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CIRCULATORY ADJUSTMENTS OF FEMALES TO

INTERVAL TRAINING AND DETRAINING

DISSERTATION

**Presented in Partial Fulfillment of the Requirements for
the Degree Doctor of Philosophy in the Graduate
School of The Ohio State University**

By

Carol Jean Stevens B.S., M.A.

The Ohio State University

1977

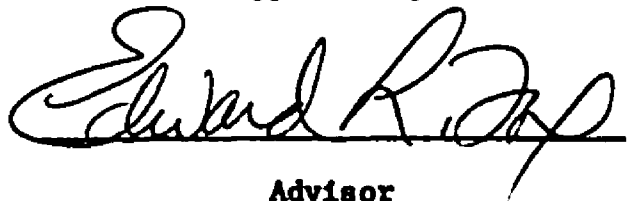
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Chapter I

INTRODUCTION

Metabolic and cardio-respiratory adaptations to physical training have been established and documented. There is agreement that the most noted adaptations with training is an increase in aerobic power. By increasing aerobic power one is able to perform greater amounts of work than before training. It has also been shown that following training submaximal work is performed more efficiently. In other words, the work is performed a little easier. This training response is important not only to the athlete, but to all individuals for most of the work performed during daily living is performed at submaximal level. Despite the importance of this information, few data are available concerning the retention of these physiological variables following training. Answers are needed to resolve some questions of practical significance concerning the type of maintenance/detraining program necessary to retain high levels of cardio-respiratory fitness.

Much of the data obtained on training and detraining have dealt primarily with the male organism. Relatively few data are available concerning the females physiological response. Today as more and more women take advantage of opportunities to participate in physically demanding activities, exercise physiologists must be

prepared to assess, and evaluate the capacity of the female to perform submaximal and maximal work.

With these thoughts in mind, the present study was designed to determine 1) the effects of an interval training program on one's ability to perform submaximal work 2) what frequency of detraining program is necessary to retain high levels of fitness.

PURPOSE OF THE STUDY

The purpose of this study was to determine the circulatory adjustments of young females, first, to training and secondly, to three different frequencies of detraining.

DELIMITATIONS OF THE STUDY

1. Eighteen healthy untrained females, students aged 18-21, from the population at The Ohio State University served as subjects.
2. The training period was of eight weeks duration, three days a week.
3. The detraining period was for ten weeks consisting of either two, one, or zero detraining workouts.
4. Training protocol consisted of an equal number of low, medium, and high intensity work bouts.

STATEMENT OF THE PROBLEM

The intention of this study was to determine circulatory adaptations maintained by three frequencies of exercise (twice per

week, once per week, zero per week) subsequent to an eight week interval training program. Following the training program subjects were assigned to one of the following three groups, matched on the basis of their maximal aerobic power ($\max \dot{V}O_2$ ml/kg.-min).

Group I (GI). Six subjects continued training, once per week. Subjects performed exactly 1/3 of the initial training program. The intensity and duration of the training sessions equaled that performed during the final week of training.

Group II (GII). Six subjects continued to train, twice per week. Subjects performed exactly 2/3 of the initial training program. The intensity and duration of the training sessions equaled that performed during the final week of training.

Group III (GIII). Six subjects ceased training completely. Normal activity patterns were resumed.

The following variables were measured before and after training and again during the fifth and tenth weeks of detraining:

1. Submaximal cardiac output
2. Submaximal $\dot{V}O_2$
3. Submaximal heart rate
4. Submaximal stroke volume
5. Submaximal arterial-venous difference.

DEFINITIONS

PaCO_2 : The partial pressure of carbon dioxide in arterial blood. This value is converted to CO_2 content (CaCO_2) in volumes percent by use of the dissociation curve for arterialized blood assuming a normal and constant O_2 content of 97.5 percent.

$\text{P}\bar{\text{VCO}}_2$: The partial pressure of carbon dioxide in mixed venous blood.

CO_2 Rebreathing: A noninvasive method of determining cardiac output.

Interval training program: A system of conditioning or training, consisting of a series of repeated bouts of exercise alternated with periods of relief (48).

Detraining: A reduction in the frequency of chronic stress, characterized by a decreased retention of physiological variables.

Training: An eight week interval program consisting of high, medium, and low intensity work bouts, three days per week.

Chapter II

REVIEW OF LITERATURE

INTRODUCTION

The review of literature has been divided into three sections. First, literature concerning the rationale of CO₂ re-breathing for the assessment of cardiac output will be discussed. Second, a brief summary concerning the submaximal circulatory adjustments of females to training, and third, literature pertaining to the cardio-respiratory response of individuals to detraining programs will be evaluated.

CO₂ REBREATHING ASSESSMENT

The CO₂ rebreathing technique is a simple adaptation of the Fick principle. This principle states that the flow of blood through an organ can be determined by measuring the amount of substance removed by, or added to the organ per minute by dividing the change in concentration of the substance in the blood. Utilizing the Fick equation, if the amount of CO₂ removed from the blood per minute is known along with the concentration differences resulting from this removal, cardiac output can be calculated as follows:

$$Q = \frac{V\text{CO}_2}{\text{C}\bar{\text{V}}\text{CO}_2 - \text{C}_a\text{CO}_2}$$

5

Where \dot{Q} = cardiac output (liters/minute), $\dot{V}\text{CO}_2$ = CO_2 output (liters/minute), $\bar{C}\text{VCO}_2$ = concentration of CO_2 in mixed venous blood (ml/liters) and CaCO_2 = concentration of CO_2 in arterial blood (ml/liters).

Christiansen et al. (12) were the first to introduce the rebreathing method to determine cardiac output. Prior to this technique the direct Fick procedure and Stewart's dye-dilution method (56, 57) were the only techniques available. Both of these procedures require catheterization of veins and arteries, which introduces trauma and anxiety to the subject which may alter physiological functions. A noninvasive technique is advantageous if it can be proven valid and reproducible.

Christiansen, Douglas, and Haldane in 1914 (12) developed a method for determining venous CO_2 pressure which became the basis for modifications by researchers. After a maximal expiration, a maximal breath was inspired from a bag or spirometer containing a mixture of CO_2 and air or CO_2 and O_2 . The breath was held for five seconds, a liter was then expired and an alveolar gas sample taken. After a few more seconds, another liter was expired and an alveolar sample taken. Occasionally, a third sample was taken following another four to five second interval. When the CO_2 pressures were the same or nearly the same in successive samples, alveolar CO_2 pressure was assumed to be in equilibrium with that of mixed venous blood. One criticism of this method was that there was inadequate

mixing with only one respiration. Henderson and Prince (34) attempted to overcome some of the criticisms of the method of Christensen et al. by lowering the breath holding time thereby reducing the chance of recirculated blood affecting the results.

The procedures described thus far have involved breath holding. Laurens (42) compared the rise of CO_2 in the lungs when the breath was held with the rise when air was rebreathed without breath holding. The rate of increase was faster and the final value higher with rebreathing. Laurens' work represents the first use of a rebreathing technique in which the breath was not held.

One of the assumptions underlying all CO_2 methods is that virtually no pressure gradient exists between alveolar and arterial CO_2 . Suskind et al. (59) concluded that no significant PCO_2 gradient exists during exercise. At rest, the mean PCO_2 gradient was 0.2 ± 1.27 mm Hg. West (64) found differences in ventilation/perfusion ratios in different parts of the lung. The overall effect, he concluded was a pressure gradient of 1 mm Hg between arterial and alveolar CO_2 .

The method for $\text{P}\bar{\text{V}}\text{CO}_2$ determination in this study is based on the theory that the PCO_2 in breath by breath samples of rebreathed air increases exponentially. Dubois, Britt, and Fenn (22), using a rapid infrared analyzer, plotted PCO_2 against time for the first twenty seconds of breath holding. Their procedure involved breath holding, after a normal expiration for periods of five, ten,

fifteen and twenty seconds. Following each period, the breath was expired and samples collected and analyzed. The PCO_2 values of these samples followed a logarithmic curve whose asymptote was oxygenated venous PCO_2 . Defares (15, 16, 17) showed that the CO_2 time course during rebreathing involved two exponential terms, one of which becomes negligible after the first respiration. Defare's work was the beginning of a new approach to determining $\overline{P\dot{V}CO_2}$. Previous methods, as well as some more recent ones, require equilibration of alveolar and mixed venous PCO_2 . Defare's (17) method does not require equilibration. He discovered that the point where two successive breaths would yield the same PCO_2 values (PCO_2 of oxygenated venous blood) was determined with the formula

$$P = \frac{a}{1 - b}.$$

Where $P = PCO_2$ at equilibrium, $a =$ slope, and $b = y$ intercept.

Jernerus, Lundin and Thomson (35) used a modification of this method. To determine $\overline{P\dot{V}CO_2}$, a P vs $P_n + 1$ plot of breath by breath CO_2 concentration was made. A second line was drawn, which was equidistant at all points from the ordinate and abscissa. This line represented all points where the CO_2 concentrations in succeeding breaths were the same. By drawing a straight line through the plotted points and extrapolating to intersect the second line, the equilibrium point between CO_2 pressure in alveolar air and in oxygenated mixed venous blood was determined. The PCO_2 value was

determined by multiplying the CO_2 concentration times barometric pressure (-47 mmHg). The concentration of CO_2 in blood was obtained from a standard CO_2 dissociation curve. Cardiac outputs were determined at rest and during exercise.

Klausen (41) further modified the method of Jernerus, Lundin and Thomson (35). In determining $\overline{\text{P}\text{VCO}_2}$, he plotted CO_2 concentrations of the breath by breath samples against time. A best fitting curve was applied to these points. CO_2 concentrations falling on this line were taken at equal time intervals and converted to their respective CO_2 pressures (PCO_2). A second modification was the use of the Bohr formula for determining PaCO_2 . The use of the Bohr formula for determining PaCO_2 required an estimate of subjects physiological dead space. Assussen and Nielsen (2) found that the average physiological dead space of their subjects varied from 170 ml at rest to 350 ml during work at a tidal volume of 3.3 liters. Their data have been used in investigations using the Bohr formula. If tidal volume is large, according to these authors even a considerable error in dead space estimation will have little effect on PaCO_2 determination.

