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Oxygen Consumption, Heart Rate and Blood Lactate Levels as Affected by Exercise Intensity, Meals and Diets in Humans During Rest, Exercise and Recovery

By
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ABSTRACT<br>Oxygen Consumption, Heart Rate and Blood Lactate Levels as Affected by Exercise Intensity, Meals and Diets in Humans During Rest, Exercise and Recovery<br>By<br>Kristian Lindsted

One hundred linear regression equations (HROXCR) between HR (HR) and oxygen consumption (OXC) were developed based on 10 incremental exercise tests in each of 10 subjects. Both slopes and $y$-intercepts were found to be significantly inhomogenous within as well as among subjects. The inhomogeneity of the HROXCR was increased rather than decreased by using incremental HR. The HROXCR was singificantly affected by previous exercise but not by diet. The repeatability $(R=0.7)$ for the slopes and $y$-intercepts of the HROXCR indicates that the variation among subjects was larger than the variation within the subjects.

The HROXCR observed during exercise was not followed during recovery. OXC returned to resting values faster than HR during early recovery (3-4 min). Duration rather than intensity was related to the $H R$ recovery lag at greater than $65 \% \max$ OXC. After 40 min of recovery the OXC had returned to the preexercise level, but the $H R$ was still elevated 20 beats/min.

The effects of 700 kcal meals from either fat, protein or carbohydrate on OXC during rest was investigated in five male subjects. The postabsorptive state served as control for each subject. The
mean OXC for control, fat, protein and carbohydrate meal respectively was $3.6,3.8,4.3$ and $4.1 \mathrm{ml} / \mathrm{minute} / \mathrm{kg}$. The thermogenic responses were as follows: fat - borderline significance; protein - significant > 6 hrs; carbohydrate - significant > 3 hrs. Only following the carbohydrate meal was there a significant HROXC correlation ( $r=0.5$ ). It may be that the regulatory thermogenic response is related to carbohydrate.

Only the carbohydrate meal affected the RQ during rest ( $\bar{X}=0.84$ compared to 0.75 ). There were no significant treatment effects upon exercise RQs. There was no effect of meals or diets on the substrate utilization during exercise at $80 \%$ maxOXC. During rest, the carbohydrate meal caused a 3 to 4 fold increase in the blood lactate concentration. During 40 min of recovery from exercise at $80 \%$ maxOXC after the control, protein and fat meals, the x for lactate decreased from 3 to 1 mM . However, following the carbohydrate meal, the $\times$ for lactate remained about 2 mM .

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## TABLE OF CONTENTS

Page
INTRODUCTION ..... 1
CHAPTER 1 - LITERATURE REVIEW ..... 5
Introduction ..... 6
The Energy Equation ..... 6
Heart Rate as a Measure of Energy Expenditure ..... 7
Relation Between HR, SV, and Circulation. ..... 10
Thermogenes is ..... 13
Non-shivering thermogenesis ..... 13
Cold-induced thermogenesis. ..... 13
Dietary-induced thermogenesis ..... 14
Control mechanisms ..... 16
Thermogenesis and exercise. ..... 17
Substrate Utilization ..... 18
Glycogen ..... 18
Intramuscular Lipids ..... 18
Control of Substrate Utilization. ..... 19
Effect of meals and exercise on substrate utiliza- tion. ..... 22
Postexercise Lactate ..... 28
Effect of exercise ..... 28
Effect of diet. ..... 31
CHAPTER 2 - THE VARIABILITY OF THE HEART RATE-OXYGEN CONSUMP- TION RELATIONSHIP WITH TIME AND COMPARISON OF INDIVIDUAL REGRESSION LINES VS A COMMON REGRES- SION LINE FOR ALL SUBJECTS. ..... 33
Synopsis ..... 34
Introduction ..... 35
Methods ..... 37
Subjects ..... 37
Measurements of oxygen consumption (OXC) and heart rate (HR) ..... 37
Test protocol ..... 39
Measurements of resting HR and OXC. ..... 40
Treatment of data and statistical procedures ..... 40
Results ..... 41
Correlation between HR and OXC at constant workload ..... 49
Discussion. ..... 51
Page
CHAPTER 3 - EFFECT OF DIET, PREVIOUS EXERCISE, AND RECOVERY UPON THE LINEAR REGRESSION BETWEEN HEART AND OXYGEN CONSUMPTION. ..... 58
Synopsis ..... 59
Introduction ..... 60
Methods ..... 62
Subjects. ..... 62
Informed consent. ..... 62
Diets ..... 62
Measurements ..... 66
Exercise tests. ..... 66
Experimental protocol ..... 68
Statistical analysis. ..... 69
Results ..... 71
Effect of diet on the HROXCR during exercise. ..... 71
Effect of previous exercise on HROXCR ..... 71
Effect of recovery on HROXCR. ..... 74
Discussion. ..... 104
Effect of diet and previous exercise. ..... 104
Recovery from exercise and HROXCR ..... 105
CHAPTER 4 - DIETARY INDUCED THERMOGENESIS DURING REST AND EXERCISE: EFFECT OF MEALS AND DIET ON OXYGEN CONSUMPTION AND ENERGY SUBSTRATE UTILIZATION DURING REST AND EXERCISE ..... 110
Synopsis ..... 111
Introduction. ..... 112
Methods ..... 115
Subjects ..... 115
Informed consent. ..... 115
Diets ..... 115
Measurements ..... 119
Exercise tests ..... 120
Experimental protocol ..... 121
Statistical analysis ..... 124
Results ..... 125
Experiments during rest ..... 125
Exercise experiments ..... 132
Discussion. ..... 136
OXC during rest ..... 136
OXC during exercise ..... 139
Energy substrate utilization during exercise. ..... 140
Page
CHAPTER 5 - EFFECT OF MEALS AND DIET ON POSTEXERCISE BLOOD LACTATE LEVELS. ..... 143
Synopsis. ..... 144
Introduction. ..... 145
Methods ..... 147
Subjects ..... 147
Informed consent ..... 147
Diet. ..... 147
Measurements ..... 151
Exercise tests ..... 152
Experimental protocol ..... 153
Statistical analysis. ..... 156
Results ..... 157
Discussion. ..... 166
CHAPTER 6 - CONCLUSIONS ..... 171
BIBLIOGRAPHY ..... 175
APPENDICES ..... 197

## LIST OF TABLES

Page
CHAPTER 2 - THE VARIABILITY OF THE HEART RATE-OXYGEN CONSUMP- TION RELATIONSHIP WITH TIME AND COMPARISON OF INDIVIDUAL REGRESSION LINES VS A COMMON REGRES- SION LINE FOR ALL SUBJECTS ..... 33
Age, height, weight, $V 02$ max for the ten subjects.The slopes $\bar{B}_{1}$ and $y$-intercepts $\bar{B}_{0}$ and the corre-sponding variances based on all ten trials ineach subject is given . . . . . . . . . . . . . . 38
Calculated F-ratio for test of homogenity withinthe ten trials in each subject with observed HR(F-1) and with corrected HR (F-2)463 Repeatability R, calculated and critical F-ratio( $\alpha=0.05$ ) for the slope $B_{1}$, the $y$-intercept $B_{0}$,the $y$-intercept corrected for resting heart rate$\mathrm{B}_{0}$ Cor, and $\mathrm{B}^{-1}$ and $\mathrm{BO}^{-1}$ Cor when the subjectwith a large deviation in the max V02 isdisregarded47
CHAPTER 3 - EFFECT OF DIET, PREVIOUS EXERCISE, AND RECOVERY UPON THE LINEAR REGRESSION BETWEEN HEART RATE AND OXYGEN CONSUMPTION ..... 58
1 Physical characteristics of the two groups offive adult male subjects. . . . . . . . . . . . .63
2 Relative intensity of exercise for various tread- mill settings used in incremental exercise tests. ..... 633 Composition of meals and diets. The energy con-tent of the diets varied between 2,100 and$2,400 \mathrm{kcal}$ for the different subjects64
4 Test of significant difference between theregression equations between HR and OXC for thefive treatments and the regression equation forcontrol72

Test of significance between the regression equations for two subjects exercised twice, one hour apart. Results are included with the regression lines corrected for the resting heart rate (cor)73

Comparison of mean recovery values and their respective standard deviation (SD) for eight trials in each of five subjects . . . . . . . . 79

Mean recovery increamental heart rates after exercise on a treadmill at 4 different speeds, each speed maintained for 10 and 30 minutes, respectively. Mean of four subjects. . . . . . 89

Mean recovery oxygen consumption in four subjects after exercise on a treadmill at four different speeds, two runs at each speed for 10 and 30 minutes, respectively90

Mean predicted oxygen consumption during recovery following exercise on a treadmill at four different speeds in four subjects. Each speed is repeated twice for 10 and 30 minutes, respectively. Time is given in minutes. Standard deviation (SD) is also given91

Mean difference ( 4 subjects) between the predicted oxygen consumption and the actual oxygen consumption during recovery after running on a treadmill at four different speeds. The subjects ran at each speed twice for 10 and 30 minutes, respectively92
Mean values of 95\% lower confidence limit (LCL) for predicted oxygen consumption (POXC), recovery oxygen consumption (ROXC), and the difference (DLCL) between LCL and ROXC in $\mathrm{ml} / \mathrm{kg} / \mathrm{min}$ follow- ing exercise on a treadmill at four different speeds (S) for 10 and 30 minutes (D), respec- tively. Mean of four subjects ..... 101
Test of significance for IRHR, ROXC and DOXC ..... 103
Page
CHAPTER 4 - DIETARY INDUCED THERMOGENESIS DURING REST AND EXERCISE: EFFECT OF MEALS AND DIET ON OXYGEN CONSUMPTION AFD ENERGY SUBSTRATE UTILIZATION DURING REST AND EXERCISE. ..... 110
1 Physical characteristics of the two groups offive adult male subjects. . . . . . . . . . . . . 1162 Composition of meals and diets. The energy con-tent of the diets varied between 2,100 and$2,400 \mathrm{kcal}$ for the different subjects117
3 Mean oxygen consumption following the ingestionof various meals for 5 male subjects in theresting state. Control was the postabsorptivestate126
4
Mean respiratory quotient following the ingestionfollowing ingestion of various meals for 5 malesubjects in the resting state. Control was thepostabsorptive state.127
5 Mean correlation coefficient (4) between HR andOXC during control, following a fat meal, pro-tein meal, and a carbohydrate meal. Number ofsubjects equal to 6 for control, 7 for fat meal,and 8 for carbohydrate meal. $n=30$ in each treat-subjects completing the treatment . . . . . . . .135
CHAPTER 5 - EFFECT OF MEALS AND DIET ON POSTEXERCISE BLOOD LACTATE LEVELS ..... 143
1 Physical characteristics of the two groups of five adult male subjects. ..... 1482 Composition of meals and diets. The energy con-tent of the diets varied between 2,100 and 2,400kcal for the different subjects149
3 Blood lactate in arterialized blood for 6 hoursfollowing a carbohydrate meal, and in the post-absorptive state following an overnight fast ascontrol. Mean of five subjects . . . . . . . . . 158

4 Mean blood lactate concentration during 40 minutes recovery from 20 minutes of exercise at $80 \%$ of maximal OXC in 5 subjects. . . . .163

5 Bonferroni $t$ for test of significance of postexercise blood lactate concentration. $\mathrm{CH}-\mathrm{M}$ is tested for interaction (between 5 and 40 minutes) against C and $\mathrm{F}-\mathrm{M}$. Total (40 minute) response to $\mathrm{CH}-\mathrm{M}$ is tested against total response to $\mathrm{F}-\mathrm{M}$; and the postabsorptive ( $P$ ) trials ( $C, F-D, C H-D$ ) are tested against the absorptive (A) trials ( $F-M$ and $P-M$ ) at 5 minutes postexercise. There are four contrasts ( $n=4$ ) . . . . . . . . . . . . . . . . . . 164

## LIST OF FIGURES

Page
CHAPTER 2 - THE VARIABILITY OF THE HEART RATE-OXYGEN CON- SUMPTION RELATIONSHIP WITH TIME AND COMPARI- SON OF INVIDIDUAL REGRESSION LINES VS A common regression line for all subjects ..... 33
1 Regression line between the OXC and the HR for atypical subject (\#8) based on a single incre-mental exercise test. . . . . . . . . . . . . . .42
2 Regression line between OXC and HR in a typicalsubject (\#8) based on ten incremental exercisetests . . . . . . . . . . . . . . . . . . . . . . 44
3 Regression lines between $H R$ and OXC for ahypothetical cases53
4 Incremental and corrected HR. ..... 55
CHAPTER 3 - EFFECT OF DIET, PREVIOUS EXERCISE, AND RECOVERY UPON THE LINEAR REGRESSION BETWEEN HEART RATE AND OXYGEN CONSUMPTION. ..... 58
1 Two regression lines based on exercise trials one hour apart in one subject ..... 75
2 Predicted OXC (POXC), lower confidence limits(LCL) and recovery OXC (ROXC) as a function ofrecovery HR77
3 HR during 40 -minute recovery. Mean of five subjects with eight exercise tests in each. ..... 804 Predicted OXC (POXC), upper curve. Lower 95\%confidence level for OXC (LCL), middle curve.Recovery OXC (ROXC), lower curve, as a functionof time82
5 Difference between predicted OXC and recoveryOXC (DOXC) as a function of time during 40 min -ute recovery. . . . . . . . . . . . . . . . . . . 84
Page
6 DOXC as \% of POXC vs recovery time (40-minute recovery). . . . . . . . . . . . . . . 877 Mean incremental HR (IHR) for four subjectsduring 40-minute recovery after exercise for10 minutes at speed $4(8.8 \mathrm{~km} / \mathrm{hr})$, lowercurve; and speed 6 ( $12.5 \mathrm{~km} / \mathrm{hr}$ ), upper curve. . . 938 Incremental HR during 40-minute recovery (RIHR)after exercise at speed 5 ( $10.5 \mathrm{~km} / \mathrm{hr}$ ).95
9 Recovery OXC (ROXC) as a function of time ..... 97
10 Mean DOXC (difference between predicted and recovery OXC for four subjects. ..... 99
CHAPTER 4 - DIETARY INDUCED THERMOGENESIS DURING REST AND EXERCISE: EFFECT OF MEALS AND DIET ON OXYGEN CONSUMPTION AND ENERGY SUBSTRATE UTILIZATION DURING REST AND EXERCISE. ..... 110
$1 \quad 0 \times C$ as a function of time ..... 128
2 Respiratory Quotient (R.Q.) as a function oftime for $\mathrm{CH}-\mathrm{M}$ and Control (C)130
3 Blood lactate as a function of time after a CH-M and C ..... 133
CHAPTER 5 - EFFECT OF MEALS AND DIET ON POSTEXERCISE BLOOD LACTATE LEVELS. ..... 1431 Blood lactate concentration (mean of five sub-jects) in the postabsorptive state (followingan overnight fast) as control (lower line) andfollowing a 2.94 MJ carbohydrate meal (bananas). 159
2 Blood lactate concentration during recovery fromexercise at $80 \%$ of maximal oxygen consumption for20 minutes (mean of five subjects).161

## INTRODUCTION

In order to evaluate the energy balance, it is necessary to have an accurate method for measuring both the energy intake and energy expenditure in free living individuals; but at present there is no generally accepted method for measuring the energy expenditure (Acheson et al, 1980). The oxygen consumption (OXC) is an indirect method for measuring the energy expenditure. The heart rate (HR) has been used as a predictor of OXC in many studies (Bradfield, 1971) because it is simple to use. As pointed out by Durnin (1977), the correlation between the HR and OXC has not been thoroughly evaluated. The possible variation between tests of the linear regression between the HR and OXC has generally not been considered since the regression coefficient between the HR and OXC in a given experiment is very high. This was also true in our experiments where a continuous test with stepwise increased work rate was evaluated $r=0.99$. However, this does not mean that one will obtain the same correlation line if the test is repeated. Furthermore, it does not guarantee that $H R$ and OXC will be correlated if only small changes in workload are considered, such as that observed in the resting state. Since many people spend most of their time at relatively low levels of activity it is important to know whether the HR can predict the OXC at rest and during low work loads.

Another question that has not been answered is whether the regression equation for HR and OXC obtained during exercise is also valid during the recovery period following exercise. This is important,
since most subjects probably spend more time recovering from physical activity than actually engaged in it. If the regression equation obtained during exercise is still valid during recovery, this implies that $H R$ and $O X C$ return to the resting state at the same speed. While the OXC in low intensity exercise has been shown to return to resting values within 1-3 minutes, the HR recovery was slower (Linnarsson, 1974). Thus, it is important to evaluate the time relationship between HR and OXC during recovery and the effects of intensity and duration of the exercise.

Diet is often mentioned as a factor that may alter the HROXCR (Durnin, 1978). Physical activity, however, rather than diet, was found by Lundgren (1946) to be the factor responsible. The effect of exercise and dietary modifications was therefore included in the study.

The term "specific dynamic effect" goes back to Rubner (1902). Recently, however, Garrow and Hawes (1972) have forwarded the view that all the energy sources (fat, carbohydrates and proteins) have an equal thermogenic effect. Based primarily on overfeeding experiments in pigs, Gurr et al. (1980) have concluded that it is carbohydrate rather than protein that exerts the primary thermogenic effect. There is presently much research activity (James and Trayhurn, 1981; Rothwell and Stock, 1979) in dietary induced thermogenesis (DIT). This is because variations in DIT are thought to be an explanation for obesity in many people. However, a criticism of many of these studies is that physical activity is not well controlled. When a thermogenic response is measured as an increase in OXC or heat production, it takes very little activity to exceed the thermogenic effect of a meal. The
thermogenic response to a fat, protein and carbohydrate meal was therefore determined in human subjects by measuring the increase in OXC in the resting state.

Many investigators believe that exercise will augment the thermogenic response to meals and/or overeating (Bray et al, 1974); however, others (Norgan and Durwin, 1980; Glick et al, 1977) have found no such effect. It is possible, as pointed out by Bray, et al (1974) that there is a critical meal size below which no effect is seen. An increased thermogenic response to a normal sized meal could have important implications for weight control and was therefore investigated in the present study.

It has been shown that a carbohydrate-free diet increases fat utilization during exercise compared to a carbohydrate-rich diet (Bergstrom et al, 1967). It is therefore generally assumed that diet can modify substrate utilization; but the experimental evidence is conflicting. Hurni et al (1982) found an effect at rest but not during exercise. Ahlborg and Felig (1977) found an effect during low intensity exercise. Costill et al (1977) by elevating plasma FFA with fat meal and heparin found an effect during high intensity exercise; whereas Bergstrom et al (1969) found an effect during low but not high intensity exercise. Finally, Brooke (1981) found no effect regardless of the exercise intensity. However, it has been shown that when glycogen is depleted there is an effect of carbohydrate upon the carbohydrate utilization during exercise (Maughan et al, 1978; BOnen et al, 1981). It was therefore decided to study the effects of fat, carbohydrates and protein meals upon the substrate utilization without
affecting the glycogen stores during exercise at $80 \%$ of the maximal OXC. The concept of a percentage of maximal oxygen consumption as an equalizer of the exercise load is almost universally used. It is, however, often criticized because people with the same maximal oxygen consumption may be in different degrees of endurance training. For this reason, many investigators have considered the use of blood lactate as an equalizer of the work load (Katz et al, 1978). If the exercise intensity is increased gradually, the blood lactate will increase slowly at first until it reaches about $2 \mathrm{mM} / 1$ (40-60\% of maximal oxygen consumption). It then increases somewhat faster until it reaches about $4 \mathrm{mM} / 1$ (65-90\% of maximal oxygen consumption), at which time it begins to increase very rapidly. Two and $4 \mathrm{~mm} / 1$ lactate is arbitrarily defined as the aerobic and anaerobic thresholds, respectively (Skinner and McLellan, 1980). These levels of blood lactate, however, are of the same order as one could expect following a carbohydrate load (Owen et al, 1980); and diet, therefore, presumably could affect these thresholds without having any effect upon the aerobic/anaerobic metabolism. For this reason, blood lactate was investigated during recovery from exercise at $80 \%$ of maximal oxygen consumption following different dietary treatments.

CHAPTER 1

LITERATURE REVIEW

## LITERATURE REVIEW

## INTRODUCTION

The measurement of the work output component by the heart rate (HR) method and some aspects of dietary induced thermogenesis will be the subject of this review. It will also include the effect of diet upon substrate utilization during rest and exercise, and dietary effects upon whole blood lactate production during rest and exercise.

## THE ENERGY EQUATION

A simplified energy balance can be written as follows (Brobeck, 1981):

Food Intake $=$ Work Output + Heat Loss + Change in Energy Stores

As Brody (1945) pointed out, provided an animal stays alive, body weight will eventually stabilize at some level. At this level, food intake has equilibrated with the other three components of the equation. The conventional concept that the body energy stores are controlled by a single set point, such as the size of the fat stores, has recently been challenged (Brobeck, 1981). It is argued that any set point depends upon a number of factors: (1) dietary composition, (2) work output, and (3) environmental temperature. Regardless of the set point, the above equation remains true at all times, provided all the terms are determined at the same point in time.

It should be mentioned that the change in energy stores are not necessarily equivalent to a change in body mass, because different energy stores have different energy density (Garrow, 1974). The term "heat loss" is conveniently divided into several distinct components:

$$
\text { Heat Loss }=\text { RMR + CIT + DIT + TISE + LUXC }
$$

where:
RMR = Resting metabolic rate
CIT = Cold induced thermogenesis
DIT = Dietary induced thermogenesis
TISE = Energy required to build new tissue and store energy LUXC $=$ Luxus consumption

Most of these terms are well known, but a few require further comment. The basal metabolic rate (BMR) is well known, but because of difficulties in measuring it, RMR is often used instead (Dauncey, 1979). One cannot expect deposition of energy and synthesis of new tissue to be 100 percent effective, and consequently, Norgan and Durnin (1980) have proposed the TISE to account for the cost of change in tissue and energy stores. The term LUXC was proposed to account for the fact that overfeeding did not result in the expected weight gain (Apfelbaum, et al, 1971). The term, however, is very controversial (Norgan and Durnin, 1980).

## HEART RATE AS A MEASURE OF ENERGY EXPENDITURE

It is well known that the HR is linearly related to the oxygen consumption (OXC) (Astrand and Rodahl, 1977). This should not be
interpreted as a causative relationship, since other factors affect the delivery of oxygen to the peripheral tissue. These factors include arterial-venous oxygen difference (AVOD), stroke volume (SV), and hemoglobin concentration (HEMC).

$$
O X C=H R \times S V \times A V O D
$$

Astrand, et al (1964) found that the AVOD increased gradually from about $7 \mathrm{ml} \mathrm{O}_{2} / 100 \mathrm{ml}$ blood at rest to $17 \mathrm{ml} \mathrm{O}_{2} / 100 \mathrm{ml}$ blood during maximal exercise, and that the SV increased from $63 \%$ of maximal volume at rest to $100 \%$ at $40 \%$ of maximal OXC, and then remained constant as the exercise load was increased further.

It has been shown that the body position affects the relation between HR and OXC (HROXCR) (Andrews, 1969; Sato and Tanaka, 1973; Vokac, et al, 1975). The primary factor here appears to be whether or not the legs are involved in the exercise, because of the effect venous pooling in the legs has on the venous return, and thereby, presumably, on the SV.

Another factor that affects the $H R O X C R$ is environmental temperature (Williams, et al, 1962; Rowell, et al, 1966; Myhre, et al, 1979; and Studier, et al, 1975). The HR increases at both lower and higher temperatures with a minimum $H R$ at about $20^{\circ} \mathrm{C}$. While the cardiac output (CO) is unchanged at elevated temperatures, the SV is decreased with a corresponding increase in the HR.

Training also appears to affect the HROXCR (Saltin, et al, 1968; Pannier, et al, 1980; Taylor, et al, 1963). Training generally decreases the resting $H R$ and increases the blood volume, and thereby
presumably the SV. Its effect upon the $H R O X C R$ is to increase the slope and decrease the OXC at a given HR.

Sex and age are two factors that affect the HROXCR (Dill and Consolazio, 1962; Montoye, et al, 1968; and Sheffield, et al, 1978). HR generally decreases with age in both sexes and it is higher in females than in males, which may partly be a training effect. As a result, women generally have less slope on the HROXCR than men. The effect of age is unknown because both $H R$ and OXC decrease with age.

