

Sheffield Hallam University

Oxygen uptake in the frequency domain as a test for cardiorespiratory fitness.

PAGGIOSI, Margaret A.

Available from the Sheffield Hallam University Research Archive (SHURA) at:

<http://shura.shu.ac.uk/20158/>

A Sheffield Hallam University thesis

This thesis is protected by copyright which belongs to the author.

The content must not be changed in any way or sold commercially in any format or medium without the formal permission of the author.

When referring to this work, full bibliographic details including the author, title, awarding institution and date of the thesis must be given.

Please visit <http://shura.shu.ac.uk/20158/> and <http://shura.shu.ac.uk/information.html> for further details about copyright and re-use permissions.

015186371

100369535 3



25421

Sheffield Hallam University
REFERENCE ONLY



Sheffield Hallam University
Learning and IT Services
Adsetts Centre City Campus
Sheffield S1 1WB

ProQuest Number: 10697465

All rights reserved

INFORMATION TO ALL USERS

The quality of this reproduction is dependent upon the quality of the copy submitted.

In the unlikely event that the author did not send a complete manuscript and there are missing pages, these will be noted. Also, if material had to be removed, a note will indicate the deletion.



ProQuest 10697465

Published by ProQuest LLC (2017). Copyright of the Dissertation is held by the Author.

All rights reserved.

This work is protected against unauthorized copying under Title 17, United States Code
Microform Edition © ProQuest LLC.

ProQuest LLC.
789 East Eisenhower Parkway
P.O. Box 1346
Ann Arbor, MI 48106 – 1346

**Oxygen Uptake Kinetics in the Frequency Domain as a
Test for Cardiorespiratory Fitness**

Margaret Anne Paggiosi

A thesis submitted in partial fulfilment of the requirements
of Sheffield Hallam University
for the degree of Doctor of Philosophy

September 1998

ABSTRACT

Oxygen Uptake Kinetics in the Frequency Domain as a Test for Cardiorespiratory Fitness

Margaret A Paggiosi (nee Cooke)

For the degree of Doctor of Philosophy at Sheffield Hallam University

Oxygen uptake kinetics describe the characteristics of the rate of change of $\dot{V}O_2$ in response to the onset of exercise or a change in work rate. There is a lack of knowledge concerning the use of $\dot{V}O_2$ kinetics in the frequency domain as a test for cardiorespiratory fitness. The PRBS exercise test has been developed to study the dynamic responses of the cardiorespiratory system to random changes in submaximal work rate. This exercise test technique provides a multi-frequent assessment of $\dot{V}O_2$ kinetics that can be expressed in terms of amplitude ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$) or phase shift (degrees) over a frequency range of 0.0022 to 0.0089 Hz.

The $\dot{V}O_2$ kinetics of young women were investigated using this submaximal test during which the work rate was alternated between two levels. The upper work rate level was chosen to be below the ventilatory threshold.

In the first experiment, the variability of replicate tests was investigated in a cohort of eight moderately active women (age = 22.6 ± 0.8 years). Although there were wide limits of agreement between the two tests there was no significant difference between test 1 and test 2.

In a second experiment to test the discriminant ability, oxygen uptake kinetics were compared to $\dot{V}O_{2\text{peak}}$ in twenty-eight sedentary or moderately active young women (age = 22.9 ± 3.1 years). The PRBS exercise test technique was able to discriminate between a group of subjects with lower $\dot{V}O_{2\text{peak}}$ ($\dot{V}O_{2\text{peak}} = 32.3 \pm 3.3 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) and a group of subjects with higher $\dot{V}O_{2\text{peak}}$ ($\dot{V}O_{2\text{peak}} = 41.1 \pm 3.2 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$).

Differences in $\dot{V}O_2$ kinetics occurred at frequencies of 0.0022 Hz for amplitude, and at frequencies of 0.0022 Hz to 0.0067 Hz for phase shift. Significant relationships were found to exist between $\dot{V}O_{2\text{peak}}$ and $\dot{V}O_2$ kinetics at frequencies of 0.0022Hz, 0.0044 Hz and 0.0067 Hz. The following model explained the highest proportion of the variation between $\dot{V}O_{2\text{peak}}$ and $\dot{V}O_2$ kinetics ($r = -0.72$, $P < 0.001$):

$$\dot{V}O_{2\text{peak}} \text{ (in ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}\text{)} = 0.503(\text{phase shift at 0.0067 Hz (in degrees)} + 72.24$$

In a third experiment to test the sensitivity to detect change, both $\dot{V}O_2$ kinetics and $\dot{V}O_{2\text{peak}}$ were measured before, during and after an eight week endurance-type training programme completed by fifteen young women (age = 21.6 ± 1.9 years). Thirteen young women (age = 24.3 ± 3.5 years) acted as a non-training control group. Faster $\dot{V}O_2$ kinetics were measured at a frequency of 0.0044 Hz for amplitude and at frequencies of 0.0022Hz to 0.0067 Hz for phase shift following the training programme. Increases in $\dot{V}O_{2\text{peak}}$ also occurred as a result of the exercise regimen. No changes in either $\dot{V}O_2$ kinetics or $\dot{V}O_{2\text{peak}}$ were observed in the non-training group. This study showed that the PRBS exercise test technique was sensitive to short-term endurance-type training adaptations.

In conclusion, the parameters measured during the PRBS exercise test provide valuable information that can not be gained from a standard assessment of $\dot{V}O_2$ kinetics in the time domain. It is proposed that this exercise test technique has potential as a means of assessing cardiorespiratory fitness within the area of sports science and within the clinical environment.

**PART OF THE RESULTS REPORTED HERE WERE PUBLISHED
IN THE FOLLOWING:**

FYSH M.L., CHAPMAN J.H., COOKE M.A., CLAXTON D.B., AND JARVIS D.R. (1996). Sensitivity of a pseudo random binary sequence exercise test to detect training induced adaptations in young, female subjects. In: First Annual Congress, Frontiers in Sports Science, the European Perspective: Book of Abstracts. Marconnet P., Gaulard J, Margaritus I and Tessier F (eds). European College of Sports Science. May 28-31, Nice, France. pp592-593.

**OTHER PUBLISHED WORK RELEVANT TO THIS THESIS
INCLUDES:**

COOKE M.A., CHAPMAN J.H. AND FYSH M.L. (1995). The validity of graded exercise testing as a means of prescribing intermittent activity performed during Phase II (early-outpatient) cardiac rehabilitation programmes. *Journal of Sports Sciences* 13; 5: 418

COOKE M.A., CHAPMAN J.H., AND FYSH M.L. (1995). Exercise prescription for the cardiac patient. In: Atkinson G and Reilly T (eds). *Sport, Leisure and Ergonomics*. E and F N Spon: London. pp287-292.

CLAXTON D. B., CHAPMAN J. H., COOKE M. A., FYSH M. L., AND JARVIS D. R. (1996). Reliability of the pseudo random binary sequence technique to measure oxygen uptake kinetics. In: First Annual Congress, Frontiers in Sports Science, the European Perspective: Book of Abstracts. Marconnet P., Gaulard J, Margaritus I and Tessier F (eds). European College of Sports Science. May 28-31, Nice, France. pp510-511.

ACKNOWLEDGEMENTS

I am grateful to the following people for their assistance:

- The Health Research Institute (Sheffield Hallam University) for providing financial support to investigate exercise testing and training in relation to cardiac rehabilitation exercise programmes. I am especially grateful to the Health Research Institute for providing the funding which enabled me to participate in a study visit to the Department of Kinesiology, University of Waterloo, Ontario, Canada.
- Dr RL Hughson for his kind invitation to participate in a study visit to the Department of Kinesiology, University of Waterloo, Ontario, Canada. I would like to acknowledge RL Hughson for his enthusiasm, which helped to initiate this study, and for supplying the software with which to analyse the data collected during the PRBS exercise test, and also for providing laboratory space within which a number of preliminary experiments were conducted. I would like to thank RL Hughson and D Northey (Department of Kinesiology, University of Waterloo, Ontario, Canada) for providing tuition time during which I received instruction concerning PRBS exercise testing and respiratory mass spectrometer methodology. I am especially grateful to Ms S Tuck for the hospitality shown during my study visit.
- Dr M Lloyd for his kind invitation to visit the Department of Cardiology, Stoke City General Hospital, North Staffordshire during which I observed stress testing procedures and gained knowledge concerning cardiac rehabilitation exercise prescription methods.
- Dr S Mawson for negotiating a study visit to view cardiac rehabilitation exercise sessions held by the Physiotherapy Department at the Northern General Hospital, Sheffield.
- Mr DB Claxton (School of Leisure and Food Management, Sheffield Hallam University) and Mrs AM Flowers (School of Science, Sheffield Hallam University) for their technical assistance and invaluable support.
- Mrs M Rangecroft (School of Computing and Management Science, Sheffield Hallam University) for statistical advice.
- Ms J Sixsmith and Mr P Mulliner (Marquette Electronics (GB) Ltd., Manchester) for technical advice. I am especially grateful to Ms J Sixsmith for her help in negotiating the study visits to the Department of Kinesiology, University of Waterloo, Ontario, Canada and the Department of Cardiology, Stoke City General Hospital, North Staffordshire.
- The students at Sheffield Hallam University who volunteered to participate in these studies.
- I am especially grateful to my supervisors Dr JH Chapman (School of Leisure and Food Management, Sheffield Hallam University), Dr ML Fysh (School of Leisure and Food Management, Sheffield Hallam University) and Dr ME Blair (School of Science, Sheffield Hallam University) for their continual advice and support.
- Finally, thank you to my husband Vin, my parents Janet and Derek, and the rest of my family for being there when I needed you most.

TABLE OF CONTENTS

CHAPTER ONE : INTRODUCTION	1
1.1) INTRODUCTION	2
1.2) PHYSIOLOGICAL BASIS FOR OXYGEN UPTAKE KINETICS	3
1.2.1) Cardiovascular and cardiorespiratory responses to the onset of exercise	3
1.2.1.1) Neural response	3
1.2.1.2) Mechanical response	5
1.2.1.3) Metabolic response	5
1.2.1.4) Oxygen utilisation by the muscle	6
1.2.2) The oxygen uptake response at the onset of exercise.....	6
1.2.2.1) Phase I.....	7
1.2.2.2) Phase II	8
1.2.2.3) Phase III.....	9
1.3) MEASUREMENT OF OXYGEN UPTAKE KINETICS	9
1.3.1) Square wave work rate forcings	11
1.3.2) Sinusoidal work rate forcings.....	15
1.3.3) Pseudo random work rate forcings	20
1.4) THE INFLUENCE OF RATE LIMITING FACTORS ON PHASE II OXYGEN UPTAKE KINETICS	27
1.4.1) Acute alterations of the oxygen delivery process.....	27
1.4.1.1) β -adrenergic receptor blockade	27
1.4.1.2) Hypoxia and hyperoxia.....	28
1.4.1.3) Circulatory occlusion.....	29
1.4.1.4) Supine and upright exercise.....	29
1.4.1.5) Lower body negative and positive pressure	30
1.4.1.6) Summary.....	31
1.4.2) Identification of the physiological factor with the same response characteristics as the oxygen uptake response	31
1.4.2.1) Heart rate and cardiac output kinetics	31
1.4.2.2) Blood flow kinetics.....	32
1.4.2.3) Phosphocreatine kinetics	33
1.4.2.4) Summary.....	36
1.4.3) Conclusion.....	36
1.5) APPLICATION OF PRBS EXERCISE TESTING.....	36
1.6) AIMS AND OBJECTIVES.....	39
CHAPTER TWO : MATERIALS AND METHODS	40
2.1) INTRODUCTION	41
2.2) EQUIPMENT	41
2.2.1) Respiratory measurements.....	41
2.2.1.1) Equipment description.....	41
2.2.1.2) Calibration procedures.....	43
2.2.1.3) Breath-by-breath calculation of oxygen uptake.....	44
2.2.2) Heart rate measurements	47
2.2.2.1) Electrocardiography.....	47
2.2.3) Ergometry	48
2.2.3.1) Equipment description.....	48

2.2.3.2) Calibration procedure	48
2.2.4) Anthropometry	49
2.3) EXERCISE PROTOCOLS	49
2.3.1) PRBS exercise test.....	49
2.3.2) Progressive exercise test to exhaustion	51
2.4) DATA ANALYSIS	53
2.4.1) Heart rate data.....	53
2.4.1.1) PRBS exercise test.....	53
2.4.1.2) Progressive exercise test to exhaustion	53
2.4.2) Oxygen uptake data	53
2.4.2.1) Analysis of PRBS data	54
2.4.2.1.1) Frequency domain analysis	54
2.4.2.2) Analysis of data from the progressive exercise test	55
2.5) RECRUITMENT AND MONITORING METHODS	56
2.5.1) Health and activity questionnaires.....	56
2.5.2) Consent forms.....	57
2.5.3) Assessment of habitual activity levels.....	57
2.5.4) Monitoring of activity levels during the eight week training study	59

CHAPTER THREE : THE VARIABILITY OF THE RESPIRATORY RESPONSES TO THE PRBS EXERCISE TEST 61

3.1) INTRODUCTION	62
3.2) METHODOLOGY	67
3.2.1) Subjects.....	67
3.2.2) Exercise testing.....	68
3.2.3) Data and statistical analysis.....	68
3.3) RESULTS	70
3.3.1) Overall results.....	70
3.3.2) Oxygen uptake kinetics	72
3.3.2.1) Descriptive statistics.....	72
3.3.2.2) Two-way analysis of variance	73
3.3.2.3) Limits of agreement.....	74
3.4) DISCUSSION.....	80
3.4.1) Overall response	80
3.4.2) Oxygen uptake kinetics comparisons	80
3.4.3) Variability.....	80
3.5) CONCLUSIONS	83

CHAPTER FOUR : THE RELATIONSHIP BETWEEN OXYGEN UPTAKE KINETICS AND AEROBIC POWER 84

4.1) INTRODUCTION	85
4.2) METHODOLOGY	89
4.2.1) Subjects.....	89
4.2.2) Exercise testing.....	90
4.3) DATA AND STATISTICAL ANALYSIS.....	90
4.3.1) Attainment of maximal oxygen uptake or peak oxygen uptake	90
4.3.2) Descriptive statistics.....	90
4.3.3) Oxygen uptake kinetics in subjects differing in aerobic power	91
4.3.4) Relationship between oxygen uptake kinetics and aerobic power	91

4.4) RESULTS	92
4.4.1) Progressive exercise test.....	92
4.4.2) Oxygen uptake kinetics	93
4.4.2.1) Mean response	93
4.4.2.2) Frequency domain analysis	93
4.4.3) Oxygen uptake kinetics in subjects differing in aerobic power	94
4.4.3.1) Amplitude	95
4.4.3.2) Phase shift.....	97
4.4.4) Relationship between oxygen uptake kinetics and peak oxygen uptake.....	98
4.4.4.1) Amplitude	98
4.4.4.2) Phase shift.....	100
4.5) DISCUSSION.....	103
4.5.1) Progressive exercise test.....	103
4.5.2) Oxygen uptake kinetics in the two sub-groups with high and low aerobic power	104
4.5.3) The relationship between oxygen uptake kinetics and aerobic power	106
4.5.4) Physiological mechanisms.....	107
4.6) CONCLUSIONS	111

CHAPTER FIVE : THE EFFECT OF ENDURANCE-TYPE TRAINING ON THE OXYGEN UPTAKE KINETICS OF YOUNG WOMEN..... 112

5.1) INTRODUCTION.....	113
5.2) METHODOLOGY.....	117
5.2.1) Recruitment	117
5.2.2) Group assignment.....	118
5.2.3) Training sub-group methodology	118
5.2.3.1) Subjects.....	118
5.2.3.2) Exercise testing.....	119
5.2.3.3) Exercise training.....	119
5.2.3.4) Data analysis.....	120
5.2.3.5) Statistical analysis	121
5.2.3.5.1) Training work rate	121
5.2.3.5.2) Training effects.....	121
5.2.4) Non- training sub-group methodology	122
5.2.4.1) Subjects.....	122
5.2.4.2) Exercise testing.....	122
5.2.4.3) Activity logs	123
5.2.4.4) Data analysis.....	123
5.2.4.5) Statistical analysis	123
5.2.4.5.1) Activity logs	123
5.2.4.5.2) Assessment of short term variation	124
5.3) RESULTS.....	124
5.3.1) Training group	124
5.3.1.1) Training	124
5.3.1.1.1) Adherence.....	124
5.3.1.1.2) Training work rate	125
5.3.1.2) Progressive exercise testing.....	126
5.3.1.2.1) Training adaptations detected from peak responses.....	128
5.3.1.3) PRBS exercise test.....	131
5.3.1.3.1) Dynamic gain and overall responses	131

5.3.1.3.2 Oxygen uptake kinetics.....	131
5.3.1.3.2.1) Training adaptations in oxygen uptake kinetics	132
5.3.2) Non-training group	138
5.3.2.1) Activity logs	138
5.3.2.2) Progressive exercise testing.....	138
5.3.2.2.1) Short term variation in peak responses.....	140
5.3.2.3) PRBS exercise test.....	140
5.3.2.3.1 Dynamic gain and overall responses	140
5.3.2.3.2) Oxygen uptake kinetics	140
5.3.2.3.2.1) Short-term variation in oxygen uptake kinetics.....	141
5.4) DISCUSSION.....	142
5.4.1) Adherence.....	143
5.4.2) Training work rate	143
5.4.3) Training adaptations	144
5.4.3.1) Progressive exercise test.....	144
5.4.3.2) Oxygen uptake kinetics	146
5.4.4) Physiological mechanisms.....	148
5.4.4.1) Time course and extent of training adaptations in aerobic power.....	148
5.4.4.2) Time course and extent of adaptations in oxygen uptake kinetics	149
5.4.4.3) Comparison of the time courses of aerobic power and oxygen uptake kinetics	150
5.4.4.4) Mechanisms of training adaptations due to endurance exercise.....	150
5.5) CONSTRAINTS IMPOSED BY THE STUDY DESIGN.....	152
5.6) IMPLICATIONS OF THE VARIABILITY OF THE RESPIRATORY RESPONSES TO THE PRBS EXERCISE TEST	153
5.7) CONCLUSIONS	157

CHAPTER SIX : FUTURE DIRECTIONS 158

6.1) INTRODUCTION.....	159
6.2) POTENTIAL APPLICATIONS OF THE PRBS EXERCISE TEST	159
6.2.1) Assessment of the effects of different training models	159
6.2.2) Evaluation of the effect of different athletic status	159
6.2.3) Clinical application.....	160
6.3) MODIFICATION OF THE PRBS EXERCISE TEST	162

REFERENCES 166

APPENDICES A-195

A.1) APPENDIX 1 - PUBLICATIONS.....	A-196
A 1.1) The validity of graded exercise testing as a means of prescribing intermittent activity performed during Phase II (early-outpatient) cardiac rehabilitation programmes.	A-196
A.1.2) Exercise prescription for the cardiac patient.	A-198
A.1.3) Sensitivity of a pseudo random binary sequence exercise test to detect training induced adaptations in young, female subjects.	A-207
A.1.4). Reliability of the peusdo random binary sequence technique to measure oxygen uptake kinetics.....	A-211
A.2) APPENDIX 2 - ENVIRONMENTAL CONDITIONS.....	A-215

A.3) APPENDIX 3 - RECRUITMENT MATERIAL.....	A-216
A.3.1) Health and activity questionnaire (variability)	A-216
A.3.2) Consent form (variability)	A-220
A.3.3) Health and activity questionnaire (training and non-training).....	A-222
A.3.4) Consent form (training group).....	A-226
A.3.5) Consent form (non-training group).....	A-229
A.3.6) A weekly activity log as completed by each subject in the non-training sub- group.	A-232
A.4) APPENDIX 4 - CRITERIA FULFILLED DURING THE PROGRESSIVE EXERCISE TEST TO EXHAUSTION.	A-233
A.4.1) The criteria fulfilled by the total subject cohort (n = 28) during the progressive exercise test to exhaustion when investigating the relationship between peak oxygen uptake and oxygen uptake kinetics.....	A-233
A.4.2) Criteria fulfilled by training subjects during the progressive exercise test to exhaustion, when investigating the effect of endurance-type training on the oxygen uptake kinetics of healthy, young females.....	A-234
A.4.3) Criteria fulfilled by the non-training subjects during the progressive exercise test to exhaustion, when investigating the effect of endurance-type training on the oxygen uptake kinetics of healthy, young females.....	A-235
A.5) APPENDIX 5 - INDIVIDUAL SHORT-TERM TRAINING ADAPTATIONS.	A-236

LIST OF FIGURES

	PAGE
1.1) The $\dot{V}O_2$ response at the onset of exercise, adapted from Barstow and Mole (1991), and Hamar (1991)._____	7
1.2) Square wave work rate forcings, described by Yoshida et al. (1993) with work rate transitions between rest and (A) 100 W, (B) 75 W and (C) 50 W._____	13
1.3) A sinusoidal work rate forcing described by Casaburi et al. (1977)._____	17
1.4) A single sequence from a PRBS work rate forcing described by Eßfeld et al. (1987)._____	22
1.5) A single sequence from a PRBS work rate forcing described by Hughson et al. (1990a)._____	22
1.6) A simplified schematic diagram of the reactions by which O_2 consumption is coupled to extramitochondrial ATP hydrolysis in muscle. Taken from Mahler (1985)._____	35
2.1) A schematic representation of the mouthpiece, saliva trap, volume turbine and digital volume transducer._____	42
2.2) Diagram of the sites used for lead attachment and placement of the four electrodes._____	48
2.3) The fifteen unit PRBS exercise test protocol with work rate changes between 25 and 75 W._____	50
2.4) The progressive cycle ergometer exercise test protocol involving ramp work rate increase of $20 \text{ W}\cdot\text{min}^{-1}$ to exhaustion._____	52
3.1) The PRBS exercise test; (A) work rate sequences, (B) a typical heart rate response and (C) a typical $\dot{V}O_2$ response._____	71
3.2) Mean values (mean \pm SD) for amplitude ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$) obtained during replicate PRBS exercise tests._____	72
3.3) Mean values (mean \pm SD) for phase shift obtained during replicate PRBS exercise tests._____	73
3.4) The difference between replicate measures of amplitude (t_2-t_1) (y-axis) and mean results of the two tests (x-axis) for each subject \blacklozenge at harmonic numbers 1 and 2._____	75
3.5) The difference between replicate measures of amplitude (t_2-t_1) (y-axis) and mean results of the two tests (x-axis) for each subject \blacklozenge at harmonic numbers 3 and 4._____	76
3.6) The difference between replicate measures of phase shift (t_2-t_1) (y-axis) and mean results of the two tests (x-axis) for each subject \blacklozenge at harmonic numbers 1 and 2._____	78
3.7) The difference between replicate measures of phase shift (t_2-t_1) (y-axis) and mean results of the two tests (x-axis) for each subject \blacklozenge at harmonic numbers 3 and 4._____	79
4.1) Amplitude ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$), at harmonic numbers 1 to 4, for the two study sub-groups categorised according to relative $\dot{V}O_{2\text{peak}}$ (mean \pm SD)._____	96
4.2) Phase shift (degrees) for the two study sub-groups categorised according to relative $\dot{V}O_{2\text{peak}}$ (mean \pm SD)._____	98
4.3) Linear regression equation for the relationship between relative $\dot{V}O_{2\text{peak}}$ and amplitude at harmonic number 2._____	100
4.4) Linear regression equations for the relationships between relative $\dot{V}O_{2\text{peak}}$ and phase shift at harmonic numbers 1, 2 and 3._____	102

5.1)	A box and whisker plot showing the training work rate (W) used to elicit a 90% maximal heart rate exercise prescription at weeks 0, 4 and 8 for the training study sub-group.	125
5.2)	Box and whisker plots showing (A) relative $\dot{V}O_{2peak}$ ($ml \cdot kg^{-1} \cdot min^{-1}$), (B) absolute $\dot{V}O_{2peak}$ ($ml \cdot min^{-1}$) and (C) peak work rate (W) at weeks 0, 4 and 8 for the training study sub-group.	130
5.3)	A box and whisker plot showing amplitude at harmonic number 2 ($ml \cdot min^{-1} \cdot W^{-1}$) at weeks 0, 4 and 8 for the training study sub-group	133
5.4)	Box and whisker plots showing (A) phase shift at harmonic number 1 (degrees), (B) phase shift at harmonic number 2 (degrees), and (C) phase shift at harmonic number 3 (degrees), at weeks 0, 4 and 8 for the training study sub-group.	135
5.5)	Training adaptations in (A) amplitude ($ml \cdot min^{-1} \cdot W^{-1}$) and (B) phase shift (degrees) resulting from the eight week cycle ergometry exercise programme for the training study sub-group.	136
5.6)	Changes (%) in $\dot{V}O_{2peak}$ and $\dot{V}O_2$ kinetics between (A) weeks 0 and 4, (B) weeks 4 and 8, and (C) week 0 and 8.	137
5.7)	Short-term variation in (A) amplitude ($ml \cdot min^{-1} \cdot W^{-1}$) and (B) phase shift (degrees) before (week 0) and after (week 8) the study period for the non-training study sub-group.	142
6.1)	A graphical representation of the modified PRBS exercise test protocol consisting of 20 s work rate changes between upper and lower work rates of 50 and 25 W respectively.	164

LIST OF TABLES

	PAGE
1.1) Amplitude ($\text{ml}\cdot\text{min}^{-1}$) and phase shift (degrees) at different frequencies of work rate change in young and elderly women taken from Cunningham et al. (1993).	19
1.2) Amplitude values ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$) at harmonic numbers 1 to 4, determined by the PRBS exercise test technique, and categorised according to relative $\dot{V}O_{2\text{max}}$ ($\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) (mean \pm SD).	38
1.3) Phase shift values (degrees) at harmonic numbers 1 to 4, determined by the PRBS exercise test technique, and categorised according to relative $\dot{V}O_{2\text{max}}$ ($\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) (mean \pm SD).	38
2.1) The characteristics (frequency in Hz and period in s) of the first four harmonics.	55
2.2) The six point activity scale used to determine the habitual activity levels of each subject.	58
2.3) The six point activity scale used to determine the activity levels of each subject in the non-training sub-group.	60
3.1) Descriptive statistics for amplitude ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$) determined during replicate PRBS exercise tests (mean \pm SD) ($n = 8$).	72
3.2) Descriptive statistics for phase shift (degrees) determined during replicate PRBS exercise tests (mean \pm SD) ($n = 8$).	73
3.3) Mean difference (t_2-t_1) $\pm 2S_{\text{diff}}$ and 95% limits of agreement for amplitude ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$).	74
3.4) Mean difference (t_2-t_1) $\pm 2S_{\text{diff}}$ and 95% limits of agreement for phase shift (degrees).	77
4.1) The $\dot{V}O_{2\text{max}}$, and $\dot{V}O_2$ kinetics of the male subjects categorised according to age (Babcock et al., 1994b) (mean \pm SD).	88
4.2) The cardiorespiratory values at exhaustion (mean \pm SD) ($n = 28$).	92
4.3) $\dot{V}O_2$ kinetics, amplitude ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$) and phase shift (degrees), at the first four harmonic numbers (mean \pm SD) ($n = 28$).	93
4.4) The characteristics of the two sub-groups of subjects categorised according to relative $\dot{V}O_{2\text{peak}}$ of below (group I) or above $37.0 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ (group II) (mean \pm SD) ($n = 28$).	94
4.5) Amplitude ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$) at harmonic numbers 1 to 4, and categorised according to relative $\dot{V}O_{2\text{peak}}$ of below (group I) or above $37.0 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ (group II) (mean \pm SD) ($n = 28$).	95
4.6) Phase shift (degrees) at harmonic numbers 1 to 4, and categorised according to relative $\dot{V}O_{2\text{peak}}$ of below (group I) or above $37.0 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ (group II) (mean \pm SD) ($n = 28$).	97
4.7) Correlation coefficients (r values) for the relationships between amplitude ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$) at harmonic numbers 1 to 4 and (I) relative $\dot{V}O_{2\text{peak}}$ ($\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$), and (II) absolute $\dot{V}O_{2\text{peak}}$ ($\text{ml}\cdot\text{min}^{-1}$).	99
4.8) Correlation coefficients (r values) for the relationships between phase shift (degrees) at harmonic numbers 1 to 4 and (I) relative $\dot{V}O_{2\text{peak}}$ ($\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$), and (II) absolute $\dot{V}O_{2\text{peak}}$ ($\text{ml}\cdot\text{min}^{-1}$).	101
5.1) Adaptations in aerobic fitness and $\dot{V}O_2$ kinetics following endurance training programmes.	114

5.2)	Progressive exercise test results at weeks 0, 4 and 8 of the exercise training programme for the training study sub-group (mean \pm SD) (n = 15).	127
5.3)	$\dot{V}O_2$ kinetics at weeks 0, 4 and 8 of the exercise training programme for the training study sub-group (mean \pm SD) (n = 15).	132
5.4)	Progressive exercise test results at weeks 0, 4 and 8 of the study period for the non-training study sub-group (mean \pm SD) (n = 13).	139
5.5)	$\dot{V}O_2$ kinetics determined during the PRBS exercise tests at weeks 0, 4 and 8 for the non-training study sub-group (mean \pm SD) (n = 13).	141
5.6)	Standard deviation (SD) of the replicate PRBS exercise tests, minimal detectable changes and actual changes in amplitude ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$) at harmonic numbers 1 to 4 following the 8 week training programme.	156
5.7)	Standard deviation (SD) of the replicate PRBS exercise tests, minimal detectable changes and actual changes in phase shift (degrees) at harmonic numbers 1 to 4 following the 8 week training programme.	156

LIST OF ABBREVIATIONS

ABBREVIATION	TERM
τ	Time constant
APMHR	Age predicted maximal heart rate
(a-v)O ₂ difference	Arterio-venous oxygen difference
BTPS	Body temperature and pressure, saturated with water vapour
dFN ₂	Change in fractional concentration of nitrogen
dFO ₂	Change in fractional concentration of oxygen
dVL	Change in lung volume
fb	Breathing frequency
FN ₂	Fractional concentration of nitrogen
FO ₂	Fractional concentration of oxygen
FpN ₂	Fractional concentration of nitrogen in previous breath
FpO ₂	End tidal fractional concentration of oxygen in previous breath
FRC	Functional residual capacity
METs	Metabolic equivalents
LBNP	Lower body negative pressure
LBPP	Lower body positive pressure
NLV	Nominal lung volume
OBLA	Onset of blood lactate
PCr	Phosphocreatine
PCr/Cr	Phosphocreatine to creatine ratio
PO ₂	Partial pressure of oxygen
PRBS	Pseudo random binary sequence
\dot{Q}	Cardiac output
\dot{Q}_{\max}	Maximal cardiac output
RER	Respiratory exchange ratio
STPD	Standard temperature and pressure, dry
t _{1/2}	Half time
VEN ₂	Volume of expired nitrogen
VEO ₂	Volume of expired oxygen
V _{ET}	Ventilatory threshold
\dot{V}_I	Inspiratory rate
VIN ₂	Volume of inspired nitrogen
VIO ₂	Volume of inspired oxygen
VN ₂	Volume of nitrogen
$\dot{V} \text{ CO}_2$	Rate of carbon dioxide output
\dot{V}_E	Minute ventilation
$\dot{V} \text{ O}_2$	Oxygen uptake
$\dot{V} \text{ O}_{2\max}$	Maximal oxygen uptake
$\dot{V} \text{ O}_{2\text{peak}}$	Peak oxygen uptake



GLOSSARY OF TERMS

This glossary of terms has been compiled using Wasserman et al. (1994), Kerlin (1974), Beaver et al. (1986), Eßfeld et al. (1987), Hughson (1990), Hughson et al. (1990a), Hughson et al. (1990b) and Hughson et al. (1993).

TERM	DESCRIPTION
Aerobic:	Having molecular oxygen present; describes any metabolic process utilising oxygen.
Adherence:	Used to describe a person's continuation in an exercise programme.
Aerobic power:	The amount of oxygen extracted from the inspired gas in a given period of time, expressed in millilitres or litres per minute. For the purpose of this thesis aerobic power is used synonymously with maximal oxygen uptake and peak oxygen uptake (c.f. maximal oxygen uptake and peak oxygen uptake).
Amplitude:	Amplitude, measured in $\text{ml} \cdot \text{min}^{-1} \cdot \text{W}^{-1}$, describes the magnitude of the $\dot{V}O_2$ response in relation to the work rate applied.
Anaerobic:	Lacking or inadequate molecular oxygen; describes any metabolic process that does not use molecular oxygen.
Analogue to digital (A/D) converter:	A device for transforming continuously changing information into discrete units over some small time frame, within which the value is considered to be relatively constant. This transforms continuous signals to a form that can be analysed by a digital computer.
Arteriovenous oxygen content difference ($(a-\bar{v}) O_2$ difference):	The difference in the oxygen content of the arterial and venous blood, usually expressed in millilitres of oxygen per decilitre or litre of blood.
Body temperature and pressure, saturated with water vapour (BTPS):	Gas volume conditioned to body temperature and the ambient atmospheric pressure and fully saturated with water vapour at the subject's body temperature. These conditions are 310 K, ambient pressure and saturated water vapour at a partial pressure 47 mmHg which is the vapour pressure of water at a body temperature of 37 °C.
Breath-by-breath:	Describes a method for the measurement of respiratory gas exchange in which respired gas volume and simultaneously measured expired gas concentration are integrated and reported.

Carbon dioxide output ($\dot{V}CO_2$):	The amount of carbon dioxide exhaled from the body into the atmosphere per unit time, expressed in millilitres or litres per minute, (standard temperature and pressure).
Cardiac output (\dot{Q}):	The flow of blood from the heart in a particular period of time, usually expressed as litres per minute. It is the product of the average stroke volume per beat and the heart rate.
Dynamic gain:	This represents the overall oxygen uptake response over the duration of the pseudo random binary sequence and is expressed in terms of $ml \cdot min^{-1}$.
Ergometer:	A device that can measure work consistently and reliably. Examples include the cycle ergometer and the treadmill.
Exercise capacity:	The potential to do work. In this thesis exercise capacity is used synonymously with maximal oxygen uptake and peak oxygen uptake (c.f. maximal oxygen uptake and peak oxygen uptake) in the field of cardiac rehabilitation.
Exponential:	A process in which the instantaneous rate of change of a variable is proportional to the 'distance' from steady state or required level. If the process is known to be, or may be exponential, the time to reach 63% of the final value, is termed the time constant (τ) of the response (c.f. time constant). This time constant is related to the half time (c.f. half time) by the following equation: $t_{1/2} = 0.693 \times \tau$
Fourier analysis:	Fourier analysis may be used to convert functions of time into functions of frequency. The frequency response characterises the input-output relationship in the frequency domain, i.e. where the input is work rate and the output is oxygen uptake. Measures of oxygen uptake kinetics are produced.
Half time ($t_{1/2}$):	The half time is a simple description of the time to reach half of the change to the final value, regardless of the function of the process. This is expressed in terms of seconds.
Intensity:	The rate of performing work. A function of energy output per unit time.
Mass spectrometer:	A device that separates and measures molecules of gas of a particular type, in a mixing stream, on the basis of their charge to mass ratio.

Maximal oxygen uptake ($\dot{V}O_{2\max}$):	The highest oxygen uptake obtainable for a given form of ergometry despite further work rate increases and effort by the subject. This is characterised by a plateau of oxygen uptake despite further increases in work rate (c.f. aerobic power).
Oxygen deficit:	The oxygen equivalent of the total energy utilised to perform the work that did not derive from reactions utilising atmospheric oxygen taken into the body after the start of exercise. For moderate intensity exercise, this oxygen deficit represents the energy equivalent of the depletion of the high-energy phosphate stores and oxygen stored in the body at the start of exercise. For heavy exercise, the oxygen deficit includes the energy equivalent of the anaerobic processes.
Oxygen delivery:	The amount of oxygen delivered to a tissue per unit time. It is the product of the oxygen content of the arterial blood and the blood flow to that tissue.
Oxygen uptake ($\dot{V}O_2$):	The amount of oxygen extracted from the inspired gas in a given period of time, expressed in millilitres or litres per minute.
Oxygen uptake kinetics:	Oxygen uptake kinetics describe the characteristics of the rate of change of oxygen uptake in response to the onset of exercise or a change in work rate. In the time domain they can be described in terms of half time or time constant (c.f. half time and time constant). In the frequency domain they can be described in terms of amplitude and phase shift (c.f. amplitude and phase shift).
Peak oxygen uptake ($\dot{V}O_{2\text{peak}}$):	The highest oxygen uptake obtainable for a given form of ergometry despite further work rate increases and effort by the subject when a plateau in the relationship between oxygen uptake and work rate is not seen (c.f. aerobic power). For the purpose of this thesis, peak oxygen uptake has been characterised using the following criteria: <ul style="list-style-type: none"> 1) a respiratory exchange ratio (RER) of 1.15 or above was apparent, or; 2) observed heart rate was within 10 $\text{beats}\cdot\text{min}^{-1}$ of age-predicted maximal heart rate (APMHR), calculated as 220 minus age.
Phase shift:	Phase shift, measured in degrees, represents the systems delay between the onset of exercise, or a change in work rate, and the increase in oxygen uptake.
Progressive exercise test:	An exercise test during which gradually increasing

stresses are placed on the cardiorespiratory system of the subject. The work rate is increased over uniform periods of time (step) or continuously (ramp).

Pseudorandom binary sequence (PRBS) exercise test:

During this exercise test, the input work rate is switched between an upper and a lower work rate level over a pre-defined period of time according to the output of a computer algorithm known as a shift register. The length of each sequence is determined by the number of work rate switches used (N) and the way they are connected ($2^N - 1$). This results in a multi-frequent assessment of oxygen uptake kinetics. The pattern of response is such that, as the frequency of the input work rate forcing is increased, the oxygen uptake output response is characterised by slower oxygen uptake kinetics. Oxygen uptake kinetics are given in the frequency domain as amplitude and phase shift (c.f. amplitude and phase shift).

Respiratory exchange ratio (RER):

The ratio of the carbon dioxide output to the oxygen uptake per unit time. This reflects tissue metabolic exchange of gases and the influence of transient changes in gas storage of oxygen and carbon dioxide.

Standard temperature and pressure, dry (STPD):

Gas volume at standard conditions of temperature and pressure, free of water vapour. The standard conditions are 0°C, 76 mmHg and dry gas.

Steady state:

This is a characteristic of a physiological system in which its functional demands are being met such that its output per unit time becomes constant.

Submaximal:

Less than maximum. Submaximal exercise requires less than maximal oxygen uptake, heart rate or anaerobic power.

Time constant (τ):

This is the time required to reach 63% of the increase in oxygen uptake from baseline, during either rest or unloaded cycling, to steady state. This is expressed in seconds.

Transducer:

A device that transforms energy from one form to another.

V-slope method:

A technique that allows the detection of the onset of lactic acidosis (ventilatory threshold) during a progressive exercise test when an accelerated rate of carbon dioxide output is generated from bicarbonate buffering of lactic acid. When plotting $\dot{V}CO_2$ against $\dot{V}O_2$, the point at which the line inflects upwards is said

to be the ventilatory threshold. This has shown to give similar results to the Ventilatory Equivalent method, which involves plotting the ratio of ventilation rate to the rate of oxygen uptake ($\dot{V}E \cdot \dot{V}O_2^{-1}$) against work rate, the ventilatory threshold is located where the curve inflects upwards, having been flat or decreasing. An upwards inflection of the curve for the ratio of ventilation rate to the rate of carbon dioxide production ($\dot{V}E \cdot \dot{V}CO_2^{-1}$) against work rate does not occur until higher work rates are reached and reflects the start of respiratory compensation for the metabolic acidosis (c.f. ventilatory threshold).

Ventilatory threshold (V_{ET}): This is defined as the level of exercise oxygen uptake above which aerobic energy production is supplemented by anaerobic mechanisms and is reflected by an increase in lactate, and the lactate/pyruvate ratio in muscle or arterial blood. The V-slope method and the Ventilatory Equivalent method can be used to determine the ventilatory threshold (c.f. V-slope method).

Work rate: This reflects the rate at which work is performed, i.e. work per unit time. Work rate is usually measured in watts (W) or kilogram metres per minute ($kg \cdot m \cdot min^{-1}$), where $1 W = 6.1 kg \cdot m \cdot min^{-1}$.

CHAPTER ONE

INTRODUCTION

1.1) Introduction

Oxygen uptake kinetics describe the characteristics of the rate of change of oxygen uptake ($\dot{V}O_2$) in response to the onset of exercise or a change in work rate. The measurement of $\dot{V}O_2$ kinetics during exercise is a technique that has been applied to the study of the physiological responses to submaximal exercise (Hagberg et al., 1980, Cochrane and Hughson, 1992, Yoshida and Whipp, 1994, and Chilibeck et al., 1997), to the physiological mechanisms that determine the time course of training adaptations (Yoshida et al., 1992 and Phillips et al., 1995), and clinically to the assessment of the severity of cardiac disease (Sietsema, et al., 1994, Koike et al., 1994, Shephard et al., 1995 and Lok and Lau, 1997) and to determine the effects of drug therapy (Koike et al., 1995).

One possible approach is to use the pseudo random binary sequence (PRBS) exercise test technique to determine $\dot{V}O_2$ kinetics. This technique has been shown to be submaximal (Hughson et al., 1990b, Hughson et al., 1990c, and Hoffmann et al., 1992), although few experiments have validated its use as a test of cardiorespiratory fitness.

There is insufficient evidence, however, concerning

- 1) the variability of the respiratory responses to the PRBS exercise test,
- 2) the relationship between $\dot{V}O_2$ kinetics determined during the PRBS exercise test and measures of aerobic power,
- 3) the sensitivity of the PRBS exercise test to detect training adaptations.

These factors must be addressed before the technique can be applied further.

As yet, $\dot{V}O_2$ kinetics, determined by the PRBS exercise test, have not been used to evaluate the effects of different training models, differentiate between groups of subjects with different athletic status, and evaluate the effects of cardiac rehabilitation exercise programmes.

The aim of this study therefore, is to evaluate the test using a population of healthy, female subjects. The findings from these investigations may eventually lead to the application of the PRBS exercise test as a means of evaluating cardiorespiratory fitness within the clinical environment and within the area of sports science.

1.2) Physiological basis for oxygen uptake kinetics

1.2.1) Cardiovascular and cardiorespiratory responses to the onset of exercise

Cardiovascular and cardiorespiratory responses to the onset of exercise fall under three broad headings: neural, mechanical and metabolic.

1.2.1.1) Neural response

Krogh and Lindhart (1913) described an initial neural mechanism, involving an irradiation of impulses from the motor cortex to the respiratory centres in the brain. This mechanism affects the autonomic respiratory centre controlling respiratory movements. This response is initiated by activity in the motor cortex and sensory information from joint and muscle receptors (proprioceptors). As a consequence of the increased activity of these centres there is an increase in the rate of ventilation of the lungs ($\dot{V}E$). D'Angelo and Torelli (1971) investigated the origin of the initial rapid

increase in $\dot{V}E$ in four young males performing treadmill walking. Ventilation rate and carbon dioxide output ($\dot{V}CO_2$) were measured using a Tissot spirometer and an infrared carbon dioxide meter. Rapid changes in $\dot{V}E$ were seen to be independent of the partial pressure of carbon dioxide in the lungs and were thus attributed to a neural mechanism. Cerretelli et al. (1995) also demonstrated that this initial adaptation to the onset of exercise was of neural origin. Male subjects performed cycle ergometry exercise at 50, 100 and 150 W in normocapnic and hypocapnic conditions. The study concluded that the concentration of carbon dioxide flowing to the lungs from the exercising muscles did not appear to affect this initial rapid rise in $\dot{V}E$. Cerretelli et al. (1995) supported the hypothesis that the impulses arising from the mechanoreceptors in the exercising muscles and joints produced an 'exercise reflex'.

The neural control of heart rate is also modified following the onset of exercise. After the initial adaptation of $\dot{V}E$, the parasympathetic neural influence on the heart is reduced. A greater neural influence arises from the sympathetic nervous system, resulting in an increase in heart rate and force of contraction of the heart. Hughson (1990) described the control of heart rate in response to light exercise as being mainly based on parasympathetic withdrawal, with sympathetic activity being increased when heart rate approaches 100 beats.min⁻¹.

Adaptations in blood flow also occur within the circulatory system with increased sympathetic stimulation to the vasculature resulting in venoconstriction and vasoconstriction. Krogh and Lindhart (1913) stated that increased blood flow was not primarily due to acceleration of the heart rate. An increase in venous return occurs due

to a number of changes in blood flow. Firstly, peripheral vasoconstriction of the vessels occurs to reduce blood flow to non-essential organs. Sympathetic neural impulses initiate vasodilation of the central blood vessels providing an increased delivery of oxygen to the exercising muscles (D'Angelo and Torreli, 1971 and Wasserman, 1982). Then a decrease in the volume of blood held on the venous side of the circulatory system results from constriction of the veins.

1.2.1.2) Mechanical response

Venous return, and therefore stroke volume, is also increased by the action of the exercising muscle or the 'muscle pump'. These circulatory adaptations result in a decrease in the pressure gradient in the veins draining the muscle bed which in turn leads to a greater pressure gradient across the muscle bed and thus increased blood flow to the exercising muscles.

1.2.1.3) Metabolic response

Changes in the concentrations of metabolites, for example increases in adenosine, potassium ions, hydrogen ions, osmolarity and decreases in partial pressure of oxygen (PO_2), cause vasodilation in the arterioles of the active skeletal muscle. These changes however do not fully explain the hyperaemia which must also involve dilation of the feed arteries. Nitric oxide released by endothelial cells has been implicated as a mediator in this response.

The overall effect of these neural, mechanical and metabolic responses is an increased cardiac output (\dot{Q}) and a redistribution of blood into the active muscle bed.

1.2.1.4) Oxygen utilisation by the muscle

Oxygen is utilised at the level of the mitochondria to produce ATP by aerobic metabolism. The amount of O₂ utilised by the muscle increases following an increase in work rate. The rate of increase may be determined by either the rate of increase of O₂ delivery to the muscle or by the activity of the enzymes which control oxidative metabolism.

1.2.2) The oxygen uptake response at the onset of exercise

At the onset of moderate exercise the oxidative processes that provide energy for the resynthesis of ATP cannot match the demand for ATP. Blood oxygen stores (oxyhaemoglobin in venous blood), physically dissolved oxygen and oxyhaemoglobin in the muscles contribute little to the overall energy demand, and therefore the energy difference must be supplied by other sources, for example the depletion of muscle creatine phosphate and also the oxygen spared by lactate accumulation in the body. The difference between the total oxygen that would have been consumed, to meet the energy demands of the contracting muscle, and the actual oxygen consumed at the lung is known as the oxygen deficit. It follows that the magnitude of the oxygen deficit is related to $\dot{V}O_2$ kinetics. The oxygen deficit will be large if $\dot{V}O_2$ kinetics for a given work rate are slow. Conversely those individuals with rapid $\dot{V}O_2$ kinetics will be less likely to deplete their high energy phosphates and are thus less likely to use anaerobiosis to supplement energy requirements (Whipp and Wasserman, 1986).

Explicit modelling procedures have revealed that the $\dot{V}O_2$ response at the onset of submaximal exercise comprises three distinct temporal stages, or phases. These phases

are illustrated in figure 1.1.

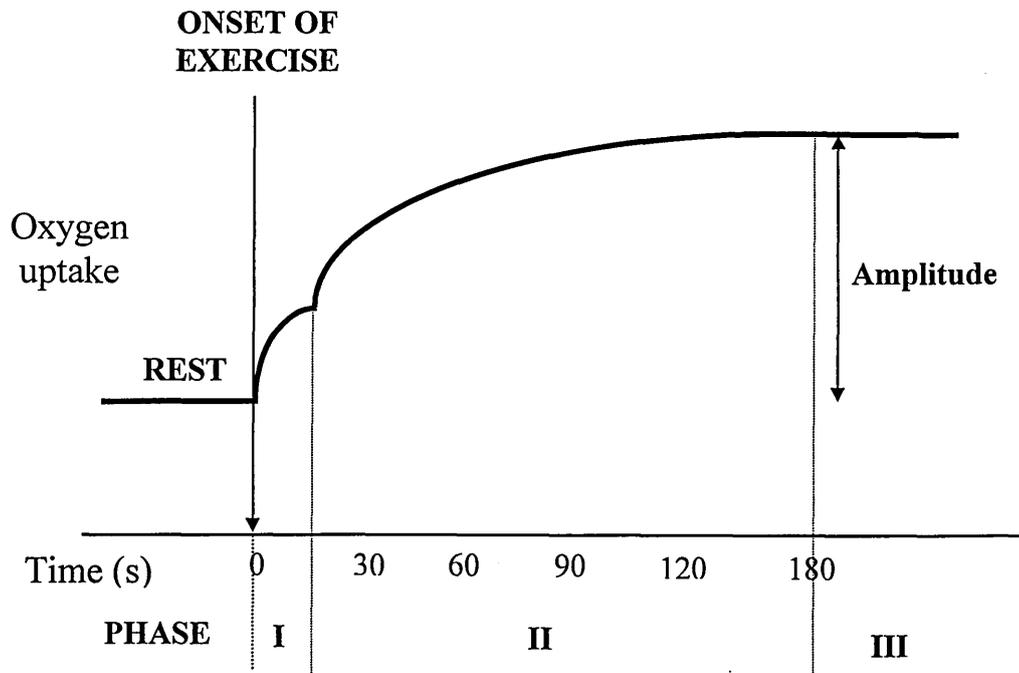


Figure 1.1) The $\dot{V}O_2$ response at the onset of exercise, adapted from Barstow and Mole (1991), and Hamar (1991).

1.2.2.1) Phase I

Krogh and Lindhart (1913) originally documented the rapid initial cardiorespiratory responses associated with the onset of muscular work. This phase I response, frequently described as the cardiodynamic phase (Whipp et al., 1982, Lamarra et al., 1989), is characterised by a rapid increase in $\dot{V}O_2$ and $\dot{V}E$, and where venous blood from the active muscles has not yet reached the lungs. This immediate response to exercise has a duration of 15 to 20 s and is elicited by the irradiation of impulses from the motor cortex to the respiratory centres. Phase I represents increased pulmonary blood flow due to an increase in \dot{Q} (Hamar, 1991, and Whipp et al., 1982) but with minimal extraction of O_2 (Cochrane et al., 1989). Extra non-saturated venous blood is returned to the lungs, from

the muscles. The increase in $\dot{V}O_2$ occurs within the first and second breath (Hamar, 1991). The nature of this phase I response has been well documented by Whipp et al. (1982), Hughson and Swanson (1989) and Cochrane et al. (1989). Since the magnitude of phase I is dependent on the magnitude of the change in work rate, and the $\dot{V}O_2$ response observed at the lungs during phase I does not reflect that at the muscles, most exercise test protocols are designed to eliminate phase I by employing work-to-work transitions rather than rest-to-work transitions.

1.2.2.2) Phase II

A slower, exponential increase in $\dot{V}O_2$ proceeds these rapid, initial adaptive responses. Hughson and Swanson (1989) suggested that this phase II (metabolic phase) response was due to a further increase in pulmonary blood flow, specifically that of the venous blood from the exercising muscles to the lungs. A concomitant increase in O_2 extraction results in a widening of the difference between the arterial and venous oxygen content ($(a-\bar{v})O_2$ difference) as blood arrives at the lungs from the exercising muscles. This blood has a lower O_2 content than the blood arriving at the lungs during phase I. The resulting increased $\dot{V}O_2$ reflects the increased muscle $\dot{V}O_2$ and continues to rise until a steady state is reached at moderate exercise intensities. A depletion in the O_2 stores, within the venous blood, can be measured during this second phase in the $\dot{V}O_2$ response (Inman et al., 1987). Oxygen uptake at the lungs reflects that within the muscles and therefore muscle $\dot{V}O_2$ kinetics can be measured during phase II of the $\dot{V}O_2$ response to exercise (Hoffmann et al., 1994).

1.2.2.3) Phase III

During moderate intensity exercise, phase III is referred to as the steady state phase, where $\dot{V}O_2$ is precisely coupled with the cellular metabolism within the exercising muscle (Wasserman, 1982), i.e. $\dot{V}O_2$ is equal to the O_2 demands of the muscle.

Provided the work rate is below the ventilatory threshold (V_{ET}), and little or no blood lactate is present ($<2\text{mM}$), this phase is reached after approximately 3 min of exercise (Whipp and Wasserman, 1972, and Barstow and Mole, 1991).

During exercise above V_{ET} , steady state is not attained, rather the $\dot{V}O_2$ continues to rise slightly. This is known as the slow component, where blood lactate will be $>2\text{mM}$. It is also known that $\dot{V}O_2$ kinetics measured during phase II are slowed by the accumulation of lactate (Paterson and Whipp, 1991). It is important therefore that an exercise test protocol, designed to elicit phase II responses has an upper work rate which is below the V_{ET} .

1.3) Measurement of oxygen uptake kinetics

The rate of increase in $\dot{V}O_2$ is well characterised in the time domain and is a description of the time constant of the response (Eßfeld et al., 1987, Connett and Honig, 1989). The time constant (τ) is the time required to reach 63% of the increase in $\dot{V}O_2$ from baseline, during either rest or unloaded cycling, to steady state (Hughson et al., 1993).

Whipp and Wasserman (1972) reported time constants of 25 to 39 s for the exponential phase II rise in $\dot{V}O_2$, when performing exercise at constant work rates within a range of 50 to 100 W.

Step work rate forcings have also been analysed using frequency domain analysis

techniques. Rather than a time constant, this method describes $\dot{V}O_2$ kinetics in terms of amplitude and phase shift. In this case the pattern of response is such that as the frequency of the input work rate forcing is increased, the $\dot{V}O_2$ output response has a decreased amplitude and a larger phase shift (Hughson et al., 1990b). Amplitude, measured in $\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$, describes the magnitude of the $\dot{V}O_2$ response in relation to the work rate applied (Eßfeld et al., 1987). Phase shift, measured in degrees, represents the systems delay between the onset of exercise, or a change in work rate, and the increase in $\dot{V}O_2$ (Eßfeld et al., 1987). Individuals with slower $\dot{V}O_2$ kinetics have larger phase shifts and attenuated amplitudes compared to individuals with faster $\dot{V}O_2$ kinetics.

Breath-by-breath technology, in the form of fast response gas analysers, has provided a tool with which to assess $\dot{V}O_2$ kinetics, and the mechanisms which control the physiological adaptations to the onset of exercise (Hamar, 1991). Open circuit systems have been employed, but poor sensitivity during the detection of rapid physiological adaptations has previously limited research within this field (Hughson and Swanson, 1989).

Various methods have been advocated for the measurement of $\dot{V}O_2$ kinetics, the most common of which utilise square wave work rate forcings, sinusoidal work rate forcings and pseudo random work rate forcings. The abrupt work rate changes, which are required to measure $\dot{V}O_2$ kinetics, are normally introduced during cycle ergometry exercise.

1.3.1) Square wave work rate forcings

Traditionally, square wave work rate forcings were the sole means by which $\dot{V}O_2$ kinetics could be measured. These protocols consist of a short period of exercise at a lower work rate immediately followed by an abrupt step increase in work rate. Exercise at the upper work rate level is maintained for an equivalent time period before an abrupt decrease in work rate to the original level. When designing a square wave work rate forcing an upper work rate which results in steady state $\dot{V}O_2$ values below V_{ET} should be chosen. By adopting this constraint, non-linearities in the relationship between $\dot{V}O_2$ and work rate, due to the presence of lactate within the blood, can be minimised (Whipp et al., 1982), thus preventing the slowing of $\dot{V}O_2$ kinetics.

The effect of exercise intensity has been investigated by means of the square wave work rate forcing. The work of Whipp and Wasserman (1972) revealed that the time constant of the exponential rise in $\dot{V}O_2$ was dependent on the magnitude of the step change in work rate. Barstow et al. (1993) confirmed the findings of Whipp and Wasserman (1972) through the measurement of time constants ranging from 32.3 ± 10 s to 75.7 ± 15.6 s for step changes in work rate ranging from 18.1 ± 4.4 W to 165.3 ± 47.1 W, respectively. A slowing of the kinetics of $\dot{V}O_2$ during large changes in work rate was demonstrated.

Yoshida et al. (1992) adopted square wave work rate forcings comprising 6 min exercise periods at an intensity equivalent to the lactate threshold and the onset of blood lactate accumulation (OBLA). The study was designed to assess the effect of strenuous endurance training on the day-to-day changes in $\dot{V}O_2$ kinetics at the onset of exercise. Following four weeks of stationary cycle ergometry performed for 30 min sessions on

six days each week, $\dot{V}O_2$ kinetics were found to be significantly faster (pre-training $\dot{V}O_2$ kinetics = 58.0 ± 10.9 s, and post-training $\dot{V}O_2$ kinetics = 36.1 ± 4.8 s, $P < 0.05$) in this healthy male population.