Ferguson et al. (28) obtained good reproducibility in exercise cardiac output determinations using the Bohr formula and a dead space estimate from Assussen and Nielsen's data (2,3) for estimating PaCO_2 . $\overline{\text{P}\text{VCO}_2}$ was estimated by the method of Defares (15) using the continuous sampling procedure of Jernerus, Lundin and

Thomson (35) and the graphical analysis described by Klausen (41). Studies by Jernerus (35), Klausen (41), Asmussen and Nielsen (2, 3), Mufesan (50), and Magel and Anderson (46, 47), have shown that the rebreathing technique is a valid and reproducible method for the determination of cardiac output.

SUBMAXIMAL CIRCULATORY ADJUSTMENTS OF FEMALES TO TRAINING

Studies have shown that training may result in an increased (3, 23, 32, 60), decreased (1, 44), or unchanged (40, 54) cardiac output during exercise. For example, Kilbom (40) found no significant reductions in \dot{Q} , $\dot{V}O_2$, and $a-\bar{v}O_2$ difference at a given submaximal workload in middle-aged women following training. Saltin and others (54) obtained similar results on male subjects. Other studies (1, 3) have shown that training results in decreased heart rate, with increases in \dot{Q} , $\dot{V}O_2$, and $a-\bar{v}O_2$ difference during exercise. Still others (54) have found that \dot{Q} and $\dot{V}O_2$ also decrease at a given level of submaximal exercise after training.

Van Handel et al. (66) found a reduction in \dot{Q} and $\dot{V}O_2$ at a given submaximal workload, with an increase in $a-\bar{v}O_2$ diff. Clausen and others (13) suggest this may be due to a redistribution of blood flow to both the nonworking areas and the active muscles after training.

Rowell (52) indicated that at any given level of oxygen consumption, the $a-\bar{v}O_2$ diff depends upon the percentage of cardiac output going to the working muscles. That is, if the percentage

perfusing nonworking tissues is large, the $a-\bar{v}O_2$ diff will be small, and vice versa. Several studies have shown that blood flow to the muscle is significantly decreased with training at the same absolute submaximal workload (48, 53). This lower blood flow is compensated by extraction of more oxygen by the working muscles (48).

Cotes and Meade (14) suggested that the bradycardia seen following training may be ascribed to increased mechanical efficiency, resulting in a lower oxygen consumption at a given workload.

CARDIORESPIRATORY RESPONSES OF INDIVIDUALS TO DETRAINING PROGRAMS

Literature concerning the effects of detraining on the cardio-respiratory system during submaximal work is limited. It seems logical to suspect that the longer a person abstains from chronic exercise following training, the more the decrease in cardio-respiratory fitness.

Interest in maintenance of fitness or detraining resulted from clinical experience in hospitals with soldiers during World War II. The first investigators studied total bed rest in relation to detraining effects. They found significant decreases in maximal $\dot{V}O_2$, blood volume, hemoglobin, and hematocrit, in as little as fourteen days of confinement. No change was found in resting cardiac output.

In 1972, Drinkwater and Horvath published (20) one of the few studies dealing with detraining effects on women. They observed max $\dot{V}O_2$, max \dot{V}_E and oxygen pulse after completing a track season and

three months thereafter. Seven girls ages 15-17 showed a decrease from 47.8 ml/kg.-min to 40.4 ml/kg.-min in max $\dot{V}O_2$, 77.5 liters/min to 69.5 liters/min in maximum ventilation, and an oxygen pulse decrease from 12.7 ml/beat to 10.9 ml/beat. A slight increase in maximally heart rate failed to be statistically significant. Blood pressure, hemoglobin, hematocrit, and plasma protein volumes remained the same. During the course of the work, heart rate, ventilatory volumes and oxygen uptake were all significantly higher in the detrained state.

Evert (26) conducted a detraining study on seven female track athletes, through a seven week post season period. She found no significant changes in ventilatory volumes, oxygen uptake or heart rate at a standard workload. The author attributed this lack of change to the short post-training period and the relatively high activity level of the girls even when they were not actively training.

Stoner (58) reported that in a time period of two weeks subsequent to a four week training period, the only change his middle-aged subjects demonstrated after two weeks of detraining was a reduction in cardiovascular efficiency.

In 1974, Fringer and Stull (31) re-examined the time course of detraining immediately following the ten week program of two weekly maximal bicycle rides. Increases in maximal oxygen consumption,

maximal ventilation, heart rate, and total work were noted at the conclusion of the training program. Twenty-two subjects retested after five weeks of detraining displayed a 35% retention of $\max \dot{V}O_2$, 14% retention of $\max \dot{V}_E$, 59% retention of total work capacity and an increased resting heart rate. An equal number of subjects were retested ten weeks post-training and exhibited a 43% retention of $\max \dot{V}_E$, with an increased resting heart rate. These data suggest the loss of training effects as reflected by the decreases in various physiological variables.

Chaloupka (11) investigated the physiological response of eleven male subjects to two frequencies of maintenance training. An interval training program of three weekly sessions for eight weeks elicited increases in $\max \dot{V}O_2$ and $\max \dot{V}_E$. Maintenance programs of once per week or once every two weeks produced reductions in measured parameters. Maintenance evaluation at six, twelve, and sixteen weeks post-training supplied valuable information concerning the time course of retention. At six weeks the two times per week maintenance program returned to pre-training values, however, the weekly group required sixteen weeks to return all parameters to initial levels.

Kendrick (36) in 1971 evaluated twenty-one sedentary middle-aged males. Training consisted of walking and jogging, twice per week. All subjects showed increased cardio-respiratory efficiency as reflected by heart rate and blood pressure measurements. Maintenance protocol involved three groups; Group A trained less than one mile weekly, Group B trained between two and five miles weekly, and

Group C covered more than six miles weekly, for a total of twelve weeks. Re-evaluation at the conclusion of the maintenance program displayed a 50% retention of initial gains by both groups A and B, while group C showed an increase in cardio-respiratory efficiency.

Michael et al. (49) examined the physiological changes of ten teenage girls from a high school track team during five months of detraining. Utilizing a three minute step test and a treadmill run, detraining tests were administered at one, three, five, seven, and twenty-three weeks post-training to insure time course plots of detraining. The heart rate response to the work increased significantly by the third week of detraining. The heart rate response at a particular work load was increased during the detraining period. The absolute $\dot{V}O_2$ did not change with any work load.

Lewis (45) studied seven subjects during thirteen weeks of training and twenty-two weeks of detraining. Subjects were tested at light, moderate, and heavy work loads on a bicycle ergometer. Measurements for $\dot{V}O_2$, \dot{V}_E , exercise heart rate, and recovery heart rate were obtained. These data indicated no change in the submaximal $\dot{V}O_2$ following training except for an increase at the heaviest work load indicating a reduced capacity to perform at near maximal conditions. Exercise heart rates were reduced during all work loads at the conclusion of training. By thirteen weeks of detraining the heart rates were equal to pre-testing values.

In 1969, Fardy (27) obtained measurements on eleven college soccer players during ten weeks of soccer training and five weeks of detraining. Significant increases in $\max \dot{V}O_2$, $\max \dot{V}_E$ and work time were found at both five and ten weeks of training. Heart rate changes in detraining paralleled miles/weeks of training. At the conclusion of five weeks of detraining all of the increases had returned back to pretraining values.

Williams and Edwards (65) in 1971 evaluated several groups participating in daily training sessions for five weeks followed by four weeks of detraining. Performances in The Ohio State University Step Test and twelve minute run time disclosed significant increases in all groups during the post training evaluation. Retesting at the conclusion of detraining revealed a significant decrease from post-training values, yet retention was noted in relation to pre-testing performances. A time course for maintaining physiological parameters appears to be indicated by these preceding data.

Case (10) examined nine male subjects during seven weeks of training and twelve weeks of detraining to determine the effects of training frequency upon retention of training effects. $\max \dot{V}O_2$ and physical work capacity displayed increases, while exercise heart rate, recovery heart rate, and one mile run time decreases significantly in the post training evaluation, regardless of two or four day per week training sessions. A significant decrease in maximal oxygen consumption by eight-weeks and a complete return to pre-

training values by the conclusion of detraining were found. A much more rapid return of exercise and recovery heart rate to the pre-training level occurred at four weeks detraining. The only parameter which failed to exhibit a detraining effect was the mile performance time. Case's study showed a longer detraining retention than the preceding authors which may be attributed in part to the longer duration of the training program.

It is evident that relatively few data are available concerning the detraining response of males and females. Even less information exists concerning the time course of important cardio-respiratory variables such as heart rate, cardiac output, stroke volume, and the $a-\bar{v}O_2$ difference.

Chapter III

METHODS AND PROCEDURES

SUBJECTS

Eighteen untrained female subjects, ages 18-21, at The Ohio State University volunteered as subjects. A thorough screening was performed to eliminate any subjects who were extremely physically active or suffering from ill-health. Prior to the commencement of the study, each student was required to complete a medical examination conducted by University Health Service of The Ohio State University. Physical characteristics are presented in Table 1.

ORIENTATION

All testing was performed in the Laboratory of Work Physiology. In order to familiarize each subject with the equipment used for testing, an orientation program was devised. This program consisted of a minimum of two sessions in the laboratory and involved practice of the rebreathing technique as well as practicing walking on the treadmill. At the commencement of the pre-testing period the subjects were at ease with the rebreathing apparatus and familiar with the procedures and equipment involved in the tests.