Stress and added weight are also known to have an effect on the HROXCR (Taylor, et al, 1963; Saltin, et al, 1968; and Borghols, et al, 1978). As expected, the effect of added weight would cause a linear increase in both the $H R$ and OXC, and presumably there will be no change in the regression line if the extra weight is taken into account. The effect of stress, in general, is not well studied, but presumably it will have an effect upon both HR and OXC. However, it may still change the regression line, and in certain situations, it has been shown to have an effect upon the HROXCR.

It is still controversial whether it is better to use an individual calibration line for the $H R O X C R$ for each subject or to use a common calibration line for all subjects (Bradfield, et al, 1970; Andrews, 1971; Bradfield, 1971; Payne, et al, 1971; and Astrand and Rodahl, 1977). Andrews (1971) reported that using incremental HR (Actual HR Resting HR) removed the differences among the subjects.

The effects of meals and exercise on HROXCR are little studied, but it has been reported that previous exercise affects the HROXCR, presumably by decreasing the SV; however, the duration of this effect is not
known. Conflicting results have been reported for the effect of meals on HROXCR; thus, Lundgren (1946) found it to be due to physical activity, but Schutz, et al (1981) have recently reported an effect of meals.

## RELATION BETWEEN HEART RATE, STROKE VOLUME, AND CIRCULATION

There are many factors controlling the HR, but the central nervous system (CNS) plays a dominant role (Nyberg, 1981). Thus, during supine rest, the sympathetic nervous system contributes about $25 \%$ to the control of the HR, while the parasympathetic system dominates; but at maximal exercise, the sympathetic nervous system's contribution is virtually $100 \%$. The parasympathetic contribution decreases simultaneously to almost zero at maximal exercise. During acute circulatory stress, such as exercise, the arterial pressure is controlled almost entirely by nervous reflex mechanisms (Guyton, 1981). However, it has been shown (Billman, et al, 1981) that the baroreflex control of the heart rate is reduced by central blood volume shifts (i.e., a smaller decrement of $H R$ is obtained by a given systolic blood pressure increase, when central blood volume is increased).

It is well known that $H R$ decreases with training (Lewis, et al, 1980). These authors found that the bradycardia of training was not due to an autonomic component, but that the decrease in HR paralleled a decrease in OXC during constant workload; and they concluded that it might be due to the cardiac enlargement. However, it has also been shown by some (Ekblom, et al, 1972; Gulbring, et al, 1960; and Pace, et al, 1947) that an increase in blood volume (as also occurs during exercise training) increases the oxygen delivering capacity, which
could operate through an effect on the SV. Apparently, however, this is not the case (Ekblom, et al, 1976); and the necessary adjustments are made in the oxygen extraction.

It has been observed that dehydration, whether caused by heat exposure or exercise, causes a decrease in plasma volume (Saltin, 1964a; Saltin, 1964b; and Costill and Fink, 1974). In submaximal exercise following the dehydration, SV was decreased and the HR increased, but at maximal exercise, there was no difference. Damoto, et al (1966) found that a change in body position from supine to standing caused a decrease in SV and an increase in $H R$, with only a slight decrease in cardiac output.

Roberts and Wenger $(1979,1980)$ have recently obtained similar results for the SV and HR. Additionally, they found that at high environmental temperatures, the skin blood flow increases, compared to cooler temperatures. This leads to an increased cutaneous blood flow in order to divert the metabolic heat. However, at high levels of skin blood flow, peripheral vascular pooling and fluid losses by filtration leads to reduced central venous pressure. This, in turn, lowers SV and increases HR. Reflexes which arise from receptors in working muscles produce vasoconstriction in a number of central and peripheral vascular beds. In the long term, physical conditioning and heat acclimation lead to increases in sweat output during thermal stress, which decreases the loss of vascular volume due to exercise and heat stress (because of more effective cooling and less cutaneous pooling of blood).

Senay (1970) and Gaebelein and Senay (1980) have likewise found that endurance training results in modification of the vascular dynamics
during exercise; that is, vascular volume becomes stabilized. In the untrained individual, heat exposure exaggerates body fluid shifts during exercise. With training, stability of the vascular volume is attained during heat exposure, but maximum protective responses towards exercise in heat are only gained upon heat acclimation.

It has been shown (Nadel, 1980; and Nadel, et al, 1980) that plasma volume is lost continuously throughout exercise, particularly at higher intensities, but the loss is most drastic during the first six minutes (at $70 \%$ of maximal $0 X C$ ). This may be the reason why the HR continues to increase throughout exercise ( $80 \%$ maximal OXC), but most drastically at the beginning (Tanaka, et al, 1979) and does not return to preexercise levels until after 45 minutes of recovery. There are mechanisms to compensate for loss of vascular volume during exercise. Thus, vasoconstriction in the splanchnic (Rowell, et al, 1968) and renal (Grimby, 1965) vascular beds has been observed during exercise. Further reduction of blood flow to the non-exercising muscle also occurs (Johnson and Rowell, 1975). Finally, it has been demonstrated that during heavy exercise, as it becomes increasingly difficult to compensate for loss of vascular volume by increased HR, a cutaneous vasoconstriction superimposes itself upon the vasodilator drive at a certain point as skin blood flow increases (Brengelmann, et al, 1977; Johnson, et al, 1974; and Nadel, et al, 1979).

At a given intensity of exercise, it has been demonstrated (Nielsen, et al, 1971; Greenleaf and Castle, 1971; and Nadel, et al, 1980) that the internal body temperature is higher in dehydrated subjects compared to normally hydrated subjects at a given work
intensity. It has also been demonstrated that dehydration causes a decrease in SV during exercise (Saltin, 1964b; and Nielsen, et al, 1971). Thus, the circulatory instability during exercise depends upon the initial state of hydration (Nadel, et al, 1980). Therefore, the variation in total body water with muscle glycogen changes may become important (Olsson and Saltin, 1970).

## THERMOGENESIS

Thermogenesis has recently received considerable interest because of its possible role in the regulation of body weight or body energy stores. It is believed that the overweight of genetically obese mice (ob/ob) is primarily due to decreased energy expenditure (Trayhurn, et al, 1979); and the ability of young lean rats to prevent obesity is probably due to increased dietary induced thermogenesis (DIT) (Rothwell and Stock, 1979).

Non-shivering Thermogenesis
Shivering involves the contraction of the muscles in order to produce heat. Non-shivering thermogenesis (NST), on the other hand, takes place without muscular contraction. Jansky (1973) has divided NST into basal NST and regulatory NST. The basal NST constitutes the heat production involved in BMR, while the regulatory NST is produced in response to specific thermoregulatory requirements. It has not been determined if DIT is regulatory or basal (Dauncey, 1979). Cold-induced Thermogenesis

It is sometimes assumed that cold induced thermogenesis (CIT) need not be considered in man, since he does not normally expose himself to
cold severe enough to induce a metabolic response (Garrow, 1978). However, Dauncy (1979) found a significant thermogenic response in man due to slight $\left(6^{\circ} \mathrm{C}\right)$ changes in ambient temperature, which indicates that man at least has the capacity for CIT (NST). Obese people have more thermal insulation than lean counterparts, which results in a reduced heat loss. And obese individuals appear to have a lower metabolic rate in.the cold, compared to normal individuals (Buskirk, et al, 1963; Jequier, et al, 1974; and Keatinge, 1960). By analogy to obese mice (ob/ob), this, however, may be due to a decreased CIT (Trayhurn, et aT, 1979). The increased metabolic rate of Eskimos in the cold may be a result of their high protein diet (Heinbecker, 1931). Dietary-induced Thermogenesis (DIT)

A number of terms have been used for DIT: post-prandial thermogenesis, specific dynamic action, and luxus-consumption. However, each covers more or less well-defined aspects of DIT. Thus, postprandial thermogenesis refers to the increased metabolic rate which follows a meal; specific dynamic action is the response in metabolic rate to a protein meal; and luxus consumption relates to the presumed increase in heat production following overeating. DIT covers both the immediate and long-term increase in heat production directly related to feeding, and this term is commonly used today. The basal DIT consists of the inevitable energy cost of digesting, absorbing, and processing or storing substrates, such as conversion of glucose to glycogen or fat and its storage in tissue, which may need to be synthesized. LUXC, on the other hand, is purely regulatory, if it exists at all (Trayhurn and James, 1981; James and Trayhurn, 1981).

The most convincing evidence in support of LUXC comes from a study in pigs (Miller and Payne, 1962) fed a low protein diet. On the low protein diets, the piglets ate five times as much energy as the ones on a high protein diet, without gaining any more weight. These studies have recently been repeated by Gurr, et al (1980). Similar experiments have also been carried out in rats fed a "cafeteria diet" (Rothwell and Stock, 1979). These authors also found that insulin plays a role in the DIT (Rothwell and Stock, 1981). This phenomenon has also been found in adult human subjects (Miller and Mumford, 1967). In some of the animal studies, there is reason to seriously question if the activity of the animals was controlled. Thus, Gurr, et al (1980) states that activity could not be involved, since the pigs were chained. However, it is easy to conceive that the chaining could cause the animals to have even more muscular activity; and there is evidence that diet may affect activity (Hart, 1978; Schemmel, 1967). The study in piglets (Gurr, et al, 1980) was also carried out in older pigs, and there, the excess energy intake on a low carbohydrate diet was stored as fat. In the cafeteria-fed rats (Rothwell and Stock, 1979), an increased metabolic response to norepinephrine is also seen in cold acclimatized rats; and warm adapted rats cease shivering much sooner when exposed to cold after being fed a "cafeteria diet" compared to a chow diet. This has led to the view that CIT and DIT are based on a similar mechanism (Trayhurn and James, 1981).

## Control Mechanisms

## Brown Adipose Tissue

According to recent studies by Foster and Frydman (1978a, 1978b, and 1979), brown adipose tissue (BAT) is the major site of NST, while skeletal muscle plays little or no role. According to the works of Nicholls (1979), thermogenesis is initiated by the release of norepinephrine, which binds to a receptor on the plasma membrane of the brown adipocyte. Adenyl cyclase is then activated and C-AMP is produced. This in turn activates the triacylglycerol lipase which causes release of FFA and glycerol. When the FFA's are oxidized in the mitochondria, protons $\left(\mathrm{H}^{+}\right)$are produced which pass through the mitochondrial membrane. Normally the passage of $\mathrm{H}+$ back through the membrane is linked to the synthesis of ATP, but in BAT, the $\mathrm{H}+$ gradient can be dissipated by the movement of $\mathrm{H}+$ through a proton conductance pathway, without ATP synthesis in association with a specific protein (uncoupling protein) unique to BAT. Joy (1963) has demonstrated a metabolic response of cold acclimatized subjects to NE.

## Hormones

Kuroshima and Yahata (1979) have shown that alucagon has twice the thermogenic response of norepinephrine, and that unlike the response to $N E$, the glucagon response is not affected by cold acclimatation (although reduced by heat acclimatation).

Danforth and Burger (1981) state that the two hormones involved in regulation of thermogenesis are thyroid hormone ( $\mathrm{T}_{3}$ ) and centrally released catecholamines (NE). They seem to work in combination to regulate cellular thermogenesis. $T_{3}$ appears to be responsible for
slow adjustments, while NE with its fast disappearance, appears to regulate the fast adjustment in thermogenesis. Thyroid hormones appear to regulate the sensitivity of BAT to NE. Shetty, et al (1979) found that during energy restriction, when the calorigenic hormones ( $T_{3}$ and NE) decrease, there was a decrease in thermogenesis. But when levadopa (a precursor of NE) was given, there was no decrease in BMR, despite a fall in $T_{3}$. This indicates that NE is the primary short-term regulator of thermogenesis.

## Thermogenesis and Exercise

Miller, et al (1967) found an almost $60 \%$ increase in energy expenditure during exercise following a 4.75 MJ meal. The subjects participated in a mild stepping exercise (mounting an 11-inch step 12 times per minute for 30 minutes). Apfelbaum, et al (1971) likewise found an increase in energy expenditure ( $20-30 \%$ ) during exercise following a 6.2 MJ supplement per day for 15 days. Bray, et al (1974) reported that breakfasts of 4.2 or 12.6 MJ increased the RMR by $10 \%$ and the metabolic rate following exercise by $20 \%$. Neither response was affected by the size of the meal. Bray, et al (1974) found no thermic effect during exercise after overeating ( $16 \mathrm{MJ} /$ day) for 30 days. In contrast, Swindells (1972), found no effect of exercise following meals of 2.5-3.8 MJ. Bray, et al (1974) has concluded that this may be due to the smaller size of their meals. Norgan and Durnin (1980), during a six-week over-feeding study, found no thermic effect during exercise, when the weight gain was taken into consideration.

## SUBSTRATE UTILIZATION

It is well established both from the respiratory quotient (RQ) (Christensen and Hansen, 1939) and from ${ }^{14} \mathrm{C}$ turnover studies (Ahlborg, et al, 1974) that in the resting postabsorptive state FFA is the major energy substrate. During rest as well as exercise, it is, however, necessary to consider the availability of substrate. During exercise it also becomes necessary to account for both the type of exercise, and its duration and intensity. Furthermore, the two major substrates (fat and carbohydrate) can be supplied intramuscularly or through the blood.

## Glycogen

Above $90 \%$ of maximal oxygen consumption, glycogen is the most important substrate, but it is far from depleted at exhaustion due to the relatively short intensity which exercise of this duration can be sustained (Hultman \& Bergstrom, 1973). At $70-80 \%$ of maximal oxygen consumption, glycogen concentration decreases in a curvilinear fashion and the time of exhaustion often coincides with glycogen depletion (Hultman and Bergstrom, 1973). Below $60 \%$ of maximal oxygen consumption, muscle glycogen, however, is not depleted at exhaustion; but liver glycogen may become a limiting factor because blood glucose is a very important substrate (Hultman and Nilsson, 1973). Intramuscular Lipids

Below $60 \%$ of maximal oxygen consumption, FFA is the most important substrate; however, blood born FFA only accounts for about $50 \%$ of the total lipid oxidation, and the rest is derived from local lipid stores (intra- and intermuscular lipid stores). It has also been
shown that utilization of blood born FFA is proportional to the arterial concentration (Hagenfeldt and Wahren, 1972; Essen, 1977). Intramuscular lipid stores appear to supply FFA whenever blood born substrates are not available in sufficient quantity.

Different modes of exercise involve different muscles to different degrees. The higher the work intensity, the more the type II fibers are recruited, and it has been shown that type II fibers are more likely to use glycogen as substrate than type I fibers. Furthermore, type I fibers can continue to use intramuscular fat as substrate even if the glycogen stores are depleted (Essen, 1977).

## Control of Substrate Utilization

## Glucose

Several reactions in glycolysis are nonequilibrium: glucose transport, hexokinase, phosphofructokinase, and pyruvate kinase; and none of these pathways appear to be substrate saturated. Thus, blood glucose appears to be the flux generating step; the blood glucose, in turn, provides a feedback link between the glucose utilization and the rate of glycogenolysis in the liver (Newsholme and Crabtree, 1979).

The muscle hexokinase shows product inhibition ( $G-6-P$ ), whereas inorganic phosphate $(\mathrm{Pi})$ is an activator. Thus, when $G-6-P$ builds up in the cell, hexokinase is inhibited and glucose concentration will increase until it reaches equilibrium with the plasma level. The phosphofructokinase (PFK) is activated by ADP, AMP, Pi and creatine-P and inhibited by the ATP/F-6-P ratio and creatine-P. Thus, glucose transport, hexokinase and PFK act in concert to control the flux of
glucose (Newsholme and Start, 1973; Newsholme, 1977; Newsholme and Crabtree, 1979).

## Glycogen

Glycogen utilization is regulated by the enzyme phosphorylase, which exists in two forms ( $a$ and $b$ ). Phosphorylase $b$ is under metabolite control: ATP and G-6-P inhibit, whereas AMP and Pi activate. Thus, its activity is increased in concert with PFK to insure adequate substrate supply if the demand is higher than can be supplied by blood glucose and FFA. Under the influence of epinephrine (E) and nervous impulse, phosphorylase $b$ is converted to phosphorylase $a$, which always has maximal activity; the result being that during exercise of high intensity (with release of NE from CNS and E from the adrenal medulla); particularly at the onset of exercise, glycogenolysis is fully activated (Foulkes and Cohen, 1979; Galbo, et al, 1977; Essen, 1978; Newsholme, 1977). The glycogenolysis during heavy exercise is of sufficient magnitude to prevent utilization of blood glucose and can actually lead to a net release of glucose from the working muscle (Wahren, 1970; Ahlborg, et al, 1974; Essen, 1978).

FFA
Citrate generated by the metabolism of FFA will inhibit PFK, and, therefore, glucose utilization as long as sufficient substrate (FFA) to cover the demand is present. As the intensity of exercise increases, sufficient oxidation of FFA is no longer possible and AMP, $\mathrm{NH}_{4}{ }^{+}, \mathrm{Pi}$, and FDP increase and release PFK from the citrate inhibition, thereby facilitating the increase in glucose utilization (Newsholme, 1977).

## Hormonal Control

It has been shown that E increases C -AMP which activates protein kinase that phosphorylates a phosphorylase kinase, which in turn converts the phosphorylase a to phosphorylase b. Recently, it has also been demonstrated that E also causes phosphorylation of a prote in (inhibitor-1) which inhibits the phosphorylase phosphatase that dephosphorylate phosphorylase a to phosphorylase b. The above reactions are also stimulated by CNS stimulus (Foulkes and Cohen, 1979).

Insulin decreases during exercise, and the decrease appears to be caused by the increased $\alpha$-adrenergic activity, which inhibits the insulin secretion. Glucagon, on the other hand, increases during prolonged exercise, and this increase correlates with a decrease in the blood glucose concentration. At the same time, there is an increase in adrenal $E$, and these hormones also control the output of glucose from the liver. NE and $E$ increase with the intensity of exercise, while glucagon is primarily affected by the duration of exercise when blood glucose decreases. Exercise also causes changes in growth hormone and cortisol (Galbo, et al, 1977).

## Other Factors

Caffeine has been shown to affect substrate utilization (by increased lipid oxidation, both during rest (Acheson, et al, 1980) and during exercise (Costill, et al, 1978), presumably by FFA mobilization. Training increases fat utilization (Johnson, et al, 1969). It is interesting to note that during heavy exercise, there is an increase in glucose utilization despite a decrease in the insulin/ glucagon ratio (Wahren, 1979). The fall in insulin during exercise
decreases with training (Wirth, et al, 1979). During exercise, kinin is liberated from kininogen and this has been shown to greatly increase the effect of insulin on glucose uptake by skeletal muscle in man (Dietze, et al, 1980). Nicotinic acid decreases FFA mobilization from adipose tissue, and heparin increases FFA mobilization. Costill, et al (1977) found that elevating plasma FFA with heparin at $70 \%$ maximal oxygen consumption decreased the rate of muscle glycogen utilization by $40 \%$. However, experiments where adipose tissue lypolysis was blocked with heparin, showed there was no effect on glycogen utilization at high intensity work (Bergstrom, et al, 1969). It is possible that greatly elevated FFA (by heparin in combination with a fat meal) does increase, even though a decreased blood FFA does not affect fat oxidation at high intensity work.

## Effect of Meals and Exercise on Substrate Utilization

Hurni, et al (1982) found that during rest the diet quickly affects the substrate utilization, but during exercise of relatively low intensity, it had no effect upon the substrate used. As will be described in the following, many investigators have found an effect of diet upon substrate utilization during exercise; however, it is necessary to consider whether or not the glycogen stores were affected, as well as the intensity of exercise. Hurni, et al (1982) used a whole body calorimeter for determination of $R Q$, and since the subjects were fed regularly, there is no reason to expect a significant effect upon the glycogen stores.

## Effect of Meals on Blood Substrate Levels

Stock (1980) found that a 1.67 MJ meal after one day of fasting significantly decreased the blood FFA level and increased blood glucose. Owen, et al (1980) similarly found that after a typical American breakfast (3.2 MJ), blood glucose, lactate, and triglycerides increased for 2-3 hours; whereas blood levels of FFA were depressed for about 4 hours. Alanine and total amino acids were also increased, while urea nitrogen was depressed for at least four hours. Crapo, et al (1981) likewise found an increase in blood glucose and insulin, while FFA were depressed following a standard meal.

Low Intensity Exercise Without Any Change in Muscle Glycogen
Luyckx, et al (1978) studied the effect of 100 g of orally ingested glucose upon plasma glucagon, FFA, and insulin. During rest, they found the expected increase in blood glucose and insulin, and a decrease in FFA and glucagon. The glucose was ingested during exercise ( $50 \%$ maximal oxygen consumption) or before the start of exercise. When preexercise glucose was compared to no glucose intake, blood glucose was elevated the first two hours of the exercise period. From 2 to about 3 $\frac{1}{2}$ hours, the blood glucose was depressed compared to control (no glucose), with a rebound after about 4 hours. As expected, plasma insulin fell in the control trial, but after the preexercise glucose meal, insulin rose in the same manner as in the resting state. After glucose, the plasma FFA was depressed in the same manner as during rest, whereas in the control trial, the FFA continued to rise throughout exercise. Preexercise glucose ingestion also prevented the expected increase in glucagon. When the glucose
was ingested after 15 minutes of exercise, the raise in blood glucose was reduced by about $1 / 3$ compared to the preexercise glucose trial. Plasma insulin neither rose nor fell, and plasma FFA and glucagon rose, although less than during the control experiment.

Using the same experimental protocol with $100 \mathrm{~g}{ }^{13} \mathrm{C}$-glucose as metabolic tracer, Pirnay, et al (1977) found that ingestion of glucose after 15 minutes of walking on the treadmill did not affect protein utilization ( $1-2 \%$ ), but increased the percentage of energy derived from glucose. It significantly reduced the utilization of endogenous glucose.

Ahlborg and Felig (1977) studied the effect of ingestion of 200 g of glucose on substrate utilization during exercise at $30 \%$ maximal oxygen consumption for 4 hours. They found that arterial glucose increased $35 \%$, arterial glycerol decreased $65 \%$, and FFA failed to increase for 2 hours of exercise in the glucose-fed group compared to control. Plasma insulin increased two- to three-fold, but glucagon levels decreased $70 \%$ compared to control. Glucose uptake in the leg (by catheterization) was increased $55 \%$ compared to $35 \%$ in controls, and splanchnic glucose output was about $110 \%$ increased, whereas splanchnic uptake of gluconeogenic precursors was decreased ( $80 \%$ ) compared to control. So at low levels of exercise ( $<60 \%$ of maximal oxygen consumption), preexercise glucose increases glucose utilization and decreases fat oxidation, and decreased hepatic gluconeogenesis. The ingestion of glucose, while increasing total glucose utilization, spares the endogenous glucose. It appears to make a difference if the glucose is ingested before or during exercise, and these changes
are hormonally determined.
Low Intensity Exercise with Glycogen Depletion
It has been observed that a greater proportion of an oral glucose load escapes the hepatic retention in glycogen depleted subjects compared to control (Maughan, et al, 1978). This glucose is taken up preferentially by the depleted muscle. Rennie and Holloszy (1977), however, found that a high plasma FFA level, which occurs after glycogen depletion, inhibits muscular glucose uptake as well as its oxidation. Ravussin, et al (1979) studied the effect of exercise one hour following 100 g orally ingested ${ }^{13} \mathrm{C}$-glucose at $40 \%$ of maximal oxygen consumption in normal controls and glycogen depleted subjects. The major difference between the two groups was that the glycogen depleted subjects used primarily fat as substrate ( $70 \%$ ), whereas the control subjects used primarily carbohydrate ( $65 \%$ ). The exogenous glucose represented $20 \%$ and 24\%, respectively. In the depleted subjects, plasma FFA remained 2-3 times higher than in control. So despite glycogen depletion, these subjects did not use exogenous carbohydrate to a higher extent.

During exercise following glycogen depletion (by prior exhaustive exercise) and either a normal-, low-, or high-carbohydrate diet, a lower RQ, blood lactate, blood glucose, and blood triglycerides were found compared to elevated plasma FFA and plasma glycerol following a low carbohydrate diet than a high CHO diet. The control (normal) was very similar to the high carbohydrate diet in all aspects studied (Maughan, et al, 1973).