Oxygen uptake kinetics at the onset and offset of exercise have been studied by Yoshida et al. (1993). Five healthy males performed rest-exercise-recovery work rate transitions with upper work rates of 50, 75 and 100 W during three square wave work rate forcings. Time constants for $\dot{V}O_2$ kinetics ranged from 19 to 43 s. The square wave work rate forcings adopted by Yoshida et al. (1993) are displayed in figure 1.2.

Square wave work rate forcings have also been applied to the elucidation of the $\dot{V}O_2$ response at the onset of exercise, and more specifically to the modelling of alveolar $\dot{V}O_2$ kinetics. The work of Cochrane and Hughson (1992) revealed the complex nature of the mechanisms controlling $\dot{V}O_2$ kinetics and the difficulty in formulating a computer model on which this $\dot{V}O_2$ response could be based. Whipp and Ward (1993) adopted the square wave work rate forcing to characterise the time delay for blood flow between the exercising muscles and the lungs, following the onset of exercise. A number of essential components, including phosphocreatine (PCr) degradation to form high energy phosphate, blood gas stores, pulmonary blood flow, \dot{Q} and $\dot{V}E$, comprised

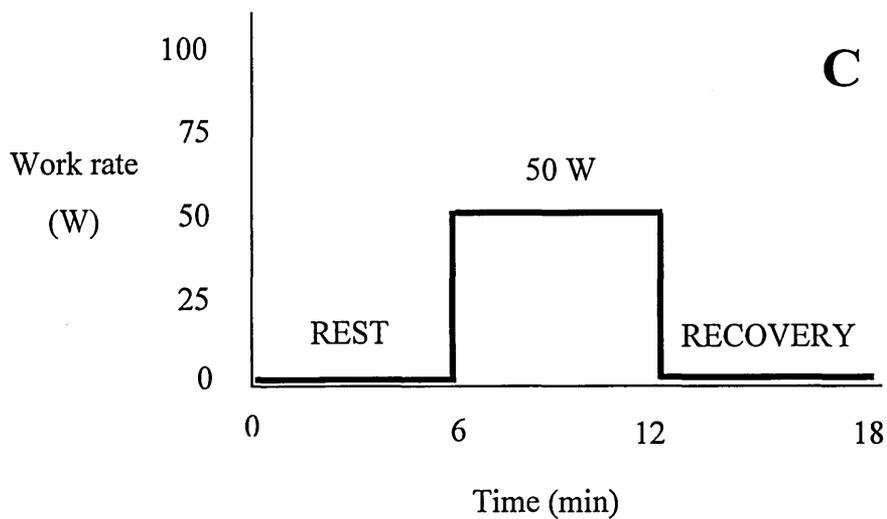
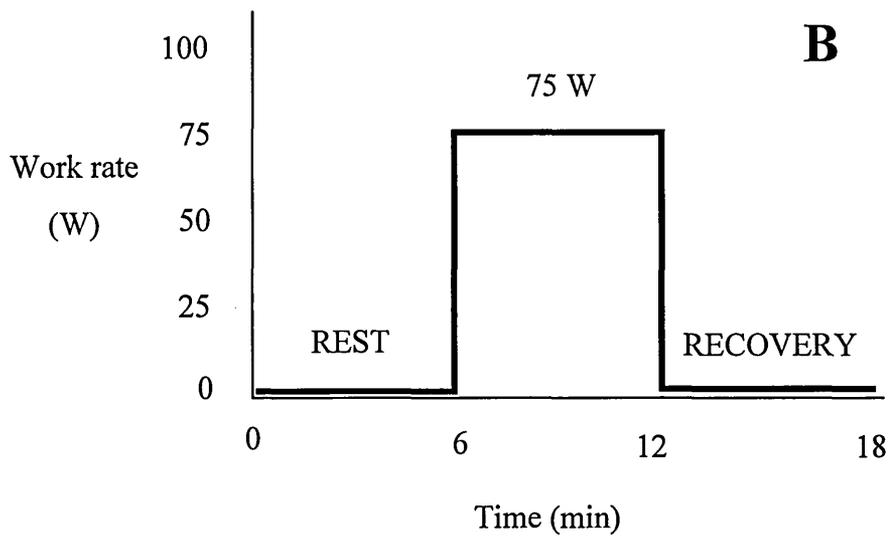
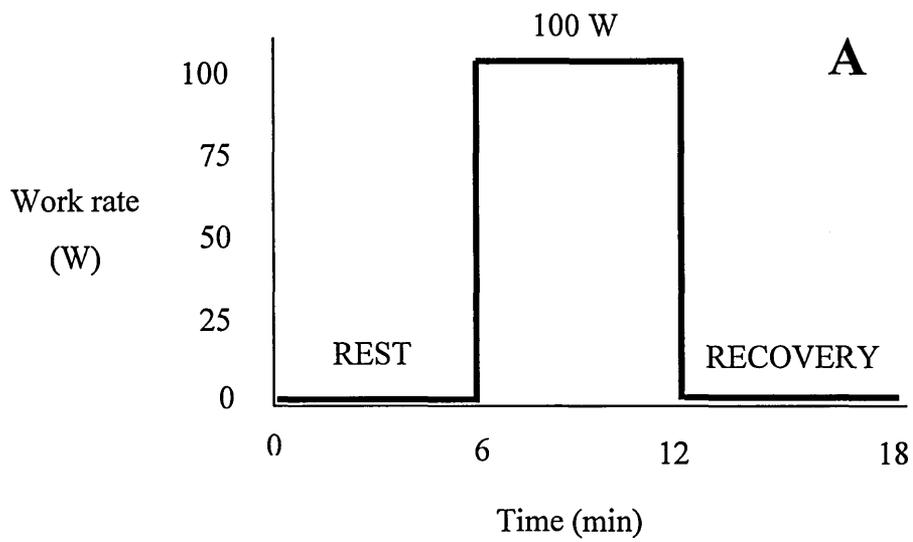


Figure 1.2) Square wave work rate forcings, described by Yoshida et al. (1993) with work rate transitions between rest and (A) 100 W, (B) 75 W and (C) 50 W.

this exponential model. Again, however, the complex nature of these mechanisms and their interaction has resulted in a speculative model for this $\dot{V}O_2$ response.

Numerous other investigators have adopted square wave work rate forcings to investigate $\dot{V}O_2$ kinetics at the onset and offset of exercise (Hagberg et al., 1980, Berry and Moritani, 1985, Hayashida et al., 1993, and Williamson et al., 1996).

Square wave work rate forcings have led to a greater understanding of the $\dot{V}O_2$ response, and the physiological adaptations, at the onset of exercise. Such assessments, however, can only provide a measure of $\dot{V}O_2$ kinetics at a single frequency of work rate change. The relatively long duration of the step changes in work rate associated with the square wave work rate forcings, has resulted in the study of $\dot{V}O_2$ kinetics at low frequencies, i.e. during slow work rate changes.

Explicit mathematical modelling techniques must be applied to the $\dot{V}O_2$ data to yield measures of $\dot{V}O_2$ kinetics (Eßfeld et al., 1987). Although modelling can be complex, the disadvantage of such an approach lies within the lack of consensus concerning the precise nature of the exponential model which should be applied to the $\dot{V}O_2$ data.

During heavy exercise, the presence of lactic acid in the blood is associated with a more complex interaction between cardiorespiratory and metabolic mechanisms. Barstow and Mole (1991) demonstrated a second, slower component in the $\dot{V}O_2$ response, when performing square wave work rate forcings designed to elicit $\dot{V}O_2$ values above V_{ET} (between 85% maximal oxygen uptake ($\dot{V}O_{2max}$) and 100% $\dot{V}O_{2max}$). Recently, Ward et al. (1996) confirmed the bi-exponential nature of the $\dot{V}O_2$ response during high intensity exercise, but maintained that the physiological mechanisms which characterise

this delay are yet unknown. Eßfeld et al. (1987) implied that the nature of the exponential model, for the $\dot{V}O_2$ response, may in fact be altered by the subject population which is being assessed. Hayashida et al. (1993) and Riley et al. (1994) have recently observed increased time constants for the exponential rise in $\dot{V}O_2$ for patients with left ventricular dysfunction and chronic heart failure respectively.

An additional disadvantage associated with the square wave work rate forcing is concerned with the extraction of the $\dot{V}O_2$ response from the noise arising from equipment and inherent biological variability. Masking of the $\dot{V}O_2$ response may occur when performing a single step change in work rate. Data collected during a number of repeated square waves can be averaged to reduce noise and aid in the identification of the $\dot{V}O_2$ response (Ward et al., 1996). The performance of repeated step changes in work rate however, results in a time consuming approach to the measurement of $\dot{V}O_2$ kinetics.

1.3.2) Sinusoidal work rate forcings

The application of sinusoidal work rate forcings to exercise testing has reduced the need for explicit mathematical modelling of the $\dot{V}O_2$ response (Eßfeld et al., 1987). This approach has partially eliminated the need for a consensus concerning the exact nature of the $\dot{V}O_2$ response, at the onset of exercise, thus providing a more direct means of measuring $\dot{V}O_2$ kinetics.

Hamar (1991) described inherent biological variability, due to irregularities associated

with measurements made at the mouth, within the breath-by-breath $\dot{V}O_2$ data collected during square wave work rate forcings. Previously, Beaver et al. (1973) implied that these irregularities were normally distributed and thus mathematical modelling methods could be applied to reduce this breath-by-breath noise. A sinusoidal wave has the additional advantage of comprising a continuously fluctuating response (Casaburi et al, 1977). The components of such a work rate forcing can be separated and averaged to yield a definite physiological response (Hamar, 1991), so theoretically providing a more accurate and reliable measure of $\dot{V}O_2$ kinetics than can be obtained from a single square wave work rate forcing.

Sinusoidal work rate forcings have been designed to oscillate between an upper and a lower work rate. The sinusoidal wave can be characterised by its frequency, defining the rate at which the changes in work rate occur, and amplitude, describing the magnitude of the work rate change. Again, work rates which elicit $\dot{V}O_2$ values below V_{ET} should be chosen to reduce non-linearities in the relationship between $\dot{V}O_2$ and work rate. Through the application of frequency analysis techniques, including Fourier analysis, the amplitude and the phase shift of sinusoidal $\dot{V}O_2$ responses can be quantified, thus producing measures of $\dot{V}O_2$ kinetics. The sinusoidal work rate forcing results in a $\dot{V}O_2$ response which is also sinusoidal in nature, but the amplitude and the phase shift of the output response will be dependent on the initial work rate input.

Casaburi et al. (1977) adopted five different exercise test protocols whilst investigating the ventilatory and gas exchange dynamics in response to sinusoidal work rate forcings. Each exercise test was initiated by a 4 min rest period where the subject remained seated

on the cycle ergometer, this was followed by the sinusoidal work rate forcing and terminated in a square wave work rate change to a level equivalent to the mean work rate of the sinusoid, giving a test duration of 30 min. The breath-by-breath responses to the sinusoidal work rate forcings with periods of 10, 4, 2, 1 and 0.7 min were studied. An example of one of the sinusoidal work rate forcings adopted by Casaburi et al. (1977) is shown in figure 1.3.

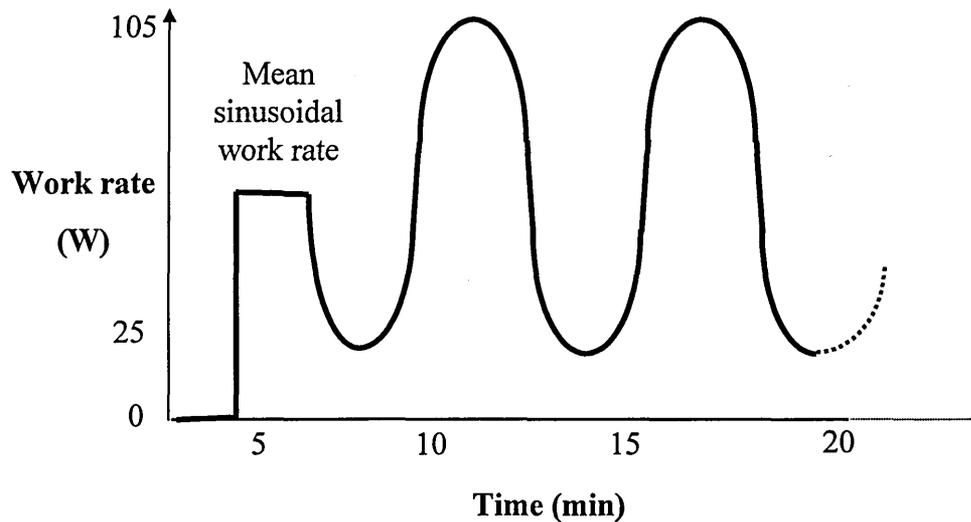


Figure 1.3) A sinusoidal work rate forcing described by Casaburi et al. (1977).

The $\dot{V}O_2$ responses which resulted from the sinusoidal work rate forcings were said to be well described by first-order linear dynamics with a time constant of 0.8 min. Phase lag times ranged from 18 to 34 degrees and from 51 to 90 degrees during sinusoids with a period of 10 min and 0.7 min respectively. Amplitudes ranged from 170 to 470 $\text{ml}\cdot\text{min}^{-1}$ and from 23 to 101 $\text{ml}\cdot\text{min}^{-1}$ during sinusoids with a period of 10 min and 0.7 min respectively. The time consuming nature of this approach therefore is the major disadvantage associated with the use of sinusoidal work rate forcings.

The work of Casaburi et al. (1977) concerning ventilatory and gas exchange kinetics, has led to a greater understanding of the interaction between respiratory variables during exercise. Significantly slower kinetics were observed for $\dot{V}E$ and $\dot{V}CO_2$ when compared to those for $\dot{V}O_2$ and heart rate. On closer analysis, the kinetics of $\dot{V}E$ and $\dot{V}CO_2$ were closely matched (correlation coefficient, $r = 0.97$). Thus it was concluded that the hyperpnea observed at the onset of exercise was associated with $\dot{V}CO_2$.

Hauozi et al. (1993) investigated $\dot{V}O_2$ kinetics below and above V_{ET} by means of sub-threshold and supra-threshold sinusoidal work rate forcings. The sinusoidal work rate forcings adopted during this study had the same frequency of work rate changes. Significantly slower $\dot{V}O_2$ kinetics, signified by reduced amplitudes (sub-threshold amplitude = $7.6 \pm 1.9 \text{ ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$, and supra-threshold amplitude = $5.4 \pm 1.9 \text{ ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$, $P < 0.01$) and increased phase shifts (sub-threshold phase shift = -52 ± 10 degrees, supra-threshold phase shift = -62 ± 11 degrees, $P < 0.05$), were observed during the supra-threshold protocol. These results compared favourably with the findings of Whipp and Wasserman (1972) and Barstow et al. (1993) for square wave work rate forcings. The reduced amplitudes and increased phase shifts can be partially explained by the presence of non-linearities in the relationship between $\dot{V}O_2$ and work rate, due to the presence of lactate within the blood (Whipp et al., 1982). A number of rate-limiting mechanisms have been proposed which could account for the introduction of this slow component into the $\dot{V}O_2$ response to supra-threshold exercise. These mechanisms will be discussed in Chapter 1, Section 1.4. The use of sinusoidal work rate forcings of the same frequency limits the knowledge which can be gained from this investigation. By adopting a number of sinusoidal work rate forcings with different frequencies of work

rate changes, a more in-depth, multi-frequent assessment of $\dot{V}O_2$ kinetics could have been attained.

Cunningham et al. (1993) conducted a multi-frequent assessment of $\dot{V}O_2$ kinetics using six sinusoidal work rate forcings of different frequencies. Nineteen young ($\dot{V}O_{2max} = 36.9 \pm 4.7 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) and elderly female subjects ($\dot{V}O_{2max} = 23.7 \pm 5.4 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) performed each of the sinusoidal work rate forcings. Gas analysis was performed using a respiratory mass spectrometer to collect breath-by-breath data for CO_2 , O_2 and N_2 . Lower amplitudes and larger phase lags (phase shifts) were seen for the elderly subject population (c.f. table 1.1).

Table 1.1) Amplitude ($\text{ml}\cdot\text{min}^{-1}$) and phase shift (degrees) at different frequencies of work rate change in young and elderly women taken from Cunningham et al. (1993).

Period of sinusoid (s)	Young subjects		Elderly subjects	
	Amplitude ($\text{ml}\cdot\text{min}^{-1}$)	Phase lag (degrees)	Amplitude ($\text{ml}\cdot\text{min}^{-1}$)	Phase lag (degrees)
45	19 ± 13	-154 ± 95	27 ± 14	-95 ± 57
60	$56 \pm 28^*$	-117 ± 47	26 ± 22	-152 ± 45
90	$77 \pm 35^*$	-115 ± 31	41 ± 31	-117 ± 36
180	$159 \pm 44^*$	-70 ± 27	54 ± 30	-76 ± 33
360	$192 \pm 71^*$	$-40 \pm 8^*$	86 ± 25	-64 ± 16
600	$187 \pm 57^*$	-23 ± 8	90 ± 31	-31 ± 12

*= significant difference ($P < 0.05$) between $\dot{V}O_2$ kinetics for young and elderly subject populations.

Cunningham et al. (1993) concluded that there was a strong link between the slowing of the components of the gas transport system and increasing age, and suggested that one single factor may be reflected in the slowing of all components in elderly subjects.

Although the repetitive nature of this type of work rate forcing has led to the reduction of equipment noise and inherent biological variability, a single sinusoidal wave can only provide a mono-frequent assessment of $\dot{V}O_2$ kinetics. A number of sinusoidally fluctuating work rate forcings, of different frequencies, must be performed to obtain measures of $\dot{V}O_2$ kinetics for both rapid and slow changes in work rate. Again, this leads to a time consuming assessment of the $\dot{V}O_2$ response to work rate changes.

1.3.3) Pseudo random work rate forcings

More recently, there has been a trend towards the use of pseudo random work rate forcings for the measurement of $\dot{V}O_2$ kinetics. The PRBS exercise technique, based on pseudo random work rate forcings, was originally designed to determine exercise capacity and the effects of weightlessness on physiological mechanisms, during space travel (Eßfeld et al., 1987). This exercise testing technique provides an assessment of $\dot{V}O_2$ kinetics which reflects $\dot{V}O_{2max}$ values, within a relatively short time period. To evaluate the reduction in $\dot{V}O_{2max}$ associated with the removal of gravitational effects, the PRBS work rate forcing was designed to incorporate submaximal work rates which would not provide a significant training effect (Stegemann, 1991).

This type of work rate forcing comprises a number of pseudo random changes in work rate between upper and lower levels, selected to elicit $\dot{V}O_2$ values below V_{ET} . The

number of work rate changes and the time interval, during which these changes occur, is controlled by a computerised random number generator (Hampton, 1965). Each pseudo random binary sequence is designed to incorporate a number of different frequencies, comprising harmonics of the slowest frequency of work rate change, thus including slow and rapid changes in work rate. This approach therefore eliminates the need for performing repeated exercise tests, as associated with square wave and sinusoidal work rate forcings, so providing a multi-frequent assessment of $\dot{V}O_2$ kinetics from a single exercise test (Eßfeld et al., 1987, Stegemann, 1991).

Typically, a single PRBS exercise test would involve the performance of a number of identical pseudo random binary sequences of work rate changes. Again, the components of such a work rate forcing can be separated and analysed using well established mathematical techniques, including ensemble averaging and Fourier analysis. The $\dot{V}O_2$ response to these pseudo random changes in work rate can be characterised in terms of amplitude and phase shift, as described in Chapter 1, Section 1.2.3.

Commonly described examples of such work rate forcings include pseudo random binary sequences consisting of:

- fifteen work rate changes (units), between 20 and 80 W, occurring at time intervals of 30 s, with a total sequence duration of 450 s or 7.5 min (Eßfeld et al., 1987 and Hoffmann et al., 1994). The total test comprised three pseudo random binary sequences, giving a total test duration of 1350 s or 22.5 min. This example is shown in figure 1.4.

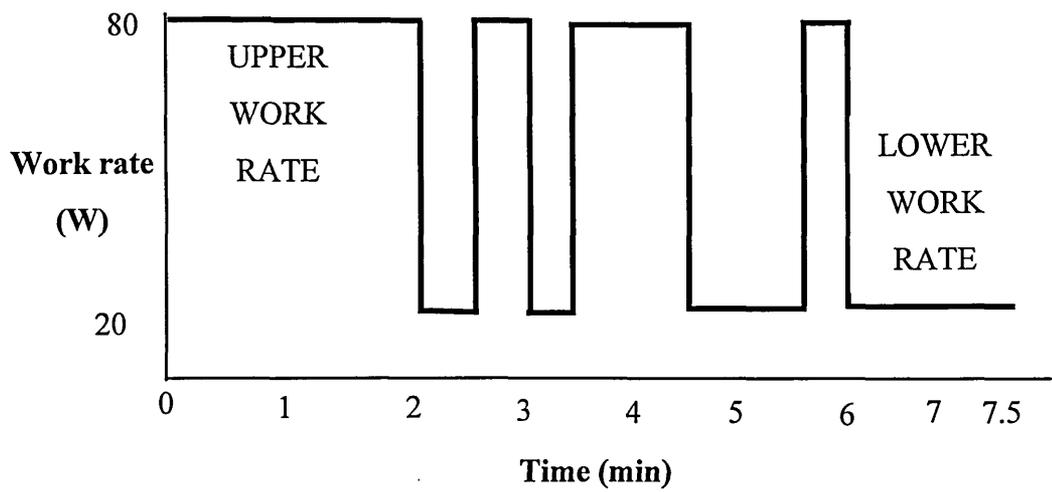


Figure 1.4) A single sequence from a PRBS work rate forcing described by Eßfeld et al. (1987).

- sixty-three work rate changes (units), between 25 and 105 W, occurring at time intervals of 5 s, with a total sequence time of 300 s or 5 min (Hughson et al., 1990a). The total test comprised six pseudo random binary sequences, giving a total test duration of 1800 s or 30 min. This example is shown in figure 1.5.

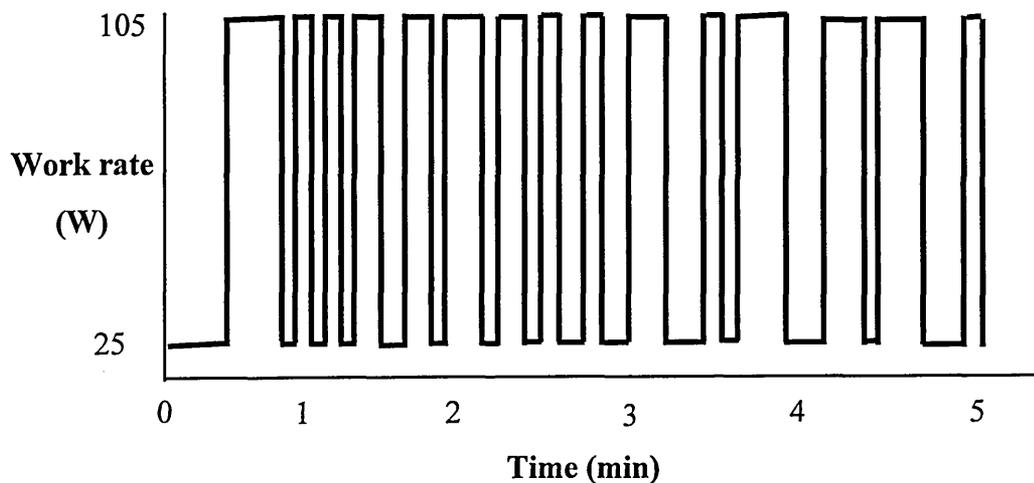


Figure 1.5) A single sequence from a PRBS work rate forcing described by Hughson et al. (1990a).

Greco et al. (1986) studied transient ventilatory and heart rate responses to moderate nonabrupt pseudo random exercise in a population of young males. Each subject performed non-abrupt dynamic variations in exercise intensity in the form of a low-pass filtered pseudo random binary sequence. The exercise intensity of the test was selected to produce a mean $\dot{V}O_2$ response of less than 50% $\dot{V}O_{2max}$, determined during a prior maximal exercise test. A constant lower work level of 25 W was used, with upper work rates ranging from 115 to 165 W. Mean exercise intensities ranged from 70 to 95 W. A rapid phase I response followed by a slower phase II response were identified when a third order, two-compartment model was fitted to the $\dot{V}O_2$ response. This study showed that the faster components in the ventilatory response to the onset of exercise can be stimulated when performing a pseudo random work rate forcing.

The PRBS exercise test technique has also been used to study the effects of posture on the physiological mechanism which may limit $\dot{V}O_2$ kinetics (Hughson et al., 1990a). Young male subjects performed a sixty-three unit PRBS exercise test with 5 s work rate changes between 25 and 105 W, and a square wave work rate forcing with work rate transitions between 25 and 105 W in the supine and upright position. Significantly slower $\dot{V}O_2$ kinetics ($P < 0.05$) were observed in the supine position (time constant for supine exercise = 40.1 ± 2.7 s) than when performing upright exercise (time constant for upright exercise = 32.0 ± 1.2 s). Hughson et al. (1990a) found that $\dot{V}O_2$ kinetics determined by means of the PRBS exercise test technique were comparable to those measured during the square wave work rate forcing ($r = 0.99$).

Hoffmann et al. (1994) reported similar findings to those of Hughson et al. (1990a)

when male and female subjects performed 1) square wave work rate forcings with work rate transitions between 20 and, 40, 80, 120 or 160 W, and 2) a sixty-three unit PRBS exercise test with 5 s work rate changes between 20 and 80 W. The study revealed that there was a tendency for slower $\dot{V}O_2$ kinetics in the supine position during both square wave and pseudo random work rate forcings, but no significant postural effect on cardiorespiratory changes could be found. Hughson et al. (1990b) also investigated the effect of different PRBS exercise test protocols on $\dot{V}O_2$ kinetics in a population of young, healthy male subjects. Each subject performed 1) a fifteen unit PRBS exercise test with 30 s work rate changes between 25 and 105 W, and 2) a sixty-three unit PRBS exercise test with 5 s work rate changes between 25 and 105 W. In a comparison of the $\dot{V}O_2$ kinetics measured during the two protocols, amplitude and phase shift were found to be unchanged. The study revealed that the PRBS exercise test with 30 s work rate changes between 25 and 105 W was sensitive to the measurement of $\dot{V}O_2$ kinetics during the slower phase II component of the $\dot{V}O_2$ response to exercise. Conversely, the PRBS exercise test with 5 s work rate changes between 25 and 105 W was found to be sensitive to the measurement of the rapid physiological adaptations which characterise the phase I response of the exponential rise in $\dot{V}O_2$ at the onset of exercise.

Comparisons of the $\dot{V}O_2$ kinetics determined by the PRBS exercise technique and sinusoidal work rate forcings have been made (Hoffmann et al., 1992, and Hoffmann et al., 1994). Hoffmann et al. (1992) used 1) a sixty-three unit PRBS exercise test with 5 s work rate changes between 20 and 80 W, 2) a fifteen unit PRBS exercise test with 30 s work rate changes between 20 and 80 W, and 3) three sinusoidal work rate forcings oscillating at different frequencies between different work rates, to investigate the

dynamic linearity of the relationship between $\dot{V}O_2$ and work rate during the measurement of $\dot{V}O_2$ kinetics. Oxygen uptake kinetics determined by the multi-frequent PRBS exercise test technique and the mono-frequent sinusoidal work rate forcings were not found to be significantly different. The agreement between the two types of test was poorer at higher frequencies of the work rate change but was not different. Hoffmann et al. (1992) stated that the relationship between $\dot{V}O_2$ and work rate was linear at work rate changes with periods of 100 s or above. i.e. harmonic numbers 1 to 4 of the fifteen unit PRBS exercise test and harmonic numbers 1 to 3 of the sixty-three unit PRBS exercise test.

The PRBS exercise test technique has particular advantages over square wave and sinusoidal work rate forcings for the determination of $\dot{V}O_2$ kinetics.

Firstly, this type of exercise test can provide a multi-frequent assessment of $\dot{V}O_2$ kinetics. As high and low frequency work rate changes are included in the protocol, both slow and fast components of the $\dot{V}O_2$ response to exercise can be stimulated during a single test performance. In addition each test comprises a series of identical pseudo random binary sequences which can be concatenated to help discriminate between the actual $\dot{V}O_2$ response, and biological and instrumental noise. Multiple square wave work rate forcings would need to be performed to identify the actual $\dot{V}O_2$ response. From these two factors it can be seen that the PRBS exercise test technique provides a less time consuming approach to the measurement of $\dot{V}O_2$ kinetics than square wave and sinusoidal work rate forcings.

Secondly, the PRBS exercise test is by definition submaximal in nature (Hughson et al.,

1990b, Hughson et al., 1990c and Hoffmann et al., 1992). Hughson et al. (1990b) observed $\dot{V}O_2$ values of $1205 \pm 11 \text{ ml}\cdot\text{min}^{-1}$ and $1190 \pm 11 \text{ ml}\cdot\text{min}^{-1}$ for fifteen unit PRBS exercise tests and sixty-three unit PRBS exercise tests with work rate changes between 25 and 105 W respectively when performed by male subjects. Hoffmann et al. (1992) reported $\dot{V}O_2$ values of $936 \pm 83 \text{ ml}\cdot\text{min}^{-1}$ for a fifteen unit PRBS exercise test with work rate changes between 20 and 80 W, when performed by a male and female study population. Inbar et al. (1994) observed $\dot{V}O_{2\text{max}}$ of $2880 \pm 570 \text{ ml}\cdot\text{min}^{-1}$ for 20 to 30 year old male subjects, during progressive exercise to exhaustion. When comparing these normative values with the $\dot{V}O_2$ values observed during the pseudo random work rate forcings, the studies of Hughson et al. (1990b), Hughson et al. (1990c) and Hoffmann et al. (1992) resulted in exercise intensities equivalent to 41.8%, 41.3 % and 32.5% $\dot{V}O_{2\text{max}}$. The submaximal nature of this exercise test technique means that the presence of blood lactate is reduced or eliminated, and consequently non-linearities, which cause errors in the assessment of $\dot{V}O_2$ kinetics, are minimised.

Finally, as with sinusoidal work rate forcings, the PRBS exercise test technique relies on well established mathematical techniques such as Fourier analysis, so eliminating the difficulties encountered when using explicit mathematical modelling.

The PRBS exercise test technique has led to a greater knowledge of the nature of $\dot{V}O_2$ kinetics. It is anticipated that this type of exercise test will enable more in-depth research into the physiological response at the onset of exercise and the mechanisms influencing $\dot{V}O_2$ kinetics.

1.4) The influence of rate limiting factors on Phase II oxygen uptake kinetics

Phase II of the $\dot{V}O_2$ response is described by first order exponential modelling suggesting that there is one rate limiting step for $\dot{V}O_2$ kinetics. There are two theories concerning where this rate limiting step lies:

- in the O_2 delivery to the working muscle (central)
- in the ability of the muscle to utilise O_2 (peripheral).

As far as $\dot{V}O_2$ kinetics are concerned, central factors are those which relate to the delivery of O_2 to the mitochondria, and peripheral factors relate to the other factors which control mitochondrial respiration (Walsh, 1992).

Two approaches have been used to provide support for these theories. One is to alter one or more steps in the O_2 delivery process, and the other is to identify the physiological parameter with the same response characteristics as the $\dot{V}O_2$ response.

1.4.1) Acute alterations of the oxygen delivery process

This approach involves changing the rate of O_2 delivery to the working muscle through manipulations involving β -adrenergic receptor blockade, hypoxia and hyperoxia, circulatory occlusion, upright and supine exercise, and finally techniques which create negative and positive lower body pressures. If O_2 transport is limiting then any change in O_2 transport should be directly linked to changes in $\dot{V}O_2$ kinetics.

1.4.1.1) β -adrenergic receptor blockade

Drugs which block β -adrenergic receptors inhibit the effects of sympathetic nervous stimulation. Cardio-selective drugs have their main effect on the β_1 -adrenergic

receptors in the heart, whereas non-selective β -adrenergic receptor blocking drugs have a more general effect on β_2 -adrenergic receptors throughout the body (Yeo et al., 1991). The effects that non-selective β -adrenergic receptor blocking drugs have on the exercise response include a reduction in \dot{Q} , decreased fat and carbohydrate mobilisation and an impairment in the normal redistribution of blood flow which occurs during exercise. It might be anticipated that $\dot{V}O_2$ kinetics should be slower due to the lower \dot{Q} and poorer blood flow redistribution with β -adrenergic receptor blockade. This has been shown in various studies (Hughson and Smyth, 1983, Hughson, 1984, Kowalchuk and Hughson, 1990). It made no difference whether the drug administered was a cardio-selective or a non-selective β -adrenergic receptor blocking agent. Although this suggests a central limitation for $\dot{V}O_2$ kinetics, these studies have been criticised because the reduced exercise capacity of healthy people receiving β -adrenergic receptor blocking agents may have resulted in the subjects exercising at an upper work rate which was no longer truly aerobic (Hoffmann et al., 1994). This may explain the slower $\dot{V}O_2$ kinetics during β -adrenergic receptor blockade.

1.4.1.2) Hypoxia and hyperoxia

Hypoxia has been shown to cause a significant slowing in the $\dot{V}O_2$ response to ramp, step and pseudo random work rate forcings (Murphy et al., 1989, Hughson and Kowalchuk, 1995) when compared to normoxia, suggesting that O_2 transport acts as a rate limiting step in oxidative metabolism. If O_2 transport is limiting then hyperoxia should result in faster $\dot{V}O_2$ kinetics. Administering gas mixtures containing 70% O_2 did not affect $\dot{V}O_2$ kinetics (Hughson and Kowalchuk, 1995) suggesting that O_2

transport can act as a rate limiting step if inspired O_2 is reduced, but under normal conditions of arterial O_2 content, oxygen transport does not become limiting.

1.4.1.3) Circulatory occlusion

Circulatory occlusion of both legs, after elevation to improve venous drainage, increases central blood volume. This manoeuvre should temporarily increase \dot{Q} , and since it also speeds up $\dot{V}O_2$ kinetics of the arms (Hughson and Inman, 1986), it can be suggested that arm blood flow may be a limiting factor in determining arm $\dot{V}O_2$ kinetics.

1.4.1.4) Supine and upright exercise

Variations in body position bring about circulatory changes which may have opposing effects on muscle perfusion. In the supine position, venous return, and therefore \dot{Q} , increase due to the removal of the normal gravitational effects seen in upright exercise. Even though \dot{Q} is increased during supine exercise (Hughson et al., 1991) it may not be reflected by an increased muscle perfusion for two reasons. Firstly, during supine exercise there may be a decrease in sympathetic tone to the splanchnic beds that would cause a reduction in the normal vasoconstriction seen during exercise. When compared to upright exercise, less blood would flow to the active skeletal muscle during supine exercise as more blood flows to the splanchnic area. Secondly, the decreased arterial pressure during supine exercise may decrease the perfusion pressure across the muscle capillary beds and consequently decrease muscle blood flow. This decrease in muscle perfusion may explain the slower $\dot{V}O_2$ kinetics, in response to a step change in work rate, observed during supine exercise when compared to upright exercise (Hughson et al., 1990a, and Convertino et al., 1984). Other studies, using the PRBS exercise test

technique, have failed to demonstrate a significant slowing of $\dot{V}O_2$ kinetics in the supine position (Hoffmann et al., 1991 and Hoffmann et al., 1994). Hughson et al. (1991) showed a trend towards slower $\dot{V}O_2$ kinetics in the supine position but significant differences were only found at a frequency of 0.0067 Hz.

In summary, supine and upright experiments have produced conflicting results which is not surprising considering the partially opposing circulatory effects brought about by changing body position. Measurement of muscle perfusion during upright and supine exercise may explain the role of O_2 transport in determining $\dot{V}O_2$ kinetics during submaximal exercise.

1.4.1.5) Lower body negative and positive pressure

Application of lower body negative pressure (LBNP) has been used as a technique to increase leg muscle blood flow (Eiken, 1988, Hughson et al., 1993). Lower body negative pressure increases muscle blood flow by decreasing the pressure in the capacitance vessels, thereby increasing the arterio-venous O_2 pressure gradient across the muscle bed. In supine exercise, the application of LBNP increased $\dot{V}O_2$ kinetics to values that were similar to those seen in upright exercise. This would suggest that the decrease in $\dot{V}O_2$ kinetics seen in supine exercise is due to a decrease in O_2 delivery to the muscle, since it can be reversed by a technique which increases O_2 delivery.

Evidence from LBNP studies supports the central limitation theory for $\dot{V}O_2$ kinetics.

Conversely, lower body positive pressure (LBPP) decreases muscle blood flow (Sundberg and Kaisjer, 1992) and it would be expected that application of LBPP would

decrease $\dot{V}O_2$ kinetics. The application of a LBPP of 45 torr, however, failed to alter $\dot{V}O_2$ kinetics (Williamson et al., 1996) casting doubt on the suggestion that $\dot{V}O_2$ kinetics under normal conditions are limited by blood flow to the working muscle.

1.4.1.6) Summary

Although some manipulations, which decrease O_2 delivery to the muscle, have been shown to decrease $\dot{V}O_2$ kinetics, the lack of a consistent effect on $\dot{V}O_2$ kinetics, of increasing O_2 delivery, suggests that O_2 delivery may not limit $\dot{V}O_2$ kinetics under normal conditions.

1.4.2) Identification of the physiological factor with the same response characteristics as the oxygen uptake response

1.4.2.1) Heart rate and cardiac output kinetics

If the rate of O_2 delivery to the muscle is the limiting factor for $\dot{V}O_2$ kinetics then the rate of response of the O_2 delivery system should be similar to the $\dot{V}O_2$ kinetics. The rate of increase in $\dot{V}O_2$ is often found to be slower than the rate of increase in either heart rate or \dot{Q} at the onset of exercise (Cerretelli et al., 1966, Linnarsson, 1974, De Cort et al., 1991, Yoshida and Whipp, 1994). Although these studies might suggest that cardiac function does not limit $\dot{V}O_2$ kinetics, the effect of redistribution of blood flow within the muscle is not known and therefore these studies do not preclude the possibility of O_2 delivery limiting $\dot{V}O_2$ kinetics.

Hughson and Morrissey (1983) used the approach of comparing the change in heart rate kinetics with changes in $\dot{V}O_2$ kinetics, when work rate protocols were altered. The similar changes in both $\dot{V}O_2$ and heart rate kinetics were taken as evidence of a central limitation for $\dot{V}O_2$ kinetics.

1.4.2.2) Blood flow kinetics

Muscle blood flow increases very rapidly at the beginning of exercise (Walloe and Wesche, 1988, Eriksen et al., 1990, Grassi et al., 1996). In order to establish the role of muscle blood flow and, therefore O_2 delivery as a limiting factor for $\dot{V}O_2$ kinetics, the $\dot{V}O_2$ kinetics of the working muscle have been measured simultaneously with muscle blood flow (Hughson et al., 1996 and Grassi et al., 1996). Grassi et al. (1996) measured the $\dot{V}O_2$ kinetics of the leg and $\dot{V}O_2$ kinetics at the mouth during leg exercise (cycling at 50 W). As $\dot{V}O_2$ kinetics measured at the mouth and leg blood flow had the same time constants, it might be concluded that blood flow was limiting $\dot{V}O_2$ kinetics. As the time delay between an increase in muscle $\dot{V}O_2$ reflects an increase in pulmonary $\dot{V}O_2$, it is important to compare $\dot{V}O_2$ at the muscle with leg blood flow. This comparison shows that leg muscle $\dot{V}O_2$ increased only modestly in the first 10 to 15 seconds of exercise even though blood flow increased markedly. This suggests that early on in the exercise response, bulk delivery of O_2 to the working muscle is not limiting $\dot{V}O_2$ kinetics of the leg but it is still not possible to discriminate between maldistribution of blood flow within muscle and more peripheral mechanisms.

There is some evidence that blood flow limits $\dot{V}O_2$ kinetics in the arms when blood flow is reduced by carrying out arm exercise above the level of the heart (Hughson et

al., 1996) but similar studies using leg exercise have not been carried out. Until it is possible to measure the blood flow distribution to the active muscle fibres the role of blood flow limiting $\dot{V}O_2$ kinetics will remain controversial.

1.4.2.3) Phosphocreatine kinetics

Another technique, using the same approach, is to compare $\dot{V}O_2$ kinetics with the kinetics of PCr degradation. It has been proposed that $\dot{V}O_2$ kinetics of the muscle and therefore $\dot{V}O_2$ kinetics measured at the lung, reflect the metabolic processes that control mitochondrial respiration in the exercising muscle (Mahler, 1985, Meyer, 1988, Whipp and Ward, 1990). The rate of PCr degradation has been implicated as the rate limiting step in the control of mitochondrial respiration (Connett and Honig, 1989). Studies which have measured $\dot{V}O_2$ kinetics and PCr kinetics have therefore provided some insight into the role of more peripheral mechanisms in limiting $\dot{V}O_2$ kinetics.

Phosphocreatine breakdown can be estimated by nuclear magnetic resonance (P^{31} NMR) and by muscle biopsy techniques, both in isolated muscle preparations and *in situ* during voluntary muscle contractions in humans (Coggan et al., 1993). Barstow et al. (1994) measured time constants during two different exercise modes which provided indirect evidence of this link since the time constants for phase II $\dot{V}O_2$ kinetics were similar to those for PCr degradation. In a more recent study, McCreary et al. (1996) measured $\dot{V}O_2$ kinetics and PCr kinetics during plantar flexion and found time constants of 44.5 s for $\dot{V}O_2$ and 47 s for PCr. The similarity between these time constants supports the contention that $\dot{V}O_2$ kinetics are controlled by muscle oxidative function with PCr degradation controlling mitochondrial oxidative phosphorylation by the “creatine phosphate shuttle”.

The $\dot{V}O_2$ of the skeletal muscle during exercise is both preceded by, and controlled by, the energy transferring reactions of the intramuscular high energy pool. Although the particular details of the controlling mechanisms remain controversial, mitochondrial creatine kinase appears to play a pivotal role, since the proportional exponential increase in muscle $\dot{V}O_2$ parallels the decrease in the levels of PCr. The diagram presented by Mahler (1985) shows that $\dot{V}O_2$ is controlled by the availability of creatine, which in turn, is determined by how quickly PCr degradation occurs at the myofibril (c.f. figure 1.6).

The activity of the mitochondrial creatine kinase enzyme is controlled by the phosphocreatine to creatine ratio (PCr/Cr). In this model, describing the control of mitochondrial respiration during moderate, submaximal exercise, O_2 is not thought to be limiting until the partial pressure of oxygen within the mitochondria (mitochondrial PO_2) falls below 1 mmHg. The O_2 supply to the mitochondria may become limiting when the partial pressure of oxygen in the capillary (capillary PO_2) falls below 15 to 20 mmHg (Wittenberg and Wittenberg, 1989) because of the physical factors limiting O_2 diffusion. In moderate exercise the partial pressure of oxygen in the veins (venous PO_2) remains above this level (Doll et al., 1968) suggesting that O_2 supply to the mitochondria does not become critical unless, as previously discussed, there is some maldistribution of the blood flow within the capillary bed.

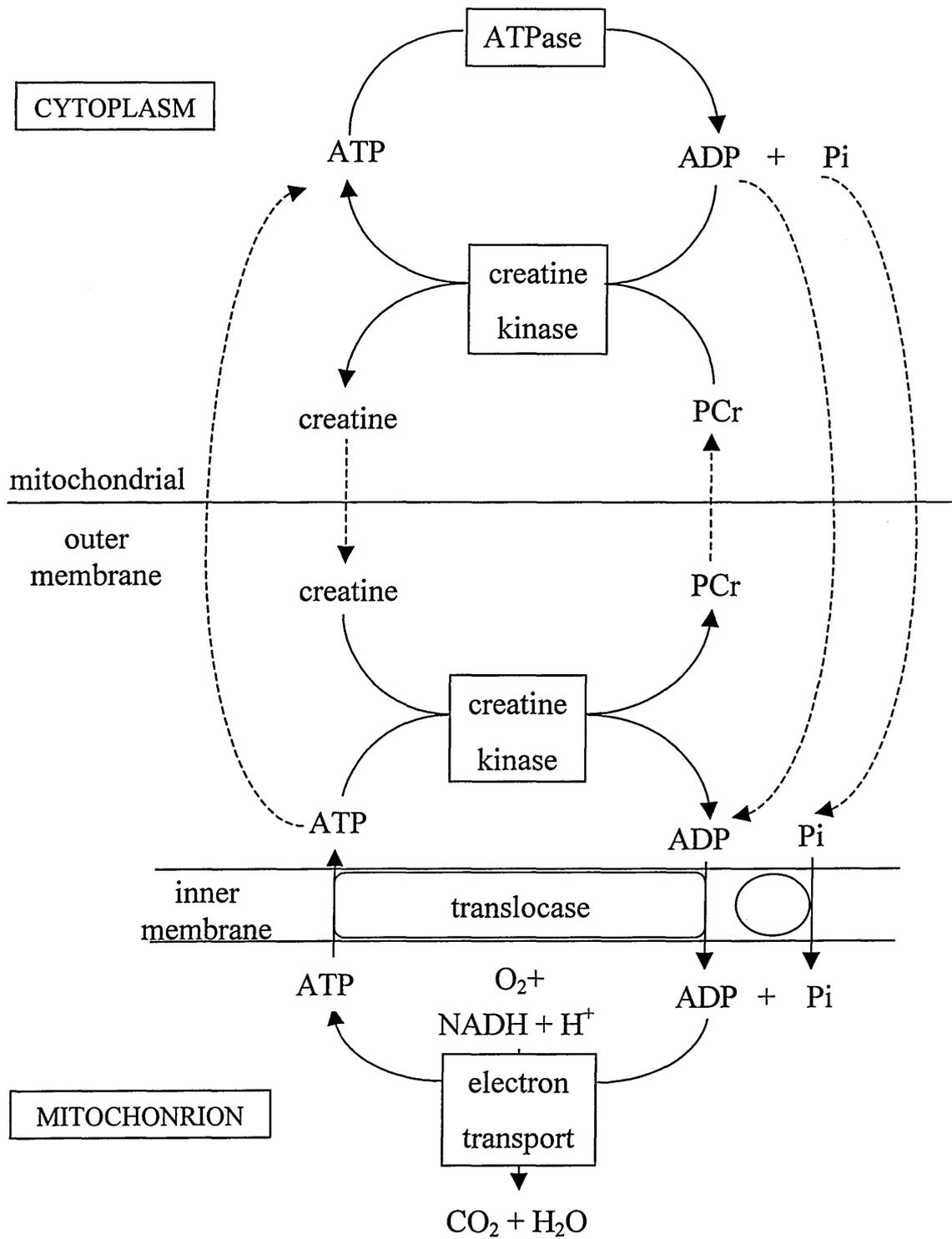


Figure 1.6) A simplified schematic diagram of the reactions by which O_2 consumption is coupled to extramitochondrial ATP hydrolysis in muscle. Dashed lines indicate diffusion. Taken from Mahler (1985).

1.4.2.4) Summary

Of the physiological parameters studied, PCr kinetics are most closely aligned to $\dot{V}O_2$ kinetics and this, taken together with the current theories on control of mitochondrial respiration (c.f. figure 1.6), provide some evidence for a peripheral site for the factor limiting $\dot{V}O_2$ kinetics.

1.4.3) Conclusion

Whether the limiting factor determining $\dot{V}O_2$ kinetics during submaximal exercise is due to central or peripheral factors remains controversial. Theoretically it could be argued that the control of $\dot{V}O_2$ kinetics is driven by ATP demand at the muscle (i.e. peripherally) rather than the supply of O_2 to the muscle. Until it becomes technically possible to measure PO_2 at the level of the muscle fibre it seems likely that this controversy will remain.

1.5) Application of PRBS exercise testing

The unique feature of the PRBS exercise technique, originally designed by Kerlin (Kerlin, 1974) for engineering purposes, is that the input pattern contains a number of frequencies of work rate changes and the output $\dot{V}O_2$ pattern can be mathematically processed to provide measures of $\dot{V}O_2$ kinetics in the frequency domain (Stegemann, 1992). The potential application to space medicine has been instrumental in the development of the PRBS exercise test technique (Stegemann, 1991, and Stegemann, 1992). Two main groups have applied the technique to the study of exercise control mechanisms. The Canadian group, led by R.L Hughson, has used this test to evaluate

the effects of various environmental, postural and pharmacological interventions on the physiological mechanisms which determine the kinetics of $\dot{V}O_2$ (Hughson et al., 1989, Hughson et al., 1990a, Hughson et al., 1990c, and Xing et al., 1991).

Similar investigations have been conducted by the German group (U Hoffman and D Eßfeld) who have demonstrated that subjects with varying $\dot{V}O_{2max}$ values can be differentiated, on the basis of $\dot{V}O_2$ kinetics, by means of a PRBS exercise test. Twenty-nine male and nine female subjects performed an exercise protocol with the following three consecutive parts:

- three 5 min periods of 20W, 80W and 20W for the determination of steady state $\dot{V}O_2$ values during the last minute of each period,
- three identical fifteen unit pseudo random binary sequences with 30 s work rate changes between 20 and 80 W for the determination of $\dot{V}O_2$ kinetics,
- a progressive exercise test with ramp changes in work rate of 10 W every 30 s to exhaustion, for the determination of $\dot{V}O_{2max}$.

Breath-by-breath gas analysis was performed using a respiratory mass spectrometer. Data collected during the pseudo random binary sequences were subjected to Fourier analysis in order to transform the data from the time to the frequency domain (c.f. Chapter 2, Section 2.4.2.1). Subjects attaining higher values of $\dot{V}O_{2max}$, during the progressive exercise test to exhaustion, were found to achieve higher amplitude values at different frequencies of work rate changes. There was a wider variation in phase shift values, however, when comparing these values to the distribution of the amplitude values. The amplitude and phase shift values reported by Eßfeld et al. (1987) are

displayed in tables 1.2 and 1.3 respectively.

Table 1.2) Amplitude values ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$) at harmonic numbers 1 to 4, determined by the PRBS exercise test technique, and categorised according to relative $\dot{V}O_{2\text{max}}$ ($\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) (mean \pm SD). The results are adapted from the work of Eßfeld et al. (1987).

Relative $\dot{V}O_{2\text{max}}$ ($\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$)	Harmonic number			
	1	2	3	4
<50	9.55 \pm 1.15	7.76 \pm 1.15	6.17 \pm 1.66	3.89 \pm 1.74
50 to 60	10.72 \pm 1.15	9.55 \pm 1.15	8.13 \pm 1.23	5.25 \pm 1.20
60 to 70	9.33 \pm 1.17	9.77 \pm 0.32	8.71 \pm 1.26	6.31 \pm 1.20
>70	10.96 \pm 1.17	9.55 \pm 1.41	9.33 \pm 1.45	6.61 \pm 1.35

Table 1.3) Phase shift values (degrees) at harmonic numbers 1 to 4, determined by the PRBS exercise test technique, and categorised according to relative $\dot{V}O_{2\text{max}}$ ($\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) (mean \pm SD). The results are adapted from the work of Eßfeld et al. (1987).

Relative $\dot{V}O_{2\text{max}}$ ($\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$)	Harmonic number			
	1	2	3	4
<50	-28 \pm 8	-55 \pm 12	-80 \pm 15	-103 \pm 43
50 to 60	-27 \pm 8	-48 \pm 13	-60 \pm 14	-88 \pm 12
60 to 70	-34 \pm 10	-52 \pm 10	-72 \pm 12	-93 \pm 21
>70	-32 \pm 9	-52 \pm 14	-59 \pm 11	-89 \pm 28

Eßfeld et al. (1987) have shown that the PRBS exercise test technique provides a means of differentiating between subjects attaining different relative $\dot{V}O_{2\max}$ values on the basis of a submaximal assessment of relative $\dot{V}O_2$ kinetics.

It would appear that the PRBS exercise test technique has not previously been used to differentiate between subjects attaining lower $\dot{V}O_{2\max}$ or to evaluate the $\dot{V}O_2$ kinetics of young, healthy females. Similarly no studies have investigated the effect of endurance-type training on $\dot{V}O_2$ kinetics measured in the frequency domain.

1.6) Aims and objectives

The overall aim of the study is to consider whether a test used to measure $\dot{V}O_2$ kinetics in the frequency domain, could theoretically be applied to the field of sports science, and primarily to the evaluate of the cardiorespiratory fitness of patients participating in cardiac rehabilitation exercise programmes.

In order to achieve this the following objectives have been specified:

- To assess the variability of $\dot{V}O_2$ kinetics, determined by the PRBS exercise test, in young, healthy women,
- To establish the relationship between $\dot{V}O_2$ kinetics and the aerobic power of young, healthy women with moderate to low habitual activity levels,
- To evaluate the effects of an eight week (twenty-four session) endurance-type training programme on the $\dot{V}O_2$ kinetics of young, healthy women.

CHAPTER TWO

MATERIALS AND METHODS

2.1) Introduction

The equipment and methods described in Section 2.2 are common to all the exercise testing procedures. The exercise test protocols described in Section 2.3 however, are specific to the different investigations and will therefore be linked to particular chapters. Data analysis techniques are explained within Section 2.4, and Section 2.5 covers the general recruitment and monitoring of subjects.

2.2) Equipment

2.2.1) Respiratory measurements

2.2.1.1) Equipment description

Oxygen uptake measurements were made on a breath-by-breath basis using the cardiorespiratory exercise testing system. This system used dedicated breath-by-breath software (First Breath software, First Breath Inc., St Agatha, Ontario, Canada) to integrate a digital volume transducer connected to a ventilation measurement module (VMM-2, Interface Associates, Laguna Niguel, CA, USA), a volume turbine (adult volume turbine, Interface Associates, Laguna Niguel, CA, USA), and a respiratory mass spectrometer (MGA-1100, Marquette Electronics Inc., Milwaukee, WI, USA), with a PC-compatible computer forming a rapid response gas analyser. Subjects breathed into a volume turbine surrounded by a precision digital volume transducer, using a mouthpiece with bite blocks (c.f. figure 2.1). Flow rate and flow direction signals were detected by means of the volume transducer and a light-weight impeller blade housed within the turbine.

Finally, after the signals had been processed by the ventilation measurement module, flow rate and flow direction data were relayed to the computer via an interface and an analogue to digital (A/D) converter. As subjects breathed through the mouthpiece, expiratory gases were sampled via a sample line. The respiratory mass spectrometer analysed O₂, CO₂ and N₂ simultaneously. All the components of the sampled gas were separated on the basis of charge to mass ratio within an ion gun and then detected by an array of sensors. Expiratory gas concentration data were then relayed to the computer via the interface and A/D converter. The mouthpiece, flow cartridge, volume transducer and sample line were all housed within a saliva trap. A plastic vial was attached to the base of this trap and acted as a receptacle for saliva produced during the exercise tests. All the apparatus was supported on the head of the subject by means of an adjustable harness.

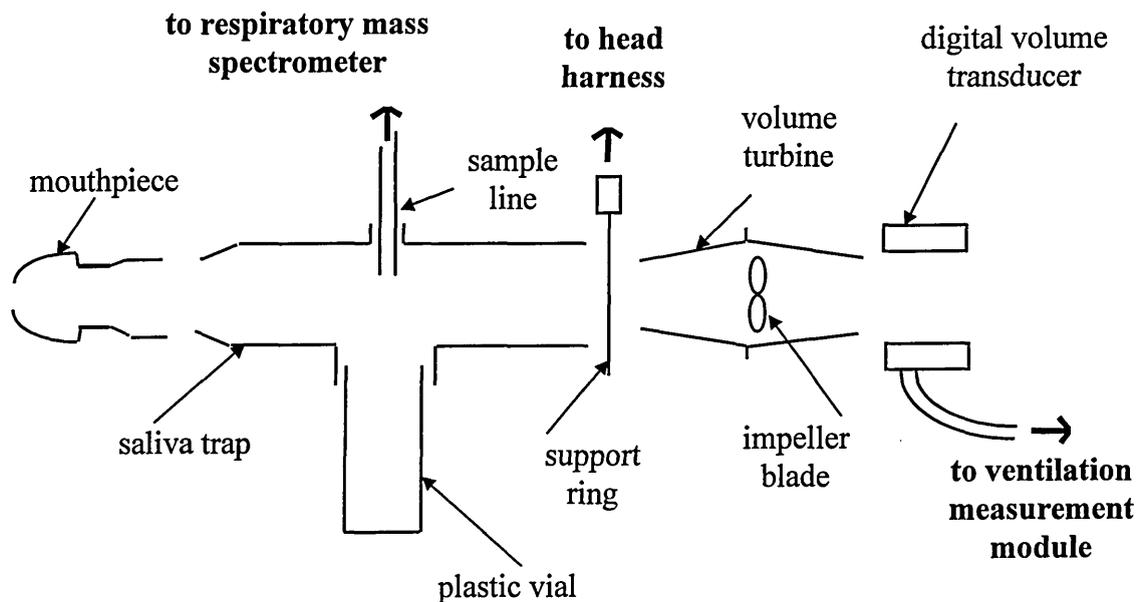


Figure 2.1) A schematic representation of the mouthpiece, saliva trap, volume turbine and digital volume transducer.

