/104	140/ 90	-10/-14	150/ 90	+10/ 0	148/ 90	- 2/ 0
	H.G.	126/ 76	120/ 78	- 6/+ 2	110/ 75	-10/- 3	126/ 78	+16/+ 3
Mean ±SD		135/ 94 ±17/ 13	128/ 80 ±14/ 10		128/ 81 ±23/ 13		132/ 83 ±17/ 15	
3	J.D.	122/ 78	108/ 70	-14/- 8	120/ 70	+12/ 0	126/ 70	+ 6/ 0
	W.F.	124/ 82	130/ 80	+ 6/- 2	130/ 90	0 /+10	132/92	+ 2/+ 2
	E.K.	133/ 86	130/ 70	- 3/-16	140/ 70	+10/ 0	140/ 90	0 /+20
	B.Q.	110/ 70	110/ 70	0 / 0	110/ 70	0 / 0	98/ 60	-12/-10
Mean ±SD		122/ 79 ±10/ 7	120/ 73 ±12/ 5		125/ 75 ±13/ 10		124/ 78 ±18/ 16	

TABLE 13 (continued)

Exercise Blood Pressure - Systolic/Diastolic (mmHg)								
Initial		10 wk		Δ		10 wk →		
180/ 95 (750)		160/ 86 (750)		-20/- 9 (750)		170/ 90 (900)		
145/ 90 (900)		142/ 78 (900)		- 3/-12 (900)			140/ 60 (900)	
176/105 (600)	176/ 85 (600)		210/120 (750)	0 /-20 (600)		175/100 (750)		
120/ 70 (600)	120/ 70 (750)		120/ 70 (750)	0 / 0 (750)		130/ 50 (900)	143/ 60 (1050)	
147/ 93 (600)	135/ 80 (600)		172/ 82 (750)	-12/-13 (600)			180/100 (750)	
150/105 (450)	142/102 (450)	160/ 83 (750)	160/ 90 (750)	- 8/- 3 (450)		- 2/+ 7 (750)	150/ 80 (830)	
126/ 95 (750)	132/ 82 (750)		140/ 80 (900)		+ 6/-13 (750)		142/ 80 (900)	200/ 90 (975)
182/102 (600)	150/ 90 (600)	180/ 90 (750)	188/100 (750)	-32/-12 (600)		+ 8/+10 (750)		174/110 (750)
188/112 (600)	190/116 (600)	194/124 (750)	210/130 (750)	+ 2/+ 4 (600)		+16/+ 6 (750)		175/100 (750)
141/ 80 (450)	166/ 90 (600)	164/ 90 (600)	180/ 80 (750)		- 2/+10 (600)			175/ 90 (750)
139/ 89 (450)	130/ 80 (450)	130/ 90* (675)	178/ 80 (675)	- 9/- 9 (450)		+48/-10 (675)		148/110 (675)
150/100 (600)	134/ 70 (600)	158/100 (750)	170/ 90 (750)	-16/-30 (600)		+12/-10 (750)	160/ 70 (750)	190/100 (900)
165/ 75 (750)		160/ 88 (750)			- 5/+13 (750)		168/ 70 (750)	160/ 80 (830)
152/ 80 (750)	130/ 50 (750)		140/ 60 (900)		-22/-30 (750)		138/ 50 (900)	170/ 68 (975)

* Incomplete test

Exercise Blood Pressure (continued)

20 wk	Δ	20 wk → 24 wk	Δ
170/ 90 (900)	0 / 0 (900)	180/ 90 (975)	-
	- 2/-18 (900)	172/100 (1050) → 164/ 92 (1050)	- 8/- 8 (1050)
192/100 (900)	-35/-20 (750)	166/ 92 (900)	-26/- 8 (900)
148/ 70 (1050)	+ 5/+10 (1050)	150/ 70 (1050)	+ 2/ 0 (1050)
	+ 2/+18 (750)	147/ 82 (750)	-33/-18 (750)
180/100 (830)	+30/+20 (830)	210/110 (830)	+30/+10 (830)
170/ 78 (975)	-30/-12 (975)	148/ 80 (975)	-22/+ 2 (975)
	-14/+10 (750)	170/ 90 (750)	- 4/-20 (750)
	-35/-30 (750)	230/140 (830) → 227/138 (830)	- 3/- 2 (830)
	- 5/+10 (750)	150/ 90 (750)	-25/ 0 (750)
	-30/+30 (675)	160/ 80 (750) → 160/ 86 (750)	0 /+ 6 (750)
188/108 (900)	-10/-20 (750) - 2/+ 8 (900)	170/ 80 (900)	-18/-28 (900)
180/ 80 (830)	+ 8/-18 (750) +20/ 0 (830)	170/ 72 (830)	-10/- 8 (830)
155/ 76 (975)	+ 2/-10 (900) -15/+ 8 (975)	136/ 66 (975)	-19/-10 (975)

TABLE 14

WARM-UP AND EXERCISE MYOCARDIAL OXYGEN DEMAND VALUES DURING A 24 WEEK TRAINING PROGRAM. THE EXERCISE VALUES ARE FOR LAST MINUTE OF THIRD EVALUATION WORK RATE. THE WORK RATES ARE GIVEN IN PARENTHESES

		Warm-up Myocardial Oxygen Demand (mmHg X HR)						
Group	Subject	Initial	10 wk	Δ	20 wk	Δ	24 wk	Δ
1	M.B.	10929	9296	-1633	7700	-1596	9768	+2068
	L.M.	8400	9230	+ 830	8424	- 806	8590	+ 166
	N.S.	13965	7300	-6665	10000	+2700	9782	- 218
	W.S.	7500	7874	+ 374	7644	- 230	8300	+ 656
	M.W.	10220	9204	-1016	10488	+1284	12600*	+2112*
Mean ±SD		10203 ±2512	8581 ± 930	-1622	8851 ±1319	+ 270	9110 ± 777	+ 668
2	I.B.	11900	10780	-1120	9424	-1356	12750*	+3326*
	D.B.	8779	6696	-2083	6272	- 424	6270	- 2
	F.C.	12700	12740	+ 40	16350	+3610	10496	-5854
	H.C.	14840	8260	-6580	11100	+2840	11400	+ 300
	H.G.	13436	11400	-2036	10010	-1390	11844	+1834
Mean ±SD		12331 ±2261	9975 ±2450	-2356	10631 ±3666	+ 656	10003 ±2551	- 930
3	J.D.	10173	7668	-2505	8760	+1092	10206	+1446
	W.F.	10800	9880	- 920	10270	+ 390	10560	+ 290
	E.K.	9241	8970	- 271	7434	-1536	8400	+ 966
	B.Q.	8324	8360	+ 36	7480	- 880	7350	- 130
Mean ±SD		9635 ±1083	8720 ± 939	- 915	8486 ±1339	- 233	9129 ±1517	+ 643

* This value is not included in group means due to 4 week absence from training.

TABLE 14 (continued)

Exercise Myocardial Oxygen Demand (mmHg X HR)									
Initial		10 wk		Δ		10 wk	→	20 wk	
24640		22880		-1760		24600		23970	
(750)		(750)		(750)		(900)		(900)	
22585		22950		+ 365			19320		
(900)		(900)		(900)			(900)		
29568	24534		33600	-5034		24150		29704	
(600)	(600)		(750)	(600)		(750)		(900)	
19305	18120		15290	-2830		18480	21420	21904	
(600)	(750)		(750)	(750)		(900)	(1050)	(1050)	
22500	19305		25800	-3195			26100		
(600)	(600)		(750)	(600)			(750)		
19775	21250	22842	21120	+1475		-1722	27550		25920
(450)	(450)	(750)	(750)	(450)		(750)	(830)		(830)
20815	19140		20720		-1675		20306	32200	20150
(750)	(750)		(900)		(750)		(900)	(975)	(975)
27300	20700	28440	29704	-6600		+1264		28536	
(600)	(600)	(750)	(750)	(600)		(750)		(750)	
29140	26790	29682	29610	-2350		- 72		23630	
(600)	(600)	(750)	(750)	(600)		(750)		(750)	
20930	24900	23452	27900		-1448			26075	
(450)	(600)	(600)	(750)		(600)			(750)	
16597	14820	18850	23970	-1777		+5120		19720	
(450)	(450)	(675)	(675)	(450)		(675)		(675)	
21900	18224	23700	23970	-3676		+ 270	22080	30020	29704
(600)	(600)	(750)	(750)	(600)		(750)	(750)	(900)	(900)
23365		23200			- 165		24024	20300	28800
(750)		(750)			(750)		(750)	(830)	(830)
22400	17160		21000		-5240		19734	24650	22940
(750)	(750)		(900)		(750)		(900)	(975)	(975)

Exercise Myocardial Oxygen Demand (continued)

Δ		20 wk	→	24 wk	Δ
- 630			26820		-
(900)			(975)		
-3630		28208		25092	-3116
(900)		(1050)		(1050)	(1050)
-9450			26726		-2978
(750)			(900)		(900)
+ 484			21750		- 154
(1050)			(1050)		(1050)
+ 300			22050		-4050
(750)			(750)		(750)
-1630			30240		+4320
(830)			(830)		(830)
- 414	-12050		21164		+1014
(900)	(975)		(975)		(975)
-1168			23800		-4736
(750)			(750)		(750)
-5980		34986		33600	-1386
(750)		(830)		(830)	(830)
-1825			21300		-4775
(750)			(750)		(750)
-4250		22800		23200	+ 400
(675)		(750)		(750)	(750)
-1890	- 316		26860		-2844
(750)	(900)		(900)		(900)
+ 824	+8500		26350		-2450
(750)	(830)		(830)		(830)
- 266	-1710		18800		-4140
(900)	(975)		(975)		(975)

CHAPTER V

DISCUSSION

The Rehabilitation Program

General

This study has investigated fourteen post myocardial infarct patients who participated in a six month exercise rehabilitation program. Specifically, it has measured the cardiovascular, respiratory and serum lipid changes over a six month period and also evaluated two types and variations of training. It was shown that continuous ergometry, at 50 per cent of the current physical working capacity for a heart rate (estimated) of 190 beats per minute (PWC_{190} est.), led to the most consistent improvement and largest changes in work capacity. In one subject (MW), however, at the outset of training, thirty minutes of continuous ergometry was too long and it was two weeks before he was able to complete the half hour. In this situation, the cyclic type of training at 40 per cent and 60 per cent of his PWC_{190} was advantageous. The mean change above initial in PWC_{170} was 54.9 per cent.

Group 1

This group started on cyclic training and early improvement was observed. After ten weeks of this training a mean significant increase of 26.4 per cent for the PWC_{170}, above the initial value, was observed. Continuous training then led to a further mean increase of 28.2 per cent (26.4 → 54.6 per cent above the initial values). In most subjects it was a more effective training stimulus - a fact which can be noted particularly by the slopes of the PWC curves during the appropriate phase of training (see Figure 20).