## High Work Intensity and Glycogen Depletion

Bonen, et al (1981) used a regimen consisting of exhaustive exercise followed by about 40 hours of fast to deplete muscle and liver glycogen. The authors then studied the response to ingestion of carbohydrate ( $1.5 \mathrm{~g} / \mathrm{kg}$ ) 15 minutes prior to exercise or $3-5$ minutes after the start of exercise. An exercise group with no glucose intake, and a rest group with glucose were used as control. As one could expect, the exercising control had no increase in blood glucose, and the increase during exercise was less than during rest, both in regard to blood glucose and insulin. During these workloads ( $80 \%$ maximal oxygen consumption), the preexercise glucose group had an initially sharp increase in both glucose and insulin (prior to exercise) followed by a sharp decrease during exercise ( 25 minutes) and a moderate increase during recovery. The during exercise glucose group of course did not have the preexercise increase, but both blood glucose and insulin was very similar to the pre-exercise glucose group, during exercise, with a more sharp increase during recovery.

Maughan and Poole (1981) compared subjects with depleted, normal, and supercompensated glycogen stores at $105 \%$ of maximal oxygen consumption. Because of the rapid production of lactate at this high workload, it was not possible to determine substrate utilization (invalid RQ), but depleted subjects had lower blood glucose than normal and supercompensated subjects.

Martin, et al (1978) studied the effect of exhaustive exercise following a normal-, high carbohydrate-, and high-fat-diet for 3 days in
humans. The high fat diet provided $90 \%$ of energy from fat and the high carbohydrate diet, $75 \%$ of energy from carbohydrate. The effect of these dietary regimens on muscle glycogen are uncertain, but they may very well have had a significant effect, particularly since the exercise was exhausting. However, the high fat-protein diet had significantly lower RQ than either the mixed or high carbohydrate diet, and the blood concentration of FFA was elevated.

## High Work Intensity Without Glycogen Depletion

Jones, et al (1980) compared heavy exercise ( $70 \%$ maximal oxygen consumption) with light exercise ( $40 \%$ maximal oxygen consumption) during 40 minutes. As expected, relatively less fat was used during the heavy work. During light work, palmitate turnover rate $\left({ }^{14} \mathrm{C}\right)$ was unchanged from rest, but a $40 \%$ decrease was observed during heavy work, as well as a fall in plasma FFA. However, during heavy work, plasma glycerol was increased ( 5 -fold) compared to light work. This was interpreted to mean that heavy work caused a shift from adipose tissue lipolysis to muscle lipolysis.

Foster, et al (1979) compared the effect of 75 g of glucose taken before exercise with a standard liquid meal (composition similar to a normal American diet)(water was used as control) during exercise to exhaustion at 80 and $100 \%$ of maximal oxygen consumption. The authors concluded that FFA mobilization was impeded by glucose ingestion. They also found a significant decrease in serum glucose following the glucose meal after 30 minutes of exercise compared to control, despite a higher initial level. They further reported an increase in the RQ following the oral glucose load compared to control. This difference, however, was not
significant. Mainly on the strength of the decreased serum FFA, the authors concluded that a glucose meal increased utilization of glucose as substrate; it is, however, necessary to also consider intramuscular FFA mobilization, and the plasma level of substrates is no certain indication of substrate utilization during heavy exercise.

## Conclusions

During rest, it is well established that the diet affects the substrate utilization, and this probably extends to exercise of low intensities ( $<60 \%$ of maximal oxygen consumption). However, during heavy exercise, there is little support for this hypothesis. During rest and low intensity exercise, blood borne substrates (FFA and glucose) are of major importance in the supply of energy, but during heavy exercise, intramuscular substrates assume an increasing role and this change is mediated through hormones (insulin, glucagon, and E) and CNS. As a consequence, one would not expect diet to have a major effect upon the substrate utilization unless intramuscular substrate is depleted; but the experimental evidence is inconclusive at present.

## POSTEXERCISE LACTATE

## Effect of Exercise

At the start of low level exercise, there is a rapid release of lactate from the exercising muscle (100-150 mmol/min/100 ml), which gradually decreases toward resting level during 1 hour exercise. At the same time, there is net release of glucose (the first 2-3 minutes), indicating rapid glyconeogenesis. Soon, however, net uptake of glucose from the working muscle is observed.

As the work intensity increases, so does the lactate production. However, when a given work intensity is maintained for a sufficiently long time, the lactate concentration is generally considered to remain fairly constant once equilibrium has been reached (Hermansen, 1971). Freund and Gendry (1978) have fitted mathematic functions to the lactate response during recovery at rest. The blood lactate during recovery continues to increase during the first 2-6 minutes (depending on work intensity) of recovery, while there is a rapid decrease in the intramuscular lactate. After 10 minutes, there is very little difference between muscle and blood lactate (Freund and Gendry, 1978).

Skinner \& McLellan (1980) have divided exercise into three phases (1-III). During phase I, the RQ is between 0.7 and 0.8 and little or no lactate is formed during this low intensity steady state exercise. Between 40 and $60 \%$ of maximal oxygen consumption, phase II is reached and blood lactate level is about doubled. When the work intensity is increased further ( $65-90 \%$ maximal oxygen consumption), the blood lactate will rise above 4 mM , and then begin to increase rapidly as the subject approaches his maximal oxygen consumption. Above a subject's maximal oxygen consumption, blood lactate continues to increase with time and no steady state is reached. (Therefore, RQ is no longer a valid measurement of the substrate utilization.) The sharp rise in blood lactate with the increase in work intensity after a blood level of about 4 mM is reached (MacDougal1, 1978; Green, et al, 1979), corresponds to the anaerobic threshold.

The early research by Hill, et al (1924) concluded that lactate was produced when there was an insufficient oxygen supply. It is well known
that training decreases lactate production during exercise at the same absolute work load (Hermansen, 1971; Johnson, et al, 1969). The argument is used that since the total oxygen consumption is not increased following training or breathing a high oxygen gas (60-100\% oxygen) (Holloszy, 1976; Welch, et al, 1977; Skinner \& McLellan, 1980; Graham, 1978), hypoxia cannot be the cause of lactate production. However, this is probably incorrect, since under conditions where lactate is produced, it is reconverted to glucose in the liver or oxidized in other muscle fiber that is not hypoxic and therefore probably uses the oxygen saved during anaerobic glycolysis (for example, to reconvert lactate to pyruvate). Bylund-Fellenius et al. (1981) recently presented strong evidence that the oxygen partial pressure in the exercising muscle indeed determines the lactate production.

It has been shown that the muscle respiratory capacity is of primary importance in determining the work rate at which blood lactate accumulation begins (Ivy, et al, 1980), and it is well known that type I fibers have higher respiratory capacity and capillary density than type II B fibers (Essen, 1978). It is well known that type I fibers are preferentially recruited at low work intensity, and both the recruitment pattern, respiratory capacity, and fiber composition may be affected by training (Saltin, et al, 1977; Baldwin and Winder, 1977; Booth, 1977). The mode of exercise (intermittent vs continuous) also affects the lactate production (Essen, 1978). Intermittent exercise of twice the exercise intensity produced the same blood lactate level as continuous exercise. This difference is best explained by a greater oxygen availability during intermittent exercise due to reloading of the muscle
myoglobin stores and to the decreased utilization of muscle glycogen during the intermittent exercise. Furthermore, it is well demonstrated that during low intensity exercise, lactate is a substrate for the exercising muscle (McGrail, et al, 1978; Stamford, et al, 1981; Poortmans, et al, 1978).

## Effect of Diet

It has been shown that following a meal containing carbohydrates, blood glucose and lactate becomes elevated, while FFA are depressed (Owen, et al, 1980; Capro, et al, 1981). On this basis, one would expect an interaction between carbohydrate intake and exercise following a carbohydrate meal. However, it has also been shown that the prime precursor of lactate is muscle glycogen (Wahren, et al, 1971); furthermore, that the muscle glycogen concentration affects the lactate production (Jacobs, 1981). Lactate production was significantly reduced when glycogen levels fell below about 40 mmol glycosyl units $/ \mathrm{kg}$. When considering dietary effects upon lactate production, it is therefore important to also consider the dietary effect upon the glycogen stores.

During low intensity work after depletion of muscle glycogen stores, a high carbohydrate diet increased blood lactate compared to a low carbohydrate diet, both during exercise ( $50 \%$ maximal oxygen consumption) and recovery. As expected at these workloads, the lactate production is very low (about 1 mil ), and it may very well be a dietary effect unrelated to exercise.

In high intensity work ( $105 \%$ maximal oxygen consumption) following glycogen depletion and either a carbohydrate-free or carbohydrate-rich diet, Maughan and Poole (1981) found that carbohydrate loading resulted
in highly significant increased blood lactate following exercise compared to the carbohydrate-free regimen. Bonen, et al (1981) exercised their subjects at $80 \%$ of maximal oxygen consumption following glycogen depletion and either during control (no glucose) or $1.5 \mathrm{~g} / \mathrm{kg}$ glucose before (15 minutes) or during (3-5 minutes) exercise. They found that preexercise glucose gave the highest blood lactate during exercise and recovery, while the lowest values were observed during the control treatment. Apparently, the effect of diet when muscle glycogen is unaffected has not been studied.

## CHAPTER 2

# THE VARIABILITY OF THE HEART RATE-OXYGEN CONSUMPTION RELATIONSHIP WITH TIME AND COMPARISON OF INDIVIDUAL REGRESSION LINES VS A COMMON REGRESSION LINE FOR ALL SUBJECTS 

## Synopsis

The variation with time of the heart rate oxygen consumption relationship (HROXCR) was studied in ten subjects with 10 trials in each subject and 40 -minute recovery. A significant inhomogeneity was found in all but one subject. The significance was not decreased by using corrected heart rate (correctedHR = Actual $H R+60$ - Resting HR). The slope differed little in subjects with similar maximal oxygen consumption, although significant differences existed. The $y$-intercepts were also significantly different, and the significance did not disappear when using correctedheart rate, unless only subjects with similar maximal oxygen consumptions were considered.

It is concluded that it is best to use individual regression lines, but because of the variability within subjects, it should be based on more than one trial. Even the relatively small variations in the oxygen consumption from day to day is positively correlated with the HR.

## Introduction

The heart rate (HR) is often used as a predictor of the oxygen consumption (OXC) and/or degree of physical activity (Andrews, 1971; Payne et al, 1971; Bradfield, 1971; Bradfield et al, 1971; Astrand, 1971; Dauncy \& James, 1979). One reason for this is the good linear correlation between HR and OXC in any given experiment (Londeree \& Ames, 1976). This implies that the $H R$ is a major determinant of the oxygen supply. However, besides the HR, the stroke volume (SV) and the arterial-venous oxygen difference (AVD) are other factors used to determine oxygen supply, and all three are known to vary with the intensity of exercise (Astrand et al, 1964; Damato et al, 1966; Williams et al, 1962).

The relationship between the HR and the OXC (HROXCR) has in fact, been shown to depend upon physical training (Saltin, et al, 1968; Pannier, et al, 1980), age (Sheffield et al, 1978; Montoye, et al, 1968; Dill \& Consolazio, 1962), body position (Andrews, 1971; Vokzc, et al, 1975; Sato \& Tanaka, 1973), and environmental temperature (Dill \& Consolazio, 1962; Rowel et al, 1966; Taylor et al, 1963; Myhre et al, 1979). While sex, age and training are easily controlled, the effects of body position and environmental temperature cannot always be easily controlled. Since the effects of temperature and position on the $H R O X C R$ in general are not very large, they are probably safely ignored for many applications (Andrews, 1971).

It has not been determined if the $H R O X C R$ remains constant for a reasonable time period, although this assumption forms the basis for the use of HR to predict the OXC. Furthermore, it is controversial whether a conmmon regression equation can be used for all subjects, or groups of subjects of similar age, sex, and physical condition (Andrews, 1971), or an individual equation should be used for each subject (Bradfield et al., 1970).

The purpose of this study was to determine if the linear relationship between the HR and OXC is constant with time when no training effect is introduced. Andrews (1971) has suggested that the use of incremental HR will remove any significant differences in the regression equation between subjects, and this was also tested in this study. Furthermore, this study addressed the question of whether individual or common regression lines best predict OXC. Durnin (1978) has argued that since most activity occurs at low level, the normal variability could easily obscure true differences. It is therefore necessary to test whether the variability in OXC at resting level of activity is correlated with the HR.

## Methods

## Subjects

Ten male students, 20-30 years of age, who were moderately fit and accustomed to exercise on a treadmill participated in this study. The subjects did not participate in regular physical training or athletic competition. None of the subjects were obese, that is, they were within $\pm 10 \%$ of desirable weight (Metropolitan Life Insurance Company, 1959), or received any medication during, and at least a month prior to, the trials. Anthropometric data and maximal oxygen consumption for the subjects are given in Table 1. The subjects gave informed consent, and the protocol used was approved by the University Committee on Research Involving Human Subjects (UCRIHS) at Michigan State University.

Measurement of Oxygen Consumption (OXC) and Heart Rate (HR)
The OXC was determined through a modified Douglas method (Consolazio et al, 1963). The expired air was collected through a low resistance (Daniels) valve and collected in light-weight (neoprene) bags. The composition of the collected air was immediately determined using the Beckman LB-2 carbon dioxide and Beckman OM-11 oxygen analyzers. The air volume was determined by metering through (using constant flow) a Singer dry gas meter. The HR was determined from a 3-lead electrocardiogram, and recorded continuously on a Sargent
Table 1. Age, height, weight, and $\dot{V}_{0}{ }_{2}$ max for the ten subjects. The slopes $\mathrm{B}_{1}$ and $y$-intercepts

| Subject Nr. | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | Mean ${ }^{3}$ |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Age (years) | 20 | 26 | 25 | 23 | 30 | 30 | 29 | 20 | 29 | 26 | 26.8 |
| Height ( cm ) | 198 | 168 | 156 | 183 | 187 | 194 | 192 | 165 | 167 | 180 | 179 |
| Weight (kg) ${ }^{1}$ | 84.9 | 80.8 | 70.8 | 93.3 | 82.0 | 90.0 | 100.1 | 63.2 | 57.2 | 78.0 | 80.0 |
| $\operatorname{Max~} \dot{V}_{02}{ }^{2}$ ( $\mathrm{ml} / \mathrm{kg} / \mathrm{min}$ ) | 50.1 | 49.0 | 45.7 | 47.0 | 51.8 | 52.1 | 53.3 | 54.6 | 66.0 | 53.9 | 52.4 |
| $\bar{B}_{1}{ }^{\text {a }}$ | . 344 | . 352 | . 345 | . 344 | . 326 | . 364 | . 359 | . 375 | . 484 | . 368 | . 366 |
| $\operatorname{Var}_{1}{ }^{\text {b }}$ | . 0017 | . 0004 | . 0008 | . 0005 | . 0003 | . 0008 | . 0013 | . 0009 | . 0014 | . 0004 | . 0017 |
| $\bar{B}_{0}{ }^{c}$ | -19.5 | -15.8 | -16.1 | -13.8 | -11.5 | -16.6 | -8.6 | -21.5 | -27.6 | -17.4 | -16.8 |
| $\operatorname{var}_{0}{ }^{d}$ | 21.8 | 2.8 | 13.9 | 5.1 | 2.3 | 10.2 | 2.3 | 11.4 | 18.8 | 7.4 | 25.2 |
| $\bar{B}_{0}$ Cor ${ }^{\text {e }}$ | -17.1 | -17.1 | -16.9 | -17.1 | -16.7 | -18.8 | -18.8 | -17.5 | -27.8 | -18.2 | -18.6 |
| $\operatorname{Var}_{0}{ }^{\text {d }}$ | 7.4 | 2.4 | 3.0 | 1.4 | 2.3 | 2.8 | 5.4 | 2.4 | 5.6 | 1.4 | 9.9 |

> 'Mean of ten determinations, one prior to each exercise test.
${ }^{2}$ Mean of two determinations (in week one and ten respectively).
$3_{\text {The means of }} \overline{\mathrm{B}}_{1}$ and $\overline{\mathrm{B}}_{0}$ are obtained by calculating the regression equation based on all trials in all subjects, and the corresponding variances are the between subjects variances.
${ }^{a_{\bar{B}}}{ }^{1}$ is the mean slope of the regression equation for the ten trials in each subject.
$b_{V a r l}$ is the variance for $\bar{B}_{1}$.
${ }^{C_{B_{0}}}$ is the mean $y$-intercept of the regression equation for the ten trials in each subject.
${ }^{\mathrm{V}_{\mathrm{Var}}^{0}}$ is the variance for $\bar{B}_{0}$.
${ }^{\mathrm{e}} \overline{\mathrm{B}}_{0}$ Cor is the $y$-intercept after correction for the resting heart rate.
recorder. The mean $H R$ for the time corresponding to each bag of expired air was determined. In an attempt to reduce error due to variation in resting $H R$, a corrected $H R$ was calculated by adding or subtracting the difference between 60 and the resting $H R$ from each $H R$ measurement. 60 being the mean $H R$ is arbitrarily chosen as standard.

## Test Protocol

The ten subjects completed an incremental exercise test once a week for ten consecutive weeks. Each subject was assigned a specific day and time, on which he would arrive in the laboratory at least 5 hours postabsorptive (usually after an overnight fast). If a subject could not meet his appointment for a particular week, he would be rescheduled for another day the same week. On the test day, the subjects were instructed not to consume coffee or tea and not to engage in heavy exercise.

The imcremental exercise test was performed on a treadmill. The steps consisted of resting and the following speeds (S-2 to S-7): $4.8,6.8,8.7,9.7,12.5$, and 14.3 (miles/hour). At rest $(S=0)$ the subjects were sitting relaxed in an armchair on the treadmill, but HR and expired air was collected as during exercise. During rest, three 5 -minute bags were collected; for S-2 and S-3, three 2-minute bags; and for the remaining speeds ( $\mathrm{S}-4$ to $\mathrm{S}-7$ ), three 1 -minute bags were collected. Not all subjects completed all the steps, but the trial was terminated when the subject approached his maximal oxygen consumption as determined in two separate tests.

Measurements of Resting HR and OXC
Five different subjects (mean age $\pm S D=26.6$ years $\pm 1.7$, mean weight $\pm S D=78.2 \mathrm{~kg} \pm 11.3$ and mean height $\pm S D=182 \mathrm{~cm} \pm 6.1$ ) were used to determine OXC and HR once an hour for 9 hours. The subjects were seated in an armchair while 3 consecutive 5 minute bags of expired air were collected and the mean HR corresponding to each bag determined from the HR-recordings. The correlation coefficient between HR and OXC was calculated for a 9 hour period in each subject.

The resting values for $H R$ and OXC for 9 of the 10 subjects used for the determinations of the regression equations as described above, were used for calculation of the correlation coefficients. These correlation coefficients are therefore based on simultaneous measurements of OXC and HR on ten different days one week apart.

## Treatment of Data and Statistical Procedures

A computer program was used to calculate OXC from the composition of the expired air on a standard temperature, pressure, dry basis. The OXC for each collection period and the corresponding mean HR were used to calculate a regression equation for each trial in each subject. A regression equation for each subject, based on all ten trials, was calculated, as was a common regression equation based on all 100 trials in the ten subjects. The slopes $B_{j}$ and the $y$-intercepts ( $B_{0}$ ) for the individual trials were used as independent variables in a one-way analysis of variance (random model). The repeatability
$R=\frac{\hat{\sigma}_{\beta}{ }^{2}}{\hat{\sigma}_{\beta}{ }^{2}+\hat{\sigma}_{\omega}{ }^{2}}$ is calculated to evaluate the relative contributions of
within and between subjects variance: $\hat{\sigma}_{\omega}{ }^{2}=m S_{E}$ and $\hat{\sigma}_{B}{ }^{2}=\frac{m S_{S}-m S_{E}}{10}$ where $m S_{E}$ is the mean square error term, and $m S_{S}$ is the mean square treatment term from the analysis of variance (Gill, 1978). The sum of squares (SSEi) for the individual regression equation and the total sum of squares for the summation regression equation in each subject (SSET) are used to test for homogeneity of regression within a subject and between subjects (Gill, 1978). The critical value is $t_{\alpha}, 2(t-1)(r-2)$, where $t=$ trials and $r=n u m b e r$ of observations in a trial. Correlation coefficients were determined in the usual manner and mean and confidence intervals are calculated by transformation to $z=0.5 \ln \frac{1+r}{1-4}$ (Gill, 1978).

## Results

The regression line for a single incremental exercise test is shown in Figure 1 for a representative subject. The correlation coefficient between HR and OXC is very high ( $>0.99$ ), which is partly due to the use of mean (integrated) HR. The regression line based on all ten incremental exercise tests in a typical subject is plotted in Figure 2.

The calculated F-statistics for test of homogeneity of regression between independent samples are given in Table 2. The critical $F$ value is 1.94 ( $\alpha=0.05$ ) and in all but one subject, there is significant inhomogeneity. Also shown are the calculated F-statistics after correcting for resting $H R$ by adding $60-r e s t i n g ~ H R ~ t o ~ a l l ~ H R ~ m e a s u r e-~$ ments. The critical F-value is again 1.94 and it is seen that rather

Figure 1. Regression line between the OXC and the HR for a typical subject (\#8) based on a single incremental exercise test. The slope $B_{1}-0.377$, the $y$-intercept $B_{0}=22.5$, and the standard error of estimate $S E=0.86 \mathrm{ml} 0_{2} / \mathrm{min} / \mathrm{kg} . \mathrm{S} 2-7$ is the different speeds in the incremental exercise tests.


FIGURE 1

Figure 2. Regression line between OXC and HR in a typical subject (\#8) based on ten incremental exercist tests. The slope $\mathrm{B}_{1}=$ 0.372 , the $y$-intercept $B_{0}=21.1$, and $S E=2.03 \mathrm{ml} 0_{2} / \mathrm{min} / \mathrm{kg}$.


FIGURE 2

Table 2. Calculated F-ratio for test of homogenity within the ten trials in each subject with observed HR ( $F-1$ ) and with corrected HR (F-2). ${ }^{\text {a }}$

| Subject No. | F-1 | $F-2$ |
| :---: | ---: | ---: |
| 1 | 20.01 | 28.37 |
| 2 | 12.50 | 6.36 |
| 3 | 9.95 | 22.30 |
| 4 | 2.49 | 14.60 |
| 5 | 1.58 | 3.51 |
| 6 | 13.70 | 12.74 |
| 7 | 11.65 | 13.04 |
| 8 | 11.16 | 27.49 |
| 9 | 3.17 | 9.55 |
| 10 | 24.78 | 25.71 |
| Total ${ }^{b}$ | 245.53 | 107.00 |

a The critical F-ratio is 1.96
$(\alpha=0.05)$.
bTotal is the F-ratio for inhomogenity between subjects.

Table 3. Repeatability R, calculated and critical F-ratio ( $\alpha=0.05$ ) tor the slope $\mathrm{b}_{1}$, the y -intercept $\mathrm{B}_{0}$, the y -intercept corrected for resting heart rate $\mathrm{B}_{0}$ Cor, and $\mathrm{B}_{1}-1$ and $\mathrm{B}_{\mathrm{O}}{ }^{-1}$ Cor when the subject with a large deviation in the $\max \mathrm{V}_{2}$ is disregarded. ${ }^{\text {a }}$

|  | $\mathrm{B}_{1}$ | $\mathrm{~B}_{1}^{-1}$ | $\mathrm{~B}_{0}$ | $\mathrm{~B}_{0}^{-1}$ | $\mathrm{~B}_{0}$ Cor | $\mathrm{B}_{0}{ }^{-1} \mathrm{Cor}$ |
| :--- | :---: | :---: | :---: | :---: | :---: | :---: |
| Repeatibility R | 0.696 | 0.158 | 0.741 | 0.632 | 0.755 | 0.092 |
| Calculated F | 22.66 | 2.91 | 25.5 | 18.21 | 31.85 | 2.02 |
| Criticial F | 1.99 | 2.06 | 1.99 | 2.06 | 1.99 | 2.06 |
| aHR is corrected by adding 60-resting HR to each observed HR. |  |  |  |  |  |  |

than decreasing the inhomogeneity, it generally increases, resulting in significant inhomogeneity in all subjects. The critical Fvalue for test of inhomogeneity between subjects $1.88(\alpha=0.05)$ and the calculated $F$ is highly significant; but in this case, the inhomogeneity is drastically reduced by correcting for resting $H R$, although it is still highly significant (the F-ratio decrease from 246 to 107).

