Acute and Chronic Physiological Changes to High Intensity Training

PATRICIA K. DOYLE
Bachelor of Science, University of Victoria, 1982

A THESIS SUBMITTED IN PARTIAL FULFILLMENT

OF THE REQUIREMENTS FOR THE DEGREE OF

MASTER OF ARTS

in the Department

of

Physical Education

ACCEPTED
FACULTY OF GRADUATE STUDIES
[redacted]
DEAN
DATE 1986-12-12

We accept this thesis as conforming
to the required standard

[redacted]
Howard A. Wenger, Ph.D.

[redacted]
David Docherty, Ph.D.

[redacted]
Frank Robinson, Ph.D.

[redacted]
John E. Peterson, M.D.

UNIVERSITY OF VICTORIA

July 1986

Supervisor: Professor Howard A. Wenger

ABSTRACT

Six healthy male varsity oarsmen from the University of Victoria volunteered to participate in a 14 week training program. This study was designed to investigate the acute and chronic responses of hemoglobin (Hb), hematocrit (Hct), white blood cell (WBC), and creatine phosphokinase (CPK), to a 4 week control period and an 8 week training program of increasing intensity and to relate these to changes in a performance test (PT). Mean maximum oxygen consumption ($\dot{V}_{O2}max$) increased from 4.4 ± 0.1 to 4.9 ± 0.1 $l \cdot min^{-1}$ and body weight and skinfolds decreased significantly during the study. No change occurred in maximal or recovery heart rates. The chronic response of Hct showed a 4 % decrease during both the control period (weeks 1-4) and over weeks 5-8 of training but increased 4 % from weeks 9-13. Hb decreased 7 % during weeks 1-4 and a further decrease of 1.9% occurred during weeks 5-8. The acute values of Hct and Hb were slightly smaller than the chronic values but followed similar trends. The mean difference between the chronic and acute values during the weeks of training was 2.4 % for Hct (48.5 ± 0.9, 47.6 ± 0.9 %) and 2.9 % for Hb (14.6 ± 0.97, 14.5 ± 0.4 $g \cdot dl^{-1}$) respectively. The only significant changes in WBC occurred in the acute fluctuations of the chronic response. CPK increased from week 1 to week 13 both chronically (63.9 ± 0.9 to 98.9 ± 0.4 $U \cdot l^{-1}$) and acutely (63.7 ± 1.3 to 109.0 ± 1.3 $U \cdot l^{-1}$). Weeks 5-8 showed the greatest change with a two fold increase

in the chronic ($129.9 \pm 1.2\ U \cdot l^{-1}$) and a four fold increase in the acute response ($253.7 \pm 2.3\ U \cdot l^{-1}$). All subjects improved in the PT over the 13 weeks with the greatest improvement occurring between weeks 4-7 ($p<0.05$). In conclusion the acute and chronic responses of Hct and Hb elicit similiar changes to repeated bouts of training ($r=0.571$). CPK levels in venous blood were significantly different in the acute and chronic response to exercise. This study was not able to demonstrate a decrease in the PT with changes in the acute and chronic levels of Hb, WBC, and CPK. However, a decrease in Hct was related to an increase in performance despite the low N and homogeneity of the sample ($r=0.340$).

Examiners:

[redacted]
Howard A. Wenger, Ph.D.

[redacted]
David Docherty, Ph.D.

[redacted]
Frank Robinson, Ph.D.

[redacted]
John E. Peterson, M.D.

CONTENTS

TABLES

FIGURES

ACKNOWLEDGEMENTS

I would like to extend my thanks to my supervisor, Dr. Howard A. Wenger who first introduced me into the world of Human Performance and because of his active interest, guidance in analytical writing and support has made my masters program in Exercise Physiology an extremely valuable experience.

Many thanks to Drs. Frank Robinson, Dave Docherty, and John Peterson for their assistance as members of my advisory committee.

Thanks to Eugene, Jim, and Ise who were instrumental in speeding up the process of completing my graphs and tables in my thesis and to Ashley for doing my slides.

For making my life as a grad student easier I would like to thank Gord M., Jim H., Kathryn, and Sheryl of Athletic and Recreation; Georgina, Gladys, and Jill of the Physical Education office; Doug, Rob, and Rene of the equipment room; fellow Grad Students; and all the individuals who helped with my testing.

A sincere thank you to coaches Al Morrow and Howie Campbell for doing an excellent job of implementing and following the 8 week training program and for giving me the opportunity to use 10 of their oarsmen in my thesis study. To Carson, Dave, Jason, Ian, Jim, Jon, Mike, Nick, Ralph, and Steve, I thank you for contributing 14 weeks of your summer to my thesis and no doubt I will always have fond memories of your antics and how you looked after completing 10 x 10, 3' minute pieces.

Finally it is with great pleasure that I can say; "Howie, you can make your own CHEESE CAKE now, because I am giving you the recipe!".

DEDICATION

To my Mum, for her love and constant support throughout my masters thesis.

To my Dad, who would have wanted this so very much.

Chapter I

INTRODUCTION

In recent years coaches have begun to use changes in blood chemistry to help monitor training programs (Rushall & Busch, 1980) and to establish the general state of health of the athlete. Abnormal levels in blood chemistry can occur because of the stress of continual training which can eventually become a limiting factor in optimal performance (Ekblom et al., 1972).

The appearance of staleness and fatigue in athletes is regularly associated with a marked fall in hemoglobin (Hb) concentration in the blood (Sutton, 1971). Ekblom et al., (1972) point out that the total amount of circulating Hb correlates well with maximum oxygen consumption ($\dot{V}_{O2}max$) and a decrease in Hb concentration would result in a decrease in physical performance which would tax $\dot{V}_{O2}max$. Several studies (Astrand & Rohadl, 1977; DeWijn et al., 1971; Burke et al., 1985) have shown that subnormal Hb concentrations inhibit performance in aerobic events because oxygen is transported by the Hb. Reduced Hb due to iron deficiency can occur without anemia and involves an exhausted store of iron and diminished iron bearing tissue enzymes (DeWijn et al., 1971).

The stress of endurance exercise decreases blood and plasma volume resulting in a decrease in hematocrit (Hct) levels. Likewise the ability of the circulatory system to support aerobic work increases as the Hct rises towards a certain level (Hct above 60.0%) and declines beyond that value (Guyton, 1981). These changes depend on the severity of the exercise and the duration (Wilkerson et al., 1977).

Therefore, it would be desirable to have both Hb and Hct values at their maximum for serious competition in activities that rely on oxygen transport (Rushall & Busch, 1980). Problems of low Hb and Hct have been found to occur in Olympic athletes, with a particularly high incidence in rowers and runners (Clement et al., 1977).

One of the changes that occur in blood chemistry as a consequence of endurance training is the increased presence of the muscle enzyme, creatine phosphokinase (CPK). It has also been shown that among serum enzymes studied in the blood, CPK activity is the most sensitive to changes in the exercise load (LaPorta et al., 1978; Magazanik, 1974) and is the most specific indicator of pathological changes in skeletal muscle. Heavy exercise and continual training have been demonstrated to increase serum CPK provided that the exertion is of sufficient severity (Hunter & Critz, 1971).

Both short term and endurance exercise have also been found to rapidly increase the circulating white blood cells (WBC) (Burke et al., 1985).

Competitive rowing is one of the most physically demanding endurance sports as it utilizes the major muscles in the body at high rates and relatively long durations (Strydom et al., 1967; Hagerman et al., 1979). It was considered to be an ideal activity to study the acute and chronic responses to progressively increasing training loads because: the adaptation to training and change in work performance could be easily measured and evaluated with the use of a rowing ergometer which is specific to the training mode of exercise (Martindale & Robertson, 1984); the oarsmen would be participating in identical workouts and therefore the absolute intensity and duration would be equal for all subjects; and rowing has already

established a land based ergometer performance test (PT) which is nationally and internationally accepted (Hagerman et al., 1979).

The purpose of this study was to investigate the acute and chronic responses of Hb, Hct, WBC, and CPK, to a progressively more difficult training program and to relate these to changes in a rowing ergometer PT.

Chapter II

METHODS

Six healthy male varsity oarsmen from the University of Victoria volunteered and signed informed consent (Appendix A). Prior to testing, the subjects were familiarized with the procedures and the physical demands of the testing and training. The subject characteristics are presented in Table 1.

2.1 Physiological Testing

$\dot{V}_{O2}$max was assessed prior to and following the 4 week control period and 8 weeks of training (Figure 1). The $\dot{V}_{O2}$max was assessed by open spirometry using a continuous multistage test on a rowing ergometer (Gjessing Ergorow). The subjects were seated and connected to the electrocardiograph and the Beckman Metabolic Measurement Cart (MMC) via a Rudolph low resistance valve (Model 65). The loads for the incremental test are given in Table 2.

Physiological measures including volume of expired air ($\dot{V}_E$), oxygen consumption ($\dot{V}_{O2}$), and respiratory exchange ratio (R) were recorded and analyzed every 30 seconds. The gas analyzers were calibrated using gases of known concentrations before and after each test. Heart rates were recorded every minute during exercise and the 6 minute recovery.

To confirm that $\dot{V}_{O2}$max was attained, all subjects met at least one of the following criteria; a plateau or decline in $\dot{V}_{O2}$max with increasing power outputs, maximal heart rate and/or respiratory exchange ratio ≥ 1.15.

Table 1

Mean Age, Weight (WT), Height (HT) and Skinfold (SK) Measurements of the Oarsmen

	Age (yrs)	WT (kg)	HT (cm)	SK (mm)
Males (N=6)	22.5 (±0.5)	82.0 (±2.0)	186.4 (±1.4)	12.8 (±1.2)

Note: Standard error shown in parentheses.

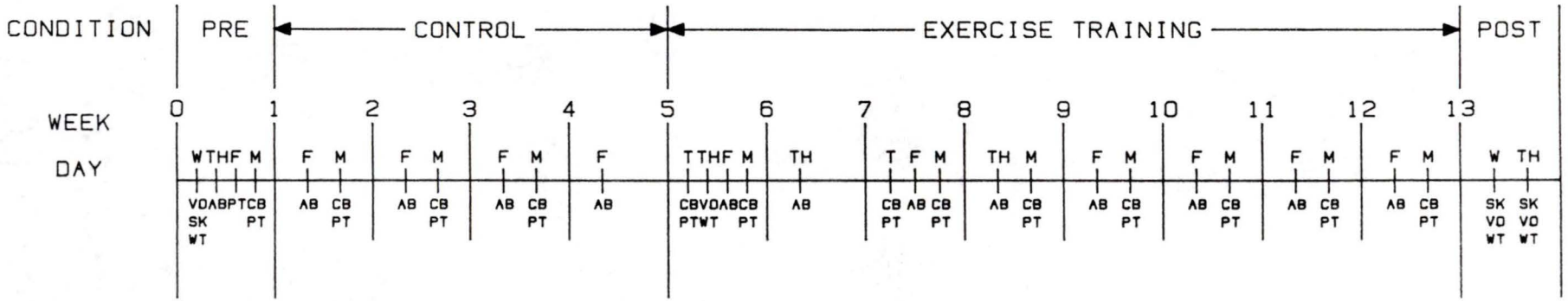
Table 2

The Loads on the Gjessing Ergometer for the Incremental Maximum Oxygen Capacity Test

Resistance (kp)	Duration (minutes)	RPM	Stroke · min^{-1}
1.50	2	600	30
1.75	2	600	30
2.00	1	600	30
2.25	1	600	30
2.50	1	600	30
3.00	1	max	max
3.50	1	max	max

Prior to the first and last $\dot{V}_{O2}max$, skinfold measurements (SK) were performed using the protocol of Durnin and Womersley (1974).

Figure 1: Experimental Protocol



WEIGHT – WT
SKINFOLDS – SK
VO_2 MAX – VO
PERFORMANCE TEST – PT
ACUTE BLOOD SAMPLE – AB
CHRONIC BLOOD SAMPLE – CB

2.2 Blood Testing

Antecubital venous blood samples were collected without stasis, twice weekly. The first sample was obtained immediately following a training session so that the values from the blood analysis would reflect the acute response to exercise. The second sample was obtained following a minimum 24 hour rest period so as to reflect the chronic status of the subjects. The blood was analyzed for Hct, Hb, WBC, and CPK.

Hct was measured using the microhematocrit centrifuge with the Adams readocrit and no correction was made for trapped plasma. Hemoglobin concentration was measured by the cyanomethemoglobin technique as described by Drabkin (1949). The activity of CPK was determined as per Sigma Chemical Technical Bulletin # 45-UV at 37°C. The Unipette microcollection system (Becton-Dickson Recorder # 5855), was used to determine the whole white blood cell counts. The reproducibility of the WBC counting technique was ± 5.0% (standard deviation of the difference as determined between 2 counters in the analysis of 20 samples with a range from 1900 to 16000). Samples were taken every 3 weeks to the Royal Jubilee Hospital and analyzed to compare the reliability of the tester. All samples were found to be within ± 5.0 %.

2.3 Performance Testing

Changes in performance were monitored weekly on the morning after the rest day through the use of the adapted land based PT of the Canadian Amateur Rowing Association (CARA). The PT was designed to simulate the intensity and duration normally experienced under race conditions (Hagerman et al., 1979). The

PT was designed to be exhaustive where the athlete achieved maximal or near maximal metabolic levels. The subjects completed a PT for 13 consecutive weeks of the study in which they were encouraged to give an all out effort and to improve their score from week to week. The normal 6 minute ergometer PT (Hagerman et al., 1979; Hagerman & Howie, 1966) was considered too long and stressful for each week and might predispose the athletes to a poor mental set throughout the study. Also, because rowers tend to follow a unique style of pacing, the most significant responses are often reached during the first minute or two of each exercise (Hagerman et al., 1979). With these factors in mind a modified 4 minute PT was used to achieve the objectives of a PT.

During the PT the feet of the subject were placed on the foot boards and strapped down. The customary rowing start was given "Sit up. Are you ready? Row". The PT consisted of a 2 stage, discontinuous submaximal and maximal protocol. The first stage involved a 2 minute warm up at the load of 1.5 kp at 600 revolutions per minute (RPM). At the end of the warm up, the subject was given time to check his foot placement and compose himself. The load was pre-set at 3.0 kp and the verbal start given again. The second stage lasted for 4 minutes and verbal feedback was given on stroke rate and RPM. However, there was no predetermined stroke rate or RPM.

2.4 The Training Program

Subjects trained on an average of five and a half days per week for eight consecutive weeks. The training program was designed to increase in intensity and duration as the program progressed. The majority of the workouts maintained

$\dot{V}_{O2}$max pace with emphasis on eliciting heart rates at approximately 175-185 beats per minute. The training program also included the following components of a rowing program but, to a lesser extent; steady state (SS), sprint work (SP), weight training (Wt. Train.) etc.... These workouts are outlined in detail in Appendix B and C.

2.5 Statistical Analysis

A multivariate analysis of variance (MANOVA) was used to determine any significant differences between the dependent and independent variables and a one way analysis of variance (ANOVA) was used to determine any interaction effects. A Pearson Product Moment Correlation was computed between the following variables; Hct, Hb, WBC, CPK and PT. Pre-test and post-test variables were compared with a non-parametric Wilcoxon test. The alpha level for significance was set at $p<0.05$. Descriptive statistics in the form of mean $\pm$ SEM and percentages were calculated for dependent variables.

Chapter III

RESULTS

The effect of 8 weeks of training on the cardiorespiratory system are shown in Table 3. Mean $\dot{V}_{O2}$max increased significantly from week 1 to week 13. Body WT was decreased following training from a mean of 82.0 ± 2.0 to 80.0 ± 1.6 kg. SK measurements decreased from a mean of 31.5 ± 3.2 to 27.0 ± 2.4 mm ($p<0.05$). However, maximal and recovery heart rates showed no changes.