Group 2

This group started on continuous training and substitution of cyclic training produced only a maintenance of the gains made previously or a small decrement. The mean increase above the initial PWC_{170} after continuous training was 42.1 per cent. This mean increment had declined 0.7 per cent at the conclusion of the cyclic training period (42.1 → 41.4 per cent). The final month of continuous training yielded a further mean increase of 33.4 per cent in their PWC_{170} (41.4 → 74.8 per cent). The better quantitative improvement in the PWC_{170}, during the continuous phase of training, was supported by the subjective feelings of the patients. A possible reason for the relative ineffectiveness of the cyclic training was that training at 60 per cent of the patient's current work capacity was not severe enough to tax his cardio-respiratory system. Astrand et al. (1960) found that with athletes intermittent exercise of three minutes followed by three minutes rest was a successful type of training if the subject's oxygen transport system is stressed maximally. When dealing with cardiac patients, maximal training is contraindicated and cyclic training therefore cannot assume its full potential of stressing the oxygen transport system.

Group 3 - Calisthenics-Walk-Jog Program

The calisthenics-walk-jog program led to small but significant gains in PWC_{170} and the later change to one month of continuous training showed further gains. The mean relative increases for this group were 15.5 per cent and 22.7 per cent, above the initial values, after ten and twenty weeks, respectively, of the calisthenics-walk-jog program. With ergometry, the average increase was a further 10.5 per cent (22.7 → 33.2 per cent). Group 3 trained intensively only three times per week and walked on two other days. The walk-jog program was probably not as successful because

the heart rate was not consistently sustained at 130-150 beats per minute for extended periods since it was manditory that the subjects stop to take pulse counts. If, after an initial period of training, they were to have begun this type of program the results probably would have been better because frequent stops for pulse counting would have been obviated. The patient by virtue of his earlier training would have become fitter and more experienced in guaging his heart rate without counting. One inherent disadvantage with jogging is that when a subject becomes tired he naturally slows down which results in a lowering of the heart rate and thereby removes the training effect.

The jogging type program, however, although not as successful as the bike has many advantages. One was the total body involvement in pre-jogging warm-up calisthenics. Another advantageous feature of jogging is the lack of boredom with training which might possibly lead to a high dropout rate in other types of training (bicycle, treadmill). This boredom and high dropout has indeed been the experience of many programs (Hellerstein, 1968 and Heller, 1969).

Control of Training

In the jogging program, adequate control of the training situation was maintained through heart rate monitoring. In some cases ECG telemetry was warranted, based on the results of the initial evaluation tests. The criteria for close monitoring in training were angina pains, ST segment depression or ectopic beats present in testing sessions. A positive feature of the bicycle ergometry training was that more subjects could be closely monitored, with only one training supervisor, than in the jogging program. Patients with marked coronary insufficiency or those who developed repeated ectopic

ventricular contractions could be more closely monitored in an ergometry program.

Appropriateness of Training

In retrospect, the calisthenic-walk-jog program was probably inappropriate for subjects JD and EK because of their overweight condition. This was especially true in the case of subject JD who developed severe angina upon exertion. When jogging his excess weight added considerably to the energy cost and as a result he developed angina at a heart rate of 115 to 125 beats per minute and he had to stop jogging. This was very discouraging to him and limited the benefit of the program because a true exercise cardiovascular stress could not be developed. When both of these subjects were placed on the ergometry program much better results were seen. As discussed earlier, subject JD could exercise at a heart rate of 150 beats per minute for a period of twenty minutes with no angina.

These two subjects illustrate an important consideration in further rehabilitation studies with cardiac patients. This feature is that an appropriate type of rehabilitation program should be selected individually for each patient. If a patient is overweight then the ergometry program would be the best selection. In a jogging program an overweight condition not only places stress on the cardiovascular system but also adds excess stress to body joints especially the knees and ankles. Also, in the case of a very weak subject (MW), the first month of training could be most advantageously spent with cyclic training, changing to the continuous method of ergometry later. Subjects BQ and WF appreciated the total body involvement with the warm-up calisthenics and this feature could easily and profitably be added to the continuous ergometry program.

Group Training and Retention of Participants

The probable reason for the retention of all subjects in this program was the companiable atmosphere of the group training sessions. Other probable reasons for the 100 per cent retention include the regular (every two weeks) measurement of the patients' fitness and up-grading of their training exercise, the close supervision of training and knowledge of their test results.

Group training proved to be a very important feature of the program. It was especially important for new subjects entering the program. It became apparent that new subjects were very apprehensive and somewhat hesitant to stress themselves, however, when they could see others training at considerable work loads they gained confidence. Group training was helpful for those who had angina, when they could see subjects (especially JD) riding through (like the walk-through phenomena discussed earlier) the bouts of angina. Although psychological tests were not included in this study it was obvious that all subjects showed a remarkable improvement in mood. Using psychological tests, Rechnitzer et al. (1967) found that a twenty-four week rehabilitation program also led to a significant subjective improvement in the mood of their patients. The cardiac-exercisers felt more confident and less anxious. In the present study the fact that all subjects were capable of returning to their work was a very important factor in their improvement in mood and feeling of well-being. All subjects returned early to work except MW who as mentioned previously was capable of work but his age was a factor in his unemployment. The fact that he could not find employment was very disturbing to him and in fact he took an unsuccessful month's absence from training to find employment. At the time of completion of this writing subject MW had indeed become employed

once more. Besides office jobs, many of the subjects returned to very strenuous work such as truck driving, including loading and unloading, bus driving, warehouse work and working on a tug boat with a local lumber mill.

Changes in Work Capacity (Table 4)

In order to evaluate the changes in physical work capacity and also to reassess the subject's training work rate the PWC_{190} was estimated. In order to make a comparison with other studies the more conventional PWC_{170} was also determined. These two values were determined by a test session which required three increasing submaximal loads, each of which lasted six minutes, to be performed by the patient. This method of evaluation meets both physiological and clinical criteria because the test can be easily applied to patients and can be interrupted quickly when an abnormal electrocardiogram appears or when a heart rate of 170 beats per minute was reached. These features were recognized earlier by Denolin et al. (1967).

All subjects showed a significant increase in their physical work capacity over the period of 24 weeks. The differences in the magnitude of the change appears to be related to the type of exercise training and also to their initial level of fitness. The three graphs in Figure 20 support the view that while all forms of training produced improvement in PWC_{170}, continuous ergometry was the best method and the improvement with the continuous ergometry could only be maintained with the cyclic effort, not enhanced. The calisthenic-walk-jog program produced the smallest changes. The fact that this group trained under supervision three times per week and walked on the days between compared to five times per week for the ergometry trained subjects could have been a factor influencing the small change although total training time (in minutes) per week was the same for all

groups. Further investigations should be carried out to compare other forms and methods of training.

In a recent discussion on current levels of fitness, Cumming (1967) reviewed three studies which quoted values for PWC_{170} of Canadian men twenty to forty years of age. These studies included medical students, managers and factory workers; the average PWC_{170} was 1032 kpm/min. The present group of fourteen subjects had an initial value of 769 kpm/min which increased with training to an average value of 1151 kpm/min. The mean value for the ten subjects who trained by bicycle ergometry, increased to 1200 kpm/min which is considerably greater than the "healthy" men that Cumming has discussed.

Circulatory Response to Training

General

A controlled warm-up as a routine part of the test session was used because a more stable and reproducible baseline resulted. This was found to be the case in the circulatory, respiratory and acid-base balance parameters.

The observed decreases in resting and exercise heart rate for a standard work rate, decreases in resting and exercise arterial blood pressure and decreases in warm-up and exercise myocardial oxygen consumption are similar to the changes seen in healthy sedentary or athletic subjects as discussed by Frick (1968). Similar changes resulting from exercise rehabilitation of patients with healed myocardial infarction have been reported by Naughton et al. (1966), Varnauskas et al. (1966), Hellerstein et al. (1967a, 1967b, 1968) and Frick and Katila (1968). These findings indicate that the convalescent cardiac patient responds to training similarly to healthy

untrained individuals. The magnitude and rate of change outlined in the results of this study appeared to be related to the type of training followed. The largest changes occurred with the continuous bicycle ergometry. A lower degree of anxiety, observed subjectively, in the patients as a result of training might also be a contributory factor to the decrements in heart rate, blood pressure and myocardial oxygen demand. In addition, the response of various hemodynamic variables to training are probably related to the patient's initial fitness level. Fortunately, in the present investigation, this was uniformly low.

Bradycardia at Rest and Exercise (Table 10)

Resting bradycardia (in this study, warm-up bradycardia) is a functional change and the evidence suggests that this is due to increased acetylcholine content in the sinus node area (Tipton, 1965). Frick, Elovainio and Somer (1967) have shown, by using experiments with beta-adrenergic antagonists, that reduced heart rates upon exertion after training implies diminished sympathetic drive.

Tension Time Index (TTI) (Table 14)

Bradycardia during rest and exercise, together with the decreased systolic pressures, significantly reduces the tension time index or myocardial oxygen demand. The decrease indicated that after training the subject can perform more external work before oxygen consumption of the heart attains a given level. This is encouraging since the training of cardiac patients is aimed at improving the ratio of oxygen supply to myocardial oxygen demand. This could signify that the myocardium has become "more efficient". A similar reduction had been observed by Hellerstein (1968), Frick and Katila (1968) and Kasch and Boyer (1969).

Oxygen Pulse (Table 10)

Oxygen pulse ($\dot{V}O_2$/HR) increased for all subjects of Groups 1 and 2 except in one instance (FC). In the calisthenics-walk-jog program a small increase was seen in only one subject (JD) and this occurred in the later stages of this program. Increases occurred, however, after a change of training from jogging to bicycling. The largest increases in oxygen pulse occurred with the higher evaluation work rates. Benestad (1968) has shown that untrained cardiac patients have characteristic low maximal exercise oxygen pulse values. The present study has shown that following appropriate training this situation can be improved. An increase in oxygen pulse is even more significant in this study because accompanying the decrease in exercise heart rate was a decrease in exercise oxygen uptake, nevertheless oxygen pulse ratio was increased with training. This data indicates a more economic or efficient system, where a larger oxygen extraction occurs per heart beat during a specific task. It is especially pertinent to the cardiac patient when a submaximal task can be performed adequately at a lower heart rate and still supply the adequate energy ($\dot{V}O_2$).

Blood Pressure (Table 13)

For most subjects exercise systolic blood pressure decreased. Resting values also decreased for many subjects, however, an increase was observed after an absence from training greater than two weeks (IB and MW). Resting diastolic blood pressure was significantly reduced with training in Groups 1 and 2. In three subjects (MB, LM and NS) who started the program with "normal" blood pressure there was a decrease for a considerable portion of training, however, blood pressure then increased to the initial values. In other parameters, the effects of holidays were also evident. Varnauskas

et al. (1966) felt that lowering the cardiac output in both rest and exercise may have contributed to this change in blood pressure. The pressure drop, together with the decreased blood-flow that they found in their studies, resulted in a significant lowering of the left ventricular work. They concluded that the hemodynamic effect of training which was responsible for the demonstrated increased working capacity was the significantly lowered left ventricular work. Frick and Katila (1968) also found an improvement in left ventricular function along with a significant increase in the rate of rise of right ventricular pressure. They attributed these results to myocardial hypertrophy, which through reduced compliance also accounted for the increased ventricular filling pressures.

