

# **HEART RATE AS A MARKER OF TRAINING STATUS**

University of Cape Town

***Miriam R Sinclair.***

The copyright of this thesis vests in the author. No quotation from it or information derived from it is to be published without full acknowledgement of the source. The thesis is to be used for private study or non-commercial research purposes only.

Published by the University of Cape Town (UCT) in terms of the non-exclusive license granted to UCT by the author.

# **HEART RATE AS A MARKER OF TRAINING STATUS.**

**BY**

**Miriam Rosemary Sinclair.**

**This thesis is presented for the Degree of**

**DOCTOR OF PHILOSOPHY**

**In the Department of Human Biology**

**UNIVERSITY OF CAPE TOWN.**

**SOUTH AFRICA.**

**September 2006.**

**MRC / UCT Research Unit for**

**Exercise Science and Sports Medicine.**

**Sports Science Institute of South Africa.**

**Boundary Road**

**Newlands**

**7700.**

**South Africa.**

## DEDICATION

I would like to dedicate this thesis to each and every person who has ever touched my life, even if only in a very small way. Each one of you has taught me something and enriched my life, even if you can't remember or thought our meeting insignificant.

I would especially like to dedicate this thesis to my mother. She sacrificed unselfishly, encouraged, supported, accepted and loved me unconditionally and I would not have been the person I am if it wasn't for her. Mom, I love you dearly and THANK YOU for you.

To Pearly Beach which I had to sacrifice to be able to work toward this degree.

## ACKNOWLEDGEMENTS

I would like to thank the following people for all their help, support and encouragement throughout this thesis.

Most importantly, **Professor Mike Lambert**. Mike, without your help, support and empathic assistance, I would not have been here today. As a mature student, my needs were different from those of the average student and it requires a very special person to recognize that and be able to deal with it in a kind and sensitive manner. I thank you from the bottom of my heart !

**Professor Tim Noakes**. Tim, you are the epitome of a kind and humble gentleman and a brilliant scientist at that. You always make time for the small person and that makes you BIG in my book. Thank you.

**My subjects**. Without your willingness to participate in sometimes grueling fitness testing, this thesis would not have been possible ! Thank you and I hope I was able to give something back to you all.

**All the staff and students at ESSM**. A very big thank you to each and every one for your interest, help and support throughout the years. A special thanks to **Lesa Siveright** and **Paul Rossouw**. Dankie Paultjie !

A very special thanks goes to **Michele van Rooyen** and **Wayne Viljoen**. Without the endless crazy bantering, life would have been a lot more painful. It is only a pleasure to know you both. Wayne, you nearly killed me with that training programme but wow, did I lift some serious weights !

**Mariam Hassen**. Thanks girl ! I appreciate all your help with my testing of the adventure racers. Your friendship is special to me.

**Manda Nkuhlu**. Your contribution toward Chapter 7 is gratefully acknowledged. Thank you.

**Justin Durandt, Stephan du Toit, Hennie Kriel and Mike Lambert**. Your great contribution toward Chapter 8 is gratefully acknowledged. Thank you very much !

The **Harry Crossley and Nellie Atkinson Research Fund** of the **University of Cape Town** for financial assistance toward the research.

To the very best sister in the world, **Prissilla**. A very big thank you for everything you have done for me over the years and your unflinching love and support. I really appreciate it. Love you lots. **Lex**, how can I even begin to say thank you for all the help and encouragement you have given me. Thank you for your very special friendship. Love you.

**John**. You are a very special person in my life. Always remember that you are much loved. Thank you very much for everything you have done for me and being there for me always !

**Doctoral Degrees Board**

University of Cape Town  
Private Bag Rondebosch  
7701 South Africa

Tel: (021) 650-2155

Fax: (021) 650-2138

vnaidoo@bremner.uct.ac.za

---

***Please complete and return to the Doctoral Degrees Board,  
University of Cape Town, when submitting your thesis for  
examination***

**PHD THESIS TITLE: Heart rate as a marker of training status.**

**I, Miriam Rosemary Sinclair**

hereby

- (a) grant the University of Cape Town free licence to reproduce the above thesis in whole or in part, for the purpose of research;
- (b) declare that:
  - (i) the above thesis is my own unaided work, both in concept and execution, and that apart from the normal guidance from my supervisor, I have received no assistance except as stated below:

---

---

---

- (ii) neither the substance nor any part of the above thesis has been submitted in the past, or is being, or is to be submitted for a degree at this University or at any other university. except as stated below

---

---

---

I am now presenting the thesis for examination for the degree of PhD.

SIGNED: \_\_\_\_\_

DATE: \_\_\_\_\_

## ABSTRACT

### Heart rate as a marker of training status.

Miriam Rosemary Sinclair.

September 2006.

It is generally accepted that a linear relationship exists between heart rate / workload and oxygen consumption and that heart rate thus accurately reflects workload and exercise intensity. As such, coaches and athletes commonly use heart rate to prescribe exercise and monitor changes in training status. Some studies have however, indicated that heart rate may not always be an accurate indicator of training status under all conditions, due to the possible influence of other variables. However, as measuring heart rate has been shown to be a reliable, accurate, inexpensive and practical method to monitor changes in training status, the purpose of this study was therefore to further explore and clarify the heart rate / workload relationship under a variety of different training and testing conditions.

The studies were conducted under well controlled vs. field testing conditions, with and without changes in training status, under controlled and uncontrolled training conditions and over varying time periods. Subjects' training status varied from generally trained to elite. Two studies consisted of monitoring heart rate during sleep using portable heart rate monitors. Five training studies were completed during which heart rate and heart rate recovery and their relationship to changes in performance was monitored. The main test used to measure heart rate and heart rate recovery (except for the heart rate during sleep studies), was a submaximal heart rate test, designed by the Sports Science Institute. The repeatability of this test is high ( $r = 0.94 - 0.99$ ) (Lamberts et al., 2004). Performance parameters were assessed using previously validated methods such as the 20-m Multi-Stage Shuttle Test (Léger et al., 1988), 5-m

multiple shuttle test (Boddington et al., 2001) Agility test (Adapted from Semenick, 1994). 10 and 20-m sprints and muscle power (squat jumps) (Jennings et al., 2005). The appropriate statistical methods were employed for each of the studies.

The most important finding was that changes in heart rate and heart rate recovery were not necessarily related to changes in training status or performance. Even though the submaximal heart rate test is effective in monitoring the general trend in changes in training status in a group of subjects, intra-variability is high when applied to individual athletes.

Changes in heart rate during sleep did not track changes in training status in a strength trained subject who improved performance by up to 79 %. In a group of habitually physically active subjects, heart rate measured during sleep has precision to detect changes in heart rate of about 8 beats.min<sup>-1</sup>. This level of precision is not sufficient for use in highly trained athletes as the heart rate changes experienced in this group is generally small.

In conclusion, even though measuring heart rate is valid, reliable, simple and inexpensive to use, the many variables that may potentially affect its relationship to training status should be kept in mind when interpreting heart rate / training status changes. As such, every subject should ideally undergo baseline heart rate testing, using a mode-specific, graded exercise test over several sessions to determine that specific individuals' intrinsic day –to day variation prior to interpreting heart rate and performance data.

## SPECIFIC AIMS / RESEARCH QUESTIONS

The aims of this thesis were to:

1. To further explore and clarify the heart rate / workload relationship under differing conditions such as well controlled testing vs. field testing conditions, controlled vs. uncontrolled training, changed training status vs. unchanged training status and average trained vs. elite athletes over varying time periods.
2. Furthermore, a goal of this study was to ascertain if measuring heart rate and heart rate recovery under these different conditions would be sufficiently precise in tracking changes in training status to determine whether this is a viable and practical marker that can be used by athletes and coaches to monitor changes in training status and for exercise prescription.

Chapter 1 examines the relevant literature pertaining to the thesis.

In the subsequent experimental section, the research questions addressed are:

1. *Is measuring heart rate during sleep repeatable under free-living conditions in a group of subjects whose training status remained unchanged ?*

The first study (Chapter 2) of the thesis assessed the repeatability of measuring heart rate during sleep under free-living conditions in a group of average endurance trained subjects over a short 3-week period during which time their training status remained unchanged.

- 2. Is measuring heart rate during sleep repeatable under free-living, but well controlled conditions in a subject whose training status changed ?*

The second study (Chapter 3) was designed to examine if measuring heart rate during sleep would be repeatable under controlled but free-living conditions in a national level, strength-trained subject whose training status changed over a period of 18 weeks.

- 3. Do changes in submaximal heart rate during exercise reflect changes in training status under well controlled testing conditions in a subject whose training status changed ?*

The third study (Chapter 4) examined the submaximal heart rate, heart rate recovery and performance responses of an international level field hockey referee to uncontrolled increased training over an 8-week period during which time training status changed. Testing took place under well controlled conditions.

- 4. Do changes in submaximal heart rate during exercise reflect changes in training status under field-testing conditions in a group of subjects whose training status changed ?*

The fourth study (Chapter 5) aimed to further examine the same parameters as measured in study 3. The aim was to further clarify the relationship between changes in the heart rate / workload relationship and performance after training under field-testing conditions when training status changed over an 8-week period in a large group of sub-elite rugby and cricket players. In this study the environmental conditions also changed.

5. *Do changes in the heart rate / workload relationship accurately reflect changes in training status under well controlled testing conditions in a group of extreme adventure racers undergoing heavy multi-modality training ?*

Study five (Chapter 6) assessed a group of sub-elite extreme adventure racers over a period of 18 weeks to examine heart rate, heart rate recovery and performance responses to an uncontrolled regime of high level, multi-modality type training. A secondary aim was to assess if racers trained and competed at similar heart rate levels. Testing took place under controlled conditions.

6. *Do changes in the heart rate / workload relationship accurately reflect changes in training status under a controlled regime of progressive training overload under controlled testing conditions when training status changed ?*

Study six (Chapter 7) aimed at examining the heart rate and heart rate recovery responses in a group of average trained subjects over a short period of 4 weeks. Subjects underwent a

controlled progressive training regime with the specific aim of inducing changes in training status and testing took place under controlled conditions.

- 7. Does perception of fatigue and changes in heart rate recovery relate to training time and intensity under well controlled training and testing conditions in a group of subjects whose training status changed ?*

Study seven (Chapter 8) aimed to determine if changes in heart rate, heart rate recovery and perception of fatigue related to training time and intensity over a period of 20 weeks in a group of elite rugby players whose training status changed.

The final chapter (Chapter 9) summarizes the most important findings of the study and presents a proposal as to possible mechanisms at work when measuring heart rate recovery and performance changes, as well as an indication of direction for future studies.

## LIST OF PUBLICATIONS

Waldeck, M.R. and Lambert, M.I. (2003). \*

Heart rate during sleep: implications for monitoring training status.  
*electronic Journal of Sports Science and Medicine*. 2, 133-138.

Boddington, M.K., Lambert, M.I. and Waldeck, M.R. (2003).

The analysis of skilled performance and game parameters during league Field Hockey Matches. *International Journal of Performance Analysis in Sport*. 3 (2), 121-129.

Boddington, M.K., Lambert, M.I. and Waldeck, M.R. (2004).

Validity of a 5-m Multiple Shuttle Run Test for Assessing Fitness of Female Field Hockey Players. *Journal of Strength and Conditioning Research*. 18(1) 97-100.

Lambert, M.I., Dugas, J.P., Kirkman, M.C., Mokone, G.G and Waldeck, M.R. (2004).

Changes in running speeds in a 100 km ultra-marathon race.  
*electronic Journal of Sports Science and Medicine*. Sept; 3 (3),

\* Directly from thesis data. Other studies are peripheral to the thesis.

## PROFESSIONAL PRESENTATION

Waldeck, M.R. and Lambert, M.I. (**Poster presentation**) (2002).

Heart rate during sleep: implications for monitoring training status.  
Proceedings of Southern Africa Physiology Conference.  
University of Stellenbosch, South Africa.

## CONTENTS

	PAGE
DEDICATION	ii
ACKNOWLEDGEMENTS	iii
DECLARATION	v
ABSTRACT	vi
AIMS AND RESEARCH QUESTIONS	viii
LIST OF PUBLICATIONS	xii
PROFESSIONAL PRESENTATION	xii
CONTENTS	xiii
LIST OF TABLES	xviii
LIST OF FIGURES	xx
<b>CHAPTER 1 INTRODUCTION TO THESIS.</b>	
<b>1.1 INTRODUCTION.</b>	1
<b>1.2 PRINCIPLES OF TRAINING ADAPTATION.</b>	4
<b>1.3 TRAINING STATUS</b>	6
<u>1.3.1 OVERREACHING.</u>	8
<u>1.3.2 OVERTRAINING SYNDROME.</u>	9
1.3.2.1 <i>PHYSIOLOGICAL FACTORS AND THE OVERTRAINING SYNDROME.</i>	10
<b>1.4 METHODS OF MEASURING TRAINING STATUS.</b>	14
<u>1.4.1 BLOOD BORNE MARKERS.</u>	16
1.4.1.1 <i>BIOCHEMICAL</i>	16
1.4.1.1.1 Blood lactate.	16

	<b>PAGE</b>
1.4.1.1.2 Other selected biochemical markers.	20
1.4.1.1.2.1 Hemoglobin and Hematokrit.	20
1.4.1.1.2.2 Blood enzyme activity.	22
1.4.1.1.2.3 Urea and metabolites.	22
1.4.1.1.2.4 Immunological markers.	24
1.4.1.2 <i>HORMONAL.</i>	25
1.4.1.2.1 Epinephrine and Norepinephrine.	26
1.4.1.2.2 Testosterone and Cortisol.	27
1.4.1.2.3 Pituitary hormones.	29
1.4.1.2.4 Other hormones: Insulin and Thyroid Stimulating Hormone (TSH).	30
<u>1.4.2 PSYCHOLOGICAL MARKERS.</u>	31
1.4.2.1 <i>PROFILE OF MOODS QUESTIONNAIRE (POMS).</i>	32
1.4.2.2 <i>RATING OF PERCEIVED EXERTION (RPE).</i>	33
<u>1.4.3 PHYSIOLOGICAL MARKERS.</u>	36
1.4.3.1 <i>MAXIMAL OXYGEN UPTAKE</i>	36
<b>1.4.3.2 HEART RATE .</b>	39
1.4.3.2.1 Introduction.	39
1.4.3.2.2 Measuring heart rate.	39
1.4.3.2.3 Heart rate during exercise.	40
<b>1.4.3.2.4 General physiology.</b>	43
<u>1.4.3.2.4.1 Autonomic (Extrinsic) factors.</u>	44
1.4.3.2.4.1.1 Plasma volume.	44
1.4.3.2.4.1.2 Baroreceptor reflex function.	46
1.4.3.2.4.1.3 Sympathetic nervous system.	47
1.4.3.2.4.1.4 Parasympathetic nervous system.	48
1.4.3.2.4.1.5 Endocrine system.	50
<u>1.4.3.2.4.2 Non-Autonomic (Intrinsic) factors.</u>	50
1.4.3.2.4.2.1 Heart volume.	50
1.4.3.2.4.2.2 Myocardial cell metabolism.	52
1.4.3.2.4.2.3 Electrophysiology of sinoatrial node.	53
1.4.3.2.4.2.4 $\beta$ -adrenergic receptors.	53

	<b>PAGE</b>
<b>1.4.3.2.5 Factors that may affect heart rate.</b>	54
1.4.3.2.5.1 <i>Cardiac drift.</i>	55
1.4.3.2.5.2 <i>Circadian rhythm.</i>	58
1.4.3.2.5.3 <i>Competition.</i>	61
1.4.3.2.5.4 <i>Environmental factors.</i>	64
1.4.3.2.5.5 <i>Medication.</i>	66
1.4.3.2.5.6 <i>Mode of exercise.</i>	67
1.4.3.2.5.7 <i>Training status.</i>	69
<b>1.4.3.2.6 Heart rate during sleep.</b>	70
1.4.3.2.6.1 <i>Types of sleep.</i>	72
1.4.3.2.6.2 <i>Effect of exercise on heart rate during sleep.</i>	73
<b>1.4.3.2.7 Heart rate variability.</b>	75
1.4.3.2.7.1 <i>Control mechanisms.</i>	76
1.4.3.2.7.2 <i>Technical and other measurement problems.</i>	76
1.4.3.2.7.3 <i>Length of training period.</i>	77
1.4.3.2.7.4 <i>Autonomic Nervous System.</i>	77
1.4.3.2.7.5 <i>Age and Gender.</i>	79
1.4.3.2.7.6 <i>Training intensity.</i>	80
<b>1.4.3.2.8 Heart rate recovery.</b>	81
1.4.3.2.8.1 <i>Autonomic Nervous System.</i>	82
1.4.3.2.8.2 <i>Training status and other variables.</i>	85
1.4.3.2.8.3 <i>Heart rate recovery and sudden death.</i>	85
<b>1.4.3.2.9 Potential of using heart rate as a marker of training status.</b>	88
<b>1.5 SUMMARY.</b>	91
<b>CHAPTER 2 HEART RATE DURING SLEEP: IMPLICATIONS FOR MONITORING TRAINING STATUS.</b>	
2.1 Abstract	94
2.2 Introduction	95
2.3 Methodology	96
2.3.1 Subjects	96
2.3.2 Experimental design	97
2.3.3 Statistical analysis	98
2.4 Results	99
2.5 Discussion	107

	<b>PAGE</b>
<b>CHAPTER 3 REPEATABILITY OF MEASURING HEART RATE DURING SLEEP: A CASE STUDY.</b>	
3.1 Abstract	110
3.2 Introduction	111
3.3 Methodology	111
3.3.1 Subjects	111
3.3.2 Experimental design	112
3.3.3 Statistical analysis	114
3.4 Results	115
3.5 Discussion	121
<b>CHAPTER 4 DO CHANGES IN SUBMAXIMAL HEART RATE DURING EXERCISE REFLECT CHANGES IN TRAINING STATUS: A CASE STUDY.</b>	
4.1 Abstract	126
4.2 Introduction	127
4.3 Methodology	128
4.3.1 Subjects	128
4.3.2 Experimental design	129
4.3.3 Statistical analysis	133
4.4 Results	134
4.5 Discussion	137
<b>CHAPTER 5 DO CHANGES IN SUBMAXIMAL HEART RATE DURING EXERCISE REFLECT CHANGES IN TRAINING STATUS: A FIELD STUDY.</b>	
5.1 Abstract	141
5.2 Introduction	142
5.3 Methodology	143
5.3.1 Subjects	143
5.3.2 Experimental design	144
5.3.3 Statistical analysis	146
5.4 Results	147
5.5 Discussion	158
<b>CHAPTER 6 HEART RATE, HEART RATE RECOVERY AND PERFORMANCE CHANGES IN A GROUP OF ADVENTURE RACERS.</b>	
6.1 Abstract	163
6.2 Introduction	164

	<b>PAGE</b>
6.3 Methodology	167
6.3.1 Subjects	167
6.3.2 Experimental design	167
6.3.3 Statistical analysis	171
6.4 Results	171
6.5 Discussion	181
<b>CHAPTER 7</b>	<b>RELATIONSHIP BETWEEN VARIOUS MARKERS OF TRAINING STATUS, INCLUDING SUBMAXIMAL HEART RATE AND TRAINING VOLUME.</b>
7.1 Abstract	189
7.2 Introduction	190
7.3 Methodology	191
7.3.1 Subjects	191
7.3.2 Experimental design	191
7.3.3 Statistical analysis	194
7.4 Results	194
7.5 Discussion	205
<b>CHAPTER 8</b>	<b>RELATIONSHIP BETWEEN HEART RATE RECOVERY, FATIGUE AND TRAINING TIME.</b>
8.1 Abstract	212
8.2 Introduction	213
8.3 Methodology	214
8.3.1 Subjects	214
8.3.2 Experimental design	214
8.3.3 Statistical analysis	215
8.4 Results	215
8.5 Discussion	220
<b>CHAPTER 9</b>	<b>SUMMARY AND CONCLUSIONS.</b>
	223
<b>REFERENCES</b>	231
<b>APPENDIXES</b>	280

## LIST OF TABLES

### CHAPTER 1

- 1.1 Summary of different methods and specific characteristics used to monitor training status 91

### CHAPTER 2

- 2.1 Descriptive characteristics of subjects (n = 10) 100
- 2.2 Total minutes in-bed time (uncorrected) per subject per session (n = 10) 101
- 2.3 Descriptive statistics of the average, minimum and maximum heart rate variation (beats.min<sup>-1</sup>) for sleep for each of the three sessions for each of the ten subjects 103
- 2.4 Descriptive statistics of the average, minimum and maximum heart rate (beats.min<sup>-1</sup>) for sleep and during the day for each of the three sessions 104
- 2.5 Intraclass Correlation Coefficient and 95% confidence intervals for the average, minimum and maximum heart rates for the three sessions for each of the 10 subjects. The data are divided into sleep and day time 105

### CHAPTER 3

- 3.1 Body mass (kg), training load (kg<sup>-1</sup>. wk<sup>-1</sup>) and average, minimum and maximum heart rate (beats.min<sup>-1</sup>) during sleep for 18 weeks 116

### CHAPTER 4

- 4.1. Mean heart rate (beats.min<sup>-1</sup>), coefficient of variation (%) and 95% confidence intervals for the heart rates for each stage of the submaximal heart rate test (n = 1; average for 8 tests) 135
- 4.2. Mean heart rate (beats.min<sup>-1</sup>) (n=1) for each stage and recovery period during the submaximal heart rate test during test 1 and test 8 136

	<b>PAGE</b>
<b>CHAPTER 5</b>	
5.1 General descriptive characteristics of subjects (n = 84)	147
5.2 Mean heart rate (beats.min <sup>-1</sup> ), (n = 84), for each stage and recovery period during the submaximal heart rate test conducted before and after 8 weeks	150
<b>CHAPTER 6</b>	
6.1 Weekly schedule of testing of adventure racers over a period of 18 weeks (n = 9)	167B
6.2 General descriptive characteristics of subjects (n = 9)	172
6.3 Average heart rate (beats.min <sup>-1</sup> ) (n=9) for each stage and recovery period during the submaximal heart rate test during week 1 and week 18	173
6.4 Average one week pre-and post race performance data after two adventure races	177
<b>CHAPTER 7</b>	
7.1 Description of performance parameter changes between week 1 and week 4 (n = 21)	196
7.2 Fatigue and Muscle Soreness ratings for weeks 2, 3 and 4 (n = 21)	197
7.3 Mean heart rate (beats.min <sup>-1</sup> ) for each stage and recovery period during the submaximal heart rate test conducted for four weeks	199
<b>CHAPTER 8</b>	
8.1 The average body mass, subjective rating of exercise intensity, perception of fatigue, average maximal heart rate during the submaximal heart rate test and heart rate recovery percentage for a 20-week period	217

## LIST OF FIGURES

## CHAPTER 2

- 2.1 A sample of the raw tracings of heart rate (beats.min<sup>-1</sup>) recorded for each of the three sessions over 24 hours for subject 1 102
- 2.2 The limits of agreement (LOA) (Bland and Altman, 1986) for minimum heart rate during sleep for test 1 vs. 2, test 2 vs. 3 and test 1 vs. 3. 106

## CHAPTER 3

- 3.1 Average heart rate (beats.min<sup>-1</sup>) and 95 % confidence intervals (CI) (a) and normalized average heart rate (%) (b) during sleep for 18 weeks 117
- 3.2 Training load (kg / session) and rating of perceived exertion (units / session) over an 18- week period 118
- 3.3 Fatigue rating (units / session) and muscle soreness (units / session) over an 18-week period 119

## CHAPTER 5

- 5.1 Group data (n = 84) for (a) average distance (m) covered during the 5-m multiple shuttle test (MST) and (b) rating of perceived exertion (RPE) recorded during the MST over two test sessions 149
- 5.2 a The relationship between changes in the delta heart rate (beats.min<sup>-1</sup>) and distance covered (m) during the MST during the stages of the submaximal heart rate test 151
- 5.2 b The relationship between changes in the delta heart rate (beats.min<sup>-1</sup>) and the distance covered (m) during the MST during recovery of the submaximal heart rate test 152
- 5.3 Average descriptive statistics of the environmental factors over two test sessions for all eight groups 154
- 5.4 The effect of delta temperature (C°), delta humidity (%) and delta windspeed (m.s<sup>-1</sup>) on heart rate (beats.min<sup>-1</sup>) during stage 4 and recovery 4 of the submaximal heart rate test 155

	PAGE
<b>CHAPTER 6</b>	
6.1 Average heart rate (beats.min <sup>-1</sup> ) and standard deviation during stage 4 and recovery 4 of the submaximal heart rate test over 18 weeks	174
6.2 Normalized values (%) for stage 4 and recovery 4 of the submaximal heart rate test over a period of 18 weeks	175
6.3 Average training time (minutes / week <sup>-1</sup> ) over 18 weeks	176
6.4 Percentage time during both races 1 and 2 spent within different heart rate ranges	179
<b>CHAPTER 7</b>	
7.1 Number of individual shuttles run during the 20-m Multi-Stage shuttle run for weeks 1 to 4	195
7.2 Heart rate recovery percentage (%) for weeks 1 to 4	198
7.3 a, b Average heart rate (beats.min <sup>-1</sup> ) for each of the Stages and Recovery periods recorded during the submaximal heart rate test	201
7.4 a The relationship between changes in the delta heart rate (beats.min <sup>-1</sup> ) during Stages 1 to 4 of the submaximal heart rate test and the change in the number of shuttles completed during the 20-m Multi-Stage shuttle test	202
7.4 b The relationship between changes in the delta heart rate (beats.min <sup>-1</sup> ) during Recovery 1 to 4 of the submaximal heart rate test and the change in the number of shuttles completed during the 20-m Multi-Stage shuttle test	203
<b>CHAPTER 8</b>	
8.1 The normalized data over the season for training time (A), players' perception of fatigue (B), players' subjective rating of training intensity (C) and heart rate recovery (D)	218
8.2 The difference in points – a positive score indicates that the team which was studied won the match	219

**CHAPTER 9**

9.1 Proposed scheme of possible scenarios of an athletes' heart rate recovery status during different stages of training	228 B
--	-------

University of Cape Town

# CHAPTER 1

## INTRODUCTION TO THE THESIS

University of Cape Town

## 1.1 INTRODUCTION.

In society today sport is big business with top performers in certain sporting disciplines earning exorbitant salaries. Competition for the available funds is intense. It is thus becoming increasingly important for top sportsmen and women to become and remain highly competitive for as long as possible. Athletes and coaches continuously look for strategies that will give them the competitive edge in an attempt to achieve peak performances coinciding with important competitions and are attempting to apply science more consistently.

The difference between success and failure at an elite level is extremely small. Snyder and Foster (1994) found that the difference between the gold and silver medalists at the 1988 Olympic speed skating competition was 0.3 %, with the mean difference between the gold and 4<sup>th</sup> place competitors a mere 1.3 %. Similarly a more recent study (Hopkins et al., 1999) examined the question of what the smallest variation would need to be for an elite athlete to have a chance at winning a gold medal. According to Hopkins et al (1999) the variation for elite athletes was approximately 1 % for running, 2 % for jumping and 3 % for throwing events. An analysis on Olympic triathletes showed similar results with a variation of 1.5 % for the top athletes, leading them to conclude that an athlete would have to improve at least 0.8 % in overall time to increase their chance of victory (Paton and Hopkins, 1999). This implies that small changes in performance may be significant, depending on the specific event. This however, imposes a challenge for monitoring performance changes with training with sufficient precision to detect these small but significant changes in performance and competition. It is certainly clear that the margin for success is extremely small, especially at the elite level. At this elite level, consistent, hard training is one of the prerequisites for success. However, the hard training which is necessary in the quest for peak performance has led to symptoms

associated with the overtraining syndrome (Foster, 1997) and overreaching (Halson and Jeukendrup, 2004).

Sporting performances have improved significantly in the last century. This can be attributed to many different factors, for example, the science behind training, sport equipment and dietary or nutritional intakes have improved dramatically in recent years (Tipton, 1997). Dietary intakes in Olympic athletes were only studied for the first time in 1952. Nutritional intakes and studies are now part of the holistic approach to sport (Tipton, 1997).

Sport physiology was only applied to sport in 1979, incorporating specific training principles into the various methodologies currently used by athletes regardless of training status (Tipton, 1997). Previously training methods incorporated a composite of Galen's concepts, personal observations, experience and word-of-mouth theories (Park, 1992). Galen was a Greek physician, born in the middle ages, who pioneered the concept of anatomy, physiology and therapeutics (<http://galenmedical.com/www/docs/8>).

The overriding factor however, that will affect physical performance is training. Optimal physical performance requires that the athlete, through training, disturbs the body's inherent homeostasis by progressively overloading the different physiological and metabolic systems to enable adaptation to occur. The exact amount of overload needed however, is difficult to predict and needs to be adapted to each individuals' specific needs. Individuals respond and thus adapt differently to rest periods and training loads (Hellard et al., 2005).

Bouchard and Rankinen (2001) reviewed several studies addressing the question of inter-individual variation in response to regular exercise training and the contributions of age,

gender, race and pre-training level. They showed that although several studies indicated marked differences in the way individuals responded to exercise training, age, gender and race had no impact on these differences. For example, they found that maximal oxygen uptake responses after training, varied from almost nothing at all to 100 % in large groups of sedentary individuals. Pre-training levels of fitness and other familial factors however, were important determinants in the way individuals responded to exercise training.

Differences in metabolism may also account for differences in the way athletes adapt to training. For example, Goedecke et al (2000) examined the variability in respiratory exchange ratio (RER) at rest and during exercise in a group of 61 trained cyclists. Their RER was measured after a 10 – 12 hour fasting period at rest and during exercise testing at 25, 50 and 70 % of peak power output (W). They found that there was a large inter-individual variability in resting RER (0.7 – 0.9) which persisted during exercise of increasing intensity. It is logical to assume that these differences in metabolism influence the response and adaptability to a training session.

It is generally accepted that an increase in training volume and intensity is directly related to increases in performance (Foster et al., 1996; Snyder and Foster, 1994). This concept of training progression is embodied in the legend of the young farmer who lifted a growing young bullock every day and so became one of the strongest people in the world (Foster et al., 1996). Kuipers and Keizer (1988) demonstrated the principle of training progression and performance in an inverted U – shaped curve model where optimal training is at the top of the curve and flanked by either under or overtraining. Since optimal training cannot yet be accurately defined, the athlete remains precariously balanced between under and overtraining, success and failure.

To this end athletes and coaches use a multitude of performance and physiological tests to optimize training with the goal of peaking at the appropriate time, whilst avoiding the negative side-effects of training. Regular performance tests are also used by coaches for prescribing exercise intensities and evaluating athletic progress (Lambert, 2006). Optimal athletic performance requires precise overloading with sufficient rest periods, so that the athlete neither under or over-trains.

Before one can examine the different performance and physiological tests available to measure training status and changes in training status, it is important to review how and when adaptation occurs in the athlete when exposed to a training stimulus.

## **1.2 PRINCIPLES OF TRAINING ADAPTATION.**

To induce any physiological, metabolic or structural changes (adaptation) associated with improved performance, the athlete needs to subject the body to positive stress, i.e a training stimulus. Positive stress in the form of regular physical activity increases the body's capability to produce more energy, tolerate physical stress better and most importantly improve exercise or sporting performance (Wilmore and Costill, 1994).

The major physical changes or adaptations occur within the first 6 to 10 weeks after the start of a training programme. The magnitude of the changes is dependent on the volume and intensity of exercise training (Foster et al., 1996; Snyder and Foster, 1994; Wilmore and Costill, 1994). Hickson et al (1981) found that several physiological variables (maximum oxygen uptake, heart rate and blood lactate responses) changed within the first two to three weeks of two different

training periods during a submaximal exercise test. The exercise intensity was increased after the first four weeks and kept at the higher level for an additional five week period. They concluded that unless the exercise intensity is adjusted regularly, no further improvement in heart rate, blood lactate or  $VO_2\text{max}$  will be experienced. This finding links up with the concept of progressive overload to ensure continued adaptation.

Foster et al (1996) found that a 10- fold increase in training load may lead to a 10 % improvement in performance from "keep – fit" maintenance levels. He came to this conclusion after a large number of athletes were tested before and after training loads were increased. A smaller number of athletes were also tested after several changes in training loads. The results indicated a systemic relationship between performance and training load. This principle has encouraged many athletes and coaches to embrace the concept of "*more is better*" with the perception that the athlete who trains the most will necessarily be the better athlete.

However, each athlete is unique, has a different rate of adaptation (Avalos et al., 2003; Mujika, 1998) and responds very differently to the same training stress (Hellard et al., 2005). What may be perceived as light training for one individual may cause another to overtrain. Lambert and Keytel (2000) conducted a study on the top male and female runners in the 56 km Two Oceans Marathon with the aim of establishing if a relationship existed between training volume and running performance. They found an inverse relationship ( $r = 0.75$ ) between the two variables but more interestingly it became clear that the annual training volume of some of the male runners were four times higher compared to other male runners of similar ability and performance. This then begs the question – what would happen if some of these athletes trained more and some less ?

The quest for anyone striving to train to produce peak performance is to identify the optimal amount of physical stress and recovery time between training sessions to provide the stimulus for adaptation which translates into sporting performance. If this is not determined precisely, the athlete is likely to either be undertrained or become over-stressed, leading to a decrement in performance and/or injury.

### **1.3 TRAINING STATUS.**

It is generally accepted that to ensure sporting success, a system of progressive exposure to an increasing training load, the "overload principle" is required to stimulate adaptation (Harre, 1973), coupled with an adequate amount of rest and recuperation between training sessions. The effectiveness of sports training essentially depends on the interplay between three important components namely intensity, volume and rest periods (the principle of periodisation) (Fry et al., 1992). The optimal net training load, taking into account the balance between overload and rest for each athlete needs to be determined.

However, the optimal net training load for each athlete is extremely difficult to calculate as each individual athlete reacts differently to the increased training stimulus (Barron et al., 1985; Costill et al., 1988; Hellard et al., 2005; Kajiura et al., 1995; Mujika, 1998) and recovery periods.

Studies performed by Lehmann et al (1992 a, b) on experienced middle and long distance runners clearly show that individuals respond very differently to similar increases in training volume and intensity. In this study when the training volume was increased, runners developed signs and symptoms of the overtraining syndrome but when training intensity was increased

with adequate day -to-day variation, the runners were able to improve on their performance without developing any symptoms of overtraining.

The balance between training and overtraining is difficult to define and often coaches and athletes only recognize that there may be a problem when performance starts deteriorating. Compounding the problem is the fact that athletes experience transient fatigue and reduced performance as a normal consequence of training (Kuipers and Keizer, 1988) and this holds especially true for elite athletes who regard a degree of overreaching as a normal consequence of intensified training (Halsen and Jeukendrup, 2004). Hooper et al (1995) makes an important point that athletes are often classified as being stale (overtrained) or not stale (not overtrained) whereas that the whole overtraining syndrome should be seen as a continuum with all athletes falling somewhere on this line.

It is thus important to realize that athletes will experience day-day variations in performance and feelings of fatigue but that this may not necessarily indicate overtraining or overreaching *per se* (Kuipers and Keizer, 1988). If the balance between training and rest is optimal, the athlete should be recovered before the next training session. All too often however, athletes and coaches react to poor performance by increasing the training stimulus even more, pushing the athlete even further into decline (Foster, 1997).

Overtraining describes the process of undergoing training net loads that are in excess of what the athlete is accustomed to. Foster (1997) includes in this description, factors or stressors in the athletes' life, such as travel, occupation and inadequate sleep. Callister et al (1989, pg 816) defines overtraining as "*the process of performing an abnormally large quantity of intense physical training*". This is often accomplished by altering training intensity, frequency and

duration as well as decreasing the rest or recovery periods. This may lead to the development of symptoms of the overreaching or overtraining syndrome.

There is still much controversy as to what defines each of these two syndromes and a recent study by Halson and Jeukendrup (2004) poses the question: Does overtraining exist? They also postulate that it is not presently possible to distinguish between the two states of overreaching and overtraining to the fatigue and lowered performance experienced as a result of normal training. This may be due to a lack of appropriate and valid diagnostic tools (Urhausen and Kindermann, 2002), the great variability in the results of different studies, poorly controlled studies and not least of all the very specific individual reactions of athletes to the training stimulus (Halson and Jeukendrup, 2004).

### 1.3.1 OVERREACHING.

The symptoms of overreaching are a consequence of an intensified training load and are often experienced as normal symptoms in elite and top level athletes (Halson and Jeukendrup, 2004). The symptoms of overreaching are transient in nature and normally resolves within a few days after sufficient rest. Fatigue is one of the main symptoms of overreaching and performance may or may not decline.

Millet et al (2005) investigated the relationship between training loads as measured from exercise heart rate and fatigue and anxiety in four elite triathletes over a 40 week training period. The study found a significant relationship between training load and fatigue ( $r = 0.30$ ;  $P < 0.001$ ) and training load and anxiety ( $r = 0.32$ ;  $P < 0.001$ ). They concluded that tracking these

variables may be helpful in identifying overreaching / overtraining and adjusting tapering periods appropriately.

Halson et al (2002) subjected 8 endurance cyclists to 2 weeks of normal, intensified and recovery training to study the cumulative effect of exercise stress on performance and fatigue. They found that the large decrease in performance was accompanied by a 9 % reduction in maximal heart rate, a 5% decrease in maximal oxygen uptake and a 9 % increase in perception of effort. They concluded that the symptoms of overreaching may occur after only 7 days of intensive exercise with inadequate rest.

### 1.3.2 OVERTRAINING SYNDROME.

Foster (1997, pg 1164) defines the overtraining syndrome as a *“complex condition characterized by a variable group of symptoms and pathophysiologic abnormalities that always include performance incompetence refractory to normal regeneration cycles”*. Hooper et al (1995, pg 106) states that overtraining *“occur in response to large volumes and / or high intensities with inadequate recovery periods between workouts”*.

Overtraining and the overtraining syndrome are detrimental to the athletes' health and capacity to perform. Overtraining presents with a sudden and severe decline in performance and persistent fatigue for prolonged periods (weeks to months) that does not resolve even with rest (Lehmann et al., 1991). In their study, 8 experienced middle and long distance runners were not able to improve or even reach their own previous personal records in the subsequent

season after only a 4 week period of intensified training, aimed at inducing the overtraining syndrome.

Most of the symptoms of the overtraining syndrome are subjective in nature and specific to each individual athlete and are often only diagnosed once performance deteriorates. It is however, still unclear whether overtraining can affect performance prior to the manifestation of symptoms, or in contrast whether symptoms of overtraining may present prior to any performance decrements (Callister et al., 1989). According to Urhausen et al (1995) the evaluation of a performance decrement as a symptom of overtraining is particularly troublesome and requires very specific standardized testing. These tests will be discussed in more detail in section 1.4.

#### *1.3.2.1 PHYSIOLOGICAL FACTORS AND THE OVERTRAINING SYNDROME.*

The physiological factors associated with the development of the overtraining syndrome, are still not well understood, suffice to say that overtraining is associated with changes in the neurological, hormonal and immune systems (Kuipers and Keizer, 1988; Wilmore and Costill, 1994). Some of the symptoms of the overtraining syndrome include loss of weight, increased heart rates at rest and during submaximal exercise, changes in mood, sleep disorders, chronic fatigue (Parker et al., 1996), increased susceptibility to illness (Shephard et al., 1991) and decrements in performance (Callister et al., 1989). Urhausen and Kindermann (2002) also report impaired anaerobic lactacid performance, a decreased time to fatigue, decreased respiratory exchange ratio and changes in some blood markers and hormones as other possible indicators of overtraining / overreaching.

Halson et al (2003) examined the immunological responses to overtraining / overreaching. Eight male endurance cyclists completed 6 weeks of intensified training and all subjects displayed symptoms of overtraining / overreaching. Performance deteriorated and mood changes were observed in all subjects. Plasma cytokines were not related to the decreased performance. However, the glutamine / glutamate ratio decreased after the period of intensified training and may possibly be used as a marker of overtraining / overreaching. The extent to which this ratio predicts or tracks changes in performance associated with overtraining is not known.

Jeukendrup et al (1992) also found significant increases in heart rate during sleep, decreased maximal heart rates and impaired performance in a group of male competitive cyclists who were subjected to a two week period of intensified training that caused all of the subjects to show symptoms of overtraining. After a two week recovery period these variables had all returned to normal but athletes still reported feeling fatigued.

Dressendorfer et al (1985) examined the changes in morning heart rate as a possible indicator of overtraining in a group of 12 healthy marathon runners competing in a 500 km race over a period of 20 days. The runners ran for 10 days, rested for 70 hours and then completed the race over the next 8 days. Runners on average completed 28 km per day which was about twice their normal training distance. Heart rate was measured manually by either counting the radial pulse or by auscultation after the subject had been seated for at least 5 minutes at approximately the same time of day. As was expected, morning heart rate decreased in the first week from an average of 59 beats.min<sup>-1</sup> to an average of 53 beats.min<sup>-1</sup>. From day 8 onwards to the end of the race, morning heart rates started increasing steadily, changing by an average of 10 beats.min<sup>-1</sup> ( $P < 0.01$ ). Changes in heart rate ranged between 1 to 20 beats.min<sup>-1</sup>.

Performance, as measured by daily average running speed, did not change significantly but was on average 15 % slower than their best marathon and training runs. All of the runners complained of feeling chronically fatigued and some runners reported persistent leg muscle soreness. The other variables tested, (blood pressure, oral temperature, body weight, sweat loss, blood glucose, lactate, insulin and cortisol) did not change significantly and were not related to the changes in morning heart rate. The authors concluded that increases in morning heart rate may be a valid sign of overtraining in endurance trained athletes and that an increase of more than 10 beats.min<sup>-1</sup> from baseline may indicate an impending overtraining response (Dressendorfer et al., 1985). This recommendation has not been tested systematically in any subsequent trials.

Uusitalo et al (1998) subjected nine female endurance athletes to a progressive regime of exercise training over a period of 6 – 9 weeks with the aim of inducing a state of overtraining. Five subjects did in fact display some symptoms of the overtraining syndrome and also had a decreased VO<sub>2</sub>max ( $P < 0.01$ ), but no changes were observed in resting heart rate, intrinsic heart rate and cardiac autonomic modulation using pharmacological blockade.

In a study by Callister et al (1989) 15 national and international elite judo athletes were subjected to 10 weeks of progressively increased training volume. Analysis of the training intensity and volume indicated that these athletes were in fact subjected to high volumes of training. Muscle strength performance, as measured by concentric isokinetic testing, showed some improvement during phase I (regular training), no improvement during phase II (increased resistance and interval training) and a decrease during phase III (highest training volume). However, peak and submaximal VO<sub>2</sub>, submaximal, maximal and resting heart rate and body mass did not change. They concluded that even though the athletes did not in fact

present with the symptoms of overtraining, performance was adversely affected. The study surmises that the fact that the athletes were all elite may in part have protected them from developing the overtraining syndrome as they are accustomed to heavy workloads and their ability to tolerate these heavy workloads for limited periods of time may in part be responsible for them becoming top athletes in the first place.

Kuipers and Keizer (1988) speculate that athletes adapted to endurance exercise may display different overtraining responses compared to resistance trained athletes. As such they concluded that non-endurance athletes would present with sympathetic type responses such as increased resting heart rate and blood pressure whilst endurance trained athletes would display more parasympathetic type symptoms such as decreased resting heart rates and deteriorating performances. However, other studies (Barron et al., 1985; Dressendorfer et al., 1985) of overtrained endurance athletes have also identified sympathetic nervous system type responses in these athletes. Israel (1958, 1967 and 1976) distinguishes between Addisonoid (parasympathetic) and Basedowian (sympathetic) overtraining. The two types of overtraining are characterized by either inhibition of excitement. This controversy has thus not been resolved as yet and further studies are needed to clarify this dichotomy.

According to Hooper et al (1995) the process or recovery of athletes from fatigue is not well researched although the technique of tapering is employed by most athletes and coaches in an attempt to recover. It is not known if the tapering period is in fact adequate for sufficient recovery from overtraining and the markers for monitoring the athlete during the recovery period is also not well researched. In an attempt to clarify the recovery period, 14 elite swimmers were studied over a 6 month period during the training, tapering and competition phases (Hooper et al., 1995). The study concluded that the tapering period (2 – 3 weeks) was

in fact not adequate to allow the overreached athletes to recover sufficiently as muscle soreness and fatigue rating remained significantly higher for the overreached athletes in the week prior to taper and after the competition. Their swimming performance was also adversely affected. Similarly, in another study, two weeks of recovery after a period of 2 weeks of intensified training was also not sufficient for athletes to recover, as athletes remained fatigued. This occurred despite the return to normal of physiological variables (Jeukendrup et al., 1992).

In summary, each individual athlete responds differently to the same training stimulus, making it difficult to reach general guidelines about training and overtraining. Furthermore, the physiological factors associated with the overtraining syndrome are poorly understood. A consensus on the definition of what constitutes overtraining and overreaching needs to be reached to ensure consistency in diagnosis as a lack of appropriate diagnostic tools, variability in study results and poorly controlled studies confounds the recognition and acceptance of the overtraining / overreaching syndrome.

However, it may be concluded that two major symptoms of overtraining are fatigue and a decrease in performance. Therefore, it is logical to assume that if these variables are measured frequently, symptoms of overreaching or impending overtraining syndrome may be identified and appropriate strategies adopted. This will be discussed in more detail.

#### **1.4 METHODS OF MEASURING TRAINING STATUS.**

A variety of methods are commonly used by athletes and coaches to measure performance and training status. Measuring performance and training status regularly, either in the field or

laboratory setting, allows the coach to evaluate training programs and the athletes' progress toward a predetermined goal as well as the presence of negative factors that may lead to symptoms of overreaching or overtraining.

However, the relationship between performance during physiological testing and during actual competitive events has not been adequately researched (Hopkins et al., 1999). After reviewing the available literature they concluded that the data presented after physiological testing may not necessarily apply to top level athletes under competitive conditions. Factors that may affect the reliability and validity of the data are: too small sample sizes; tests not valid; athletes not of high enough standard and their behaviors may not be representative of normal training and competition conditions (Hopkins et al., 1999). They concluded that physiological tests should only be used if a test is valid and more reliable than the actual competition or event and the scientist is sure that the test in question produces similar improvements compared to the actual event. To ensure this validity, the within-subject variation in performance should be a key factor when estimating an appropriate sample size. Furthermore, the estimation of performance improvement should preferably be done during the actual event and training and dietary habits should be replicated for the studies (Hopkins et al., 1999). This however is difficult to accomplish as elite athletes normally follow a strict regimented and periodised training program that makes them reluctant to participate in studies that may affect their preparation and they are even more loath to allow interference during a competition that they perceive will affect their performance and medal winning opportunity.

As a result of these practical difficulties, a variety of different methods are used as indirect markers to test training status. These tests may be blood borne markers (biochemical and

hormonal), physiological ( $\text{VO}_2\text{max}$ , heart rate) or psychological (POMS and RPE). These will be discussed in more detail in the next section.

#### 1.4.1 BLOOD BORNE MARKERS.

##### *1.4.1.1 BIOCHEMICAL.*

The changes in certain biochemical markers are often used to determine the training status of athletes and as a diagnostic tool to detect overtraining/overreaching (Urhausen et al., 1995). Rowbottom et al (1995) concluded that the interpretation of blood markers as markers of training status should be treated with care as endurance training especially causes large expansion of blood plasma volume and unless this change is taken into account, incorrect assumptions may be made.

##### *1.4.1.1.1 Blood lactate.*

Lactate as a consequence of oxygen-independent metabolism of glycogen through the glycolytic pathway (Swart and Jennings, 2004) and blood lactate concentration is frequently used as a marker of training status and exercise intensity (Billat, 1996). The normal value of blood lactate for a healthy adult at rest is  $< 1.5 \text{ mmol.l}^{-1}$ . Blood lactate testing used to be impractical for the majority of athletes and coaches, as they needed to have access to a laboratory. However, technical advances have made available small compact instruments that are able to analyze blood obtained from a finger prick within a few minutes (Hopkins, 1991).

These portable lactate analyzers show fairly good reliability but inaccuracies of up to 7 % have been reported with portable monitors differing from laboratory testing by approximately 1 – 2 mmol.l<sup>-1</sup> (Swart and Jennings, 2004).

The relationship between steady state lactate values and work load during exercise is curvilinear (Dennis et al., 1992) and reproducible (Hopkins, 1991), providing various factors are controlled (Swart and Jennings, 2004). The lactate or anaerobic threshold, as first postulated by Hill et al (1923), is seen by some physiologists as a good indicator of an athletes' potential for endurance training although the concept of an anaerobic threshold has recently been challenged (Dennis et al., 1992; Noakes, 1998).

The lactate threshold is defined as the point at which lactate starts to rise above the baseline level when exercising and is arbitrarily set at 2 to 4 mmol.l<sup>-1</sup> (Kindermann et al., 1979; Wilmore and Costill, 1994), though Billat (1996) have shown a large variation among different athletes with values ranging anywhere between 2 – 8 mmol.l<sup>-1</sup>. According to Wilmore and Costill (1994) the lactate threshold, when expressed as a percentage of VO<sub>2</sub>max, is one of the best indicators of an athletes' pace in endurance type activities. It has been postulated that training at, and above, the lactate threshold will consequently lower the level of lactate in the blood at both maximal and submaximal levels (Weltman et al., 1992). These theories have led to lactate regularly being used to assess the training status and exercise intensities of athletes. Blood lactate concentration will only rise above baseline level when the exercise intensity is above 60% of maximum and does not reach a steady state prior to the athlete being exhausted and as such blood lactate levels are not accurate measures of exercise intensity above the anaerobic threshold (Hopkins, 1991). The lactate threshold in untrained athletes occurs at 50 to

60 % of  $VO_{2max}$  , with endurance trained top level athletes only reaching this threshold at 70 or 80%  $VO_{2max}$  (Fay et al., 1989; Wilmore and Costill, 1994).

A study by Pyne et al (2001) examined the changes in performance in eight male and four female national level swimmers over an 8-month period leading up to the Commonwealth Games, to determine if lactate threshold would be sufficiently sensitive to detect small changes in endurance fitness. Lactate threshold was determined by calculating the threshold as a function of the slope and y-intercept of the lactate-velocity curve. The swimmers completed 7 x 200-m incremental swimming step tests on four occasions during the 8-month period. Results indicated that 200-m maximal effort time, lactate tolerance and swimming velocity at lactate threshold all improved with changes in training status, but was not directly related to performance during competition.

The blood lactate concentration / heart rate relationship was further examined in a study by Verges et al (2003), during which ten national level male cross country skiers were tested during a maximal laboratory treadmill running test and a field roller skiing test. The field test consisted of four loops (4 km long) at different exercise intensities, from warm-up to maximal aerobic speed. Results showed that lactate values during the field test were significantly higher ( $P < 0.05$ ) than the values measured during the laboratory test, at a similar heart rate. They conceded that the different modes used in the testing may have contributed to the difference in the results and again emphasized the importance of exercise specificity when testing and interpreting results.

In a study conducted by Pelayo et al (1996), six elite swimmers were studied over a 23 week period. The first 10 weeks consisted of mostly endurance training with the last thirteen weeks

mainly high intensity training. Subjects performed several maximal effort lactic tests at regular intervals. Lactate recovery values showed an increase during the first ten weeks of endurance training and decreased significantly during the last high intensity training period leading them to conclude that lactate recovery values are useful as practical indicators for the two different types of training.

Jeukendrup et al (1992) examined the response of seven male competitive cyclists after a period of two weeks of intensified training with the aim of inducing a short-term state of overtraining. Maximal and submaximal (at 200, 250 and 300 Watt) lactate values were significantly lower during the ergometer test after the period of intensified training. This pattern of lower maximal and submaximal levels after intensified training have been confirmed by several other studies (Foster et al., 1988; Lehmann et al., 1991; Lehmann et al., 1992 a b).

There are also several factors that may affect the accuracy and interpretation of steady state blood lactate concentration such as; i) Rate of change in exercise intensity, ii) Carbohydrate depletion, iii) Mode of exercise, iv) Precision of monitoring blood lactate concentration, v) Ambient temperature, vi) Overtraining syndrome, and vii) Muscle damage (Swart and Jennings, 2004).

In conclusion, much controversy remains regarding the exact mechanism of lactate production and utilization and this makes it difficult to use as a marker of training status. The definition of the lactate threshold needs to be clarified with standardized criteria. Furthermore, the various factors that may affect the blood lactate concentration during exercise need to be considered when blood lactate concentrations are analyzed and interpreted.

#### 1.4.1.1.2 Other selected biochemical markers.

##### 1.4.1.1.2.1 Hemoglobin and Hematocrit

Hemoglobin and hematocrit values are often measured, especially in endurance trained athletes to assess training status and as a diagnostic marker to explain fatigue associated with training. Endurance performance is dependent on the ability of the red blood cells to transport sufficient oxygen to the working muscles (Calbet et al., 2006). Endurance training increases blood plasma volume (Rowbottom et al., 1995) and red cell mass whilst decreasing hemoglobin and hematocrit values, primarily due to the increased plasma volume (Schumacher et al., 2002).

Hematological parameters (hemoglobin, hematocrit, ferritin and transferrin) were measured by Banfi et al (2006) in a group of elite rugby players over a competitive season. Even though all of the parameters were well within the normal physiological range over the whole season, there was some variability, mostly related to training and competition workloads. Hemoglobin and hematocrit levels were higher at the beginning of the season and decreased in the latter half of the season when the physical training load was highest. Ferritin levels increased towards the end of the season.

A study by Tokudome et al (2004) found lower levels of hemoglobin, ferritin and white blood cell counts in a group of ultra-marathon runners. Some of these runners displayed symptoms of the overreaching / overtraining syndrome and were running in excess of 300 kilometers per month. Low levels of hemoglobin and hematocrit levels have been associated with overreaching and overtraining (Lehmann et al., 1991 and 1993). A study by Rietjens et al

(2002) on elite Olympic distance triathletes over a period of 3 years found that 46 % of the athletes presented with hematological values below the normal range during the off-season. This percentage increased from 55 % of athletes during the training season to 72 % during the race season. These elite athletes were thus constantly training and performing under the lower limits of normal hematological values. Hemoglobin and ferritin levels were most frequently below the normal values and only the red blood cell count decreased significantly ( $P < 0.05$ ) between the race and training season.

A study by Schumacher et al (2002) looked at the characteristics of the red blood system and iron metabolism in athletes in different sporting disciplines and at different performance levels. They found lower levels of hemoglobin, hematocrit and red blood cells in the endurance trained athletes compared to the power and mix-trained athletes with no difference found in hemoglobin and hematocrit levels between the athletes and controls. They concluded that the specific type and duration of activity is the main cause of adaptation in the blood cell system and iron metabolism. This was attributed mainly to plasma volume expansion in all the groups and to a measure of hemolysis in the runners.

In summary, although hemoglobin and hematocrit values change with changes in training status, it is unlikely that they can be used with any accuracy to predict or track changes in training status, as values may still fall within normal physiological ranges. The role of plasma volume expansion and its effect on hematological parameters still need further clarification.

#### 1.4.1.1.2.2 *Blood enzyme activity*

The measurement of blood enzyme activity has a limited value in measuring training status, especially that of the overtraining syndrome (Gleeson, 2002; Wilmore and Costill (1994). The enzymes often measured as markers are CPK (creatine phosphokinase), LDH (lactate dehydrogenase) and SGOT (serum glutamic oxalic transaminase), all of which are associated with metabolic pathways and are important in the production of muscle energy. High levels of these enzymes in the blood suggest changes in cell membrane permeability and may be increased 2 to 10 times above normal after periods of intensified training (Wilmore and Costill, 1994).

#### 1.4.1.1.2.3 *Urea and metabolites*

High levels of blood urea may indicate an increased rate of protein catabolism and could account for the weight loss experienced by overtrained athletes (Kindermann, 1986; Wilmore and Costill, 1994). A study by Lehmann et al (1991) however, found levels of urea, creatinine and uric acid, unchanged in a group of eight well trained runners who were subjected to increased training volume with the aim of inducing the overtraining syndrome.

Hartmann and Mester (2000) examined the serum urea concentration and serum creatine kinase activity in 6,981 international level athletes during training in an attempt to identify if these markers were potentially useful in identifying the overtraining syndrome. Values for both these markers were consistently lower in the women. Both the markers exhibited extremely

high individual variability. They concluded that if these markers were to be used, measurements should be done every 3<sup>rd</sup> day under standardized conditions.

Urhausen et al (1998) studied the effect of increased training load on the pituitary hormonal response in a group of 17 male endurance athletes. Symptoms of the overtraining syndrome were induced in fifteen of the athletes, primarily by increasing the frequency of high-intensity exercise bouts without increasing the total amount of training. Performance, as measured by time to exhaustion during the cycle ergometer test, decreased significantly by 27 % on average. Serum urea and uric acid however, was unchanged in both the normal and overtrained states.

Filaire et al (2004) conducted a study on 12 national level cyclists over an 8 month period. Urinary levels of methoxyamines (metanephrine, normetanephrine) and 3-methoxy-4-hydroxyphenylglycol sulphate (MHPG-S) was measured after a period of unspecified training, 48 hours prior to the start of 4 days of intensified training, at the end of the increased 4-day training (+ 187 %) and again after 4 months of training. Salivary levels of cortisol and testosterone were also measured. The Profile of Moods (POMS) questionnaire was also recorded at the same time periods to examine if mood changes followed the pattern of hormonal changes and to identify symptoms of overtraining. Levels of normetanephrine were significantly increased ( $P < 0.05$ ), with the testosterone - cortisol ratio decreased ( $P < 0.05$ ) and no change noted in the MHPG-S levels after the 4 day intensified training period. After 8 months of training there were significant changes noted in the levels of metanephrine and MHPG-S ( $P < 0.05$ ), although mood as measured by the POMS remained unchanged through all of the different periods, leading them to conclude that there may be a dissociation between the neural and endocrine catecholaminergic systems.

In conclusion, the increase in blood plasma volume after training is a major confounding factor when interpreting changes in blood markers and may lead to incorrect assumptions being made regarding an athletes' training status. It is also difficult to ascertain whether changes in blood markers are due to the normal adaptation process after training or due to negative changes in training status such as the overtraining syndrome. Results from studies are currently still too variable and inconsistent to be seen as accurate markers of changes in training status. Furthermore, the cost and expertise required to measure enzymes and other blood and biochemical markers may not be of practical use for the measurement of training status in athletes, especially in the field testing situation. The interpretation of results may also not be within the scope of most coaches and therefore the measurement of markers in the blood has limited practical value in monitoring training status.