2.2.1.2) Calibration procedures

The volume turbine was calibrated prior to the study using a 3000 ml volume calibration syringe (Hans Rudolf Inc., Kansas City, Mo, USA). Flow rates approximating those which would occur during the exercise testing session were used to calibrate the turbine. Murphy et al. (1989) described flow rates of 1 to 2 l·s⁻¹ and 6 to 8 l·s⁻¹ for submaximal and maximal exercise respectively. A flow rate of 3 l·s⁻¹ was considered suitable for the calibration of the turbine when testing female subjects performing a PRBS exercise test followed by a progressive exercise test to exhaustion. Daily calibration checks ensured that the turbine measured gas volumes within ± 30 ml (± 1%) of the mean value (Murphy et al., 1989).

High tolerance (within ± 0.03 volume %) calibration gases (12% O₂ and 5% CO₂ - tank 1, and 21% O₂ and 0% CO₂ - tank 2) were used to calibrate the respiratory mass spectrometer on a daily basis.

Ambient temperature, humidity and barometric pressure were measured prior to each exercise test using a hygrometer (Griffin, England) and a barometer (Griffin and George Ltd., England) (c.f. appendix 2). In order to make comparisons between tests carried out under different atmospheric conditions a correction factor was applied to account for the effects of temperature, pressure and water vapour on the measured volumes. All volume measurements were corrected to Body Temperature and Pressure Saturated with water vapour (BTPS), that is a temperature of 310 K, ambient pressure and saturated water vapour at a partial pressure of 47 mmHg which is the vapour pressure of water at a body temperature of 37 °C (Fox et al., 1993). Standard Temperature and Pressure, Dry (STPD) was used for all metabolic calculations of $\dot{V} O_2$ and was calculated as a dry gas at a temperature of 273 K and a pressure of 760 mmHg.

An analyser lag-time calibration was performed daily to ensure correct alignment between the ventilation signal and the measurement of fractional gas concentrations. The ventilation measurement module produced a signal almost instantaneously. The signal from the respiratory mass spectrometer however was delayed by the time required to transport the gas to the analysers and the time response of the individual analysers. The time delay between the two signals is known as the lag-time. The lag-time measured during the lag-time calibration was used to align gas volume and concentration on a breath-by-breath basis.

2.2.1.3) Breath-by-breath calculation of oxygen uptake

The generation of breath-by-breath $\dot{V} O_2$ data is required to investigate the physiological responses to the rapid and slow work rate changes incorporated into the fifteen unit PRBS exercise test. The sensitivity of such a system to detect changes in $\dot{V} O_2$ is dependent on the response time of the gas analyser. As a result of its rapid sampling time (200 Hz), the respiratory mass spectrometer (MGA-1100, Marquette Electronics Inc., Milwaukee, WI, USA) is capable of generating $\dot{V} O_2$ data on a breath-by-breath basis and measuring the changes in $\dot{V} O_2$ which result from a pseudo random work rate forcing, thus providing an accurate assessment of $\dot{V} O_2$ kinetics.

Beaver et al. (1981) described a means of calculating true alveolar gas exchange which relies on the ability of a gas analysis system to measure both inspiratory ($\dot{V} I$) and expiratory ($\dot{V} E$) rates, and requires the use of a respiratory mass spectrometer. This method employs an estimate of the nominal lung volume (NLV) which represents the volume of the lung involved in gas exchange. Nominal lung volume however, does not represent any physical quantity, therefore functional residual capacity (FRC), which can

be estimated from age, height, weight and sex, is used as an equivalent to NLV. The NLV does not significantly affect the measurement of mean $\dot{V} O_2$, but does affect breath to breath fluctuations in $\dot{V} O_2$, thus reducing physiological noise.

The calculation of breath-by-breath $\dot{V} O_2$ at the alveolar surface, described by Beaver et al. (1981) is as follows:

$$\dot{V} O_2 = [(VIO_2 - VEO_2) - (dVL \times FO_2) - (dFO_2 \times NLV)] \times fb \dots \dots \dots (1)$$

when

- $\dot{V} O_2$ = rate of oxygen uptake
- VIO_2 = volume of inspired oxygen
- VEO_2 = volume of expired oxygen
- dVL = change in lung volume
- FO_2 = fractional concentration of oxygen
- dFO_2 = change in fractional concentration of oxygen
- NLV = nominal lung volume
- fb = breathing frequency

and where

$$dVL = [(VN_2 - NLV) \times dFN_2] / FN_2 \dots \dots \dots (2)$$

represents the change in lung volume on a breath-by breath basis,

when

- dVL = change in lung volume
- VN_2 = volume of nitrogen
- NLV = nominal lung volume
- dFN_2 = change in fractional concentration of nitrogen
- FN_2 = fractional concentration of nitrogen

On a breath-by-breath basis $\dot{V} N_2$ will fluctuate about a mean of zero and is calculated as follows:

$$VN_2 = VIN_2 - VEN_2 \dots \dots \dots (3)$$

when

VN_2 = volume of nitrogen

VIN_2 = volume of inspired nitrogen

VEN_2 = volume of expired nitrogen

A positive VN_2 value results when the VIN_2 is greater than VEN_2 and a negative VN_2 when VEN_2 is greater than VIN_2 .

The changes in fractional gas concentrations, for example dFN_2 and dFO_2 are estimated from changes in end tidal gas concentrations where:

$$dFN_2 = FN_2 - FpN_2 \dots \dots \dots (4)$$

when

dFN_2 = change in fractional concentration of nitrogen

FN_2 = fractional concentration of nitrogen

FpN_2 = fractional concentration of nitrogen in previous breath

and where:

$$dFO_2 = FO_2 - FpO_2 \dots \dots \dots (5)$$

when

dFO_2 = change in fractional concentration of oxygen

FO_2 = end tidal fractional concentration of oxygen

FpO_2 = end tidal fractional concentration of oxygen in previous breath

This method of calculating alveolar estimates of $\dot{V} O_2$ appreciably reduces the breath-

by-breath noise and enhances the underlying response characteristics of the signal (Barstow et al., 1996).

2.2.2) Heart rate measurements

2.2.2.1) Electrocardiography

A III-lead electrocardiogram (ECG) system (CASE 15 stress system, Marquette Electronics Inc., Milwaukee, WI, USA) and an acquisition module were used to collect heart rate data (in $\text{beats}\cdot\text{min}^{-1}$) at a digital sampling rate of $32,000\ \text{samples}\cdot\text{min}^{-1}$, over a measurement range of 30 to $300\ \text{beats}\cdot\text{min}^{-1}$. Left arm, left leg, right arm and right leg (earth) leads with grabber adapters were used to connect the subject to the acquisition module. Heart rate data was then relayed to the computer via the interface and the A/D converter. Before the attachment of the electrodes and leads to the chest, ethanol was used to clean any oil from the skin of the subject. An abrasive pad was then used to remove the epidermal skin layer at the electrode sites improving contact between the adhesive surface of the electrode and the skin. Four disposable, diaphoretic monitoring electrodes (Red Dot (Ag/AgCl), 3M, London, Ontario, Canada) were positioned on the chest (c.f. figure 2.2).

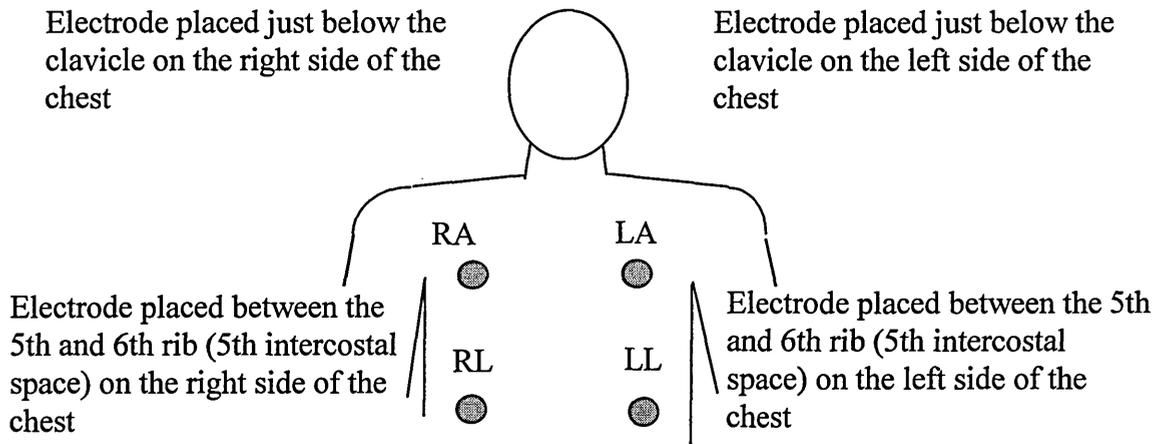


Figure 2.2) Diagram of the sites used for lead attachment and placement of the four electrodes. (RA = right arm lead, LA = left arm lead, RL = right leg lead (earth) and LL = left leg lead).

2.2.3) Ergometry

2.2.3.1) Equipment description

All exercise tests were performed in an upright position on an electrically-braked cycle ergometer, with a dynamic load range of 25 to 400 W, and at a cadence of 60 rev·min⁻¹ (Erg 550, Bosch, Berlin, Germany). The work rate was computer controlled by dedicated software (First Breath software, First Breath Inc., St Agatha, Ontario, Canada). The seat of the cycle ergometer was adjusted to a height equivalent to 96 to 100% trochanteric height in order to achieve optimal mechanical efficiency during cycling (Price et al., 1996). This seat height was maintained during subsequent exercise testing sessions.

2.2.3.2) Calibration procedure

Prior to commencement of the studies, the cycle ergometer was calibrated by applying a

voltage curve where one volt (1 V) applied to the cycle ergometer was equal to 100 W on the work rate controller (First Breath software). Following this initial procedure, daily checks were performed whereby the cycle ergometer was pedalled at a fixed cadence and against a fixed work rate in order to ensure that the signal from the ergometer was reported accurately to the work rate controller.

2.2.4) Anthropometry

The height (in m to the nearest 0.01 m) and body mass (in kg to the nearest 0.1 kg) of each subject were measured using a stadiometer and a set of upright scales (Avery, Birmingham, England) respectively.

2.3) Exercise protocols

An initial familiarisation session was conducted to introduce the subjects to the equipment and procedures adopted during the investigations. During this session, subjects were advised to abstain from strenuous exercise and the consumption of food within the two hour period before exercise testing. Loose sports clothing and training shoes were worn during all exercise tests.

2.3.1) PRBS exercise test

During the subsequent investigations, the PRBS exercise test technique involving 30 s changes between an upper and lower work rate was used to determine $\dot{V}O_2$ kinetics. A submaximal, four sequence PRBS protocol was programmed into the PC-compatible

computer by means of the work rate controller. This protocol consisted of a number of possible work rate changes between 25 and 75 W. The lower work rate level of 25 W was chosen to match the lower work rate limit of the cycle ergometer. An upper work rate of 75 W was chosen to elicit $\dot{V} O_2$ values below V_{ET} (Hoffmann et al., 1994).

Ventilatory threshold was measured using the V-slope method described by Beaver et al. (1986). Ventilatory threshold was located at the point where the curve of $\dot{V} CO_2$ against $\dot{V} O_2$ inflected upwards. This was equivalent to the point where $\dot{V} E \cdot \dot{V} O_2^{-1}$ inflected upwards whereas the curve for $\dot{V} E \cdot \dot{V} CO_2^{-1}$ remained constant or decreased, when using the Ventilatory Equivalent method. A four bit random number generator within the computer created a 30 s interval, pseudo random switching pattern between these pre-defined work rates. Each PRBS sequence consisted of fifteen possible work rate changes, or units, producing a sequence duration of 450 s (7.5 min), and thus a total test time of 1800 s (30 min). Due to the pseudo random nature of these protocols, subjects were unaware of the impending work rate changes. The work rate changes comprising this exercise test protocol are show in figure 2.3.

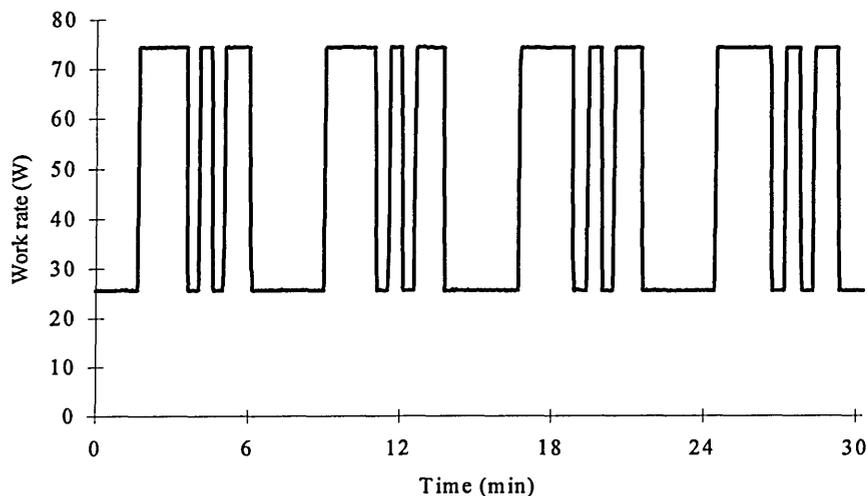


Figure 2.3) The fifteen unit PRBS exercise test protocol with work rate changes between 25 and 75 W.

One sequence of the PRBS exercise test protocol can be described in terms of the following seven stages:

- Stage 1 120 s (2 min) at a work rate of 25 W,
- Stage 2 120 s (2 min) at a work rate of 75 W,
- Stage 3 30 s (0.5 min) at a work rate of 25 W,
- Stage 4 30 s (0.5 min) at a work rate of 75 W,
- Stage 5 30 s (0.5 min) at a work rate of 25 W,
- Stage 6 60 s (1 min) at a work rate of 75 W,
- Stage 7 60 s (1 min) at a work rate of 25 W.

This exercise test protocol was used to investigate:

- a) the variability of the PRBS exercise test (c.f. Chapter 3),
- b) the relationship between aerobic power and $\dot{V} O_2$ kinetics (c.f. Chapter 4),
- c) the effect of endurance-type training on the $\dot{V} O_2$ kinetics of young, healthy women (c.f. Chapter 5).

2.3.2) Progressive exercise test to exhaustion

A ramp progressive exercise test to exhaustion was used to determine aerobic power (Zeballos and Weisman, 1994). This protocol began with a 3 min warm-up at 40 W, followed by ramp increases in work rate of $20 \text{ W}\cdot\text{min}^{-1}$ until volitional fatigue had been attained. This test end-point was reached when the pedal cadence fell below $40 \text{ rev}\cdot\text{min}^{-1}$ (Hughson et al., 1991). Strong verbal encouragement was given as the subjects approached exhaustion.

The gradient of the ramp was designed to result in a test duration of 8 to 12 min, with the incremental part of the protocol being completed within 6 to 12 min (Wasserman et al., 1994). The protocol for the progressive exercise test to exhaustion is shown in figure 2.4.

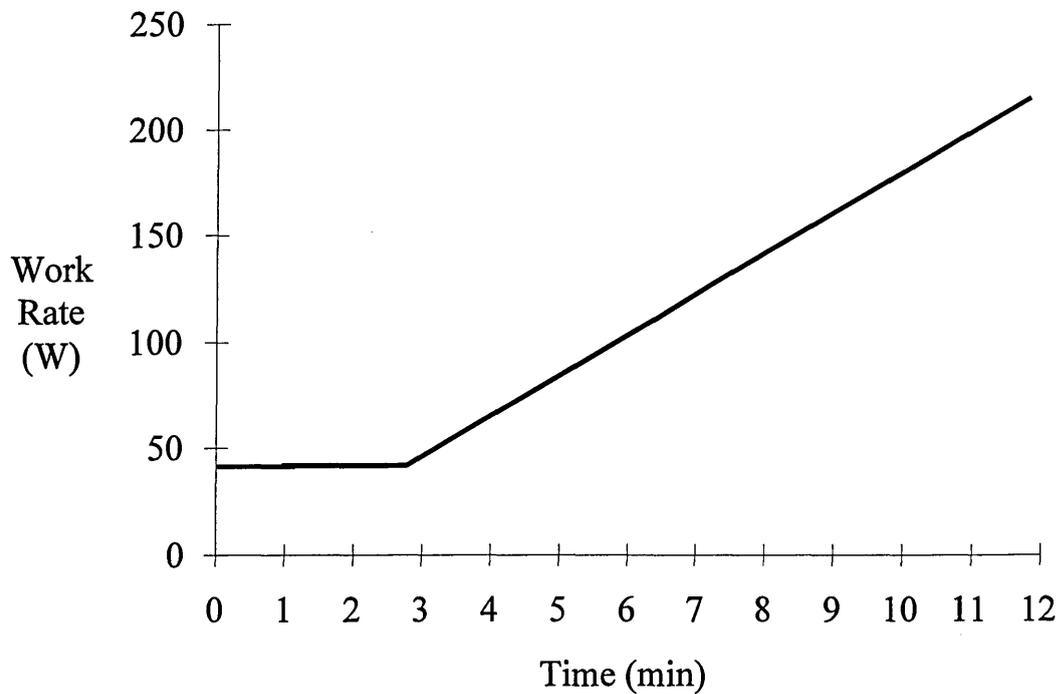


Figure 2.4) The progressive cycle ergometer exercise test protocol involving ramp work rate increase of $20 \text{ W}\cdot\text{min}^{-1}$ to exhaustion.

Finally, after performing the ramp progressive exercise test, all subjects completed a 5 min cool down period on the electrically braked cycle ergometer.

2.4) Data analysis

2.4.1) *Heart rate data*

2.4.1.1) PRBS exercise test

Heart rate data were calculated on a $\text{beats}\cdot\text{min}^{-1}$ basis. The heart rate response for the PRBS exercise test was calculated as the mean response to the last three sequences of this submaximal test.

2.4.1.2) Progressive exercise test to exhaustion

Heart rate data were calculated on a $\text{beats}\cdot\text{min}^{-1}$ basis and then averaged on a 30 s time basis using a spreadsheet software package (Excel V5, Microsoft Corporation, Redmond, WA, USA).

2.4.2) *Oxygen uptake data*

Preliminary analysis consisted of the removal of non-physiological breaths (incomplete or abnormal breaths) from the breath-by-breath data. Non-physiological breaths were incomplete breaths caused by coughing or swallowing. This process was performed using specialised data analysis software (First Breath software, First Breath, St Agatha, Ontario, Canada). All data files were stored on the fixed disk of the PC-compatible computer prior to further analysis.

2.4.2.1) Analysis of PRBS data

Software provided by the Department of Kinesiology at the University of Waterloo was used to analyse the data collected during the PRBS exercise tests (Hughson RL - personal communication).

The breath-by-breath data collected during each PRBS exercise test was linearly interpolated to produce data points at every 1 s interval within the time domain. The interpolated data were then split into the four pseudo random work rate sequences, which comprised each PRBS exercise test, and stored on the fixed disk of the PC-compatible computer as separate files containing 450 s of data.

In order to minimise the non-linearities previously associated with rest to work transitions in work rate (Hughson and Morrissey, 1983), the first sequence produced during the splitting procedure was used as a warm-up period and discarded from further data and statistical analyses.

Each of the remaining sequences were time-aligned prior to an ensemble averaging procedure. This mathematical process resulted in a single file consisting of 450 s of $\dot{V} O_2$ data. Ensemble averaging enhanced the signal to noise ratio, thus providing a more defined physiological response to the pseudo random work rate changes.

2.4.2.1.1) FREQUENCY DOMAIN ANALYSIS

The 450 s of $\dot{V} O_2$ data were transformed from the time to the frequency domain using a standard Fourier analysis technique (Eßfeld et al., 1987, and Hughson et al., 1989).

Oxygen uptake kinetics, namely phase shift (in degrees) and amplitude (in $\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$), were calculated for the relationship between the work rate input and

the resultant $\dot{V} O_2$ response. Harmonic number 0 (dynamic gain) represented the overall $\dot{V} O_2$ response over the 450 s of the pseudo random binary sequence. Only the harmonics of the original work rate forcing with periods of approximately 100 s or greater, i.e. harmonic numbers 1 to 4, were subjected to statistical analyses. The characteristics of these harmonics can be described in terms of the frequency and period of the sinusoidal wave of which they comprise (c.f. table 2.1).

Table 2.1) The characteristics (frequency in Hz and period in s) of the first four harmonics.

Harmonic number	Frequency of sinusoidal wave (Hz)	Period of sinusoidal wave (s)
1	0.0022	450
2	0.0044	225
3	0.0067	150
4	0.0089	112.5

By adopting this criterion, non-linearities in the relationship between $\dot{V} O_2$ and work rate, associated with higher frequencies of work rate changes (Eßfeld et al., 1991, Hoffmann et al., 1992, and Hoffmann et al., 1994), were minimised.

2.4.2.2) Analysis of data from the progressive exercise test

The breath-by-breath data were time-averaged on a 30 s basis using a spreadsheet software package (Excel V5, Microsoft Corporation, Redmond, WA, USA. Maximal oxygen uptake was said to have been achieved during the progressive exercise test to

exhaustion when a plateau in the relationship between $\dot{V} O_2$ and work rate was apparent (Zeballos and Weisman, 1994).

Peak oxygen uptake was said to have been attained if there was no plateau in the relationship between $\dot{V} O_2$ and work rate, but:

- a respiratory exchange ratio (RER) of 1.15 or above was apparent, or
- observed heart rate was within 10 beats·min⁻¹ of APMHR, calculated as 220 minus age (Zeballos and Weisman, 1994).

A plateau in the relationship between $\dot{V} O_2$ and work rate was identified using the method advocated by Rowland and Cunningham (1992). The mean of the increases in $\dot{V} O_2$ values were calculated during the final minute and during the previous 4 to 5 min of submaximal exercise. A plateau was said to have been attained when the change in $\dot{V} O_2$ during the final minute of exercise was less than 2 standard deviations (2 SD) below the mean of the increase in $\dot{V} O_2$ during the 4 to 5 min of submaximal exercise. Peak or maximal oxygen uptake was then defined as the highest 30 s time averaged $\dot{V} O_2$ achieved by each individual (Zeballos and Weisman, 1994).

2.5) Recruitment and monitoring methods

2.5.1) Health and activity questionnaires

Health and activity questionnaires were used to screen subjects for cardiovascular, respiratory and musculoskeletal disorders prior to their inclusion within the study population. These health and activity questionnaires were also used to ascertain

information concerning the amount and type of physical activity performed on a regular basis. The health and activity questionnaires used are shown in Appendix A.3.1 (the variability of the PRBS exercise test technique - c.f. Chapter 3) and A.3.3 (the effect of endurance-type training on the $\dot{V} O_2$ kinetics of healthy, young women - c.f. Chapter 5).

2.5.2) Consent forms

Prior to commencement of the studies, brief introductory sessions were conducted. During these sessions the study requirements were explained and written informed consent was obtained from each subject. The consent forms used are shown in Appendix A.3.2 (the variability of the PRBS exercise test technique - c.f. Chapter 3), A.3.4 and A.3.5 (the effect of endurance-type training on the $\dot{V} O_2$ kinetics of healthy, young women - c.f. Chapter 5).

2.5.3) Assessment of habitual activity levels

During the investigation into the effect of endurance-type training on the $\dot{V} O_2$ kinetics of healthy, young women (c.f. Chapter 5), the level of habitual activity performed by each subject was calculated from the health and activity questionnaire (c.f. appendix A.3.3). A variation of the guidelines and method advocated by the Allied Dunbar National Fitness Survey (1992), for the calculation of physical activity levels, was adopted to provide an indication of the activity levels of the total subject cohort.

Subjects provided an indication of the physical activities performed on a regular weekly basis. Each activity was then classified as being of a light (including walks consisting

of two or more miles at an average or slow pace, and social dancing where the individual was not out of breath or sweaty), moderate (including walks consisting of two or more miles at an brisk or fast pace, and football, aerobics, swimming, tennis and cycling where the individual was not out of breath or sweaty) or vigorous intensity (including hill walking at a brisk pace, and squash, aerobics, cycling, running, football and tennis, where the individual was out of breath or sweaty). A variation of the six point activity level scale, devised during the Allied Dunbar National Fitness Survey (1992), was used to determine the number of occasions during which each subject regularly performed moderate or vigorous activities lasting 20 min or more. The variation of the six point activity scale adopted to determine habitual activity levels is shown in table 2.2.

Subjects allocated an activity score of 5 were deemed to have high habitual activity levels. Conversely, subjects allocated activity scores of 1 or 0, performed little or no physical exercise.

Table 2.2) The six point activity scale used to determine the habitual activity levels of each subject.

Activity level	Activity of 20 min duration performed on a regular weekly basis
5	3 or more occasions of vigorous activity
4	3 or more occasions of a mix of moderate and vigorous activity
3	3 or more occasions of moderate activity
2	2 occasions of a mix of moderate and vigorous activity
1	1 occasion of moderate or vigorous activity
0	None

2.5.4) Monitoring of activity levels during the eight week training study

During the investigation into the effect of endurance-type training on the $\dot{V} O_2$ kinetics of healthy, young women (c.f. Chapter 5), the level of activity performed by non-training subjects during the eight week study period, was calculated from the weekly activity logs. An example of a weekly activity log is shown in Appendix A.3.6. The guidelines and methods advocated by the Allied Dunbar National Fitness Survey (1992), for the calculation of physical activity levels, was adopted to provide an indication of the activity levels of the non-training sub-group.

Subjects provided an indication of the physical activities performed on a weekly basis (recorded from Sunday to Sunday of each week). Each activity was then classified as being of a light, moderate or vigorous intensity, as previously described in Chapter 2, Section 2.5.3. The six point activity level scale, devised during the Allied Dunbar National Fitness Survey (1992), was used to determine the number of occasions, within a four week period, during which each subject performed moderate or vigorous activities lasting 20 min or more. The eight week study period was divided into four week periods consisting of weeks 1 to 4 and weeks 5 to 8 in order to apply the Allied Dunbar six point activity scale (Allied Dunbar National Fitness Survey, 1992). This six point activity scale is shown in table 2.3.

Subjects allocated an activity score of 5 were deemed to have high activity levels.

Conversely, the subjects allocated activity scores of 1 or 0 performed little or no physical activity during the eight week study period.

Table 2.3) The six point activity scale used to determine the activity levels of each subject in the non-training sub-group.

Activity level	Activity of 20 min duration performed during a four week period
5	12 or more occasions of vigorous activity
4	12 or more occasions of a mix of moderate and vigorous activity
3	12 or more occasions of moderate activity
2	5 to 11 occasions of a mix of moderate and vigorous activity
1	1 to 4 occasions of a mix of moderate and vigorous activity
0	None

CHAPTER THREE

THE VARIABILITY OF THE RESPIRATORY RESPONSES TO THE PRBS EXERCISE TEST

3.1) Introduction

The variability of $\dot{V}O_2$ measurements during exercise has been assessed using a variety of statistical methods (Froelicher et al., 1974, Nordrehaug et al., 1991, Shaw et al., 1994 and Atkinson, 1995).

Traditionally coefficients of variation (%CV), accompanied by a Student's t-test, have been used to show no significant test / re-test variability for replicate measures of aerobic fitness determined during maximal exercise tests. An extension of this approach is the application of the two-way analysis of variance with repeated measures technique, which provides a means of investigating any interaction between the test results and the number of tests performed (Altman, 1991).

Froelicher et al. (1974) studied the variability of relative $\dot{V}O_{2max}$ determined during two frequently adopted maximal exercise tests, the Bruce (Bruce et al., 1973) and the Balke (Balke and Ware, 1959) treadmill exercise tests. Healthy male volunteers performed each of the two exercise tests a total number of three times in an individually randomised order. Each subject completed one treadmill test per week on approximately the same day and at the same time. Gas volume measurements were made at intervals of 1 min using a Tissot spirometer, corrected for temperature and pressure. A Student's paired t-test revealed no significant difference between replicate measurements of relative $\dot{V}O_{2max}$ determined during either the Bruce or the Balke treadmill exercise tests. Froelicher et al. (1974) reported therefore that the measures of relative $\dot{V}O_{2max}$ made during the two tests were reproducible. Low variability, in the form of small coefficients of variation, was apparent for both exercise tests (%CV for Bruce test = 4.4%, %CV for Balke test = 5.8%) although some degree of intra-subject

variability was observed (%CV range for Bruce test = 1.2 to 8.5%, %CV range for Balke test = 0.6 to 11.1%). The larger coefficient of variation and greater degree of intra-subject variability associated with the Balke treadmill exercise test could indicate that this test produced less reliable measures of relative $\dot{V}O_{2max}$.

Nordrehaug et al (1991) investigated the variability of absolute $\dot{V}O_{2max}$ determined during the Bruce treadmill exercise test (Bruce et al., 1973). Two maximal Bruce exercise tests were performed by healthy male subjects at least two weeks apart.

Expiratory gas analysis was performed continuously using an ergospirometer to produce values for O_2 , CO_2 and respiratory volumes on a 30 s time basis. In agreement with the findings of Froelicher et al. (1974), the Bruce exercise test was found to be a reproducible method of determining absolute $\dot{V}O_{2max}$ (%CV = 5%). Again despite the small coefficient of variation associated with this method of measuring absolute $\dot{V}O_{2max}$, there was some degree of intra-subject variability, with Nordrehaug et al. (1991) reporting an intra-subject variability range of 1 to 13%.

Although coefficients of variation have been used previously to describe the variation in exercise test results, they are only valid if the measurement error is dependent upon the magnitude of the results (Bland, 1995). If that relationship is not found, other techniques should be used to assess variability.

Correlation coefficients (r) and intra-class correlations (R) have also been used to assess the variability of exercise tests by examining the relationship between replicate test results.

Nordrehaug et al. (1991) examined the intra-subject variability of absolute $\dot{V}O_{2max}$ values determined during replicate Bruce treadmill exercise tests. The Pearson Product

Moment correlation coefficient, representing the association between replicate absolute $\dot{V}O_{2\max}$ values, was given as $r = 0.94$ ($P < 0.05$).

Cohen-Solal et al. (1991) investigated the variability of relative $\dot{V}O_{2\text{peak}}$ measured during 1) a symptom-limited progressive cycle ergometer exercise test involving step changes in work rate of 20 W every 3 min, and 2) a symptom-limited cycle ergometer exercise test involving ramp changes in work rate of $10 \text{ W}\cdot\text{min}^{-1}$. Patients with mild, stable chronic congestive heart failure and exercise limitation due to fatigue or dyspnoea performed the two cycle ergometer tests on two occasions during an eight day period. Respiratory gas analysis was performed using a variety of fast response gas analysers at a number of different research centres. Following the determination of relative $\dot{V}O_{2\text{peak}}$, defined as the highest $\dot{V}O_2$ value reached at the end of exercise, the variability of the two exercise tests was examined. No significant differences were found to exist between replicate measures of relative $\dot{V}O_{2\text{peak}}$ determined by either of the two cycle ergometer tests. A close association was found to exist between replicate measures of relative $\dot{V}O_{2\text{peak}}$ measured during the step exercise test ($r = 0.93$) and the ramp exercise test ($r = 0.97$). Cohen-Solal et al. (1991) concluded that highly reproducible relative $\dot{V}O_{2\text{peak}}$ values resulted from both exercise tests when performed by patients with mild, stable chronic congestive heart failure.

The usefulness of the information gained by describing variability in terms of correlation coefficients or intra-class correlations is questionable. These methods provide a measure of the extent of a relationship between data sets but do not provide a good assessment of the agreement between the results produced during identical protocols (Bland and Altman, 1995). The inability of these approaches to provide reliable assessments of the agreement between test results arises from the fact that

correlation coefficients and intraclass correlations can be influenced by the characteristics of a subject population.

Bland and Altman (1986) advocated the use of the 95% limits of agreement method to assess the repeatability of test results obtained during method comparison studies (Lee, 1992, Shaw et al., 1994, and Liehr et al., 1995). Altman (1991) and Bland (1995) have also advocated this method as a means of assessing the extent of the agreement between the test and re-test results obtained during the performance of identical protocols.

The usefulness of the different statistical methods which have been applied to the assessment of the variability of replicate test results has been investigated by Atkinson (1995). Two repeated measurements of isometric leg strength were taken in two different study populations using a portable leg and back dynamometer. Population one was a homogenous cohort of young athletic subjects (age = 18 to 28), whereas population two was a heterogenous cohort of older subjects (age = 47 to 65 years). Coefficients of variation, intraclass correlations and 95% limits of agreement were calculated for the test / retest results. Greater variability in leg strength was found within the older subject population (%CV = 21.1%) when compared to the young athletic population (%CV = 18.6%). Contrasting findings were evident when considering Pearson Product Moment correlation coefficients and intraclass correlations. Greater variability in repeated leg strength measurements was found to occur in the homogenous young athletic population ($r = 0.01$, $P = 0.78$, and $R = 0.26$) when compared to the heterogenous older population ($r = 0.69$, $P = 0.03$, and $R = 0.83$). This finding emphasises the limitations of correlation coefficients and intraclass correlations to evaluate the variability of replicate leg strength measurements, and

demonstrates the effect of population characteristics on these two statistical methods. When considering the 95% limits of agreement for the two subject populations, greater variability in replicate test results was observed in the older subject population. The 95% limits of agreement were wider for the heterogenous older population (95% limits of agreement = -657 to +743 N) than for the homogenous young athletic population (95% limits of agreement = -610 to +613N). Atkinson (1995) concluded that as agreement limits were not affected by population heterogeneity, this statistical method could be adopted as a universal means of assessing the intra-subject variability of replicate test results.

There is limited knowledge however concerning the variability of replicate measures of $\dot{V}O_2$ kinetics determined by the PRBS exercise test technique. Berry and Moritani (1985) assessed the variability of $\dot{V}O_2$ kinetics measured during two identical square wave work rate forcings. Ten young male college students performed a single step change from rest to a work rate of 150 W at a pedal cadence of 50 rpm, to determine the time constant of $\dot{V}O_2$. Replicate measures of $\dot{V}O_2$ kinetics were made using an identical protocol two weeks after performing the initial exercise test. A Pearson Product Moment correlation coefficient was calculated to examine the variability of the $\dot{V}O_2$ kinetics measured during the step change in work rate. The reproducibility of this exercise test was described as being satisfactory with a correlation coefficient of $r = 0.87$ ($P < 0.01$) existing between replicate measures of $\dot{V}O_2$ kinetics.

The reproducibility of $\dot{V}O_2$ kinetics from ramp work rate tests was investigated by Hughson and Inman (1986) in five male subjects. Individual subjects showed approximately two-fold differences in estimates of $\dot{V}O_2$ kinetics (%CV from individuals

repeating the test six times ranged from 18.5 to 29.3%). It was concluded that several repetitions should be pooled prior to estimation of kinetic parameters.

Claxton et al. (1996) (Appendix A.1.4) examined the reliability of a sixty-three unit PRBS exercise test with 5 s work rate changes between 25 and 105 W to measure $\dot{V}O_2$ kinetics. Twenty healthy male subjects performed two identical PRBS exercise tests on an electrically braked cycle ergometer. Respiratory gas exchange was measured on a breath-by-breath basis using a respiratory mass spectrometer. An assessment of intra-subject variability for amplitude and phase shift was made using the analysis of variance technique, and limits of agreement were calculated in order to assess how well the results agreed. Wide limits of agreement were observed at each harmonic number for both amplitude and phase shift, although there was no significant difference between the $\dot{V}O_2$ kinetics resulting from the two replicate tests.

In a review of the literature no evidence could be found of any study which described the intra-subject variability of the test / re-test results determined during a PRBS exercise test with 30 s work rate changes between 25 and 75 W.

The aim of this investigation was to:

- investigate the variability of replicate measures of $\dot{V}O_2$ kinetics made during a fifteen unit PRBS exercise test protocol with 30 s work rate changes between 25 and 75W.

3.2) Methodology

3.2.1) Subjects

Eight healthy, moderately active, female subjects (age = 22.6 ± 0.8 years, height = 1.65

± 0.06 m and body mass = 65.5 ± 4.5 kg) were recruited from the student population at Sheffield Hallam University. Prior to inclusion into the study population, all subjects were screened for cardiovascular, respiratory and musculo-skeletal disorders by means of a health and activity questionnaire (c.f. appendix A.3.1). Written informed consent was obtained from each individual prior to commencement of the study (c.f. appendix A.3.2).

3.2.2) Exercise testing

An eight (subjects) by two (observations) study design was adopted in order to investigate the variability of the PRBS exercise test. All eight subjects completed two replicate PRBS exercise tests (c.f. Chapter 2, Section 2.3.1) to determine $\dot{V}O_2$ kinetics. The exercise tests were performed on the same day of the week and at the same time for each subject (Froelicher et al., 1974) in order to reduce diurnal variation. Each subject completed one PRBS exercise test per week and all tests were performed within a period of two weeks.

Oxygen uptake and heart rate were measured throughout this exercise testing procedure (c.f. Chapter 2, Section 2.2.1 and 2.2.2). Individual seat height was recorded during the initial PRBS exercise test and maintained for the subsequent assessment of $\dot{V}O_2$ kinetics.

3.2.3) Data and statistical analysis

The breath-by-breath data collected during the PRBS exercise tests were subjected to

Fourier analysis to produce $\dot{V}O_2$ kinetics at harmonic numbers 1 to 4 (c.f. Chapter 2, Section 2.4.2.1).

Descriptive statistics, i.e. the mean and standard deviation (mean \pm SD), were calculated for $\dot{V}O_2$ kinetics determined during each PRBS exercise test. Histograms were plotted to ensure that the data points were normally distributed and occurred within ± 2 SD of the mean value.

An assessment of intra-subject variability of the PRBS exercise test was made by means of a repeated measures two-way analysis of variance with replication. The observed F statistics were calculated in each case and compared to tabulated values for the F distribution. Significant differences in the replicate measures of $\dot{V}O_2$ kinetics were said to exist if the observed F statistics were greater than the tabulated values (F_{crit}). A significance level of $\alpha \leq 0.05$ was selected.

An error of linearity test was performed where Pearson Product Moment correlation coefficients were used to assess the extent of the relationship between the measurement error and the measurement size. If a significant association was found, indicating that the error of the measurement was dependent on the size of the result, a coefficient of variation was calculated to quantify the variability of the test results.

Limits of agreement (95%) were calculated to provide an assessment of the repeatability of this PRBS exercise test (Bland and Altman, 1995, and Atkinson, 1995). The 95% limits of agreement method involved the calculation of the mean difference and the standard deviation of the differences (mean $\pm S_{diff}$) between the replicate measures of $\dot{V}O_2$ kinetics. This statistical process provided a range of values within which 95% of

the differences between the two tests would occur.

3.3) Results

3.3.1) Overall results

A typical response to the PRBS exercise test is shown in figure 3.1.

Dynamic gains for $\dot{V}O_2$ of $1046 \pm 60 \text{ ml}\cdot\text{min}^{-1}$ and $1024 \pm 60 \text{ ml}\cdot\text{min}^{-1}$ resulted from test 1 and test 2 respectively. No significant difference was found between the dynamic gain for $\dot{V}O_2$ measured during test 1 and test 2. A mean overall heart rate response of $115 \pm 5 \text{ beats}\cdot\text{min}^{-1}$ and $115 \pm 3 \text{ beats}\cdot\text{min}^{-1}$ resulted from test 1 and test 2 respectively.

No significant difference was found between the mean heart rate response resulting from test 1 and test 2.

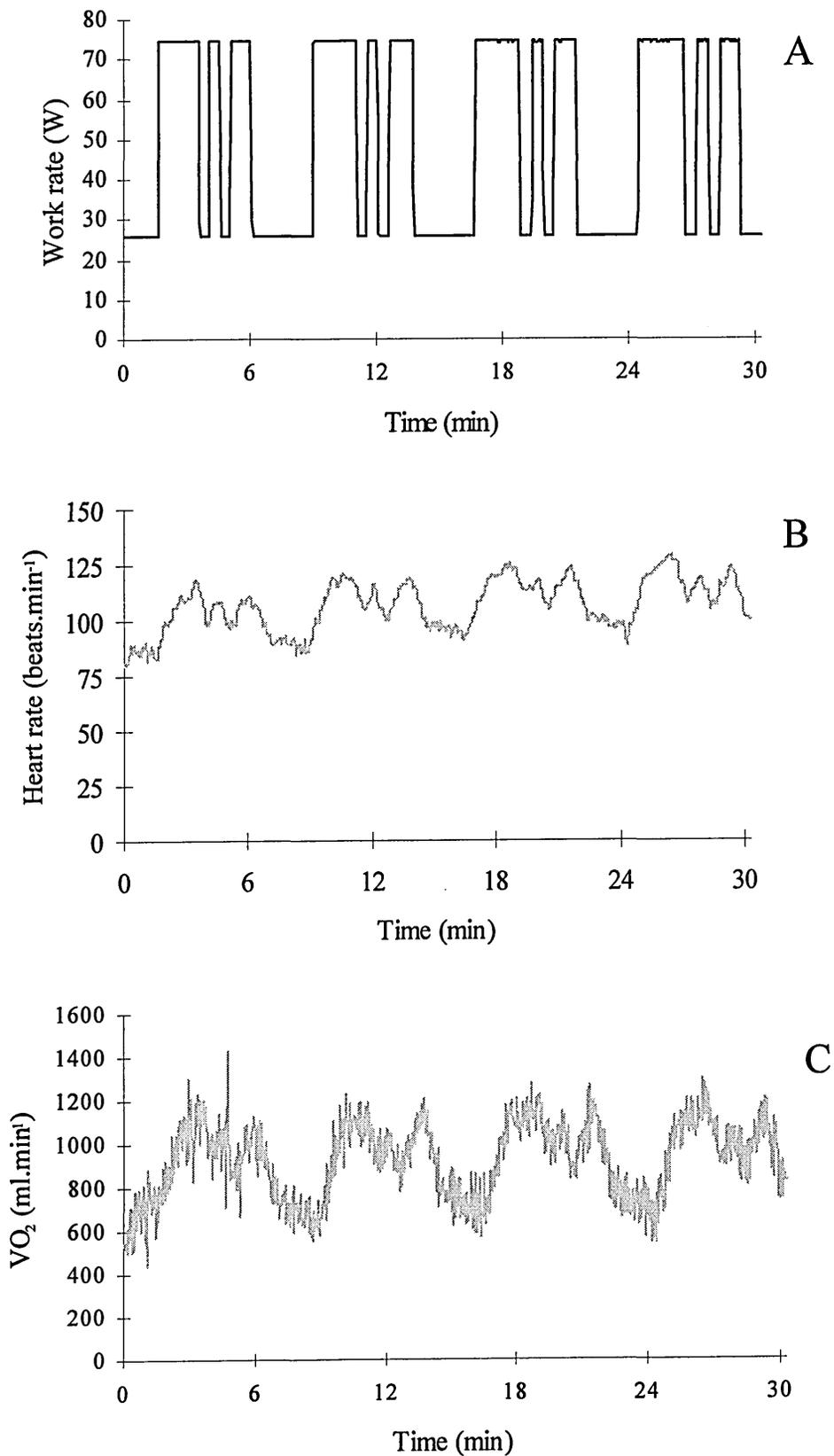


Figure 3.1) The PRBS exercise test; (A) work rate sequences, (B) a typical heart rate response and (C) a typical $\dot{V}O_2$ response.

3.3.2) Oxygen uptake kinetics

3.3.2.1) Descriptive statistics

The descriptive statistics for the $\dot{V}O_2$ kinetics determined during replicate PRBS exercise tests are displayed in table 3.1 and 3.2 and presented graphically in figures 3.2 and 3.3.

Table 3.1) Descriptive statistics for amplitude ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$) determined during replicate PRBS exercise tests (mean \pm SD) (n = 8).

Harmonic number	Amplitude ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$)	
	Test 1	Test 2
1	9.498 \pm 0.490	9.812 \pm 0.686
2	6.752 \pm 0.434	6.550 \pm 0.326
3	7.958 \pm 0.912	7.836 \pm 0.851
4	8.053 \pm 0.491	8.105 \pm 0.707

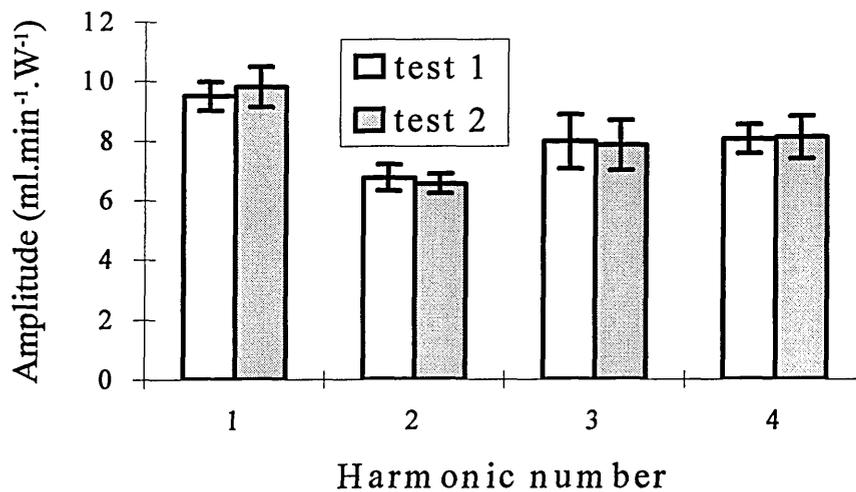


Figure 3.2) Mean values (mean \pm SD) for amplitude ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$) obtained during replicate PRBS exercise tests.

Table 3.2) Descriptive statistics for phase shift (degrees) determined during replicate PRBS exercise tests (mean \pm SD) (n = 8).

Harmonic number	Phase shift (degrees)	
	Test 1	Test 2
1	-23.3 \pm 4.2	-22.5 \pm 4.5
2	-40.8 \pm 4.3	-40.8 \pm 4.9
3	-71.9 \pm 9.4	-67.3 \pm 5.3
4	-76.0 \pm 7.9	-76.9 \pm 8.3

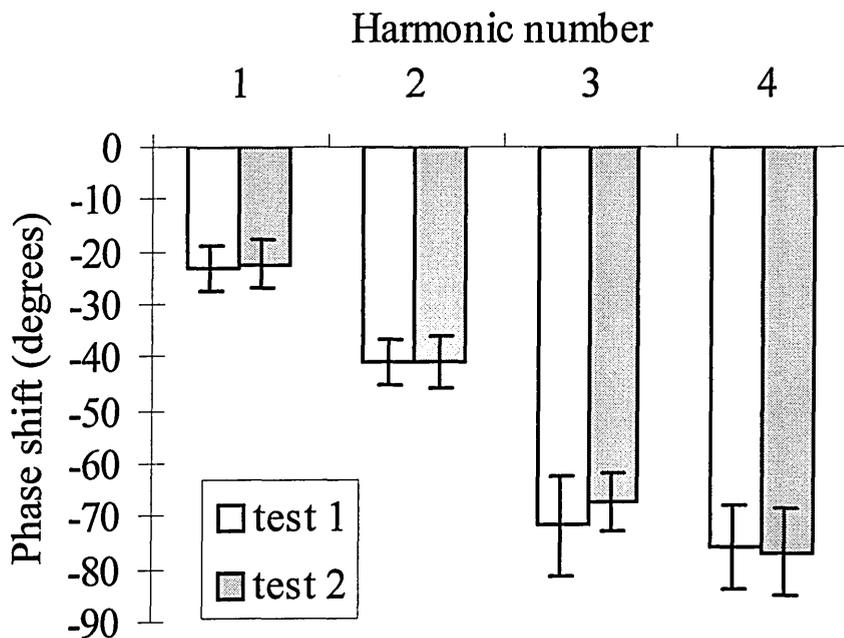


Figure 3.3) Mean values (mean \pm SD) for phase shift obtained during replicate PRBS exercise tests.

3.3.2.2) Two-way analysis of variance

A repeated measures two-way analysis of variance with replication revealed no significant differences between test 1 and test 2 at harmonic numbers 1 to 4.

3.3.2.3) Limits of agreement

An error of linearity test showed no significant relationship between the measurement error and the measurement size for either amplitude or phase shift. Therefore coefficients of variation were not calculated.

The 95% limits of agreement for amplitude are summarised in table 3.3.

Table 3.3) Mean difference $(t_2-t_1) \pm 2S_{diff}$ and 95% limits of agreement for amplitude ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$).

Harmonic number	1	2	3	4
Amplitude:	0.314	-0.202	-0.122	0.052
mean difference	\pm	\pm	\pm	\pm
($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$)	0.890	0.718	0.476	1.512
95% limits of	-0.576	-0.920	-2.598	-1.459
agreement	to	to	to	to
($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$)	1.204	+0.515	+2.354	+1.564

The difference between the upper and lower 95% limits of agreement ranged from 1.435 $\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$ at harmonic number 2 to 4.952 $\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$ at harmonic number 3. The 95% limits of agreement for amplitude are presented graphically (Bland, 1995) in figures 3.4 and 3.5.

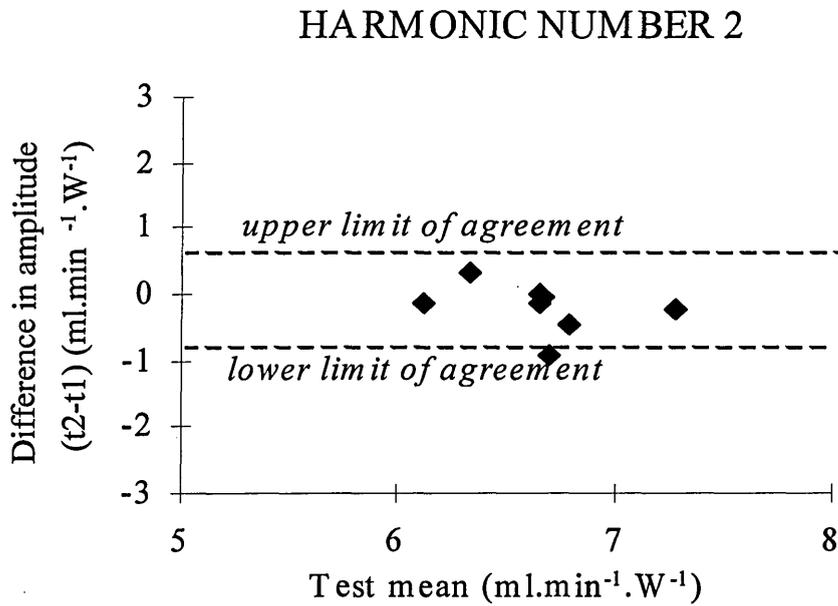
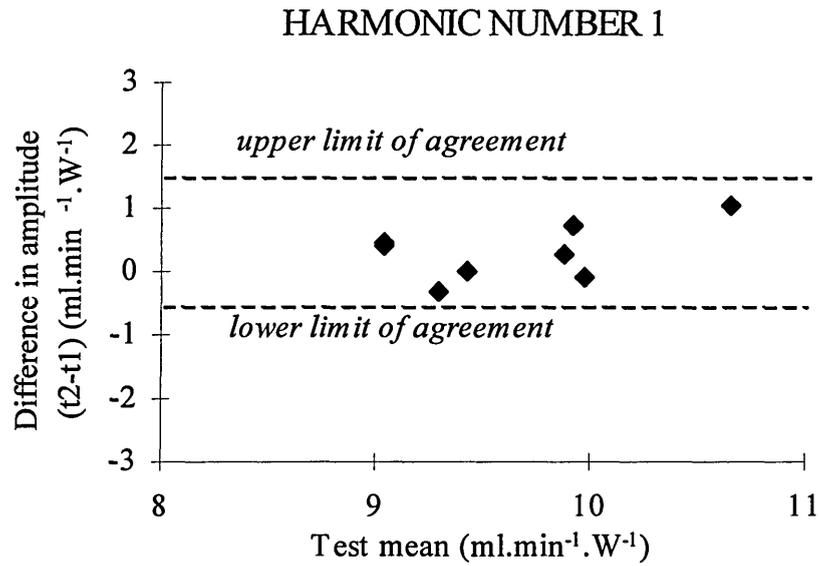


Figure 3.4) The difference between replicate measures of amplitude (t_2-t_1) (y-axis) and mean results of the two tests (x-axis) for each subject \blacklozenge at harmonic numbers 1 and 2.

95% limits of agreement for amplitude ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$) at harmonic numbers 1 and 2, where the upper and lower limits of agreement represent $\text{mean} + 2S_{\text{diff}}$ and $\text{mean} - 2S_{\text{diff}}$ respectively.

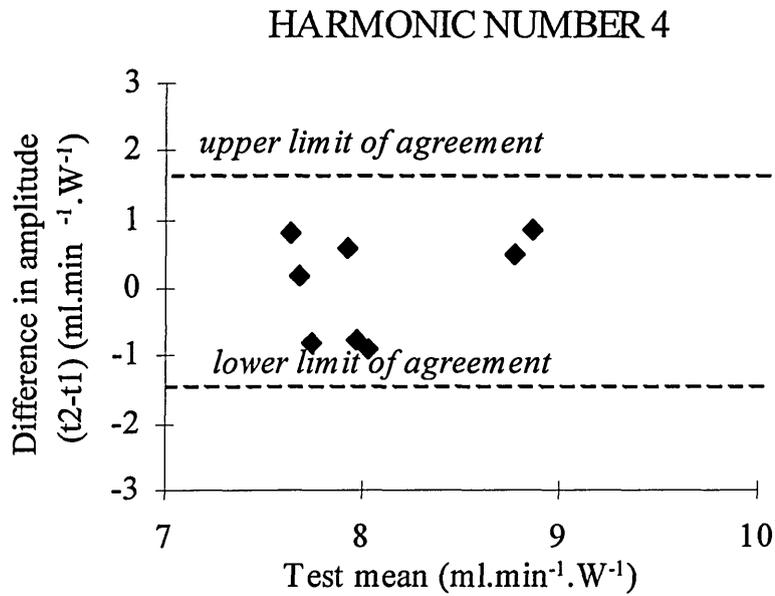
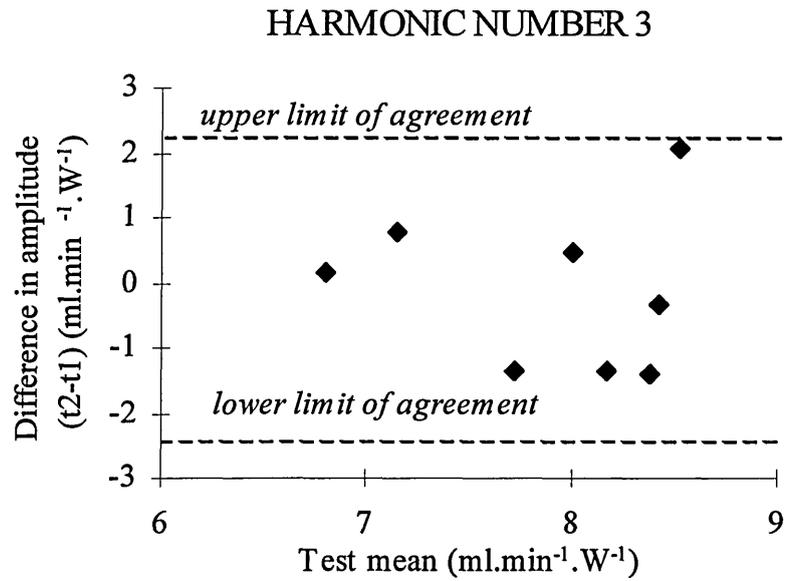


Figure 3.5) The difference between replicate measures of amplitude (t_2-t_1) (y-axis) and mean results of the two tests (x-axis) for each subject \blacklozenge at harmonic numbers 3 and 4.

95% limits of agreement for amplitude ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$) at harmonic numbers 3 and 4, where the upper and lower limits of agreement represent $\text{mean} + 2S_{\text{diff}}$ and $\text{mean} - 2S_{\text{diff}}$ respectively.

The 95% limits of agreement for phase shift are summarised in table 3.4.

Table 3.4) Mean difference $(t_2-t_1) \pm 2S_{diff}$ and 95% limits of agreement for phase shift (degrees).

Harmonic number	1	2	3	4
Phase shift:	-1.1	0	-4.8	1
mean difference	\pm	\pm	\pm	\pm
(degrees)	8.1	8.2	12.7	17.1
95% limits of	-9.2	-8.2	-17.5	-16.1
agreement	to	to	to	to
(degrees)	+7.0	+8.2	+7.9	+18.1

The difference between the upper and lower 95% limits of agreement ranged from 16.2 degrees at harmonic number 1 to 34.2 degrees at harmonic number 4. The 95% limits of agreement for phase shift are presented graphically (Bland, 1995) in figures 3.6 and 3.7.

