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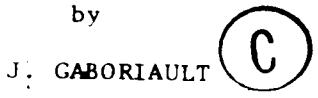
THE UNIVERSITY OF ALBERTA

HEMODYNAMIC EFFECTS OF EXERCISE TRAINING IN PATIENTS WITH ISCHEMIC HEART DISEASE.

by

REAL J. GABORIAULT 

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES

IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE DEGREE

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research, for acceptance, a thesis entitled: " Hemodynamic Effects of Exercise Training in Patients with Ischemic Heart Disease ", submitted by Réal J. Gabori'ault in partial fulfilment for the requirements for the degree of Master of Science (Physical Education).

Dr. B. J. Macnab
Supervisor

Howard P. Unger
Professor

Simon Lee
Professor

Date March 25

DEDICATION

A mes parents et beaux-parents,
qui malgré leur éloignement,
ont favorisé et facilité
mes années d'études.

ABSTRACT

The purpose of this non-invasive study was to compare the hemodynamic changes evoked by exercise training with that in a reference group. Six healed myocardial infarct patients (mean age, 54 years) were tested on an electrically braked bicycle ergometer prior to a three month training program and monthly thereafter.

Following a preliminary test consisting of a treadmill test at maximal work capacity, the subjects were randomly assigned to either group. The training was conducted four times a week, one hour per session. The subjects were monitored all the time and alternatively trained on a treadmill and on a bicycle ergometer. The 70% and 90% of the predetermined maximal heart rate served as testing intensities. The subjects were invited to participate in the experiment, three months after their myocardial infarction.

The main statistical analyses were done by a four way analysis of variance with repeated measures on three factors. Following training, there was no significant ($P < .05$) difference between the groups at submaximal work capacity. An unchanged cardiac output concomitant with a significant ($P < .05$) decrease in heart rate resulted in an increased stroke volume. After training, the maximal oxygen consumption was higher in the experimental group.

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STATEMENT OF THE PROBLEM

INTRODUCTION

Post coronary exercise programs have attracted a great deal of attention in recent years. It is generally agreed that exercise training has beneficial effects on myocardial infarct survivors. A number of clinical studies (1-13) have emphasized the benefit of physical exertion as a form of therapy after a recent myocardial infarction. Although the sample size has tended to be small in such programs, physicians are aware of the feasibility and safety demonstrated by different rehabilitation programs (14-20).

The valuable aspect of physical exertion following a myocardial infarction lies in its relation both to cardiac work and to social functions. By preventing the deterioration of physical condition during the convalescent period, the exercise training enhances the possibilities of an early return to work and of a better enjoyment of the patient's leisure activities (21). The physical demands are often minimized and the patient regains more tolerance toward the total amount of work output during his daily routine and above all, his self-confidence is often restored after he has gone through a disease with such a high mortality rate (12). During the early phase after an acute myocardial infarction, there are good grounds for enforced rest. The duration of the immobilization of the patient used to be several weeks (22), but since the re-

commendation of Levine (23), there is a tendency for an early prescription of exercise treatment after a myocardial infarction in order to improve the patients' conditions (24-28).

THE PROBLEM

The problem in this non-invasive study was to investigate the effect of short-term continuous training on the cardiorespiratory functions of cardiac patients. The specific parameters measured were:

1- Maximal oxygen consumption

2- Maximal heart rate

3- Oxygen consumption relative to body weight, cardiac output, heart rate and stroke volume at submaximal work loads corresponding to a warm-up level (200 Kpm), 70% of maximal heart rate and 90% of maximal heart rate.

RATIONALE FOR THE STUDY

Coronary heart disease patients on programs of exercise have been reported to have an increased physical work capacity, an increased feeling of general well-being, a reduced incidence of recurrent myocardial infarction, angina pectoris and ischemic electrocardiographic changes as well as a reduced fatality rate (29-37). In these studies there is no clear evidence that properly selected patients are endangered by exercise programs; in fact, they appear to derive both symptomatic and psychological benefits. The fact that they are under the supervision of trained medical personnel helps to reduce their anxiety and apprehension

associated with physical effort.

The use of functional responses to maximal exercise provides a more accurate prediction of future cardiac events in a specific person than the assessment of risk factors identified by examining this person at rest (38-40). Identification of risk factors such as hypertension, hyperlipemia and cigarette smoking may be helpful (41) but are often absent in patients who experience myocardial infarction or sudden death (42).

In the evaluation of the functional impairment of the cardiac patient by physical exertion, we may find a more valuable form of treatmeant that will enhance a better recovery and possibly prevent or minimize other heart attacks.

AIM OF THE STUDY

Previous studies (1, 7, 12, 31, 33-35, 43) have reported that physical working capacity can be increased by exercise training in patients limited by coronary insufficiency. In these studies, the patients have been selected according to various characteristics such as angina pectoris, medications used, hypertension or non-recent myocardial infarction. In the present experiment an attempt was made to evaluate the circulatory parameters for several months after the start of the training, in patients who had recently experienced a myocardial infarction and who did not develop angina pectoris during effort.

The general aim of this non-invasive experiment was to compare

the circulatory changes evoked by short-term physical reconditioning at submaximal work levels, with that in a reference group from the same series, with special emphasis on left ventricular function namely cardiac output (using carbon dioxide rebreathing method), heart rate and stroke volume. These patients have usually an impaired heart condition which minimize their tolerance for maximal effort. Nevertheless, their oxygen consumption was measured both at submaximal and maximal work level.

DELIMITATIONS OF THE STUDY

Seven (7) male cardiac patients under the age of 65, participated voluntarily in this non-invasive study. They were randomly assigned to either the experimental (n=4) or the reference (n=3) group.

LIMITATIONS OF THE STUDY

1. One drop-out from the experimental group.
2. Small groups (n=3) mainly due to the rejection of candidates who developed angina pectoris during physical effort.
3. It was impossible to control the patients' activities outside the laboratory hours and the absences of the training sessions due to temporary illness, holiday or work out of town.
4. The seasonal effect on the parameters assessed.
5. The anxiety and the physical state of the patients during the testing sessions.

DEFINITION OF TERMS

Bruce treadmill test: is the test used to assess the patient's physical condition. The speed and slope elevation of the treadmill determine different stages (see protocol in Appendix 2).

Carbon dioxide rebreathing method: is based on the Fick principle. Knowledge of the amount of carbon dioxide in a unit of arterial and venous blood and its rate of removal, allows a calculation of the blood flow passing through an organ.

Cardiac output: is the amount of blood ejected by the heart in the circulatory system usually expressed as liters per minute.

Cardiac patient: is a person having a documented myocardial infarction diagnosed by: ECG changes with appearance of a pathological Q wave, arrythmias, ST segment changes or serum enzyme alterations (SGOT, LDH, CPK).

Maximal heart rate: is the highest heart rate obtained during the Bruce treadmill test and measured electronically by means of electrodes registering the number of heart depolarisations (QRS complex) per minute.

Maximal oxygen consumption: is the volume of oxygen utilized, just before exhaustion or symptoms, from the inspired air when the cardiorespiratory functions cannot make any further adjustments to increase the work loads.

Oxygen consumption: is the volume of oxygen utilized from the inspired air usually expressed as liters per minute or as a function of body

weight in milliliters per kilogram per minute at STPD i.e. standard temperature and pressure, dry ($0^{o}C$ and 760 mmHg). Oxygen uptake or oxygen intake have the same meaning.

Percentage of maximal heart rate: is the percent of the maximal heart pulse obtained during the Bruce treadmill test.

Stroke volume: is the volume of blood ejected per beat after a heart contraction usually expressed as milliliters per beat.

CHAPTER II

REVIEW OF THE LITERATURE

PHYSICAL EXERTION IN CORONARY PATIENTS

The effects of physical exertion after myocardial infarction have been well documented (1, 2, 4-6, 8, 9, 18-21, 32, 38, 44-46). The main goal of such exercise programs has been to improve the functional capacity of an impaired heart, not necessarily to the extent of the marathon runners (3), but enough to result in fewer and less severe reinfarctions (10).

In addition to the favourable influence upon man's well-being, physical training seems to act as a preventive measure against coronary heart disease (47). A slight decrease of the number of abnormal ECG findings at rest and at work (premature beats and ST-changes) was also found after training (48).

CARDIORESPIRATORY ALTERATIONS

Although the respiratory functions are age dependent, there is a tendency for an improvement after training leading to an increased maximum oxygen uptake and hence physical working capacity. The ability to increase oxygen uptake with increasing work is a function of the many variables which comprise the oxygen transport system mainly cardiac output and arteriovenous oxygen difference. Since the latter is not markedly restricted by heart disease, the relatively low values of maximal

oxygen intake reveals the functional severity of disease in cardiac patients (44).

The maximum oxygen uptake has been widely accepted to represent the functional capacity of the circulatory system and the aerobic physical work capacity. It reflects both the performance of the heart as a pump and the efficiency of distribution of blood flow (44).

Backer et al. (1) observed the subjective maximal physical working capacity 6-8 weeks and 12-14 weeks after myocardial infarction. Sixty-nine patients (mean age, 54.0 years) were randomly ascribed to a control group (33 subjects) or to a rehabilitated group (36 subjects) which performed interval training three times a week, at a target heart rate corresponding to 70% of the maximum heart rate previously determined at the first exercise test. The tests were performed on an electrically braked bicycle ergometer. Physical performance increased favorably in the control group by 19.5% (from 513 to 613 Kpm/min), however, the improvement in the rehabilitated group was much more important and statistically significant: 31.8% (from 566 to 746 Kpm/min). Physical working capacity at a pulse rate of 120 beats per minute (PWC 120) showed an increase of 26.4% (from 375 to 474 Kpm/min) in the rehabilitated group after 12-14 weeks of training while the control group demonstrated an increase of 12.1% (from 388 to 435 Kpm/min). No values were reported for oxygen consumption and heart rate at different work levels.

In an extensive study of two years, Kavanagh et al. (49) trained a group of thirty-one post coronary patients divided in a high intensity

(more than 30 points/week according to Cooper's scheme) and a low intensity group (less than 30 points/week). The training protocol consisted of supervised (1½ hour once a week) and non-supervised (4 times per week) activities such as calisthenic, walking, jogging and interval training. Patients were exercised on a bicycle ergometer during testing sessions. Inevitably, many of the low intensity group (8/14) were patients over the age of 50 while many of the high intensity group (10/13) were under the age of 50. Nevertheless, the aerobic power predicted from the oxygen scale of the Astrand nomogram increased 14.8% for the high intensity group (from 27.8 to 31.9 ml/kg/min) after the first year and 49.2% (from 27.8 to 41.5 ml/kg/min) after the second year of training, the low intensity group increased 19.7% (from 25.8 to 30.9 ml/kg/min) after one year and 12.8% (from 25.8 to 29.1 ml/kg/min) after two years of training. The large improvement in aerobic power for the high intensity group could be explained by a relatively low initial work capacity level, the distance (20 miles/week) and the intensity (2 miles in 16 minutes or less) ran during training while the decrease in aerobic power between the first and the second year for the low intensity group, despite the fact that they lost 3.8 kg., could be explained by their "physiological older" condition; many were older than the other group and had complications such as angina and diastolic hypertension.

Kavanagh and Shephard (4) compared the continuous and interval training in 41 sedentary patients (mean age, 47 years) recovering from proven myocardial infarction over a period of one year. The predicted aerobic power based on the work and oxygen scales of the Astrand nomo-

gram showed a substantial increase for all patients of 30.8% (from 26.9 to 35.2 ml/kg/min) when considering the work scale and an increase of 31.2% after one year of training (from 23.7 to 32.1 ml/kg/min) when considering the oxygen scale. The gains were somewhat larger for the continuous exercise regimen. Although the subjects had followed a preliminary walking and jogging program for up to one year, the percent increment of aerobic power seems quite large. Weight loss occurred over the program which would have accounted for a portion of these large increases, but exact figures were not given so the precise contribution of the weight loss to improvement of aerobic power cannot be determined. Hemodynamic values were reported in the study.

Kasch and Boyer (50) evaluated the changes in maximal work capacity resulting from six month training in patients with ischemic heart disease. Eleven patients (mean age, 50 years) participated in the study. The training consisted of interval training one hour per day, four days per week. The working capacity, determined on a bicycle ergometer in a multi-stage test procedure, increased by 38.2% (from 19.9 to 27.5 ml/kg/min) at three months and 53.8% (from 19.9 to 30.6 ml/kg/min) at six months even though the mean drop of body weight was 2.6 pounds. The mean resting heart rates were reduced by 11.7 beats/min., the maximal heart rate increased by 17 beats/min. and the mean resting blood pressures were reduced by 9 mmHg systolic and 6 mmHg diastolic.

A six week program of intensive training in seven patients with angina pectoris, aged 39 to 60 years (average, 48) was carried out by

Redwood et al. (51). The patients exercised twice a day, five days a week. The training consisted of an uninterrupted bicycle ergometer exercise with increment of 20 watts every three minutes until angina occurred. After a 15-minute rest period, exercise was repeated. The patients were measured at the beginning, after three and six weeks of training. Exercise capacity showed a progressive and marked improvement of 6.8 minutes represented by an average increase of 40 watts throughout the training period. Maximal oxygen consumption was measured during peak subanginal exercise bouts and showed an improvement of 56% after six weeks of training (from 9.6 to 15.0 ml/kg/min). The great percent improvement can be explained by the use of nitroglycerine during the testing periods and the low values prior the commencement of training. The response of the product of arterial-blood pressure and heart rate showed a marked diminution of 18.1% (from 4300 to 3521) to the same external work load. The heart rate showed a decrease of 19.3% (from 119 to 96 beats/min). The improvement of the circulatory response to exercise might have been confounded by the use of nitroglycerin during the "maximal" effort.

In a study at maximal exercise with fifteen men ans one women (mean age, 55 years), McDonough et al. (52) investigated the fundamental indicator of the degree of impairment resulting from coronary disease. The results demonstrated that the maximal cardiac output (using the direct Fick method) paralleled the maximal oxygen intake and was reflected by a plateau during the final minutes of exercise. Significant decreases in stroke volume (from 96 to 89 ml/beat) and increases in mean pulmonary arterial pressure (from 22 to 25 mmHg) suggest that acute left ventricular

dysfunction is the mechanism limiting the maximal cardiac output.

In Bergstrom et al. (32) study, a serie of 63 consecutive male patients (mean age, 54.6 years) with myocardial infarction were divided in two groups and each group in two subgroups corresponding to an age cutting point of 55 years. The physical training was carried out during a three-month period starting three months after the onset of the infarction, and comprised three 1-hour training sessions a week. Following the training period, there was an increase in maximal work performed of 24.1% (from 625 to776 Kpm/min) and in W130 of 23.9% (from 593 to 735 Kpm/min) in the training group; the greatest increase occurring in the younger age group. In the reference group, there was a significant increase of 7.2% (from 566 to 607 Kpm/min) for W130. The reference patients exhibited no appreciable change in heart rate (from 132 to 128 beats/min) at the same submaximal work load while the training patients showed a 11% decrease (from 136 to 121 beats/min). The stroke volume during the same submaximal exercise, measured at the right heart catheterization, increased 6.8% (from 88 to 94 ml/beat) in the training group; the largest increase was noted in the younger (under 55 years) patients without angina pectoris and no change occurred in the reference group.