#### *1.4.1.1.2.4 Immunological markers*

Athletes who display symptoms of the overtraining syndrome often also present with a compromised immune system (Gleeson, 2002). As several of the markers of a suppressed immune system are also affected by both acute and chronic exercise stress, it has been suggested that these markers be used to identify possible symptoms of the overtraining syndrome (Gleeson, 2002).

Reasons for the lowered immune status of athletes undergoing heavy training loads are still unclear and several reasons have been put forward to explain this phenomenon. Khansari et al (1990) suggested that the cumulative effect of repeated bouts of exercise may cause the immunosuppressive reaction, specifically due to the increased levels of stress hormones such

as the glucocorticoids. Other possible reasons cited are changes in plasma glutamine levels (Parry-Billings et al., 1992) and lowered levels of serum complement concentration levels (Mackinnon, 1998). Robson et al (1999) suggested that repeated bouts of exercise over extended periods of time, combined with the adverse effect of acute exercise sessions on the neutrophil count may cause athletes to become more vulnerable to infection.

Smith (2004) studied the effect of repeated tissue trauma as a possible cause for the development of the overtraining syndrome. This study proposed the mechanism that chronic injuries due to excessive heavy training loads may cause the up-regulation of the humeral arm of the adaptive immune system (IL-4, IL-6 and IL-10), with a corresponding down regulation of the cell-mediated arm (IL-12) and thus make the athlete more vulnerable to opportunistic infections.

Problems associated with using immunological markers as indicators of imminent overtraining are the fact that it is expensive to do and may only measure one aspect of a multi-dimensional system (Gleeson, 2002). Another problem may be the fact that these markers normally change in response to an infection and it may be difficult to conclusively identify the origin of the immunosuppression in athletes so affected (Gleeson, 2002).

#### 1.4.1.2 HORMONAL.

Endogenous hormones are directly involved in exercise –induced adaptations and also play a part in the recovery process through the regulation of anabolic and catabolic processes after

exercise (Urhausen et al., 1995). It is surmised that changes in these hormones also seem to be partly involved in the origin of the overtraining syndrome (Fry et al., 1991; Meeusen et al., 2004; Steinacker et al., 2004; Steinacker et al., 2005; Urhausen and Kindermann, 1994).

The endocrine system has an important role in maintaining homeostasis in the body. An exercise session normally disrupts the homeostasis and causes several systems, including the endocrine system, to react and thus adapt to the specific stimulus (Virta, 1985). Various blood hormones may change in response to the exercise stress. For example, blood concentrations of thyroxine, testosterone, cortisol, urea, epinephrine and norepinephrine are routinely measured as changes may either indicate adaptation or overreaching / overtraining (Meeusen et al., 2004; Steinacker et al., 2004; Steinacker et al., 2005; Urhausen et al., 1995).

#### 1.4.1.2.1 Epinephrine and Norepinephrine.

The measurement of free plasma catecholamines, namely epinephrine and norepinephrine are often used to evaluate the sympatho-adrenergic system during and after exercise (Kaji et al., 1989). High levels of epinephrine and norepinephrine at rest have been measured in athletes undergoing heavy training (Kirwan et al., 1988). These two hormones may affect physiological variables such as heart rate and blood pressure, causing both to increase. The measurement of these hormonal levels may however be affected by the individual variation encountered, plasma volume and duration of the exercise (Péronnet et al., 1986).

Levels of catecholamines are also affected by physiological stress in response to exercise and competition. A study by Weiler et al (1994) found similar levels of epinephrine and

norepinephrine during competition and high intensity training in a group of badminton players. At the same time however, the lactate levels were approximately three times lower during the competition compared to the high intensity training condition.

Several factors may affect the results when free plasma catecholamines are measured and these should be kept in mind. There may be intra-individual variability when using different assays (Péronnet et al., 1986), and the half-life of catecholamines in free plasma is only about 2 minutes, so blood needs to be sampled quickly after exercise (Urhausen et al., 1995). Compared to venous blood samples, capillary blood samples may be more practical, however, catecholamines measured in venous blood samples can be measured with more precision and therefore it remains the sampling method of choice.

#### 1.4.1.2.2 Testosterone and Cortisol.

Changes in plasma testosterone and cortisol concentrations depend on the intensity and duration of the exercise bout (Busso et al., 1992). The specific type (mode) of exercise may also affect testosterone and cortisol levels in athletes (Izquierdo et al., 2004; Tremblay et al., 2004).

Izquierdo et al (2004) found that the basal serum total testosterone and free testosterone concentrations were lower in elite amateur cyclists compared to age-matched weightlifters and untrained subjects. Tremblay et al (2004) also examined the hormonal response (androgens and cortisol) in endurance and resistance trained subjects of different training status, subjected to exercise training of similar volume. Results indicated that endurance-trained subjects

presented with smaller changes in hormone concentrations during exercise compared to the resistance trained subjects. They concluded that the mode of exercise and intensity is more pertinent in the hormone profile changes seen in male athletes compared to exercise volume as measured by caloric expenditure. Similarly, Paccotti et al (2005) deduced that changes in cortisol are more likely affected by training status and mode of exercise. Results from a study by Tremblay et al (2005) indicated that the duration of exercise has an independent effect on hormonal changes in response to endurance training.

Kirwan et al (1988) found serum cortisol increased in response to athletes increasing their training volume 2-fold. Cortisol levels were  $18 \pm 2$  and  $21 \pm 1$  milligrams.dl<sup>-1</sup> pre-exercise and at the end of the intensified training period on day 11 respectively. However, in another study (Kirwan et al., 1990) decreased (-10%) serum cortisol levels were found after a five day period of intensified training in both dietary conditions, when carbohydrate intake was controlled (either EQ-CHO; 8 g.kg<sup>-1</sup>.d<sup>-1</sup> or LO-CHO; 4g.kg<sup>-1</sup>.d<sup>-1</sup>). Both testosterone and cortisol are important hormones involved in protein and carbohydrate metabolism and compete with each other at the muscle cell receptor level (Urhausen et al., 1995). A study by Mujika et al (1996 a, b) found blood levels of cortisol and total testosterone unchanged during 12 weeks of training and 4 weeks of tapering in 8 well trained competitive swimmers. However, changes in performance during both the training and tapering periods were positively correlated with changes in the total testosterone : cortisol ratio ( $r = 0.86$ ,  $P < 0.01$  and  $r = 0.81$ ,  $P < 0.05$  respectively).

The ratio of testosterone to cortisol has been studied as an indicator of a change in training status or even a probable cause of the overtraining syndrome as its function is the regulation of the anabolic processes during recovery (Kuipers and Keizer, 1988). Filaire et al (2004) found a

significantly ( $P < 0.05$ ) reduced testosterone-cortisol ratio after a four day period of intensified training in a group of national cyclists. Urhausen et al (1995) however suggest that the testosterone / cortisol ratio is an indicator of training strain rather than the overtraining syndrome as there were essentially no significant changes in testosterone and cortisol levels detected at rest in numerous studies on overtraining (Fry et al., 1992; Kirwan et al., 1988; Urhausen and Kindermann, 1994) and no direct link with performance.

#### 1.4.1.2.3 Pituitary Hormones.

The hypothalamus integrates various signals (metabolic, hormonal, sensory afferents and central stimuli) and because of this function, the status of the pituitary hormones, are representative of the functional status of the athlete (Steinacker et al., 2004 and 2005).

Pituitary hormones that may be affected by exercise are Luteinising Hormone (LH) and Follicle-Stimulating Hormone (FSH) (Urhausen et al., 1995). Barron et al (1985) found decreased levels of Luteinising Hormone and an unchanged response of LH to the Gonadotrophic Releasing Hormones (GRH) after acute physical exercise. They also found a reduced increase in corticotrophin (ACTH), growth hormone and cortisol after an insulin-induced hypoglycaemia in 4 overtrained marathon runners. At the same time the pituitary hormones responded adequately after the LH-releasing hormone (LHRH) was administered. This led them to conclude that there was a hypothalamic dysfunction present in the sense of a central depression in the overtraining syndrome. The Follicle-Stimulating Hormone does not change significantly following exercise (Urhausen et al., 1995). Corticotrophin secretion is also exercise

dependent particularly with regard to the duration and intensity of the exercise bout and may be affected by low blood glucose levels (Heitkamp et al., 1993).

Several studies (Elias and Wilson, 1993; MacIntyre, 1987; Sutton and Lazarus, 1976) found the levels of hypothalamic hormones such as growth hormone-releasing and inhibiting hormone to be influenced by various factors such as the neurotransmitters (catecholamines), hormones (adrenal), amino acids, exercise, stress, sleep and low blood sugar levels.

#### 1.4.1.2.4 Other hormones: Insulin and Thyroid Stimulating Hormone (TSH),

Insulin (Urhausen and Kindermann, 1994) and Thyroid-Stimulating Hormones (Lehmann et al., 1992 a, b) have also been measured in athletes after training and with reference to the overtraining syndrome but thus far results are inconclusive and more studies are needed.

Again, as with other hematological parameters, there is a problem as changes due to normal adaptation in response to a training stimulus may be indistinguishable to the changes occurring due to maladaptation (overtraining syndrome). Most of the hormones either directly affect or are affected by changes in other hormones and this interplay make it an extremely complex system to study and difficult to identify cause and effect. High intra- and inter-assay differences that occur with laboratory testing also complicates interpretation of hormonal changes as does the fact that hormonal changes are reliant on circadian and / or pulsatile rhythms and may also be affected by psychological factors (Urhausen et al., 1995).

In summary, while various hormones do change in response to changes in training status, the measurement of hormones as a marker of training status is impractical in the field situation. This is in part due to the variable half-life of different hormones, the expense and the lack of precision in the relationship between hormone concentration and training status.

#### 1.4.2 PSYCHOLOGICAL MARKERS.

Scientists are increasingly recognizing the importance of psychological testing when assessing performance. Some of the signs and symptoms of overreaching and overtraining may present as altered mood states, such as depression, increased anxiety and anger. Morgan et al (1987) found that physically active subjects and elite athletes tended to present with better mood states than the population in general. However, several studies (Morgan et al., 1988; Raglin et al., 1991), found altered mood states after periods of intense training, even in well trained endurance athletes.

A recent study by Millet et al (2005) examined the relationship between training, anxiety and fatigue in a group of elite athletes over a 40 week period. They found a significant relationship ( $P < 0.001$ ) between training load and anxiety as well as a significant relationship ( $P < 0.001$ ) between training load and perceived fatigue. Monitoring these factors may well aid in the early detection of overreaching (staleness).

#### 1.4.2.1 PROFILE OF MOODS QUESTIONNAIRE (POMS).

The Profile of Mood State (POMS) questionnaire was designed by McNair (1971) and is often used when testing athletes. The questionnaire consists of 65 items which are answered by the athlete and provides a global measure of mood and scores for tension, anger, depression, fatigue, confusion and the only positive mood state, vigor.

In a study by Verde et al (1992) ten highly trained runners increased their training load by an average of 38 % over a three week period. Results indicated a significant increase in global disturbance and fatigue ( $P = 0.03$ ) and a significant decrease in vigor ( $P = 0.02$ ). During the subsequent recovery period most of the negative mood scores returned to normal, however, the only positive mood scale, vigor, stayed at below the initial values tested.

A recent study by Rietjens et al (2005) used of a shortened version (10 items) of the POMS questionnaire on a group of well trained male cyclists during a two week period of intensified training. Five of the subjects presented with continuous increased mood and fatigue ratings, but these were not significant, and in contrast to the study by Millet et al (2005), no relationship was found with training load. They concluded that the shortened POMS may not be sufficiently accurate to detect early signs of overreaching.

In conclusion, the POMS questionnaire is useful in identifying changes in mood states but there is still no consensus as to what changes are required to diagnose an athlete as overtrained. It may also not be practical to expect athletes to complete an extensive questionnaire on a daily basis as would be required if this was going to be used to monitor mood, and by implication, training status. At this stage the shortened POMS as used by Rietjens et al (2005) may not be useful in the early stages of overreaching. There still remains uncertainty regarding the

relationship between training load and changes in the POMS questionnaire and this needs to be clarified in future studies.

#### *1.4.2.2. RATING OF PERCEIVED EXERTION (RPE).*

The concept of measuring subjective feelings of exertion using a rating scale was first introduced by Borg in 1961 (Borg, 1961 and 1973). The idea that this scale could be used to prescribe exercise intensity was seen as a potentially interesting application (Borg and Linderholm, 1970) and the 15 point Borg scale was developed (Borg et al., 1987).

The rating of perceived exertion (RPE) scale has been widely used to determine the subjective feeling of "strain" during exercise (Dunbar et al., 1992).

Stephenson et al (1982) examined six female athletes on five different days of the menstrual cycle over a period of a month. Subjects cycled on a bicycle ergometer at a maximum workload of 184 W and at four submaximal exercise intensities (45, 83, 121 and 154 W). They found that there was a linear relationship between RPE and heart rate ( $r = 0.87$ ) at all exercise intensities and regardless of the day during the menstrual cycle. A later study by Dunbar et al (1992) found the RPE scale valid when regulating exercise intensity using a graded exercise test on a cycle ergometer and treadmill.

Carton and Rhodes (1985) examined the then available literature on the RPE scale. They found that there was a wide variability in the findings in the literature and concluded that inter-subject variability, type of exercise and nutritional status of the subject may influence

performance variations. Trained athletes also tended to report lower RPE values than untrained subjects.

Koltyn and Morgan (1992) examined the effectiveness of using RPE and heart rate to measure exercise intensity in two groups of 38 subjects each over a 14 week period. Training was similar for both groups. Both groups improved their cardiovascular endurance and performance as measured by the distance they were able to run in 15 minutes. However, the group using heart rate as a measure of training intensity only improved by an average of 6 % whilst the RPE group improved by 11 %.

The relationship between the perception of effort during exercise and fatigue still needs to be clarified however, and no study thus far has been able to identify a single factor that could consistently explain exertion ratings. It is surmised that a variety of perceptual clues are needed to integrate multiple afferent signals to initiate a sense of perception of effort. Hampson et al (2001) propose that a mechanism exists to channel afferent signals to allow exercise performance to continue within the biomechanical and metabolic limitations of the body through a process called teloanticipation. They postulate that there are various mechanisms at play that indicates that some sort of regulatory mechanism exist, such as the decreased muscle recruitment that occurs just before the onset of fatigue, the accuracy with which subjects can use RPE to regulate exercise intensity and the fact that hypnosis and biofeedback training can change perceived exertion and heart rate. This is a new field of study using RPE and more research is needed.

A more recent development in the use of the RPE scale is that of Foster et al (1995), where the original Borg scale has been slightly modified in order to better quantify training load and

athletic performance. This in response to the fact that they were able to demonstrate that this exercise rating system (session RPE) corresponds closely to the average heart rate reserve and to the percentage of the training session during which heart rate and blood lactate values are closely related (Foster et al., 1995). Subjects are asked to give a global intensity rating or overall perception of effort for an entire exercise session as if responding to a simple question by an interested mother, "*How was your workout, honey ?*". This session RPE is then multiplied by the duration of the exercise session (minutes), and once added together at the end of a week, provides the total training load (units / week) for that week.

Day et al (2004) studied the reliability of the session RPE in quantifying exercise intensity in a group of nine male and 10 female resistance trained athletes. Exercise intensity was divided into a high-intensity (90 % of 1 RM), moderate (70 % of 1 RM) and light-intensity (50 % of 1 RM) exercise session. Each athlete completed each exercise intensity twice and the protocol consisted of five exercises (back squat, bench press, overhead press, bicep curl and tricep pushdown). RPE was measured following the completion of each set and the session RPE was measured 30 minutes after the end of the exercise. As was expected, the session RPE was higher for the high-intensity exercise session compared to the moderate and low-intensity sessions ( $P < \text{or} = 0.05$ ). Subjects also found fewer repetitions and higher intensity training more stressful than more repetitions at a lower intensity. The intraclass correlation coefficient for the session RPE was  $r = 0.88$ . They concluded that the session RPE is a reliable indicator of exercise intensity during resistance training. Similarly, a study by Sweet et al (2004) found that the session RPE increased as the exercise intensity increased in a group of resistance trained athletes, even when the total workload decreased ( $P < 0.05$ ). They concluded that session RPE is a viable method for quantifying training load and training intensity.

Impellizzeri et al (2004) used the session RPE to quantify training load based on heart rate response to exercise in a group of nineteen soccer players. The players were tested using an incremental treadmill test before and after a seven week training period. The training load was quantified using the method as described in Foster et al (1995). The session RPE scores were correlated with training load by using heart rate measures. Correlations between the various heart rate based training load measures and session RPE were significant ( $r = 0.50$  to  $r = 0.85$ ;  $P < 0.01$ ). This led them to conclude that session RPE is potentially a good indicator of training load in soccer players.

In summary, the RPE scale is a valuable adjunct to measuring training intensity and by implication training status, but the high inter-subject variability and other potentially confounding variables such as type of exercise and nutritional status should be kept in mind. Session RPE seems to be a reliable measurement of exercise intensity under varying conditions. An advantage of both RPE and session RPE is that they are easy and practical to administer and inexpensive.

### 1.4.3 PHYSIOLOGICAL MARKERS.

#### *1.4.3.1 MAXIMAL OXYGEN UPTAKE.*

Maximal oxygen uptake ( $VO_2\text{max}$ ) is defined as the highest rate of oxygen consumption reached during maximal exercise (Wilmore and Costill, 1994). The relationship between oxygen consumption and power output is also fairly linear over a range of intensities from rest to maximum steady state (Åstrand and Rodahl, 1986). In general, oxygen consumption is a good

measurement of training intensity and Daniels et al (1984) found a coefficient of variation of only 3 % in a group of trained runners over a 6-month period, running on a treadmill at a set speed. It is generally accepted that  $\text{VO}_2\text{max}$  is a good indicator of aerobic or endurance performance.  $\text{VO}_2\text{max}$  is largely genetically determined (Bouchard and Rankinen, 2001) but significant changes can be induced by an appropriate training stimulus.

Pollock and Wilmore (1990) found an average increase in  $\text{VO}_2\text{max}$  of 20% after untrained subjects trained for 6 months. This magnitude of change was also previously demonstrated in a study by Hickson et al (1981). However, it is also acknowledged that  $\text{VO}_2\text{max}$  has some restrictions when used as a measure of endurance performance. Well trained endurance athletes specifically may still be able to improve on their performance despite no further improvements in  $\text{VO}_2\text{max}$  and continued training (Arts and Kuipers, 1994; Åstrand and Rodahl, 1986). A further aspect of concern is the fact that there is a large inter-variability in  $\text{VO}_2\text{max}$  between athletes of similar training status (Arts and Kuipers, 1994; Daniels et al., 1978). Kuipers et al (1985) also found that  $\text{VO}_2\text{max}$  shows a much larger day-to-day variation than maximal power output measures ( $W_{\text{max}}$ ). According to Bassett and Howley (1997) there are several factors limiting  $\text{VO}_2\text{max}$ , namely i)  $\text{O}_2$  pulmonary diffusion capacity; ii) maximal cardiac output; iii) peripheral circulation; and iv) metabolic capacity of skeletal muscle.

Vella and Robergs (2005) studied the relationship between cardiovascular variables such as cardiac output, stroke volume and heart rate and oxygen uptake during a continuous incremental cycle test to exhaustion in twenty moderately to highly trained subjects. Results indicated a significant ( $P < 0.01$ ) non-linear relationship between all of the cardiac variables measured and  $\text{VO}_2$ . They speculate that in some athletes  $\text{VO}_2\text{max}$  may be limited by cardiac output as evidenced by the plateau at  $\text{VO}_2\text{max}$  displayed by three of the subjects and that

peripheral factors may also account for a limited  $\text{VO}_2\text{max}$  in some subjects. These factors all point to the cardiovascular system and thus oxygen delivery as being the prime factors in limiting  $\text{VO}_2\text{max}$ . However, in a study by Doherty et al (2003), a  $\text{VO}_2\text{max}$  plateau was only identified in 39 % of elite males and 25 % of elite females during a graded treadmill test to exhaustion. The three criteria used to determine maximal effort were i) a plateau in the  $\text{VO}_2$ , defined as an increase of less than  $1.5 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ; ii) a final RER of 1.1 or higher; and iii) a final heart rate above 95 % of the age related maximum.

Noakes (1988, 1998 and 2006) however, has challenged this conventional belief that the rate of oxygen delivery was the crucial issue in the developing of a  $\text{VO}_2\text{max}$ . He instead suggests that athletes with a higher  $\text{VO}_2\text{max}$  have muscles with superior contractile ability which enables them to tolerate and achieve higher workloads, thus utilizing more oxygen at maximal workloads. A recent study by Noakes et al (2001), postulates that the brain acts as a "central governor", regulating skeletal muscle recruitment during exercise as a protective measure to prevent myocardial ischaemia and so determining performance.

In summary, although  $\text{VO}_2\text{max}$  may be a useful indicator of cardiovascular fitness, it does not seem to be useful for identifying imminent signs of overtraining, as the measurement of true  $\text{VO}_2\text{max}$  is complex, expensive, exhaustive and not readily accessible to all athletes and coaches in the field situation.

### **1.4.3.2 HEART RATE.**

#### 1.4.3.2.1 Introduction.

There is an accepted linear relationship between heart rate / workload and oxygen consumption (Arts and Kuipers, 1994). This relationship makes it possible to use heart rate to predict training intensity. With the advent of portable heart rate monitors it has become easy to monitor heart rate accurately and under a variety of conditions (Léger and Thivierge, 1988; Seaward et al., 1990).

#### 1.4.3.2.2 Measuring heart rate.

Heart rate can easily be monitored by either electrocardiogram (ECG) recording or radiotelemetry (portable heart rate monitors). The development of these portable heart rate monitors has changed the application of exercise testing and prescription (Laukkanen and Virtanen, 1998).

Several studies have been done to validate portable heart rate monitors (Léger and Thivierge, 1988; MacFarlane et al., 1989; Seaward et al., 1990). Léger and Thivierge (1988) found a strong relationship ( $r = 0.99$ ) between heart rate measured by the better of 13 commercially available portable heart rate monitors and simultaneous recording by conventional electrocardiogram (ECG) machines. Seaward et al (1990) also found these two devices were equally reliable in measuring heart rate under a variety of different aerobic conditions (walking, running, aerobic dancing, Nordic Track simulated cross-country skiing) and exercise intensities

(rest, light, moderate, high and maximal intensity) with a correlation coefficient of  $r = 0.99$ . The fact that these portable heart rate monitors can be used in a variety of exercise situations makes it an extremely valuable and practical tool for researchers, athletes and coaches. Another advantage is that the athlete can receive immediate feedback and heart rate monitors with memory capacity can be used to record and store data for analysis at a later stage (Gilman, 1996).

#### 1.4.3.2.3 Heart rate during exercise.

Athletes are increasingly using heart rate, expressed as a percentage of their maximal heart rate (%HRmax), to monitor their own training intensity (Karvonen and Vuorimaa, 1988), whilst coaches use target heart rate as a means of establishing an athlete's training status and for exercise prescription (Karvonen, 1975; Zavorsky, 2000). According to Londeree et al (1995), using a regression equation relating percent of maximal oxygen consumption (%  $VO_2$ max) to percent of maximal heart rate (% HRmax), heart rate can easily be used for exercise prescription instead of having to rely on gas analysis. Due to the fact that resting heart rate increases with age and maximal heart rate decreases, %HR max is a better indicator of exercise intensity as the effect of other variables and age itself is diminished (Karvonen and Vuorimaa, 1988). The use of the formula: (maximal heart rate =  $220 - \text{age}$ ) alone to predict an appropriate training intensity is imprecise, but may be improved by using maximal heart rate and age using a multiple regression model (Roecker et al., 2002).

In a study by Gilman and Wells (1993), the heart rates of six female runners were monitored during 6 days of training and an 8 kilometer race. They were also requested to provide

information about their perception of their training intensity and race intensity. This subjective information was correlated to the heart rate recordings. Even though subjects reported on average only one moderate intensity training session, heart rate data indicated that subjects in fact spent about 45 % of training time at a moderate intensity. Similarly subjects reported one or two sessions doing high intensity training, with heart rate data indicating only 9 % of time spent within this training zone. More than 70 % of the race was run at a high intensity with subjects averaging 96 % of maximal heart rate. This clearly shows that subjects tend to overestimate the intensity at which they train and that heart rate monitoring provides a more accurate estimation of training intensity. As such it can be a valuable training tool to athletes and coaches alike.

Hills et al (1998) concluded that it is reasonable to assume that heart rate accurately reflects workload and intensity. There is speculation that changes in the heart rate / workload relationship can be used to accurately track changes in training status. This originates from the belief that there is a linear relationship between heart rate / workload and oxygen consumption (Arts and Kuipers, 1994). It is thus possible to use heart rate to predict training intensity due to this linear relationship between heart rate and oxygen uptake ( $VO_2\text{max}$ ) between values of 60 to 90% of maximum oxygen uptake, but caution should be exercised at values of > 90%  $VO_2\text{max}$  as the relationship becomes more unstable and less well defined (Ballarin et al., 1996; Bunc et al., 1988; Conconi et al., 1996; Gilman, 1996).

Cardiovascular adaptations and changes during resistance training may however, differ from the changes experienced during aerobic/ endurance type training. As such, Cooke and Carter (2005) examined the effect of high-intensity strength training on vagal-cardiac control and cardiovagal baroreflex sensitivity using heart rate variability measures. Twenty-two subjects

were either assigned to a strength training regime or as a recreationally active control group over a period of 8 weeks. They concluded that resistance training did not affect either vagal-cardiac control or cardiovagal baroreflex sensitivity in healthy young adults.

A study by Izquierdo et al (2005) found significant decreases in average heart rate after 16 weeks of training at workloads of 150 and 180 watts in the combined resistance and endurance and endurance only trained groups, with no changes recorded in the strength training only group.

Other studies found heart rate responses to resistance training related to the duration of the exercise (Lamotte et al., 2005), prolonged onset of peak cardiovascular responses, decreased cardiovascular responses to exercise and improved recovery from maximal exertion (Vincent et al., 2003).

The effects of resistance training on changes in heart rate however, still needs to be studied further before consensus can be reached as to the specific changes and the magnitude thereof in resistance trained athletes.

A brief discussion on the general physiology of the heart will follow as an understanding of this is necessary before the more applied aspects of heart rate and training status can be discussed.

#### 1.4.3.2.4 General physiology.

The heart has the unique ability to generate its own electrical signal (autoconduction) that enables it to contract without neural stimulation. Without neural or hormonal stimulation the heart has an intrinsic rate of approximately 70-80 beats per minute. The sinoatrial (SA) node is the pacemaker of the heart, as the impulses for cardiac contraction is generated from here. It has an established contraction rate called sinus rhythm. These impulses spread through the atria and are picked up by the atrioventricular node that then conducts the impulses through the Bundle of His, down the ventricular septum to the Purkinje fibers that transmits the impulses through the ventricles (Ross and Wilson, 1990).

Even-though the heart has its own intrinsic control mechanism, it may be affected by three extrinsic systems:

- Parasympathetic nervous system
- Sympathetic nervous system
- Endocrine system (Vander et al., 1975).

The parasympathetic nervous system (autonomic nervous system), acts on the heart through the vagus nerve, predominating at rest. The vagus nerve has a depressant effect, slowing the heart rate and increasing the force of the cardiac contraction. The sympathetic nervous system has the opposite effect and is responsible for increasing the heart rate whilst decreasing the force of the cardiac contraction. The sympathetic nervous system reacts during periods of physical and emotional stress. The endocrine system releases the catecholamines, adrenalin and noradrenalin through the adrenal medulla. These hormones have a stimulating effect on

the heart, increasing its rate and prolonging the effects of the sympathetic nervous action (Ross and Wilson, 1990).

Control of the cardiovascular system is effected by the brain through the autonomic nervous system (sympathetic and parasympathetic nervous system) (Hainsworth, 1998), baroreceptors, chemoreceptors and other circulating hormones (Levy and Martin, 1979). The autonomic nervous system functions entirely on a subconscious level and cannot be influenced by conscious volition (Aubert et al., 2003). Even though heart rate is relatively easy to measure accurately there are many factors that may affect heart rate. Factors affecting heart rate may be classified as either intrinsic (within the heart itself) or extrinsic (outside of the heart itself) in nature.

#### *1.4.3.2.4.1 Autonomic (Extrinsic) factors.*

##### *1.4.3.2.4.1.1 Plasma volume.*

In response to especially endurance exercise, the body adapts by expanding the plasma volume and as a consequence also the total blood volume (Fellmann, 1992; Selby and Eichner, 1994). This occurs in an effort to decrease the effect of post exercise plasma depletion. The increase in blood volume is dependent on the frequency and intensity of the exercise.

According to Convertino et al (1991) and Selby and Eichner (1994), the plasma volume in novice joggers may increase by nearly 300 milliliters, consequently decreasing the haemoglobin concentration by 5%. In contrast the plasma volume in elite runners may increase by nearly 1000 ml with a decrease in haemoglobin concentration of up to 20%.

The increase in plasma volume is due to two mechanisms. The first mechanism relates to the release of the hormones aldosterone and anti-diuretic hormone (ADH) and the second mechanism to the increased excretion of plasma proteins, specifically albumin (Convertino et al., 1980). The hormones cause the kidneys to increase the retention of water and the increased levels of plasma proteins lead to an increased osmotic pressure (Wilmore and Costill, 1994). According to Scatchard et al (1944), endurance training may increase the total plasma protein content by as much as 28 g and as 1 g of protein binds to 14 to 15 milliliters of water, plasma volume can be increased by as much as 427 milliliters.

In an early study by Convertino (1983), during which he studied the effect of changes in the relationship between plasma volume and heart rate with exercise training over an 8-day period, the conclusion was that changes in this relationship could be indicative of the effect of the elevated blood volume on stroke volume and cardiac output. In this study Convertino (1983) was able to show that a 1 % increase in plasma volume was associated with a decrease in heart rate of 1 % during exercise at 65 % of  $\text{VO}_2\text{max}$ . Thus the attempt at maintenance of venous return and stroke volume may be partly responsible for the decrease in maximal and submaximal heart rate. It has been found that when training is reduced for a period of two to four weeks after several months of running and cycling training, the blood volume can be reduced by up to 9 %, with a reduction of 12 % in both the stroke volume and plasma volume (Wilmore and Costill, 1994).

Rotstein et al (1998) investigated the effect of supramaximal short duration intermittent exercise in both hot (35° C, 30% relative humidity) and neutral conditions (22° C, 40% relative humidity) on plasma volume changes. They determined that plasma volume during short duration cycling is not affected by different environmental conditions and that plasma volume returned to its pre-

exercise levels within a 40 minute period regardless of whether the subjects had an active or passive recovery period.

Green et al (1990) attempted to ascertain if the training induced changes in plasma volume also affected cardiac function during prolonged submaximal cycling. Seven male cyclists were tested prior to and after a short three day training period. Testing took place over a 2 hour period at 65 % of  $VO_2$ max. As was expected the plasma volume increased by 20 % ( $P < 0.05$ ) and the total blood volume by 12 % ( $P < 0.05$ ). The cardiac output also increased ( $P < 0.05$ ) after training and was accompanied by an average decrease in heart rate of 13  $\text{beats}\cdot\text{min}^{-1}$  and an increase in stroke volume of 22 milliliters ( $P < 0.05$ ). They concluded that these three changes are the early adaptive responses to exercise training.

In summary, large changes in plasma volume occur in response to especially endurance type exercise training and may affect stroke volume and cardiac output leading to changes in submaximal and maximal heart rate. These changes should be kept in mind when interpreting changes in heart rate.

#### 1.4.3.2.4.1.2 Baroreceptor Reflex Function.

Baroreceptors are found in the aortic arch and the carotid sinuses (arterial baroreceptors). These receptors detect changes in the arterial blood pressure and react accordingly. Changes in the arterial blood pressure may cause changes in heart rate (stroke volume and peripheral resistance) (Berne and Levy, 1997).

Prolonged aerobic training can increase the sensitivity of the baroreceptors, causing an increased inhibition of the sympathetic nervous system and leading to a reduced arterial blood pressure and a lowered heart rate (Kingwell et al., 1992). This mechanism may also be used to explain the reduced maximum heart rate in trained athletes (Kingwell et al., 1992). There are also mechanical and metabolic receptors present in the active skeletal muscles that could have an influence on the cardiovascular system, also known as “exercise pressor reflex” and thus affect heart rate and arterial blood pressure (McCloskey and Mitchell, 1972). This mechanism could also be inhibited as it has been shown that the mean arterial pressure decreases with training during maximal exercise (Mier et al., 1997).

#### 1.4.3.2.4.1.3 Sympathetic nervous system.

The sympathetic nervous system forms part of the autonomic nervous system and together with the parasympathetic nervous system influence various physiological systems. The sympathetic nervous system is seen as the “fight or flight” mechanism in response to physiological stress. Reactions to stress include increased heart and respiratory rates, constricting peripheral blood vessels, reducing gastrointestinal movement and constricting sphincters (Aubert et al., 2003). Both the afferent and efferent nerve endings of the sympathetic nervous system end on the myocardium (Aubert et al., 2003).

The functions of the sympathetic nervous system on the cardiac system specifically are varied and include increasing the heart rate, cause dilation of the coronary vessels to increase blood supply to the heart and causes vasoconstriction in other non-essential tissues to ensure that the blood is diverted to the heart (Aubert et al., 2003; Wilmore and Costill, 1994).

It is generally accepted that during exercise heart rate increases due to an increased sympathetic and decreased parasympathetic nervous system response and that the role of each of these systems depend in large part on the intensity of the exercise performed (Collet et al., 1999; Malpas, 2002). Smith et al (1989) found that physical training inhibits the sympathetic response in athletes at a set exercise level, resulting in a decreased submaximal heart rate after training.

#### 1.4.3.2.4.1.4 Parasympathetic nervous system.

The parasympathetic system functions in direct opposition to that of the sympathetic nervous system. It forms part of the autonomic nervous system and stimulates the heart via the vagus nerve. The afferent and efferent nerve endings of the parasympathetic system end on the sinoatrial node, atrial myocardium and the atrioventricular node (Aubert et al., 2003). The parasympathetic system is dominant at rest and has a depressant effect on the heart. It slows the conduction of impulses and as such decreases the heart rate (Wilmore and Costill, 1994).

Smith et al (1989) demonstrated that in endurance trained subjects, the parasympathetic system dominates at rest ( $P < 0.04$ ), with a lesser sympathetic influence ( $P < 0.05$ ) compared to untrained subjects, by using atropine and metoprolol as a selective pharmacological blockade. They concluded that the exercise bradycardia displayed in these trained subjects was due to a lower intrinsic heart rate and resting autonomic balance coupled with a stronger parasympathetic influence.

Katona et al (1982) conducted a study on 10 non-athletes and 8 world-class oarsmen by using dual pharmacological blockades (propranolol and atropine respectively) on two occasions under similar circumstances to determine if the bradycardia normally observed in trained athletes, are due to changes in the intrinsic heart rate, sympathetic or parasympathetic nervous systems. Findings indicated that changes are unlikely to be caused by changes in acetylcholine or catecholamine levels and changes in the sensitivity of the pacemaker to these specific transmitters and blocking agents, as the effects of the transmitters were negated by full blocking doses of atropine and propranolol. They concluded that the resting bradycardia observed in athletes is entirely due to a reduction in the intrinsic heart rate, due to metabolic and mechanical effects, and not due to an increased parasympathetic tone.

A more recent study by Carter et al (2003 a) postulates that the training induced effect on the autonomic system i.e increased parasympathetic and decreased sympathetic activity together with an altered intrinsic heart rate, is responsible for the decreased resting heart rate in long term endurance trained athletes and that this type of training also leads to a decreased submaximal exercise heart rate.

The different findings of these studies relating to the specific roles of the parasympathetic and sympathetic nervous system serve to highlight the uncertainty that still exists regarding the exact mechanisms at work in athletic bradycardia. These factors should be taken into consideration when interpreting heart rate data as monitoring may have been affected by a variety of different circumstances. Ideally these factors should be controlled for when measuring heart rate.

#### 1.4.3.2.4.1.5 Endocrine system.

A variety of different hormones regulate cellular functions after being released into the blood circulation (Vander et al., 1975). For example, the adrenal medulla releases the hormones, epinephrine and norepinephrine (catecholamines) in response to certain stimuli, including exercise. The catecholamines mimic and activate the sympathetic nervous system and as such affects the metabolic rate directly, thus also increasing the heart rate (Ross and Wilson, 1990).

The relationship between heart rate and catecholamine excretion was studied by Platen et al (2000) who conducted a 4-week intensive training program with the aim of inducing overtraining. Mean nightly heart rate (2 – 5 am) and morning heart rate was measured. 24-hour urine was collected every night for the duration of the training for the measurement of catecholamine excretion. The urinary catecholamine excretion increased during this period but the mean night heart rate decreased and the mean morning heart rate remained constant. They concluded that night and morning heart rate is independent of the increased levels of urinary catecholamine excretion.

#### 1.4.3.2.4.2 Non-Autonomic (Intrinsic) factors.

##### 1.4.3.2.4.2.1 Heart volume.

According to Wilmore and Costill (1994), intense aerobic type exercise increases the need for higher circulating blood volume that in turn increases the demands on the left ventricle. The left

ventricle responds by increasing its internal diameter size, thus effectively increasing the size of the chamber, and wall thickness. This hypertrophy however, is different to the pathological changes seen in cardiac disease (Fagard et al., 1983 and 1984).

A study by Milliken et al (1988), using magnetic resonance imaging (MRI), showed that highly trained athletes (runners, cyclists and skiers) each had a greater left ventricular mass than that found in the untrained controls. They also concluded that the left ventricular mass was highly correlated to  $VO_2$  max. Urhausen and Kindermann (1998) showed that the hearts of endurance athletes had higher values for all the variables measured (heart volume, left ventricular mass, end-diastolic diameter and left ventricular septal and posterior wall thickness) than bodybuilders after correcting for body mass. Similar results were found by Bonaduce et al (1997) when they compared the cardiac (heart volume) characteristics of non-athletes to previously highly trained cyclists after a one month period of detraining. Gyimes et al (2004) concluded that long term high intensity endurance training had the biggest effect on left ventricular cavity size and that strength training did not necessarily induce ventricular wall thickening.

Lewis et al (1980) postulated that the tonic stretch that accompanies an enlarged ventricle after prolonged endurance training could inhibit the acute stretch response (Bainbridge reflex) and cause a lower intrinsic heart rate. Katona et al (1982) concluded that the cardiac hypertrophy present in athletes could have a direct mechanical effect on the pacemaking mechanism of the heart and thus cause changes in heart rate. Perrault and Turcotte (1994) argue that the cardiac hypertrophy found in athletes could be due to ventricular dilatation from increased plasma volumes and not necessarily due to myocardial enlargement.

Sundstedt et al (2004) studied the effect of changes in heart volume on the predicted increased cardiac output in a group of 24 male endurance athletes. Change in left ventricular volume was measured by contrast echocardiography during an upright cycling test. Results indicated that the end-diastolic volume was increased by 18 % ( $P < 0.001$ ), end-systolic volume decreased by 21 % ( $P = 0.002$ ), stroke volume increased by 45 % ( $P < 0.001$ ) and the increased end-diastolic volume was responsible for 73 % of the increased stroke volume.

In summary, any change in cardiac dimensions, regardless of the origin, is bound to have an effect on heart rate and therefore needs to be considered when heart rate measurements are recorded over a long period, particularly after training.

#### 1.4.3.2.4.2.2 Myocardial cell metabolism.

According to Katona et al (1982), any changes in the cardiac cell metabolism will result in increased energy production and utilization and this could lead to changes in heart rate. It is generally accepted that increased acto-myosin ATPase activity indicates that the myocardium has adapted to the training stimulus (Bhan and Scheuer, 1972 and 1975). From this, Zavorsky (2000) concluded that the higher energy production and utilization in the myocardium could lead to a lowered maximal heart rate during maximal exercise.

#### 1.4.3.2.4.2.3 Electrophysiology of sinoatrial node.

The sino-atrial node (SA) is a concentration of highly specialized cardiac cells situated in the upper posterior portion of the atrium. It is called the pacemaker of the heart and the electrical impulses for the heart to contract is generated from here (intrinsic control). It is responsible for maintaining sinus rhythm (Ross and Wilson, 1990).

Several studies have indicated that changes in the electrophysiology of the SA node could alter maximal heart rate by causing changes in the resting membrane potential, the threshold potential or the slope of polarization during the 4<sup>th</sup> phase of the action potential (Brorson et al., 1976; Hughson et al., 1977; Lewis et al., 1980). Boddy et al (1974) demonstrated that highly trained individuals exhibit higher total body potassium (K<sup>+</sup>) levels with lower serum potassium levels than sedentary individuals.

#### 1.4.3.2.4.2.4 $\beta$ – adrenergic receptors.

The adrenergic nerves are influenced and stimulated by the sympathetic nervous system through the neurotransmitter norepinephrine. Norepinephrine is released through neural impulses and crosses the synapses to bind with the adrenergic receptors on the target cells. Adrenergic receptors are classified into two groups, namely alpha and beta –adrenergic receptors. Beta-receptors ( $\beta$ ) are again divided into beta-1 and beta-2 receptors. Beta-1 receptors are found mainly in the heart, with beta-2 found in the blood vessels, lungs, liver, intestines and skeletal muscle (Wilmore and Costill, 1994).

Werle et al (1990) demonstrated that intensive exercise reduces the number of  $\beta$ -receptors in the cell membrane through movement to the intracellular compartment. They postulated that this could be due to increased levels of catecholamines, hormonal or metabolic factors during acute exercise. Several studies (Hammond et al., 1987; Hammond et al., 1988; Werle et al., 1990) have found reduced numbers of  $\beta$ -receptors in animals due to the down-regulation of receptor synthesis after prolonged exercise training. Zavorsky, (2000) speculated that this decrease in receptor activity could be responsible for the decreased maximal heart rate in humans after exercise training.

#### **1.4.3.2.5 Factors that may affect heart rate during exercise.**

Under certain conditions heart rate may not be an accurate marker of training intensity. There are various factors that may affect the heart rate / workload relationship. Some factors include environmental temperatures (Claremont et al., 1975; Gisolfi and Cohen, 1979), state of hydration, mode of exercise, duration of exercise, cardiac drift and competition (Gilman, 1996; Lambert et al., 1998). Other factors include certain medications, acute changes in body mass and other environmental factors such as humidity (Fisher et al., 1999) and windchill factors (Wilmore and Costill, 1994).

Lower heart rate at rest or athletic bradycardia is often observed in highly trained endurance athletes. These lower heart rates are believed to be due to an increased parasympathetic nervous system response (vagal tone) (Kenny, 1985; Seals and Chase, 1998) rather than a reduced sympathetic nervous system response (Lin and Horvath, 1972; Smith et al., 1989). Katona et al (1982) speculates that this bradycardia is due to a decreased intrinsic heart rate

whilst Carter et al (2003 a) concluded that all of the above factors are in fact involved in regulating the lowered heart rate.

There is thus still uncertainty regarding the exact mechanism of athletic bradycardia. Although heart rate generally reflects the amount of work or exercise intensity, there are several factors which can alter the heart rate / workload relationship. Heart rate during exercise may be influenced by several factors. These will be discussed in more detail.

#### 1.4.3.2.5.1 Cardiac drift.

Cardiac drift can be defined as a progressively increasing heart rate as the exercise duration increases (Montain and Coyle, 1992) and may or may not be accompanied by a decrease in blood volume (Coyle and Hamilton, 1990; Heaps et al., 1994). Shaffrath and Adams (1984) and Kindermann et al (1979) also included in this definition decreases in mean arterial pressure and maintenance of cardiac output during prolonged exercise. Cardiac drift can be interpreted as a compensation in heart rate when homeostasis can no longer be maintained. The mechanism for cardiac drift is unclear and researchers have postulated various possible mechanisms to explain this phenomenon. The most common explanation is that dehydration after prolonged exercise is associated with cardiac drift.

To test this theory, Heaps et al (1994) studied 9 subjects who cycled at 65% of peak  $O_2$  consumption while euhydrated and also in a state of hypohydration at 21° C. Subjects were rested for 2 hours after the exercise induced dehydration and the dehydration was then either

not reversed (no water) or only partially reversed (by 65 %) during the rest period (water trial). This study showed that the magnitude of cardiac drift at 21° C is proportionate to the level of hydration and not due to a reduction in blood volume as the blood volume was the same for the different hydration states (hypohydrated and euhydrated). During the “water trial” subjects were hypohydrated by 0.9 % of bodyweight. Heart rate increased significantly ( $P < 0.05$ ) by  $10 \pm 2$  beats.min<sup>-1</sup> whilst the stroke volume decreased by 9 milliliter / bodyweight compared to the euhydrated state. When the subjects were hypohydrated by 2.8 % of bodyweight during the “no water trial”, the heart rate increased significantly ( $P < 0.05$ ) by  $18 \pm 2$  beats.min<sup>-1</sup> and stroke volume decreased by 18 milliliter / bodyweight compared to the euhydrated state. This study thus showed that a loss of fluid from the intracellular and interstitial spaces could have a significant effect on cardiac drift during exercise even when the blood volume remained the same. Several other studies have also confirmed that cardiac drift during exercise could occur without reductions in blood volume (Coyle and Hamilton, 1990; Raven and Stevens, 1988; Sawka et al., 1979).

Montain and Coyle (1992) found that an athletes' hydration status played a role in cardiac drift. For every 1% fluid loss due to dehydration, the athletes' heart rate increased by 7 beats.min<sup>-1</sup>. They also determined that the magnitude of dehydration was related to increases in oesophageal temperature ( $r = 0.98$ ), heart rate ( $r = 0.99$ ) and a decrease in stroke volume ( $r = 0.99$ ).

In a study by Schabert et al (1998), during which athletes ran a 60-minute time trial, it was found that the cardiac drift was large (158 beats.min<sup>-1</sup> at the start of trial to 177 beats.min<sup>-1</sup> at the end of the trial) and that this factor could significantly affect the heart rate – running speed relationship. Even though the subjects increased their running speed towards the end of the

trial, it was still not possible to account for the higher heart rate. This leads one to conclude that heart rate is not predictably related to running speed particularly towards the end of long duration exercise.

Other factors that could affect cardiac drift are ambient temperature and humidity. Booth et al (1997) conducted a study during which running speed remained relatively constant at a temperature of 32° C and 60% humidity. Heart rate showed an increase from 168 to 188 beats.min<sup>-1</sup> during the 30-minute trial. Coyle and Gonzalez-Alonso (2001) suggest that cardiac drift, accompanied by a decrease in stroke volume after 10-20 minutes of exercise, is mainly due to an increased heart rate and not due to increased cutaneous blood flow due to increasing body temperature.

Wingo et al (2005) studied the effect of cardiovascular drift in nine male cyclists on maximal oxygen uptake in a hot (35° C) environment during prolonged, constant, moderate intensity cycling at 60 % of VO<sub>2</sub>max, after which VO<sub>2</sub>max was measured. During one protocol, subjects ingested fluid with body weight remaining unchanged. A second protocol was performed where no fluids were ingested and subjects became dehydrated. It was found that taking in fluids did not affect the cardiovascular drift that occurred or altered the VO<sub>2</sub>max. Heart rate increased by 12 %, stroke volume decreased by 16 %, with VO<sub>2</sub>max decreasing by 19 % (P < 0.05) despite a higher maximal heart rate at 45 minutes compared to 15 minutes. They concluded that cardiovascular drift after 45 minutes in a hot environment is associated with a decrease in VO<sub>2</sub>max and that changes in heart rate may be a valid marker to identify relative changes in metabolic intensity in these conditions.

There is also speculation that there is a lesser incidence of cardiovascular drift in more highly trained individuals, especially with endurance type training, but this has not been scientifically proven (Lambert et al., 1998). According to Gilman (1996), heart rate reflects work intensity inaccurately as can be seen with cardiac drift where the heart rate increases slowly when exercise exceeds 20 minutes. This upward drift may be as much as 20 beats.min<sup>-1</sup> regardless of whether lactate levels remain the same or decrease.

In summary, cardiac drift occurs in exercise of increasing duration, which causes heart rate to increase by up to 20 beats.min<sup>-1</sup> at the same workload. It is affected by factors such as level of hydration, environmental (temperature and humidity) variables and possibly training status. There is still controversy about the specific mechanism responsible for cardiac drift and studies have also reported different results regarding the effect of changes in the blood volume on cardiac drift. Cardiac drift may alter the heart rate-workload relationship, therefore this needs to be considered if the heart rate / workload relationship is used to monitor training status.

#### *1.4.3.2.5.2 Circadian rhythm.*

Under normal conditions the body has a 24-hour or circadian rhythm (Atkinson and Reilly, 1996). The circadian rhythm is interconnected to various physiological alterations that take place in the body such as changes in heart rate, temperature, oxygen uptake, flexibility, muscle strength, power output, short-term memory and other performance parameters (Atkinson and Reilly, 1996; Winget et al., 1985). Pickering (1988) noted that circadian rhythms in heart rate and blood pressure could be affected by factors such as sleep, posture, food ingestion and activity.

These circadian rhythms are endogenously controlled and will remain present even if the subject is removed from the influence of external variations (Atkinson and Reilly, 1996; Halberg et al., 1970). In studies performed by Aschoff (1965) and Wever, (1979), subjects were isolated from all external time signals, however, circadian rhythm patterns only showed minor deviations between 24 to 25 hours. Normally, these endogenous rhythms are controlled by factors or time-givers (*zeitgebers*) such as the light-dark cycle and social influences (Atkinson and Reilly, 1996).

There is some indirect evidence that performance is improved in the early evening when the body temperature is at its highest. This co-incidently, is the time when most world records are broken (Atkinson and Reilly, 1996; Atkinson et al., 1994; Conroy and O'Brien, 1974).

According to Reilly et al (1984) a significant circadian rhythm for resting heart rate occurs with a peak at 15h00, with the circadian pattern persisting during both submaximal and maximal training loads. Atkinson and Reilly (1996) found that heart rate presents with a definite ultradian rhythm. The term ultradian refers to any rhythm presenting with periods of less than 20 hours, as can be seen in for example, the 90 minute cycle of sleep stages. Throughout the 24-hour circadian cycle heart rate displays variations in amplitude of between 5-15% of the 24 hour mean (Reilly, 1990; Smolensky et al., 1976). Maximal heart rate displayed definite circadian variations with a study by Ilmarinen et al (1980) reporting a 15.6 % difference between trough and peak values compared to the 3.5 % reported by Reilly et al (1984).

However, a study done by Cohen (1980), in contrast, found no rhythm in maximal heart rate when performing maximal incremental exercise during seven testing periods throughout the solar day. Exogenous factors such as sleep, posture, food and activity also seems to have a big effect on the circadian rhythms of blood pressure and heart rate (Pickering, 1988). Heart

rate responses to exercise in the mornings however, seem to be minimal. Variations in amplitude is increased in physically fit compared to sedentary or unfit individuals when tested under controlled laboratory conditions (Atkinson et al., 1993; Harma et al., 1982).

Bonaduce et al, (1997) found the circadian pattern of day-night variations did not change in top level athletes after vigorous training. This day-night variation did not differ between trained and untrained athletes, with higher values of heart rate variability (this will be discussed in more detail under 1.4.3.2.7) measures recorded at night, possibly indicating some measure of parasympathetic control. In a study conducted by Wahlberg and Åstrand (1973) on 20 subjects, heart rates were consistently lower (3 to 5 beats.min<sup>-1</sup>) at night (03h00) compared to the day (15h00), regardless of whether the workload was submaximal or maximal. Cohen and Muehl (1977) similarly found night time heart rates to be at its lowest between 04h00 and 08h00. Dalton et al (1997) however, suggests that individuals may have an adaptive response that allows them to train and perform at different times, regardless of the inherent circadian rhythm.

According to Atkinson and Reilly (1996) circadian variations in metabolic responses to submaximal exercise are less well defined than when heart rate is measured, with a definite lack of rhythmicity at maximal exercise intensities. In studies by Reilly and Brooks (1982) and Faria and Drummond (1982) it was demonstrated that VO<sub>2</sub>max remained stable regardless of the time of testing, and therefore there was no evidence of any circadian variation. This was an expected outcome as the amplitude of the resting rhythm of VO<sub>2</sub> during maximal exercise normally would be less than 0.5 % of VO<sub>2</sub>max, making it extremely difficult to measure (Atkinson and Reilly, 1996).

In summary, there is no consensus on the magnitude of the effect of the circadian rhythm on the various performance parameters and other physiological changes such as blood pressure and heart rate that occurs during exercise. It is however an important factor that should be taken into consideration when using heart rate as an indicator of training status as heart rate responses to exercise at different times may vary and performance may also be affected depending on the time of day that testing takes place. This is one of the main reasons why exercise testing should preferably be done at the same time of day so as to minimize any circadian effects on heart rate and performance.

#### *1.4.3.2.5.3 Competition.*

This section addresses the effects of competition on heart rate.

The first study to show that there is a difference between heart rate during training and competition at the same running speed was that of Selley et al (1995). They studied 16 males in a 10- and 21 km race situation as well as in a field test at predetermined running speeds which allowed them to measure heart rate at the same speeds in a non-competitive bout that the subjects ran during the races. The results indicated that % HR max for each subject was related ( $r = 0.98$ ) to running speed in the field test and was repeatable ( $r = 0.98$ ). The % HRmax also remained similar during both the 10- and 21- km races even though the 10 km race was completed at a faster average pace than the 21 km race. The % HRmax in the field test however was consistently lower compared to that achieved during the 10-km ( $P < 0.01$ ) and 21-km ( $P < 0.001$ ) races. This led them to conclude that factors other than running speed, possibly increased sympathetic stimulation, may influence heart rate during competition. It may thus be inappropriate to use heart rate to prescribe running speeds for a competitive race

compared to a non-competitive situation. These findings have important implications for athletes and coaches using heart rate as an indicator of pace during competitions as it may well lead to an erroneous pacing strategy.

Similarly, Mbambo and Lambert (2000), found a correlation of  $r = 0.99$  ( $P < 0.001$ ) for the heart rate – running speed relationship during non-competitive training compared to a correlation of  $r = 0.02$  during a race. This in essence means that heart rate responds differently to increasing exercise intensity during competitions compared to during training. In the study by Foster et al (1993), athletes were tested under simulated competition conditions using a 5-km time trial test on a racing bicycle attached to a windload simulator and results were compared to a standard graded exercise protocol frequently used to test athletes to further elucidate the physiological responses during competition compared to laboratory testing. They speculate that the pattern of power output employed by athletes during competition differs substantially from that to using graded exercise protocols during routine laboratory testing. For example, higher  $\text{VO}_2$  ( $3.5 \pm 0.8$  l.min<sup>-1</sup> vs.  $3.3 \pm 0.8$  l.min<sup>-1</sup>), maximal heart rate ( $184 \pm 11$  beats.min<sup>-1</sup> vs.  $175 \pm 11$  beats.min<sup>-1</sup>) and blood lactate values ( $14.8 \pm 3.7$  mM vs.  $11.9 \pm 2.1$  mM) were measured during the 5-km time trial (simulated competition) compared to the cycle ergometer test respectively ( $P < 0.05$ ). However, in this study the peak heart rate during the 5-km time trial was not significantly different compared to the cycle ergometer test. They concluded that the physiological responses of athletes differ substantially under competitive compared to normal training circumstances.

Palmer et al (1994) studied the heart rate of seven cyclists. They determined that six of the seven cyclists displayed higher peak heart rates during racing compared to testing under maximal laboratory conditions. However, a more recent study performed by Boudet et al

(2002), in which 16 subjects were tested under three different conditions (laboratory, field and competition) to test the variability of maximal heart rate, showed that heart rate peaks were not statistically different ( $P = 0.62$ ). Mean peak heart rates were (laboratory)  $194 \pm 8$ , (field)  $194 \pm 12$  and (competition)  $192 \pm 10$  beats.min<sup>-1</sup>. They also found a large intra-individual variation ( $\pm 6$  beats.min<sup>-1</sup>) in all three conditions (laboratory, field, competition) with the absolute median maximal heart rate 8 beats.min<sup>-1</sup> lower than the heart rate peak. Both the median maximal heart rate and heart rate peak were highly related ( $\rho = 0.89$ ,  $z = 3.5$ ,  $P = 0.0006$ , Spearman test). They concluded that median maximal heart rate had a higher inter-condition relationship, was more stable and provided more value than an isolated peak, and could therefore potentially decrease the risk of under or over estimating heart rate and exercise intensity (Boudet et al., 2002).

In summary, there are data that shows that competition could potentially affect the heart rate-running speed relationship and that heart rate as measured during competition may not be an accurate measurement of running speed (Mbambo and Lambert, 2000). Therefore, if heart rate is used during races as a pacing mechanism, it could cause athletes to perform worse than expected. However, there is conflicting evidence as provided by the recent study of Boudet et al (2002) where athletes were tested under different conditions (laboratory, field and competition) with the results not indicating significant differences in heart rate and thus further studies will be needed to clarify the dichotomy.

#### 1.4.3.2.5.4 Environmental factors.

Environmental factors such as temperature and humidity can affect the body's response during both exercise and rest (Wilmore and Costill, 1994) and the cardiovascular system in particular may experience strain, exhibiting changes in heart rate, stroke volume and cardiac output (Adams et al., 1975; Galloway and Maughan, 1997; Nielsen et al., 1993). A normal body temperature may range between 36.1 to 37.8 ° C, with convulsions occurring at  $\pm 40$  ° C and death following at approximately 42.2 ° C or when the rectal temperature decreases to approximately 24.2 – 25.7 ° C (Wilmore and Costill, 1994).

During exercise in the heat, the total blood volume (González-Alonso et al., 1997) may be affected due to competition from the muscles and skin (Marino and Booth, 2001). This may cause a lowered blood and plasma volume. Cardiovascular disturbances such as haemoconcentration and a reduction in blood volume (González-Alonso et al., 1997), increased heat storage and core temperature, decreased stroke volume and cardiac output and increased heart rate may follow if the fluid loss is 10 % or more (Fortney et al., 1981). Wind can affect the rate of heat loss via convection and conduction, also known as windchill.

Humidity also affects heat loss in the form of evaporation. If the humidity content is high it may limit the amount of heat loss by inhibiting sweating. If the surrounding environmental temperature is low but the humidity is high, no evaporation can take place either and heat stress can occur. During prolonged exercise when the temperature and the humidity are high, the individual will not be able to lose the build-up of excessive heat and heat stress will occur rapidly (Wilmore and Costill, 1994).

Claremont et al (1975), states that when the environmental heat stress is high, heart rate will be higher at a given exercise intensity. She found that male cyclists, cycling at either 0 or 35° C for a period of 30 to 60 minutes at 52 – 59% of  $\text{VO}_2\text{max}$ , had an average increase in heart rate of 13  $\text{beats}\cdot\text{min}^{-1}$  at the higher temperature. A study by Galloway and Maughan (1997) showed a significantly higher ( $P < 0.05$ ) heart rate during exercise at a temperature of 31° C than at 4°, 11° or 21° C.