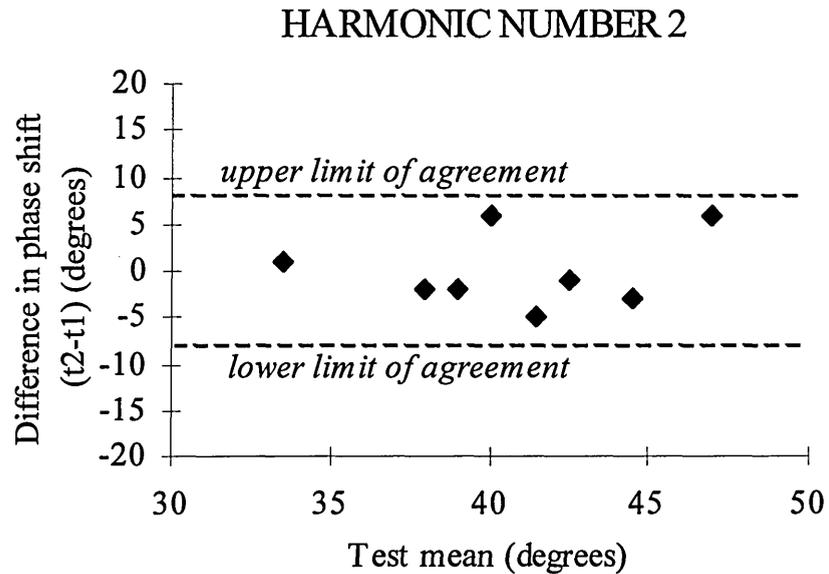
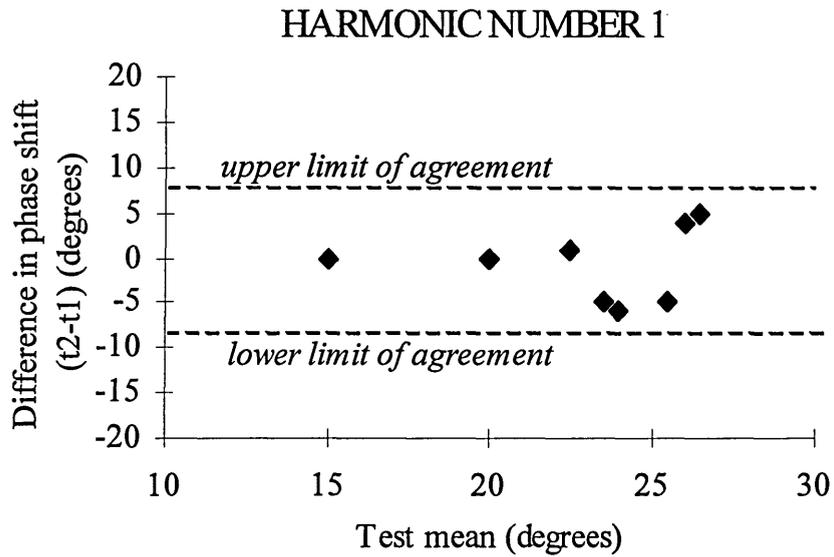


Figure 3.6) The difference between replicate measures of phase shift (t_2-t_1) (y-axis) and mean results of the two tests (x-axis) for each subject \blacklozenge at harmonic numbers 1 and 2. 95% limits of agreement for phase shift (degrees) at harmonic numbers 1 and 2, where the upper and lower limits of agreement represent $\text{mean} + 2S_{\text{diff}}$ and $\text{mean} - 2S_{\text{diff}}$ respectively.

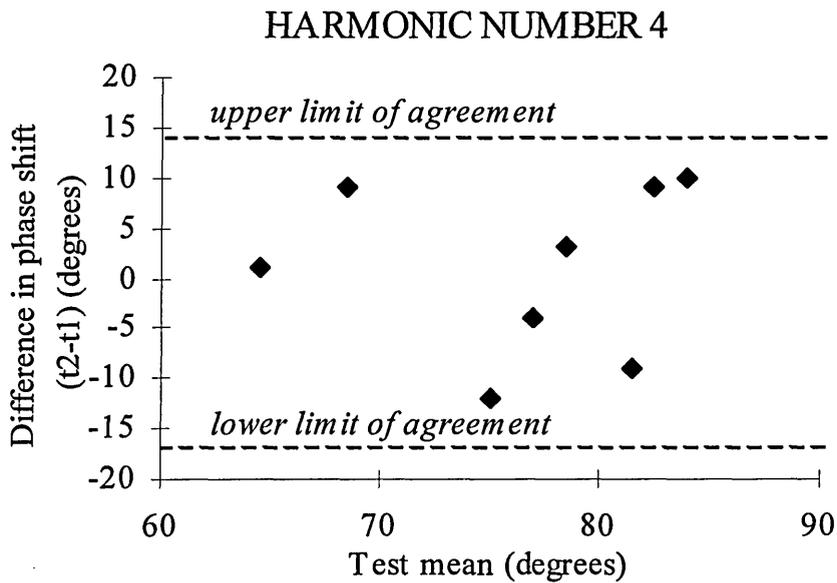
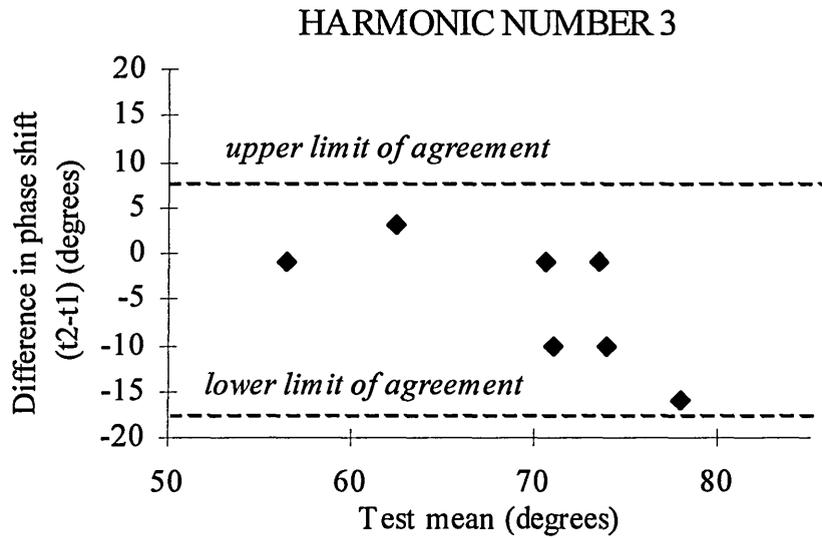


Figure 3.7) The difference between replicate measures of phase shift (t_2-t_1) (y-axis) and mean results of the two tests (x-axis) for each subject \blacklozenge at harmonic numbers 3 and 4. 95% limits of agreement for phase shift (degrees) at harmonic numbers 3 and 4, where the upper and lower limits of agreement represent $\text{mean} + 2S_{\text{diff}}$ and $\text{mean} - 2S_{\text{diff}}$ respectively.

3.4) Discussion

3.4.1) Overall response

All subjects completed two replicate PRBS exercise tests with 30 s work rate changes between 25 and 75 W. The dynamic gain for $\dot{V}O_2$ ranged from 889 to 1176 ml.min⁻¹.

This means that the overall exercise intensity was likely to be less than 50% $\dot{V}O_{2max}$ for this female population (Henritze et al., 1985, McCord and Paterson, 1989, and Astrand, 1960). The heart rate responses were also submaximal being approximately 55% APMHR.

3.4.2) Oxygen uptake kinetics comparisons

Although there are no comparative $\dot{V}O_2$ kinetics data, determined during the PRBS exercise test, for female subjects, the results are in a similar broad range to the values reported from other studies (Eßfeld et al., 1987, Cunningham et al., 1993 and Hughson – personal communication).

3.4.3) Variability

In assessing variability, the mean difference between measurements obtained in the two tests is important since it is an estimate of the average bias of the measurement. Large mean differences, which would indicate the existence of undesirable trends (Katch et al., 1982), were not detected in this study. A previous study (unpublished data) investigated the reproducibility of the measurement of $\dot{V}O_2$ kinetics in four subjects who performed four replicate PRBS exercise tests. A repeated measures two-way analysis of variance

with replication revealed no significant difference between any of the parameters measured during the four tests. Further examination of both individual and mean differences did not identify any particular upward or downward trends. It was assumed therefore that the variability of this study was not due to the nature of the testing procedure or to the influence of habituation.

Although no significant difference was found to exist between the replicate test results at any of the harmonic numbers, wide limits of agreement, indicating a large degree of variability between the test / re-test results, were evident. This would suggest that it is more appropriate to apply the PRBS exercise test to the study of groups of subjects than to draw conclusions from a single test in individual subjects.

A similar conclusion was drawn in a study by Claxton et al. (1996) (c.f. Appendix A.1.4) for male subjects performing sixty-three unit PRBS exercise tests. Other studies have also shown substantial degrees of individual variation in both steady state $\dot{V}O_2$ and $\dot{V}O_2$ kinetics (Armstrong and Costill, 1985, Hughson and Inman, 1986, Nordrehaug et al., 1991, and Morgan et al., 1991).

One explanation for the wide intra-subject variability in replicate measures of $\dot{V}O_2$ kinetics is biovariation (Katch et al., 1982) which can affect even highly regimented exercise testing procedures. Biovariation can comprise a number of factors which may dictate the contribution made by random error to the process of determining $\dot{V}O_2$ kinetics. Psychological factors including anxiety prior to, and during the performance of unfamiliar procedures (British Association of Sports Science, 1988) may have altered the cardiovascular responses to the PRBS exercise test protocols. Theoretically, a lessening of the contribution made by these factors to the error inherent within the

measurement procedure, should have occurred with the performance of subsequent tests. Greco et al. (1986) suggested that sudden alterations in exercise intensity, even when incorporated into a pseudo random work rate forcing, could act as a signal to the subject to voluntarily influence the resultant cardiorespiratory responses. This would ultimately contribute to the random variation within the $\dot{V}O_2$ response to replicate PRBS exercise test protocols.

Complex analysis procedures were adopted to produce $\dot{V}O_2$ kinetics from the $\dot{V}O_2$ response to the pseudo random work rate forcings. Although the removal of the first PRBS sequence reduced the initial transients, characteristic of rest to work transitions (Krogh and Linhard, 1913), variability between repeated measures of $\dot{V}O_2$ kinetics may have been introduced through breath-by-breath noise (Lamarra et al., 1987).

In order to reduce breath-by-breath noise, non-physiological breaths were removed and three sequences were overlaid to produce an average response prior to Fourier analyses. In spite of this, the interpolation of the $\dot{V}O_2$ data must be considered as a source of possible variation. The interpolation process resulted in the production of a data point at every 1 s interval. Each data point was calculated from the $\dot{V}O_2$ during the previous and following breath. Thus it can be seen that this stage of the analysis process was dependent on the breathing pattern of each individual. It is proposed that a greater degree of variability between replicate measures of $\dot{V}O_2$ kinetics may have existed for slower breathing individuals. When fewer breaths were taken, more data points were introduced by the process of interpolation. Variations within the breathing pattern will have occurred during the performance of each PRBS exercise test. Therefore when comparing the $\dot{V}O_2$ kinetics produced during two separate PRBS exercise tests, slightly different measures of amplitude and phase shift will have resulted.

3.5) Conclusions

- The individual variability of the PRBS exercise test has been shown to be considerable for young, adult female subjects.
- The results from several repetitions of the PRBS exercise test should be pooled to give a more reliable estimate of $\dot{V}O_2$ kinetics for any individual subject.
- The test could be usefully applied to the assessment of group $\dot{V}O_2$ kinetics.

CHAPTER FOUR

THE RELATIONSHIP BETWEEN OXYGEN UPTAKE KINETICS AND AEROBIC POWER

4.1) Introduction

It has been suggested for some time that subjects with greater aerobic power have faster rates of adaptation to step changes in work rate, i.e. faster $\dot{V}O_2$ kinetics (Whipp and Wasserman, 1972, Weltman and Katch, 1976, Hickson et al., 1978 and Hagberg et al., 1978). Weltman and Katch (1976), for example, were able to demonstrate a difference in $\dot{V}O_2$ kinetics between two groups of subjects, where one group had attained a higher mean relative $\dot{V}O_{2max}$ ($60 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) than the other group ($50 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$). The subjects attaining lower aerobic power were found to have a mean half time for $\dot{V}O_2$ ($t_{1/2}$) which was 6 s greater than that for the subjects attaining higher aerobic power. At the time of these studies however, the concept of including a time delay in the analysis of $\dot{V}O_2$ kinetics was not well accepted. The half times for $\dot{V}O_2$ published by the above authors may therefore describe both the initial, rapid cardiodynamic response (phase I), where $\dot{V}O_2$ at the lungs does not reflect that at the muscles, and the exponential increase in $\dot{V}O_2$ (phase II), where $\dot{V}O_2$ at the lungs does reflect that at the muscles.

It was not until 1985 that a relationship was established between phase II $\dot{V}O_2$ kinetics and aerobic power (Powers et al., 1985). Oxygen uptake kinetics were assessed in ten endurance trained male athletes using a step change in work rate requiring approximately 50% of their aerobic power, determined during a progressive exercise test to exhaustion. The relative $\dot{V}O_{2max}$ and $\dot{V}O_2$ kinetics ranged from 50 to 70 $\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ and 36.0 to 21.6 s, respectively. They described a significant correlation between $\dot{V}O_{2max}$ and $\dot{V}O_2$ kinetics ($r = -0.80, P < 0.05$) and successfully demonstrated

that subjects with greater aerobic power had faster $\dot{V}O_2$ kinetics. The following model was found to describe the half time for $\dot{V}O_2$:

$$\dot{V}O_2 \text{ kinetics (t/2, in s)} = -0.544(\text{relative } \dot{V}O_{2\text{max}}(\text{in ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1})) + 59 \quad (6)$$

The coefficient of determination for the model ($1-r^2$) revealed that 36% of the common variance between $\dot{V}O_{2\text{max}}$ and $\dot{V}O_2$ kinetics could not be explained by this model. As subjects differed in aerobic power but had performed similar training routines in the ten weeks prior to testing, it was suggested that the difference in $\dot{V}O_2$ kinetics was due to initial fitness level rather than training.

Eßfeld et al. (1987) present the only data where the PRBS exercise test technique has been used to investigate the $\dot{V}O_2$ kinetics of subjects differing in aerobic power. Male and female subjects were grouped into four categories of fitness (group 1: $\dot{V}O_{2\text{max}} < 50 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$, group 2: $\dot{V}O_{2\text{max}} = 50 \text{ to } 60 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$, group 3: $\dot{V}O_{2\text{max}} = 60 \text{ to } 70 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$, and group 4: $\dot{V}O_{2\text{max}} > 70 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$). Phase shift parameters demonstrated a trend towards faster $\dot{V}O_2$ kinetics in those subjects with greater aerobic power. Interestingly, amplitude parameters were shown to discriminate between subjects in different aerobic power groupings.

Since previous investigations have predominantly used trained or highly active subjects, the study by Zhang et al. (1991) is very important to the application of $\dot{V}O_2$ kinetics to fitness assessment, as sedentary subjects were used. The relationship between $\dot{V}O_{2\text{peak}}$ and $\dot{V}O_2$ kinetics was determined in nine sedentary male subjects (age range = 31 to 45

years) achieving relative $\dot{V}O_{2\text{peak}}$ ranging from 31 to 67 $\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$. Oxygen uptake kinetics were quantified by the time taken to reach 75% ($t_{0.75}$) of the response to a 3 min step change in work rate. Work rates of 45, 60 or 90 W were used depending on the $\dot{V}O_{2\text{peak}}$ achieved. The size of the step change in work rate was adjusted to ensure that steady state was attained during the 3 min test. Faster $\dot{V}O_2$ kinetics were observed in subjects with higher $\dot{V}O_{2\text{peak}}$. The following model described the relationship between $\dot{V}O_{2\text{peak}}$ and $\dot{V}O_2$ kinetics ($r = 0.75$, $P < 0.05$):

$$\dot{V}O_2 \text{ kinetics } (t_{0.75}, \text{ in s}) = -1.04(\text{relative } \dot{V}O_{2\text{peak}}(\text{in } \text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1})) + 99.3 \quad (7)$$

Zhang et al. (1991) concluded that the kinetics of aerobically produced ATP flux relative to the anaerobic ATP flux are greater in those subjects attaining higher $\dot{V}O_{2\text{max}}$ values, and thus give rise to faster $\dot{V}O_2$ kinetics.

Most of the subjects studied by Zhang et al. (1991) however had achieved $\dot{V}O_{2\text{peak}}$ of $>40 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$. For a method of assessing $\dot{V}O_2$ kinetics to be applicable to cardiac patients, subjects with low aerobic power must be studied. Cardiac patients participating in cardiac rehabilitation programmes typically have $\dot{V}O_{2\text{peak}}$ values ranging from 13.6 to 23.8 $\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ (Riley et al., 1994). Babcock et al. (1994b) investigated the $\dot{V}O_2$ kinetics of subjects with poor fitness levels. The male subjects ($n = 46$, age range = 30 to 80 years) studied were grouped as a function of age. Oxygen uptake kinetics were assessed using a step change in work rate between 80% of the work rate needed to elicit $\dot{V}O_2$ at the V_{ET} and loadless cycling (0 W). The $\dot{V}O_{2\text{max}}$ and $\dot{V}O_2$ kinetics of the study sub-groups categorised according to age are shown in table 4.1.

The results showed that $\dot{V}O_2$ kinetics were significantly slowed in older, less fit subjects.

Table 4.1) The $\dot{V}O_{2max}$ and $\dot{V}O_2$ kinetics of the male subjects categorised according to age (Babcock et al., 1994b) (mean \pm SD).

	Group 1	Group 2	Group 3
Number of subjects	17	17	12
Age (years)	36.7 \pm 5.0	51.0 \pm 4.7	71.4 \pm 4.1
Absolute $\dot{V}O_{2max}$ ($l \cdot min^{-1}$)	3.17 \pm 0.5	2.58 \pm 0.4	1.74 \pm 0.1
$\tau \dot{V}O_2$ (s)	38.8 \pm 9.5	48.6 \pm 11.2*	60.8 \pm 17.6 [§]

* denotes significant difference between group 2 and group 1 ($P < 0.05$)

[§] denotes significant difference between group 3 and group 1, and group 3 and group 2 ($P < 0.05$).

Factors have been suggested which may explain the slowing of $\dot{V}O_2$ kinetics with age, including the specific changes that occur with ageing, for example the relative slowing of the components of the gas transport system (Cunningham et al., 1993), and changes in skeletal muscle morphology (Overend et al., (1992). The effects of ageing however cannot totally explain the slowing of $\dot{V}O_2$ kinetics, since long-term inactivity may also have contributed. In order to discriminate between subjects with lower levels of fitness, on the basis of $\dot{V}O_2$ kinetics, studies must be controlled for age.

Chilibeck et al. (1996) used multiple linear regression analysis to demonstrate that cardiorespiratory fitness was the most significant explanatory variable for the variance in the time constant for oxygen uptake (τ). This variable explained 31% of the variance

within the model relating $\dot{V}O_2$ kinetics with $\dot{V}O_{2\max}$. Habitual activity levels for the subjects however were not included amongst the variables used to explain τ .

The aims of this investigation were to use the PRBS exercise test technique to:

- examine the relationship between aerobic power and $\dot{V}O_2$ kinetics in a homogenous group of young, healthy females,
- discriminate between the $\dot{V}O_2$ kinetics of two groups of subjects, with one group attaining higher than average aerobic power for the total subject cohort, and one group attaining lower than average aerobic power for the total subject cohort.

4.2) Methodology

4.2.1) Subjects

The subject cohort studied, and the data collected, during this investigation comprise the preliminary data set for the effect of endurance-type training on the $\dot{V}O_2$ kinetics of healthy, young women (c.f. Chapter 5).

Twenty-eight female subjects (age = 22.9 ± 3.1 years, height = 1.64 ± 0.05 m and body mass = 62.9 ± 7.9 kg) were recruited from the student population at Sheffield Hallam University. Prior to inclusion in the study population, all subjects were screened for cardiovascular, respiratory and musculo-skeletal disorders, by means of a health and activity questionnaire (c.f. appendix A.3.3). Written informed consent was obtained from each individual prior to commencement of the study (c.f. appendix A.3.4 and A.3.5).

4.2.2) Exercise testing

All subjects completed a PRBS exercise test with 30 s work rate changes between 25 and 75 W, to determine $\dot{V}O_2$ kinetics (c.f. Chapter 2, Section 2.3.1). The upper work rate was set to be below that at V_{ET} for all subjects. A progressive exercise test with ramp increases in work rate of $20 \text{ W}\cdot\text{min}^{-1}$ to exhaustion, was then performed (c.f. Chapter 2, Section 2.3.2). Oxygen uptake and heart rate were measured throughout these exercise testing procedures (c.f. Chapter 2, Sections 2.2.1 and 2.2.2). Individual seat height was recorded (c.f. Chapter 2, Section 2.2.3).

4.3) Data and statistical analysis

The breath-by-breath data collected during the submaximal PRBS exercise test were subjected to Fourier analysis to produce measures of $\dot{V}O_2$ kinetics at harmonic numbers 1 to 4 (c.f. Chapter 2, Section 2.4.2.1). The data collected during the progressive exercise test to exhaustion were averaged on a 30 s time basis (c.f. Chapter 2, Section 2.4.2.2).

4.3.1) Attainment of maximal oxygen uptake or peak oxygen uptake

The number of criteria, described previously in Chapter 2, Section 2.4.2.2, which were fulfilled by each subject during the progressive exercise test to exhaustion was documented to determine whether $\dot{V}O_{2\text{max}}$ or $\dot{V}O_{2\text{peak}}$ had been achieved.

4.3.2) Descriptive statistics

The mean and standard deviation (mean \pm SD) were calculated for each data set.

Histograms were plotted to ensure that the data points were normally distributed and occurred within ± 2 SD of the mean.

4.3.3) Oxygen uptake kinetics in subjects differing in aerobic power

The subject cohort was sub-divided into two categories, according to relative $\dot{V}O_{2\text{peak}}$ values. These sub-groups consisted of subjects with values below and above the mean value of the total subject cohort. Analysis of variance was used to detect any differences in $\dot{V}O_2$ kinetics between the two study sub-groups. A significance level of $\alpha \leq 0.05$ was selected.

4.3.4) Relationship between oxygen uptake kinetics and aerobic power

Pearson Product Moment Correlation analysis and linear regression analysis were used to determine the relationships between the $\dot{V}O_2$ kinetics and aerobic power for the total subject cohort. Correlation coefficients and linear regression equations were calculated for the relationships between $\dot{V}O_2$ kinetics at harmonic numbers 1 to 4 and (I) relative $\dot{V}O_{2\text{peak}}$ and (II) absolute $\dot{V}O_{2\text{peak}}$. A significance level of $\alpha \leq 0.05$ was selected. Adjusted R^2 (Adj. R^2) was used to describe the goodness of fit of the model, by measuring the correlation between the observed values and those predicted by the regression equation (Altman, 1991).

4.4) Results

4.4.1) Progressive exercise test

The average maximum work rate achieved at the end of the test by the twenty-eight subjects was 189.5 ± 23.1 W. The values ranged from 149.4 to 245.2 W.

Five subjects, 18% of the total population, attained $\dot{V}O_{2\max}$ during the maximal exercise test by achieving a plateau in the relationship between $\dot{V}O_2$ and work rate. The remaining subjects attained $\dot{V}O_{2\text{peak}}$ by fulfilling one or both of the criteria for $\dot{V}O_{2\text{peak}}$ (c.f. Chapter 2, Section 2.4.2.2). Of these two criteria, all subjects attained an RER of 1.15 or higher, and 29% of subjects attained a maximal heart rate within $10 \text{ beats}\cdot\text{min}^{-1}$ of APMHR (c.f. appendix A.4.1). Since only 18% of the subjects achieved $\dot{V}O_{2\max}$, the term $\dot{V}O_{2\text{peak}}$ was preferred for the group results. The results for the progressive exercise test to exhaustion are displayed in table 4.2.

Table 4.2) The cardiorespiratory values at exhaustion (mean \pm SD) (n = 28).

Variable	Mean \pm SD
Relative $\dot{V}O_{2\text{peak}}$ ($\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$)	37.0 ± 5.5
Absolute $\dot{V}O_{2\text{peak}}$ ($\text{ml}\cdot\text{min}^{-1}$)	2297 ± 245
Peak heart rate ($\text{beats}\cdot\text{min}^{-1}$)	183 ± 10
Peak RER	1.16 ± 0.06

Mean relative $\dot{V}O_{2\text{peak}}$ ranged from 26.6 to $47.9 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ and absolute $\dot{V}O_{2\text{peak}}$ ranged from 1982 to $2958 \text{ ml}\cdot\text{min}^{-1}$.

4.4.2) Oxygen uptake kinetics

4.4.2.1) Mean response

A dynamic gain for $\dot{V}O_2$ of $1056 \pm 80 \text{ ml}\cdot\text{min}^{-1}$ and a mean overall heart rate response of $120 \pm 16 \text{ beats}\cdot\text{min}^{-1}$ resulted from the PRBS exercise test for the twenty-eight subjects. This represents an average exercise intensity of 46% $\dot{V}O_{2\text{peak}}$ and 66% peak heart rate achieved during the ramp progressive exercise test.

4.4.2.2) Frequency domain analysis

The $\dot{V}O_2$ kinetics which resulted from the PRBS exercise test are displayed in table 4.3.

Table 4.3) $\dot{V}O_2$ kinetics, amplitude ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$) and phase shift (degrees), at the first four harmonic numbers (mean \pm SD) (n = 28).

Variable	Mean \pm SD
Amplitude at harmonic number 1 ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$)	10.304 ± 0.432
Amplitude at harmonic number 2 ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$)	6.761 ± 0.735
Amplitude at harmonic number 3 ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$)	8.716 ± 0.881
Amplitude at harmonic number 4 ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$)	8.168 ± 0.881
Phase shift at harmonic number 1 (degrees)	-24.9 ± 2.8
Phase shift at harmonic number 2 (degrees)	-42.2 ± 4.9
Phase shift at harmonic number 3 (degrees)	-70.0 ± 7.8
Phase shift at harmonic number 4 (degrees)	-78.0 ± 6.7

4.4.3) Oxygen uptake kinetics in subjects differing in aerobic power

Relative $\dot{V}O_{2\text{peak}}$ values of $32.3 \pm 3.3 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ and $41.1 \pm 3.2 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ were achieved by the sub-groups with relative $\dot{V}O_{2\text{peak}}$ below and above $37.0 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ respectively. The characteristics of the two sub-groups of subjects are described in table 4.4.

Table 4.4) The characteristics of the two sub-groups of subjects categorised according to relative $\dot{V}O_{2\text{peak}}$ of below (group I) or above $37.0 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ (group II) (mean \pm SD) (n = 28).

Variable	Group I (<37.0 ml·kg⁻¹·min⁻¹)	Group II (>37.0 ml·kg⁻¹·min⁻¹)
Age (years)	23.3 \pm 3.2	22.4 \pm 2.8
Height (m)	1.65 \pm 0.06	1.63 \pm 0.03
Weight (kg)	67.8 \pm 8.3	58.5 \pm 3.6
Absolute $\dot{V}O_{2\text{peak}}$ (ml·min⁻¹)	2171 \pm 165	2406 \pm 242
Habitual activity level	3.2 \pm 2.0	3.2 \pm 1.8

4.4.3.1) Amplitude

The mean \pm SD of the amplitudes and the difference between the two study sub-groups are displayed in table 4.5.

Table 4.5) Amplitude ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$) at harmonic numbers 1 to 4, and categorised according to relative $\dot{V}O_{2\text{peak}}$ of below (group I) or above $37.0 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ (group II) (mean \pm SD) (n = 28).

Relative $\dot{V}O_{2\text{peak}}$ ($\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$)	Subject number	Amplitude ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$)			
		Harmonic number 1	Harmonic number 2	Harmonic number 3	Harmonic number 4
Group I <37.0	13	10.263 \pm 0.421	6.455 \pm 0.496**	8.681 \pm 1.070	7.916 \pm 1.013
Group II >37.0	15	10.407 \pm 0.588	7.462 \pm 1.009**	9.213 \pm 0.782	8.488 \pm 0.853

** = significant difference between study sub-groups at $P < 0.01$.

Faster $\dot{V}O_2$ kinetics were found in those subjects with greater aerobic power (c.f. Figure 4.1 and 4.2). The amplitude values at harmonic number 2 were found to be significantly greater ($P < 0.01$) for the sub-group with relative $\dot{V}O_{2\text{peak}}$ above $37.0 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ (group II) than those for the sub-group with relative $\dot{V}O_{2\text{peak}}$ below $37.0 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ (group I). Although a trend towards higher amplitudes at harmonic numbers 1, 3 and 4, was observed in those subjects with greater aerobic power, no significant difference in amplitude was seen to exist between the two sub-groups. The mean \pm SD of amplitude, at harmonic numbers 1 to 4, for the two study groups categorised according to relative

$\dot{V}O_{2peak}$ are shown in figure 4.1.

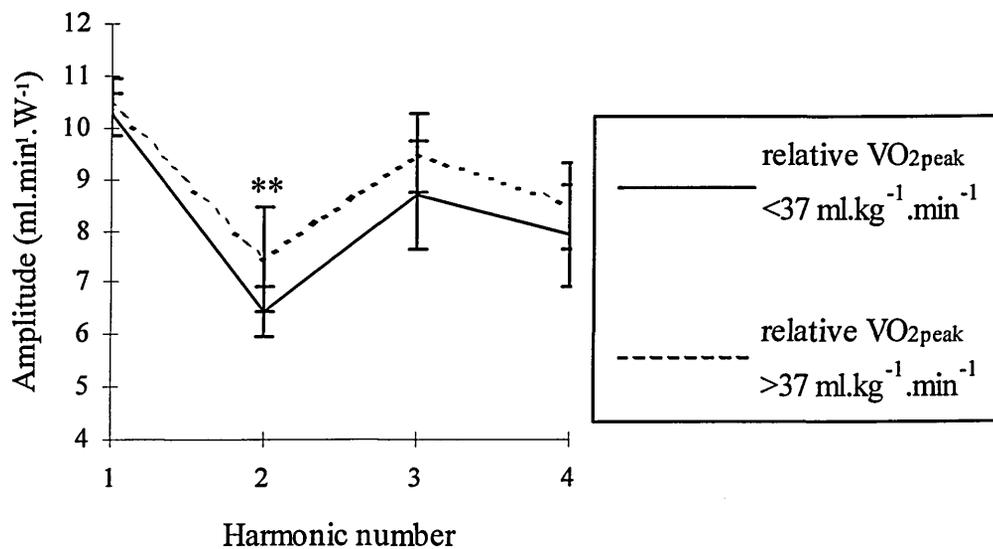


Figure 4.1) Amplitude ($\text{ml} \cdot \text{min}^{-1} \cdot \text{W}^{-1}$), at harmonic numbers 1 to 4, for the two study sub-groups categorised according to relative $\dot{V}O_{2peak}$ (mean \pm SD). (** = significant difference between groups at $P < 0.01$).

4.4.3.2) Phase shift

The mean \pm SD of the phase shifts and the difference between the two study sub-groups are displayed in table 4.6.

Table 4.6) Phase shift (degrees) at harmonic numbers 1 to 4, and categorised according to relative $\dot{V}O_{2\text{peak}}$ of below (group I) or above $37.0 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ (group II) (mean \pm SD) (n = 28).

Relative $\dot{V}O_{2\text{peak}}$ ($\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$)	Subject number	Phase shift (degrees)			
		Harmonic number 1	Harmonic number 2	Harmonic number 3	Harmonic number 4
Group I <37.0	13	-26.3 \pm 2.5**	-44.7 \pm 5.2**	-75.0 \pm 7.2***	-80.3 \pm 7.9
Group II >37.0	15	-23.6 \pm 2.5**	-40.1 \pm 3.4**	-65.7 \pm 5.4***	-76.1 \pm 4.9

** = significant difference between study sub-groups at $P < 0.01$.

*** = significant difference between study sub-groups at $P < 0.001$.

At harmonic numbers 1, 2 and 3, the phase shifts of the sub-group with relative $\dot{V}O_{2\text{peak}}$ above $37.0 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ (group II) were found to be significantly smaller than those for the sub-group with relative $\dot{V}O_{2\text{peak}}$ below $37.0 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ (group I).

The mean \pm SD of phase shifts, at harmonic numbers 1 to 4, for the two study groups categorised according to relative $\dot{V}O_{2\text{peak}}$ are shown in figure 4.2.

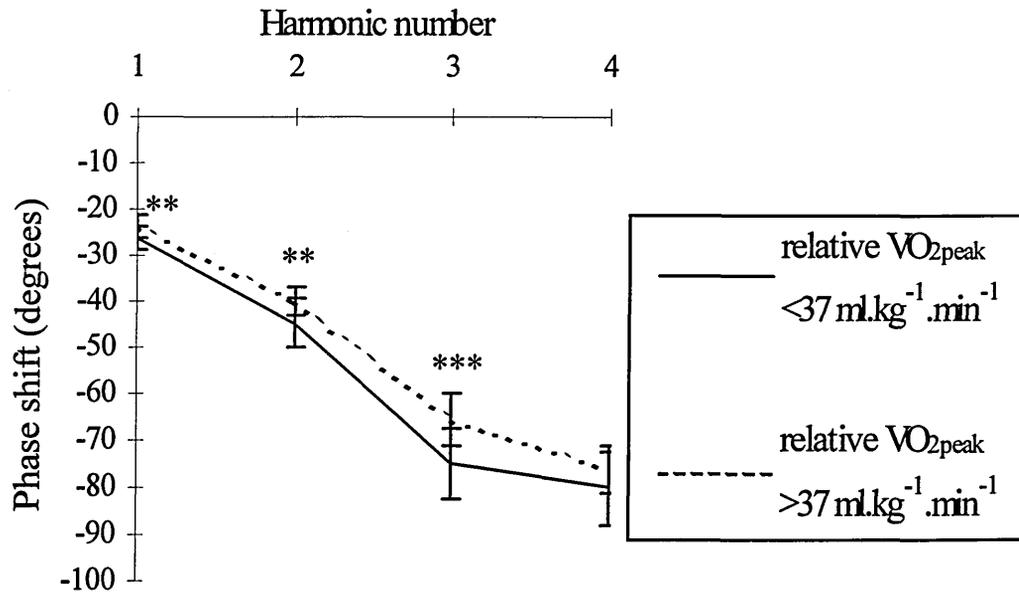


Figure 4.2) Phase shift (degrees) for the two study sub-groups categorised according to relative $\dot{V}O_{2\text{peak}}$ (mean \pm SD). (** and *** = significant difference between groups at $P < 0.01$ and $P < 0.001$ respectively).

4.4.4) Relationship between oxygen uptake kinetics and peak oxygen uptake

4.4.4.1) Amplitude

The Pearson Product Moment correlations (r values) used to assess the extent of the associations existing between amplitude and (I) relative $\dot{V}O_{2\text{peak}}$, and (II) absolute $\dot{V}O_{2\text{peak}}$ are displayed in table 4.7.

Table 4.7) Correlation coefficients (r values) for the relationships between amplitude ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$) at harmonic numbers 1 to 4 and (I) relative $\dot{V}O_{2\text{peak}}$ ($\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$), and (II) absolute $\dot{V}O_{2\text{peak}}$ ($\text{ml}\cdot\text{min}^{-1}$).

Amplitude ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$)	Relative $\dot{V}O_{2\text{peak}}$ ($\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$)	Absolute $\dot{V}O_{2\text{peak}}$ ($\text{ml}\cdot\text{min}^{-1}$)
	r value	r value
Harmonic number 1	0.13	0.18
Harmonic number 2	0.61**	0.40*
Harmonic number 3	0.19	0.34
Harmonic number 4	0.22	0.27

* = significant correlation ($P<0.05$).

** = significant correlation ($P<0.01$).

Significant positive relationships were found to exist between relative $\dot{V}O_{2\text{peak}}$ and amplitude at harmonic number 2 ($r = 0.61$, $P<0.01$), and absolute $\dot{V}O_{2\text{peak}}$ and amplitude at harmonic number 2 ($r = 0.40$, $P<0.05$). High amplitudes at harmonic number 2 were apparent for those subjects attaining high relative $\dot{V}O_{2\text{peak}}$ and high absolute $\dot{V}O_{2\text{peak}}$.

The linear regression equation for the relationship between relative $\dot{V}O_{2\text{peak}}$ and amplitude is shown in figure 4.3.

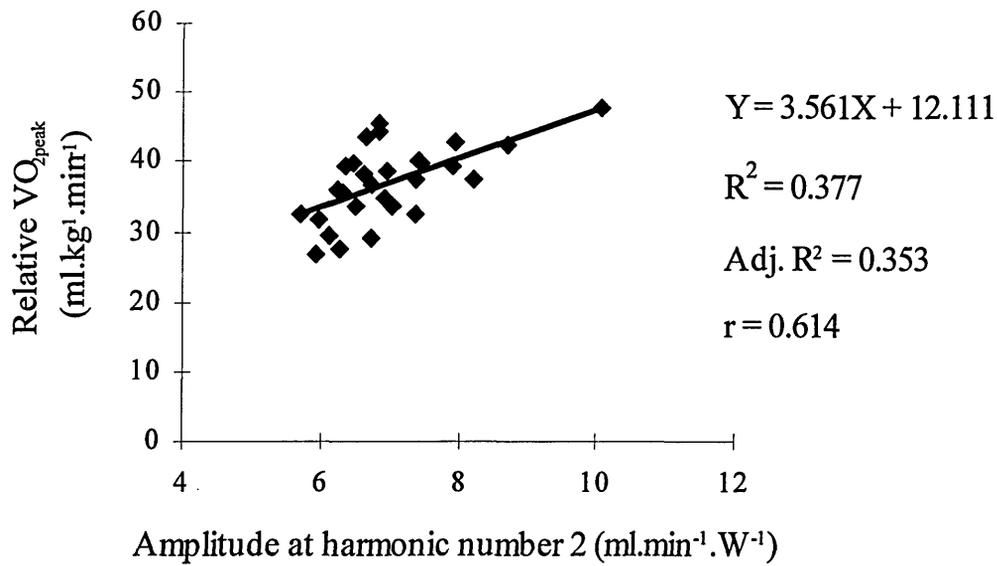


Figure 4.3) Linear regression equation for the relationship between relative $\dot{V}O_{2peak}$ and amplitude at harmonic number 2.

4.4.4.2) Phase shift

The Pearson Product Moment correlations (r values) used to assess the extent of the associations existing between phase shift and (I) relative $\dot{V}O_{2peak}$, and (II) absolute $\dot{V}O_{2peak}$ are displayed in table 4.8.

Table 4.8) Correlation coefficients (r values) for the relationships between phase shift (degrees) at harmonic numbers 1 to 4 and (I) relative $\dot{V}O_{2\text{peak}}$ ($\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$), and (II) absolute $\dot{V}O_{2\text{peak}}$ ($\text{ml}\cdot\text{min}^{-1}$).

Phase shift (degrees)	Relative $\dot{V}O_{2\text{peak}}$ ($\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$)	Absolute $\dot{V}O_{2\text{peak}}$ ($\text{ml}\cdot\text{min}^{-1}$)
	r value	r value
Harmonic number 1	-0.66***	-0.48**
Harmonic number 2	-0.60***	-0.33
Harmonic number 3	-0.72***	-0.45**
Harmonic number 4	-0.24	-0.28

** = significant correlation ($P < 0.01$),

*** = significant correlation, $P < 0.001$.

Significant negative relationships were found to exist between relative $\dot{V}O_{2\text{peak}}$ and phase shift at harmonic numbers 1 ($r = -0.66$, $P < 0.001$), 2 ($r = -0.60$, $P < 0.001$) and 3 ($r = -0.72$, $P < 0.001$). Weaker negative associations were apparent between absolute $\dot{V}O_{2\text{peak}}$ and phase shift at harmonic numbers 1 ($r = -0.48$, $P < 0.01$) and 3 ($r = -0.45$, $P < 0.01$). Smaller phase shifts were apparent for those subjects attaining high relative $\dot{V}O_{2\text{peak}}$ at harmonic numbers 1, 2 and 3, and high absolute $\dot{V}O_{2\text{peak}}$ at harmonic numbers 1 and 3.

The linear regression equations for the relationships between relative $\dot{V}O_{2\text{peak}}$ and phase shift are shown in figure 4.4.

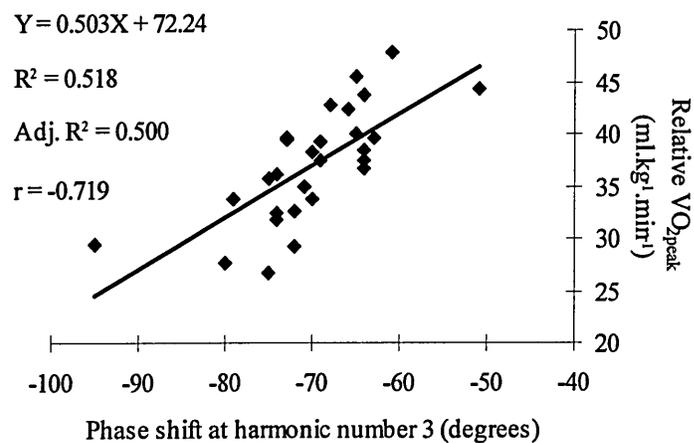
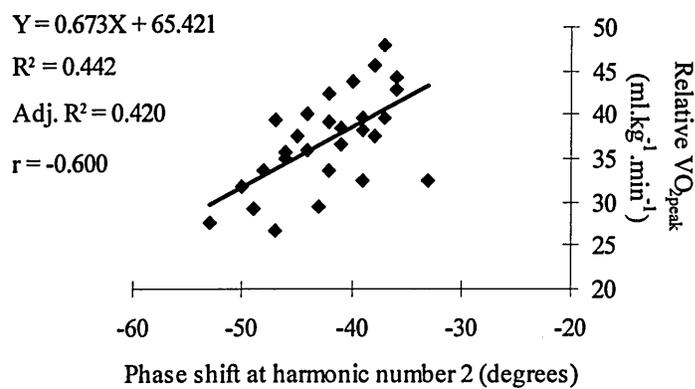
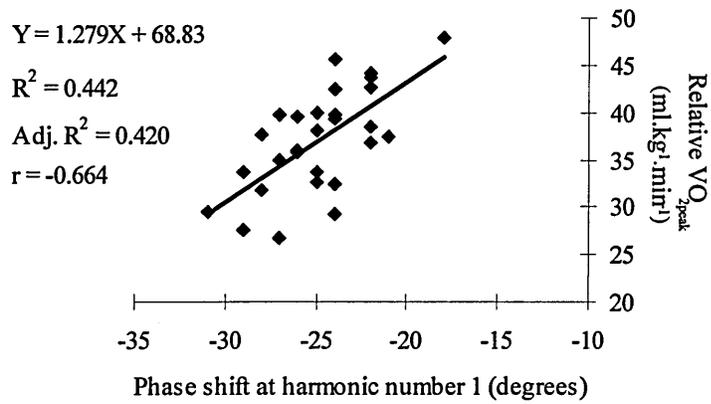


Figure 4.4) Linear regression equations for the relationships between relative $\dot{V}O_{2\text{peak}}$ and phase shift at harmonic numbers 1, 2 and 3.

4.5) Discussion

The purpose of this investigation was to study the $\dot{V}O_2$ kinetics, determined by the PRBS exercise test technique, of a population of female subjects in relation to the established measure of aerobic power.

4.5.1) Progressive exercise test

During this maximal assessment, mean relative and absolute $\dot{V}O_{2\text{peak}}$ values of $37.0 \pm 5.5 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ and $2297 \pm 245 \text{ ml}\cdot\text{min}^{-1}$ respectively were attained by this young female population. Aerobic power values determined during maximal cycle ergometry exercise are known to be significantly lower than those measured during treadmill exercise (Glassford et al., 1965, Pollock et al., 1982, and Hellerstein and Franklin, 1984).

Aerobic power values were found to be comparable to the $\dot{V}O_{2\text{max}}$ results presented by Pollock et al. (1982) for healthy women (relative $\dot{V}O_{2\text{max}} = 36.6 \pm 4.9 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) performing progressive cycle exercise to exhaustion comprising step changes in work rate. When comparing this mean aerobic power value ($\dot{V}O_{2\text{peak}} = 37.0 \pm 5.5 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) to the mean $\dot{V}O_{2\text{max}}$ values reported by Pollock et al. (1982), the female subjects studied within the present investigation were more comparable to those subjects classified as sedentary (relative $\dot{V}O_{2\text{max}} = 33.3 \pm 5.2 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$). These aerobic power values were also comparable to those reported by McCord and Paterson (1989) (relative $\dot{V}O_{2\text{max}} = 38.4 \pm 4.3 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$). Cunningham et al. (1993) used a ramp progressive exercise test to exhaustion to measure $\dot{V}O_{2\text{max}}$ in a population of young females. This protocol resulted in the attainment of $\dot{V}O_{2\text{max}}$ values (relative $\dot{V}O_{2\text{max}} =$

$36.9 \pm 4.7 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) which were identical to those $\dot{V}O_{2\text{peak}}$ values reported during the present study. Maximal heart rates reported by Cunningham et al. (1993) (maximal heart rate = $181 \pm 8 \text{ beats}\cdot\text{min}^{-1}$) were also similar to the peak heart rates measured during the present study. As these actual peak heart rates were lower than APMHR ($194 \text{ beats}\cdot\text{min}^{-1}$) it is likely that the subjects did not achieved 'true $\dot{V}O_{2\text{max}}$ ' according to the strict definition by Zeballos and Weisman (1994) but did attain $\dot{V}O_{2\text{peak}}$.

4.5.2) Oxygen uptake kinetics in the two sub-groups with high and low aerobic power

The two sub-groups were formed on the basis of the aerobic power results, with all subjects included in the groupings. Fifteen subjects had relative $\dot{V}O_{2\text{peak}}$ greater than the mean value for the total subject cohort ($37.0 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) and thirteen subjects had relative $\dot{V}O_{2\text{peak}}$ below the mean value. The mean relative $\dot{V}O_{2\text{peak}}$ for the higher fitness group was $41.1 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$, and the mean relative $\dot{V}O_{2\text{peak}}$ for the lower fitness group was $32.2 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$. Coincidentally these values correspond with the two female sub-groups identified by Pollock et al. (1982), where the subjects were categorised according to activity levels rather than by $\dot{V}O_{2\text{max}}$. Pollock et al. (1982) described twenty-nine sedentary women as having an average $\dot{V}O_{2\text{max}}$ of $33.3 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$, which corresponds with the lower fitness group of the present study. Similarly the twenty active women studied by Pollock et al. (1982) were reported to have an identical average $\dot{V}O_{2\text{max}}$ ($41.4 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) to the higher fitness level group of the present study. Examination of the activity questionnaires (c.f. Chapter 2, Section 2.5) revealed no significant difference between the levels of activity performed by the two study sub-groups (mean activity score = 3.2). It could be suggested therefore that

neither of the groups were trained. The activity scores show that the young women of two moderately active groups were compared. The present study adopted a slightly different approach to that of Pollock et al. (1982), as sub-group classification was on the basis of aerobic power and not habitual activity levels. In this case, the difference in aerobic power values cannot be attributed to differences in habitual activity levels.

In spite of the fact that there was only a difference of $8.9 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ between the mean relative $\dot{V}O_{2\text{peak}}$ for the two study sub-groups, significant differences in $\dot{V}O_2$ kinetics were demonstrated for phase shift at harmonic numbers 1, 2 and 3, and amplitude at harmonic number 2. The significant slowing of the $\dot{V}O_2$ kinetics of young women, at these harmonic numbers, for the lower fitness group is in agreement with studies in men using step changes in work rate to determine $\dot{V}O_2$ kinetics (Powers et al., 1985, Babcock et al., 1994b and Chilibeck et al., 1996).

The results from the present study partially agree with the findings of Eßfeld et al. (1987), where the PRBS exercise test technique was also used to differentiate between subject groups differing in aerobic power. The same general findings apply, in that the groups of higher fitness level exhibited faster $\dot{V}O_2$ kinetics, but Eßfeld et al. (1987) could only differentiate between subject groups on the basis of amplitude. Although it was demonstrated that phase shift tended to be smaller in the fitter groups of subjects, the finding was not statistically significant. Eßfeld et al. (1987) was able to show significant differences between subjects in adjacent groups using amplitude values at higher harmonic numbers, i.e. above the fourth harmonic number, whereas in the present study significant differences in amplitude were only found at harmonic number

2. The small differences between the findings of the present study and those presented by Eßfeld et al. (1987) can be explained by differences in the populations studied, in the equipment used, in the relative work rate levels and work rate patterns applied to the cycle ergometer, and also in the analysis procedures used.

4.5.3) The relationship between oxygen uptake kinetics and aerobic power

The correlation coefficient for the relationship between $\dot{V}O_2$ kinetics and aerobic power was calculated at each harmonic number. At harmonic numbers where a significant correlation was found, this relationship was represented by a linear regression equation. Oxygen uptake kinetics were found to be more frequently and more highly correlated with relative $\dot{V}O_{2peak}$, a finding in keeping with that of Chilibeck et al. (1996). Simple independent linear regression equations resulted in four separate prediction models for relative $\dot{V}O_{2peak}$. The percentage variability accounted for by the models was 35% for amplitude at harmonic number 2, 42% for phase shift at harmonic number 2, 42% for phase shift at harmonic number 1 and 50% for phase shift at harmonic number 3. It can be seen therefore that for this cohort of twenty-eight young females, the best predictor of relative $\dot{V}O_{2peak}$ was phase shift at harmonic number 3, since this parameter explains 50% of the variation in the relationship between $\dot{V}O_2$ kinetics and aerobic power. The varying degrees of variability accounted for by the models suggest that no mono-frequent measure of $\dot{V}O_2$ kinetics, determined by the PRBS exercise test, should be seen as a perfect predictor of aerobic power.

4.5.4) *Physiological mechanisms*

Maximal oxygen uptake represents the maximal amount of O₂ utilised by the tissues per minute, whereas $\dot{V}O_2$ kinetics are measures of the rate of change of O₂ utilisation in response to a change in work rate. Both $\dot{V}O_2$ kinetics and aerobic power may be limited by the ability of the cardiovascular system to transport O₂ to the site of mitochondrial respiration (central factors), and/or the ability of the muscle cell to utilise O₂ (peripheral factors) (Sahlin et al., 1988, Stainsby et al., 1989, Sutton, 1992a, Sutton, 1992b, Yoshida and Whipp., 1994, Wagner 1992). In the literature, there is some discrepancy concerning where the dividing line exists between central and peripheral factors. As far as $\dot{V}O_2$ kinetics are concerned, central factors are those which relate to the delivery of O₂ to the mitochondria, and peripheral factors relate to the other factors which control mitochondrial respiration (Walsh, 1992). When discussing aerobic power, central factors determine O₂ delivery only as far as the muscle arterioles, whereas peripheral factors are those factors determining the extraction of O₂ from capillary blood (Sutton, 1992a, Sutton, 1992b).

Many studies and review articles have concluded that the major limiting factor for aerobic power is the delivery of O₂ to the muscle cell (Rowell, 1986, Sutton, 1992a, Sutton, 1992b, Wasserman, 1982, Wasserman, 1994, Wagner, 1992, Bassett and Howley, 1997), whereas more uncertainty exists concerning the primary limiting factor for $\dot{V}O_2$ kinetics (Hughson and Morrissey, 1983, Walsh, 1992, Mahler, 1985).

In maximal aerobic exercise, the major sites of potential limitation in the O₂ transport system have been identified as follows:

- pulmonary diffusion capacity for O₂,

- maximal cardiac output (\dot{Q}_{\max})
- peripheral circulation.

In elite endurance athletes, a pulmonary diffusion limitation has been shown to be one factor which affects aerobic power (Inbar et al., 1993). In some elite endurance athletes, the arterial haemoglobin saturation drops and this may be linked to a ventilation/perfusion mismatch (Hopkins et al., 1994), although pulmonary diffusion capacity is not thought to limit maximal aerobic performance in groups other than elite endurance athletes.

The consensus of opinion is that the capacity of the central cardiovascular system, determined by \dot{Q}_{\max} , to transport O_2 to the muscle cell is the main factor limiting aerobic power in healthy subjects (Bassett and Howley, 1997). The studies that have led to this conclusion are based on exercise involving either large and small muscle masses, or arm and leg exercise. Studies in which maximal blood flow has been measured in small muscle groups, during knee extensor exercise, have shown that the maximal muscle blood flow is two to three times higher than the maximal flow achieved during two-legged cycle exercise (Saltin, 1985). This shows that the capacity of the heart to deliver O_2 to the exercising muscles is the limiting factor in whole body exercise when a large muscle mass is being used.

Several studies have shown that the aerobic power for combined arm and leg exercise is similar to that for leg exercise alone (Astrand and Saltin, 1961, Secher et al., 1974).

Blood flow measurements during arm and leg exercise show that \dot{Q} is unable to supply the O_2 demands of the combined muscle mass at high exercise intensities (Secher et al., 1977). During exercise using small muscle groups, for example arms, aerobic power is

determined by the oxidative capacity of the muscle since the ability of the muscle blood flow to increase is not limited by \dot{Q} as it is when larger muscle groups are employed. In contrast, Gleser et al. (1974) have shown that adding arm exercise to maximal leg exercise increased $\dot{V}O_{2\max}$ by 10% suggesting that aerobic power is limited by the muscle mass employed and not by \dot{Q}_{\max} . In this study however, leg exercise was performed with the arms hanging by the sides of the subject. This has been shown to reduce both the power output and the aerobic power that can be achieved during conventional leg cycling, when the arms are used to stabilise the body (Nagle et al., 1984). This may account for the lower aerobic power achieved during leg only exercise. The consensus of opinion therefore is that aerobic power is determined by the maximum capacity of the cardiovascular system to supply blood to the working muscle.

The other line of evidence, suggesting a central limitation, is based on the relationships between aerobic power and \dot{Q} in trained and untrained subjects. It has been shown that the two-fold difference in $\dot{V}O_{2\max}$ between the two groups is largely due to differences in \dot{Q} , and specifically in stroke volume (Rowland, 1974, Bassett and Howley, 1997). The role of peripheral mechanisms in reducing the venous O_2 content is thought to be less important. Some studies suggest that venous O_2 content is similar in trained and untrained subjects, whilst others have shown that training results in an increase in $(a-\bar{v})O_2$ difference (Spina et al., 1992).

Oxygen utilisation by the muscle mitochondria depends on the oxidative enzyme activity, provided that the PO_2 within the mitochondria is above 0.5 mmHg. A PO_2 of 15 to 20 mmHg is required to provide a sufficient diffusion gradient between the capillary and the muscle cell, and to maintain mitochondrial PO_2 above this critical

threshold value of 0.5 mmHg. During maximal exercise, capillary PO_2 is thought to fall below 15 to 20 mmHg and therefore O_2 delivery becomes limiting (Wasserman, 1994).

There are marked differences in the muscle oxidative enzyme capacity of trained and untrained subjects (Holloszy and Coyle, 1984), however this is not thought to limit aerobic power as the biochemical changes are much greater than the increase in $\dot{V}O_{2max}$. Experiments using isolated muscle suggest that increased enzymatic activity is required in order to maintain the electron flux despite very low PO_2 values at the mitochondrial level (Honig et al., 1992, Robinson et al., 1994). This means that biochemical adaptations might have a role in determining aerobic power when capillary PO_2 falls below the critical threshold.

Some of the more recent evidence (c.f. Chapter 1) supports a peripherally limiting site for $\dot{V}O_2$ kinetics. This may explain the relationship found between aerobic power and $\dot{V}O_2$ kinetics in the present study, when considering that subjects with greater aerobic power are characterised by having increased skeletal muscle oxidative enzyme capacity. It is difficult to argue that O_2 transport becomes limiting during submaximal exercise however, as diffusion at the capillary surface is theoretically greater than the $\dot{V}O_2$ response (Walsh, 1992). This could explain why there was only a moderate relationship between phase shift at harmonic number 3 and relative $\dot{V}O_{2max}$ in the present study. Only 50% of the variation within the relationship between aerobic power and $\dot{V}O_2$ kinetics was accounted for by the model.

4.6) Conclusions

- Faster $\dot{V}O_2$ kinetics were related to higher relative $\dot{V}O_{2peak}$ values in healthy young women.
- Two groups of healthy young women differing in relative $\dot{V}O_{2peak}$ can be differentiated on the basis of $\dot{V}O_2$ kinetics.
- Significant correlations were apparent between $\dot{V}O_2$ kinetics and $\dot{V}O_{2peak}$ relative to body mass, although the relationship between $\dot{V}O_2$ kinetics and absolute responses for $\dot{V}O_{2peak}$ was weaker.
- The following model relating $\dot{V}O_2$ kinetics and relative $\dot{V}O_{2peak}$ explains 50 % of the variability in the data:

$$\dot{V}O_{2peak} \text{ (in ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}\text{)} = 0.503(\text{phase shift at harmonic number 3} \\ \text{(in degrees)}) + 72.24 \quad (8)$$

- It is concluded that $\dot{V}O_2$ kinetics are reflecting the increase in oxidative capacity of the muscle, coupled with improvement in O_2 delivery at the muscular level, i.e. increased capillarisation,
- This method of measuring $\dot{V}O_2$ kinetics in the frequency domain could be used as a test for cardiorespiratory fitness.