Rousseau et al. (11) have been interested in the hemodynamic determinants of maximal oxygen intake and mainly in the factors limiting the maximal oxygen intake. They compared seven patients (mean age, 47.9 years) as the control group with seven patients (mean age, 48.7 years) who followed a physical training program which involved individually graded exercises for 45 minutes, 3 times a week for a period of 13.5 months. They

were evaluated on an uninterrupted multistage treadmill test before and after the training period. The control group was considered to have a lower maximum oxygen uptake than the trained group at the pre-test but their age and maximal heart rate did not differ significantly. Following the training period, the maximal oxygen uptake increased 30.2% (from 25.5 to 33.4 ml/kg/min) with an increase of 4.3% (from 168.5 to 175.5 beats/min) in maximal heart rate. The major difference between the two groups was the significantly higher maximal arteriovenous oxygen difference of the trained patients (16.1 vs 14.4 ml/100ml). The values in stroke volume was always lower at maximal exercise level than the submaximal one. The fall in stroke volume at maximal oxygen uptake level was of the same magnitude in untrained (15%; from 100 to 85 ml/beat) and in trained subjects (18%; from 106 to 87 ml/beat). Their findings suggest that maximal oxygen uptake is limited by the decrease in stroke volume at maximal exercise level. In trained patients the maximal arteriovenous oxygen difference is greater and results in maximal oxygen uptake that is higher than in untrained patients.

Frick and Katila (36) studied the hemodynamic consequences of physical training after myocardial infarction. Seven patients participated in the training program consisting of bicycle ergometer work, three times a week, for one to two months. The training was followed by reduction in exercise heart rate of 8.3% (from 133 to 122 beats/min) at the higher exercise work load. Stroke volume was increased by 14.9% (from 109.8 to 126.2 ml/beat) at the same work load while cardiac output showed no change.

Clausen et al. (13) studied the effects of 4 to 6 weeks training program in nine patients (mean age, 52 years) with coronary heart disease. The training program consisted of intermittent work (effective work time 30 min.), five days a week and performed on a mechanically braked bicycle ergometer. Their working capacity was measured before and after the training period; the mean increase was 174 Kpm/min or 33.3% (from 522 to 696 Kpm/min). The maximal oxygen consumption assessed just below the threshold for angina pectoris increased by 32.5% (from 16.0 to 21.2 ml/kg/min). The mean cardiac output was unchanged for the same oxygen uptake. The stroke volume, calculated as the ratio of cardiac output and heart rate simultaneously recorded, was greater after training. This is mainly due to a 9.3% decrease (from 117 to 107 beats/min) in heart rate; the changes in heart rate during the exercise period suggest, however, that in some cases the greater stroke volume could be preserved only for short periods. The increased stroke volume does not inevitably involve an enhanced myocardial contractility but might be secondary to a decreased total peripheral resistance.

Detry et al. (33) evaluated the physical capacity of 12 male patients (mean age, 47.8 years) with coronary heart disease after a three month training program. Six of the patients had angina pectoris during effort and six with prior myocardial infarction but without angina. The training program involved three sessions of 45 minutes per week and was conducted at two different places with two different test procedures (one used a multistage treadmill test while the other a multistage bicycle exercise test). The "maximal" oxygen uptake increased 22.5% (from

23.0 to 28.2 ml/kg/min) after physical training. This increase was greater in patients with angina pectoris (30.7%; from 18.6 to 24.3 ml/kg/min) who had lower pre-training values than in patients with healed myocardial infarction but no angina (17.7%; from 27.3 to 32.2 ml/kg/min); similarly, maximal heart rate increased by 8% in angina patients, while the change was only 0.8% in patients without angina. At rest and at two submaximal exercise levels, corresponding to 45% and 75% of the pre-training "maximal" oxygen uptake, heart rate and cardiac output decreased by 13.1% (from 107 to 93 beats/min) and 11% respectively (from 9.1 to 8.1 l/min). The arteriovenous oxygen difference increased by 9.4% (from 8.5 to 9.3 ml/100 ml), while the stroke volume remained unchanged. The lower cardiac output was attended by an increased arteriovenous oxygen difference, an unchanged stroke volume, and lower heart rate.

Kirchheiner et al. (7) compared two groups of heart patients (mean age, 57.3 years), one of whom went through a physical training program which comprised cycling, walking, running, skipping and ball games. The training took place 3 times a week, for a period of six months. The working tests were carried out on an electrically braked bicycle before and after the training period. In both tests the measurements were carried out on the same submaximal work load, and the mean values were calculated. The highest obtainable steady state conditions during physical working capacity were obtained until the subjects developed angina, severe dyspnoea or exhaustion. As the result of training, the physical work capacity increased, the mean stroke volume increase 16% (from 81 to 94 ml/beat), the mean heart rate decreased 13% (from

119 to 104 beats/min) while the cardiac output was unchanged. The cardiac output determinations were made according to the Fick principle in carbon dioxide measurements.

Bjernulf (30) compared a trained group (mean age, 55.5 years) with a reference group (mean age, 52.5 years). After exclusions, both groups comprised 21 patients. The training program consisted of interval training on bicycle, three times per week for three months. For the same work load, there was no significant change in oxygen consumption after the training period (0.9 liters/min). A significant reduction in heart rate (from 107 to 100 beats/min) was reported for the trained group as well as for the reference group (from 107 to 103 beats/min) after training. The low heart rate values seem to reflect a low work test level. No values were reported for the intensity of the work test (it was referred to a publication in german). An increase in stroke volume of 6.6% (from 88 to 94 ml/beat) was reported after the training period, while cardiac output remained the same. The low values and the non-significant difference in oxygen uptake after training could be explained by the low work test level as judged by the low heart rate values.

Clausen and Trap-Jensen (53) evaluated the distribution of cardiac output in seven patients (mean age, 53 years) with coronary heart disease who participated in an intermittent training program on bicycle ergometer, five days a week for 4 to 10 weeks. As the result of training, the physical work capacity increased 32% (from 633 to 830 Kpm/min) corresponding to approximately 20 to 26 ml/kg/min. Heart rate decreased at rest,

at submaximal load and heavy load by 7.5% (from 71 to 66 beats/min), 15.5% (from 119 to 103 beats/min) and 9.8% (from 145 to 132 beats/min) respectively. During heavy exercise, cardiac output was increased 5.5% (from 12.99 to 13.71 liters/min) while stroke volume was increased 14.1% (from 91.8 to 104.7 ml/beat). These effects of training could be explained as peripheral regulatory alterations without implying primary improvement in myocardial performance.

Bergman and Varnauskas (59) studied the hemodynamic effects of physical training in five males, aged 44-55 years. The training period lasted 30 minutes on the bicycle ergometer, three times a week, for six months. There was tendency for a decrease in oxygen consumption after six months of training, both at rest and during exercise at the same submaximal work load. The mean decrease in heart rate after six months of training was 15% during early exercise and 12% during late exercise. The cardiac output was lowered after training; the mean decrease was 6% at rest and about 13% in both of the exercise measurements. The stroke volume was unchanged but the mean arteriovenous oxygen difference increased 9% during early exercise and 11% during late exercise. The lowering of cardiac output with the increase in arteriovenous oxygen difference after training, is explained by a redistribution of blood flow to the exercising muscles.

Haggendal et al. (54) explained the mechanism involved in the lowering of heart rate by a lower sympathetic drive subsequent to a lower noradrenaline level in arterial blood. Furthermore, Clausen et al. (55)

suggested that the reduction in heart rate may be influenced by extracardiac factors, probably in the trained muscles themselves, modifying the sympathetic stimulation of the sino-atrial node.

This lower heart rate at submaximal exercise has to be compensated by either a greater stroke volume or a wider arteriovenous oxygen difference (33). Stroke volume is not increased to the same extent in cardiac patients (11, 30, 33) as occurs in healthy subjects (56). This difference may reflect the extent of the disease involvement. It has been found (11, 33), however, that there is a greater arteriovenous oxygen difference after a training period. This improvement generates a more complete extraction of oxygen from the blood at the muscular level enabling a more effective regulation of the blood flow (57). Depending upon the initial fitness level, the contribution of both the stroke volume and the arteriovenous oxygen difference is equally important; the percentage increments of the latter being greater in the least fit (58).

Conflicting opinions have been expressed concerning the possible effect of physical conditioning on cardiac output at submaximal work loads. Some investigations (7, 13, 30, 36) reported that the increase in stroke volume concomitant with the decrease in heart rate, after a period of training, result in an unchanged cardiac output (Table I). While others (33, 53, 59) demonstrated a reduced cardiac output at a given submaximal exercise level resulting in a diminished muscle perfusion paralleled by a more effective vasoconstriction in "non-working" muscles or tissues and an increase arteriovenous oxygen difference. This suggests not necessarily an improvement in cardiac performance but an alteration

TABLE I Summary table for the review of the literature

Authors		N	A	C	MA	TP	E	F	WC	VO2	CO	HR	SV
Backer et al.	(1)	69		o	54.0	4	B	3	+				
Kavanagh et al.	(49)	31	o			24	B	1		+			
Kavanagh et al.	(4)	41	o		47.0	12	B	2	+	+			
Kasch et al.	(50)	11	o		50.0	6	B	4		+*		+*	
Redwood et al.	(51)	7	o		48.0	1.5	B	5	+*	+*		-	
McDonough et al.	(52)	16			55.0		T						-*
Bergstrom et al.	(32)	63	o	o	54.6	3	B	3	+*			-	+
Rousseau et al.	(11)	14		o	48.3	13.5	T	3		+*		+*	-*
Frick et al.	(36)	7	o		47.3	2	B	3			=	-	+
Clausen et al.	(13)	9	o		52.0	1.5	B	5	+*	+*	=	-	+
Detry et al.	(33)	12	o		47.8	3	BT	3	*	+*	-	-	=
Kirchheiner et al.	(7)	27	o	o	57.3	6	B	3	+		=	-	+
Bjernulf et al.	(30)	41	o	o	54.0	3	B	3		=	=	-	+
Clausen et al.	(53)	7	o		53.0	2	B	5	+*	+	-	-	+
Bergman et al.	(59)	5	o		50.0	6	B	3		=	-	-	=
Present study		6		o	54.0	3	BT	4	+*	+*	=	-	+

o included in N
- decrease
+ increase
= unchanged
* maximal

N: Number of subjects; A: Angina; C: Control; MA: Mean age in years; TP: Training period in months; E: Ergometry; B:Bicycle; T: Treadmill; F: Frequency per week; WC: Work capacity; VO2: Oxygen consumption; CO: Cardiac output; HR: Heart rate; SV: Stroke volume.

of the peripheral circulatory regulation and possibly enzymatic adaptation (62-64) leading to a more economical heart work.

Several studies (13, 30, 33, 36) reported a significant reduction in arterial blood pressure after a period of physical training. Both the lower heart rate and the lower blood pressure reduce the oxygen consumption of the myocardium (63, 64). This is of great importance for heart patients considering the imbalance between the oxygen demands of the myocardium and its supply.

The coronary artery collateralization observed in animal studies (65-67) after a physical training period does not seem to follow the same pattern in men (68). This may be explained by the species difference (69).

PSYCHOLOGICAL ADAPTATION

The critical combination of emotions and coronary disease is difficult to evaluate but when emotions become equated to stress it may be detrimental. The physician must be aware of the importance of a psychological reorientation as well as a physical one. The hypnotherapy may be the treatment of choice in extreme situations (70). With the loss of illusion that life is of infinite duration, the patients who had recently recovered from a myocardial infarction need to be encouraged. Regular exercise is a mean to re-establish a feeling of security in a growing society where many cardiac patients but still capable people are excluded from economic and social life. To some patients, physical exertion provides a profound emotional satisfaction which leads to the acceptation of

their hidden impaired condition; while to others, it may be the feeling of security necessary to adjust themselves to the demands of daily living

CARBON DIOXIDE REBREATHING METHOD

The carbon dioxide rebreathing method is based on the Fick principle. Knowledge of the amount of carbon dioxide in a unit of arterial and venous blood and its rate of removal, allows a calculation of the blood flow passing through an organ. The evidence for the validity of the measurement of cardiac output using soluble gases has been well demonstrated by Butler (71). Comparisons have been done with the direct Fick (72), dye dilution (73, 74) and acetylene methods (75). A wide use of the carbon dioxide rebreathing method has been applied to exercise in healthy subjects (73, 76-80) as well as to cardiac patients (81, 82), with satisfactory results for the cardiac output measurements, since the development and improvement of rapid gas analysers. The reproducibility of the method (83-85) is reported to increase with the intensity of exercise (73).

CHAPTER III

METHODS AND PROCEDURES

MATERIAL

Seven male patients treated for a recent myocardial infarction were invited to participate in this non-invasive study. The age range of the subjects was forty nine to sixty one years with a mean age of 54.0 years. The time range for their last myocardial infarction was four to eight months with a mean time of 5.6 months.

No patients with heart failure, with unstable angina, with uncontrolled hypertension, with more than two episodes of myocardial infarction, with manifest diabetes mellitus, with disabling diseases were included as subjects. All patients 65 years of age or older were also excluded.

For a diagnosis of myocardial infarction, the following criteria were required: ECG changes with appearance of a pathological Q wave, arrhythmias or segment changes, serum enzyme alterations (SGOT, LDH, CPK). The diagnosis was made by an experienced cardiologist.

TESTING ENVIRONMENT

The subjects came to the Clinical Sciences Building adjacent to the University Hospital, three months after the myocardial infarction, to be evaluated in a bicycle screening test and in a preliminary treadmill

test within two weeks after the screening test. Before the start of the experiment, they were tested in a pre-test and monthly thereafter at the same time of the day.

The testing and training room temperature was maintained at 23 $\pm$ 2^{o}C but the relative humidity was not controlled. The elevation was approximately 2000 feet above sea level and the atmospheric pressure was 700 $\pm$ 10 mmHg.

CALIBRATION OF GASES AND INSTRUMENTS

Reference gases were checked with a Scholander apparatus prior to the testing according to the Scholander technique described by Consolazio, Johnson and Pecora (87).

The rapid paramagnetic oxygen analyser (Rapox) was calibrated with a 100 per cent oxygen concentration and room air while the rapid carbon dioxide analyser (Godart, NV, Capnograph) (Plate 1) was calibrated with the checked gas concentrations before every test with a constant procedure. High concentrations of carbon dioxide were employed (12 and 10%) and necessitated the use of a quadratic equation in order to correct the alinearity of the carbon dioxide values over a 10% gas concentration. The correction factor (for barometric pressure and temperature) for converting the gas volume to STPD was taken before every testing period.

A ten-liter Parkinson Cowan volume meter was used to calculate the inspired air and a correction factor of 1.02 was applied to the inspired volume registered.