In a study by Cochrane and Sleivert (1999), eight male distance runners were tested over a two-hour period on a treadmill at 70 % maximum oxygen consumption. The mean heat load was kept the same at 22.2° C wet bulb temperature but the dry bulb temperature was either increased (24 to 27.5° C) or decreased (27.5 to 24° C). The heart rate, rectal temperature, rating of perceived exertion (RPE), oxygen consumption and performance was similar for both tests. The whole body sweat rate was higher and plasma volume loss was greater in the cooling versus the warming pattern test. They concluded that the mean heat load is a more important factor to consider than changing temperature and humidity when assessing an individual's response to exercise in the heat.

Marino and Booth (2001) studied seven competitive runners during a 30- minute self-paced run on a treadmill under a warm (32° C, 60 % relative humidity) condition. Subjects were either whole body precooled or not in the control condition. Resting heart rate was significantly lower ( $P < 0.05$ ) at  $62 \pm 4 \text{ beats}\cdot\text{min}^{-1}$  following precooling compared to  $75 \pm 3 \text{ beats}\cdot\text{min}^{-1}$  during the control condition (no cooling). The heart rate at 5 minutes into the run differed significantly ( $P < 0.05$ ) from the precooled ( $158 \pm 10 \text{ beats}\cdot\text{min}^{-1}$ ) compared to the control condition ( $166 \pm 10 \text{ beats}\cdot\text{min}^{-1}$ ), after which there was no difference in heart rate for the remainder of the run under both conditions, with heart rate at the end of the exercise similar. The researchers could not

explain why heart rate was similar at the end of the exercise as the precooling subjected the runners to less thermoregulatory strain, increased heat storage capacity and higher work outputs in this study.

In conclusion, environmental factors such as high temperature ( $> 30^{\circ}\text{C}$ ) and humidity will affect the cardiac system by causing changes in heart rate, stroke volume, cardiac output, decreased blood volume, haemoconcentration and an increase in the athlete's core temperature. All of the above changes will certainly affect an athlete's performance and may also be detrimental to their health in extreme conditions. It is also possible that the mean heat load may be even more important than the actual temperature and humidity but more studies are needed to confirm this.

When testing athletes in the field situation it may be difficult to control environmental variables but possible changes in these conditions should be kept in mind when interpreting heart rate data. Certainly these variables should be controlled during laboratory testing.

#### *1.4.3.2.5.5 Medication.*

There are a large number of different categories of pharmacological substances that may have an effect on heart rate. These medications may either increase or decrease the heart rate at rest or during exercise (ACSM, 1995).

Beta-blockers and anti-adrenergic medications slow and strengthen the heart beat whilst medications such as amphetamines, ephedrine, pseudoephedrine will increase the heart rate with a concomitant decrease in effectivity (ACSM, 1995). As beta-blockers depress the heart

rate response during exercise, specifically at lower heart rate levels, care should be taken when prescribing exercise intensity. Target heart rates should be adapted accordingly and other methods should be employed to ascertain exercise intensity such as rating of perceived exertion (RPE) (Derman and Schwelinnuss, 1998).

The possible effects of medication on heart rate should be considered when testing athletes. Medication use should preferably be reported by athletes and if possible controlled for prior to exercise testing. Reporting of medication use by some athletes may be a problem especially if the drugs in question are on the banned list.

#### *1.4.3.2.5.6 Mode of exercise.*

Under certain conditions, such as with different modes of exercise, heart rate may not be an accurate marker of exercise intensity and the heart rate / workload relationship can be compromised.

Londeree et al (1995) tested the %  $\text{VO}_2\text{max}$  and %  $\text{HRmax}$  regressions on several different exercise modes (treadmill, skier, shuffle skier, stepper, rower) to clarify this relationship. Ten male subjects were introduced and familiarized to each of the different modes and were then required to perform an incremental test to maximum on each. They concluded that there are definite mode-specific differences and that the upright and weight-bearing modes had a similar relationship whilst in contrast the weight-supported and arm exercise modes were not accurate in following this relationship. Hauber et al (1997) tested 11 fitness swimmers to determine if the indicators of exercise intensity (heart rate and oxygen consumption) used in land-based

exercise (treadmill running) could also be used in swimming. They found that peak heart rate ( $171 \pm 3$  vs.  $183 \pm 3$  beats.min<sup>-1</sup>) and O<sub>2</sub> uptake ( $3.6 \pm 0.2$  vs.  $4.0 \pm 0.2$  l.min<sup>-1</sup>) appear to be mode specific for swimming and treadmill running respectively, but that when exercising at a set submaximal O<sub>2</sub> uptake level, heart rate remains similar regardless of the mode. They concluded that target heart rates used in land-based exercises could also be used for swimmers.

In a study by Weston et al (2001), it was found that whilst the relationship between VO<sub>2</sub> (ml.min<sup>-1</sup>) and heart rate (beats.min<sup>-1</sup>) during treadmill running was linear ( $r = 0.89$ ), the relationship between heart rate and VO<sub>2</sub> during trampoline exercise showed a different pattern. There was a marked elevation of heart rate before any change in the VO<sub>2</sub>, which led them to conclude that the traditional heart rate zones need to be reassessed when prescribing for mini trampoline jogging.

After evaluating the Conconi Test in a group of rowers, Bourgois and Vrijens (1998) concluded that the anaerobic threshold for heart rate is not relevant for monitoring endurance training in rowing. Conconi et al (1996) in his revised study, states that the speed/heart rate relationship is linear at low to moderate speed and curvilinear from submaximal to maximal speeds. Several subjects ( $n = 670$ ) were tested by applying the Conconi Test in studies from different sports, between the periods 1980 to 1993. The studies consisted of the following sports: running, cycling, race-walking, rowing, canoeing, roller-skating, ice-skating, roller-skiing, swimming and cross-country skiing. Subjects were of varying athletic ability and the test was conducted both indoors in the laboratory and outdoors as well as during training and competition.

In summary, different exercise modalities may compromise the heart rate / workload relationship. This obviously needs to be considered when heart rate is either used to prescribe exercise or monitor training status.

#### *1.4.3.2.5.7 Training status.*

There are numerous cardiac adaptations that take place in response to training and some of the parameters that may be affected include heart rate, stroke volume and cardiac output as described previously. According to Wilmore and Costill (1994), a higher level of cardio-respiratory fitness produces a lower heart rate at a specified work rate during submaximal exercise.

Some highly trained and conditioned athletes may present with resting heart rates of lower than 40 beats.min<sup>-1</sup> or even 30 beats.min<sup>-1</sup>. As discussed earlier in Section 1.4.3.2.4.1.4, this decrease is thought to be due to increased parasympathetic activity in the heart with a concomitant decrease in sympathetic activity and a decreased intrinsic heart rate.

Several studies (Pollock, 1973; Seals and Chase, 1989; Shi et al., 1995; Spina et al., 1993; Wilmore et al., 1996) studied the effect of endurance training on various categories of subjects over differing periods of time and all found a decreased heart rate at rest of between – 2.7 and – 10 beats.min<sup>-1</sup>. However, there were several similar studies (Martin et al., 1987; Meredith et al., 1989; O'Connor et al., 1993; Sedgwick et al., 1974) that found no change in heart rate at rest after 12 weeks of endurance training.

Uusitalo et al (1998) progressively increased the training load in a group of female endurance athletes over a period of 6 – 9 weeks. The increased training load and symptoms of overtraining in some subjects did not influence the intrinsic heart rate or cardiac autonomic modulation significantly. They found that the resting heart rate decreased with the heavy endurance training and overtraining. Verde et al (1992) found that after a 3-week period of high volume training, the peak heart rate decreased somewhat and approached statistical significance in 9 of their ten subjects. He also found that after the 3 week period of increased training and a 3 week recovery period, waking heart rates only changed by  $< 2 \text{ beats} \cdot \text{min}^{-1}$  and concluded that a change in heart rate as small as this would be of very little value as an indicator of imminent overtraining.

In conclusion, it can be seen that there is still no consensus on the mechanism responsible for the decreased heart rate or athletic bradycardia observed in endurance-trained athletes.

Different studies have also reported different magnitude of changes in resting heart rate after endurance training, with changes ranging from nothing at all to minus ten beats per minute.

The problem is that studies are conducted over differing periods of training, at different exercise intensities and with athletes of differing training status, making it difficult to compare results.

#### **1.4.3.2.5 Heart rate during sleep.**

As there is an accepted relationship between heart rate and training status, it is conceivable that this relationship will be more evident during sleep when the factors that may affect heart rate during the day are minimized. To this end several studies have examined heart rate during sleep and its relationship to training status (Bevier et al., 1987; Bunnell et al., 1983; Rousset

and Buguet, 1982) and found that heart rate during sleep was increased after submaximal daytime walking exercise. These studies also only reported average heart rate values during sleep. Results from these and other studies (Jeukendrup et al., 1992; O'Connor et al., 1993) are diverse and further studies are needed to clarify the exact nature of the heart rate during sleep and training status relationship.

Jeukendrup et al (1992) suggested that heart rate measured during sleep may be a more accurate assessment of resting heart rate compared to palpated resting heart rate measured for 15 seconds upon waking. It is logical to assume that if there is a relationship between heart rate and training status that this will be more evident during sleep when extraneous factors that influence heart rate are reduced. Jeukendrup et al (1992) conducted a study on seven male competitive cyclists and showed that the palpated morning resting heart rate did not change during a two-week period of high volume training, while the mean heart rate during sleep showed a significant ( $P < 0.05$ ) increase and may therefore be a viable marker of symptoms of overtraining.

O'Connor et al (1993) conducted a study on nine untrained males to assess the influence of daytime exercise on heart rate during sleep. Subjects cycled at 75 % of  $VO_2$ max, starting at 30 minutes per day, for a period of 12 weeks. The exercise duration was increased by 5 minutes every 4 weeks up to a maximum of 40 minutes. Heart rate was measured on two separate occasions, before and after the 12-week training period. Heart rate was measured on both a training and a non-training day. They found no significant difference in heart rate during sleep on both training ( $P = 0.14$ ) and non-training days, before and after the 12 week training period. They did however, conclude that endurance training in young males speeds up the achievement of baseline heart rate during sleep and that endurance training did not moderate

the relationship between an acute episode of exercise during the day and heart rate during sleep.

#### *1.4.3.2.6.1 Types of sleep.*

There are two major types of sleep, namely non-rapid eye movement (NREM), also known as slow-wave sleep and rapid eye movement (REM) or paradoxical sleep. These two sleep patterns alternate and follow a regular 30 to 90 minute cycle. REM sleep occurs after about 30 minutes of NREM sleep and lasts 10 to 15 minutes. During REM sleep, respiration and heart rate becomes irregular and blood pressure may increase or decrease whilst these same changes are much smaller in magnitude during NREM sleep (Vander et al., 1975).

Ninety minutes after the start of sleep, electroencephalograph (EEG) changes take place in the brain and as sleep deepens the EEG waves become slower, larger and irregular. The EEG patterns are substantially different between the two different types of sleep. Blood flow and oxygen consumption by the brain do not show any changes or decrease during sleep (Vander et al., 1975).

The level of neurotransmitters in the brain may vary during sleep. Marieb (1992) recorded an increased level of noradrenalin during REM sleep. Jeukendrup et al (1992) found that recorded sleeping heart rates increased significantly following high intensity interval training. He hypothesized that this increase may be due to an increase in sympathetic resting tone. Snyder et al (1964) noted decreased heart rates during slow wave sleep (NREM) that occurs primarily in the first few hours of sleep. It is believed that especially aerobic exercise increases the incidence of slow wave sleep, and Home (1981) hypothesized that daytime exercise could thus

influence heart rates more during the first few hours of sleep as this is when slow wave sleep is more prevalent. In the study by Roussel and Buguet (1982), three of the subjects displayed increases in heart rate during paradoxical sleep (REM), compared to the preceding slow wave sleep, with one subject displaying the opposite.

Heart rate is also affected when a person is in the supine position and this factor should be taken into account when considering sleeping heart rate as a possible indicator of training status. Whilst in the supine position, blood does not pool in the lower extremities and thus returns more easily to the heart. This means that the resting stroke volume values are much higher in the supine than in the upright position with a concurrent decrease in heart rate (Wilmore and Costill, 1994).

#### *1.4.3.2.6.2 Effect of exercise on heart rate during sleep.*

An oversight by some of the earlier studies on heart rate during sleep was that these studies did not report if the measurement of heart rate changes were performed on days following exercise or on non-exercise days. Thus, O'Connor et al (1993) examined the influence of daytime exercise on heart rate during sleep as previously discussed. Even though they found no difference between heart rate during sleep on both training and non-training days, they conceded that there could be potential interactive effects on heart rate during sleep such as level of fitness, exercise intensity, duration and mode. Previous studies (Bevier et al., 1987; Bunnell et al., 1983; Roussel and Buguet, 1982) reported increased mean sleeping heart rates on the day following submaximal walking exercise.

Other studies reported increased heart rates during sleep in fit, but not unfit subjects (Walker et al., 1978), reduced sleeping heart rates (Sadamoto et al., 1986) or unchanged heart rates (Sedgwick et al., 1974) after a period of 8 – 12 weeks of training. A study by Roussel and Buguet (1982) measured the heart rate during sleep of 4 volunteers. After a 5 day control period, the subjects marched for 6 consecutive days followed by an immediate 5 day recovery period. Heart rate measurements were taken during sleep over all three periods. Compared to the control and recovery period, heart rate during sleep displayed an average increase of about 10% during the exercise period.

A more recent study of four middle and long distance runners over a whole season found that the individual variability for sleeping heart rate was high (mean  $\pm$  SD of  $50 \pm 4$  beats.min<sup>-1</sup>), making the assessment of training status difficult (Schultz et al., 2000, unpublished data). The average heart rate during sleep (between 2 and 5 am) of four athletes was measured twice per week. The subjects underwent daily training at one of 6 different intensity levels, compared to regular treadmill step tests, with the training load categorized according to blood lactate concentrations (regeneration:  $\sim 1$  mmol.l<sup>-1</sup> lactate, endurance 1:  $< 1.5$  mmol.l<sup>-1</sup>, endurance 2:  $1.5 - 3$  mmol.l<sup>-1</sup>, endurance 3:  $> 3 - 6$  mmol.l<sup>-1</sup>, high-intensity:  $> 6$  mmol.l<sup>-1</sup> and competition). Training load had no significant effect on heart rate during sleep in three athletes and the variation in heart rate during sleep could only partly be explained by training load in one of the tested athletes.

Heart rate during sleep shows a measure of intrinsic variation and some studies (Brisswalter and Legros, 1994) have demonstrated changes in the day-to day variation of between 5 to 8 beats.min<sup>-1</sup> under controlled conditions during a submaximal test. If this is then the range of variability one can normally expect to find, it would suggest that changes in the minimum heart

rate during sleep would need to be greater than 10 beats.min<sup>-1</sup> to be of value in detecting changes in training status.

In conclusion, the accepted relationship between heart rate and training status may be more evident during sleep as many of the variables that may affect heart rate are minimized. The studies that have examined heart rate during sleep have used a variety of different training and testing protocols and the findings of these studies are quite diverse, with some reporting increased, decreased or unchanged heart rates. The use of palpated resting (morning) heart rate is a rather inaccurate measurement to track changes in training status, due to lack of control and especially in elite athletes where the magnitude of changes in heart rate may be quite small. Heart rate measurements during sleep under less well controlled, free-living and field testing conditions have not been researched and as this is the level of practicality one would hope to achieve when measuring heart rate and changes in training status, it is important to examine this aspect further, although the many variables influencing heart rate during sleep should certainly be taken into consideration when interpreting data.

#### **1.4.3.2.6 Heart rate variability.**

As described previously, it is widely accepted that the cardiovascular system is regulated by the autonomic nervous system and physical activity is accompanied by increased sympathetic activation and parasympathetic withdrawal, causing heart rate to increase during exercise. The autonomic nervous system again is responsible for the decreased heart rate at rest and during exercise as observed in especially endurance trained athletes. There are however, some authors who challenge the belief that the autonomic nervous system is solely responsible for

the changes in heart rate observed during and after exercise training (Bonaduce et al., 1997; Katona et al., 1982; Smith et al., 1989; Stein et al., 2000; Stein et al., 2002). All of these researchers hypothesize that in addition to the autonomic changes that take place after exercise training, intrinsic adaptations occur in the electrical conduction system, leading to a slower heart rate or athletic bradycardia.

#### *1.4.3.2.7.1 Control mechanisms.*

Several recent studies have examined the process of heart rate variability in an attempt to clarify the control mechanism of the autonomic nervous system during and after exercise training. Heart rate variability can be measured using electrocardiogram (ECG) tracings (R-R intervals), and is analyzed in time (statistical studies) and frequency domains (power spectrum) (Aubert et al., 2003). Power in the different frequency bands represents either sympathetic (0.04 – 0.15 Hz) or parasympathetic (0.15 – 0.4 Hz) activity, though the authors state that other feedback mechanisms are also involved, mostly in the low frequency band (Aubert et al., 2003). It is important that high quality ECG data be obtained and as many of the heart rate variability indices depend on the duration of the recording, recording time should be between a minimum of 10 minutes to 24 hours (Aubert et al., 2003).

#### *1.4.3.2.7.2 Technical and other measurement problems*

A problem experienced whilst measuring heart rate variability is technical in nature and has been identified by nearly all the studies on heart rate variability. Heart rate normally increases

steadily depending on the exercise intensity and a steady state is required when performing spectral analysis (Aubert et al., 2003). Other problems experienced are the fact that in the few studies on heart rate variability, the methodologies differ substantially, especially as far as training / exercise intensity is concerned and thus the interpretation of data may be skewed as well as the fact that studies performed for different time periods cannot be compared to one another (Aubert et al., 2003).

#### *1.4.3.2.7.3 Length of training period.*

Stein et al (2002) speculates that the length of time of training may be an important factor to consider when evaluating the contribution of the autonomic nervous system and the intrinsic electrical conduction adaptations to changes in heart rate. They propose that short term training may be responsible for the autonomic changes experienced. Aubert et al (2003) further speculates that long term aerobic training then may be responsible for the more intrinsic electrophysiological adaptations due to its dilatory effect on the atrium and ventricle.

#### *1.4.3.2.7.4 Autonomic Nervous System.*

A study performed by Javorka et al (2002) attempted to determine if there was an association between heart rate variability parameters and the rate of slowing of heart rate after exercise. They showed that the rate of decrease in heart rate after exercise was independent of the heart rate variability parameters during rest. However, there was a relationship between heart rate

variability parameters in the early post-exercise period and during the phase of the recovery, indicating a parasympathetic influence.

Power spectral analysis of heart rate variability was performed on a junior cross-country skier before, with symptoms of the overtraining syndrome and once recovered (Hedelin et al., 2000). They found increased heart rate variability during the overtraining period, specifically in the higher frequency spectrum, coupled with a decreased resting heart rate that led them to conclude that there was significant parasympathetic modulation in the cardiac autonomic nervous system during the overtraining phase.

In a study by Winsley et al (2005) twenty sedentary and twenty active females were subjected to a 2 week overtraining regime during which they trained seven days a week for 40 minutes per session at an exercise intensity of 70 to 90 % of heart rate reserve. In contrast to the study by Hedelin et al (2000) they found no significant changes in resting heart rate in both groups. However, heart rate variability indices (low and high frequency power) changed significantly ( $P \leq 0.05$ ) in the sedentary group when compared to the active subjects. The active group also exhibited changes in the low and high power frequency, but these were not significant, after the overreaching phase.

Portier et al (2001) also found a decreased resting heart rate variability ( $P < 0.001$ ) during a period of intensive training in 8 elite runners and concluded that spectral analysis could be a useful tool in identifying impending autonomic system imbalance and fatigue that could lead to overtraining.

Pichot et al (2002) tested 6 sedentary subjects over a period of 2 months of intensive training, 1 month of overtraining and 2 weeks of recovery. As was to be expected, performance as well as heart rate variability indices improved during the 2 months of intensive training, with the parasympathetic system initially dominant. However, during the overtraining period, there was an increase in sympathetic activity with a concomitant decrease of the parasympathetic indices. During the recovery period the parasympathetic system again increased, with all of the heart rate variability indices back to normal pre-study values after 7 weeks of recovery. They concluded that the status of the autonomic nervous system depends on the accumulation of physical fatigue due to increased training loads and that heart rate variability measurements could potentially be useful to identify changes.

#### *1.4.3.2.7.5 Age and Gender.*

Carter et al (2003 b) examined whether age and gender had any effect on heart rate variability after a 12 week running program. The subjects were divided into two groups; a young group aged 19 – 21 years and a middle-aged group of 40 – 45 years. Each group consisted of 6 males and 6 females. Both groups followed a standardized training program and heart rate variability was measured prior to the start of training and also post training. Heart rate decreased significantly in both age groups after training during rest ( $3 \pm 1$  beats.min<sup>-1</sup>) and also during submaximal exercise ( $8 \pm 1$ beats.min<sup>-1</sup>). Results indicated significant increases in heart rate variability indices in both groups.

In contrast a study by Catai et al (2002) found no significant changes in heart rate variability after training in a young (median 21 years) and older (median 53 years) group of healthy

sedentary male subjects. They also concluded that the resting bradycardia observed in both groups after short term aerobic training pointed more to an intrinsic change in the sinus node rather than changes in vagal – sympathetic modulation.

#### *1.4.3.2.7.6 Training intensity.*

The effect of training intensity on heart rate variability is still unclear. Parekh and Lee (2005) tested thirteen male subjects at two different exercise intensities; low (50%) and high (80%) of  $\text{VO}_2$  reserve on a treadmill. Electrocardiogram tracings were recorded for 5 minutes pre-exercise and for 30 minutes after completion of the test whilst in the supine position. Heart rate variability (R-R intervals) and spectral analysis was performed. Results indicated that there was less change in cardiac autonomic balance with low intensity exercise compared to high intensity exercise that displayed a faster vagal modulation restoration time. Similarly, a study by (Pigozzi et al., 2001) showed that trained subjects displayed a reduced effect on changes in heart rate at submaximal workloads.

Madden et al (2006) examined the response on heart rate variability of endurance and strength training on 45 healthy subjects in a randomized cross-training study over a period of 6 months. They found that strength training had no significant effect on heart rate variability, in contrast to endurance training that effected definite changes in most of the time domains and all of the frequency domains. They concluded that it may be possible that aerobic and strength training operate through different mechanisms to reduce the risk of cardiac mortality.

In summary, although heart rate variability may be a useful laboratory tool to attempt to clarify the respective contributions of the autonomic nervous system and intrinsic cardiac adaptations, many aspects still need to be clarified;

-The specific roles of the sympathetic and parasympathetic system within the autonomic nervous system and the intrinsic adaptations in the electrical conduction system that occur with physical training need to be clarified.

- There are still a variety of technical problems associated with measuring heart rate variability; amongst others the problem of reaching a steady state before spectral analysis can be employed, quality of electrocardiogram tracings and recording times.

- Methodologies also differ substantially and would need to be standardized before heart rate variability data between different studies can be accurately interpreted.

Aubert et al (2003) strongly suggest that any future studies include detailed descriptions as to the specific methodologies used, population group tested, training programmes, intensity and duration of training.

In conclusion, heart rate variability measurement as a tool to measure changes in heart rate and training status currently presents with numerous technical and other deficiencies and lacks the required precision and level of practicality necessary to correctly interpret data.

#### **1.4.3.2.8 Heart rate recovery.**

Heart rate increases in proportion to the exercise intensity. Upon cessation of exercise heart rate does not immediately return to its pre-exercise level, but remains slightly elevated for a

period of time before returning to its resting level. Darr et al (1988) divides heart rate recovery into two phases, fast (15 -120 seconds) and slow (120 – 240 seconds). The rate at which the recovery process occurs depends partly on the fitness level of the athlete and the exercise intensity (O'Connor et al., 1993; Pierpont and Voith, 2004). The time that it takes the heart rate to return to its resting (baseline) level is called the heart rate recovery period, but there are different definitions as to when the cut-point occurs (Carnethon et al., 2005; Jouven et al., 2005; Seshadri et al., 2004; Shetler et al., 2001).

There is still much controversy about the exact mechanism responsible for the decrease in heart rate post-exercise particularly with regards to the autonomic nervous system and the role of the parasympathetic and sympathetic system in the recovery process.

#### *1.4.3.2.8.1 Autonomic Nervous System.*

An earlier study by Savin et al (1982) attempted to clarify the contribution of the autonomic nervous system to heart rate recovery following exercise. Six male subjects were tested with four treatments after peak treadmill exercise; (i) using a parasympathetic blockade atropine sulphate, which has a direct effect on the parasympathetic nervous system by blocking the action of the neurotransmitter, acetylcholine (Lee and Leroux, 1992); (ii) a sympathetic blockade propranolol hydrochloride, which has a similar blocking effect on the sympathetic nervous system; (iii) double blockade using both drugs; and (iv) no drugs. They concluded that the exponential character of the decrease in recovery heart rate after peak exercise is due to an intrinsic factor of the circulatory system as this occurred during all four experimental treatments.

A study by Kannankeril et al (2004) examined the influence of the parasympathetic system on heart rate recovery after high intensity exercise. They defined the parasympathetic effect on the heart rate by the difference in heart rate with and without atropine. They found that the parasympathetic effect on heart rate during maximal exercise was between 3 to 6 beats.min<sup>-1</sup> ( $P < 0.05$ ). This in effect means that the parasympathetic nervous system exerts a protective effect on the heart during maximal exercise by slowing the heart rate down. The largest effect of the parasympathetic nervous system on heart rate was seen in the 1<sup>st</sup> minute of recovery (23 beats.min<sup>-1</sup>;  $P < 0.0002$ ). This contribution from the parasympathetic system increased until 4 minutes and then stabilized until 10 minutes. They concluded that the parasympathetic effect remains important especially in the early phases of recovery after high intensity exercise and that these parasympathetic effects may be important in preventing sudden death during exercise.

A longitudinal study by Carnethon et al (2005) tested whether physical activity was associated with higher parasympathetic activity in a diverse group of subjects. They defined heart rate recovery as the difference between the maximal heart rate and heart rate 2 minutes after exercise stopped to provide an estimation of parasympathetic activity. Heart rate recovery rates were measured whilst performing a symptom-limited graded exercise treadmill test at baseline and again at the end of a 7 year period. Subjects who had high levels of self-reported habitual physical activity had faster heart rate recovery rates (45 beats.min<sup>-1</sup>) compared to the subjects in the lowest tertile who were less physically active (42 beats.min<sup>-1</sup>). Overall, heart rate recovery decreased by about 3 beats.min<sup>-1</sup> over a 7 year period and decreased the least in the group whose physical activity levels had increased (-1 beat.min<sup>-1</sup>) compared to the group whose physical activity levels had decreased (- 4 beats.min<sup>-1</sup>;  $P < 0.01$ ). All of the subjects had remained physically active to some extent over the 7 year period. They concluded that regular

physical activity may be responsible for blunting the age –related decline normally seen in the autonomic nervous system.

A study by Pierpont et al (2000) applied the heart rate during recovery after exercise to a first order exponential decay curve to calculate the time constant, believed to be an index of parasympathetic activity. Results indicated that time constants only stabilized 3 minutes after exercise stopped and varied unacceptably in the first 5 seconds of monitoring following maximal exercise; however, during recovery after submaximal exercise, time constant values were much more stable. They concluded that first order decay does not have sufficient precision to measure heart rate recovery after maximal exercise but could be useful during submaximal exercise bouts.

Quantifying heart rate recovery is difficult as the rate of recovery varies depending on the exercise level (Pierpont and Voth, 2004). To this end they designed a model for heart rate recovery that may be applied to multiple exercise intensities simultaneously. They used the Levenberg-Marquardt method for nonlinear models for 4 levels of exercise, using one first order constant for parasympathetic reactivation and one first order constant for sympathetic reduction. Results indicated a parasympathetic reactivation time constant of  $44 \pm 37$  seconds i.e the parasympathetic system reacted faster and started influencing changes in heart rate before the sympathetic system withdrawal at  $65 \pm 56$  seconds. The designed model was also able to explain up to  $99.7 \pm 0.1\%$  of variance in the data. This model may assist in further refining and quantifying heart rate recovery and the distinct coordinative roles of the parasympathetic and sympathetic systems.

#### *1.4.3.2.8.2 Training Status and other variables.*

It is generally accepted that especially endurance trained athletes have a much quicker heart rate recovery than untrained subjects and that this pertains to both submaximal and maximal exercise (Wilmore and Costill, 1994). However there are other factors, other than training status, that may affect heart rate recovery times such as environmental factors, altitude and altered sympathetic nervous system responses (Wilmore and Costill, 1994).

Slow heart rate recovery rates were also identified in older individuals after exercise. Darr et al (1988) investigated this observation and designed a study in which the level of fitness and work intensity were controlled in four different groups, divided according to age and fitness level (young trained, old trained, young untrained, old untrained). No age dependent effects were identified in any of the groups after a regression analysis of the fast (15-120 seconds) and slow (120-240 seconds) recovery heart rates. The trained subjects however, all showed a faster heart rate recovery ( $P < 0.005$ ) within the first minute regardless of age. These results indicate that the slower heart rate recovery identified in older subjects may have been due to other variables that were not controlled for.

#### *1.4.3.2.8.3 Heart rate recovery and sudden death.*

Recently heart rate recovery has been described as a potential indicator for sudden death syndrome, associated with physical activity. As changes in heart rate during exercise and recovery are normally mediated by the autonomic nervous system (sympathetic and

parasympathetic), any changes in the neural control of the heart may lead to a risk of sudden death (Jouven et al., 2005).

Slow heart rate recovery time after exercise, possibly due to reduced parasympathetic activity, may also be indicative of an increased risk of all-cause mortality although the mechanism remains unclear (Huang et al., 2005). Seshadri et al (2004) defines a slow or abnormal heart rate recovery as a decrease in heart rate of less or equal to 12 beats.min<sup>-1</sup> within the first minute after cessation of exercise. It is also only recently that heart rate recovery was seen to have prognostic value apart from its value as an indicator of training status. However, several issues about the relationship between heart rate recovery and sudden death remain unresolved. For example, the effect of beta blockers, the most appropriate cut-point at which to assess the magnitude of the decrease in heart rate and the optimal time point at which to measure heart rate recovery still needs to be resolved (Shetler et al., 2001).

A study by Shetler et al (2001) determined that the best prognostic cut-point to measure heart rate recovery was at 2 minutes with a value of 22 beats.min<sup>-1</sup> being optimal at that time point. However, their study also failed to explain the pathophysiology of an abnormal heart rate recovery. Initially vagal reactivation was presumed to be the main factor determining the rate of decrease in heart rate during the first 30 seconds of recovery, however, subjects with a slow heart rate recovery rate also presented with lower exercise workloads, shorter duration of exercise and fewer subjects attaining 90% of their target heart rate (Huang et al., 2005).

According to Jouven et al (2005) subjects are at greater risk from sudden death due to myocardial infarction when presenting with abnormal heart rate profiles during exercise and recovery, even in apparently healthy subjects. After testing 5713 apparently healthy subjects, 81 subjects died suddenly during the 23-year follow-up period. The subjects at increased risk

presented with resting heart rates of more than 75 beats.min<sup>-1</sup>, an increase in heart rate during exercise of less than 89 beats.min<sup>-1</sup> and a recovery heart rate decrease of less than 25 beats.min<sup>-1</sup> after cessation of the exercise bout. Heart rate recovery was defined as the decrease in heart rate from the peak heart rate attained during the exercise bout to the heart rate at one minute after exercise stopped. This led them to conclude that the heart rate profile during exercise and recovery may be a potential indicator of sudden death.

The short term test-retest reproducibility of heart rate recovery was examined in a study by Yawn et al (2003) who studied the retrospective data of 90 patients who underwent 2 exercise stress tests within an 18 week period. They defined an abnormal heart rate recovery as a decrease of less than 12 beats.min<sup>-1</sup> within the first minute after the cessation of exercise. The results showed great individual variability between the first and second test with less than 55 % agreement between the two tests regardless of the definition of abnormal heart rate recovery used. They concluded that heart rate recovery has limited short test-retest reproducibility. However, this preliminary data was obtained from a retrospective comparison of medical information, is the only study currently available and long term stability under more controlled conditions have not been tested.

In summary, heart rate recovery values may thus not only be indicative of a subject's training status but may also be of value in identifying individuals at risk of sudden death or altered parasympathetic and sympathetic nervous system function. During periods of overtraining for example, an increased sympathetic and decreased parasympathetic system response is noted, leading to an increased heart rate that may lead to an altered heart rate recovery pattern.

- The exact mechanism and pathophysiology of slow or abnormal heart rate recovery still remains to be resolved. The mechanism for the decrease in heart rate after exercise as a function of the autonomic nervous system is still controversial with different findings.
- There are many factors that may affect heart rate recovery, such as exercise intensity, training status, environmental factors, individual variability and altitude that should be controlled for when measuring and interpreting heart rate recovery.
- The optimal value and precise cut-point also need to be clarified as well as to how all of the information regarding heart rate recovery may be applied to medical patients compared to an athletic population.

#### **1.4.3.2.9 Potential of using heart rate as a marker of training status.**

Although numerous studies have examined the effect of exercise training and heart rate (Bevier et al., 1987; Blomquist and Saltin, 1983; Bunnell et al., 1983; Horne, 1981; Karvonen and Vuorimaa, 1988; Roussel and Buguet, 1982) and other studies have examined the relationship between morning resting heart rate patterns and training status and overtraining, (Callister et al., 1989; Dressendorfer et al., 1985; Wilmore et al., 1996), there is no consensus on whether heart rate can predict training status with any degree of accuracy. However, it remains important that when heart rate is used to determine the training status of an athlete, that the heart rate / workload relationship be established for that individual athlete using an incremental test (Arts and Kuipers, 1994).

The varied response of the heart rate / workload relationship under differing conditions as well as the many factors that may influence this relationship makes it extremely difficult to

conclusively prove that this relationship does indeed have the capacity to accurately reflect changes in training status. However, as it is easy and relatively cheap to measure heart rate accurately, compared to the other methods described previously to measure changes in training status, heart rate remains the most viable practical method to potentially measure changes in training status. The test-retest repeatability of measuring heart rate during submaximal and maximal exercise under controlled conditions has been reported to be  $r = 0.96$  to  $0.98$  (Lambert et al., 1998; Selley et al., 1995) and  $r = 0.86$  (St Clair Gibson et al., 1998) respectively, which means that the test has sufficient precision to detect relatively small changes.

The biggest challenge that remains is to define and refine the interpretation of the heart rate data collected so that the information gleaned is accurate, reliable and of practical use to athletes and coaches.

To this end the submaximal heart rate test used throughout this thesis was conceptualized and designed at the Sports Science Institute of South Africa (Lamberts et al., 2004). The design of the test incorporates the principle of the linear heart rate / workload / oxygen consumption relationship as set out by Arts and Kuipers (1994). The test has a high repeatability ( $r = 0.94$  to  $0.99$ ) for submaximal and heart rate recovery on a day-to-day basis as demonstrated by Lamberts et al (2004). The test is designed to be submaximal in nature as several studies (Ballarin et al., 1996; Conconi et al., 1996; Gilman, 1996) have identified the fact that the heart rate / workload and oxygen consumption principle becomes unstable and less defined at workloads higher than 90% of  $VO_2$ max. Furthermore, the test was designed to be non-aversive to athletes as it ideally needs to be done at regular intervals. Lastly it was important that the

test was easy to administer, inexpensive and practical and can be performed both in the laboratory and field testing situation.

The test has four stages of two minutes each of increasing workload, designed to progressively increase the heart rate of the athlete, interspersed with four recovery phases of one minute duration each. Heart rate is measured every 5 seconds for the duration of the test using portable heart rate monitors. The stages are separated by the rest period to allow some recovery and to maintain the submaximal quality of the test. As the workloads are fixed at pre set levels for every stage (8.4, 9.6, 10.8, and 12.0 km.hour<sup>-1</sup>) it can be used on a regular basis to compare changes in fitness levels. The fourth and last recovery period is designed so that heart rate recovery may be measured for a period of one to two minutes after cessation of the test. Heart rate recovery is controlled by the autonomic nervous system through the relationship of the sympathetic and parasympathetic system to one another, although the exact mechanism is still unclear. The heart rate recovery percentage can also be calculated by subtracting the lowest heart rate (beats.min<sup>-1</sup>) attained during the last one minute recovery period from the highest heart rate (beats.min<sup>-1</sup>) achieved during the fourth stage, divided by the highest heart rate (beats.min<sup>-1</sup>) during stage 4 x 100. The % HRmax, maximal, minimum and mean heart rate for the test can be determined.

One test thus has the potential to provide the athlete and coach with a multitude of information about the athletes' heart rate response to exercise that is readily available and does not necessarily require expensive facilities, equipment or expertise.

## 1.5 SUMMARY.

Competition at the elite level is particularly intense in the quest for fame and fortune. As sport has progressed through the ages, mostly due to improved scientific training methodology and equipment, so the difference between individual top athletes have diminished.

As athletes become more highly trained, the magnitude of changes in performance decreases. This essentially means that a small improvement in performance might be significant in the race for gold, but it also means that it becomes more difficult to track or monitor these minute changes in training status. The margin of error is thus small when measuring training status and for an elite athlete the precision of tests to monitor training status is non-negotiable. The majority of elite athletes in the developed world will have access to laboratory-based physiological testing, but this is not universally the case and field-testing will remain the only option for the rest of the elite athletes and for the general athletic population at large. Methods of physiological field-testing, therefore, need to be precise, valid, reliable, robust, inexpensive and practical.

Individuals however, present with different training adaptations to similar training stimulus and this confounds the problem of identifying the optimal training load for each individual athlete. Generally, a progressive increase in training load in conjunction with sufficient rest and recuperation periods seem to be the desired recipe, keeping in mind individual responses. If this balance is not optimal however, overreaching or symptoms of the overtraining syndrome may occur, leading to decrements in performance.

There are a multitude of methods currently available to monitor changes in training status. However, generally, there are a lack of appropriate and valid diagnostic tools, great variability in the results of different studies, often studies are poorly controlled and the very specific individual reactions of athletes to training makes it difficult to identify the optimal testing method.

Methods currently in use to monitor training status include measuring blood borne (biochemical and hormonal), psychological (POMS and RPE) and physiological markers (VO<sub>2</sub>max and heart rate) markers. Whilst most of these tests are sufficiently precise to be of value in measuring changes in training status, some require specialized laboratory facilities and staff, are expensive and not practical enough to be of use in field-testing. A summary of the methods to monitor training status with a checklist of their characteristics are shown in Table 1.1.

**Table 1.1 Summary of different methods and specific characteristics used to monitor training status.**

		Precision	Cost effectiveness	Field-testing	Practical
<b>Blood borne markers.</b>	Biochemical	Moderate	Low	Low	Low
	Hormonal	Moderate	Low	Low	Low
<b>Psychological markers.</b>	POMS	High	High	High	Moderate
	RPE	High	High	High	High
<b>Physiological markers.</b>	VO <sub>2</sub> max	High – untrained Low - trained	Low	Low	Low
	Heart rate	High	High	High	High

As can be seen in the above summary, heart rate and RPE are the methods that are most likely to be of use for the majority of athletes, regardless of level of training status. Various different aspects of heart rate, heart rate recovery and the effect of exercise training have been examined by numerous studies over the years. The test-retest repeatability of measuring heart rate during maximal and submaximal exercise under controlled conditions have been shown to be high, which makes it extremely useful to detect even relatively small changes, as is often found in elite athletes.

There are however, some factors that may affect the accepted heart rate / workload relationship and which makes it difficult to conclusively prove that this relationship does indeed have the capacity to be an accurate measure of changes in training status. It is imperative that these factors be studied further to enhance the use of an already well established method.

Measuring heart rate remains an easy, viable, practical and inexpensive method to accurately measure changes in training status, compared to the other methods currently employed. Heart rate measurements are already commonly used on a daily basis by athletes and coaches alike to monitor changes in training status, exercise intensity and for exercise prescription. However, the interpretation of the data are at this stage incomplete. Further studies are needed to refine the interpretation of the heart rate data collected so that coaches and athletes are able to gain maximally from this information. Accordingly, the collective aim of the studies in this thesis is to provide further information so that heart rate data can be interpreted with a greater degree of understanding.

# CHAPTER 2

**HEART RATE DURING SLEEP:  
IMPLICATIONS FOR MONITORING  
TRAINING STATUS**

University of Croydon

## 2.1 ABSTRACT

Changes in resting heart rate have been associated with changes in training status. It is reasonable to assume that the relationship between heart rate and training would be more evident during sleep when the extraneous factors that may affect heart rate are reduced. Therefore the aim of the study was to assess the repeatability of monitoring heart rate during sleep when training status remained unchanged, to determine if this measurement had sufficient precision to be used as a marker of training status. The heart rate of ten female subjects ( $35 \pm 8$  years) was monitored for 24 hours on three occasions over three weeks. Average, minimum and maximum heart rate during sleep and day- time activities was calculated. The average heart rate of the group during sleep was similar on each of the three tests ( $65 \pm 9$ ,  $63 \pm 6$  and  $67 \pm 7$  beats.min<sup>-1</sup> respectively). However, the range in minimum heart rate variation during sleep for all subjects over the three testing sessions ranged from 0 to 10 beats.min<sup>-1</sup> (mean =  $5 \pm 3$  beats.min<sup>-1</sup>), with the range for the maximum heart rate variation being 2 to 31 beats.min<sup>-1</sup> (mean =  $13 \pm 9$  beats.min<sup>-1</sup>). The intraclass correlation coefficient (ICC: 95% confidence intervals) for average, minimum and maximum heart rate during sleep were  $r = 0.91: 0.76 - 0.97$ ;  $r = 0.92: 0.79 - 0.98$  and  $r = 0.55: 0.17 - 0.84$  respectively. On an individual basis the minimum heart rate during sleep varies by about 8 beats.min<sup>-1</sup>. This amount of intrinsic day-to-day variation needs to be considered when changes in heart rate that may occur with changes in training status are interpreted.

## 2.2 INTRODUCTION

As discussed in the literature review (Chapter 1), the accurate measurement and interpretation of training status is of extreme importance.

Studies have examined the relationship between morning resting heart rate patterns and training status and overtraining (Callister et al., 1989; Dressendorfer et al., 1985; Wilmore et al., 1996). Several studies have examined heart rate during sleep and its relationship to training status (Bevier et al., 1987; Bunnell et al., 1983; Jeukendrup et al., 1992; O'Connor et al., 1993; Roussel and Buguet, 1982).

Jeukendrup et al (1992) suggested that measuring heart rate during sleep may be a more viable marker of overtraining compared to palpated morning resting heart rate and as such may be a more accurate assessment of changes in heart rate. In their study on seven male competitive cyclists the palpated morning heart rates did not change after a two-week period of intensified training, while the mean heart rate during sleep showed a significant ( $P < 0.05$ ) increase and as such may be a viable indicator of imminent overtraining. Some longitudinal studies have found heart rate during sleep to be reduced (Sadamoto et al., 1986) or unchanged (Sedgwick et al., 1974) after several weeks of exercise training. It is logical to assume that if there is a relationship between heart rate and training status that this will be more evident during sleep when extraneous factors that may influence heart rate are reduced.

However, even though heart rate during sleep may seem to be of value as an indicator of training status when compared to that of palpated morning heart rate values, the many variables influencing heart rate during sleep should be taken into consideration when

interpreting data. The results from all the studies are fairly diverse and further studies are needed to clarify the specific relationship between heart rate during sleep and training status.

Therefore, the aim of this study was to assess the repeatability of measuring heart rate during sleep under free-living but controlled conditions in a group of subjects whose training status did not change over a period of three weeks. The results from this study will serve as a guide for the next step in developing a protocol for monitoring training status.

## **2.3 METHODOLOGY**

### *2.3.1 Subjects*

Ten female subjects between the ages of 18 to 45 years ( $35 \pm 8$  years) were recruited to participate in the study through advertising at a local health club.

Each potential participant completed a Health Screen/Activity Questionnaire with questions on personal details, medical history, physical activity and sleep profiles. This was done to ensure eligibility of subjects for the study, to exclude any potential variables such as certain medications that may influence heart rate and to gather information regarding the subject's training status and general sleep profile.

All subjects were pre-menopausal, regular exercisers with varying levels of training ( $7 \pm 3$  hrs.week<sup>-1</sup>), and with no known cardiac pathology. Subjects trained on average  $4 \pm 1$  times per week and had done so for more than five years. Subjects were mostly recreational athletes

and participated in aerobic type training (circuit, treadmill, aerobics, cycling and swimming).

Two of the athletes competed at a provincial level in speedwalking and long distance running respectively. Four subjects also did some weight training as part of their general exercise program. The subjects' level of fitness was not tested at the start of the study.

None of the subjects smoked although two subjects had previously smoked. None of the women were on any medication that could influence their heart rate. The only inclusion/exclusion criteria specified was that subjects should be between the ages of 18 and 45, with no known cardiac pathology.

A resting electrocardiogram (ECG) was performed on each subject prior to acceptance into the study to exclude any subjects with unknown cardiac pathology.

Prior to the start of the trial an introductory session was held with each participant during which the aim and protocol of the study was explained. All subjects gave their informed consent. The study was approved by the Research Ethics Committee of the University of Cape Town.

### *2.3.2 Experimental design*

The trial consisted of a 24-hour monitoring session conducted on three occasions over a three-week period. Monitoring took place on the same day of each week. Each session consisted of a 24 hour monitoring day that started upon waking before the subject got out of bed. The subjects fitted and activated the heart rate monitors that were set up next to their bed before getting out of bed. Heart rate was recorded every minute over the 24-hour period (in total 1440 min) and stored in the heart rate monitor for analysis at a later stage (Polar Vantage XL,

Kempele, Finland). The highest and lowest heart rate recorded during this period was defined as the maximum and minimum heart rate respectively. The starting time of sleep was determined through self-reporting by the subjects and verified by checking the trend of the heart rate during the first 30 minutes prior to the start of the reported sleep time.

Subjects were encouraged to maintain their normal lifestyle whilst being monitored and were also requested to replicate their activity and dietary intakes as closely as possible on each of the three monitoring sessions by using the activity and nutritional records which were given to them as reminders at the orientation session. The subjects' compliance to the protocol was assessed at each testing session by comparing records of the previous week to the current week and discussing this with the subject. None of the subjects reported any extreme deviations from the dietary intake and training sessions during the study. After checking, the subjects were given a copy of the record and reminded to replicate this at the next test session. Subjects were requested not to change their training program in any way during the trial. This was verified through means of the activity records kept.

Subjects self-selected the day of testing to fit in with their own lifestyle. They were then however compelled to test on the same day for the duration of the testing. The study was designed to represent a typical field study situation. Therefore the conditions of the study were matched to the conditions in which the monitoring would usually take place with athletes.

### 2.3.3 STATISTICAL ANALYSIS

Results are expressed as mean  $\pm$  standard deviation (SD) unless otherwise noted. Heart rate data outside the ranges  $< 30$  and  $> 220$  beats.min<sup>-1</sup> were accepted as noise and were excluded

from analysis. The average, minimum and maximum heart rate variation during sleep for each of the ten subjects was calculated for each of the three test sessions. The average, minimum and maximum heart rate during sleep and during the daytime for the group was calculated for each of the three trials. The measurement error, defined as the within subject coefficient of variation, [(standard deviation/mean) x 100] was calculated for the average, minimum and maximum heart rate during sleep. An analysis of variance (ANOVA) with repeated measures was used to determine if there were any differences for the variables on each of the testing days. The intraclass correlation coefficient (ICC) and 95% confidence interval was calculated for the average, minimum and maximum heart rate for the three sessions for all subjects and divided into sleep and daytime periods. The limits of agreement for minimum heart rate was calculated for tests 1 and 2, tests 2 and 3 and tests 1 and 3 (Bland and Altman, 1986). Statistical significance was accepted when  $P < 0.05$ .

## **2.4 RESULTS**

Subject characteristics such as age, mass, blood pressure, physical activity ( $\text{hr}\cdot\text{wk}^{-1}$ ) and sleep ( $\text{hr}\cdot\text{night}^{-1}$ ) are described in Table 2.1.

**TABLE 2.1 Descriptive characteristics of subjects (n = 10).**

<b>Variable.</b>	<b>Mean <math>\pm</math> SD</b>
Age (years)	35 $\pm$ 8
Mass (kg)	62 $\pm$ 9
Systolic BP (mmHg)	112 $\pm$ 13
Diastolic BP (mmHg)	70 $\pm$ 13
Physical Activity $\blacktriangle$ (hr.wk <sup>-1</sup> )	7 $\pm$ 3
Sleep $\bullet$ (hr.night <sup>-1</sup> )	7 $\pm$ 1

All values are expressed as Mean  $\pm$  Standard Deviation.

$\blacktriangle$  Physical activity denotes actual time spent training as reported by the subjects in the questionnaire.

$\bullet$  Hours sleep per night as reported by the subjects in the questionnaire.

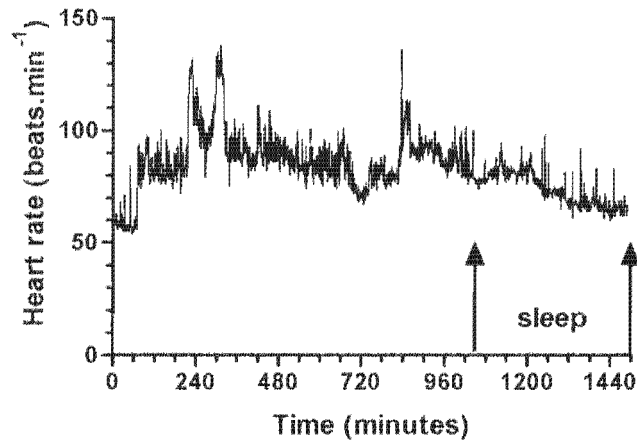
Self-reported physical activity levels and average sleeping time ranged from 1 to 12 hours per week and 6 to 9 hours per night respectively. Records of current exercise times were compared to hours reported on the initial questionnaire and found to correlate. Subjects reported a mean total in-bed time of 467  $\pm$  27 minutes for the three test sessions. Total in-bed time was different for each of the three test sessions at 472  $\pm$  52, 493  $\pm$  39 and 437  $\pm$  50 minutes respectively (Table 2.2). Intra-subject variability of in-bed time between sessions was high. The average difference between the highest and lowest amount of sleep for each subject was 17  $\pm$  9 %.

TABLE 2.2 Total minutes in-bed time (uncorrected) per subject per session (n = 10).

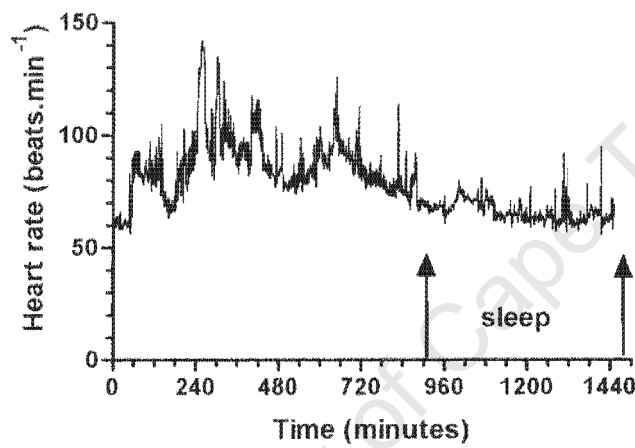
Subject	Day 1.	Day 2.	Day 3.	Mean $\pm$ SD
1	447	560	397	468 $\pm$ 84
2	540	502	551	531 $\pm$ 26
3	452	461	378	430 $\pm$ 46
4	512	500	390	467 $\pm$ 67
5	467	507	445	473 $\pm$ 31
6	511	461	419	464 $\pm$ 46
7	523	447	434	468 $\pm$ 48
8	392	456	461	436 $\pm$ 38
9	393	551	422	455 $\pm$ 84
10	480	481	470	477 $\pm$ 6
Mean $\pm$ SD	472 $\pm$ 52	493 $\pm$ 39	437 $\pm$ 50	467 $\pm$ 27

Heart rate data of 504 minutes in total for all subjects and sessions were excluded during analysis as the values fell outside the range of 30 to 220 beats.min<sup>-1</sup>. This represents a mean loss of data of  $1 \pm 1\%$  (n = 30). No specific pattern could be identified to explain lost heart rate data. The average heart rate during the 3 days, preceding the 3 nights during which heart rate was recorded during sleep was  $83 \pm 10$ ,  $83 \pm 5$  and  $87 \pm 8$  beats.min<sup>-1</sup> respectively. There were no differences between any of these testing days. The minimum and maximum heart rate during the day was also similar for all three testing days. Figure 2.1 depicts a sample of the raw tracing of heart rate recorded over 24 hours for one of the subjects. Similar tracings were recorded for all 10 subjects.

## SESSION 1.



## SESSION 2.



## SESSION 3.

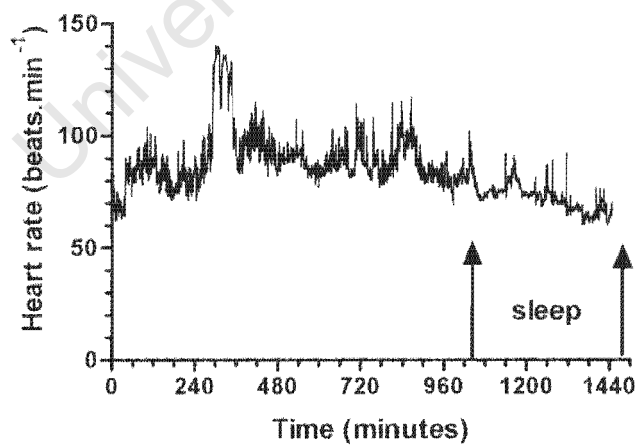


Figure 2.1 A sample of the raw tracings of heart rate (beats.min<sup>-1</sup>) recorded for each of the three sessions over 24 hours for subject 1.

Table 2.3 describes the average, minimum and maximum heart rate during sleep for each of the ten subjects for each of the three sessions. There were no significant differences in these measurements between the three test days. Minimum heart rate during sleep varied from 36 to 65 beats.min<sup>-1</sup> (mean = 53 ± 7 beats.min<sup>-1</sup>). Maximum heart rate during sleep varied from 82 to 116 beats.min<sup>-1</sup> (mean = 99 ± 8 beats.min<sup>-1</sup>). The range for minimum heart rate variation for all the subjects over the 3 testing sessions was 0 to 10 beats.min<sup>-1</sup> (mean = 5 ± 3 beats.min<sup>-1</sup>), with the range for average heart rate variation between 1 and 10 beats.min<sup>-1</sup> (mean = 6 ± 3 beats.min<sup>-1</sup>). The range for maximum heart rate showed a greater variation of 2 to 31 beats.min<sup>-1</sup> (mean = 13 ± 9 beats.min<sup>-1</sup>). There were no differences for any of the heart rate measurements. The measurement error was 5 ± 3 % for minimum, 5 ± 3 % for average and 7 ± 5 % for maximum heart rate during sleep.

**TABLE 2.3 Descriptive statistics of the average, minimum and maximum heart rate variation (beats.min<sup>-1</sup>) for sleep for each of the three sessions for each of the ten subjects.**

Variable	Average Heart Rate			Minimum Heart Rate			Maximum Heart Rate		
	Session 1	2	3	Session 1	2	3	Session 1	2	3
Subject 1	74	66	72	60	56	60	98	95	92
Subject 2	45	53	51	36	39	42	82	113	85
Subject 3	66	59	69	56	52	56	101	98	90
Subject 4	69	63	65	59	53	56	106	107	105
Subject 5	63	63	64	53	53	53	94	116	100
Subject 6	73	76	77	57	64	65	106	100	104
Subject 7	62	62	65	51	50	53	103	97	91
Subject 8	65	66	69	47	52	57	100	104	107
Subject: 9	70	69	66	58	57	55	89	100	94
Subject: 10	58	58	67	47	48	40	91	100	110

Table 2.4 describes the average, minimum and maximum heart rates during sleep and during the day for each of the three sessions for all the subjects. Average and minimum heart rate variation during sleep for all three sessions were similar with greater variation for maximum heart rate. Average, minimum and maximum heart rates during the day were similar for all three sessions.

**TABLE 2.4 Descriptive statistics of the average, minimum and maximum heart rate (beats.min<sup>-1</sup>) for sleep and during the day for each of the three sessions (n = 10 subjects).**

Variable	Session	Sleep	Day
Average Heart Rate	1	65 ± 9	83 ± 10
	2	63 ± 6	83 ± 5
	3	67 ± 7	87 ± 8
Minimum Heart Rate	1	52 ± 7	55 ± 5
	2	52 ± 6	56 ± 5
	3	54 ± 8	57 ± 8
Maximum Heart Rate	1	97 ± 8	148 ± 24
	2	103 ± 7	148 ± 21
	3	98 ± 9	158 ± 30

All values are expressed as Mean ± Standard Deviation.

Minimum heart rate during sleep for the group had the highest intraclass correlation coefficient value (ICC = 0.92 : 95 % CI of ICC: 0.79 – 0.98) and maximum heart rate during sleep had the lowest value (ICC = 0.55 : 95 % CI of ICC: 0.17 – 0.84) (Table 2.5).

**TABLE 2.5 Intraclass Correlation Coefficient and 95% confidence intervals for the average, minimum and maximum heart rates for the three sessions for each of the 10 subjects. The data are divided into sleep and day time.**

Variables	r for Sleep		r for Day Time	
		(95% C.I.)		(95% C.I.)
Average Heart Rate	0.91	(0.76 – 0.97)	0.73	(0.41 – 0.92)
Minimum Heart Rate	0.92	(0.79 – 0.98)	0.79	(0.52 – 0.94)
Maximum Heart Rate	0.55	(0.17 – 0.84)	0.84	(0.61 – 0.95)

The limits of agreement (LOA) for the minimum heart rate for tests 1 and 2, 2 and 3, and 1 and 3 were  $0 \pm 4.1$ ;  $-1.3 \pm 3.9$  and  $-1.3 \pm 5.3$  beats.min<sup>-1</sup> respectively (Figure 2.2).