CHAPTER FIVE

THE EFFECT OF ENDURANCE-TYPE TRAINING ON THE OXYGEN UPTAKE KINETICS OF YOUNG WOMEN

5.1) Introduction

Endurance-type training is known to increase whole body aerobic power due to physiological adaptations that improve maximal $\dot{V}O_2$ delivery and improve O_2 extraction. There is also evidence to suggest that endurance type-training can speed up the rate of adjustment of $\dot{V}O_2$ at the onset of constant load, submaximal exercise, i.e. $\dot{V}O_2$ kinetics (Hagberg et al., 1980, Berry and Moritani, 1985, Babcock et al., 1994a, Yoshida et al., 1992 and Phillips et al., 1995).

The relationship between aerobic power and $\dot{V}O_2$ kinetics has been established through cross-sectional studies (Powers et al., 1985, Eßfeld et al., 1987 and Zhang et al., 1991). Chilibeck et al. (1996) have shown that relative fitness was the strongest significant predictor of τ followed by sex and age. Multiple linear regression equations were calculated for young (age = 26.3 ± 2.5 years) and for old subjects (age = 68.9 ± 5.8 years) resulting in the following models:

Young: τ (in s) = $99.9 - 1.39(\dot{V}O_{2max}$ (in $ml \cdot kg^{-1} \cdot min^{-1}$)), $R = -0.85$, $P < 0.0001$),

Old: τ (in s) = $97.8 - 1.74(\dot{V}O_{2max}$ (in $ml \cdot kg^{-1} \cdot min^{-1}$)), $R = -0.59$, $P < 0.001$).

In longitudinal studies, vigorous endurance type-training has resulted in faster $\dot{V}O_2$ kinetics in both older (Babcock et al., 1994a) and younger subjects (Yoshida et al., 1992, and Phillips et al., 1995) (c.f. table 5.1). Endurance-type training performed at a frequency of three to four days per week, and at an intensity set to elicit $\dot{V}O_2$ at the V_{ET} plus 50% of the difference between the V_{ET} and $\dot{V}O_{2max}$, over a twenty-four week period, dramatically accelerated ($\dot{V}O_2$ kinetics were found to be 49% faster) the $\dot{V}O_2$ kinetics (pre-training $\tau \dot{V}O_2 = 62.2 \pm 15.5$ s, post-training $\tau \dot{V}O_2 = 31.9 \pm 6.3$ s, $P < 0.05$)

Table 5.1) Adaptations in aerobic fitness and $\dot{V}O_2$ kinetics following endurance training programmes.

Reference	Subject number	Gender	Age (years)	Training period	Training frequency	Training duration	Training Intensity	Change in aerobic power (%)	Step change for τ	Change in τ (%)
Hagberg et al. (1980)	8	Not known	30	9 weeks	6 days/week	30 to 40 min	6 x 5 min at $\dot{V}O_{2max}$ and cycling to fatigue	24	a) 0 to 50% $\dot{V}O_{2max}$ b) 0 to 70% $\dot{V}O_{2max}$	a) 21 b) 33
Berry and Moritani (1985)	a) 10 b) 10	Male	22	5 weeks	5 days/week	Given as distance (1.6 to 4.8 km)	a) 60 to 70% maximal heart rate reserve (steady state) b) 85 to 95% maximal heart rate reserve (interval)	Not known	Rest to 105 W	a) 8 b) 11
Yoshida et al. (1992)	4	Male	24	20 days	6 days/week	30 min	$\dot{V}O_2$ at the onset of blood lactate to exhaustion	23	a) 20W to onset of blood lactate b) 20 W to lactic acid threshold	a) 37 b) 27
Babcock et al. (1994a)	8	Male	72	24 weeks	3 to 4 days/week	30 min	$\dot{V}O_2$ at $V_{ET} + 50\%$ difference between V_{ET} and $\dot{V}O_{2max}$	20	0 to 80% $\dot{V}O_2$ at V_{ET}	49
Phillips et al. (1995)	7	Male	23	30 days	5 days in every 6 days	2 hours	60% $\dot{V}O_{2peak}$	11	25W to 60% $\dot{V}O_{2peak}$	58

of older men. A concomitant 20% increase in $\dot{V}O_{2\max}$ was observed as a result of this cycle ergometry training programme. Oxygen uptake kinetics for older men after training were similar to those for sedentary younger subjects (τ for trained older men = 31.9 ± 6.3 s, τ for sedentary younger men = 38.8 ± 9.5 s (Babcock et al., 1994a)). Interestingly, the mean post-training aerobic power of the older male subjects ($\dot{V}O_{2\max}$ = 2.12 ± 0.1 l·min⁻¹, (Babcock et al., 1994a)) was considerably lower than that for younger individuals ($\dot{V}O_{2\max}$ = 3.17 ± 0.5 l·min⁻¹, (Babcock et al., 1994b)). Endurance-training in sedentary, younger subjects has also resulted in increases in $\dot{V}O_{2\max}$ (11 to 24%) and decreases in τ (6 to 37%) (Hagberg et al., 1980, Berry and Moritani, 1985, Yoshida et al., 1992, Babcock et al., 1994a and Phillips et al., 1995).

The degrees of effectiveness of these endurance-training programmes could be explained by the differences between the training models employed. Contrasting methods of quantifying τ from work rate steps of differing magnitude may also have contributed to the range of training effects observed during these studies. All the training models involved short-term exercise (three to nine weeks) with training intensities or session durations resulting in very aggressive regimen which are more applicable to athletic training programmes than for the improvement of general fitness. The aggressive nature of these programmes is particularly apparent when reviewing the studies conducted by Yoshida et al. (1992) and Phillips et al. (1995). High intensity training, where subjects exercised at a power output equivalent to the $\dot{V}O_2$ at the onset of blood lactate until exhaustion, accompanied by very frequent exercise sessions (six days per week) of moderate duration (30 min) was chosen by Yoshida et al. (1992). Phillips et al. (1995) prescribed very long exercise sessions (two hours) at an initial intensity of 60% $\dot{V}O_{2\text{peak}}$ which resulted in fatigue at the end of the session. Training

was performed on a very frequent basis (five days in every six days) to achieve training adaptations in aerobic fitness, $\dot{V}O_2$ kinetics and biochemical markers. After four days (eight hours of training), Phillips et al. (1995) demonstrated a significant decrease (23%) in τ (pre-training $\tau = 37.2 \pm 4.8$ s, τ after four days of training = 28.8 ± 5.1 s, $P < 0.01$). Less pronounced training adaptations in $\dot{V}O_{2peak}$ (10.5% increase) were only seen after thirty days of training (pre-training relative $\dot{V}O_{2peak} = 44.3 \pm 2.4$ ml·kg⁻¹·min⁻¹, relative $\dot{V}O_{2peak}$ after thirty days of training = 49.6 ± 2.1 ml·kg⁻¹·min⁻¹, $P < 0.05$). Yoshida et al. (1992) described a close relationship between τ and training day, demonstrating a gradual decrease in τ with training.

An alternative to the aggressive training approach adopted by Yoshida et al. (1992) and Phillips et al. (1995) has been demonstrated by Berry and Moritani (1985). A more progressive training model involving exercise programmes of longer duration, e.g. nine to ten weeks (Hickson et al., 1978, Hickson et al., 1981 and Govindasamy et al., 1992) have been shown to be effective for sedentary subjects. Such programmes have resulted in increases in aerobic power of 14 to 38%. The decreases in τ reported by Berry and Moritani (1985) however were more modest (8% after a programme of steady state running, or 12% after high intensity interval training) than those reported by Phillips et al. (1995). This can be partially explained by training specificity since the training mode was running but the method of assessing $\dot{V}O_2$ kinetics involved cycling.

Several studies have shown that vigorous short-term training can result in faster $\dot{V}O_2$ kinetics and increased aerobic power. All these studies however have investigated the effects of endurance-type training on male subjects. There is at present no knowledge

concerning the effects of less vigorous training programmes on the $\dot{V}O_2$ kinetics of young women, for example training regimens based on the American College of Sports Medicine (1995) guidelines for exercise prescription designed to promote general fitness.

Although the effect of endurance type-training on the $\dot{V}O_2$ kinetics of males has been quantified in the time domain using mono-frequent assessment methods, there is only circumstantial evidence to suggest that similar effects will be found in the frequency domain (Stegemann et al., 1985, Hughson et al., 1990b). A multi-frequent assessment of $\dot{V}O_2$ kinetics in the frequency domain has yet to be used to detect adaptations resulting from endurance-type training.

The aim of this investigation was to:

- determine the effects of short-term endurance type-training on $\dot{V}O_2$ kinetics in the frequency domain determined by the PRBS exercise test technique, in a population of young healthy females.

5.2) Methodology

5.2.1) Recruitment

Twenty-eight female subjects (age = 22.9 ± 3.1 years, height = 1.64 ± 0.05 m and body mass = 62.9 ± 7.9 kg) were recruited from the student population at Sheffield Hallam University. Prior to inclusion into the study population, all subjects were screened for cardiovascular, respiratory and musculo-skeletal disorders, and provided information

concerning habitual activity levels, by means of a health and activity questionnaire (c.f. Appendix A.3.3). No subjects were on restricted diets. Written informed consent was obtained from each individual prior to commencement of the study (c.f. Appendix A.3.4 and A.3.5). Habitual activity levels were calculated for the subject cohort using a variation on the guidelines of the Allied Dunbar National Fitness Survey (1992) (c.f. Chapter 2, Section 2.5.3).

5.2.2) Group assignment

Subjects were non-randomly assigned to either a training or non-training sub-group on the basis of an interview to ascertain information concerning personal preference and degree of commitment to the investigation. As the likelihood of young females to participate continually in regular sporting activities or exercise programmes has been shown to be lower than for their male counterparts (Lynch and Main, 1993 and, Cauley et al., 1991), sub-group assignment on a non-random basis was deemed to be the most effective method of reducing drop-out and enhancing adherence. It was hoped that this would eliminate the problems reported by Marcus and Stanton (1993), where 33% of the women were not complying with American College of Sports and Medicine (1995) guidelines for exercise prescription at the end of nine weeks of exercise training.

5.2.3) Training sub-group methodology

5.2.3.1) Subjects

Fifteen subjects (age = 21.6 ± 1.9 years, height = 1.65 ± 0.05 m and body mass = 65.0 ± 9.8 kg) were assigned to the training sub-group.

5.2.3.2) Exercise testing

All subjects completed a PRBS exercise test to determine $\dot{V}O_2$ kinetics (c.f. Chapter 2, Section 2.3.1) and a progressive exercise test to exhaustion (c.f. Chapter 2, Section 2.3.2) at weeks 0, 4 and 8, thus allowing the assessment of training adaptations. Oxygen uptake and heart rate were measured throughout the exercise testing procedures (c.f. Chapter 2, Section 2.2.1 and 2.2.2). Individual seat height was recorded during the first PRBS exercise test and then maintained during subsequent exercise testing sessions (c.f. Chapter 2, Section 2.2.3).

5.2.3.3) Exercise training

The design of the exercise prescription was based on the guidelines advocated by the American Heart Association (Fletcher et al., 1990, and McHenry et al., 1990) and the American College of Sports Medicine (1995) for exercise training.

Training consisted of eight weeks of endurance exercise. Forty minute training sessions were attended on three occasions per week and involved cycle ergometry exercise. Each training session consisted of a 5 min warm-up period at 50% of the training work rate, followed by 30 min of exercise at the prescribed intensity, and concluded with a 5 min cool-down stage at 50% of the training work rate. A training intensity of 90% maximal heart rate ($90.5 \pm 3.1\%$ actual peak heart rate), determined during the progressive exercise test to exhaustion and equivalent to $83.8 \pm 2.9\% \dot{V}O_{2peak}$, was selected in accordance with the upper limit of the training intensity range advocated by the American College of Sports Medicine (1995) for a healthy population. This exercise intensity was achieved by altering the resistance applied to the cycle ergometer. All subjects cycled at a cadence of $60 \text{ rev}\cdot\text{min}^{-1}$. Group exercise training sessions were

scheduled at the convenience of the subjects. In order to achieve maximal adherence, this exercise prescription comprised high intensity exercise but minimal exercise frequency and duration.

To ensure that all subjects conformed to the exercise prescription, heart rate telemetry was used to monitor heart rate throughout each training session. Heart rate data were collected on a 5 s time-averaged basis using sports watches (Polar Sports Tester PE4000, Polar Electro Oy, Kempele, Finland). These were wireless monitors which consisted of a transmitter unit worn on the chest and a receiver unit worn on the wrist. Electrodes placed against the chest relayed impulses from the heart to the receiver unit where they were displayed. Following each training session, heart rate data were downloaded from each receiver unit using an interface unit (Polar Computer Interface Unit, Polar Electro Oy, Kempele, Finland) and specialised software (Polar Heart rate Analysis Software Version 3, Polar Electro Oy, Kempele, Finland).

Programme charts were used to monitor the progress of the subjects over the eight week training period. During each training session the work rate used to elicit the 90% maximal heart rate exercise prescription was recorded and used as a reference for the next training session. When the training heart rate fell below the 90% maximal heart rate exercise prescription the training work rate was increased by 25 W for the subsequent session.

5.2.3.4) Data analysis

The breath-by-breath data collected during the PRBS exercise tests were subjected to Fourier analysis to produce measures of $\dot{V}O_2$ kinetics at harmonic numbers 1 to 4 (c.f.

Chapter 2, Section 2.4.2.1). The data collected during the progressive exercise tests to exhaustion were averaged on a 30 s time basis (c.f. Chapter 2, Section 2.4.2.2).

The number of criteria fulfilled by each training subject during the progressive exercise test to exhaustion (c.f. Chapter 2, Section 2.4.2.2) was documented to ensure the attainment of $\dot{V}O_{2\max}$ or $\dot{V}O_{2\text{peak}}$.

5.2.3.5) Statistical analysis

5.2.3.5.1) Training work rate

The mean \pm SD were calculated for the training work rates at weeks 0, 4 and 8.

Histograms were plotted to ensure that the data points were normally distributed and occurred within 2 SD of the mean.

Changes in training work rate over the training period were assessed using a two-way repeated measures analysis of variance without replication. A significance level of $\alpha \leq 0.05$ was selected. Significant differences between training work rate at 0, 4 and 8 weeks were investigated further by means of *post hoc* Tukey tests. The changes in training work rate over the eight week period were calculated and expressed as percentage changes.

A box and whisker plot was used to display any significant changes in training work rate.

5.2.3.5.2) Training effects

The mean \pm SD were calculated for each data set at weeks 0, 4 and 8. Histograms were plotted to ensure that the data points were normally distributed and occurred within 2

SD of the mean.

Adaptations in aerobic power and $\dot{V}O_2$ kinetics due to the endurance-type training programme were assessed using a two-way repeated measures analysis of variance without replication. A significance level of $\alpha \leq 0.05$ was selected. Significant differences between aerobic power and $\dot{V}O_2$ kinetics at 0, 4 and 8 weeks were investigated further by means of *post hoc* Tukey tests. The adaptations in aerobic power and $\dot{V}O_2$ kinetics over the eight week period were calculated and expressed as percentage changes.

Box and whisker plots were used to display any significant training adaptations.

5.2.4) Non- training sub-group methodology

5.2.4.1) Subjects

Thirteen subjects (age = 24.3 ± 3.6 years, height = 1.63 ± 0.05 m and body mass = 60.3 ± 3.9 kg) were assigned to the non-training sub-group.

5.2.4.2) Exercise testing

All subjects completed a PRBS exercise test to determine $\dot{V}O_2$ kinetics (c.f. Chapter 2, Section 2.3.1) and a progressive exercise test to exhaustion (c.f. Chapter 2, Section 2.3.2) at weeks 0, 4 and 8, thus allowing the assessment of variation in submaximal and maximal parameters. Oxygen uptake and heart rate were measured throughout the exercise testing procedures (c.f. Chapter 2, Section 2.2.1 and 2.2.2). Individual seat height was recorded during the first PRBS exercise test and then maintained during subsequent exercise testing sessions (c.f. Chapter 2, Section 2.2.3).

5.2.4.3) Activity logs

All subjects assigned to the non-training study sub-group were required to complete weekly activity logs (c.f. Appendix A.3.6). The guidelines advocated by the Allied Dunbar National Fitness Survey (1992) were used to calculate the activity levels of the non-training group.

5.2.4.4) Data analysis

The breath-by-breath data collected during the PRBS exercise tests were subjected to Fourier analysis to produce measures of $\dot{V}O_2$ kinetics at harmonic numbers 1 to 4 (c.f. Chapter 2, Section 2.4.2.1). The data collected during the progressive exercise tests to exhaustion were averaged on a 30 s time basis (c.f. Chapter 2, Section 2.4.2.2).

The number of criteria fulfilled by each non-training subject during the progressive exercise test to exhaustion (c.f. Chapter 2, Section 2.4.2.2) was documented to ensure the attainment of $\dot{V}O_{2max}$ or $\dot{V}O_{2peak}$.

5.2.4.5) Statistical analysis

The mean \pm SD were calculated for each data set at weeks 0, 4 and 8. Histograms were plotted to ensure that the data points were normally distributed and occurred within 2 SD of the mean.

5.2.4.5.1) Activity logs

To ensure that the non-training subjects had maintained their habitual activity levels, activity log scores for weeks 0 to 4, and 5 to 8 were compared by means of a two-way

repeated measures analysis of variance without replication. In addition, these activity scores were compared to the habitual activity levels calculated from the health and activity questionnaires of the non-training subjects. A significance level of $\alpha \leq 0.05$ was selected.

5.2.4.5.2) Assessment of short term variation

An assessment of the variation in aerobic power and $\dot{V}O_2$ kinetics over the study period was made using a two-way repeated measures analysis of variance without replication. A significance level of $\alpha \leq 0.05$ was selected. Any variation in aerobic power and $\dot{V}O_2$ kinetics over the eight week period were calculated and expressed as percentage changes.

5.3) Results

5.3.1) *Training group*

5.3.1.1) Training

5.3.1.1.1) Adherence

All subjects completed the prescribed twenty-four exercise training sessions over the eight week period at the required exercise intensity.

5.3.1.1.2) Training work rate

During the exercise sessions, training work rate increased significantly (31.7%, $P < 0.001$) between week 0 (training work rate = 90.5 ± 17.1 W) and week 4 (training work rate = 117.0 ± 23.2 W). No further significant increases in training work rate were observed between week 4 and 8. Overall, a 42.5% ($P < 0.001$) increase in training work rate was observed between week 0 (training work rate = 90.5 ± 17.1 W) and week 8 (training work rate = 125.1 ± 23.1 W). A box and whisker plot displaying training work rate at week 0, 4 and 8 is shown in figure 5.1.

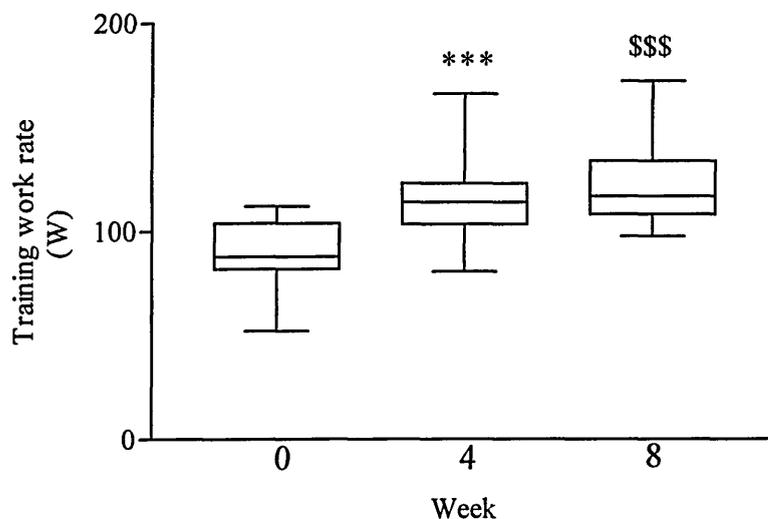


Figure 5.1) A box and whisker plot showing the training work rate (W) used to elicit a 90% maximal heart rate exercise prescription at weeks 0, 4 and 8 for the training study sub-group. The box represents the upper and lower quartiles (inter-quartile range), with the dividing line indicating the median. The whiskers show the ranges of values with the ends of the whiskers representing the 2.5% and the 97.5% values (Altman, 1991).

*** and \$\$\$ = significant difference between training work rates at weeks 0 and 4

($P < 0.001$) and at weeks 0 and 8 ($P < 0.001$) respectively.

At week 0, a narrower range of training work rates was apparent, between extreme values, when compared to the distribution of the training work rates adopted at week 4 and 8. The median was nearer to the lower limit of the inter-quartile range, with training work rates in the lower quartile falling into a wider range than those values in the upper quartile (c.f. figure 5.1).

At week 4, a wider range of training work rates was apparent, between extreme values, when compared to the distribution of the training work rates adopted at week 4 and 8. The median value was nearer to the upper limit of the inter-quartile range with training work rates in the lower quartile falling into a narrower range than those values in the upper quartile (c.f. figure 5.1).

At week 8, a wider range of training work rates was apparent when compared to the distribution of training work rates adopted at week 0. The training work rate range at week 8 was narrower than that at week 4. The median value was nearer to the lower limit of the inter-quartile range, with training work rates in the lower quartile falling into a narrower range than those values in the upper quartile (c.f. figure 5.1).

5.3.1.2) Progressive exercise testing

At week 0, five subjects (33.3% of the training study sub-group) attained $\dot{V}O_{2max}$ during the progressive exercise test to exhaustion by achieving a plateau in the relationship between $\dot{V}O_2$ and work rate. At weeks 4 and 8, two subjects (13.3% of the training study sub-group) and three subjects (20% of the training study sub-group) achieved $\dot{V}O_{2max}$ respectively. The remaining subjects all attained $\dot{V}O_{2peak}$ at weeks 0, 4 and 8 by fulfilling one or both of the criteria for $\dot{V}O_{2peak}$ (c.f. Chapter 2, Section 2.4.2.2). Of these criteria all subjects achieved an RER of 1.15 or above at weeks 0, 4 and 8. Only

46.7% (7 subjects), 33.3% (5 subjects) and 40.0% (6 subjects) attained a maximal heart rate within 10 beats·min⁻¹ of APMHR (c.f. Appendix A.4.2) at weeks 0, 4 and 8 respectively. As a minority of subjects fulfilled the criteria for $\dot{V}O_{2max}$, the term $\dot{V}O_{2peak}$ was used to describe the response of the training cohort.

The results for the progressive exercise test to exhaustion performed at weeks 0, 4 and 8 are displayed in table 5.2.

Table 5.2) Progressive exercise test results at weeks 0, 4 and 8 of the exercise training programme for the training study sub-group (mean ± SD) (n = 15).

	Week 0	Week 4	Week 8
Body mass	65.0 ±	65.0 ±	65.2 ±
(kg)	9.8	9.8	9.3
Relative $\dot{V}O_{2peak}$	34.2 ±	35.7 ±	37.0 ±***
(ml·kg⁻¹·min⁻¹)	4.5	4.1***	3.5\$\$\$
Absolute $\dot{V}O_{2peak}$	2189 ±	2289 ±	2385 ±***
(ml·min⁻¹)	170	197***	203\$\$\$
Peak heart rate	186 ±	185 ±	188 ±
(beats·min⁻¹)	9	9	9
Peak work rate	178.9 ±	202.6 ±	214.0 ±***
(W)	13.9	17.4***	14.4\$\$\$

***= significant difference between weeks 0 and 4, or weeks 4 and 8 at P < 0.001.

\$\$\$ = significant difference between weeks 0 and 8 at P < 0.001 (overall change).

At week 0 relative $\dot{V}O_{2peak}$ ranged from 29.1 to 40.0 ml·kg⁻¹·min⁻¹ and absolute $\dot{V}O_{2peak}$ values ranged from 1980 to 2595 ml·min⁻¹. At week 4 relative $\dot{V}O_{2peak}$ ranged from 28.2 to 40.7 ml·kg⁻¹·min⁻¹ and absolute $\dot{V}O_{2peak}$ values ranged from 2032 to 2813 ml·min⁻¹. At week 8 relative $\dot{V}O_{2peak}$ ranged from 30.8 to 42.7 ml·kg⁻¹·min⁻¹ and absolute $\dot{V}O_{2peak}$ values ranged from 2085 to 2876 ml·min⁻¹.

5.3.1.2.1) Training adaptations detected from peak responses

Training adaptations in relative $\dot{V}O_{2peak}$ occurred between week 0 (relative $\dot{V}O_{2peak} = 34.2 \pm 4.5$ ml·kg⁻¹·min⁻¹) and week 4 (relative $\dot{V}O_{2peak} = 35.7 \pm 4.1$ ml·kg⁻¹·min⁻¹). A 4.7% (P<0.001) increase in relative $\dot{V}O_{2peak}$ was observed during the first four weeks of the training programme. Further adaptations in relative $\dot{V}O_{2peak}$ were seen to occur between weeks 4 and 8 (relative $\dot{V}O_{2peak} = 37.0 \pm 3.5$ ml·kg⁻¹·min⁻¹). A 4.0% (P<0.001) increase in relative $\dot{V}O_{2peak}$ occurred during the last four weeks of the training period. Overall, an 8.7% (P<0.001) increase in relative $\dot{V}O_{2peak}$ was observed as a result of the eight week training programme. Training adaptations in absolute $\dot{V}O_{2peak}$ occurred between week 0 (absolute $\dot{V}O_{2peak} = 2189 \pm 170$ ml·min⁻¹) and week 4 (absolute $\dot{V}O_{2peak} = 2289 \pm 197$ ml·min⁻¹). A 4.6% (P<0.001) increase in absolute $\dot{V}O_{2peak}$ was observed during the first four weeks of the training programme. Further adaptations in absolute $\dot{V}O_{2peak}$ were seen to occur between weeks 4 and 8 (absolute $\dot{V}O_{2peak} = 2385 \pm 203$ ml·min⁻¹). A 4.5% (P<0.001) increase in absolute $\dot{V}O_{2peak}$ occurred during the last four weeks of the training period. Overall, a 9.1% (P<0.001) increase in absolute $\dot{V}O_{2peak}$ was observed as a result of the eight week training programme. Box and whisker plots displaying relative $\dot{V}O_{2peak}$ and absolute $\dot{V}O_{2peak}$ at weeks 0, 4 and 8 are

shown in figure 5.2. Body mass was found to be unchanged following this short-term training programme. Although similar maximal heart rates were attained by the training study sub-group at weeks 0, 4 and 8, significant increases in peak work rate resulted from the training programme. Improvements in peak work rate were observed between week 0 (peak work rate = 178.9 ± 13.9 W) and week 4 (peak work rate = 202.6 ± 17.4 W). During the first four weeks of the programme a 13.4% ($P < 0.001$) increase in peak work rate occurred.

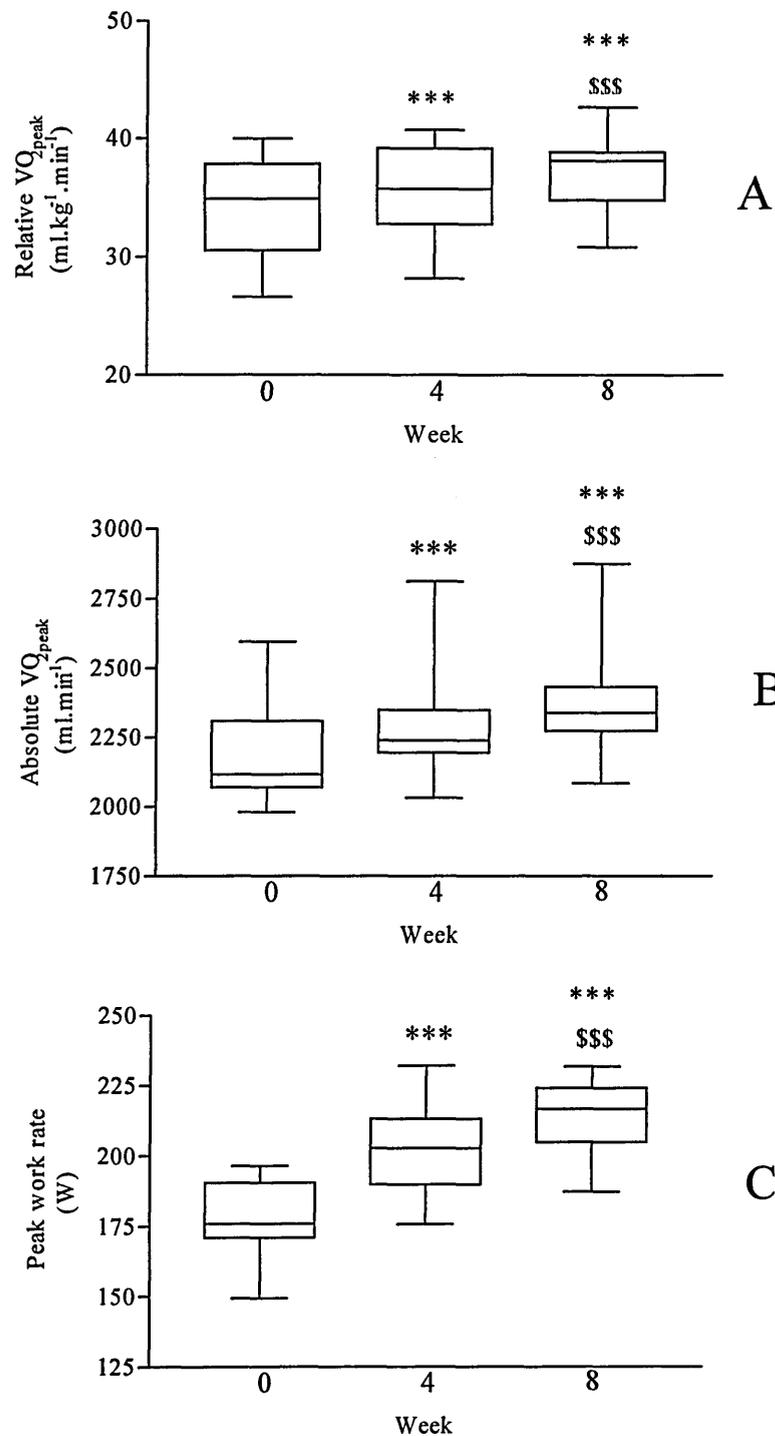


Figure 5.2) Box and whisker plots showing (A) relative $\dot{V}O_{2peak}$ ($ml \cdot kg^{-1} \cdot min^{-1}$), (B) absolute $\dot{V}O_{2peak}$ ($ml \cdot min^{-1}$) and (C) peak work rate (W) at weeks 0, 4 and 8 for the training study sub-group. The box represents the upper and lower quartiles (interquartile range), with the dividing line indicating the median. The whiskers show the ranges of values with the ends of the whiskers representing the 2.5% and the 97.5% values (Altman, 1991). *** = significant difference between weeks 0 and 4, or weeks 4 and 8 ($P < 0.001$). \$\$\$ = significant difference between weeks 0 and 8 ($P < 0.001$).

Further improvements in peak work rate were seen between weeks 4 and 8 (peak work rate = 214 ± 14.4 W). A 6.6% ($P < 0.001$) increase in peak work rate occurred during the last four weeks of the programme. Overall a 20.0% ($P < 0.001$) increase in peak work rate was observed over the duration of the study period. A box and whisker plot displaying peak work rate at weeks 0, 4 and 8 is shown in figure 5.2.

5.3.1.3) PRBS exercise test

5.3.1.3.1 DYNAMIC GAIN AND OVERALL RESPONSES

All subjects completed a PRBS exercise test to determine $\dot{V}O_2$ kinetics at weeks 0, 4 and 8. At week 0 a dynamic gain for $\dot{V}O_2$ of 1062 ± 82 ml·min⁻¹ and a mean overall heart rate response of 128 ± 13 beats·min⁻¹ resulted from the PRBS exercise test. At week 4 a dynamic gain for $\dot{V}O_2$ of 1024 ± 75 ml·min⁻¹ and a mean overall heart rate response of 120 ± 15 beats·min⁻¹ resulted from the PRBS exercise test. At week 8 a dynamic gain for $\dot{V}O_2$ of 999 ± 86 ml·min⁻¹ and a mean overall heart rate response of 115 ± 12 beats·min⁻¹ resulted from the PRBS exercise test. Significant differences in the dynamic gain for $\dot{V}O_2$ ($P < 0.05$) and overall mean heart rate response ($P < 0.01$) to the PRBS exercise test were seen between weeks 0 and 8.

5.3.1.3.2 OXYGEN UPTAKE KINETICS

The results for the PRBS exercise test at weeks 0, 4 and 8 are displayed in table 5.3.

Table 5.3) $\dot{V}O_2$ kinetics at weeks 0, 4 and 8 of the exercise training programme for the training study sub-group (mean \pm SD) (n = 15).

	Week 0	Week 4	Week 8
Amplitude at harmonic number 1 (ml·min ⁻¹ ·W ⁻¹)	10.306 \pm	10.012 \pm	10.150 \pm
	0.431	0.503	0.523
Amplitude at harmonic number 2 (ml·min ⁻¹ ·W ⁻¹)	6.762 \pm	7.230 \pm	7.487 \pm
	0.735	0.704	0.762 ^{\$}
Amplitude at harmonic number 3 (ml·min ⁻¹ ·W ⁻¹)	8.716 \pm	8.845 \pm	9.245 \pm
	0.881	0.832	1.269
Amplitude at harmonic number 4 (ml·min ⁻¹ ·W ⁻¹)	8.172 \pm	8.331 \pm	8.469 \pm
	0.886	1.236	1.057
Phase shift at harmonic number 1 (degrees)	-25.7 \pm	-22.3 \pm	-21.9 \pm
	3.0	2.4***	2.6 ^{\$\$\$}
Phase shift at harmonic number 2 (degrees)	-43.9 \pm	-42.5 \pm	-39.3 \pm
	4.4	3.8	6.0 ^{\$}
Phase shift at harmonic number 3 (degrees)	-72.3 \pm	-67.7 \pm	-64.7 \pm
	8.1	5.6	10.1 ^{\$}
Phase shift at harmonic number 4 (degrees)	-78.5 \pm	-77.2 \pm	-77.1 \pm
	5.6	7.2	4.9

*** = significant difference between weeks 0 and 4 (P<0.001). ^{\$} and ^{\$\$\$} = significant

difference between weeks 0 and 8 (P<0.05) and (P<0.001) respectively.

5.3.1.3.2.1) Training adaptations in oxygen uptake kinetics

Training adaptations in $\dot{V}O_2$ kinetics were observed as higher amplitudes and smaller

phase shifts. A significant increase (7.6%, $P < 0.05$) in amplitude at harmonic number 2 was seen between week 0 (amplitude at harmonic number 2 = 6.762 ± 0.735 ml·min⁻¹·W⁻¹) and week 8 (amplitude at harmonic number 2 = 7.487 ± 0.762 ml·min⁻¹·W⁻¹). Significant changes in amplitude at harmonic number 2 were not apparent during the first four weeks of the training programme. A box and whisker plot displaying amplitude at harmonic number 2 at weeks 0, 4 and 8 is shown in figure 5.3.

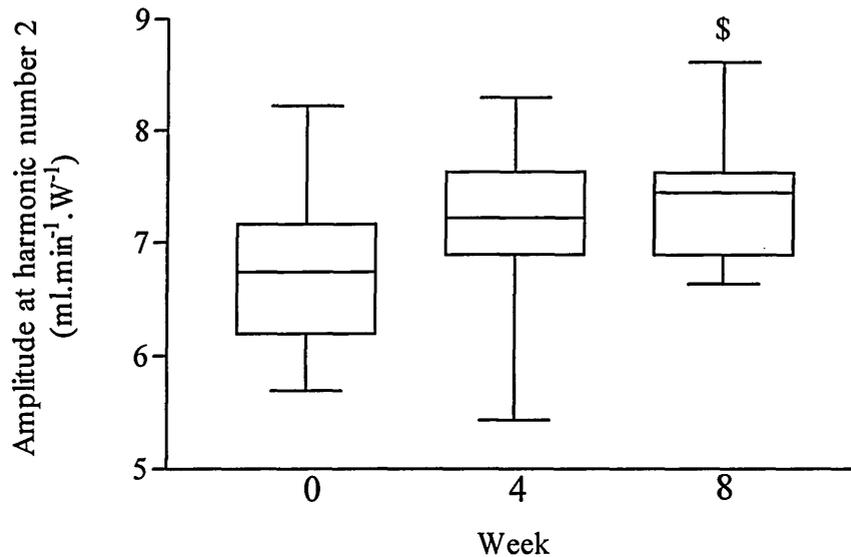


Figure 5.3) A box and whisker plot showing amplitude at harmonic number 2 (ml·min⁻¹·W⁻¹) at weeks 0, 4 and 8 for the training study sub-group. The box represents the upper and lower quartiles (inter-quartile range), with the dividing line indicating the median. The whiskers show the ranges of values with the ends of the whiskers representing the 2.5% and the 97.5% values (Altman, 1991). \$ = significant difference between weeks 0 and 8 ($P < 0.05$).

A significant reduction in phase shifts at harmonic number 1 occurred between week 0 (phase shift at harmonic number 1 = -25.7 ± 3.0 degrees) and week 4 (phase shift at harmonic number 1 = -22.3 ± 2.4 degrees). During the first four weeks of the training programme a 12.3% ($P < 0.001$) decrease in phase shift at harmonic number 1 was

observed. No further significant change occurred between week 4 (phase shift at harmonic number 1 = -22.3 ± 2.4 degrees) and week 8 (phase shift at harmonic number 2 = -21.9 ± 2.6 degrees). Overall a 14.1% ($P < 0.001$) reduction in phase shift at harmonic number 1 occurred during the eight week period. Significantly smaller phase shifts at harmonic numbers 2 and 3 were only observed after eight weeks of cycle ergometry training. A 9.6 % ($P < 0.05$) decrease in phase shift occurred at harmonic number 2 between week 0 (phase shift at harmonic number 2 = -43.9 ± 4.4 degrees) and week 8 (phase shift at harmonic number 2 = -39.3 ± 6.0 degrees). Similarly a 9.6% ($P < 0.05$) decrease in phase shift at harmonic number 3 was detected between week 0 (phase shift at harmonic number 3 = -72.3 ± 8.1 degrees) and week 8 (phase shift at harmonic number 3 = -64.7 ± 10.1 degrees). Box and whisker plots displaying phase shift at harmonic numbers 1, 2 and 3 at week 0, 4 and 8 are shown in figure 5.4.

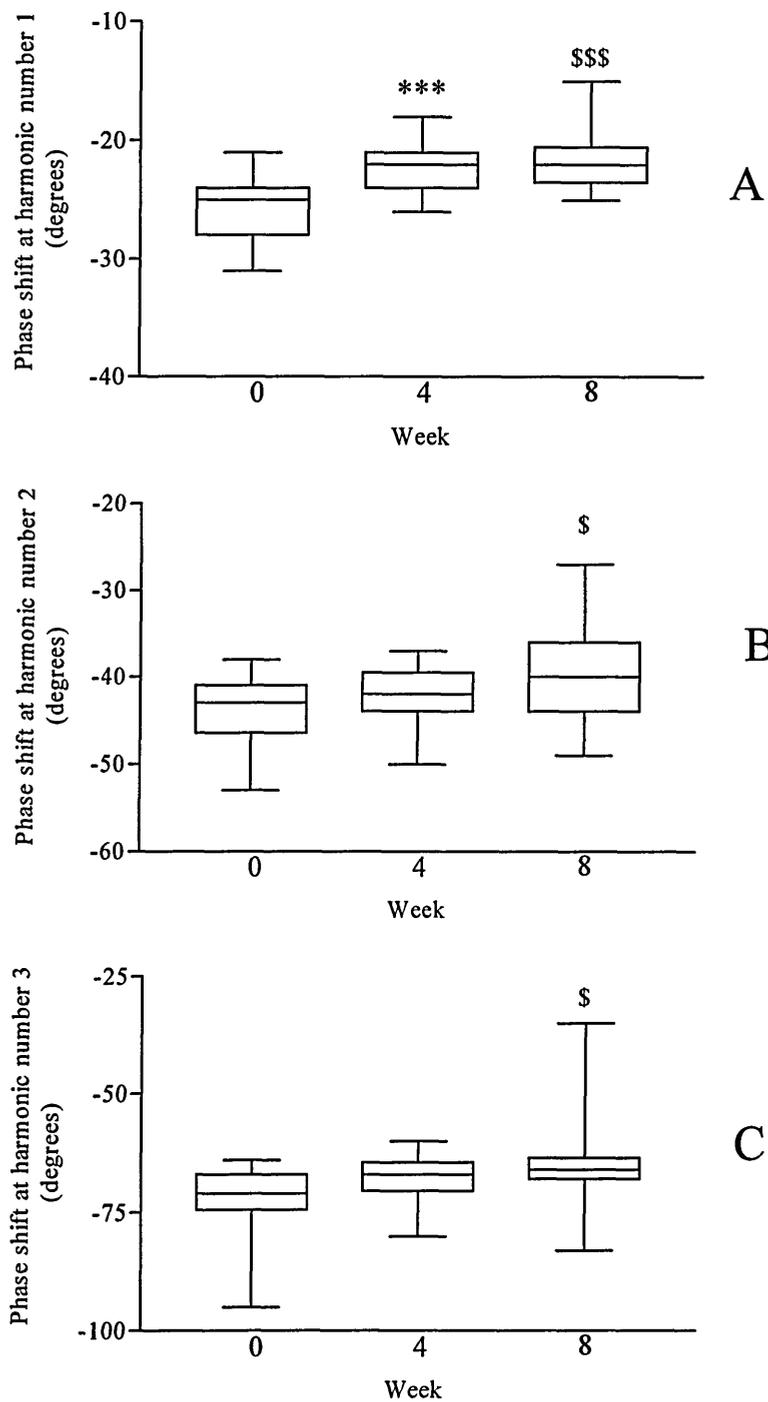


Figure 5.4) Box and whisker plots showing (A) phase shift at harmonic number 1 (degrees), (B) phase shift at harmonic number 2 (degrees), and (C) phase shift at harmonic number 3 (degrees), at weeks 0, 4 and 8 for the training study sub-group. The box represents the upper and lower quartiles (inter-quartile range), with the dividing line indicating the median. The whiskers show the ranges of values with the ends of the whiskers representing the 2.5% and the 97.5% values (Altman, 1991). *** = significant difference between weeks 0 and 4 ($P < 0.001$). \$ = significant difference between weeks 0 and 8 ($P < 0.05$). \$\$\$ = significant difference between weeks 0 and 8 ($P < 0.001$).

Training adaptations in amplitude and phase shift for the training study sub-group are summarised in figure 5.5.

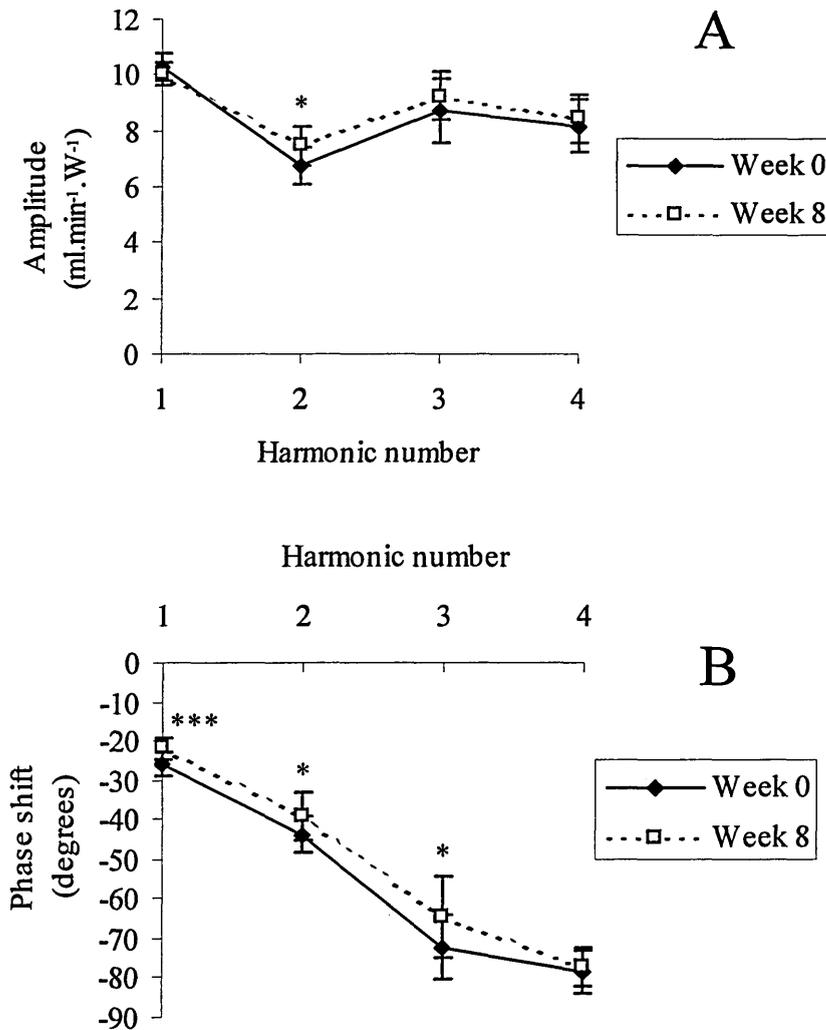


Figure 5.5) Training adaptations in (A) amplitude ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$) and (B) phase shift (degrees) resulting from the eight week cycle ergometry exercise programme for the training study sub-group. * = significant difference between weeks 0 and 8 ($P<0.05$).

*** = significant difference between weeks 0 and 8 ($P<0.001$).

The training adaptations in aerobic power and $\dot{V}O_2$ kinetics observed over the eight week training programme are summarised in figure 5.6. Individual training adaptations in $\dot{V}O_2$ kinetics are shown in Appendix 5.

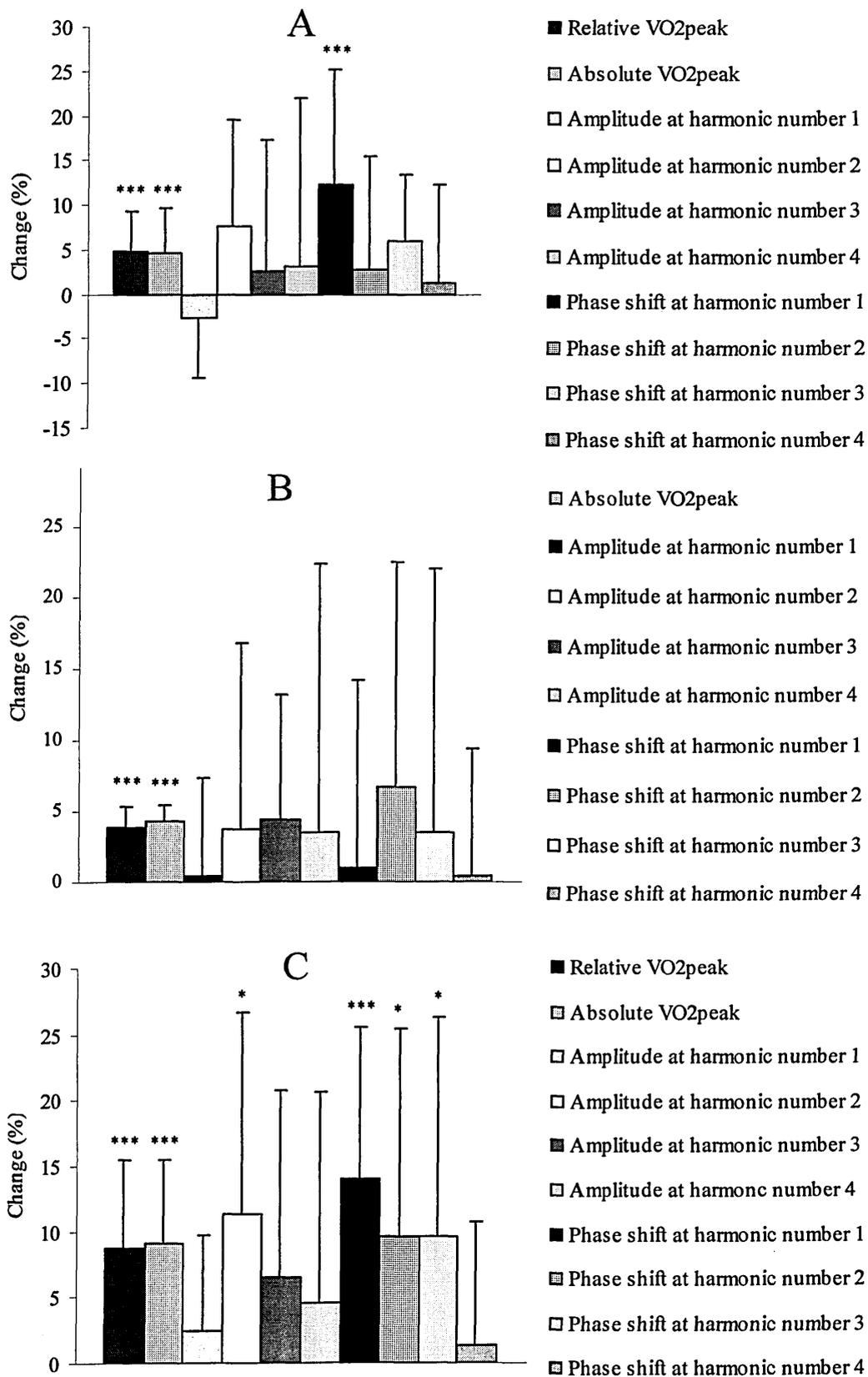


Figure 5.6) Changes (%) in $\dot{V}O_{2peak}$ and $\dot{V}O_2$ kinetics between (A) weeks 0 and 4, (B) weeks 4 and 8, and (C) weeks 0 and 8. * and *** = significant changes at $P < 0.05$ and $P < 0.001$ respectively.

5.3.2) *Non-training group*

5.3.2.1) Activity logs

During the eight week study period there was no significant change in the activity levels of the non-training sub-group. Activity scores of 3.6 ± 2.2 and 4.0 ± 1.9 were calculated for weeks 0 to 4 and weeks 5 to 8 respectively. When the activity scores were compared to the habitual activity scores (3.5 ± 2.0), no significant difference was found to exist between activity levels. Thus, the subjects assigned to the non-training study sub-group did not appear to alter their activity habits during the study period. Any variation in maximal responses or $\dot{V}O_2$ kinetics therefore were unlikely to be due to a training stimulus.

5.3.2.2) Progressive exercise testing

At week 0, the non-training subjects did not achieved a plateau within the relationship between $\dot{V}O_2$ and work rate, and therefore did not attain 'true $\dot{V}O_{2max}$ '. At week 4 and week 8, three subjects (23.1% of the non-training population) achieved a plateau within the relationship between $\dot{V}O_2$ and work rate. The remaining subjects all attained $\dot{V}O_{2peak}$ during the progressive exercise tests to exhaustion performed at weeks 0, 4 and 8 by fulfilling one or both of the criteria for $\dot{V}O_{2peak}$. Of these two criteria all subjects achieved an RER of 1.15 or above. Only four subjects (30.1% of the non-training population), three subjects (23.1% of the non-training population) and two subjects (15.4% of the non-training population) attained maximal heart rates within 10 $\text{beats}\cdot\text{min}^{-1}$ of APMHR at weeks 0, 4 and 8 respectively. Although $\dot{V}O_{2max}$ had been achieved by a minority of the non-training study sub-group, the term $\dot{V}O_{2peak}$ was

adopted to describe both parameters of aerobic power.

The results of the progressive exercise tests to exhaustion performed at weeks 0, 4 and 8 are displayed in table 5.4.

At week 0, relative $\dot{V}O_{2peak}$ values ranged from 32.5 to 47.9 ml·kg⁻¹·min⁻¹ and absolute $\dot{V}O_{2peak}$ values ranged from 1986 to 2958 ml·min⁻¹. At week 4, relative $\dot{V}O_{2peak}$ values ranged from 31.9 to 47.2 ml·kg⁻¹·min⁻¹ and absolute $\dot{V}O_{2peak}$ values ranged from 1896 to 2732 ml·min⁻¹. At week 8, relative $\dot{V}O_{2peak}$ values ranged from 31.0 to 45.5 ml·kg⁻¹·min⁻¹ and absolute $\dot{V}O_{2peak}$ values ranged from 1868 to 2889 ml·min⁻¹.

Table 5.4). Progressive exercise test results at weeks 0, 4 and 8 of the study period for the non-training study sub-group (mean ± SD) (n = 13).

	Week 0	Week 4	Week 8
Body mass	60.3 ±	60.8 ±	61.2 ±
(kg)	3.9	4.4	4.2
Relative $\dot{V}O_{2peak}$	40.2 ±	39.4 ±	39.4 ±
(ml·kg⁻¹·min⁻¹)	4.7	5.1	5.6
Absolute $\dot{V}O_{2peak}$	2420 ±	2386 ±	2405 ±
(ml·min⁻¹)	265	274	319
Peak heart rate	180 ±	179 ±	178 ±
(beats·min⁻¹)	11	11	11
Peak work rate	201.8 ±	205.7 ±	208.9 ±
(W)	25.8	27.3	30.4

5.3.2.2.1) Short term variation in peak responses

No significant variation in maximal responses was apparent between weeks 0 and 4, weeks 4 and 8, or over the duration of the eight week study period.

5.3.2.3) PRBS exercise test

5.3.2.3.1 DYNAMIC GAIN AND OVERALL RESPONSES

All thirteen subjects completed a PRBS exercise test to determine $\dot{V}O_2$ kinetics at weeks 0, 4 and 8. At week 0 a dynamic gain for $\dot{V}O_2$ of $1050 \pm 79 \text{ ml}\cdot\text{min}^{-1}$ and a mean overall heart rate response of $110 \pm 12 \text{ beats}\cdot\text{min}^{-1}$ resulted from the PRBS exercise test. At week 4 a dynamic gain for $\dot{V}O_2$ of $1025 \pm 55 \text{ ml}\cdot\text{min}^{-1}$ and a mean overall heart rate response of $110 \pm 12 \text{ beats}\cdot\text{min}^{-1}$ resulted from the PRBS exercise test. At week 8 a dynamic gain for $\dot{V}O_2$ of $1014 \pm 60 \text{ ml}\cdot\text{min}^{-1}$ and a mean overall heart rate response of $105 \pm 10 \text{ beats}\cdot\text{min}^{-1}$ resulted from the PRBS exercise test. No significant difference was found to exist between the dynamic gain for $\dot{V}O_2$ and the mean overall heart rate response over the eight week study period.

5.3.2.3.2) OXYGEN UPTAKE KINETICS

Oxygen uptake kinetics for weeks 0, 4 and 8 are displayed in table 5.5.

Table 5.5) $\dot{V}O_2$ kinetics determined during the PRBS exercise tests at weeks 0, 4 and 8 for the non-training study sub-group (mean \pm SD) (n = 13).

	Week 0	Week 4	Week 8
Amplitude at harmonic number 1 (ml·min ⁻¹ ·W ⁻¹)	10.372 \pm 0.601	10.236 \pm 0.421	9.936 \pm 0.784
Amplitude at harmonic number 2 (ml·min ⁻¹ ·W ⁻¹)	7.250 \pm 1.106	7.374 \pm 1.029	7.335 \pm 0.926
Amplitude at harmonic number 3 (ml·min ⁻¹ ·W ⁻¹)	9.257 \pm 0.976	9.103 \pm 0.976	8.769 \pm 2.386
Amplitude at harmonic number 4 (ml·min ⁻¹ ·W ⁻¹)	8.273 \pm 1.065	8.289 \pm 0.923	8.478 \pm 0.955
Phase shift at harmonic number 1 (degrees)	-24.0 \pm 2.4	-23.2 \pm 34	-21.4 \pm 2.7
Phase shift at harmonic number 2 (degrees)	-40.2 \pm 4.8	-38.0 \pm 6.4	-39.9 \pm 4.5
Phase shift at harmonic number 3 (degrees)	-67.3 \pm 6.7	-68.7 \pm 8.1	-64.4 \pm 4.8
Phase shift at harmonic number 4 (degrees)	-77.5 \pm 7.9	-76.8 \pm 5.4	-74.5 \pm 5.6

5.3.2.3.2.1) Short-term variation in oxygen uptake kinetics

No significant variation in $\dot{V}O_2$ kinetics was apparent between weeks 0 and 4, weeks 4 and 8 or over the duration of the eight week study period.

Short-term variations in amplitude and phase shift for the non-training study sub-group

are summarised in figure 5.7.