Table 1
PHYSICAL CHARACTERISTICS

SUBJECTS	AGE YEAR	HEIGHT CM.	WEIGHT KG.
1	18	172.7	64.2
2	18	172.7	68.1
3	18	168.9	56.8
4	18	158.8	55.3
5	20	167.6	64.7
6	18	141.0	55.3
7	19	143.5	69.8
8	21	175.3	66.5
9	20	177.8	62.5
10	19	166.4	55.4
11	18	158.8	50.1
12	18	167.6	59.5
13	19	172.7	56.8
14	19	175.9	63.8
15	19	160.0	49.9
16	18	154.3	54.3
17	20	167.6	62.3
18	18	171.5	66.1
\bar{X}	18.8	165.2	60.1
$\pm SD$	0.9	10.6	6.1

CONTROL GROUP

Eight untrained subjects served as a control group. This group was evaluated during a previous study by Lesmes, et al. (43). The authors revealed no significant change in the measured parameters during an eight week interim. In an earlier study performed at our laboratory, Lesmes et al. (44) demonstrated reliability of the CO₂ rebreathing procedure to be $r = 0.86$. Consequently attributing any measured changes to training and detraining seems justifiable.

EXPERIMENTAL DESIGN

Eighteen subjects participated in a two phase program. Phase one consisted of running in an interval training program, three days per week for eight weeks. Upon completion of the training program subjects were assigned to one of three detraining groups, matched on the basis of their maximal oxygen consumption (ml/kg.-min). Detraining or phase II continued for ten weeks.

Experimental Groups.

	Number of Subjects	Frequency of Training	Amount of Work done
Group I	6	1 day per week	1/3 of initial training
Group II	6	2 days per week	2/3 of initial training
Group III	6	0 days per week	normal activity, no training

A submaximal work performance test was performed on each subject before and after training, and during the fifth and tenth week of detraining. The following physiological variables were assessed.

1. Submaximal oxygen consumption expressed as liters per minute
2. Cardiac output expressed as liters per minute
3. Submaximal heart rate.

TRAINING

All training sessions were conducted on a 220 yard (201 m) indoor track at the French Field House, The Ohio State University. Prior to training all subjects were familiarized with the track and training protocol to be utilized throughout the study. The girls ran in shorts, tee-shirts, and a pair of running shoes to help avoid ankle and foot injuries. Warm-up exercises were encouraged prior to running. All running times were recorded throughout the training and detraining programs.

Examples of the typical workouts performed during training and detraining are as follows:

High intensity	6 X 201 meters (1:3)
Rest five (5) minutes	7 X 101 meters (1:3)
Medium intensity	3 X 404 meters (1:2½)
Rest five (5) minutes	3 X 201 meters (1:2½)
Low intensity	3 X 807 meters (1:2)
Rest five (5) minutes	1 X 605 meters

The parenthesis following the workout prescription indicates the work-relief ratio. The complete workout schedule for all forty-four workouts can be found in the Appendix. Interpreting the prescription of a workout is explained in the following example. In the low intensity workouts subjects were required to run 807 meters, three times. Between each repetition each subject was allowed a work-relief period of (1:2). In other words, for every minute of work, two minutes of rest were given. Upon completion of the set (3 X 807 m) subjects were given an additional rest period of five minutes. Following the five minute rest period, one 605 m workout would be performed. Interpretations of the other prescriptions are made in the same manner. One minute following the completion of the entire workout a carotid pulse count was obtained. These values were obtained to monitor the severity of each training session.

EXPERIMENTAL EVALUATIONS

All subjects were evaluated before and after training and during the fifth and tenth weeks of detraining. Re-test evaluations were performed in an attempt to develop a time course for changes in physiological variables.

SUBMAXIMAL OXYGEN CONSUMPTION

Subjects were required to walk on a treadmill 4 mph (0 % grade) for a period of five minutes. During the fourth and fifth minutes an expired gas sample was collected. This sample was

collected 120 liter chain compensated gasometer (Warren E. Collins). A meter stick, attached to the side of the gasometer, indicated the amount of air displacement. Initial and final readings were subtracted and multiplied by the conversion factor of 133.2 cc/mm to obtain gas volumes. The temperature of the air in the gasometer was recorded and used in the gas volume calculations. Two liter rubber aliquot sample bags were used to transport the expired gas to the analyzers. Expired gas samples were analyzed for CO_2 and O_2 with a Beckman LB-2 and OM-11 analyzers, respectively. Reference gases for calibrating the analyzers were verified with the Haldane apparatus. Heart rates were recorded during the last 15 seconds of each one minute period by direct lead electrocardiography.

CARDIAC OUTPUT

Immediately following collection of expired air, the determination of cardiac output was obtained by CO_2 rebreathing. The rebreathing technique used in this study is an adaptation of the method devised by Defares (17). The experimental set-up of the equipment is graphically illustrated in Figure 1.

Between minutes three and four, tidal volume was estimated. This was necessary in fixing the volume of gas placed in the rebreathing bag. The tidal volume plus two liters of 100% oxygen was then placed into the rebreathing bag. During the fourth minute of exercise VO_2 and tidal volume was determined.

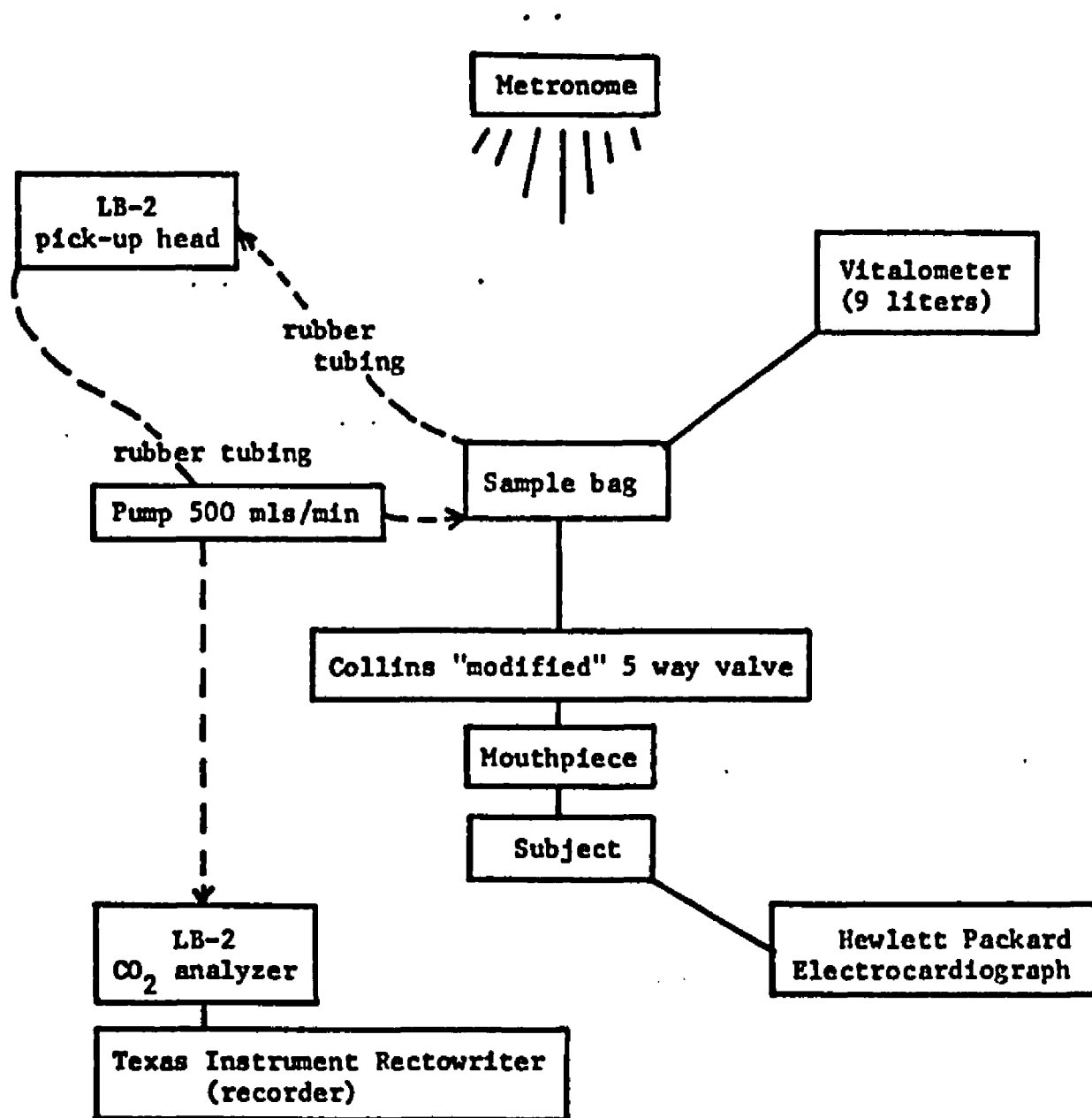


Figure 1

Rebreathing Apparatus

Following the fifth minute of exercise, the subject was instructed to exhale normally. Upon doing so, the Collins modified 5 way valve was immediately turned so that the subject's next inhalation was from the rebreathing bag. The subject then rebreathed at a constant rate of 36 breaths per minute until a plateau was reached on the CO_2 curve, generally five to ten seconds. An example of the calculations necessary to determine cardiac output are contained in the Appendix. CO_2 concentrations were analyzed with a Beckman LB-2 analyzer. Breath by breath analysis of CO_2 was recorded by a Texas Instrument Rectowriter. PaCO_2 was calculated from expired air using the Bohr formula with a dead space estimation from tidal volume according to the data of Asmussen and Nielsen (2).

STATISTICAL EVALUATION

A two factor (3 X 4) model of analysis of variance for repeated observations on the same subject was utilized for comparison of re-test groups and interaction. When significant F ratios were discovered, a Tukey multiple range test was employed to determine the location of statistical significance.

Chapter IV

RESULTS AND ANALYSIS OF THE DATA

The purpose of this study was to determine the circulatory adjustments of college age females to two distinct phases of training. During phase I, cardiovascular adaptations to an eight week interval training program were evaluated. Subsequent to training, a ten week detraining program was implemented to determine the response of the cardio-respiratory system to three different frequencies of detraining.

STATISTICAL EVALUATION

A two factor (3 X 4) model of analysis of variance for repeated observations on the same subject was utilized for comparison of retest groups and interaction. When significant F ratios were discovered, a Tukey multiple range test was employed to determine the location of statistical significance.

All of the following parameters were tested for statistical significance at the 0.05 level of confidence.