Developed Collateral Circulation

The improvement in physical work capacity may be due to the training-induced acceleration of collateral intercoronary circulation development and therefore improved blood supply to the ischemic area of the myocardium. This was first found experimentally by Eckstein (1957) and later confirmed by others, (Leon and Bloor, 1968 and Tomanek, 1970). Kattus (1968) has shown that myocardial revascularization occurs in cardiac patients through an exercise rehabilitation program. The disappearance of angina in the subjects in the present study, especially subjects FC, JD and MW, and the disappearance of premature ectopic contractions could be attributed to myocardial revascularization. The difficulty of obtaining medical approval for pre and post-training coronary angiograms denied the investigation of this possible evaluation. The elimination of premature ventricular beats by exercise conditioning was also reported by Heller (1967) and Hellerstein (1968). Many patients in the present program also could be withdrawn from or maintained

on reduced dosages of medication for cardiac arrhythmias and from cardiotonic agents.

Cardiac Output (Tables 6 and 11)

The consistent decrease shown in cardiac output in submaximal exercise in this study was also observed by Varnauskas et al. (1966) in their cardiac rehabilitation program. Clausen et al. (1969) also found a significant decrease in the cardiac output for a given oxygen uptake in two of nine patients. The conditioning response observed by Frick and Katila (1968) was a reduced heart rate/increased stroke volume pattern with cardiac output lower at the first testing but not at the second. The authors suggested that the lower cardiac output at the first load was caused by an increase in the mechanical efficiency at a load at which the subjects had mostly been trained. Some of the exercise responses evident in the study by Frick and Katila are different from the present study and others probably due to the fact that supine exercise evaluation was used by the former group and upright exercise evaluation has been used by the present and other studies. In the study by Frick and Katila (1968), resting heart rate, stroke volume and cardiac output remained at the pretraining level whereas in the study by Varnauskas et al. (1966) resting cardiac output was decreased. In the present study, warm-up heart rate generally decreased while stroke volume showed a small consistent increase and warm-up cardiac output showed a small average decrease for all groups. These changes were not however statistically significant.

At rest, the investigation of Frick and Katila (1968) showed a thirty-three per cent increase in pulmonary capillary wedge pressure. This rise was compensated by a thirty-six percent decrease in pulmonary vascular resistance

therefore allowing the pulmonary arterial pressure to remain at the pretraining level. The elevated pulmonary capillary wedge pressures were interpreted as being related to noncompliant left ventricles due to fibrosis and scarring inherent in coronary arterial disease. Training increased these pressures at rest without any increment in the radiologically determined heart volumes. Myocardial hypertrophy superimposed on the already noncompliant left ventricles was also suggested as a possible explanation.

Varnauskas et al. (1966) and Ekblom (1969) suggested the decrease in cardiac output with physical conditioning is accompanied by an alteration of its distribution. With increasing work loads a progressively greater proportion of the linearly increasing cardiac output is directed to the working muscles and the myocardium, whereas the flow to the non-exercising tissues shows a concomitant decrement. The decrease in cardiac output at submaximal work loads after training is attributed to a redistribution of blood flow from other body tissues to the exercising muscles. It is suggested that after training, the perfusion of the exercising muscles was favored by a more pronounced restriction of splanchnic and renal flow.

Clausen (1970) and Clausen et al. (1969,1970) showed that actually after physical conditioning the perfusion of these non-exercising tissues is relatively increased at a submaximal work load, however, cardiac output is decreased due to a reduction in the skeletal muscle blood flow (both myocardium and exercising muscle). The investigators suggested that such a change might be fundamental for the altered circulatory regulation by reducing general sympathetic vasoconstriction during exercise. These findings are contrary to classical theories of exercise blood flow but are further supported by investigation which has shown an increased oxygen extraction in the trained muscle (Holloszy, 1967). Biochemical studies in

animals also have shown that physical conditioning results in increased activity of oxidative mitochondrial enzymes and an increase in the number and size of the mitochondria (Holloszy, 1967; Gollnick and King, 1969; Banister, Tomanek and Cvorkov, 1971).

Clausen (1970) concluded that the beneficial effect of coronary rehabilitation programs on angina pectoris is mainly due to a "reduction at a given submaximal work level of two of the main determinants of the myocardial oxygen requirement - i.e. the heart rate and the systolic blood pressure". Decrement in these parameters is a feature of the present study. He continued by suggesting that the reduced heart rate resulting from physical training is, in normal people, due to an increased stroke volume. However, the present study shows that some patients with coronary heart disease do not increase their stroke volume in response to training. They obtain lower heart rates at a given work load because the cardiac output decreases, possibly due to lowered muscle blood flow.

In explanation of results contrary to the above theory Clausen et al. (1969) suggested that an augmentation of the flow to the non-working tissues occurred in such cases. They continued by stating that:

> "an acceptable explanation for this phenomenon is available when it is concerned that patients with coronary artery disease (CAD) generally have subnormal values for stroke volume and cardiac output for a given oxygen consumption. As a consequence a more pronounced restriction of the flow to nonworking tissues, effected by an abnormally steep rise of the general sympathetic vasoconstrictive tone, is needed at a given work load, to provide a sufficient blood supply to the working muscles ... If the same work can be performed after physical conditioning with lower muscle blood flow, a reduction of the sympathetic tone and thus of the peripheral resistance, is possible. Hereby a more adequate perfusion of the nonexercising tissues could be obtained."

In the present study the effects of physical conditioning on submaximal exercise stroke volume was variable, however, in most subjects, it either

remained unchanged or showed an increase in the early period followed by a decrease throughout the rest of the program. This decrease often occurred during the period of the most successful type of training - continuous bicycle ergometry. Varnauskas et al. (1966) also observed significantly lower values for exercise stroke volume after training. In a few subjects the decrease in cardiac output occurred primarily through a reduction in heart rate with only a small change in stroke volume.

Frick and Katila (1968) state that in the case of those subjects who show a reduced heart rate/increased stroke volume training pattern, the increased stroke volume in coronary patients is due to cardiac hypertrophy without a change in heart volume. Investigations have shown that with "normal" subjects the increased stroke volume training response is related to a heart volume increment which does not apparently occur with coronary patients.

Benestad (1968) suggests that for many post infarction patients low aerobic work power can be related to the myocardial lesion, which may have reduced the mass of contractile structures thereby imposing a cardiac limitation on maximal work output. Among other consequences this would reduce contractile power. This could be a possible reason for the failure of subjects JD, EK and MW to increase exercise stroke volume above resting levels and therefore yields low exercise cardiac outputs and limits work capacity. Other possible explanations include noncompliant ventricles, due to fibrosis and scarring, and incomplete ventricular filling due to, among other factors, poor venous return.

Studies by Clausen et al. (1969, 1970) and Frick et al. (1968) have calculated the exercise effect on total peripheral vascular resistance. From

the values of mean arterial blood pressure ($P_{A\ mean}$) and cardiac output (C.O.) the total peripheral vascular resistance (TPVR) can be calculated ($TPVR = \frac{P_{A\ mean}\ (mmHg)}{C.O.\ l/min}$). From the changes induced by training in this study it seems feasible that a decrease in TPVR, during testing work rates, would have occurred in many patients and indeed they were (e.g. Grp 1, Subj NS: 10.3 → 9.30 mmHg/l/min, (900 kpm/min); Grp 2, Subj DB: 8.52 → 8.06 mmHg/l/min, (875 kpm/min); Grp 3, Subj BQ: 6.27 → 5.36 mmHg/l/min, (750 kpm/min)). However, resting values for TPVR remained unchanged (Group Means: Initial → 24 weeks, Grp 1: 17.58 → 17.52 mmHg/l/min; Grp 2: 19.52 → 19.49 mmHg/l/min; Grp 3: 15.28 →15.96 mmHg/l/min). In the four to ten week rehabilitation study of Clausen and Trap-Jensen (1970) TPVR was unchanged at rest and during moderate submaximal work (60 per cent of pretraining maximum) but was significantly lower at the heavy work load which was an average of 633 kpm/min (a load the subject could tolerate before training for twelve to fifteen minutes). Due to the short training period used, the magnitude of this change was much less than in the present study. Frick et al. (1968) did not find a significant change with training. These differences in observed training effects are probably due to the fact that the patients in former studies exercised in the upright position whereas those in the study of Frick et al. (1968) were examined in the supine position.

Red blood cell count, hematocrit, mean cell volume and hemoglobin, which were were routinely measured, did not change significantly in any of the fourteen subjects.

Respiratory and Metabolic Responses to Training

Pulmonary Function

The pulmonary function tests of resting tidal volume and respiratory rate did not change with training. Vital capacity and values for FEV_1 showed over-all small significant increases (Table 8). In all groups a significant increase in vital capacity occurred after ten weeks of training however no further increase occurred afterwards.

Some subjects (IB, DB, MB, WF and WS) showed considerable increases in their FEV_1. The increases usually occurred in the early part of the training except in the case of WF who showed an increase in the later stage when training was changed to the ergometer. These early increases in the "dynamic spirometer parameters" indicate increased mechanical properties of the lungs and chest wall.

Respiratory Gas Exchange and Metabolic Responses (Tables 7, 9, 10 and 12)

The maximal oxygen uptake of these patients were not measured because it was not considered safe. The large increases in physical work capacity indicate increased maximal oxygen uptake. Direct measurements of oxygen uptake and carbon dioxide elimination were measured routinely for the three evaluation work rates. Benestad (1968) found that the mean values for oxygen uptake for twenty-four cardiac patients were higher than for a comparable group especially at lower rates of work. The investigation also found that the pulmonary ventilation efficiency for the cardiac patients was lower than for the normals. The deterioration was not so distinct as in a group of old people (70-83 years).

The training response shown in the present rehabilitation study was identified by reduced ventilation, oxygen uptake and carbon dioxide

elimination for submaximal work rates in all subjects. Frick and Katila (1968) observed that during their first exercise load both ventilation and oxygen uptake were lower in every subject after the training but at the second load the changes were not significant. Clausen et al. (1969) found a significant decrease in ventilation however oxygen uptake did not change. No changes were seen in either ventilation or oxygen uptake for submaximal work loads either in the program of Varnauskas et al. (1966). It must be remembered however, when comparing any of the results of the present study to others in the literature, that this study occurred over six months as compared to four to six weeks for Varnauskas et al. (1966), Frick and Katila (1968) and Clausen et al. (1969). The training response of normal subjects in terms of submaximal ventilation and oxygen uptake varies from study to study. Astrand and Rodahl (1970) presented evidence that in one group the two decreased and in another ventilation decreased whereas oxygen uptake was unchanged.