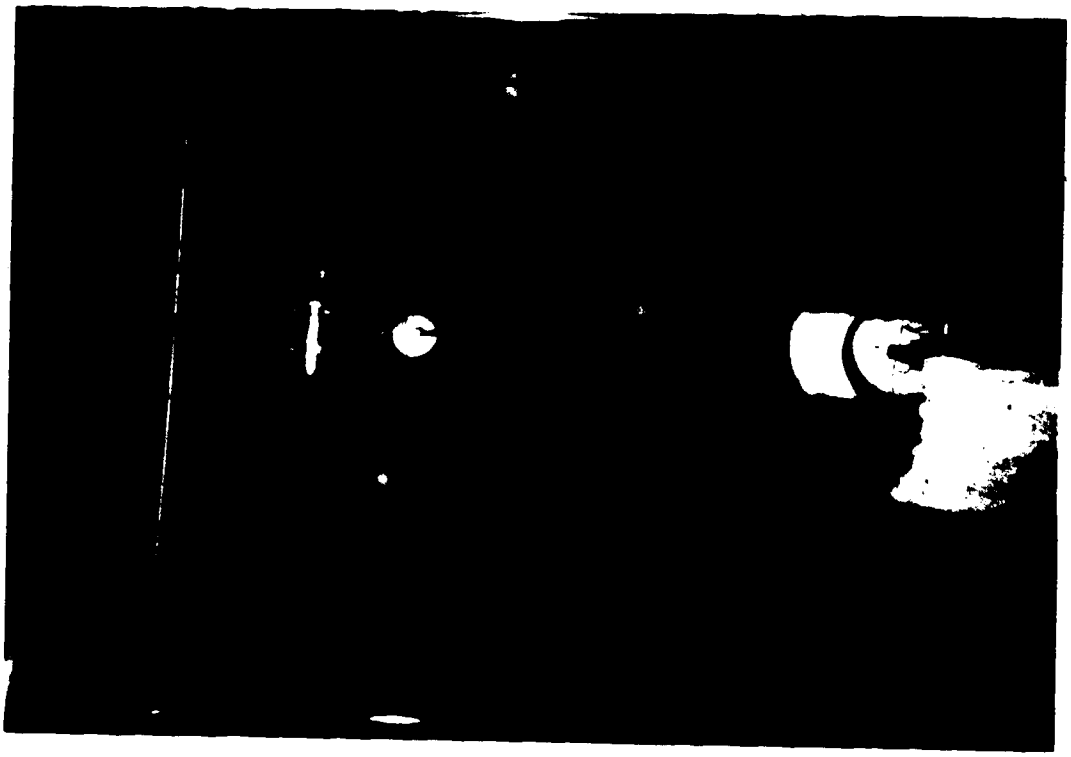
A four channel Beckman dynograph (type RB, 374) was used to simultaneously record the carbon dioxide from the capnograph, the oxygen from the Rapox and the volume inspired from the volume meter. The paper speed was verified with a stop-watch.

The electrically braked ergometer bicycle (Quinton, model 844) had a reported factory variation of 2Rpm but this was not verified.

COLLECTION AND ANALYSIS OF GASES

A two way Godart stopcock with a fitted sterilized Collins rubber mouthpiece was sealed to a Collins triple "T" high velocity valve containing 180 c.c. of dead space and was firmly suspended to a transverse bar (Plate 2). A low-resistant, flexible plastic hose (1½" diameter) connected the valve to the Parkinson Cowan volume meter and to the collecting bag. The air passed through the volume meter for the calculation of the inspired volume and was mixed in a meteorological rubber bag for the analysis of the expired gases. The subjects' noses were clamped with a soft foam clip. The mixing bag was tightly fixed to a two way metal valve which permitted the collection of the expired air at the desired time.

The inspired volume was measured by a rotating heliport (Beckman potentiometer) situated on the needle axis of the Parkinson Cowan volume meter. The expired carbon dioxide and oxygen concentrations were analysed from the mixing rubber bag which was shaken before gas analysis. The readouts from the Godart capnograph, the Rapox and the volume meter were simultaneously recorded on a curvilinear, four channel paper by a



Beckman dynograph (type RB, 374)

REBREATHING METHOD

Campbell's method (74), which is based on the Fick equation with carbon dioxide values, was used to determine cardiac output. This may be demonstrated to be:

$$\dot{Q} = \frac{\dot{V}CO2}{C_{\bar{v}}CO2 - C_{a}CO2}$$

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

$\dot{V}CO2$ = CO2 output in milliliters per minute, STPD

$C_{\bar{v}}CO2$ = milliliters of CO2 per liter of mixed venous blood

$C_{a}CO2$ = milliliters of CO2 per liter of arterial blood

The inspired ventilation was recorded and at the same time the expired gas was collected for thirty seconds after a work time of 3½ minutes which corresponded to a steady state as judged by a variation of less than ± 2% of CO2 in the end-tidal sample or of less than ± 2 heart beats per minute. With the volume and concentration of the expired gas, the $\dot{V}CO2$ was measured.

Immediately after the gas collection, the expired gas was sampled at the mouth (tidal gas) in order to determine the $C_{a}CO2$. When a constant end-tidal sample was recorded, the subject rebreathed a mixture of CO2 in O2 and the $C_{\bar{v}}CO2$ was then determined within thirty seconds of

the end of the expired gas collection.

REBREATHING TECHNIQUE

The apparatus used for rebreathing is shown on Plate 2. It served to obtain estimates of mixed venous P_{CO2} and consisted of a five-liter bag fitted to one port of the Godart stopcock just distal to the mouthpiece. The bag was filled with a known concentration of CO2 in O2 using a big cylinder tank which had a security valve. The mixtures were already prepared (4%, 7%, 10% and 12% CO2 in O2). The tap of the Godart stopcock was turned at the end of a normal expiration and the subject rebreathed deeply and rapidly for 5-8 seconds (rate 50/min). When the gas concentration was well chosen the equilibrium between the bag and the lungs gases was rapidly obtained and followed by a plateau value recorded on the Beckman dynograph at a speed of 2.5 millimeters per second. The gas was continually sampled near the mouth level and monitored by a rapid carbon dioxide analyser at a flow rate of 1.5 liter per minute.

The equilibrium pattern was recognized when the P_{CO2} differences were within $\pm$ 1 millimeter of Hg on an inspiration followed by an expiration (the plateau). It was imperative that the plateau was obtained before the recirculation raised the $C\bar{v}_{CO2}$, especially at high work load levels. Because of the high concentration of O2 in the rebreathing mixture, it was assumed that the blood was fully oxygenated. The CO2 tensions were converted to CO2 contents using the standard CO2 dissociation curve. The following equation was used as a more convenient way to express this curve:

$$C_{CO2} = Log_e C_{CO2} = (0.396 \times Log_e P_{CO2}) + 2.4$$

$$C_{CO2} = \text{Antilog } (Log_e C_{CO2})$$

where: C_{CO2} = CO2 content

P_{CO2} = CO2 tension (partial pressure)

Jones' downstream correction factor (86), $(P\bar{v}_{CO2} \times 0.24) - 1.1$, was applied before the conversion to the $C_{\bar{v}CO2}$. This correction factor accounts for the alveolar-to-blood P_{CO2} difference during rebreathing of CO_2 in O_2 mixtures during exercise.

TESTING PROCEDURES

Subject selection

All patients were examined by the same experienced cardiologist who supervised all work tests and followed the subjects during the course of the experiment.

Once the patient was transferred from the coronary unit to the ward at the University Hospital in Edmonton, he received general information about the program. The patients usually agreed and responded favorably with the consent of their family doctor. The subject was asked to return for readmission approximately three months later for a screening test.

Screening and preliminary tests

The subjects came to the laboratory and were weighed on a medi-

cal scale. During the bicycle screening test, the patient was made familiar with the equipment and procedures. An attempt was made to evaluate his working capacity with the aid of the electrocardiogram and the sphigmomanometer. If he demonstrated moderate to severe angina pectoris, pathological arrhythmias or unusual ST-segment changes, the subject was excluded from the experiment.

Within two weeks after the screening test, a preliminary Bruce treadmill test (44) (see Appendix 2) was performed to evaluate his symptom limited maximum work capacity. The blood pressure was monitored sphygmomanometrically every minute while the electrocardiogram showed a constant reading of the heart rate per minute and the ST-segment changes every 5 heart beats (Quinton ST-segment computer, model 730). The maximal heart rate thus obtained, served as the selected percentage of the possible training and testing levels. The electrodes used for the ECG were placed in a modified lead I position (Plate 2). The maximum oxygen consumption was measured as well and the gas was collected during the last twenty seconds before the cessation of the effort due to exhaustion and/or symptoms.

Pre-test

A bicycle pre-test consisting of three different work loads, corresponding firstly to a warm-up level (200 Kpm), secondly to a work load corresponding to 70% of the maximal heart rate and thirdly to a work load corresponding to 90% of the maximal heart rate previously determined, was used. These three work loads were separated by a three minute rest period

on the bike and followed by a twenty rest period on a chair.

A second determination was performed immediately following the 20 minute rest period. The parameters such as the oxygen uptake, cardiac output, heart rate and stroke volume were evaluated as previously described. The time of the experimentation lasted between 70 to 80 minutes for each patient. The same procedure was repeated every month during three months. All the data were computed and transcribed on a data sheet (Appendix 1).

GROUP ASSIGNMENT

Following the preliminary test, the subjects were randomly allocated to either group; four patients were assigned to the experimental group and three to the reference group. Because of an early return to work, one experimental subject dropped the experiment after ten weeks of training, reducing his group to three subjects. The random assignment of the subjects to the groups was done with an odd and even number table. For each patient an envelope containing the group designation was then taken and it was noted whether the subject was experimental or reference.

TRAINING MODEL

The subjects in the reference group were encouraged to walk one mile per day in order to be able to perform the monthly stress test without any harmful consequences. A record of their daily exercise routine was kept by asking them to note every exercise done. The purposes of this record were to increase their motivation for the experiment by keeping interest in them and to simulate the usual physician's recommendations.

The physical training for the other group was carried out four times per week at the laboratory. Each training session was led by a qualified exercise physiologist and a registered nurse and lasted approximately one hour. Each patient was trained individually, as the number of patients was too small to form a group.

The first two to three weeks served as a general conditioning in order to increase the tolerance of the cardiac patients. Gradually the duration and intensity of the training stimulus were increased on an individual basis up to about 80% of their maximum heart rate.

Figure 1 shows the schematic arrangement of a training session. It started with a five minute warm-up followed by a 35 minute continuous exercise, a 5 minute active recovery period and a 10 minute passive recovery period. The patients were monitored all the time and trained alternatively on the treadmill and on the electrically braked ergometer bicycle. A physician was always available in an adjacent room i[redacted] in the same room, and an emergency equipment including a defibrillator were present in the room and kept in readiness.

No complications such as angina, arrhythmias or reinfarction occurred during the training and testing periods even though the patients worked near the maximal capacity. The mean percentage of participation for the experimental group was 81% when excluding excused absences such as work out of town, vacation, holiday or temporary illness and 77% when including these absences.

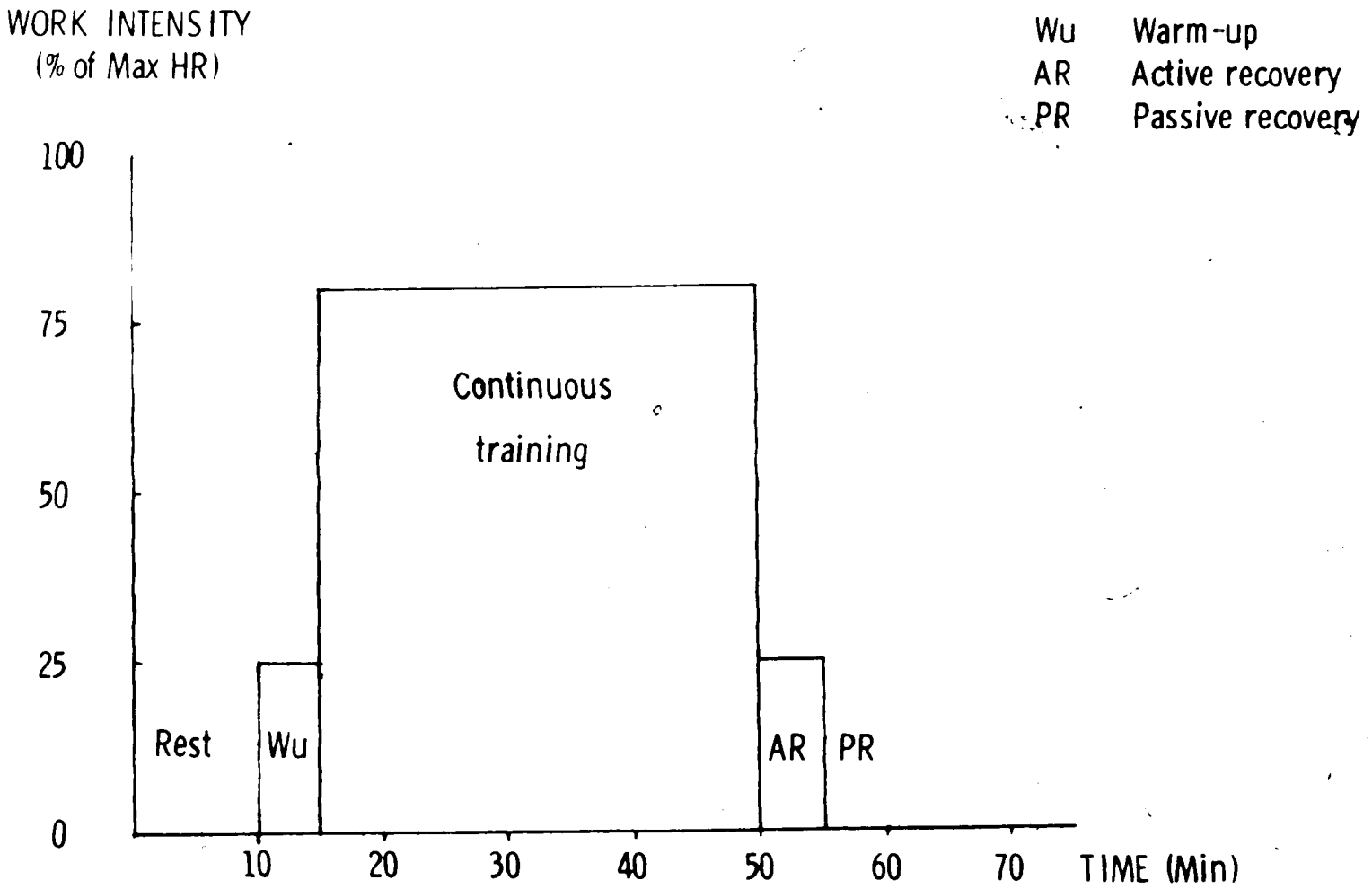


Figure 1 Schematic arrangement of the training sessions

STATISTICAL PROCEDURES AND EXPERIMENTAL DESIGN

The design

A 2x4x3x2 factorial design, with repeated measures on factor R, factor M and factor L was used. The two levels of factor R were the repetitions:

a) first repetition values

b) second repetition values

The four levels of factor M were the months:

a) beginning values

b) first month values

c) second month values

d) third month values

The three levels of factor L were the loads:

a) warm-up level (200 Kpm)

b) 70% of maximal heart rate

c) 90% of maximal heart rate

The two levels of factor G were the groups:

a) experimental group

b) reference group

Statistical analysis

The data on each parameter were analysed initially by a four way analysis of variance with repeated measures on factors R, M and L. If significant F ratios were obtained on the simple main effect, a Newman Keuls test was used as a comparison between ordered means.