Table 3

Mean Change in Maximum Oxygen Consumption from Pre-Control Through the Training Period

$\dot{V}_{O2}$max Test		
	$ml \cdot kg^{-1} \cdot min^{-1}$	$l \cdot min^{-1}$
Pre-control	54.4 (±1.0)[a,b]	4.4 (±0.1)[a,b]
Post-control	58.4 (±0.8)[a]	4.7 (±0.1)[a]
Post-training	60.4 (±1.9)[b]	4.9 (±0.1)[b]

Note: Standard error shown in parentheses.
Paired letters indicate significance at $p<0.05$.

The chronic response in Hct (Figure 2) decreased 4.0 % between week 1 (51.4 ± 0.6 %) and week 4 (49.3 ± 0.6 %) and between week 5 (48.5 ± 0.5 %) and week 8 (46.8 ± 0.8 %). During weeks 9 (48.2 ± 0.9 %) to 13 (49.8 ± 1.3 %), Hct increased by 4.0 %. The acute response mimicked this pattern but with slightly

Table 4

Correlation Matrix of the Dependent Variables

	Hct	Hb	WBC	CPK
Hct				
Hb	0.571			
WBC	0.148	0.001		
CPK	-0.299	-0.111	-0.077	
PT	-0.342	0.120	-0.218	0.252

smaller differences. The mean difference between the chronic (48.5 ± 1.0 %) and the acute (47.6 ± 0.9 %) response during the 8 weeks of training was 2.4 %.

Hb showed a more stable response to training than Hct (Figure 3). The mean chronic value showed a decrease of 7.0 % from the pre-control value of week 1 (14.5 ± 0.6 $g \cdot dl^{-1}$) to week 4 (13.5 ± 0.8 $g \cdot dl^{-1}$). This remained stable until week 8 when Hb decreased to a low of 13.3 ± 0.4 $g \cdot dl^{-1}$ and a drop of 8.9 % from week 1. The acute response followed the same pattern but with lower values. The mean difference between the chronic (14.4 ± 0.4 $g \cdot dl^{-1}$) and the acute (13.9 ± 0.4 $g \cdot dl^{-1}$) response during training was 2.9 % ($p<0.05$). However, the difference between the chronic (14.6 ± 0.7 $g \cdot dl^{-1}$) and the acute (14.5 ± 0.4 $g \cdot dl^{-1}$) response at week 13 was only 0.3 %.

During weeks 1-4 the chronic levels of WBC were significantly higher than the acute levels. The only significant changes that occurred in the training period of weeks 5-13 were weekly fluctuations in the acute response (Figure 4).

The greatest change in all 4 variables occurred in the response of CPK (Figure 5). The mean serum enzyme level significantly increased from week 1 to 13, both chronically (63.9 ± 0.9 to 98.9 ± 1.7 $U \cdot l^{-1}$) and acutely (63.9 ± 1.3 to

Figure 2: Mean Hct Values During the Control Period and the Progressively More Difficult Training Program

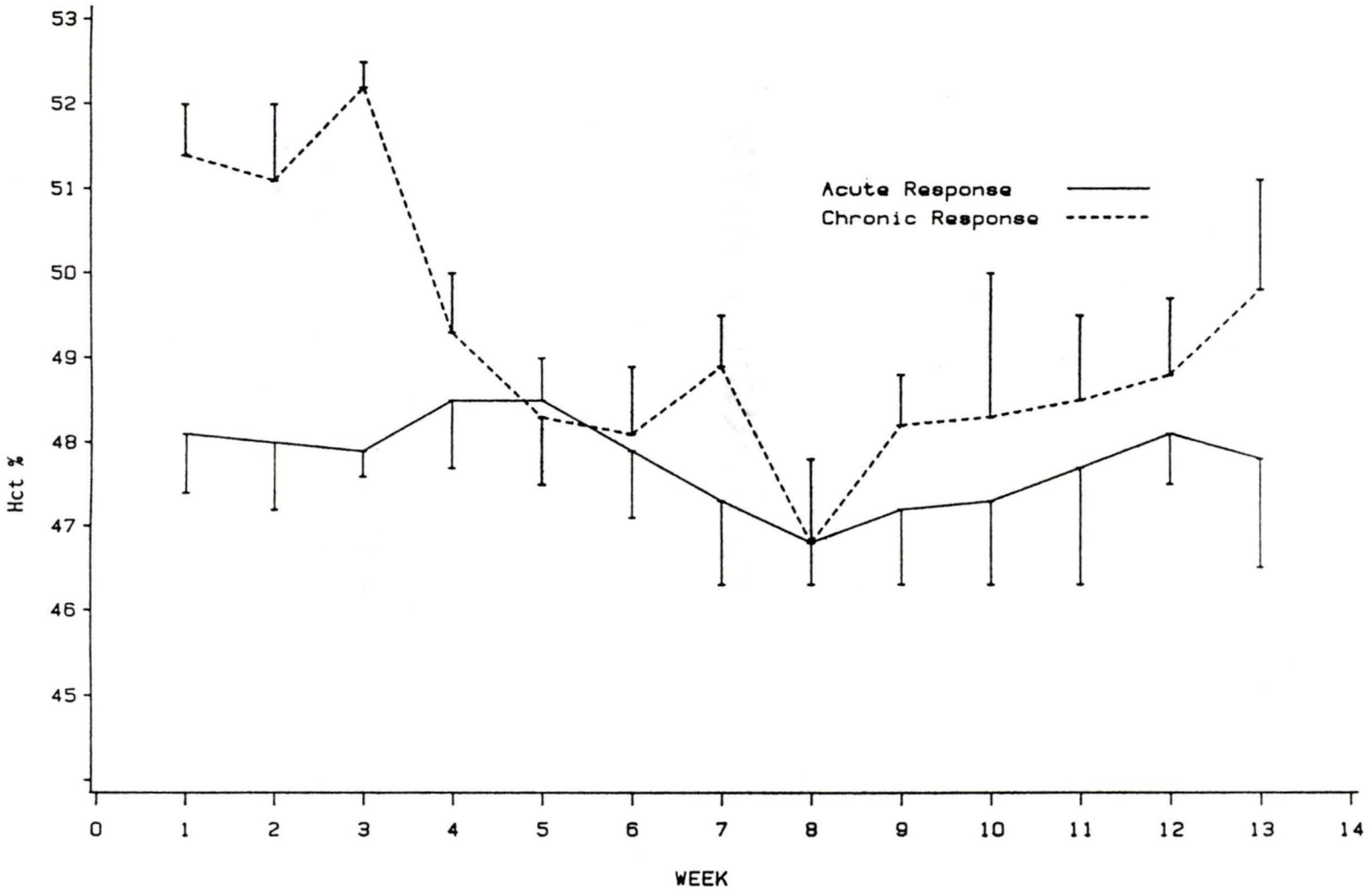

Acute Response
Chronic Response
Hct %
WEEK
53
52
51
50
49
48
47
46
45
0
1
2
3
4
5
6
7
8
9
10
11
12
13
14

Figure 3: Mean Hb Values During the Control Period and the Progressively More Difficult Training Program

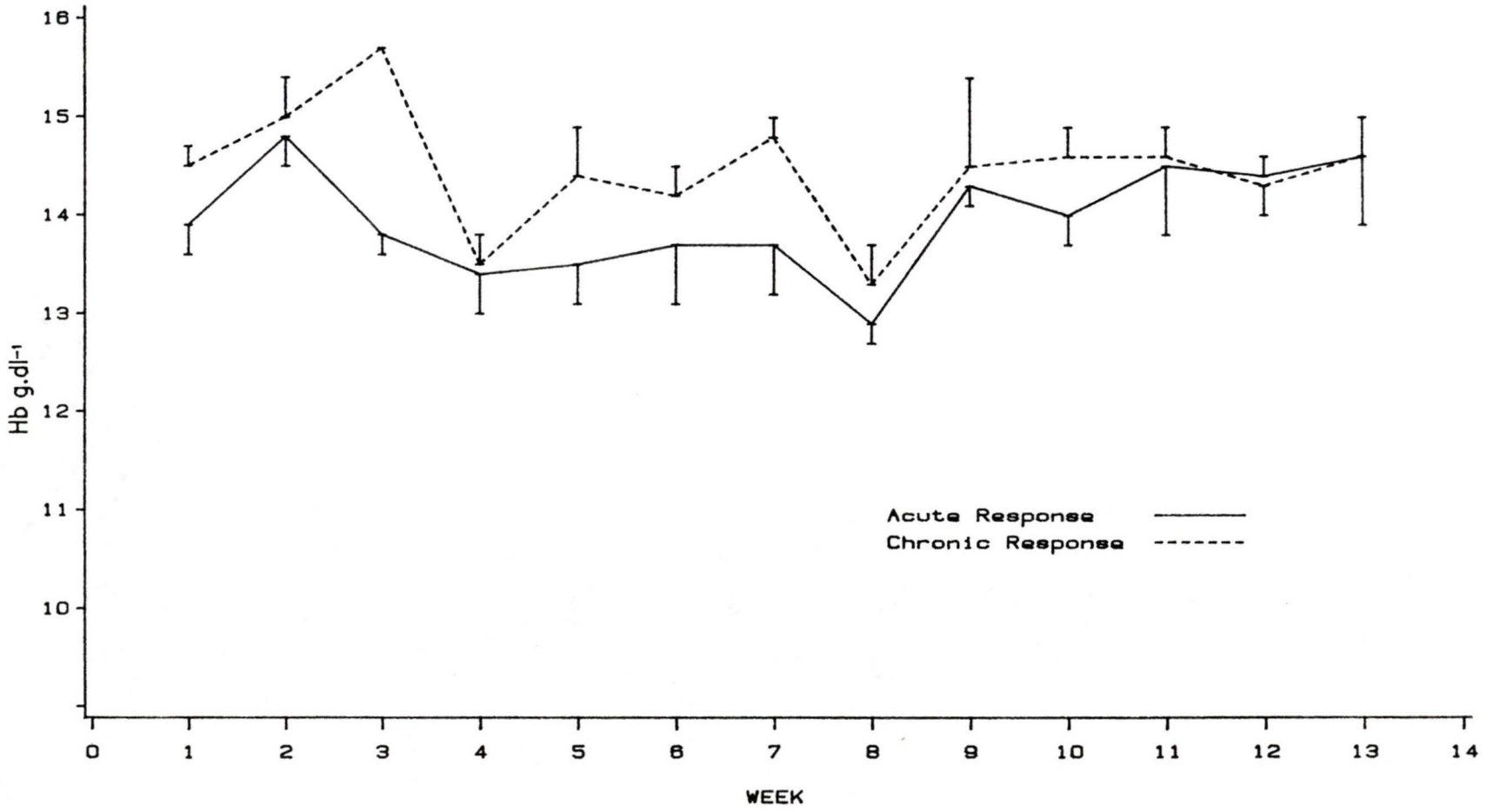
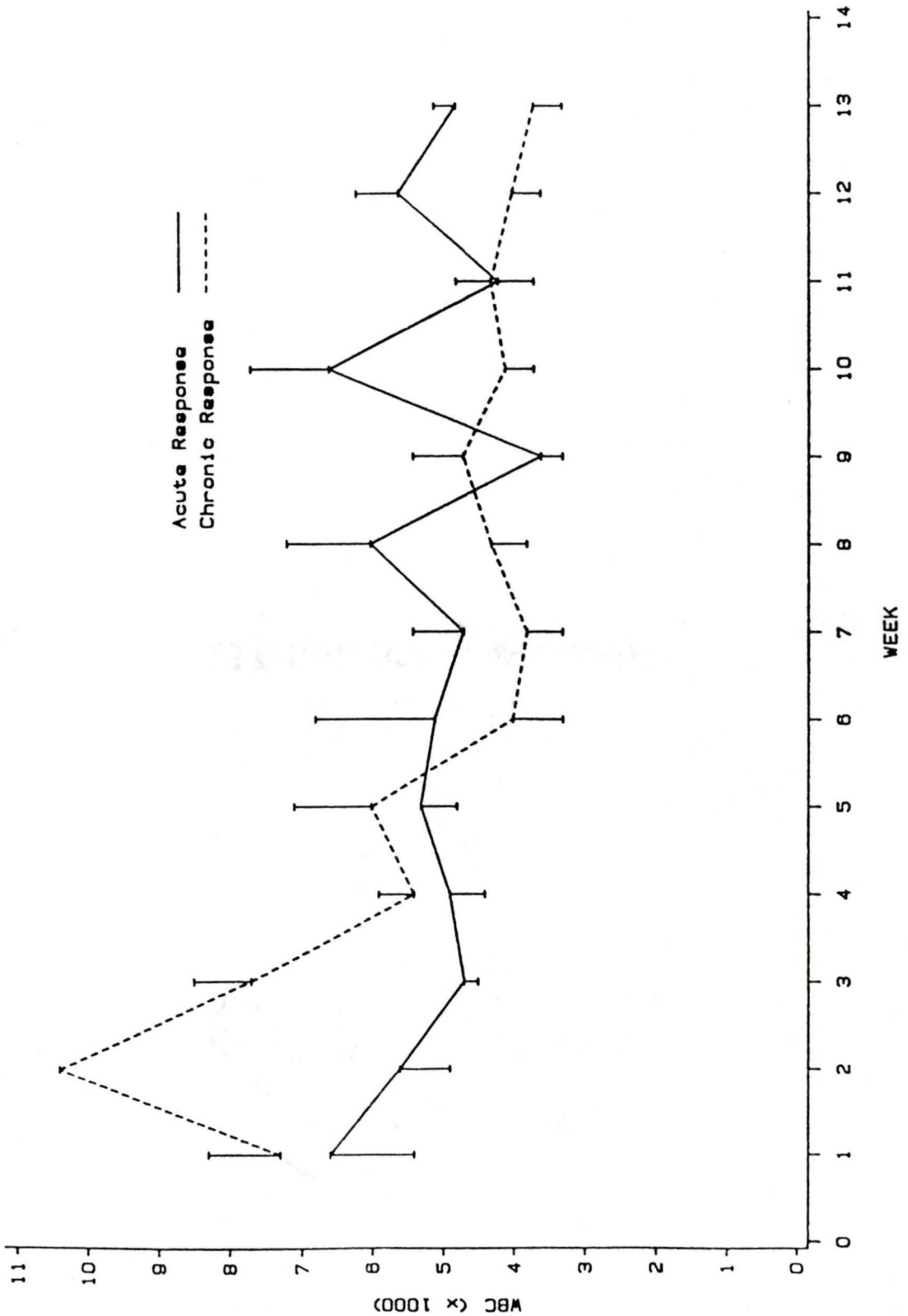

16
15
14
13
12
11
10
Hb g.dl-1
Acute Response
Chronic Response
0
1
2
3
4
5
6
7
8
9
10
11
12
13
14
WEEK

Figure 4: Mean WBC Values During the Control Period and the Progressively More Difficult Training Program


Acute Response
Chronic Response
WBC (x 1000)
WEEK
0
1
2
3
4
5
6
7
8
9
10
11
12
13
14

109.0 ± 1.3 $U \cdot l^{-1}$). The greatest difference between the 2 responses was a 49.0 % higher level during weeks 5-7 in the mean acute response of 253.7 ± 2.3 $U \cdot l^{-1}$ compared to the chronic level 129.9 ± 1.2 $U \cdot l^{-1}$ ($p<0.05$).

The PT (Figure 6), showed that all subjects improved over the 13 weeks with the greatest improvement between weeks 4-7 ($p<0.05$). Weeks 8-13 showed some fluctuation in mean PT scores but remained considerably higher than the pre-control values of week 1.

The degree of correlation between the variables are shown in Table 4. These values indicate a low degree of commonalty between the variables measures and a poor predictive relationship over the 13 weeks.