- A. Submaximal oxygen consumption (liters/min).
- B. Submaximal oxygen consumption (ml/kg.-min).
- C. Submaximal cardiac output (liters/min).
- D. Submaximal heart rate (bpm).
- E. Submaximal stroke volume (mls).

F. Submaximal $a-\bar{v}O_2$ difference (ml/100 ml).

G. Total body weight (kg).

Mean circulatory changes during training and detraining are shown in Tables 2 and 3.

SUBMAXIMAL OXYGEN CONSUMPTION

No significant change occurred in submaximal oxygen consumption expressed as either liters/min or ml/kg.-min following training and during detraining. These data appear in Figure 2.

HEART RATE

A significant bradycardia occurred following training. Mean heart rate dropped from 147 bpm to 133 bpm, representing a 10.2% decrease. This value remained relatively unchanged throughout the detraining program. It is interesting to note that the mean heart rate during the tenth week of detraining (130 bpm) is significantly lower than the initial pre-training value. Analysis of variance indicated significant differences among mean group values. This difference is evident when comparing the mean heart rate for Group II (129 bpm) with Group I (140 bpm) and Group III (140 bpm). Statistical significance did not occur with interaction, therefore, no differences in heart rate were found among groups or trials. These results are graphically represented in Figure 3.

Table 2
CIRCULATORY CHANGES DURING TRAINING

	PRE			POST		
$\dot{V}O_2$ (liters/min)	1.278 \pm	0.17	GI	1.212 \pm	0.14	
	1.424 \pm	0.28	GII	1.321 \pm	0.08	
	1.333 \pm	0.14	GIII	1.318 \pm	0.22	
Cardiac output (liters/min)	15.56 \pm	3.09	GI	13.94 \pm	0.68	
	13.33 \pm	2.49	GII	11.76 \pm	1.43	
	14.44 \pm	3.24	GIII	11.23 \pm	1.55	
Stroke volume (mls)	105.98 \pm	16.90	GI	86.66 \pm	4.13	
	97.08 \pm	21.23	GII	88.60 \pm	9.58	
	94.54 \pm	20.76	GIII	86.81 \pm	4.26	
Heart rate (bpm)	147.30 \pm	15.0	GI	138.0 \pm	8.50	
	139.00 \pm	18.3	GII	131.5 \pm	11.50	
	153.30 \pm	13.3	GIII	129.2 \pm	16.60	
$a-\bar{v}O_2$ (diff. mls/100 mls)	8.44 \pm	1.83	GI	10.15 \pm	1.20	
	11.12 \pm	3.39	GII	11.41 \pm	2.09	
	9.63 \pm	2.50	GIII	11.76 \pm	1.05	

Values are mean \pm SD

Table 3
CIRCULATORY CHANGES DURING DETRAINING

	Fifth Week			Tenth Week		
$\dot{V}O_2$ (liters/min)	1.144 \pm 0.186	GI		1.192 \pm 0.170		
	1.305 \pm 0.159	GII		1.234 \pm 0.159		
	1.263 \pm 0.172	GIII		1.245 \pm 0.113		
Cardiac output (liters/min)	12.28 \pm 1.230	GI		11.97 \pm 1.230		
	11.08 \pm 0.392	GII		10.71 \pm 0.665		
	11.89 \pm 1.470	GIII		11.54 \pm 0.773		
Stroke volume (mls)	89.20 \pm 6.690	GI		86.48 \pm 6.57		
	89.30 \pm 7.430	GII		90.05 \pm 9.14		
	84.48 \pm 7.840	GIII		88.54 \pm 6.08		
Heart rate (bpm)	138.0 \pm 17.00	GI		139.0 \pm 17.20		
	125.0 \pm 7.68	GII		119.0 \pm 5.53		
	141.0 \pm 17.40	GIII		131.0 \pm 12.70		
a- $\bar{v}O_2$ diff. (mls/100 ml)	9.49 \pm 1.83	GI		11.91 \pm 1.23		
	11.83 \pm 1.79	GII		10.71 \pm 0.66		
	10.67 \pm 1.95	GIII		11.53 \pm 0.77		

Values are mean \pm SD.