During submaximal work pulmonary ventilation is primarily determined by the CO_2 production which is directly related to the oxygen utilization (Astrand and Rodahl, 1970). Since the blood lactate level has been shown to be lower after submaximal work following training in this and other studies, (Astrand and Rodahl, 1970), the respiratory drive is reduced. This results in a reduction of pulmonary ventilation. The decreases in ventilation and oxygen uptake may be accounted for by consideration of the effects of coronary heart disease and physical conditioning on the efficiency of muscular work. Whipp and Wasserman (1969) state that patients with aortic stenosis and other forms of coronary heart disease require supplemental energy (substrate and oxygen) to perform a given workload, since cardiac work is increased. The increased cardiac work is not part of the measured external

work but does result in an increase in oxygen uptake. They suggest that the "work efficiency" of these subjects is thereby reduced. Abramovich (1948) has shown that oxygen consumption in man with compensated cardiac hyperfunction is increased by an average of twelve per cent under basal metabolic conditions. Levine and Wagman (1962) studied myocardial oxygen consumption in twelve healthy individuals and in seven individuals with overt heart failure secondary to valvular lesion or hyperfunction. The hypertrophied heart of the cardiac patient utilized sixteen per cent of the entire oxygen consumption of the body whereas the normal heart accounted for only five to six per cent of this total. The authors concluded that the hypertrophied heart acts as an "oxygen trap". As training progresses it is possible that this extra cardiac work will be reduced and the $\dot{V}O_2$ for a standard work rate will decrease (i.e. "work efficiency" increases). The biochemical and mitochondrial changes induced by exercise training will allow greater oxygen extraction and decrease myocardial oxygen uptake (Holloszy, 1967; Gollnick and King, 1969; Banister, Tomanek and Cvorkov, 1971). Whipp and Wasserman continue in their discussion of the efficiency of muscular work by stating that physical training will allow the performance of a task with less stress on an organ system. For example, pulmonary ventilation necessary for a given task will be less in a trained than in an untrained subject. They describe this as an increased "physiological efficiency" which is specific to the organ system under consideration. They have described another form of efficiency which has to an extent taken place with all subjects in the early part of training. This form, they classify as "motor efficiency" which relates to the actual work output in performing a work task. This is increased when a more skillful performance decreased the work required to perform the task. Therefore, as a result of the twenty-four week rehabilitation program in this

study all subjects have shown increased work, physiological and motor efficiency.

The ventilation equivalent ($\dot{V}_E/\dot{V}O_2$) for eight of the fourteen subjects decreased for a standard work rate after training. This decrease indicates an increase in pulmonary ventilation efficiency ("physiological efficiency"). The largest decreases were associated with the low and intermediate work rates. Three subjects in Group 1, four in Group 2 and only one in Group 3 showed this decrease which suggests that ergometry training induced the largest change in pulmonary efficiency. In the remaining subjects the ventilation equivalent remained unchanged indicating that pulmonary ventilation and oxygen uptake decreased proportionally with training. Within the twenty-four week period two subjects showed small increases in the ratio of $\dot{V}_E/\dot{V}O_2$ which resulted after an absence from training. However, further training decreased the values once more.

Carbon dioxide elimination was also observed to be significantly reduced as a result of training. The effect and magnitude of the reduced $\dot{V}CO_2$ may be seen in the reduced respiratory exchange ratios. Blood lactates and also base excess values were less at a submaximal task after training. This accounts partly for the lower observed values for $\dot{V}CO_2$. Carbon dioxide elimination will also be decreased due to the decrease in pulmonary ventilation as a result of the increased "physiological efficiency". Other cardiac rehabilitation programs have observed the lowered lactate values with training (Varnauskas et al., 1966; Frick and Katila, 1968 and Clausen et al., 1969). Decreased lactate, base excess and R values can indicate increased ability for oxidative metabolism after training which would support the theory of increased oxygen extraction allowing for the reduced muscle blood flow that Clausen (1970) claims is responsible for the decreased cardiac output after training. Frick and Katila (1968) found reduced lactate and excess lactate

concentrations after training and suggested that this is a result of a lower degree of anaerobic metabolism and more effective splanchnic removal of lactate after the training. Rowell et al. (1966) have shown that throughout the course of muscular exercise a large fraction of lactate (estimated as 50 per cent) is removed by the liver, kidneys, heart and skeletal muscles. It is possible that after training the heart of these cardiac patients could remove proportionally more lactate for its metabolism especially in the light of better coronary vascularization.

Serum Lipid Response to Training (Table 5)

Body weight remained unchanged in nine of the subjects, increased slightly in three (MB, HG, JD) and decreased in two (DB, FC). In all subjects serum cholesterol levels were significantly reduced with training and with no manipulation of diet. The subject who showed the largest weight loss (DB) also had the largest reduction in his serum cholesterol value (302 → 145 mg%), however, those subjects who increased weight also showed decreased cholesterol with training. Malinov, Perley and McLaughlin (1968a, 1968b, 1969) have shown in animals that the oxidation of cholesterol increases with muscular contraction. The liver and adrenals have been shown to be mainly responsible for splitting the side chain of cholesterol during muscular exercise. However, conflicting evidence appears in the literature on the effect of exercise on the reduction of serum lipids. Often, where physical conditioning was not accompanied by a decrease in body weight, there was no reduction in cholesterol levels (Skinner et al., 1967 and Pollock et al., 1969). Siegel et al. (1970) recently have shown a significant reduction in cholesterol without any loss in total body weight.

The mean serum triglyceride levels of the fourteen subjects in this

study showed a small but not significant decrease with training. Individually, some subjects showed large reductions. As in the study by Siegel et al. (1970) the decrease was variable in many subjects. In only one subject (HG), however, did a consistent increase occur. Any absence from training increased the current triglyceride level. Skinner et al. (1967) observed a similar situation and concluded that the decrease in the fasting serum triglycerides which occurred as a result of an exercise program was an acute short-term effect that persisted for at least forty-four hours. This effect also appeared to be cumulative when a regular endurance conditioning program was performed. In some subjects an early response to exercise was an increase in serum triglycerides suggesting a mobilizing effect by exercise before a reduction could take place. It should be mentioned that in the present study all serum lipid values were not fasting values. These determinations were taken two hours after a light breakfast and it was felt that these values are more meaningful than an artificial twelve hour fasting value in that they reflect the true level that the patient would possess throughout his daily living.

It is concluded that the hemodynamic and emotional changes which have occurred in these fourteen patients form a rational physiological basis for the use of physical training in the rehabilitation of the post myocardial infarct patient.

Future Considerations - A Logical Progression

As a result of this study and the large number of post-infarction patients in this area it has become apparent that a more elaborate community involvement is necessary. Various stages in the rehabilitation seem to be indicated. The first stage occurs within the hospital's Intensive Care Units. The bed exercise and types of physical activities appropriate during this time are outlined by

Kottke (1967). Once the patient leaves the hospital, exercise evaluation tests should be performed to indicate whether exercise training is considered appropriate. These tests could be performed in local test centres. On the basis of these results an individualized exercise program can be prescribed for the patient to be done under close supervision in the test centre with continuous cardiac monitoring in the initial months. The present study indicated that close supervision is easiest done with the use of bicycle ergometers. Warm-up calisthenics should be postponed for a period of time until confidence in the subject is achieved. After that time close attention should be placed on those overweight. The cyclic form of ergometry appears appropriate for those weak or with marked coronary insufficiency. After a period of time, dependent on the individual, continuous ergometry could be successfully used. Routine evaluation every two weeks, besides continuous training monitoring, is essential during this period to update the exercise program and determine the level of medication necessary.

When the patients attain a high exercise tolerance with freedom from arrhythmias and decreased dependence on drug therapy, the need for close supervision and monitoring decreases. The patient can then be placed in the hands of an associated institution such as the YMCA which can handle larger numbers but are specially trained to supervise the management of these patients. Evaluation tests could be performed monthly and then on a three to four month basis as his condition improves. With such a scheme it is hopeful that the present five year figures of mortality, after an infarction, can be reduced.

In summary, the management of post myocardial infarct patients is a large and involved undertaking, however, as this and other similar programs have shown, carefully controlled exercise therapy can progressively restore

and maintain the mental and physical state of the patient.

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APPENDIXES

APPENDIX 1

Computer program for cardio-respiratory parameters
for exercise evaluation sessions.

```
                C PROGRAM TO CALCULATE VARIOUS RESPIRATORY AND CARDIOVASCULAR PARAMETERS
                C NAMELY: VEATPSVESTPD VCO2 VO2 R TRUEO2 C2PULSE VENT EQUIV HR
                C ALSO    CARDIAC OUTPUT MYOCARDIAL O2 DEMAND ARTERIAL DISTENSIBILITY.
0001                  INTEGER SYS,DIA,PULS,BARO,HR,INRT,NAME,DATE,LOADA,LOADB,LOADC
CC02                  INTEGER SERCHA,SERCHB,SERCHC
C003                  DIMENSION VCL(20),TEMP(20),CXY(600),CDIC(600),TABLE(41,18),HR(20)
0004                  DIMENSION CAL(3),SYS(2),DIA(2),PACO(2),REBR(2),INRT(20)
CC05                  DIMENSION PULS(2),NAME(20),DATE(8)
CC06                  DIMENSION VESTPD(21),VCO(21)
0007                  DIMENSION CORR(71)
CCC8                  DIMENSION VENT(17),VENTT(17),VENTTT(17)
CCC9                  DIMENSION HRR(17),HRRR(17),HRRRR(17)
CC10                  DIMENSION VVO(17),VVVO(17),VVVVO(17)
C011                  DIMENSION DENT(17),DENTT(17),DENTTT(17)
C012                  DIMENSION DRR(17),DRRR(17),DRRRR(17)
CC13                  DIMENSION DVO(17),DVVO(17),DVVVO(17)
CC14                  DIMENSION ARRAYX(17)
CC15                  READ(5,500)CORR
CC16                500 FORMAT(16F5.1)
C017                  DO 203 I=1,41
CC18                  READ(5,204)(TABLE(I,J),J=1,18)
CC19                204 FORMAT (16F5.3/2F5.3)
CC20                203 CONTINUE
0C21                  NWEEK=0
CC22                  NCONT=0
CC23                  NCOUT=0
0024                  NCOUNT=0
0025                  NARRAY=-6
CC26                  READ(5,1000)SERCHA,SERCHB,SERCHC
CC27               1000 FORMAT(3I5)
C028                  DO 2000 II=1,15
0029                  4 READ (5,200)NAME,DATE,LOADA,LOADB,LOADC
CC30                200 FORMAT(20A4,/8A4,3I5)
0C31                  NWEEK=NWEEK+1
CC32                  NARRAY=(NARRAY+2)
CC33                  ARRAYX(NWEEK)=NARRAY
C034                  5 WRITE (6,201)
0035                201 FORMAT('1',T40,'RESULTS OF CARDIOVASCULAR AND RESPIRATORY DATA'
                       1       //6X,'TAKEN FROM')
                C HEADING
0036                  6 WRITE (6,202) NAME,DATE,LOADA,LOADB,LOADC
0037                202 FORMAT('+',T20,20A4,T100,8A4,//11X,' LOAD1=',I5,/11X,' LOAD2=',I5,
                       1 /11X,' LOAD3=',I5)
                C TITLE CONDITIONS SET
0038                  9 READ(5,205)CAL(1),CAL(2),CAL(3)
C039                205 FORMAT(3F10.4)
C040                 10 READ(5,206)((SYS(M),DIA(M),PULS(M),REBR(M),PACO(M)),M=1,2)
0041                206 FORMAT(3I10,2F10.2)
0042                 11 READ (5,207)BARO
CC43                207 FORMAT (I5)
                C ABOVE TO 8 IS DATA READ.
0044                 12 WRITE (6,208)
```