Ordinary statistical methods were used for the calculation of the means, standard deviations (SD) and standard errors of the mean (SEM) (88).

The error of a single determination was calculated from duplicate determinations according to the formula:

$$\text{error} = \sqrt{(d^2)/2n} \quad (88)$$

Regression lines were calculated to determine a relationship between various parameters. The significance of the relationship was assessed by testing the regression coefficient.

The two significant levels ($P < .01$ and $P < .05$) were used. All computations were done via the IBM 360 computer at the University of Alberta.

CHAPTER IV

RESULTS AND DISCUSSION

As regards the anthropometric measurements (Table II), the experimental group had a significantly higher period after LMI (last myocardial infarction). Both groups revealed approximately the same age, height, weight and infarct site. Despite minor differences, the groups may be considered to be comparable.

RESULTS AT SUBMAXIMAL WORK CAPACITY

The experimental and reference group values for the various hemodynamic parameters are reported in Appendix 4. The statistical analysis of each parameter is summarized in Appendix 5.

Oxygen consumption

Following the training period, oxygen consumption relative to body weight at the same given submaximal work loads increased slightly (Table III, Figure 2) by 4.6% (from 18.23 to 19.11 ml/kg/min) and 3.3% (from 19.79 to 20.45 ml/kg/min) for the experimental and the reference group respectively. However a four way analysis of variance (Appendix 5-1, a) failed to show any significant treatment or interaction effects between the groups and the months. The two groups responded the same way to the different loads, the reference group being always superior in his oxygen consumption. Body weight decreased by 2.6% (from 81.0 to

TABLE II Clinical and anthropological data

Group	S	Age	Ht	Wt	LMI	Infarct site
EXP	1	50	173	90.9	8	Anteroseptal
	2	50	163	76.7	8	Inferior
	3	60	178	75.3	7	Anteroseptal
	Mean	53	171	81.0	8	
	SEM	3.3	4.4	5.0	0.3	
	N	3	3	3	3	
REF	1	61	174	69.1	4	Anteroseptal
	2	54	176	91.8	4	Inferior
	3	54	178	76.4	4	Anteroseptal
	Mean	56	176	79.1	4	
	SEM	2.3	1.2	6.7	-	
	N	3	3	3	3	

EXP: Experimental; REF: Reference; S: subject; SEM: standard error of the mean; AGE in years; Ht: Height in cm.; Wt: weight in kg.; LMI: Last myocardial infarction.

TABLE III Data for OXYGEN CONSUMPTION relative to body weight in milliliters per kilogram per minute at submaximal work loads

WARM-UP LEVEL		MONTHS			
Group		0	1	2	3
	Mean	12.32	11.18	12.23	11.94
EXP	SEM	0.76	1.19	0.97	0.87
	N	6	6	6	6
	Mean	12.26	13.12	11.34	13.53
REF	SEM	0.38	2.84	0.46	0.79
	N	6	6	6	6
70% OF MAXIMAL HEART RATE					
	Mean	19.35	18.95	21.26	20.20
EXP	SEM	0.88	2.46	1.64	2.03
	N	6	6	6	6
	Mean	21.47	19.40	19.41	21.38
REF	SEM	1.15	1.25	1.06	1.06
	N	6	6	6	6
90% OF MAXIMAL HEART RATE					
	Mean	23.01	22.98	27.12	25.19
EXP	SEM	1.15	2.76	1.52	1.89
	N	6	6	6	6
	Mean	25.64	23.83	23.08	26.43
REF	SEM	0.69	1.34	1.24	1.22
	N	6	6	6	6

EXP: Experimental; REF: Reference; SEM: Standard error of the mean.

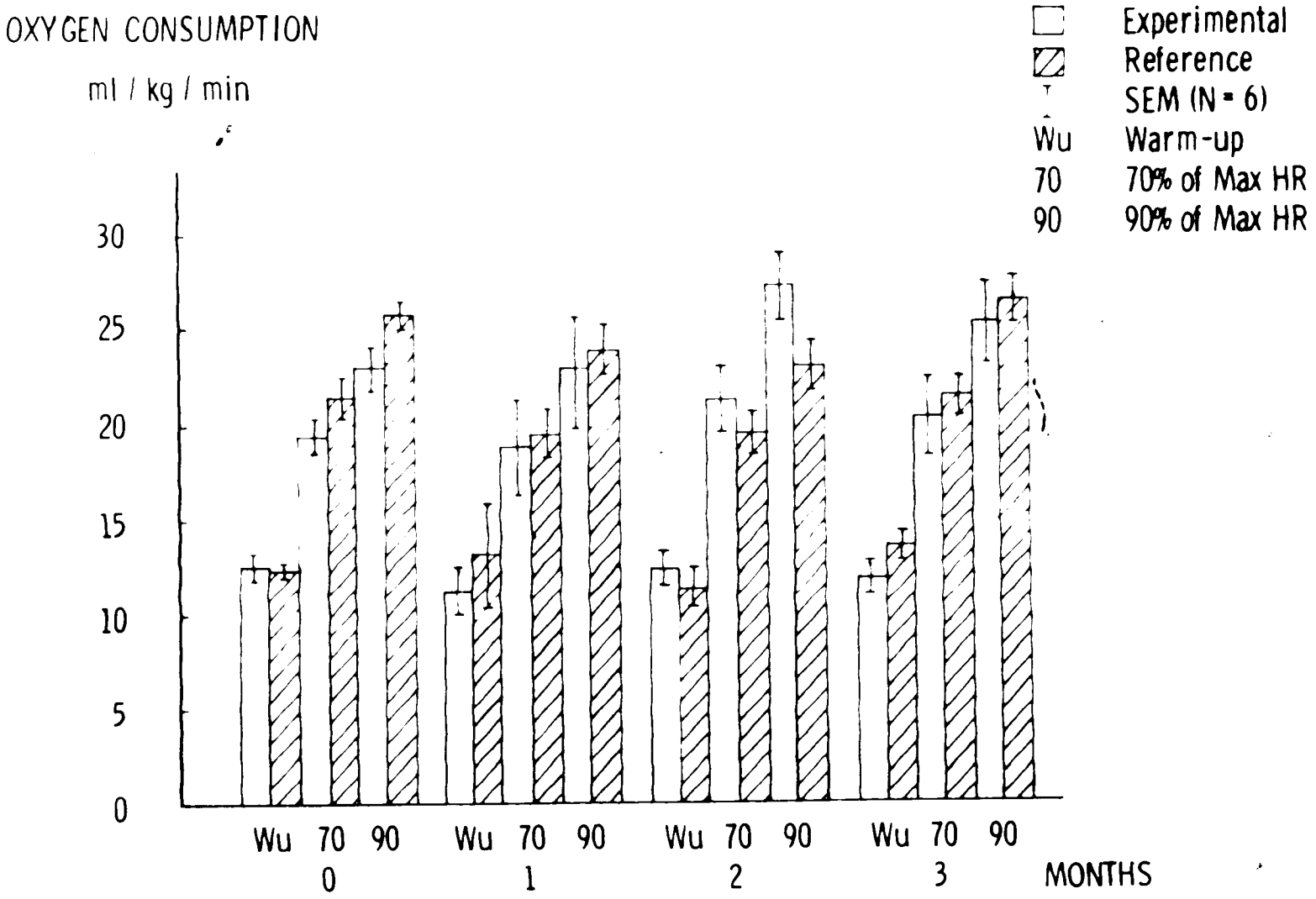


Figure 2 Oxygen consumption at submaximal work loads

78.8 kg) and by 1.4% (from 78.8 to 77.7 kg) (Figure 7) for the experimental and the reference group respectively. No significant change has been noted.

A significant ($P < .01$) difference between the loads has been found (12.24, 20.18, 24.66 ml/kg/min for L1, L2 and L3 respectively). Therefore, a Newman Keuls test for ordered means (Appendix 5-1, b) has been applied and revealed a significant difference between all means for loads.

Cardiac output

Cardiac output showed a slight decrease of 4.3% (from 14.38 to 13.78 l/min) for the experimental group whereas the reference group showed a minor increase of 6.9% (from 12.63 to 13.51 l/min) after the training period (Table IV, Figure 3). A four way analysis of variance (Appendix 5-2, a) showed a significant ($P < .05$) difference between the months. A Newman Keuls test was then applied (Appendix 5-2, b) to look at the difference between ordered means (13.51, 13.98, 14.87, 13.64 l/min for M0, M1, M2, and M3 respectively). The results showed a significant ($P < .05$) difference between the third measurement (M2) and the other measurements; however no significant difference has been found between the other measurements (M0, M1 and M3).

A four way analysis of variance (Appendix 5-2, a) revealed also a significant ($P < .01$) F ratio for the simple main effect on factor work (L) (10.37, 15.60, 16.03 l/min for L1, L2 and L3 respectively),

TABLE IV Data for CARDIAC OUTPUT in liters per minute at sub-maximal work loads

WARM-UP LEVEL		MONTHS			
Group		0	1	2	3
EXP	Mean	10.92	11.50	10.95	9.96
	SEM	0.73	0.54	0.76	0.31
	N	6	6	6	6
REF	Mean	8.97	9.35	10.34	10.95
	SEM	0.34	0.34	0.30	0.69
	N	6	6	6	6
70% OF MAXIMAL HEART RATE					
EXP	Mean	15.81	16.88	16.12	15.17
	SEM	0.65	0.96	0.52	0.53
	N	6	6	6	6
REF	Mean	14.09	14.43	17.65	14.67
	SEM	1.21	1.11	1.62	1.29
	N	6	6	6	6
90% OF MAXIMAL HEART RATE					
EXP	Mean	16.41	16.43	18.09	16.20
	SEM	1.15	1.05	0.27	0.69
	N	6	6	6	6
REF	Mean	14.83	15.27	16.08	14.92
	SEM	1.33	1.61	1.80	1.77
	N	6	6	6	6

EXP: Experimental; REF: Reference; SEM: Standard error of the mean.

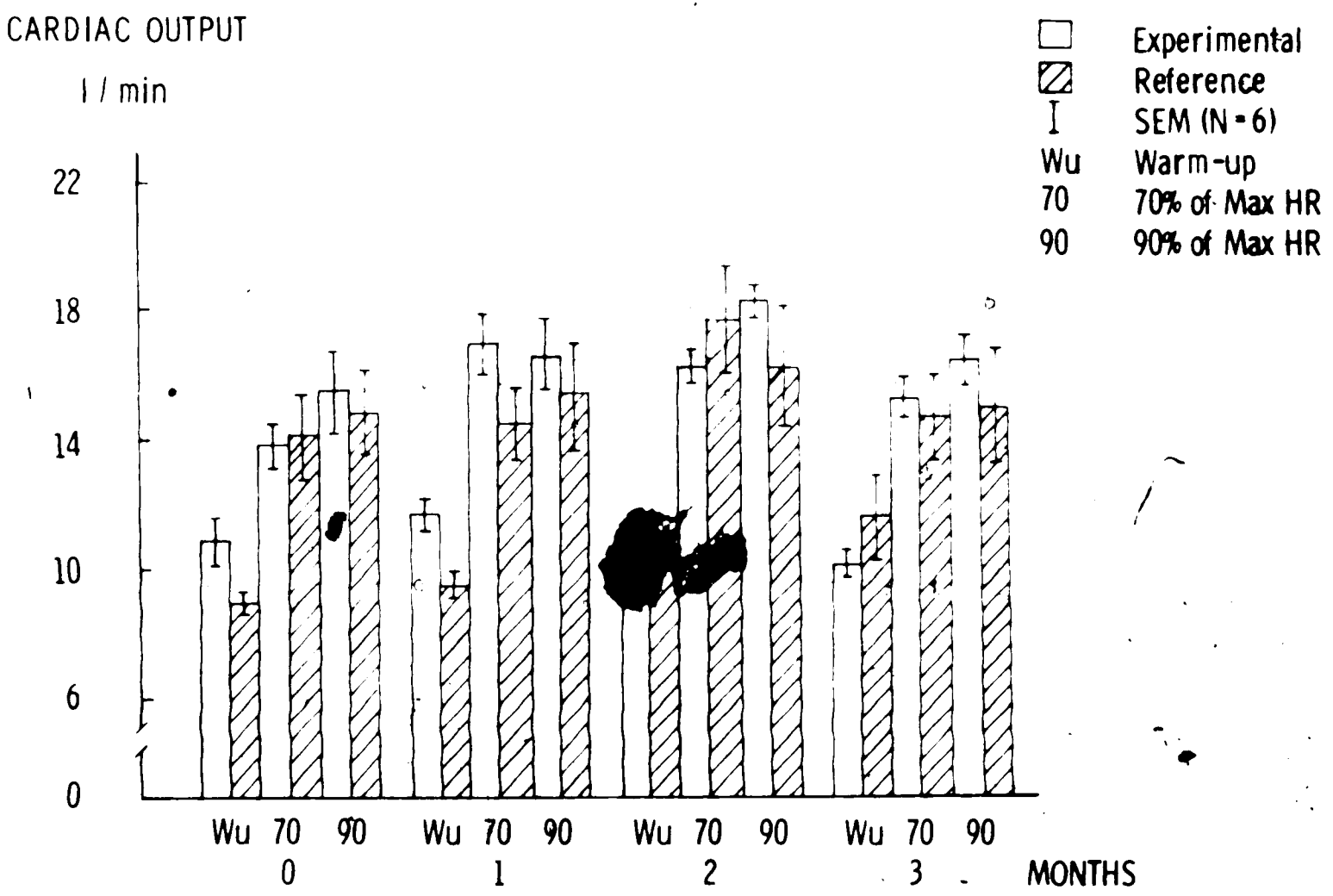


Figure 3 Cardiac output at submaximal work loads

therefore, a Newman Keuls test (Appendix 5-2, c) was carried out and showed that cardiac output for L1 was significantly ($P < .01$) lower than cardiac output for L2 and L3 but the difference was not statistically significant between L2 and L3.

Heart rate

The heart rate decreased (Table V, Figure 4) by 8% (from 130.9 to 121.0 beats/min) for the experimental group and by 1.6% (from 132.7 to 130.5 beats/min) for the reference group over the training period. A four way analysis of variance (Appendix 5-3, a) revealed a significant ($P < .01$) F ratio for the simple main effect on factor time (M) (131.81, 127.44, 125.61, 125.75 beats/min for M0, M1, M2 and M3 respectively). Therefore, a Newman Keuls comparison between ordered means (Appendix 5-3, b) was applied and showed a significant ($P < .05$) higher heart rate at M0 over the three other months. Although there was no significant interaction effect between the months and the groups, the experimental group showed a constant decrease in heart rate over the training period whereas the reference group showed some fluctuations (Figure 4).