Figure 5: Mean CPK Values During the Control Period and the Progressively More Difficult Training Program

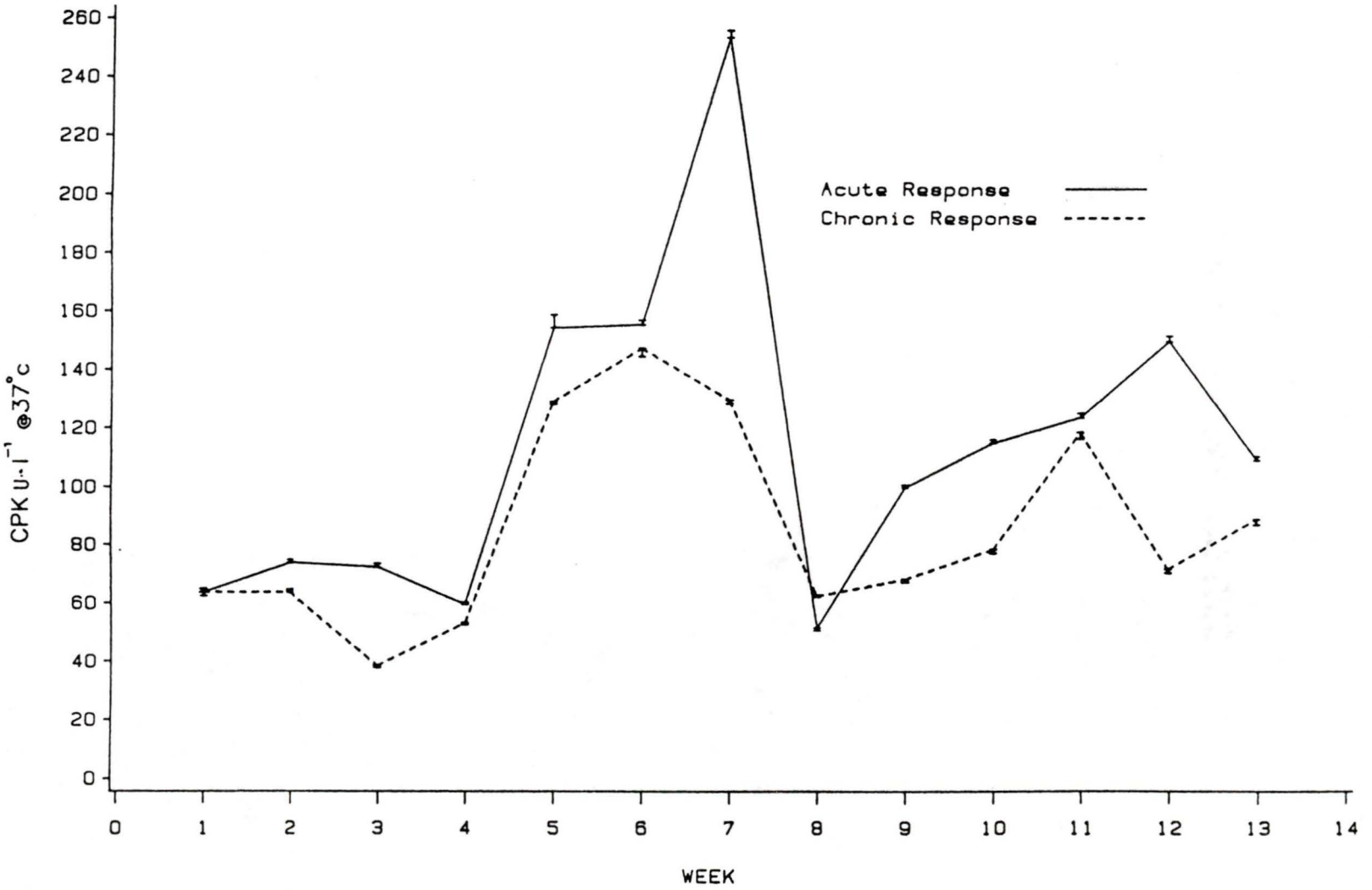
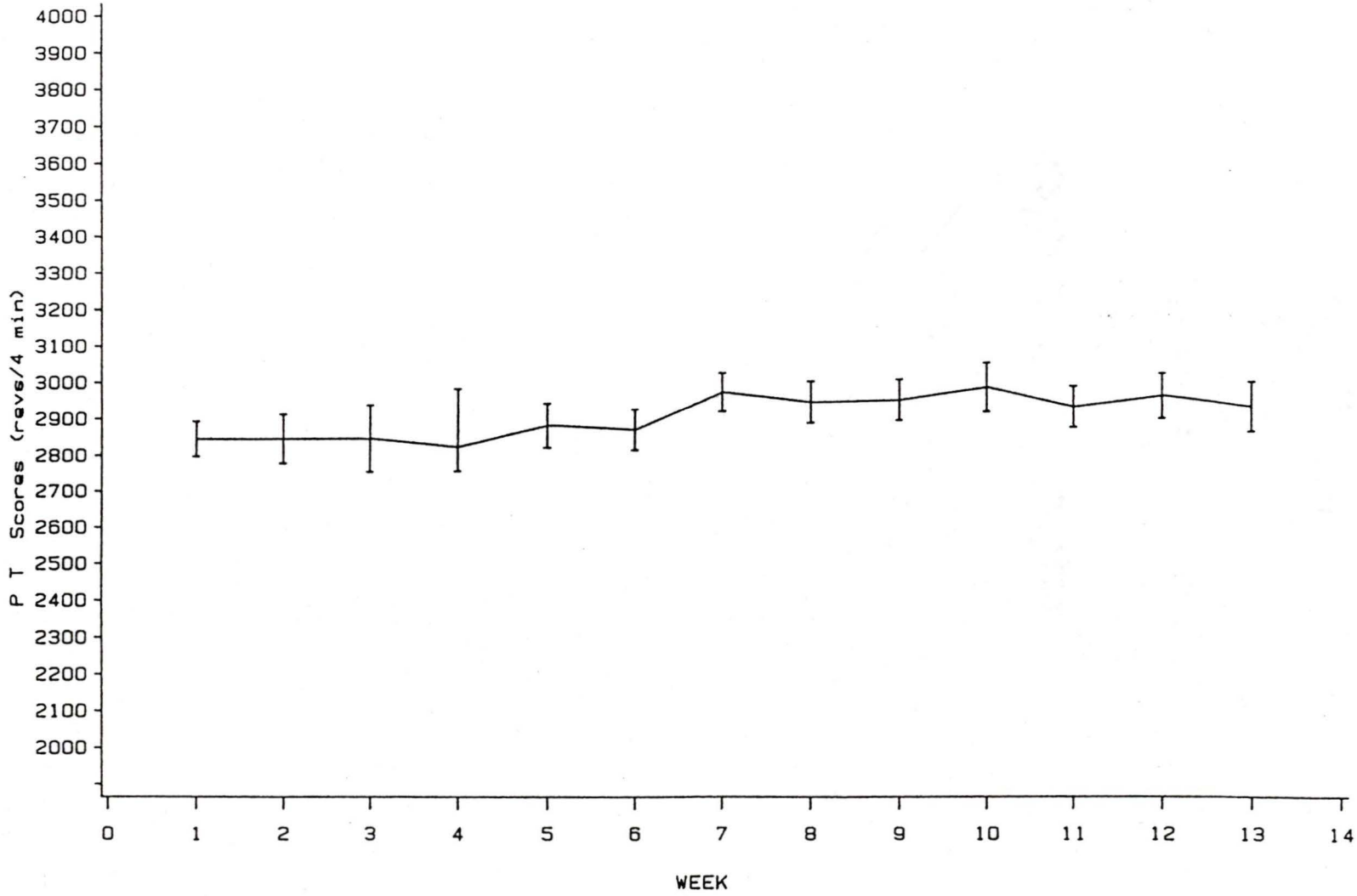

Acute Response
Chronic Response
CPK U·l⁻¹ @37°c
WEEK
0
20
40
60
80
100
120
140
160
180
200
220
240
260
0
1
2
3
4
5
6
7
8
9
10
11
12
13
14

Figure 6: Mean PT Values During the Control Period and the Progressively More Difficult Training Program


4000
3900
3800
3700
3600
3500
3400
3300
3200
3100
3000
2900
2800
2700
2600
2500
2400
2300
2200
2100
2000
P T Scores (revs/4 min)
0
1
2
3
4
5
6
7
8
9
10
11
12
13
14
WEEK

Chapter IV

DISCUSSION

Co-operation in the study by the athletes and their coaches was very good. Motivation was maintained at a high level by frequent meetings and discussion of the results. Compliance in reporting training data, and in conducting regular criterion PT, and attendance for blood sampling by every subject was excellent.

The acute and chronic response in the 4 variables employed were similar at the start of the training program (week 5). The six subjects who participated were of similar rowing experience, functional capacities (as measured by the $\dot{V}_{O2}max$ and the PT), age and anthropometric data.

The control period coincided with final exams and late night studying. This package of stressors required considerable emotional and adaptive demands which may account for the control period fluctuations in the hematological responses and the low $\dot{V}_{O2}max$ values at the beginning of the control period.

The 6.4 % increase in $\dot{V}_{O2}max$ over the control period (Table 3) may be the result of a learning effect as none of the subjects had experienced a $\dot{V}_{O2}max$ test on the rowing ergometer prior to this study. Secondly, the subjects were instructed not to compensate for the absence of a training program during the control period with more intense physical exercise and during this period training was not supervised.

$\dot{V}_{O2}$max showed a 4.1 % improvement during the 8 week training program. Similiar investigations involving endurance training (Coyle et al., 1986; Ekblom et al., 1968) have showed that an increase in $\dot{V}_{O2}$max was due mainly to an enlarged stroke volume (SV) and maximal cardiac output resulting in more oxygen transported arterially during maximal exercise. The aerobic capabilities of these oarsmen are comparable with other oarsmen in other countries using a rowing ergometer (Cunningham et al., 1975: USA, 4.69 $l \cdot min^{-1}$).

The mean acute and chronic Hb responses during the pre-control, control and training periods (Figure 3) were below the average for the Canadian male population (16.0 $g \cdot dl^{-1}$; range 14-18 $g \cdot dl^{-1}$) (Clement et al., 1977; Pate, 1983). The levels in this study are considered low because several authorities have proposed that athletes should have Hb higher than the normal (Bevegard et al., 1963; Clement et al., 1977 Kjellberg et al., 1949).

During the first 4 weeks of training (weeks 5-8) an initial decline in mean Hb response occurred. Reports on the iron status of athletes have documented low Hb values ("sports anemia") in those involved in heavy endurance training and with an absence of disease (Davis & Brewer, 1935; Dressendorfer et al., 1981; Lindemann, 1978; Oscai et al., 1968; Pate, 1983; Williamson, 1981; Yoshimura, 1970). Since the clinically accepted criterion for anemia has been a Hb concentration below 14 $g \cdot dl^{-1}$ in men (Wintrobe, 1981), sports anemia becomes a clinical anemia in an athlete (Pate, 1983). It can occur when the athlete experiences an increased destruction of erythrocytes (RBC) from the acute stress of exercise (Clement & Sawchuk, 1984; Ekblom et al., 1972). This can result from a weakening of the structural integrity of the cell membrane, possibly due to the

trauma of increased circulatory rate, increased body temperature and compression of the RBC by muscular activity and acute exercise acidosis (Clement et al., 1984). Yoshimura (1970), quoted evidence which suggested during strenuous muscular exercise, adrenalin secretion is elevated and the contraction of the spleen releases a hemolysing factor which increases the fragility of the RBC and initiates sports anemia. This anemia which occurs in response to the acute training process could be an adaptation to training and therefore advantageous to the athlete. Yoshimura (1970) states that the destruction of RBC allows the protein from Hb to be used to "promote growth or hypertrophy of the muscles and regeneration of new strong RBC".

Another contributing cause of sports anemia is the increase in blood volume (BV) that occurs with training. Several investigators (Dill & Costill, 1974; Oscai et al., 1968; Brotherhood et al., 1975), suggested that this anemia represents an apparent, rather than absolute anemia, and is the result of exercise induced increases in total BV without an equivalent rise in the number of RBC. Oscai et al., (1968) found that BV increased 6 %, total body Hb was unchanged, and Hb concentration decreased from 15.2 to 14.4 $g \cdot dl^{-1}$ over 16 weeks of running and Brotherhood et al., (1975) demonstrated a significant increase in plasma volume (PV) and total Hb content in a group of runners with no apparent increase in Hb concentration. Thus a lower Hb concentration does not rule out an equal or higher Hb content in the body.

The accumulated training up to week 8 produced a slight drop in the PT and both of the Hb responses attained their lowest values and their largest decreases during this period where the acute Hb response dropped down to 12.9 ± 0.2 $g \cdot dl^{-1}$

from 13.7 ± 0.5 $g \cdot dl^{-1}$ and the chronic response dropped down to 13.3 ± 0.4 $g \cdot dl^{-1}$ from 14.8 ± 0.2 $g \cdot dl^{-1}$. Clement & Sawchuck, (1984) have stated that even with only minor decrements in Hb, anemic subjects have shown impaired physical performance. However, sports anemia would not be expected to affect power output until Hb dropped to less than 12.0 $g \cdot dl^{-1}$ and previous research has used a criterion measure of ± 2.5 $g \cdot dl^{-1}$ to indicate whether changes in values were noteworthy (Rushall & Busch, 1980). Since the difference between the chronic and acute values was not of this magnitude and nor did they go as low as 12.0 $g \cdot dl^{-1}$, the PT were not significantly impaired.

Hb rose during weeks 9-11 as compared to weeks 5-8 and the control period. This suggested that cumulative endurance training can increase the mean Hb concentration and that a transient anemia, as seen in weeks 5-8, may occur at the onset of endurance training.

A difference of only 0.3 % occurred during weeks 11-13 between the mean chronic and acute Hb response. This suggested that endurance athletes will reach and maintain a steady state in the middle to low levels of the normal Hb range. The mean Hb concentration at week 13 was 14.6 ± 0.5 $g \cdot dl^{-1}$ which is clearly within the normal range but slightly below the male population mean.

The chronic response showed higher mean values than the acute response in 11 of the 13 weeks. This could have been due to the minimum 24 hour rest period which re-established the losses of RBC during the exercise or when the chronic response were sampled. Changes in Hct have often been used to indicate the degree of hemodilution or hemoconcentration of the blood in healthy individuals (Senay et al., 1965 and Poortmans, 1964). Intense high level endurance training is

typically reflected by an increase in PV (Williamson, 1981; Lindemann, 1978; Ross et al., 1983) and lower Hct levels which mimic anemia (Maron et al., 1975, 1977). These lower Hct levels are associated with normal levels of RBC or even slightly above normal but diluted in a larger PV.

During the first 4 weeks of training (weeks 5-8), a hemodilution effect occurred with a decrease of (3.0 %) in the acute response and a slightly larger change (4.0 %) in the chronic response from the pre-control period. The total BV closely relates to the level of conditioning and that trained individuals usually have a naturally expanded BV (Brotherhood et al., 1975, Coyle et al., 1986; Dill & Costill, 1974). Therefore, it would be difficult to produce a large increase in BV in individuals who are already in good condition with exercise induced hypervolemia already present (Oscai et al., 1968).

A decrease in Hct occurred during weeks 5-8 which was likely due to an increase in PV whereas an increase in Hct and Hb occurred during weeks 9-11 which was probably due to a maintenance of BV and an increase in RBC or Hb production. Thus Hct would increase. Most studies have demonstrated that PV like BV is decreased as a result of the repeated exercise loads causing Hct and Hb concentration to increase (Costill & Fink et al., 1974; Greenleaf et al., 1977; Novosadova, 1977). This hemoconcentration effect may also be partially attributed to environmental influences towards the end of the study which increased sweating and dehydration of the subjects and which possibly decreased the PV.

A comparison of the results of this study following the acute and chronic exercise indicates the importance of work intensity in stimulating the

hypervolemic response during repeated days of exercise. Maximal exercise intervenes more deeply and stimulates outward movement of fluid from the vascular space resulting in a decrease in PV (Convertino, 1980) thereby affecting the magnitude of the Hct.