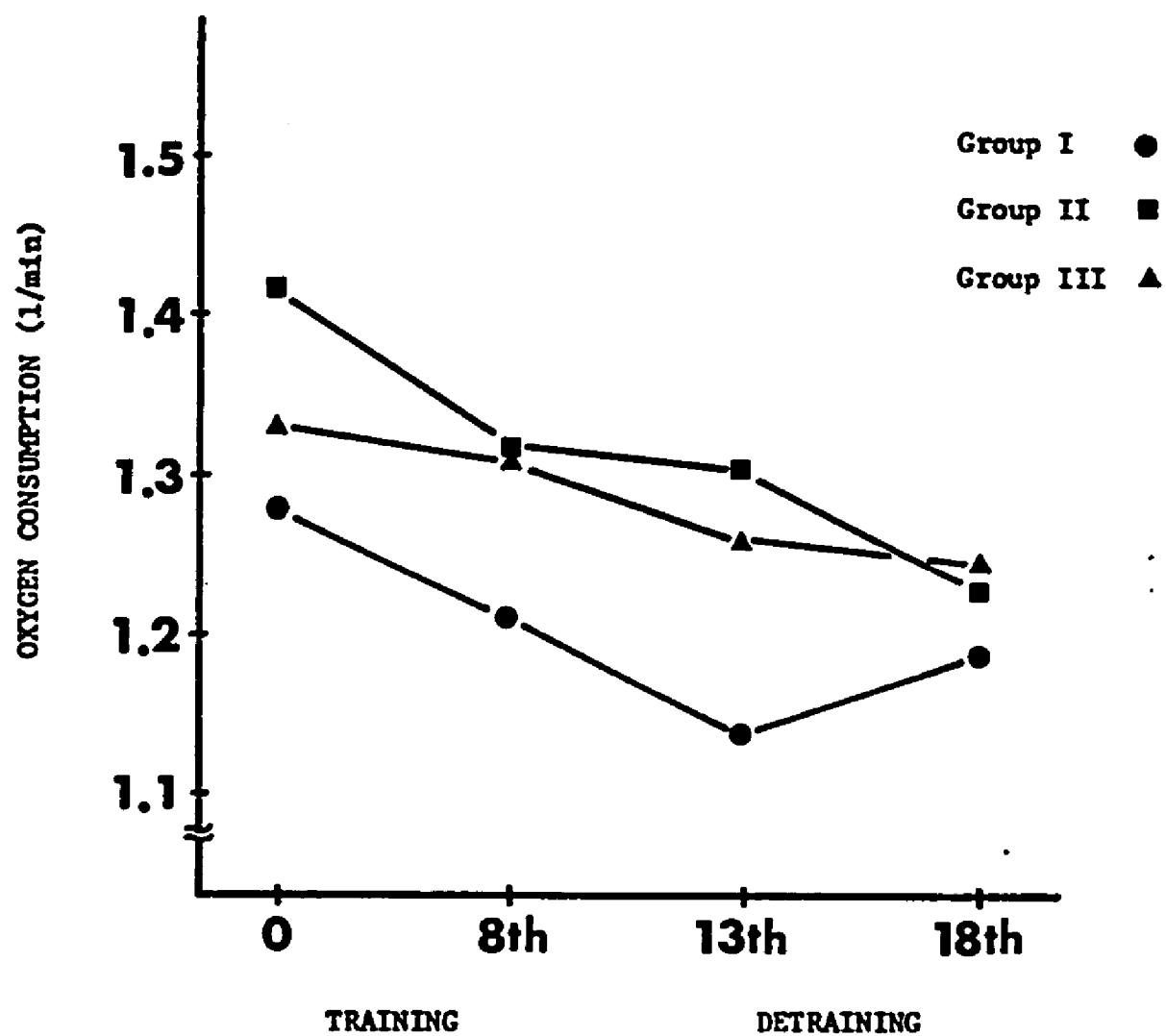


FIGURE 2. SUBMAXIMAL $\dot{V}O_2$, TRAINING AND DETRAINING

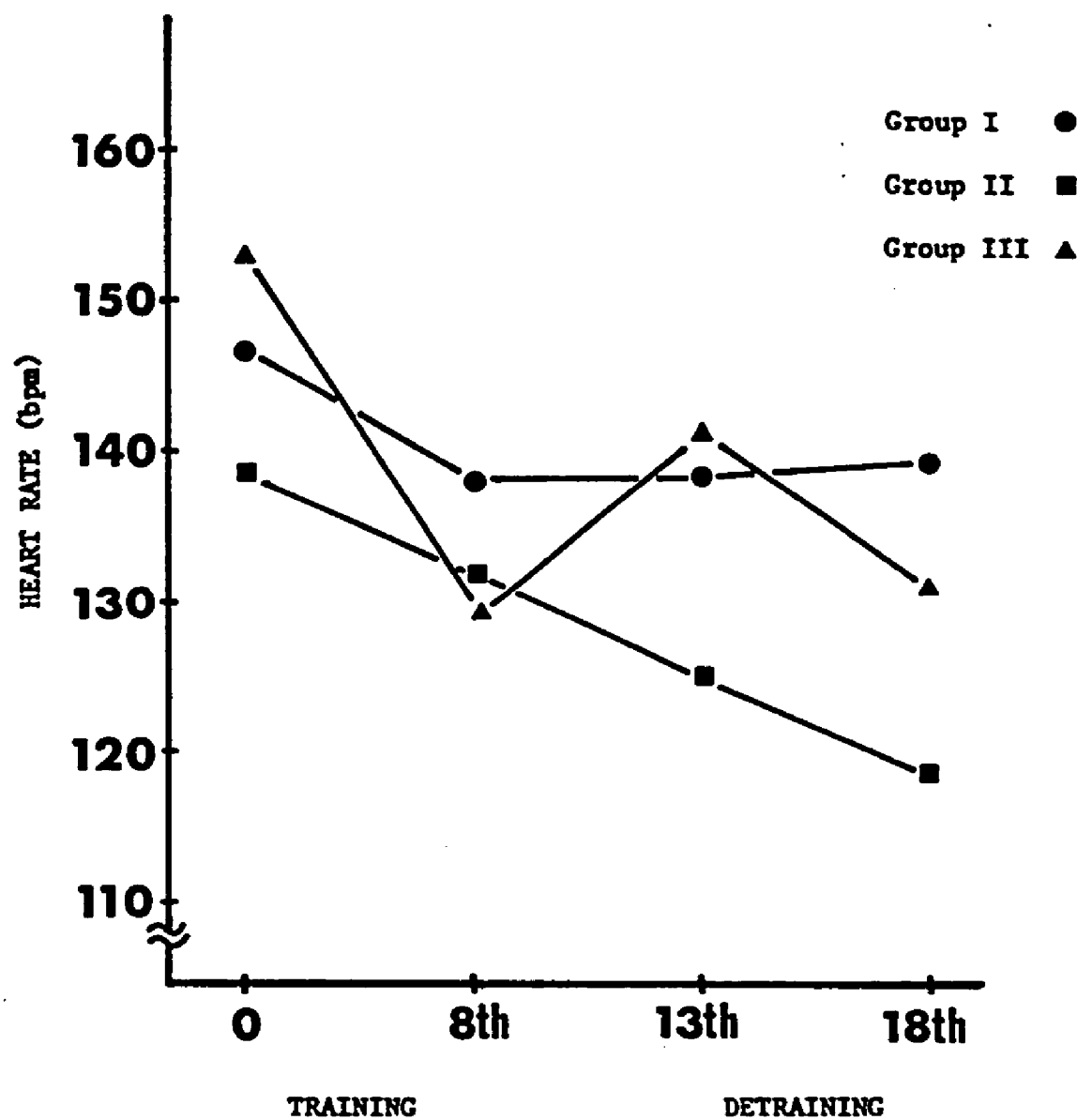


FIGURE 3. SUBMAXIMAL HEART RATE, TRAINING AND DETRAINING

STROKE VOLUME

Training produced a significant decrease in stroke volume. Mean values dropped from 99.2 mls at the beginning of training to 86.8 mls at the end of training. This represents a 12.5% decrease. Although the mean stroke volume is relatively unchanged, throughout the trials of detraining, 87.9 mls (fifth week) and 88.3 mls (tenth week), both are significantly lower when compared to the initial mean pre-training value. Analysis of variance demonstrated no significant differences among groups or interaction. See Figure 4.

CARDIAC OUTPUT

Training produced a significant decrease in stroke volume and heart rate. Therefore, it is not surprising to see a 19.4% decrease in cardiac output following training. Mean cardiac output at pre-training was 14.45 liters/min as compared to 11.65 liters/min post-training. During the fifth and tenth week of detraining the mean cardiac outputs were 11.79 liters/min and 11.40 liters/min respectively. These values are significantly lower than the initial pre-training value of 14.45 liters/min. Analysis of variance demonstrated no statistical significance among groups or interaction. These results are graphically represented in Figure 5.

ARTERIAL - VENOUS OXYGEN DIFFERENCE

Analysis of variance for this variable indicated no statistical differences in either trials or interaction. Although

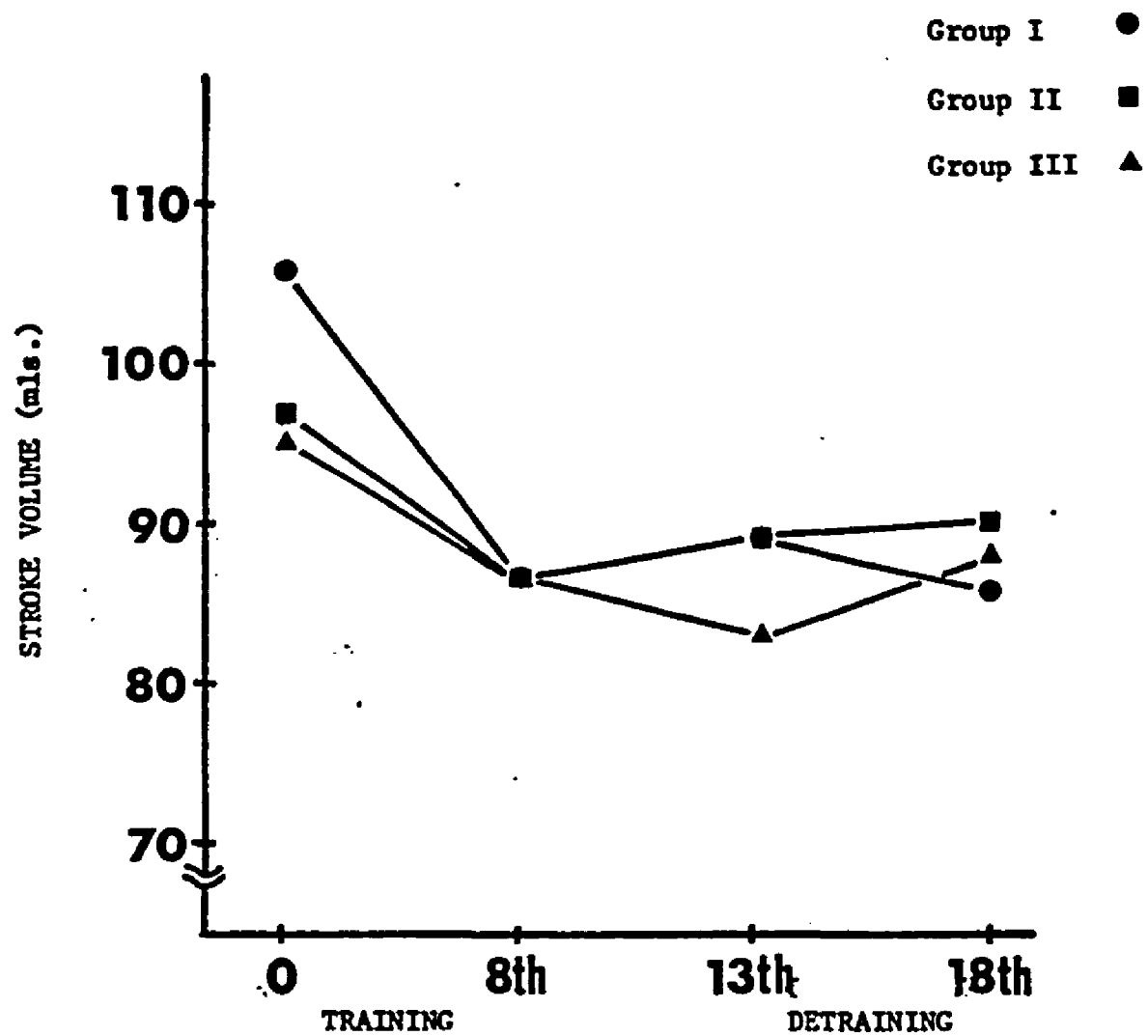


FIGURE 4. SUBMAXIMAL STROKE VOLUME, TRAINING AND DETRAINING

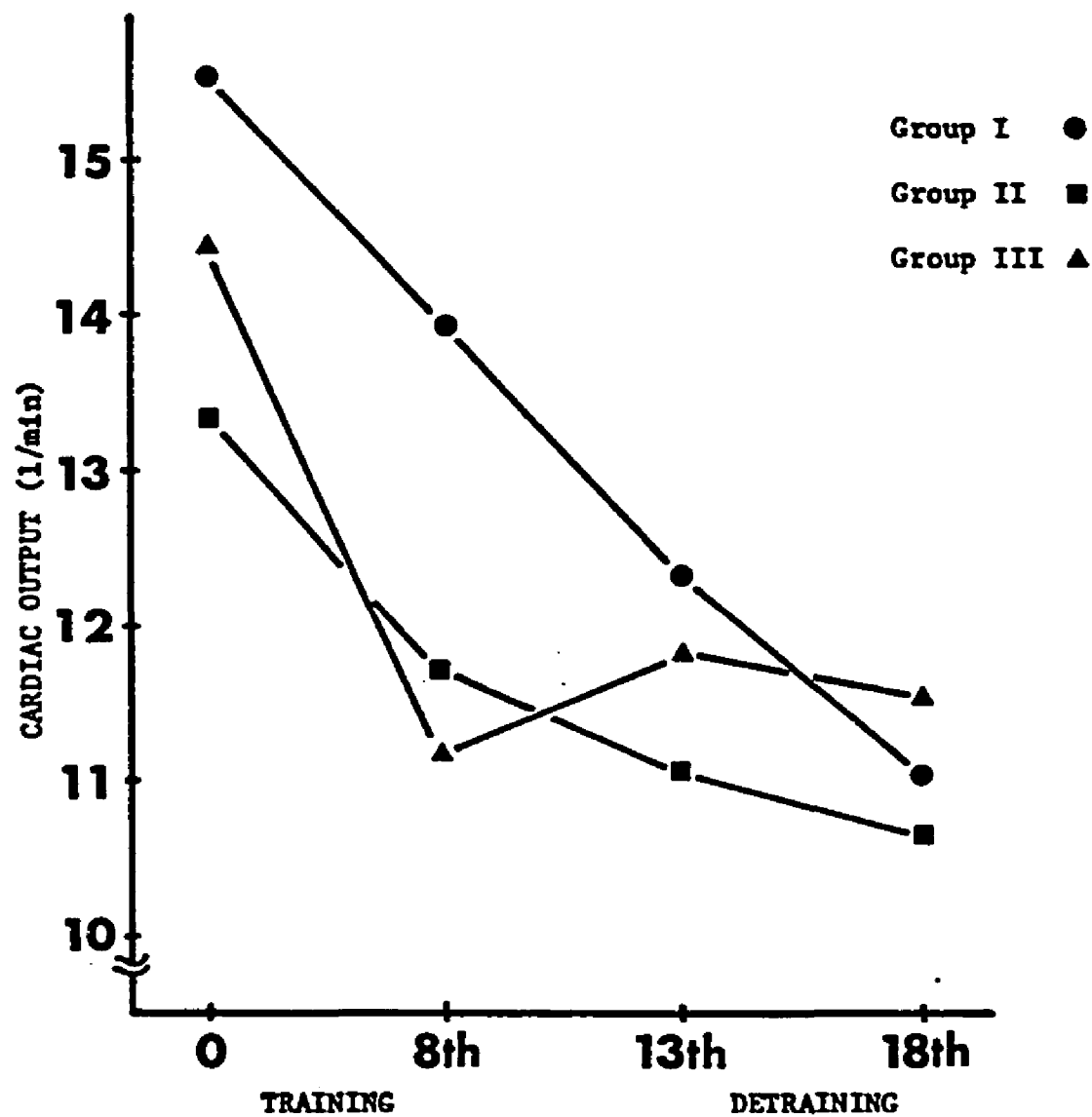


FIGURE 5. SUBMAXIMAL CARDIAC OUTPUT, TRAINING AND DETRAINING

there was a slight increase in the mean $a-\bar{v}O_2$ difference following training, (9.72 mls/100 mls as compared to 11.10 mls/100 mls) this increase was not statistical different. It is interesting to note, however, that mean differences were significant among groups. The mean $a-\bar{v}O_2$ difference for Groups I, II, and III was 10.03, 11.46, and 10.90 respectively. Group II was significantly greater than Group I. Group II's mean change was 12.5% higher than Group I. No significant differences were found between Group II and Group III. See Figure 6.