The four way analysis of variance (Appendix 5-3, a) also revealed a significant ($P < .01$) F ratio for the simple main effect on factor loads (L) (121.7, 130.7, 148.8 beats/min for L1, L2 and L3 respectively). A Newman Keuls test (Appendix 5-3, c) then revealed significant ($P < .01$) differences between all the loads.

A further analysis (Appendix 5-3, a) indicated a significant

TABLE V Data for HEART RATE in beats per minute at submaximal work loads

WARM-UP LEVEL		MONTHS			
Group		0	1	2	3
EXP	Mean	105	102	103	99
	SEM	4.4	2.9	2.9	2.8
	N	6	6	6	6
REF	Mean	105	102	97	102
	SEM	8.1	7.5	3.8	6.1
	N	6	6	6	6
70% OF MAXIMAL HEART RATE					
EXP	Mean	136	128	127	125
	SEM	5.0	3.8	4.9	5.0
	N	6	6	6	6
REF	Mean	137	133	127	133
	SEM	3.8	3.8	2.5	2.4
	N	6	6	6	6
90% OF MAXIMAL HEART RATE					
EXP	Mean	152	145	140	140
	SEM	4.5	4.0	6.1	5.8
	N	6	6	6	6
REF	Mean	156	153	149	156
	SEM	2.2	3.3	3.0	1.4
	N	6	6	6	6

EXP: Experimental; REF: Reference; SEM: Standard error of the mean.

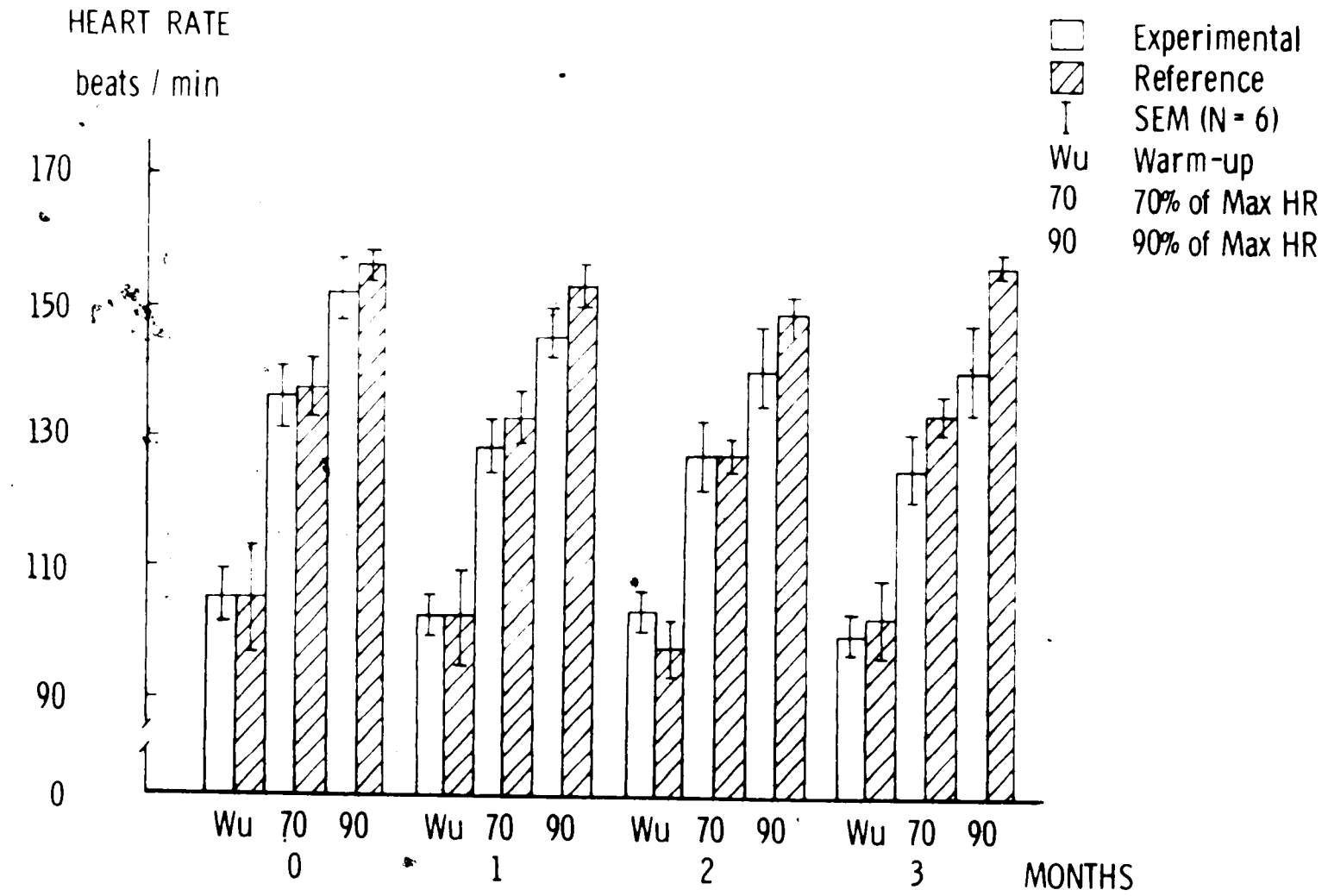


Figure 4 Heart rate at submaximal work loads

($P < .05$) F ratio for the simple main effect on factor repetitions (R) (124.9, 129.2 beats/min for R1 and R2 respectively). R2 being significantly ($P < .05$) higher than the first one.

Stroke volume

Following the training period, stroke volume at the same given submaximal work loads increased slightly (Table VI, Figure 5) by 3.2% (from 110.54 to 114.17 ml/beat) and by 11.0% (from 95.24 to 105.72 ml/beat) for the experimental and the reference group respectively. A four way analysis of variance (Appendix 5-4, a) showed a significant ($P < .01$) simple main effect on factor time (M) (102.89, 110.92, 120.33, 109.94 ml/beat for M0, M1, M2, and M3 respectively). Therefore, a Newman Keuls test (Appendix 5-4, b) was applied and showed a significant ($P < .05$) [illegible] stroke volume in M0 over M1, M2 and M3; but M1 was not significantly different ($P < .05$) from M3.

A four way analysis of variance (Appendix 5-4, a) revealed a significant ($P < .05$) main effect on factor work (L) (103.25, 120.72, 109.10 for L1, L2 and L3 respectively). A Newman Keuls procedure for ordered means (Appendix 5-4, c) showed a significant ($P < .05$) difference between L2 and L1 only.

This four way analysis of variance also revealed a significant ($P < .05$) main effect on factor repetitions (R) (113.64, 108.41 ml/beat for R1 and R2 respectively) R2 being significantly ($P < .05$) lower than R1.

TABLE VI Data for STROKE VOLUME in milliliters per beat at sub-maximal work loads

WARM-UP LEVEL		MONTHS			
Group		0	1	2	3
EXP	Mean	104.8	113.2	106.5	101.3
	SEM	8.95	5.41	5.71	4.76
	N	6	6	6	6
REF	Mean	86.9	94.4	108.4	110.6
	SEM	4.92	6.73	6.69	11.99
	N	6	6	6	6
70% OF MAXIMAL HEART RATE					
EXP	Mean	117.4	133.4	128.3	123.4
	SEM	6.97	10.53	7.01	8.27
	N	6	6	6	6
REF	Mean	103.7	109.7	139.1	110.7
	SEM	9.88	9.88	13.14	10.74
	N	6	6	6	6
90% OF MAXIMAL HEART RATE					
EXP	Mean	109.4	115.1	130.9	117.8
	SEM	9.76	10.12	7.26	9.15
	N	6	6	6	6
REF	Mean	95.1	99.8	108.7	96.0
	SEM	8.53	10.39	13.00	12.02
	N	6	6	6	6

EXP: Experimental; REF: Reference; SEM: Standard error of the mean.

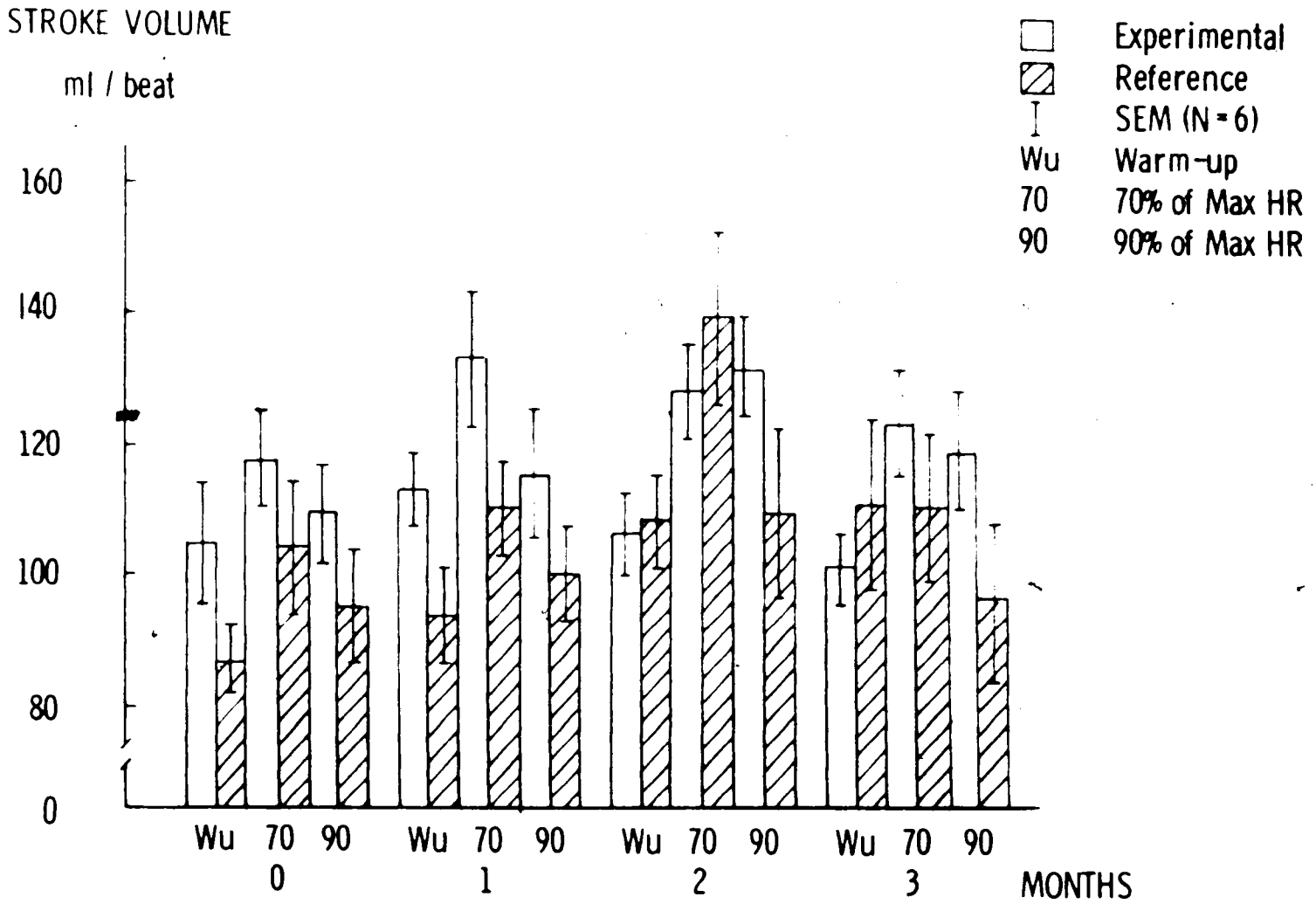


Figure 5 Stroke volume at submaximal work loads

RESULTS AT MAXIMAL WORK CAPACITY

The values at maximal work capacity are reported in Table VII.

Maximal oxygen consumption

Following the training period, the experimental group showed an improvement of 30.9% (from 29.57 to 38.71 ml/kg/min) (Table VII, Figure 6) while the reference group demonstrated a slight increase of 1.7% (from 28.70 to 29.18 ml/kg/min). Body weight decreased by 2.6% (from 81.0 to 78.8 kg) and 1.4% (from 78.8 to 77.7 kg) (Figure 7) for the experimental and the reference group respectively. No significant ($P < .05$) changes have been noted.

Maximal heart rate

Maximal heart rate (Table VII, Figure 8) did not change to a great extent in both groups after training (from 164.3 to 163.7 beats/min and from 161.0 to 159.7 beats/min in the experimental and the reference group respectively).

Maximal work capacity

Even though the means of the groups in terms of stages have not been calculated (Table VII), both the experimental and the reference group improved appreciably their tolerance to maximal effort in terms of total time during the continuous treadmil test (from 11.7 to 12.3 minute and from 9.3 to 11.0 minute for the experimental and the reference group respectively) after the training period.

TABLE VII Data at maximal work capacity on treadmill

Group	S	Pre-training					Post-training				
		MHR	MVO2	Wt	St	T	MHR	MVO2	Wt	St	T
EXP	1	150	31.79	90.9	3	3	164	39.32	88.1	4	1
	2	175	35.81	76.7	4	1	167	39.07	73.2	4	1
	3	168	21.12	75.3	3	1	160	37.75	75.5	3	2
	Mean	164.3	29.57	81.0			163.7	38.71	78.9		
	SEM	7.4	4.4	5.0			2.2	0.5	4.6		
	N	3	3	3			3	3	3		
REF	1	160	23.01	69.1	2	3	145	23.88	67.0	2	3
	2	161	32.77	92.8	3	1	155	26.84	93.5	3	2
	3	162	30.33	74.5	2	3	179	36.83	72.5	4	1
	Mean	161.0	28.70	78.8			159.7	29.18	77.7		
	SEM	0.6	2.9	7.2			10.1	3.9	8.1		
	N	3	3	3			3	3	3		

EXP: Experimental; REF: Reference; S: Subject; SEM: Standard error of the mean; MHR: Maximal heart rate in beats per minute; MVO2: Maximal oxygen consumption in milliliters per kilogram per minute; Wt: Weight in kilograms; St: Stage (ref. Appendix 2); T: Time in minutes.