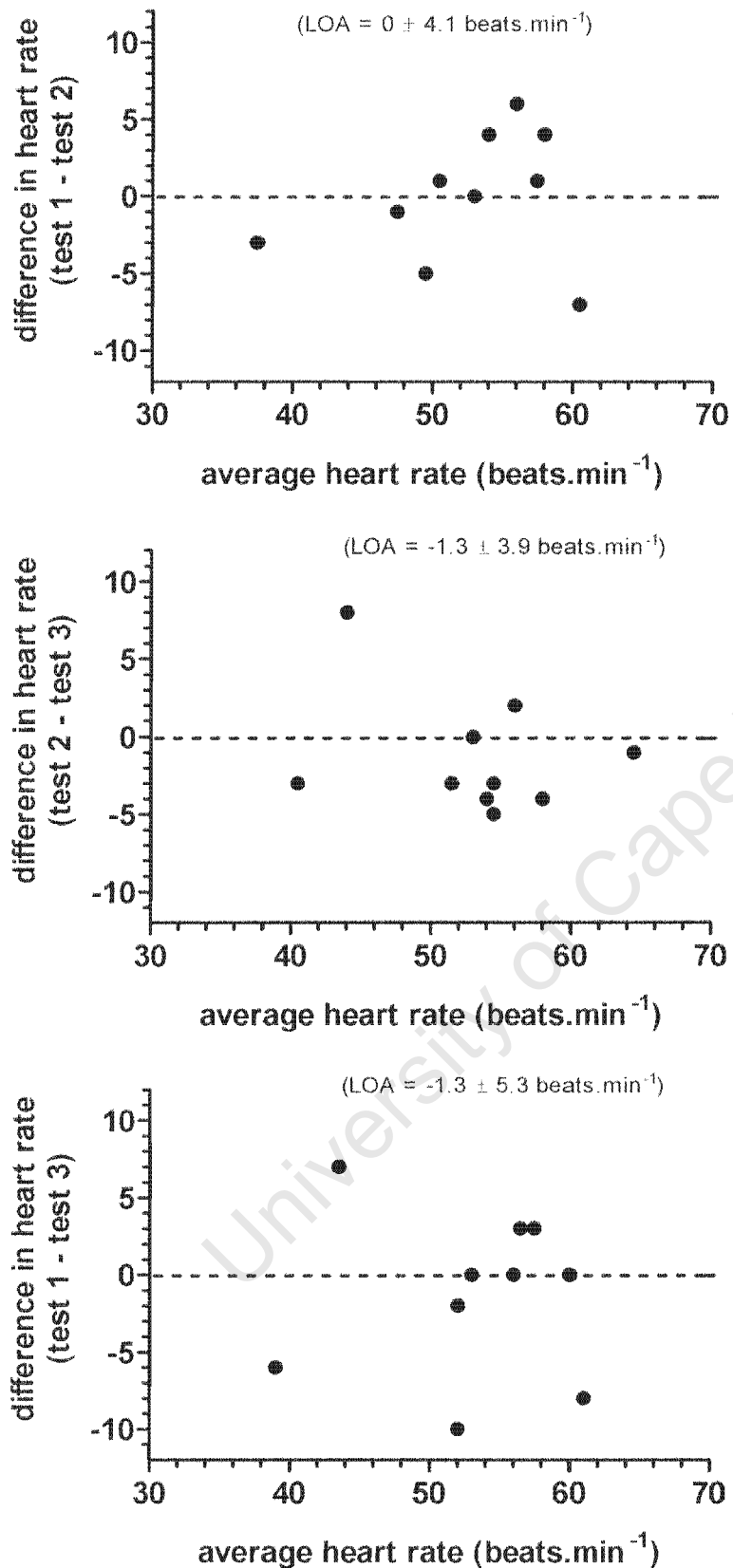


Figure 2.2 The limits of agreement (LOA) (Bland and Altman, 1986) for minimum heart rate during sleep for test 1 vs. 2, test 2 vs. 3 and test 1 vs. 3.

## 2.5 DISCUSSION.

The aim of this study was to assess the repeatability of heart rate during sleep to determine whether this measurement had sufficient precision to use as a marker of training status. An important factor to consider before changes in heart rate data can be interpreted with any degree of accuracy is knowing the intrinsic day-to-day variation in heart rate during sleep. In accordance with this aim the study was designed to determine the intrinsic variation of heart rate during sleep on three different testing occasions within a short period of time, during which the training status of the subjects remained unchanged. Although training status was not measured in this study it can be assumed that it did not change as the subjects were requested to keep their training habits constant during the 3 weeks of the study. Their compliance to training was verified whenever they were tested. All of the subjects had been following a set exercise regime for several years and none were competing at the present time even though five of the subjects had previously competed at a provincial level in their chosen sport.

The most important finding of this study was that monitoring changes in heart rate during sleep has precision to detect changes in heart rate during sleep of about 8 beats.min<sup>-1</sup>. This was calculated by determining the range of the minimum heart rate during sleep for each of the subjects over each of the three testing sessions. This suggests that changes in the minimum heart rate during sleep needs to be greater than about 10 beats.min<sup>-1</sup> to be detected with any degree of confidence. A study by Lambert et al (1998) found that the day-to-day variation in heart rate under controlled, submaximal exercise conditions was 6 beats.min<sup>-1</sup>. A more recent study by Lamberts et al (2004) found similar values with the day-to-day heart rate variation being about 5 to 8 beats.min<sup>-1</sup> under controlled conditions during a submaximal test.

Although the intraclass correlation coefficient (average heart rate during sleep) was 0.91 (95% C.I. of 0.76 – 0.97) suggesting that the measurement has “good reliability” (Vincent, 1995), from a clinical perspective it is debatable whether this is sufficiently precise to detect relatively small changes in heart rate during sleep that may occur with a change in training status.

The study design may be criticized for the lack of rigor in controlling factors that may have influenced the heart rate. However, the aim of the study was to determine whether heart rate during sleep could be monitored as a marker of training status. Any practical monitoring system needs to be done under relatively free-living conditions. Therefore the subjects were requested to replicate their activities, including sleep as closely as possible on the three monitoring occasions. Subjects were able to replicate their training activities accurately but were less successful in maintaining their exact sleeping habits. No information was obtained on sleeping conditions. This is the degree of control that one might expect in a situation where athletes are monitored under free-living conditions.

It may be argued that the repeatability would have been better had the subjects been tested in a controlled laboratory environment and had their stage of the menstrual cycle been controlled. However, several studies (Gamberale et al., 1975; Jurkowski et al., 1981; Petrofsky et al., 1976; Princi et al., 2005) found no difference in heart rate during the different phases of the menstrual cycle and thus the researchers did not control for this variable as this would then lack the practical relevance that is needed for athletes.

Heart rate spiked above 90 beats.min<sup>-1</sup> intermittently throughout sleep for short periods in all subjects. These higher heart rate ranges may have been due to positional changes or may be related to the normal circadian pattern and rapid eye movement (REM) sleep periods. Pickering

(1988) noted that circadian rhythms in heart rate and blood pressure could be affected by factors such as sleep, posture, food ingestion and activity.

In summary, on an individual basis minimum heart rate during sleep varies by about 8 beats.min<sup>-1</sup> in a group of moderately aerobically trained subjects. This amount of intrinsic day-to-day variation needs to be considered when changes in heart rate that may occur with changes in training status are interpreted.

Following on from this study, the next chapter examines the heart rate during sleep in a national class powerlifter under free-living, but well controlled conditions, over a period of 18 weeks during which time her training status changed. The aim is to further examine the implications of measuring heart rate during sleep and its relationship to training status, so that decisions can be made about a research protocol to monitor changes in training status.

# CHAPTER 3

**REPEATABILITY OF MEASURING  
HEART RATE DURING SLEEP: A  
CASE STUDY**

University of Pretoria

### 3.1 ABSTRACT

The aim of this study was to determine whether changes in heart rate during sleep under free-living but well controlled conditions tracks changes in performance in a subject whose training status changed over a period of 18 weeks. The heart rate during sleep of a national class powerlifter was measured once a week during which time her training status changed significantly. During the study the training status, as determined by measuring the 1 RM (repetition maximum) improved by 79 % for the squat (59 pre vs. 105 kg post), 70 % for the deadlift (71 pre vs. 120 kg post) and 46 % for bench press (48 pre vs. 71 kg post). Average heart rate during sleep was  $66 \pm 7$  beats.min<sup>-1</sup> during week 1 and  $67 \pm 8$  beats.min<sup>-1</sup> during week 18, with the average sleeping heart rate for the total test period of 18 weeks  $66 \pm 2$  beats.min<sup>-1</sup>. The lowest average sleeping heart rate was 61 and the highest average was 70 beats.min<sup>-1</sup>. In summary, measuring heart rate during sleep under free-living conditions was repeatable but did not track changes in training status in a national class powerlifter. This may be due to the fact that either; i) heart rate changes are less pronounced in strength type training compared to endurance training; or ii) the changes in heart rate during sleep lacks the precision to track changes in performance associated with increased fitness.

## **3.2 INTRODUCTION**

Changes in heart rate during sleep may be a viable marker of training status as there is less influence from extraneous factors that could influence heart rate. The previous study (Chapter 2) has shown that on an individual basis heart rate varies by about 8 beats.min<sup>-1</sup> in a group of moderately trained subjects, over a short 3-week period when training status did not change. Heart rate during sleep has been extensively discussed in Chapters 1 and 2.

The aim of this study was therefore to determine whether changes in heart rate during sleep tracks changes in performance associated with changes in fitness. A national level powerlifter was studied over 18 weeks, under free-living but well controlled testing conditions when training status changed. This study serves as a pilot to determine whether there is sufficient evidence to justify a more in depth study on heart rate during sleep in subjects undergoing training.

## **3.3 METHODOLOGY**

### *3.3.1 Subject*

The subject, a 45 year old female, volunteered to participate in a study during which she was tested weekly over an 18 week period. The subject was a national class powerlifter and during this period followed her own normal training programme leading up to the national championships. The subject has never smoked and was not any medication that could have affected her heart rate. A previous resting electrocardiogram (ECG) was normal and excluded any cardiac pathology.

The subject completed a health questionnaire / activity profile and gave written informed consent prior to the start of testing. The study was approved by the Ethics and Research Committee of the Faculty of Health Sciences of the University of Cape Town.

### *3.3.2 Experimental Design*

The subject had an eight week detraining period before the start of the study. The trial consisted of a weekly heart rate monitoring session during sleep for a period of 18 weeks. The first heart rate monitoring session took place four days after the first training session and the last heart rate monitoring session four days after the last training session prior to the championships.

The subjects' training status was measured at the start and completion of the study by using an estimated 1 RM (repetition max) measurement as described in Baechle et al (2000) for each of the three powerlifts (squat, bench press, deadlift). The subject trained 3 times per week throughout the study, except for weeks 4, 12 and 18, which were rest weeks as per the periodization plan and week 11 when the subject was away on holiday.

#### *Heart rate monitoring during sleep*

Heart rate monitoring took place once a week for the duration of the study. The day was self selected by the subject and all monitoring took place on the same day for the duration of the study. The same heart rate monitor was used for all the monitoring sessions and was not used for any other subject. Monitoring started at the same time each night at 20h00 and terminated

at the same time each morning at 07h00 before getting out of bed. The subject activated the heart rate monitor at 20h00.

The subject then measured the dry and wet room temperatures and determined the relative humidity using a sling psychrometer and method as described in Fox et al (1993) immediately prior to going to bed. The subject was in bed by 22h00. Bedding and bedclothes were kept similar for the duration of the study. Heart rate was recorded every minute over the 11-hour period (in total 660 minutes) and stored in the heart rate monitor for analysis at a later stage (Polar Vantage XL, Kempele, Finland). The average, minimum and maximum heart rate for each of the test sessions was calculated by using the appropriate statistical analysis.

The subject maintained a normal lifestyle whilst being monitored and kept an activity and food intake diary in order to replicate this on every testing day. No deviation in activity on testing days was noted and only minimal deviations in food intake were recorded. The subject refrained from ingesting caffeine or alcohol after 16h00 and did no training on the day of testing.

### *Training*

The subject trained 3 times per week for approximately two-and half hours per session. All training took place indoors in an air-conditioned studio. The subject followed a specific periodized training programme, incorporating rest weeks to avoid overtraining. The programme started with lower weights and higher repetitions, progressively increasing the weights being lifted whilst simultaneously decreasing the number of repetitions. The subject kept a training diary that was completed at every training session.

Prior to the start of each training session the subject recorded the ambient temperature as supplied by the South African Weather Services. Body mass was recorded using the same digital scale (Pentronic Digital Scale, Japan). The subject then recorded perception of fatigue and muscle soreness prior to the start of training. A continuous scale from 0 to 10 was used, where 0 = "nothing at all" to 10 = "maximal pain" (Appendix A).

Training sessions consisted of one of the main disciplines, squat, bench press or deadlift with the corresponding assistance exercises. The number of sets and repetitions for each exercise and the weight lifted were recorded immediately following completion of the exercise. Training load was later quantified using these data by multiplying the number of repetitions with the weight lifted for each of the exercise and training sessions. Data were expressed as kilograms lifted per session and per week. Immediately following the completion of the session the subject recorded the overall session rating of perceived exertion (session RPE) (Foster et al., 1995) prior to exiting the gym (Appendix B).

### 3.3.3 STATISTICAL ANALYSIS

Results are expressed as mean  $\pm$  standard deviation unless otherwise noted. Average, minimum and maximum heart rate during sleep was calculated. Data for average heart rate was normalized by setting the value at week 1 to 100 and adjusting the subsequent values accordingly. The coefficient of variation (%) was calculated for the average heart rate during sleep. Average, minimum and maximum values for rating of perceived exertion, fatigue and training load was calculated for each of the training sessions over the entire test period.

### 3.4 RESULTS

The subject was 152.5 cm tall and weighed 58.5 kg at the start of the study. Mass decreased to 56.0 kg (- 2.5 kg) over the 18 week test period (Table 3.1).

Average heart rate during sleep was  $66 \pm 7$  beats.min<sup>-1</sup> during week 1 and  $67 \pm 8$  beats.min<sup>-1</sup> during week 18 (Table 3.1 and Fig 3.1 a). The average sleeping heart rate for the test period of 18 weeks was  $66 \pm 2$  beats.min<sup>-1</sup>. The lowest average sleeping heart rate was 61 and the highest 70 beats.min<sup>-1</sup>. The minimum sleeping heart rate varied from 51 to 57 beats.min<sup>-1</sup> (average  $54 \pm 2$  beats.min<sup>-1</sup>) with the maximum heart rate during sleep varying from 94 to 110 beats.min<sup>-1</sup> (average  $100 \pm 5$  beats.min<sup>-1</sup>) (Table 3.1). The normalized data (Fig 3.1 b) confirms the small variation that occurred with the average, minimum and maximum heart rate during sleep with the coefficient of variation ranging from 3.6, 3.0 and 4.5% respectively.

**Table 3.1 Body mass (kg), training load ( $\text{kg}^{-1} \cdot \text{wk}^{-1}$ ) and average, minimum and maximum heart rate ( $\text{beats} \cdot \text{min}^{-1}$ ) during sleep for 18 weeks.**

Weeks	Training load / week	Body mass	Average heart rate $\pm$ SD	Minimum heart rate	Maximum heart rate
1	20,515	58.5	66 $\pm$ 7	54	94
2	17,992	58.0	61 $\pm$ 7	53	100
3	23,336	58.0	66 $\pm$ 7	55	100
4	0	58.0	68 $\pm$ 8	54	101
5	22,373	58.0	70 $\pm$ 9	54	95
6	18,803	57.9	64 $\pm$ 6	54	94
7	24,635	57.8	70 $\pm$ 9	56	110
8	17,670	57.5	62 $\pm$ 7	53	97
9	16,719	57.6	66 $\pm$ 7	53	99
10	10,222	57.8	68 $\pm$ 9	54	99
11	0	57.0	65 $\pm$ 8	56	104
12	0	56.8	67 $\pm$ 6	56	99
13	7,392	56.5	65 $\pm$ 7	51	98
14	12,390	56.0	67 $\pm$ 8	54	107
15	8,880	55.8	68 $\pm$ 7	57	105
16	4,890	55.3	66 $\pm$ 6	55	103
17	3,890	54.3	64 $\pm$ 7	51	99
18	0	53.5	67 $\pm$ 8	54	95

Data are expressed as the mean, except where indicated otherwise.

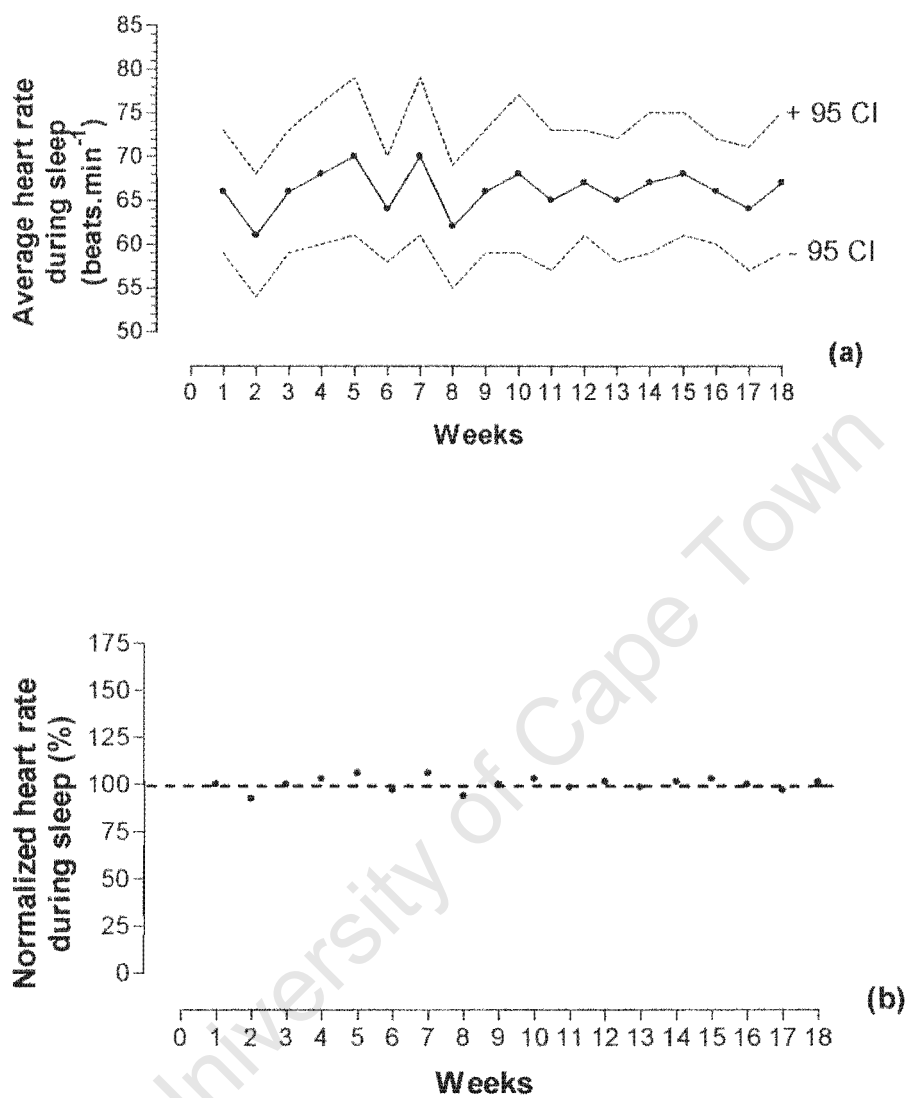


Figure 3.1 Average heart rate (beats.min<sup>-1</sup>) and 95 % confidence intervals (CI) (a) and normalized average heart rate (%) (b) during sleep for 18 weeks.

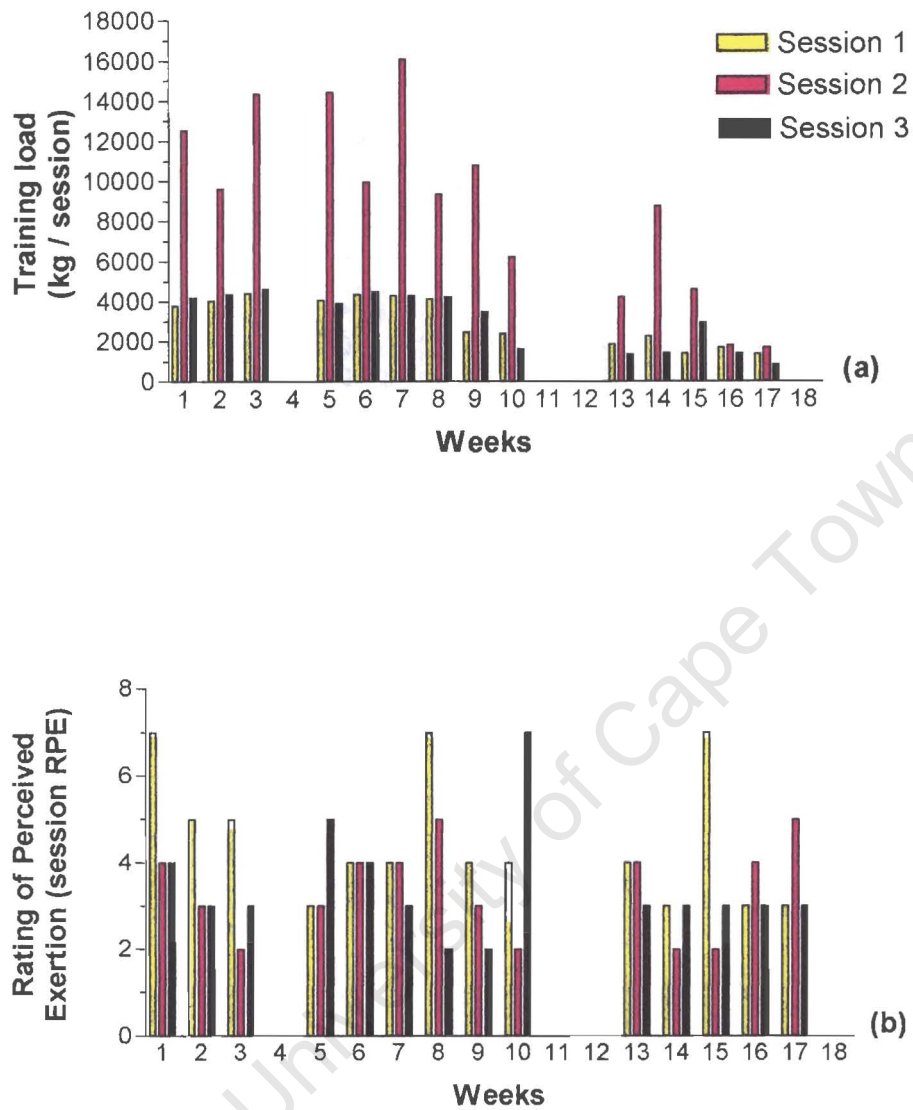


Figure 3.2 Training load (kg / session) and session rating of perceived exertion (units / session) over an 18-week period.

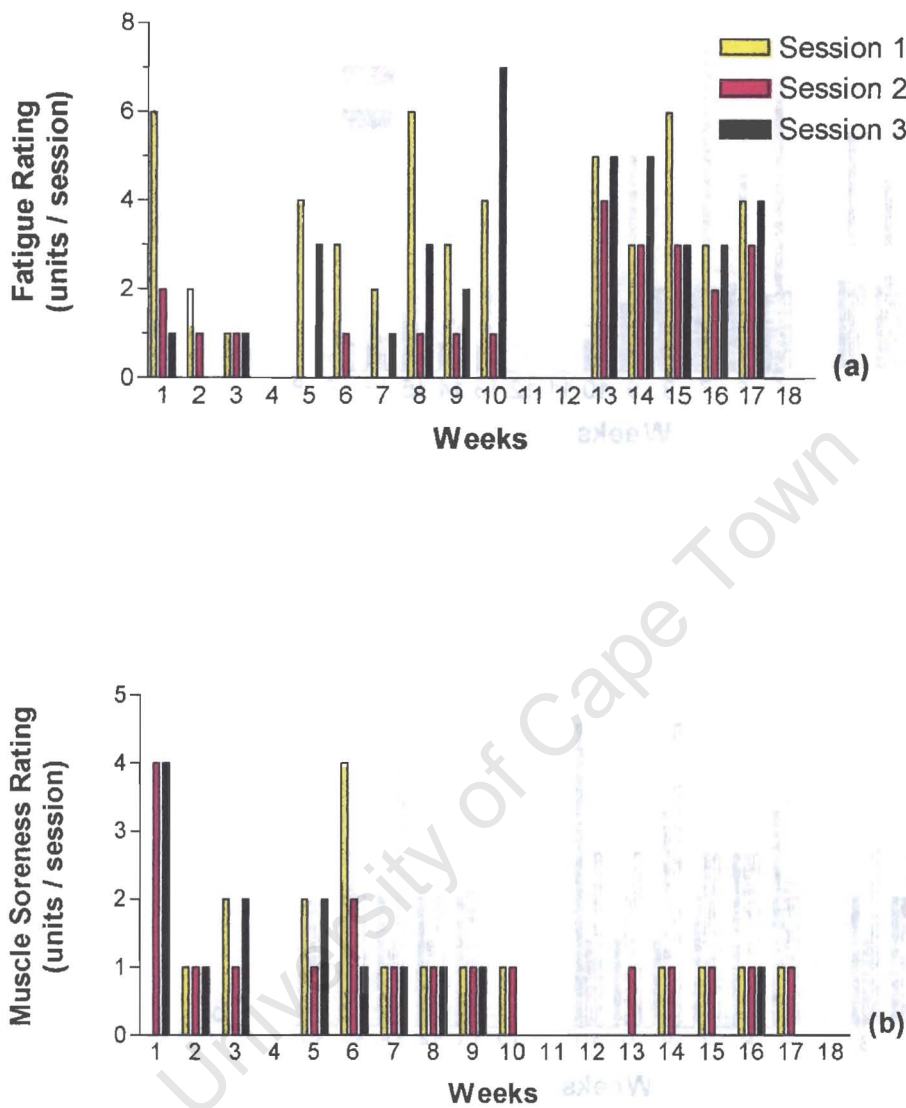


Figure 3.3 Fatigue rating (units / session) and muscle soreness (units / session) over an 18-week period.

Dry temperature during sleep varied between 14 and 22° C with a mean temperature of  $18 \pm 3^\circ\text{C}$ . Humidity ranged from 57 to 81 % with a mean humidity of  $70 \pm 8\%$ .

Average heart rate during sleep during the different phases of the menstrual cycle as measured on days 1, 8, 15 and 22 remained stable throughout and was measured at 66, 65, 67 and 66 beats.min<sup>-1</sup> respectively.

Training load per week varied greatly with a high of 24,635 kilograms (kg) during week 3 and a low of 3,890 kg in the week prior to the national championships. Weeks 4, 11, 12 and 18 were rest weeks with no training taking place (Fig 3.2 a). Average training load per training session for each of the three disciplines (squat, deadlift, bench press) over the study period varied from  $3,027 \pm 1210$  kg for the squat,  $8,876 \pm 4,639$  kg for the deadlift and  $3,078 \pm 1,430$  kg for the bench press. The subject performed more exercises, sets and repetitions on the bench press day - thus the relatively high load compared to the squat days. The average training load per session for each of the three disciplines over the last 5 week period prior to the championships decreased to  $1,702 \pm 362$  kg,  $4,205 \pm 2,861$ kg and  $1,587 \pm 792$  kg for the squat, deadlift and bench press respectively.

Each session was given a global rating of perceived exertion (session RPE) (Foster et al., 1995). RPE ranged from a low of 2 (easy) to a high of 7 (really hard) (Fig 3.2 b). Average RPE rating over the whole study period ranged from  $5 \pm 0$  for the squat,  $3 \pm 0$  for the deadlift and  $3 \pm 0$  for the bench press sessions. RPE ratings over the last 5 weeks were similar at  $4 \pm 0$ ,  $3 \pm 0$  and  $3 \pm 0$  for the squat, deadlift and bench press respectively.

Performance as determined by the heaviest weight lifted during training for each of the three disciplines improved. Direct measurement of the squat showed an improvement from 50 to 110 kg's, bench press from 45 to 60 kg's and deadlift from 60 to 120 kg's between weeks 1 and 18. Estimated 1 RM measurements for the squat showed a change from 59 to 105 kg's (79 %

improvement), bench press 48 to 60 kg's (46 % improvement) and the deadlift from 71 to 120 kg's (70 % improvement).

The subjective rating of fatigue ranged from 0 (nothing at all) to a high of 7 (severe) for week 10 just prior to a scheduled rest week (week 11). Average fatigue rating for the 18 week period was  $4 \pm 2$  for the squat,  $2 \pm 1$  for the deadlift and  $3 \pm 2$  for the bench press sessions. Fatigue ratings for the last five weeks were only slightly higher (Squat:  $4 \pm 1$ ; Deadlift:  $3 \pm 1$ ; Bench press:  $4 \pm 1$ ) than the initial training period. This however, occurred after a 2 week rest period, possibly indicating the increased strain of training with maximal weights (Fig 3.3 a).

Muscle soreness ranged from 0 (nothing at all) to a maximum of 4 (mild) in week 6. The average muscle soreness rating stabilized at 0 to 1 for the rest of the test period (Fig 3.3 b).

### **3.5 DISCUSSION**

The aim of this study was to determine whether changes in heart rate during sleep would be able to track changes in performance associated with changes in fitness. A national level powerlifter was tested under free-living but well controlled conditions for 18 weeks. The free-living conditions under which the subject was tested was well controlled for, insofar as the environmental conditions were measured and bedclothes and bedding were kept similar for the duration of the study period. No deviation from the routine followed on the testing day was noted and food intake only differed marginally. This is probably far more control than one would expect to find when testing athletes outside of the laboratory setting, but not as limiting as laboratory testing would be. This may be seen as a limitation to the study but is far more practical in nature and closer to the reality of real world testing. The testing was conducted

solely on non-training days to further minimize any possible effects on heart rate during sleep. The stringent control was to ensure that any changes in heart rate during sleep would be detected and not missed because of extraneous noise in the measurement of heart rate.

Horne (1981) surmised that especially aerobic type training would affect heart rate more during the first few hours of sleep as endurance type exercise has been shown to increase the incidence of slow wave (NREM) sleep (Snyder et al., 1964). Walker et al (1978) stated that daytime exercise elevates heart rate during sleep in fit, but not unfit individuals. However, in a study by O'Connor et al (1993) no difference was found in heart rate during sleep on both training and non-training days after a 12 week training period. The above studies however, were done on endurance / aerobic type training. Even though the training in our study was power / resistance type training and not endurance training, we felt it prudent to avoid this possible confounding factor by testing only on a non-training day.

The first important finding was that changes in heart rate during sleep under free-living conditions did not track changes in training status in a power athlete. Average heart rate was  $66 \pm 2$  beats.min<sup>-1</sup> with a minimum and maximum average heart rate of 61 and 70 beats.min<sup>-1</sup> respectively over the 18 week test period. When looking at the normalized average, minimum and maximum heart rate data it is clear that the heart rate during sleep did not change significantly over the 18 week period even though the subject's training status changed fairly dramatically (+ 65 % average for 3 lifts combined). This finding differs from other studies that examined sleeping heart rate after several weeks of endurance type training and found sleeping heart rate to be reduced (Sadamoto et al., 1986), decreased (Sedgwick et al., 1974) or increased (Bevier et al., 1987; Bunnell et al., 1983; Roussel and Buguet, 1982). These studies, however, were all conducted on endurance athletes and not on power athletes. It is generally accepted that heart rate changes in power / resistance trained athletes respond

differently compared to endurance trained athletes and is dependent on the training volume, intensity, duration (Lamotte et al., 2005), length of rest periods between sets and the amount of muscle mass utilized (Stone et al., 1991), although the exact mechanism is still unclear. Some studies found heart rate to be reduced (Kang et al., 2005; Vincent et al., 2003) or unchanged (Barauna et al., 2005; Cooke and Carter, 2005; Izquierdo et al., 2005) after resistance type training.

The average dry temperature measured in the bedroom was  $18 \pm 3$  °C (range 14 to 22° C), average wet temperature  $14 \pm 2$ ° C and average humidity  $70 \pm 8\%$  (range 57 – 81 %). The environmental factors can thus be presumed not to have affected heart rate during sleep as no extreme variations were encountered and the temperature and humidity changes occurred slowly over the 18 week period.

Bodyweight decreased from 58.5 kg during week 1 and stabilized at 56.0 kg's during the last 5 weeks of training. The weight loss was a deliberate effort by the subject who wanted to compete in a lower weight class. A loss of 2.5 kg over this extended period will not be sufficient to adversely affect heart rate and no difference was noted in heart rate during the period of weight loss and the last 5 weeks when weight stabilized.

Heart rate during the different menstrual phases did not differ significantly and varied between 65 to 67 beats.min<sup>-1</sup> for each of the phases within the 27 day cycle over the entire test period. There is still no consensus on the effect of the different phases of the menstrual cycle on heart rate. Princi et al (2005) found no correlation between heart rate and the follicular and luteal phases of the menstrual cycle in 6 healthy young women. They did however find some correlation between the female sex hormones concentrations and some of the heart rate variability parameters with higher heart rate variability during the luteal phase. In contrast Leicht

et al (2003) concluded that the normal cyclic variations in the female sex hormones were not significantly associated with cardiac autonomic changes as measured by heart rate variability.

Jurkowski et al (1981) studied nine young healthy women during the mid-follicular and mid-luteal phase at different exercise intensities (33, 66 and 90 % of maximum power output) and found no difference in heart rate or cardiac output during any of the phases. Other studies also found no difference in heart rate during any of the phases of the menstrual cycle (Gamberale et al., 1975; Petrofsky et al., 1976). These results confirm the finding in this study that heart rate is not affected by the different phases of the menstrual cycle.

Fatigue ratings varied with the highest rating of 7 recorded during week 10 just prior to a rest week after 6 weeks of consistent training. Overall, however, only slightly higher average values of fatigue were recorded during the last 5 weeks of training, even though this coincided with the weekly total training load decreasing from an average of 18,403 to 7,488 kg's and followed a 2-week rest period. Again this may be related to the fact that near maximal weights were being lifted but may also be due to accumulated fatigue over the entire training period. Millet et al (2005) found a significant relationship ( $P < 0.001$ ) between training load and perceived fatigue in four elite triathletes during a 40-week training study, by calculating training load from exercise heart rate. This differs from the finding in our study but may be due to the very different training loads experienced by triathletes, whose training is aerobic in nature.

In summary, measuring heart rate during sleep under free-living but controlled conditions was repeatable (about 8 beats.min<sup>-1</sup>; Chapter 2) but not able to track changes in training status in a national class powerlifter. This may be due to the fact that heart rate changes are less pronounced in strength type training compared to endurance type training where it is generally

accepted that improvements in fitness levels are accompanied by a lowered heart rate (Pollock, 1973; Wilmore et al., 1996). It may also be possible that even though the subject had an 8 week detraining period prior to the start of testing, the athlete may have still been sufficiently conditioned not to display significant changes in heart rate when training recommenced as she normally performs at a sub-elite level (elite vs. non-elite). These factors may have affected the results in this study. Another explanation for the lack of change in heart rate during sleep was that the changes lack the precision to track changes in performance associated with increased fitness.

As measuring heart rate during sleep seemed to lack precision in tracking changes in training status, we decided to pursue another strategy. Therefore, the next chapter attempts to determine if submaximal heart rate during exercise under controlled testing conditions accurately reflects changes in training status.

# CHAPTER 4

**DO CHANGES IN SUBMAXIMAL  
HEART RATE DURING EXERCISE  
REFLECT CHANGES IN TRAINING  
STATUS: A CASE STUDY**

University of Cape Town

#### **4.1 ABSTRACT**

The aim of this study was to clarify the relationship between changes in the heart rate / workload relationship and performance after training and subsequent testing under well controlled conditions when training status changed. The subject was an International and SA national field hockey umpire. The subject's heart rate was measured weekly during a submaximal heart rate test over a period of 8 weeks. The subject followed a progressive training programme and changes in training status were measured by performing a 20-m Multi-Stage shuttle test, Agility test, and 10 and 20 meter sprint tests. Heart rate was measured continuously during the submaximal heart rate test and the maximum and minimum heart rate during stage 4 (minutes 10 to 11) and recovery 4 (minute 12) was used to calculate the heart rate recovery percentage. The submaximal heart rate test, measuring heart rate recovery percentage, improved from 21 % during test 1 to 34 % (+ 13 %) in test 8. The average minimum heart rate decreased from 64 to 59 beats.min<sup>-1</sup> between tests 1 and 8 with the average maximal heart rate also decreasing from 172 to 158 beats.min<sup>-1</sup>, a change of 5 and 13 beats.min<sup>-1</sup> respectively. The 20-m Multi-Stage shuttle test increased from an individual shuttle count of 68 during test 1 to 80 shuttles at the last test during week 8 (mean 75 ± 5 shuttles). Sprint times for the 10 and 20 meters improved between tests 1 and 8 from 2.6 to 2.3 seconds and 4.3 to 3.9 seconds respectively. The time for the agility (T-test) improved from 13.5 to 12.6 seconds between tests 1 and 4. Improvements in heart rate recovery, measured during a submaximal heart rate test, seemed to track changes in performance under well controlled testing conditions.

## 4.2 INTRODUCTION.

The development of and easy availability of portable heart rate monitors has changed the application of exercise testing and prescription (Laukkanen and Virtanen, 1998). Athletes are increasingly using heart rate, expressed as a percentage of their maximal heart rate, to monitor their own training intensity whilst coaches use heart rate to prescribe exercise and establish an athlete's training status. There is speculation that changes in the heart rate / workload relationship can be used to accurately track changes in training status. This originates from the belief that there is a linear relationship between heart rate / workload and oxygen consumption (Arts and Kuipers, 1994). Hills et al (1998) concluded that it is reasonable to assume that heart rate accurately reflects workload and intensity.

Numerous studies have examined the effect of exercise training on heart rate (Bevier et al., 1987; Blomquist and Saltin, 1983; Bunnell et al., 1983; Karvonen and Vuorimaa, 1988) with several studies examining the relationship between morning resting heart rate, training status and overtraining (Callister et al., 1989; Dressendorfer et al., 1985; Wilmore et al., 1996). Wilmore et al (1996) found that heart rate during steady-state submaximal exercise at similar workloads decreases, specifically with aerobic type training. This phenomenon was confirmed by several other studies (Blomquist and Saltin, 1983; Dowell, 1983; Pollock, 1973).

However, under certain conditions heart rate may not be an accurate marker of training intensity. There are various factors that may affect the heart rate / workload relationship. Some factors include environmental temperatures, state of hydration, mode of exercise, duration of exercise, cardiac drift and competition (Lambert et al., 1998). Other factors include certain medications, severe body mass changes and other environmental factors such as humidity and

windchill (Wilmore and Costill, 1994). It is thus important to control for these variables when assessing changes in heart rate and training status.

Another important factor to consider when interpreting heart rate data is the naturally occurring intrinsic day-to-day variation in heart rate within individuals. This was demonstrated in the study by Lambert et al (1998) that found that heart rate during submaximal exercise under controlled conditions varied by 1 – 6 beats.min<sup>-1</sup>. In a more recent study (Lamberts et al., 2004) the day-to-day variation in heart rate, when training status remained unchanged, was 5 – 8 beats.min<sup>-1</sup>. In this study the specific workloads and other factors as described previously by Lambert et al (1998) were also well controlled. Knowledge of the variation of heart rate under specific conditions is essential before changes in submaximal heart rate with training can be interpreted correctly.

Therefore the aim of this study was to clarify the relationship between changes in the heart rate / workload relationship and changes in performance after training and subsequent testing under well controlled conditions when training status changed. As these research designs are labour intensive, we decided to firstly do a case study to determine whether there is sufficient evidence to justify doing a trial with more subjects.

## **4.3 METHODOLOGY**

### *4.3.1 Subject*

The subject, an international and South African field hockey umpire, volunteered to participate in a study in which she was tested weekly over an eight week period while she progressively

increased her training load as she prepared for the Athens Olympic Games where she was due to officiate.

The subject completed a health questionnaire / activity profile and gave written informed consent prior to the start of testing. The study was approved by the Ethics and Research Committee of the Faculty of Health Sciences of the University of Cape Town.

#### *4.3.2 Experimental Design*

The trial consisted of a submaximal heart rate test performed weekly for the duration of the study. A 20-m Multi-Stage shuttle test and agility (T-test) was performed alternatively every second week as a measure of performance to track changes in training status. Sprint times over 10 and 20 meters were also measured six times over the study period.

Height was measured at the first session. The sum of seven skinfolds (biceps, triceps, sub-scapular, abdominal, supra-iliac, thigh and calf) as described by Ross and Marfell-Jones (1991) was measured at the first, fourth and last session. Fat % was determined using the formula as described in Durnin and Womersley (1974). Body mass was recorded at each testing session using the same digital scale (Seca, Germany).

The subject's perception of fatigue and muscle soreness was recorded prior to the start of each testing session (Appendix A). If the subject answered in the affirmative she was asked to rate her perception on a continuous scale ranging from 0 – 10, where 0 = "nothing at all" to 10 = "maximal pain".

Training was not monitored or prescribed and the subject followed her own normal training programme. She was however, provided with additional training options that she was free to use and verbally followed up and encouraged prior to each testing session. The subject was highly motivated however as she had to fulfill certain fitness criteria at an international training camp before being allowed to officiate in Athens.

The subject was requested to refrain from doing any training on the day of testing, to control exercise intensity on the day prior to testing, to abstain from ingesting any caffeine or alcohol for three hours before testing and to avoid taking any medication on the day of testing unless medically prescribed and reported. These prescriptions were verified before every testing session. All testing took place indoors at the Sports Science Institute on a rubberized floor. Testing took place at the same time and same day of the week for the duration of the study.

#### *Submaximal Heart Rate Test*

The test was performed prior to the start of the study and weekly thereafter for 8 weeks. The subject was fitted with a Polar Vantage XL (Kempele, Finland) portable heart rate monitor before the test. Heart rate was measured every five seconds for the duration of the test and stored for later analysis.

The test was a submaximal shuttle test with periods of increasing intensity interspersed with recovery periods as described by Lamberts et al (2004). A pre-recorded auditory signal guided the subject through the test. The subject was required to run between two markers, 20 meters apart. After the signal that started the test, the subject had to run from marker A to marker B within a set time. Another signal marked the return run and so on. Running speed progressively

increased through four stages (8.4 km.h<sup>-1</sup>, 9.6 km.h<sup>-1</sup>, 10.8 km.h<sup>-1</sup>, 12.0 km.h<sup>-1</sup>). Each stage lasted two minutes followed by a one-minute rest. The subject was only allowed to stand upright with her arms next to her body during the rest period to minimize affects on the heart rate. A two-minute rest period followed the fourth stage. The test continued until the subject voluntarily stopped the test or until the test reached its conclusion at 13 minutes.

For this study, heart rate recovery was calculated by taking the maximum heart rate obtained during stage 4 minus the minimum heart rate reached during the one-minute recovery period (recovery 4), divided by the maximum heart rate during stage 4, multiplied by 100, thus calculating the percentage heart rate recovery.

The subject was then required to indicate her rating of perceived exertion (RPE) for the test on the Borg scale (Borg, 1973) (Appendix C).

The subject could then rest before the start of the next test that occurred about 10 minutes later.

#### *20-m Multi-Stage Shuttle Test*

The 20-meter Multi-Stage shuttle test (Léger and Lambert, 1988) was used as a measure of performance for this study and to assess the predicted maximal aerobic power of the subject. The test was conducted prior to the start of the study and then every second week.

Two markers were set 20 meters apart and the subject was guided by a pre-recorded audiotape. The subject was required to run at a given pace and had to touch the line with her

foot at either end coinciding with the sound of the pre-recorded tape. The initial running speed started at 8.5 km.h<sup>-1</sup> and increased by 0.5 km.hr<sup>-1</sup> every minute. This was a maximal effort test and the subject had to continue running until she stopped voluntarily or was asked to stop after being given two warnings for failing to reach the line in time with the bleep. The level and number of shuttles were then recorded.

### *10 and 20 meter Sprints*

A 10 and 20 meter sprint was performed prior to the start of the study period and every second week for the duration of the study. The purpose of this test was to measure performance associated with muscle power. The subject underwent a 10 minute warm-up prior to the start of the test.

An electronic sprint timer with photo-electric sensors was set at chest height and placed at the 10 and 20 meter intervals from the start line. The subject started in a crouched position, 30 cm behind the start line. The subject then had to sprint maximally through the 20 meter track. The subject completed two maximal efforts, separated by a 5 minute recovery period. The instantaneous times at the 10 and 20 meter marks were recorded and the fastest times for both distances recorded.

### *T-test (Agility)*

The T-test (Semenick, 1994) was used as a marker of performance of motor coordination and speed and conducted every second week.

Markers were placed in the formation of a 'T' with the long arm of the T being 10 meters in length. The two arms were each 5 meters from the center marker. The subject crouched behind the marker, jumped up and sprinted to the center marker, touched the base then ran sideways to the end marker on one side, touched the base and ran sideways to the marker on the other side and touched the base. The subject then had to run sideways back to the center marker, touch and run backwards to the start. Two maximal runs were completed with a 5 minute recovery period between attempts. The fastest of the 2 attempts were recorded.

#### 4.3.3 STATISTICAL ANALYSIS

Results are expressed as mean  $\pm$  standard deviation unless otherwise noted. The heart rate recovery percentage was calculated for each test session. The average heart rate during each of the different stage and recovery periods for all eight test sessions was calculated. The coefficient of variation (%) for each of the stage and recovery periods during the submaximal heart rate test was calculated for the average heart rate over the eight test sessions. The 95% confidence intervals for the average heart rate for each stage and recovery period over the eight test sessions of the submaximal heart rate test were also calculated. Average values and standard deviations for each of the performance parameters for each of the test sessions were calculated.

#### 4.4 RESULTS

The subject was 39 years old, 166 cm tall and weighed 65 kg at the start of the study. Mass decreased from 65.0 to 63.9 kg (- 1.1 kg) between tests 1 and 8 (Table 4.1) with the body fat % decreasing from 29.5 to 27.9 % (- 1.6%) during the same period.

The submaximal heart rate test, measuring heart rate recovery percentage, improved from 21 % during test 1 to 34 % (+ 13 %) in test 8. This indicated an increased recovery of heart rate after the test. The rating of perceived exertion (RPE) similarly improved from 12 to 10 units during tests 1 and 8 respectively, confirming the change in training status. An RPE rating of 16 was recorded during week 3, coinciding with day 1 of menses. However, an RPE rating of only 12 was recorded during week 6, again coinciding with day 1 of menses.

The 20-m Multi-Stage shuttle test increased from an individual shuttle count of 68 during test 1 to 80 shuttles at the last test during week 8 (mean  $75 \pm 5$  shuttles).

The subjective rating of fatigue over the 8 week period ranged between 1 (very, very slight) and 6 (moderate to severe) with a mean of  $2 \pm 2$  units. The rating of six occurred during week 3 that coincided with day 1 of menses. A fatigue rating of 2 was recorded during week 6 when the subject was also menstruating.

The muscle soreness rating varied between 0 (nothing at all) to 3 (slight) with a mean of  $1 \pm 1$  units. Again week 3 (menses) elicited a rating of 3, with week 6 (menses) recording no muscle soreness at all.

Sprint times for the 10 and 20 meters improved between tests 1 and 8 from 2.6 to 2.3 seconds and 4.3 to 3.9 seconds respectively. The time for the Agility (T-test) improved from 13.5 to 12.6 seconds between tests 1 and 4. Both these tests were used as performance parameters and confirm the change in training status.

Table 4.1 shows the mean heart rate (beats.min<sup>-1</sup>) for each of the four stages and recovery periods recorded during the submaximal heart rate test. Mean heart rate increased as expected, except during stage 3 when the mean heart rate was  $154 \pm 3$  beats.min<sup>-1</sup> compared to  $156 \pm 17$  beats.min<sup>-1</sup> during stage 2. The higher heart rate during stage 2 occurred in three of the eight testing sessions. The 95% confidence intervals for stages 2 and 3 indicated a wider range in heart rate during stage 2 compared to that of stage 3. The mean heart rate during the four recovery periods increased progressively as expected. The coefficient of variation (%) was larger during stages 1 and 2 (14 and 10 % respectively) compared to stages 3 and 4 (2 and 2 % respectively).

**Table 4.1. Mean heart rate (beats.min<sup>-1</sup>), coefficient of variation (%) and 95% confidence intervals for the heart rates for each stage of the submaximal heart rate test (n = 1; average for 8 tests).**

	Heart rate (beats.min <sup>-1</sup> ) Mean $\pm$ SD	95% confidence intervals for heart rate	Coefficient of variation (%)
Stage 1	138 $\pm$ 19	114 – 162	14
Recovery 1	75 $\pm$ 8	66 – 85	10
Stage 2	156 $\pm$ 17	141 – 172	11
Recovery 2	93 $\pm$ 7	87 – 100	7
Stage 3	154 $\pm$ 3	152 – 157	2
Recovery 3	112 $\pm$ 6	107 – 117	5
Stage 4	166 $\pm$ 4	162 – 169	2
Recovery 4	122 $\pm$ 9	114 – 130	8

Heart rate during stage 4 and recovery 4 of the submaximal heart rate test decreased by 9 and 28 beats.min<sup>-1</sup> respectively between tests 1 and 8 (Table 4.2).

**Table 4.2. Mean heart rate (beats.min<sup>-1</sup>) (n=1) for each stage and recovery period during the submaximal heart rate test during test 1 and test 8.**

	Test 1	Test 8	% Change
Stage 1	172	130	32
Recovery 1	70	70	0
Stage 2	170	145	17
Recovery 2	88	90	- 2
Stage 3	156	150	4
Recovery 3	118	100	18
Stage 4	168	159	6
Recovery 4	133	105	27
Heart rate recovery %	21	34	13
Maximal heart rate (% HRmax)	96	88	8

The average minimum heart rate decreased from 64 to 59 beats.min<sup>-1</sup> between tests 1 and 8 with the average maximal heart rate also decreasing from 172 to 158 beats.min<sup>-1</sup>, a change of 5 and 13 beats.min<sup>-1</sup> respectively.

The percentage of maximal heart rate (% HRmax) decreased from 96 % during week 1 to 88 % during week 8.

#### 4.5 DISCUSSION

The aim of this study was to clarify the relationship between changes in the heart rate / workload relationship and performance, with testing conducted under well-controlled conditions when training status changed. A case study was done as a pilot to determine whether there were sufficient data to warrant a more comprehensive study.

The first finding was that the subject had improved her level of fitness as defined by the number of individual shuttles completed during the 20-m Multi-Stage shuttle test. The subject completed 68 individual shuttles during test 1 that was done prior to the start of the study and 80 individual shuttles during test 8. This can be regarded as a meaningful improvement. The subject trained twice a week for 30 minutes prior to the start of the study and increased her training frequency to four times a week for the duration of the study. Even though the subject improved on the number of shuttles run, motivation may have been a limiting factor as the subject only had to complete 80 shuttle runs to qualify her to officiate as an umpire. The subject was verbally encouraged throughout the test and similarly for all of the tests. She was the only subject performing the test, which is maximal in nature. Furthermore, performance in the 10 and 20 meter sprint tests and agility (T-test) tests also improved, confirming that fitness characteristics associated with sport also improved.

The subjects' heart rate recovery percentage improved from 21 to 34 % between tests 1 and 8 respectively, further indicating an improved level of cardiovascular fitness. The heart rate recovery percentage progressively increased during the study, culminating in a heart rate recovery of 34 %. The rating of perceived exertion (RPE) similarly improved from 12 (between light and somewhat hard) during test 1 to a rating of 10 (light) during test 8. The subjective fatigue and muscle soreness unit ratings remained low for the duration of the study indicating

the subject was not overtrained. The higher unit rating for both fatigue (6 = moderate / severe) and muscle soreness (3 = slight) occurred during week 3 that coincided with day 1 of menses. During this test the subjects' heart rate recovery improved from 21 to 24 % but elicited an RPE rating of 16 (hard), the highest recorded. The subject declined to do any performance testing on this day as she felt dizzy and weak. However, during week 6 the subject was again tested during menses. During this test her heart rate recovery percentage was the same as the previous week (27 %) but more interestingly her RPE was only 12, with a fatigue rating of 2 (very slight) and muscle soreness rating of 0 (nothing at all). The subject was also able to perform both the 20-m Multi-Stage shuttle test and agility test and improved on both compared to the previous test. In a study by Stephenson et al (1982) during which he tested six females on five different days of their menstrual cycle (days 2, 8, 14, 20 and 26; day 1 = onset of menses) at maximal (184 W) and four submaximal workloads (45, 83, 121 and 154 W), RPE did not change significantly ( $P > 0.05$ ) at any of the exercise intensities on any of the cycle days. RPE was linearly correlated with heart rate ( $r = 0.87$ ) at all exercise intensities on all menstrual cycle days. They concluded that females could safely be used in any psychophysiological testing without regard to the menstrual cycle phase. This study presents with conflicting results but as the subject was the only participant, it is not possible to really reach any conclusion.

Average heart rate increased during the four stage and recovery periods of the submaximal heart rate test as expected, however the heart rate during stage 2 was higher than stage 3 on three test occasions. No explanation can be given for this phenomenon, although when questioned, the subject indicated that the speed during stage 2 was slower than her normal comfortable running pace. Heart rate decreased from 168 to 159  $\text{beats}\cdot\text{min}^{-1}$  (- 9  $\text{beats}\cdot\text{min}^{-1}$ ) during stage 4 and from 133 to 105  $\text{beats}\cdot\text{min}^{-1}$  (- 28  $\text{beats}\cdot\text{min}^{-1}$ ) between tests 1 and 8 respectively. This is different from a study done by Lamberts et al (2004) in which the same test

was used under well controlled conditions over a five-day period during which training status did not change. In this study mean heart rate changed by  $5 \pm 2$  beats.min<sup>-1</sup> during stage 4 and by  $8 \pm 3$  beats.min<sup>-1</sup> during recovery 4. The difference in heart rate changes between the two studies may well be due to the short period of their testing and the fact that the subjects' training status did not change in the study of Lamberts et al (2004).

Overall the average minimum and maximum heart rate decreased by 5 and 13 beats.min<sup>-1</sup> respectively over the eight week period which is consistent with increased cardiovascular fitness as demonstrated by the improved performance results in our subject.

Maximal heart rate (% HRmax), as calculated by heart rate measured during the submaximal heart rate test as a percentage of predicted maximum heart rate ( $220 - \text{age}$ ), ranged between 100 % during week 4 to a low of 88 % during week 8. The maximal heart rate percentage during week 1 was 96 % (Table 4.2). This again indicates improved cardiovascular fitness as the workloads remain unchanged during the submaximal heart rate test. However, all the maximal heart rate percentage values except for week 8 were above 90 %, indicating that the subject remained relatively stressed by the submaximal heart rate test for most of the study period. This is also much higher than the average maximal heart rate percentage recorded in studies during actual match situations examining the heart rate of top international football, rugby and ice-hockey referees. A study by Helsen and Bultynck (2004) on football referees and assistant referees found that the average maximal heart rate during matches varied between  $85 \pm 5$  % and  $77 \pm 7$  % respectively. Similarly a study on ice-hockey referees indicated maximal heart rates above 70 % for over 70 % of the total match time (Wilkins et al., 1991) and a study by Krstrup et al (2002) on 15 assistant top-class soccer referees found maximal heart rate percentage values of 73 % (range 60 – 88 %). Unfortunately no studies on heart rate and field hockey referees or assistant referees could be found, so it is difficult to assess the

difference in heart rate found in our study, plus the test was not performed during a match situation as the only possible match during the study period was rained out. It is however conceivable that the psychological stress associated with officiating at a top-level Olympic Games match would put even more stress on the heart rate response and thus lead to higher heart rates. This underlines the need for hockey referees to have a good level of cardiovascular fitness to meet the demands of the game and to minimize cardiovascular stress.

In summary, heart rate recovery improvements as measured by the submaximal heart rate test seemed to track changes in performance during the 20-m Multi-Stage shuttle and other performance tests under well controlled testing conditions in an international level subject whose training status changed over a period of 8 weeks. There were sufficient positive findings from this study to warrant doing a similar study with more subjects.

Therefore, in the next chapter the submaximal heart rate test will again be used to measure the heart rate / workload relationship in a bigger sample of subjects whose training status change over the same period of time to ascertain the accuracy of the test under less well controlled testing conditions. Testing will be conducted under field conditions as this is the level of practicality required by athletes and coaches.

# CHAPTER 5

**DO CHANGES IN SUBMAXIMAL  
HEART RATE DURING EXERCISE  
REFLECT CHANGES IN TRAINING  
STATUS: A FIELD STUDY**

## 5.1 ABSTRACT

The aim of this study was to clarify the relationship between changes in the heart rate / workload relationship and performance after training under free-living, field-testing conditions when training status changed. The heart rate of 84 club level rugby and cricket subjects was measured whilst performing a submaximal heart rate test before and after eight weeks of training during which the training status changed. A 5-m multiple shuttle test (MST) was used to measure performance changes. An analysis of variance (ANOVA) with repeated measures, T-test for independent samples and a multiple regression analysis was performed. All the athletes improved on their level of fitness as defined by the distance covered during the multiple shuttle test. The total distance increased from an average of  $672 \pm 104$  to  $726 \pm 58$  m between tests 1 and 2 ( $P < 0.01$ ) with an average improvement of  $12 \pm 29\%$  (95% CI 5 – 18 %). Mean heart rate during stage 4 of the submaximal heart rate test decreased from  $184 \pm 11$  to  $180 \pm 11$  beats.min<sup>-1</sup> between tests 1 and 2 respectively. The increase in distance covered during the MST and the decrease in submaximal heart rate can be interpreted as representing an improvement in fitness. However, there is only a weak inverse relationship ( $r = -0.2$ ) between these variables that was not significant ( $P = 0.2$ ). The variation in environmental conditions on the days of the tests was a confounding factor to the study. In conclusion, heart rate responses to submaximal exercise varied greatly under free-living, field-testing conditions and changes in the heart rate / workload relationship was not an accurate marker of changes in training status. Even though submaximal heart rate decreased after training, the changes in heart rate were not related to changes in performance. The effect of environmental variables, especially humidity, on heart rate and performance must be considered when the heart rate data are interpreted.

## 5.2 INTRODUCTION

In Chapters 1 and 4 the various factors that may affect heart rate and the various studies that were performed to assess changes in heart rate and its relationship to training status were discussed.

A recent study by Boudet et al (2002) examined the maximal heart rate response in 16 subjects under three different conditions (laboratory, field, competition) and found no significant difference in maximal heart rate in all three conditions. They did however find a large intra-individual variation of  $\pm 6$  beats.min<sup>-1</sup> in all three conditions.

The test-retest repeatability of measuring heart rate during submaximal and maximal exercise under controlled conditions have been reported to be between  $r = 0.96$  to  $0.98$  (Lambert et al., 1998; Selley et al., 1995) and  $r = 0.86$  (St Clair Gibson et al., 1998).