From Table 1 it is seen that one subject (\#9) has significantly higher maximal oxygen consumption than the other subjects ( 66 vs a mean of $52 \pm 2.8$ ). Since the regression equation can be expected to vary with maximal oxygen consumption, the analysis of variance is done with and without this subject. Using corrected HR does not affect the slope $\left(B_{1}\right)$, but the $y$-intercept $\left(B_{0}\right)$. The repeatability, calculated $F$, and critical $F$ are given in Table 3. $R$ can vary between 0 and 1: $R=0$ means that all the variability lies within the subjects, and $R=1$ means that all the variability lies between the subjects. From Table 3, it is seen that correction for differences in resting HR has no effect on $R$ for the $y$-intercept ( 0.741 vs 0.755 ), and removing the inhomogenous subject (\#9) has no effect in itself ( 0.741 vs 0.632 ); but using corrected $H R$ and removing subject \#9 causes practically all the variation to be within subjects ( 0.755 vs 0.092 ) . As seen from Table 2, using corrected HR drastically reduces the variation within subjects (from a mean of 25.2 to 9.9 ); but since it has no effect on $R$ and $B_{0}$ Corr is highly significant ( $F=31.9$ ) as seen in Table 3, it means that it reduces the variability equally within and among the subjects. When removing subject \#9 from the analysis and
using corrected $H R$, there is no longer evidence of inhomogeneity ( $F=$ 2.02). $R$ for the slopes is likewise reduced by removing subject \#9 ( 0.696 vs 0.158 ); but in this case, the slopes are still significantly different between subjects ( $F=2.91$ ).

In conclusion, it can be said that each subject has a characteristic $B_{1}$ and $B_{0}$ around which the individual incremental exercise test values vary. When subjects are homogenous with respect to maximal oxygen consumption, the individual variation is far more important than the variation among subjects for the slope, and also for $B_{0}$ if corrected HR is used.

## Correlation Between HR and OXC at Constant Workload

The resting HR within a subject generally varies by $\pm 10$ beats/ minute from minute to minute. But, during exercise, the short-term variation is decreased to $\pm 2$ to 3 beats/minute at high workloads. The mean HR corresponding to each bag of expired air has a short-term (15 $\min )$ variation of $\pm 1$ beat/minute or less; however, over extended test periods of up to 10 hours, there is variation in the mean HR of $\pm 5$ beats/minute. There is also a variation in the resting HR from day to day of about 15-20 beats/minute.

The mean correlation between HR and OXC taken at rest at 9 different times throughout a day for the 5 subjects was -0.032 and the confidence interval -0.199 to 0.135 . The mean standard deviations for $H R$ and OXC were 2.91 and 0.19 respectively. This means at a given workload (rest), HR and OXC vary independently of each other in the postabsorptive state. In 9 different subjects the correlation between

HR and OXC was determined from measurements taken in the resting state on ten different days. In this case the mean SD for $H R$ and OXC were 4.55 and 0.47 respectively, that is approximately doubled due to day-to-day variation. The mean correlation coefficient $\bar{r}$ was 0.478 with the confidence interval from 0.375 to 0.570 . This means that the relatively small variation in OXC observed from day to day in the resting state is significantly correlated with the $H R$.

## Discussion

The HR is frequently used as a predictor of OXC (Bradfield, 1971). The excellent regression between HR and OXC found in this study ( $r>0.99$ ) when several different workloads are considered, gives support for the use of HR as a predictor of OXC. The mean standard error of estimate (SE) for the 100 regression equations calculated in this study was 1.40 ml oxygen $/ \mathrm{min} / \mathrm{kg}$ which corresponds to $0.49 \mathrm{kcal} /$ min. This compares favorably with the value of 0.37 reported by Andrews (1971), since we generally used a wider range of workloads; and the SE tends to increase with increasing workload.

As discussed by Dauncy and James (1979), in most applications the HR varies within a relative narrow range ( $60-100$ beat/min or less). It is therefore important to see how well HR and OXC is correlated within a narrow workload. The correlation ( $r$ ) was calculated between HR and OXC in five subjects from periodic measurements throughout a day with no physical activity. Under these conditions there was no correlation between HR and OXC; and the standard deviations for HR and OXC were 2.9 and 0.19 respectively. When the correlation was based on measurements of HR and OXC on 10 different days (sub.jects again in the resting state) the correlation ( $r=0.478$ ) was statistically different from $0(\alpha=0.05)$ and could explain about $25 \%$ of the variation, the remaining variation being due to random variation in $H R$ and OXC. In the measurements from day to day, the SD for $H R$ and OXC were 4.6 and 0.47 respectively. Thus even relatively small changes in

HR are positively correlated with the OXC.
Andrews (1971) reported that substituting HR for incremental HR (IHR) (actual $H R$ - resting $H R$ ) removes the differences in $y$-intercept for the regression lines between $H R$ and OXC. Using IHR is equivalent to having all the individual regression lines going through a common point (IHR = 0 and $O X C=$ Resting $O X C$ ), since the resting OXC generally varies very little compared to the SE of the regression equation (SE $\sim 0.3$ and 1.4 respectively). When the IHR is used the $y$-intercept corresponds to the resting OXC; since this, in our experience, invariably lies on the regression line; and an analysis of variance of the $y$-intercept therefore becomes meaningless. The corrected $H R$ ( $C H R=$ actual $H R+60$ Resting HR) was therefore used in the present study. The effects of using corrected and incremental HR is illustrated in Figure 3 and Figure 4 for a hypothetical case. Like the IHR, the CHR adjust all the regression lines to a common resting HR; but instead of this being 0 it is 60 , and the variability of the $y$-intercept due to variation in slope is thus retained. Using CHR has no effect upon the slope; and only where very homogenous subjects (with respect to maximal OXC) were considered did it decrease the variability in the $y$-intercept between subjects. However, as seen from Table 2, using CHR does not decrease the variability in the $y$-intercept within a subject. It is therefore concluded that there is no advantage in using CHR (or IHR).

Whether to use a common regression equation between $H R$ and OXC for all the subjects as recommended by Andrews (1971) or an individual determined regression equation as recommended by Bradfield et al. (1970) and Astrand and Rodahl (1977) is still controversial. Since

Figure 3. Regression lines between HR and OXC for a hypothetical case: Training decrease $H R$ and increase slope. Line $a$ and $b$ illustrate variation in the HROXCR such as caused by exercise.


FIGURE 3

Figure 4. Incremental and Corrected HR. IHR = Actual HR - Resting HR CHR $=$ Actual HR + 60 - Resting HR. Both adjustments reduce the variability of the $y$-intercept; but not necessarily the difference between the regression equations.


FIGURE 4
both the slope and the $y$-intercept are significantly different even within homogenous subjects, it is always better to use individually determined regression lines. This can also be seen from the repeatability $R$ ( $R=0.70$ for the slope and 0.74 for the $y$-intercept), which means that most of the variability is between subjects even when only homogenous subjects are considered ( $\mathrm{R}=0.63$ ). The finding of significant inhomogeneity among the regression lines makes it necessary to determine the regression based on data from at least 3 exercise test spaced at least 2 days apart, in order to ascertain that the regression line is typical for the subject.

Payne et al. (1971) reported that HR and OXC obtained during sitting and standing rest did not fall on the regression equation obtained during exercise. They did find, however, that HR and OXC obtained during supine rest fell on the regression line. In the present study 15 minutes of sitting rest was used as the first step in the incremental exercise test, and the HR and OXC obtained during rest almost invariably lay on the regression line. The reason for this discrepancy may lie in how the resting measures are conducted. As described by Dauncey and James (1979) it is important that the subjects moves the legs periodically in order to avoid venous pooling.

As reported by Hirsch and Bishop (1981) the breathing pattern affects the HR and causes a cyclic variation. In the present study this effect was clearly seen at rest ( $\pm 10$ beats $/ \mathrm{min}$ ), but it disappeared at higher workloads. Since the HR was recorded continuously, and the mean $H R$ corresponding to each bag of expired air was calculated, this variation had no effects on the results.

## CHAPTER 3

> EFFECT OF DIET, PREVIOUS EXERCISE, AND
> RECOVERY UPON THE LINEAR REGRESSION
> BETWEEN HEART RATE AND OXYGEN CONSUMPTION

## Synopsis

We have studied the effect of diet composition upon the regression between heart rate (HR) and oxygen consumption (OXC) during an incremental exercise test. Our results do not give any evidence for a dietary effect. Repeated exercise one hour apart did, however, have a significant effect upon both the slope and $y$-intercept of the regression equation between HR and OXC. During recovery, the HR did not predict the OXC based on the regression equation obtained during exercise. Both the exercise intensity ( $S$ ) and duration ( $D$ ) had a significant effect upon the recovery $H R$ (RHR), whereas only $S$ had a significant effect upon recovery OXC (ROXC). Consequently, RHR lagged behind the ROXC the first three minutes of recovery, but then returned toward resting values at the same rate as the OXC. The difference between ROXC and predicted OXC (POXC) is primarily determined by D.

## Introduction

The heart rate (HR) is often used as a predictor of the oxygen consumption (OXC) and/or degree of physical activity (Andrews, 1971; Payne, et al, 1971; Bradfield, 1971; Bradfield, 1971; Astrand, 1971; Dauncy \& James, 1979). One reason for this is the high linear correlation between HR and OXC in any given experiment (Londeree and Ames, 1976). This implies that the HR is a major determinant of the oxygen supply. However, besides the HR, the stroke volume (SV) and the arterial-venous oxygen difference are other factors that determine the oxygen supply; and all three are known to vary with the intensity of exercise (Astrand et al, 1964; Damato et al, 1966; Williams et al, 1962).

The relationship between the $H R$ and OXC (HROXCR) has been shown to vary with physical training (Saltin et al, 1968; Pannier et al, 1980), age (Sheffield, et al, 1978; Montoye et al, 1968; Dill \& Consolzio, 1962), body position (Andrews, 1971; Vokzc et al, 1975; Sato \& Tanaka, 1973), and environmental temperature (Dill \& Consolazio, 1962; Rowell et al, 1966; Taylor et al, 1963; Myhre et al, 1979). While it is relatively easy to control for sex, age, and physical training, in most applications, it is difficult to control for body position and environmental temperature. However, in most experiments, it may be safe to assume that body position and environmental temperature have an equal effect upon control and experimental groups.

The effects of diet, previous exercise or stress on HROXCR are not well studied (21). However, stress has been shown to increase the HR at constant OXC (Taylor et al, 1963; Sonne \& Galbo, 1980) and previous physical activity does affect the regression equation (Lundgren, 1946), whereas conflicting results are reported for dietary effects (Lundgren, 1946; Schutz et al, 1981). Since the average subject will probably spend more time recovering from physical activity than he/she will spend exercising, it is also important to know if the HROXCR obtained during exercise is followed during recovery.

The objectives of the present study were (1) to determine the effects of dietary composition upon the regression equation between $H R$ and OXC, (2) to determine if the HROXCR obtained during an incremental exercise test also was valid during recovery, and (3) to determine what effect previous exercise might have upon the HROXCR.

## Methods

The study was divided into two parts. The first part examined the relationship between HR and OXC during recovery following 20 min of exercise at $80 \%$ of maximal OXC. In the second part the effects of duration( $D$ ) and intensity ( $S$ ) of the exercise period upon the relationship between the HR and OXC during recovery was studied.

## Subjects

Two groups of five healthy male college students volunteered for the study. The physical characteristics of the subjects are given in Table 1. None of the subjects took any medication; and on the day of the experiment, they were requested not to drink any caffeine-containing beverages. They also did not participate in any strenuous physical activity on the day before the experiments were conducted.

## Informed Consent

Each subject signed an informed consent form after the details of the experiments were described to him. The form stated the experimental procedures, identified possible risks, and noted that a subject could terminate his participation at any time.

## Diets

The composition of the diets and meals is given in Table 3. Each meal contained 700 kcal . The fat meal consisted of 288 ml whipping cream, which the subjects drank in less than 10 minutes. The protein

Table 1. Physical characteristics of the two groups of five adult male subjects. (SD = Standard Deviation.)

| Physical | Group I ( $n=5$ ) |  | Group II ( $\mathrm{n}=4$ ) |  |
| :---: | :---: | :---: | :---: | :---: |
| Characteristics | Mean | SD | Mean | SD |
| Ages (years) | 24.8 | 3.1 | 28.8 | 2.2 |
| Body Weight (kg) | 74.5 | 11.9 | 81.9 | 7.0 |
|  | 173 | 7.4 | 184 | 16.5 |
| $\mathrm{VO}_{2}-\max (\mathrm{ml} / \mathrm{min} / \mathrm{kg})^{\text {a }}$ | 54.1 | 6.6 | 49.9 | 2.6 |
| Mean running speed ${ }^{\text {b }}$ | 10.9 | 0.8 |  |  |
| $\%_{20}$-max (mean) ${ }^{\text {b }}$ | 78 | 0.6 |  |  |

$$
\text { a Maximal oxC } \quad b_{\text {final treadmill setting }}
$$

Table 2. Relative intensity of exercise for various treadmill settings used in incremental exercise tests.

| Setting | Speed <br> $(\mathrm{km} / \mathrm{hr})$ | $\% \mathrm{vo}_{2}$-maxa | Duration (D) of Exercise |
| :--- | :--- | :---: | :---: |
| $(\mathrm{min})$ |  |  |  |

${ }^{\text {a Maximal OXC: mean of four subjects. }}$
bThe time given is for an intermediate step. When 4-7 was the final speed D was either 10 or 30 minutes.
Table 3. Composition of meals and diets. The energy content of the diets varied between 2,100 and 2,400

| Diets or Meals | Method | $\begin{gathered} \text { Total } \\ \text { Energy (Kcal) } \end{gathered}$ | \% of total energy |  |  | \% by weight |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  |  |  | Fat | Protein | Carbohydrate | Water | Ash |
| Fat Meal | Food Tables ${ }^{\text {a }}$ | 700 | 92 | 3 | 5 |  |  |
| Protein Meal | Food Tables | 700 | 22 | 76 | 2 |  |  |
| Carbohydrate Meal | Food Tables | 700 | 2 | 5 | 93 |  |  |
| Fat Diet | Food Tables | 2,150 | 65 | 15 | 20 |  |  |
| Carbohydrate Diet | Food Tables | 2,150 | 20 | 15 | 65 |  |  |
| Fat Diet | Chemical Analysis | 2,150 | 66 | 16 | 19 | 68.0 | 4.0 |
| Carbohydrate Diet | Chemical Analysis | 2,150 | 22 | 15 | 63 | 72.8 | 3.4 |

[^0]meal consisted of 94 g creamed cottage cheese and 545 g (raw weight) skinned chicken breast, with all visible fat removed. The subjects ate the protein meal in 30 minutes. The carbohydrate meal consisted of 824 g ripe bananas (peeled), which were eaten in less than 10 minutes. The subjects drank 340 cc of ginger ale ( 1 kcal ) with the meals (Vernors, Detroit, MI), and were allowed salt, pepper, and water ad lib.

During the experimental periods the subjects ate 2,100 to 2,400 kcal per day either as a high fat or as a high carbohydrate diet. The composition is given in Table 3. The subjects were fed one of the two experimental diets in random order for three days prior to the exercise test, which was performed in the postabsorptive state the fourth day. The diet and test meals were prepared and eaten in a kitchen adjacent to the exercise laboratory. Two menus were used for each diet: one for breakfast and one for dinner and supper. The menus were repeated for all three days. The subjects were supervised during the meals and ate all the allotted food. Duplicate portions were used for chemical analysis. Fat was analyzed by ether extraction, protein by microkjeldahl, minerals as ash $\left(600^{\circ} \mathrm{C}\right)$ and carbohydrate by difference.

For both diets regular food items were used. Sources of fat were: margarine, mayonnaise, cheese, eggs, bacon and olives; while sources of carbohydrate were: bananas, bread, potatoes, sweet potatoes, beans, carrots and peas. These foods contain some protein. Additional Protein came from soy protein. For the fat diet imitation chicken, and for the carbohydrate diet imitation beef (Worthington Foods, Worthington, OH ) were used.

## Measurements

Heart rate (HR) was obtained from an electrocardiogram (lead 2 with the positive lead in V5 position). The EKG signal was converted to heart rate through a cardiotachometer (built in our lab) and continuously recorded by a calibrated Sargent Recorder, Model DTM-115-4 (Sargent and Company, Chicago, IL). The mean HR in beats/minute corresponding to each collection of expired air was calculated from the recording.

The oxygen consumption (OXC) was determined by a modified Douglas method (Consolazio, et al, 1963). The expired air was collected through a low resistance valve (Otis-McKerrow, Warren Collins, Inc., Braintree, MA) in light-weight neoprene bags. The composition ( $\mathrm{O}_{2}$ and $\mathrm{CO}_{2}$ contents) of the collected air was immediately determined using the Beckman LB-2 carbon dioxide and Beckman OM-11 oxygen analyzers, respectively (Beckman Instruments, Schiller Park, IL). The air volume was determined by metering through (using a constant flow of $501 / m i n$ ) a Singer dry gas meter (American Meter Company, Philadelphia, PA).

The mean OXC for the control experiment was compared to the baseline (mean resting OXC prior to the ingestion of the meal). To correct for the day to day variation in the resting OXC in each subject the difference between the control and baseline values were added or subtracted from the OXC for that day's treatment.

## Exercise Tests

The maximal OXC was determined in a continuous incremental test with the subjects exercising on the treadmill until exhaustion. The speed was increased stewpise at $8.8,10.5,12.5$ and $14.3 \mathrm{~km} / \mathrm{hour}$. When
the subjects reached the highest speed they could comfortably run ( 12.5 or $14.3 \mathrm{~km} / \mathrm{hr}$ ) further increases in the workload were accomplished by increasing the treadmill inclination in steps of $2 \%$. The subjects ran 3 minutes at each step, the HR was recorded continuously, three one-minute bags of expired air were collected at each step, and the oxygen consumption calculated for each minute.

The OXC following the different treatments was determined through an incremental exercise test with the final workload corresponding to $80 \%$ of the subjects' maximal OXC. Three collections of expired air were made at each step and the mean HR corresponding to each air collection was recorded. The test started at rest with the subject seated in an armchair, while 3 consecutive 5 -minute collections of expired air were made. During the following steps the subjects would walk or run on the treadmill. At the $4.8 \mathrm{~km} / \mathrm{hr}$ and $6.8 \mathrm{~km} / \mathrm{hr}$ levels the subjects exercised for 6 minutes each, and at the other levels they exercised for 3 minutes each; except when they reached the final speed (the one corresponding closest to $80 \%$ of their maximal $0 \times C$ ), they continued the exercise for 20 minutes. During the final stage, one-minute bags of expired air were collected, every second minute starting with the second minute.

Following the exercise, the $H R$ and OXC were determined during 40 minutes of recovery. Two one-minute bags, one three-minute and one five-minute bag of expired air were collected during the first 10 minutes of recovery. During the next 30 minutes, 3 five-minute bags of expired air were collected every second 5 -minute period. During the alternate 5 -minute periods, when no collections were made, the subject
remained seated in the armchair, but was relieved of the face mask with air-collection valve and the noseclip. Arterialized blood samples were collected from one of the fingers at $5,15,25$ and 40 minutes of recovery for determination of the blood lactate concentration.

In the second group (group II) of subjects the final setting (S) of the treadmill was varied between 4 and 7 in order to investigate the effect of exercise intensity and the duration (D) of each speed was either 10 or 30 min . The various treatment combinations were administered to each subject in random order. The speeds, duration and mean \% of maximal OXC are given in Table 2. The collection of expired air during recovery was slightly modified in this group. Instead of collecting 2 one min bags and one 3 min during the first 5 min of recovery, one 1 min bag and 2 two min bags were collected.

## Experimental Protocol

In the second part of the experiment four subjects (group II) participated. The final setting of the treadmill was varied between 4 and 7. .The corresponding speeds and \% maximal OXC (mean) are given in Table 2. The duration at this setting was either 10 or 30 min . From these data the effect ò speed and intensity on HR and OXC during recovery was evaluated. Not all subjects completed the higher exercise intensities; but statistical analysis is based only on the settings completed by all subjects.

## Exercise

The exercise experiments were carried out over a 10 -week period with each test given one week apart. During the first and last tests the maximal OXC were determined. The second test was always a
control (postabsorptive state) prior to which the subjects ate their normal diet. Prior to the third test, the subjects consumed one of the experimental diets for three days (two started with the high fat diet and three with the high carbohydrate diet). Then followed another control test before the opposite experimental diet was introduced, three days prior to the fifth test. Following both dietary treatments, the test was carried out in the postabsorptive state. The three meals and a final control were introduced in random order so that all the subjects would not consume the meals in the same order. The exercise tests began 30 minutes after termination of the carbohydrate meals and 2-3 hours following the fat and protein meals, which we had found to correspond to the maximal thermogenic response.

## Statistical Analysis

The incremental recovery $H R($ IRHR), recovery OXC (ROXC) and the difference between the predicted OXC (POXC) based on the regression equation obtained during the preceding exercise in each subject (DOXC) were analyzed by a split block (block = subjects) repeated measurements design (each subject serves as his own control).

In the first part of the experiment the HROXCR during recovery was compared to the linear regression line during exercise by calculating the $95 \%$ confidence limits. In the second part of the experiment the treatment means for ROXC, IRHR and DOXC were compared by designed contrasts (Bonferroni t statistics; Miller, 1966).

The regression lines between HR and OXC during exercise were calculated for the 5 subjects in part I of the study. A common regression line was calculated for the 3 control experiments. Dietary
effects on the HROXCR were evaluated by testing for differences in slopes and $Y$-intercepts between the regression lines; but because the regression equation can vary with time random significant results can be expected in some cases. Incremental HR as used by Andrews (1971) is the actual $H R-$ resting $H R$ in the individual subjects. By using incremental HR the $Y$-intercept of the regression line corresponds to the resting $0 \times C$; and it therefore does not have much meaning to test for differences in the $Y$-intercept. A "corrected" HR is therefore used in this study instead. The corrected $H R$ is obtained by adding 60 -resting $H R$ to the measured $H R$.

## Results

Effect of diet on the HROXCR during exercise
There was no evidence that any of the dietary treatments had an effect upon the linear regression between HR and OXC during exercise. The calculated slopes and $y$ intercepts are given in Table 4 for the 5 dietary treatments and control. The control line was calculated from HR and OXC for 3 separate experiments in order to compensate for possible variation in the regression equation with time. All the treatments are compared to the control line for each subject separately. In 5 cases, the regression line for a treatment differed from that of control, but in no case did it occur twice in the same subject. It is therefore not a treatment effect, but a change occurrence due to the variation of the regression line.