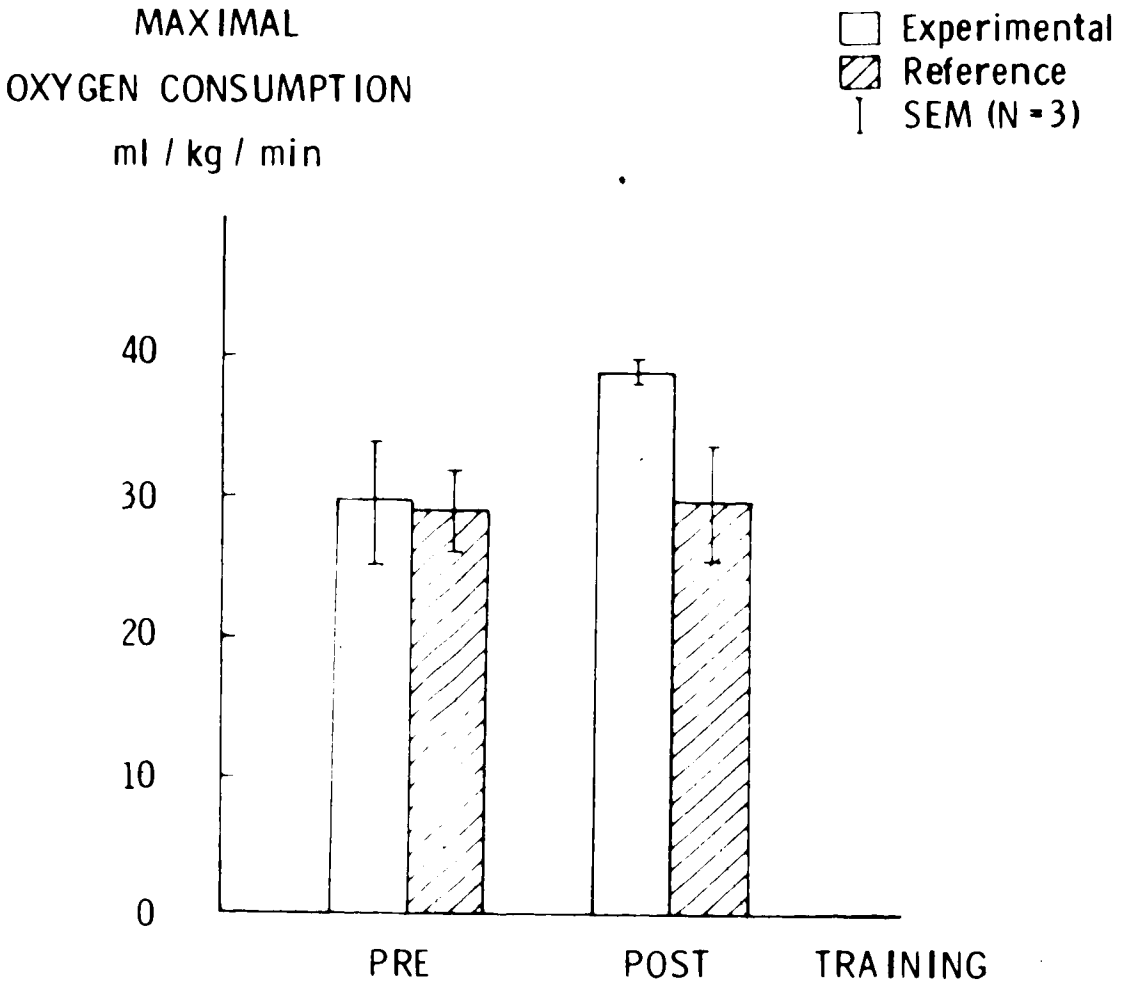


Figure 6 Oxygen consumption at maximal work capacity

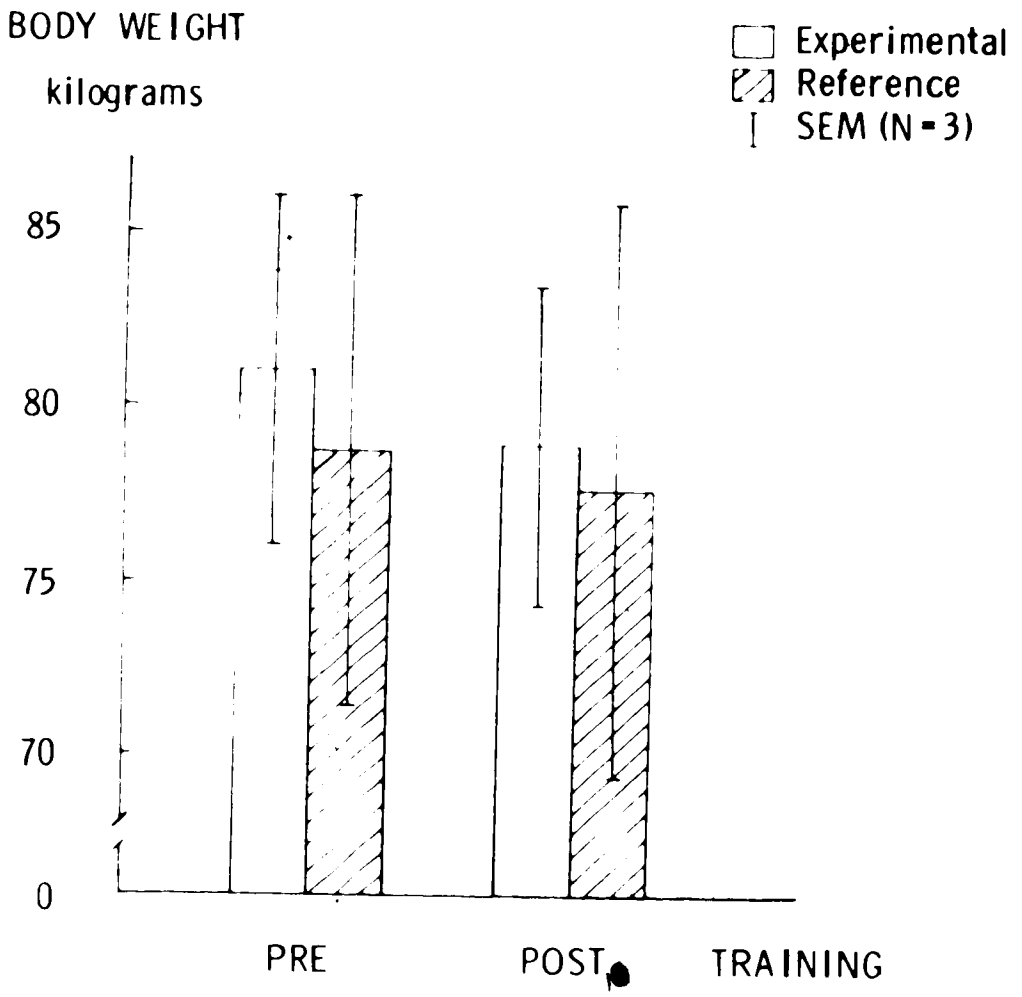


Figure 7 Body weight before and after training

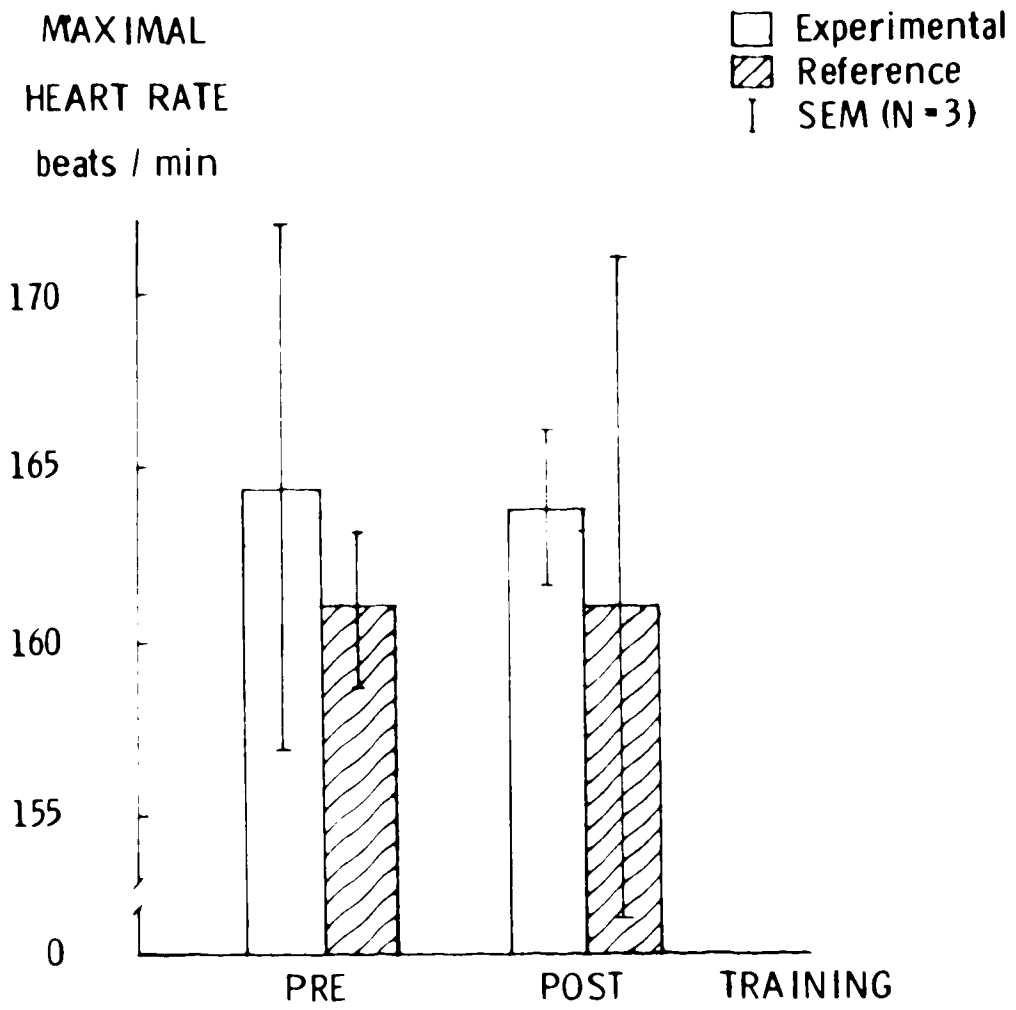


Figure 8 Heart rate at maximal work capacity

CORRELATION COEFFICIENT

The individual correlations between oxygen consumption and cardiac output are summarized in Table VIII. In most cases, both in the experimental and the reference group, oxygen consumption seems to be a relatively good predictor of cardiac output with an overall correlation coefficient of 0.52 and 0.61 for the experimental and the reference group respectively.

The individual correlations between heart rate and cardiac output are summarized in Table IX and revealed an overall correlation coefficient of 0.57 for the experimental group and 0.46 for the other group.

REPRODUCIBILITY

In order to look at the reproducibility of the carbon [illegible] rebreathing method, double determinations were performed with a 20 minute rest interval between each set of determinations. The percentage of error (Table X) was relatively small specially for cardiac output where the t-test did not show any significant ($P < .05$) difference between double determinations. However, oxygen consumption, heart rate and stroke volume showed some significant differences.

DISCUSSION

The effects of short-term physical training on cardiac patients can be visualized in two ways: firstly, by cardiorespiratory adaptations to a standard submaximal work load; secondly, by partial inhibition of

TABLE VIII Individual correlations between OXYGEN CONSUMPTION and CARDIAC OUTPUT

Group	S	N	B	A	R
EXP	1	24	0.60	5.08	0.89
	2	24	0.29	6.53	0.82
	3	24	0.40	8.67	0.66
	Total	72	0.25	9.79	0.52
REF	4	24	0.09	9.06	0.31
	5	24	0.43	6.07	0.80
	6	24	0.59	4.04	0.88
	Total	72	0.41	5.50	0.63

EXP: Experimental; REF: Reference;
S: Subjects; N: Number of measurements;
B: Slope of CARDIAC OUTPUT predicted from OXYGEN CONSUMPTION; A: Intercept;
R: Correlation coefficient.

TABLE IX Individual correlations between HEART RATE and CARDIAC OUTPUT

Group	S	N	B	A	R
EXP	1	24	0.09	-6.83	0.91
	2	24	0.08	1.79	0.78
	3	24	0.14	-1.83	0.71
	Total	72	0.09	3.36	0.57
REF	4	24	0.02	7.58	0.28
	5	24	0.09	2.31	0.81
	6	24	0.15	-2.62	0.84
	Total	72	0.07	3.91	0.46

EXP: Experimental; REF: Reference; S: Subjects; N: Number of measurements; B: Slope of CARDIAC OUTPUT predicted from HEART RATE; A: Intercept; R: correlation coefficient.

TABLE X Reproducibility of different variables at different work intensities estimated from double determinations for the two groups

GRP	Load	N	M 1	M 2	SD1	SD2	t	ER	% ER
Oxygen consumption									
	W-up	12	11.87	11.97	2.4	2.2	-0.15	0.66	0.8
EXP	70%	12	19.31	20.56	4.5	4.2	-1.61	0.83	6.5
	90%	12	23.62	25.53	5.2	4.3	-2.63*	0.89	8.1
	W-up	12	12.53	12.59	4.6	2.3	-0.06	0.96	0.5
REF	70%	12	19.95	20.87	2.8	2.8	-2.09	0.50	4.6
	90%	12	24.17	25.32	3.1	2.8	-3.31**	0.47	4.8
Cardiac output									
	W-up	12	11.06	10.60	1.7	1.4	0.81	0.56	4.2
EXP	70%	12	16.14	15.84	1.5	1.9	0.90	0.33	2.0
	90%	12	16.75	16.82	2.3	2.0	-0.20	0.34	0.4
	W-up	12	10.05	9.76	1.5	1.1	0.83	0.35	3.0
REF	70%	12	15.14	15.27	3.1	3.7	-0.25	0.47	0.9
	90%	12	15.10	15.44	3.8	3.9	-1.09	0.32	2.3
Heart rate									
	W-up	12	103.5	103.3	6.3	9.4	-1.03	2.43	2.6
EXP	70%	12	126.6	130.8	10.9	12.4	-2.89*	1.87	3.3
	90%	12	142.7	145.2	12.2	13.9	-2.70*	1.14	1.8
	W-up	12	96.7	105.9	16.9	12.6	-5.03**	3.20	9.5
REF	70%	12	130.1	134.9	9.2	6.3	-3.29**	1.98	3.7
	90%	12	152.2	155.1	8.1	4.6	-2.04	1.56	1.9
Stroke volume									
	W-up	12	110.3	102.9	17.7	12.7	1.42	5.46	7.2
EXP	70%	12	138.8	122.5	18.7	21.5	1.80	3.78	5.1
	90%	12	119.1	117.6	23.2	22.4	0.58	2.40	1.3
	W-up	12	106.8	93.3	23.9	15.7	3.31**	5.50	14.5
REF	70%	12	117.5	114.1	27.3	31.3	0.95	3.56	3.0
	90%	12	99.8	100.1	26.3	26.7	-0.18	1.53	0.3

* .05 level
** .01 level

EXP: Experimental; REF: Reference; M: Mean; SD: Standard deviation; ER: Error of the double determination

certain risk factors. In many previous investigations, angina patients have been included as subjects (4, 7, 13, 30, 32, 33, 36, 49, 50, 51, 53, 61). In a desire to keep this study as homogeneous as possible, the angina subjects were referred to another rehabilitation program. The healed myocardial infarct patients were exposed to a more intensive exercise program which comprised four-day training sessions a week. The object of the rehabilitation program was to improve physical capacity in an early period after myocardial infarction. Work capacity, oxygen consumption, cardiac output, heart rate and stroke volume were the parameters selected to evaluate adaptation to physical effort.

The experimental group (with training) was compared to a reference group (without scheduled training) who was asked to walk one mile per day in order to be able to perform the monthly stress test without any harmful effect. Only one patient discontinued his laboratory training in the experimental group because of an early return to work which was nevertheless physically demanding.

The 30.9% improvement (Table VII, Figure 6) at maximal oxygen consumption for the experimental group after the training program is in agreement with many other studies (4, 13, 32, 33, 49, 50, 51). Such improvement is concomitant with a more efficient performance at any given work level; the subjects performed more work at higher intensity. As the result of a low initial fitness level, the rapid increase in work capacity is usually paralleled by a reduction of anxiety in the working situation and a positive attitude toward physical exercise (49).