TOTAL BODY WEIGHT

Total body weight did not significantly change throughout the training and detraining program.

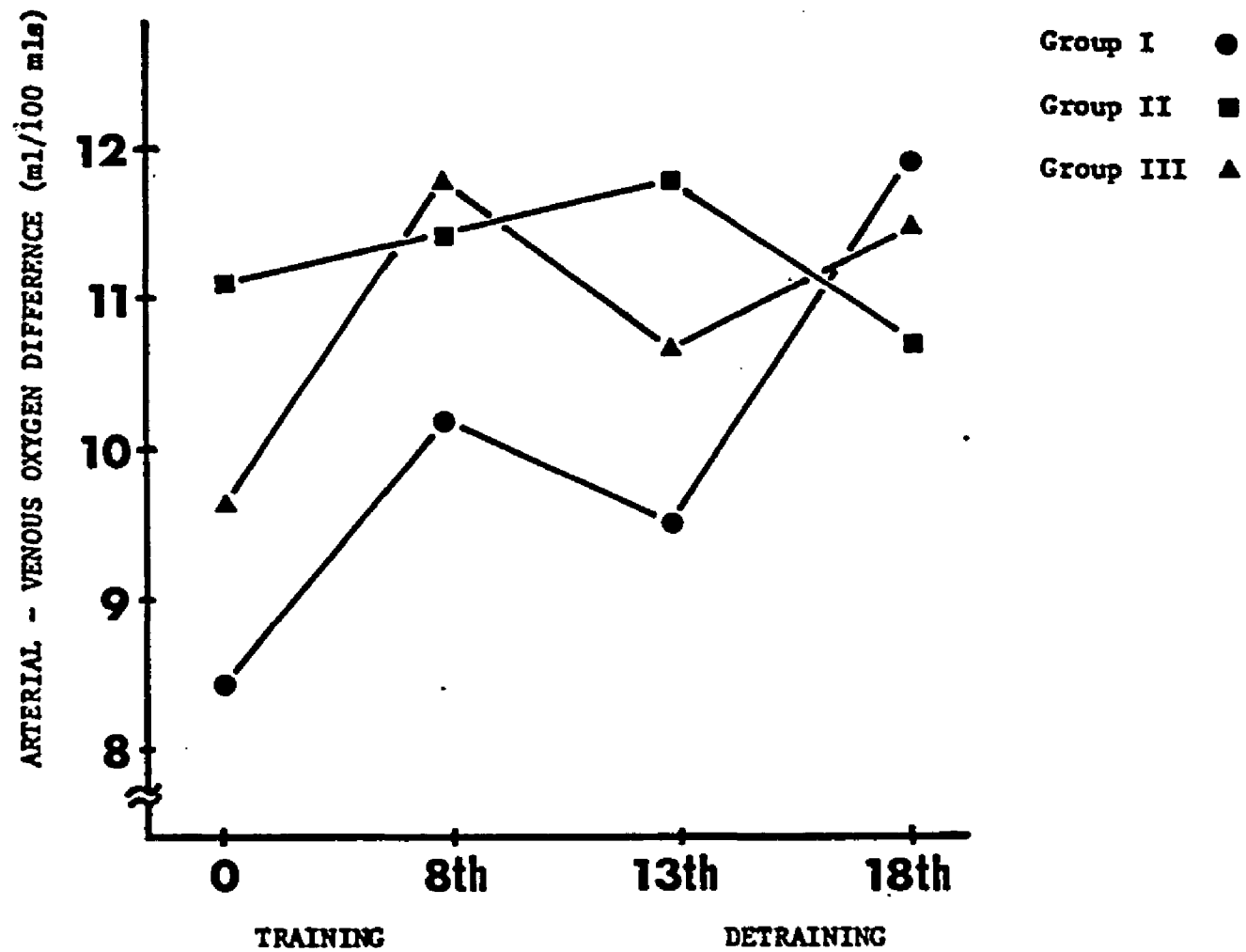


FIGURE 6. SUBMAXIMAL ARTERIO-VENOUS DIFFERENCE, TRAINING AND DETRAINING

Chapter V

DISCUSSION

The discussion will be broken down into two sections. Section one will deal with the circulatory adaptations which occurred during the eight weeks of training. Section two will focus on how these adaptations were altered within the ten week detraining program.

CIRCULATORY ADAPTATIONS TO TRAINING

SUBMAXIMAL OXYGEN CONSUMPTION (submax $\dot{V}O_2$)

Irrespective of units (ml/kg.-min or liters/min), submaximal oxygen consumption did not change with training. Studies (18, 40, 44, 54) performed on both male and female subjects have reported similar results. Still others, (23, 29) have noted a decrease in submax $\dot{V}O_2$. This decrease is attributable to an increased mechanical efficiency. Perhaps this phenomenon is best observed while training on a bicycle ergometer where subjects learn to become more efficient with riding.

Although the absolute submax $\dot{V}O_2$ did not change with training, maximal oxygen consumption was increased by twenty percent (51). Therefore, the percentage of max $\dot{V}O_2$ utilized during this submaximal work task was decreased.

SUBMAXIMAL HEART RATE (submax HR)

Submaximal heart rate dropped significantly with training. A mean decrease of 14 bpm was evident in the present study. Previous investigators (23, 29, 30, 40, 55) have considered this variable to be the most pronounced and consistent change during submaximal exercise following training.

Since no changes were found in submax $\dot{V}O_2$, one can rule out that an improved mechanical efficiency played a major role in the reduction of submax HR. The mechanism for a training bradycardia has been described by Frick et al. (30) as a decrease in sympathetic drive. This decrease can be due to, an increase in stroke volume reducing sympathetic drive, fewer afferent impulses arising from the trained muscles, or descending impulses from the motor cortex. Regardless of the mechanism, less submaximal circulatory stress is of an important and practical benefit to the trained individual.

SUBMAXIMAL STROKE VOLUME (submax SV)

A significant decrease of 12.4 mls was found in submaximal stroke volume with training. This finding is surprising since it has been shown that training increases stroke volume during submaximal exercise at a given work load (29, 30, 40, 55). Perhaps this decrease can be attributed to the fact that the subjects were all working at approximately 36% of their maximal $\dot{V}O_2$. Astrand (6) has shown that maximal stroke volume occurs at approximately 40% of one's maximal $\dot{V}O_2$.

Andrew et al. (1) examined eight subjects at four different submaximal workloads (350, 550, 750, and 900 kpm), and found no significant change in submaximal stroke volume.

Lesmes et al. (44), who utilized subjects comparable to those in the present study, obtained similar decreases in submaximal stroke volume following training. The exact mechanism as to why this occurred is not immediately apparent. It appears, however, that more research is needed in regards to how submaximal stroke volume is altered following training.

SUBMAXIMAL CARDIAC OUTPUT (submax \dot{Q})

Studies have shown that training may result in an increased, (2, 23, 32, 60) decreased, (44, 54) or unchanged, (40, 55) cardiac output during submaximal exercise. For example, Kilbom (40) found no significant reductions in \dot{Q} , $\dot{V}O_2$, and $a-\bar{v}O_2$ diff. at a given submaximal workload in middle-aged women following training. Saltin and others (54) obtained similar results with male subjects. Other studies (2, 60) have shown that training results in a decreased heart rate, but increased \dot{Q} , $\dot{V}O_2$, and $a-\bar{v}O_2$ diff. during moderate work. Still others (13, 54) have found that \dot{Q} and $\dot{V}O_2$ decrease at a given level of submaximal exercise after training.

Since heart rate and stroke volume were significantly decreased with training in this study, it is not surprising that submax \dot{Q} (HR X SV) also decreased significantly. Mean submax \dot{Q} dropped from 14.45 liters/min at the beginning of training to

11.65 liters/min at the end of training. These results compare with those of Lesmes et al. (44) who did a similar training study on females of comparable ages.

SUBMAXIMAL ARTERIAL - VENOUS OXYGEN DIFFERENCE (submax $a-\bar{v}O_2$ diff.)

Although there was a slight increase in the mean $a-\bar{v}O_2$ difference (9.72 mls/100 ml to 11.11 mls/100 ml) following training, this difference was not significant.

Training leads to a larger $a-\bar{v}O_2$ diff. in young men (2,23). This increase is characterized by a greater extraction of oxygen per se by the skeletal muscles (53) and/or a redistribution of blood flow. Redistribution of blood flow occurs from areas of low extraction such as the splanchnic bed to areas of high extraction such as the working skeletal muscles.

The present data and those of Kilbom (40) in young women found no significant increases in the $a-\bar{v}O_2$ diff. following training. The reason for this is not known.

CIRCULATORY ADAPTATIONS TO DETRAINING

When an athlete, male or female, begins to curtail one's training activities, one regresses into a state of detraining. The entire pattern of physiological change during detraining is less understood, in either males and females, than is the pattern of increased physiological efficiency. Few investigators have studied detraining responses to work tests, and their efforts have simply

been concerned with measuring the athletes following an interval of time to see what fitness losses have occurred. Submaximal measurements of cardiac output, stroke volume, and the $a-\bar{v}O_2$ diff. in females following an interval training program has not been evaluated by anyone.