The importance of Hb and Hct concentration for human physical performance, has been discussed (Astrand & Rodahl, 1977; Kjellberg et al., 1949). An optimal Hb concentration for athletes requires consideration of the relationship between Hct and blood viscosity. Hct is highly correlated with Hb concentration (Pate, 1983) and this study showed a correlation of r=0.571. Exceptionally high Hct, such as those in polycythemia, can result in a reduced oxygen delivery to the peripheral tissues because of the high oxygen carrying capacity of the blood is more than offset by a reduced rate of blood flow. The optimal Hct has been suggested to be near the upper end of the normal range; i.e. near 45 % (Stone et al., 1968). However, this generalization has been challenged. Studies on experimental enhancement of Hb concentration by removal and subsequent reinfusion of RBC (blood doping) have indicated that such treatments are associated with increases in maximal aerobic power and endurance performance (Ekblom et al., 1972). Recent animal model investigations indicate that an optimal Hct for oxygen delivery during exercise may be high as 50 % (Buick et al., 1980). The mean acute Hct value in this study was 47.8 ± 0.8 % and the mean chronic value for over the 13 weeks was 49.2 ± 0.9 %. Both of these values are closer to the upper end of the normal range and they are in agreement with Astrand and Rodahl, (1977) norms for trained athletes.

Many would assume that any decrease in Hb or Hct would inhibit performance because it would retard oxygen delivery to the tissues. This study was not able to show a decrease in the PT through changes in the level of Hb and the correlation was low (r=0.120). This may be because there is no relationship or because a correlation with a low N and a homogeneous group will likely be low. However, the increase in performance was related (r=-0.340) to the decreased Hct in spite of the low N and homogeneity of the sample.

The total amount of circulating Hb correlates well with maximal oxygen uptake (Ekblom et al., 1972) and a physiological increase in Hb concentration is associated with an increased capacity for physical endurance (Astrand & Rodahl, 1977; Kjellberg et al., 1949; Oscai et al., 1968). This in turn may indicate a readiness for maximal physical performance (Clement et al., 1977) and is also associated with reaching a peak in physical training (Falsetti, 1983).

A summary of the results has shown that the acute and chronic reponses of Hb and Hct elicit similar changes to repeated bouts of training. It would appear that both Hb and Hct had risen to optimal levels by the end of this study which increased the ability of their aerobic systems to support aerobic work. The increase in the PT and the improvement in the $\dot{V}_{O2}$max of the oarsmen that resulted from an 8 week vigorous training program is an example of a long term adaptation to increased chronic stress. Physiologically, no decrement in performance output was shown with such a training program. This latter effect would be advantageous to athletes whose performance requires such loads. It is therefore possible that the subjects in this study may have reached their physical peak during the training period which would be desirable for serious competition.

The highest mean values in WBC counts, in both the chronic (7356 ± 624.0) and the acute (5424 ± 856.0) conditions occurred during the control period. These elevated values may be indicative of the "package of stressors" that accompanied the subjects during the control period. During the training period the mean values of both WBC responses were at the low end of the normal male range (4000-11000), and 38.0 % lower than the pre-control values. Similiar results (a decrease of 15 %) were reported by Dressendorfer et al., (1981) in his study of 12 male endurance runners.

WBC counts showed weekly fluctuations in the acute response to training. Physical exercise is said to have a rapid effect on circulating WBC causing an increase in both short term and endurance exercise (Burke et al., 1985). This phenomena is commonly cited in the literature (Dressendorfer et al., 1981; Lamb, 1978; Guyton, 1981) and is usually explained as an effect of increased circulation during exercise; a "washing out" of the WBC, specifically the neutrophils, which adhere to the walls of the capillaries. This increased rapid flow of blood through the capillaries, pulls the WBC off the walls, mobilizing them through the circulation. The WBC count will return to its normal value within 1-2 hours after completing the physical exercise because the majority of them will again be sequestered in the capillaries (Guyton, 1981; Lamb, 1978).

Therefore, what appears to be an increase in the production of WBC due to inflammation or an increase in the training load as shown by the small amplitude changes in the acute response, is merely an increase in the amount of circulating WBC after a hard workout. This study was not able to show a decrease in PT with an increase in the number of WBC as shown by the low correlation (r=-0.218).

CPK activity both at rest (the chronic response) and after exercise (the acute response) has been the subject of considerable interest. The mean chronic values for CPK during this study fall within the ranges established for upper limits of the normal population, which are between 10-100 $U \cdot l^{-1}$ (Rutledge et al., 1978). The increase of serum enzyme activities tends to be maintained for several hours or even days after exercise, particularly in the case of CPK (Fowler et al., 1962; Rose et al., 1970; Gimenez & Florentz, 1984; Haralambie et al., 1976). This may contribute to the "chemically" higher chronic value observed during the training as compared to the control period. Elevated serum CPK has been attributed to a variety of causes. In downhill running it has been suggested (Schwane et al., 1983) that prolonged repeated impact on hard surfaces contributes to a substantial elimination of intracellular CPK. Rowing is characterized not only by an absence of such trauma but also by reduced force production. Forces of 130-275 kg are reportedly exerted during the striking phase of running (Bates' study cited in Symanski et al., 1983). The magnitude of the force that is needed to develop dynamic strength in rowing is equal to about 84.0 kg (Secher, 1983) which is considerably less than at foot strike. Energy demands and forces placed on an oarsmen are somewhat different than those placed on other endurance athletes. Rowing utilizes arm, back, and leg muscles whereas running involves the use of the legs almost entirely. Additionally, rowing is a weight-supported activity in contrast to a non weight-supported activity such as marathon running (Cunningham et al., 1975). Exercise induced muscle soreness is known to be greater in muscles which were lengthened while they were active (eccentric contractions) than in muscles which were allowed to shorten (concentric

contractions), (Assmussen 1956; Edwards & Harrison, 1984) This may be why higher levels of CPK are found in the landing phase of running activities due to the amount of muscle damage that occurs with eccentric contractions as opposed to rowing which involves concentric contractions.

During weeks 5-7 a 4 fold increase in the acute (253.7 ± 2.4 $U \cdot l^{-1}$) and a 2.5 fold increase in the chronic (147.7 ± 2.7 $U \cdot l^{-1}$) levels of CPK from the pre-control value (63.9 ± 1.1 $U \cdot l^{-1}$) occurred. Since this was the onset of training, these levels reflect a substantial increase in muscle load. During the weeks 8-11 the level of CPK decreased as the muscles began to tolerate the exercise loads. Following a marathon run, plasma CPK peaks some 6-24 hours after the end of the run (Young, 1984). Siegel et al., (1981) have found the largest increase in CPK, of 20 fold, following marathon running at 8-24 hours post exercise. Similar findings have been observed after cycling (Ahlborg & Brohult, 1967), crosscountry skiing (Stromme et al., 1978), bobsledding (Haralambie et al., 1976), and rowing (Hansen et al., 1982). If our samples of CPK were assessed at 6, 18, and 24 hours, it is possible that the magnitude of change for CPK may have been even higher (Brynes et al., 1985).

Muscle fibre type has also been implicated with CPK efflux. Type 1 (slow twitch) fibres in contrast to type 2 (fast twitch) fibers are characterized by a high oxidative capacity and lower CPK activity (Haralambie, 1978). Muscle samples obtained from rowers have revealed that they have approximately 70 % slow twitch fibres and the presence of very few fast-twitch fibres (Hagerman & Staron, 1983; Larsson & Forsberg, 1980). This inability to elevate serum CPK following rowing workouts may be the result of the reduced mechanical trauma or a smaller number of type 2 fibres utilized during the activity of rowing.

Other studies have reported that following an exercise training program, the serum CPK response was reduced (Hunter & Critz, 1971; Maxwell & Bloor, 1981). Since these studies used exercises requiring less of an eccentric component, a direct comparison with the present study is quite possible. The mechanism responsible for lower CPK following training is not apparent (Fox et al., 1977). There are several possibilities, one of which is the state of physical conditioning of the athlete and two, the susceptibility to skeletal muscle insult which subsequently may limit the degree of CPK release.

The enzyme response in the present study may reflect exceptional training status of the oarsmen. Trained skeletal muscle has an increased availability of ATP which may better maintain the integrity of the cell membrane during work and thus reduce enzyme efflux (Hunter & Critz, 1971). When an athlete starts a specific exercise load his CPK may reach 250 $U \cdot l^{-1}$ and then, when he becomes better trained, the same load may elicit only 200 $U \cdot l^{-1}$ (Brown, 1983). The mean value of CPK at week 11 (121.4 ± 1.7 $U \cdot l^{-1}$) was only a 2 fold increase but the intensity and duration of the work loads was much higher than week 7 (Table 5; Appendix 2). Millard et al., (1981) reported a steady decline in mean post-exercise CPK among collegiate swimmers during a 5 month training season, despite progressive increases in yardage.

Rowing performed by highly trained oarsmen at high levels of intensity or prolonged durations may impose sufficient degrees of trauma producing muscular stress to disrupt the structural integrity of the cell membrane. This will result in a loss of intracellular CPK to the bloodstream. The muscles respond to the initial change in CPK from the intense exercise stress and eventually adapt to the

chronic heavy intensity exercise by eliminating smaller amounts of CPK. This study was not able to show decreases in the PT with changes in CPK and the correlation was low (r=0.252). However, CPK levels in venous blood are significantly different in the acute and chronic response to exercise.

In summary, the results of this study have shown that the acute and chronic levels of Hb and Hct are similiar in their response to repeated bouts of training. From the comparison of the levels obtained following acute and chronic exercise it maybe concluded that acute exercise intervenes more severely in the PV affecting the Hct. The acute response shows a higher Hct than is found in the chronic response and this supports a decrease in PV. The onset of heavy training can cause a transient anemia during the initial weeks due to the chronic increase in PV. This anemic process could be one of adaptation and is advantageous to the athlete. Therefore during chronic exercise the Hb concentration is maintained at a steady state in the middle to lower levels of the normal range.

The largest increase in both the acute and chronic levels of CPK occurred in the first weeks of training and may be attributed to the acute stress of the exercise load and not to muscle damage. Following this response the muscle adapted to the CPK through increased permeability which resulted in smaller amounts of CPK being eliminated.

Increasing WBC is an effect of circulation during exercise and has no special benefit towards the performance of the athlete. The response of WBC to training does not seem to be an effective variable to predict physical fitness.

The improvement in the performance of the oarsmen and in the physical performance capacity that resulted from a 13 week vigorous training program is

an example of a long term adaptation to increased chronic stress. The low values of Hb and Hct are normal for highly trained athletes and thus sports anemia does not produce a decrement in the performance of these athletes during a progressively more difficult training program.

In conclusion, Hb, Hct and CPK could provide, on the basis of this study reliable information on physical performance and could be an index of the response of the oarsmen adapting to varying amounts of exercise load during training. Hct and CPK appear to be the more sensitive of these three variables and an increase in the level of CPK might also be predictive of the level of physical fitness. However, further studies are still necessary for a better understanding of the adaptation of muscle to training and the relationship with the observed blood parameters.

REFERENCES

Ahlborg, B., & Brohult, B. (1967). Capacity for prolonged exercise in man. *Försvarsmedicin*, 3, (Suppl 1.)

Assumssen, E. (1979). Muscle fatigue. *Medicine and Science in Sports*, 11(4), 313-321.

Astrand, P.O., & Rodahl, K. (1977). *Textbook of Work Physiology*. New York: McGraw-Hill Book Company.

Bevegard, S., Holmgren, A. & Jonnson, B. (1963). Circulatory studies in well trained athletes at rest and during heavy exercise with special reference to stroke volume and influence of body position. *Acta Physiologica Scandinavica*, 57, 43-50.

Brotherhood, J., Brozovic, B., & Pugh, L.G. (1975). Hematological studies of middle and long distance runners. *Clinical Science and Molecular Medicine*, 48, 139-145.

Brown, R.L. (1983). Overtraining of athletes. *The Physician and Sportsmedicine*, 11(6), 93-110.

Burke, E.R., Falsetti, H.L., Feld, R.D., Patton, G.S., & Kennedy, C. (1985). Blood testing to determine overtraining in swimmers. In J. Joy (Ed.), *Coaching Notes*, Canadian Amateur Rowing Association, 5(1), 44-46.

Brynes, W.C., Clarkson, P.M., White, J.S., Hsieh, S.S., Frykman, P.N. & Maughan, R.J. (1985). Delayed onset muscle soreness following repeated bouts of downhill running. *Journal of Applied Physiology*, 59(3), 710-715.

Buick, F.J., Gledhill, N., & Frosse, A.B. (1980). Effect of induced erythrocytes on aerobic work capacity. *Journal of Applied Physiology*, 48, 636-642.

Clement, D.B., Asmundson, R.C., & Medhurst, C.W. (1977). Hemoglobin values: a comparative survey of the 1976 Canadian Olympic team. *Canadian Medical Association Journal*, 177, 614-617.

Clement, D.B. & Sawchuk, L.L. (1984). Iron status and sports performance. *Sports Medicine*, 1, 65-74.

Convertino, V.A., Brock, P.J., Kiel, L.C., Bernauer, E.M., & Greenleaf, J.E. (1980). Exercise training-induced hypervolemia role of plasma albumin, renin and vasopressin. *Journal of Applied Physiology*, 48(4), 665-669.

Convertino, V.A., Kiel, L.C., Bernauer, E.M. & Greenleaf, J.E. (1981). Plasma volume, osmolality, vasopressin, and renin activity during graded exercise in man. *Journal of Applied Physiology*, 50(1), 123-128.

Costill, D.L., & Fink, W.J. (1974). Plasma volume changes following exercise and thermal dehydration. *Journal of Applied Physiology*, 37, 521-525.

Costill, D.L. (1977). Sweating: its composition and effects on body fluids. *Annals of New York Academy of Science*, 301, 160-174.

Coyle, E.F., Hemment, M.K., & Coggan, A.R. (1986). Effects of detraining on cardiovascular responses to exercise: role of blood volume. *Journal of Applied Physiology*, 60(1), 95-99.

Crowell, J.W., & Smith E.E. (1967). Determinant of the optimal hematocrit. *Journal of Applied Physiology*, 22(3), 501-504.

Cunningham, D.A., Goode, P.B., & Critz J.B. (1975). Cardiorespiratory response to exercise on a rowing and bicycle ergometer. *Medicine and Science in Sports*, 7(1), 37-43.

Davis, J.E., & Brewer, N. (1935). Effect of physical training on blood volume, hemoglobin, alkali reserve and osmotic resistance of erythrocytes. *American Journal of Physiology*, 13,586-591.

DeWijn, J., DeJongste, J.L., Mosterd, W., & Willebrand, D., (1971). Hemoglobin, packed cell volume, serum iron and iron binding capacity of selected athletes during training. *Nutrition and Metabolism*, 13, 126-139.

Dill, D.B, & Costill, D.L. (1974). Calculation of percentage change in volumes of blood, plasma, and red cells in dehydration. *Journal of Applied Physiology*, 37, 247-248.

Drabkin, D.L. (1948). The standardization of hemoglobin measurement. *American Journal of the Medical Sciences*, 215, 110-111.

Drabkin, D.L. (1949). The standardization of hemoglobin. *American Journal of the Medical Sciences*, 217, 710-711.

Dressendorfer, R.H., & Wade, C.E. (1983). The muscular overuse syndrome in long distance runners. *The Physician and Sportsmedicine*, 11(11), 116-130.

Dressendorfer, R.H. Wade, C.E., & Amsterdam, E.A. (1981). Development of pseudoanemia in marathon runners during a 20-day road race. *Journal of the American Medical Association*, 246, 1215-1218.

Dufaux, B., Hoederath, A., Streitberger, L., Hollmann, W., & Assman, G. (1981). Serum ferritin, transferrin, haptoglobin, and iron in middle and long distance runners, elite rowers and professional cyclists. *International Journal of Sports Medicine*, 2(1), 43-46.