Duration, frequency and intensity of exercise are important factors to consider as pointed out by Kavanagh and Shephard (4) who did a comparison study between continuous and interval training. Gains in aerobic power were larger for the continuous exercise regimen; but as mentioned by Wenger (89) in a study on healthy subjects, the amount of work performed may be of significance when comparing groups with different training regimens.

Following the training period, a significant decrease of heart rate was observed both in the experimental and reference group at submaximal work capacity. This would seem to imply that physical rehabilitation and/or placebo effect took place in the two groups (55, p.48), but that training also had an effect. It could be possible that the total work performed by the reference group at home elicited a beneficial effect as shown by a slight heart rate decrease. Furthermore, Clausen et al. (55), tend to show that reduction in heart rate is not dependent on intracardiac factors but changes in extracardiac factors, probably the result of peripheral changes modifying the sympathetic stimulation of the sino-atrial node. The positive attitude toward the monthly stress tests could have influenced the heart rate decrease in both groups.

As shown in Figure 8, no significant ($P < .05$) changes occurred in maximal heart rate for both groups. This is in agreement with the Detry et al (33) study which compared angina with healed myocardial infarct patients; the latter group did not show any appreciable change after training compared with the former group who did not obtain a true maximal

heart rate level at the pre-training test because of a symptom limited maximal heart rate. For the same reason, Kasch et al. (50) reported the same observation with a group of angina patients. On the other hand, in an experiment which did not include angina patients, Rousseau et al. (11), reported a small 4.3% increase which was , however, not statistically significant ($P < .05$).

Cardiac output tended to remain the same after the training period except at month 2 (Figure 3) where a minor fluctuation in the two groups appeared; this fluctuation could be possibly due to some seasonal effect (month of May) (90). The same pattern at M2 has been observed in oxygen consumption (Figure 2) values as well as heart rate (Figure 4) and stroke volume (Figure 5) values.

A small but significant increase of stroke volume was noted in both groups. Some studies (7, 13, 30, 32, 36, 53) reported a stroke volume increase whereas others (33, 59) reported an unchanged stroke volume. These findings of an unchanged cardiac output, lower heart rate and increased stroke volume during exercise, correspond well with the results obtained by Kirchheiner et al. (7), Clausen et al. (13), Frick et al. (36) and Bjernult et al. (30).

Clausen et al. (13) mentioned that the increase in stroke volume, calculated as the ratio of cardiac output and heart rate, was mainly due to a 9.3% decrease in heart rate. This finding did not necessarily imply an enhanced myocardial contractility but the alteration of the peripheral circulatory regulation may be an important factor mediating those

hemodynamic changes. Furthermore, it has been noticed, a lower systolic blood pressure concomitant with a lower heart rate causing a reduction in myocardial work. These observations suggest that physical training leads to a physiological improvement in the management of coronary disease.

Frick and Katila (36) have reported the reduced heart rate-increased stroke volume pattern as a training response in patients with coronary heart disease. The data suggested that the increased stroke output can be due to cardiac hypertrophy without change in volume. The 14.9% increase in stroke volume paralleled the 8.3% decrease in heart rate leading to an unchanged cardiac output. They also mentioned a reduced tension-time indices secondary to the relative bradycardia which could improve the ratio of oxygen supply to demand of the heart.

In a six-month training period, Kirchheiner et al. (7) compared two groups of cardiac patients. As the result of training, heart rate decreased 13% and stroke volume increased 16% while cardiac output remained unchanged. This study could not determine if physical training causes central or peripheral hemodynamic changes. Bjernulf (30) reported a similar experiment and the same results with the exception of lower testing intensities.

The above findings seem to support the view that the increase in work capacity and reduction of cardiac work after physical training in patients with coronary heart disease are due to the effects on peripheral circulation.

Contrary to the studies (7, 13, 30, 36) reporting an unchanged

cardiac output, Bergman et al. (59) found that cardiac output was lowered after training, stroke volume was unchanged but the mean arteriovenous oxygen difference increased. The lowering of cardiac output with the increase in arteriovenous oxygen difference after training is explained by a redistribution of blood flow to the exercising muscles.

Although Detry et al. (33) found the same qualitative changes in cardiac output, heart rate and stroke volume as Bergman et al. (59), they did not adhere to the concept of a redistribution of blood flow to the exercising muscles due to the lowering of cardiac output. The post-training bradycardia was considered to be compensated by an increased arteriovenous oxygen difference but the decreased cardiac output being secondary to the improved peripheral utilization of the oxygenated blood.

This concept is in agreement with the findings of Clausen et al. (13, 53) with the ^{133}Xenon-clearance method which demonstrated that muscular blood flow is reduced when measuring at identical submaximal work loads before and after training suggesting a more complete extraction of the oxygenated blood in the working muscles.

The biochemical changes and specially the improvement in the activity of the oxidative mitochondrial enzymes (62-64) observed in muscle tissue after physical conditioning, seem to support the concept of Detry et al. (33) and Clausen et al. (13, 53) of an increased oxygen utilization at the muscular level after training.

The factors responsible for this divergence of opinions in car-

diac output findings could be explained by the difference in age, method employed, severity of myocardial necrosis and ischemia, time elapsed after myocardial infarction, cardiovascular functional capacity at the commencement of training and the magnitude of physical exertion (intensity, duration and frequency) (11, 33, 49). Body position may influence the magnitude of measurable changes (13, 36).

Clausen et al. (13) have examined the patients in the sitting position whereas Frick and Katila (36) in the supine posture. The latter gives optimal conditions for filling the heart chambers, greater venous return, a larger cardiac output, a higher blood pressure, a lower heart rate and a greater stroke volume (30).

The non-significant difference between L2 and L3 for cardiac output (Appendix 5-2, c) may reflect the extent of the myocardial necrosis affecting the blood ejection after a determined work intensity. This is also visualized by a constantly lower stroke volume at the higher work load (L3). This is supported by a recent study done by McDonough et al. (52) on cardiac patients at maximal exercise. They found that the significant decreases in stroke volume were limiting the maximal cardiac output because of the acute left ventricular dysfunction in these patients. Furthermore, they found that the maximal cardiac output paralleled the maximal oxygen consumption with a correlation coefficient of 0.88 ($\dot{Q}$ = 3.45 + 5.90 VO2). The lower values reported in Table VIII ($\dot{Q}$ = 9.79 + 0.25 VO2, r = 0.52) for the experimental group and ($\dot{Q}$ = 5.50 + 0.41 VO2, r = 0.63) for the reference group could be explained by the parameters

assessed at submaximal loads, the different method, number of measure-- ments and subjects used.

The primary reason for double determinations was to obtain reliable results with the carbon dioxide rebreathing method at submaximal work capacity. As shown in Table X, the percentage of error values tend to decrease with the increase in work intensity. This is in agreement with the findings of Ferguson et al. (73) who reported a better reproducibility of the carbon dioxide method with increasing work load. Repeated testing also gave further information about symptoms and limiting factors.

The significant higher values at the second repetition for heart rate as well as the significant lower values at the second repetition for stroke volume in both the experimental and the reference group, strongly suggest that the period of rest between the two determinations was not sufficient enough to allow a proper recovery for the subjects. This could explain the relatively low correlations between oxygen consumption and cardiac output (Table VIII) or between heart rate and cardiac output (Table IX). This observation could be of significance for these subjects considering their impaired heart condition. Fortunately, no serious complications (such as ventricular fibrillation or myocardial infarction) occurred during these 144 tests in patients with healed myocardial infarction.

SUMMARY AND CONCLUSION

Six healed myocardial infarct patients (mean age, 54 years) were divided in two groups after a random assignment. No significant ($P < .05$) difference was observed between the experimental and the reference group. Physical working capacity improved appreciably over the training period.

The three subjects in the experimental group could exercise to fatigue and performed an average of 80% - 90% of their maximal heart rate without any cardiac symptoms. Their maximal oxygen consumption showed a substantial increase of 30.9% (from 29.57 to 38.71 ml/kg/min) which was not seen to the same extent in the reference group who became exhausted earlier during the tests despite their prescribed daily activity. With these subjects having no angina pectoris during effort, the main deterioration of the physical work capacity could be ascribed to the relative coronary insufficiency and not to the impaired myocardium.

The exercise testing and the small experimental group made it possible to individualize the training intensity according to the goal and to the patient's tolerance. It was suitable to use a certain percentage of the pre-determined maximal heart rate to evaluate the subject's loads.

The heart rates at the given submaximal work intensities were significantly ($P < .05$) lower for the two groups following the three month training program. No significant changes following training were found

for the oxygen consumption relative to body weight and cardiac output at submaximal work loads. As a result of a decreased heart rate and unchanged cardiac output, stroke volume increased significantly for both groups.

Following training, maximal work capacity and maximal oxygen consumption relative to body weight showed substantial improvement in the experimental group whereas the reference group demonstrated minor changes. Finally, no significant changes were found with training in any of the groups for heart rate at maximal work capacity.

CONCLUSION

Since the two groups are not significantly different for the parameters assessed at submaximal work level, it seems probable that the total amount of work performed by the reference group was high enough to elicit beneficial effects. The main difference for the two groups would be at maximal work intensity where the experimental group performed more intense work after the training period.

Exercise training does not seem to elicit cardiac output changes as measured by the carbon dioxide rebreathing method after a three month training program but seems to decrease appreciably the submaximal heart rate at the same given work load. As the result of an unchanged cardiac output and a decreased heart rate, stroke output increased favorably. This not necessarily imply an increased myocardial contractility but may be explained by a muscular peripheral adaptation and better oxygen uti-

lization or a lower sympathetic drive to the sino atrial node. Nevertheless, these hemodynamic effects of exercise training reduced favorably the cardiac work resulting in a more economical energy expenditure specially for these patients suffering of such a hidden impaired condition.

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APPENDICES

APPENDIX 1

DATA SHEET

APPENDIX I Data sheet

Name ..... Date ...../... Work load .....

(1) B. Pressure	=	(2) Room temp.	.....
(3) Factor (STPD)	=	(4) Total vol. Insp.	.....
(5) (4) ..... x 1.02	=	(6) .7904 x (5).../.80	= .....
(7) % CO_2 in exp. air	=	(8) Minute volume	.....
(9) % O_2 in exp. air	=	(10) Vol. corrected	.....
(11) % CO_2 end-tidal	=	(12) $\dot{V}O_2$	= .....
(13) % CO_2 rebreathing	=	(14) RQ	.....

(15) $\dot{V}CO_2$ = (7) ... - 0.03 x (10) .../100 .....

(16) $P\bar{v}_{CO_2}$ = %CO_2 reb. x (BP - P_{H_2O} Torr/100) = ...

(17) Downstream correction factor: (0.24 x $P\bar{v}_{CO_2}$) - 11 = ...

(18) True P_{CO_2} (16) ... - (17) ... = ...

(19) $C\bar{v}_{CO_2}$ - (conversion in the table) = ...

(20) P_{aCO_2} - (11) ... x (BP - P_{H_2O} Torr/100) = ...

(21) C_{aCO_2} = (conversion in the table) = ...

(22) Q (15) ... / (19) ... - (21) ... (l/min) = ...

(23) Heart rate (beats/min) = ...

(24) Stroke volume (ml/beat) = ...

(25) Body weight (kg) .....

APPENDIX 2

MULTISTAGE STRESS TEST PROTOCOL

APPENDIX 2 Multistage stress test protocol

Stages	Speed (mph)	Elevation (%)	Time (min.)
0	1.7	5	0-3
1	1.7	10	3-6
2	2.5	12	6-9
3	3.4	14	9-12
4	4.2	16	12-15

APPENDIX 3

EXPERIMENTAL DESIGN

APPENDIX 3 Experimental design

			MONTHS							
			0		1		2		3	
Group	Work load	S	R1	R2	R1	R2	R1	R2	R1	R2
	Warm-up	1	.	.	.	.	.	.	.	.
		2	.	.	.	.	.	.	.	.
		3	.	.	.	.	.	.	.	.
EXP	70% of Max HR	1	.	.	.	.	.	.	.	.
		2	.	.	.	.	.	.	.	.
		3	.	.	.	.	.	.	.	.
	90% of Max HR	1	.	.	.	.	.	.	.	.
		2	.	.	.	.	.	.	.	.
		3	.	.	.	.	.	.	.	.
	Warm-up	4	.	.	.	.	.	.	.	.
		5	.	.	.	.	.	.	.	.
		6	.	.	.	.	.	.	.	.
REF	70% of Max HR	4	.	.	.	.	.	.	.	.
		5	.	.	.	.	.	.	.	.
		6	.	.	.	.	.	.	.	.
	90% of Max HR	4	.	.	.	.	.	.	.	.
		5	.	.	.	.	.	.	.	.
		6	.	.	.	.	.	.	.	.

EXP: Experimental; REF: Reference; Max HR: Maximal heart rate; S: Subjects; R: Repetitions.

APPENDIX 4

RAW SCORES AT SUBMAXIMAL WORK LOADS FOR THE DEPENDENT VARIABLES

APPENDIX 4 Raw scores at submaximal work loads for the dependent variables

APPENDIX 4-1 Raw data for OXYGEN CONSUMPTION relative to body weight in milliliters per kilogram per minute

WARM-UP LEVEL		MONTHS							
		0		1		2		3	
Group	S	R1	R2	R1	R2	R1	R2	R1	R2
EXP	1	10.67	10.67	9.50	8.40	9.68	8.90	10.67	10.10
	2	15.65	11.99	13.32	13.46	14.93	13.47	14.40	14.95
	3	11.95	13.01	7.86	14.51	12.95	13.47	10.83	10.70
REF	1	12.01	13.89	26.24	15.60	10.92	12.46	14.46	16.99
	2	11.22	12.64	8.06	8.94	10.24	10.24	11.94	12.69
	3	11.78	12.04	10.27	9.60	11.29	12.90	11.98	13.09
% OF MAXIMAL HEART RATE									
EXP	1	18.59	19.03	15.03	16.57	18.24	18.58	19.18	18.96
	2	21.38	22.16	22.82	28.63	26.40	26.40	26.89	25.51
	3	16.20	18.23	11.72	18.91	19.55	18.36	15.72	14.93
REF	1	18.81	17.66	21.56	21.27	17.79	18.63	19.08	19.08
	2	20.92	22.98	15.45	16.11	16.59	18.53	20.15	20.79
	3	23.69	24.24	19.07	22.93	21.10	23.79	25.21	23.97
% OF MAXIMAL HEART RATE									
EXP	1	20.13	21.99	17.02	19.01	23.25	23.14	21.79	21.79
	2	23.86	27.93	30.87	31.00	31.73	31.20	29.22	32.65
	3	20.44	22.71	15.98	23.97	27.08	26.29	22.06	23.65
REF	1	24.51	26.06	25.39	27.23	21.57	24.23	25.63	26.23
	2	23.64	24.23	19.87	19.54	18.86	21.55	23.33	23.45
	3	27.11	27.85	24.93	26.00	24.87	27.42	30.44	29.48

EXP: Experimental; REF: Reference; S: Subject; R: Repetitions

APPENDIX 4-2 Raw data for CARDIAC OUTPUT in liters per minute at submaximal work loads

WARM-UP LEVEL		MONTHS							
		0		1		2		3	
GROUP	S	R1	R2	R1	R2	R1	R2	R1	R2
EXP	1	14.27	10.09	12.59	10.30	10.55	8.87	10.08	9.06
	2	8.93	10.89	9.74	13.23	10.04	9.81	9.66	9.53
	3	10.58	10.87	11.48	11.65	13.72	12.80	11.32	10.13
REF	1	8.97	10.52	10.50	8.42	10.79	8.89	8[illegible]	9.79
	2	9.03	8.34	9.52	8.91	10.83	10.26	11.26	10.42
	3	8.24	8.69	8.67	10.13	10.60	10.64	13.47	12.06
70% OF MAXIMAL HEART RATE									
EXP	1	13.01	15.20	14.96	16.96	15.48	14.74	16.07	16.43
	2	14.54	14.49	15.38	14.65	16.26	15.18	14.78	13.02
	3	18.05	17.58	18.93	20.42	18.17	16.86	16.10	14.59
REF	1	10.76	10.83	12.48	10.12	14.41	13.75	10.90	11.20
	2	13.13	15.88	14.99	15.26	15.82	17.06	14.41	15.34
	3	15.27	16.16	17.89	15.82	20.91	23.89	18.25	17.89
90% OF MAXIMAL HEART RATE									
EXP	1	16.60	18.75	20.09	18.52	18.60	18.71	18.00	18.26
	2	12.22	13.75	14.30	13.58	17.25	17.30	13.96	14.86
	3	18.97	18.19	17.13	14.94	18.07	18.60	15.80	16.32
REF	1	10.87	11.04	11.86	10.98	10.59	11.18	10.17	10.95
	2	15.32	15.43	14.49	14.29	16.13	17.81	13.59	14.57
	3	18.27	18.04	20.81	19.17	19.12	21.62	19.96	20.26

EXP: Experimental; REF: Reference; S: Subject; R: Repetitions.