## Effect of previous exercise on HROXCR

Exercise changes both the slope and $y$-intercept of the regression equation between HR and OXC. Two of the subjects in part I were exercised as usual at $80 \%$ of their maximal OXC, and after one hour rest the same exercise protocol was repeated. One hour after the first exercise test, the OXC had returned to preexercise levels, but the HR was still elevated ( 20 beats $/ \mathrm{min}$ ). Table 5 gives the values for the slopes $\left(B_{1}\right), y$-intercepts ( $B_{0}$ ) and standard error of estimate (SE) for the two regression lines. The calculated t-statistics for the test of significance is also given. Both the slope and $y$-intercept are

Table 4. Test of significant difference between the regression equations between HR and OXC for the five treatments and the regression equation for control. Critical $T=1.99$.

| Subject | Treatment | Control ${ }^{\text {a }}$ | Diets ${ }^{\text {b }}$ |  | Meals ${ }^{\text {b }}$ |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  |  |  | Fat D | CHO D | Fat M | Pro M | CHO M |
| 1 | Slopes | 0.372 | 0.337 | 0.347 | 0.385 | 0.372 | 0.371 |
|  | Y-Intercept | -20.6 | -23.4 | -17.2 | -23.2 | -22.0 | -21.3 |
|  | S.E.C | 1.59 | 2.71 | 1.34 | 1.25 | 0.84 | 1.49 |
|  |  |  | 0.36 | 2.21* | 1.02 | 0.01 | 0.07 |
|  | $\mathrm{T}_{0}^{1 d}$ |  | 1.41 | 2.38* | 1.59 | 0.90 | 0.43 |
| 2 | Slopes | 0.364 | 0.365 | 0.374 | 0.367 | 0.369 | 0.405 |
|  | Y-Intercept | -17.1 | -17.4 | -18.7 | -13.8 | -15.6 | -22.9 |
|  | S.E. | 2.03 | 0.74 | 1.13 | 1.10 | 1.07 | 0.91 |
|  | $\mathrm{T}_{1}$ |  | 0.07 | 0.73 | 0.52 | 0.35 | 2.72* |
|  | $T_{0}^{1}$ |  | 0.21 | 0.92 | 1.98 | 0.86 | 3.28* |
| 3 | Slopes | 0.474 | 0.461 | 0.473 | 0.436 | 0.495 | 0.498 |
|  | Y-Intercept | -26.1 | -23.5 | -23.5 | -22.6 | -31.6 | -29.1 |
|  | S.E. | 4.03 | 2.86 | 3.11 | 2.72 | 3.10 | 2.65 |
|  | $\mathrm{T}_{1}$ |  | 0.44 | 0.05 | 1.23 | 0.59 | 0.68 |
|  | $\mathrm{T}_{0}$ |  | 1.37 | 1.32 | 1.83 | 2.34* | 1.43 |
| 4 | Slopes | 0.366 | 0.331 | 0.372 | 0.386 | 0.345 | 0.387 |
|  | Y-Intercept | -16.6 | -12.8 | -18.1 | -18.7 | -15.3 | -18.6 |
|  | S.E. | 1.99 | 1.30 | 1.38 | 1.55 | 1.57 | 1.46 |
|  | T1 |  | 2.80* | 0.42 | 1.49 | 1.67 | 1.64 |
|  | $\mathrm{T}_{0}^{1}$ |  | 2.53* | 0.98 | 1.32 | 0.81 | 1.27 |
| 5 | Slopes | 0.321 | 0.307 | 0.305 | 0.330 | 0.290 | 0.348 |
|  | $Y$-Intercept | -12.6 | -12.0 | -12.5 | -14.2 | -11.0 | -15.7 |
|  | S.E. | 1.74 | 0.95 | 0.96 | 1.44 | 1.13 | 2.13 |
|  | $\mathrm{T}_{1}$ |  | 1.34 | 1.53 | 0.79 | 2.92* | 1.98 |
|  | $\mathrm{T}_{0}$ |  | 0.49 | 0.05 | 1.10 | 1.20 | 1.90 |

$a_{n}=54 \quad b_{n}=18$
${ }^{\text {C Standard }}$ error of means in ml oxygen $/ \mathrm{kg} / \mathrm{min}$.
${ }^{d} T_{1}$ and $T_{0}$ are the calculated $t$-values for test of difference between the particular regression line and the control line.
*Indicates significance ( $\alpha=0.05$ ).

Table 5. Test of significance between the regression equations for two subjects exercised twice, one hour apart. Results are included with the regression lines corrected for the resting heart rate (cor).

| Subject | Trial | Slope | Y-Intercept | $S E^{\text {a }}$ | $T_{1}{ }^{\text {b }}$ | $T_{0}{ }^{\text {b }}$ |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| 1 | 1 | 0.293 | -13.4 | 1.27 | 4.34 | 6.21 |
|  | 2 | 0.347 |  | 131 |  |  |
|  | 2 | 0.347 | -23.6 | 1.31 |  |  |
|  | 1 cor | 0.293 | -13.4 | 1.27 |  | 2.92 |
|  | 2 cor | 0.348 | -18.2 | 1.31 |  |  |
| 2 | 1 | 0.359 | -16.1 | 1.39 | 3.17 | 4.88 |
|  | 2 | 0.404 | -25.5 | 2.14 |  |  |
|  | 1 cor | 0.358 | -18.1 | 1.39 |  | 1.19 |
|  | 2 cor | 0.405 | -20.7 | 2.14 |  |  |

${ }^{a_{S E}}=$ standard error of estimate.
$\mathrm{b}_{1}$ and $\mathrm{T}_{0}$ are the calculated t -statistics for slope and Y -Intercept, respectively. The critical t-value for both $T_{1}$ and $T_{0}$ are 2.04 for subject 1 and 2.02 for subject 2. ( $n=18$ and 22 respectively.)
significantly different between the two regression equations in each subject. Using corrected HR decreases of the $y$-intercepts, but in the one subject the difference is still significant. The regression lines are shown in Figure 1.

## Effect of recovery on HROXCR

The linear regression equation between HR and OXC obtained during exercise is not followed during the 40 min of recovery. The ROXC is lower than the lower confidence limit (LCL) during the entire 40 min recovery period; and the predicted OXC (POXC) can be as much as $300 \%$ above the recovery OXC (ROXC). These results are based on 8 trials in each of 5 subjects, and are shown in Figure 2 and Table 6. After 40 min of recovery the RHR was still elevated 20 beats/min, and is only decreasing 1 beat/10 minutes. At the end of recovery the OXC has returned to the resting level (Figure 4). Two subjects were followed to complete recovery of the HR, which took at least 3 hours; and appeared to depend on the training of the subject. The RHR is shown in Figure 3 and POXC, LCL and ROXC in Figure 4. Since POXC and LCL are both derived from the RHR and the linear regression equation between HR and OXC obtained during exercise, it is not surprising that RHR, POXC and LCL have a very similar time course, although the values for LCL of course are somewhat lower. As seen from Figure 4 and Table 6, the ROXC falls much faster toward the resting preexercise OXC (the x-axis in Figure 4). The difference between POXC and ROXC (DOXC) quickly reaches a maximum at about 3 min of recovery and then decreases very slowly (Figure 5). Expressed as percentage of POXC, DOXC, however, reaches a maximum of $62 \%$ at 3 min , and then remains constant for the

Figure 1. Two regression lines based on exercise trials one hour apart in one subject. The second line begins at a higher HR. Both slopes and y-intercepts are significantly different. The regression lines for the first and second test is marked 1 and 2 respectively.


FIGURE 1

Figure 2. Predicted OXC (POXC), lower confidence limits (LCL) and recovery OXC (ROXC) as a function of recovery HR. Mean of five subjects with eight exercise tests in each.

(6у/и!ш/ィІш) $9 \times 0$

FIGURE 2

Table 6. Comparison of mean recovery values and their respective standard deviation (SD) for eight trials in each of five subjects.

| Time ${ }^{\text {a }}$ (min) | 0.5 | 1.5 | 3.5 | 7.5 | 17.5 | 27.5 | 37.5 |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| RHR ${ }^{\text {b }}$ | 142 | 111 | 101 | 92 | 84 | 81 | 80 |
| SD | 12.4 | 7.8 | 7.0 | 8.9 | 5.6 | 4.6 | 4.4 |
| ROXC ${ }^{\text {C }}$ | 26.4 | 11.3 | 7.4 | 5.7 | 5.1 | 4.6 | 4.4 |
| SD | 2.5 | 0.85 | 1.4 | 0.54 | 0.59 | 0.57 | 0.59 |
| LCL ${ }^{\text {d }}$ | 31.0 | 19.7 | 15.7 | 12.1 | 9.4 | 8.2 | 7.7 |
| SD | 3.3 | 2.1 | 2.0 | 2.4 | 2.2 | 2.1 | 2.2 |
| POXC ${ }^{\text {e }}$ | 34.6 | 23.2 | 19.3 | 15.9 | 13.3 | 12.0 | 11.3 |
| SD | 3.0 | 2.0 | 2.2 | 1.8 | 1.2 | 0.7 | 0.8 |
| DLCL ${ }^{\text {f }}$ | 4.6 | 8.4 | 8.3 | 6.4 | 4.3 | 3.6 | 3.3 |
| DOXC9 ${ }^{9}$ | 8.2 | 12.1 | 11.9 | 10.2 | 8.2 | 7.4 | 6.9 |
| \%DOXCh | 24 | 52 | 62 | 64 | 62 | 62 | 61 |

[^1]Figure 3. HR during 40-minute recovery. Mean of five subjects with eight exercise tests in each. The $x$-axis intersects the $y$-axis at the preexercise HR.
(u!u/sieeq) yH

FIGURE 3

Figure 4. Predicted OXC (POXC), upper curve. Lower 95\% confidence level for OXC (LCL), middle curve. Recovery OXC (ROXC), lower curve, as a function of time. The x-axis represents resting value. Mean of five subjects with eight exercise tests in each.


FIGURE 4

Figure 5. Difference between predicted OXC and recovery OXC (DOXC) as a function of time during 40 minute recovery. Mean of eight trials in each of five subjects.


FIGURE 5
remainder of the 40 min recovery (Figure 6). This means that during the first 3 minutes of recovery, the $H R$ progressively lags behind the ROXC, but thereafter, returns toward the resting levels at the same speed until ROXC reaches the resting level or slightly below. The RHR does not return to resting level until three or more hours later.

The effect of exercise intensity (S) and duration (D) on this lag in RHR was investigated in four subjects. Because not all subjects completed all the S-levels, only the first five treatments are included in the statistical analysis, while the data are reported for any treatment completed by at least one subject. In order to reduce intrasubject variability, the IHR(HR-RHR) is used. The means for IHR are shown in Table 7, the mean ROXC in Table 8 mean POXC in Table 9, and mean DOXC in Table 10. IHR is plotted in Figure 7 for 2 exercise intensities and in Figure 8 for 10 and 30 min of exercise, while Figure 9 shows ROXC at 2 exercise intensities and Figure 10, DOXC as a function of time. LCL, ROXC and DOXC are shown for all the treatments in Table 11. Except for speed 4 at 10 minutes, ROXC is always less than LCL and the relationship is similar to that already discussed for recovery after exercise at constant speed (Figure 2). The effect of duration of exercise appears stronger than the effect of intensity of exercise on DOXC. Thus, the mean (over the 40 min recovery) DOXC for 30 minutes is $10 \mathrm{ml} / \mathrm{min} / \mathrm{kg}$ and for 10 minutes is $6.6 \mathrm{ml} / \mathrm{min} / \mathrm{kg}$.

The results of the analysis of variance show that both treatment and time have a highly significant effect upon IRHR, ROXC, and DPOXC.

Figure 6. DOXC as \% of POXC vs recovery time (40-minute recovery). Mean of eight trials in five subjects.


FIGURE 6

Table 7. Mean recovery incremental ${ }^{\text {a }}$ heart rates after exercise on a treadmill at 4 different speeds, each speed maintained for 10 and 30 minutes, respectively. Mean of four subjects.

| Setting | $\begin{aligned} & \text { Time } \\ & (\min ) \end{aligned}$ | Recovery time ${ }^{\text {c }}$ |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  |  | 0.5 | 3.0 | 7.5 | 17.5 | 27.5 | 37.4 |
| 4 | 10 | 57 | 25 | 17 | 14 | 11 | 10 |
| 4 | 30 | 73 | 38 | 28 | 24 | 20 | 14 |
| 5 | 10 | 71 | 32 | 26 | 22 | 18 | 16 |
| 5 | 30 | 84 | 47 | 36 | 31 | 27 | 24 |
| 6 | 10 | 90 | 45 | 32 | 30 | 26 | 22 |
| 6d | 30 | 100 | 56 | 40 | 32 | 29 | 21 |
| $7{ }^{\text {d }}$ | 10 | 103 | 51 | 39 | 34 | 31 | 22 |
| 7 e | 30 | 112 | 63 | 54 | 44 | 41 | 37 |

${ }^{\text {a }}$ Incremental $=$ measured $H R-$ resting $H R$ in each subject.
bTime refers to duration of exercise at the final setting.
CRecovery time refers to the midpoint for collection of expired air.
dThree subjects only.
eone subject, only.
Standard errors of individual treatment means at one time are irrelevant for comparisons or reliability.

Standard error for difference between 2 treatments at one time is 4.74 (beats/min).

Standard error of difference between 2 times for one treatment is 5.58 (beats/min).

Table 8. Mean recovery oxygen consumption in four subjects after exercise on a treadmill at four different speeds, two runs at each speed for 10 and 30 minutes, respectively.

| Setting | Recovery time (min) ${ }^{\text {b }}$ |  |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | $\begin{aligned} & \text { Time }{ }^{\mathrm{a}} \\ & (\min ) \end{aligned}$ | 0.5 | 3.0 | 7.5 | 17.5 | 27.5 | 37.5 |
| 4 | 10 | 19.6 | 6.1 | 4.2 | 4.0 | 3.9 | 3.8 |
| 4 | 30 | 19.0 | 6.2 | 4.6 | 4.7 | 4.0 | 4.0 |
| 5 | 10 | 22.9 | 6.9 | 4.8 | 4.5 | 4.2 | 4.3 |
| 5 | 30 | 24.3 | 7.4 | 5.0 | 4.3 | 4.3 | 3.8 |
| 6 | 10 | 26.9 | 8.3 | 5.4 | 4.7 | 4.1 | 3.8 |
| $6^{C}$ | 30 | 29.3 | 10.2 | 6.5 | 5.5 | 5.2 | 4.9 |
| 7 C | 10 | 30.9 | 8.9 | 6.6 | 4.9 | 4.7 | 4.3 |
| 7 d | 30 | 31.2 | 8.9 | 5.8 | 5.2 | 4.4 | 3.5 |
| a Duration $D$ of final treadmill setting. |  |  |  |  |  |  |  |
| brefers to midpoint of collection of expired air. |  |  |  |  |  |  |  |
| ${ }^{\text {C }}$ Three subjects only. |  |  |  |  |  |  |  |
| ${ }^{\text {d One subject only }}$. |  |  |  |  |  |  |  |
| Standard errors of individual treatment means at one time are irrelevant for comparisons or reliability. |  |  |  |  |  |  |  |
| Standard error for difference between 2 treatments at one time is 0.698 ( $\mathrm{ml} / \mathrm{min} / \mathrm{kg}$ ). |  |  |  |  |  |  |  |
| Standard error for difference between 2 times for one treatment is 2.13 ( $\mathrm{ml} / \mathrm{min} / \mathrm{kg}$ ). |  |  |  |  |  |  |  |
| Resting $0 \times C=3.6 \mathrm{ml} / \mathrm{min} / \mathrm{kg}$. |  |  |  |  |  |  |  |

Table 9. Mean predicted oxygen consumption during recovery following exercise on a treadmill at four different speeds in four subjects. Each speed is repeated twice for 10 and 30 minutes, respectively. Time is given in minutes. Standard deviation (SD) is also given.

| Setting |  | $\begin{aligned} & \text { Time }^{\mathrm{a}} \\ & (\min ) \end{aligned}$ | Recovery time (min) ${ }^{\text {b }}$ |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  |  |  | 0.5 | 3.0 | 7.5 | 17.5 | 27.5 | 37.5 |
|  |  |  | POXC in ml/min/kg. |  |  |  |  |  |
| 4 |  | 10 | 23.9 | 13.1 | 9.7 | 9.0 | 7.5 | 7.1 |
|  | SD |  | 5.5 | 4.4 | 3.5 | 3.7 | 2.6 | 2.0 |
| 4 |  | 30 | 27.3 | 17.2 | 12.9 | 11.4 | 9.9 | 8.3 |
|  | SD |  | 5.1 | 3.8 | 3.2 | 3.5 | 2.1 | 2.4 |
| 5 |  | 10 | 29.8 | 15.6 | 12.2 | 10.9 | 9.3 | 8.6 |
|  | SD |  | 6.8 | 3.8 | 3.7 | 3.2 | 1.8 | 2.1 |
| 5 |  | 30 | 32.6 | 19.5 | 15.6 | 14.0 | 12.5 | 11.5 |
|  | SD |  | 4.3 | 4.7 | 3.5 | 4.1 | 4.4 | 4.1 |
| 6 |  | 10 | 33.4 | 19.1 | 14.0 | 13.2 | 12.0 | 10.7 |
|  | SD |  | 5.8 | 3.6 | 2.5 | 3.4 | 3.1 | 1.9 |
| $6^{C}$ |  | 30 | 38.8 | 23.4 | 18.0 | 15.2 | 14.2 | 11.5 |
|  | SD |  | 4.2 | 3.4 | 3.5 | 2.2 | 2.4 | 3.2 |
| $7^{C}$ |  | 10 | 38.1 | 20.4 | 17.7 | 15.0 | 13.6 | 10.7 |
|  | SD |  | 2.2 | 2.4 | 2.6 | 2.2 | 2.2 | 2.3 |
| 7 d |  | 30 | 40.0 | 23.8 | 20.9 | 17.6 | 16.6 | 15.3 |

${ }^{\text {a }}$ Duration ( $D$ ) of final exercise step.
$\mathrm{b}_{\text {Refers }}$ to midpoint for collection of expired air.
CThree subjects only. done subject only.

Table 10. Mean difference (4 subjects) between the predicted oxygen consumption and the actual oxygen consumption during recovery after running on a treadmill at four different speeds. The subjects ran at each speed twice for 10 and 30 minutes, respectively.

| Setting | $\begin{aligned} & \text { Time }^{\mathrm{a}} \\ & (\mathrm{~min}) \end{aligned}$ | Recovery time (min) ${ }^{\text {b }}$ |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  |  | 0.5 | 3.0 | 7.5 | 17.5 | 27.5 | 37.5 |
| 4 | 10 | 4.3 | 6.7 | 5.5 | 4.6 | 3.6 | 3.3 |
| 4 | 30 | 8.8 | 11.4 | 9.4 | 7.3 | 6.9 | 5.4 |
| 5 | 10 | 6.9 | 8.5 | 7.5 | 6.3 | 5.1 | 4.4 |
| 5 | 30 | 8.4 | 11.9 | 10.6 | 9.7 | 8.2 | 7.6 |
| 6 | 10 | 6.1 | 9.7 | 8.7 | 8.5 | 7.1 | 6.8 |
| $6^{\text {c }}$ | 30 | 9.5 | 13.3 | 11.6 | 9.8 | 9.0 | 6.5 |
| $7{ }^{\text {c }}$ | 10 | 8.2 | 11.8 | 10.1 | 9.1 | 9.4 | 6.6 |
| $7{ }^{\text {d }}$ | 30 | 8.8 | 15.0 | 15.1 | 12.4 | 12.2 | 11.8 |
| Mean |  | 7.6 | 11.1 | 9.8 | 8.4 | 7.8 | 6.6 |
| $\begin{aligned} & \mathrm{D} 10^{e} \\ & \mathrm{D} 30^{f} \end{aligned}$ |  | 6.4 8.9 | 9.2 12.9 | 8.0 11.7 | 7.1 9.8 | 6.3 9.1 | 5.3 7.8 |

aduration (D) of final exercise step.
brefers to midpoint for collection of expired air.
CThree subjects only. done subject only.
emean for 10 min . fmean for 30 min .
DOXC in $\mathrm{ml} / \mathrm{min} / \mathrm{kg}$.
Standard errors of individual treatment means at one time are irrelevant for comparisons or reliability.

Standard error for difference between 2 treatments at one time is $1.8(\mathrm{ml} / \mathrm{min} / \mathrm{kg})$.

Standard error for difference between 2 times for one treatment is 0.97 ( $\mathrm{ml} / \mathrm{min} / \mathrm{kg}$ ).

Figure 7. Mean incremental HR (IHR) for four subjects during 40-minute recovery after exercise for 10 minutes at speed $4(8.8 \mathrm{~km} / \mathrm{hr})$, lower curve; and speed 6 ( $12.5 \mathrm{~km} / \mathrm{hr}$ ), upper curve. Significant difference at $0.5,3.0,7.5,17.5$, and 27.5 minutes. Mean values for four subjects. Standard error for difference of 2 treatments at one time is 4.74 (beats/ min). Standard error for difference of 2 times for one treatment is 5.58 (beats $/ \mathrm{min}$ ).


FIGURE 7

Figure 8. Incremental HR during 40-minute recovery (RIHR) after exercise at speed $5(10.5 \mathrm{~km} / \mathrm{hr})$. Upper curve for $D=30$ minutes, and lower curve for $D=10$ minutes.
(u!u/speeq) yHI

FIGURE 8

Figure 9. Recovery OXC (ROXC) as a function of time. As there is no difference in ROXC due to the duration of exercise, 10- and 30-minute periods are combined. Upper curve is for speed $4(8.8 \mathrm{~km} / \mathrm{hr})$ and lower curve for speed 6 ( $12.5 \mathrm{~km} / \mathrm{hr}$ ). Significant differences for the first minute of recovery only. Mean values for four subjects. Standard error for difference of two treatments at one time is 0.698 ( $\mathrm{ml} / \mathrm{min} / \mathrm{kg}$ ). Standard error for difference of two times at one treatment is $2.13(\mathrm{ml} / \mathrm{min} / \mathrm{kg})$.


FIGURE 9

Figure 10. Mean DOXC (difference between predicted and recovery OXC) for four subjects. Significant differences at 3.0 and 7.5 minutes. Since there was no significant speed effects, the mean for 30 minutes for Speed 4 - Speed 7 (upper curve) is compared to the mean for 10 minutes of exercise for Speed 4 - Speed 7 (middle curve). The lower curve is for the lowest speed ( $8.8 \mathrm{~km} / \mathrm{hr}$ ) which is significantly different from $S 6$ when all time points are considered.


FIGURE 10

Table 11. Mean values of $95 \%$ lower confidence limit (LCL) for predicted oxygen consumption (POXC), recovery oxygen consumption (ROXC), and the difference (DLCL) between LCL and ROXC in ml/kg/min following exercise on a treadmill at four different speeds (S) for 10 and 30 minutes (D), respectively. Mean of four subjects.

|  | $s^{\text {a }}$ | D ${ }^{\text {b }}$ | Recovery time $(\min )^{\text {c }}$ |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  |  |  | 0.5 | 3.0 | 7.5 | 17.5 | 27.5 | 37.5 |
| LCL | 4 | 10 | 20.5 | 9.6 | 6.2 | 4.9 | 3.8 | 3.4 |
| ROXC |  |  | 19.6 | 6.1 | 4.2 | 4.0 | 3.9 | 3.8 |
| DLCL |  |  | 0.9 | 3.5 | 2.0 | 0.9 | -0.1 | -0.4 |
| LCL | 4 | 30 | 25.2 | 15.2 | 10.8 | 8.8 | 7.8 | 6.2 |
| ROXC |  |  | 19.0 | 6.2 | 4.6 | 4.7 | 4.0 | 4.0 |
| DLCL |  |  | 6.2 | 9.0 | 6.2 | 4.1 | 3.8 | 2.2 |
| LCL | 5 | 10 | 27.4 | 13.2 | 9.8 | 8.5 | 6.9 | 6.2 |
| ROXC |  |  | 22.9 | 6.9 | 4.8 | 4.5 | 4.2 | 4.3 |
| DLCL |  |  | 4.5 | 6.3 | 5.0 | 4.0 | 2.7 | 1.9 |
| LCL | 5 | 30 | 29.8 | 16.6 | 12.8 | 11.2 | 9.6 | 8.6 |
| ROXC |  |  | 24.3 | 7.4 | 5.0 | 4.3 | 4.3 | 3.8 |
| DLCL |  |  | 5.5 | 9.2 | 7.8 | 6.9 | 5.3 | 4.8 |
| LCL | 6 | 10 | 30.6 | 16.5 | 11.7 | 10.4 | 9.0 | 8.3 |
| ROXC |  |  | 26.9 | 8.3 | 5.4 | 4.7 | 4.1 | 3.8 |
| DLCL |  |  | 3.7 | 8.2 | 6.3 | 5.7 | 4.9 | 4.5 |
| LCL | $6^{\text {d }}$ | 30 | 35.7 | 20.5 | 15.2 | 12.3 | 11.3 | 8.5 |
| ROXC |  |  | 29.3 | 10.2 | 6.5 | 5.5 | 5.2 | 4.9 |
| DLCL |  |  | 6.4 | 10.3 | 8.7 | 6.8 | 6.1 | 3.6 |
| LCL | $7{ }^{\text {d }}$ | 10 | 34.8 | 17.2 | 13.3 | 11.5 | 10.4 | 7.5 |
| ROXC |  |  | 30.9 | 8.9 | 6.6 | 4.9 | 4.7 | 4.3 |
| DLCL |  |  | 3.9 | 8.3 | 6.7 | 6.6 | 5.7 | 3.2 |
| LCL | $7{ }^{\text {e }}$ | 30 | 36.3 | 20.4 | 17.4 | 14.1 | 13.1 | 11.8 |
| ROXC |  |  | 31.2 | 8.9 | 5.8 | 5.2 | 4.4 | 3.5 |
| DLCL |  |  | 5.1 | 11.5 | 9.6 | 8.9 | 8.7 | 8.3 |
| ${ }^{\text {a }}$ Treadmill setting. |  |  |  | b Duration of final setting. |  |  |  |  |
| ${ }^{\text {Refers }}$ to midpoint for collection of expired air. |  |  |  |  |  |  |  |  |
| ${ }^{\text {d }}$ Three subjects only. ${ }^{\text {One subject only }}$ |  |  |  |  |  |  |  |  |

Whereas the time response for IRHR and ROXC depends on the treatment, this is not the case for DPOXC. The results of designed contrast using Bonferroni $t$ (Miller, 1966) are shown in Table 12. There is no interaction between speed and duration of exercise for either IRHR, ROXC, or DOXC, although there is a strong trend for DOXC at all six recovery times. IRHR is significantly affected by both speed and duration of exercise, whereas ROXC is only affected by speeds and only at 0.5 minutes. There is a strong trend for ROXC during the first 10 minutes of recovery. DOXC is significantly affected by duration of exercise only although there is a strong trend for exercise intensity ( S ), which becomes significant when the total response during recovery (over all time points) is considered.