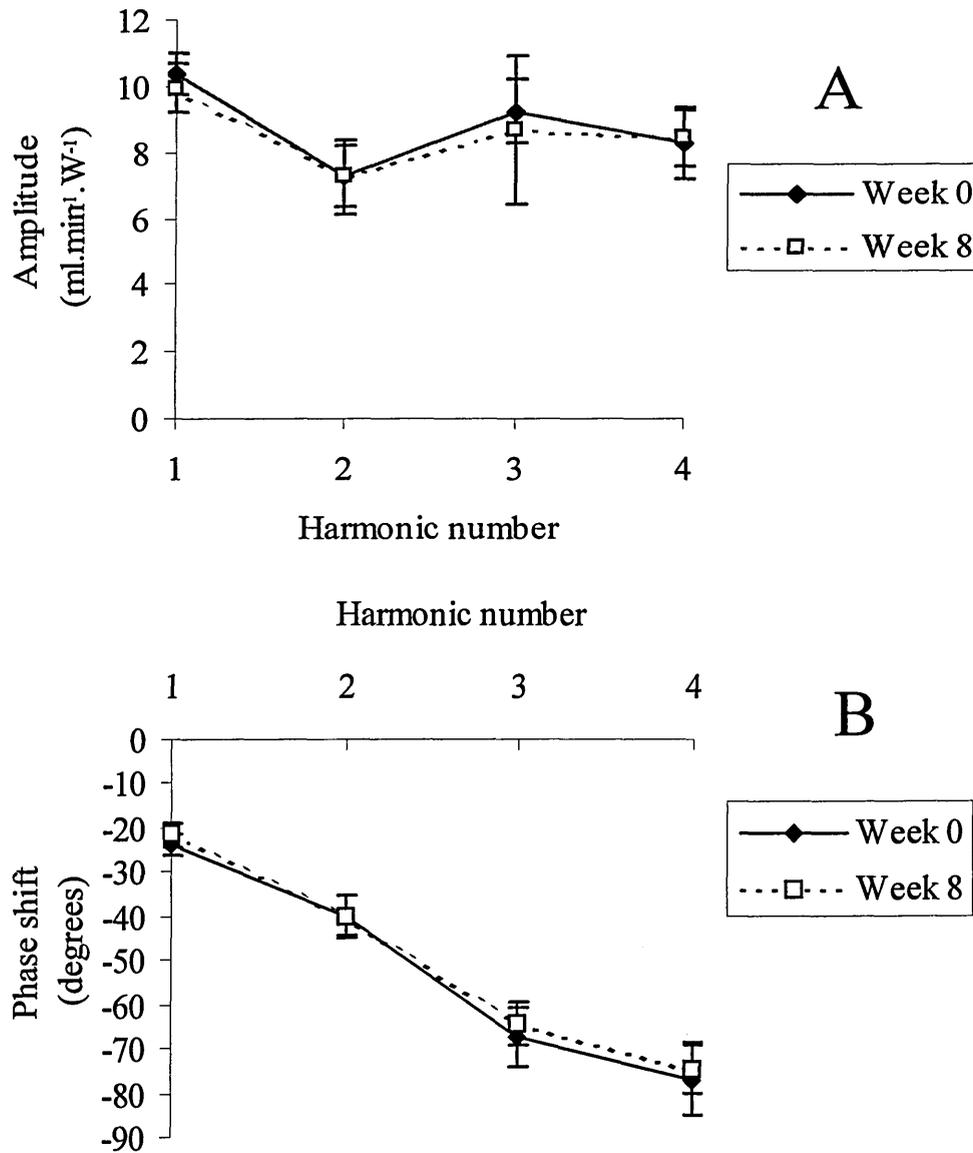


Figure 5.7) Short-term variation in (A) amplitude ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$) and (B) phase shift (degrees) before (week 0) and after (week 8) the study period for the non-training study sub-group.

5.4) Discussion

The purpose of this investigation was to assess the effects of short-term endurance-type training on the $\dot{V}O_2$ kinetics, in the frequency domain, of young, healthy females.

5.4.1) Adherence

In contrast to the findings of Marcus and Stanton (1993), which reported reduced adherence rates for the continual participation of young females in exercise programmes, a 100% adherence rate was achieved during the present study. The enhanced adherence and lack of drop-out may have partially resulted from the non-random allocation of subjects to study sub-groups, the short-term nature of the exercise programme and arrangement of the training sessions at the convenience of the participants. The present study involved the use of a training model which was comparable to the minimum requirements for frequency and duration with high intensity exercise (American College of Sports Medicine, 1995). The high adherence rate achieved during this investigation could have resulted from the non-random assignment of subjects to the study sub-groups.

5.4.2) Training work rate

During the eight week training period significant increases in the training work rate were needed to maintain the 90% maximal heart rate (90.5% actual peak heart rate) exercise prescription. This training adaptation was a direct result of a decrease in submaximal exercising heart rate. Evidence for a reduction in submaximal exercising heart rate has been described by Smith et al. (1976), Saltin et al. (1976), McCord and Paterson (1989) and Govindasamy et al. (1992). Smith et al. (1976) reported a reduction in submaximal exercising heart rate for young, healthy females participating in a nine week programme of cycle ergometry exercise. A 33% increase in training work load (training work load at week 0 = $426 \text{ kpm}\cdot\text{min}^{-1}$, training work load at week 9 = $639 \text{ kpm}\cdot\text{min}^{-1}$) was needed to maintain an exercise intensity of 70% maximal heart rate

range. In agreement with the findings of Smith et al. (1976), greater increases in training work rate were needed to maintain the exercise intensity during the first few weeks of the exercise programme, with more modest increases in work rate being applied to the cycle ergometer between weeks 4 to 8. In agreement with the findings of Hickson et al., 1978, and Hickson et al., 1981), the present study has shown that significant short-term training adaptations can result from a progressive training model.

5.4.3) Training adaptations

5.4.3.1) Progressive exercise test

Adaptations in the peak responses of the training subjects were seen as significant increases in relative (8.7%) and absolute $\dot{V}O_{2peak}$ (9.1%). The magnitude of these training adaptation was greater than the 7.6% ($P<0.05$) and the 5.2% ($P<0.05$) increases in relative and absolute $\dot{V}O_{2max}$ described by McCord and Paterson (1989). Although the session duration and frequency of these two training programmes were similar, McCord and Paterson (1989) preferred to prescribe a programme of low impact aerobic dance at an intensity of 70 to 85% maximal heart rate reserve to healthy young females for twelve weeks. As training load is related to the fitness level of an individual, the less pronounced increase in relative and absolute $\dot{V}O_{2max}$, observed by McCord and Paterson (1989), may have been due to the higher baseline exercise capacity of their subject population. Relative and absolute $\dot{V}O_{2peak}$ measured during the present study was lower suggesting that these subjects were less active than those studied by McCord and Paterson (1989).

The training adaptations demonstrated during the eight week exercise programme are in

contrast to the findings of Henritze et al. (1985) for young healthy females participating in a twelve week programme of cycle ergometry training. Subjects in this study trained at or above the lactate threshold on five occasions per week. No significant increases in relative $\dot{V}O_{2\max}$ were seen for either group of subjects following training.

Weltman et al. (1992) described the effects of a fifty-two week progressive programme of running on healthy, female subjects. Subjects in this study trained at or above the lactate threshold on three to five occasions per week and covered a distance of five to thirty-five miles per week. Significant increases in relative $\dot{V}O_{2\max}$ were seen after the eighth menstrual cycle for those subjects training at the lactate threshold, with an increase of 11% being observed by the twelfth menstrual cycle (fifty-two weeks) (pre-training relative $\dot{V}O_{2\max} = 42.7 \pm 6.2 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$, post-training relative $\dot{V}O_{2\max} = 47.4 \pm 8.0 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$, $P < 0.05$). Significant increases in relative $\dot{V}O_{2\max}$ were seen between baseline and the fourth menstrual cycle for those subjects training above the lactate threshold, with an increase of 13% being observed by the twelfth menstrual cycle (fifty-two weeks) (pre-training relative $\dot{V}O_{2\max} = 44.2 \pm 5.5 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$, post-training relative $\dot{V}O_{2\max} = 50.1 \pm 4.6 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$, $P < 0.05$).

The exercise capacity (before training) of the subject populations studied by Henritze et al. (1985) and Weltman et al. (1992) was higher than that in the present study and that reported by McCord and Paterson (1989). This may partially explain the absence of training adaptations following the exercise regimen prescribed by Henritze et al. (1985), and the length of the training programme required to produce increases in aerobic power, of a similar magnitude to those observed in the present study, in the subjects studied by Weltman et al. (1992).

5.4.3.2) Oxygen uptake kinetics

The PRBS exercise test technique has been shown to be able to detect adaptations in $\dot{V}O_2$ kinetics, in the frequency domain, following short-term, endurance-type training. Oxygen uptake kinetics for young, females were significantly faster after completing an eight week programme of cycle ergometry. This finding complements those studies which have demonstrated faster $\dot{V}O_2$ kinetics, measured in the time domain, following endurance-type training (Berry and Moritani, 1985, Yoshida et al., 1992, Babcock et al., 1994a, and Phillips et al., 1995). No significant changes in $\dot{V}O_2$ kinetics were apparent for the non-training sub-group during the study period.

Faster $\dot{V}O_2$ kinetics were seen as a significant increase in amplitude at harmonic number 2, and significant decreases in phase shift at harmonic numbers 1, 2 and 3. From these results it can be seen that the PRBS exercise test is sensitive to adaptations in $\dot{V}O_2$ kinetics at a number of harmonics. This emphasizes the importance of using a technique which provides a multi-frequent assessment of $\dot{V}O_2$ kinetics. Training adaptations in $\dot{V}O_2$ kinetics were not apparent at higher harmonic numbers. This can be partially explained when reviewing the increased variability of $\dot{V}O_2$ kinetics at higher harmonic numbers (c.f. Chapter 3).

Different time courses can be seen for these adaptations. Significant decreases in phase shift at harmonic number 1 were observed after four weeks of endurance training, whereas adaptations in $\dot{V}O_2$ kinetics at other harmonic numbers were apparent only after the whole of the training programme had been completed. Yoshida et al. (1992) reported a similar time course for adaptations in $\dot{V}O_2$ kinetics, determined during step changes in work rate, with significant decreases in τ occurring by week 3 of the training programme. In contrast, Phillips et al. (1995) observed significant decreases in τ by

day 4. The difference between these time courses may be partially due to the very aggressive nature of the training model adopted by Phillips et al. (1995) and particularly to the very long duration of the training sessions.

When considering the magnitude of the changes in $\dot{V}O_2$ kinetics following this training programme, only a limited comparison can be made with other studies due to differences in the techniques adopted to determine $\dot{V}O_2$ kinetics. Training adaptations in phase shift, representing an increase in the rate of adaptation to the pseudo random changes in work rate, ranged from 14.1% to 9.6%. The magnitude of these changes are in agreement with those calculated from the work of Berry and Moritani (1985). Berry and Moritani (1985) demonstrated an increase in the rate of adaptation to a single step change in work rate following five weeks of steady state running at an intensity of 60 to 70% of maximal heart rate reserve. The percentage change for the decrease in the time constant for $\dot{V}O_2$ of young, healthy males was equivalent to 11.2%. More pronounced changes in $\dot{V}O_2$ kinetics following endurance training were reported by Babcock et al. (1994a), Yoshida et al. (1992) and Phillips et al. (1995). Following a programme of endurance cycle ergometry exercise, Babcock et al. (1994a) described significant increases (equivalent to 49%) in the rate of adaptation to a square wave work rate forcing. Such pronounced training adaptations in $\dot{V}O_2$ kinetics can be explained to some extent when the older and more sedentary nature of the subjects, and the relatively high intensity of the exercise prescription, are considered. Phillips et al. (1995) reported a 38% decrease in the time constant for phase I of the $\dot{V}O_2$ response following four days of cycle ergometry exercise, and observed a 58% decrease in the time constant for phase II of the $\dot{V}O_2$ response following thirty days of training. Again the aggressive

nature of the training programme and the long duration of the exercise sessions could have directly influenced the magnitude of these training adaptations. Whilst the training adaptations observed during the present investigation were more modest, the less intensive training regimen ensured high adherence (100%).

5.4.4) Physiological mechanisms

5.4.4.1) Time course and extent of training adaptations in aerobic power

Many studies have shown that endurance-type training results in improvements in aerobic power but the extent and the time course of these improvements depends on the nature of the training stimulus (Govindasamy et al., 1992). In the present study, improvements of 4.6% after four weeks of training and 9.1% after eight weeks of training were found for $\dot{V}O_{2peak}$ when using a training model with a frequency of three times per week and an intensity equivalent to 90% maximal heart rate. These results show a similar time course to older, less fit subjects (Govindasamy et al., 1992), who trained at an identical frequency but at an intensity of 70 to 75% $\dot{V}O_{2max}$. Other studies, using a greater frequency, duration and intensity of training, have shown greater improvements in aerobic power (Hickson et al., 1981, Spina et al., 1992) but over a similar time course (Hickson et al., 1981). Although most of these studies have been conducted on men, there appears to be no difference between the training response of men and women (Spina et al., 1992).

Other studies have found a longer time course for improvements in aerobic power (Weltman et al., 1992, and Phillips et al., 1995) or no improvements at all following

endurance-type training (Henritze et al., 1985, and Green et al., 1992). It would appear that improvements in aerobic power are sensitive to the training model, since no improvements were found after a twelve week training programme of five days per week, at either 44% or 59% $\dot{V}O_{2max}$ (Henritze et al., 1985). Neither were improvements found after training for two hours per day, for five to seven days at 67% $\dot{V}O_{2max}$ (Green et al., 1992) although improvements were evident after ten days using the same training model (Shoemaker et al., 1996).

5.4.4.2) Time course and extent of adaptations in oxygen uptake kinetics

Improvements of 9.6% to 14.1% were found after eight weeks of training, in phase shifts at harmonic numbers 1 to 3, and amplitude at harmonic number 2. Only phase shift at harmonic number 1 showed a training induced adaptation after four weeks. There are no directly comparable studies using the PRBS exercise test technique, however a number of studies have used a step work rate input to determine $\dot{V}O_2$ kinetics before and after endurance-type training (Hagberg et al., 1980, Berry and Moritani, 1985, Yoshida et al., 1992, Babcock et al., 1994a, and Phillips et al., 1995). Studies involving younger subjects have shown changes in $\dot{V}O_2$ kinetics of between 6% (Berry and Moritani, 1985) and 26% (Phillips et al., 1995) after training. Greater improvements have been shown in much older subjects (Babcock et al., 1994b) and in studies where step changes in work rate may have introduced a slow component (Hagberg et al., 1980, and Yoshida et al., 1992) into the resultant $\dot{V}O_2$ kinetics. Faster $\dot{V}O_2$ kinetics have been demonstrated after only four days of high intensity training, undertaken for two hours per day (Phillips et al., 1995) although other studies have not shown improvements until the completion of fifteen days of training (Yoshida

et al., 1992). The difference in improvements may be due to the differences in the duration of training, since the subjects in the study of Yoshida et al., 1992) trained for thirty minutes per day. In the present study, the first training response (phase shift at harmonic number 1) was evident after only six hours of training, which suggests that these results are comparable to the time courses for $\dot{V}O_2$ kinetics of eight hours and seven and a half hours found in previous studies (Phillips et al., 1995, and Yoshida et al., 1992).

5.4.4.3) Comparison of the time courses of aerobic power and oxygen uptake kinetics

Of the two studies which compare the time courses of aerobic power and $\dot{V}O_2$ kinetics, one showed that the rate of adaptation in maximal and submaximal parameters were similar and changed significantly after approximately two weeks of training (Yoshida et al., 1992). The other study (Phillips et al., 1995) showed that the improvement in $\dot{V}O_2$ kinetics precedes the changes in $\dot{V}O_{2max}$, with $\dot{V}O_2$ kinetics improving after four days of training while $\dot{V}O_{2max}$ was still unchanged after nine days of training. In the present study, both $\dot{V}O_2$ kinetics and $\dot{V}O_{2peak}$ had increased after twelve days of training (after four weeks) which supports the findings of Yoshida et al. (1992).

5.4.4.4) Mechanisms of training adaptations due to endurance exercise

Endurance training results in improvements in cardiovascular function and in muscle oxidative capacity, with increases in aerobic power having been linked to these changes (Ekblom et al., 1968, Saltin et al., 1976, Spina et al., 1992, Coggan et al., 1993, and Phillips et al., 1995). Since these factors are also implicated in the control of $\dot{V}O_2$

kinetics, it would be expected that these improvements would result in faster $\dot{V}O_2$ kinetics.

Phillips et al. (1995) however have shown that $\dot{V}O_2$ kinetics improve before changes in $\dot{V}O_{2peak}$ or muscle oxidative potential are seen, although in the present study it was not possible to show a dissociation between improvements in $\dot{V}O_{2peak}$ and $\dot{V}O_2$ kinetics. Training brings about an improvement in the mitochondrial oxidative response to a given decrease in PCr, in effect tighter metabolic control, which means that a given increase in $\dot{V}O_2$ requires a smaller decrease in PCr (Cadefau et al., 1994). Phillips et al. (1996) have shown that after five days of training (approximately ten hours), muscle lactate concentration, PCr hydrolysis and glycogen depletion are all reduced. Since this occurred before any increase in succinic dehydrogenase, and improvements in $\dot{V}O_2$ kinetics also occurred before other mitochondrial changes (Phillips et al., 1995), it is suggested that the training adaptations for $\dot{V}O_2$ kinetics are linked to increased O_2 availability and tighter metabolic control in the muscle (Cadefau et al., 1994).

If this is the case, then the increased O_2 availability and faster $\dot{V}O_2$ kinetics may be linked to a more rapid increase in total $\dot{V}O_2$ kinetics and blood flow to the working muscles. Evidence for this hypothesis can be found when reviewing the work of Shoemaker et al. (1996). This study has shown that the kinetics of blood flow to the exercising muscle, measured by the mean blood velocity of the femoral artery using non-invasive Doppler techniques, increased after eight to ten days of training. In this study ten days of training equates to approximately twenty hours of vigorous training. Interestingly, although $\dot{V}O_{2max}$ had also significantly improved by this time, the kinetics of \dot{Q} were not affected by training.

The improved blood flow is unlikely to be due to increased capillarisation because although this is a well documented training response (Klausen et al., 1982, Kiens et al., 1993), the time course of the adaptations is too long. Shoemaker et al. (1996) have suggested that the increased blood flow may be due to increased vasodilation, as suggested by the decreased vascular conductance seen in their study. Unfortunately, since blood velocity measurements were not made earlier (i.e. when four days of training had been completed), and even though the same training protocol was used as that adopted by Phillips et al. (1995), it is not possible to decide whether the improvements in $\dot{V}O_2$ kinetics are linked to improvements in blood flow to the muscle.

5.5) Constraints imposed by the study design

In the training study, non-random allocation was used to assign the subjects to either a training or a non-training sub-group. This method of assignment was based on the degree of commitment shown by the subjects for the study. Non-random assignment of the subjects was chosen to increase adherence and reduce dropout. Studies have shown a high incidence of non-compliance of female subjects when participating in exercise programmes (Marcus and Stanton, 1993). The likelihood of female subjects to participate in exercise programmes is lower than for male subjects (Cauley et al., 1991, and Lynch and Main, 1993).

Ideally the subjects should have been assigned to the training and non-training study sub-groups on a random basis. One common way of randomising subjects is to use a random number table (Altman, 1991). Randomisation prevents bias. There is an increased likelihood of bias occurring within the results when subjects are not

randomised. If the design of a study is biased then the validity of the application of the findings to the general population is questionable. During an intervention study randomisation ensures that the treatment of the study sub-groups is not dependent on the characteristics of the subjects. Altman (1991) described a 'volunteer bias' where volunteers had a better prognosis than refusers. In the case of this training study, those subjects who wanted to participate in the training programme would be more likely to comply to the requirements of the study than those who did not want to train. As statistical theory depends on randomisation, the results of the training study may be biased due to the non-randomisation of subjects on the basis of subject preference and therefore the inclusion of a volunteer bias.

The findings of the training study, therefore, should only be assumed to have occurred within that subject population, and it should not be presumed that these effects would occur within other populations of subjects. Since a non-equivalent control group design was not used, changes in $\dot{V}O_2$ kinetics cannot be solely attributed to the effects of the training intervention.

5.6) Implications of the variability of the respiratory responses to the PRBS

exercise test

The variability of the respiratory responses to the PRBS exercise test must be considered in relation to the changes in $\dot{V}O_2$ kinetics that resulted from training. Wide limits of agreement were seen for amplitude and phase shift, with wider limits of agreement occurring at higher harmonic numbers (c.f. Chapter 3).

Atkinson and Nevill (1998) described the difficulty in determining the reliability of measurement tools using limits of agreement, and highlighted the fact that few studies in the area of sports science had employed this method. The greater the variability, i.e. the wider the limits of agreement, the larger the minimal detectable change that would be needed for a given sample size. In the case of the PRBS exercise test, a large minimal detectable change would be needed to ensure that adaptations in $\dot{V}O_2$ kinetics were solely due to the training programme.

Although significant differences in $\dot{V}O_2$ kinetics were detected following the 8 week training programme, the power of the study was at best 60 % (power ranged from 35 to 60 % over harmonic numbers 1 to 4). This was calculated using the nomogram described by Altman (1991) which relates the standardised difference to power or sample size. As no data were available concerning the expected change in $\dot{V}O_2$ kinetics with training, measured using the PRBS exercise test, power calculations could not be used to determine the necessary sample size when designing this training study.

Therefore the standardised difference was calculated as the ratio of the change in $\dot{V}O_2$ kinetics to the standard deviation of this change following completion of the training programme. Using the sample number and the standardised difference, the power of the study was calculated from the nomogram at harmonic numbers 1 to 4 for amplitude and phase shift.

The variability of the respiratory responses to the PRBS exercise test reduced the power of the study. The power of the study could have been improved by increasing the subject sample size. This would have reduced the variability of the respiratory

responses to the PRBS exercise test. Ideally, a study should be designed to have a power of 80 to 90 % (Altman, 1991). To achieve this a total of 90 subjects would need to be studied (45 training subjects and 45 non-training subjects). This would ensure that there was a high chance of detecting a significant training effect.

Alternatively, variability could have been reduced by making the PRBS exercise test more repeatable. Narrower limits of agreement and smaller minimal detectable changes would be expected with a more repeatable test. The minimisation of systematic errors and the possible contribution of inherent biological variability of the respiratory responses to the PRBS exercise test have already been discussed (c.f. Chapter 4).

The large degree of variability associated with the PRBS exercise test means that on an individual basis the minimal detectable changes are large. This negates the use of this test as a means of assessing the effects of training on $\dot{V}O_2$ kinetics of individual subjects. Individual minimal detectable changes were calculated for this using the following equation:

$$\text{Minimal detectable change} = 2 \times \sqrt{2} \times \text{SD (of the replicate tests)} \dots \dots \dots (9)$$

The minimal detectable changes for amplitude and phase shift at harmonic numbers 1 to 4 are presented in tables 5.6 and 5.7 respectively.

Table 5.6) Standard deviation (SD) of the replicate PRBS exercise tests, minimal detectable changes and actual changes for amplitude ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$) at harmonic numbers 1 to 4 following the 8 week training programme.

	Harmonic number			
	1	2	3	4
SD of the replicate tests ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$)	0.445	0.359	1.238	0.756
Minimal detectable change ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$)	1.259	1.015	2.828	2.138
Actual change ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$)	-0.281	0.682	0.519	0.271

Table 5.7) Standard deviation (SD) of the replicate PRBS exercise tests, minimal detectable changes and actual changes for phase shift (degrees) at harmonic numbers 1 to 4 following the 8 week training programme.

	Harmonic number			
	1	2	3	4
SD of the replicate tests (degrees)	4.2	4.1	6.5	8.5
Minimal detectable change (degrees)	11.9	11.6	18.4	24.0
Actual change (degrees)	4	5	8	1

As the minimal detectable changes are greater than the training effects seen it is not

possible to say that any individual had shown a significant training adaptation.

5.7) Conclusions

- Faster $\dot{V}O_2$ kinetics and increased aerobic capacity were apparent following the completion of the eight week endurance-type training programme.
- No significant change in submaximal and maximal parameters were seen over the eight week period for the non-training sub-group.
- As the design of the study involved the non-random allocation of subjects to the two study sub-groups, a degree of bias may have been introduced into the results. This will limit the application of the findings to this population of healthy, young, female subjects.

CHAPTER SIX

FUTURE DIRECTIONS

6.1) Introduction

The studies presented within this thesis have led to a greater knowledge concerning the submaximal nature of the PRBS exercise test technique, and its application to the differentiation of subjects attaining different levels of aerobic fitness and to the detection of short-term training adaptations in young, healthy females. There are other potential applications for the PRBS exercise test technique. The aim of this chapter is to assess the further contribution that this test might make within the area of sports science and within the clinical environment.

6.2) Potential applications of the PRBS exercise test

6.2.1) Assessment of the effects of different training models

As yet the effect of different training models on $\dot{V}O_2$ kinetics, measured in the frequency domain is unknown. Further research in this area could include assessing the effects of different training intensities, training modes, training frequencies and durations on $\dot{V}O_2$ kinetics.

6.2.2) Evaluation of the effect of different athletic status

There is only limited knowledge concerning the effect of athletic status on $\dot{V}O_2$

kinetics. Edwards et al. (in press) used the PRBS exercise test technique to examine the differences between the $\dot{V}O_2$ kinetics of elite endurance male runners and elite male sprinters. Significantly greater amplitude values were observed for the endurance runners, and phase shift components were significantly shorter in the endurance runners compared to the sprinters. The study concluded that $\dot{V}O_2$ kinetics were differentially faster in elite endurance runners than in elite sprinters.

Further research into the use of the PRBS exercise test technique as a test of sports performance could be conducted.

6.2.3) Clinical application

The PRBS exercise test has a potential application within the clinical environment. It is proposed that this test could provide a suitable means of evaluating the effects of cardiac rehabilitation exercise programmes.

The effects of cardiac rehabilitation exercise programmes are not always reviewed. In order to evaluate the physiological benefits induced by such interventions, cardiac patients should be assessed before, during and after participation in a prescribed exercise programme. Currently graded exercise test methodology (Balke and Ware, 1959, Naughton et al., 1963 and Bruce et al., 1973) limits the extent of this evaluation as cardiac patients may be unable to complete these tests safely (Fletcher et al., 1990, and McConnell, 1996).

Improvements in exercise capacity, due to training, are usually determined from $\dot{V}O_{2peak}$ or exercise time measured at the end of a graded exercise test (Kozlowski and Ellestad, 1984). Neither may be reliable parameters of exercise capacity as patient motivation and the subjective nature of the end-points of the test (Hellerstein and Franklin, 1984, and DeBusk et al., 1979) can influence these results. The appropriateness of using heart rate information, obtained during a graded exercise test, has also been questioned by Cooke et al. (1995). It has been shown that a target heart rate prescribed from such a test can result in lower $\dot{V}O_2$ values than expected when performing intermittent circuit-type activities that typically comprise cardiac rehabilitation exercise programmes.

As a consequence of the stressful nature of these graded exercise tests, a number of tests have been devised for the prediction of $\dot{V}O_{2max}$ from parameters measured during submaximal exercise (Astrand and Ryhming, 1954, and Legge and Banister, 1986). The use of heart rate measurements for the prediction of $\dot{V}O_{2max}$ should not be advocated for cardiac patients. Many medications prescribed for the management of cardiovascular disease, for example beta-adrenergic receptor blocking agents, are known to affect the physiological response to exercise (Chick et al., 1988, Gordon et al., 1987, Yeo et al., and Peel and Mossberg, 1995).

Consequently there is a need for a new exercise test technique which will more accurately reflect the type of exercise regimen currently being prescribed to cardiac patients. It is proposed that the PRBS exercise test could provide a suitable means of evaluating the effects of cardiac rehabilitation exercise programmes. This proposal can

be substantiated when both the submaximal nature of the PRBS exercise test and the sensitivity of the test to detect short-term training adaptations are considered.

At present however, the variability of the respiratory responses to the PRBS exercise test limits its application to the assessment of $\dot{V}O_2$ kinetics in groups of patients. This negates the use of this test as a means of evaluating the response of individual patients to a cardiac rehabilitation exercise programme. As the PRBS exercise test can be applied to the measurement of group $\dot{V}O_2$ kinetics, it is proposed that this test could be used to evaluate the effects of different types of cardiac rehabilitation exercise programmes on groups of patients. Alternatively, this test could be used to assess the effects of the same type of cardiac rehabilitation exercise programme on groups of patients with various classifications of cardiac disease, for example following bypass surgery or myocardial infarction.

6.3) Modification of the PRBS exercise test

The following practical issues must be addressed however before the PRBS exercise test can be applied to other populations, particular cardiac patients. Alterations have been made to the PRBS exercise test protocol following consideration of the potentially reduced exercise capacities of cardiac patients.

Firstly, the exercise test duration should be minimised to account for the decreased exercise capacity of cardiac patients. It may be possible to determine $\dot{V}O_2$ kinetics using a PRBS exercise test comprising only two sequences. A warm-up period,

comprising the last 2 min of the pseudo random binary sequence, should be incorporated into the beginning of the exercise test in order to reduce non-linearities and the subsequent introduction of errors into the test results. Following breath-by-breath data collection, the warm-up period should be discarded and the two pseudo random binary sequences analysed using the method described in Chapter 2, Section 2.4.2. A preliminary study involving 12 young, healthy females (age = 22.4 ± 3.3 years, height = 1.63 ± 1.5 m and body mass = 64.8 ± 9.7 kg) showed that there was no significant difference between the $\dot{V}O_2$ kinetics determined from two or three pseudo random binary sequences.

Secondly, the upper work rate should be reduced to reflect the potentially decreased exercise capacities of cardiac patients. Further research will be needed to find a common upper work rate that can be maintained below the V_{ET} . Ventilatory thresholds of 19.9 ± 3.4 ml·kg⁻¹·min⁻¹ and 18.4 ± 4.4 ml·kg⁻¹·min⁻¹ has been described by Hayashida et al. (1993) for male and female patients with left ventricular dysfunction respectively. Using the guidelines produced by the American College of Sports Medicine (1995), an upper work rate of 50 W would result in an $\dot{V}O_2$ of 17.9 to 9.1 ml·kg⁻¹·min⁻¹ for subjects with body masses ranging from 50 to 100 kg. An upper work rate of 50 W performed by cardiac patients should therefore result in submaximal cardiovascular responses. Lavie and Milani. (1994) stated that cardiac patients could have very compromised exercise capacities, therefore caution should be used if this upper work rate was to be performed by cardiac patients with severely compromised exercise capacities.

- Stage 1 80 s at a work rate of 50 W,
- Stage 2 20 s at a work rate of 25 W,
- Stage 3 20 s at a work rate of 50 W,
- Stage 4 20 s at a work rate of 25 W,
- Stage 5 40 s at a work rate of 50 W,
- Stage 6 40 s at a work rate of 25 W,
- Stage 7 20 s at a work rate of 50 W,
- Stage 8 60 s at a work rate of 25 W,

In conclusion, the PRBS exercise test technique produces a multi-frequent assessment of $\dot{V}O_2$ kinetics that results in amplitude and phase shift values at a number of harmonics. These parameters provide valuable information that could not be gained from a mono-frequent assessment of $\dot{V}O_2$ kinetics. The test has potential as a means of assessing cardiorespiratory fitness within the area of sports science and within the clinical environment.

ALTMAN DG (1991). Practical Statistics for Medical Research. Chapman and Hall: London. pp331-333 and pp399-401.

ALLIED DUNBAR NATIONAL FITNESS SURVEY (1992). A report on activity patterns and fitness levels. Commissioned by the Sports Council and Health Education Authority. Main Findings. The Sports Council: London. pp 47-48.

AMERICAN COLLEGE OF SPORTS MEDICINE (ACSM) (1995). The ACSM'S Guidelines for Exercise Testing and Prescription. Lea and Febiger: London. pp156-168.

ARMSTRONG LE AND COSTILL DL (1985). Variability of respiration and metabolism: responses to submaximal cycling and running. Research Quarterly for Exercise and Sport 56; 2: 93-96.

ASTRAND I (1960). Aerobic work capacity in men and women with special reference to age. Acta Physiologica Scandanavica 49: S169.

ASTRAND P-O AND RYHMING I (1954). A normogram for calculation of aerobic capacity (physical fitness) from pulse rate during submaximal work. Journal of Applied Physiology 7:218-222.

ASTRAND P O AND SALTIN B (1961). Maximal oxygen uptake and heart rate in various types of muscular activity. Journal of Applied Physiology 16: 977 – 981.

ATKINSON G (1995). A comparison of statistical methods for assessing measurement repeatability in ergonomics research. In: Sport, Leisure and Ergonomics. Atkinson G and Reilly T (eds.). E and FN Spon: London. pp 218-222.

ATKINSON G AND NEVILL AM (1998). Statistical methods for assessing measurement error (reliability) in variables relevant to sports medicine. Sports Medicine 26; 4: 217-238.

BABCOCK MA, PATERSON DH AND CUNNINGHAM DA (1994a). Effects of aerobic endurance training on gas exchange kinetics of older men. Medicine and Science in Sports and Exercise 26; 4: 447-452.

BABCOCK MA, PATERSON DH, CUNNINGHAM DA AND DICKINSON JR (1994b). Exercise on-transient gas exchange kinetics are slowed as a function of age. Medicine and Science in Sports and Exercise 26; 4: 440-446.

BALKE B AND WARE RW (1959). An experimental study of physical fitness of Air Force personnel. U.S Armed Forces Medical Journal 10: 675-688.

BARSTOW TJ AND MOLE PA (1991). Linear and non-linear characteristics of oxygen uptake kinetics during heavy exercise. Journal of Applied Physiology 71; 6: 2099-2106.

BARSTOW TJ, CASABURI R AND WASSERMAN K (1993). O₂ uptake kinetics and the O₂ deficit as related to exercise intensity and blood lactate. Journal of Applied Physiology

BARSTOW TJ, BUCHTHAL SZ AND COOPER DM (1994). Muscle energetics and pulmonary oxygen uptake kinetics during moderate exercise. *Journal of Applied Physiology* 77: 1742- 1749.

BARSTOW TJ, JONES AM, NGUYEN PH AND CASABURI R (1996). Influence of muscle fibre type and pedal frequency on oxygen uptake kinetics of heavy exercise. *Journal of Applied Physiology* 81; 4: 1642-1650.

BASSETT DR JR. AND HOWLEY ET (1997). Maximal oxygen uptake: “classical” versus “contemporary” viewpoints. *Medicine and Science in Sports and Exercise* 29; 5: 591-603.

BEAVER WL, WASSERMAN K AND WHIPP BJ (1973). On-line computer analysis and breath-by-breath graphical display of exercise function tests. *Journal of Applied Physiology* 34; 1: 128-132.

BEAVER WL, LAMARRA N AND WASSERMAN K (1981). Breath-by-breath measurement of true alveolar gas exchange. *Journal of Applied physiology: Respiration, Environment and Exercise Physiology* 51; 6: 1662-1675.

BEAVER W, WASSERMAN K AND WHIPP BJ (1986). A new method form detecting anaerobic threshold by gas exchange. *Journal of Applied Physiology* 60; 6: 2020-2027.

BERRY M AND MORITANI T (1985). The effects of various training intensities on the kinetics of oxygen consumption. *Sports Medicine and Physical Fitness* 25; 3: 77-83.

BLAND JM (1995). *An Introduction to Medical Statistics*. Oxford Medical Publications: Oxford. pp265-273.

BLAND JM AND ALTMAN DG (1986). Statistical methods for assessing agreement between two methods of clinical measurement. *Lancet*; I: 307-310.

BLAND JM AND ALTMAN DG (1995). Comparing two methods of clinical measurement: a personal history. *International Journal of Epidemiology* 24; 3: S7-S14.

BRITISH ASSOCIATION OF SPORTS SCIENCE (1988). Position statement on the physiological assessment of the elite competitor. White Line Press: Leeds. p 3.1 and p 4.1.

BRUCE RA, KUSUMI F AND HOSMER D (1973). Maximal oxygen intake and nomographic assessment of functional aerobic impairment in cardiovascular disease. *American Heart Journal* 85: 546-562.

CADEFAU J, GREEN HJ, CUSSO R, BALL-BURNETT M AND JAMIESON G (1994). Coupling of muscle phosphorylation potential to glycolysis during work after short term training. *Journal of Applied Physiology* 76: 2586-2593.

CASABURI R, WHIPP BJ, WASSERMAN, BEAVER WL AND KOYAL SN (1977).

Ventilatory and gas exchange dynamics in response to sinusoidal work. *Journal of Applied Physiology: Respiration, Environment and Exercise Physiology* 42; 2: 300-311.

CAULEY JA, DONFIELD SM, LAPORTE RE AND WARHAFTIG NE (1991). Physical activity by socio-economic status in two population based cohorts. *Medicine and Science in Sports and Exercise* 23; 3: 343-351.

CERRETELLI P, SIKAND R AND FARHI L (1966). Readjustment in cardiac output and gas exchange during onset of exercise and recovery. *Journal of Applied Physiology* 21: 1345-1350.

CERRETELLI P, GRASSI B, XI L, SCHENA F, MARCONI C, MEYER M AND FERRETTI G (1995). The role of pulmonary CO₂ flow in the control of the phase I ventilatory response to exercise in humans. *European Journal of Applied Physiology* 71: 287-294.

CHICK TW, HALPERIN AK AND GACEK EM (1988). The effect of antihypertensive medications on exercise performance: a review. *Medicine and Science in Sports and Exercise* 20; 5: 447-454.

CHILIBECK PD, PATERSON DH, PETRELLA RJ AND CUNNINGHAM DA (1996). The influence of age and cardiorespiratory fitness on kinetics of oxygen uptake. *Canadian Journal of Applied Physiology* 21; 3: 185-196.

CHILIBECK PD, PATERSON DH, CUNNINGHAM DA, TAYLOR AW AND NOBLE EG (1997).

Muscle capillarization, O₂ diffusion distance and $\dot{V}O_2$ kinetics in old and young individuals. *Journal of Applied Physiology* 82; 1: 63-69.

CLAXTON DB, CHAPMAN JH, COOKE MA, FYSH ML AND JARVIS DR (1996). Reliability of the pseudo random binary sequence technique to measure oxygen uptake kinetics. In: First Annual Congress, *Frontiers in Sports Science, the European Perspective: Book of Abstracts*. Marconnet P, Gaulard J, Margaritus I and Tessier F (eds.). European College of Sports Science. May 28-31, Nice, France. pp510-511.

COCHRANE JE, HUGHSON RL AND MURPHY PC (1989). On alveolar oxygen uptake kinetics. *In: Respiratory Control: A Modeling Perspective*. Swanson GD, Grodins FS and Hughson RL (eds.) Plenum Press: New York. pp 147-153.

COCHRANE JE AND HUGHSON RL (1992). Computer simulation of O₂ transport and utilisation mechanisms at the onset of exercise. *Journal of Applied Physiology* 73; 6: 2382-2388.

COGGAN AR, ABDULJALIL AM, SWANSON SC, EARLE MS, FARRIS FW, MENDENHALL LA AND ROBITAILLE P-M (1993). Muscle metabolism during exercise in young and older untrained and endurance-trained men. *Journal of Applied Physiology* 75; 5: 2125-2133.

COHEN-SOLAL A, ZANNAD F, KAYANAKIS J-G, GUERRT P, AUPETIT JF AND KOLSKY H

(1991). Multicentre study of the determination of peak oxygen uptake and ventilatory threshold during bicycle exercise in chronic heart failure: comparison of graphical methods, interobserver variability and influence of the exercise protocol. *European Heart Journal* 12: 1055-1063.

CONNETT RJ AND HONIG CR (1989). Regulation of $\dot{V}O_2$ in red muscle: do current biochemical hypotheses fit in vivo data? *American Journal of Physiology* 256 (Regulatory Integrative Comprehensive Physiology 25): R898-R906.

CONVERTINO VA, GOLDWATER DJ AND SANDLER H (1984). Oxygen uptake kinetics of constant-load work : upright vs. supine exercise. *Aviation, Space and Environmental Medicine* 55: 501-505.

COOKE MA, CHAPMAN JH AND FYSH ML (1995). Exercise prescription for cardiac rehabilitation. In: *Sport, Leisure and Ergonomics*. E and FN Spon: London. pp 287-292.

CUNNINGHAM DA, HIMANN JE, PATERSON DH AND DICKINSON JR (1993). Gas exchange dynamics with sinusoidal work in young and elderly women. *Respiratory Physiology* 91: 43-56.

D'ANGELO E AND TORELLI G (1971). Neural stimuli increasing respiration during different types of exercise. *Journal of Applied Physiology* 30; 1: 116-121.

DEBUSK RF, HOUSTON N, HASKELL W, FRY G AND PARKER M (1979). Exercise training soon after myocardial infarction. *The American Journal of Cardiology* 44; 7: 1223-1229.

DE CORT SC, INNES JA, BARSTOW TJ AND GUZ A (1991). Cardiac output, oxygen consumption and arteriovenous oxygen difference following a sudden rise of exercise levels in humans. *Journal of Physiology* 441: 502-512.

DOLL E, KEUL J AND MAIWALD C (1968). Oxygen tension and acid base equilibria in venous blood of working muscle. *American Journal of Physiology* 215: 23-29.

EDWARDS AM, CHALLIS NV, CHAPMAN JH, CLAXTON DB AND FYSH ML (in press). $\dot{V}O_2$ kinetics determined by PRBS techniques differentiate elite endurance runners from elite sprinters. *International Journal of Sports Medicine*.

EIKEN O (1988). Effects of increased muscle perfusion pressure on responses to dynamic leg exercise in man. *European Journal of Applied Physiology and Occupational Physiology* 57: 772-776.

EKBLOM B, ASTRAND P-O, SALTIN B, STENBERG J AND WALLSTROM B (1968). Effect of training on circulatory response to exercise. *Journal of Applied Physiology* 24: 518-528.

ERIKSEN M, WAALER BA, WALLOE L AND WESCHE J (1990). Dynamics and dimensions

of cardiac output changes in humans at the onset and at the end of moderate rhythmic exercise. *Journal of Physiology (London)* 426: 423-437.

EBFELD D, HOFFMANN U AND STEGEMANN J (1987). $\dot{V} O_2$ kinetics in subjects differing in aerobic capacity: investigation by spectral analysis. *European Journal of Applied Physiology* 56: 508-515.

EBFELD D, HOFFMAN U AND STEGEMANN J (1991). A model for studying the distortion of muscle oxygen uptake patterns by circulation parameters. *European Journal of Applied Physiology* 62: 83-90.

FLETCHER GF, FROELICHER VF, HARTLEY H, HASKELL WL AND POLLOCK ML (1990). Exercise standards: a statement for health professionals from the American Heart Association. *Circulation* 82: 2286-2322.

FOX E, BOWERS R AND FOSS M (1993). *The Physiological Basis for Exercise and Sport*. WCB Brown and Benchmark Publishers: Dubuque, USA. pp 641-646.

FROELICHER VF, BRAMMELL H, DAVIS G, NOGUERA I, STEWART A AND LANCASTER MC (1974). A comparison of the reproducibility and physiologic response to three maximal treadmill protocols. *Chest* 65; 5:512-517.

GLESER M, HORSTMAN D AND MELLO R (1974). The effect on VO_{2max} of adding arm work to maximal leg work. *Medicine and Science in Sports and Exercise* 6: 104-107.

GLASSFORD RG, BAYCROFT GHY, SEDGEWICK AW AND MACNAB RBJ (1965).

Comparison of maximal oxygen uptake values determined by predicted and actual methods. *Journal of Applied Physiology* 20; 3: 509-519.

GORDON NF, MYBURGH DP, SCHWELLNUS MP AND VAN RENSBERG JP (1987). Effect of β -blockade on exercise core temperature in coronary artery disease patients. *Medicine and Science in Sports and Exercise* 19; 6: 591-596.

GOVINDASAMY D, PATERSON DH, POULIN MJ AND CUNNINGHAM DA (1992).

Cardiorespiratory adaptation with short term training in older men. *European Journal of Applied Physiology* 65: 203-208.

GRASSI B, POOLE DC, RICHARDSON RS, KNIGHT DR, ERICKSON BK AND WAGNER PD (1996). Muscle O₂ uptake kinetics in humans: implications for metabolic control. *Journal of Applied Physiology* 80; 3: 988-998.

GRECO FC, BAIER H AND SAEZ A (1986). Transient ventilatory and heart rate responses to moderate non-abrupt pseudorandom exercise. *Journal of Applied Physiology* 60; 5: 1524-1534.

GREEN HJ, HELYAR R, BALL-BURNETT M, KOWALCHUCK, N SYMON S AND FARRANCE B (1992). Metabolic adaptations to training precede changes in muscle mitochondrial capacity. *Journal of Applied Physiology* 72: 484-491.

HAGBERG JM, NAGLE FJ AND CARLSON JL (1978). Transient O₂ uptake response at the onset of exercise. *Journal of Applied Physiology: Respiration, Environment and Exercise Physiology* 44: 90-92.

HAGBERG JM, HICKSON RC, EHSANI AA AND HOLLOSZY JO (1980). Faster adjustment to and recovery from submaximal exercise in the trained state. *Journal of Applied Physiology: Respiration, Environment and Exercise Physiology* 48; 2: 218-224.

HAMAR D (1991). Advances in the computerised breath-by-breath method. *In: Advances in Ergometry*. Bachl N, Graham TE and Lollgen H (eds). Springer-Verlag: Berlin.

HAMPTON R L (1965). Experiments using pseudo-random noise. *Simulation* April: 246-254.

HAOUZI P, FUKUBA Y, CASBURI R, STRINGER W AND WASSERMAN K (1993). O₂ uptake kinetics above and below the lactic acidosis threshold during sinusoidal exercise. *Journal of Applied Physiology* 75; 4: 1683-1690.

HAYASHIDA W, KUMADA T, KOHNO F, NODA M, ISHIKAWA N, KAMBAYASHI M AND KAWAI C (1993). Post-exercise oxygen uptake kinetic in patients with left ventricular dysfunction. *International Journal of Cardiology* 38; 1: 63-72.

HELLERSTEIN HK AND FRANKLIN BA (1984). Evaluating the cardiac patient for exercise

therapy. Clinics in Sports Medicine 3; 2: 371-393.

HENRITZE J, WELTMAN A, SCHURRER RL AND BARLOW K (1985). Effects of training at and above the lactate threshold and maximal oxygen uptake. European Journal of Applied Physiology 54: 84-88.

HICKSON RC, BOMZE HA AND HOLLOSZY JO (1978). Faster adjustment of O₂ uptake to the energy requirement of exercise in the trained state. Journal of Applied Physiology: Respiration, Environment and Exercise Physiology 44: 877-881.

HICKSON RC, HAGBERG JM, EHSANI AA AND HOLLOSZY JO (1981). Time course of the adaptive responses of aerobic power and heart rate to training. Medicine and Science in Sports and Exercise 13: 17-20.

HOFFMANN U, EBFELD D, STEGEMANN J AND SCHUTZE H (1991). Comparison of $\dot{V}O_2$ kinetics in upright and supine position. Acta Astronautica 23: 135-137.

HOFFMANN U, EBFELD D, WUNDERLICH H-G AND STEGEMANN J (1992). Dynamic linearity of $\dot{V}O_2$ responses during aerobic exercise. European Journal of Applied Physiology 64: 139-144.

HOFFMANN U, EBFELD D, WUNDERLICH H-G AND STEGEMANN J (1994). Prediction of individual oxygen uptake on-step transients from frequency responses. European Journal of Applied Physiology and Occupational Therapy 69: 93-97.

HOLLOSZY JO AND COYLE EF (1984). Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. *Journal of Applied Physiology*. 56; 4: 831-838.

HONIG CR, CONNETT RJ AND GAYESKI TEJ (1992). O₂ transport and its interaction with metabolism; a systems view of aerobic capacity. *Medicine and Science in Sports and Exercise* 24; 1: 47-53.

HOPKINS SR, MCKENZIE DC, SCHOENE RB, GLENNY RW AND ROBERTSON HT (1994). Pulmonary gas exchange during exercise in athletes. I - Ventilation perfusion mismatch and diffusion limitation. *Journal of Applied Physiology* 77: 912-917.

HUGHSON RL AND MORRISSEY MA (1983). Delayed kinetics of $\dot{V}O_2$ in the transition from prior exercise. Evidence for oxygen transport limitation of $\dot{V}O_2$ kinetics: a review. *International Journal of Sports Medicine* 4: 31-39.

HUGHSON RL AND SYMTHE GA (1983). Slower adaptation to steady state of submaximal exercise with β -blockers. *Journal of Applied Physiology* 52: 107-110.

HUGHSON RL (1984). Alterations in the oxygen deficit-oxygen debt relationships with β -adrenergic receptor blockade in man. *Journal of Physiology (London)* 349: 375-387.

HUGHSON RL AND INMAN MD (1986). Oxygen uptake kinetics from ramp work tests: variability of single test values. *Journal of Applied Physiology* 61; 1: 373-376.

HUGHSON RL AND SWANSON GD (1989). Breath-by-breath gas exchange: data collection and analysis. In: Respiratory Control: A Modeling Perspective. Swanson GD, Grodins FS and Hughson RL (eds.) Plenum Press: New York. pp179-190.

HUGHSON RL, WINTER DA, PATLA AE, COCHRANR JE, CUERVO LA AND SWANSON GD (1989). Kinetics of oxygen uptake studied with two different pseudorandom binary sequences. In: Respiratory Control : A Modeling Perspective. Swanson GD, Grodins FS and Hughson RL (eds.) Plenum Press: New York.

HUGHSON RL (1990). Exploring cardiorespiratory control mechanisms through gas exchange dynamics. *Medicine and Science in Sports and Exercise* 22; 1: 72-79.

HUGHSON RL, XING H, BORKHOFF C AND BUTLER GC (1990a). Cardiorespiratory responses to maximal and submaximal exercise in supine and upright positions. *The Physiologist* 33; 1: S38-S39.

HUGHSON RL, WINTER DA, PATLA AE, SWANSON GD AND CUERVO LA (1990b). Investigation of $\dot{V}O_2$ kinetics in humans with pseudorandom binary sequence work rate change. *Journal of Applied Physiology* 68; 2: 796-801.

HUGHSON RL, XING HC, BUTLER GC AND NORTHEY DR (1990c). Effect of hypoxia on $\dot{V}O_2$ kinetics during pseudorandom binary sequence exercise. *Aviation, Space and Environmental Medicine* 61: 236-239.

HUGHSON RL, XING CH, BORKHOFF C AND BUTLER GC (1991). Kinetic of ventilation and gas exchange during supine and upright cycle exercise. *European Journal of Applied Physiology* 63: 300-307.

HUGHSON RL, COCHRANE JE AND BUTLER GC (1993). Faster O₂ uptake kinetics at onset of supine exercise with than without lower body negative pressure. *Journal of Applied Physiology* 75; 5: 1962-1967.

HUGHSON RL AND KOWALCHUK JM (1995). Kinetics of oxygen uptake for submaximal exercise in hyperoxia, normoxia and hypoxia. *Canadian Journal of Physiology* 20; 2: 198-210.

HUGHSON R L, SHOEMAKER JK, TSCHAKOVSKY ME AND KOWALCHUK JM (1996). Dependence of muscle $\dot{V}O_2$ on blood flow dynamics at onset of forearm exercise. *Journal of Applied Physiology* 81; 4: 1619-1626.

INBAR O, WEINSTEIN Y, KOWALSKI A, EPSTEIN S AND ROTSTEIN A (1993). Effects of increased ventilation and improved pulmonary gas-exchange on maximal oxygen uptake and power output. *Scandinavian Journal of Medicine and Science in Sports* 3: 81-88.

INMAN MD, HUGHSON RL, WEISIGER KH AND SWANSON GD (1987). Estimate of mean tissue O₂ consumption at onset of exercise in males. *Journal of Applied Physiology* 63; 4: 1578-1585.

KATCH VL, SADY SS AND FREEDSON P (1982). Biological variability in maximum aerobic power. *Medicine and Science in Sports and Exercise* 14: 21-25.

KERLIN TW (1974). Mathematical background. In: *Frequency Response Testing in Nuclear Reactors*. Parsegian VL (editor). Academic Press: New York. pp5-51.

KIENS B, ESSEN-GUSTAVSSON B, CHRISTENSEN N AND SALTIN B (1993). Skeletal muscle substrate utilisation during submaximal exercise in man: effect of endurance training. *Journal of Physiology* 469: 459-478.

KLAUSEN K, SECHER N H, CLAUSEN J P, HARTLING O AND TRAP-JENSEN J (1982). Central and regional circulatory adaptations to one-leg training. *Journal of Applied Physiology: Respiration, Environment and Exercise Physiology* 52; 4: 976-983.

KOIKE A, HIROE M, ADACHI H, YAJIMA T, YAMAUCHI Y, NOGAMI A, ITO H, MIYAHARA Y, KOREHAGA M AND MARUMO F (1994). Oxygen uptake kinetics are determined by cardiac function at onset of exercise rather than peak exercise in patients with prior myocardial infarction. *Circulation* 90; 5: 2324-2332.

KOIKE A, HIROE M, YAJIMA T, ADACHI H, SHIMIZU N, KANO H, SUGIMOTO K, MIYAHARA Y, KORENAGA M AND MARUMO F (1995). Effects of nicorandil on kinetics of oxygen uptake at the onset of exercise in patients with coronary artery disease. *American Journal of Cardiology* 76; 7: 449-452.

KOWALCHUK JM AND HUGHSON RL (1990). Effect of β -adrenergic blockade on $\dot{V}O_2$ kinetics during pseudorandom binary sequence exercise. *European Journal of Applied Physiology and Occupational Physiology* 60: 365-369.

KOZLOWSKI YH AND ELLESTAD MH (1984). The exercise test as a guide to management and prognosis. *Clinics in Sports Medicine* 3; 2: 395-416.

KROGH A AND LINHARD (1913). The regulation of respiration and circulation during the initial stages of muscular work. *Journal of Physiology (London)* 47: 112-136.

LAMARRA N, WHIPP BJ, WARD SA AND WASSERMAN K (1987). Effect of interbreath fluctuations on characterizing exercise gas exchange kinetics. *Journal of Applied Physiology* 62; 5: 2003-2012.

LAMARRA N, WARD SA AND WHIPP BJ (1989). A general- purpose model for investigating dynamic cardiopulmonary responses during exercise. In: *Respiratory Control: A Modeling Perspective*. Swanson GD, Grodins FS and Hughson RL (eds.) Plenum Press: New York. pp155-164.

LAVIE CJ AND MILANI RV (1994). Patients with high baseline exercise capacity benefit from cardiac rehabilitation and exercise training programs. *American Heart Journal* 128; 6; 1: 1105-1109.

LEE J (1992). Evaluating agreement between two methods for measuring the same

quantity: a response. *Computing in Biology and Medicine* 22; 5: 369-371.

LEGGE BJ AND BANISTER (1986). The Astrand and Ryhming normogram revisited. *Journal of Applied Physiology* 61; 3: 1203-1209.

LIEHR P, DEDO YL, TORRES S AND MEINIGER JC (1995). Assessing agreement between clinical measurement methods. *Heart and Lung* 24: 240-245.

LINNARSSON D (1974). Dynamics of pulmonary gas exchange and heart rate changes at start and end of exercise. *Acta Physiologica Scandanavica* S415: 1-68.

LOK NS AND LAU CP (1997). Oxygen uptake kinetics and cardiopulmonary performance in lone atrial fibrillation and the effects of sotalol. *Chest* 111; 4: 934-940.

LYNCH WD AND MAIN DS (1993). Frequency of exercise and dropouts in a work-site program: correlates of 6-month activity patterns. *Journal of Occupational Medicine* 35; 11: 1147-1151.

MCCONNELL TR (1996). Exercise prescription: when the guidelines do not work. *Journal of Cardiopulmonary Rehabilitation* 16: 34-37.

MCCORD P AND PATERSON JNP (1989). The effect of low impact dance training on aerobic capacity, submaximal heart rates and lean body composition of college-aged females. *Sports Medicine and Physical Fitness* 29; 2: 184-188.

MCCREARY CR, CHILIBECK PD, MARSH GD, PATERSON DH, CUNNINGHAM DA AND THOMPSON RT (1996). Kinetics of pulmonary oxygen uptake and muscle phosphates during moderate-intensity calf exercise. *Journal of Applied Physiology* 81; 3: 1331-1338.

MCHEMRY PL, ELLESTAD MH, FLETCHER GF, FROELICHER V, HARTLEY H, MITCHELL JH AND SIVARAJAN FROELICHER ES (1990). A position statement for health professionals by the committee on exercise and cardiac rehabilitation of the council on clinical cardiology, American Heart Association. *Circulation* 81; 1: 396-398.

MAHLER M (1985). First-order kinetics of muscle oxygen consumption, and an equivalent proportionality between $\dot{V}O_2$ and phosphorylcreatine level: Implications for the control of respiration. *Journal of General Physiology* 86: 135-165.

MARCUS BH AND STANTON A (1993). Evaluation of relapse prevention and reinforcement interventions to promote exercise adherence in sedentary females. *Research Quarterly for Exercise and Sport* 64; 4: 447-452.

MEYER RA (1988). A linear model of muscle respiration explains monoexponential phosphocreatine changes. *American Journal of Physiology* 254 (Cell Physiology 23): C548-C553).

MORGAN DW, MARTIN PE, KRAHENBUHL GS AND BALDINI FD (1991). Variability in running economy and mechanics among trained male runners. *Medicine and Science in*

Sports and Exercise 23; 3: 378-383.