However, no study has examined the submaximal heart rate response under free-living, field-testing conditions when training status changed. These are the conditions under which most athletes would be tested and as such it is important to examine how field-testing could affect the heart rate – workload relationship under less well-controlled circumstances.

The data currently being used by athletes and coaches alike were generated under controlled laboratory conditions that may not be representative of the changes that occur in submaximal heart rate in the field-testing situation. This then raises the question of how training-induced changes in heart rate can be interpreted when the data are generated under field-testing conditions.

The varied response of the heart rate / workload relationship under differing conditions as well as the many factors that may influence this relationship makes it extremely difficult to conclusively prove that this relationship does indeed have the capacity to accurately reflect changes in training status. In the previous study (Chapter 4), the subjects' heart rate recovery percentage improved from 21 to 34 % and even though performance parameters improved as demonstrated by the increased number of shuttles completed during the 20-m Multi-Stage shuttle test (68 to 80 shuttles), the relationship seemed indifferent.

Therefore the aim of this study was to clarify the relationship between changes in the heart rate / workload relationship and performance after training and under free-living field-testing conditions in a bigger sample of subjects when training status changed.

### **5.3 METHODOLOGY**

#### *5.3.1 Subjects*

One hundred and forty-eight subjects (n = 143 male and 5 female), consisting of rugby and cricket players from eight different clubs were recruited to participate in the study. The players were selected from the first, second and under 21 teams of the respective clubs. Sixty-four players withdrew from the study for various reasons. The remaining 84 subjects (male; n = 5) were tested on two occasions separated by eight weeks. All the subjects completed a health questionnaire/ activity profile and gave written informed consent prior to the start of testing. The study was approved by the Ethics and Research Committee of the Faculty of Health Sciences of the University of Cape Town.

### 5.3.2 Experimental Design

The trial consisted of a sub-maximal heart rate test to measure heart rate changes and a 5-m multiple shuttle test to measure performance changes during weeks 0 and 8. All tests were performed at the same time of day ( $\pm$  30 minutes).

The sum of seven skinfolds, and fat percentage was recorded at the first and last session using the procedures as described in Chapter 4, pg 128. Body mass was recorded at each testing session.

The subject's perception of fatigue and muscle soreness was recorded prior to each testing session using the subjective rating scale as described in Chapter 4, pg 128 (Appendix A).

Training was not monitored, but all the subjects played for a club and trained twice a week during the on-season under a designated rugby or cricket coach. It was thus assumed that their fitness levels would improve over the eight-week period as the first test was conducted immediately prior to the start of the season. As all of the players were serious recreational athletes who did not necessarily train all year, their fitness levels were fairly low at the beginning of the on-season.

Players were requested to refrain from any other training on the day of testing, to control exercise intensity on the day prior to testing, to abstain from ingesting any caffeine or alcohol products for three hours before testing and to avoid taking any medication on the day of testing unless medically prescribed and reported.

All testing took place outside at the respective club fields. Subjects were subdivided into 8 groups for testing. Ambient temperature, humidity, wind-speed and direction were obtained from the South African Weather Office for each of the sixteen test sessions.

### *Submaximal Heart Rate Test*

The test has been described in detail in Chapters 1 and 4.

### *5-m Multiple Shuttle Test*

The submaximal heart rate test served as a warm-up prior to the 5-m multiple shuttle test (MST). This test was used to measure changes in training status. This test was designed specifically to test the match-related fitness in various sports characterized by intermittent, short-duration, high-intensity bouts of exercise such as rugby (Duthie et al., 2003), field hockey (Reilly and Borrie, 1992) and soccer (Reilly and Gilbourne, 2003) and therefore it was decided to use this test as a measure of performance, rather than the 20-m Multi-Stage shuttle test used in the previous chapter. Subjects were familiarized with the test prior to starting.

The test was described and found to be reliable and valid by Pendleton (1997) and Boddington et al (2001). Subjects were requested to perform the test at maximal effort and were verbally encouraged by the tester and fellow players.

The test comprised of six markers (A, B, C, D, E, F), placed 5 meters apart. A period of 30 seconds' running was followed by a 35 second rest period. Subjects started at marker A and

upon an auditory signal sprinted to the second marker (B), 5 meters away, touched the base with their hands and sprinted back to marker A. This process was repeated to the remaining markers (C, D, E, F), always returning to marker A. The distance covered by the subject during the 30-second running period was recorded to the nearest two and a half meters. Whilst returning to the starting point subjects were required to indicate their rating of perceived exertion (RPE) for each shuttle on the Borg scale (Borg, 1973) (Appendix C). A complete test consisted of six 30-second running and 35-second rest periods.

### 5.3.3 STATISTICAL ANALYSIS

Results are expressed as mean  $\pm$  standard deviation (SD) unless otherwise noted. A T-test for dependent samples was performed on all the variables to determine differences between tests 1 and 2. A Pearson's product moment correlation was calculated to determine relationships between variables. The 95% confidence intervals (CI) were calculated where appropriate. A multiple regression analysis was used to determine changes in heart rate with changing environmental factors. A T-test for independent samples was performed to determine the effect of environmental factors on subjects assigned to groups based on their performance during the multiple shuttle test and the submaximal heart rate test. This will be explained in more detail in the results section. Statistical significance was accepted when  $P < 0.05$ .

## 5.4 RESULTS

The general descriptive characteristics of the 84 subjects (male  $n = 79$  and female  $n = 5$ ) are shown in Table 5.1. The average fat percentage, sum of 7 skinfolds and mass decreased significantly ( $P < 0.01$ ) between Tests 1 and 2 that were conducted eight weeks apart. Mass changes ranged from  $-2$  to  $+3$  kg.

**Table 5.1** General descriptive characteristics of subjects ( $n = 84$ ).

Variable	Test 1	Test 2
Age (years)	$24 \pm 5$	
Stature (cm)	$180 \pm 9$	
Fat %	$17 \pm 5$	$16 \pm 5^*$
Mass (kg)	$87 \pm 15$	$87 \pm 14^*$
Sum of 7 skinfolds (mm)	$84 \pm 31$	$80 \pm 30^*$

\*  $P < 0.01$  before versus after 8 weeks.

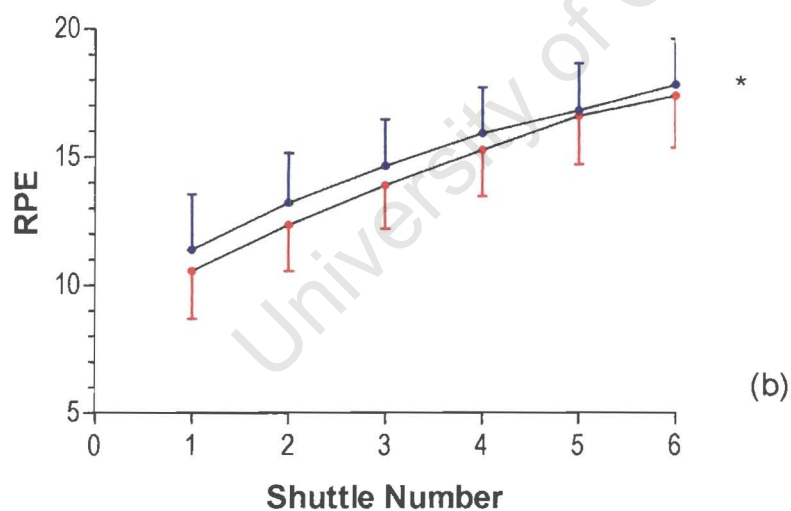
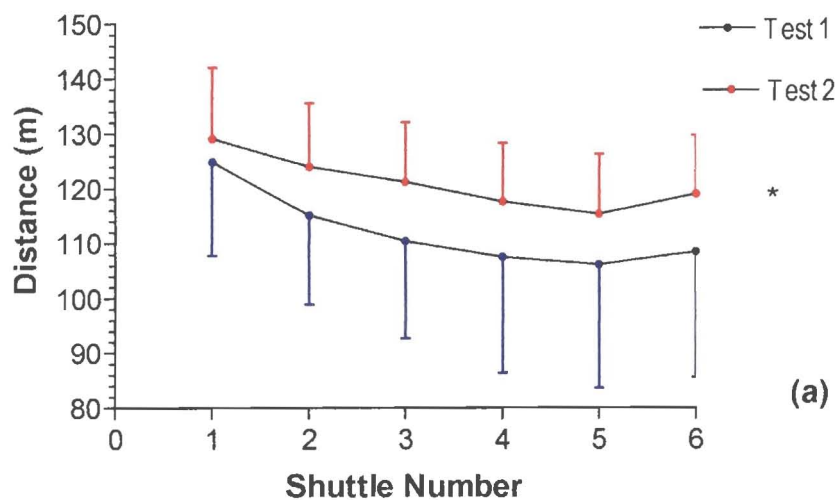
All values are expressed as Mean  $\pm$  Standard Deviation.

All the subjects improved their level of fitness, associated with performance, as defined by the total distance in meters covered during the 5-m multiple shuttle test (MST). The total distance increased from an average of  $672 \pm 104$  to  $726 \pm 58$  meters between tests 1 and 2 ( $P < 0.01$ ) with an average improvement of  $12 \pm 29\%$  (95% CI  $5 - 18\%$ ). The lowest and highest percentage improvement was 0.3 and 212% respectively.

The distance for each of the six shuttles during the multiple shuttle test for both tests 1 and 2 is shown in Figure 5.1 a. Subjects covered more distance at each shuttle after 8 weeks compared to the test before training ( $P < 0.0001$ ), confirming an improvement in their fitness.

Figure 5.1 b shows the rating of perceived exertion (RPE) recorded for each of the six shuttles over the two testing sessions. RPE increased significantly ( $P < 0.0001$ ) between shuttles. The average RPE was also lower during the second test ( $14 \pm 2$ ) compared to the first test ( $15 \pm 2$ ) ( $P < 0.0001$ ). The increased workload at a lower perception of effort once again confirmed that the subjects incurred training adaptations as a result of their training.

Table 5.2 shows the mean heart rate ( $\text{beats} \cdot \text{min}^{-1}$ ) for each of the stages and recovery periods recorded during the submaximal heart rate test. Mean heart rate increased progressively during the four different stages and recovery periods as expected. Mean heart rate decreased significantly ( $P < 0.0001$ ) between tests at all workloads.



\* All shuttles are significantly different at  $P < 0.0001$ .

Data are expressed as the Mean  $\pm$  Standard Deviation.

Figure 5.1 Group data ( $n = 84$ ) for (a) average distance (m) covered during the 5-m multiple shuttle test (MST) and (b) rating of perceived exertion (RPE) recorded during the MST over two test sessions.

**Table 5.2 Mean heart rate (beats.min<sup>-1</sup>), (n = 84), for each stage and recovery period during the submaximal heart rate test conducted before and after 8 weeks.**

	Test 1	Test 2	% Change
Stage 1	152 ± 13	148 ± 12 *	3
Recovery 1	104 ± 19	96 ± 21 *	8
Stage 2	165 ± 1	160 ± 12 *	3
Recovery 2	119 ± 1	111 ± 20 *	7
Stage 3	176 ± 1	171 ± 11 *	3
Recovery 3	131 ± 21	125 ± 19 *	5
Stage 4	184 ± 11	180 ± 11 *	2
Recovery 4	142 ± 19	135 ± 16 *	5
Heart rate recovery %	23 ± 7	25 ± 7	2
% of maximum predicted heart rate	94 ± 5	92 ± 5	2

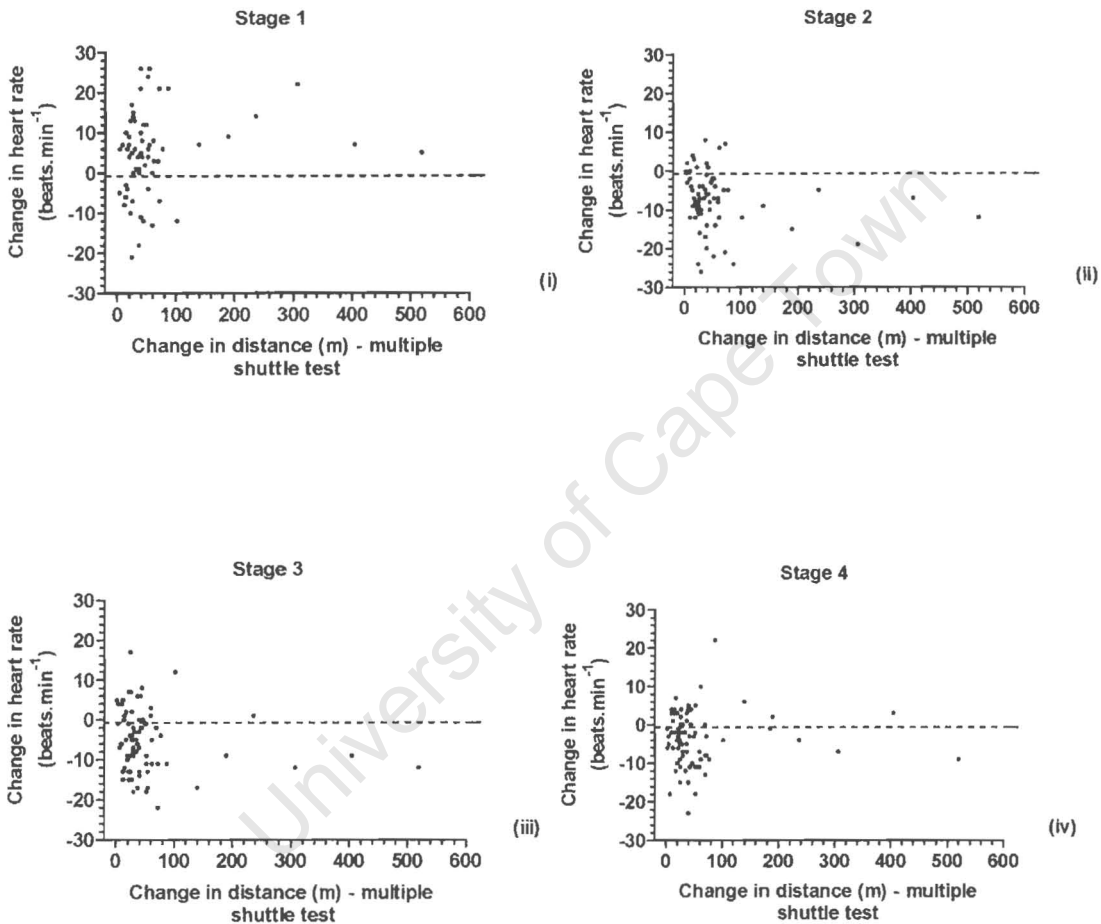
\* P < 0.0001 test 1 vs. test 2.

Heart rate is expressed as Mean ± Standard Deviation.

Mean heart rate during stage 4 of the submaximal heart rate test decreased from 184 ± 11 to 180 ± 11 beats.min<sup>-1</sup> between test 1 and test 2 respectively (Table 5.2). The increase in distance covered during the MST and the decrease in submaximal heart rate can be interpreted as representing an improvement in fitness. However, there is only a weak inverse relationship ( $r = -0.2$ ) between these variables (delta heart rate and distance covered during the stage and recovery periods of the MST) that was not significant ( $P = 0.2$ ) (Figure 5.2 a,b).

Figure 5.2 a (i,ii,iii,iv) depicts the relationship between the change in heart rate (beats.min<sup>-1</sup>) during the four stages of the submaximal heart rate test versus the change in distance

(meters) covered during the multiple shuttle test with Figure 5.2 b (i,ii,iii,iv) showing a similar pattern during the four recovery phases.



**Figure 5.2 a** The relationship between changes in the delta heart rate (beats.min<sup>-1</sup>) during each of the stages of the submaximal heart rate test and distance covered (m) during the MST.

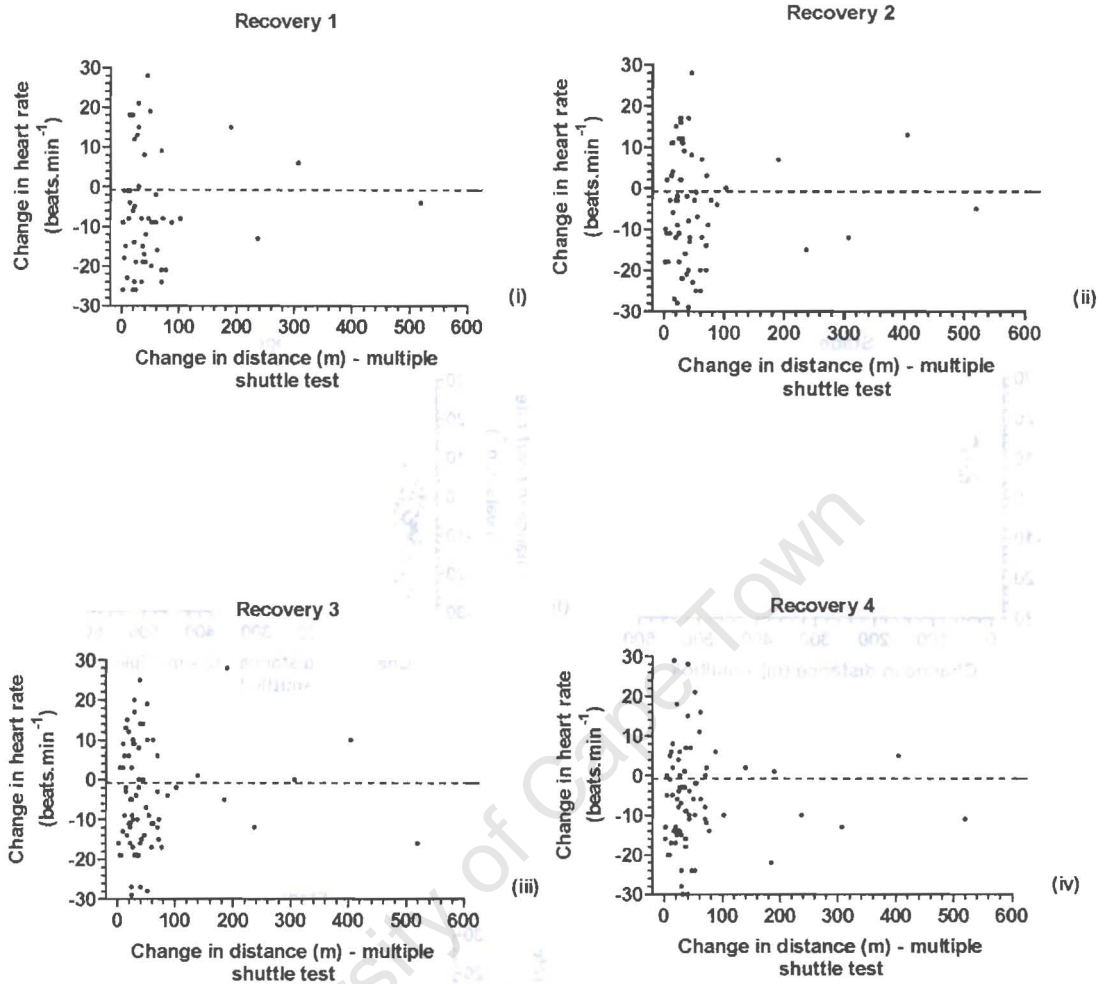


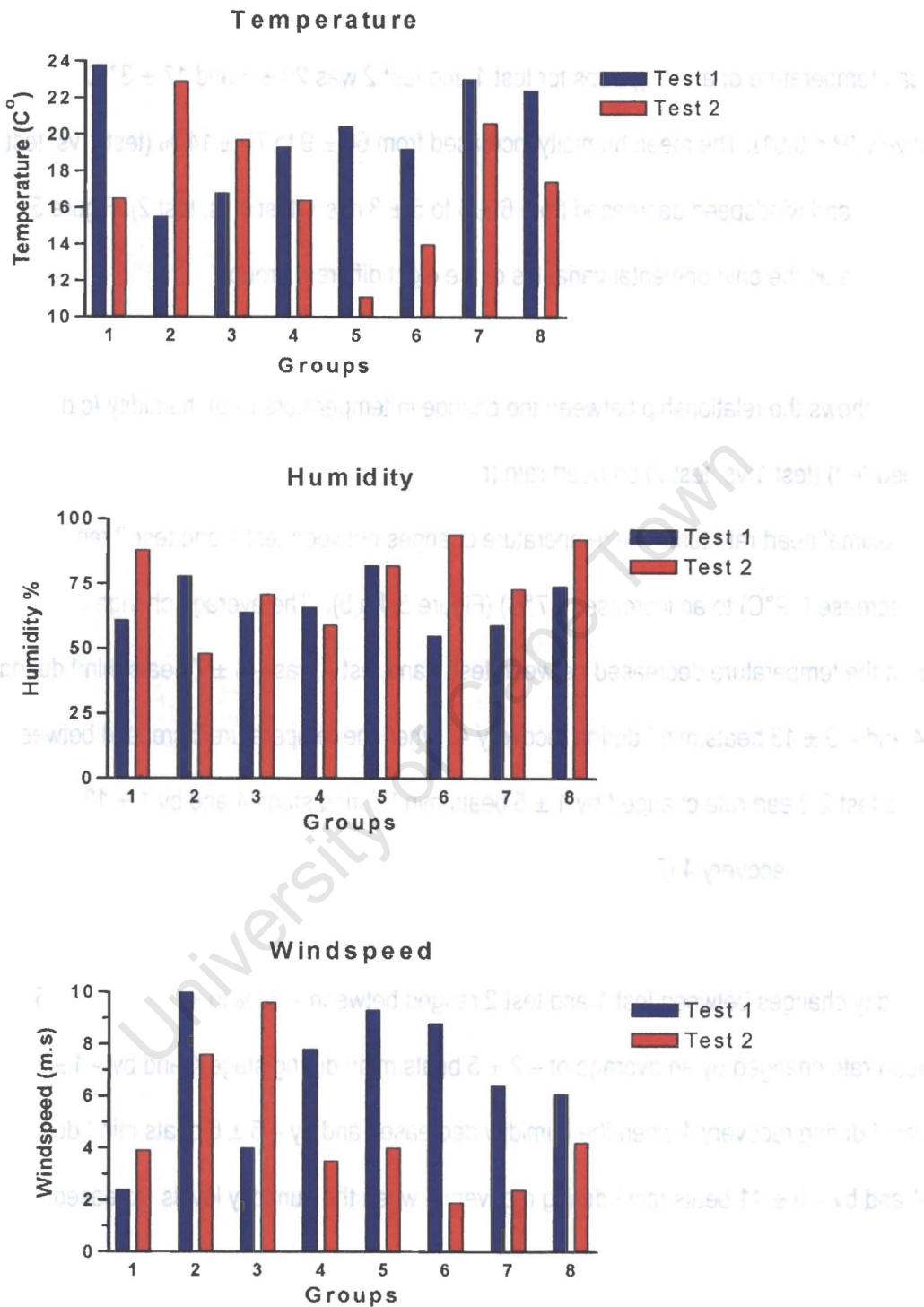
Figure 5.2 b The relationship between changes in the delta heart rate (beats.min<sup>-1</sup>) during each of the recovery phases of the submaximal heart rate test and the distance covered (m) during the MST.

Subjects were divided into 8 groups for pre and post testing. They were tested on different days, in different geographical locations, and with differing environmental conditions.

The mean temperature of all the groups for test 1 and test 2 was  $20 \pm 3$  and  $17 \pm 3^\circ \text{C}$  respectively ( $P < 0.01$ ). The mean humidity increased from  $66 \pm 9$  to  $77 \pm 14\%$  (test 1 vs. test 2) ( $P < 0.01$ ) and windspeed decreased from  $6 \pm 3$  to  $5 \pm 3 \text{ m.s}^{-1}$  (test 1 vs. test 2). Figure 5.3 further elucidates the environmental variables of the eight different groups.

Figure 5.4 shows the relationship between the change in temperature (a,b), humidity (c,d) and windspeed (e,f) (test 1 vs. test 2) on heart rate ( $\text{beats.min}^{-1}$ ) during stage 4 and recovery 4 of the submaximal heart rate test. The temperature changes between test 1 and test 2 ranged from a decrease ( $-9^\circ\text{C}$ ) to an increase ( $+7^\circ\text{C}$ ) (Figure 5.4 a,b). The average change in heart rate when the temperature decreased between test 1 and test 2 was  $-4 \pm 7 \text{ beats.min}^{-1}$  during stage 4 and  $-9 \pm 13 \text{ beats.min}^{-1}$  during recovery 4. When the temperature increased between test 1 and test 2, heart rate changed by  $1 \pm 5 \text{ beats.min}^{-1}$  during stage 4 and by  $1 \pm 13 \text{ beats.min}^{-1}$  during recovery 4 ( $P = 0.5$ ).

The humidity changes between test 1 and test 2 ranged between  $-30\%$  to  $+39\%$  (Figure 5.4 c,d). Heart rate changed by an average of  $-2 \pm 5 \text{ beats.min}^{-1}$  during stage 4 and by  $-1 \pm 13 \text{ beats.min}^{-1}$  during recovery 4 when the humidity decreased and by  $-5 \pm 6 \text{ beats.min}^{-1}$  during stage 4 and by  $-9 \pm 11 \text{ beats.min}^{-1}$  during recovery 4 when the humidity levels increased ( $P < 0.5$ ).



**Figure 5.3** Average descriptive statistics of the environmental factors over two test sessions for all eight groups (n = 84).

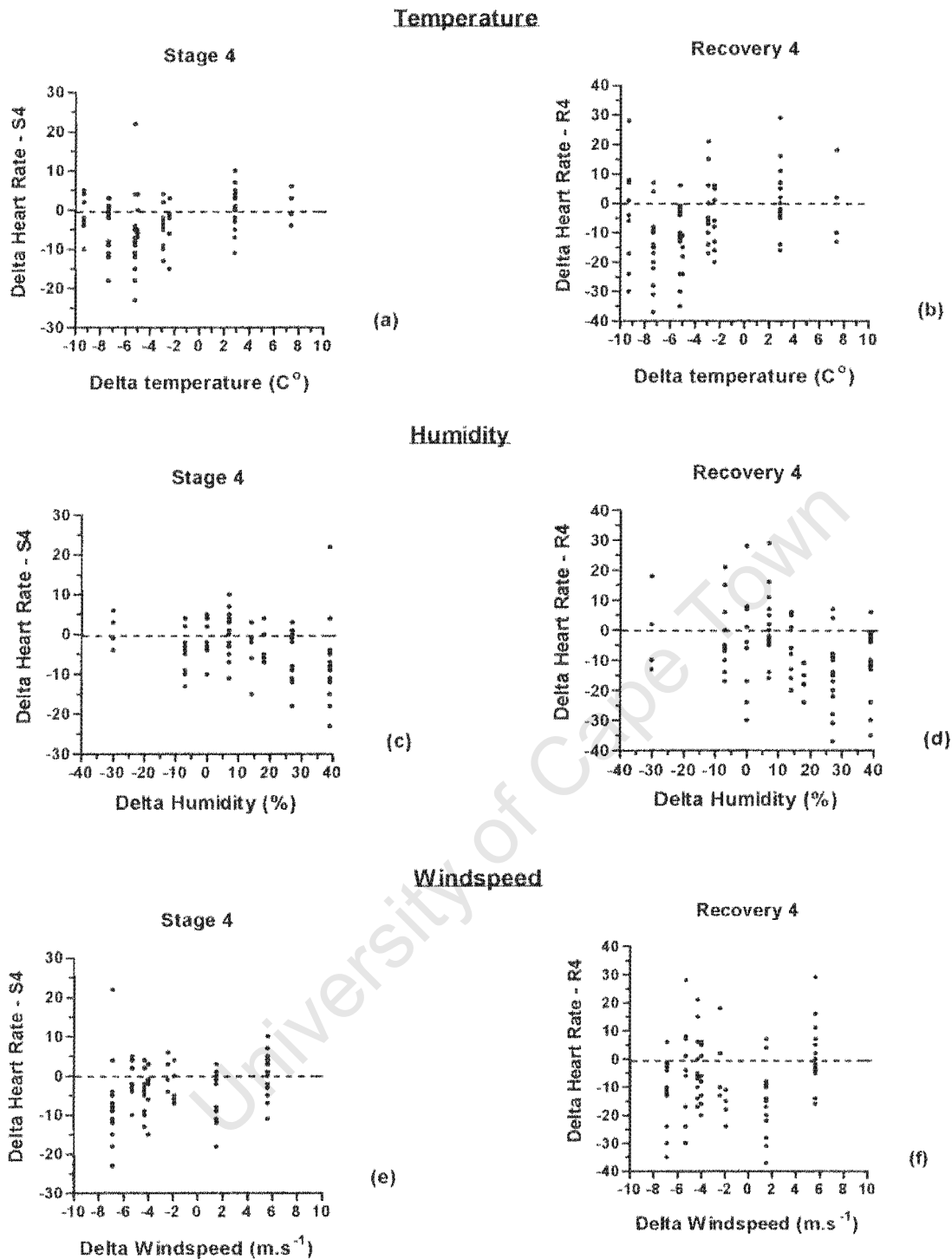


Figure 5.4 The effect of delta temperature ( $^{\circ}\text{C}$ ), delta humidity (%) and delta windspeed ( $\text{m}\cdot\text{s}^{-1}$ ) on heart rate ( $\text{beats}\cdot\text{min}^{-1}$ ) during stage 4 and recovery 4 of the submaximal heart rate test.

The windspeed changes ranged between  $-6 \text{ m.s}^{-1}$  to  $+5 \text{ m.s}^{-1}$  between test 1 and test 2 (Figure 5.4 e,f). The average change in heart rate when the windspeed decreased was  $-3 \pm 6 \text{ beats.min}^{-1}$  during stage 4 and  $-7 \pm 12 \text{ beats.min}^{-1}$  during recovery 4. When the windspeed increased, heart rate changed by  $-3 \pm 6 \text{ beats.min}^{-1}$  during stage 4 and by  $-7 \pm 12 \text{ beats.min}^{-1}$  during recovery 4 ( $P > 0.5$ ).

For an alternative interpretation of the data the subjects were then divided into two groups according to whether their heart rate decreased (HR - 5;  $n = 33$ ) or increased (HR + 5;  $n = 26$ ) between the two tests (pre vs. post training). These groups were determined based on the understanding that a change in heart rate of more than  $5 \text{ beats.min}^{-1}$  can be considered a real change which is greater than the day-to-day variation.

The RPE differed significantly ( $P < 0.05$ ) between the groups for shuttles 2 to 4. Shuttle 1 was also different, but not significant ( $P = 0.2$ ). Group 1 (HR - 5) had higher average RPE values (15) for test 1, compared to the average of 14 for group 2 (HR + 5). Group 1 (HR - 5) showed a bigger decrease in test 2 with an average RPE value of 14 compared to that of group 2 (HR + 5) of 15. There was a tendency for performance changes as measured by the delta distance changes during the multiple shuttle test to be different between the groups ( $67 \pm 104$  and  $40 \pm 46 \text{ m}$ ) as well as between test 1 and test 2 but this was not significant ( $P = 0.2$ ).

The change in maximum heart rate was significantly different ( $P < 0.000001$ ) between groups 1 (HR - 5) and 2 (HR + 5) ( $-9 \pm 5$  and  $0.4 \pm 6 \text{ beats.min}^{-1}$ ) respectively. Delta minimum heart rate differed significantly ( $P < 0.01$ ) between groups 1 (HR - 5) and 2 (HR + 5) ( $-9 \pm 12$  and  $4 \pm 15 \text{ beats.min}^{-1}$ ). Delta percentage of predicted maximum heart rate also differed ( $-5 \pm 2$  and  $0.2 \pm 3 \%$ ) between the groups and was significant at  $P < 0.000001$ ). Delta heart rate during stage 4 of the submaximal heart rate test was significantly different ( $P < 0.000001$ ) between the

groups ( $-10 \pm 4$  and  $4 \pm 4$  beats.min<sup>-1</sup>, HR - 5 and HR + 5 respectively), with heart rate during recovery 4 also significantly different ( $P < 0.0001$ ) between the groups ( $-14 \pm 11$  and  $1 \pm 13$  beats.min<sup>-1</sup>).

The effect of the environmental factors on the two groups with different heart rates was then compared.

The delta temperature ( $-4 \pm 3$  and  $-2 \pm 6$  °C) and delta windspeed ( $-2 \pm 4$  and  $0.01 \pm 5$  m.s<sup>-1</sup>) variables differed between the two groups (HR - 5 and HR + 5) but were not significant ( $P > 0.05$ ). Delta humidity however was significantly different ( $P < 0.01$ ) between groups 1 (HR - 5) and 2 (HR + 5) at  $21 \pm 16$  and  $9 \pm 17$  % respectively.

Subjects were then again divided into two groups, depending on whether their performance during the 5-m multiple shuttle test increased or decreased. A change in distance of 25 meters was defined as being a "meaningful change" in performance. Group 1 (MST - 25; n = 24) consisted of the subjects whose improvement was less than 25 meters, with group 2 (MST + 25; n = 60) showing an improvement of more than 25 meters. RPE was not significantly different ( $P = 0.5$  to  $0.8$ ) between the two groups for all six shuttles.

The delta MST total for the two groups was significantly different ( $P = 0.001$ ) at  $14 \pm 7$  and  $71 \pm 84$  m, MST - 25 and MST + 25 respectively.

The percentage of predicted maximal heart rate ( $92 \pm 3$  and  $96 \pm 3$  %) was significantly different between the groups (MST - 25 and MST + 25) for both tests 1 and 2 ( $P < 0.001$ ).

Minimum heart rate differed significantly ( $P < 0.001$ ) during test 1 ( $73 \pm 13$  and  $85 \pm 14$  beats.min<sup>-1</sup>) but was not significant for test 2 ( $P = 0.1$ ) ( $73 \pm 16$  and  $79 \pm 14$  beats.min<sup>-1</sup>, MST -

25 and MST + 25 respectively). Delta maximum heart rate was different between the two groups ( $-5 \pm 6$  and  $-4 \pm 6$ ) but not significant ( $P = 0.9$ ). Delta heart rate during stage 4 differed between the groups ( $-3 \pm 6$  and  $-4 \pm 7$  beats.min<sup>-1</sup>) but was not significant ( $P = 0.5$ ), with delta heart rate during recovery 4 not significantly different ( $P = 0.1$ ) at  $-3 \pm 15$  and  $-8 \pm 13$  beats.min<sup>-1</sup> MST - 25 and MST + 25 respectively.

Delta humidity was significantly different ( $P < 0.05$ ) between the groups ( $4 \pm 15$  and  $14 \pm 20$  %). Delta temperature and delta windspeed were different between the groups at  $-3 \pm 6$  and  $-3 \pm 5$  °C and  $-1 \pm 4$  and  $-2 \pm 4$  m.s<sup>-1</sup> respectively but were not significant ( $P > 0.5$ ).

## **5.5 DISCUSSION**

The aim of this study was to examine the heart rate response to bouts of increasing submaximal exercise under free-living, field-testing conditions when the training status of the subjects changed over a period of 8 weeks.

The first finding was that all of the subjects improved their level of fitness as defined by their improved performance (distance covered) during the 5-m multiple shuttle test (MST). Subjects generally displayed minor improvements of less than 100 meters, regardless of whether their heart rates increased or decreased. The highest increase in distance during the MST was 520 meters with a decrease in heart rate of  $-18$  beats.min<sup>-1</sup> while the smallest change in distance covered was 2.5 meters with a heart rate decrease of  $-11$  beats.min<sup>-1</sup>. Changes in heart rate were thus not necessarily related to changes in performance ( $r = -0.2$ ) as can be seen in Figure 5.2 a,b.

The next finding was that the rating of perceived exertion (RPE) for all subjects was lower during test 2 compared to test 1. This again confirmed an increase in the subjects' level of fitness, also taking into consideration the general improvement in performance during the MST. When subjects were divided into two groups, based on whether heart rate increased (HR + 5) or decreased (HR - 5), RPE was not different at  $15 \pm 2$  and  $14 \pm 3$  respectively. When the subjects were divided into two groups whose performance during the MST differed (MST + 25 meters and MST - 25 meters), the overall RPE value was the same at  $15 \pm 3$  and  $15 \pm 2$  respectively. Changes in RPE and changes in performance during the MST are thus not necessarily related.

Average heart rate increased during all four stage and recovery periods of the submaximal heart rate test, as was expected. The average change in heart rate during stage 4 for all subjects ( $n = 79$ ) was  $-4 \pm 7$  beats.min<sup>-1</sup> and  $-7 \pm 13$  beats.min<sup>-1</sup> during recovery 4. This change was expected as the subjects' training status improved due to the training intervention. The number of subjects may vary as some heart rate data was lost during the submaximal heart rate test due to technical problems experienced with the heart rate monitors.

Fourteen (18 %) of the subjects ( $n = 79$ ) presented with delta heart rate changes during stage 4 of more than 10 beats.min<sup>-1</sup>. The highest change in heart rate was  $-23$  beats.min<sup>-1</sup> and  $+22$  beats.min<sup>-1</sup> respectively. Five subjects' heart rate did not change at all between test 1 and test 2. Twenty-one subjects' (27 %) heart rate increased and showed an average change of 5 beats.min<sup>-1</sup> with fifty-three subjects' (67 %) heart rate decreasing showing an average change of 7 beats.min<sup>-1</sup>. As can be seen, heart rate variations were quite diverse with no specific discernable pattern. Similarly, the changes in performance were equally diverse and could not be matched to the delta heart rate changes that occurred. The majority of subjects showed heart rate changes of at least 7 beats.min<sup>-1</sup> with another significant percentage showing

changes of  $>10$  beats.min<sup>-1</sup>. This may indicate that heart rate changes would generally need to be between 7 and 10 beats.min<sup>-1</sup> before changes in training status would be detected. This however, may preclude higher trained fitter subjects who may present with much smaller changes in heart rate. Some subjects' heart rate increased even though their performance improved, possibly indicating influence from environmental variables.

Environmental conditions varied to a large extent as the eight groups were tested on different days and in different geographical locations. Temperature, wind speed and humidity were significantly different for all of the groups between test 1 and test 2.

According to Wilmore and Costill (1994) certain factors such as high humidity and ambient temperature can raise heart rate. Increasing temperature increases heart rate during prolonged exercise (Lambert et al., 1998). This phenomenon was confirmed in a study by Booth et al (1997) during which there was increasing cardiac drift due to higher temperatures and humidity. A study by Galloway and Maughan (1997) also found significantly ( $P < 0.05$ ) higher heart rates during exercise at 31° C compared to exercise during lower temperatures of 4, 11 and 21° C. The highest temperature recorded during our study was 23.8° C with the lowest at 11.1° C, so it was anticipated that temperature would not have played a major role in the heart rate variation seen in this study, especially as the test was of a relatively short duration.

When the subjects were divided into two groups based on whether heart rate had either decreased or increased by more than 5 beats.min<sup>-1</sup>, it was found that the change in humidity was significantly ( $P < 0.005$ ) higher in the group who had a decrease in heart rate. This was a paradoxical finding as one would have expected the heart rate to increase as postulated by Wilmore and Costill (1994) with increasing humidity.

However, in a study by Cochrane and Sleivert (1999) the influence of changing patterns of heat and humidity on thermoregulation and endurance performance was tested. They concluded that it was in actual fact the mean heat load that influenced the subjects' physiological responses (heart rate, rectal temperature, RPE and oxygen consumption) more and not so much the changing pattern of temperature and humidity. The lowest humidity percentage recorded during our test was 48 % with the highest 94 %, and although the mean heat load was not tested in our study its influence may have been negligible as our tests were of short duration.

The subjects were then divided into two groups whose performance had differed during the MST. Group 1 had improved by less than 25 meters (lower quartile) with group 2 showing improvements of greater than 25 meters. Again the environmental factors (temperature and humidity) were different between the two groups but only the change in humidity was significantly ( $P < 0.05$ ) higher in Group 2 (MST + 25). One would have expected the higher humidity levels to have influenced the subjects' performance negatively and this may have been the case. One can only surmise that their improvement in performance would have been greater had the humidity levels been lower.

In summary, heart rate responses to submaximal exercise varied greatly under free-living, field-testing conditions and changes in the heart rate / workload relationship was not an accurate marker of changes in training status, as defined by changes in performance in the 5-m multiple shuttle test. Even though on average heart rate decreased with training, the changes in heart rate were not directly related to changes in performance. The effect of environmental variables, especially humidity, on heart rate and performance may have affected the results and therefore should be considered when the data are interpreted.

# CHAPTER 6

HEART RATE, HEART RATE  
RECOVERY AND PERFORMANCE  
CHANGES IN A GROUP OF  
ADVENTURE RACERS

University of Cape Town

## 6.1 ABSTRACT

The aim of this study was to assess if changes in the heart rate / workload relationship can track changes in training status in a group of adventure racers ( $n = 9$ ) after strenuous multi-modality training, under well controlled testing conditions over a period of 18 weeks. In addition heart rate was recorded during two adventure races of varying distances and two racers' heart rate was recorded over a period of five weeks during normal training to assess differences between heart rate during training and competition. The trial consisted of a submaximal heart rate test to measure changes in heart rate. Muscle power and a 20-meter Multi-Stage shuttle test were performed to detect changes in performance. Subjects participated in two adventure races during which their heart rates, body mass, muscle soreness and fatigue ratings were monitored. Two male subjects recorded their average and maximal heart rates during training for a period of 5 weeks. A T-test for dependent samples was performed on all the variables to determine differences between test 1 and test 18, as well as between pre and post race tests results for both races. Performance as measured by the 20-m Multi-Stage shuttle test increased from  $96 \pm 23$  to  $100 \pm 26$  shuttles between weeks 1 and 18. The squat jump also improved minimally. The heart rate recovery percentage increased from 32 to 41 % between weeks 1 and 18 respectively, indicating improved aerobic fitness levels. Only body mass was significantly different ( $P < 0.05$ ) before and after race 1. Subjects trained at higher average heart rates (76 % of HRmax) compared to average race heart rates (65 % of HRmax). In conclusion, performance parameters remained unchanged throughout the study, with heart rate changes similarly not significant. Despite training at higher levels of maximal heart rate compared to races, the training level and intensity was far below what was expected. Large inter and intra-variability in both performance and heart rate variables made it difficult to interpret changes.

## 6.2 INTRODUCTION

Due to the very nature of their sport, adventure racers are a unique group of athletes that have yet to be studied comprehensively. Adventure racing is a multi-disciplinary sport, consisting of hiking, mountain biking, flat, white water or sea rowing or kayaking, trail running, abseiling, caving, orienteering and technical or fixed-line mountaineering (Townes, 2005) or any other sport as the organizers may wish to include. These sports may be combined in any way. The aim of the race is to cover a specific distance within the shortest possible time, performing a variety of different tasks over a multitude of different terrains and using different sport modalities. Races may vary between short ( $\pm 6$  hours), or long (several days duration) and may cover hundreds of kilometers over a variety of different terrains, mostly remote wilderness and with changes in altitude. The participants are unassisted for large parts of the race and have to carry all technical equipment, first-aid and medical supplies and food for several days by themselves. Racers often choose not to sleep to gain a time advantage and sleep deprivation is common in competitors. However, few studies have been done on adventure racers, partly due to the fact that it is a relatively unknown sport. There are also logical problems of testing these subjects during competitions due to the multi-modality nature of the competition and the mostly inhospitable terrain in which the competitions are held.

The few studies on adventure racers have focused on injuries (Fordham et al., 2004), medical support and strategies to support adventure racers (Greenland, 2004; Townes, 2005), and sleep deprivation, energy expenditure and cardiorespiratory function (Scott and McNaughton, 2004).

Studies on heart rate have been done on triathletes (O'Toole et al., 1998), ultraendurance cycling (Neumayr et al., 2003) and male and female orienteers (Bird et al., 2003 a, b). Due to the shortage of literature on adventure racing, these sports are often used as reference points and results from these studies applied to adventure racing.

Neumayr et al (2003) studied the effect of an ultraendurance race, over a distance of 460 km with a cumulative altitude difference of 11,000 meters, on the heart rate of a well trained amateur cyclist. They found that the overall race intensity was moderate with an average heart rate of 130 beats.min<sup>-1</sup>. Only 0.4 % of the total race was performed at a high intensity.

Performance as measured by the average speed declined in the second half of the race with the average heart rate decreasing by approximately 10 % (138 to 124 beats.min<sup>-1</sup>).

Bird et al (2003 a) in his study on male orienteers found that the average heart rate during competition was  $86 \pm 6$  % of maximal heart rate regardless of age and level of the competitor. The international standard competitors however showed less variability in heart rate responses but this was assumed to be due to better navigation techniques that led to fewer changes of speed than the more inexperienced competitors. Average heart rate during the race was  $159 \pm 13$  beats.min<sup>-1</sup>, regardless of training status. In a similar study on female orienteers, the maximal heart rate (%) during a race was  $99 \pm 8$  % for national and  $88 \pm 9$  % for club standard athletes. The average heart rate during the race was higher in the national standard group ( $170 \pm 11$  beats.min<sup>-1</sup>) compared to the club standard athletes ( $158 \pm 11$  beats.min<sup>-1</sup>) (Bird et al., 2003 b).

The above data indicates that subjects competing in these types of races have to train very hard at a high intensity as the races are run at a consistently high intensity. They may also be vulnerable to suffering from symptoms of the overtraining syndrome due to the intense training regimes and the fact that they have to train and prepare simultaneously for several different

exercise modalities. One can assume that this would similarly apply to adventure racers with the added stress of the multi-modality nature of the competition, type of terrain and psychological stressors.

Adventure racers often go without sleep for prolonged periods to gain a time advantage over competitors. Scott and McNaughton (2004) studied the effect of sleep deprivation and intermittent physical exercise on changes in heart rate in six adventure racers. Subjects cycled on a cycle ergometer at 50 % of  $VO_2$ peak for 20 minutes out of every two hours during a 30 hour period of sleep deprivation. Results indicated a significantly ( $P < 0.05$ ) lower heart rate with sleep deprivation, with no effect noticed on other respiratory gas exchange variables. Similarly, a study by Chen (1991) after a 30-hour sleep deprivation period showed that resting heart rate and maximal performance parameters, such as maximal heart rate and time to fatigue decreased ( $P < 0.05$ ). However, other studies have also examined the effect of sleep deprivation on cardiovascular function and presents with some conflicting results. A study by Goodman et al (1989) found peak heart rate and endurance time to exhaustion unchanged after a period of 60 hours without sleep. Similarly, a study by Martin (1981) also found exercise heart rate and metabolic rate unchanged after 36 hours without sleep.

The aim of this study was therefore to assess if changes in the heart rate / workload relationship can track changes in training status in a group of adventure racers after continuous high intensity training under well controlled testing conditions over a period of 18 weeks.

In addition heart rate was recorded during two adventure races of varying time lengths (long and short duration) and two racers' heart rate was recorded over a period of five weeks during normal training to assess differences between heart rate during training and competition.

## **6.3 METHODOLOGY**

### *6.3.1 Subjects*

Eleven subjects (n = 7 males, n = 4 females), all active adventure racers, were recruited at the beginning of their competitive training season to participate in the study. Six of the subjects aimed to compete in an International competition later in the year. Two male subjects withdrew after the start of the study due to injuries sustained during races.

The subjects completed a health / activity questionnaire and gave written informed consent prior to the start of testing. The study was approved by the Ethics and Research Committee of the Faculty of Health Sciences of the University of Cape Town.

### *6.3.2 Experimental Design*

See Table 6.1 for detailed map of testing protocol for all the different performance and submaximal heart rate variables.

Height was measured at the first session. Body composition, (skinfold measurements, bodyfat percentage) following the procedures as described in Chapter 4, was assessed at the first session, every second week thereafter, pre and-post races and at the last testing session. Body mass was recorded at each testing session using the same digital scale (Seca, Germany).

The controlled testing trial consisted of a submaximal heart rate test to measure changes in heart rate and the heart rate recovery percentage and was conducted weekly over a period of

18 weeks. The test was not performed during weeks 6 and 17 as these were race weekends. The tests were performed indoors at the Sports Science Institute of South Africa, on a rubberized floor under controlled environmental conditions, and were conducted at the same time of day ( $\pm$  15 minutes). This test was conducted first as it is a submaximal test with the two subsequent tests being maximal in nature. This test was conducted according to the protocol as described in Chapter 4, pg 129.

Muscle power was measured every 3<sup>rd</sup> week to measure changes in performance and was conducted prior to the 20-m Multi-Stage shuttle test. This test was included as the sport demands a good level of muscle power to enable racers to complete the very physically demanding tasks. Kuipers et al (1985) also found that maximal power output measures ( $W_{max}$ ) had a lower day-to-day variability than  $VO_{2max}$ .

The 20-meter Multi-Stage shuttle test was performed approximately every 3<sup>rd</sup> week as a measure of performance. This test has been described in detail in Chapter 4, pg 130.

Each subject completed a daily training log for the duration of the study, detailing the type of training, intensity and the duration of each activity. The subjects were requested to maintain their normal training and no training interventions were offered.

Subjects recorded their daily perception of fatigue and muscle soreness in the training log in the mornings prior to any training. These ratings were also recorded for non-training days.

(Appendix A).

Subjects were requested to refrain from any training on the day of testing, to avoid ingesting any caffeine or alcohol for three hours prior to testing and to report any medication ingested on

Table 6.1 Weekly schedule of testing of adventure racers over a period of 18 weeks (n = 9).

Weeks	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
Dates	5/7	12/7	19/7	26/7	2/8	Race 1	16/8	23/8	30/8	6/9	13/9	20/9	27/9	4/10	11/10	18/10	Race 2	1/11
Body composition	*				*		*			*			*			*		*
Body mass	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
Muscle soreness / Fatigue	Daily																	
Submaximal heart rate test	*	*	*	*	*		*	*	*	*	*	*	*	*	*	*		*
20m Multi-Stage shuttle test	*				*		*			*			*			*		*
Muscle power / Strength	*				*		*			*			*			*		*
Training log	Daily																	

\* Tests conducted on these days.

the day of testing. Subjects were requested to control exercise intensity on the day prior to testing but this was impossible to control as most of the subjects used weekend races in a variety of disciplines as part of their training regime.

### *Muscle Power Measurement*

Muscle power was measured with a FitroDyne Device (FitroDyne, Fitronic, Bratislava, Slovakia). This test was found to have a high reliability ( $r = 0.97$ ) (Jennings et al., 2005).

Subjects did this exercise within ten minutes of the submaximal heart rate test and were already well warmed up. They were allowed to do their own additional warm up and stretching before the start of the test. Subjects were familiarized with the protocol prior to the start of testing.

The FitroDyne Device was placed on the floor, with the nylon cord attached to an Olympic bar. The subject had to stand next to the FitroDyne with the Olympic bar (20 kg) placed on the back, with the cord perpendicular to the ground. The subject then had to bend down in a controlled manner till the knees were at a  $90^\circ$  angle, pause momentarily in this squat position before jumping up as fast and high as possible. Two spotters ensured the safety of the subject. Each subject performed three squat jumps in a rotation fashion with the other subjects. The upward velocity of muscle contraction was recorded for each jump. The best of the three attempts were used for analysis. Peak power was calculated as force (body mass (kg) + 20 kg)  $\times$   $9.8 \text{ m}\cdot\text{s}^{-2}$   $\times$  speed of contraction ( $\text{m}\cdot\text{s}^{-1}$ ). The subjects could then rest until the start of the next test about 10 minutes later.

### *Competition / Races*

Subjects participated in two adventure races during which their heart rates, body mass, sleep and muscle soreness and fatigue ratings were monitored. Heart rate was measured, using Polar Accurex Plus (Kempele, Finland) portable heart rate monitors. Heart rate was recorded every minute continuously throughout the race and stored for analysis at a later time. Body mass, muscle soreness and fatigue ratings were taken at the start of the race, when subjects clocked into a transition checkpoint and at the conclusion of the race.

The first race took place in early August over a distance of 150 kilometers and the second race late in October over a distance of 190 kilometers. The major differences between the two tests, apart from time, were the fact that race 1 involved a variety of different exercise modalities (trail running, paddling, abseiling, swimming, mountain biking, hiking, orienteering) and partly took place in the dark and in a completely inhospitable wilderness terrain. The weather was extremely poor, cold and rainy. Racers also had to carry all their own equipment, safety gear, food and clothing, as large parts of the race was unassisted. Race 2 only involved hiking, trail running and biking within the city limits and was completed within a relatively short period of 16 hours. Most of the race consisted of road cycling on tarred roads and subjects were able to stop for food. They also carried minimal equipment.

### *Training heart rates*

Two male subjects volunteered to record their heart rate at every training session over a period of five weeks. Average and maximal heart rates were recorded.

### 6.3.3 STATISTICAL ANALYSIS

Results are expressed as mean  $\pm$  standard deviation unless otherwise noted. A T-test for dependent samples was performed on all the variables to determine differences between test 1 and test 18, as well as between pre and post race tests for both races. The 95% confidence intervals (CI) were calculated where appropriate. Statistical significance was accepted when  $P < 0.05$ .

### **6.4 RESULTS**

Eleven subjects were recruited to participate in the study. Two male subjects however, withdrew due to injuries / aggravating chronic injuries during races. Average age was  $28 \pm 4$  years. Average height was  $171 \pm 4$  cm. Body mass was  $70 \pm 11$  kg at the start of the study and  $70 \pm 13$  kg after 18 weeks. Body fat percentage and sum of skinfolds changed (Table 6.2), but this was not significant.

Performance as measured by the 20-m Multi-Stage shuttle test, stayed the same ( $96 \pm 23$  vs.  $100 \pm 26$  shuttles) over the study. Similarly, muscle power, as measured by the squat jump also did not change over the study (Table 6.2).

The heart rate recovery percentage increased from 32 to 41 % between weeks 1 and 18 respectively, indicating improved fitness levels (Table 6.2) ( $P = 0.06$ ).

**Table 6.2 General descriptive characteristics of subjects (n=9).**

<b>Variable</b>	<b>Week 1</b>	<b>Week 18</b>
Fat %	20 ± 4	20 ± 3
Sum of 7 skinfolds (mm)	97 ± 11	88 ± 22
Mass (kg)	70 ± 11	70 ± 13
Heart rate recovery (%)	32 ± 10	41 ± 12
20-m Multi-Stage shuttle test (Shuttles)	96 ± 23	100 ± 26
Squat jump – Peak Power (W)	1255 ± 376	1261 ± 228
Squat jump – Speed/Peak power (m.s <sup>-1</sup> )	1.4 ± 0.2	1.5 ± 0.1

All values are expressed as Mean ± Standard Deviation.

Table 6.3 shows the average heart rate (beats.min<sup>-1</sup>) for each of the four stage and recovery periods during the submaximal heart rate test. Average heart rate decreased over the 18 week period even though fitness levels did not change, but was not significant. The change in heart rate during stage 4 was - 3 beats.min<sup>-1</sup> and during recovery 4, - 15 beats.min<sup>-1</sup>. The average heart rate over 18 weeks during stage 4 of the submaximal heart rate test was 182 beats.min<sup>-1</sup> and 116 beats.min<sup>-1</sup> during recovery 4 (Figure 6.1). The normalized data of the average heart rate during stage 4 and recovery 4 over the study period shows the variability in especially recovery 4 (Figure 6.2).

**Table 6.3 Average heart rate (beats.min<sup>-1</sup>) (n=9) for each stage and recovery period during the submaximal heart rate test during week 1 and week 18.**

	<b>Week 1</b>	<b>Week 18</b>
Stage 1	150 ± 13	141 ± 9
Recovery 1	77 ± 10	78 ± 12
Stage 2	162 ± 13	156 ± 12
Recovery 2	92 ± 18	87 ± 17
Stage 3	175 ± 11	169 ± 11
Recovery 3	114 ± 23	99 ± 23
Stage 4	184 ± 11	181 ± 11
Recovery 4	123 ± 29	108 ± 26

All values are expressed as Mean ± Standard Deviation.

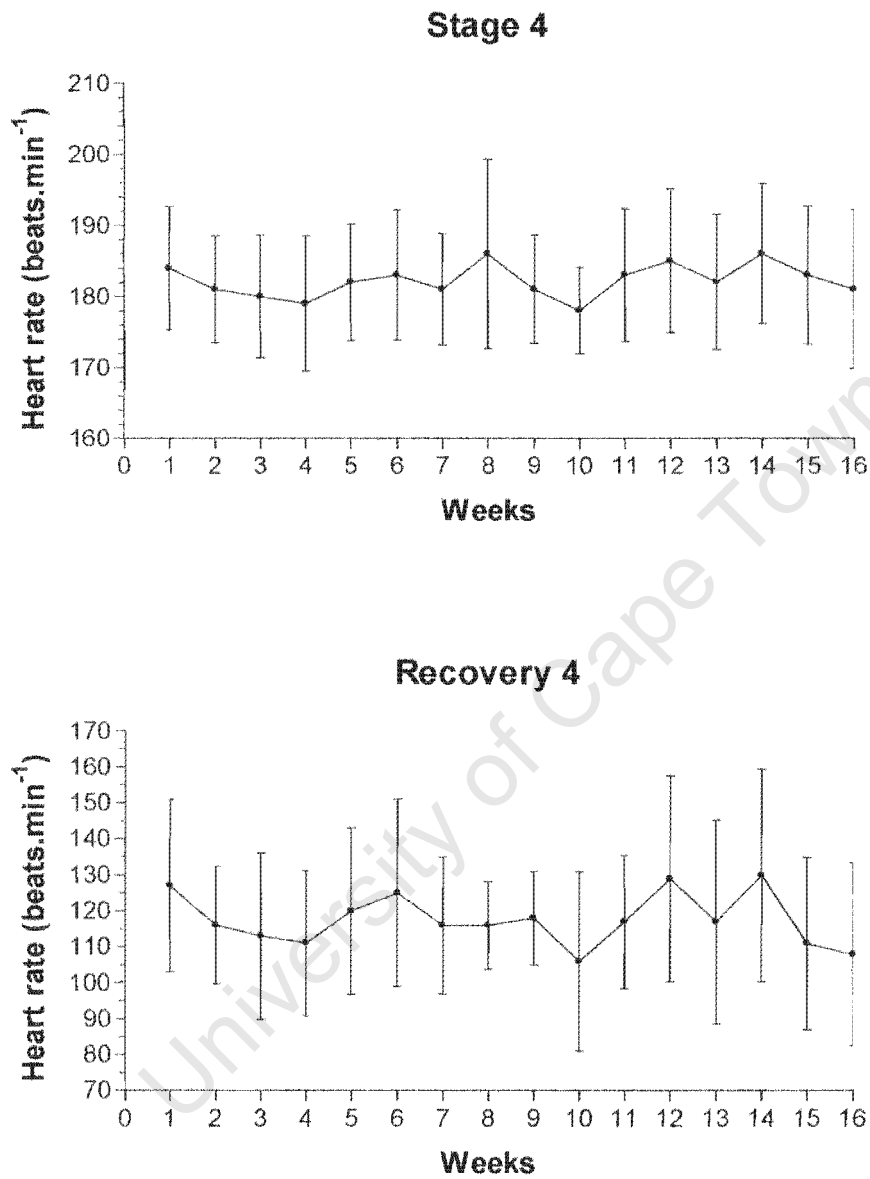


Figure 6.1 Average heart rate (beats.min<sup>-1</sup>) and standard deviation during stage 4 and recovery 4 of the submaximal heart rate test over 18 weeks (n = 9).