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FORTRAN IV G LEVEL  18                  MAIN                DATE = 70336          18/08/07

0045            208 FORMAT(/7X,'VEATPS(L/MIN)',T23,'VESTPD(L/MIN',T37,'VCO2(L/MIN)',
                   1T51,'VO2(L/MIN)',T65,'R',T71,'TRUE O2',T81,'O2 PULSE(L/BEAT)',
                   2T100,'VENT EQUIV',T114,'HR',/T40,'WARM-UP')
                C PLOT PARAMETERS TO BE WORKED,ACROSS THE PAGE.
0046             13 DO 209  K=1,20
0047             14 READ (5,210)VOL(K),TEMP(K),HR(K),INRT(K)
0048            210 FORMAT(F5.1,F10.1,2I5)
                C WRITING OUT UNCORRECTED VOLUME OF GAS BREATHED IN L/MIN.
0049             16 I=TEMP(K)
0050             17 J=BARO
0051             18 VESTPD(K)=VOL(K)*TABLE((J-698)/2,I-14)
                C WRITING OUT CORRECTED VOLUME IN L/MIN.
0052                INRTE=INRT(K)
0053             20 DO 213 L=1,INRTE
0054             21 READ (5,214)CO10(L)
0055            214 FORMAT(10F8.3)
0056            213 CONTINUE
0057             22 SUM=0
0058                INRTE=INRT(K)
0059             23 DO 215 M=1,INRTE
0060             24 SUM=SUM+CO10(M)
0061            215 CONTINUE
0062             25 MEAN=SUM/INRT(K)
0063             26 FECO=((MEAN*CAL(1)*.7)+.03 )
                C CALCULATING MEAN % OF EXPIRED CO2 IN 1 MIN
0064                INRTE=INRT(K)
0065             27 DO 216 I=1,INRTE
0066             28 READ (5,217)CXY(I)
0067            217 FORMAT(10F8.3)
0068            216 CONTINUE
0069             29 SUM=0
0070                INRTE=INRT(K)
0071             30 DO 218 I=1,INRTE
0072             31 SUM=SUM+CXY(I)
0073            218 CONTINUE
0074             32 MEAN=SUM/INRT(K)
0075             33 FECO=(20.93-(MEAN*CAL(2)*.7) )
                C CALCULATING MEAN % OF EXPIRED O2 OVER 1MIN
0076             34 FENN=100-(FECO+FCCO)
0077             35 VCCO=(VESTPD(K)*.01)*(FCOO-(FENN*.0003795))
0078             36 IF(K.LQ.2) VCOOO=VCOO
0079             37 IF(K.EQ.20)VCO=VCOO
                C CALCULATING VOLUME OF EXPIRED CO2.
0080             40 VCO(K)=(VESTPD(K)*.01)*((FENN*.2648)-FCOO)
                C CALCULATING VOLUME O2 USED IN 1MIN.
0081             42 R=VCCO/VCO(K)
                C CALCULATING R RATIO OVER 1MIN
0082             43 TRUE=(VCO(K)*100)/VESTPD(K)
                C CALCULATE  TRUE O2
0083             45 COPL=VOO(K)/HR(K)
                C CALCULATE  O2 PULSE IN L/BEAT.
0084             47 VINT =1/TRUE
```

```
FORTRAN IV G LEVEL  18                  MAIN                DATE = 70336          18/08/07

0085                WRITE(6,41)VOL(K),VESTPD(K),VCOO,VOO(K),R,TRUE,OOPL,VINT,HR(K)
0086              41 FORMAT  (/11X,F5.1,T28,F5.1,T39,F9.3,T51,F9.3,T64,F5.3,T74,F5.3,
                   1T8°,F5.4,T104,F5.2,T112,I5)
0087                IF (K.EQ.8) GO TO 1100
0088                IF (K.EC.14)GO TO 1101
0089                IF (K.EC.20)GO TO 1102
0090                GO TO 51
0091             1100 IF (LOADA.EQ.SERCHA)GO TO 1103
0092                IF (LOADA.EC.SERCHB) GO TO 1104
0093                IF (LCADA.EC.SERCHC) GO TO 1105
0094                GO TO 1CO
0095             1101 IF(LOADB.EQ.SERCHA) GO TC 1103
0096                IF(LOADB.EQ.SERCHB) GO TO 1104
0097                IF(LOADB.EQ.SERCHC) GO TO 1105
0098                GO TO 100
0099             1102 IF(LOADC.EQ.SERCHA) GO TO 1103
0100                IF(LOADC.EQ.SERCHB) GO TO 1104
0101                IF(LOADC.EQ.SERCHC) GO TO 1105
0102                GO TO 100
0103             1103 NCONT=NCONT+1
0104                VENT (NWEEK)=VESTPD(K)
0105                HRR  (NWEEK)=HR (K)
0106                VVO  (NWEEK)=VOO(K)
0107                GO TO 1CO
0108             1104 NCOUT=NCOUT+1
0109                VENTT(NWEEK)=VESTPD(K)
0110                HRRR(NWEEK)=HR(K)
0111                VVVO(NWEEK)=VOO(K)
0112                GO TC 100
0113             1105 NCOUNT=NCOUNT+1
0114                VENTTT(NWEEK)=VESTPD(K)
0115                HRRRR  (NWEEK)=HR(K)
0116                VVVVO (NWEEK)=VCO(K)
0117                GC TC 1CC
0118              100 IF(K.LT.20) GO TO 51
0119             1106 IF(NCONT.EQ.NWEEK)GO TO 1107
0120                VENT (NWEEK)=0
0121                HRR  (NWEEK)=0
0122                VVO  (NWEEK)=0
0123                NCONT=NWEEK
0124             1107 IF(NCOUT.EQ.NWEEK)GO TO 1108
0125                VENTT(NWEEK)=0
0126                HRRR (NWEEK)=0
0127                VVVO (NWEEK)=0
0128                NCOUT=NWEEK
0129             1108 IF(NCOUNT.EQ.NWEEK)GO TO 1109
0130                VENTTT(NWEEK)=0
0131                HRRRR(NWEEK)=0
0132                VVVVO (NWEEK)=0
0133                NCOUNT=NWEEK
0134                IF(II.EQ.1) GO TO 5C
0135             1109 DENT (NWEEK)=VENT (NWEEK)-VENT (NWEEK-1)
```

APPENDIX 2

Computer plot program to give graphs of heart rate, ventilation, oxygen uptake and carbon dioxide elimination for specific work rates throughout the training period.

```
FORTRAN IV G LEVEL 18                    MAIN                    DATE = 70336          18/08/07

C0095               WRITE(6,41)V(K),VESTPD(K),VCOO,VOO(K),R,TRUE,OOPL,VINT,HR(K)
C0096            41 FORMAT  (/11X,F5.1,T20,F5.1,T39,F9.3,T51,F9.3,T64,F5.3,T74,F5.3,
                   1T89,F5.4,T104,F5.2,T112,I5)
C0097               IF (K.EQ.P) GO TO 1100
C0098               IF (K.EQ.14)GO TO 1101
                    IF (K.EQ.20)GO TO 1102
                    GO TO 51
C0091            1100 IF (LOADA.EQ.SERCHA)GO TO 1103
C0092                 IF (LOADA.EQ.SERCHB) GO TO 1104
C0093                 IF (LOADA.EQ.SERCHC) GO TO 1105
C0094                 GO TO 100
C0095            1101 IF(LOADB.EQ.SERCHA) GO TO 1103
C0096                 IF(LOADB.EQ.SERCHB) GO TO 1104
C0097                 IF(LOADB.EQ.SERCHC) GO TO 1105
C0098                 GO TO 100
C0099            1102 IF(LOADC.EQ.SERCHA) GO TO 1103
C0100                 IF(LOADC.EQ.SERCHB) GO TO 1104
C0101                 IF(LOADC.EQ.SERCHC) GO TO 1105
C0102                 GO TO 100
C0103            1103 NCONT=NCONT+1
0104                  VENT (NWEEK)=VESTPD(K)
C0105                 HRR  (NWEEK)=HR (K)
C0106                 VVO  (NWEEK)=VOO(K)
C0107                 GO TO 100
C0108            1104 NCOUT=NCOUT+1
C0109                 VENTT(NWEEK)=VESTPD(K)
C0110                 HRRR(NWEEK)=HR(K)
0111                  VVVO(NWEEK)=VOO(K)
C0112                 GO TO 100
C0113            1105 NCOUNT=NCOUNT+1
C0114                 VENTTT(NWEEK)=VESTPD(K)
C0115                 HRRRR  (NWEEK)=HR(K)
C0116                 VVVVO (NWEEK)=VOO(K)
C0117                 GO TO 100
C0118             100 IF(K.LT.20) GO TO 51
C0119            1106 IF(NCONT.EQ.NWEEK)GO TO 1107
C0120                 VENT (NWEEK)=0
C0121                 HRR  (NWEEK)=0
C0122                 VVO  (NWEEK)=0
C0123                 NCONT=NWEEK
C0124            1107 IF(NCOUT.EQ.NWEEK)GO TO 1108
C0125                 VENTT(NWEEK)=0
C0126                 HRRR (NWEEK)=0
C0127                 VVVO (NWEEK)=0
C0128                 NCOUT=NWEEK
C0129            1108 IF(NCOUNT.EQ.NWEEK)GO TO 1109
C0130                 VENTTT(NWEEK)=0
C0131                 HRRRR(NWEEK)=0
C0132                 VVVVO (NWEEK)=0
C0133                 NCOUNT=NWEEK
C0134                 IF(II.EQ.1) GO TO 50
C0135            1109 VENT (NWEEK)=VENT (NWEEK)-VENT (NWEEK-1)
```