MURPHY PC, CUERVO LA AND HUGHSON RL (1989). A study of cardiorespiratory dynamics with step and ramp exercise tests in normoxia and hypoxia. Cardiovascular Research 23: 825-832.

NAGLE F, RICHIE J AND GIESE M (1984). $\dot{V}O_{2max}$ responses in separate and combined arm and leg air braked ergometer exercise. Medicine and Science in Sports and Exercise 16: 563-566.

NAUGHTON J, SEVELLUS G AND BALKE B (1963). Physiological responses of normal and pathological subjects to a modified work capacity test. Journal of Sports Medicine 3: 201-206.

NORDREHAUG JE, DANIELSEN R, STANGELAND L, ROSLAND GA AND VIK-MO H (1991). Respiratory gas exchange during treadmill exercise testing: reproducibility and comparison of different exercise protocols. Scandanavian Journal of Clinical and Laboratory Investigation 51: 655-658.

OVEREND TJ, CUNNINGHAM DA, PATERSON DH AND LEFCOE MS (1992). Thigh composition in young and elderly men determined by computer tomography. Clinics in Physiology (Oxford) 12: 629-640.

PATERSON DH AND WHIPP BJ (1991). Asymmetries of oxygen uptake transients at the

on- and offset of heavy exercise in humans. *Journal of Physiology (London)* 443: 575-586.

PEEL C AND MOSSBERG KA (1995). Effects of cardiovascular medications on exercise responses. *Physical Therapy* 75; 5: 387-396.

PHILLIPS SM, GREEN HJ, MACDONALD MJ, AND HUGHSON RL (1995). Progressive effect of endurance training on $\dot{V}O_2$ kinetics at the onset of submaximal exercise. *Journal of Applied Physiology* 79; 6: 1914-1920.

POLLOCK ML, FOSTER C, SCHMIDT D, HELLMAN C, LINNERUD AC AND WARD A (1982). The comparison of physiologic responses to three different maximal graded exercise test protocols in healthy women. *American Heart Journal* 103: 363-373.

POWERS SK, DODD S AND BEADLE RE (1985). Oxygen uptake kinetics in trained athletes differing in $\dot{V}O_{2max}$. *European Journal of Applied Physiology* 54:306-308.

PRICE D, DONNE B AND M O'BRIEN (1996). The effect of variation in seat height on submaximal cycling performance in man. *British Journal of Sports Medicine* 30: 370 [abstract].

RILEY M, PORSZASZ J, STANFORD CF AND NICHOLLS DP (1994). Gas exchange responses to constant work rate exercise in chronic cardiac failure. *British Heart Journal* 72: 150-155.

ROBINSON D M, OGILVIE RW, TULLSON PC AND TERJUNG RL (1994). Increased peak oxygen consumption of trained muscle requires increased electron flux capacity. *Journal of Applied Physiology* 77: 1941-1952.

ROWELL LB (1986). In: *Human Circulation: Regulation During Physical Stress*. New York: Oxford University Press.

ROWLAND LB (1974). Human cardiovascular adjustments to exercise and thermal stress. *Physiology Reviews* 54:75-159.

ROWLAND TW AND CUNNINGHAM LN (1992). Oxygen uptake plateau during maximal treadmill exercise in children. *Chest* 101; 2: 485-489.

SAHLIN K, REN JM AND BROBERG S (1988). Oxygen deficit at the onset of submaximal exercise is not due to delayed oxygen transport. *Acta Physiologica Scandanavica* 134: 175-181.

SALTIN B, NAZAR K, COSTILL DL, STEIN E, JANSSON E, ESSEN B AND GOLLNICK PD (1976). The nature of the training response: peripheral and central adaptations to one-legged exercise. *Acta Physiologica Scandanavica* 96: 289-305.

SALTIN B (1985). Haemodynamic adaptations to exercise. *American Journal of Cardiology* 55: 42D - 47D.

SECHER NH, RUBERG-LARSEN N, BINKHORST RA AND BONDE-PETERSEN F (1974).

Maximum oxygen uptake during arm cranking and combined arm plus leg exercise.

Journal of Applied Physiology 36: 515-518.

SECHER NH, CLAUSEN JP, CLAUSEN K, NOER I AND TRAP-JENSEN J (1977). Central and

regional circulatory effects of adding arm exercise to leg exercise. Acta Physiologica

Scandinavica 100: 288-297.

SHAW DE, JONES HS AND MOSELEY MJ (1994). Analysis of method-comparison data.

Ophthalmology, Physiology and Optics 14: 92-96.

SHEPHARD RJ, KAVANGH T, MERTENS D AND QURESHI I S (1995). Kinetics of the

transplanted heart: implications for the choice of exercise protocol. Journal of

Cardiopulmonary Rehabilitation 15: 288-296.

SHOEMAKER JK, PHILLIPS SM, GREEN HJ AND HUGHSON RL (1996). Faster femoral

artery blood velocity kinetics at the onset of exercise following short term training.

Cardiovascular Research 31: 278-286.

SIETSEMA KE, BEN-DOV I, ZHANG YY, SULLIVAN C AND WASSERMAN K (1994).

Dynamics of oxygen uptake for submaximal exercise and recovery in patients with

chronic heart failure. Chest 105; 6: 1693-1700.

SMITH DP, BYRD RJ AND PURCELL WK (1976). The effect of physical training on the

cardiac output and physical work capacity of young women. *American Corrective Therapy Journal* 30; 4: 115-119.

SPINA RJ, OGAWA T, MARTIN III WH, COGGAN AR, HOLLOSZY JO AND EHSANI AA (1992). Exercise training prevents decline in stroke volume during exercise in young healthy subjects. *Journal of Applied Physiology* 72; 6: 2458-2462.

STAINSBY WN, BRECHUE WF, O'DROBINAK DM AND BARCLAY JK (1989). Oxidation/reduction state of cytochrome oxidase during repetitive contractions. *Journal of Applied Physiology*. 67: 2158 - 2162.

STEGEMANN J, EBFELD D AND HOFFMANN U (1985). Effects of a 7-day head down tilt (-6°) on the dynamics of oxygen uptake and heart rate adjustment in upright position. *Aviation, Space and Environmental Medicine* 56: 410-414.

STEGEMANN J (1991). Ergometry in space and during parabolic flights. In: *Advances in Ergometry*. Bachl N, Graham TE and Lollgen H (eds.) Springer-Verlag: Berlin.

STEGEMANN J (1992). The influence of different space-related physiological variations on exercise capacity determined by oxygen uptake kinetics. *Acta Astronautica* 27: 65-69.

SUNDBERG CJ AND KAISJER L (1992). Effects of graded restriction of perfusion on circulation and metabolism in the working leg; quantification of human ischaemia –

model. Acta Physiologica Scandanavica 146: 1-9.

SUTTON JR (1992A). Limitations of maximal oxygen uptake. Sports Medicine 13; 2: 127-133.

SUTTON JR (1992B). $\dot{V} O_{2\max}$ - new concepts on an old theme. Medicine and Science in Sports and Exercise 24; 1: 26-29.

WAGNER PD (1992). Gas exchange and peripheral diffusion limitation. Medicine and Science in Sports and Exercise 24; 1: 54-58.

WALLOE L AND WESCHE J (1988). Time course and magnitude of blood flow changes in the human quadriceps muscle during and following rhythmic exercise. Journal of Physiology (London) 405: 257-274.

WALSH ML (1992). Possible mechanisms of oxygen uptake kinetics. Annals of Physiology and Anthropometry 11; 3: 215-223.

WARD SA, LAMARRA N AND WHIPP BJ (1996). The control components of oxygen uptake kinetics during high intensity exercise in humans. *In: First Annual Congress, Frontiers in Sports Science, the European Perspective: Book of Abstracts.* Marconnet P, Gaulard J, Margaritus I and Tessier F (eds.) European College of Sports Science. May 28-31, Nice, France. pp268-269.

WASSERMAN K (1982). Dyspnea on exertion: is it the heart of the lungs? *Journal of the American Medical Association* 248; 18: 2039-2043.

WASSERMAN K, HANSEN JE, SUE DY, WHIPP BJ AND CASABURI R (1994). Measurements During Integrative Cardiopulmonary Exercise Testing. *In: Principles of Exercise Testing and Interpretation*. Lea and Febiger: Philadelphia. pp 52-79.

WASSERMAN K (1994). Coupling of external to cellular respiration during exercise: the wisdom of the body revisited. *American Journal of Physiology* 266 (Endocrinology and Metabolism 29): E519-E539.

WELTMAN A AND KATCH V (1976). Min-by-min respiratory exchange and oxygen uptake kinetics during steady-state exercise in subjects of high and low max $\dot{V}O_2$. *The Research Quarterly* 47; 3: 490-198.

WELTMAN A, SEIP RL, WELTMAN JY, HASKVITZ EM, EVANS WS, VELDHUIS JD AND ROGOL AD (1992). Exercise training at and above the lactate threshold in previously untrained women. *International Journal of Sports Medicine* 13; 3: 257-263.

WHIPP BJ AND WASSERMAN K (1972). Oxygen uptake kinetics for various intensities of constant-load work. *Journal of Applied Physiology* 33; 3: 351-356.

WHIPP BJ AND WASSERMAN K (1986). Effect of anaerobiosis on the kinetics of oxygen uptake during exercise. *Federation Proceedings* 45: 2942-2947.

WHIPP BJ, WARD SA, LAMARRA N, DAVIS JA AND WASSERMAN K (1982). Parameters of ventilatory and gas exchange during exercise. *Journal of Applied Physiology: Respiration, Environment and Exercise Physiology* 52; 6: 1506-1513.

WHIPP B J AND WARD S A (1990). Physiological determinants of pulmonary gas exchange kinetics during exercise. *Medicine and Science in Sports and Exercise* 22: 62-71.

WILLIAMSON JW, RAVEN PB AND WHIPP BJ (1996). Unaltered oxygen uptake kinetics at exercise onset with lower-body positive pressure in humans. *Experiments in Physiology* 81; 4: 695-705.

WITTENBERG BA AND WITTENBERG JB (1989). Transport of oxygen in muscle. *Annals of Reviews in Physiology* 51: 857-878.

XING HC, COCHRANE JE, YAMAMOTO Y AND HUGHSON RL (1991). Frequency domain analysis of ventilation and gas exchange kinetics in hypoxic exercise. *Journal of Applied Physiology* 71; 6: 2394-2401.

YEO WW, RAMSAY LE AND JACKSON PR (1991). Anti-anginal agents. *The Pharmaceutical Journal* August 3, 160-162.

YOSHIDA T, UDO M, OHMORI T, MATSUMOTO Y, URAMOTO T AND YAMAMOTO K (1992). Day-to-day changes in oxygen uptake kinetics at the onset of strenuous

endurance training. *European Journal of Applied Physiology* 64: 78-83.

YOSHIDA T, YAMAMOTO K, NAKA T AND UDO M (1993). Cardiac output and oxygen uptake kinetics at the onset and offset of exercise. *Journal of Thermal Biology* 18; 5: 609-615.

YOSHIDA T AND WHIPP BJ (1994). Dynamic asymmetries of cardiac output transients in response to muscular exercise in man. *Journal of Physiology* 480; 2: 355-359.

ZEBALLOS RJ AND WEISMAN IM (1994). Behind the scenes of cardiopulmonary exercise testing. *Clinics in Chest Medicine* 15; 2: 193-213.

ZHANG Y-Y, JOHNSON MC, CHOW N AND WASSERMAN K (1991). The role of fitness on $\dot{V} O_2$ and $\dot{V} CO_2$ kinetics in response to proportional step increases in work rate. *European Journal of Applied Physiology* 63: 94-100.

A.1) Appendix 1 - Publications

A 1.1) The validity of graded exercise testing as a means of prescribing intermittent activity performed during Phase II (early-outpatient) cardiac rehabilitation programmes.

Cooke M.A., Chapman J.H., Fysh M.L. (1995). Journal of Sports Sciences 13; 5: 418
Health Research Institute, Sheffield Hallam University, Pearson Building, 27
Broomgrove Road, Sheffield, S10 2NA, UK

Graded exercise testing (GXT) is commonly used as an indicator for the inclusion of patients in Phase II (early-outpatient) cardiac rehabilitation exercise programmes. This investigation compared the intensity of each activity performed during a circuit prescribed by physiotherapists. The oxygen uptake ($\dot{V}O_2$) and heart rate relationships during continuous and intermittent exercise were also compared to validate the appropriateness of GXT in the prescription of intermittent exercise to cardiac patients.

Four healthy females (mean age = 20.75 years, range 20-22 years, mean height = 166.0 cm, range 160-171 cm, mean body mass = 59.0 kg, range 50-65 kg) performed a Bruce treadmill GXT to 85 % age-predicted maximum heart rate (APMHR), and a circuit consisting of activities of increasing intensities. Heart rate ($\text{beats}\cdot\text{min}^{-1}$) and $\dot{V}O_2$ ($\text{l}\cdot\text{min}^{-1}$) were measured using a miniaturised, radio telemetry, gas analysis system (Cosmed K2, Cosmed, Rome, Italy).

One-way analysis of variance (ANOVA) was used to compare heart rate and $\dot{V}O_2$ for the different activities performed during the circuit. Significant differences were found between activities for heart rate ($P < 0.001$) and $\dot{V}O_2$ ($P < 0.01$) which were significantly

higher during star-jumps. Regression equations of $\dot{V}O_2$ against heart rate, for continuous and intermittent exercise, were calculated for each subject. Heart rates were used to calculate $\dot{V}O_2$ values corresponding to the limits of an effective training zone (70 and 85 % APMHR). The $\dot{V}O_2$ values were found to be significantly higher during continuous treadmill walking than intermittent circuit training at 70 % ($P < 0.01$) and 85 % APMHR ($P < 0.005$), when compared using one-way ANOVAs.

Table showing heart rate and $\dot{V}O_2$ (mean \pm SD) for activities in the circuit.

	Step-ups	Sit/ stand	Cycling	Medicine ball	Star jumps
Heart rate (beats.min⁻¹)	151 \pm 18	143 \pm 6	143 \pm 10	145 \pm 11	175 \pm 8
$\dot{V}O_2$ (l.min⁻¹)	0.91 \pm 0.22	1.08 \pm 0.25	0.93 \pm 0.10	0.76 \pm 0.14	1.33 \pm 0.17

These results have implications for setting target heart rates during cardiac rehabilitation exercise programmes that are essentially intermittent in nature. It is probable that a target heart rate prescribed from GXT will result in a lower $\dot{V}O_2$ value during intermittent, circuit-type activities.

**[Presented at the 3rd International Conference on Sport Leisure and Ergonomics,
Cheshire, UK, 1995.]**

A.1.2) Exercise prescription for the cardiac patient.

Cooke M.A., Chapman J.H., Fysh M.L. (1995). In: Atkinson G and Reilly T (eds). Sport, Leisure and Ergonomics. E and F N Spon: London. pp287-292.

Health Research Institute, Sheffield Hallam University, Pearson Building, 27 Broomgrove Road, Sheffield, S10 2NA, UK

1 Introduction

Exercise prescription based on age-predicted maximum heart rate (APMHR) is both a practical and an effective method of exercise programming (McArdle et al., 1994). To induce cardiovascular benefits, aerobic activity of an intensity of 70 to 85% APMHR is recommended three times per week, for a duration of 20 to 30 min (American College of Sports Medicine, 1991).

The various activities which make up rehabilitation exercise programmes (REP) for cardiac patients are often prescribed on the basis of a circuit (Stewart et al., 1988 and Haennel et al., 1991). Within REP, the resistive exercises suggested by Verrill et al. (1992) can be performed alongside the more dynamic exercises of cycling and stair-climbing recommended by Williams (1994). For the cardiac patient myocardial oxygen demand is an important consideration. It increases during exercise due to an interaction between changes in myocardial contractility, increases in heart rate and changes in arterial blood pressure (McArdle et al., 1994). Those activities with associated high myocardial oxygen demand but low oxygen uptake ($\dot{V}O_2$) need to be carefully monitored (Coplan et al., 1986).

Previous physiological evaluations of REP (Greer et al., 1980), have used bulky gas analysers and electrocardiograph (ECG) systems with restrictive leads which have prevented normal movement patterns. The Cosmed K2 is a miniaturised, telemetric gas

analysis system designed for measuring heart rate and $\dot{V}O_2$ in the field (Concu et al., 1992 and Lucia et al., 1993) and allows unrestricted movement during an exercise programme.

Graded exercise testing (GXT) is commonly used to assess the cardiorespiratory response and exercise capacity of cardiac patients prior to inclusion in Phase II (early-outpatient) REP (Fletcher et al., 1990). Such assessments consist of continuous, multi-stage exercise tests, performed on a treadmill or cycle ergometer which place progressively greater demands on the cardiorespiratory system. The end-point of continuous graded exercise tests is varied and includes muscle fatigue, angina, dyspnea, exercise hypotension, arrhythmias and S-T segment depression (Hellerstein and Franklin, 1984). If clinical symptoms do not appear, the test may be terminated on achieving a heart rate of 85% APMHR.

Graded exercise testing acts as a guide for setting the intensity of exercise for outpatient programmes (Hall, 1993). The validity of using heart rate information obtained from GXT as a tool for prescribing the exercise intensity for REP depends, to some extent, on the relationships between heart rate and $\dot{V}O_2$ during the continuous exercise of GXT and the intermittent activity of REP. By using the Cosmed K2 it is possible to compare the $\dot{V}O_2$ and heart rate relationships during REP with the responses to the GXT.

The aims of this investigation were to use the Cosmed K2 in a pilot study to a) identify activities which might induce a greater myocardial oxygen demand with little concomitant increase in aerobic metabolism and b) assess the appropriateness of using heart rate information obtained from the GXT in the prescription of REP.

2 Methods

2.1 Subjects

Four healthy, non-smoking, female volunteers (mean age = 20.8 years, range 20-22 years) gave verbal informed consent prior to the investigation. Anthropometric data including height (mean height = 1.66 m, range 1.60-1.71 m) and body mass (mean body mass = 59.0 kg, range 50-65 kg) were collected. Age-predicted maximum heart rate was calculated for each of the subjects using the following equation (McArdle et al., 1994):

$$220 - \text{age in years} \quad (1)$$

2.2 Cardiac Rehabilitation Exercise Programme

Activities were chosen which would result in a range of cardiovascular and musculoskeletal demands and were typical of activities found in cardiac rehabilitation programmes. Cycling and step-ups are examples of dynamic activities which are non-weight bearing and weight bearing, respectively. Sit to stand and medicine ball passing involve muscle groups used in everyday activities whilst star jumps are classified as a whole-body activity. Each activity formed part of a circuit (Figure 1) which was continuously monitored. All activities were performed for 2 min, followed by a rest period of 30 s, before progressing to the next station. The range of intensities was achieved either through an increase in the number of repetitions or by an increase in work rate during cycling.

2.3 Graded exercise testing

A graded exercise test was performed on a treadmill (Ergo ES1, Woodway GmbH, Weil am Rhein, Germany) until a test end-point of 85% APMHR had been reached. The Bruce protocol, of an initial grade of 10% and $0.76 \text{ m}\cdot\text{s}^{-1}$ for 3 min, followed by

increments in the grade and/or speed every 3 min, was adopted during the exercise test. The 0% and 5% grade were omitted in accordance with Bruce (1971) as all subjects originated from a healthy population.

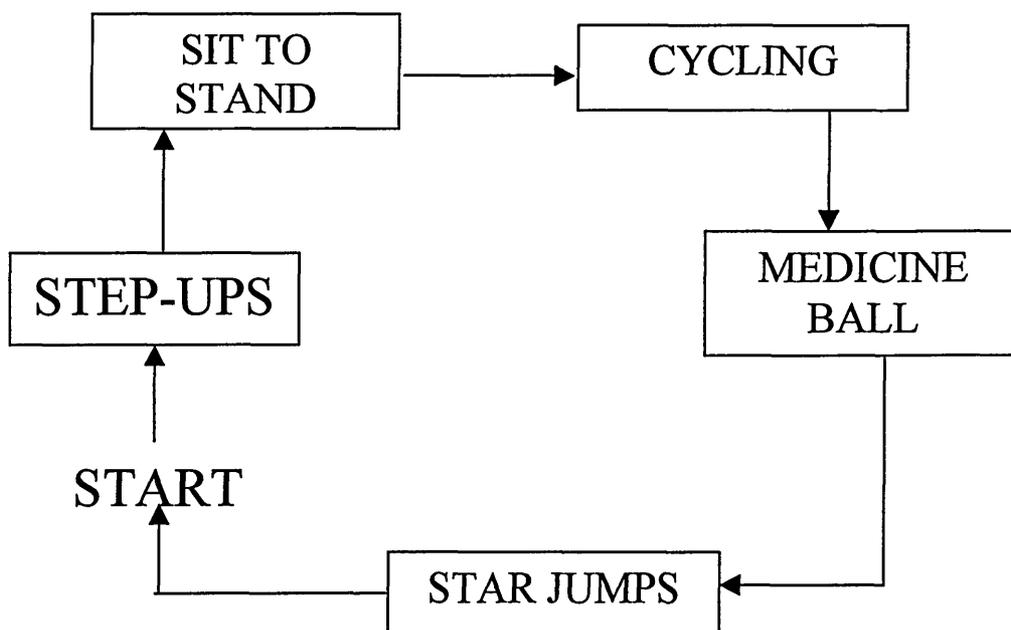


Figure 1. Activities performed during the circuit.

2.4 Metabolic Evaluation

Heart rate ($\text{beats}\cdot\text{min}^{-1}$) and $\dot{V}\text{O}_2$ ($\text{l}\cdot\text{min}^{-1}$) were measured throughout, using the Cosmed K2, (Cosmed, Rome, Italy). The system uses a face mask containing a 28 mm photoelectric gas turbine for volume measurement and an oxygen electrode for the determination of expired oxygen content. Signals were transmitted by radio-telemetry, from a light-weight portable transmitter unit worn around the chest and back.

3 Data analysis

For each activity, the mean heart rate, $\dot{V}\text{O}_2$ and oxygen pulse were calculated. A one-way analysis of variance (ANOVA) was used to evaluate inter-activity differences in

HR, $\dot{V}O_2$ and oxygen pulse values recorded during the circuit. A post hoc Tukey test was then performed to identify differences between activities.

Heart rate and $\dot{V}O_2$ relationships during GXT and REP were expressed as linear regression equations for each subject. Heart rates corresponding to the upper and lower limits of an effective training zone (70 to 85% APMHR) were used to calculate the corresponding values of $\dot{V}O_2$ during these two different types of exercise. A one-way ANOVA compared $\dot{V}O_2$ values for GXT and REP at 70% and 85% APMHR.

4 Results

During REP, significant differences in heart rate, $\dot{V}O_2$ and oxygen pulse were observed between activities (Table 1).

Table 1). Heart rate, oxygen uptake and oxygen pulse (mean \pm standard deviation) during REP

Activity	Heart rate (beats \cdot min $^{-1}$)	Oxygen Uptake (l.min $^{-1}$)	Oxygen pulse (ml.beat $^{-1}$)
Medicine ball	145 \pm 10	0.76 \pm 0.14	5.28 \pm 0.99
Step-ups	151 \pm 18	0.91 \pm 0.22	5.92 \pm 0.69
Cycling	143 \pm 10	0.93 \pm 0.10	6.49 \pm 0.06
Sit to stand	143 \pm 6	1.08 \pm 0.25	7.35 \pm 0.15
Star-jumps	175 \pm 8	1.33 \pm 0.17	7.60 \pm 0.96

Table 2). Oxygen uptake (mean \pm standard deviation) at 70% and 85% APMHR during REP and GXT

	Oxygen uptake at 70% APMHR ($l \cdot \text{min}^{-1}$)	Oxygen uptake at 85% APMHR ($l \cdot \text{min}^{-1}$)
REP	0.79 ± 0.07	1.11 ± 0.08
GXT	0.94 ± 0.09	1.45 ± 0.13

Star-jumps resulted in significantly higher heart rates ($P < 0.001$) than all other activities.

A significantly higher $\dot{V}O_2$ ($P < 0.01$) was only observed when star-jumps were compared with medicine ball passing. Oxygen pulse was found to be significantly higher during sit to stand and star-jumps ($P < 0.01$) when compared to step-ups and medicine ball passing, respectively.

Heart rates ranged from 111 to 179 $\text{beats} \cdot \text{min}^{-1}$ during the GXT and from 129 to 186 $\text{beats} \cdot \text{min}^{-1}$ during REP. Significantly higher values of $\dot{V}O_2$ were found to occur during GXT when compared to REP at 70% ($P < 0.01$) and 85% APMHR ($P < 0.005$) (Table 2).

5 Discussion

According to Williams (1994) even seemingly innocent activities should be carefully considered before they are included in REP. By measuring $\dot{V}O_2$ during an unrestricted REP, more information can be gained about the relative benefits and risks of different components of the programme.

The low $\dot{V}O_2$ and oxygen pulse of medicine ball passing was as expected, due to the small muscle mass involved in this activity (Chapman and Elliott, 1988). Myocardial oxygen demand is further increased in arm exercise by the associated blood pressure rises which are greater than those observed during lower body exercise (Miles et al.,

1989). The risks associated with medicine ball passing need to be balanced with the benefits of arm strengthening as part of the rehabilitation process (Stewart et al., 1988).

The dynamic activity of cycling is generally recommended for cardiac patients (Williams, 1994). The $\dot{V}O_2$ measured during this non-weight bearing activity was not significantly different from any other activity. The highest oxygen pulse was observed during star-jumps which also resulted in the most elevated heart rates. The strenuous nature of this activity needs careful monitoring by the exercise therapist.

The high heart rates which occurred during star-jumps represented 87% APMHR for these subjects. All heart rate and $\dot{V}O_2$ results from REP were compared to the heart rate and $\dot{V}O_2$ relationships from GXT to see if by prescribing exercise at a percentage APMHR, this would result in different oxygen uptakes. Significantly higher $\dot{V}O_2$ values were observed during continuous exercise at 70% and 85% APMHR. These results have implications for prescribing exercise heart rates to patients undergoing cardiac rehabilitation exercise programmes which are essentially intermittent in nature. It is possible that a target heart rate prescribed from a graded exercise test will result in lower $\dot{V}O_2$ values than expected while performing intermittent circuit-type activities.

6 Acknowledgements

Acknowledgement is given to the valuable assistance of Claire Kettlewell and Ceri-Lynne Williams during this investigation.

7 References

American College of Sports Medicine (1991) Guidelines for Exercise Testing and Prescription. Lea and Febiger: Philadelphia, p. 106.

Bruce, R.A. (1971) Exercise testing of patients with coronary artery disease. Annals of

Clinical Research, 3, 323-332.

Chapman, J.H. and Elliott, P.W.R. (1988) Cardiovascular effects of static and dynamic exercise. *European Journal of Applied Physiology*, 58, 152-157.

Coplan, N.L. Gleim, G.W. and Nicholas, J.A. (1986) Principles of exercise prescription for patients with coronary artery disease. *American Heart Journal*, 112, 145-149.

Concu, A. Marcello, C. Rocchitta, A. Ciutu, C. and Esposito, A. (1992) Telemetric measurement of heart-rate-matched oxygen consumption during volleyball games. *Medicine and Science Research*, 20, 243-245.

Fletcher, G.F. Froelicher, V.F. Hartley, L.H. Haskell, W.L. and Pollock, M.L. (1990) Exercise standards: A statement for health professionals from the American Heart Association. *Circulation*, 82, 2286-2321.

Greer, M. Weber, T. Dimick, S. and Ratliff, R. (1980) Physiological responses to low intensity cardiac rehabilitation exercise. *Physical Therapy*, 60, 1147-1151.

Haennel, R.G. Quinney, H.A. and Kappacoda, C.T. (1991) Effects of hydraulic circuit training following coronary artery by-pass surgery. *Medicine and Science in Sports and Exercise*, 23, 158-165.

Hall, L.K. (1993) Exercise prescription for cardiac patients (ed L.K. Hall). *Developing and Managing Cardiac Rehabilitation Programs*. Human Kinetics, Illinois, pp. 73-83.

Hellerstein, H.K. and Franklin, B.A. (1984) Evaluating the cardiac patient for exercise therapy : role of exercise testing. *Clinics in Sports Medicine*, 3, 317-393.

Lucia, A. Fleck, S.J. Gotshall, R.W. and Kearney, J.T. (1993). Validity and reliability of the Cosmed K2 instrument. *International Journal of Sports Medicine*, 14, 380-386.

McArdle, W.D., Katch, F.I. and Katch, V.L. (1994) *Exercise Physiology: Energy Nutrition and Human Performance*. Lea and Febiger, Philadelphia, p. 250 and

- Miles, D.S. Cox, and H. Bomze, J. (1989) Cardiovascular responses to upper body exercise in normals and cardiac patients. *Medicine and Science in Sport and Exercise*, 21, S126-S131.
- Stewart, K. Mason, M. and Kelemen, N. (1988) Three year participation in circuit weight training improves muscular strength and self efficacy in cardiac patients. *Journal of Cardiopulmonary Rehabilitation*, 8, 292-296.
- Verill, D. Shoup, E. McElveen, G. Witt, K. and Berhey, D. (1992) Resistive exercise training in cardiac patients. *Sports Medicine*, 13, 171-193.
- Williams, M. (1994) Exercise Testing and Training in the Elderly Cardiac Patient, *Current Issues in Cardiac Rehabilitation Series*. Human Kinetics, Leeds, p. 31.

A.1.3) Sensitivity of a pseudo random binary sequence exercise test to detect training induced adaptations in young, female subjects.

Fysh M.L*., Chapman J.H.* , Cooke M.A.** , Claxton D.B.* , Jarvis D.R.*** (1996). In: First Annual Congress, Frontiers in Sports Science, the European Perspective: Book of Abstracts. Marconnet P., Gaulard J, Margaritus I and Tessier F (eds). European College of Sports Science. May 28-31, Nice, France. pp592-593.

* School of Science, Sheffield Hallam University, Sheffield, UK,

** Health Research Institute, Sheffield Hallam University, Sheffield, UK,

*** School of Leisure and Food Management, Sheffield Hallam University, Sheffield, UK.

INTRODUCTION

Training adaptations to aerobic exercise programmes are usually identified by increases in maximal oxygen uptake and peak oxygen uptake ($\dot{V}O_{2\text{peak}}$) which are normally assessed using incremental exercise tests to exhaustion. Training adaptations in oxygen uptake ($\dot{V}O_2$) kinetics are less well documented. Babcock (1994) reported a significant decrease in the time constant for $\dot{V}O_2$ kinetics in older men following a 6 month programme of aerobic cycle training.

Oxygen uptake kinetics can be assessed using a number of techniques. One such technique is the pseudo random binary sequence (PRBS) exercise test. The PRBS test has been developed to study the dynamic responses of the cardiorespiratory system to random changes in submaximal work rate (Hughson, 1990a), and may be suitable to assess training adaptations.

The aim of this study was to evaluate the sensitivity of the PRBS test to detect short term training induced adaptations in a young, female population.

METHODS

Twenty-eight female students (age = 22.9 ± 3.1 years, height = 164.2 ± 4.9 cm, weight 63.3 ± 7.6 kg) were assigned to either a training or a control group. The training group (N=15) completed a supervised exercise programme of cycle ergometry. Three exercise sessions a week were performed at an intensity of 85% age-predicted maximum heart rate ($220 - \text{age in years}$), for a duration of 8 weeks. Heart rate telemetry was used to monitor the exercise intensity during all exercise sessions. The control group (N = 13) maintained their normal levels of activity (confirmed by daily activity diaries).

Two exercise tests were undertaken before and after the experimental period using respiratory mass spectrometry and electrocardiography to measure $\dot{V}O_2$ ($\text{ml}\cdot\text{min}^{-1}$) and heart rate ($\text{beats}\cdot\text{min}^{-1}$) respectively.

Test 1 - PRBS exercise test. This test consisted of 4 sequences, each of 450s duration divided into 15 submaximal work rate changes (25 to 80 W) of 30 s intervals. The $\dot{V}O_2$ response, observed during the PRBS exercise test, was related to the original work rate pattern, using a standard Fourier analysis which produced values of phase shift (degrees) and amplitude ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$) for harmonics 1 to 4 (Hughson, 1990b).

Test 2 - incremental exercise test. This test consisted of an initial warm-up of 3 min at 40W followed by ramp increases in work rate of $20 \text{ W}\cdot\text{min}^{-1}$ until exhaustion. Peak oxygen uptake was defined as the highest 30s time-averaged $\dot{V}O_2$ value recorded during the test (Zeballos 1994).

Both tests were completed on an electrically-braked cycle ergometer at a pedal frequency of 60 rev·min⁻¹. One-way analysis of variance (ANOVA) was used to evaluate any adaptations which had occurred during the 8 week training programme.

RESULTS

The 8 week training period resulted in significant increases in $\dot{V}O_{2peak}$ ($P < 0.05$), as assessed by the incremental exercise test (from 2189 ± 170 ml·min⁻¹ to 2385 ± 203 ml·min⁻¹). No significant changes were observed in the control group (2420 ± 265 ml·min⁻¹ to 2405 ± 319 ml·min⁻¹). The effects of the training programme on $\dot{V}O_2$ kinetics, as assessed by the PRBS exercise test, are shown in figure 1.

No significant differences in the $\dot{V}O_2$ kinetics were observed in the control group. Significant decreases ($P < 0.01$) in heart rate during the PRBS test were observed in the training group following the 8 week programme (from 128 ± 13 beats·min⁻¹ to 115 ± 12 beats·min⁻¹). No significant changes were observed in the control group (from 110 ± 12 beats·min⁻¹ to 105 ± 11 beats·min⁻¹).

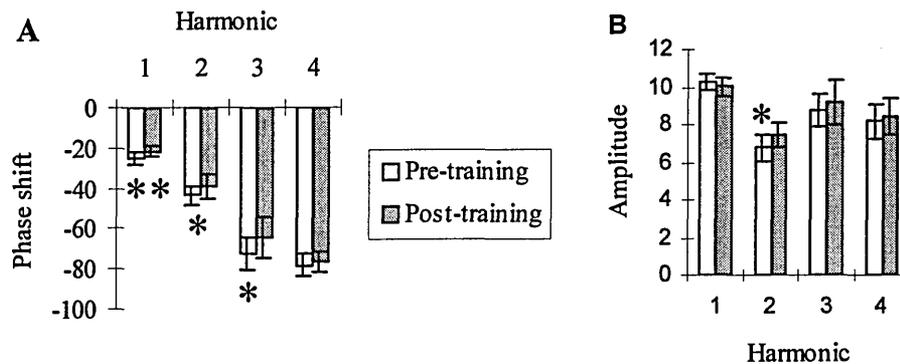


Figure 1: (A) phase shift (degrees) and (B) amplitude (ml·min⁻¹·W⁻¹) before and after the 8 week training programme. * significant at the $P < 0.05$ level and ** significant at the $P < 0.01$ level.

DISCUSSION

This 8 week study brought about expected training adaptations as shown by increases in $\dot{V}O_{2peak}$. Training adaptations were also observed in the $\dot{V}O_2$ kinetics, as measured by the PRBS exercise test. These were more pronounced in phase shift than amplitude. Since phase shift is related to the speed of response, the observed decreases in phase shift indicate faster $\dot{V}O_2$ kinetics following a short programme of cycle ergometry training. These results show that this PRBS exercise test is sensitive to training adaptations, and as such provides a submaximal exercise test which may be suitable for use with various populations.

REFERENCES

- BABCOCK, M. A.; PATERSON, D. H.; CUNNINGHAM, D. A.: Effects of aerobic endurance training on gas exchange kinetics of older men. *Med. Sci. Sports Exerc.* 26: 447-452, 1994.
- HUGHSON, R.L.: Exploring cardiorespiratory control mechanisms through gas exchange dynamics. *Med. Sci. Sports Exerc.* 22: 72-79, 1990a.
- HUGHSON, R.L.; WINTER, D.A.; PATLA, A.E.; SWANSON, G.D.; CUERVO, L.A.: Investigation of $\dot{V}O_2$ kinetics in man with pseudorandom binary sequence work rate changes. *J. Appl. Physiol.* 68: 769-801, 1990b.
- ZEBALLOS, R. J.; WEISMAN, I. M.: Behind the scenes of cardiopulmonary exercise testing. *Clin. Chest Med.* 15: 193-213, 1994.

[Presented at the First Annual Congress of European Colleges of Sport-Science, Nice, France, 28 to 31 May, 1996.]

A.1.4). Reliability of the pseudo random binary sequence technique to measure oxygen uptake kinetics.

Claxton D. B.*, Chapman J. H.*, Cooke M. A.** , Fysh M. L.* , Jarvis D. R. *** (1996).
In: First Annual Congress, Frontiers in Sports Science, the European Perspective: Book of Abstracts. Marconnet P., Gaulard J, Margaritus I and Tessier F (eds). European College of Sports Science. May 28-31, Nice, France. pp510-511.

* School of Science, Sheffield Hallam University, Sheffield, UK,

** Health Research Institute, Sheffield Hallam University, Sheffield, UK,

*** School of Leisure and Food Management, Sheffield Hallam University, Sheffield, UK.

INTRODUCTION

In clinical and exercise physiology the assessment of oxygen uptake ($\dot{V}O_2$) kinetics can provide valuable information about functional capacity. Various methods have been employed in the assessment of $\dot{V}O_2$ kinetics. One such method uses pseudo random binary sequence (PRBS) changes in work rate (Bennett 1981; Hughson 1990). In a review of literature no evidence could be found of any studies that assessed the test / re-test reliability of the PRBS technique. The aim of this experiment was therefore to assess the test / re-test reliability of a PRBS test protocol by examining intra-subject variability in $\dot{V}O_2$ kinetics.

METHODOLOGY

A test / re-test study was carried out using two identical PRBS protocols. Twenty healthy male subjects, average age 26.21 ± 3.98 years (mean \pm standard deviation (SD)),

participated in the study. Each subject performed the tests on an electrically braked cycle ergometer. Respiratory gas exchange was measured on a breath-by-breath basis using a respiratory mass spectrometer. The test protocol incorporated a series of six identical PRBS sequences. Each sequence consisted of 63 units of 5 s duration, giving a total period of 315 s. During each sequence the work rate was automatically alternated between 25W and 105W. Throughout the tests, subjects maintained a pedalling rate of 60 rpm. No information was given on impending changes in workload.

Following each test, breath-by-breath data was subjected to standard Fourier transform.

The transform yielded the parameters of $\dot{V}O_2$ kinetics, i.e. phase shift and amplitude, for a range of harmonics. Only harmonics with a period over 100 s were deemed suitable for assessment (Hoffman 1994).

Assessment of intra-subject variability in the parameters was made using the analysis of variance (ANOVA) technique. Limits of agreement (Altman 1991) were calculated in order to assess how well the results obtained in the two tests agreed. Statistical significance for all analyses was set at $P < 0.05$.

RESULTS

All subjects completed two identical PRBS exercise tests. The mean heart rate recorded during the tests was 102 ± 11 beats.min⁻¹. Mean values for the parameters of $\dot{V}O_2$ kinetics are shown in table 1.

One-way analysis of variance revealed no significant intra-subject variability in any of the parameters as measured during the two tests. Wide 95% limits of agreement were, however, observed at each harmonic for both parameters (table 2).

Table 1: Mean values for the parameters obtained for the relationship between oxygen uptake and work rate: mean values (\pm SD)

Harmonic	1		2		3	
Period (s)	315		157.5		105	
Test number	Test 1	Test 2	Test 1	Test 2	Test 1	Test 2
Mean phase shift (degrees)	29.6 (\pm 4.85)	30.3 (\pm 5.73)	43.3 (\pm 4.28)	42.35 (\pm 4.00)	63.75 (\pm 7.83)	62.2 (\pm 7.66)
Mean amplitude (ml.min ⁻¹ .W ⁻¹)	10.68 (\pm 0.94)	10.47 (\pm 0.89)	8.06 (\pm 1.04)	7.83 (\pm 0.96)	6.24 (\pm 0.91)	5.97 (\pm 0.84)

Table 2: 95% Limits of agreement - Test 2 minus Test 1: mean difference (\pm 2SD)

Harmonic	1	2	3
Phase shift: mean difference (degrees)	+0.7 (\pm 7.37)	-0.95 (\pm 9.59)	-1.55 (\pm 11.32)
95% limits of agreement (degrees)	-6.67 to +8.07	-10.54 to +8.64	-12.87 to +9.77
Amplitude: mean difference (ml.min ⁻¹ .W ⁻¹)	-0.21 (\pm 1.42)	-0.23 (\pm 0.88)	-0.27 (\pm 0.98)
95% limits of agreement (ml.min ⁻¹ .W ⁻¹)	-1.63 to +1.21	-1.99 to +1.53	-1.25 to +0.71

DISCUSSION

The aim of this study was to investigate the test / re-test reliability of a specific PRBS protocol. The results generated followed a similar pattern to those obtained by Hughson (1990) in an identical single PRBS test.

Statistical analysis revealed no significant difference between the test and re-test data.

Although this suggests that the PRBS protocol is reliable for group data, the wide limits

of agreements still need to be considered. One possible explanation for these limits is inherent biological variability. This factor is exhibited by all complex biological systems, and can affect even highly regimented exercise protocols. It may, however, be possible to reduce some of this variability by applying appropriate smoothing techniques to the data, for example auto- and / or cross-correlations (Hoffmann 1994).

REFERENCES

- ALTMAN, D.G: Practical statistics for medical research. Chapman and Hall, 1991.
- BENNETT, F.M.; REISCHL, P.; GRODINS, F.S.; YAMASHIRO, S.M.; FORDYCE, W.E.: Dynamics of ventilatory response to exercise in humans. Journal of Applied Physiology 51: 194-203, 1981.
- HOFFMAN, U.; EBFELD, D.; WUNDERLICH, H.G; STEGEMANN, J.: VO₂ kinetics determined by PRBS-technique and sinusoidal testing. Zeitschrift für Kardiologie 83: 57-60 1994.
- HUGHSON, R.L.; WINTER, D.A.; PATLA, A.E.; COCHRANE, J.E.; CUERVO, L.A.; SWANSON, G.D.: Kinetics of oxygen uptake studied with two different pseudorandom binary sequences. In: Respiratory control a modelling perspective. 179-191, 1990.

[Presented at the First Annual Congress of European Colleges of Sport-Science, Nice, France, 28 to 31 May, 1996.]

A.2) Appendix 2 - Environmental conditions

Table showing the environmental conditions (mean \pm SD) during the evaluation of the variability of the PRBS exercise test with 30 s work rate changes between 25 and 75 W.

Test number	Temperature ($^{\circ}\text{C}$)	Barometric pressure (mmHg)
1	21.3 ± 0.8	753.4 ± 4.5
2	21.5 ± 0.4	757.8 ± 8.1

Table showing the environmental conditions (mean \pm SD) during the evaluation of the effect of endurance-type training on the $\dot{V}\text{O}_2$ kinetics of healthy, young females.

Week	Temperature ($^{\circ}\text{C}$)	Barometric pressure (mmHg)
0	20.2 ± 1.4	746.7 ± 13.2
4	20.5 ± 1.1	742.5 ± 5.4
8	21.1 ± 1.4	741.1 ± 7.5

A.3) Appendix 3 - Recruitment material

A.3.1) Health and activity questionnaire (variability)

An evaluation of the variability of a pseudo random binary sequence exercise test

Purpose of this investigation.

The pseudo random binary sequence (PRBS) exercise test has been used to assess the dynamic responses of the cardiorespiratory system to exercise. A preliminary investigation where the PRBS exercise test was applied to a healthy, female population showed the test to be submaximal and could therefore be applied to a general population. Parameters (oxygen uptake kinetics) measured during this test were found to relate to maximal oxygen uptake or cardiorespiratory fitness.

Further research is now needed to assess the variability of this PRBS exercise test when performed by a population of young female volunteers.

In order to assess the suitability of each volunteer for this investigation, you are asked to complete this health and exercise questionnaire and return it to the investigator.

~~~~~

###### *1). Personal details.* (Please print in black or blue ink).

Surname \_\_\_\_\_ First name \_\_\_\_\_

Date of Birth \_\_\_\_\_ Age \_\_\_\_\_

Current address \_\_\_\_\_  
\_\_\_\_\_

Postcode \_\_\_\_\_ Telephone number \_\_\_\_\_

Course Title \_\_\_\_\_

2). **Medical details.**

i). Have you been told that you have any of the following health problems ?

|                            | YES                      | NO                       |
|----------------------------|--------------------------|--------------------------|
| a) asthma                  | <input type="checkbox"/> | <input type="checkbox"/> |
| b) high blood pressure     | <input type="checkbox"/> | <input type="checkbox"/> |
| c) diabetes                | <input type="checkbox"/> | <input type="checkbox"/> |
| d) anaemia                 | <input type="checkbox"/> | <input type="checkbox"/> |
| e) cardiovascular diseases | <input type="checkbox"/> | <input type="checkbox"/> |

ii). Have you had any of the following in the last 6 months ?

|                                               | YES                      | NO                       |
|-----------------------------------------------|--------------------------|--------------------------|
| a) viral or bacterial infections              | <input type="checkbox"/> | <input type="checkbox"/> |
| b) musculoskeletal injuries                   | <input type="checkbox"/> | <input type="checkbox"/> |
| c) operations involving a general anaesthetic | <input type="checkbox"/> | <input type="checkbox"/> |

iii). Are you taking any medications prescribed by your G.P. ?

| YES                      | NO                       |
|--------------------------|--------------------------|
| <input type="checkbox"/> | <input type="checkbox"/> |

If yes, please give details. \_\_\_\_\_

\_\_\_\_\_

iv). Do you have any other disabilities that may impair your exercise performance in these tests ? If yes, please give details. \_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

3). *Exercise participation.*

i). How many days per week do you exercise ?

- a) none
- b) 1 to 2 days
- c) 3 to 4 days
- d) 5 to 6 days
- e) everyday

ii). How long do you exercise for during each session ?

- a) up to 20 minutes
- b) 20 to 40 minutes
- c) 40 to 50 minutes
- d) up to 60 minutes
- e) longer than 60 minutes

iii). Please indicate which of the following activities you participate in regularly.

- a) aerobics
- b) archery
- c) badminton
- d) canoeing
- e) circuit training
- f) cycling
- g) dancing
- h) fencing

- i) football
- j) hiking
- k) horseback riding
- l) martial arts
- m) netball
- n) running
- o) skiing
- p) squash
- q) swimming
- r) tennis
- s) volleyball

t) other (please specify) \_\_\_\_\_

Additional comments (if applicable) \_\_\_\_\_

---

---

---

---

### *A.3.2) Consent form (variability)*

#### **An evaluation of the variability of a pseudo random binary sequence exercise test**

##### ***Purpose of this investigation.***

The pseudo random binary sequence (PRBS) exercise test has been used to assess the dynamic responses of the cardiorespiratory system to exercise. A preliminary investigation where the PRBS exercise test was applied to a healthy, female population showed the test to be submaximal and could therefore be applied to a general population. Parameters (oxygen uptake kinetics) measured during this test were found to relate to maximal oxygen uptake or cardiorespiratory fitness.

Further research is now needed to assess the reproducibility and repeatability of this PRBS exercise test when performed by a population of young female volunteers.

##### ***Experimental requirements***

All participants are required to complete repeated PRBS exercise tests

##### ***Special instructions.***

Participants are asked to:

- abstain from consuming large quantities of alcohol, caffeinated products such as tea, coffee and chocolate prior to the study . These factors may cause the heart rate to be elevated and an alteration of your cardiorespiratory response to the above exercise tests.
- have consumed a light meal at least 2 hours before the start of the tests.
- wear suitable clothing (training shoes, shorts/tracksuit and T-shirt) during **all** exercise sessions.

***Expected benefits from these exercise tests.***

The exercise test provide an assessment of your response to submaximal exercise, scientifically. Records are strictly confidential.

Questions concerning any of the procedures involved in these sessions are encouraged.

If you have any queries, please ask.

***Freedom of consent.***

Your permission to perform the previously described exercise programme and exercise tests is required. Your participation in this experiment is strictly voluntary. You are free to deny consent and withdraw from this investigation at any time.

Name of participant \_\_\_\_\_(please print)

Signature of participant \_\_\_\_\_ Date \_\_\_\_\_

Signature of witness \_\_\_\_\_ Date \_\_\_\_\_

**An evaluation of the effect of endurance-type training on the  $\dot{V}O_2$  kinetics of young, healthy females**

***Purpose of this investigation.***

The pseudo random binary sequence (PRBS) exercise test has been used to assess the dynamic responses of the cardiorespiratory system to exercise. A preliminary investigation where the PRBS exercise test was applied to a healthy, female population showed the test to be submaximal and could therefore be applied to a general population. Parameters (oxygen uptake kinetics) measured during this test were found to relate to maximal oxygen uptake or cardiorespiratory fitness.

Further research is now needed to assess the effect of endurance-type training on the  $\dot{V}O_2$  kinetics of young, healthy females.

In order to assess the suitability of each volunteer for this investigation, you are asked to complete this health and exercise questionnaire and return it to the investigator.

~~~~~  
1). *Personal details.*

(Please print in black or blue ink).

Surname _____ First name _____

Date of Birth _____ Age _____

Current address _____

Postcode _____ Telephone number _____

Course Title _____

2). *Medical details.*

i). Have you been told that you have any of the following health problems ?

	YES	NO
a) asthma	<input type="checkbox"/>	<input type="checkbox"/>
b) high blood pressure	<input type="checkbox"/>	<input type="checkbox"/>
c) diabetes	<input type="checkbox"/>	<input type="checkbox"/>
d) anaemia	<input type="checkbox"/>	<input type="checkbox"/>
e) cardiovascular diseases	<input type="checkbox"/>	<input type="checkbox"/>

ii). Have you had any of the following in the last 6 months ?

	YES	NO
a) viral or bacterial infections	<input type="checkbox"/>	<input type="checkbox"/>
b) musculoskeletal injuries	<input type="checkbox"/>	<input type="checkbox"/>
c) operations involving a general anaesthetic	<input type="checkbox"/>	<input type="checkbox"/>

iii). Are you taking any medications prescribed by your G.P. ?

YES	NO
<input type="checkbox"/>	<input type="checkbox"/>

If yes, please give details. _____

iv). Do you have any other disabilities that may impair your exercise performance in these tests ? If yes, please give details. _____

3). **Exercise participation.**

i). How many days per week do you exercise ?

- a) none
- b) 1 to 2 days
- c) 3 to 4 days
- d) 5 to 6 days
- e) everyday

ii). How long do you exercise for during each session ?

- a) up to 20 minutes
- b) 20 to 40 minutes
- c) 40 to 50 minutes
- d) up to 60 minutes
- e) longer than 60 minutes

iii). Please indicate which of the following activities you participate in regularly.

- a) aerobics
- b) archery
- c) badminton
- d) canoeing
- e) circuit training
- f) cycling
- g) dancing
- h) fencing

- i) football
- j) hiking
- k) horseback riding
- l) martial arts
- m) netball
- n) running
- o) skiing
- p) squash
- q) swimming
- r) tennis
- s) volleyball

t) other (please specify) _____

Additional comments (if applicable) _____

An evaluation of the effect of endurance-type training on the $\dot{V}O_2$ kinetics of young, healthy females

Purpose of this investigation.

The pseudo random binary sequence (PRBS) exercise test has been used to assess the dynamic responses of the cardiorespiratory system to exercise. A preliminary investigation where the PRBS exercise test was applied to a healthy, female population showed the test to be submaximal and could therefore be applied to a general population. Parameters (oxygen uptake kinetics) measured during this test were found to relate to maximal oxygen uptake or cardiorespiratory fitness.

Further research is now needed to assess the effect of endurance-type training on the $\dot{V}O_2$ kinetics of young, healthy females.

Experimental requirements

All participants are required to complete:

- an 8-week training programme consisting of cycling against a resistance to produce heart rates equivalent to 90% maximal heart rate. Three exercise test sessions will be held per week each of a duration of 40 minutes during which heart rate will be monitored using heart rate telemetry.
- a total of 6 exercise tests consisting of progressive exercise tests to exhaustion and submaximal PRBS exercise test during the study period.

Special instructions.

Participants are asked to:

- abstain from consuming large quantities of alcohol, caffeinated products such as tea, coffee and chocolate prior to the study . These factors may cause the heart rate to be elevated and an alteration of your cardiorespiratory response to the above exercise tests.
- have consumed a light meal at least 2 hours before the start of the tests.
- wear suitable clothing (training shoes, shorts/tracksuit and T-shirt) during **all** exercise sessions.

Discomforts.

Some of the tests will involve exercise to exhaustion. You may experience some discomfort during exercise. If you are unable to complete the session the test will be terminated at your request, but you will be encouraged to continue.

Expected benefits from training.

Benefits such as increase maximal oxygen uptake (fitness), decreased heart rate during any submaximal activity and increased psychological well being are known to occur as a result of training.

The exercise test provide an assessment of your maximal oxygen uptake (fitness), and your response to maximal and submaximal exercise, scientifically. Records are strictly confidential.

Questions concerning any of the procedures involved in these sessions are encouraged.

If you have any queries, please ask.

Freedom of consent.

Your permission to perform the previously described exercise programme and exercise

tests is required. Your participation in this experiment is strictly voluntary. You are free to deny consent and withdraw from this investigation at any time.

Name of participant _____ (please print)

Signature of participant _____ Date _____

Signature of witness _____ Date _____

An evaluation of the effect of endurance-type training on the $\dot{V}O_2$ kinetics of young, healthy females

Purpose of this investigation.

The pseudo random binary sequence (PRBS) exercise test has been used to assess the dynamic responses of the cardiorespiratory system to exercise. A preliminary investigation where the PRBS exercise test was applied to a healthy, female population showed the test to be submaximal and could therefore be applied to a general population. Parameters (oxygen uptake kinetics) measured during this test were found to relate to maximal oxygen uptake or cardiorespiratory fitness.

Further research is now needed to assess the effect of endurance-type training on the $\dot{V}O_2$ kinetics of young, healthy females.

Experimental requirements

All participants are required to complete:

- a total of 6 exercise tests consisting of progressive exercise tests to exhaustion and submaximal PRBS exercise test during the study period.
- daily activity diaries to ensure normal activity and lifestyle patterns are maintained.

Special instructions.

Participants are asked to:

- abstain from consuming large quantities of alcohol, caffeinated products such as tea, coffee and chocolate prior to the study . These factors may cause the heart rate

to be elevated and an alteration of your cardiorespiratory response to the above exercise tests.

- have consumed a light meal at least 2 hours before the start of the tests.
- wear suitable clothing (training shoes, shorts/tracksuit and T-shirt) during **all** exercise sessions.

Discomforts.

Some of the tests will involve exercise to exhaustion. You may experience some discomfort during exercise. If you are unable to complete the session the test will be terminated at your request, but you will be encouraged to continue.

Expected benefits from testing.

The exercise test provide an assessment of your maximal oxygen uptake (fitness), and your response to maximal and submaximal exercise, scientifically. Records are strictly confidential.

Questions concerning any of the procedures involved in these sessions are encouraged.

If you have any queries, please ask.

Freedom of consent.

Your permission to perform the previously described exercise tests is required. Your participation in this experiment is strictly voluntary. You are free to deny consent and withdraw from this investigation at any time.

Name of participant _____(please print)

Signature of participant _____

Date _____

Signature of witness _____

Date _____

A.3.6) A weekly activity log as completed by each subject in the non-training sub-group.

Name: _____

Subject Number: _____

Week number _____

Date from _____ to _____

Activity	Number of times in last week	Average time	Were you out of breath of sweaty?
Aerobics			
Badminton			
Basketball			
Canoeing			
Circuit training			
Cycling			
Dancing			
Football			
Golf			
Hiking			
Keep fit			
Martial arts			
Netball			
Running			
Squash			
Swimming			
Tennis			
Volleyball			
Other please indicate			

A.4) Appendix 4 -Criteria fulfilled during the progressive exercise test to exhaustion.

A.4.1) The criteria fulfilled by the total subject cohort (n = 28) during the progressive exercise test to exhaustion when investigating the relationship between peak oxygen uptake and oxygen uptake kinetics.

Subject n = 28	Plateau of the relationship between VO₂ and work rate	RER ≥ 1.15	Heart rate within 10₁ beats·min⁻¹ APMHR	Total number criteria fulfilled
1	x	✓	✓	2
2	x	✓	✓	2
3	✓	✓	✓	3
4	x	✓	✓	1
5	✓	✓	x	2
6	x	✓	x	1
7	✓	✓	✓	3
8	x	✓	✓	2
9	✓	✓	x	2
10	✓	✓	✓	3
11	x	✓	x	1
12	x	✓	x	1
13	x	✓	x	1
14	x	✓	x	1
15	x	✓	x	1
16	x	✓	✓	2
17	x	✓	✓	2
18	x	✓	x	1
19	x	✓	x	1
20	x	✓	✓	2
21	x	✓	x	1
22	x	✓	x	1
23	x	✓	x	1
24	x	✓	x	1
25	x	✓	✓	2
26	x	✓	x	1
27	x	✓	x	1
28	x	✓	x	1

A.4.2) Criteria fulfilled by training subjects during the progressive exercise test to exhaustion, when investigating the effect of endurance-type training on the oxygen uptake kinetics of healthy, young females.