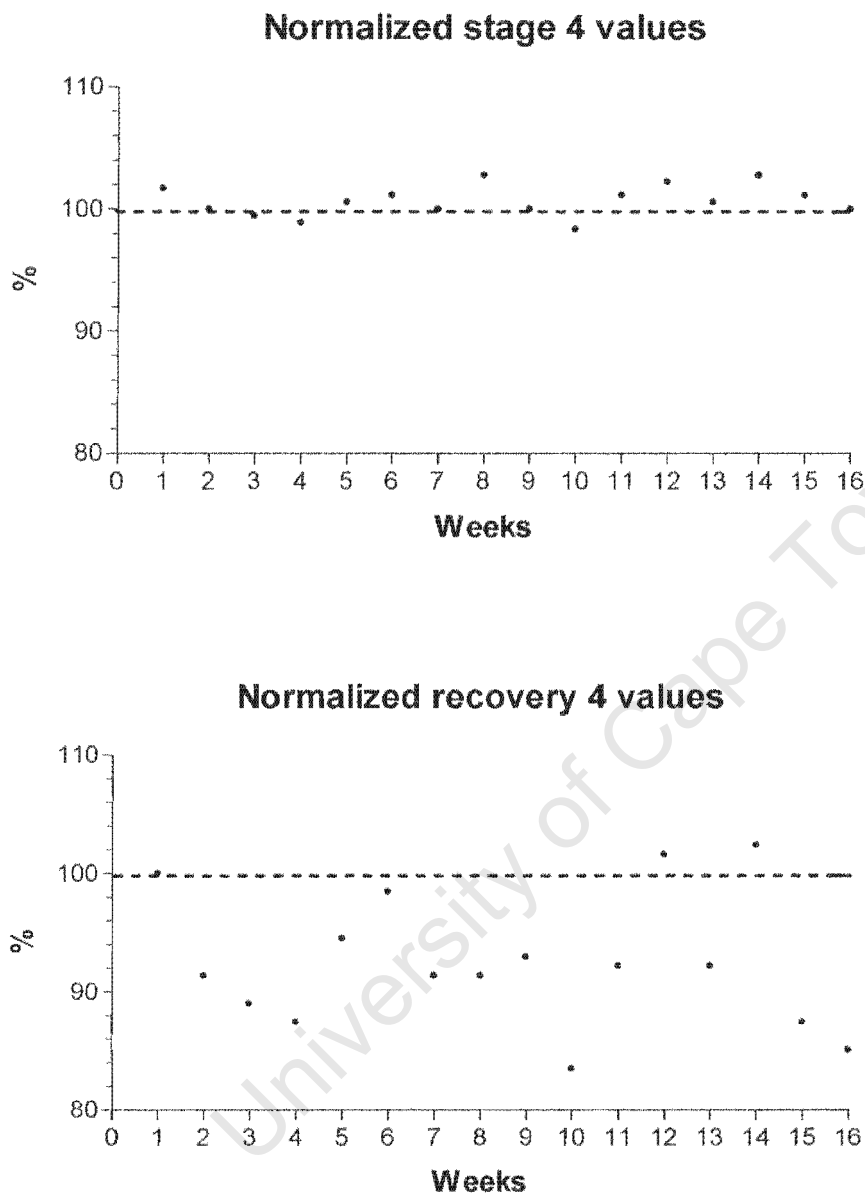


Figure 6.2 Normalized values (%) for stage 4 and recovery 4 of the submaximal heart rate test over a period of 18 weeks (n = 9).

Average training time ( $\text{min}^{-1} \cdot \text{wk}^{-1}$ ) varied greatly with no specific training plan being followed (Figure 6.3). Training time ranged from a low of  $88 \pm 117$  minutes per week during week 7, to a high of  $786 \pm 468$  minutes per week during week 14. Training times did however decrease to  $211 \pm 615 \text{ min}^{-1} \cdot \text{wk}^{-1}$  prior to race 1 and to  $161 \pm 172 \text{ min}^{-1} \cdot \text{wk}^{-1}$  in the week prior to race 2. Subjects were divided into high and low volume trainers. High volume trainers on average reported training times of 679 minutes per week whilst the low volume trainers reported an average of 459 minutes per week.

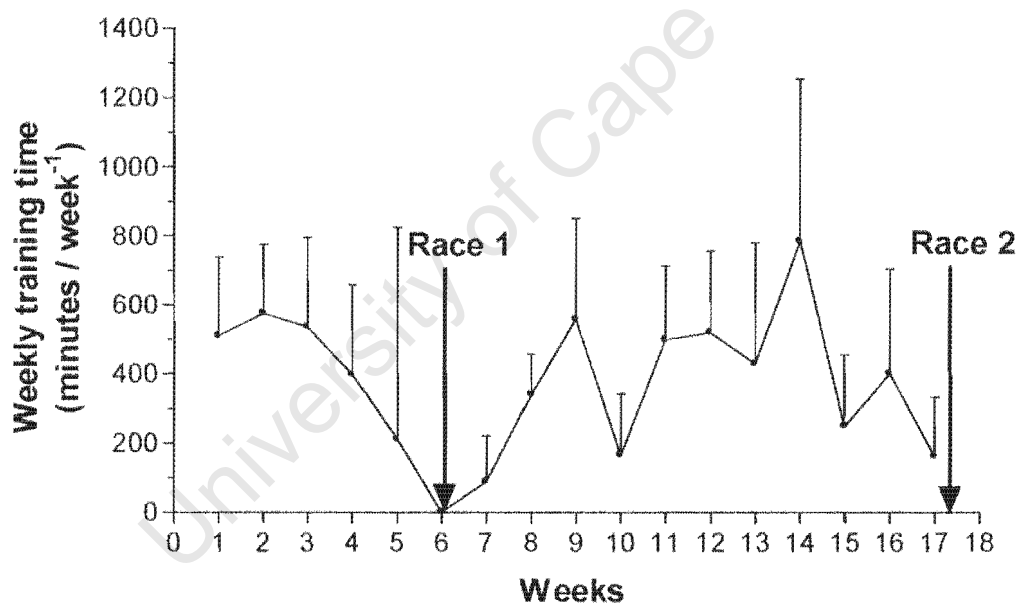


Figure 6.3 Average training time (minutes / week<sup>-1</sup>) over 18 weeks (n = 9).

Average muscle soreness and fatigue ratings remained similar over the entire study period at  $1 \pm 1$  to  $4 \pm 3$  units and  $1 \pm 2$  and  $3 \pm 2$  units respectively.

### *Race results*

Body mass, heart rate recovery percentage, 20-m Multi-Stage shuttle and squat jump tests were performed in the week prior to the races and again one week after the races to assess the effects of the races on the various variables (Table 6.4).

**Table 6.4 Average one week pre-and post race performance data after two adventure races.**

Variables	Race 1 (n=4)		Race 2 (n=5)	
	Pre-race	Post race	Pre-race	Post race
Body mass (kg)	74 ± 13	69 ± 12 *	71 ± 13	71 ± 14
Heart rate recovery (%)	33 ± 11	28 ± 11	42 ± 10	38 ± 13
20-m MST (shuttles)	104 ± 22	102 ± 26	93 ± 23	97 ± 29
Squat Jump (W)	1226 ± 374	1205 ± 328	1155 ± 351	1205 ± 220

\* P < 0.05 between pre and post race.

All values are expressed as Mean ± Standard Deviation.

As can be seen, only body mass was significantly different (P < 0.05) before and after race 1. During this race, one subject lost a total of 11 kg over the race, with the smallest loss being 2.5 kg. Average heart rate for the subject who lost 11 kg was 112 beats.min<sup>-1</sup> (58 % of HRmax) for the whole race compared to 108 beats.min<sup>-1</sup> (56 % of HRmax) for the subject who lost 2.5 kg.

Another subject lost 7.5 kg (average heart rate of 95 beats.min<sup>-1</sup>; 48 % of HRmax). Race 1 was completed over a period of three nights and approximately three and a half days and a distance of 150 kilometers.

During race 2 the smallest weight loss was 1 kg (average heart rate 119 beats.min<sup>-1</sup>; 60 % of HRmax) and the biggest loss 2.5 kg (average heart rate 128 beats.min<sup>-1</sup>; 65 % of HRmax).

Race 2 was contested over a longer distance (190 km) but completed within 16 hours, thus no definitive changes in body mass was observed.

Heart rate recovery percentage was meaningfully lower after both races but not statistically significant (Table 6.4). The decrease could be considered a meaningful difference even in the absence of statistical significance. Even though the races differed substantially, both in duration and intensity, the heart rate recovery percentage decreased by a similar (- 5 %) margin.

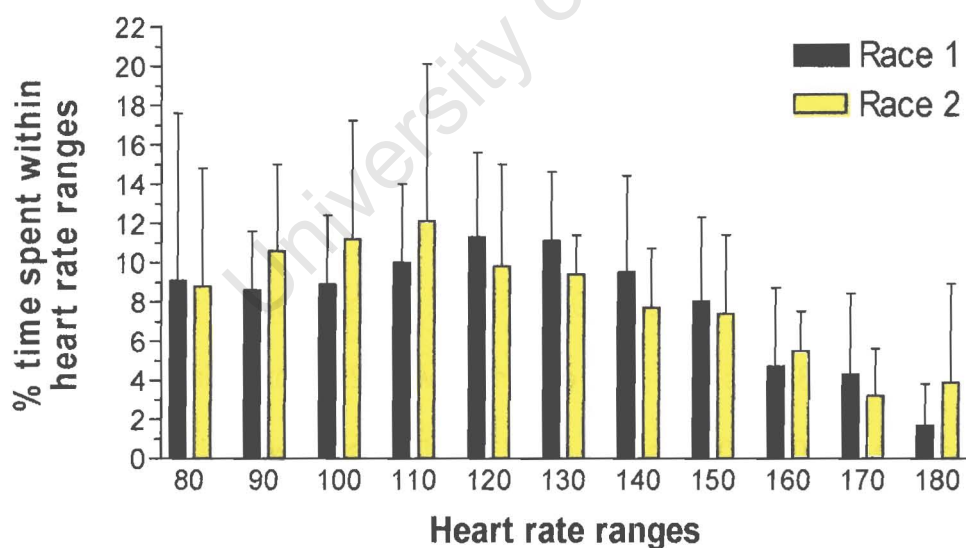
Performance as measured by the 20-m Multi-Stage shuttle test and squat jumps decreased after race 1 but improved after race 2 (Table 6.4). This contradictory finding may be due to the fact that the second race was of shorter duration and therefore the subjects did not build up accumulated fatigue during the race and recovered quicker even though this is not reflected in the heart rate recovery percentage results.

Muscle soreness ratings during both races ranged between 0 and 6 units, with all subjects recording increased ratings. Even though the second race was shorter in duration, the muscle soreness ratings remained similar.

Fatigue rating showed a similar pattern for both races, with fatigue increasing during the race as measured at the transition points, reaching a high post race. Ratings varied from 2 units pre-race to 7 at transition and 6 units post race.

An attempt was made to record sleep time for race 1 only, but because the researcher could not accompany the racers due to the terrain and the inaccurate and incomplete data provided by the racers themselves, this data was not used.

The percentage time spent within the different heart rate ranges during both races is illustrated in Figure 6.4. As can be seen, the majority of time was spent in the lower to middle heart rate ranges of 80 to 130 beats.min<sup>-1</sup>, with less time spent in the ranges 140 to 180 beats.min<sup>-1</sup> during both races.



**Figure 6.4** Percentage time during both races 1 and 2 spent within different heart rate ranges (Race 1: n = 4) (Race 2: n = 5). Data are expressed as Mean  $\pm$  Standard deviation.

The average heart rate for all 4 subjects who completed race 1 was 114 beats.min<sup>-1</sup>, ranging between an average heart rate of 95 to 140 beats.min<sup>-1</sup>. During race 2 the average heart rate was 119 beats.min<sup>-1</sup> (n = 5), ranging between 112 and 128 beats.min<sup>-1</sup>. Compared to this the average heart rate over 18 weeks during stage 4 of the submaximal heart rate test was 182 beats.min<sup>-1</sup> and 116 beats.min<sup>-1</sup> during recovery 4.

### *Training heart rates*

Two male subjects recorded their average and maximal heart rates during all training sessions for a period of 5 weeks.

Subject 1 trained at an average heart rate of 152 beats.min<sup>-1</sup> (range 130 to 168 beats.min<sup>-1</sup>) and at an average maximal heart rate of 176 beats.min<sup>-1</sup> (range 163 to 190 beats.min<sup>-1</sup>). This corresponded to 77 and 89 % of predicted maximum heart rate (HRmax) respectively.

During race 1 the subjects' average heart rate was 108 beats.min<sup>-1</sup> (55 % of HRmax), with only 3.5 % of time spent within the heart rate ranges of 150 – 160 beats.min<sup>-1</sup> and 0.2 % of time spent within the heart rate ranges of 170 = 180 beats.min<sup>-1</sup>. During race 2 the subjects' average heart rate was 128 beats.min<sup>-1</sup> (65 % of HRmax), with 12.8 % of the time spent within the heart rate ranges of 150 – 160 beats.min<sup>-1</sup> and 1.3 % of time spent within the 170 – 180 beats.min<sup>-1</sup> range.

Subject 2 trained at an average heart rate of 143 beats.min<sup>-1</sup> (range 115 – 167 beats.min<sup>-1</sup>) (76 % of HRmax) and an average maximal heart rate of 173 beats.min<sup>-1</sup> (range 151 – 187 beats.min<sup>-1</sup>) (92 % of HRmax).

This subject only competed in race 2 after he had to withdraw during race 1 because of an injury. Average heart rate during the race was 118 beats.min<sup>-1</sup> (65 % of HRmax). Time spent between the heart rate ranges of 140 to 150 beats.min<sup>-1</sup> and 170 to 180 beats.min<sup>-1</sup> was 7.9 %, and 2.5 % respectively.

## **6.5 DISCUSSION**

The aim of this study was to assess if changes in the heart rate / workload relationship can track changes in training status, under well controlled testing conditions, in a group of adventure racers whose training status changed over a period of 18 weeks. Six of the subjects indicated that they were training with the aim of competing at an International competition in November. It was therefore assumed that subjects would train consistently hard at high intensities. Also, studies on similar sporting activities (orienteers) reported subjects competing at high exercise intensities (86 % to 99 % of HRmax; Bird et al., 2003 a,b). A further aim was to assess heart rate during field-testing and competitive conditions and to examine if training heart rates mirrored heart rates during competition.

### *Training (18 weeks) and general monitoring.*

The first finding was that the subjects increased their state of training as indicated by the increase in heart rate recovery percentage from 32 to 41 % ( $P = 0.06$ ) over the whole study period. An increase of more than 5 % is seen as a meaningful change.

Performance as measured by the 20-m Multi-Stage shuttle test and muscle power (squat jumps) remained similar for the duration of the study. The small changes in performance also did not correlate with the improvements in the heart rate recovery percentage. This is an important finding as it again indicates a dissociation between heart rate recovery and performance changes. Intra-variability was high when looking at individual racers. One subject with a heart rate recovery of 32 % completed 132 shuttles on one testing occasion, and at another session completed only 135 shuttles at a heart rate recovery of 43 %. Similarly, another subject completed 98 and 106 shuttles at a heart rate recovery of 27 and 35 % respectively.

The average heart rate decreased by 3 beats.min<sup>-1</sup> during stage 4 and by 15 beats.min<sup>-1</sup> during recovery 4 over the 18 week period, indicating a measure of improvement in training status, but this change was insignificant. In the study on rugby and cricket players (Chapter 5), average heart rate decreased by 4 and 7 beats.min<sup>-1</sup> during stage 4 and recovery 4 respectively. This study was however, conducted over a short 8-week period, and as such it is difficult to compare the results.

The second most important finding was the fact that training time and intensity varied greatly from day-to day and even week to week. After discussions with subjects and inspection of the daily training log kept by the subjects, it emerged that all subjects trained without a definite structured periodised training plan. This held true for both the higher and lower level performers. The three teams consisted of subjects who were rated as either provincial or national standard competitors. The difference between the low and high level groups as regards training however, only pertained to the volume of training performed. The higher level competitors trained an average of 679 minutes per week compared to the lower level competitors who trained an average of 459 minutes per week. Training intensity was reported

as low to high by all subjects, with a fair mix over all the different modalities. However, if one examines the unchanged performance parameters, one can assume that their subjective reported training intensities may be overrated. The two male subjects who reported their training heart rates, trained at average heart rate values of 76 and 77 % of HRmax. Both these males were national class racers.

As the racers were all full-time employees, training had to be completed either before or after work. Other considerations were the weather, the presence or absence of a training partner, work and social commitments and generally depended on what they felt like doing.

Convenience races in the various disciplines, for example running, triathlons etc were used over the weekends as training sessions. Subjects sometimes trained for seven days without a rest day or rested for a week with no apparent goal in mind. This was in stark contrast to what was expected from this standard of competitor, competing in very physically and mentally grueling races. The only common trait seen was that all of the subjects decreased their training time and intensity in the week prior to the races (Fig 6.3).

Muscle soreness and fatigue ratings did not vary greatly during normal training and test sessions and ranged between 1 and 4 units and 1 and 3 units respectively. Higher fatigue and muscle soreness ratings occurred after weekends when subjects competed in a variety of training races such as marathons, triathlons etc. Sometimes subjects competed on both weekend days, depending on when and where races were being held.

#### *Adventure races.*

Subjects competed in two adventure races during the study period. Subjects were tested in the week prior to the races and again a week after the races. During both races subjects were

weighed before, during transition and immediately after the race. Subjects then indicated muscle soreness and fatigue ratings and heart rate monitors were checked.

#### *Pre and Post race.*

Only body mass changes were significantly different ( $P < 0.005$ ) during pre and post race 1 measurements. One subject lost 11 kg during the race, another 8 kg, with the least amount of weight lost being 2.5 and 4.5 kg by the two female competitors. This was despite the fact that competitors were weighed at the halfway transition point and advised to increase fluid and food intake to compensate for weight loss up to that point. Despite these weight losses however, average heart rates remained fairly stable. The subject who lost the most weight had an average heart rate of 112 beats.min<sup>-1</sup> (58 % of HRmax). The subject who lost the least weight had an average heart rate of 108 beats.min<sup>-1</sup> (56 % of HRmax). Some measure of cardiac drift (O'Toole et al., 1998; Montain and Coyle, 1992; Schabort et al., 1998) coupled with dehydration (Heaps et al., 1994) also occurs over the longer race periods, so one can surmise that this would also have been the case in our study. The loss of bodyweight is mostly related to loss of fluid and Montain and Coyle (1992) showed that for every 1 % loss of body mass, heart rate during exercise increased by about 7 beats.min<sup>-1</sup>. The results from our study however, do not support this finding. It is obvious that the subjects were not severely stressed cardiovascularly if one looks at the average heart rates and percentage of maximal heart rate maintained during the race. We are not able to explain this finding in our study. The average heart rate for all 4 subjects over the race period was 115 beats.min<sup>-1</sup> (59 % of HRmax).

During race 2, weight loss was minimal with the most weight lost being 2.5 kg. Average heart rates for the 5 subjects were 119 beats.min<sup>-1</sup> (61 % of HRmax) for the race period. The male

subject who lost the most weight had an average heart rate of 128 beats.min<sup>-1</sup> (65 % of HRmax). Despite the very different nature and exercise modalities utilized in the two races, subjects' competed at very similar average heart rates and maximal heart rate percentages.

The percentage time spent within the different heart rate ranges for both races can be seen in Figure 6.4. The majority of race time was spent in the lower to middle heart rate ranges of 80 to 130 beats.min<sup>-1</sup> with the most time spent between 110 to 120 beats.min<sup>-1</sup>. This is similar to a study done by Neumayr et al (2003) in which he found that the average heart rate for a cyclist over a distance of 460 km was 130 beats.min<sup>-1</sup>, with the second half of the race performed at an average heart rate of 124 beats.min<sup>-1</sup>. However, a study by Bird et al (2003 a,b) found much higher average heart rates in a group of male and female orienteers. Average heart rates for their studies during competition were 159 and 170 beats.min<sup>-1</sup> respectively. When one compares the results from the submaximal heart rate test in our study, where the average heart rate during stage 4 was 182 beats.min<sup>-1</sup>, compared to the average race heart rates of 115 and 119 beats.min<sup>-1</sup> for races 1 and 2 respectively, it becomes clear that the adventure racers employ some sort of pacing strategy to ensure that they make it to the end of the race. This may have something to do with the fact that they are totally uninformed as to which exercise modalities will be used during races, and modalities may be changed at short notice depending on weather conditions etc. The "uncertainty" factor may play a big role in the psyche of these racers and hence the pacing strategy.

The mode of exercise can also influence the heart rate / workload relationship and as these adventure races employ a variety of different exercise modes over varying periods of time it makes it extremely difficult to define the effect on heart rate. The subjects were requested to indicate on their heart rate monitors when they changed from one exercise mode to another,

but they either forgot or it was difficult to do with all the equipment they were carrying, so that the data collected were too inaccurate and we were not able to use it for this study.

The same problem was experienced in the recording of sleep time or rather lack of sleep time and so these confounding variables could not be examined further. However, there is still conflicting evidence on the effect of lack of sleep on heart rate with some studies indicating definitive effects on heart rate (Chen, 1991; Scott and McNaughton, 2004) and others finding no effects (Goodman et al., 1989; Martin, 1981).

The heart rate recovery percentage decreased from the pre to post race testing sessions for both races as was expected. Both however, decreased by the same margin of - 5 %, despite the difference in race time.

At the same time, performance parameters differed between the two races for the pre and post race testing sessions. After race 1, performance in the 20-m Multi-Stage shuttle test and squat jump decreased in the week after the race. Race 2 showed the opposite, where performance improved slightly in both the tests in the week after the race. The shorter race may have left the subjects with less accumulated fatigue, whereas the first race would have surely led to a build-up of fatigue over the 4 day period.

Again however, there was great intra-variability between pre and post race testing session results. One subject had a heart rate recovery of 35 % in the week prior to the race and completed 134 shuttles during the MST test. In the test one week post race his heart rate recovery had decreased to 34 % (- 0.8 %) but he only managed to complete 115 shuttles (- 19 shuttles). In contrast, another subjects' heart rate recovery was 48 and 39 % (- 9.1 %) but she ran 106 and 101 shuttles (- 5 shuttles) pre and post race respectively. This again indicates that

athletes react very differently to similar racing and testing conditions and data interpretation should be specific to each individual athlete.

Training heart rates.

Several studies have examined heart rate during training and competition (Foster et al., 1993; Lambert et al., 1998; Palmer et al., 1994; Selley et al., 1995). All of these studies indicated that subjects do not train at the same heart rates compared to that attained during competitions. Lambert et al (1998) found a correlation of  $r = 0.99$  for the heart rate / running speed relationship during non-competitive training compared to a correlation of  $r = 0.02$  during a competition. A more recent study by Boudet et al (2002) however, found no difference ( $P = 0.62$ ) between heart rates during testing under laboratory, field and competition conditions.

Two male adventure racers recorded their average and maximal heart rates during every training session over a period of 5 weeks. Both subjects consistently trained at higher average heart rate values (77 and 76 % of maximal heart rate) and higher maximal heart rates (89 and 92 % of HRmax). One subject completed race 1 at an average heart rate of 55 % of HRmax and race 2 at an average of 65 % of HRmax. The second subject completed race 2 only and at an average of 65 % of HRmax.

Again, this leads one to surmise that adventure racers follow a pacing strategy during races to ensure they complete the race itself. During training sessions they concentrate on improving fitness levels as they only train one modality at a time under controlled conditions and training sessions seldom last longer than 2 hours. They thus feel comfortable pushing themselves during training but not necessarily during races due to the many unknown variables they have to face during races. Often racers are only given an indication of which modalities will be used

during a race but no specifics as regards time, distance etc. They often only find out what the next section will comprise of as they clock into the next check point.

In summary, performance parameters remained the same and heart rate changes were not significant. Training generally followed a very haphazard pattern, with racers seemingly not training as hard and intense as we expected, considering the very grueling nature of their sport. There was also large intra-variability in performance and heart rate parameters when assessing individual athletes. Due to the many variables that could potentially affect heart rate during adventure races, it was difficult to accurately assess the effect of these many variables on heart rate during competition. It is also clear that adventure racers train at higher heart rate values than that recorded during races, possibly due to the result of some sort of pacing strategy to enable them to complete these extreme races.

In the light of the above findings, the next chapter will again examine changes in submaximal heart rate and its relationship to changes in training status. However, in an attempt to control confounding variables, training will take place under the supervision of a sports scientist at a central, environmentally well controlled venue to ensure all of the subjects are exposed to similar conditions and training workloads. Testing will also take place in a well controlled environment.

# CHAPTER 7

**RELATIONSHIP BETWEEN VARIOUS  
MARKERS OF TRAINING STATUS,  
INCLUDING SUBMAXIMAL HEART  
RATE AND TRAINING VOLUME**

University of Pretoria

## 7.1 ABSTRACT

The aim of this study was to further examine the heart rate / workload relationship and changes in performance under a controlled regime of progressive training overload under well controlled training and testing conditions when training status changed. Twenty-four physically active subjects were tested weekly for a period of 4 weeks. Performance tests and a submaximal heart rate test were conducted during this time. An analysis of variance (ANOVA) with repeated measures was performed to determine differences between variables on the four different testing occasions. A T-test for independent samples was performed to determine differences between the groups divided by differences in heart rate response in the submaximal test and also by differences in performance during the 20-meter Multi-Stage shuttle test. The number of individual shuttles completed during the 20-meter Multi-Stage shuttle test increased progressively between tests each week, indicating an increased level of endurance fitness. The total number of shuttles completed increased from an average of  $77 \pm 20$  to  $84 \pm 18$  shuttles between weeks 1 and 4 ( $P < 0.05$ ). The heart rate recovery percentage increased significantly from  $23 \pm 5$  (95% CI 20 – 27) during week 1 to  $28 \pm 7$  % (95% CI 24 – 32) for week 4 ( $P < 0.05$ ). Heart rate recovery improvements however were not correlated with changes in performance measures. Measuring submaximal heart rate recovery is effective in monitoring the general trend in changes in training status in a group of athletes but intra-variability is high when examining individual subjects.

## 7.2 INTRODUCTION

It is generally accepted that an increase in training volume and intensity is directly related to improvements in performance (Foster et al., 1996; Gilman, 1996), with the major physical changes normally occur within the first 6 to 10 weeks.

In the previous study (Chapter 6), the training volume and intensity was far less than expected, which led to no changes being recorded in both performance and heart rate recovery parameters. This precluded the demonstration of an acceptable model to study the relationship between heart rate recovery, performance parameters and other symptoms of fatigue.

Therefore this study was designed with the aim of inducing performance and heart rate changes, as well as symptoms of fatigue, through a controlled, progressive increase in training load over a short three week period. The type of training selected was designed to induce reasonable muscle stress and by implication, fatigue. The specific time period was chosen to enable us to expose subjects to hard training by increasing the training load (volume) over a very short period, minimize the risk of injury that may occur over longer training periods and to decrease the risk of subjects not complying to the stringent programme.

The aim of this study was therefore to further examine the heart rate / workload relationship and changes in several performance parameters over a short 3-week period under a controlled and supervised regime of progressive training overload (volume) under well controlled testing conditions when training status changed, with all subjects exposed to similar testing and training conditions.

## **7.3 METHODOLOGY**

### *7.3.1 Subjects*

Subjects were recruited from the University and the Sports Science Institute to participate in the study. Inclusion criteria specified were that subjects should be healthy and free of any orthopedic problems and between the ages of 18 and 35 years old. They also had to be involved in sport or recreational training for at least three days per week.

All subjects completed a health questionnaire / activity profile and gave written consent prior to the start of testing. The study was approved by the Ethics and Research Committee of the University of Cape Town.

### *7.3.2 Experimental Design*

The trial consisted of a pre-test (week 1) to determine baseline values for all the parameters that were tested in the subsequent three weeks (weeks 2, 3 and 4). Testing took place on the same day of each week (Saturday) and at a similar time in the morning. The tests took place in a predetermined order that was replicated at each testing session.

The training sessions were conducted indoors at the Sports Science Institute over a period of three weeks. Training sessions lasted approximately sixty minutes with a 15-minute warm-up period and 45 minutes of training. Training days were Monday, Tuesday, Thursday and Friday with rest days on Wednesday and Sunday. Testing took place on Saturdays. The training was of an intermittent, high intensity type with frequent episodes of acceleration and deceleration,

designed to induce muscle stress. The volume of training was increased every week. The first programme was a circuit type, conducted on Mondays and Thursdays, with the second programme mostly of a running nature, completing shuttles on Tuesdays and Fridays. (Appendix D).

Subjects were requested to refrain from any other exercise on the testing days and to abstain from any alcohol or caffeine intake for at least three hours before testing. Subjects were also asked not to take any medication on the day of testing unless medically prescribed and reported.

Height, body mass, skinfold measurements and fat percentage were measured weekly according to the protocol as described in Chapter 4, pg 128.

Perception of muscle soreness and general fatigue were recorded for each subject prior to each testing session using the subjective rating scale as described in Chapter 4 (Appendix A). The assessment of muscle soreness was done according to special charts for the anterior and posterior regions of the body (Appendix E).

The dry and wet ambient temperature was measured with a sling psychrometer using a procedure described in Fox et al (1993) to determine the dry temperature and relative humidity prior to each testing session.

A blood sample was taken prior to the start of testing to assess serum creatine kinase activity. The blood was collected, placed in ice and centrifuged as soon as possible at 3000 rpm for 10 minutes at 4°C. The separated serum was then stored in Eppendorf tubes at -20 °C for later analysis. The serum creatine kinase assay was performed using the CK NAC-activated Kit

(Boehringer and Mannheim, Montreal, Quebec, Canada). This test was included to assess muscle stress due to increased training intensity and is normally used as a marker of muscle damage.

#### *Submaximal Heart Rate Test*

This test was conducted weekly according to the protocol as described in Chapter 4, pg 129.

#### *10 and 20 meter Sprints*

A 10 and 20 meter sprint was performed on all the testing days to measure changes in performance. This test was described in Chapter 4, pg 131.

The subjects were required to warm-up before the test. The warm-up included 10 minutes of submaximal running, appropriate stretching and some familiarization sprints. Each subject did a minimum of 10 minutes of submaximal running, followed by appropriate stretching and some acceleration sprints.

#### *20-m Multi-Stage Shuttle Test*

This test was conducted weekly according to the protocol as described in Chapter 4, pg 130.

#### *Muscle Power test*

This test was conducted weekly as described in Chapter 6, pg 168. However, in addition, a second vertical squat jump was also performed with the barbell with an additional 20 kg attached to the bar.

### 7.3.3 STATISTICAL ANALYSIS

Results are expressed as the mean and standard deviation unless otherwise noted. An analysis of variance (ANOVA) with repeated measures was performed to determine differences between variables on the four different testing occasions. A Tukey post-hoc analysis was done when significant differences were detected in the ANOVA. A T-test for independent samples was performed to determine differences between the groups divided by differences in heart rate response in the submaximal test and also by differences in performance during the 20 meter multi-stage shuttle test. A multiple regression analysis was used to determine changes in heart rate with changes in performance and environmental factors. A one-way analysis of variance (ANOVA) was performed to determine the effect of variables (humidity, temperature, fatigue) on the heart rate during stage 4 and recovery 4. The 95% confidence intervals (CI) were calculated where appropriate. Statistical significance was accepted when  $P < 0.05$ .

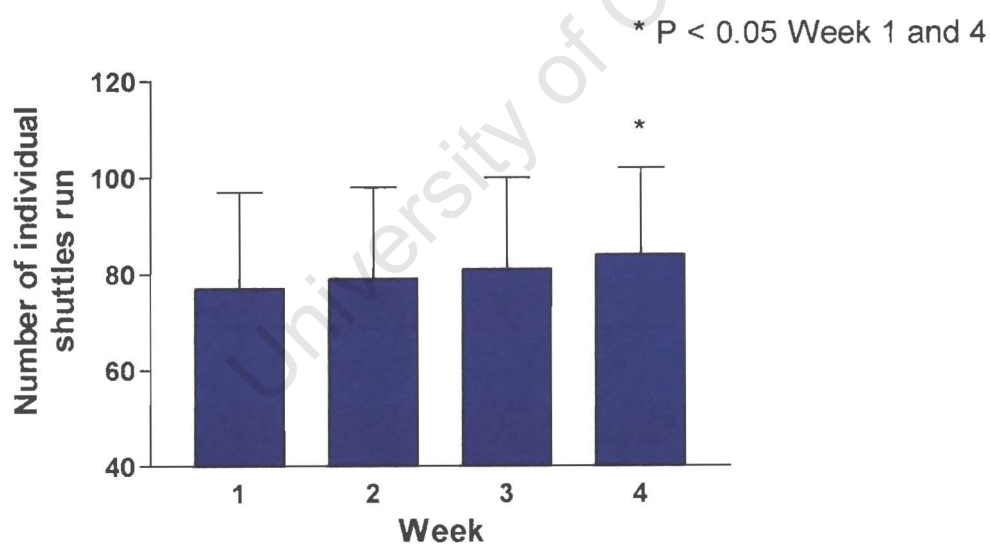
### **7.4 RESULTS.**

Thirty-seven subjects were recruited for the study. However, thirteen subjects withdrew due to various reasons. Twenty-four subjects completed the three-week training period but the data from only 21 subjects were used for this study, due to incomplete data in the submaximal heart rate test for 3 subjects. Eight female and thirteen male subjects completed the study.

Their average age, mass and height were  $25 \pm 3$  years,  $73 \pm 10$  kg and  $176 \pm 9$  cm respectively. The average change in body mass for the group ( $0.2 \pm 1.1$  kg) during the study was not significant. However, the individual changes in body mass ranged between + 2.3 kg to

– 2.0 kg. Body fat percentage did not change ( $0.1 \pm 0.8\%$ ). The range in body fat changes was from + 2.0 % to – 1.4 %.

The number of individual shuttles completed during the 20-meter Multi-Stage shuttle test increased progressively between tests each week, indicating an increased level of endurance fitness (Figure 7.1). The total number of shuttles completed increased significantly from an average of  $77 \pm 20$  to  $84 \pm 18$  shuttles between weeks 1 and 4 ( $P < 0.05$ ) (Table 7.1). On an individual basis, fifteen subjects improved (week 1 vs. week 4), with the number of shuttles completed ranging from 2 to 50 shuttles. One subject had no change while five subjects completed fewer shuttles with a range of – 3 to – 14 shuttles.



**Figure 7.1** Number of individual shuttles run during the 20-m Multi-Stage shuttle run for weeks 1 to 4 (n = 21).

Performance as measured by the 10 and 20- meter sprint times, was similar between weeks 1 to 4, at  $2 \pm 0$  and  $2 \pm 0$  seconds<sup>-1</sup> (Delta  $0 \pm 0$  seconds<sup>-1</sup>) and  $3 \pm 0$  and  $3 \pm 0$  seconds<sup>-1</sup> (Delta  $3 \pm 0$  seconds<sup>-1</sup>) respectively (Table 7.1).

Muscle power as determined by the vertical squat jump did not change ( $1326 \pm 306$  watts vs.  $1362 \pm 258$  watts) (Delta  $48 \pm 87$  watts) ( $P = 0.2$ ) (Table 7.1).

Serum Creatine Kinase (CK) activity was significantly higher at week 2 ( $196 \pm 178$  U.L<sup>-1</sup>) compared to week 1 ( $113 \pm 143$  U.L<sup>-1</sup>) ( $P = 0.009$ ). The CK activity decreased in week 3 ( $126 \pm 118$  U.L<sup>-1</sup>) compared to week 2 ( $P < 0.05$ ). There was no significant difference between weeks 1 and 4 ( $P = 0.7$ ) (Table 7.1).

**Table 7.1 Description of performance parameter changes between week 1 and week 4 (n = 21).**

	Week 1	Week 4
20-m Multi-Stage test (shuttles)	$77 \pm 20$	$84 \pm 18^*$
10-m Sprint (seconds)	$2 \pm 0$	$2 \pm 0$
20-m Sprint (seconds)	$3 \pm 0$	$3 \pm 0$
Muscle power (watts)	$1326 \pm 306$	$1362 \pm 258$
Creatine Kinase (U.L <sup>-1</sup> )	$113 \pm 143$	$132 \pm 132$

\* Significant at  $P < 0.05$ .

Data are expressed as Mean  $\pm$  Standard Deviation.

As was expected, fatigue levels increased from an average of  $3 \pm 2$  units during week 2 to  $4 \pm 1$  units during week four ( $P < 0.005$ ). Eight subjects had an increased delta fatigue rating of 3 units. The subjects with an increased perception of fatigue increased their average shuttle count in the 20-m Multi-Stage shuttle test by  $2 \pm 8$  shuttles while their average heart rate during stage 4 decreased by  $10 \pm 11$  beats.min<sup>-1</sup> and during recovery 4 by  $17 \pm 18$  beats.min<sup>-1</sup>. Five subjects whose delta fatigue rating decreased by  $-1$  unit, showed increases in their shuttle count of  $6 \pm 6$  shuttles, while their mean heart rate decreased during stage 4 by  $7 \pm 5$  beats.min<sup>-1</sup> and during recovery 4 by  $12 \pm 10$  beats.min<sup>-1</sup> (Table 7.2)

Muscle soreness levels decreased from  $19 \pm 33$  units during week 2 to  $14 \pm 15$  units during week 4. The change was not significant ( $P = 0.5$ ) (Table 7.2). The higher values for the muscle soreness ratings compared to all the other studies are due to the fact that we experimented with a more detailed assessment chart that incorporated separate charts for the anterior and posterior views of the body (Appendix D).

**Table 7.2 Fatigue and Muscle Soreness ratings for weeks 2, 3 and 4 (n = 21).**

		<b>Week 1</b>	<b>Week 2</b>	<b>Week 3</b>	<b>Week 4</b>
Fatigue (units)	#		$3 \pm 2$	$4 \pm 2$	$4 \pm 1$ *
Muscle soreness (units)	#		$19 \pm 33$	$15 \pm 22$	$14 \pm 15$

\*  $P < 0.005$  between week 2 and week 4.

# Data for fatigue and muscle soreness for the baseline testing during week 1 were not recorded due to unforeseen circumstances.

The heart rate recovery percentage increased significantly from  $23 \pm 5$  (95% CI 20 – 27) during week 1 to  $28 \pm 7$  % (95% CI 24 – 32) for week 4 ( $P < 0.05$ ) (Table 7.3 and Figure 7.2). The percentage increase between weeks 1 and 4, which suggests an improvement in recovery, ranged from 5 to 99 %. The biggest decrease (deterioration) was – 28%. In total, 17 subjects had an improved heart rate recovery percentage of  $6 \pm 4$  %, while four subjects had an average deterioration of  $3 \pm 3$  %.

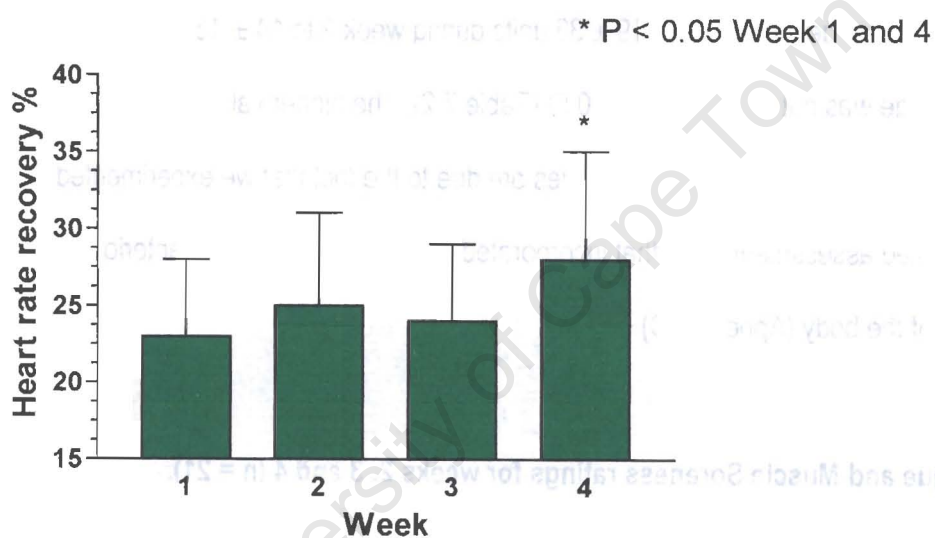


Figure 7.2 Heart rate recovery percentage (%) for weeks 1 to 4 (n = 21).

**Table 7.3 Mean heart rate (beats.min<sup>-1</sup>) for each stage and recovery period during the submaximal heart rate test conducted for four weeks.**

	<b>Week 1</b>	<b>Week 2</b>	<b>Week 3</b>	<b>Week 4</b>	<b>n</b>
Stage 1	146 ± 16	144 ± 17	149 ± 17	142 ± 14	8
Recovery 1	97 ± 27	90 ± 21	99 ± 24	88 ± 19	8
Stage 2	163 ± 15	159 ± 15	161 ± 17	152 ± 14	9
Recovery 2	120 ± 18	109 ± 22	114 ± 24	99 ± 21 *	10
Stage 3	179 ± 15	171 ± 17	174 ± 15	167 ± 15 *	10
Recovery 3	141 ± 22	128 ± 22	133 ± 21	118 ± 20 ● ■	13
Stage 4	192 ± 16	183 ± 14 #	185 ± 14 ▲	180 ± 14 □	14
Recovery 4	148 ± 18	138 ± 19	141 ± 17	130 ± 18 □	14
Heart rate recovery (%)	23 ± 5	25 ± 6	24 ± 5	28 ± 7 *	13
% of maximum predicted heart rate	98 ± 6	93 ± 8 ◇	92 ± 11 ▲	91 ± 7 △	12

# P < 0.005 week 1 vs. week 2

◇ P < 0.05 week 1 vs. week 2

▲ P < 0.05 week 1 vs. week 3

\* P < 0.05 week 1 vs. week 4

■ P < 0.05 week 3 vs. week 4

● P = 0.0005 week 1 vs. week 4

□ P < 0.0005 week 1 vs. week 4

△ P < 0.005 week 1 vs. week 4

Data are expressed as the Mean ± Standard Deviation.

Table 7.3 and Figure 7.3 a,b shows the average heart rate (beats.min<sup>-1</sup>) for each of the four stages and recovery periods recorded during the submaximal heart rate test. As expected the average heart rate increased progressively during the four stages and recovery periods of each test as the workload increased. As fitness levels increased, the average heart rate decreased progressively from weeks 1 and 2 to week 4. At week 3, however, heart rate was slightly increased from week 2, but this was not significant ( $P = 0.8$ ). The heart rate decreased again to week 4. Average heart rate during stage 4 of the submaximal heart rate test decreased from  $192 \pm 16$  to  $180 \pm 14$  beats.min<sup>-1</sup> between weeks 1 and 4 ( $P < 0.0005$ ), with the average heart rate during recovery 4 decreasing from  $148 \pm 18$  to  $130 \pm 18$  beats.min<sup>-1</sup> ( $P < 0.0005$ ) between weeks 1 and 4 respectively.

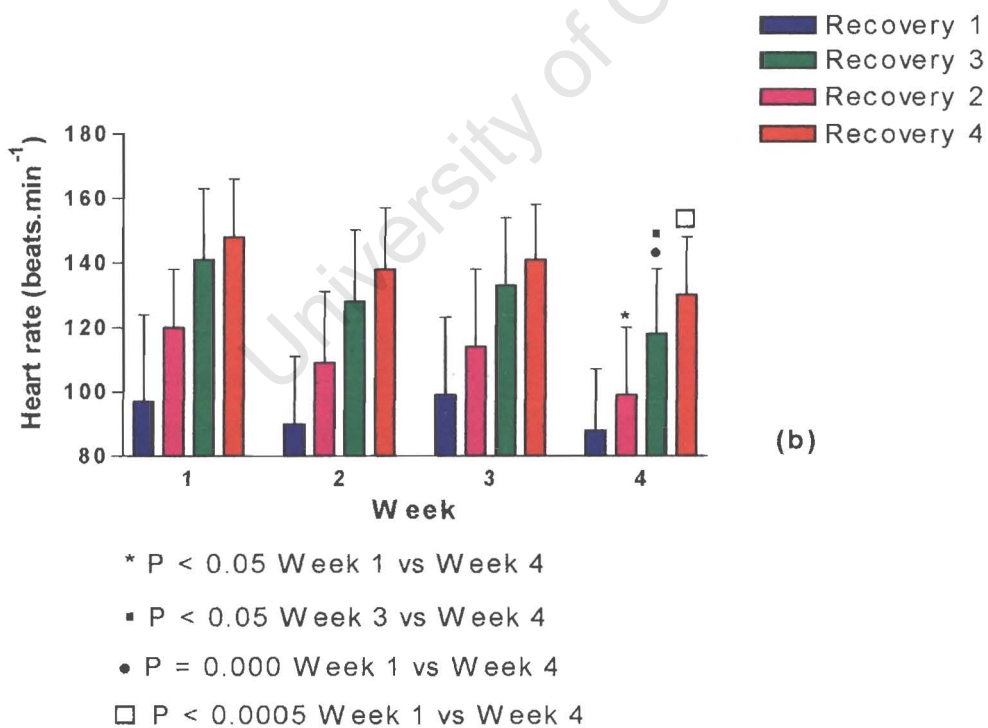
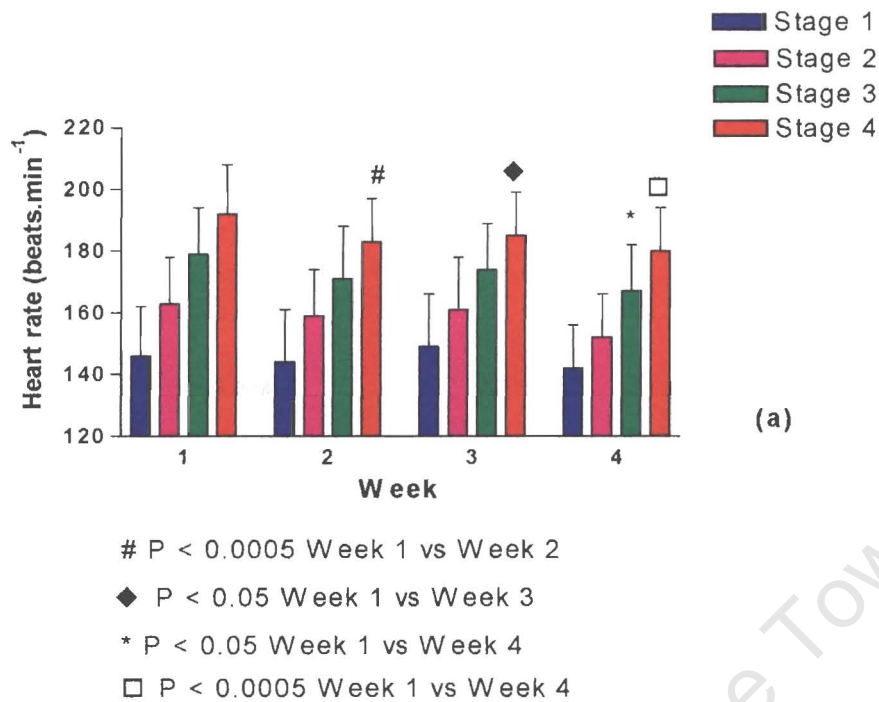
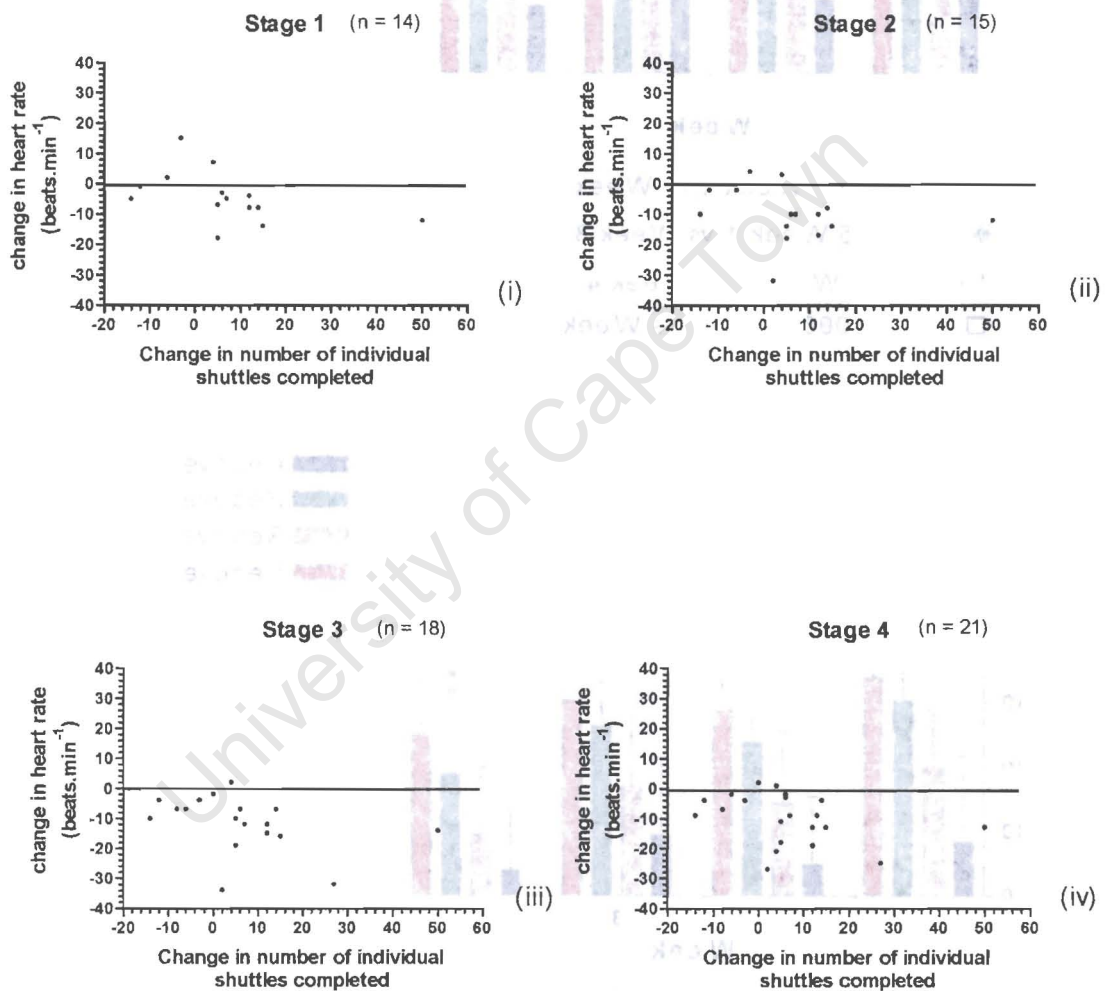
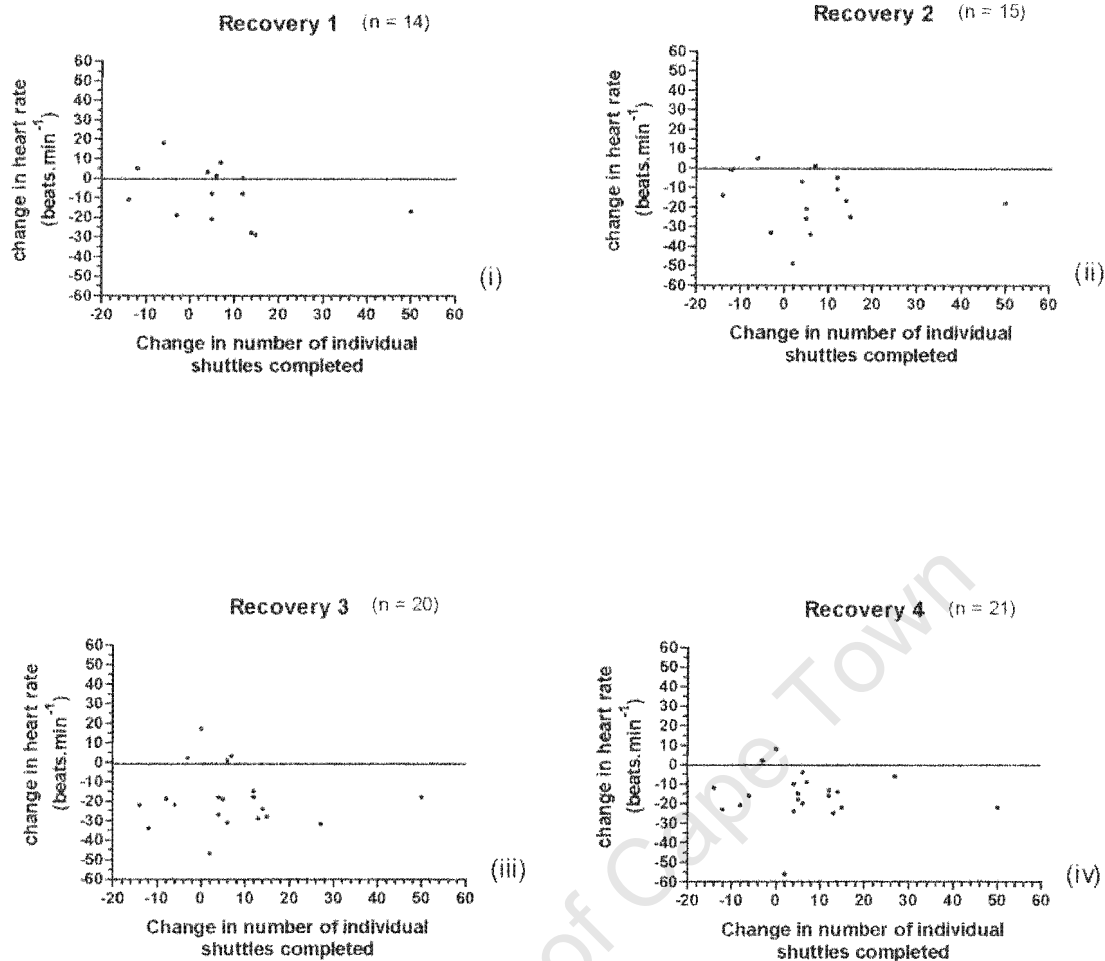


Figure 7.3 a, b Average heart rate (beats.min<sup>-1</sup>) for each of the Stages and Recovery periods recorded during the submaximal heart rate test.

Figure 7.4 a (i,ii,iii,iv) depicts the relationship between the change in heart rate during the four stages of the submaximal heart rate test versus the change in the number of shuttles run during the four weeks. Figure 7.4 b (i,ii,iii,iv) shows a similar pattern during the four recovery phases.



**Figure 7.4 a** The relationship between changes in the delta heart rate (beats.min<sup>-1</sup>) during Stages 1 to 4 of the submaximal heart rate test and the change in the number of shuttles completed during the 20-m Multi-Stage shuttle test.



**Figure 7.4 b** The relationship between changes in the delta heart rate (beats.min<sup>-1</sup>) during Recovery 1 to 4 of the submaximal heart rate test and the change in the number of shuttles completed during the 20-m Multi-Stage shuttle test.

The submaximal heart rate test showed a non-significant change in the average minimum heart rate from  $75 \pm 15$  to  $76 \pm 11$  beats.min<sup>-1</sup> between weeks 1 and 4.

The average maximal heart rate attained during the submaximal heart rate test changed from  $190 \pm 12$  to  $181 \pm 11$  beats.min<sup>-1</sup> between weeks 1 and 4 respectively ( $P < 0.0005$ ). Two subjects increased their maximal heart rate between week 1 and 4, with the biggest increase

being  $14 \text{ beats}\cdot\text{min}^{-1}$ . The biggest decrease was  $-27 \text{ beats}\cdot\text{min}^{-1}$  (between week 1 and 4) with an average change in maximal heart rate of  $11 \text{ beats}\cdot\text{min}^{-1}$ .

To further enhance the interpretation of the data, the subjects were divided into two groups according to the level of change experienced in humidity.

The environmental dry temperature was well controlled and differed by  $1 \pm 1^\circ\text{C}$ . The mean temperature varied between  $15$  to  $16^\circ\text{C}$  between weeks 1 and 4. The mean humidity (%) ranged from  $87 \pm 4$  to  $78 \pm 5$  and  $66 \pm 8$  % respectively ( $P < 0.0001$ ). One group consisted of 15 subjects who experienced a delta humidity change of  $-19$  % between weeks 1 and 4. This group displayed an average change in the number of shuttles completed of  $9 \pm 15$  shuttles, a mean heart rate change during stage 4 of  $-13 \pm 8 \text{ beats}\cdot\text{min}^{-1}$  and during recovery 4 of  $-17 \pm 13 \text{ beats}\cdot\text{min}^{-1}$ . The second group consisted of six subjects who experienced a delta humidity change of  $-27$  %. This group showed an average change in the number of shuttles completed of  $2 \pm 8$  shuttles, a change in the mean heart rate during stage 4 of  $-4 \pm 4 \text{ beats}\cdot\text{min}^{-1}$  and during recovery 4 of  $-13 \pm 13 \text{ beats}\cdot\text{min}^{-1}$ .

To further enhance the interpretation of the data, the subjects were divided into two groups according to whether their heart rate increased or decreased ( $-5 \text{ beats}\cdot\text{min}^{-1}$ ) between week 1 and 4 during stage 4 of the submaximal heart rate test.

Only two subjects' heart rate increased (deteriorated) by an average of  $2 \pm 1 \text{ beats}\cdot\text{min}^{-1}$ , with 13 subjects showing a decreased heart rate (improvement) of  $15 \pm 7 \text{ beats}\cdot\text{min}^{-1}$  that was significant ( $P < 0.005$ ). The change in the number of shuttles completed during the 20 m multi-stage shuttle run increased by an average of  $10 \pm 16$  shuttles in the group whose heart rate had decreased compared to  $2 \pm 3$  shuttles for the group whose heart rate had increased ( $P =$

0.5). The group whose heart rate had decreased during the submaximal heart rate test also showed improvements in maximal heart rate ( $-13 \pm 7$  beats.min<sup>-1</sup>) ( $P < 0.005$ ) and % HRmax ( $-7 \pm 3$  %) ( $P < 0.001$ ). The group whose heart rate had increased presented with an increased maximal heart rate of  $8 \pm 9$  beats.min<sup>-1</sup> and % HRmax of  $4 \pm 5$ %.