```
FORTRAN IV G LEVEL  18                  MAIN                    DATE = 70336          18/08/07

C136                DRR(NWEEK)=HRR(NWEEK)-HRR(NWEEK-1)
C137                DVO(NWEEK)=VVO(NWEEK)-VVO(NWEEK-1)
C138              1110 DENTT(NWEEK)=VENTT(NWEEK)-VENTT(NWEEK-1)
C139                DRRR(NWEEK)=HRRR(NWEEK)-HRRR(NWEEK-1)
C140                DVVO(NWEEK)=VVVO(NWEEK)-VVVO(NWEEK-1)
C141                DENTTT(NWEEK)=VENTTT(NWEEK)-VENTTT(NWEEK-1)
C142                DRRRR (NWEEK)=HRRRR(NWEEK)-HRRRR(NWEEK-1)
C143                DVVVO(NWEEK)=VVVVO(NWEEK)-VVVVO(NWEEK-1)
C144                IF(VENT(NWEEK-1).EQ.0) DENT(NWEEK)=20
C145                IF(VENT(NWEEK).EQ.0)DENT(NWEEK)=20
C146                IF (HRR(NWEEK-1).EQ.0) DRR(NWEEK)=20
C147                IF(HRR(NWEEK).EQ.0) DRR(NWEEK)=20
C148                IF (VVO(NWEEK-1).EQ.0) DVO(NWEEK)=1
C149                IF (VVO(NWEEK).EQ.0) DVO(NWEEK)=1
C150                IF(VENTT(NWEEK-1).EQ.0) DENTT(NWEEK)=20
C151                IF (VENTT(NWEEK).EQ.0) DENTT(NWEEK)=20
C152                IF (HRRR(NWEEK-1).EQ.0) DRRR(NWEEK)=20
C153                IF (HRRR(NWEEK).EQ.0) DRRR(NWEEK)=20
C154                IF (VVVO(NWEEK-1).EQ.0)DVVO(NWEEK)=1
C155                IF (VVVO(NWEEK).EQ.0) DVVO(NWEEK)=1
C156                IF (VENTTT(NWEEK-1).EQ.0) DENTTT (NWEEK)=20
C157                IF (VENTTT(NWEEK).EQ.0) DENTTT(NWEEK)=20
C158                IF (HRRRR(NWEEK-1).EQ.0) DRRRR(NWEEK)=20
C159                IF (HRRRR(NWEEK).EQ.0) DRRRR(NWEEK)=20
C160                IF (VVVVO(NWEEK-1).EQ.0) DVVVO(NWEEK)=1
C161                IF (VVVVO(NWEEK).EQ.0) DVVVO(NWEEK)=1
C162                WRITE (6,2222)
C163              2222 FORMAT (/7X,'VENT1',T14,'VENT2',T22,'VENT3',T30,
                   1 'HR1',T35,'HR2',T40,'HR3',T45,'VO2 1',T52,'VO2 2',T61,'VO2 3')
0164                WRITE(6,2223)DENT(NWEEK),DENTT(NWEEK),DENTTT(NWEEK) ,
                   1 DRR(NWEEK),DRRR(NWEEK),DRRRR(NWEEK),
                   2DVO(NWEEK),DVVO(NWEEK),DVVVO(NWEEK)
0165              2223 FORMAT (/7X,F5.1,T14,F5.1,T22,F5.1,T30,F4.1,T35,F4.1,T40,F4.1,
                   1T45,F5.3,T52,F5.3,T61,F5.3)
0166                GO TO 51
C167                50  DENT(NWEEK)=20
C168                    DENTT(NWEEK)=20
C169                    DENTTT(NWEEK)=20
C170                    DRR(NWEEK)=20
C171                    DRRR(NWEEK)=20
C172                    DRRRR (NWEEK)=20
C173                    DVO(NWEEK)=1
C174                    DVVO(NWEEK)=1
C175                    DVVVO(NWEEK)=1
0176                51 IF(K.EQ.1)GO TO 209
0177                52 IF(K.EQ.2)GO TO 226
C178                53 GO TO 55
C179               226 WRITE (6,227)
C180               227 FORMAT(T40,'LOAD 1')
C181                54 GO TO 209
C182                55 IF(K.GT.2.AND.K.LT.8)GO TO 209
0183                56 IF(K.EQ.8)GO TO 228
```

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FORTRAN IV G LEVEL  18                  MAIN                DATE = 70336        18/08/07

0184                  IF(K.GT.8)GO TO 58
0185               228 WRITE (6,229)
0186               229 FORMAT(T40,'LOAD 2')
0187                57 GO TO 209
0188                58 IF(K.GT.8.AND.K.LT.14)GO TO 209
0189                59 IF (K.EQ.14) GO TO 230
0190                   IF (K.GT.8)GO TO 60
0191               230 WRITE (6,231)
0192               231 FORMAT(T40,'LOAD 3')
0193                   GO TO 209
                 C  FROM 60TO 51 BRANCHES INTRODUCED FOR DIFFERENT LOADS.
0194                60 IF(K.GT.14.AND.K.LT.20)GO TC 209
0195                   IF (K.EQ.20)GO TO 209
0196               209 CONTINUE
0197                61 WRITE (6,232)
0198               232 FORMAT(//7X,'CARDIAC OUTPUT(L/MIN)',T30,'SYSTOLIC',T44,'DIASTOLIC'
                      1 ,T60,'MYOCARDIAL DEMAND',T82,'ARTERIAL DISTENSIBILITY',
                      2T114,'HR',//T40,'WARM-UP')
                 C WRITING CARDIC PARAMETES ACROSS PAGE
0199                62 DO 233 N=1,2
0200                   IF(N.EQ.1)GO TO 1111
0201                   GO TO 63
0202              1111 PVCOO=(REBR(N)*BARO*CAL(1))/100
0203                   GO TO 1112
0204                63 PVCOO=(RFBR(N)*BARO*CAL(3))/100
0205              1112 CVCO=CORR(PVCOO-9)
0206                   ICVCO=CVCO+.5
0207                64 IF(N.EQ.1) GO TO 234
0208                   IF(N.GT.2) GO TO 233
0209                65 GO TO 235
0210               234 CACO=CORR(PACO(N)-9)
0211                   CO=(VCOOO*100)/(ICVCO-CACC)
0212                   GO TO 67
0213               235 CACC=CORR(PACO(N)-9)
0214                   CO=(VCO*100)/(ICVCO-CACO)
0215                67 MYOD=SYS(N)*PULS(N)
0216                69 ARTD=(CO/HR(N))/(SYS(N)-DIA(N))
0217                   WRITE(6,68)CO,SYS(N),DIA(N),MYOD,ARTD,PULS(N)
0218                68 FORMAT (/T12,F5.2,T33,I5,T48,I5,T65,I5,T90,F7.6,T112,I5)
0219                   IF(N.EQ.1)GO TO 241
0220                   GO TO 233
0221               241 WRITE(6,242)
0222               242 FORMAT(T40,'LAST MINUTE')
0223               233 CONTINUE
0224              2000 CONTINUE
                 C     PLOTTER PROGRAM
                 C     PLOT FOR VENT.
0225                   SERHA=SERCHA
0226                   SERHB=SERCHB
0227                   SERHC=SERCHC
0228                   CALL PLOTS
0229                   CALL SYMBOL (1.0,4.1,0.14,'LOAD=',0.0,5)
```

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FORTRAN IV G LEVEL  18                  MAIN                    DATE = 70336          18/08/07