APPENDIX 4-3 Raw data for HEART RATE in beats per minute at submaximal work loads

WARM-UP LEVEL		MONTHS							
		0		1		2		3	
Group	S	R1	R2	R1	R2	R1	R2	R1	R2
	1	96	97		90	92	96	94	93
EXP	2	95	118	103	102	105	104	108	107
	3	107	118	104	112	111	107	95	96
	1	130	128	124	124	103	110	114	124
REF	2	88	97	82	93	84	99	84	98
	3	84	104	87	99	90	93	90	102
70% OF MAXIMAL HEART RATE									
	1	122	128	125	121	119	117	118	122
EXP	2	142	156	139	140	141	143	138	141
	3	128	140	120	122	117	123	110	118
	1	145	144	140	142	130	134	140	139
REF	2	123	130	117	127	116	129	124	134
	3	134	145	135	135	126	128	131	132
90% OF MAXIMAL HEART RATE									
	1	138	144	136	136	126	127	130	128
EXP	2	160	168	157	156	156	161	157	159
	3	148	152	138	144	134	135	132	132
	1	159	159	160	160	154	158	162	158
REF	2	148	151	140	147	138	151	153	157
	3	158	162	158	155	143	150	154	153

EXP: Experimental; REF: Reference; S: Subject; R: Repetitions.

APPENDIX 4-4 Raw data for STROKE VOLUME in milliliters per beat at submaximal work loads

WARM-UP LEVEL		MONTHS							
		0		1		2		3	
Group	S	R1	R2	R1	R2	R1	R2	R1	R2
	1	148.6	104.0	129.9	144.4	113.6	92.4	107.3	97.4
EXP	2	94.0	92.3	94.6	129.7	95.6	94.3	89.4	89.0
	3	97.9	92.1	110.4	104.0	123.6	119.7	119.2	105.5
	1	69.0	82.2	84.7	67.9	104.7	80.8	76.4	78.9
REF	2	102.6	86.0	116.0	95.8	128.9	103.7	134.1	106.3
	3	98.1	83.6	99.6	102.4	117.7	114.4	149.6	118.0
70% OF MAXIMAL HEART RATE									
	1	123.0	118.8	119.7	140.2	130.1	126.0	136.2	134.7
EXP	2	102.4	93.5	110.6	104.6	115.3	106.2	107.1	92.3
	3	141.1	125.6	157.8	167.4	155.3	137.1	146.4	123.6
	1	74.2	75.2	89.1	71.3	110.8	102.6	77.9	80.6
REF	2	106.8	122.2	128.1	120.1	136.4	132.2	116.2	114.5
	3	132.6	111.5	132.5	117.2	166.3	186.6	139.3	135.5
90% OF MAXIMAL HEART RATE									
	1	120.3	130.2	147.7	136.2	147.7	147.4	138.5	142.7
EXP	2	76.4	81.9	91.1	87.1	110.6	107.4	88.9	93.5
	3	128.2	119.7	124.1	104.1	134.9	137.8	119.7	123.6
	1	68.4	69.4	74.1	68.6	68.8	70.7	62.8	69.3
REF	2	103.5	102.2	103.5	97.2	116.9	118.0	88.8	92.8
	3	115.6	111.4	131.7	123.7	133.7	144.1	129.6	132.4

EXP: Experimental; REF: Reference; S: Subject; R: Repetitions.

APPENDIX 5

STATISTICAL ANALYSIS

LEGEND FOR APPENDIX 5:

G	Group
L	Load
R	Repetition
M	Month
SS	Sum of Squares
DF	Degree of Freedom
MS	Mean Square
F	F ratio

Appendix 5-I, a Four way analysis of variance for OXYGEN CONSUMPTION relative to body weight (milliliters per kilogram per minute) at submaximal work loads

Summary table:

Source of variation	SS	DF	MS	F	Prob
G	6.66	1	6.66	0.034	0.8627
Error	784.43	4	196.11		
L	3797.00	2	1898.50	63.394	0.0000
LG	1.37	2	0.69	0.023	0.9774
Error	293.58	8	29.95		
R	28.98	1	28.98	7.620	0.0508
RG	1.31	1	1.31	0.344	0.5892
Error	15.21	4	3.80		
M	42.69	3	14.23	0.728	0.5547
MG	87.75	3	29.25	1.496	0.2655
Error	234.56	12	19.55		
RM	6.61	3	2.20	0.628	0.6106
RMG	42.51	3	14.17	4.039	0.0337
Error	42.09	12	3.51		
LM	18.94	6	3.16	1.458	0.2346
LMG	30.27	6	5.04	2.330	0.0648
Error	51.96	24	2.17		
LR	13.20	2	6.60	5.433	0.0323
LRG	0.79	2	0.39	0.324	0.7326
Error	9.72	8	1.22		
LRM	21.62	6	3.60	2.043	0.0988
LRMG	13.64	6	2.27	1.289	0.2994
Error	42.32	24	1.76		

Appendix 5-1, b Newman Keuls comparison between ordered means for OXYGEN CONSUMPTION relative to body.weight (ml/kg/min) at different loads

	Load means	L1 12.24	L2 20.18	L3 24.66
L1	12.24	-	7.94**	12.42**
L2	20.18		-	4.68**
L3	24.66			-

* .05
** .01

Critical value	r = 2	r = 3
$Q_{.95}$ (r,8)	2.58	3.19
$Q_{.99}$ (r,8)	3.74	4.45

$$\text{Critical value} = (r,8) \times \sqrt{EMS/N_L}$$

where: EMS = 29.95

N_L = 48

Appendix 5-2, a Four way analysis of variance for CARDIAC OUTPUT (liters per minute) at submaximal work loads

Summary table:

Source of variation	SS	DF	MS	F	Prob
G	41.77	1	41.77	0.402	0.5607
Error	416.00	4	104.00		
L	953.78	2	476.89	22.05	0.0006
LG	3.54	2	1.77	0.08	0.9223
Error	172.99	8	21.62		
R	0.27	1	0.27	0.289	0.6195
RG	0.75	1	0.75	0.796	0.4227
Error	3.75	4	0.94		
M	40.55	3	13.52	5.131	0.0164
MG	21.02	3	7.01	2.660	0.0956
Error	31.62	12	2.63		
RM	2.11	3	0.70	0.876	0.4807
RMG	5.21	3	1.74	2.166	0.1451
Error	9.62	12	0.80		
LM	11.38	6	1.90	0.818	0.5670
LMG	26.44	6	4.41	1.900	0.1222
Error	55.67	24	2.32		
LR	2.07	2	1.03	0.490	0.6296
LRG	0.99	2	0.50	0.024	0.9767
Error	16.85	8	2.11		
LRM	8.07	6	1.34	2.123	0.0878
LRMG	6.84	6	1.14	1.801	0.1415
Error	15.20	24	0.63		

Appendix 5-2, b Newman Keuls comparison between ordered means for CARDIAC OUTPUT (l/min) at different months

	Month	M0	M4	M2	M3
	means	13.51	13.64	13.98	14.87
M0	13.51		0.13	0.47	1.36*
M4	13.64		-	0.34	1.23*
M2	13.98			-	0.89*
M3	14.87				-

* .05
** .01

Critical value	r = 2	r = 3	r = 4
$Q_{.95}$ (r,12)	0.83	1.02	1.13
$Q_{.99}$ (r,12)	1.17	1.36	1.49

Critical value = $(r,12) \times \sqrt{EMS/N_M}$

where: EMS = 2.63

N_M = 36

Appendix 5-2, c Newman Keuls comparison between ordered means for CARDIAC OUTPUT (l/min) at different loads

	Load means	L1 10.37	L2 15.60	L3 16.03
L1	10.37	-	5.23**	5.66**
L2	15.60		-	0.43
L3	16.03			-

* .05
** .01

Critical value	r = 2	r = 3
$Q_{.95}$ (r,8)	2.19	2.71
$Q_{.99}$ (r,8)	3.18	3.78

$$\text{Critical value} = (r,8) \times \sqrt{EMS/N_L}$$

where: EMS = 21.62

N_L = 48

Appendix 5-3, a Four way analysis of variance for HEART RATE (beats per minute) at submaximal work loa

Summary table:

Source of variation	SS	DF	MS	F	Prob
G	650.25	1	650.25	0.301	0.6123
Error	8632.00	4	2158.00		
L	54172.0	2	27086.0	83.957	0.0000
LG	670.54	2	335.27	1.039	0.3970
Error	2580.90	8	322.62		
R	684.69	1	684.69	10.206	0.0331
RG	58.78	1	58.78	0.876	0.4023
Error	268.36	4	67.09		
M	1301.10	3	433.68	6.950	0.0058
MG	383.47	3	127.82	2.048	0.1608
Error	748.80	12	62.40		
RM	140.47	3	46.82	2.719	0.0911
RMG	125.39	3	41.80	2.427	0.1160
Error	206.64	12	17.22		
LM	67.99	6	11.33	0.582	0.7408
LMG	105.74	6	17.62	0.906	0.5072
Error	466.94	24	19.46		
LR	62.35	2	31.17	5.084	0.0376
LRG	79.26	2	39.63	6.463	0.0214
Error	49.06	8	6.13		
LRM	61.49	6	10.25	1.367	0.2679
LRMG	49.57	6	8.26	1.102	0.3900
Error	179.94	24	7.50		

Appendix 5-3, b Newman Keuls comparison between ordered means for HEART RATE (beats/min) at different months

	Month means	M2 123.61	M3 125.75	M1 126.94	M0 131.81
M2	123.61	-	2.14	3.33	8.20**
M3	125.75		-	1.19	6.06*
M1	126.94			-	4.87*
M0	131.81				-

* .05
** .01

Critical value	r = 2	r = 3	r = 4
$Q_{.95}$ (r,12)	4.05	4.98	5.54
$Q_{.99}$ (r,12)	5.70	6.65	7.26

$$\text{Critical value} = (r,12) \times \sqrt{EMS/N_M}$$

where: EMS = 62.40

N_M = 36

Appendix 5-3, c **Newman Keuls comparison between ordered means for HEART RATE (beats/min) at different loads**

Load	means	L1 101.69	L2 130.60	L3 148.79
L1	101.69	-	28.91**	47.10**
L2	130.60		-	18.19**
L3	148.79			-

* .05
** .01

Critical value	r = 2	r = 3
$Q_{.95}$ (r,8)	8.44	10.46
$Q_{.99}$ (r,8)	12.28	14.58

$$\text{Critical value} = (r,8) \times \sqrt{EMS/N_L}$$

where: EMS = 322.62

N_L = 48

Appendix 5-4, a Four way analysis of variance for STROKE VOLUME (milliliters per beat) at submaximal work loads

Summary table

Source variat	SS	DF	MS	F	[redacted]b
G	4791.60	1	4791.60	0.459	0.5354
Error	41789.0	4	10447.0		
I	7584.30	2	3792.10	5.055	0.0381
IG	921.48	2	460.74	0.614	0.5648
Error	6001.80	8	750.23		
R	985.54	1	985.54	15.935	0.0162
RG	3.72	1	3.72	0.060	0.8182
Error	247.39	4	61.85		
M	5542.80	3	1846.60	12.092	0.0006
MG	1372.90	3	457.63	2.996	0.0731
Error	1833.60	12	152.80		
RM	2.81	3	0.94	0.012	0.9981
RMG	361.28	3	120.43	1.557	0.2508
Error	927.89	12	77.32		
IM	889.46	6	148.24	0.910	0.5042
IMG	2503.00	6	417.17	2.562	0.0463
Error	3908.20	24	162.84		
IR	566.36	2	283.18	1.816	0.2237
IRG	150.34	2	75.17	0.482	0.6343
Error	1247.40	8	155.93		
IRM	577.24	6	96.21	2.128	0.0872
IRMG	641.78	6	106.96	2.366	0.0615
Error	1084.90	24	45.21		

Appendix 5-4, b Newman Keuls comparison between ordered means for STROKE VOLUME (ml/beat) at different months

	Month means	M0 102.89	M3 109.94	M1 110.92	M2 120.33
M0	102.89	-	7.05*	8.03*	17.45**
M3	109.94		-	0.98	10.39**
M1	110.92			-	9.41**
M2	120.33				-

* .05
** .01

Critical value	r = 2	r = 3	r = 4
$Q_{.95}$ (r,12)	5.50	6.71	7.48
$Q_{.99}$ (r,12)	7.69	8.97	9.79

$$\text{Critical value} = (r,12) \times \sqrt{EMS/N_M}$$

where: EMS = 152.80

N_M = 36

Appendix 5-4, c Newman Keuls comparison between ordered means for STROKE VOLUME (ml/beat) at different loads

	Load means	L1 103.25	L3 109.10	L2 120.72
L1	103.25	-	5.85	17.47*
L3	109.10		-	11.62
L2	120.72			-

* .05
** .01

Critical value	r = 2	r = 3
$Q_{.95}$ (r,8)	12.89	15.96
$Q_{.99}$ (r,8)	18.72	22.24

$$\text{Critical value} = (r,8) \times \sqrt{EMS/N_L}$$

where: EMS = 750.23

N_L = 48