Subjects were again divided into two groups depending on whether they had improved (+ 5; n = 12) or deteriorated (- 5; n = 4) on the number of shuttles completed during the 20-m Multi-Stage shuttle test.

Group 1 increased the number of shuttles completed by an average of  $14 \pm 13$  shuttles compared to group 2 who had decreased the number of shuttles completed by  $10 \pm 4$  shuttles ( $P < 0.005$ ). The delta heart rate during the submaximal heart rate test for stage 4 and recovery 4 was not significantly different between group 1 ( $-12 \pm 7$  beats.min<sup>-1</sup> and  $-15 \pm 7$  beats.min<sup>-1</sup>) and group 2 ( $-6 \pm 3$  beats.min<sup>-1</sup> and  $-18 \pm 5$  beats.min<sup>-1</sup>) ( $P = 0.1$ ).

## **7.5 DISCUSSION**

The aim of this study was to determine whether changes in submaximal heart rate could track changes in training status as measured by several performance parameters over a relatively short 3-week training period. Training was tightly controlled and supervised by a sports scientist and took place under well controlled conditions. Training was of a progressive nature, increasing in intensity (volume) every week and was designed to stress subjects and induce a measure of fatigue. Testing also took place under well controlled conditions. For this study subjects were exposed to similar training loads and testing conditions.

The study period was kept short as it is generally accepted that the major physiological changes (maximum oxygen uptake, heart rate) normally occur in the first few weeks of training (Hickson et al., 1981; Wilmore and Costill, 1994). It was also a more practical arrangement to ensure compliance as the subjects were required to attend training four times a week plus a further day for fitness testing over the full study period. Minimizing the risk of injury was a prerequisite as subjects were exposed to a heavy exercise training load.

Although the average change in delta body mass was only  $0.2 \pm 1.1$  kg, large individual changes (+ 2.3 to - 2.0 kg) in body mass occurred in 4 subjects. The changes however, did not affect the heart rates in these individuals and no explanation could be found for the weight changes.

The first finding was that as a group the subjects improved their aerobic level of fitness as can be seen in the number of shuttles completed. The number of shuttles increased significantly from an average of  $77 \pm 20$  to  $84 \pm 18$  shuttles over the study period. During this period 15 subjects improved on the number of shuttles completed, with 6 subjects showing a decrease in performance. Thus, the majority of the subjects were able to adapt to the increased training load. It is also possible that the training protocol was insufficient or of too short a duration to induce fatigue and symptoms of the overtraining syndrome. Other factors that could have affected the results were that subjects may have previously been exposed to similar training protocols or subjects' previous training histories.

The second finding of this study was that the heart rate recovery percentage for the group as a whole improved concurrently with the improved shuttle test results ( $23 \pm 5$  to  $28 \pm 7$  %), again indicating a degree of increased cardiovascular fitness.

However, when the data were related to individual subjects it became clear that the heart rate recovery percentage was not necessarily related to the number of shuttles completed ( $r = -0.16$ ). The subject who completed the most number of shuttles (122 shuttles) during the 20-m Multi-Stage shuttle test after training did so at a recovery heart rate of 31 % during week 3. The subject's heart rate recovery percentage increased to 40% during week 4 even though the number of completed shuttles decreased to 108. This phenomenon was detected in several other subjects. The researchers hypothesized that this finding may be due to a lag period during which time performance already started to deteriorate before the effect is seen in heart rate recovery measurements. The female subject who completed the lowest number of shuttles (37 shuttles) during week 1 recorded a heart rate recovery percentage of 14 %. Another male subject with a similar heart rate recovery percentage (15 %) at the same time however, completed 72 shuttles. This leads one to surmise that other factors may also have an important effect on heart rate changes and performance. The exact mechanism responsible for the decrease in heart rate immediately following exercise, particularly with regard to the autonomic nervous system and the role of the sympathetic and parasympathetic system is still controversial and needs to be further clarified (Carnethon et al., 2005; Kannankeril et al., 2004; Lee et al., 1992; Savin et al., 1982). Motivation may be an important factor as especially the male subjects were perceived to be very competitive during the 20-m Multi-Stage shuttle test and this may well have caused some to perform above their potential.

The average heart rate during the 4<sup>th</sup> stage and recovery periods of the submaximal heart rate test decreased by 12 and 18 beats.min<sup>-1</sup> respectively from week 1 to 4 as was expected with improved fitness levels. This finding is thus consistent with that of Hickson et al (1981) who found that the biggest changes in some physiological parameters occurred in the first two to three weeks after increases in training intensity. Interestingly, the changes in our study occurred in a group of subjects who were all already engaged in some form of training at least

three times a week, so one would have expected heart rate changes of a smaller magnitude. It may well be that the starting level of fitness for the group as a whole was lower than expected.

The 10 and 20-m sprint times remained similar for the duration of the study whilst muscle power as determined by the squat jump increased somewhat from week 1 ( $1326 \pm 306$  watts) to week 4 ( $1362 \pm 258$  watts) ( $P = 0.2$ ). One would have expected these two performance parameters to change concurrently as both are anaerobic type activities. The difference may well be that the squat jump is initially more technically difficult to perform and that the difference may be due to a learning effect rather than an increase in muscle power. The 10-m sprint times especially are very dependent on the subjects' initial reaction time to the signal to start and this skill may take longer to develop. Again the changes were not significant and if the study had continued for another week or so, one may have been able to elicit more definitive changes. The improvements experienced in these two performance parameters may have been more a function of learning and less of improvements in cardiovascular fitness, and as such is difficult to interpret in the context of this study.

Creatine Kinase (CK) activity, as a measure of muscle damage, increased significantly from week 1 to week 2 ( $P = 0.009$ ), indicating a sudden increased training stress, decreased significantly ( $P < 0.05$ ) from week 2 to week 3 as the subjects adapted to the training regime, and then increased again to week 4 as the training volume was increased. Again the duration of the study may have been a limiting factor as increased levels of CK may indicate imminent fatigue and overreaching.

Fatigue levels increased significantly ( $P < 0.01$ ) over the four week period, as was expected.

The eight subjects who had the highest fatigue levels managed to increase the average

number of shuttles by 2 shuttles whilst at the same time decreasing their average heart rate during stage 4 by 10 beats.min<sup>-1</sup> and during recovery 4 by 17 beats.min<sup>-1</sup>. These subjects were probably more unfit than the other subjects and found it difficult to tolerate the training load imposed on them. Five subjects presented with lower levels of fatigue at the end of the 4 week period. On average these subjects increased the number of shuttles run by 6, whilst the average heart rate decreased by much smaller margins than the less well trained group. Average heart rate during stage 4 decreased by 7 beats.min<sup>-1</sup> and by 12 beats.min<sup>-1</sup> during recovery 4. This is in line with what one would expect and is similar to the results from the 18 published studies reviewed by Pollock (1973) in which an average decrease of 7 beats.min<sup>-1</sup> were found.

The environmental dry temperature was well controlled and did not change significantly over the study period. The average humidity (%) however did change significantly and affected the two groups differently. The two groups experienced a change of approximately - 19 % and - 27 % respectively. The first group completed more shuttles and average heart rate decreased more compared to the second group. This finding is consistent with the results in Chapter 5. This confirms that humidity is indeed an important variable that should be considered when interpreting changes in heart rate.

Some of the subjects were identified as not having adapted to the training regime as per their differences in the physiological test results. It was thus decided to divided the group into 2 groups (adapters vs. non-adapters) to try and identify why some subjects improved and others deteriorated under similar training and testing conditions.

When the subjects were divided into two groups depending on whether their heart rate increased (deteriorated) or decreased (improved) by 5 beats.min<sup>-1</sup> or more, it was found that

only two subjects had increased heart rates, with 13 subjects presenting with decreased heart rates. Again the performance during the 20-m Multi-Stage shuttle test was higher in the group whose heart rate had decreased compared to the subject's whose heart rate had increased. This serves to confirm that increased fitness levels are normally accompanied by improved (lowered) heart rates in the majority of athletes, as is generally accepted.

Subjects were also divided into two groups depending on the change in performance during the 20-m Multi-Stage shuttle test. Group 1 consisted of 12 subjects who had completed more shuttles compared to group 2 (n = 4) who had completed fewer shuttles. The delta heart rate changes, however, was not significantly different ( $P = 0.1$ ) between the two groups. This is different to what one would have expected and again highlights the dichotomy in heart rate and training results.

In summary, the submaximal heart rate test is effective in monitoring the general trend in changes in training status in average trained subjects, but is not an accurate measure on an individual basis, with high intra-variability. Subjects in our study responded very differently to similar training volume and testing conditions. This was a somewhat expected result as some subjects would adapt quicker than others when exposed to similar training stress. This observation has been confirmed by various other studies (Barron et al., 1985; Costill et al., 1988; Kajiura et al., 1995; Lehman et al., 1992). The results may have been different if the study had been of a longer duration and of higher exercise intensity, as it seems that the three week period and level of intensity imposed on the athletes were insufficient to induce enough physiological changes to cause athletes to become fatigued and start showing signs of overreaching or the overtraining syndrome.

In the light of the above findings the researchers decided to continue to investigate the changes in heart rate recovery, measures of fatigue and its relationship to changes in training status in a group of elite rugby players over a much longer period of 20 weeks, during which time they were subjected to similar high intensity / high volume training and matches.

University of Cape Town

# CHAPTER 8

RELATIONSHIP BETWEEN HEART  
RATE RECOVERY, FATIGUE AND  
TRAINING TIME

University of Cape Town

## **8.1 ABSTRACT**

This study monitored a group of elite rugby union players ( $n = 15$ ), subjected to a regime of titrated training volume, weekly over a period of 20 weeks. Training volume was adjusted depending on whether recovery heart rate decreased or increased. The aim of the study was to determine whether this approach could maintain constant symptoms of subjective fatigue throughout the season. Data were analyzed using a one-way analysis of variance with a Tukey post-hoc test. The line of best fit was calculated for the normalized training time, subjective rating of training intensity, perception of fatigue and heart rate recovery over the 20 weeks to establish trends in the variables over time. Data were normalized where appropriate. Heart rate recovery data only showed a general improvement as the season progressed ( $R = 0.76$ ). The training time decreased from  $450 \text{ min.wk}^{-1}$  (wk 2) to  $145 \text{ min.wk}^{-1}$  (wk 19) and the subjective intensity of training decreased from  $7 \pm 1$  (wk 1) to  $4 \pm 2$  units (wk 19) ( $P < 0.05$ ). In contrast there was no significant change in their perception of fatigue throughout the season (ranged from  $4 \pm 2$  to  $6 \pm 1$  units). In conclusion, this study shows that titrating training loads in response to changes in recovery heart rate may be a useful tool for maintaining levels of subjective fatigue in a group of elite rugby players.

## **8.2 INTRODUCTION**

Rugby union has been a professional sport since 1995, and players are involved in about 30 games per year (Keohane, 2004). It is a highly competitive international sport and financial gains and losses play an important part in the approach to the game and thus by implication the players.

Although there are studies describing the training volume and fitness parameters of professional rugby union (Gamble, 2004; Holmyard and Hazeldine, 1993; Noakes and Du Plessis, 1997) and amateur rugby league teams (Gabbett, 2005), the length of season, levels of fatigue and the number of matches a player can play is based more on anecdotal information.

It is however, important for players to remain highly competitive throughout the season. As a result players are subjected to high intensity / high volume training and matches almost continuously from the start of the season with very little time to recover. As these are elite athletes they are also expected to perform prescribed pre-season training programmes, mostly strength type of training. This creates a situation where players are at high risk of overtraining (Foster, 1998) and fatigue (Fry et al., 1994), leading to impaired performance. Another potential problem is that players peak and adapt at different rates (Hellard et al., 2005). The demands of the game are also different for players in the different positions (Mashiko et al., 2004).

In the previous study (Chapter 7), we were unable to demonstrate a reasonable model for heart rate recovery, performance and fatigue. This may have been due to the short period over which the study was conducted as well as the fact that the training intensity may not have been sufficiently high to induce the changes wanted.

The aim of this study was therefore to monitor changes in heart rate recovery in a group of elite rugby players over an extended 20-week training period, during which time they were subjected to titrated training levels, to determine if the subjective symptoms of fatigue could be maintained throughout the study period.

### **8.3 METHODOLOGY**

#### *8.3.1 Subjects*

Forty-two players in a professional first division rugby team in South Africa were monitored throughout a 20-week season, starting 5 weeks before the first match and ending on the completion of the tournament which the team won.

All subjects completed pre-season training programmes as prescribed by their own fitness coaches. The subjects' level of fitness was not tested at the start of the study. There were no exclusion criteria at the start of the study, but subjects withdrew if they became injured or were dropped from the squad. Subjects gave informed consent. The study was approved by the Research and Ethics Committee of the University of Cape Town.

### 8.3.2 Experimental design

The trial consisted of a submaximal heart rate test (described in Chapter 4) conducted on a weekly basis throughout the 20-week study period. Testing took place 2-3 days after each match. Body mass was recorded weekly, using the same digital scale.

Total weekly field training time, including warm-up and cool-down time was recorded by the fitness trainer. The rating of training intensity and fatigue was recorded daily by each player, using the subjective rating scale as previously described (Appendix A).

Training was prescribed and monitored by designated fitness coaches. Training followed a periodized plan and was adapted depending on the matches to be played. The heart rate recovery results were given to the fitness coaches on the day of testing, with an interpretation of each player's individual response and recommendations for the next weeks training volume. The fitness coach attempted to regulate the training load based on whether the recovery heart rate increased or decreased. There were no firm guidelines on how the training volume was regulated in response to the change in heart rate recovery; however it is reasonable to assume that if the player's recovery heart rate decreased that the training load would be decreased for the following week.

Training and testing took place at the same time of day. Testing was conducted outside on a grass field.

### 8.3.3 STATISTICAL ANALYSIS

Data are expressed as mean  $\pm$  standard deviation. Data were analyzed using a one-way analysis of variance with a Tukey post-hoc test. The line of best fit was calculated for the normalized training time, subjective rating of training intensity, perception of fatigue and heart rate recovery over the 20 weeks to establish trends in the variables over time. Data were normalized by setting the value at week 1 to 100 and adjusting the subsequent values accordingly. Based on previous work (Lamberts et al., 2004), a sample size of  $n = 7$  results in statistical power of 80%. Statistical significance was accepted as  $P < 0.05$ .

## **8.4 RESULTS**

Several players in the original forty-two man squad were injured at various stages during the season and could not complete all the tests, or were removed from the squad and had to be replaced. Therefore, only the data from 15 players were used for this study.

The average mass of the players at the start of the season was  $107 \pm 15$  kg (range 80 - 124 kg). Body mass did not change throughout the season (Table 8.1).

The first finding was that the heart rate recovery percentage did not change significantly throughout the 20-week period ( $14 \pm 4$  % (week 1) to  $20 \pm 6$  % (week 17) (Table 8.1). When these data were normalized there was a progressive increase in heart rate recovery ( $R = 0.76$ ; Figure 8.1D), suggesting that there was a trend for the heart rate recovery percentage to improve through the season.

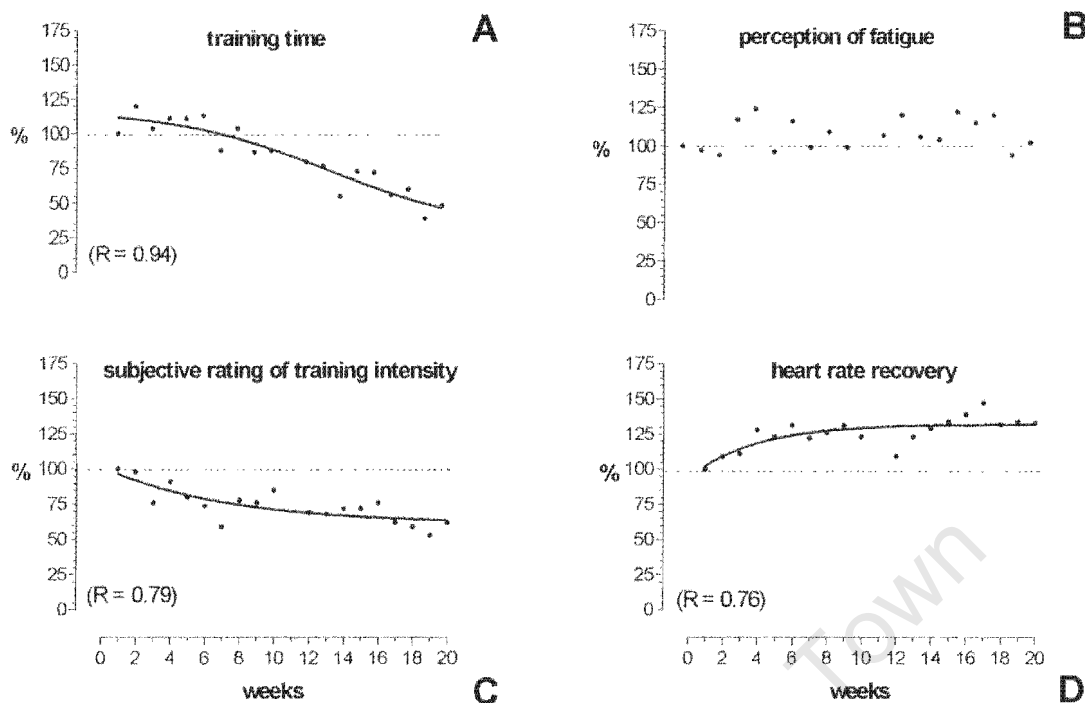
Similarly, the average maximal heart rate during the submaximal heart rate test was not significantly different throughout the 20-week period, ranging from  $168 \pm 11$  beats.min<sup>-1</sup>(week 16) to  $179 \pm 9$  beats.min<sup>-1</sup> (week 2) (Table 8.1).

**Table 8.1** The average body mass, subjective rating of exercise intensity, perception of fatigue and average maximal heart rate during the submaximal heart rate test and heart rate recovery percentage for a 20-week period.

week	n	Body mass (kg)	Training time per week (min.wk <sup>-1</sup> )	Subjective intensity of training (1 - 10)	Perception of fatigue (1 - 10)	Average maximal heart rate (beats.min <sup>-1</sup> )	Average heart rate recovery (%)
1	10	107 ± 15	375	7 ± 1	5 ± 2	174 ± 8	14 ± 4
2	11	106 ± 14	450	7 ± 1	4 ± 2	179 ± 9	15 ± 4
3	9	102 ± 14	390	5 ± 1	4 ± 2	174 ± 12	15 ± 7
4	11	103 ± 15	415	6 ± 1	5 ± 2	173 ± 12	17 ± 6
5	12	103 ± 14	415	5 ± 2	6 ± 1	172 ± 11	17 ± 6
6	15	103 ± 14	425	5 ± 1	4 ± 1	174 ± 10	18 ± 6
7	9	100 ± 15	330	4 ± 0 *	5 ± 1	170 ± 11	16 ± 3
8	13	105 ± 13	390	5 ± 1	5 ± 1	177 ± 8	17 ± 6
9	12	104 ± 14	325	5 ± 1	5 ± 1	170 ± 11	18 ± 5
10	11	107 ± 13	330	6 ± 1	5 ± 1	174 ± 11	17 ± 5
11		-	-	-	-	-	-
12	11	104 ± 15	300	5 ± 1	5 ± 1	172 ± 11	15 ± 5
13	10	104 ± 13	290	5 ± 1	5 ± 1	169 ± 11	17 ± 6
14	8	102 ± 14	205	5 ± 1	5 ± 1	172 ± 11	17 ± 6
15	9	105 ± 15	275	5 ± 0	5 ± 1	171 ± 10	18 ± 6
16	10	101 ± 15	270	5 ± 1	6 ± 2	168 ± 11	19 ± 8
17	11	104 ± 12	210	4 ± 1 *	5 ± 1	169 ± 12	20 ± 6
18	12	102 ± 14	225	4 ± 1 *	5 ± 2	170 ± 10	18 ± 6
19	8	100 ± 16	145	4 ± 2 *	4 ± 1	171 ± 10	18 ± 7
20	12	103 ± 15	180	4 ± 1 *	5 ± 1	168 ± 10	18 ± 5

\* vs. starting values, P < 0.05

Values are expressed as the Mean ± Standard Deviation.



**Figure 8.1** The normalized data over the season for training time (A), players' perception of fatigue (B), players' subjective rating of training intensity (C) and heart rate recovery (D). (n ranged from 8 to 15).

The longest training time was  $450 \text{ min}\cdot\text{wk}^{-1}$  which coincided with week 2 (three weeks before the first match) and the shortest training time was  $180 \text{ min}\cdot\text{wk}^{-1}$  which coincided with week 19 (the week preceding the final match) (Table 8.1). The normalized training data showed a progressive curvilinear decline ( $R = 0.94$ ; Figure 8.1A). After the data were normalized it was shown that at the end of the season, coinciding with the playoff matches, the team trained only 39% of the time compared to the training time at the start of the season.

The players' perception of fatigue remained fairly constant throughout the season, ranging from  $4 \pm 2$  units (week 3) to  $6 \pm 2$  units (week 15). There was no trend in the data when it was normalized (Figure 8.1B).

The subjective intensity of training rating ranged from  $7 \pm 1$  units during week 1 to  $4 \pm 2$  units during week 19. The subjective intensity of training were significantly lower than the starting value ( $P < 0.05$ ) at weeks 7, 17, 18, 19 and 20 (Table 8.1). The normalized data showed a progressive curvilinear decline ( $R = 0.79$ , Figure 8.1C).

The team won 9 of their matches and lost only 2. The 2 matches were lost in consecutive weeks (14 and 15) but were then followed up by 4 consecutive match wins, of which the last game was the final (Figure 8.2).

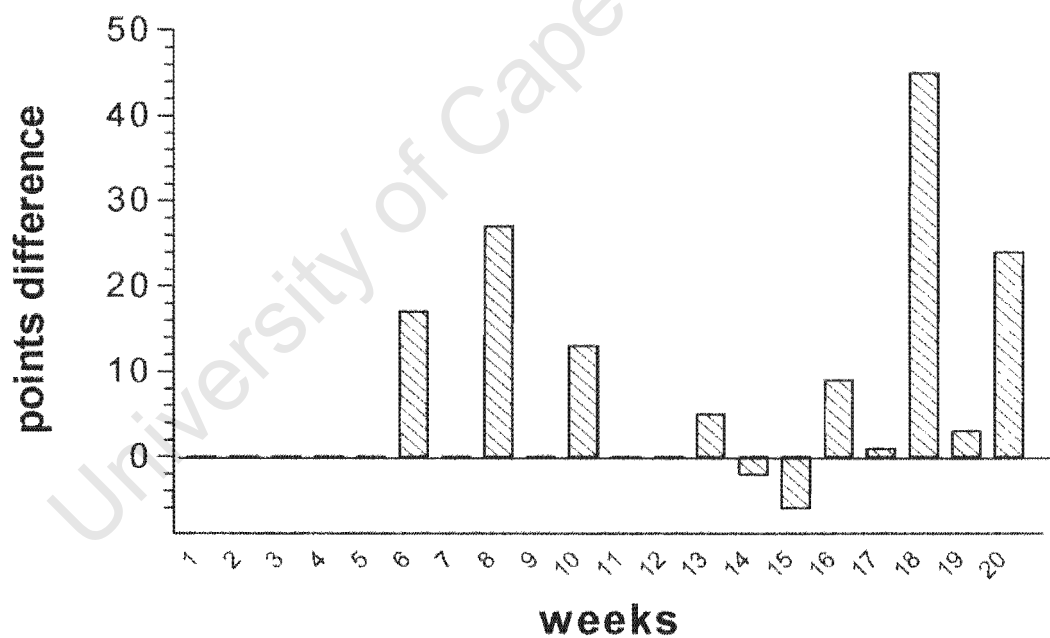


Figure 8.2 The difference in points in matches played. A positive score indicates that the team that was studied won the match.

## 8.5 DISCUSSION

The aim of this study was to monitor changes in heart rate recovery and subjective rating of fatigue in a group of elite rugby players over an extended 20-week training period, during which time they were subjected to a regime of titrated training loads, depending on whether their recovery heart rate increased or decreased. The goal was to determine if this approach would be successful in maintaining a constant rating of subjective fatigue throughout the study period in this group of subjects.

The first finding was that although the average maximal heart rate remained fairly constant throughout the season, the heart rate recovery percentage increased somewhat over the 20-week period, coinciding with the phase in the season when the team became dominant and started winning matches comfortably (Figure 8.1d). Even though heart rate changes were smaller than expected, this may in part be due to the fact that as elite players, they maintained their training in the pre-season and thus they were already more trained at the beginning of the season. An increase in heart rate recovery can be interpreted as a positive response from the player's perspective, as it showed that heart rate was recovering at a faster rate, possibly as a result of tighter regulation by the autonomic nervous system, in particular the result of vagal reactivation (Imai et al., 1994). It may be argued that the quicker recovery of the elevated heart rate is associated with increased "fitness" (Tiukinhoy et al., 2003). Although this is a fairly broad term, it may be applied to rugby union where a player's fitness is highly dependent on quick recovery from short duration, high intensity bouts of exercise. Typically rugby union consists of repetitive bouts of short duration (less than 60 seconds) high intensity exercise, followed by short rest periods (less than 60 seconds) (Duthie et al., 2003). Therefore, a test which monitors the rate of recovery of heart rate has some relevance to the fitness demands of the sport. It is important to note that these changes occurred at the end of the season coinciding with the

important play-off matches, perhaps explaining, in part, the fact that the team won the tournament.

The second finding from this study was that the players' perception of fatigue remained fairly constant throughout the 20-week season. This occurred however, despite a decrease in training time of over 60% throughout the season and significant reductions in the subjective intensity of training ratings. This is an interesting finding which suggests that the perception of effort can be a "protected" outcome variable regulated by the training load. It is reasonable to assume that the stress incurred from continuous training and playing matches would have resulted in symptoms of overreaching and impaired performance (Budgett, 1990; Fry et al., 1994; Fry et al., 1991; Fry and Kraemer, 1997) had the training load not been reduced. The fact that this team won the tournament also suggests that the manipulation of training load by the coaching staff to keep the perception of effort constant may have partly contributed to the team's success. It would have been interesting to have repeated the experiment with one of the other teams in the tournament, particularly the higher ranked teams who faded badly as the tournament progressed. It is tempting to speculate that the perception of effort / training load of the players in these other teams had not been regulated as tightly as it had been in the team the researchers studied.

As the season progressed, the training load was reduced by approximately 60%. It is logical to assume that this reduced training load was not sustainable in the long run and that players would have started losing their training adaptations had the season progressed for much longer. A more recent study on rugby league players during a competitive season showed that muscle power and maximal aerobic power decreased and skinfold thickness increased towards the end of the season when the training loads were the lowest and match loads the highest. This period also coincided with an increased risk of injury (Gabbett, 2005). An earlier study on

elite rugby union players confirmed similar findings (Holmyard and Hazeldine, 1993). This emphasizes the importance of players following periodized training programmes and an adequate pre-season phase of training before the next competition

In conclusion, these data support the importance of gathering subjective information from the players about their perception of fatigue during the season as these measurements have been shown to be accurate predictors of overtraining (Hooper et al., 1995). In our study players did not display symptoms of the overtraining syndrome and subjective ratings of fatigue remained constant as the training load decreased during the season. This may have been different had the training load not been adjusted in response to heart rate recovery changes.

This study also suggests that training, titrated in response to changes in recovery heart rate, is a useful objective measure in a group of elite rugby players with heart rate recovery increases coinciding with when the team was performing at its best during the season.

# CHAPTER 9

## SUMMARY AND CONCLUSIONS

University of Cape Town

## 9.1 SUMMARY AND CONCLUSIONS.

The purpose of this thesis was to further explore and clarify the heart rate / workload relationship under differing conditions such as well controlled testing vs. field testing conditions, controlled vs. uncontrolled training, changed training status vs. unchanged training status and average trained vs. elite athletes over varying time periods.

The main goal was to determine if measuring heart rate and heart rate recovery under these different conditions has sufficient precision to track changes in training status and therefore be considered a viable and practical marker that can be used by athletes and coaches to monitor changes in training status and for exercise prescription.

A summary of the thesis research questions and answers follows:

1. *Is measuring heart rate during sleep repeatable under free-living conditions in a group of subjects whose training status remained unchanged ? (Chapter 2).*

Answers:

- Average heart rate for the group during sleep remained similar over the three week period, however, the individual variation was about 8 beats.min<sup>-1</sup>.
- Minimum heart rate during sleep had the least variation (0 – 10 beats.min<sup>-1</sup>; average  $5 \pm 3$  beats.min<sup>-1</sup>), with maximal heart rate during sleep the most (2 – 31 beats.min<sup>-1</sup>; average  $13 \pm 9$  beats.min<sup>-1</sup>).

2. *Does heart rate during sleep change under free-living, but well controlled conditions in a subject whose training status changed ? (Chapter 3).*

Answers:

- Average heart rate during sleep remained the same over the full 18 week study period, despite performance improving by nearly 70 % and a large decrease in training load.

3. *Do changes in submaximal heart rate during exercise reflect changes in training status under well controlled testing conditions in a subject whose training status changed ? (Chapter 4).*

Answers:

- Submaximal heart rate changes tracked changes in training status.
- Heart rate recovery improved concurrently with improvements in training status.
- Average minimum and maximum heart rate decreased progressively as training status improved.

4. *Do changes in submaximal heart rate during exercise reflect changes in training status under field-testing conditions in a group of subjects whose training status changed ? (Chapter 5).*

Answers:

- Changes in submaximal heart rate and heart rate recovery seemed to track the general trend of changes in training status in this group of subjects. However, once the data were examined on an individual basis, the heart rate changes were quite diverse with no specific relationship to changes in performance.
- Changes in RPE during the 5-m Multiple shuttle test and performance were not related.
- Environmental conditions were a confounding factor in the study.

5. *Do changes in the heart rate / workload relationship accurately reflect changes in training status under well controlled testing conditions in a group of extreme adventure racers undergoing heavy multi-modality training ? (Chapter 6).*

Answers:

- Training time and intensity varied and the training volume of the subjects was lower than expected, considering the intensive nature of the sport.

- Even though heart rate recovery improved meaningfully over the study period, performance parameters remained the same.
- Fatigue and muscle soreness levels also remained similar and did not correlate with heart rate, training load and performance changes.
- Subjects seem to train at a higher intensity compared to the intensity at which they compete. This is in contrast to most other sporting disciplines but may just be a reflection of the type of competition protocol.

*6. Do changes in the heart rate / workload relationship accurately reflect changes in training status under a controlled regime of progressive training overload under controlled testing conditions when training status changed ? (Chapter 7).*

Answers:

- Subjects responded differently to similar training and testing conditions.
- The majority of the subjects adapted well to the training regime and performance parameters improved. However, again heart rate changes were not necessarily related to the changes in performance.
- Environmental conditions, especially humidity, affected heart rate.

7. Does perception of fatigue and changes in heart rate recovery relate to training time and intensity under well controlled training and testing conditions in a group of subjects whose training status changed ? (Chapter 8).

Answers:

- Perception of fatigue remained constant throughout the study.
- The training load was titrated according to heart rate recovery results and decreased to approximately 39 % of the training load compared to at the start of the study period.
- Heart rate recovery showed an upward trend over the study period, indicating an improvement in training status.

## Conclusions

The main finding of this study was that a dissociation exists between changes in heart rate and changes in performance parameters. This seems to be the case regardless of the training and testing conditions, training status of the athlete or changes in training status (fitness levels).

A second finding was that rating of perceived exertion, fatigue and muscle soreness ratings were also not sufficiently correlated to changes in performance and training load variations under the different conditions.

Another finding was that environmental conditions, especially humidity, seem to have an effect on heart rate during exercise.

Collectively, these findings should be interpreted in the context that heart rate and heart rate recovery changes are a function of a well balanced autonomic nervous system and that any imbalance or dysfunction of the autonomic nervous system will translate into indifferent heart rate changes independent of performance changes.

The performance of athletes should be viewed in a holistic manner, with no single physiological test being definitive in explaining training status. As such the measurement of heart rate should be seen as part of the holistic approach when evaluating athletes in conjunction with other available and applicable physiological tests.

Heart rate, heart rate recovery and performance should be seen to function on a continuum and an athlete at any one time will function at different points on this continuum depending on various factors. As such there may be a lag period during which heart rate and performance changes may not necessarily correspond to each other at the same point in time. At this time there are insufficient data to state with certainty whether heart rate recovery changes precedes changes in performance or *vice versa*.

To further illustrate the above, a scheme of possible scenarios has been proposed to indicate where an athlete may find him / herself during different stages of training.

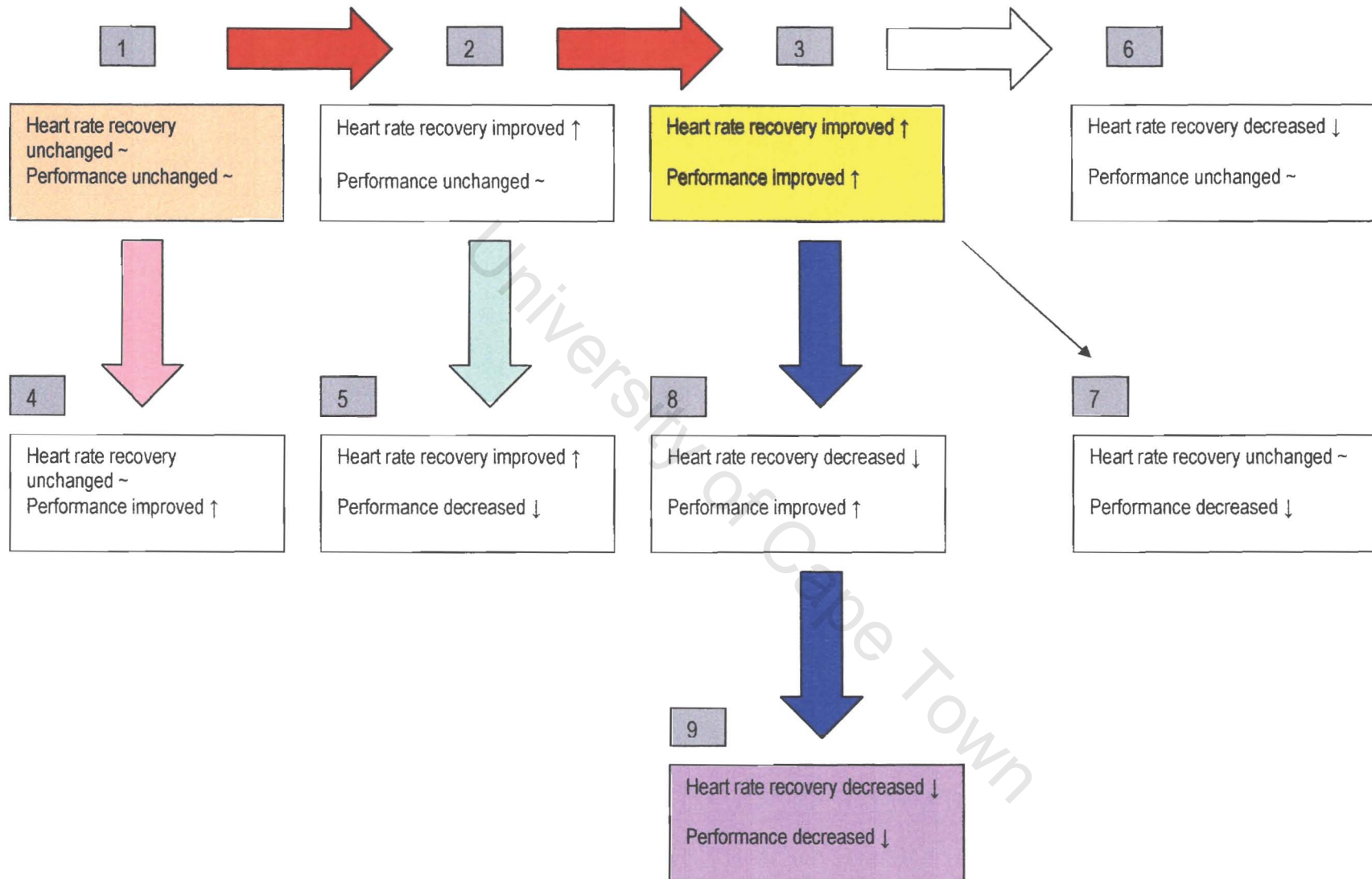


Figure 9.1 Proposed scheme of possible scenarios of an athletes' heart rate recovery status during different stages of training.

At the start of training one can assume athletes to be at phase 1 where heart rate and performance are related and set at a certain level.

After several weeks of training, one of two scenarios may unfold. Athletes may progress to phase 2, where heart rate recovery measurements start showing an improvement, due to cardiac autonomic changes, but this is not yet reflected in performance measures. Another scenario may be where heart rate recovery changes are not yet evident, but performance start improving (phase 4). As mentioned earlier, it is still not evident whether heart rate or performance changes occur first.

Once the athlete reaches phase 2, one of two processes may be followed. If heart rate and performance changes occur as expected and the athlete adapts optimally, the athlete would then progress to phase 3. This phase (3) is characterized by improved heart rate and performance parameters and would be the desired scenario for athletes. However, the athlete may present with improved heart rate recovery measurements, but with a decreased performance (phase 5). This is a less likely scenario and other factors would have to be eliminated such as injuries, poor motivation etc.

If training and recovery are ideal, athletes should remain at phase 3. From here however, several different scenarios may follow:

Athletes may progress to phase 6, where heart rate recovery measurements start decreasing as the autonomic nervous system becomes stressed, but performance is not yet affected. The

athlete could also present with unchanged heart rate recovery measurements, but performance is adversely affected (phase 7). During these phases one may need to assess the athlete in respect of possible overreaching.

From phase 3, the athlete may present with a reduced heart rate recovery as the autonomic nervous system becomes overstressed, but performance still improves (phase 8). Here the athlete may still be sufficiently conditioned not to display reductions in performance. The worst case scenario would be for athletes to start showing signs of the overtraining syndrome as reflected in decreased heart rate recovery and performance measurements (phase 9). This phase is indicative of severe cardiac autonomic imbalance.

In retrospect we may have employed a different research design at some point to further examine individual heart rate, heart rate recovery and performance changes, taking the above scheme and findings into consideration. Future studies should examine individual responsiveness to training load, fatigue and heart rate responses.

The take home message for coaches and athletes is that even though the measurement of heart rate and heart rate recovery is repeatable and reliable, it is not sufficiently correlated to changes in performance to be used as an independent marker of training status in all athletes. However, there are sufficient data to recommend that athletes should undergo baseline heart rate testing, using mode-specific, graded exercise testing to determine their intrinsic day-to-day variation. Thereafter, regular heart rate measurements, in conjunction with other measurements (training load, perception of effort, muscle soreness) may provide useful information for ensuring that the athletes' training progresses at the expected rate.

# REFERENCES

University of Cape Town

## REFERENCES.

- Adams, W.C., Fox, R.H., Fry, J. and MacDonald, I.C. (1975). Thermoregulation during marathon running in cool, moderate and hot environments. *Journal of Applied Physiology*. 38, 1030 – 1037.
- Aschoff, J. (1965). Circadian rhythms in man. *Science*. 148, 1427 - 1432.
- Åstrand, P.O. and Rodahl, K. (1986). Textbook of Work Physiology. Physiological Bases of Exercise. (3rd edition). McGraw – Hill Publishers, New York.
- American College of Sports Medicine. (1995). Guidelines for Exercise Testing and Prescription. (Fifth edition). Williams and Wilkins. London, Munich.
- Arts, F.J.P., Kuipers, H. (1994). The relation between power output, oxygen uptake and heart rate in male athletes. *Journal of Sport Sciences*. 9,183-189
- Atkinson, G. and Reilly, T. (1996). Circadian variation in sports performance. *Sports Medicine*. Apr; 21 (4), 292 – 312.
- Atkinson, G., Coldwells, A., Reilly, T. et al (1993). A comparison of circadian rhythms in work performance between physically active and inactive subjects. *Ergonomics*. 36, 273 – 281.
- Atkinson, G., Coldwells, A., Reilly, T. et al. (1994). The influence of age on diurnal variations in competitive cycling performance. *Journal of Sports Science*. 12, 127 – 128.

- Aubert, A.E., Seps, B. and Beckers, F. (2003). Heart rate variability in athletes. *Sports Medicine*. 33 (12), 889 – 919.
- Avalos, M., Hellard, P. and Chatard, J.C. (2003). Modeling the training-performance relationship using a mixed model in elite swimmers. *Medicine and Science in Sports and Exercise*. May; 35 (5), 838 – 846.
- Baechle, T.R., Earle, R.E. and Wathen, D. (editors). (2000). *Essentials of Strength Training and Conditioning*. Chp 18, NSCA. Human Kinetics, Champaign, IL.
- Ballarin, E., Sudhues, U., Borsetto, C., Casoni, I., Grazi, G., Guglielmini, C., Manfredinini, F., Mazzoni, G. and Conconi, F. (1996). Reproducibility of the Conconi Test: Test repeatability and observer variations. *International Journal of Sports Medicine*. 17, 520 – 524.
- Banfi, G., Del Fabbro, M., Mauri, C., Corsi, M.M. and Melegati, G. (2006). Haematological parameters in elite rugby players during a competitive season. *Clinical and Laboratory Haematology*. Jun; 28 (3), 183 – 188.
- Barauna, V.G., Junior, M.L., Costa Rosa, L.F., Casarini, D.E., Krieger, J.E. and Oliveira, E.M. (2005). Cardiovascular adaptations in rats submitted to a resistance-training model. *Clinical and Experimental Pharmacology and Physiology*. Apr; 32 (4), 249 – 254.
- Barron, J.L., Noakes, T.D., Levy, W., Smith, C. and Millar, R.P. (1985). Hypothalamic dysfunction in overtrained athletes. *Journal of Clinical Endocrinology and Metabolism*. 60, 803 – 806.

Berne, R.M. and Levy, M.N. (1997). Cardiovascular physiology. (7<sup>th</sup> edition). St Louis (MO). Mosby.

Bevier, W.C., Bunnell, D.E. and Horvarth, S.M. (1987). Cardiovascular function during sleep of active older athletes and the effects of exercise. *Experimental Gerontology*. 22, 329 – 337.

Bassett, D.R. and Howley, E.T. (1997). Maximal oxygen uptake: “classical” versus “contemporary” viewpoints. *Medicine and Science in Sports and Exercise*. 591 – 603.

Bhan, A.K. and Scheuer, J. (1972). Effects of physical training on cardiac actomyosin triphosphate activity. *American Journal of Physiology*. 223, 1486 – 1490.

Bhan, A.K. and Scheuer, J. (1975). Effects of physical training on cardiac myosin ATPase activity. *American Journal of Physiology*. 228, 1178 – 1182.

Billat, L.V. (1996). Use of blood lactate measurements for prediction of exercise performance and for control of training. Recommendations for long-distance running. *Sports Medicine*. 22, 157 – 175.

Bird, S., George, M., Theakston, S., Balmer, J. and Davison, R.C. (2003 a). Heart rate responses of male orienteers aged 21 -67 years during competition. *Journal of Sports Science*. Mar; 21 (3), 221 – 228.

Bird, S., George, M., Balmer, J. and Davison, R.C. (2003 b). Heart rate responses of women aged 23 – 67 years during competitive orienteering. *British Journal of Sports Medicine*. Jun; 37 (3), 254 – 257.

Bland, J.M. and Altman, D.G. (1986). Statistical methods for assessing agreement between two methods of clinical measurement. *The Lancet* . Feb 8, 307 – 311.

Blomqvist, C.G. and Saltin, G. (1983). Cardiovascular adaptations to physical training. *Annual Review of Physiology*. 45,169-189

Boddington, M.K., Lambert, M.I., St Clair Gibson, A. and Noakes, T.D. (2001). Reliability of a 5-m multiple shuttle test. *Journal of Sport Sciences*.19, 223-228

Boddy, K., Hume, R., King, P.C., Weyers, E. and Rowan, T. (1974). Total body, plasma erythrocyte potassium and leucocyte ascorbic acid in "ultra-fit" subjects. *Clinical Science and Molecular Medicine*. 46, 449 – 455.

Bonaduce, D., Petretta, M., Cavallaro, V., Apicella, C., Ianniciello, A., Romano, M., Breglio, R. and Marciano, F. (1997). Intensive training and cardiac autonomic control in high level athletes. *Medicine and Science in Sport and Exercise*. 691 – 696.

Booth, J., Marino, F. and Ward, J.J. (1997). Improved running performance in hot humid conditions following whole body precooling. *Medicine and Science in Sports and Exercise*. 29, 943 – 949.

Borg, G.A.V. (1973). Perceived exertion: a note on history and methods. *Medicine and Science in Sports*. 5, 90 – 99.

Borg, G. (1961). Interindividual scaling and perception of muscular force. *Kungliga Fysiografiska Sällskapet I Lund Forhandlingar*. 31, 117 – 125.

Borg, G., Hassmen, P. and Lagerstrom, M. (1987). Perceived exertion related to heart rate and blood lactate during arm and leg exercise. *European Journal of Applied Physiology*. 56, 679 – 685.

Borg, G. and Linderholm, H. (1970). Exercise performance and perceived exertion in patients with coronary insufficiency, arterial hypertension and vasoregulatory asthenia. *Acta Medica Scandinavica*. 187, 17 – 26.

Bouchard, C. and Rankinen, T. (2001). Individual differences in response to regular physical activity. *Medicine and Science in Sports and Exercise*. Jun; 33 (6 Suppl), S 446 – S451.

Boudet, G., Garet, M., Bedu, M., Albuissou, E. and Chamoux, A. (2002). Median maximal heart rate for heart rate calibration in different conditions: laboratory, field and competition. *International Journal of Sports Medicine*. May; 23 (4), 290 – 297.

Bourgois, J. and Vrijens, J. (1998). The Conconi Test: A controversial concept for the determination of the anaerobic threshold in young rowers. *International Journal of Sports Medicine*. 19, 553 – 559.

Brisswalter, J. and Legros, P. (1994). Daily stability in energy cost in running, respiratory parameters and stride rate among well-trained middle distance runners. *International Journal of Sports Medicine*. 15, 238 – 241.

Brorson, L., Conradson, T.B., Olsson, B. et al. (1976). Right atrial monophasic action potential and effective refractory periods in relation to physical training and maximal heart rate. *Cardiovascular Research*. 10, 160 – 168.

Budgett, R. (1990). Overtraining syndrome. *British Journal of Sports Medicine*. 24, 231 – 236.

Bunc, V., Heller, J. and Leso, J. (1988). Kinetics of heart rate responses to exercise. *Journal of Sports Science*. 6 (1), 39 – 48.

Bunnell, D.E., Bevier, W.C. and Horvarth, S.M. (1983). Nocturnal sleep, cardiovascular function and adrenal activity following maximum-capacity exercise. *Electroencephalography and Clinical Neurophysiology*. 56, 186-189.

Busso, T., Häkkinen, K., Pakarinen, A., Kauhanen, H., Komi, P.V. and Lacour, J.R. (1992). Hormonal adaptations modelled responses in elite weightlifters during 6 weeks of training. *European Journal of Applied Physiology*. 64, 381 – 386.

Calbet, J.A., Lundby, C., Koskolou, M. and Boushel, R. (2006). Importance of hemoglobin concentration to exercise: acute manipulations. *Respiratory Physiology and Neurobiology*. Apr; 28 151 (2-3), 132 – 140.

Callister, R., Callister, R.J., Fleck, S.J. and Dudley, G.A. (1989) Physiological and performance responses to overtraining in elite judo athletes. *Medicine and Science in Sports and Exercise*. 22, 816-824.

Carnethon, M.R., Jacobs, D.R Jr., Sidney, S., Sternfeld, B., Gidding, S.S., Shoushtari, C. and Liu, K. (2005). A longitudinal study of physical activity and heart rate recovery: CARDIA, 1987 – 1993. *Medicine and Science in Sports and Exercise*. Apr; 37 (4), 606 – 612.

Carter, J.B., Banister, E.W. and Blaber, A.P. (2003 a). Effect of endurance exercise on autonomic control of heart rate. *Sports Medicine*. 33 (1), 33 – 46.

Carter, J.B., Banister, E.W. and Blaber, A.P. (2003 b). The effect of age and gender on heart rate variability after endurance training. *Medicine and Science in Sports and Exercise*. Aug; 35 (8), 1333 – 1340.

Carton, R.L. and Rhodes, E.C. (1985). A critical review of the literature on ratings scales for perceived exertion. *Sports Medicine*. May – Jun; 2 (3), 198 – 222.

Catai, A.M., Chacon – Mikahil, M.P., Martinelli, F.S., Forti, V.A., Silva, E., Golfetti, R., Martins, L.E., Szrajter, J.S., Wanderley, J.S., Lima-Filho, E.C., Milan, L.A., Marin-Neto, J.A., Maciel, B.C. and Gallo-Junior, L. (2002). Effects of aerobic exercise training on heart rate variability during wakefulness and sleep and cardiorespiratory responses of young and middle-aged healthy men. *Brazilian Journal of Medical and Biological Research*. Jun; 35 (6), 741 – 752.

Chen, H.I. (1991). Effects of 30-h sleep loss on cardiorespiratory functions at rest and in exercise. *Medicine and Science in Sports and Exercise*. Feb; 23 (2), 193 – 198.

Claremont, A.D., Nagle, F., Reddan, W.D. and Brooks, G.A. (1975). Comparison of metabolic, temperature, heart rate and ventilatory responses to exercise at extreme ambient temperatures (0° and 35°C). *Medicine and Science in Sports*. 7 (2), 150 – 154.

Cochrane, D.J. and Sleivert, G.G. (1999). Do changing patterns of heat and humidity influence thermoregulation and endurance performance? *Journal of Science and Medicine in Sport*. Dec; 2(4), 322 – 332.

Cohen, C.J. (1980). Human circadian rhythms in heart rate response to a maximal exercise stress. *Ergonomics*. 23, 591 – 595.

Cohen, C.J. and Muehl, G.E. (1977). Human circadian rhythms in resting and exercise pulse rates. *Ergonomics*. 20, 475 – 479.

Collet, C., Roure, R., Delhomme, G., Dittmar, A., Rada, H. and Vernet-Maury, E. (1999). Autonomic nervous system responses as performance indicators among volleyball players. *European Journal of Applied Physiology and Occupational Physiology*. 80, 41 – 51.

Conconi, F., Grazi, G., Casoni, I., Guglielmini, C., Borsetto, C., Ballarin, E., Mazzoni, G., Patracchini, M. and Manfredi, F. (1996). The Conconi Test: Methodology after 12 years of application. *International Journal of Sports Medicine*. 17, 509 – 519.

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

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

Convertino, V.A., Mack, G.W. and Nadei, E.R. (1991). Elevated central venous pressure: A consequence of exercise training-induced hypervolemia? *American Journal of Physiology*. 260, R 273 – 277.

Conroy, R.T.W.L. and O'Brien, M. (1974). Diurnal variation in athletic performance. *Journal of Physiology*. 236 – 251.

Cooke, W.H. and Carter, J.R. (2005). Strength training does not affect vagal-cardiac control or cardiovagal baroreflex sensitivity in young healthy subjects. *European Journal of Applied Physiology*. Mar; 93 (5 -6), 719 – 725.

Costill, D.L., Flynn, M.G., Kirwan, J.P., Houmard, J.A., Mitchell, J.B., Thomas, R. and Park, S.H. (1988). Effects of repeated days of intensified training on muscle glycogen and swimming performance. *Medicine and Science in Sports and Exercise*. 20 (3), 249 – 254.

Coyle, E.F. and Hamilton, M.T. (1990). Fluid replacement during exercise: effects on physiological homeostasis and performance. In: *Perspectives in Exercise Science and Sports*

*Medicine*. Gisolfi, C.V and Lamb, D.R. (editors). Fluid homeostasis during exercise. Benchmark Press, Indianapolis. 3, 281 – 303.

Coyle, E.F. and Gonzalez-Alonso, J. (2001). Cardiovascular drift during prolonged exercise: new perspectives. *Exercise and Sports Science Review*. Apr; 29 (2), 88 – 92.

Dalton, B., McNaughton, L. and Davoren, B. (1997). Circadian rhythms have no effect on cycling performance. *International Journal of Sports Medicine*. 18 (7), 538 – 542.

Daniels, J., Scardina, N., Hayes, J. and Foley, P. (1984). Variations in  $\text{VO}_2$  submax during treadmill running. *Medicine and Science in Sports and Exercise*. 16, 108.

Daniels, J.T., Yarbrough, R.A. and Foster, C. (1978). Changes in  $\text{VO}_2$ max and running performance with training. *European Journal of Applied Physiology*. 39, 249 – 254.

Darr, K.C., Bassett, D.R., Morgan, B.J. and Thomas, D.P. (1988). Effects of age and training status on heart rate recovery after peak exercise. *American Journal of Physiology*. Feb; 254 (2 Pt 2), H 340 – 343.

Day, M.L., McGuigan, M.R., Brice, G. and Foster, C. (2004). Monitoring exercise intensity during resistance training using the session RPE scale. *Journal of Strength and Conditioning Research*. 18, 353 – 358.

Dennis, S.C., Noakes, T.D. and Bosch, A.N. (1992). Ventilation and blood lactate increase exponentially during incremental exercise. *Journal of Sports Science*. 10, 437 – 449.

Derman, W. and Schwelinnuss, M. (1998). *The Journal of Modern Pharmacy*. 12 – 15.

Doherty, M., Nobbs, L. and Noakes, T.D. (2003). Low frequency of the “plateau phenomenon” during maximal exercise in elite British athletes. *European Journal of Applied Physiology*. Aug; 89 (6), 619 – 623.

Dowell, R.T. (1983). Cardiac adaptations to exercise. *Exercise and Sport Science Review*. 99-117.

Dressendorfer, R.H., Wade, C.E, and Scaff, J.H. (1985). Increased morning heart rate in runners: A valid sign of overtraining ? *Physician and Sports Medicine*. 13, 77-86.

Dunbar, C.C., Robertson, R.J., Baun, R., Blandin, M.F., Metz, K., Burdett, R. and Goss, F.L. (1992). The validity of regulating exercise intensity by ratings of perceived exertion. *Medicine and Science in Sports and Exercise*. 24 (1), 94 – 99.

Durnin, J.V. and Womersley, J. (1974). Body fat assessed from the total body density and its estimation from skinfold thickness: measurements on 481 men and women aged from 16 to 72 years. *British Journal of Nutrition* .32, 77 – 97.

Duthie, G., Pyne, D. and Hooper, S. (2003). Applied physiology and game analysis of rugby union. *Sports Medicine*. 33, 973 – 991.

Elias, A.N. and Wilson, A.F. (1993). Exercise and gonadal function. *Human Reproduction*. 8, 1747 – 1761.

- Fagard, R.H., Aubert, A.E., Lysens, R., Staessen, J., Vanhees, L. and Amery, A. (1983). Noninvasive assessment of seasonal variations in cardiac structure and function in cyclists. *Circulation*. 67 (4), 896 – 901.
- Fagard, R., Aubert, A.E., Staessen, J., Eynde, E.V., Vanhees, L. and Amery, A. (1984). Cardiac structure and function in cyclists and runners: comparative echocardiographic study. *British Heart Journal*. 52 (2), 124 – 129.
- Faria, I.E. and Drummond, B.J. (1982). Circadian changes in resting heart rate and body temperature, maximal oxygen consumption and perceived exertion. *Ergonomics*. 25, 381 – 386.
- Fay, L., Londeree, B.R., LaFontaine, T.P. and Volek, M.R. (1989). Physiological parameters related to distance running performance in females. *Medicine and Science in Sport and Exercise*. 21, 319 – 324.
- Fellman, N., Bedu, M., Boudet, G., Mage, M., Sagnol, M., Pequignot, J.M., Claustrat, B., Brun, J., Peyrin, L. and Coudert, J. (1992). Inter-relationships between pituitary hormones and catecholamines during a 6-day Nordic ski race. *European Journal of Applied Physiology*. 64, 258 – 265.
- Filaire, E., Legrand, B., Lac, G. and Pequignot, J.M. (2004). Training of elite cyclists: effects on mood state and selected hormonal responses. *Journal of Sports Sciences*. Nov – Dec; 22 (11 – 12), 1025 – 1033.

- Fisher, M., Paolone, V., Rosene, J., Drury, D., Van Dyke, A. and Moroney, D. (1999). The effect of submaximal exercise on recovery hemodynamics and thermoregulation in men and women. *Research Quarterly in Exercise and Sport*. Dec; 70 (4), 361 – 368.
- Fordham, S., Garbutt, G. and Lopes, P. (2004). Epidemiology of injuries in adventure racing athletes. *British Journal of Sports Medicine*. Jun; 38 (3), 300 – 303.
- Fortney, S.M., Nadel, E.R., Wenger, C.B. and Bove, J.R. (1981). Effect of acute alterations of blood volume on circulatory performance in humans. *Journal of Applied Physiology*. 50, 292 – 298.
- Foster, C. (1998). Monitoring training in athletes with reference to overtraining syndrome. *Medicine and Science in Exercise and Sport*. July; 30 (7), 1164 – 1168.
- Foster, C., Daines, E., Hector, L., Snyder, A.C. and Welsh, R. (1996). Athletic performance in relation to training load. *Wisconsin Medical Journal*. June, 370 – 374.
- Foster, C., Green, M.A., Snyder, A.C. and Thompson, N.N. (1993). Physiological responses during simulated competition. *Medicine and Science in Sports and Exercise*. 877 – 882.
- Foster, C., Hector, L., Welsh, R., Schragar, M., Green, M. and Snyder, A.C. (1995). Effects of specific versus cross training on running performance. *European Journal of Applied Physiology*. 70, 367 – 372.

Foster, C., Snyder, A.C., Thompson, N.N. and Kuettel, K. (1988). Normalization of the blood lactate profile in athletes. *International Journal of Sports Medicine*. 9, 198 – 200.

Fox, E., Bowers, R. and Foss, M. (1993). In: *The Physiological Basis for Exercise and Sport*. (5<sup>th</sup> edition) Brown and Benchmark Publishers, Madison, Wisconsin.

Fry, A.C. and Kraemer, W.J. (1997). Resistance exercise, overtraining and overreaching. Neuroendocrine responses. *Sports Medicine*. 23, 106 – 129.

Fry, A.C., Kraemer, W.J., van Borselen, F., Lynch, J.M., Marsit, E.P., Roy, N., Triplett, T. and Knuttgen, G. (1994). Performance decrements with high-intensity resistance exercise overtraining. *Medicine and Science in Sports and Exercise*. 26, 1165 – 1173.

Fry, R.W., Morton, A.R. and Keast, D. (1992). Periodisation and the prevention of overtraining. *Canadian Journal of Applied Sport Science*. 17, 241 – 248.