0230                 CALL NUMBER (2.2,4.1,0.14,SERHA,0.0,0)
0231                 CALL SCALE(VENT,4.0,NWEEK,+1)
0232                 CALL SCALE (ARRAYX,4.0,NWEEK,+1)
0233                 CALL AXIS (0.0,0.0,'VENT L/MIN',+10,4.0,90.0,VENT(NWEEK+1),
                    1  VENT(NWEEK+2))
0234                  CALL AXIS (0.0,0.0,'TIME 2WEEKS',-11,4.0,0.0,ARRAYX(NWEEK+1),
                    1 ARRAYX(NWEEK+2))
0235                 CALL LINE(ARRAYX,VENT,NWEEK,1,-1,1)
0236                 CALL PLOT (0.0,5.0,-3)
0237                 CALL SYMBOL (1.0,4.1,0.14,'LOAD=',0.0,5)
0238                 CALL NUMBER (2.2,4.1,0.14,SERHB,0.0,0)
0239                  CALL SCALE(VENTT,4.0,NWEEK,+1)
C240                  CALL SCALE(ARRAYX,4.0,NWEEK,+1)
0241                 CALL AXIS (0.0,0.0,'VENT L/MIN',+10,4.0,90.0,VENTT(NWEEK+1),
                    1 VENTT(NWEEK+2))
0242                  CALL AXIS (0.0,0.0,'TIME 2WEEKS',-11,4.0,0.0,ARRAYX(NWEEK+1),
                    1ARRAYX(NWEEK+2))
0243                 CALL LINE(ARRAYX,VENTT,NWEEK,1,-1,1)
0244                 CALL PLOT (5.0,0.0,-3)
0245                 CALL SYMBOL (1.0,4.1,0.14,'LOAD=',0.0,5)
0246                 CALL NUMBER (2.2,4.1,0.14,SERHC,0.0,0)
C247                 CALL SCALE (VENTTT,4.0,NWEEK,+1)
C248                 CALL SCALE (ARRAYX,4.0,NWEEK,+1)
0249                 CALL AXIS (0.0,0.0,'VENT L/MIN',+10,4.0,90.0,VENTTT(NWEEK+1),
                    1 VENTTT(NWEEK+2))
0250                 CALL AXIS (0.0,0.0,'TIME 2WEEKS',-11,4.0,0.0,ARRAYX(NWEEK+1),
                    1 ARRAYX(NWEEK+2))
0251                 CALL LINE(ARRAYX,VENTTT,NWEEK,1,-1,1)
               C     PLOT HEART RATE
0252                 CALL PLOT (C.0,-5.0,-3)
0253                 CALL SYMBOL (1.0,4.1,0.14,'LOAD=',0.0,5)
0254                 CALL NUMBER (2.2,4.1,0.14,SERHA,0.0,0)
C255                 CALL SCALE (HRR,4.0,NWEEK,+1)
0256                 CALL SCALE (ARRAYX,4.0,NWEEK,+1)
0257                 CALL AXIS (0.0,0.0,'HR BEATS/MIN',+12,4.0,90.0,HRR(NWEEK+1),
                    1HRR(NWEEK+2))
0258                 CALL AXIS (0.0,0.0,'TIME 2WEEKS',-11,4.0,0.0,ARRAYX(NWEEK+1),
                    1 ARRAYX(NWEEK+2))
C259                 CALL LINE(ARRAYX,HRR,NWEEK,1,-1,1)
C260                 CALL PLOT (5.0,C.0,-3)
0261                 CALL SYMBOL (1.0,4.1,0.14,'LOAD=',0.0,5)
C262                 CALL NUMBER (2.2,4.1,0.14,SERHB,0.0,C)
0263                 CALL SCALE(HRRR,4.0,NWEEK,+1)
0264                 CALL SCALE(ARRAYX,4.0,NWEEK,+1)
C265                 CALL AXIS (0.0,0.0,'HR BEATS/MIN',+12,4.0,90.0,HRRR(NWEEK+1),
                    1 HRRR(NWEEK+2))
0266                 CALL AXIS(0.0,0.0,'TIME 2WEEKS',-11,4.0,0.0,ARRAYX(NWEEK+1),
                    1 ARRAYX(NWEEK+2))
C267                 CALL LINE(ARRAYX,HRRR,NWEEK,1,-1,1)
C268                 CALL PLOT (0.0,5.0,-3)
0269                 CALL SYMBOL (1.0,4.1,0.14,'LOAD=',0.0,5)
C270                 CALL NUMBER (2.2,4.1,0.14,SERHC,0.0,0)
```

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FORTRAN IV G LEVEL  18                  MAIN                DATE = 70336          18/08/07

0271                 CALL SCALE (HRRRR,4.0,NWEEK,+1)
0272                 CALL SCALE(ARRAYX,4.0,NWEEK,+1)
0273                 CALL AXIS (0.0,0.0,'HR BEATS/MIN',+12,4.0,90.0,HRRRR(NWEEK+1),
                    1 HRRRR(NWEEK+2))
0274                 CALL AXIS (0.0,0.0,'TIME 2WEEKS',-11,4.0,0.0,ARRAYX(NWEEK+1),
                    1 ARRAYX(NWEEK+2))
0275                 CALL LINE (ARRAYX,HRRRR,NWEEK,+1,-1,1)
                C    PLOT  VC2
0276                 CALL PLOT (5.0,0.0,-3)
0277                 CALL SYMBOL (1.0,4.1,0.14,'LOAD=',0.0,5)
0278                 CALL NUMBER (2.2,4.1,0.14,SERHA,0.0,0)
0279                 CALL SCALE (VVO,4.0,NWEEK,+1)
0280                 CALL SCALE (ARRAYX,4.0,NWEEK,+1)
0281                 CALL AXIS (0.0,0.0,'VO2 L/MIN',+9,4.0,90.0,VVO(NWEEK+1),
                    1 VVO(NWEEK+2))
0282                 CALL AXIS (0.0,0.0,'TIME 2WEEKS',-11,4.0,0.0,ARRAYX(NWEEK+1),
                    1 ARRAYX(NWEEK+2))
0283                 CALL LINE (ARRAYX,VVO,NWEEK,+1,-1,1)
0284                 CALL PLOT (0.0,-5.0,-3)
0285                 CALL SYMBOL (1.0,4.1,0.14,'LOAD=',0.0,5)
0286                 CALL NUMBER (2.2,4.1,0.14,SERHB,0.0,0)
0287                 CALL SCALE (VVVO,4.0,NWEEK,+1)
0288                 CALL SCALE (ARRAYX,4.0,NWEEK,+1)
0289                 CALL AXIS (0.0,0.0,'VO2 L/MIN',+9,4.0,90.0,VVVO(NWEEK+1),
                    1 VVVO(NWEEK+2))
0290                 CALL AXIS (0.0,0.0,'TIME 2WEEKS',-11,4.0,0.0,ARRAYX(NWEEK+1),
                    1 ARRAYX(NWEEK+2))
0291                 CALL LINE (ARRAYX,VVVO,NWEEK,+1,-1,1)
0292                 CALL PLOT (5.0,0.0,-3)
0293                 CALL SYMBOL (1.0,4.1,0.14,'LOAD=',0.0,5)
0294                 CALL NUMBER (2.2,4.1,0.14,SERHC,0.0,0)
0295                 CALL SCALE (VVVVO,4.0,NWEEK,+1)
0296                 CALL SCALE (ARRAYX,4.0,NWEEK,+1)
0297                 CALL AXIS (0.0,0.0,'VO2 L/MIN',+9,4.0,90.0,VVVVO(NWEEK+1),
                    1  VVVVO(NWEEK+2))
0298                 CALL AXIS (0.0,0.0,'TIME 2WEEKS',-11,4.0,0.0,ARRAYX(NWEEK+1),
                    1 ARRAYX(NWEEK+2))
0299                 CALL LINE(ARRAYX,VVVVO,NWEEK,1,-1,1)
                C    PLOT CHANGE VENT L/MIN/2WEEKS
0300                 CALL PLOT (0.0,5.0,-3)
0301                 CALL SYMBOL (1.0,4.1,0.14,'LOAD=',0.0,5)
0302                 CALL NUMBER (2.2,4.1,0.14,SERHA,0.0,0)
0303                 CALL SCALE (DENT,4.0,NWEEK,+1)
0304                 CALL SCALE (ARRAYX,4.0,NWEEK,+1)
0305                 CALL AXIS (0.0,0.0,'CHANGE VENT/2WEEKS',+18,4.0,90.0,
                    1 DENT(NWEEK+1),DENT(NWEEK+2))
0306                 CALL AXIS (0.0,0.0,'TIME 2WEEKS',-11,4.0,0.0,ARRAYX(NWEEK+1),
                    1 ARRAYX(NWEEK+2))
0307                 CALL LINE (ARRAYX,DENT,NWEEK,1,-1,1)
0308                 CALL PLOT (5.0,0.0,-3)
0309                 CALL SYMBOL (1.0,4.1,0.14,'LOAD=',0.0,5)
0310                 CALL NUMBER (2.2,4.1,0.14,SERHB,0.0,0)
```

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FORTRAN IV G LEVEL  18                  MAIN                DATE = 70336          18/08/07

 0311                 CALL SCALE (DENTT,4.0,NWEEK,+1)
 0312                 CALL SCALE (ARRAYX,4.0,NWEEK,+1)
 0313                 CALL AXIS (0.0,0.0,'CHANGE VENT/2WEEKS',+18,4.0,90.0,
                     1 DENTT(NWEEK+1),DENTT(NWEEK+2))
 0314                 CALL AXIS (0.0,0.0,'TIME 2WEEKS',-11,4.0,0.0,ARRAYX(NWEEK+1),
                     1 ARRAYX(NWEEK+2))
 0315                 CALL LINE (ARRAYX,DENTT,NWEEK,1,-1,1)
 0316                 CALL PLOT (0.0,-5.0,-3)
 0317                 CALL SYMBOL (1.0,4.1,0.14,'LOAD=',0.0,5)
 0318                 CALL NUMBER (2.2,4.1,0.14,SERHC,0.0,0)
 0319                 CALL SCALE (DENTTT,4.0,NWEEK,+1)
 0320                 CALL SCALE (ARRAYX,4.0,NWEEK,+1)
 0321                 CALL AXIS (0.0,0.0,'CHANGE VENT/2WEEKS',+18,4.0,90.0,
                     1 DENTTT(NWEEK+1),DENTTT(NWEEK+2))
 0322                 CALL AXIS (0.0,0.0,'TIME 2WEEKS',-11,4.0,0.0,ARRAYX(NWEEK+1),
                     1 ARRAYX(NWEEK+2))
 0323                 CALL LINE(ARRAYX,DENTTT,NWEEK,1,-1,1)
           C      PLOT  CHANGE IN HR BEATS/MIN/2WEEKS
 0324                 CALL PLOT (5.0,0.0,-3)
 0325                 CALL SYMBOL (1.0,4.1,0.14,'LOAD=',0.0,5)
 0326                 CALL NUMBER (2.2,4.1,0.14,SERHA,0.0,0)
 0327                 CALL SCALE (DRR,4.0,NWEEK,+1)
 0328                 CALL SCALE (ARRAYX,4.0,NWEEK,+1)
 0329                 CALL AXIS (0.0,0.0,'HR/MIN/2WEEKS',+13,4.0,90.0,DRR(NWEEK+1),
                     1 DRR(NWEEK+2))
 0330                 CALL AXIS (0.0,0.0,'TIME 2WEEKS',-11,4.0,0.0,ARRAYX(NWEEK+1),
                     1 ARRAYX(NWEEK+2))
 0331                 CALL LINE (ARRAYX,DRR,NWEEK,1,-1,1)
 0332                 CALL PLOT (0.0,5.0,-3)
 0333                 CALL SYMBOL (1.0,4.1,0.14,'LOAD=',0.0,5)
 0334                 CALL NUMBER (2.2,4.1,0.14,SERHB,0.0,0)
 0335                 CALL SCALE (DRRR,4.0,NWEEK,+1)
 0336                 CALL SCALE (ARRAYX,4.0,NWEEK,+1)
 0337                 CALL AXIS (0.0,0.0,'HR/MIN/1WEEKS',+12,4.0,90.0,DRRR(NWEEK+1),
                     1 DRRR(NWEEK+2))
 0338                 CALL AXIS (0.0,0.0,'TIME 2WEEKS',-11,4.0,0.0,ARRAYX(NWEEK+1),
                     1 ARRAYX(NWEEK+2))
 0339                 CALL LINE(ARRAYX,DRRR,NWEEK,1,-1,1)
 0340                 CALL PLOT (5.0,0.0,-3)
 0341                 CALL SYMBOL (1.0,4.1,0.14,'LOAD=',0.0,5)
 0342                 CALL NUMBER (2.2,4.1,0.14,SERHC,0.0,0)
 0343                 CALL SCALE (DRRRR,4.0,NWEEK,+1)
 0344                 CALL SCALE (ARRAYX,4.0,NWEEK,+1)
 0345                 CALL AXIS (0.0,0.0,'HR/MIN/2WEEKS',+13,4.0,90.0,DRRRR(NWEEK+1),
                     1 DRRRR(NWEEK+2))
 0346                 CALL AXIS (0.0,0.0,'TIME 2WEEKS',-11,4.0,0.0,ARRAYX(NWEEK+1),
                     1 ARRAYX(NWEEK+2))
 0347                 CALL LINE(ARRAYX,DRRRR,NWEEK,1,-1,1)
           C      PLOT  CHANGE IN VO2 L/MIN/2WEEKS
 0348                 CALL PLOT (0.0,-5.0,-3)
 0349                 CALL SYMBOL (1.0,4.1,0.14,'LOAD=',0.0,5)
 0350                 CALL NUMBER (2.2,4.1,0.14,SERHA,0.0,0)
```