Durnin, J.V., & Womersley, J. (1974). Body fat assessed from total body density and its estimation from skinfold thickness; measurements on 481 men and women aged 16-72 years. *British Journal of Nutrition*, 32, 77-79.

Edwards, R.J., & Harrison, M.H. (1984). Intravascular volume and protein responses to running exercise. *Medicine and Science in Sports and Exercise*, 16(3), 247-255.

Ehn, L., Carlmark, B., and Höglund, S. (1980). Iron status in athletes involved in intense physical activity. *Medicine and Science in Sport and Exercise*, 12(1), 61-64.

Ekblom, B., Astrand, P.O., Stenberg, B., & Wallström, B. (1968). Effect of training on circulatory response to exercise. *Journal of Applied Physiology*, 24, 518-528.

Ekblom, B., Goldberg, A., & Gullibring, B., (1972). Response to exercise after blood loss and reinfusion. *Journal of Applied Physiology*, 28, 175-185.

Falsetti, F.G. (1983). Overtraining of athletes. *The Physician and Sportsmedicine*, 11(6), 93-110.

Fowler, W.M., Chowdhury, S.R., Pearson, C.M., Gardner, G., & Bratton, R. (1962). Changes in serum enzyme levels after exercise in trained subjects. *European Journal of Applied Physiology*, 17, 943-946.

Fox, E.L., Bartels, R.L., Klinzing, J. & Ragg, K. (1977). Metabolic responses to interval training programs of high and low power output. *Medicine and Science in Sports*, 9(3), 191-196.

Gimenez, M., & Florentz, M. (1984). Serum enzyme variations in man during an exhaustive "square-wave" endurance exercise test. *European Journal of Applied Physiology*, 52, 219-224.

Greenleaf, J.E., Convertino, V.A., Stremel, R.W., Bernauer, E.M. Adam, W.C., Vignau, S.R., & Brock, P.J. (1977). Plasma {Na+}, {Ca+}, and volume shifts and thermoregulation during exercise in man. *Journal of Applied Physiology*, 43, 1026-1032.

Guyton, A.C. (1981). *Textbook of Medical Physiology*, (6th ed., p. 71). Saunders Co.: Philadelphia.

Hagerman, F.C. (1984). Applied physiology of rowing. *Sports Medicine*, 1, 303-321.

Hagerman, F.C., & Howie, G.A. (1966). Use of certain physiological variables in the selection of 1967 New Zealand crew. *Research Quarterly*, 42(3), 264-273.

Hagerman, F.C., Hagerman, G.R., & Mickelson, T.C. (1979). Physiological profiles of the elite rowers. *The Physician and Sportsmedicine*, 7(7), 74-83.

Hagerman, F.C. & Staron, R.S. (1983). Seasonal variations among physiological variables in elite oarsmen. *Canadian Journal of Applied Sport Sciences*, 8, 143-148.

Hansen, K.N., Bjerre-Knudsen, J., Brodthagen, U., Jordal, R., & Paulev, P.E. (1982). Muscle cell leakage due to long distance training. *Journal of Applied Physiology*, 48, 177-188.

Haralambie, G.B. (1978). Creatine kinase and hexose phosphate isomerase activity in skeletal muscles of healthly male athletes. *Enzyme*, 23.

Haralambie, G.B., Cerny, F.J., & Huber, G. (1976). Serum Enzyme Levels after bobsled racing. *Journal of Sports Medicine & Physical Fitness*, 16, 54-56.

Hunter, B.J., & Critz, J.B. (1971). Effect of training on plasma enzyme levels in man. *Journal of Applied Physiology*, 31(1), 20-23.

Kjellberg, S.R., Rudhe, U., & Sjöstrand, T. (1949). Increase of the amount of hemoglobin and blood volume in connection with physical training. *Acta Physiologica Scandinavica*, 19, 146-151.

Lamb, D.R. (1978). *Physiology of Exercise*, New York: Macmillan Publishing Company.

Lamb, D.R. (1984). *Physiology of Exercise Responses & Adaptations*, New York: Macmillan Publishing Company.

LaPorta, M.A., Linde, H.W., Bruce, D.L., & Fitzsimons, E.J. (1978). Elevation of creatine phosphokinase in young men after recreational exercise. *Journal of the American Medical Association*, 129, 2685-2686.

Larsson, L., & Forsberg, A. (1980). Morphological muscle characteristics in rowers. *Canadian Journal of Applied Sport Sciences*, 5, 239-244.

Lindemann, R. (1978). Low hematocrit during basic training; athlete's anemia? (letter). *New England Journal of Medicine*, 229, 1191-1192.

Magazanik, A., Shapiro, Y., Meytes, D., & Meytes, I. (1974). Enzyme blood levels & water balance during a marathon race. *Journal of Applied Physiology*, 36(2), 214-217.

Maron, M.B., Horvath, S.M., & Wilkerson, J.E. (1975). Acute blood biochemical alterations in response to marathon running. *European Journal of Applied Physiology*, 34, 173-181.

Maron, M.B., Horvath, S.M., & Wilkerson, J.E. (1977). Blood biochemical alterations during recovery from competition marathon running. *European Journal of Applied Physiology*, 36, 231-238.

Martindale, W.O., & Robertson, D.G. (1984). Mechanical energy in sculling and in rowing an ergometer. *Canadian Journal of Applied Sport Sciences*, 9(3), 153-163.

Maxwell, J.H., Bloor, C.M. (1981). Effects of conditioning on exertional rhabdomyolysis and serum creatine kinase after severe exercise. *Enzyme* 26, 177-181.

Millard, M.L., Zauner, C.W., & Cade, R.J. (1981). Effects of intense training on blood parameters in national class collegiate swimmers. *Medicine and Science in Sports and Exercise*, 13(2), 110.

Misner, J.E. Massey, B.H., & Williams, B.T. (1973). The effect of physical training on the response of serum enzymes to exercise stress. *Medicine and Science in Sports and Exercise*, 5(2), 86-88.

Nanji, A.A. (1983). Serum creatine kinase isoenzymes: A review. *Muscle and Nerve*, 6, 83-90.

Novosadova, J. (1977). The changes in hematocrit, hemoglobin, plasma volume and proteins during and after different types of exercise. *European Journal of Applied Physiology*, 36(3), 223-230.

Nowacki, P., Krause, R., & Adam, K. (1969). Maximal oxygen uptake by a rowing crew winning Olympic gold medal 1968. *Pflugers Archives*, 312, R66.

Nutrition Canada. (1975). *British Columbia Survey*, Ottawa: Health and Welfare Canada.

Oscai, L.B., Williams, B.T., & Hertig, B.A. (1968). Effect of exercise on blood volume. *Journal of Applied Physiology*, 24(5), 622-624,

Pate, R.R. (1983). Sports Anemia: a review of the current research literature. *The Physician and Sportsmedicine*, 11(2), 115-131.

Poortmans, J.R. (1964). Serum protein determination during short exhaustive physical activity. *Journal of Applied Physiology*, 19, 1125-1132.

Prampero, P.E., Cortili, D.I., & Certelli, P. (1971). Physiological effects of rowing. *Journal of Applied Physiology*, 31, 852- 857.

Rhodes, E.C., Mosher, R.E., McKenzie, D.C., Franks, I.M., Potts, J.E., & Wenger, H.A. (1986). Physiological profiles of the Canadian Olympic Soccer Team. *Canadian Journal of Applied Sport Sciences*, 11(1), 31-36.

Rose, L.T., Bousser, J.E., & Cooper, K.H. (1970). Serum enzymes after marathon running. *Journal of Applied Physiology*, 29(3, 355-357.

Ross, J.H., Attwood, E.C., Atkin, G.C., & Villar, R.N. (1983). A study of the effects of severe repetitive exercise on serum myoglobin, creatine kinase transaminases, and lactate dehydrogenase. *Quarterly Journal of Medicine, New Series L11*, 206, 268-279.

Rowell, L.B. (1974). Human cardiovascular adjustments to exercise and thermal stress. *Physiological Reviews*, 54(1), 75-159.

Rushall, B.S., & Busch, J.D. (1980). Hematological responses to training in elite swimmers. *Canadian Journal of Applied Sports Science*, 5(3), 164-169.

Rutledge, J., Clayson, K.J., & Strandjord, P.E. (1978). Effect of physical conditioning on serum kinase after exercise. *Journal of American Medical Association*, 240(24), 2633.

Sanders, T.M. and Bloor, C.M., (1975). Effects of endurance exercise on serum enzyme activities in well conditioned males. *Medicine and Science in Sports and Exercise*, 7(1), 4-47.

Schwane, J,A., Johnson, S.R., Vandenakker, C.B., & Armstrong, R.B. (1983). Delayed-onset muscular soreness and plasma CPK, and LDH activities after downhill running. *Medicine and Science in Sports and Exercise*, 15(1), 51-56.

Secher, N.H. (1983). The physiology of rowing. *Journal of Sports Sciences*, 1, 23-53.

Senay, L.C., & Christensen, M.L. (1965). Changes in blood plasma during progressive dehydration. *Journal of Applied Physiology*, 20, 1136-1140.

Siegel, A.J., Silverman, L.M., & Holman, L. (1981). Elevated creatine kinase MB isoenzyme levels in marathon runners. *Journal of the American Medical Association*, 246(18), 2049-2051.

Sigma Chemical Company. (1981). *Sigma Technical Bulletin, No45-UV*, Creatine phosphokinase (CPK) in serum, plasma, and cerebrospinal fluid at 340 NM. St. Louis: Missouri.

Stone, H.O., Thompson, H.K., & Schmidt-Nielson, K. (1968). Influence of erythrocytes on blood viscosity. *American Journal of Physiology*, 214, 913-918.

Strydom, N.B., Wyndham, C.H., Greyson, J.S., & Atkins, A.T. (1967). A scientific approach to the selection & training of oarsmen. *South African Medical Journal*, 41, 1100-1102.

Sutton, J. (1971). Pathology in the limitation of performance. *Australian Journal of Sport Medicine*, 4, 6-14.

Symanski, J.D., McMurray, R.G., Silverman, L.M., Smith, B.W., & Siegel, A.J. (1983). Serum creatine kinase and CK-MB isoenzyme responses to acute and prolonged swimming in trained athletes. *Clinica Chimica Acta*, 129, 181-187.

Thomson, W,H., Sweetin, J.C., & Hamilton, I.D, (1975). ATP and muscle energy efflux after physical exertion. *Clinica Chimica Acta*, 59,241-245.

Van Beaumont, W. (1972). Evaluation of hemoconcentration from hematocrit measures. *Journal of Applied Physiology*, 32(5), 712-713.

Wilkerson, J.E. Gutin, B., & Horvath, S.M. (1977). Exercise induced changes in blood, red cell, and plasma volumes in man. *Medicine and Science in Sports*, 9(3), 155-158.

Williamson, M. (1981). Anemia in runners and other athletes. *The Physician and Sportsmedicine*, 9(6), 73-79.

Wintrobe, M.M. (1981). *Clinical Hematology*, Philadelphia: Lea & Feliger.

Yoshimura, H. (1970). Anemia during physical training (sports review). *Nutritional Reviews*, 28(10), 251-253.

Young, A. (1984). Plasma creatine kinase after the marathon-a diagnostic dilemma. *British Journal of Sports Medicine*, 18(4), 269-272.

REVIEW OF LITERATURE

Exercise and Training

The changes in blood after a single bout of exercise (acute training) or after a period of training (chronic training), are not always easily predicted. A wide range of normal values are found for most of the blood variables at rest and the ranges increase after exercise and training. The inconsistent effects of exercise on the blood can be partially attributed to normal variations which occur at rest, to variations in the exercise or training programs, to how the analysis of the blood is conducted, and that all athletes do not response in the same manner (Lamb, 1978).

The blood constituents most often identified to help monitor and tailor training programs are: hemoglobin (Hb), hematocrit (Hct), white blood cells (WBC), and creatine phosphokinase (CPK) (Burke et al., 1985; Falsetti, 1983). Hb is the iron protein substance located in the red blood cell (RBC) and the primary function is transport of oxygen from the lungs to the tissues. Since Hb in the RBC carries oxygen, it is the number of RBC and the amount of Hb in those cells that are important in determining how much oxygen can be carried to the working muscle of the athlete. Serum Hb increases when the RBC are destroyed and in certain diseases. Low levels are recorded in anemia, after large blood loss, and sometimes in trained athletes. Total Hb levels in man range from 14 to 18 grams per decilitre of blood ($g \cdot dl^{-1}$) (Clement et al., 1977; Lamb, 1978; Pate, 1983). Hct is the percentage of RBC in the plasma (the water portion of the blood),

comprising the total blood volume (BV). Low values are seen after excess fluid intake and severe bleeding and in RBC anemia. High concentrations of RBC are seen in athletes after exercise in hot weather. Sweating causes a loss of fluids from the plasma which will increase the concentration of RBC. Normal Hct values for men are 45-55 % (Lamb, 1978; Stone et al., 1968).

WBC are used in fighting infections. The number of WBC increase in response to infections or inflammations and help destroy the causative agents. Physical exercise has a rapid effect on circulating WBC causing an increase in both short term and endurance exercise. Short term exercise results in lymphocytosis, where as endurance exercise leads to a preferential increase in circulating neutrophils. The increased WBC that is often seen after exercise is explained as an effect of greater circulation during exercise, "washing out" the WBC from their storage places. Emotional stress has also been seen to raise WBC (Burke et al., 1985). Normal values range from 4000-10000 per ml of blood (Guyton, 1981; Lamb, 1978).

CPK is a muscle enzyme that is released into the circulation during and after strenuous exercise (Young, 1984). Hard exercise, particularly eccentric contractions, can cause large increases in CPK (Byrnes et al., 1985). This release after exercise is thought to be analogous to the release of cardiac enzymes after an infarct where the magnitude of the release reaches a maximum some 24 hours later (Newham et al., 1983). Similarly, the release of skeletal muscle CPK enzyme is thought to be proportional to the intensity and duration of the exercise (Apple et al., 1985). It should also be noted that high levels of CPK may be due to an accumulation effect (i.e. a delayed enzyme response may increase for successive days) (Haralambie et al., 1976). The normal range is between 10-100 international

units per liter ($U \cdot l^{-1}$) (Rutledge et al., 1978). Clinically significant differences are found between the trained and the untrained muscle; with the untrained muscle having greater rises in CPK than the trained muscle (Byrnes et al., 1985; Fowler et al., 1962). The physiological mechanism allowing the enzyme to enter the blood is currently unclear (Burke et al., 1985; Young, 1984). Increased activity of CPK may be related to transient changes in the permeablity of the membrane of the muscle, allowing a greater flow of the enzyme out of the muscle (Sanders & Bloor, 1975; Thomson, 1975).

Training Studies

Several authors have used an experimental model to observe the effects of heavy training on hematological variables. Yoshimura (1970) has suggested that a transient anemia may occur with the onset of endurance training and this relatively mild, apparently functional pseudoanemia condition has been termed "sports anemia". This hypothesis does not appear to be extensively documented (Pate, 1983). Davis and Brewer, (1935) observed a transient anemia during the first few weeks of endurance training in dogs. In humans, Lindemann (1978) and Oscai et al., (1968) reported reduced Hb concentrations with training. Dressendorfer et al., (1981) observed a substantial decrease in Hb concentrations in runners participating in a 20 day road race, although Glass et al., (1969) reported no change in Hb in cyclists who underwent a marked increase in training load.

Therefore, heavy training may cause a transient anemia during the inital weeks of exercise. In the longer term, Hb concentration in endurance athletes

seem to maintain a steady state in the middle to lower levels of the normal range (Pate, 1983).

Possible Causes of Sports Anemia

Reports of anemia and suboptimal Hb levels in athletes have generated much speculation regarding the causes of sports anemia. First, a relative anemia could develop through a hemodilution effect of an expanded PV. Second, reduced Hb synthesis and/or erythropoiesis could result in an actual decrease in total Hb. Third, an increased rate of destruction of RBC and degradation of Hb could decrease the concentration of Hb in the blood (Ekblom et al., 1972; Pate, 1983; Williamson, 1981).