Subject	Week 0				Week 4				Week 8			
	Plateau of VO ₂ / work rate	RER ≥ 1.15	Heart rate within 10 ₁ beats·min ⁻¹ APMHR	Total number criteria met	Plateau of VO ₂ / work rate	RER ≥ 1.15	Heart rate within 10 ₁ beats·min ⁻¹ APMHR	Total number criteria met	Plateau of VO ₂ / work rate	RER ≥ 1.15	Heart rate within 10 ₁ beats·min ⁻¹ APMHR	Total number criteria met
1	X	✓	✓	2	X	✓	X	1	X	✓	X	1
2	X	✓	✓	2	X	✓	✓	2	X	✓	✓	2
3	✓	✓	✓	3	X	✓	X	1	X	✓	X	1
4	X	✓	✓	1	X	✓	✓	2	X	✓	✓	2
5	✓	✓	X	2	X	✓	X	1	X	✓	✓	2
6	X	✓	X	1	X	✓	X	1	X	✓	✓	2
7	✓	✓	✓	3	✓	✓	✓	3	✓	✓	✓	3
8	X	✓	✓	2	X	✓	X	1	X	✓	X	1
9	✓	✓	X	2	X	✓	X	1	X	✓	X	1
10	✓	✓	✓	3	X	✓	✓	2	✓	✓	✓	3
11	X	✓	X	1	X	✓	X	1	X	✓	X	1
12	X	✓	X	1	X	✓	✓	2	X	✓	X	1
13	X	✓	X	1	X	✓	X	1	X	✓	X	1
14	X	✓	X	1	✓	✓	X	2	✓	✓	X	2
15	X	✓	X	1	X	✓	X	1	X	✓	X	1

A.4.3) Criteria fulfilled by the non-training subjects during the progressive exercise test to exhaustion, when investigating the effect of endurance-type training on the oxygen uptake kinetics of healthy, young females.

Subject	Week 0				Week 4				Week 8			
	Plateau of VO_2 / work rate	RER ≥ 1.15	Heart rate within 10_1 beats·min ⁻¹ APMHR	Total number criteria met	Plateau of VO_2 / work rate	RER ≥ 1.15	Heart rate within 10_1 beats·min ⁻¹ APMHR	Total number criteria met	Plateau of VO_2 / work rate	RER ≥ 1.15	Heart rate within 10_1 beats·min ⁻¹ APMHR	Total number criteria met
1	x	✓	✓	2	x	✓	✓	2	x	✓	x	1
2	x	✓	✓	2	x	✓	x	1	x	✓	x	1
3	x	✓	x	1	x	✓	x	1	x	✓	x	1
4	x	✓	x	1	✓	✓	x	2	x	✓	x	1
5	x	✓	✓	2	x	✓	x	1	✓	✓	x	2
6	x	✓	x	1	x	✓	x	1	x	✓	x	1
7	x	✓	x	1	x	✓	x	1	x	✓	x	1
8	x	✓	x	1	x	✓	✓	2	x	✓	✓	2
9	x	✓	x	1	x	✓	x	1	✓	✓	x	2
10	x	✓	✓	2	x	✓	✓	2	x	✓	✓	2
11	x	✓	x	1	x	✓	x	1	x	✓	x	1
12	x	✓	x	1	x	✓	x	1	x	✓	x	1
13	x	✓	x	1	✓	✓	x	2	✓	✓	x	2

A.5) Appendix 5 - Individual short-term training adaptations.

Figures showing the individual changes, in $\dot{V}O_2$ kinetics and relative $\dot{V}O_{2peak}$ due to the 8 week training period.

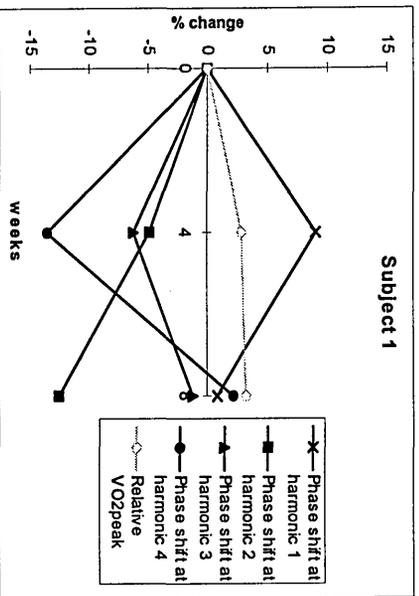
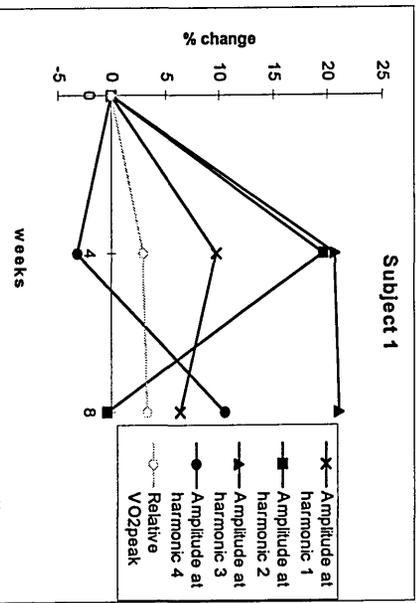


Table showing individual adaptations, in terms of change and % change, resulting from the 8 week training period.

	0 to 4 weeks		4 to 8 weeks		0 to 8 weeks	
	Change	% change	Change	% change	Change	% change
Relative $\dot{V}O_{2peak}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	1.87	2.9	-0.15	0.3	1.17	3.2
Amplitude at harmonic 1 ($ml \cdot min^{-1} \cdot W^{-1}$)	0.96	9.7	-0.33	-3.0	0.63	6.7
Amplitude at harmonic 2 ($ml \cdot min^{-1} \cdot W^{-1}$)	1.36	19.6	-1.38	-16.7	-0.3	2.9
Amplitude at harmonic 3 ($ml \cdot min^{-1} \cdot W^{-1}$)	1.64	20.6	0.03	0.3	1.67	20.9
Amplitude at harmonic 4 ($ml \cdot min^{-1} \cdot W^{-1}$)	-0.24	-3.3	1.00	14.2	0.76	10.9
Phase shift at harmonic 1 (degrees)	1.09	9.1	-2.00	-8.3	0	0.8
Phase shift at harmonic 2 (degrees)	0.95	-4.9	-3.00	-7.7	-5.00	-12.6
Phase shift at harmonic 3 (degrees)	0.93	-6.3	3.00	5.0	-1.00	-1.3
Phase shift at harmonic 4 (degrees)	0.86	-13.5	10.00	15.6	0	2.1

Figures showing the individual changes, in $\dot{V}O_2$ kinetics and relative $\dot{V}O_{2peak}$ due to the 8 week training period.

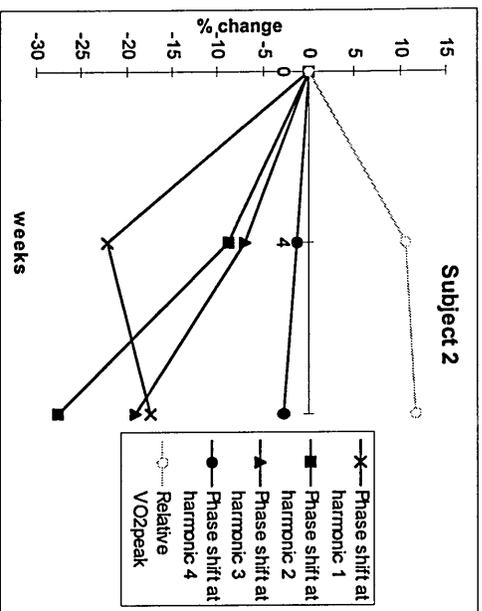
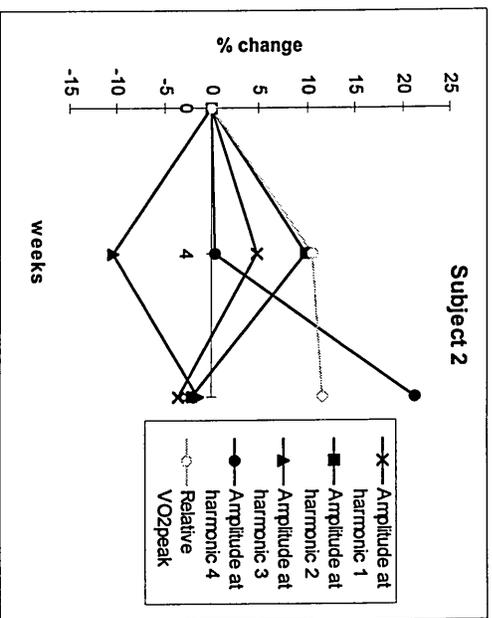


Table showing individual adaptations, in terms of change and % change, resulting from the 8 week training period.

	0 to 4 weeks		4 to 8 weeks		0 to 8 weeks	
	Change	% change	Change	% change	Change	% change
Relative $\dot{V}O_{2peak}$ (ml·kg ⁻¹ ·min ⁻¹)	3.36	10.6	0.52	0.9	4.38	11.7
Amplitude at harmonic 1 (ml·min ⁻¹ ·W ⁻¹)	0.46	4.7	-0.84	-8.2	-0.38	-3.5
Amplitude at harmonic 2 (ml·min ⁻¹ ·W ⁻¹)	0.68	9.8	-0.90	-11.9	-0.22	-2.1
Amplitude at harmonic 3 (ml·min ⁻¹ ·W ⁻¹)	-0.96	-10.5	0.74	9.0	-0.22	-1.5
Amplitude at harmonic 4 (ml·min ⁻¹ ·W ⁻¹)	0.02	0.3	1.62	21.0	1.64	21.3
Phase shift at harmonic 1 (degrees)	0.78	-22.2	1.00	4.8	-5.00	-17.4
Phase shift at harmonic 2 (degrees)	0.91	-8.7	-8.00	-19.0	-12.00	-27.7
Phase shift at harmonic 3 (degrees)	0.93	-7.0	-8.00	-12.1	-13.00	-19.1
Phase shift at harmonic 4 (degrees)	0.99	-1.3	-1.00	-1.4	-2.00	-2.7

Figures showing the individual changes, in $\dot{V}O_2$ kinetics and relative $\dot{V}O_{2peak}$ due to the 8 week training period.

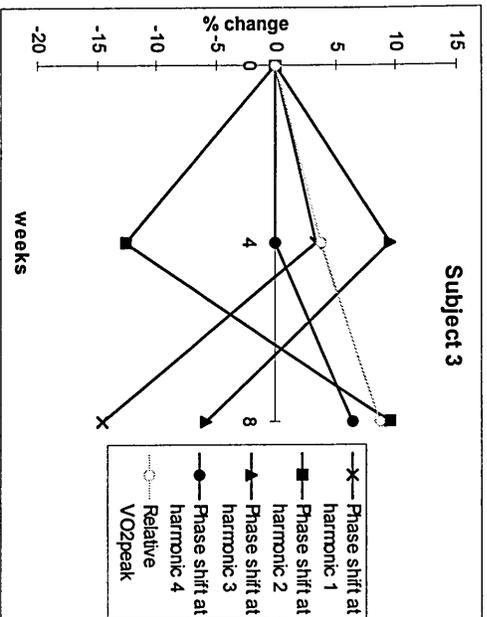
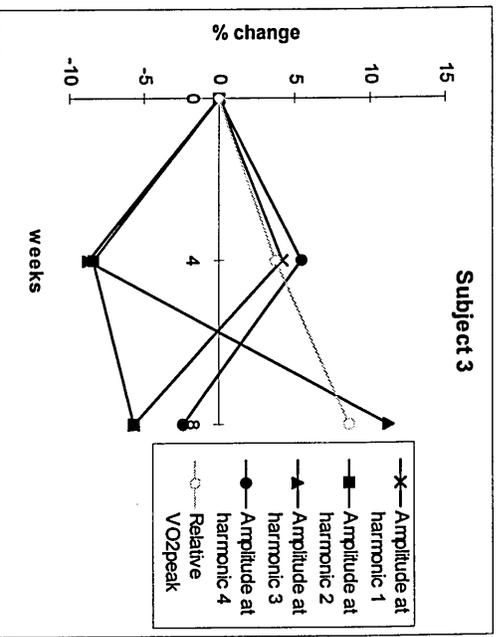


Table showing individual adaptations, in terms of change and % change, resulting from the 8 week training period.

	0 to 4 weeks		4 to 8 weeks		0 to 8 weeks	
	Change	% change	Change	% change	Change	% change
Relative $\dot{V}O_{2peak}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	1.2	3.8	2.17	5.0	3.37	8.6
Amplitude at harmonic 1 ($ml \cdot min^{-1} \cdot W^{-1}$)	-0.92	-8.4	0.27	2.7	-0.65	-5.7
Amplitude at harmonic 2 ($ml \cdot min^{-1} \cdot W^{-1}$)	-0.70	-8.8	0.22	3.1	-0.47	-5.7
Amplitude at harmonic 3 ($ml \cdot min^{-1} \cdot W^{-1}$)	0.49	5.4	0.54	5.8	1.03	11.2
Amplitude at harmonic 4 ($ml \cdot min^{-1} \cdot W^{-1}$)	0.28	3.4	-0.50	-5.8	-0.22	-2.4
Phase shift at harmonic 1 (degrees)	0.88	-12.5	1.00	4.8	-2.00	-14.5
Phase shift at harmonic 2 (degrees)	1.10	9.5	0	0	4.00	9.5
Phase shift at harmonic 3 (degrees)	1.00	0	-4.00	-5.8	-4.00	-5.8
Phase shift at harmonic 4 (degrees)	1.03	2.6	3.00	3.8	5.00	6.4

Figures showing the individual changes, in $\dot{V}O_2$ kinetics and relative $\dot{V}O_{2peak}$ due to the 8 week training period.

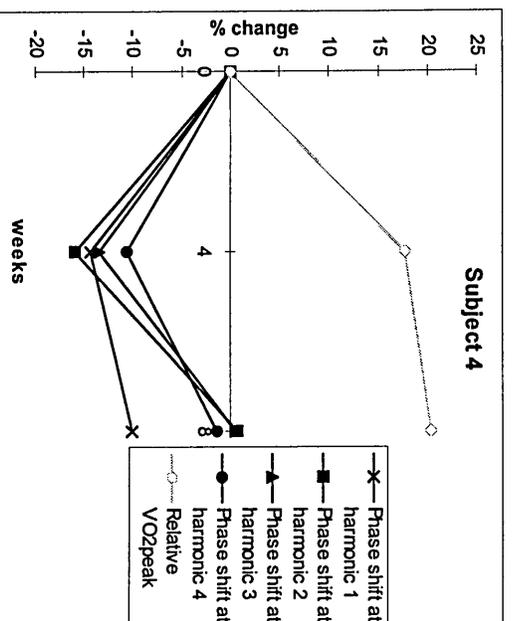
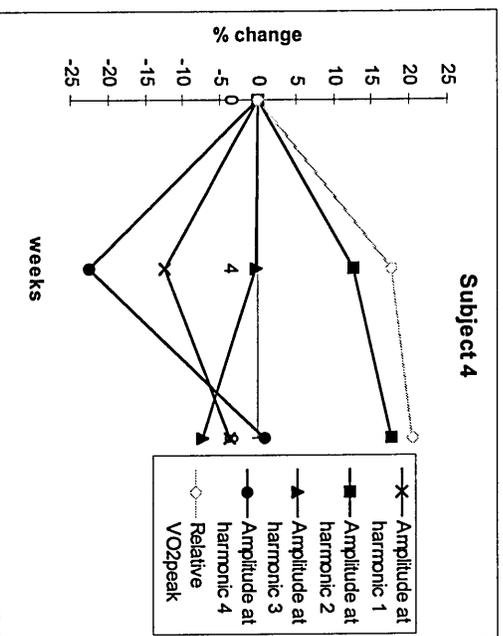


Table showing individual adaptations, in terms of change and % change, resulting from the 8 week training period.

	0 to 4 weeks		4 to 8 weeks		0 to 8 weeks	
	Change	% change	Change	% change	Change	% change
Relative $\dot{V}O_{2peak}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	4.00	17.3	3.63	3.0	7.63	20.3
Amplitude at harmonic 1 ($ml \cdot min^{-1} \cdot W^{-1}$)	-1.31	-12.4	0.87	9.4	-0.44	-3.6
Amplitude at harmonic 2 ($ml \cdot min^{-1} \cdot W^{-1}$)	0.76	12.6	0.34	5.1	1.10	17.7
Amplitude at harmonic 3 ($ml \cdot min^{-1} \cdot W^{-1}$)	-0.03	-0.3	-0.61	-7.0	-0.64	-7.3
Amplitude at harmonic 4 ($ml \cdot min^{-1} \cdot W^{-1}$)	-2.10	-22.5	1.70	23.4	-0.41	0.9
Phase shift at harmonic 1 (degrees)	0.86	-14.3	1.00	4.2	-3.00	-10.1
Phase shift at harmonic 2 (degrees)	0.84	-16.0	7.00	16.7	-1.00	0.7
Phase shift at harmonic 3 (degrees)	0.86	-13.5	9.00	14.1	-1.00	0.6
Phase shift at harmonic 4 (degrees)	0.89	-10.6	7.00	9.2	-2.00	-1.4

Figures showing the individual changes, in $\dot{V}O_2$ kinetics and relative $\dot{V}O_{2peak}$, due to the 8 week training period.

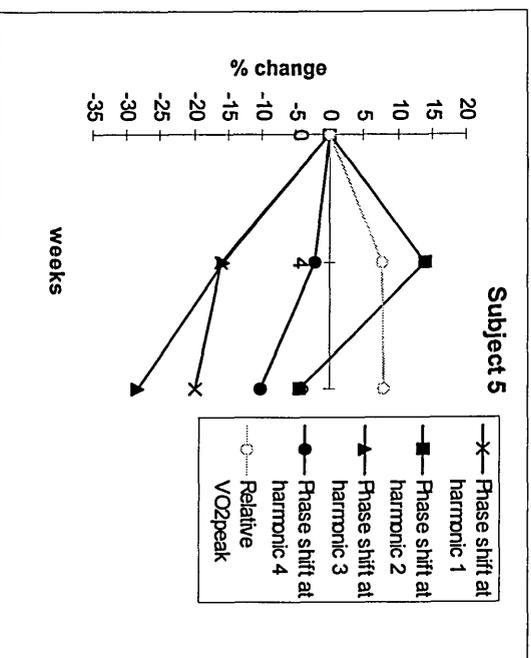
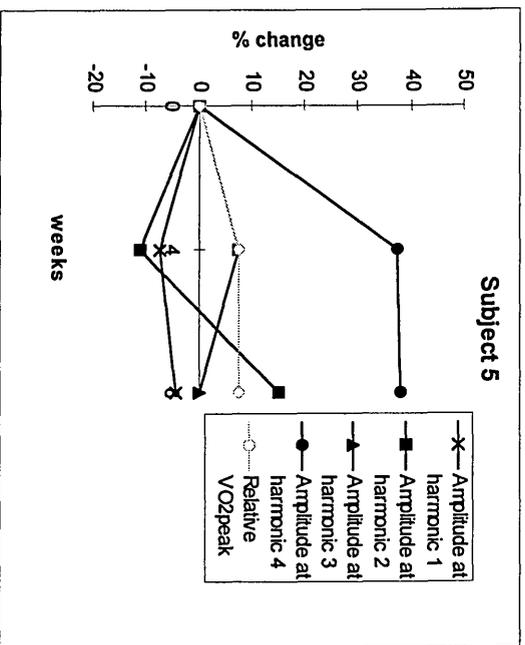


Table showing individual adaptations, in terms of change and % change, resulting from the 8 week training period.

	0 to 4 weeks		4 to 8 weeks		0 to 8 weeks	
	Change	% change	Change	% change	Change	% change
Relative $\dot{V}O_{2peak}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	1.93	7.7	0.22	0.1	2.15	7.8
Amplitude at harmonic 1 ($ml \cdot min^{-1} \cdot W^{-1}$)	-0.76	-7.4	0.30	3.2	-0.45	-4.4
Amplitude at harmonic 2 ($ml \cdot min^{-1} \cdot W^{-1}$)	-0.68	-11.1	1.43	26.3	0.75	12.3
Amplitude at harmonic 3 ($ml \cdot min^{-1} \cdot W^{-1}$)	0.60	7.5	-0.63	-7.4	-0.04	-0.4
Amplitude at harmonic 4 ($ml \cdot min^{-1} \cdot W^{-1}$)	2.24	37.5	0.04	0.5	2.29	38.2
Phase shift at harmonic 1 (degrees)	0.84	-16.1	-1.00	-3.8	-6.00	-19.4
Phase shift at harmonic 2 (degrees)	1.14	14.0	-9.00	-18.4	-3.00	-7.0
Phase shift at harmonic 3 (degrees)	0.84	-15.8	-10.00	-12.5	-25.00	-26.3
Phase shift at harmonic 4 (degrees)	0.98	-2.2	-7.00	-8.0	-9.00	-10.1

Figures showing the individual changes, in $\dot{V}O_2$ kinetics and relative $\dot{V}O_{2peak}$ due to the 8 week training period.

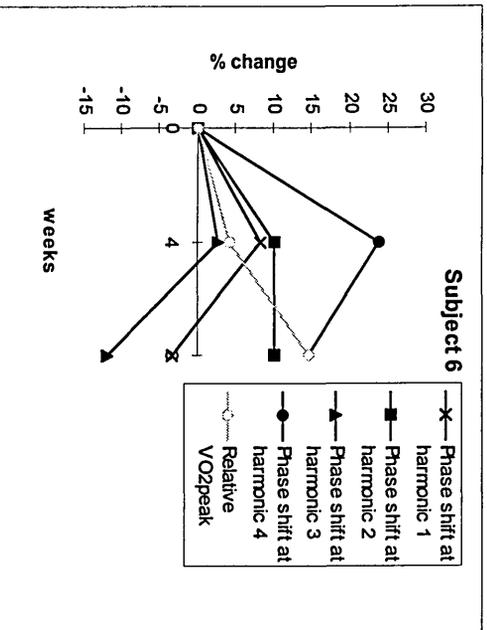
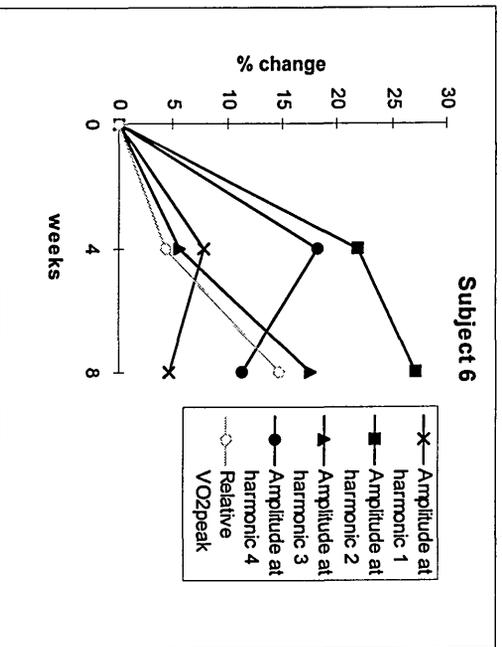


Table showing individual adaptations, in terms of change and % change, resulting from the 8 week training period.

	0 to 4 weeks		4 to 8 weeks		0 to 8 weeks	
	Change	% change	Change	% change	Change	% change
Relative $\dot{V}O_{2peak}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	1.56	4.3	3.83	9.9	5.39	14.7
Amplitude at harmonic 1 ($ml \cdot min^{-1} \cdot W^{-1}$)	0.78	7.8	-0.35	-3.2	0.44	4.6
Amplitude at harmonic 2 ($ml \cdot min^{-1} \cdot W^{-1}$)	1.25	21.9	0.37	5.3	1.61	27.2
Amplitude at harmonic 3 ($ml \cdot min^{-1} \cdot W^{-1}$)	0.44	5.6	0.95	11.4	1.40	17.6
Amplitude at harmonic 4 ($ml \cdot min^{-1} \cdot W^{-1}$)	1.45	18.2	-0.64	-6.8	0.81	11.4
Phase shift at harmonic 1 (degrees)	1.08	8.3	-3.00	-11.5	-1.00	-3.2
Phase shift at harmonic 2 (degrees)	1.10	10.3	0	0	4.00	10.3
Phase shift at harmonic 3 (degrees)	1.03	2.7	-11.0	-14.5	-9.00	-11.8
Phase shift at harmonic 4 (degrees)	1.24	23.9	-8.00	-9.1	9.00	14.8

Figures showing the individual changes, in $\dot{V}O_2$ kinetics and relative $\dot{V}O_{2peak}$, due to the 8 week training period.

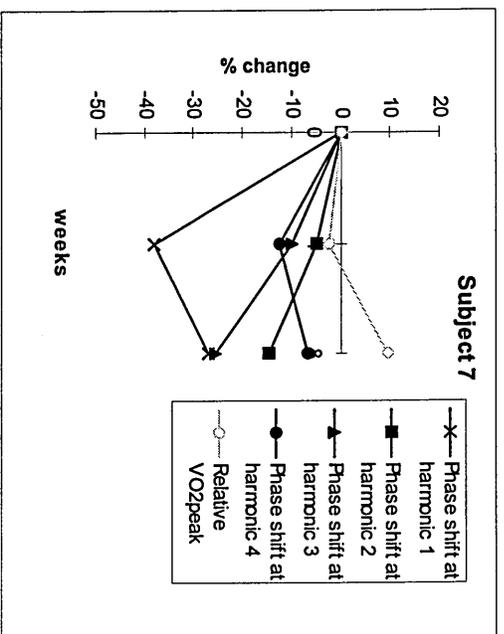
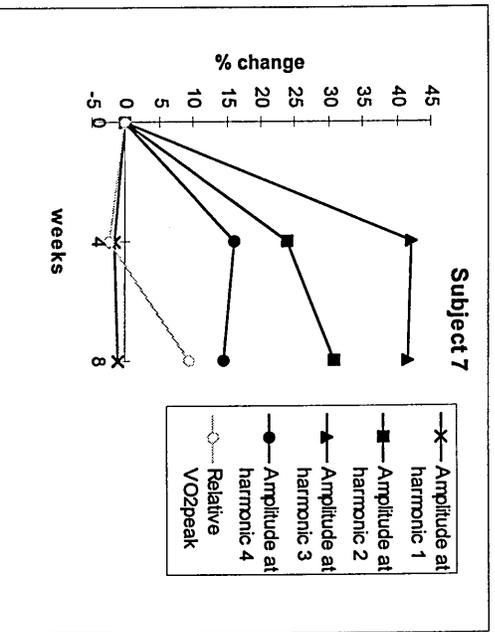


Table showing individual adaptations, in terms of change and % change, resulting from the 8 week training period.

	0 to 4 weeks		4 to 8 weeks		0 to 8 weeks	
	Change	% change	Change	% change	Change	% change
Relative $\dot{V}O_{2peak}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	2.31	-3.4	1.02	12.9	3.34	9.5
Amplitude at harmonic 1 ($ml \cdot min^{-1} \cdot W^{-1}$)	-0.18	-1.7	0.07	0.7	-0.11	-1.0
Amplitude at harmonic 2 ($ml \cdot min^{-1} \cdot W^{-1}$)	1.56	24.0	0.55	6.9	2.11	30.9
Amplitude at harmonic 3 ($ml \cdot min^{-1} \cdot W^{-1}$)	3.19	42.0	-0.05	-0.4	3.14	41.6
Amplitude at harmonic 4 ($ml \cdot min^{-1} \cdot W^{-1}$)	1.43	16.1	-0.14	-1.4	1.29	14.7
Phase shift at harmonic 1 (degrees)	0.62	-37.9	2.00	11.1	-9.00	-26.8
Phase shift at harmonic 2 (degrees)	0.95	-4.8	-4.00	-10.0	-6.00	-14.8
Phase shift at harmonic 3 (degrees)	0.90	-10.1	-11.0	-15.5	-19.00	-25.6
Phase shift at harmonic 4 (degrees)	0.88	-12.5	4.00	5.7	-6.00	-6.8

Figures showing the individual changes, in $\dot{V}O_2$ kinetics and relative $\dot{V}O_{2peak}$ s due to the 8 week training period.

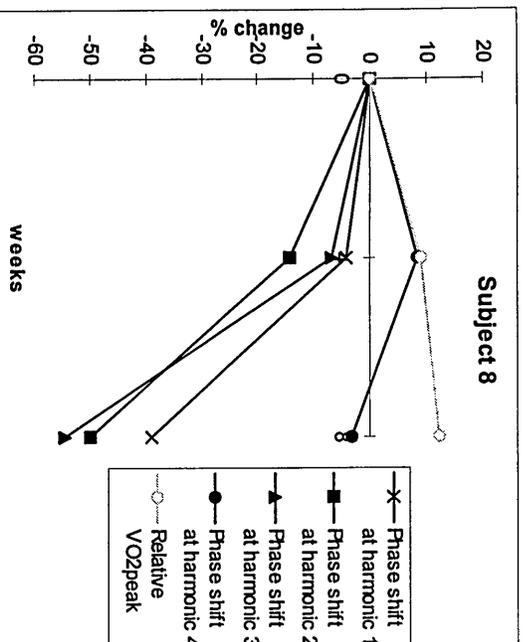
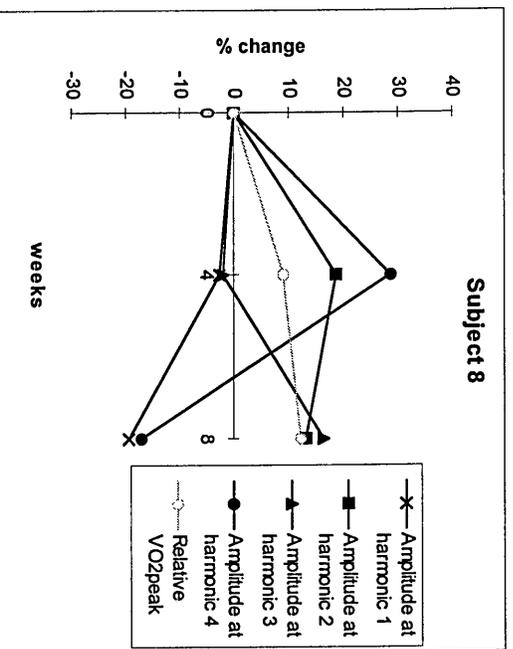


Table showing individual adaptations, in terms of change and % change, resulting from the 8 week training period.

	0 to 4 weeks		4 to 8 weeks		0 to 8 weeks	
	Change	% change	Change	% change	Change	% change
Relative $\dot{V}O_{2peak}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	2.86	9.0	1.02	2.9	3.74	12.2
Amplitude at harmonic 1 ($ml \cdot min^{-1} \cdot W^{-1}$)	-0.29	-2.6	-1.76	-16.6	-2.04	-19.2
Amplitude at harmonic 2 ($ml \cdot min^{-1} \cdot W^{-1}$)	1.25	18.6	-0.44	-5.5	0.82	13.1
Amplitude at harmonic 3 ($ml \cdot min^{-1} \cdot W^{-1}$)	-0.20	-1.8	1.89	18.3	1.70	16.5
Amplitude at harmonic 4 ($ml \cdot min^{-1} \cdot W^{-1}$)	2.49	28.9	-5.11	-45.9	-2.62	-17.0
Phase shift at harmonic 1 (degrees)	0.96	-4.2	-8.00	-34.8	-9.00	-39.0
Phase shift at harmonic 2 (degrees)	0.86	-14.3	-15.00	-35.7	-22.00	-50.0
Phase shift at harmonic 3 (degrees)	0.93	-6.9	-32.00	-47.7	-37.00	-54.6
Phase shift at harmonic 4 (degrees)	1.08	8.3	-9.00	-11.5	-3.00	-3.2

Figures showing the individual changes, in $\dot{V}O_2$ kinetics and relative $\dot{V}O_{2peak}$, due to the 8 week training period.

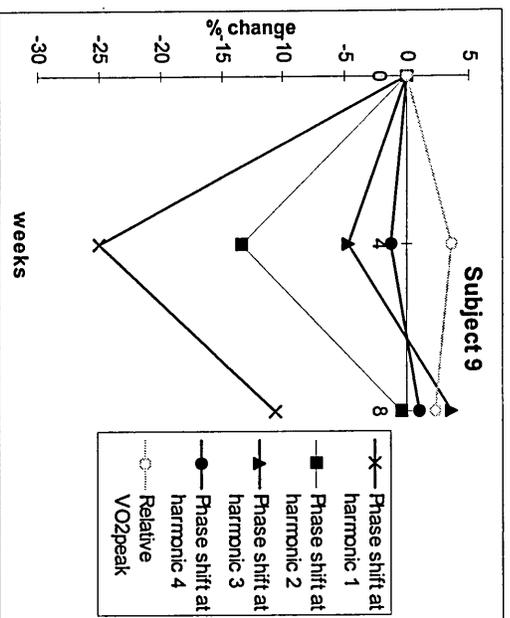
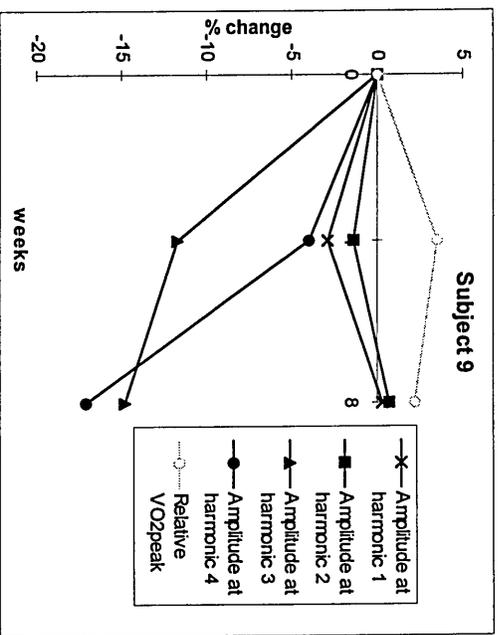


Table showing individual adaptations, in terms of change and % change, resulting from the 8 week training period.

	0 to 4 weeks		4 to 8 weeks		0 to 8 weeks	
	Change	% change	Change	% change	Change	% change
Relative $\dot{V}O_{2peak}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	1.24	3.5	-0.25	-1.3	0.99	2.2
Amplitude at harmonic 1 ($ml \cdot min^{-1} \cdot W^{-1}$)	-0.30	-2.9	-0.32	3.2	-0.62	0.3
Amplitude at harmonic 2 ($ml \cdot min^{-1} \cdot W^{-1}$)	-0.10	-1.4	0.15	2.1	0.05	0.7
Amplitude at harmonic 3 ($ml \cdot min^{-1} \cdot W^{-1}$)	-1.11	-11.7	-0.26	-3.1	-1.37	-14.8
Amplitude at harmonic 4 ($ml \cdot min^{-1} \cdot W^{-1}$)	-0.35	-4.0	-1.08	-13.1	-1.43	-17.1
Phase shift at harmonic 1 (degrees)	0.75	-25.0	3.00	14.3	-4.00	-10.7
Phase shift at harmonic 2 (degrees)	0.87	-13.3	5.00	12.8	-1.00	-0.5
Phase shift at harmonic 3 (degrees)	0.95	-4.7	5.00	8.2	2.00	3.5
Phase shift at harmonic 4 (degrees)	0.99	-1.3	1.00	1.3	0	1.0

Figures showing the individual changes, in $\dot{V}O_2$ kinetics and relative $\dot{V}O_{2peak}$, due to the 8 week training period.

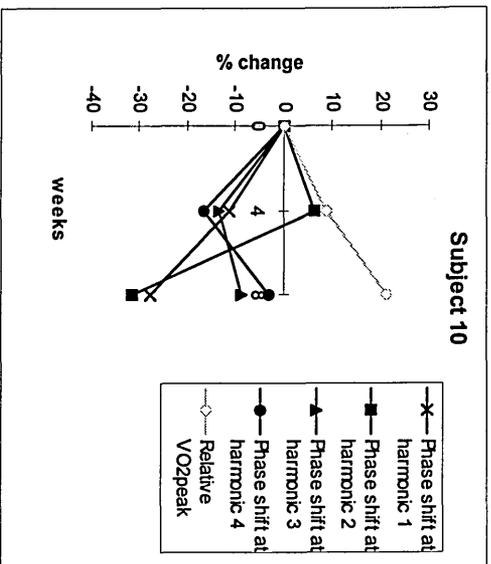
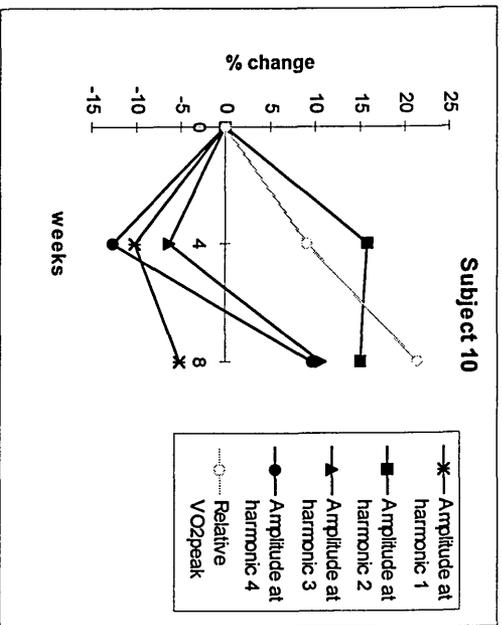


Table showing individual adaptations, in terms of change and % change, resulting from the 8 week training period.

Relative $\dot{V}O_{2peak}$ ($\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$)	0 to 4 weeks		4 to 8 weeks		0 to 8 weeks	
	Change	% change	Change	% change	Change	% change
Amplitude at harmonic 1 ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$)	2.26	9.0	3.19	11.4	5.45	21.3
Amplitude at harmonic 2 ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$)	-1.11	-10.3	0.50	5.2	-0.61	-5.1
Amplitude at harmonic 3 ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$)	0.93	15.7	-0.05	-0.7	0.88	15.0
Amplitude at harmonic 4 ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$)	-0.56	-6.4	1.40	17.0	0.83	10.6
Phase shift at harmonic 1 (degrees)	-1.06	-12.7	1.63	22.4	0.57	9.7
Phase shift at harmonic 2 (degrees)	0.89	-11.1	-4.00	-16.7	-7.00	-27.8
Phase shift at harmonic 3 (degrees)	1.06	6.4	-19.00	-38.0	-16.00	-31.6
Phase shift at harmonic 4 (degrees)	0.87	-13.3	3.00	4.6	-7.00	-8.7
Phase shift at harmonic 4 (degrees)	0.84	-16.5	9.00	13.6	-4.00	-2.9

Figures showing the individual changes, in $\dot{V}O_2$ kinetics and relative $\dot{V}O_{2peak}$, due to the 8 week training period.

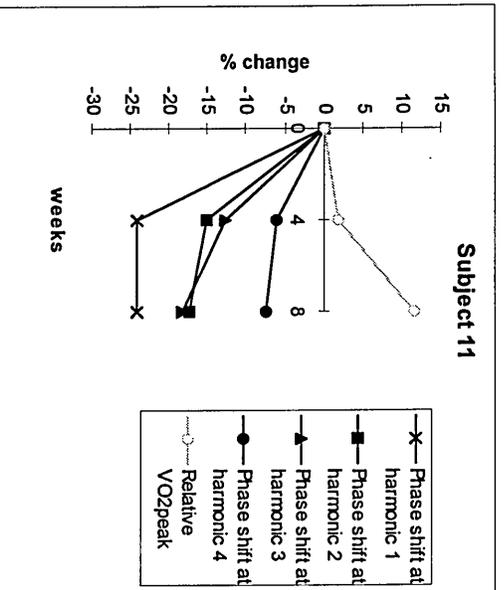
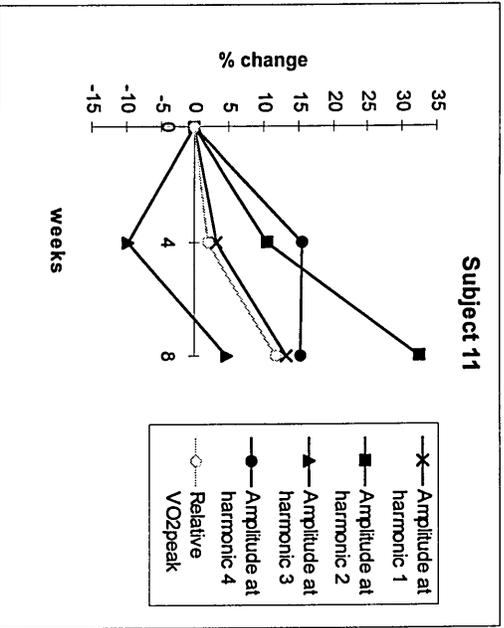


Table showing individual adaptations, in terms of change and % change, resulting from the 8 week training period.

	0 to 4 weeks		4 to 8 weeks		0 to 8 weeks	
	Change	% change	Change	% change	Change	% change
Relative $\dot{V}O_{2peak}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	0.25	2.0	1.98	9.5	2.24	11.7
Amplitude at harmonic 1 ($ml \cdot min^{-1} \cdot W^{-1}$)	0.29	3.1	0.99	10.1	1.28	13.2
Amplitude at harmonic 2 ($ml \cdot min^{-1} \cdot W^{-1}$)	0.67	10.6	1.53	22.1	2.20	32.6
Amplitude at harmonic 3 ($ml \cdot min^{-1} \cdot W^{-1}$)	-0.89	-9.6	1.19	14.3	0.31	4.7
Amplitude at harmonic 4 ($ml \cdot min^{-1} \cdot W^{-1}$)	1.22	15.6	-0.04	-0.4	1.19	15.2
Phase shift at harmonic 1 (degrees)	0.76	-24.1	0	0	-7.00	-24.1
Phase shift at harmonic 2 (degrees)	0.85	-15.1	-1.00	-2.2	-9.00	-17.3
Phase shift at harmonic 3 (degrees)	0.88	-12.5	-4.00	-5.7	-14.00	-18.2
Phase shift at harmonic 4 (degrees)	0.94	-6.1	-1.00	-1.3	-6.00	-7.4

Figures showing the individual changes, in $\dot{V}O_2$ kinetics and relative $\dot{V}O_{2peak}$, due to the 8 week training period.

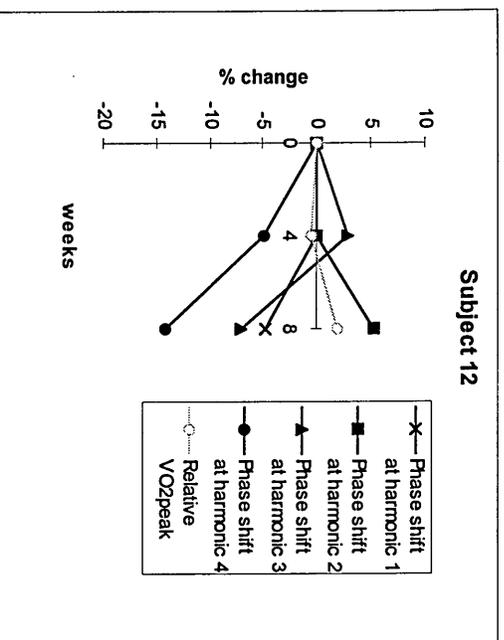
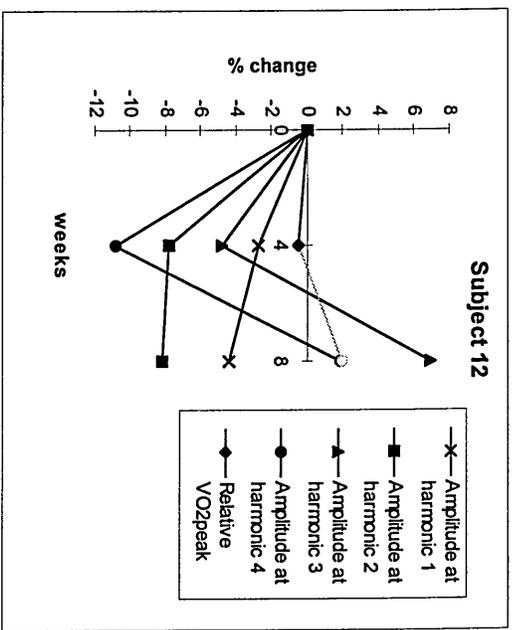


Table showing individual adaptations, in terms of change and % change, resulting from the 8 week training period.

	0 to 4 weeks		4 to 8 weeks		0 to 8 weeks	
	Change	% change	Change	% change	Change	% change
Relative $\dot{V}O_{2peak}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	-0.01	-0.5	0.46	2.4	0.45	1.9
Amplitude at harmonic 1 ($ml \cdot min^{-1} \cdot W^{-1}$)	-0.29	-2.7	-0.17	-1.6	-0.47	-4.3
Amplitude at harmonic 2 ($ml \cdot min^{-1} \cdot W^{-1}$)	-0.64	-7.8	-0.03	-0.4	-0.67	-8.2
Amplitude at harmonic 3 ($ml \cdot min^{-1} \cdot W^{-1}$)	-0.42	-4.8	0.98	11.7	0.56	6.9
Amplitude at harmonic 4 ($ml \cdot min^{-1} \cdot W^{-1}$)	-0.96	-10.8	1.02	12.7	0.05	1.9
Phase shift at harmonic 1 (degrees)	1.00	0	-1.00	-4.8	-1.00	-4.8
Phase shift at harmonic 2 (degrees)	1.00	0	2.00	5.3	2.00	5.3
Phase shift at harmonic 3 (degrees)	1.03	2.9	-7.00	-9.9	-5.00	-7.2
Phase shift at harmonic 4 (degrees)	0.95	-4.9	-8.00	-10.3	-12.00	-14.2

Figures showing the individual changes, in $\dot{V}O_2$ kinetics and relative $\dot{V}O_{2peak}$, due to the 8 week training period.

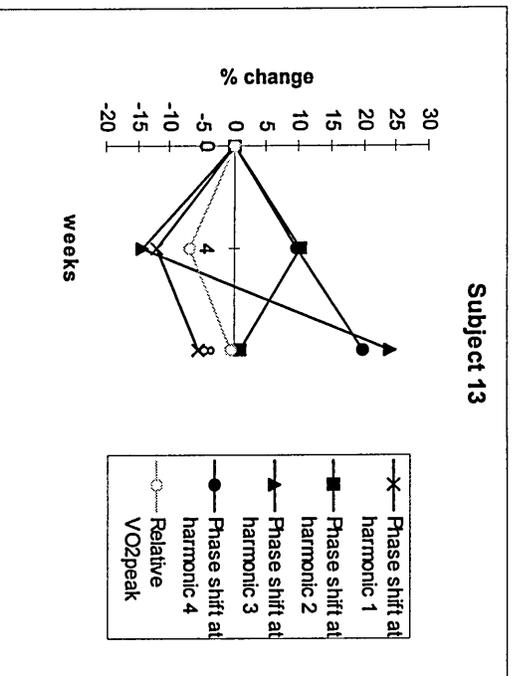
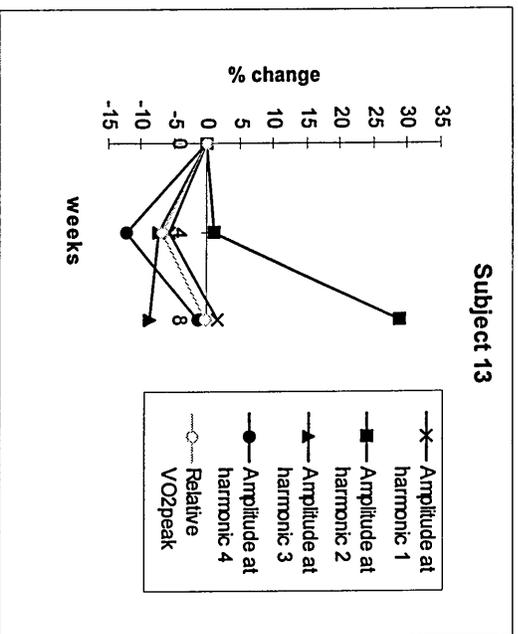


Table showing individual adaptations, in terms of change and % change, resulting from the 8 week training period.

	0 to 4 weeks		4 to 8 weeks		0 to 8 weeks	
	Change	% change	Change	% change	Change	% change
Relative $\dot{V}O_{2peak}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	-2.86	-6.9	2.71	6.8	-0.14	-0.5
Amplitude at harmonic 1 ($ml \cdot min^{-1} \cdot W^{-1}$)	-0.59	-5.7	0.68	7.1	0.09	1.4
Amplitude at harmonic 2 ($ml \cdot min^{-1} \cdot W^{-1}$)	0.06	0.9	1.88	28.0	1.93	28.9
Amplitude at harmonic 3 ($ml \cdot min^{-1} \cdot W^{-1}$)	-0.65	-7.5	-0.10	-1.2	-0.75	-8.7
Amplitude at harmonic 4 ($ml \cdot min^{-1} \cdot W^{-1}$)	-1.05	-12.4	0.81	10.9	-0.24	-1.5
Phase shift at harmonic 1 (degrees)	0.88	-12.0	1.00	4.5	-2.00	-5.5
Phase shift at harmonic 2 (degrees)	1.10	10.3	-4.00	-9.3	0	1.0
Phase shift at harmonic 3 (degrees)	0.86	-14.3	23.00	38.3	13.00	24.0
Phase shift at harmonic 4 (degrees)	1.10	9.7	8.00	10.1	15.00	19.8

Figures showing the individual changes, in $\dot{V}O_2$ kinetics and relative $\dot{V}O_{2peak}$, due to the 8 week training period.

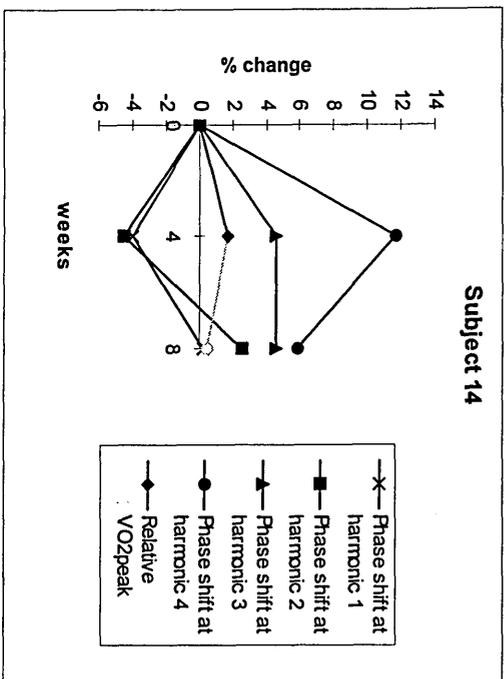
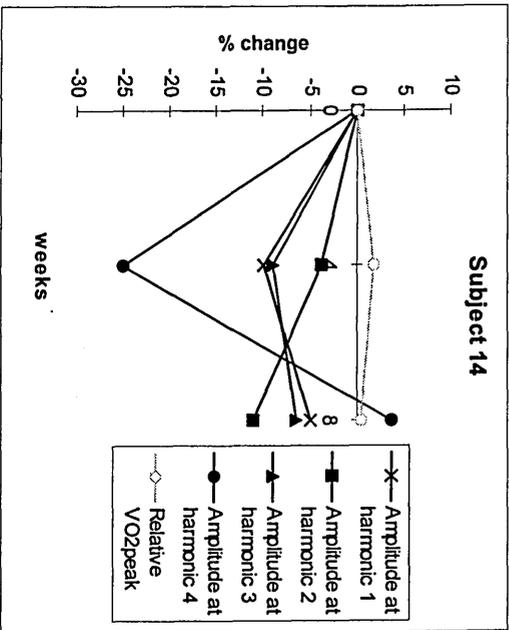


Table showing individual adaptations, in terms of change and % change, resulting from the 8 week training period.

Relative $\dot{V}O_{2peak}$ (ml·kg ⁻¹ ·min ⁻¹)	0 to 4 weeks		4 to 8 weeks		0 to 8 weeks	
	Change	% change	Change	% change	Change	% change
Amplitude at harmonic 1 (ml·min ⁻¹ ·W ⁻¹)	0.83	1.7	0.14	-1.2	0.97	0.5
Amplitude at harmonic 2 (ml·min ⁻¹ ·W ⁻¹)	-1.03	-9.9	0.46	4.9	-0.57	-5.0
Amplitude at harmonic 3 (ml·min ⁻¹ ·W ⁻¹)	-0.28	-3.8	-0.51	-7.2	-0.80	-11.0
Amplitude at harmonic 4 (ml·min ⁻¹ ·W ⁻¹)	-0.88	-9.0	0.23	2.6	-0.64	-6.4
Amplitude at harmonic 1 (degrees)	-2.28	-25.0	2.0	28.6	-0.33	3.6
Phase shift at harmonic 2 (degrees)	0.96	-4.0	1.00	4.2	0	0.2
Phase shift at harmonic 3 (degrees)	0.95	-4.5	3.00	7.1	1.00	2.6
Phase shift at harmonic 4 (degrees)	1.05	4.6	0	0	3.00	4.6
Phase shift at harmonic 4 (degrees)	1.12	11.7	-5.00	-5.8	4.00	5.9

Figures showing the individual changes, in $\dot{V}O_2$ kinetics and relative $\dot{V}O_{2peak}$, due to the 8 week training period.

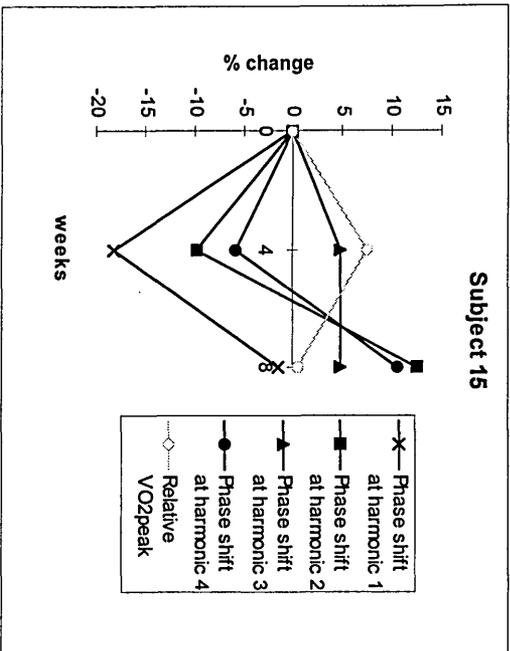
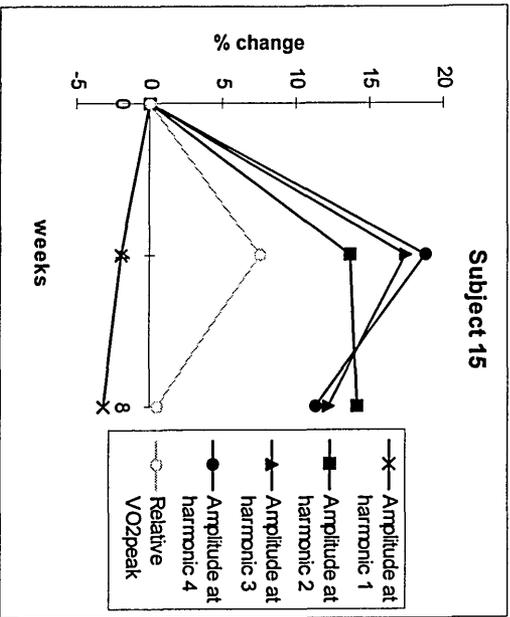


Table showing individual adaptations, in terms of change and % change, resulting from the 8 week training period.

	0 to 4 weeks		4 to 8 weeks		0 to 8 weeks	
	Change	% change	Change	% change	Change	% change
Relative $\dot{V}O_{2peak}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	0.83	7.5	-0.25	7.0	2.40	0.5
Amplitude at harmonic 1 ($ml \cdot min^{-1} \cdot W^{-1}$)	-0.20	-1.9	-0.13	-1.3	-0.32	-3.2
Amplitude at harmonic 2 ($ml \cdot min^{-1} \cdot W^{-1}$)	0.92	13.6	0.04	0.5	0.96	14.2
Amplitude at harmonic 3 ($ml \cdot min^{-1} \cdot W^{-1}$)	1.27	17.5	-0.46	-5.3	0.81	12.2
Amplitude at harmonic 4 ($ml \cdot min^{-1} \cdot W^{-1}$)	1.34	18.7	-0.62	-7.3	0.72	11.4
Phase shift at harmonic 1 (degrees)	0.82	-18.2	3.00	16.7	-1.00	-1.5
Phase shift at harmonic 2 (degrees)	0.90	-9.8	-1.00	-2.7	-5.00	12.5
Phase shift at harmonic 3 (degrees)	1.05	4.7	0	0	3.00	4.7
Phase shift at harmonic 4 (degrees)	0.94	-5.7	-4.00	-4.9	-9.00	10.6