In conclusion, it can be said that the HROXCR is different during exercise and recovery, and the reason is that the HR lags behind ROXC during the first 3 minutes of recovery. This lag in RHR is primarily effected by duration of exercise, and above $75 \%$ of maximal oxygen consumption there is little if any effect of exercise intensity.
Table 12.

| Var. | Test | Contrast | Recovery time (min) |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  |  |  | 0.5 | 3.0 | 7.5 | 17.5 | 27.5 | 37.5 |
| IRHR | Inter. | 1,4VS2,3 | -0.40 | 0.27 | -0.13 | -0.13 | 0.00 | 0.53 |
|  | Time | 1,3VS2,4 | $3.87{ }^{\text {b }}$ | $3.74{ }^{\text {b }}$ | $2.80{ }^{\text {b }}$ | 2.54 | 2.40 | 1.60 |
|  | Speed | 1,2VS3,4 | $3.34{ }^{\text {b }}$ | 2.14 | 2.27 | 2.00 | 1.87 | 2.14 |
|  | Speed | 1VS5 | $6.23{ }^{\text {b }}$ | $3.77{ }^{\text {b }}$ | $2.83{ }^{\text {b }}$ | $3.02{ }^{\text {b }}$ | $2.83{ }^{\text {c }}$ | 2.26 |
| ROXC | Inter. | 1,4VS2,3 | 2.05 | 0.41 | -0.20 | -0.91 | 0.00 | -0.71 |
|  | Time | 1,3VS2,4 | 0.81 b | -0.61 | -0.61 | 0.51 | 0.20 | -0.30 |
|  | Speed | 1,2VS3.4 | $8.71{ }^{\text {b }}$ | 2.03 | 1.01 | 0.10 | 0.61 | 0.30 |
|  | Speed | 1VS5 | $7.40{ }^{\text {b }}$ | 2.23 | 1.22 | 0.71 | 0.20 | 0.00 |
| DOXC ${ }^{\text {d }}$ | Inter. | 1,4VS2,3 | -1.17 | -0.51 | -0.31 | 0.27 | -0.08 | 0.43 |
|  | Time | 1,3vS2,4 | -2.34 | $-3.15{ }^{\text {b }}$ | $-2.72{ }^{\text {c }}$ | -2.38 | -2.50 | -2.07 |
|  | Speed | 1,2VS3,4 | -0.86 | 0.89 | 1.25 | 1.60 | 1.09 | 1.28 |
|  | Speed | 1VS5 | ---- | ---- | $4.25{ }^{\text {be }}$ | ---- |  |  |

$$
\begin{aligned}
& { }^{\text {e Contrast }} \text { based on all six recovery times. }
\end{aligned}
$$

## Discussion

## Effect of Diet and Previous Exercise

There is no evicence of any effect of the different dietary treatments upon either the slope or $y$-intercept of the regression equation between the HR and OXC. The HROXCR varies with time around one for the subject's characteristic value. One can therefore expect to find occasional significant values. As can be seen from Table 4, the significance does not occur at a particular treatment in more than one subject, but occurs at random.

As shown in Table 5, where the regression equation between HR and OXC is determined twice one hour apart in the same subject, the regression lines are significantly different, both in regard to slope and y-intercept. After one hour of rest, the OXC has returned to baseline values, but the $H R$ is still elevated by about 20 beats/minute. Using IHR will correct for the differences in resting HR, and as shown in Table 5, it does decrease the significance of the differences in the $y$-intercepts. However, since the slopes are different, this does not necessarily mean that the regression lines are more homogenous.

Schutz, et al (1981) have presented evidence that meals change the HROXCR. In their protocol, they first determined the regression equation, then fed the meals before re-establishing the regression line. Thus, the effect they found may equally well be due to an effect of the previous exercise. The older subjects presumably
were not very fit; so even though they used low intensity exercise, an effect of previous exercise cannot be excluded. The effect of previous exercise, in our experience, is dependent upon the training of the subjects.

Lundgren (1946) in his study on lumberjacks, also found an effect of breakfast on the HROXCR, but upon further study concluded that it was due to increased activity unrelated to the meal. He found a similar effect after a day of lumbering. Others have found that prolonged, heavy exercise causes an increase in HR (Saltin, 1964; Rowell, 1974).

## Recovery from Exercise and the HROXCR

The HROXCR during exercise and recovery are statistically significantly different throughout exercise at all work intensities, since the recovery OXC (ROXC) is less than the lower 95\% confidence limit (LCL) for the predicted OXC (POXC) based on the RHR and the prediction equation obtained during exercise. It is remarkable that DOXC increases rapidly during the first 3-4 min of exercise, and thereafter decreasing in parallel with the RHR. This means that the RHR falls behind the ROXC during the first few minutes of recovery, and then returns toward the resting value at the expected rate based on ROXC. During exercise there is a redistribution of the blood volume, with less blood going to the viscera and unused muscles (Saltin, 1964b; Saltin et al, 1968). During recovery the perfusion to these organs must increase which can further decrease an already compromised vascular volume and cause a decrease in SV and a compensatory increase in HR (Tanaka et al, 1979; Nadel et al, 1980).

In Figure 3, the RHR is plotted as a function of time. The HR stabilizes at a level of about 20 beats/minute above the resting level (x-axis). Figure 7 demonstrates the effect on the IHR of increasing the workload, and the differences are significant except at the last time interval (Table 12). Nandi and Spodick (1977) have obtained similar results, although they only followed the recovery for 5 minutes. However, as shown in Figure 8 for IRHR, there is also a significant effect of duration of exercise. The difference due to $D$ slowly decreases with time, and it is significant for the first 10 minutes only. When we look at the ROXC as shown in Figure 4 (lower curve), we see that the OXC decreases rapidly at first, and then slowly approaches the baseline level (the x-axis $=$ preexercise $0 \times C$ ). As shown in two subjects followed for 5 hours post-exercise, the ROXC ends approaching the POXC (Fig. 2), and both RHR and ROXC are lower than the preexercise resting level.

For the ROXC there are significant differences for the first minute only. There is no effect of $D$ upon the ROXC. This condition is clearly different from what was seen for the $H R$, where both speed and duration of exercise have significant effect. These results are in agreement with the data of Hagberg, et al (1980), who found that the OXC response was two-phasic during recovery. A rapid phase of approximately 2 minutes duration was significantly affected by the work intensity, but not by duration. Neither speed nor duration had any effect on the slow component, except for work intensities of higher than 65\% of maximal oxygen consumption. In the present study, we found highly significant effects of speed for the first minute only (fast component),
but no effect of duration. Although not significant, we found a strong positive effect of speed upon the slow component, even at $60 \%$ of the maximal oxygen consumption.

In Figure 4, the two upper lines show that POXC and 95\% lower confidence limit (LCL), and since both are based on the actual RHR, it is not surprising that they lag behind ROXC (lower curve). In Figure 2, POXC, LCL and ROXC are plotted as a function of RHR to illustrate the statistical significance. Only duration of exercise has a statistically significant effect upon DOXC, which is illustrated in Figure 10 by the two upper curves. However, as seen from Table 12, the effect of speed is significant when the lowest and highest speeds are contrasted for all times simultaneously.

It is concluded that the RHR does not predict the ROXC based on the regression equation during the preceding exercise period, because the RHR lags behind the ROXC during the first three minutes of recovery. This lag is affected by duration of exercise only above $65 \%$ of maximal oxygen consumption. At lower work intensities, there is an effect of intensity (S) also; and this lag in RHR is not observed below $50 \%$ of maximal oxygen consumption in moderately fit subjects.

Analysis of the regression equations for $H R$ and OXC during exercise following different dietary treatments provided no evidence for a dietary effect upon the HROXCR; but exercise clearly changes both the slope and $y$-intercept. It is the duration of activity which is primarily responsible for the lag in RHR, although at low work intensities (around $50 \%$ of maximal oxygen consumption, depending on the physical training of the subjects), it is not observed.

Despite the intra-subject variability, and the deviation of ROXC from POXC, the HROCSR still forms the best basis for prediction of activity and HR and OXC are still correlated when only the day-to-day variation in resting OXC is considered. The deviation during recovery will make the observed differences appear greater than they really are, which may be acceptable for many applications. Mode of work (Andrews, 1971) and environmental temperature (Dill \& Consolazio, 1962) also affect the HROXCR. It is recommended that at least three determinations of the individual regression line be made; but if that is not possible, it may be better to use a common slope.

During steady state exercise, the HR slowly increases. That is why duration of exercise has a significant effect upon RHR, as has also been found by others (Tanaka et al, 1979). This increase in HR appears to be correlated with an increase in body temperature (Tanaka et al., 1979). As the HR increases, there is a simultaneous decrease in stroke volume (Roberts \& Wenger, 1980; Nadel et al, 1980). SV, in addition to exercise, is also affected by the state of hydration (Nadel et al, 1980); and in fact, the effect of exercise is probably related to the decrease in vascular volume (Saltin, 1964b). Both thermal and metabolic dehydration cause a decrease in stroke volume and an increase in HR to compensate for the decrease in stroke volume at submaximal workload, while there is no difference in HR and OXC at maximal workload (Saltin, 1964a). Costill and Fink (1974) reported that dehydration due to heat was similar to that caused by exercise in the effect upon $H R$ and stroke volume, provided the same weight loss was obtained. Senay (1979) reported that training decreases the plasma
volume loss due to exercise, and in this study the best-trained subjects (as evidenced by lactate accumulation) had a less pronounced increase in HR during exercise and a faster recovery than less welltrained subjects.

## CHAPTER 4

DIETARY INDUCED THERMOGENESIS DURING REST
AND EXERCISE: EFFECT OF MEALS AND DIET ON OXYGEN CONSUMPTION AND ENERGY SUBSTRATE UTILIZATION DURING REST AND EXERCISE

## Synopsis

In this study the oxygen consumption was followed for 8 hours after consumption of a fat, carbohydrate, or protein meal respectively with measurements in the post-absorptive state (no food) as control. We found that each meal had a (statistically significant) different effect on the oxygen consumption during rest, whereas neither the meals nor a high fat or high carbohydrate diet (for 3 days prior to exercise) had any effect during exercise. Substrate utilization during rest (as evaluated by the $R Q$ ) was affected only by the carbohydrate meal, whereas none of the treatments had any effect during exercise. Exercise following the meals compared to control (postabsorptive state) did not show any thermogenic effect.

## Introduction

Overfeeding is generally thought to cause an increase in thermogenesis. It has been shown that subjects generally gain less weight than the excess energy intakes suggest (Dauncy, 1979). Conflicting evidence comes from Glick, et al (1977) who reported that there was no effect of overfeeding on thermogenesis. One explanation for the controversy may be that energy cost of physical activity is increased due to overfeeding, but Norgan and Durnin (1980) found no such effect. Norgan and Durnin also argue against what is termed "luxus-consumption" (Apfelbaum, et al, 1971). It has also been shown that underfeeding results in a decreased resting metabolic rate (RMR) (Apfelbaum, et al, 1971; Dauncy, 1979).

Rubner (1902) and Lusk (1928) found that protein had a specific thermogenic response; but more recently, Garrow and Hawes (1972) found a similar thermogenic effect of a test meal regardless of its nutrient content. Shetty, et al (1980) found similar results, and Owen, et al (1980) found a thermogenic response to breakfast as evaluated by the oral temperature. However, in an animal (pigs) study, Gurr, et al (1980) found a major response to a low protein diet, but none to a high protein diet. Dauncy (1979) reported a significant increase in 24-hour energy expenditure in man, when the energy intake was changed from glucose to protein.

Cold induced thermogenesis (CIT) due to epinepherine acting on brown adipose tissue and muscle is readily demonstrated in laboratory animals (Heldmeir, 1971) and also in man (Joy, 1963). This response can be reduced by $B$-adrenergic-blockade with propranolol (Dauncy and Ingram, 1979). In rats, DIT as well as CIT involves changes in the activity of the sympathetic nervous system (Rothwell and Stock, 1979; Glick, et al, 1981).

Capro, et al (1981) demonstrated that the ingestion of a standard meal caused an increase in blood insulin and glucose, but a decrease in plasma free fatty acids in man. In the present study, both protein and carbohydrate meals had a thermogenic effect (maximal response was 38 and $27 \%$ respectively). The effect of a carbohydrate meal lasted less than 6 hrs compared to more than 8 hrs for a protein meal.

The effect of diet on muscle glycogen content and therefore exercise endurance is well demonstrated (Karlson and Saltin, 1971). Under conditions where the glycogen stores are not expected to change, it has been shown that substrate concentration in the blood and the RQ are affected, whereas the OXC is not (Foster, et al, 1979) at exercise intensities of $80-100 \%$ of maximal oxygen consumption. Others have found an effect of diet upon energy substrate utilization at rest, but not during exercise (Hurni, et al, 1980). It is surprising that pre-exercise glucose feeding should have an effect on the substrate utilization during exercise, unless it affects the glycogen stores, since it has been shown that even at much lower work intensities, the major substrate is intramuscular (Essen, 1978). In the present study, there was no effect of diet or meals upon energy substrate utilization
during exercise ( $80 \%$ of maximal oxygen consumption), but carbohydrate meals increased utilization of carbohydrates during rest.

Apfelbaum, et al (1971) and Miller, et al (1967) found an increase in OXC during exercise following overeating. Bray, et al (1974) found no effect of overfeeding on OXC, but reported that a 4.2 and 12.6 MJ meal increased the OXC during exercise. However, in the present study, we found no thermogenic effect on exercise following a meal.

## Methods

The study was divided into two parts. The first part examined the effect of meals on OXC and the relative contribution of fat and carbohydrate to the energy utilization during rest. In the second part the effect of meals and diet composition upon the same parameters was investigated during exercise.

## Subjects

Two groups of five healthy male college students volunteered for the study. The physical characteristics of the subjects are given in Table 1. None of the subjects took any medication; and on the day of the experiment, they were requested not to drink any caffeine-containing beverages. They also did not participate in any strenuous physical activity on the day before the experiments were conducted.

Informed Consent
Each subject signed an informed consent form after the details of the experiments were described to him. The form stated the experimental procedures, identified possible risks, and noted that a subject could terminate his participation at any time.

## Diets

The composition of the diets and meals is given in Table 2. Each meal contained 700 kcal . The fat meal consisted of 288 ml whipping cream, which the subjects drank in less than 10 minutes. The protein

Table 1. Physical characteristics of the two groups of five adult male subjects. (SD = Standard Deviation).

| Physical Characteristics | Parts |  |  |  |
| :---: | :---: | :---: | :---: | :---: |
|  | Rest |  | Exercise |  |
|  | Mean | SD | Mean | SD |
| Age (years) | 26.6 | 1.7 | 24.8 | 3.1 |
| Body Weight (kg) | 78.2 | 11.3 | 74.5 | 11.9 |
| Height (cm) | 182 | 6.1 | 173 | 7.4 |
| $\mathrm{V}_{2}$-max ${ }^{\text {a }}$ (ml $\mathrm{O}_{2} / \mathrm{min} / \mathrm{kg}$ ) | -- | -- | 54.1 | 6.6 |

[^2]| Diets or Meals | Method | TotalEnergy (Kcal) | \% of total energy |  |  | \% by weight |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  |  |  | Fat | Protein | Carbohydrate | Water | Ash |
| Fat Meal | Food Tables ${ }^{\text {a }}$ | 700 | 92 | 3 | 5 |  |  |
| Protein Meal | Food Tables | 700 | 22 | 76 | 2 |  |  |
| Carbohydrate Meal | Food Tables | 700 | 2 | 5 | 93 |  |  |
| Fat Diet | Food Tables | 2,150 | 65 | 15 | 20 |  |  |
| Carbohydrate Diet | Food Tables | 2,150 | 20 | 15 | 65 |  |  |
| Fat Diet | Chemical Analysis | 2,150 | 66 | 16 | 19 | 68.0 | 4.0 |
| Carbohydrate Diet | Chemical Analysis | 2,150 | 22 | 15 | 63 | 72.8 | 3.4 |

${ }^{\text {a }}$ Watt and Merrill (1963); Church \& Church (1975).
meal consisted of 94 g creamed cottage cheese and 545 g (raw weight) skinned chicken breast, with all visible fat removed. The subjects ate the protein meal in 30 minutes. The carbohydrate meal consisted of 824 g ripe bananas (peeled), which were eaten in less than 10 minutes. The subjects drank 340 cc of ginger ale with the meals containing 1 kcal (Vernors, Detroit, MI), and were allowed salt, pepper, and water ad lib.

During the experimental periods the subjects ate 2,100 to 2,400 kcal per day either as a high fat or as a high carbohydrate diet. The composition is given in Table 2. The subjects were fed one of the two experimental diets in random order for three days prior to the exercise test, which was performed in the postabsorptive state the fourth day. The diet and meals were prepared and eaten in a kitchen adjacent to the exercise laboratory. Two menus were used for each diet: one for breakfast and one for dinner and supper. The menus were repeated for all three days. The subjects were supervised during the meals and ate all the allotted food. Duplicate portions were used for chemical analysis. Fat was analyzed by ether extraction, protein by microkjeldahl, minerals as ash $\left(600^{\circ} \mathrm{C}\right)$ and carbohydrate by difference.

For both diets regular food items were used. Sources of fat were: margarine, mayonnaise, cheese, eggs, bacon and olives; while sources of carbohydrate were: bananas, bread, potatoes, sweet potatoes, beans, carrots and peas. These foods contain some protein. Additional protein came from soy protein. For the fat diet it was imitation chicken, and for the carbohydrate diet it was imitation beef (Worthington Foods, Worthington, OH ).

## Measurements

Heart rate (HR) was obtained from an electrocardiogram (lead 2 with the positive lead in V5 position). The EKG signal was converted to heart rate through a cardiotachometer (built in our lab) and continuously recorded by a calibrated Sargent Recorder, Model DTM-115-4 (Sargent and Company, Chicago, IL). The mean HR in beats/minute corresponding to each collection of expired air was calculated from the recording.

The oxygen consumption (OXC) was determined by a modified Douglas method (Consolazio, et al, 1963). The expired air was collected through a low resistance valve (Otis-McKerrow, Warren Collins, Inc., Braintree, MA) in light-weight neoprene bags. The composition ( $\mathrm{O}_{2}$ and $\mathrm{CO}_{2}$ contents) of the collected air was immediately determined using the Beckman LB-2 carbon dioxide and Beckman OM-11 oxygen analyzers, respectively (Beckman Instruments, Schiller Park, IL). The air volume was determined by metering through (using a constant flow of $50 \mathrm{1} / \mathrm{min}$ ) a Singer dry gas meter (American Meter Company, Philadelphia, PA).

The mean OXC for the control experiment was compared the baseline (mean resting OXC prior to the :ingestion of the meal). To correct for the day to day variation in the resting OXC in each subject the difference between the control and baseline values were added or subtracted from the OXC for that day's treatment.

Lactic acid was determined in duplicate arterialized blood samples (Gambino, 1961; Jung et al, 1966) collected from the fingers after the hand was immersed in warm water $\left(45^{\circ} \mathrm{C}\right)$ for two minutes. The finger was dried, cleaned with alcohol and punctured with a lancet.

The first drop of blood was wiped off with sterile gauze. One hundred $\mu 1$ of blood was collected in a capillary tube centrifuged at 2000 rpm in $8 \%$ perchloric acid. The sample was then incubated at room temperature with lactate dehydrogenase for one hour. The nicotine adenine dinucleotide (NADH) generated was measured on a monocromatic spectrophotometer (Gilford Stasar II, Gilford Instruments, Inc., Oberlin, OH) at 340 nm .

## Exercise Tests

The maximal OXC was determined in a continuous incremental test with the subjects exercising on the treadmill until exhaustion. The speed was increased stepwise at $8.8,10.5,12.5$ and $14.3 \mathrm{~km} / \mathrm{hour}$. When the subjects reached the highest speed they could comfortably run (12.5 or $14.3 \mathrm{~km} / \mathrm{hr}$ ) further increases in the workload were accomplished by increasing the treadmill inclination in steps of $2 \%$. The subjects ran 3 minutes at each step, the $H R$ was recorded continuously, three one-minute bags of expired air were collected at each step, and the oxygen consumption calculated for each minute.

The OXC following the different treatments was determined through an incremental exercise test with the final workload corresponding to $80 \%$ of the subjects' maximal OXC. Three collections of expired air were made at each step and the mean HR corresponding to each air collection was recorded. The test started at rest with the subject seated in an armchair, while 3 consecutive 5-minute collections of expired air were made. During the following steps the subjects would walk or run on the treadmill. At the $4.8 \mathrm{~km} / \mathrm{hr}$ and $6.8 \mathrm{~km} / \mathrm{hr}$ levels
the subjects exercised for 6 minutes each, and at the other levels they exercised for 3 minutes each; except when they reached the final speed (the one corresponding closest to $80 \%$ of their maximal OXC), they continued the exercise for 20 minutes. During the final stage, one-minute bags of expired air were collected, every second minute starting with the second minute.

Following the exercise the HR and OXC were determined during 40 minutes of recovery. Two one-minute bags, one three-minute and one five-minute bag of expired air were collected during the first 10 minute of recovery. During the next 30 minutes, 3 five-minute bags of expired air were collected every second 5 -minute period. During the alternate 5 -minute periods, when no collections were made, the subject remained seated in the armchair, but was relieved of the face mask with air-collection valve and the noseclip. Arterialized blood samples were collected from one of the fingers at $5,15,25$ and 40 minutes of recovery for determination of the blood lactate concentration.

## Experimental Protocol

Rest
For measurements taken during rest, the subjects were seated in an armchair. The subjects were asked to restrict their movement, but at the same time, to change the leg position approximately once every minute to avoid venous pooling. The temperature of the laboratory was kept constant $\left( \pm 1^{\circ} \mathrm{C}\right)$ throughout the experiment, but it did vary somewhat between experimental days depending on the environmental
temperature. The humidity and barometric pressure could not be controlled.

The day of the experiment, the subjects arrived in the laboratory about 8:00 am in the postabsorptive state (after overnight fast), and spent the day in the laboratory resting, reading, or talking. No food or beverages except the experimental meal were consumed, but the subjects drank as much water as they wanted. The three treatments were (1) the fat meal, (2) the protein meal, and (3) the carbohydrate meal described previously, and for the control treatment, no food was given. The four treatments were done in a random order one week apart.

Before the experimental meal was given, six five-minute baseline measurements of OXC and HR were taken. The subject was seated in an armchair for three consecutive five-minute periods while expired air was collected and HR recorded. The subject was then given a fiveminute break in which he walked around in the laboratory before another series of three consecutive five-minute collections of expired air and recordings of HR were carried out. For each five minutes, one bag of expired air was collected for analysis of OXC and RQ. The mean HR was calculated for each bag of expired air. The mean of the six determinations was used as the baseline (time 0 ) for that particular treatment (meal). Duplicate arterialized blood samples for lactate determinations were collected before the meals were eaten.

After baseline determination, the subjects ate the experimental meal and three consecutive five-minute collections of expired air and recordings of $H R$ were taken at 30 minutes and at one hour following the completion of the meal and thereafter at one-hour intervals for eight
hours following the meal. The mean of the three measurements was used. During the control treatment, when no meal was given, the protocol was the same, except that no "baseline" measurements were taken. Blood was collected for lactate determination at the same times as the other measurements were taken except for the first hour, when blood samples were taken every 15 minutes. Blood collections were discontinued after 6 hours. During rest lactate was measured only during the control treatment and following the carbohydrate meal.