Blood and Plasma Volume

It is well documented that endurance athletes have a relatively large total blood volume (Brotherhood et al., 1975; Dill & Costill, 1974; Kjellberg et al., 1949) and that endurance training is accompanied by increases in plasma volume (Oscai et al., 1968). Total blood volume is defined as the plasma volume plus the total red cell volume (BV = PV + RCV). Changes in PV and/or changes in RCV therefore will affect BV.

It appears that the relative intensity at which the exercise is performed and the duration of the session are the indices of the volume changes during submaximal work. During maximum (or near maximum) intensity sustained for short periods of time, a decrease in PV occurs resulting in a hemoconcentration effect (Van Beaumont et al., 1972). Joyce and Poortmans (1970) reported a

decrease in PV of 3 % following 90 seconds (s.). high intensity exercise and Rotstein et al., (1982) report a decrease of 11 % after 30 seconds of all out cycling. Thus, the decrease in PV is attributed to an increase in capillary hydrostatic pressure brought about by the increase in arterial pressure during maximal exercise. The major effect of chronic exercise on blood seems to be an increase due primarily to an increase in PV. In studies comparing endurance athletes to sedentary people, the endurance athletes exhibited an increase in BV of 20-30 % (Dill et al, 66; Kjellberg et al., 1949). Studies comparing BV in pre and post-training have found similiar results. In previously sedentary individuals, increases up to 6 % as a result of training occurred, with total Hb unchanged and a decrease in Hb concentration from 15.2 to 14.4 $g \cdot dl^{-1}$ (Oscai et al., 1968). In fit individuals (high $\dot{V}_{O2}max$), training increased BV up to 8 % (Convertino et al., 1980) and may have some implications in work performance. Kanstrup and Ekblom, (1984) have shown that even if Hb concentration was decreased by 8 % due to an increased PV (by plasma expansion), $\dot{V}_{O2}max$ could still be maintained. Thus total Hb may be more relevant to high aerobic power than the Hb concentration. However, a reduced Hb concentration with an elevated blood volume may result in an unchanged $\dot{V}_{O2}max$ but a reduced performance time. Other findings indicate that the physical performance during high intensity exercise is somewhat dependent upon other factors besides $\dot{V}_{O2}max$ (Coyle et al., 1986) such as an increased cardiac output, which would compensate for the reduced arterial oxygen content.

With exercise, plasma is lost from the vascular space to the interstitial fluid, thus decreasing PV. When work intensity exceeds 40 % $\dot{V}_{O2}max$ a threshold is

reached at which the rate of fluid efflux exceeds the rate of solute efflux. This produces a net hypotonic plasma loss and increases the Na+ concentration and osmolality proportionate to the work load. Convertino et al., (1981) indicated that work intensity stimulated the hypervolemic response associated with chronic exercise. An exercise training intensity greater than 40 % $\dot{V}_{O2}$max may be required to stimulate arginine vasopressin (AVP) which is associated with water and salt retention following exercise, and in turn, increases water retention and PV. This was consistent with the observation that exercise training at 65 % $\dot{V}_{O2}$max produces larger AVP and hypervolemic responses than training at 50 % $\dot{V}_{O2}$max (Convertino et al., 1980). Wilkerson et al., (1977) concluded that % PV could not be expressed as a discrete linear function of work intensity over an entire range of exercise but that a definite break occurs at a relative work rate of about 65 % $\dot{V}_{O2}$max.

Destruction of RBC

Some researchers believe that the anemia response is one of adaptation and is therefore advantageous to the athlete. Yoshimura (1970) points out that sports anemia is caused by an increased destruction of RBC which is a result of an increase in fragility due to the acute stress of the exercise. This increase in the destruction of RBC is caused by a liberation of some hemolysing factor from the spleen. When an athlete does strenuous exercise muscular exercise, adrenalin secretion is promoted by the stress and the contraction of the spleen may be accelerated; and release of the hemolysing factor (Yoshimura, 1970). The Hb from the destruction of the RBC could than be utilized for muscle protein

synthesis and new red cells. This adaptive response promotes growth and hypertrophy of the muscle and regeneration of new RBC in response to strenuous physical training.

Changes in Hct have often been used to indicate the degree of hemoconcentration or hemodilution of blood in healthy individuals (Joyce & Poortmans, 1970; Saltin, 1964; Senay et al., 1965). However, direct comparison PV measurements during exercise do not always show concomitant changes in the Hct (Kaltreider & Meneely, 1940; Saltin, 1964). An extreme difference was reported by Saltin, (1964) who found a 25.7 % decrease in PV but only an 8.5 % increase in maximal exercise.

The hemoconcentration that occurs with exercise explains the observed increases in RBC count, Hb concentrations and Hct, all of which reflect the greater concentration of the RBC in a given volume of blood.

Mode of Training

During training, RBC concentrations (Hct ratio) are lowered (Dill & Costill., 1974) but the relative amount of RBC appears unaltered. When the mode or training was running, Ekblom et al., (1972) found no change whereas others have found a small decrease (Dressendorfer et al., 1981). When rowing was the mode of training, Tomaski, (1979) found a slight increase. Although it may be beneficial for endurance athletes to have a greater RCV the research shows that no appreciable change occurs as a result of training.

Impact on Performance

A substantial base of physiological information has suggested that anemia could negatively affect certain types of athletic performance. Hb plays a central role in oxygen transport. Hb concentration determines the oxygen carrying capacity of the blood and this in turn affects oxygen delivery to peripheral tissues. Since the maximal rate of oxygen delivery to the muscles is a principal determinant of maximal aerobic power, anemia could be a determinant in activities that depend on a high rate of aerobic energy yield.

The relationship between endurance performance and Hb concentration is not as clear when only clinically normal Hb values are considered (Pate, 1983). It is well established that total Hb correlates highly with $\dot{V}_{O2}$max in litres per minute. However, these correlations become nonsignificant when $\dot{V}_{O2}$max is expressed relative to body weight ($ml \cdot kg \cdot min^{-1}$) as is typically the case in studies of the functional abilities of the athletes. For example, Vellar and Hermansen, (1971) found no significant relationship between hematological variables (within the normal range) and $\dot{V}_{O2}$max expressed relative to body weight. It may be that in such studies the importance of Hb concentration is obscured by the statistical weight of the many other variables that affect $\dot{V}_{O2}$max. This is suggested by the studies that have used blood reinfusion to increase Hb levels while holding other variables constant. These exercise studies, performed in chronic anemics (Kjellberg et al., 1949) as well as in acutely-induced anemic subjects (Ekblom et al., 1972), have shown reduced maximal oxygen uptake and physical performance. After erythrocyte reinfusion (Ekblom et al., 1972), Hb concentration, $\dot{V}_{O2}$max and physical performance have all been increased.

Therefore, low serum iron values and suboptimal hemoglobin (Hb) values have been demonstrated among athletes, in particular those who are involved in heavy physical training (Yoshimura, 1970). This has been looked upon as a possible factor responsible for suboptimal oxygen transport and hence lower capacity for physical performance since those persons who exercise regularly are expected to have values that are at least average and possibly higher than the average (DeWijn et al., 1971; Kjellberg et al., 1949).

CPK Responses

Some authors have suggested that the magnitude of the increase in serum of CPK levels after exercise depends on the physical condition of the subjects and that training may prevent CPK enzyme release (Sanders & Bloor, 1975). This seems in contrast with the observations of Stromme et al., (1978) and the results of Rose et al., (1970) who showed that highly trained as well as moderately trained cross-country skiers and marathon runners had marked increases in CPK after exertion. Furthermore, Misner et al., (1973), were unable to find any reduction in the exercise induced enzyme increase in a group of subjects who participated in a progressive exercise program 3 times a week for 15 weeks. The study of Dressendorfer and Wade, (1983) of 12 runners averaging 28km per day for 10 days followed by 2 days rest showed an approximately 4 fold increase above the baseline early in the race and it remained 2 times higher than the upper limit of the normal. Serum CPK returned to normal following the two day rest period but became elevated again after running resumed. These studies may suggest that chronic elevation in CPK activity could be an enzymatic marker of exercise

related, muscle injury. Fowler et al., (1962) reported decreases after exercise and marked fluctuation of CPK. However, there was considerable intra-individual variability of CPK at rest and in response to exercise indicating the labile nature of the enzyme. The possibility that a large muscle mass is the factor that is causing the elevation has been suggested because of the difference in fibre type distribution. (Hagerman & Staron, 1983; Larsson & Forsberg, 1980). Fast-twitch fibres are known to contain more CPK than slow-twitch fibres, and the pattern of muscular contraction in different types of exercise might influence the release of CPK. Therefore, this might also cause problems in comparison of data from studies with different experimental conditions.

Many investigators have reported on the changes of serum enzyme activities after various kinds of exercise stress. These results suggest that CPK is the most sensitive index of acute stress in well-conditioned individuals (Critz & Cunningham, 1972; Hunter & Critz, 1971; Fowler et al., 1962; Laporta et al., 1978; Sanders & Bloor, 1975).

REFERENCES

Ahlborg, B. & Brohult, B. (1967). Capacity for prolonged exercise in man. *Försvarsmedicin*, 3, (Suppl 1.)

Alexander, S. (1984). Physiologic and biochemical effects of exercise. *Clinical Biochemistry*, 17(2), 126-131.

Apple, F.S., Rogers, M.A., Casal, D.C. Sherman, W.M., & Ivy, J.L. (1985). Creatine-kinase-MB isoenzyme adaptions in stressed human skeletal muscle of marathon runners. *Journal of Applied Physiology*, 59(1), 149-153.

Assumssen, E. (1979). Muscle fatigue. *Medicine and Science in Sports*, 11(4), 313-321.

Astrand, P.O., & Rodahl, K. (1977). *Textbook of Work Physiology*. New York: McGraw-Hill Book Company.

Bevegard, S., Holmgren, A. & Jonnson, B. (1963). Circulatory studies in well trained athletes at rest and during heavy exercise with special reference to stroke volume and influence of body position. *Acta Physiologica Scandinavica*, 57, 43-50.

Brown, R.L. (1983). Overtraining of athletes. *The Physician and Sportsmedicine*, 11(6), 93-110.

Brodthagen, U.A., Hansen, K.N., Knudsen, J.B., Jordal. R., Kristensen, O., & Paulev, P.E. (1985). Red cell 2,3-DPG, ATP and mean cell volume in highly trained athletes. *European Journal of Applied Physiology*, 53, 334-338.

Brotherhood, J., Brozovic, B., & Pugh, L.G. (1975). Hematological studies of middle and long distance runners. *Clinical Science and Molecular Medicine*, 48, 139-145.

Buick, F.J., Gledhill, N., & Troise, A.B. (1980). Effect of induced erythrocytes on aerobic work capacity. *Journal of Applied Physiology*, 48, 636-642.

Burke, E.R., Falsetti, H.L., Feld, R.D., Patton, G.S., & Kennedy, C. (1985). Blood testing to determine overtraining in swimmers. In J. Joy (Ed.), *Coaching Notes*, Canadian Amateur Rowing Association, 5(1), 44-46.

Brynes, W.C., Clarkson, P.M., White, J.S., Hsieh, S.S., Frykman, P.N. & Maughan, R.J. (1985). Delayed onset muscle soreness following repeated bouts of downhill running. *Journal of Applied Physiology*, 59(3), 710-715.

Clement, D.B., & Asmundson, R.C. (1982). Nutritional intake and haematological parameters in endurance runners. *The Physician and Sportsmedicine*, 10(3), 37-43.

Clement, D.B., Asmundson, R.C., & Medhurst, C.W. (1977). Hemoglobin values: a comparative survey of the 1976 Canadian Olympic team. *Canadian Medical Association Journal*, 177, 614-617.

Clement, D.B. & Sawchuk, L.L. (1984). Iron status and sports performance. *Sports Medicine*, 1, 65-74.

Convertino, V.A. (1983). Heart rate and sweatrate responses associated with exercise-induced hypervolemia. *Medicine and Science in Sports and Exercise*, 15(1), 77-82.

Convertino, V.A., Brock, P.J., Kiel, L.C., Bernauer, E.M., & Greenleaf, J.E. (1980). Exercise training-induced hypervolemia role of plasma albumin, renin and vasopressin. *Journal of Applied Physiology*, 48(4), 665-669.

Convertino, V.A., Kiel, L.C., Bernauer, E.M. & Greenleaf, J.E. (1981). Plasma volume, osmolality, vasopressin, and renin activity during graded exercise in man. *Journal of Applied Physiology*, 50(1), 123-128.

Costill, D.L. (1977). Sweating: its composition and effects on body fluids. *Annals of New York Academy of Science*, 301, 160-174.

Coyle, E.F., Hemment, M.K., & Coggan, A.R. (1986). Effects of detraining on cardiovascular responses to exercise: role of blood volume. *Journal of Applied Physiology*, 60(1), 95-99.

Crowell, J.W., & Smith E.E. (1967). Determinant of the optimal hematocrit. *Journal of Applied Physiology*, 22(3), 501-504.

Cunningham, D.A., Goode, P.B., & Critz J.B. (1975). Cardiorespiratory response to exercise on a rowing and bicycle ergometer. *Medicine and Science in Sports*, 7(1), 37-43.

Cureton, T.K. (1969). The relative value of stress indicators related to the prediction of strenuous athletic (treadmill) performance. *Biochemistry of Exercise. Medicine and Sport*, 3, 37-80.

Dagg, J.H., Marrow, J.J., MacFarlan, N. & Goldberg, A. (1967). Sideropenia (latent iron deficiency). *Quarterly Journal of Medicine*, 36, 600.

Davies C.T., & Knibbs A.V. (1971). The training stimulus: the effects of intensity, duration, and frequency of effort on maximum aerobic power output. *International Zoological Agnew*, 29, 229-305.

Davis, J.E., & Brewer, N. (1935). Effect of physical training on blood volume, hemoglobin, alkali reserve and osmotic resistance of erythrocytes. *Journal of Applied Physiology*, 13, 586-591.

DeWijn, J., DeJongste, J.L., Mosterd, W., & Willebrand, D., (1971). Hemoglobin, packed cell volume, serum iron and iron binding capacity of selected athletes during training. *Nutrition and Metabolism*, 13, 26-139.

Dill, D.B, & Costill, D.L. (1974). Calculation of percentage change in volumes of blood, plasma, and red cells in dehydration. *Journal of Applied Physiology*, 37, 247-248.

Dill, D.B., Hall, F.G., Hall, K.D., Dawson, C. & Newton, J.L. (1966). Blood, plasma, and red cell volumes: age, exercise, and environment. *Journal of Applied Physiology*, 21(2), 597-602.

Drabkin, D.L. (1948). The standardization of hemoglobin measurement. *American Journal of the Medical Sciences*, 215, 110-111.

Drabkin, D.L. (1949). The standardization of hemoglobin. *American Journal of the Medical Sciences*, 217, 710-711.

Dressendorfer, R.H., & Wade, C.E. (1983). The muscular overuse syndrome in long distance runners. *The Physician and Sportsmedicine*, 11(11), 116-130.

Dressendorfer, R.H. Wade, C.E., & Amsterdam, E.A. (1981). Development of pseudoanemia in marathon runners during a 20-day road race. *Journal of the American Medical Association*, 246, 1215-1218.

Dressendorfer, R.H., Wade, C.E., & Scaff, J.H. (1985). Increased morning heartrate in runners: A valid sign of Overtraining. *The Physician and Sportsmedicine*, 13(8), 77-86.

Dufaux, B., Hoederath, A., Streitberger, L., Hollmann, W., & Assman, G. (1981). Serum ferritin, transferrin, haptoglobin, and iron in middle and long distance runners, elite rowers and professional cyclists. *International Journal of Sports Medicine*, 2(1), 43-46.