```
0351          CALL SCALE (DVO,4.0,NWEEK,+1)
0352          CALL SCALE (ARRAYX,4.0,NWEEK,+1)
0353          CALL AXIS (0.0,0.0,'VO2 L/MIN/2WEEKS',+16,4.0,90.0,DVO(NWEEK+1),
             1 DVO(NWEEK+2))
0354          CALL AXIS (0.0,0.0,'TIME 2WEEKS',-11,4.0,0.0,ARRAYX(NWEEK+1),
             1 ARRAYX(NWEEK+2))
0355          CALL LINE (ARRAYX,DVO, NWEEK,1,-1,1)
0356          CALL PLOT (5.0,0.0,-3)
0357          CALL SYMBOL (1.0,4.1,0.14,'LOAD=',0.0,5)
0358          CALL NUMBER (2.2,4.1,0.14,SERHB,0.0,0)
0359          CALL SCALE (DVVO,4.0,NWEEK,+1)
0360          CALL SCALE (ARRAYX,4.0,NWEEK,+1)
0361          CALL AXIS (0.0,0.0,'VO2 L/MIN/2WEEKS',+16,4.0,90.0,DVVO(NWEEK+1),
             1 DVVO (NWEEK+2))
0362          CALL AXIS (0.0,0.0,'TIME 2WEEKS',-11,4.0,0.0,ARRAYX(NWEEK+1),
             1 ARRAYX(NWEEK+2))
0363          CALL LINE (ARRAYX,DVVO,NWEEK,1,-1,1)
0364          CALL PLOT (0.0,5.0,-3)
0365          CALL SYMBOL (1.0,4.1,0.14,'LOAD=',0.0,5)
0366          CALL NUMBER (2.2,4.1,0.14,SERHC,0.0,0)
0367          CALL SCALE (DVVVO,4.0,NWEEK,+1)
0368          CALL SCALE(ARRAYX,4.0,NWEEK,+1)
0369          CALL AXIS (0.0,0.0,'VO2 L/MIN/2WEEKS',+16,4.0,90.0,DVVVO(NWEEK+1),
             1 DVVVO(NWEEK+2))
0370          CALL AXIS (0.0,0.0,'TIME 2WEEKS',-11,4.0,0.0,ARRAYX(NWEEK+1),
             1 ARRAYX(NWEEK+2))
0371          CALL LINE(ARRAYX,DVVVO,NWEEK,1,-1,1)
0372          END
```

APPENDIX 3

Computer program for split-plot two-factor design analysis of variance.

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FORTRAN IV G LEVEL  18                  MAIN                    DATE = 71036          15/15/34

C**   SPLIT-PLOT TWO-FACTOR DESIGN ANALYSIS OF VARIANCE
C**   WITH UNWEIGHTED-MEANS SOLUTION FOR UNEQUAL SIZE GROUPS
C**   LEAD CARD HAS NO. OF ANALYSES IN I4 FORMAT.
C**   FIRST HEADER CARD FOR EACH SET OF DATA HAS NO. OF LEVELS
C**   OF FACTOR A, FACTOR B AND TOTAL NO. OF SUBJECTS IN
C**   I4 FORMAT AND TITLE IN 16A4 FORMAT.
C**   SECOND HEADER CARD HAS NAMES OF FACTORS A AND B EACH
C**   IN 10A4 FORMAT.
C**   DATA FOLLOWS WITH SCORES FOR FIRST SUBJ ON ALL
C**   LEVELS OF B ON SAME CARD(S)IN FIELDS OF 10
C**   WITH F10.4 FORMAT.
C**   DATA IS ORGANIZED AS FOLLOWS  A(1)S(1)B(1)..A(1)S(1)B(J)
C**   .. A(1)S(K)B(J) ... A(I)S(K)B(J).
C**   PROGRAM WRITTEN BY J.MONTGOMERY
C**   REFERENCE-KIRK-EXP.DESIGN-PP245-283
0001        DIMENSION TITLE(16),FACTA(10),FACTB(10),Y(10,10,20),
           1SUMS(10,20),SUMSSQ(10,20),AB(10,10),ABSQ(10,10),ABM(10,10),
           2ABSD(10,10),A(10),B(10),AM(10),BM(10),NS(10),HN(10)
0002        NDEK=0
0003        READ(5,1)NANAL
0004   1000 NDEK=NDEK+1
0005        READ(5,2)NA,NB,NTOT,(TITLE(I),I=1,16)
0006        READ(5,3)(FACTA(I),I=1,10),(FACTB(I),I=1,10)
0007        READ(5,4)(NS(I),I=1,NA)
0008        DO 1010 I=1,NA
0009        NTEMP=NS(I)
0010        DO 1010 K=1,NTEMP
0011   1010 READ(5,5)(Y(I,J,K),J=1,NB)
0012        SUM=0.0
0013        SUMSQ=0.0
0014        DO 1030 I=1,NA
0015        NTEMP=NS(I)
0016        DO 1030 K=1,NTEMP
0017        SUMS(I,K)=0.0
0018        SUMSSQ(I,K)=0.0
0019        DO 1020 J=1,NB
0020        SUMS(I,K)=SUMS(I,K)+Y(I,J,K)
0021        SUMSSQ(I,K)=SUMSSQ(I,K)+Y(I,J,K)**2
0022   1020 CONTINUE
0023        SUM=SUM+SUMS(I,K)
0024        SUMSQ=SUMSQ+SUMSSQ(I,K)
0025   1030 CONTINUE
0026        DO 1050 I=1,NA
0027        NTEMP=NS(I)
0028        DO 1050 J=1,NB
0029        AB(I,J)=0.0
0030        ABSQ(I,J)=0.0
0031        DO 1040 K=1,NTEMP
0032        AB(I,J)=AB(I,J)+Y(I,J,K)
0033        ABSQ(I,J)=ABSQ(I,J)+Y(I,J,K)**2
0034   1040 CONTINUE
0035        ABM(I,J)=AB(I,J)/NS(I)
```

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FORTRAN IV G LEVEL  18                  MAIN                    DATE = 71036          15/15/34

0036                  ABSD(I,J)=SQRT((ABSQ(I,J)*NS(I)-AB(I,J)**2)/(NS(I)*(NS(I)-1)))
0037            1050  CONTINUE
0038                  SSS=0.0
0039                  DO 1060 I=1,NA
0040                  NTEMP=NS(I)
0041                  DO 1060 K=1,NTEMP
0042                  SSS=SSS+SUMS(I,K)**2/NB
0043            1060  CONTINUE
0044                  SSAA=0.0
0045                  DO 1080 I=1,NA
0046                  A(I)=0.0
0047                  DO 1070 J=1,NB
0048                  A(I)=A(I)+AB(I,J)
0049            1070  CONTINUE
0050                  SSAA=SSAA+A(I)**2/(NS(I)*NB)
0051            1080  CONTINUE
0052                  SSB=0.0
0053                  DO 1100 J=1,NB
0054                  B(J)=0.0
0055                  DO 1090 I=1,NA
0056                  B(J)=B(J)+AB(I,J)
0057            1090  CONTINUE
0058                  SSB=SSB+B(J)**2/NTOT
0059            1100  CONTINUE
0060                  SSABB=0.0
0061                  DO 1110 I=1,NA
0062                  DO 1110 J=1,NB
0063                  SSABB=SSABB+AB(I,J)**2/NS(I)
0064            1110  CONTINUE
0065                  HARN=0.0
0066                  DO 1120 I=1,NA
0067                  HARN=HARN+1.0/NS(I)
0068            1120  CONTINUE
0069                  HARN=NA/HARN
0070                  SSABM=0.0
0071                  DO 1140 I=1,NA
0072                  AM(I)=0.0
0073                  DO 1130 J=1,NB
0074                  SSABM=SSABM+ABM(I,J)**2
0075                  AM(I)=AM(I)+ABM(I,J)
0076            1130  CONTINUE
0077            1140  CONTINUE
0078                  SUMM=0.0
0079                  DO 1150 I=1,NA
0080                  SUMM=SUMM+AM(I)
0081            1150  CONTINUE
0082                  XM=SUMM**2/(NA*NB)
0083                  X=SUM**2/(NB*NTOT)
0084                  SSAM=0.0
0085                  DO 1160 I=1,NA
0086                  SSAM=SSAM+AM(I)**2/NB
0087            1160  CONTINUE
```

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FORTRAN IV G LEVEL  18                  MAIN                    DATE = 71036          15/15/34

0088                  SSBM=0.0
0089                  DO 1180 J=1,NB
0090                  BM(J)=0.0
0091                  DO 1170 I=1,NA
0092                  BM(J)=BM(J)+ABM(I,J)
0093            1170  CONTINUE
0094                  SSBM=SSBM+BM(J)**2/NA
0095            1180  CONTINUE
0096                  SSA=HARN*(SSAM-XM)
0097                  SSSA=SSS-SSAA
0098                  SSB=HARN*(SSBM-XM)
0099                  SSAB=HARN*(SSABM-SSAM-SSBM+XM)
0100                  SSBSA=SUMSQ-SSABB-SSS+SSAA
0101                  NDFA=NA-1
0102                  NDFB=NB-1
0103                  NDFAB=NDFA*NDFB
0104                  NDFSA=NTOT-NA
0105                  NDFBSA=NDFSA*NDFB
0106                  AMS=SSA/NDFA
0107                  SAMS=SSSA/NDFSA
0108                  BMS=SSB/NDFB
0109                  ABMS=SSAB/NDFAB
0110                  BSAMS=SSBSA/NDFBSA
0111                  FA=AMS/SAMS
0112                  FB=BMS/BSAMS
0113                  FAB=ABMS/BSAMS
0114                  WRITE(6,6)(TITLE(I),I=1,16)
0115                  WRITE(6,7)(FACTA(I),I=1,10)
0116                  WRITE(6,8)(FACTB(I),I=1,10)
0117                  WRITE(6,9)
0118                  DO 1190 I=1,NA
0119            1190  WRITE(6,10)(ABM(I,J),J=1,NB)
0120                  WRITE(6,11)
0121                  DO 1200 I=1,NA
0122            1200  WRITE(6,10)(ABSD(I,J),J=1,NB)
0123                  WRITE(6,12)
0124                  WRITE(6,13)SSA,NDFA,AMS,FA,SSSA,NDFSA,SAMS,
                     1SSB,NDFB,BMS,FB,SSAB,NDFAB,ABMS,FAB,SSBSA,
                     2NDFBSA,BSAMS
0125                   IF(NDEK.LT.NANAL)GO TO 1000
0126                1 FORMAT(I4)
0127                2 FORMAT(3I4,16A4)
0128                3 FORMAT(2(10A4))
0129                4 FORMAT(10I4)
0130                5 FORMAT(10F15.9)
0131                6 FORMAT('1',T10,16A4)
0132                7 FORMAT('0',T10,'FACTOR A IS',10A4)
0133                8 FORMAT('0',T10,'FACTOR B IS',10A4)
0134                9 FORMAT('1',T10,'CELL MEANS')
0135               10 FORMAT('0',T10,10(F10.4,5X))
0136               11 FORMAT('1',T10,'CELL STANDARD DEVIATIONS')
0137               12 FORMAT('1',T10,'SUMMARY OF SPLIT-PLOT,UNWEIGHTED-
```

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FORTRAN IV G LEVEL  18                  MAIN                    DATE = 71036          15/15/34

                1MEANS ANALYSIS',///,T10,'SOURCE',T40,'SUM SQUARES',
                2T60,'DEGREES FREEDOM',T80,'MEAN SQUARES',T100,'F-RATIOS')
0138          13 FORMAT('0',T10,'A',T40,F12.2,T60,I4,T80,F12.2,T100,
                1F10.4,//,T10,'SUBJ.W.A',T40,F12.2,T60,I4,T80,F12.2,
                2//,T10,'B',T40,F12.2,T60,I4,T80,F12.2,T100,F10.4,
                3//,T10,'AB',T40,F12.2,T60,I4,T80,F12.2,T100,F10.4,
                4//,T10,'B X SUBJ W.A',T40,F12.2,T60,I6,T80,F12.2)
0139             STOP
0140             END
```