SUBMAXIMAL OXYGEN CONSUMPTION (submax $\dot{V}O_2$)

Submaximal oxygen consumption was not significantly altered throughout the ten week detraining program. Other studies on males and females (26, 45) have demonstrated similar results. However, in a study on seven girls, ages 15-17, Drinkwater et al. (20) found significant increases in submaximal oxygen consumption following training. In the present study, detraining was evaluated within a ten week period, whereas, the detraining period evaluated by Drinkwater was three months following the girls track season. This could account for the discrepancies found within these data.

SUBMAXIMAL HEART RATE (submax HR)

Detraining appears to have a significant effect upon both resting and exercise heart rate. Fringer and Stull (31) noted a significant increase in resting heart rate within a ten week detraining period. Other investigators (20, 27, 36, 45, 49, 58) have shown significant increases in submaximal heart rate in the detrained state when compared to their post training values. It appears by these data that the higher levels of cardiovascular efficiency which are obtained through training are lost upon the cessation of training.

In the present study submaximal heart rate was independent of detraining frequency. There was, however, a general trend for submaximal heart rate to decrease with Group II, who continued to train two days per week. At the end of the detraining program Group II's heart rate was 119 as compared to values of 139 and 131 for Groups I and III respectively. These findings are consistent with those of Fardy (27) which demonstrated that heart rate changes in detraining paralleled miles per week of training.

SUBMAXIMAL STROKE VOLUME (submax SV)

Submaximal stroke volume remained unaltered within the ten week detraining program. Frequency of detraining appeared to have little or no effect upon this variable. Since no data are available to support or refute these findings, it appears that submaximal stroke volume will not change within a ten week period following training.

SUBMAXIMAL CARDIAC OUTPUT (submax \dot{Q})

Frequency of detraining did not effect the submaximal cardiac output. Mean values throughout this period (11.79 liters/min, 11.40 liters/min) were not significantly different. Since both stroke volume and heart rate remained unchanged within the ten week session, it is not surprising to find that submax \dot{Q} also remained unchanged. Perhaps changes in this variable would have been found if a larger number of subjects had been utilized within each group.

SUBMAXIMAL ARTERIAL - VENOUS OXYGEN DIFFERENCE (submax $a-\bar{v}O_2$)

Since no statistical differences were found among trials or interaction, it appears that the submax $a-\bar{v}O_2$ diff. is unaltered within a ten week detraining period. It is also independent of detraining frequency. There was a significant F ratio found among groups. Group II was significantly higher than Group I. However, when analyzing this mean difference, it appears that Group I started the initial training program with a lower mean $a-\bar{v}O_2$ diff. and it continued to remain as such throughout the entire eighteen week period. It may be concluded that a ten week program of three distinct frequencies will have little or no effect upon submaximal arterial-venous oxygen difference.

Chapter VI

SUMMARY AND CONCLUSIONS

The purpose of this study was to determine the circulatory responses of college age females to two distinct phases of training. Phase I examined what adaptations would be elicited by an eight week training program of three days per week; Phase II consisted of a ten week detraining period. Three different frequencies of detraining were evaluated in order to understand what effects it would have on the cardio-respiratory system.

Eighteen healthy, untrained, female university students volunteered for this eighteen week study. The first part of the study consisted of an eight week interval training program, three days per week. At the conclusion of the training program subjects were assigned to one of three groups based on maximal oxygen consumption ($\text{ml/kg} \cdot \text{min}$). Group I continued to train once per week, Group II trained twice per week, and Group III ceased training for the entire ten weeks.

Circulatory evaluations in the form of a submaximal treadmill walk were administered at weeks zero, eight, thirteen and eighteen. The repeated measurements were designed to provide a time course for any possible changes. Specific details concerning the tests and procedures can be found in Chapter III.

Training provided a progressive increase in distance and intensity within an interval training format. Subjects covered distances from 110 yards to 880 yards in a single repetition. Various combinations of repetitions provided total distance workouts from 990 to 3300 yards. All workouts were conducted on Monday, Wednesday and Friday. The exact training schedule of workouts may be found in the Appendix.

The workouts performed during detraining were different for each group. Group III ceased training completely, thus returning to normal activity patterns. Group II trained on Monday and Wednesday of each week, and completed all of the twenty workouts described in the Appendix. Group I participated in only ten detraining workouts, equaling exactly half of the work performed by Group II. Detraining sessions equaled but never exceeded the intensity or duration of the workouts performed during the last week of training. Based on this premise we can assume that frequency of detraining was the only manipulated variable throughout the ten week program.

Statistical evaluation of the data was performed subsequent to the eighteen week training-detraining program. A two factor (3 X 4) model of analysis of variance for repeated observations on the same subject was utilized for comparisons of retests, groups and interaction. When statistically significant F ratios were discovered, a Tukey multiple range test was employed to determine the location of statistical variation.

CONCLUSIONS

Within the limitations of this investigation the following conclusions appear justified:

1. An eight week interval training program will produce no significant change in submaximal oxygen consumption.
2. Submaximal cardiac output will significantly decrease as a result of a three day per week interval training program.
3. Submaximal heart rate significantly decreases with training.
4. Submaximal stroke volume significantly decreases with training.
5. The $a-\bar{v}O_2$ difference is not significantly altered in females with interval training.
6. A ten week detraining program will produce no significant change in submaximal oxygen consumption.
7. Regardless of frequency of detraining, submaximal cardiac output is not significantly changed.
8. Submaximal stroke volume will not be significantly altered by a ten week detraining program.
9. A detraining program of two days per week will enhance exercise bradycardia.
10. The $a-\bar{v}O_2$ difference is independent of detraining frequency.
11. Females can successfully endure strenuous training programs, similar to their male counterparts.

APPENDIX A

Table 3
Phase One
INTERVAL TRAINING PROGRAM

<u>DAY</u>	<u>PRESCRIPTION</u>	<u>DAY</u>	<u>PRESCRIPTION</u>
1	110 time trial 4 x 220 easy (1:3)	14	4 x 220 @ :48 (2:25) 5 min rest
2	880 time trial 4 x 110 easy (1:3)	15	8 x 110 @ :22 (1:06) 2 x 880 @ 3:40 (7:20) 5 min rest
3	3 x 440 easy (1:3)	16	1 x 660 @ 2:40 4 x 220 @ :45 (2:15) 5 min rest
4	4 x 220 @ :55 (2:45) 5 min rest 2 x 110 @ :25	17	6 x 110 @ :22 (1:06) 3 min rest 1 x 330 @ 1:10
5	1 x 880 @ 4:00 (8:00) 5 min rest 1 x 660 @ 3:00	18	3 x 88 @ 3:35 (7:00) 3 x 440 @ 1:40 (3:05) 4 min rest
6	4 x 220 @ :52 (2:35) 5 min rest 4 x 110 @ :25 (1:15)	19	3 x 220 @ :42 (2:05) 3 x 660 @ 2:35 (5:00) 5 min rest
7	2 x 660 @ 2:50 (5:00) 5 min rest 1 x 220 @ :52	20	1 x 880 @ 3:35 6 x 220 @ :42 (2:05) 4 min rest
8	7 x 110 @ :23 (1:09) 4 min rest 3 x 220 @ :50 (2:30)	21	7 x 110 @ :20 (1:00) 4 x 440 @ 1:35 (2:55) 4 min rest
9	2 x 880 @ 3:50 (7:40)	22	2 x 660 @ 2:30 (5:00) 3 x 880 @ 3:30 (7:00) 7 min rest
10	2 x 440 @ 1:50 (3:00) 5 min rest 1 x 660 @ 2:45	23	1 x 660 @ 2:30 110 time trial 4 min rest
11	2 x 880 @ 3:45 (7:30) 4 min rest 1 x 220 @ :50	24	8 x 220 @ :42 (2:05) 4 min rest 3 x 110 @ :19 (:56) 880 time trial 5 min rest 4 x 660 @ 2:30 (5:00)
12	110 time trial 4 min rest 7 x 220 @ :45 (2:25)		
13	880 time trial 5 min rest 3 x 440 @ 1:45 (3:15)		

*- training sessions which group I participated in
- group II participated in all Phase Two sessions.

Table 3

Phase Two

INTERVAL DETRAINING PROGRAM

<u>DAY</u>	<u>PRESCRIPTION</u>	<u>DAY</u>	<u>PRESCRIPTION</u>
1	6 x 220 @ :42 (2:05) 4 min rest	11	110 time trial 6 x 220 @ :42 (2:05) 4 min rest
2*	7 x 110 @ :19 (:56) 4 x 660 @ 2:25 (4:50)		6 x 110 @ :19 (:56)
3	2 x 880 @ 3:30 (7:00) 7 min rest	12*	880 time trial 2 x 660 @ 2:25 (4:50)
4*	2 x 660 @ 2:30 (5:00) 11 x 110 @ :19 (:56) 4 min rest	13	4 x 220 @ :42 (2:05) 4 min rest
5	4 x 220 @ :42 (2:05) 4 x 440 @ 1:35 (2:55) 4 min rest	14*	11 x 110 @ :19 (:56) 2 x 880 @ 3:30 (7:00) 7 min rest
6*	4 x 220 @ :42 (2:05) 4 x 660 @ 2:30 (5:00) 5 min rest	15	2 x 660 @ 2:30 (5:00) 4 x 660 @ 2:30 (5:00) 5 min rest
7	1 x 440 @ 1:40 (3:05) 5 x 220 @ :42 (2:05) 4 min rest	16	1 x 440 @ 3:05 4 x 440 @ 1:35 (2:55) 4 min rest
8*	9 x 110 @ :19 (:56) 2 x 660 @ 2:25 (4:50) 5 min rest	17	4 x 220 @ :42 (2:05) 2 x 660 @ 2:30 (5:00) 5 min rest
9	3 x 440 @ 1:35 (2:55) 3 x 880 @ 3:30 (7:00) 7 min rest	18*	3 x 440 @ 1:35 (2:55) 5 x 220 @ :42 (2:05) 4 min rest
10*	1 x 440 @ 1:40 (3:05) 7 x 220 @ :42 (2:05) 4 min rest	19	9 x 110 @ :19 (:56) 7 x 220 @ :42 (2:05) 4 min rest
	5 x 110 @ :19 (:56)	20*	5 x 110 @ :19 (:56) 3 x 880 @ 3:30 (7:00) 7 min rest
			1 x 440 @ 1:40 (3:05)