Durnin, J.V., & Womersley, J. (1974). Body fat assessed from total body density and its estimation from skinfold thickness; measurements on 481 men and women aged 16-72 years. *British Journal of Nutrition*, 32, 77-79.

Edgerton, V.R., Gardner, G.W., Ohira, Y., Gunawardena, K.A., & Senwiratne, B. (1979). Iron deficiency anemia and its productivity and activity patterns. *British Medical Journal*, 2, 1546-1549.

Edwards, R.J., & Harrison, M.H. (1984). Intravascular volume and protein responses to running exercise. *Medicine and Science in Sports and Exercise*, 16(3), 247-255.

Ehn, L., Carlmark, B., and Höglund, S. (1980). Iron status in athletes involved in intense physical activity. *Medicine and Science in Sport and Exercise*, 12(1), 61-64.

Ekblom, B., Goldberg, A., & Gullibring, B., (1972). Response to exercise after blood loss and reinfusion. *Journal of Applied Physiology*, 28, 175-185.

Falsetti, R.H. (1983). Overtraining of athletes. *The Physician and Sportsmedicine*, 11(6), 93-110.

Fox, E.L., Bartels, R.L., Klinzing, J. & Ragg, K. (1977). Metabolic responses to interval training programs of high and low power output. *Medicine and Science in Sports*, 9(3), 191-196.

Fowler, W.M., Chowdhury, S.R., Pearson, C.M., Gardner, G., & Bratton, R. (1962). Changes in serum enzyme levels after exercise in trained subjects. *European Journal of Applied Physiology*, 17, 943-946.

Gilligan, D.R., Altschule, M,D, & Katersky, E.M., (1943). Physiological intravascular hemolysis of Exercise. Hemoglobinemia and hemoglobinurua following cross country runs. *Journal of Clinical Investigations*, 22, 859-869.

Gimenez, M., & Florentz, M. (1984). Serum enzyme variations in man during an exhaustive "square-wave" endurance exercise test. *European Journal of Applied Physiology*, 52, 219-224.

Glass, H.I., Edwards, R.H., & Garreta, A.C. (1969). CO red cell labelling for blood volume and total hemoglobin in athletes: effect of training. *Journal of Applied Physiology*, 26, 131-134.

Guyton, A.C. (1981). *Textbook of Medical Physiology*, (6th ed., p. 71). Saunders Co.: Philadelphia.

Hagerman, F.C. (1984). Applied physiology of rowing. *Sports Medicine*, 1, 303-321.

Hagerman, F.C., & Howie, G.A. (1966). Use of certain physiological variables in the selection of 1967 New Zealand crew. *Research Quarterly*, 42(3), 264-273.

Hagerman, F.C., Hagerman, G.R., & Mickelson, T.C. (1979). Physiological profiles of the elite rowers. *The Physician and Sportsmedicine*, 7(7), 74-83.

Hagerman, F.C. & Staron, R.S. (1983). Seasonal variations among physiological variables in elite oarsmen. *Canadian Journal of Applied Sport Sciences*, 8, 143-148.

Hansen, K.N., Bjerre-Knudsen, J., Brodthagen, U., Jordal, R., & Paulev, P.E. (1982). Muscle cell leakage due to long distance training. *Journal of Applied Physiology*, 48, 177-188.

Haralambie, G.B. (1978). Creatine kinase and hexose phosophate isomerase activity in skeletal muscles of healthy male athletes. *Enzyme*, 23.

Haralambie, G.B., & Senser, L. (1980). Metabolic changes in man during long distance swimming. *European Journal of Applied Physiology*, 43, 115-125.

Haralambie, G.B., Cerny, F.J., & Huber, G. (1976). Serum enzyme levels after bobsled racing. *Journal of Sports Medicine & Physical Fitness*, 16, 54-56.

Hermansen, L., Hultman, E., Kaijser, L., & Saltin, B. (1979). Muscle fuel for competition. *The Physician and Sportsmedicine*, 7, 48-58.

Hollmann, W., & Kastner, K. (1969). The behavior of arterial blood gases, arterial substrates, pH and hematocrit, in different ergometric work. *Biochemistry of Exercise. Medicine and Sport*, 3, 81-88.

Holmgren, A., Mossfeldt, F., Sjöstrand, T., & Strom, G. (1960). Effect of training on work capacity, total hemoglobin, blood volume, heart volume and pulse rate in recumbent positions. *Acta Physiologica Scandinavica*, 50, 72-83.

Hong, C.Z., & Lien, I.N. (1984). Metabolic effects of exhaustive training of athletes. *Archives of Physical Medicine Rehabilitation*, 65, 362-365.

Hunter, B.J., & Critz, J.B. (1971). Effect of training on plasma enzyme levels in man. *Journal of Applied Physiology*, 31(1), 20-23.

Joyce, H., & Poortmans, J. (1970). Hematocrit and serum proteins during arm exercise. *Medicine and Science in Sports and Exercise*, 2, 187-190.

Kaltreider, N.L., & Meneely, C.R. (1940). The effect of exercise on the volume of the blood. *Journal of Clinical Investigation*, 19, 627-634.

Kanstrup, I., & Ekblom, B.I. (1984). Acute Hypervolumia, cardiac performance and aerobic power during exercise. *Journal of Applied Physiology*, 52, 1186-1191.

Kim, E.K., Page, F.H., & Logan, J.E., (1978). An investigation of factors influencing the measurement of creatine phosphokinase activity in serum using a coupled enzymatic method. *Ciblia*, 11(1), 3-9.

King. S.W., Statland, B.e., & Sawory, J. (1972). The effect of a short burst of exercise on activity values of enzyme in sera of healthy young men. *Clinica Chimica Acta*, 72, 211-218.

Kjellberg, S.R., Rudhe, U., & Sjöstrand, T. (1949). Increase of the amount of hemoglobin and blood volume in connection with physical training. *Acta Physiologica Scandinavica*, 19, 152-169.

Knuttgen, H.G., Nordesjö, L.O., Ollander, B., & Saltin, B. (1973). Physical conditioning through interval training with young male adults. *Medicine and Science in Sport and Exercise*, 5(4), 220-226.

Kruse, K.J. (1985). The effect of exercise and training on blood volume and erythrocyte count: a review. *American Corrective Therapy Journal*, 39(1), 6-9.

Lamb, D.R. (1978). *Physiology of Exercise*, New York: Macmillan Publishing Company.

Lamb, D.R. (1984). *Physiology of Exercise Responses & Adaptations*, New York: Macmillan Publishing Company.

LaPorta, M.A., Linde, H.W., Bruce, D.L., & Fitzsimons, E.J. (1978). Elevation of creatine phosphokinase in young men after recreational exercise. *Journal of the American Medical Association*, 129, 2685-2686.

Larsson, L., & Forsberg, A. (1980). Morphological muscle characteristics in rowers. *Canadian Journal of Applied Sport Sciences*, 5, 239-244.

Lindemann, R. (1978). Low hematocrit during basic training; athlete's anemia? (letter). *New England Journal of Medicine*, 229, 1191-1192.

Magazanik, A., Shapiro, Y., Meytes, D., & Meytes, I. (1974). Enzyme blood levels & water balance during a marathon race. *Journal of Applied Physiology*, 36(2), 214-217.

Maron, M.B., Horvath, S.M., & Wilkerson, J.E. (1975). Acute blood biochemical alterationss in response to marathon running. *European Journal of Applied Physiology*, 34, 173-181.

Maron, M.B., Horvath, S.M., & Wilkerson, J.E. (1977). Blood biochemical alterations during recovey from competitve running. *European Journal of Applied Physiology*, 36, 231-238.

Martin, R.P., Haskell, W.L., & Wood, P.D. (1977). Blood chemistry and lipid profiles of elite distance runners. *Annals of New York Academy of Science*, 301, 346-360.

Martin, D.E., Vroon, D.H., May, D.F. & Pilbeam, S.P. (1986). Physiological changes in elite male distance runners training, *The Physician and Sportsmedicine*, 14(1), 152-171.

Martindale, W.O., & Robertson, D.G. (1984). Mechanical energy in sculling and in rowing an ergometer. *Canadian Journal of Applied Sport Sciences*, 9(3), 153-163.

Maxwell, J.H., Bloor, C.M. (1981). Effects of conditioning on exertional rhabdomyolysis and serum creatine kinase after severe exercise. *Enzyme* 26, 177-181.

Medbø J.I., & Sejersted, O.M. (1985). Acid-base and electrolyte balance after exhausting exercise in endurance trained subjects. *Acta Physiologica Scandinavica*, 125, 97-109.

Millard, M.L., Zauner, C.W., & Cade, R.J. (1981). Effects of intense training on blood parameters in national class collegiate swimmers. *Medicine and Science in Sports and Exercise*, 13(2), 110.

Misner, J.E., Massey, B.H., & Williams, B.T. (1973). The effect of physical training on the response of serum enzymes to exercise stress. *Medicine and Science in Sports and Exercise*, 5(2), 86-88.

Nanji, A.A. (1983). Serum creatine kinase isoenzymes: a review. *Muscle and Nerve*, 6, 83-90.

Newham, D.J., Jones, D.A., & Edwards, R.H. (1983). Large delayed plasma creatine kinase changes after a stepping exercise. *Muscle and Nerve*, 6, 380-385.

Newham, D.J., Mills, K.R., Quigley, B.M., & Edwards, R.H. (1982). Muscle pain and tenderness after exercise. *Australian Journal of Sports Medicine and Exercise Science*, 14(4), 129-131.

Novosadova, J. (1977). The changes in hematocrit, hemoglobin, plasma volume and proteins during and after different types of exercise. *European Journal of Applied Physiology*, 36(3), 223-230.

Nowacki, P., Krause, R., & Adam, K. (1969). Maximal oxygen uptake by a rowing crew winning the Olympic gold medal in 1968. *Pflugers Archives*, 312, R66.

Nutrition Canada. (1975). *British Columbia Survey*, Ottawa: Health and Welfare Canada.

Ohri, V.C., Chatterti, B.K., Das, M., Akhtar, S.C., Tewari, P., Bhattacharji, P., & Behl, A. (1983). Effect of submaximal exercise on hematocrit platelet aggregation and blood fibrinogen levels. *The Journal of Sport Medicine and Physical Fitness*, 23(2), 127-130.

Oscai, L.B., Williams, B.T., & Hertig, B.A. (1968). Effect of exercise on blood volume. *Journal of Applied Physiology*, 24(5), 622-624,

Pate, R.R. (1983). Sports Anemia: a review of the current research literature. *The Physician and Sportsmedicine*, 11(2), 115-131.

Pate, R.R., Maguire, M., & Van Wyk, J. (1979). Dietary Iron Supplementation in Women Athletes. *The Physician and Sportsmedicine*, 7(9), 81-86.

Pearce, J. (1965). Serum creatine kinase and exercise. *British Medical Journal*, 2, 107.

Perkkio, M.V., Jansson, L.T., Brooks, G.A., Refino, C.J. & Dallman, P.R. (1985). Work performance in iron deficiency of increasing severity . *Journal of Applied Physiology*, 58, 1477-1480.

Perkkio, M.V., Jansson, L.T., Henderson, S., Refino, C., Brooks, G.A., & Dallman, P.R. (1985). Work performance in the iron deficient rat: improved endurance with other exercise training. *American Journal of Physiology 249 (Endocrinology of Metabolism)*, 12, E306- e311.

Prampero, P.E., Cortili, D.I., & Certelli, P. (1971). Physiological effects of rowing. *Journal of Applied Physiology*, 31, 852- 857.

Rhodes, E.C., Mosher, R.E., McKenzie, D.C., Franks, I.M., Potts, J.E., & Wenger, H.A. (1986). Physiological profiles of the Canadian Olympic Soccer Team. *Canadian Journal of Applied Sport Sciences*, 11(1), 31-36.

Rogozin, V.A. (1976). The effect of the number of training sessions on skeletal muscle protein synthesis. *Medicine and Science in Sports*, 8(4), 223-225.

Rose, L.T., Bousser, J.E., & Cooper, K.H. (1970). Serum enzymes after marathon running. *Journal of Applied Physiology*, 29(3), 355-357.

Ross, J.H., Attwood, E.C., Atkin, G.C., & Villar, R.N. (1983). A study of the effects of severe repetitive exercise on serum myoglobin, creatine kinase transaminases, and lactate dehydrogenase. *Quarterly Journal of Medicine, New Series L11*, 206, 268-279.

Rotstein, A., Bar, O., & Dlin, R. (1982). Hemoglobin, hematocrit, & calculated plasma volume changes induced by a short submaximal task. *International Journal of Sports Medicine*, 3, 230-235.

Rowell, L.B. (1974). Human cardiovascular adjustments to exercise and thermal stress. *Physiological Reviews*, 54(1), 75-159.

Rushall, B.S., & Busch, J.D. (1980). Hematological responses to training in elite swimmers. *Canadian Journal of Applied Sports Science*, 5(3), 164-169.

Rutledge, J., Clayson, K.J., & Strandjord, P.E. (1978). Effect of physical conditioning on serum kinase after exercise. *Journal of the American Medical Association*, 240(24), 2633.

Ryan, A.J. (1983). Overtraining of Athletes. *The Physician and Sportsmedicine*, 11(6), 93-110.

Saltin, B. (1964). Circulatory reponse to submaximal and maximal exercise after thermal dehydration. *Journal of Applied Physiology*, 19, 1125-1132.

Sanders, T.M., and Bloor, C.M., (1975). Effects of endurance exercise on serum enzyme activites in well conditioned males. *Medicine and Science in Sports and Exercise*, 7(1), 4-47.

Schwane, J.A., Johnson, S.R., Vandenakker, C.B., & Armstrong, R.B. (1983). Delayed-onset muscular soreness and plasma CPK, and LDH activites after downhill running. *Medicine and Science in Sports and Exercise*, 15(1), 51-56.

Secher, N.H. (1983). The physiology of rowing. *Journal of Sports Sciences*, 1, 23-53.

Senay, L.C., & Christensen, M.L. (1965). Changes in blood plasma during progressive dehydration. *Journal of Applied Physiology*, 20, 1136-1140.

Senay, L.C., Rogers, G., & Jooste, P. (1980). Changes in blood plasma during progressive treadmill and cycle exercise. *Journal of Applied Physiology*, 49(1), 59-65.

Shapiro, Y., Magazanik, A., Sohar, E., & Reich, C. (1972). Serum enzyme changes in untrained subjects following prolonged march. *Canadian Journal of Physiology and Pharmacology*, 51, 271-276.

Siegel, A.J., Silverman, L.M., & Holman, L. (1981). Elevated creatine kinase MB isoenzyme levels in marathon runners. *Journal of the American Medical Association*, 246(18), 2049-2051.

Sigma Chemical Company. (1981). *Sigma Technical Bulletin, No 45-UV*, Creatine phosphokinase (CPK) in serum, plasma, and cerebrospinal fluid at 340 NM. St. Louis: Missouri.

Stone, H.O., Thompson, H.K., & Schmidt-Nielson, K. (1968). Influence of erythrocytes on blood viscosity. *American Journal of Physiology*, 214, 913-918.

Stromme, S.B., Tevit, B., & Refsum, H.E. (1978). Enzymatic changes following prolonged heavy exercise. In regulatory mechanisms in metabolism during exercise. In F. Landry & W. Orban (Ed.), *International Symposium on Biochemistry of Exercise*, Miami: Symposia Specialists Inc., 1, 243-249.

Strydom, N.B., Wyndham, C.H., Greyson, J.S., & Atkins, A.T. (1967). A scientific approach to the selection & training of oarsmen. *South African Medical Journal*, 41, 1100-1102.

Sutton, J. (1971). Pathology in the limitation of performance. *Australian Journal of Sport Medicine*, 4, 6-14.

Symanski, J.D., McMurray, R.G., Silverman, L.M., Smith, B.W., & Siegel, A.J. (1983). Serum creatine kinase and CK-MB isoenzyme responses to acute and prolonged swimming in trained athletes. *Clinica Chimica Acta*, 129, 181-187.