## Exercise

The exercise experiments were carried out over a 10-week period with each test given one week apart. During the first and last tests the maximal OXC were determined. The second test was always a control (postabsorptive state) prior to which the subjects ate their normal diet. Prior to the third test, the subjects consumed one of the experimental diets for three days (two started with the high fat diet and three with the high carbohydrate diet). Then followed another control test before the opposite experimental diet was introduced, three days prior to the fifth test. Following both dietary treatments, the test was carried out in the postabsorptive state. The three meals and a final control were introduced in random order so that all the subjects would not consume the meals in the same order. The exercise tests began 30 minutes after termination of the carbohydrate meals and 2-3 hours following the fat and protein meals, which we had found to correspond to the maximal thermogenic response.

Statistical Analysis
OXC, RQ and lactate were analyzed by a split block (block = subjects) repeated measurements design. The differences among treatment means were evaluated by Tukey's test (Tukey, 1953). The correlation coefficient between HR and OXC was calculated for each test at rest in each subject, and the mean and confidence limits for each dietary treatment and control were calculated by conversion to the Z-score (Steel and Torrie, 1960).

## Results

## Experiments During Rest

Table 3 shows resting OXC for eight hrs following the experimental meals and during control when no food was given. All treatments were compared with control and each other and the Tukey test was used to determine the significance of the treatment differences shown in Table 3. Although the fat meal caused an increased OXC compared to control from 1 to 6 hrs after the meal it was not significant. The protein meal caused a significant increase in OXC compared to control from 1 to 6 hrs after the meal; and the carbohydrate meal caused OXC to be significantly different from control to 3 hrs following the meal. The protein meal caused a significant increase in OXC from 3 to 6 hrs compared to the carbohydrate meal. The mean increases in OXC compared to control following the fat, protein and carbohydrate meals was $7.3 \%, 21.2 \%$ and $12.8 \%$ respectively, while the peak increases were $8.9 \%, 33.0 \%$ and $27.4 \%$ respectively. The different thermogenic responses for the treatments are shown in Figure 1.

Table 4 shows the effects of the treatments on the respiratory quotient (RQ). Only following the carbohydrate meal is there a significant effect upon the RQ compared to control. Although the RỌ is increased for the eight hrs observed, the increase is only significant ( $p<0.05$ ) for 3 hrs . The maximal increase in RỌ compared to control is 0.14 and the mean increase is 0.09 . The RQ for the carbohydrate meal and control is plotted in Figure 2.

Table 3. Mean oxygen consumption following the ingestion of various meals for 5 male subjects in the resting state. Control was the postabsorptive state.

| Time <br> (hrs) | Control | Fat | Protein | Carbohydrate |
| :--- | :--- | :--- | :--- | :--- |
| 0 | 3.60 | 3.56 | 3.62 | 3.56 |
| 0.5 | 3.58 | 3.62 | $4.04^{\mathrm{cf}}$ | $4.06^{\mathrm{b}}$ |
| 1.0 | 3.60 | 3.82 | $4.32^{\mathrm{af}}$ | $4.32^{\mathrm{a}}$ |
| 2.0 | 3.62 | 3.90 | $4.66^{\mathrm{ad}}$ | $4.56^{\mathrm{a}}$ |
| 3.0 | 3.58 | 3.90 | $4.76^{\mathrm{ag}}$ | $4.14^{\mathrm{bg}}$ |
| 4.0 | 3.56 | $3.90^{\mathrm{c}}$ | $3.82^{\mathrm{c}}$ |  |
| 5.0 | 3.48 | 3.70 | $4.54^{\mathrm{adg}}$ | $3.84^{\mathrm{g}}$ |
| 6.0 | 3.60 | 3.62 | $4.22^{\mathrm{ach}}$ | $3.70^{\mathrm{g}}$ |
| 7.0 | 3.56 | 3.58 | $3.96^{\mathrm{cf}}$ | $3.66^{\mathrm{h}}$ |
| 8.0 | 3.58 |  | $3.92^{\mathrm{cf}}$ | 3.64 |

$a / b / c_{V a l u e s ~ f o r ~ a n y ~ o f ~ t h e ~ m e a l s ~ a r e ~ s i g n i f i c a n t l y ~ d i f f e r e n t ~ f r o m ~}^{\text {a }}$ control at the same time ( $a, p<0.01, b p<0.05 ; c, p<0.10$ ).
$d / e / f$ Values for the carbohydrate - or protein meal are significantly different from the fat meal at the same time ( $d, p<0.01$; $e, p<0.05 ; f, p<0.10)$.
$g / h / i_{V a l u e s ~ f o r ~ t h e ~ p r o t e i n ~ m e a l ~ a r e ~ s i g n i f i c a n t l y ~ d i f f e r e n t ~ f r o m ~}^{\text {f }}$ the carbohydrate meal at the same time ( $g, p<0.01$; $h, p<0.05$; $i, p<0.10$ ).

Standard errors of individual treatment means at one time are irrelevant for comparisons or reliability.

Standard error of difference of 2 treatments at one time is 0.157.
Standard error of difference of 2 times for one treatment is 0.088 .
OXC in $\mathrm{ml} / \mathrm{min} / \mathrm{kg}$.

Table 4. Mean respiratory quotient following the ingestion following ingestion of various meals for 5 male subjects in the resting state. Control was the postabsorbtive state.

| Time, | Meals |  |  |  |
| :---: | :---: | :---: | :---: | :---: |
| (hrs) | Control | Fat | Protein | Carbohydrate |
| 0 | 0.750 | 0.762 | 0.756 | 0.760 |
| 0.5 | 0.750 | 0.756 | 0.756 | $0.856^{\mathrm{cfi}}$ |
| 1.0 | 0.746 | 0.760 | 0.760 | $0.880^{\mathrm{eh}}$ |
| 2.0 | 0.750 | 0.746 | 0.762 | $0.892^{\mathrm{adh}}$ |
| 3.0 | 0.744 | 0.736 | 0.736 | $0.876^{\mathrm{bdg}}$ |
| 4.0 | 0.746 | 0.740 | 0.754 | $0.342^{\mathrm{cf}}$ |
| 5.0 | 0.764 | 0.732 | 0.712 | $0.808^{\mathrm{i}}$ |
| 6.0 | 0.750 | 0.736 | 0.752 | 0.780 |
| 7.0 | 0.754 | 0.738 | 0.740 | 0.796 |
| 8.0 | 0.742 | 0.748 | 0.744 | 0.790 |

$a / b / c_{\text {Values }}$ for any one of the meals are significantly different from control at the same time ( $a, p<0.01 ; b, p<0.05 ; c, p<0.10$ ).
$d / e / f_{\text {Values }}$ for the carbohydrate - or protein meal are significantly different from the fat meal at the same time ( $d, p<0.01$; e, $p<0.05 ; f, p<0.10$ ).
$\mathrm{g} / \mathrm{h} / \mathrm{i}^{\mathrm{V}}$ alues for the protein and carbohydrate meals are significantly different from each other ( $g, p<0.01 ; h, p<0.05$; i, $p<0.10$ ).

Standard errors of individual treatment means at one time are irrelevant for comparisons or reliability.

Standard error of difference of 2 treatments at one time is 0.035 .
Standard error of difference of 2 times for one treatment is 0.023 .

Figure 1. OXC as a function of time. Treatments are: $C=$ control (lower curve), $\mathrm{CH}-\mathrm{M}=$ carbohydrate meal (middle curve), and $\mathrm{P}-\mathrm{M}=$ protein meal (upper curve). All treatments are the mean of 5 subjects. The $5 \%$ least significant difference (LSD) is 0.47. SE for difference between the treatment means at the same time is $0.111 \mathrm{ml} / \mathrm{min} / \mathrm{kg}$, and the $S E$ for the difference between two time means in the same treatment is $0.062 \mathrm{ml} / \mathrm{min} / \mathrm{kg}$.


FIGURE 1

Figure 2. Respiratory Quotient (R.Q.) as a function of time for $C H-M$ and Control (C). No other treatment had a significant effect. LSD is $(5 \%)=0.104$. Mean of 5 subjects. (SE for the difference between the treatment means at the same time is 0.025 and the $S E$ for difference between two time means in the same treatment is 0.016.)


FIGURE 2

As will be reported elsewhere the blood lactate concentration was measured following the carbohydrate meal and during the control. The results are shown in Figure 3. The peak lactate response of about 2.4 mM occurred 1 hr following the meal and the response was significant for 3 hrs .

The mean correlation coefficient ( $\bar{r}$ ) between the HR and OXC was calculated for each treatment. As seen from Table 5 only for the carbohydrate meal was $\bar{r}$ significantly different from $0(\bar{r}=0.516)$.

## Exercise Experiments

The mean OXC for the 20 min during exercise following the fat, protein, carbohydrate meal, the high fat; high carbohydrate diet and control were: $41.7,41.8,42.0,41.5,41.6$ and $42.4 \mathrm{ml} / \mathrm{min} / \mathrm{kg}$ body weight. The standard error for difference of 2 treatments at one time is $2.58 \mathrm{ml} / \mathrm{min} / \mathrm{kg}$. The RQ during exercise following the fat, protein, carbohydrate meals, the high fat, high carbohydrate diets and control were: $0.84,0.84,0.87,0.86,0.83,0.85$; and the standard error was 0.069 . There were no statistical significant treatment differences for either OXC or RQ.

Figure 3. Blood lactate as a function of time after a $\mathrm{CH}-\mathrm{M}$ and C (at least 5 hours postabsorptive). Mean of 5 subjects. Five percent $L S D=1.471$. (The. $S E$ for difference between the two treatment means at the same time is $0.239{ }^{\mathrm{m} M}$, and the SE for difference between the two time means in the same treatment is 0.072 mM .)
bLOOD LACTATE (REST)


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FIGURE 3

Table 5. Mean correlation coefficient (4) between $H R$ and OXC during control, following a fat meal, protein meal, and a carbohydrate meal. Number of subjects equal to 6 for control, 7 for fat meal, and 8 for carbohydrate meal. $n=30$ in each treatment for each
 the treatment.

| Treatment | Control | Fat Meal | Protein Meal | Carbohydrate <br> Meal |
| :---: | :---: | :---: | :---: | :---: |
| $r$ | -0.075 | 0.058 | 0.041 | 0.516 |
| $r_{U}{ }^{a}$ | 0.094 | 0.211 | 0.195 | 0.609 |
| $r_{L} b$ | -2.243 | -0.101 | -0.117 | 0.390 |

${ }^{a} r_{u}=$ Upper $95 \%$ confidence limit.
$\mathrm{br}_{1}=$ Lower 95\% confidence limit.

## Discussion

## OXC During Rest

CIT is caused by the action of catecholamines upon brown adipose tissue and muscles (Cannon, et al, 1977) and it is generally assumed that DIT operates in a similar manner (Dauncey, 1979; Rothwell and Stock, 1979; Glick, 1981). In the present study there was a specific effect of each energy nutrient on the OXC; however, only the carbohydrate meal caused a simultaneous change in the energy substrate utilization from fat to carbohydrate. This is further substantiated by the increase in RQ. Furthermore, only the change in OXC caused by carbohydrate meal correlates with the HR, as shown in Table 5. As shown by Sonne and Galbo (1980)., catecholamines would be expected to have a simultaneous effect upon $H R$ and OXC. The fact that this is the case for the carbohydrate meal may be related to the effect of carbohydrate upon the central nervous system. Thus, Lansberg and Young (1978) found that overfeeding stimulates and fasting suppresses the sympathetic nervous system în mice, and they state "changes in sympathoadrenal activity may, at least in part, explain the fluctuations in metabolic rate and heat production that occur with changes in diet". Crandall and Fernstrom (1980) found that carbohydrate feeding increased brain indoles in rats, whereas protein meals had no effect.

It is not surprising that fat does not have much effect upon the OXC, since the subjects (after an overnịght fast) were already primarily consuming fat, as evidenced by the relatively low RQ's of about
0.76 (Table 4). However, in one subject there was a clear response; and this subject normally did not consume fat; this raises the questions of whether customary food consumption habits affect the DIT. Stock (1980) found no effect of fasting and overeating for one day on the thermogenic response to a test meal, but that does not exclude an effect on the composition of the diet a subject has customarily consumed. One could, for example, anticipate that subjects who normally subsist on low levels of proteins may respond differently to a P-M from subjects who habitually consume large amounts of protein. Gurr, et al (1980) found that in pigs a high carbohydrate diet caused a larger DIT response than a high protein diet, and similar results have been obtained in rats (Tulp, et al, 1977); but it is important to distinguish between true DIT and effect of increased activity. The feeding pattern and presumably also the composition of the diet can affect the activity (Spangler and Johnson, 1981). The experimental protocol used in the present study was very demanding upon the subjects, and in order to make it more acceptable we tried to let the subjects listen to music. We found, however, that the music caused large variation in the OXC and HR and was therefore not used. In preliminary studies it was also found that the energy content of the meal, and the time the subjects spent eating appeared to effect the thermogenic response. For fat and carbohydrate meals, eating time of about 30 minutes could totally abolish the response; and for protein meal, reducing the energy content to half also reduced the thermogenic response to about half. These factors need to be systematically studied, but they may explain why originally, only
protein was thought to have an effect, and why mixed meals sometimes gave different responses from a meal consisting of a single nutrient (Garrow and Hawes, 1972). In the present study, smoking and caffeinecontaining beverages were excluded, since coffee has been shown to have an effect on OXC (Acheson, et al, 1980).

For the carbohydrate meal the OXC, RQ and blood lactate showed similar time responses, although the lactate response decreased about an hour earlier. The other treatments (fat and protein meals) had no effect on the $R Q$, and in the case of lactate, were only studied in two subjects (no response). The similarity in the $O X C, R Q$ and lactate response points to the possibility that DIT is a metabolic response unrelated to CIT (and catecholamines). Capro, et al (1981) found that a mixed meal caused a postprandial increase in plasma glucose and insulin that lasted about 4 hours, and a decrease in the free fatty acids for about 5 hours. Similar results were obtained by Owen, et al (1980). Rothwell and Stock (1981) found that insulin was increased during diet-induced thermogenesis in cafeteria-fed rats.

While the data (Table 3) show specific increases in OXC for the protein and carbohydrate meals, the OXC correlates with the HR only for the carbohydrate meal and may indicate a difference in the underlying mechanism for the thermogenic response. It is doubtful that activity played any role in the thermogenic responses found in the present study, but it may have played a major role in many animal studies, as discussed by Scheibel, et al (1979). Thyroid hormones are also involved in thermogenesis (Sestoft, 1980; Danforth, et al, 1979),

1979; Elkin, et al, 1980; Davidson and Chopra, 1979) but experiments of Jung, et al (1979) indicate that thyroid hormones are not involved in DIT.

## OXC During Exercise

In contrast to the situation during rest, we find that the dietary treatments have no significant effect on OXC during exercise. The mean OXC during exercise is $42 \mathrm{ml} / \mathrm{min} / \mathrm{kg}$ and the standard error for difference between treatment at the same time is $2.58 \mathrm{ml} 0_{2} / \mathrm{min} / \mathrm{kg}$, which gives a coefficient of variation (CV) of about $6 \%$. In a study designed to test the variability in the energy expenditure of standard activity, Durnin and Namyslowski (1958) found a CV of about $10 \%$ in ten male subjects, which is the same order of magnitude as in the present study.

Several authors have found an effect of diet upon the OXC during exercise (Miller, et al, 1967; Apfelbaum, et al, 1971; Bray, et al, 1974), either as a result of overfeeding or following a meal. However, Swindells (1972) did not find any effect of meals upon DIT during exercise, and Norgan and Durnin (1980) did not find any effect of overfeeding when correcting for increases in body weight (as in our study). It is possible, as suggested by Bray, et al (1974), that the size of a meal makes a difference, so that an effect is only apparent after intake of at least 4.2 MJ. Few people normally eat a meal consisting of 12.6 MJ as used by Bray, et al (1974), and using single nutrients, as we did, it is impossible for most subjects to eat more than about 3 MJ. Garby and Lambert (1977) found no effect of a 4 MJ meal.

## Energy Substrate Utilization Durina Exercise

There is no effect of any dietary treatment on the type of substrate used during exercise. The mean $R Q$ during exercise was 0.85 , the SE is 0.07 , and the CV is $7 \%$. In the present study, the subjects exercised at about $80 \%$ of their maximal oxygen consumption, and the exercise lasted for only 20 minutes in order to avoid an effect of diminishing muscle glycogen stores. When evaluating the effect of diet upon the substrate utilization during exercise, it is necessary to consider both the relative work load and the duration of exercise. The percent of substrate supplied by glucose increases from negliạible at rest to about $35 \%$ at moderate work loads for one hour (Wahren, et al, 1971); but during longer exercise times, the alucose utilization increases further and peaks at $90-180$ minutes of exercise (Alborg, et al, 1974).

At relatively low levels of activity ( $30-50 \%$ of maximal oxygen consumption), where blood borne substrates play an important role in the energy supply, pre-exercise glucose feeding increases glucose utilization during exercise. The changes appear to be hormonally mediated (insulin and glucagon) (Ahlborg and Felig, 1977; Pirnay, et al, 1977; Luyckx, et al, 1978; Maughan, et al, 1978). Martin, et al (1978) found that a high fat diet for 3 days ( $90 \%$ of energy as fat or protein) at $70 \%$ of the maximal oxygen consumption markedly decreased carbohydrate utilization, but such a diet very likely affected the muscle glycogen stores (Astrand, 1967). At 80\% and 100\% of maximal oxygen consumption, Foster, et al (1979) found that ingestion of glucose compared to water gave a small increase in the

RQ, but the variations in the R $\cap$ were not significant at 20 minutes. Hurnie, et al (1980) found that although diet affected the RQ at rest, this was not the case during exercise.

Ravussin, et al (1979) studied the effect of exercise ( $40 \%$ of maximal oxygen consumption) on glucose utilization in glycogen depleted subjects and controls following ingestion of $100 \mathrm{~g}^{13} \mathrm{C}$-glucose, and found that the use of exogenous glucose was similar, but the decreased glycogen utilization in depleted subjects was replaced by lipid oxidation, not endogenous carbohydrate.

Artificially elevating plasma FFA with heparin caused an increased utilization of fat, whereas 75 g of glucose injested 45 minutes prior to exercise increased glucose utilization (RQ) at $70 \%$ of maximal oxygen consumption (Costill, et al, 1977). Bergstrom, et al (1979) used nicotinic acid to block the release of FFA from adipose tissue. Up to about $65 \%$ of maximal oxygen consumption, it caused an increase in the RQ. Despite a marked decrease in plasma FFA, it had no effect upon the muscle substrate utilization at higher work loads. Hagenfeldt and Wahren (1972) found that at high work loads, fat oxidation exceeded FFA uptake, suggesting utilization of intramuscular lipid stores. Jones, et al (1980) likewise found that at light exercise, FFA is extracted from the circulation, whereas in heavy exercise, muscle triglycerides play a more important role. Essen (1978) has also shown a significant contribution of intramuscular substrates (glycogen and triglycerides) during exercise, and this contribution increases with the intensity of the exercise.

Bylund-Felenius, et al (1981) have presented evidence that the intramuscular oxygen tension $\left(\mathrm{pO}_{2}\right)$ determines the substrate utilization during exercise, but hormones (insulin, glucagon and catecholamines) probably also play a role (Norris, et al, 1978). At rest and during low intensity exercise, it is clear that diet affects both blood nutrient levels and substrate utilization (Ahlborg and Felig, 1977). While at high work intensities, blood levels of nutrients are still affected by diet (Foster, et al, 1979); the present study gives no support for a dietary effect upon substrate utilization at high work loads ( $>75 \%$ of maximal oxygen consumption) provided the glycogen stores are not affected. Similar results were obtained by Bergstrom, et al (1969).

## CHAPTER 5

EFFECT OF MEALS AND DIET ON POSTEXERCISE BLOOD LACTATE LEVELS

## Synopsis

The effects of a high fat diet and a high carbohydrate diet; and fat, protein and carbohydrate meals on postexercise lactate were investigated. Five subjects were exercised for 20 minutes at approximately 80\% of maximal oxygen consumption. After each of the diets and control they were exercised in the postabsorptive state, and after the meals in the absorptive state. The lactate production was also measured in the resting state for six hours following a carbohydrate meal, and during control (postabsorptive state). Following a carbohydrate meal, there was a four-fold increase in the whole blood lactate concentration compared to control. After exercise, blood lactate was measured at 5, 15,25 , and 40 minutes. There was a significant time by treatment interaction for carbohydrate meal compared to control and fat meal. The total response was also significantly different for carbohydrate meal compared to fat meal. These results do not support the hypothesis that diet affects the lactate production during exercise, although a carbohydrate meal has a significant effect upon the decrease in blood lactate concentration during recovery.

## Introduction

The widely used concept that the percentage of maximal oxygen consumption can be used to equalize the physiological work load has recently been questioned (Katch, et al, 1978). The authors showed that at the same relative percent oxygen uptake, the subjects had different anaerobic thresholds. Kinderman, et al (1979) have showed that the aerobic-anaerobic threshold is a better predictor of the physiological work load.

It is controversial whether decreased muscle oxygen tension, high rate of glycogenolysis, or both are responsible for lactate production (Bylund-Fellenius, 1981; Jacobs, 1981). However, it is clear that a number of factors affect the blood lactate. These include muscle respiratory capacity and fiber composition, mode (rest or exercise) of recovery (McGrail, et al, 1978; Poortmans, et al, 1978; Stanford, et al, 1981), as well as muscle glycogen content (Graham, 1978; Ivy, et al, 1980; Jacobs, 1981).

A number of studies have demonstrated how previous exercise and diet can affect muscle glycogen and lactate production (Jacobs, 1981; Essen, 1978; Bonen et a1, 1981; Maughan, et al, 1978; and Maughan and Poole, 1981). Thus, Maughan $(1978,1981)$ has shown that at both high and low intensity work, an exercise-diet regimen that increases muscle glycogen also increases lactate production during exercise and vice versa. The effect of lactate agrees with the explanation of Newsholme
and Crabtree (1979) that lack of substrate (glycogen) will reduce the flow through a metabolic pathway. Robin and Hance (1980) emphasize the importance of enzymes and substrates in regulation of the glycogenolytic rate, and it has been demonstrated that blood FFA's affect the glycolytic rate and lactate production (Bergstrom, et al, 1969; Foster, et al, 1979); although Bergstrom found no effect at high work loads.

In the present study we evaluated the effect of diet on the post-exercise blood lactate under conditions in which one would not expect any effect on muscle glycogen. We found that diet did effect blood lactate recovery rate but not the blood lactate concentration 5 min following exercise.

## Methods

The study was divided into two parts. The first part examined the effect of meals on OXC and the relative contribution of fat and carbohydrate to the energy utilization during rest. In the second part the effect of meals and diet composition upon the same parameters was investigated during exercise.

## Subjects

Two groups of five healthy male college students volunteered for the study. The physical characteristics of the subjects are given in Table 1. None of the subjects took any medication; and on the day of the experiment, they were requested not to drink any caffeine-containing beverages. They also did not participate in any strenuous physical activity on the day before the experiments were conducted.

## Informed Consent

Each subject signed an informed consent form after the details of the experiments were described to him. The form stated the experimental procedures, identified possible risks, and noted that a subject could terminate his participation at any time.