SAMPLE CARDIAC OUTPUT

NAME _____ AGE 19 HT 65.25 WT 150 LBS
 GROUP I TEST PRE _____ POST _____ WT 68.18 KGS
 WORK LEVEL _____ DATE 1/8/77
 FINAL GASOMETER READING 371.2 TEMP 24 C°
 INITIAL GASOMETER READING 130.2 PRESSURE 743.8 mmHg
 STPD FACTOR 0.869
 UNCORRECTED VOLUME 32.10 BTPS FACTOR 1.080
 VOLUME STPD 27.89 L PRESSURE-47mmHg _____ mmHg
 VOLUME BTPS 34.66 L HR 150 BR 21
 Vt = 1650 ml (BTPS)

GAS ANALYSIS:

% CO₂ 4.35 % O₂ 16.09 % N₂ 79.56
 % CO₂ - 0.04% = %CO₂ expired 4.31 % O₂ consumed 4.99
 VCO₂ ml/min (STPD) 1200 VO₂ ml/min (STPD) 1390
 R = 0.86

$\bar{P}\text{VCO}_2 = \text{CO}_2 \text{ (Equil)} \times (P_B - 47) = \underline{59.29}$

CaCO₂ = 47.15 ml/liter CVC0₂ = 56.73 ml/liter

$\dot{Q} = \frac{\text{VCO}_2 \text{ (STPD) ml}}{\text{CVC0}_2 \text{ ml/l} - \text{CaCO}_2 \text{ ml/l}} = \frac{1200}{9.58} = 12.50$
 $\text{SV} = \frac{\dot{Q}}{\text{HR}} = \underline{83.3} \text{ ml}$

$\text{a-vO}_2 \text{ Difference} = \frac{\text{VO}_2}{\dot{Q}} = \underline{111.20} \text{ ml/l}$

APPENDIX B

Table 5

ANALYSIS OF VARIANCE - SUBMAXIMAL VO₂

Source of Variance	SS	df	MS	F
Treatments	0.05	2	0.025	1.09
Trials	0.16	3	0.053	0.26
Interaction	0.81	6	0.135	0.67
Between Error	0.35	15	0.023	
Within Error	0.93	45	0.20	
Total		71		

* Significant ($P < .05$)

Table 6

ANALYSIS OF VARIANCE - SUBMAXIMAL CARDIAC OUTPUT

Source of Variance	SS	df	MS	F
<hr/>				
Treatments	17.92	2	8.96	2.03
Trials	109.76	3	36.59	12.66*
Interaction	8.23	6	1.37	0.47
Between Error	66.27	15	4.42	
Within Error	130.32	45	2.89	
Total		71		

* Significant ($P < .05$)

Table 7

ANALYSIS OF VARIANCE - SUBMAXIMAL HEART RATE

Source of Variance	SS	df	MS	F
<hr/>				
Treatments	9485.5	2	4742.7	7.12*
Trials	2947.6	3	982.5	3.95*
Interaction	2473.0	6	412.2	1.65
Between Error	9987.0	15	665.8	
Within Error	11177.3	45	248.4	
Total		71		

* Significant ($P < 0.05$)

Table 8

ANALYSIS OF VARIANCE - SUBMAXIMAL STROKE VOLUME

Source of Variance	SS	df	MS	F
<hr/>				
Treatments	219.2	2	109.6	0.55
Trials	1997.8	3	665.9	5.55*
Interaction	205.4	6	34.2	0.31
Between error	3007.45	15	200.5	
Within error	5039.7	45	111.9	
Total		71		

* Significant ($P < .05$)

Table 9

ANALYSIS OF VARIANCE - SUBMAXIMAL A- $\dot{V}O_2$ DIFF.

Source of Variance	SS	df	MS	F
<hr/>				
Treatments	94.2	2	4.71	12.86*
Trials	29.34	3	9.78	0.39
Interaction	7.03	6	1.17	0.47
Between Error	54.95	15	3.66	
Within Error	1101.6	45	24.48	
Total		71		

* Significant ($P < 0.05$)

Table 10

RAW DATA

PRE-TRAINING

Subject	Group days/wk	$\dot{V}O_2$ (l/min)	H.R. (bpm)	SV (mls)	A- $\dot{V}O_2$ diff ² (mls/100 ml)	\dot{Q} (l/min)
1	2	1.696	115	89.04	16.56	10.24
2	1	1.390	150	83.30	11.12	12.50
3	1	1.230	142	85.40	10.13	12.14
4	0	1.150	133	104.51	8.27	13.90
5	0	1.380	150	80.93	11.36	12.14
6	1	1.400	142	116.50	84.80	16.50
7	2	1.380	139	109.85	9.03	15.27
8	0	1.440	162	107.03	8.30	17.34
9	0	1.210	146	86.90	9.52	12.70
10	1	1.460	175	118.57	7.20	20.27
11	2	0.991	130	11.92	6.39	15.50
12	2	1.760	140	110.00	11.42	15.40
13	2	1.270	171	60.18	12.24	10.37
14	2	1.450	140	94.21	10.99	13.19
15	1	0.983	130	113.10	6.68	14.70
16	0	1.300	158	122.70	6.70	19.40
17	1	1.210	145	119.03	7.01	17.26
18	0	1.520	171	65.14	13.64	11.14
		\bar{x} 1.345	\bar{x} 146.6	\bar{x} 98.79	\bar{x} 9.72	\bar{x} 14.44

Table 11

RAW DATA

POST TRAINING

Subject	Group days/wk	$\dot{V}O_2$ (l/min)	H.R. (bpm)	SV (mls)	$A-\bar{V}O_2$ diff ² (mls/100 ml)	\dot{Q} (l/min)
1	2	1.264	115	101.50	9.43	13.40
2	1	1.395	132	90.75	11.64	11.98
3	1	1.151	142	89.43	9.06	12.70
4	0	0.991	100	82.70	11.98	8.30
5	0	1.379	136	82.35	12.31	11.20
6	1	1.145	136	89.70	9.39	12.20
7	2	1.450	143	83.20	12.23	11.90
8	0	1.353	133	92.48	11.00	12.30
9	0	1.187	120	91.33	10.83	10.96
10	1	1.277	150	83.33	10.21	12.50
11	2	1.060	125	88.40	10.80	9.80
12	2	1.127	130	96.15	9.01	12.50
13	2	1.533	146	84.93	12.36	12.40
14	2	1.493	130	78.46	14.63	10.20
15	1	0.985	126	86.50	9.03	10.90
16	0	1.341	143	86.01	10.90	12.30
17	1	1.319	142	80.28	11.57	11.40
18	0	1.662	143	86.01	13.51	12.30
		\bar{x} 1.226	\bar{x} 132.8	\bar{x} 86.86	\bar{x} 11.10	\bar{x} 11.62

Table 12

RAW DATA

FIFTH WEEK - DETRAINING

Subject	Group days/wk	$\dot{V}O_2$ (l/min)	H.R. (bpm)	SV (mls)	A- $\dot{V}O_2$ diff ² (mls/100 ml)	\dot{Q} (l/min)
1	2	1.254	115	94.43	11.54	10.86
2	1	1.132	140	85.00	9.51	11.90
3	1	1.183	136	95.95	9.06	13.05
4	0	1.168	115	80.70	12.58	9.28
5	0	1.389	143	85.94	11.30	12.29
6	1	1.415	142	82.60	13.10	11.73
7	2	1.448	136	79.41	13.41	10.80
8	0	1.501	150	79.46	12.59	11.92
9	0	1.229	136	99.04	9.12	13.47
10	1	1.237	167	83.29	8.89	13.91
11	2	1.100	120	96.58	9.49	11.59
12	2	1.149	120	96.42	9.93	11.57
13	2	1.470	130	83.38	13.56	10.84
14	2	1.408	126	85.63	13.05	10.79
15	1	0.867	115	90.17	8.36	10.37
16	0	1.283	134	89.32	10.71	11.97
17	1	1.031	130	98.23	8.07	12.77
18	0	1.008	167	78.44	7.69	13.10
		\bar{x} 1.2373	\bar{x} 134.5	\bar{x} 87.99	\bar{x} 10.66	\bar{x} 11.78

Table 13

RAW DATA

TENTH WEEK - DETRAINING

Subject	Group days/wk	$\dot{V}O_2$ (l/min)	H.R. (bpm)	SV (mls)	A- $\dot{V}O_2$ diff ² (mls/100 ml)	\dot{Q} (l/min)
1	2	1.224	111	101.17	10.89	11.23
2	1	1.479	143	86.57	11.95	12.38
3	1	1.139	136	81.98	10.22	11.15
4	0	1.047	107	94.39	10.36	10.10
5	0	1.337	142	81.26	11.58	11.54
6	1	1.154	130	85.08	10.43	11.06
7	2	1.365	125	84.72	12.89	10.59
8	0	1.368	140	81.00	12.06	11.34
9	0	1.228	136	88.53	10.19	12.04
10	1	1.222	167	77.60	9.42	12.96
11	2	1.020	115	97.30	9.12	11.19
12	2	1.078	120	90.00	9.98	10.80
13	2	1.425	125	75.52	15.09	9.44
14	2	1.295	120	91.58	11.78	10.99
15	1	0.953	115	91.91	9.02	10.57
16	0	1.276	130	92.38	10.62	12.01
17	1	1.205	143	95.73	8.80	13.69
18	0	1.224	130	93.69	10.04	12.18
		\bar{x} 1.224	\bar{x} 129.7	\bar{x} 93.65	\bar{x} 10.80	\bar{x} 11.40

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