Tiidus, P.M., & Ianuzzo, D.C. (1983). Effects of intensity and duration of muscular exercise on delayed muscle soreness and serum enzyme activities. *Medicine and Science in Sports and Exercise*, 15(6), 461-465.

Thomas, T,R., & Etheridge, G.L. (1982). The effect of acute exercise on body density, body volume, and plasma volume. *Canadian Journal of Applied Sport Sciences*, 7(4), 258-262.

Thomson, W,H., Sweetin, J.C., & Hamilton, I.D. (1975). ATP and muscle energy efflux after physical exertion. *Clinica Chimica Acta*, 59,241-245.

Tomaski, M. (1979). Physical exercise effect on erythrocyte metabolism. *ACTA Physiologica Polonica*, 30, 631-638.

Van Beaumont, W. (1972). Evaluation of hemoconcentration from hematocrit measures. *Journal of Applied Physiology*, 32(5), 712-713.

Van Beaumont, W., Greenleaf, J.E., & Johos, L. (1972). Disproportional changes in hematocrit, plasma volume, and proteins during exercise and bed rest. *Journal of Applied Physiology*, 33, 55-61.

Vellar, O.D. & Hermansen, L. (1971). Physical performance and hematological responses to heavy exercise in patients with anemia. *Journal of Clinical Investigations*, 39, 378-388.

Wasserman, D.H., Lavina, H., Lickley, A. & Vranic, M. (1985). Effect of hematocrit reduction on hormonal and metabolic response to exercise. *Journal of Applied Physiology*, 58(4), 1257-1262.

Wilkerson, J.E., Gutin, B., & Horvath, S.M. (1977). Exercise induced changes in blood, red cell, and plasma volumes in man. *Medicine and Science in Sports*, 9(3), 155-158.

Williamson, M. (1981). Anemia in runners and other athletes. *The Physician and Sportsmedicine*, 9(6), 73-79.

Yoshimura, H. (1970). Anemia during physical training (sports review). *Nutritional Reviews*, 28(10), 251-253.

Young, A. (1984). Plasma creatine kinase after the marathon-a diagnostic dilemma. *British Journal of Sports Medicine*, 18(4), 269-272.

Appendix A

INFORMED CONSENT

UNIVERSITY OF VICTORIA
P.O. BOX 1700, VICTORIA, BRITISH COLUMBIA, CANADA V8W 2Y2
TELEPHONE (604) 721-7211, TELEX 049-7222
Sport and Fitness Testing Centre

School of Physical Education
721-8373

INFORMED CONSENT
FOR
PHYSIOLOGICAL ASSESSMENTS

In order to assess physiological function(s) the following laboratory assessments will be performed:

Lab Initial	Subject Initial	
___	___	Submaximal Cardio-Respiratory Function

You will exercise on an ergometer at 75% of predicted maximum heart rate. The following indicated variables will be measured:

a) ventilatory responses ___ c) thermoregulatory responses ___

b) heart rate responses ___ d) other _____

___ ___ Maximal Cardio-Respiratory Function

You will exercise on an ergometer with progressively increasing loads to elicit maximal responses in the following indicated variables:

a) oxygen consumption ___ c) ventilation ___

b) heart rate ___ d) other _____

___ ___ Submaximal and/or Maximal Muscular Contractions

You will perform submaximal or maximal muscular contractions in the following modes:

___isometric ___isotonic ___isokinetic ___eccentric

___ ___ Blood Chemistry

Blood samples may be taken prior to, during, or post-exercise by:

a) venipuncture ___ b) finger tip prick ___

Lab Initial	Subject Initial	
		Body Composition
		Lean body mass and percent body fat may be assessed by: a) anthropometric measures ___ b) body densiometry ___
___	___	Tests will be administered by qualified personnel under the direct supervision of the investigator(s).
___	___	Blood samples will be taken by a qualified laboratory technician or registered nurse.
___	___	Training will be monitored by the investigator(s) or trained assistants.
___	___	Test and training data and results will be treated in a confidential manner and used only to describe group responses.
___	___	Absolute confidentiality of individual results will be maintained unless specific approval has been given to other use of the material by each subject, or guardian where necessary.
___	___	While it is highly unlikely that a subject should be injured or taken ill during a test or training session, lab personnel are trained in emergency procedures and emergency equipment is on-site at all times.
___	___	All laboratory activity will be completed proximal to medical and/or paramedical assistance.
___	___	The maximal exercise loads imposed will not exceed those which might be expected of an athlete during sports performance.

I have read the above and agree to participate in this research project/ fitness appraisal at my own risk. I regularly take part in strenuous physical activity at least as intense as these tests. I realize that I may expect a thorough explanation and/or demonstration of any procedures and that I may terminate participation at any time in any or all procedures of my own volition.

Having voluntarily assumed participation and the risks thereof, in the project, I hereby disclaim and release the University of Victoria, its agents, servants or employees, including all personnel involved in the research project fitness appraisal from any and all liability that might otherwise arise as a result of my participation as a research subject in this study/or fitness appraisal.

NAME: _____ DATE: _____
(please print)

SIGNATURE: _____

I, the undersigned guardian, am guardian of _____, the intended subject. I have discussed the experimentation with the subject and have read the material supplied by the experimentors. I agree on behalf of the subject to permit his/her participation on the terms and subject to the waiver and release of the University of Victoria hereinbefore set out.

GUARDIAN'S SIGNATURE: _____
(where applicable)

DL:gs
84/10/25

UNIVERSITY OF VICTORIA
P.O. BOX 1700, VICTORIA, BRITISH COLUMBIA, CANADA V8W 2Y2
TELEPHONE (604) 721-7211, TELEX 049-7222
Sport and Fitness Testing Centre

School of Physical Education
721-8373

INFORMED CONSENT

FOR

PHYSIOLOGICAL ASSESSMENTS

In order to assess physiological function(s) the following laboratory assessments will be performed:

Lab Initial	Subject Initial	
___	___	Submaximal Cardio-Respiratory Function

You will exercise on an ergometer at 75% of predicted maximum heart rate. The following indicated variables will be measured:

a) ventilatory responses ___ c) thermoregulatory responses ___

b) heart rate responses ___ d) other _____

___	___	Maximal Cardio-Respiratory Function

You will exercise on an ergometer with progressively increasing loads to elicit maximal responses in the following indicated variables:

a) oxygen consumption ___ c) ventilation ___

b) heart rate ___ d) other _____

___	___	Submaximal and/or Maximal Muscular Contractions

You will perform submaximal or maximal muscular contractions in the following modes:

___isometric ___isotonic ___isokinetic ___eccentric

___	___	Blood Chemistry

Blood samples may be taken prior to, during, or post-exercise by:

a) venipuncture ___ b) finger tip prick ___

Lab Initial	Subject Initial	
		Body Composition
		Lean body mass and percent body fat may be assessed by:
		a) anthropometric measures ___ b) body densiometry ___
___	___	Tests will be administered by qualified personnel under the direct supervision of the investigator(s).
___	___	Blood samples will be taken by a qualified laboratory technician or registered nurse.
___	___	Training will be monitored by the investigator(s) or trained assistants.
___	___	Test and training data and results will be treated in a confidential manner and used only to describe group responses.
___	___	Absolute confidentiality of individual results will be maintained unless specific approval has been given to other use of the material by each subject, or guardian where necessary.
___	___	While it is highly unlikely that a subject should be injured or taken ill during a test or training session, lab personnel are trained in emergency procedures and emergency equipment is on-site at all times.
___	___	All laboratory activity will be completed proximal to medical and/or paramedical assistance.
___	___	The maximal exercise loads imposed will not exceed those which might be expected of an athlete during sports performance.

I have read the above and agree to participate in this research project/ fitness appraisal at my own risk. I regularly take part in strenuous physical activity at least as intense as these tests. I realize that I may expect a thorough explanation and/or demonstration of any procedures and that I may terminate participation at any time in any or all procedures of my own volition.

Having voluntarily assumed participation and the risks thereof, in the project, I hereby disclaim and release the University of Victoria, its agents, servants or employees, including all personnel involved in the research project fitness appraisal from any and all liability that might otherwise arise as a result of my participation as a research subject in this study/or fitness appraisal.

NAME: _____ DATE: _____
(please print)

SIGNATURE: _____

I, the undersigned guardian, am guardian of _____, the intended subject. I have discussed the experimentation with the subject and have read the material supplied by the experimentors. I agree on behalf of the subject to permit his/her participation on the terms and subject to the waiver and release of the University of Victoria hereinbefore set out.

GUARDIAN'S SIGNATURE: _____
(where applicable)

DL:gs
84/10/25

Appendix B

CLASSIFICATION OF ROWING WORKOUTS

Table 5

Classification of Rowing Workouts

INTENSITY		
Hard	**Medium**	**Easy**
3 minute intervals on-off	4-6x3 minute intervals on-off 2000m race day Performance test VO_2 max test 10 minute intervals on-off	steady state (SS) short sprints drills; starts 400 hard strokes

DURATION		
Hard	**Medium**	**Easy**
10x3 minute interval on-off 10x90 strokes on 3 minute off 10x10 minute intervals on-off	6x3 minute intervals on-off 4x2000 meters	4x3 minute intervals on-off Performance Test Race day

WEEK NO.	WORKOUTS	INTENSITY			DURATION		
	NO. PER WEEK	Easy	Medium	Hard	Easy	Medium	Hard
6-7	10	3	3	4	10		
7-8	9	1	3	5	5	3	1
8-9	9		2	7	7	2	
9-10	10	1	4	5	4	3	3
10-11	8	3	1	4	3		4
11-12	9	1	3	5	3	1	5
12-13	8	1	2	5	2	1	5
13-14	9	1	3	5	3	1	5

Appendix C

TESTING AND TRAINING SESSIONS

Table 6

Collection of the Dependent Variables during the Control Period

Week #	Tuesday	Wednesday	Thursday	Friday	Saturday	Sunday	Monday
1							
A.M.		HT, WT, SK	A.Blood	PT			C.Blood, PT
P.M.		VO_2Max					
2							
A.M.				A.Blood			C.Blood, PT
P.M.							
3							
A.M.		Race-2000m		A.Blood			C.Blood, PT
P.M.							
4							
A.M.				A.Blood			C.Blood, PT
P.M.							
5							
A.M.				A.Blood			
P.M.							

Legend:

A.Blood	= Acute Blood	HT	= Height
C.Blood	=Chronic Blood	WT	= Weight
PT	= Performance Test	SK	= Skinfold
VO_2Max	=VO_2Max Test		

Table 7

Testing and Training Sessions During the Exercise Period

Week #	Tuesday	Wednesday	Thursday	Friday	Saturday	Sunday	Monday
6							
A.M.	C.Blood, PT	4x3'on-off 32-34 strks/min	4x3'on-off 32-34 strks/min	SS A.Blood	B.C.Champ. Regatta	Victoria Regatta	C.Blood, PT
P.M.	4x3'on-off 32-34 strks/min	4x3'on-off 32-34 strks/min	VO_2Max. Test WT, SS	SS	off	off	off
7							
A.M.	4x2.5'on-off 34-36 strks/min	4x3'on-off 32-34 strks/min	3x10'on 20' off 28 strks/min A.Blood	off	P.N.W.Champ. Regatta	P.N.W.Champ. Regatta	off
P.M.	4x3.5'on-off 32-34 strks/min	4x3'on-off 34-36 strks/min	1.0 hrs. short SP	off	P.N.W.Champ. Regatta	off	off
8							
A.M.	C.Blood, PT	4x3'on-off 32-34 strks/min	4x3'on-off 32-34 strks/min	6x3'on-off 32-34 strks/min A.Blood	6x3'on-off 32-34 strks/min	off	Victoria Regatta
P.M.	6x3'on-off 32-34 strks/min	6x3'on-off 32-34 strks/min	off Wt. Train.	4x3'on-off 32-34 strks/min	off Wt. Train.	C.Blood, PT	Victoria Regatta
9							
A.M.	6x3'on-off 32-34 strks/min	6x3'on-off 32-34 strks/min	1.5hr SS A.Blood	off Wt. Train.	Shawnigan Lake Regatta	P.N.W.Skulling Regatta	off
P.M.	4x3'on-off 32-34 strks/min	4x3'on-off 32-34 strks/min	6x3'on-off 32-34 strks/min	4x2000m Tempo	Shawnigan Lake Regatta	off	C.Blood, PT
10							
A.M.	10x3'on-off 30,32,34,32,30 strks,2 of each	10x90 strks 3'off 3-32,3-34,4-32	off Wt. Train.	10x3'on-off 30,32,34,32,30 strks,2 of each A.Blood	10x90'on 3'off 32,34,36,34,32 strks,2 of each.	off	C.Blood, PT
P.M.	SS drills	SS,ST,SP 400 hard strokes	off	SS drills	off Wt. Train.	off	10x3'on-off 32-36 2 of each

Week #	Tuesday	Wednesday	Thursday	Friday	Saturday	Sunday	Monday
11							
A.M.	10x90 strks3-32 3-34,3-36,1-38	10x3'on-off pyr 30->38,38->30	10x90 strks 3-34,4-36,3-34	10x3'on-off Tempo,A.Blood	3x2000m Race pace	off	C.Blood, PT
P.M.	off Wt. Train.	SS ST SP 400 hard strks	off Wt. Train.	SS,4x6' 24-28	off	off	10x3'on-off 2-32, 3-34,4-36,1-38
12							
A.M.	10x90 strkspyr 30->40,40->32	10x3'on-off Tempo	10x90 strks 3-32,4-34,3-32	10x3 on-off 3-32 3-34,3-36,1-38+ A.Blood	off	off	C.Blood, PT
P.M.	off Wt. Train.	SS 4x6'on-off 26->28	off Wt. Train.	SS,ST,SP 400 hard strks	off Wt. Train.	off	10x3'on-off 2-32, 3-34,4-36,1-38
13							
A.M.	10x90 strkspyr 30->40,40->32	10x3'on-off Tempo	10x90 strks 3-32,4-34,3-32	10x3'on-off 3-32 3-34,3-36,1-38+	3x2000m Race pace	off	C.Blood, PT
P.M.	off Wt. Train.	SS,4x6'on-off 26->28	off Wt. Train.	SS ST SP 400 hard strks	off Wt. Train.	off	off
14							
A.M.	off	HT, WT, SK	HT, WT, SK	off	off	off	off
P.M.	off	VO_2Max. Test	VO_2Max. Test	off	off	off	off

Legend:

SS = Steady state
ST = Starts
SP = Sprints
strks=Strokes
Wt. Train. =Weight Training

HT = height
WT = weight
SK = skinfold
PT =Performance Tests

A.Blood = Acute Blood
C.Blood =Chronic Blood
pyr =pyramid
strks/min =strokes per minute

VITA

Surname: Doyle Given Names: Patricia K.

Place of Birth: Victoria B.C. Date of Birth: Feb., 8 1958

Educational Institutions Attended, with Dates of Entering and Leaving:

University of Victoria	1976	to	1982
University of Victoria	1984	to	1986
		to	

Degrees Awarded, with Dates and Names of Institutions:

Bachelor of Science	1982	University of Victoria
Masters of Arts	1986	University of Victoria

Awards:

British Columbia Post-Secondary Scholarship

Publications:

A Documentation of the Athletes Use and Knowledge of Anabolic Steroids.

PARTIAL COPYRIGHT LICENSE

I hereby grant the right to lend my thesis (the title of which is shown below) to users of the University of Victoria Library, and to make single copies only for such users or in response to a request from the Library of any other university, or similar institution, on its behalf or for one of its users. I further agree that permission for extensive copying of this thesis for scholarly purposes may be granted by me or a member of the University designated by me. It is understood that copying or publication of this thesis for financial gain shall not be allowed without written permission.

Title of Thesis:

ACUTE AND CHRONIC PHYSIOLOGICAL CHANGES TO HIGH INTENSITY TRAINING

Author 

Patricia Katherine Doyle

July 14th 1986