Diets
The composition of the diets and meals is given in Table 2. Each meal contained 700 kcal. The fat meal consisted of 288 ml whipping cream, which the subjects drank in less than 10 minutes. The protein

Table 1. Physical characteristics of the two groups of five adult male subjects.

| Physical | Parts |  |  |  |  |
| :--- | :--- | :---: | :--- | :---: | :---: |
| Characteristics | Rest |  |  | Exercise |  |
|  | Mean | SD $^{\mathrm{a}}$ | $\underline{M e a n}$ | $\underline{\text { SD }}$ |  |
| Age (years) | 26.6 | 1.7 | 24.8 | 3.1 |  |
| Body Weight (kg) | 78.2 | 11.3 | 74.5 | 11.9 |  |
| Height (cm) | 182 | 6.1 | 173 | 7.4 |  |
| Max0xC |  |  |  |  |  |

${ }^{\text {a }}{ }^{S D}=$ Standard Deviation. $\quad b_{\text {Maximal }}$ OXC.

| Diets or Meals | Method | TotalEnergy (Kcal) | \% of total energy |  |  | \% by weight |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  |  |  | Fat | Protein | $\begin{aligned} & \text { Carbo- } \\ & \text { hydrate } \end{aligned}$ | Water | Ash |
| Fat Meal | Food Tables ${ }^{\text {a }}$ | 700 | 92 | 3 | 5 |  |  |
| Protein Meal | Food Tables | 700 | 22 | 76 | 2 |  |  |
| Carbohydrate Meal | Food Tables | 700 | 2 | 5 | 93 |  |  |
| Fat Diet | Food Tables | 2,150 | 65 | 15 | 20 |  |  |
| Carbohydrate Diet | Food Tables | 2,150 | 20 | 15 | 65 |  |  |
| Fat Diet | Chemical Analysis | 2,150 | 66 | 16 | 19 | 68.0 | 4.0 |
| Carbohydrate Diet | Chemical Analysis | 2,150 | 22 | 15 | 63 | 72.8 | 3.4 |

[^3]meal consisted of 94 g creamed cottage cheese and 545 g (raw weight) skinned chicken breast, with all visible fat removed. The subjects ate the protein meal in 30 minutes. The carbohydrate meal consisted of 824 g ripe bananas (peeled), which were eaten in less than 10 minutes. The subjects drank 340 cc of ginger ale with the meals containing 1 kcal (Vernors, Detroit, MI), and were allowed salt, pepper, and water ad lib. During the experimental periods the subjects ate 2,100 to 2,400 kcal per day either as a high fat or as a high carbohydrate diet. The composition is given in Table 2. The subjects were fed one of the two experimental diets in random order for three days prior to the exercise test, which was performed in the postabsorptive state the fourth day. The diet and meals were prepared and eaten in a kitchen adjacent to the exercise laboratory. Two menus were used for each diet: one for breakfast and one for dinner and supper. The menus were repeated for all three days. The subjects were supervised during the meals and ate all the allotted food. Duplicate portions were used for chemical analysis. Fat was analyzed by ether extraction, protein by microkjeldahl, minerals as ash $\left(600^{\circ} \mathrm{C}\right)$ and carbohydrate by difference. For both diets regular food items were used. Sources of fat were: margarine, mayonnaise, cheese, eggs, bacon and olives; while sources of carbohydrate were: bananas, bread, potatoes, sweet potatoes, beans, carrots and peas. These foods contain some protein. Additional protein came from soy protein. For the fat diet it was imitation chicken, and for the carbohydrate diet it was imitation beef (Worthington Foods, Worthington, OH ).

## Measurements

Heart rate (HR) was obtained from an electrocardiogram (lead 2 with the positive lead in V5 position). The EKG signal was converted to heart rate through a cardiotachometer (built in our lab) and continuously recorded by a calibrated Sargent Recorder, Model DTM-115-4 (Sargent and Company, Chicago, IL). The mean HR in beats/minute corresponding to each collection of expired air was calculated from the recording.

The oxygen consumption (OXC) was determined by a modified Douglas method (Consolazio, et al, 1963). The expired air was collected through a low resistance valve (Otis-McKerrow, Warren Collins, Inc., Braintree, MA) in light-weight neoprene bags. The composition ( $\mathrm{O}_{2}$ and $\mathrm{CO}_{2}$ contents) of the collected air was immediately determined using the Beckman LB-2 carbon dioxide and Beckman OM-11 oxygen analyzers, respectively (Beckman Instruments, Schiller Park, IL). The air volume was determined by metering through (using a constant flow of $50 \mathrm{l} / \mathrm{min}$ ) a Singer dry gas meter (American Meter Company, Philadelphia, PA).

The mean OXC for the control experiment was compared the baseline (mean resting OXC prior to the:ingestion of the meal). To correct for the day to day variation in the resting OXC in each subject the difference between the control and baseline values were added or subtracted from the OXC for that day's treatment.

Lactic acid was determined in duplicate arterialized blood samples (Gambino, 1961; Jung et al, 1966) collected from the fingers after the hand was immersed in warm water $\left(45^{\circ} \mathrm{C}\right)$ for two minutes. The finger was dried, cleaned with alcohol and punctured with a lancet.

The first drop of blood was wiped off with sterile gauze. One hundred $\mu 1$ of blood was collected in a capillary tube centrifuged at 2000 rpm in $8 \%$ perchloric acid. The sample was then incubated at room temperature with lactate dehydrogenase for one hour. The nicotine adenine dinucleotide (NADH) generated was measured on a monocromatic spectrophotometer (Gilford Stasar II, Gilford Instruments, Inc., Oberlin, OH ) at ${ }^{\prime} 340 \mathrm{~nm}$.

## Exercise Tests

The maximal OXC was determined in a continuous incremental test with the subjects exercising on the treadmill until exhaustion. The speed was increased stepwise at $8.8,10.5,12.5$ and $14.3 \mathrm{~km} / \mathrm{hour}$. When the subjects reached the highest speed they could comfortably run (12.5 or $14.3 \mathrm{~km} / \mathrm{hr}$ ) further increases in the workload were accomplished by increasing the treadmill inclination in steps of $2 \%$. The subjects ran 3 minutes at each step, the HR was recorded continuously, three one-minute bags of expired air were collected at each step, and the oxygen consumption calculated for each minute.

The OXC following the different treatments was determined through an incremental exercise test with the final workload corresponding to $80 \%$ of the subjects' maximal OXC. Three collections of expired air were made at each step and the mean $H R$ corresponding to each air collection was recorded. The test started at rest with the subject seated in an armchair, while 3 consecutive 5-minute collections of expired air were made. During the following steps the subjects would walk or run on the treadmill. At the $4.8 \mathrm{~km} / \mathrm{hr}$ and $6.8 \mathrm{~km} / \mathrm{hr}$ levels
the subjects exercised for 6 minutes each, and at the other levels they exercised for 3 minutes each; except when they reached the final speed (the one corresponding closest to $80 \%$ of their maximal $0 \times C$ ), they continued the exercise for 20 minutes. During the final stage, one-minute bags of expired air were collected, every second minute starting with the second minute.

Following the exercise the $H R$ and OXC were determined during 40 minutes of recovery. Two one-minute bags, one three-minute and one five-minute bag of expired air were collected during the first 10 minute of recovery. During the next 30 minutes, 3 five-minute bags of expired air were collected every second 5 -minute period. During the alternate 5 -minute periods, when no collections were made, the subject remained seated in the armchair, but was relieved of the face mask with air-collection valve and the noseclip. Arterialized blood samples were collected from one of the fingers at $5,15,25$ and 40 minutes of recovery for determination of the blood lactate concentration.

## Experimental Protocol

## Rest

For measurements taken during rest, the subjects were seated in an armchair. The subjects were asked to restrict their movement, but at the same time, to change the leg position approximately once every minute to avoid venous pooling. The temperature of the laboratory was kept constant $\left( \pm 1^{\circ} \mathrm{C}\right)$ throughout the experiment, but it did vary somewhat between experimental days depending on the environmental
temperature. The humidity and barometric pressure could not be controlled.

The day of the experiment, the subjects arrived in the laboratory about 8:00 am in the postabsorptive state (after overnight fast), and spent the day in the laboratory resting, reading, or talking. No food or beverages except the experimental meal were consumed, but the subjects drank as much water as they wanted. The three treatments were (1) the fat meal, (2) the protein meal, and (3) the carbohydrate meal described previously, and for the control treatment, no food was given. The four treatments were done in a random order one week apart.

Before the experimental meal was given, six five-minute baseline measurements of OXC and HR were taken. The subject was seated in an armchair for three consecutive five-minute periods while expired air was collected and HR recorded. The subject was then given a fiveminute break in which he walked around in the laboratory before another series of three consecutive five-minute collections of expired air and recordings of HR were carried out. For each five minutes, one bag of expired air was collected for analysis of OXC and RQ. The mean HR was calculated for each bag of expired air. The mean of the six determinations was used as the baseline (time 0 ) for that particular treatment (meal). Duplicate arterialized blood samples for lactate determinations were collected before the meals were eaten.

After baseline determination, the subjects ate the experimental meal and three consecutive five-minute collections of expired air and recordings of $H R$ were taken at 30 minutes and at one hour following the completion of the meal and thereafter at one-hour intervals for eight
hours following the meal. The mean of the three measurements was used. During the control treatment, when no meal was given, the protocol was the same, except that no "baseline" measurements were taken. Blood was collected for lactate determination at the same times as the other measurements were taken except for the first hour, when blood samples were taken every 15 minutes. Blood collections were discontinued after 6 hours. During rest lactate was measured only during the control treatment and following the carbohydrate meal.

## Exercise

The exercise experiments were carried out over a 10 -week period with each test given one week apart. During the first and last tests the maximal OXC were determined. The second test was always a control (postabsorptive state) prior to which the subjects ate their normal diet. Prior to the third test, the subjects consumed one of the experimental diets for three days (two started with the high fat diet and three with the high carbohydrate diet). Then followed another control test before the opposite experimental diet was introduced, three days prior to the fifth test. Following both dietary treatments, the test was carried out in the postabsorptive state. The three meals and a final control were introduced in random order so that all the subjects would not consume the meals in the same order. The exercise tests began 30 minutes after termination of the carbohydrate meals and 2-3 hours following the fat and protein meals, which we had found to correspond to the maximal thermogenic response.

## Statistical Analysis

Blood lactate during rest and recovery from exercise following the various treatments was analyzed by a split block (block = subject) repeat measurements design (each subject served as his own control). The differences between treatment means were evaluated by designed contrast (Bonferroni t statistics; Miller, 1966).

## Results

The resting blood lactate in the postabsorptive (after an overnight fast) resting state was about 0.5 mM and decreased slightly with time. Following the carbohydrate meal there was a rapid increase in the blood lactate concentration to a mean value of 2.3 mM , which was significantly different from control for the first 3 hrs. The results are given in Table 3 and plotted in Figure 1.

Regardless of time the postexercise blood lactate level are similar in the postabsorptive state for controls and when the subjects were fed a high fat or a high carbohydrate diet for 3 days. The mean for the 3 conditions was calculated and presented as "D" in Table 4 and plotted in Figure 2. The fat and protein meals give similar values and the mean is presented as "M" in Table 4 and plotted in Figure 2. For $D$ the blood lactate decreases from 3.0 at 5 min of recovery to 0.7 at 40 min of recovery. The values for $M$ - although lower - are not statistically different as shown in Table 5. Following the carbohydrate meal the postexercise blood lactate decreases from 2.6 mM at 5 min to 1.8 mM at 40 min of recovery and is plotted separately in Figure 2. The total blood lactate response following the carbohydrate meal is significantly different from that following the fat meal as shown in Table 5. There is also a significant interaction between the post-exercise blood lactate following the carbohydrate meal and both the control and fat meal at 5 and 40 min of recovery. Thus

Table 3. Blood lactate in arterialized blood for 6 hours following a carbohydrate meal, and in the postabsorptive state following an overnight fast as control. Mean of five subjects.

| Time <br> (hrs) | Control | Carbohydrate <br> Meal | Dunnett's Test* <br> $t_{D}$ |
| :--- | :--- | :--- | :--- |
| 0.00 | 0.51 | 0.59 | 0.37 |
| 0.25 | 0.49 | 1.44 | $4.43^{\mathrm{a}}$ |
| 0.50 | 0.48 | 1.89 | $6.58^{\mathrm{a}}$ |
| 0.75 | 0.49 | 2.21 | $8.03^{\mathrm{a}}$ |
| 1.00 | 0.53 | 2.28 | $8.17^{\mathrm{a}}$ |
| 2.00 | 0.41 | 1.66 | $5.84^{\mathrm{a}}$ |
| 3.00 | 0.40 | 0.93 | $2.47^{\mathrm{b}}$ |
| 4.00 | 0.36 | 0.64 | 1.31 |
| 5.00 | 0.35 | 0.59 | 1.12 |
| 6.00 | 0.34 | 0.52 | 0.84 |

*Significant difference between treatment means of the same time was determined by Dunnett's test (Dunnett, 1955, 1964).
${ }^{\text {a }}$ Significant at $1 \%$ level ( $t=3.36$ ).
${ }^{\mathrm{b}}$ Significant at $5 \%$ level ( $t=2.31$ ).

Standard errors of individual treatment means at one time are irrelevant for comparisons or reliability.

Standard error of difference of two treatments at one time is 0.214 (mM).

Standard error of difference of two times for one treatment is 0.102 (mM).

Figure 1. Blood lactate concentration (mean of five subjects) in the postabsorptive state (following an overnight fast) as control (lower line) and following a 2.94 MJ carbohydrate meal (bananas). The S.E. for difference between the treatment means at the same line is $0.339(\mathrm{mM})$; and the S.E. for difference between the means at two times is $0.102(\mathrm{mM})$.
blood lactate (rest)

(7/ww) efełot poola

Figure 1

Figure 2. Blood lactate concentration during recovery from exercise at $80 \%$ of maximal oxygen consumption for 20 minutes (mean of five subjects). Upper curve following $\mathrm{CH}-\mathrm{M}$; middle curve is the postabsorptive state; and lower curve is mean of $F-M$ and P-M. The S.E. for differences between the treatment means at the same time is 0.483 ( mM ); the S.E. for differences between two times is $1.13(\mathrm{mM})$, and the S.E. for comparing interaction between two treatments at two different times is 0.282 (mM).
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FIGURE 2

Table 4. Mean blood lactate concentration during 40 minutes recovery from 20 minutes of exercise at $80 \%$ of maximal $0 \times C$ in 5 subjects.

| Treatments | Blood Lactate (mM) |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: |
|  | Preexercise | 5 min | 15 min | 25 min | 40 min |
| Control | 0.782 | 2.94 | 1.76 | 1.10 | 0.68 |
| Fat Diet | 0.517 | 2.99 | 1.48 | 1.22 | 0.75 |
| Carbohydrate Diet | 0.782 | 2.99 | 1.33 | 1.03 | 0.69 |
| Fat Meal | 0.543 | 2.21 | 1.03 | 0.65 | 0.45 |
| Proteein Meal | 0.772 | 2.53 | 1:43 | 1.02 | 0.93 |
| CarDohydrate Meal | 0.698 | 2.62 | 1.99 | 1.83 | 1.82 |
| Meam $D^{\text {a }}$ |  | 2.97 | 1.52 | 1.12 | 0.71 |
| Mean $M^{b}$ |  | 2.37 | 1.23 | 0.84 | 0.69 |

$S$ Eandard errors of individual treatment means at one time are i Frelevant for comparisons or reliability.
$S$ tandard error of difference of two treatments at one time is - 520 (mM).
$S$ tandard error of difference of two times for one treatment $50.200(\mathrm{mM})$.

Table 5. Bonferroni $t$ for test of significance of postexercise blood lactate concentration. $\mathrm{CH}-\mathrm{M}$ is tested for interaction (between 5 and 40 minutes) against $C$ and $F-M$. Total (40 minute) response to $\mathrm{CH}-\mathrm{M}$ is tested against total response to $\mathrm{F}-\mathrm{M}$; and the postabsorptive ( P ) trials ( $C, F-D, C H-D$ ) are tested against the absorptive (A) trials ( $F-M$ and $P-M$ ) at 5 minutes postexercise. There are four contrasts ( $m=4$ ).

| Test | Contrasts | $T_{B}$ |
| :---: | :---: | :---: |
| Interaction | $\mathrm{CH}-\mathrm{M}$ vs C at 5 \& 40 min | $\mathrm{a}_{5.17}{ }^{1}$ |
| Interaction | CH-M vs F-M at 5 \& 40 min | $\mathrm{a}_{3.40}{ }^{1}$ |
| Total CH vs Total F | CH-M vs F-M at all times | $\mathrm{a}_{3.70}{ }^{2}$ |
| M vs D | $\begin{gathered} F-M, P-M \text { vs } C, F-D, C H-D \\ \text { at } 5 \text { min } \end{gathered}$ | $1.77^{2}$ |

[^4]the rate of return for the post-exercise blood lactate differ from the other treatments.

## Discussion

Shortly after ingestion of the carbohydrate meal there is a significant increase in the blood lactate during rest compared to control. Although blood glucose was not measured in the present study, Capro, et al (1981) found that plasma glucose and insulin were elevated for approximately four hours following standard meal, and that plasma FFA were depressed for approximately 5 hours. Similar responses to ingestion of 100 g glucose were found for blood glucose, insulin, and FFA by Ravussin, et al (1979) and Luyckx, et al (1978). Owen, et al (1980) found that a typical American breakfast elevated blood glucose, lactate, and pyruvate for about 2 hours, while triglycerides were still elevated at 3 hours. They also found that FFA, glycerol and ketone bodies were depressed for about 3 hours following the breakfast.

The blood lactate following the ingestion of the fat and protein meal was not studied except in a preliminary study in one subject, where no effect was found còmpared to control. However, one would expect a decrease in blood glucose and lactate and an increase in FFA (Seyffert, et al, 1967) if any change occurs, since fat is already the major energy substrate used in the postabsorptive state during rest.

It is controversial whether the oxygen tension in muscle tissue is the primary determinant of lactate production during exercise (Graham, 1978); but Bylund-Fellenius, et al (1981) have recently
presented evidence in support of this concept. However, the individual variation in lactate production is also determined by muscle respiratory capacity and fiber composition (Ivy, et al, 1980), which in turn is modified by training (Johnson, 1969). .

Jacobs (1981) has shown that the level of muscle glycogen affects lactate production during exercise. Thus, at low levels of muscle glycogen, glycogenolysis decreased drastically, but above about 40 $\mathrm{mM} / \mathrm{kg}$ there is no effect. It has been shown in rats (StankiewiczChoroszucha and Gorski, 1978) that when muscle glycogen is not available, muscle triglycerides become the major source of energy. They also reported that the level of blood FFA did not affect intramuscular utilization of triglycerides, but blood FFA level did affect the rate of glycogen utilization. In agreement with these findings, Maughan, et al (1978, 1981) found that a high carbohydrate diet (after glycogen depleting exercise) elevated plasma lactate during and following exercise compared to control, whereas low carbohydrate diet increased plasma FFA under these conditions. These dietary effects were seen both after low and high intensity work. Bonen, et al (1981) also found that a high carbohydrate diet increased blood lactate during high intensity work. Both these authors used a protocol designed to deplete muscle glycogen, and under such conditions, it is clear that diet has an effect upon blood lactate and substrate utilization. Costill, et al (1977) found that artificially elevating FFA (with heparin) following a fat meal increased utilization of fat as a substrate during high intensity work ( $70 \%$ of maximally oxygen consumption) compared to preexercise ingestion of 75 g glucose. Consequently,
the utilization of muscle glycogen was decreased $40 \%$ compared to control. They also found that ingestion of glucose increased glycogen utilization 17\% (partly at the expense of blood carbohydrate). Bergstrom, et al (1969), however, found that decreasing arterial FFA by nicotinic acid did affect substrate utilization, only at low intensity exercise (below about $65 \%$ of maximal oxygen consumption). In the present study, our diets and exercise were designed not to have a significant effect upon muscle glycogen stores, because there was sufficient carbohydrate present in the high fat diet and the exercise was of relatively short duration. In the case of the carbohydrate meal, there clearly was a significant interaction (Table 5), which means that the carbohydrate meal interferes with the normal post-exercise recovery of blood lactate. This is not surprising, in view of the highly significant response in blood lactate to the carbohydrate meal at rest. There are great intra-individual differences in blood lactate: one subject (more fit, as indicated by his maximal oxygen consumption of $66 \mathrm{ml} / \mathrm{min} / \mathrm{kg}$ compared to about 50 for the other subjects) had very little lactate response to exercise. Following the carbohydrate meal lactate response was decreased at five minutes following exercise, but then the blood lactate actually increased to that expected following a carbohydrate meal at rest ( 1.5 mM ). Apparently, either the liver does not produce lactate during exercise, or it is metabolized by the exercising muscle (Poortmans, 1978). These results do not support the hypothesis that diet affects the blood lactate concentration during high, intensity exercise, although a carbohydrate meal clearly affects the rate of decrease in blood
lactate during the recovery period.

CONCLUSIONS

We developed one hundred regression equations between HR and OXC from 10 exercise test in each of 10 subjects. Several aspects of the regression equation between $H R$ and OXC were investigated based on these equations. Inhomogeneity of the regression lines was found both within and between the subjects. Most (about 70\%) of the variability is between subject variability as evaluated by the repeatability $R_{1}$ and $R$ is only slightly decreased by using more homogenous subjects.

It is not valid to use the regression equation between $H R$ and OXC measured during exercise to predict recovery OXC. This can lead to as much as $300 \%$ overestimation of OXC. The reason for this was found to be a lag in the $H R$ recovery relative to the OXC recovery, which occurred during the first 3 min of the recovery period after exercise at $80 \%$ of the maximal OXC. This lag in the HR recovery is presumably caused by a loss of vascular volume, which causes a decrease in stroke volume and a compensatory increase in HR. Duration of exercise rather than the intensity of exercise was found to be primarily responsible for this lag in recovery HR. Repeated exercise was found to be one factor that could cause the regression line to change with respect to slope and $y$-intercept.

Meal feeding compared to the postabsorptive state and a high fat compared to a high carbohydrate diet did not alter the linear rearession equation between the $H R$ and OXC.

There is a need to confirm and expand the present finding in groups of different physical training, which will also allow for comparing the effect of physical training on the linear regression equation between HR and OXC. Furthermore the effects of coffee, smoking and mental stress (such as anxiety) on the regression equation between HR and OXC need to be studied.

The effects of a high fat, a high protein and a high carbohydrate meal (700 kcal) on dietary induced thermogenesis were studied at rest. Each meal differed in its thermogenic response as measured by OXC, both with respect to the maximal response and its duration. The maximal increases in OXC was $8.9 \%, 33.0 \%$ and $27.4 \%$ for the fat, protein and carbohydrate meals respectively. There was also an increase in blood lactate (about 3 mM ) and respiratory quotient following the carbohydrate meal, which indicates a shift in the metabolism. Only during the carbohydrate meal was the increase in OXC positively correlated with the $H R$, giving indirect evidence that the regulatory mechanism of DIT for the 3 meals may be different. There was no thermogenic response during exercise under our experimental procedures.

There is a need for a study of the regulatory mechanisms behind the thermogenic response to the different meals, as the results indicate that they are different. If the thermogenic response to a carbohydrate is regulated differently, it can resolve the controversy about the thermogenic response to the energy substrates and may help explain why some people develop obesity. There is also a need to study the effects of meal size, eating time and composition on habitual food intake on the thermogenic response to fat, protein and carbohydrate
meals. Furthermore, it may be of interest to study the effect of smoking on DIT since smoking cessation can lead to increased body weight. A carbohydrate meal was shown to significantly increase blood lactate concentration compared to control; and when the meal preceeded exercise it resulted in elevated blood lactate levels during recovery ( 2 mM ). However, there was no effect of either meals or diets high in fat or carbohydrate on the substrate utilization or blood lactate concentration during exercise at $80 \%$ of maximal OXC.

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APPENDICES

## Appendix A

Time $=$ duration of expired air collection (sec)
R.Q. = Respiratory Quotient
$\dot{\mathrm{V}} 02=0 \times y g e n$ consumption ( $\mathrm{ml} / \mathrm{min} / \mathrm{kg}$ )
$H R=$ Heart rate
Resting $=$ Collection of expired air samples in the resting state prior to exercise

Exercise $=$ Collection of expired air during the incremental exercise test

Recovery $=$ Collection of expired air during recovery following the exercise test


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[^0]:    ${ }^{\text {a Watt and Merrill (1963); Church \& Church (1975). }}$

[^1]:    a Time refers to midpoint for collection of expired air.
    $\mathrm{b}_{\text {Recovery }}$ heart rate in beats $/ \mathrm{min}$. Resting $H R=60 \pm 6$.
    CRecovery oxygen consumption ( $\mathrm{ml} / \mathrm{min} / \mathrm{kg}$ ). Resting $\mathrm{OXC}=4.2 \pm 0.4$. d95\% lower confidence limit for predicted oxygen consumption.
    ePredicted oxygen consumption during recovery, based on the RHR and the prediction equation for previous exercise.
    $f_{\text {Difference between LCL and ROXC (DLCL). }}$
    GDifference between POXC and ROXC (DOXC).
    $h_{\text {DOXC }}$ as \% of POXC.

[^2]:    ${ }^{\text {a Maximal OXC. }}$

[^3]:    ${ }^{\text {a }}$ Watt and Merrill (1963); Church \& Church (1975).

[^4]:    ${ }^{\text {a }}$ Significant at $5 \%$ level
    ${ }^{1}$ Tabular $T_{B}=2.66$ at $5 \%$ level
    ${ }^{2}$ Tabular $T_{B}=2.74$ at $5 \%$ level