Fry, R.W., Morton, A.R. and Keast, D. (1991). Overtraining in athletes. *Sports Medicine*. 12, 32 – 65.

Fry, R.W., Morton, A.R., Garcia-Webb, P., Crawford, G.P. and Keast, D. (1992). Biological responses to overload training in endurance sports. *European Journal of Applied Physiology*. 64, 335 – 344.

Gabbett, T.J. (2005). Changes in physiological and anthropometric characteristics of rugby league players during a competitive season. *Journal of Strength and Conditioning Research*. 19, 400 – 408.

Galen. Available at: <http://galenmedical.com/www/docs/8>. (Accessed on 30 July 2006).

Galloway, S.R. and Maughan, R.J. (1997). Effects of ambient temperature on the capacity to perform prolonged cycle exercise in man. *Medicine and Science in Sports and Exercise*. 29 (9), 1240 – 1249.

Gamble, P. (2004). Physical preparation for elite-level rugby union football. *National Strength and Conditioning Journal*. 26, 10 – 23.

Gamberale, F., Strindberg, L. and Wahlberg, I. (1975). Female work capacity during the menstrual cycle: physiological and psychological reactions. *Scandinavian Journal of Work and Environmental Health*. Jun; 1 (2), 120 – 127.

Gilman, M.B. (1996). The use of heart rate to monitor the intensity of endurance training. *Sports Medicine*. Feb; 21 (2), 73 – 79.

Gilman, M.B. and Wells, C.L. (1993). The use of heart rates to monitor exercise intensity in relation to metabolic variables. *International Journal of Sports Medicine*. 14 (6), 339 – 344.

Gisolfi, C. and Cohen, J. (1979). Relationships among training, heat acclimation and heat tolerance in men and women: the controversy revisited. *Medicine and Science in Sports*. 11 (1), 56 – 59.

Gleeson, M. (2002). Biochemical and Immunological markers of overtraining. *Journal of Sports Science and Medicine*. 1, 31 – 41.

Goedecke, J.H., St Clair Gibson, A., Grobler, L., Collins, M., Noakes, T.D. and Lambert, E.V. (2000). Determinants of the variability in respiratory exchange ratio at rest and during exercise in trained athletes. *American Journal of Physiology, Endocrinology and Metabolism*. Dec; 279 (6), E 1325 – 1334.

González-Alonso, J., Mora-Rodríguez, R., Below, P.R. and Coyle, E.F. (1997). Dehydration markedly impairs cardiovascular function in hyperthermic endurance athletes during exercise. *Journal of Applied Physiology*. 82, 1229 – 1236.

Goodman, J., Radomski, M., Hart, L., Plyley, M. and Shephard, R.J. (1989). Maximal aerobic exercise following prolonged sleep deprivation. *International Journal of Sports Medicine*. Dec; 10 (6), 419 – 423.

Green, H.J., Jones, L.L. and Painter, D.C. (1990). Effects of short-term training on cardiac function during prolonged exercise. *Medicine and Science in Sports and Exercise*. 22 (4), 488 – 493.

Greenland, K. (2004). Medical support for adventure racing. *Emergency Medicine Australasia*. Oct – Dec; 16 (5-6), 465 – 468.

Gyimes, Z., Pavlik, G. and Simor, T. (2004). Morphological and functional differences in cardiac parameters between power and endurance athletes: a magnetic resonance imaging study. *Acta Physiologica Hungarica*. 91 (1), 49 – 57.

Hainsworth, R. (1998). *Physiology of the cardiac autonomic system*. In: Clinical guide to cardiac autonomic tests. Malik, M. (editor). Kluwer Academic Publishers, Dordrecht.

Halberg, F., Vallbona, C. and Dietlin, L.F. (1970). Human circadian circulatory rhythms during weightlessness in extraterrestrial flight or bedrest with and without exercise. *Space Life Sciences*. 2, 18 – 32.

Halson, S.L., Bridge, M.W., Meeusen, R., Busschaert, B., Gleeson, M., Jones, D.A. and Jeukendrup, A.E. (2002). Time course of performance changes and fatigue markers during intensified training in trained cyclists. *Journal of Applied Physiology*. Sep; 93 (3), 947 – 956.

Halson, S.L., Lancaster, G.I., Jeukendrup, A.E. and Gleeson, M. (2003). Immunological responses to overreaching in cyclists. *Medicine and Science in Sports and Exercise*. May; 35 (5), 854 – 861.

Halson, S.L. and Jeukendrup, A.E. (2004). Does overtraining exist ? An analysis of overreaching and overtraining research. *Sports Medicine*. 34 (14), 967 – 981.

Hammond, H.K., White, F.C., Brunton, L.L. and Longhurst, J.C. (1987). Association of decreased myocardial  $\beta$ -receptors and chronotropic response to isoproterenol and exercise in pigs following chronic dynamic exercise. *Circulatory Research*. 60, 720 – 726.

Hammond, H.K., Ransnas, L.A. and Isnel, P.A. (1988). Noncoordinate regulation of cardiac Gs protein and  $\beta$ -adrenergic receptors by a physiological stimulus, chronic dynamic exercise. *Journal of Clinical Investigations*. 82, 2168 – 2171.

Hampson, D.B., St Clair Gibson, A., Lambert, M.I. and Noakes, T.D. (2001). The influence of sensory cues on the perception of exertion during exercise and central regulation of exercise performance. *Sports Medicine*. 31 (13), 935 – 952.

Hanna, M.I., Ilmarinen, J. and Yletyinen, I. (1982). Circadian variation of physiological functions in physically average and very fit dayworkers. *Journal of Human Ergology*. 11 Supp, 33 – 46.

Harre, D. (1973). *Trainingslehre*, Sportverlag, Berlin.

Hartmann, U. and Mester, J. (2000). Training and overtraining markers in selected sport events. *Medicine and Science in Sports and Exercise*. Jan; 32 (1), 209 – 215.

Hauber, C., Sharp, R.L. and Franke, W.D. (1997). Heart rate response to submaximal workloads during running and swimming. *International Journal of Sports Medicine*. 18, 347 – 353.

- Heaps, C.L., Gonzalez-Alonso, J. and Coyle, E.F. (1994). Hypohydration causes cardiovascular drift without reducing blood volume. *International Journal of Sports Medicine*. 15, 74 – 79.
- Hedelin, R., Wiklund, U., Bjerle, P. and Henriksson – Larsen, K. (2000). Cardiac autonomic imbalance in an overtrained athlete. *Medicine and Science in Sports and Exercise*. Sep; 32 (9), 1531 – 1533.
- Heitkamp, H.C., Schmid, K. and Scheib, K. (1993).  $\beta$ -Endorphin and adreno-corticotropic hormone production during marathon and incremental exercise. *European Journal of Applied Physiology*. 66, 269 – 274.
- Hellard, P., Avalos, M., Millet, G., Lacoste, L., Barale, F. and Chatard, J.C. (2005). Modeling the residual effects and threshold saturation of training: a case study of Olympic swimmers. *Journal of Strength and Conditioning Research*. Feb; 19 (1), 67 – 75.
- Helsen, W. and Bultynck, J.B. (2004). Physical and perceptual-cognitive demands of top-class refereeing in association football. *Journal of Sports Science*. Feb; 22 (2), 179 – 189.
- Hickson, R.C., Hagberg, J.M., Ehsani, A.A. and Holloszy, J.O. (1981). Time course of the adaptive responses of aerobic power and heart rate to training. *Medicine and Science in Sports and Exercise*. 13 (1), 17 – 20.
- Hill, A.V., Lupton, H. and Long, H.N.C. (1923). Muscular fatigue, lactate and oxygen supply and demand. *Proceedings of the Royal Society of London, Series B*. 97, 84 – 138.

Hills, A.P., Byrne, N.M. and Ramage, A.J. (1998). Submaximal markers of exercise intensity. *Journal of Sport Sciences*. 16, S 71-S 76

Holmyard, D.J. and Hazeldine, R.J. (1993). Seasonal variations in the anthropometric and physiological characteristics of international rugby union players. In: *Science and Football II: Proceedings of the Second World Congress of Science and Football*. Reilly, T., Clarys, A. and Stibbe, A. (editors). E and FN Son Publishers, London. Pg 21 – 26.

Hooper, S.L., MacKinnon, L.T., Howard, A., Gordon, R.D. and Bachmann, A.W. (1995). Markers for monitoring overtraining and recovery. *Medicine and Science in Sports and Exercise*. 27 (1), 106 – 112.

Hopkins, W.G. (1991). Quantification of training in competitive sports. *Sports Medicine*. 12 (3), 161 – 183.

Hopkins, W.G., Hawley, J.A. and Burke, L.M. (1999). Design and analysis of research on sport performance enhancement. *Medicine and Science in Sports and Exercise*. 31, 472 – 485.

Horne, J.A. (1981). The effects of exercise on sleep: A critical review. *Biological Psychiatry*. 12, 241 – 290.

Huang, P.H., Leu, H.B., Chen, J.W. and Lin, S.J. (2005). Heart rate recovery after exercise and endothelial function – two important factors to predict cardiovascular events. *Preventive Cardiology*. 8 (3), 167 – 170; Quiz 171.

- Hughson, R.L., Sutton, J.R., Fitzgerald, D.J. and Jones, N.L. (1977). Reduction of intrinsic sinoatrial frequency and norepinephrine response of the exercised rat. *Canadian Journal of Physiological Pharmacology*. 55, 813 – 820.
- Ilmarinen, J., Ilmarinen, R., Korhonen, O. and Nurminen, M. (1980). Circadian variation of physiological functions related to physical work capacity. *Scandinavian Journal of Work, Environment and Health*. 6, 112 – 122.
- Impellizzeri, F.M., Rampinini, E., Coutts, A.J., Sassi, A. and Marcora, S.M. (2004). Use of RPE-based training load in soccer. *Medicine and Science in Sports and Exercise*. Jun; 36 (6), 1042 – 1047.
- Israel, S. (1958). Die Erscheinungsformen des Übertrainings. *Sportmedizin*. 9, 207 – 209.
- Israel, S. (1967). Das akute Entlastungssyndrom des Leistungssportlers. *Sportmedizin*. 18 (H8), 185 – 190.
- Israel, S. (1976). Zur problematik des Übertrainings aus internistischer und leistungsphysiologischer sicht. *Medizin und Sport*. 16 (1), 1 – 12.
- Izquierdo, M., Hakkinen, K., Ibanez, J., Kraemer, W.J. and Gorostiaga, E.M. (2005). Effects of combined resistance and cardiovascular training on strength, power, muscle cross-sectional area, and endurance markers in middle-aged men. *European Journal of Applied Physiology*. May; 94 (1-2), 70 – 75.

- Izquierdo, M., Ibanez, J., Hakkinen, K., Kraemer, W.J., Ruesta, M. and Gorostiaga, E.M. (2004). Maximal strength and power, muscle mass, endurance and serum hormones in weightlifters and road cyclists. *Journal of Sports Science*. May; 22 (5), 465 – 478.
- Javorka, M., Zila, I., Balharek, T. and Javorka, K. (2002). Heart rate recovery after exercise: Relations to heart rate variability and complexity. *Brazilian Journal of Medical Biology Research*. Aug; 35 (8), 991 – 1000.
- Jeukendrup, A.E., Hesselink, M.K.C., Snyder, A.C., Kuipers, H. and Keizer, H.A. (1992). Physiological changes in male competitive cyclists after two weeks of intensified training. *International Journal of Sports Medicine*. Oct; 13 (7), 534 – 541.
- Jennings, C.L., Viljoen, W., Durandt, J. and Lambert, M.I. (2005). The reliability of the FitroDyne as a measure of muscle power. *Journal of Strength and Conditioning Research*. Nov; 19 (4), 859 – 863.
- Jouven, X., Empana, J.P., Schwartz, P.J., Desnos, M., Courbon, D. and Ducimetiere, P. (2005). Heart-rate profile during exercise as a predictor of sudden death. *New England Journal of Medicine*. May; 352 (19), 1951 – 1958.
- Jurkowski, J.E., Jones, N.L., Toews, C.J. and Sutton, J.R. (1981). Effects of menstrual cycle on blood lactate, O<sub>2</sub> delivery, and performance during exercise. *Journal of Applied Physiology*. Dec; 51 (6), 1493 – 1499.

Kaji, Y., Ariyoshi, K., Tsuda, Y., Kanaya, S., Fujino, T. and Kuwabara, H. (1989). Quantitative correlation between cardiovascular and plasma epinephrine response to mental stress.

*European Journal of Applied Physiology and Occupational Physiology*. 59, 221 – 226.

Kajiura, J.S., MacDougall, J.D., Ernst, P.B. and Younglai, E.V. (1995). Immune response to changes in training intensity and volume in runners. *Medicine and Science in Sport and Exercise*. 27 (8), 1111 – 1117.

Kang, J., Hoffman, J.R., Im, J., Spiering, B.A., Ratamess, N.A., Rundell, K.W., Nioka, S., Cooper, J. and Chance, B. (2005). Evaluation of physiological responses during recovery following three resistance exercise programs. *Journal of Strength and Conditioning Research*. May; 19 (2), 305 – 309.

Kannankeril, P.J., Le, F.K., Kadish, A.H. and Goldberger, J.J. (2004). Parasympathetic effects on heart rate recovery after exercise. *Journal of Investigative Medicine*. Sep; 52 (6), 394 – 401.

Karvonen, J. (1975). Follow-up of training of an endurance runner. *Stadion*. 6, 76 – 79.

Karvonen, J. and Vuorimaa, T. (1988). Heart rate and exercise intensity during sports activities. Practical application. *Sports Medicine*. May; 5, 303-312.

Katona, P.G., McLean, M., Dighton, D.H. and Guz, A. (1982). Sympathetic and parasympathetic cardiac control in athletes and nonathletes at rest. *American Physiological Society*. 1652 – 1657.

- Kenny, L. (1985). Parasympathetic control of resting HR: relationship to aerobic power. *Medicine and Science in Sports and Exercise*. 17, 451 – 455.
- Keohane, M. (2004). Boks must think of career longevity. *Business Day*. 21 September 2004. Available at [www.keo.co.za](http://www.keo.co.za). (Accessed on 10 March 2005).
- Khansari, D.N., Murgu, A.J. and Faith, R.E. (1990). Effects of stress on the immune system. *Immunology Today*. 11, 170 – 175.
- Kindermann, W., Simon, G. and Keul, J. (1979). The significance of the aerobic-anaerobic transition for the determination of work load intensities during endurance training. *European Journal of Applied Physiology and Occupational Physiology*. Sep; 42 (1), 25 – 34.
- Kingwell, B.A., Dart, A.M., Jennings, G.L. and Komer, P.I. (1992). Exercise training reduces the sympathetic component of the blood pressure-heart rate baroreflex in man. *Clinical Science*. 82, 357 – 362.
- Kirwan, J.P., Costill, D.L. and Flynn, M.G. (1988). Physiological response to successive days of intense training in competitive swimmers. *Medicine and Science in Sports and Exercise*. 20, 255 – 259.
- Kirwan, J.P., Costill, D.L., Houmard, J.A., Mitchell, J.B., Flynn, M.G. and Fink, W.J. (1990). Changes in selected blood measures during repeated days of intense training and carbohydrate control. *International Journal of Sports Medicine*. Oct; 11 (5), 362 – 366.

Koltyn, K.F. and Morgan, W.P. (1992). Efficacy of perceptual versus heart rate monitoring in the development of endurance. *British Journal of Sports Medicine*. Jun; 26 (2), 132 – 134.

Krustrup, P., Mohr, M. and Bangsbo, J. (2002). Activity profile and physiological demands of top-class soccer assistant refereeing in relation to training status. *Journal of Sports Science*. Nov; 20 (11), 861 – 871.

Kuipers, H. and Keizer, H.A. (1988). Overtraining in elite athletes, review and directions for the future. *Sports Medicine*. 6, 79 – 92.

Kuipers, H., Verstappen, F.T.J., Keizer, H.A., Geurten, P. and Van Kranenburg, G. (1985). Variability of aerobic performance in the laboratory and its physiologic correlates. *Sports Medicine*. 6, 197 – 201.

Lambert, M. (2006). Physiological testing for the athlete: Hype or Help? *International Journal of Sports Science and Coaching*. 1 (2), 199 – 208.

Lambert, M.I. and Keytel, L.R. (2000). Training habits of top runners in different age groups in a 56 km race. *South African Journal of Sports Medicine*. August, 17 – 32.

Lambert M.I., Mbambo, Z.H. and St. Clair Gibson, A. (1998). Heart rate during training and competition for long distance running. *Journal of Sports Science*. 16: S 85-S 90.

Lamberts, R.P., Lemmink, K.A.P.M., Durandt, J.J. and Lambert, M.I. (2004). Variation in heart rate during submaximal exercise: implications for monitoring training. *Journal of Strength and Conditioning Research*. 18 (3), 103 – 107.

Lamotte, M., Niset, G. and van de Borne, P. (2005). The effect of different intensity modalities of resistance training on beat-to-beat blood pressure in cardiac patients. *European Journal of Cardiovascular Prevention and Rehabilitation*. Feb; 12 (1), 12 – 17.

Laukkanen, R.M.T. and Virtanen, P.K. (1998). Heart rate monitors: State of art. *Journal of Sport Sciences*. 16, 143-151.

Lee, N. and Leroux, V. (editors). (1992). In: *Guide to Medicines and Drugs*. The Medical Association of South Africa. (1<sup>st</sup> SA edition). Dorling Kindersley Books. Reader's Digest Association, Inc. Cape Town.

Léger, L.A. and Lambert, J.A. (1982). A maximal multistage 20-m shuttle run test to predict  $\dot{V}O_2$ max. *European Journal of Applied Physiology and Occupational Physiology*. 49, 1 – 12.

Léger, L. and Thivierge, M. (1988). Heart rate monitors: validity, stability and functionality. *The Physician and Sportsmedicine*. 16 (5), 143 – 151.

Lehmann, M., Dickhuth, H.H., Gendrisch, G., Lazar, M., Thum, R., Kaminski, J.F., Aramendi, J.F., Peterke, E., Wieland, W. and Keul, J. (1991). Training – overtraining. A prospective experimental study with experienced middle – and long distance runners. *International Journal of Sports Medicine*. 12, 444 – 452.

Lehmann, M., Baumgartl, P., Wiesenack, C., Seidel, A., Baumann, H., Fischer, S., Spori, U., Gendrischi, G., Kamiński, R. and Keul, J. (1992). Training – overtraining: Influence of a defined increase in training volume vs. training intensity on performance, catecholamines and some metabolic parameters in experienced middle and long distance runners. *European Journal of Applied Physiology*. 64, 169 – 177.

Lehmann, M., Gastmann, U., Petersen, K.G., Bacht, N., Seidel, A., Khalaf, A.N., Fischer, S. and Keul, J. (1992). Training – overtraining: performance, and hormone levels, after a defined increase in training volume versus intensity in experienced middle- and long-distance runners. *British Journal of Sports Medicine*. 26, 233 – 242.

Lehmann, M., Foster, C. and Keul, J. (1993). Overtraining in endurance athletes: a brief review. *Medicine and Science in Sports and Exercise*. 25, 854 – 862.

Leicht, A.S., Himing, D.A. and Allen, G.D. (2003). Heart rate variability and endogenous sex hormones during the menstrual cycle in young woman. *Experimental Physiology*. May; 88 (3), 441 – 446.

Levy, M.N. and Martin, P.J. (1979). Neural control of the heart. In: Berne, R.M. (editor). *Handbook of Physiology*. Bethesda (MD): American Physiological Society. Pg 581 – 620.

Lewis, S.F., Nylander, E., Gad, E. and Areskog, N.H. (1980). Non-autonomic component in bradycardia of endurance trained men at rest and during exercise. *Acta Physiologica Scandinavica*. 109, 297 – 305.

Lin, Y-C. and Horvath, S.M. (1972). Autonomic nervous control of cardiac frequency in exercise trained rat. *Journal of Applied Physiology*. 33, 796 – 799.

Londeree, B.R., Thomas, T.R., Ziogas, G., Smith, T.D. and Zhang, Q. (1995). %  $VO_{2max}$  versus %  $HR_{max}$  regressions for six modes of exercise. *Medicine and Science in Sports and Exercise*. 27 (3), 458 – 461.

MacFarlane, D.J., Fogarty, B.A. and Hopkins, W.G. (1989). The accuracy and variability of commercially available heart rate monitors. *New Zealand Journal of Sports Medicine*. 17 (4), 51 – 53.

MacIntyre, J.G. (1987). Growth hormone and athletes. *Sports Medicine*. 4, 129 – 140.

Mackinnon, L.T. (1998). Effects of overreaching and overtraining on immune function. In: *Overtraining in Sport*. Kreider, R.B., Fry, A.C. and O'Toole, M.L. (editors). Human Kinetics. Champaign, IL. pg 219 – 241.

Madden, K.M., Levy, W.C. and Stratton, J.K. (2006). Exercise training and heart rate variability in older adult female subjects. *Clinical Investigative Medicine*. Feb; 29 (1), 20 – 28.

Malpas, S.C. (2002). Neural influences on cardiovascular variability: possibilities and pitfalls. *American Journal of Physiology and Heart Circulatory Physiology*. 282, H 6 – H 20.

Marieb, E.N. (1992). *Human Anatomy and Physiology*. (2<sup>nd</sup> edition). Benjamin / Cummings Publishing Company, INC. Pg 626 – 628.

- Marino, F.E. and Booth, J. (2001). Cardiovascular responses to self-paced running in warm humid conditions following whole body precooling. *Sports Medicine*. March; 3 – 8.
- Martin, B.J. (1981). Effects of sleep deprivation on tolerance of prolonged exercise. *European Journal of Applied Physiology and Occupational Physiology*. 47 (4), 345 – 354.
- Martin, W. H. III., Montgomery, J., Snell, P.G., Corbett, J.R., Sokolov, J.J., Buckey, J.C., Maloney, D.A. and Blomqvist, C.G. (1987). Cardiovascular adaptations to intense swim training in sedentary middle-aged men and women. *Circulation*. 75, 323 – 330.
- Mashiko, T., Umeda, T., Nakaji, S. and Sugawara, K. (2004). Position related analysis of the appearance of and relationship between post-match physical and mental fatigue in university rugby football players. *British Journal of Sports Medicine*. 38, 617 – 621.
- Mbambo, Z.H. and Lambert, M.I. (2000). Variations in the heart rate of an elite long distance runner during races of varying distances. *South African Journal of Sports Medicine*. April; (7), 17 – 20.
- Meredith, C.N., Frontera, W.R., Fisher, E.C., Hughes, V.A., Herland, J.C., Edwards, J. and Evans, W.J. (1989). Peripheral effects of endurance training in young and old subjects. *Journal of Applied Physiology*. 66, 2844 – 2849.
- McCloskey, D.I. and Mitchell, J.H. (1972). Reflex cardiovascular and respiratory responses originating from exercising muscle. *Journal of Physiology (Lond)*. 224, 173 – 186.

McNair, D.M. (1971). *Profile of Mood States Manual. Educational and Industrial Testing Service*. San Diego.

Meeusen, R., Duclos, M., Gleeson, M., Rietjens, G.J., Steinacker, J.M. and Urhausen, A. (2006). Prevention, diagnosis and treatment of the overtraining syndrome. *Journal of Sports Science*. 6, 1 – 14.

Meeuson, R., Piacentini, M.F., Busschaert, B., Buyse, L., de Schutter, G. and Stray-Gundersen, J. (2004). Hormonal responses in athletes: the use of a two bout exercise protocol to detect subtle differences in (over) training status. *European Journal of Applied Physiology*. Mar; 91 (2-3), 140 – 146.

Mier, C., Turner, M.J., Ehsani, A.A. and Spina, R.J. (1997). Cardiovascular adaptations to 10 days of exercise. *Canadian Journal of Applied Physiology*. 22, Suppl: 52P.

Millet, G.P., Groslambert, A., Barbier, B., Rouillon, J.D. and Candau, R.B. (2005). Modelling the relationship between training, anxiety and fatigue in elite athletes. *International Journal of Sports Medicine*. Jul – Aug; 26 (6), 492 – 498.

Milliken, M.C., Stray-Gundersen, J., Peshock, R.M., Katz, J. and Mitchell, J.H. (1988). Left ventricular mass as determined by magnetic resonance imaging in male endurance athletes. *American Journal of Cardiology*. Aug 1; 62 (4), 301 – 305.

Montain, S.J. and Coyle, E.F. (1992). The influence of graded dehydration on hyperthermia and cardiovascular drift during exercise. *Journal of Applied Physiology*. 73, 903 – 910.

- Morgan, W.P., Brown, D.R., Raglin, J.S., O'Connor, P.J. and Ellickson, K.A. (1987). Psychological monitoring of overtraining and staleness. *British Journal of Sports Medicine*. 21, 107 – 114
- Morgan, W.P. and Borg, G. (1976). Perception of effort in the prescription of physical activity. In: *Humanistic and Mental Aspects of Sports, Exercise and Recreation*. Craig, T.T. (editor). American Medical Association, Chicago. Pg 26 – 129.
- Morgan, W.P., Costill, D.L., Flymnn, M.G., Raglin, P.J., and O'Connor, P.J. (1988). Mood disturbance following increased training in swimmers. *Medicine and Science in Sports and Exercise*. 20, 408 – 414.
- Mujika, I. (1998). The influence of training characteristics and tapering on the adaptation in highly trained individuals: a review. *International Journal of Sports Medicine*. Oct; 19 (7), 439 – 446.
- Mujika, I., Chatard, J.C. and Geysant, A. (1996 a). Effects of training and taper on blood leucocyte populations in competitive swimmers: relationships with cortisol and performance. *International Journal of Sports Medicine*. Apr; 17 (3), 213 – 217.
- Mujika, I., Chatard, J.C., Padilla, S., Guezennec, C.Y. and Geysant, A. (1996 b). Hormonal responses to training and its tapering off in competitive swimmers: relationships with performance. *European Journal of Applied Physiology and Occupational Physiology*. 74 (4), 361 – 366.

- Neumayr, G., Pfister, R., Mitterbauer, G., Gaenzer, H., Sturm, W. and Hoertnagl, H. (2003). Heart rate response to ultraendurance cycling. *British Journal of Sports Medicine*. Feb; 37 (1), 89 – 90.
- Nielsen, B., Hales, J.R.S., Strange, S., Christensen, N.J., Warberg, J. and Saltin, B. (1993). Human circulatory and thermoregulatory adaptations with heat acclimation and exercise in a hot, dry environment. *Journal of Physiology*. 460, 467 – 485.
- Noakes, T.D. (1988). Implications of exercise testing for prediction of athletic performance: a contemporary perspective. *Medicine and Science in Sports and Exercise*. 20, 319 – 330.
- Noakes, T.D. (1997). 1996 J.B. Wolfe Memorial Lecture. Challenging beliefs: ex Africa simper aliquid novi. *Medicine and Science in Sports and Exercise*. Jan; 32 (1), 85 – 88.
- Noakes, T.D. (1998). Maximal oxygen uptake: 'classical' versus 'contemporary' viewpoints: a rebuttal. *Medicine and Science in Sports and Exercise*. 30, 1381 – 1398.
- Noakes, T.D. (2006). Comment on point: "In health and in a normoxic environment,  $\text{VO}_2\text{max}$  is / is not limited primarily by cardiac output and locomotor muscle blood flow. *Journal of Applied Physiology*. May; 100 (5), 1742 – 1743.
- Noakes, T.D. and du Plessis, M. (1996). Rugby without risk. J.L. van Schaik Publishers, Pretoria.

Noakes, T.D., Peltonen, J.E. and Rusko, H.K. (2001). Evidence that the central governor regulates exercise performance during acute hypoxia and hyperoxia. *The Journal of Experimental Biology*. 204, 3225 – 3234.

O'Connor, P.J., Crowley, M.A., Gardner, A.W. and Skinner, J.S. (1993). Influence of training on sleeping heart rate following daytime exercise. *European Journal of Applied Physiology*. 67, 39 – 42.

O'Toole, M.L., Douglas, P.S. and Hiller, W.D. (1998). Use of heart rate monitors by endurance athletes: lessons from triathletes. *Journal of Sports Medicine and Physical Fitness*. Sep; 38 (3), 181 – 187.

Paccotti, P., Minetto, M., Terzolo, M., Ventura, M., Ganzit, G.P., Borrione, P., Termine, A. and Angeli, A. (2005). Effects of high-intensity isokinetic exercise on salivary cortisol in athletes with different training schedules: relationships to serum cortisol and lactate. *International Journal of Sports Medicine*. Nov; 26 (9), 747 – 755.

Palmer, G., Hawley, J.A., Dennis, S. and Noakes, T.D. (1994). Heart rate response during a 4 day cycle race. *Medicine and Science in Sports and Exercise*. 26, 1278 – 1283.

Parekh, A. and Lee, C.M. (2005). Heart rate variability after isocaloric exercise bouts of different intensities. *Medicine and Science in Sports and Exercise*. Apr; 37 (4), 599 – 605.

Park, R.J. (1992). Athletes and their training in Britain and America, 1800 – 1914. In: *Sport and Exercise Science*. Berryman, J. W. and Park, R.J. (editors). University of Illinois Press, Urbana, IL. Pg 57 – 107.

Parker, S., Brukner, P. and Rosier, M. (1996). Chronic fatigue syndrome and the athlete. *Sports Medicine, Training and Rehabilitation*. 6, 269 – 278.

Parry-Billings, M., Budgett, R., Koutedakis, Y., Blomstrand, E., Brooks, S., Williams, C., Calder, P.C., Pilling, S., Baigre, R. and Newsholme, E.A. (1992). Plasma amino acid concentrations in the overtraining syndrome: Possible effects on the immune system. *Medicine and Science in Sports and Exercise*. 24, 1353 – 1358.

Paton, C.D. and Hopkins, W.G. (1999). Performance Enhancement at the fifth IOC World Congress on Sport Sciences. (on-line) *Sportscience* 3(3) News and Comment: Conference Report. Available at <http://sportsci.org/jour/9903/cdpwghIOC.html> (Accessed on 14 March 2006).

Pelayo, P., Mujika, I., Sidney, M. and Chatard, J-C. (1996). Blood lactate recovery measurements, training, and performance during a 23-week period of competitive swimming. *European Journal of Applied Physiology*. 74, 107 – 113.

Pendleton, M.H.W. (1997). Reliability and validity of the Welsh Rugby Union shuttle run test. Unpublished BSc dissertation, University of Wales Institute, Cardiff.

Péronnet, F., Blier, P., Brisson, G., Diamond, P., Ledoux, M. and Volle, M. (1986).

Reproducibility of plasma catecholamine concentrations at rest and during exercise in man.

*European Journal of Applied Physiology*. 54, 555 – 558.

Perrault, H. and Turcotte, R.L. (1994). Exercise-induced cardiac hypertrophy: fact or fallacy?

*Sports Medicine*. 17, 288 – 308.

Petrofsky, J.S., LeDonne, D.M., Rinehart, J.S. and Lind, A.R. (1976). Isometric strength and

endurance during the menstrual cycle. *European Journal of Applied Physiology and*

*Occupational Physiology*. Mar 9; 35 (1), 1 - 10.

Pichot, V., Busso, T., Roche, F., Garet, M., Costes, F., Duverney, D., Lacour, J.R. and

Barthelemy, J.C. (2002). Autonomic adaptations to intensive and overload training periods: a laboratory study. *Medicine and Science in Sports and Exercise*. Oct; 34 (10), 1660 – 1666.

Pickering, T. G. (1988). The influence of daily activity on ambulatory blood pressure. *American Heart Journal*. 116, 1141 – 1145.

Pigozzi, F., Alabiso, A., Parisi, A., Di Salvo, V., Di Luigi, L., Spataro, A. and Lellamo, F. (2001).

Effects of aerobic exercise training on 24 hr profile of heart rate variability in female athletes.

*Journal of Sports Medicine and Physical Fitness*. Mar; 41 (1), 101 – 107.

Pierpont, G.L. and Voth, E.J. (2004). Assessing autonomic function by analysis of heart rate

recovery from exercise in healthy subjects. *American Journal of Cardiology*. Jul 1; 94 (1), 64 –

68.

Pierpont, G.L., Stolpmann, D.R. and Gornick, C.C. (2000). Heart rate recovery post - exercise as an index of parasympathetic activity. *Journal of the Autonomic Nervous System*. May 12; 80 (3), 169 – 174.

Platen P., Wöstmann, R., Schulz, H., Hartmann, U., Bartmus, U., Grabow, V. and Heck, H. (2000). Effects of high training loads on urinary catecholamine excretion and nightly heart rates. *Deutsche Zeitschrift für Sportmedizin*. 51 (9). (Abstract – Unpublished data).

Pollock, M.L. (1973). The quantification of endurance training programs. *Exercise and Sports Science Reviews*. 1,155- 188.

Pollock, M.L. and Wilmore, J.H. (1990). *Exercise in Health and Disease*. (2 nd edition). W.B. Sanders Co, Philadelphia. Pg 319 – 355.

Portier, H., Louisy, F., Laude, D., Berthelot, M. and Guezennec, C.Y. (2001). Intense endurance training on heart rate and blood pressure variability in runners. *Medicine and Science in Sports and Exercise*. Jul; 33 (7), 1120 – 1125.

Princi, T., Parco, S., Accardo, A., Radillo, O., De Seta, F. and Guaschino, S. (2005). Parametric evaluation of heart rate variability during the menstrual cycle in young women. *Biomedical and Scientific Instruments*. 41, 340 – 345.

Pyne, D.B., Lee, H. and Swanwick, K.M. (2001). Monitoring the lactate threshold in world-ranked swimmers. *Medicine and Science in Sports and Exercise*. Feb; 33 (2), 291 – 297.

Raglin, J.S., Morgan, W.P. and O'Connor, P.J. (1991). Changes in mood states during training in female and male college swimmers. *International Journal of Sports Medicine*. 12, 585 – 589.

Raven, P.B. and Stevens, G.H.J. (1988). Cardiovascular function and prolonged exercise. In: *Perspectives in Exercise Science and Sports Medicine*. Lamb, D.R. and Murray, R. (editors) Prolonged exercise. Benchmark Press, Indianapolis. 1, 43 – 74.

Reilly, T. (1990). Human circadian rhythms and exercise. *Critical Review of Biomedical Engineering*. 18, 165 – 180.

Reilly, T. and Borrie, A. (1992). Physiology applied to field hockey. *Sports Medicine*. 14, 10 – 26.

Reilly, T. and Brooks, G.A. (1982). Investigation of circadian rhythms in metabolic responses to exercise. *Ergonomics*. 25, 1093 – 1097.

Reilly, T. and Gilbourne, D. (2003). Science and football: a review of applied research in the football codes. *Journal of Sports Science*. 21, 693 – 705.

Reilly, T., Robinson, G. and Minors, D.S. (1984). Some circulatory responses to exercise at different times of day. *Medicine and Science in Sports and Exercise*. 16 (5), 477 – 482.

Rietjens, G.J., Kuipers, H., Adam, J.J., Saris, W.H., van Breda, E., van Hamont, D. and Keizer, H.A. (2005). Physiological, biochemical and psychological markers of strenuous training-induced fatigue. *International Journal of Sports Medicine*. Jan – Feb; 26 (1), 16 – 26.

Rietjens, G.J., Kuipers, H., Hartgens, F. and Keizer, H.A. (2002). Red blood cell profile of elite Olympic distance triathletes. A three-year follow-up. *International Journal of Sports Medicine*. Aug; 23 (6), 391 – 396.

Robertson, R.J. (1982). Central signals of perceived exertion during dynamic exercise. *Medicine and Science in Sports and Exercise*. 14, 390 – 396.

Robson, P.J., Blannin, A.K., Walsh, N.P., Castell, L.M. and Gleeson, M. (1999). Effects of exercise intensity, duration and recovery on *in vitro* neutrophil function in male athletes. *International Journal of Sports Medicine*. 20, 128 – 135.

Roecker, K., Niess, A.M., Horstmann, T., Striegel, H., Mayer, F. and Dickhuth, H.H. (2002). Heart rate prescriptions from performance and anthropometrical characteristics. *Medicine and Science in Sports and Exercise*. May; 34 (5), 881 – 887.

Ross, W.D. and Marfell-Jones, M.J. (1991). Kinanthropometry. In: *Physiological Testing of the High Performance Athlete*, (2<sup>nd</sup> edition) McDougal, J.D., Wenger, H.A. and Green, H.J. (editors). Human Kinetics, Champaign, Illinois, USA. Pg 223 – 308.

Ross & Wilson. (1990). *Anatomy and Physiology in Health and Illness*. Wilson, K.J.W (editor). (7<sup>th</sup> edition). Churchill Livingstone, Edinburgh, London, Melbourne and New York.

Rotstein, A., Falk, B., Einbinder, M. and Zigel, L. (1998). Changes in plasma volume following intense intermittent exercise in neutral and hot environmental conditions. *Journal of Sports Medicine and Physical Fitness*. Mar; 38 (1), 24 – 29.

Roussel, B. and Buguet, A. (1982). Changes in human heart rate during sleep following daily physical exercise. *European Journal of Applied Physiology*. 49 (3), 409 – 416.

Rowbottom, D.G., Keast, D., Goodman, C. and Morton, A.R. (1995). The haematological, biochemical and immunological profile of athletes suffering from the overtraining syndrome. *European Journal of Applied Physiology and Occupational Physiology*. 70, 502 – 509.

Sadamoto, T., Fuchi, T., Taniguchi, Y. and Miyashita, M. (1986). Effect of 8 weeks submaximal conditioning and deconditioning on heart rate during sleep in middle-aged women. In: *Sport and Ageing*. McPherson B.D. (editor). Human Kinetics, Champaign, IL. Pg 233 – 240.

Savin, W.M., Davidson, D.M. and Haskell, W.L. (1982). Autonomic contribution to heart rate recovery from exercise in humans. *Journal of Applied Physiology*. Dec; 53 (6), 1572 – 1575.

Sawka, M.N., Knowlton, R.G. and Critz, J.B. (1979). Thermal and circulatory responses to repeated bouts of prolonged running. *Medicine and Science in Sports*. 11 (2), 112 – 116.

Scatchard, G., Batchelder, A. and Brown, A. (1944). Chemical, clinical and immunological studies on the products of human plasma fractionalization: IV. The osmotic pressure of plasma and of serum albumin. *Journal of Clinical Investigations*. 23, 458 – 464.

Schabert, E.J., Hopkins, W.G. and Hawley, J.A. (1998). Reproducibility of self-paced treadmill performance of trained endurance runners. *International Journal of Sports Medicine*. 18, 1 – 4.

Schulz, H., Hartmann, U., Platen, P., Grabow, V., Wostmann, R., Niessen, M., Bartmus, U. and Heck, H. (2000). Sleeping heart rate is little influenced by training load. *Deutsche Zeitschrift für Sportmedizin*. 51(9). (Abstract - Unpublished data).

Schumacher, Y.O., Schmid, A., Grathwohl, D., Bultermann, D. and Berg, A. (2002). Hematological indices and iron status in athletes of various sports and performances. *Medicine and Science in Sports and Exercise*. May; 34 (5), 869 – 875.

Scott, J.P. and McNaughton, L.R. (2004). Sleep deprivation, energy expenditure and cardiorespiratory function. *International Journal of Sports Medicine*. Aug; 25 (6), 421 – 426.

Seals, D.R. and Chase, P.G. (1989). Influence of physical training on heart variability and baroreflex circulatory control. *Journal of Applied Physiology*. 66, 1886 – 1895.

Seaward, B.L., Sleamaker, R.H., McAuliffe, T. and Clapp, J.F. 3 rd. (1990). The precision and accuracy of a portable heart rate monitor. *Biomedical Instrument Technology*. Jan – Feb; 24 (1), 37 – 41.

Selby, G.B. and Eichner, R.E. (1994). Hematocrit and performance: the effect of endurance training on blood volume. *Seminars in Hematology*. Apr; 31 (2), 122 – 127.

Sedgwick, A.W., Craig, R.J., Crouch, R. and Dowling, B. (1974). The effects of physical training on the day and night long-term heart rates of middle-aged men. *European Journal of Applied Physiology*. 33, 307 – 314.

Selley, E.A., Kolbe, T., van Zyl, C.G., Noakes, T.D. and Lambert, M.I. (1995). Running intensity as determined by heart rate is the same in fast and slow runners in both the 10- and 21-km races. *Journal of Sports Sciences*. 13, 405 – 410.

Semenick, D.M. (1994). Testing protocols and procedures. In: Baechle, T.R. (Ed). *Essentials of Strength Training and Conditioning*. Human Kinetics, Champaign, IL. Pg 258 – 273.

Seshadri, N., Gildea, T.R., McCarthy, K., Pothier, C., Kavuru, M.S. and Lauer, M.S. (2004). Association of an abnormal exercise heart rate recovery with pulmonary function abnormalities. *Chest*. Apr; 125 (4), 1186 – 1190.

Shaffrath, J.D. and Adams, W.C. (1984). Effects of airflow and work load on cardiovascular drift and skin blood flow. *Journal of Applied Physiology*. 56 (6), 1411 – 1417.

Shephard, R.J., Verde, T.J., Thomas, S.G. and Shek, P. (1991). Physical activity and the immune system. *Canadian Journal of Sports Science*. 16, 163 – 185.

Shetler, K., Marcus, R., Froelicher, V.F., Vora, S., Kalisetti, D., Prakash, M., Do, D. and Myers, J. (2001). Heart rate recovery: Validation and methodological issues. *Journal of the American College of Cardiology*. 38 (7), 1980 – 1987.

- Shi, X., Stevens, G.H.J., Foresman, B.H., Stern, S.A. and Raven, P.B. (1995). Autonomic nervous system control of the heart: endurance exercise training. *Medicine and Science in Sports and Exercise*. 27, 1406 – 1413.
- Smith, L.L. (2004). Tissue trauma: The underlying cause of overtraining syndrome? *Journal of Strength and Conditioning Research*. 18 (1), 185 – 193.
- Smith, M.L., Hudson, D.L., Graitzer, H.M. and Raven, P.G. (1989). Exercise training bradycardia: The role of autonomic balance. *Medicine and Science in Sports and Exercise*. 21, 40 – 44.
- Smolensky, M.H., Tatar, S.E., Bergman, S.A., Losman, J.G., Barnard, C.N., Dasco, C.C. and Kraft, I.A. (1976). Circadian rhythmic aspects of human cardiovascular function. A review by chronobiologic statistical methods. *Chronobiologia*. 3, 337 – 371.
- Snyder, A.C. and Foster, C. (1994). Physiology and nutrition for skating. In: *Perspectives in Exercise and Sports Medicine. Physiology and Nutrition of Competitive Sports*. Lamb, D.R., Knuttgen, H.G. and Murray, R. (editors). Cooper Publishing Group, Carmel, Indiana. (7), Pg 181 – 219.
- Snyder, F., Hobson, J.A., Morrison, D.F. and Goldfrank, F. (1964). Changes in respiration, heart rate and systolic blood pressure in human sleep. *Journal of Applied Physiology*. 19, 417 – 422.

Spina, R.J., Ogawa, T., Kohrt, W.M., Martin, W.H. 3<sup>rd</sup>., Holloszy, J.O. and Ehsani, A.A. (1993). Differences in cardiovascular adaptations to endurance exercise training between older men and women. *Journal of Applied Physiology*. 75, 849 – 855.

St Clair Gibson, A., Broomhead, S.A., Hawley, J.A., Lambert, M.I. and Noakes, T.D. (1998). Is heart rate during field testing reliable ? A study of repeatability of heart rate in different sporting populations. *Journal of Sports Science*. 16, S 109.

Stein, R., Moraes, R.S., Cavalcanti, A.V., Ferlin, E.L., Zimmerman, L.I. and Ribeiro, J.P. (2000). Atrial automaticity and atrioventricular conduction in athletes: contribution of autonomic regulation. *European Journal of Applied Physiology*. 82 (1 – 2), 155 – 157.

Stein, R., Medeiros, C.M., Rosito, G.A., Zimmerman, L.I. and Ribeiro, J.P. (2002). Intrinsic sinus and atrioventricular node electrophysiologic adaptations in endurance athletes. *Journal of American College Cardiology*. 39 (6), 1033 – 1038.

Steinacker, J.M., Brkic, M., Simsch, C., Nething, K., Kresz, A., Prokopchuk, O. and Liu, Y. (2005). Thyroid hormones, cytokines, physical training and metabolic control. *Hormonal and Metabolic Research*. Sep; 37 (9), 538 – 544.

Steinacker, J.M., Lormes, W., Reissnecker, S. and Liu, Y. (2004). New aspects of the hormone and cytokine response to training. *European Journal of Applied Physiology*. Apr; 91 (4), 382 – 391.

Stephenson, L.A., Kolka, M.A. and Wilkerson, J.E. (1982). Perceived exertion and anaerobic threshold during the menstrual cycle. *Medicine and Science in Sports and Exercise*. 14 (3), 218 – 222.

Stone, M.H., Fleck, S.J., Triplett, N.T. and Kraemer, W.J. (1991). Health- and performance-related potential of resistance training. *Sports Medicine*. 11, 210 – 231.

Sunstedt, M., Hedberg, P., Jonason, T., Ringqvist, I., Brodin, L.A. and Henriksen, E. (2004). Left ventricular volumes during exercise in endurance athletes assessed by contrast echocardiography. *Acta Physiologica Scandinavica*. Sept; 182 (1), 45 – 51.

Sutton, L. and Lazarus, L. (1976). Growth hormone in exercise: comparison of physiological and pharmacological stimuli. *Journal of Applied Physiology*. 41, 523 – 527.

Swart, J. and Jennings, C.L. (2004). Use of blood lactate concentration as a marker of training status. *South African Journal of Sports Medicine*. 16 (3), 3 – 7.

Sweet, T.W., Foster, C., McGuigan, M.R. and Brice, G. (2004). Quantitation of resistance training using the session rating of perceived exertion method. *Journal of Strength and Conditioning Research*. Nov; 18 (4), 796 – 802.

Tipton, C.M. (1997). Sports medicine: a century of progress. *Journal of Nutrition*. May; 127 (5 Suppl), 878 S – 885 S.

Tiukinhoy, S., Beohar, N. and Hsie, M. (2003). Improvement in heart rate recovery after cardiac rehabilitation. *Journal of Cardiopulmonary Rehabilitation*. 23 (2), 84 – 87.

Tokudome, S., Kuriki, K., Yamada, N., Ichikawa, H., Miyata, M., Shibata, K., Hoshino, H., Tsuge, S., Tokudome, M., Goto, C., Tokudome, Y., Kobayashi, M., Goto, H., Suzuki, S., Okamoto, Y., Ikeda, M. and Sato, Y. (2004). Anthropometric, lifestyle and biomarker assessment of Japanese non-professional ultra-marathon runners. *Journal of Epidemiology*. Sep; 14 (5), 161 – 167.

Townes, D.A. (2005). Wilderness medicine: strategies for provision of medical support for adventure racing. *Sports Medicine*. 35 (7), 557 – 564.

Tremblay, M.S., Copeland, J.L. and Van Helder, W. (2004). Effect of training status and exercise mode on endogenous steroid hormones in men. *Journal of Applied Physiology*. Feb; 96 (2), 531 – 539.

Tremblay, M.S., Copeland, J.L. and Van Helder, W. (2005). Influence of exercise duration on post-exercise steroid hormone responses in trained males. *European Journal of Applied Physiology*. Aug; 94 (5-6), 505 – 513.

Urhausen, A., Gabriel, H.H. and Kindermann, W. (1998). Impaired pituitary hormonal response to exhaustive exercise in overtrained endurance athletes. *Medicine and Science in Sports and Exercise*. Mar; 30 (3), 407 – 414.

Urhausen, A., Gabriel, H. and Kindermann, W. (1995). Blood hormones as markers of training stress and overtraining. *Sports Medicine*. Oct; 20 (4), 251 – 276.

Urhausen, A. and Kindermann, W. (1994). Monitoring of training by determination of hormone concentration in the blood – review and perspectives. In: Liesen, H., Weiß, M. Baum, M. (editors). *Regulations – und Repairmechanismen*, Deutscher Ärzte- Verlag, Köln. Pg 551 – 554.

Urhausen, A. and Kindermann, W. (2002). Diagnosis of overtraining: what tools do we have ? *Sports Medicine*. 32 (2), 95 – 102.

Uusitalo, A.L.T., Uusitalo, A.J. and Rusko, H.K. (1998). Exhaustive Endurance Training for 6 – 9 Weeks did not induce changes in Intrinsic Heart Rate and Cardiac Autonomic Modulation in Female Athletes. *International Journal of Sports Medicine*. 19, 532 – 540.

Vander, A.J., Sherman, J.H. and Luciano, D.S. (1975). In; *Human Physiology*. The mechanisms of body function. (2<sup>nd</sup> edition). Thomas, A.P., First, C. (editors). McGraw-Hill, Inc, New York.

Vella, C.A. and Robergs, R.A. (2005). Non-linear relationships between central cardiovascular variables and  $\text{VO}_2$  during incremental cycling exercise in endurance-trained individuals. *Journal of Sports Medicine and Physical Fitness*. Dec; 45 (4), 452 – 459.

Verde, T., Thomas, S. and Shephard, R.J. (1992). Potential markers of heavy training in highly trained distance runners. *British Journal of Sports Medicine*. 26 (3), 167 – 173.

- Verges, S., Flore, P. and Favre-Juvin, A. (2003). Blood lactate concentration / heart rate relationship: laboratory running test vs. field roller skiing test. *International Journal of Sports Medicine*. Aug; 24 (6), 446 – 451.
- Vincent, W.J. (1995). *Statistics in Kinesiology*. Champaign, IL, Human Kinetics. Pg 178 – 181.
- Vincent, K.R., Vincent, H.K., Braith, R.W., Bhatnagar, V. and Lowenthal, D.T. (2003). Strength training and hemodynamic responses to exercise. *American Journal of Geriatric Cardiology*. Mar – Apr; 12 (2), 97 – 106.
- Wahlberg, I. and Åstrand, I. (1973). Physical work capacity during the day and at night. *Work and Environmental Health*. 10, 65 – 68.
- Walker, J.M., Floyd, T.C., Fein, G., Cavness, C., Lualhati, R. and Feinberg, I. (1978). Effects of exercise on sleep. *Journal of Applied Physiology*. 44, 945 – 951.
- Weiler, B., Urhausen, A., Coen, B. et al. (1994). Changes of catecholamine and lactate during training and competition in badminton. . In: Liesen, H., Weiß, M. Baum, M. (editors). *Regulations – und Repairmechanismen*, Deutscher Ärzte- Verlag, Köln. Pg 636 – 638.
- Weltman, A., Seip, R.L., Snead, D., Weltman, J.Y., Haskvitz, E.M., Evans, W.S., Veldhuis, J.D. and Rogol, A.D. (1992). Exercise training at and above the lactate threshold in previously untrained woman. *International Journal of Sports Medicine*. 13, 257 – 263.

- Werle, E.Q., Strobel, G. and Weicker, H. (1990). Decrease in cardiac  $\beta_1$  and  $\beta_2$  adrenoreceptors by training and endurance exercise. *Life Sciences*. 46, 9 – 17.
- Weston, A.R., Khan, A. and Mars, M. (2001). Does heart rate adequately reflect exercise intensity during mini-trampoline exercise? *South African Journal of Sports Medicine*. March; 8 (1), 9 – 14.
- Wever, R. (1979). Influence of physical workload on free running circadian rhythms of man. *Pflügers Archiv: European Journal of Physiology*. 38, 119 – 126.
- Wilkins, H.A., Petersen, S.R. and Quinney, H.A. (1991). Time-motion analysis of and heart rate responses to amateur ice hockey officiating. *Canadian Journal of Sport Science*. Dec; 16 (4), 302 – 307.
- Wilmore, J.H., Stanforth, P.R., Gagnon, J., Leon, A.S., Rao, D.C., Skinner, J.S. and Bouchard, C. (1996). Endurance exercise training has a minimal effect on resting heart rate: The Heritage Study. *Medicine and Science in Sport and Exercise*. 28, 829-835.
- Wilmore, J.H. and Costill, D.L. (1994). (authors) *Physiology of Sport and Exercise*. Human Kinetics, Champaign, Illinois, USA.
- Winget, C.M., DeRoshia, C.W. and Holley, D.C. (1985). Circadian rhythms and athletic performance. *Medicine and Science in Sports*. 17 (5), 498 – 516.

Wingo, J.E., Lafrenz, A.J., Ganio, M.S., Edwards, G.L. and Cureton, K.J. (2005).

Cardiovascular drift is related to reduced maximal oxygen uptake during heat stress. *Medicine and Science in Sports and Exercise*. Feb; 37 (2), 248 – 255.

Winsley, R.J., Battersby, G.L. and Cockle, H.C. (2005). Heart rate variability assessment of overreaching in active and sedentary females. *International Journal of Sports Medicine*. Nov; 26 (9), 768 – 773.

Yawn, B.P., Ammar, K.A., Thomas, R. and Wollan, P.C. (2003). Test – retest reproducibility of heart rate recovery after treadmill exercise. *Annals of Family Medicine*. Nov – Dec; 1 (4), 236 – 241.

Zavorsky, G. S. (2000). Evidence and possible mechanisms of altered maximum heart rate with endurance training and tapering. *Sports Medicine*. Jan; 29 (1), 13 – 26.

# APPENDICES

University of Cape Town

## APPENDIX A

**GENERAL FATIGUE / MUSCLE SORENESS**

1. DO YOU FEEL THAT YOU ARE GENERALLY FATIGUED DURING NORMAL DAILY ACTIVITIES IN THE PREVIOUS 24 HOURS ?

2. HAVE YOU EXPERIENCED ANY MUSCLE SORENESS IN THE LOWER EXTREMITIES IN THE PREVIOUS 24 HOURS ?

IF YES – PLEASE RATE:

0 = NOTHING AT ALL

6 = MODERATE - SEVERE

1 = VERY VERY SLIGHT

7 = SEVERE

2 = VERY SLIGHT

8 = VERY SEVERE

3 = SLIGHT

9 = VERY VERY SEVERE

4 = MILD

10 = MAXIMAL PAIN

5 = MODERATE

## APPENDIX B

**SESSION RATING OF PERCEIVED EXERTION**

0	REST
1	REALLY EASY
2	EASY
3	MODERATE
4	SORT OF HARD HARD
5	HARD
6	
7	REALLY HARD
8	
9	REALLY, REALLY HARD
10	JUST LIKE MY HARDEST RACE

## APPENDIX C

**BORG RATING OF PERCEIVED EXERTION**

6	NO EXERTION AT ALL
7	EXTREMELY LIGHT
8	VERY LIGHT
9	
10	LIGHT
11	
12	
13	SOMEWHAT HARD
14	
15	HARD
16	
17	VERY HARD
18	EXTREMELY HARD
19	
20	MAXIMAL EXERTION

## APPENDIX D

### Training protocol

The training days were on Mondays, Tuesdays, Thursdays, and Fridays for three weeks. There were two types of training programs. The first program was a circuit type program with a number of different exercises which was conducted on Monday and Thursday. The second program was a shuttle program which mostly involved running shuttles. This program was conducted on Tuesdays and Fridays. After every week the workload of each the training sessions was increased

### Circuit program (Monday and Thursday)

This was all conducted after a warm-up and a stretching routine. For the circuit program the subjects were required to perform all the exercises listed below. The subjects were required to exercise for 30 seconds and have a 20 seconds rest period between each station. They had to exercise for the whole 30 seconds doing as many correct repetitions as possible.

### Circuit program for week 1:

1. Accumulative 5 metre shuttles
2. Ladder drill: Quick step along a ladder
3. 20 metre shuttles
4. Cycling on stationary bicycle: Males – level 10, Females – level 7
5. Bridge on ball with alternating leg raises
6. Skipping
7. Alternate arm and leg raise on ball
8. Lateral step ups
9. Push ups: Male – normal push ups, Females – ladies push ups
10. Stepping
11. Cycling on stationary bicycle: Males – level 10, Females – level 7
12. Chest press with dumbbells: Males – 10 kg weights, Females – 5 kg weights
13. Seated bicep curls: Males – 12 kg weights, Females – 6 kg weights
14. Shoulder press: Males – 10 kg weights, Females – 5 kg weights
15. Lunges

16. Squat: Males – Olympic bar with 10kg, Females – Olympic bar only
17. Bench press: Males – Olympic bar with 10kg, Females – 15 kg barbell

### **Shuttle Program (Tuesday and Friday)**

This was all conducted after a warm up and a stretching routine. The shuttle program involved a small circuit and a number of shuttle activities. For the circuit the subjects were required to exercise for 30 seconds with a rest period of 20 seconds between each exercise. For the 20 metre shuttles the subjects had to complete 10, 20 metre shuttles: Males had to complete the circuit in 50 seconds, and females in 58 seconds. For the 5 metre shuttles the subjects had to complete the accumulative 5 metre shuttles: Males had to complete the circuit in 30 seconds and females in 35 seconds.

### **Shuttle program for week 1**

1. Stepping
2. Reverse leg raises
3. Skipping
4. 10 x 20 metre shuttles
5. Accumulative 5 metre shuttles
6. 10 x 20 metre shuttles
7. Accumulative 5 metre shuttles
8. 10 x 20 metre shuttles

### **Circuit program for week 2**

1. 5 metre shuttles
2. Ladder drill: Quick step along a ladder
3. 20 metre shuttles
4. Oblique ball sit ups
5. 5 metre shuttles
6. Karaoke ladder drill
7. 20 metre shuttles
8. Cycling on stationary bicycle: Males – level 10, Females – level 7
9. Bridge on ball with alternating leg raises

10. Skipping
11. Alternate arm and leg raise on ball
12. Lateral step ups
13. Push ups: Male – normal push ups, Females – ladies push ups
14. Stepping
15. Cycling on stationary bicycle: Males – level 10, Females – level 7
16. Chest press with dumbbells: Males – 10 kg weights, Females – 5 kg weights
17. Seated bicep curls: Males – 12 kg weights, Females – 6 kg weights
18. Shoulder press: Males – 10 kg weights, Females – 5 kg weights
19. Lunges
20. Squat: Males – Olympic bar with 10kg, Females – Olympic bar only
21. Bench press: Males – Olympic bar with 10kg, Females – 15 kg barbell

### **Shuttle program for week 2**

1. Stepping
2. Reverse leg raises
3. Skipping
4. Stepping
5. Foot Changes
6. Skipping
7. 10 x 20 metre shuttles
8. Accumulative 5 metre shuttles:
9. 10 x 20 metre shuttles
10. Accumulative 5 metre shuttles
11. 10 x 20 metre shuttles
12. Accumulative 5 metre shuttles
13. 10 x 20 metre shuttles

### **Circuit program for week 3**

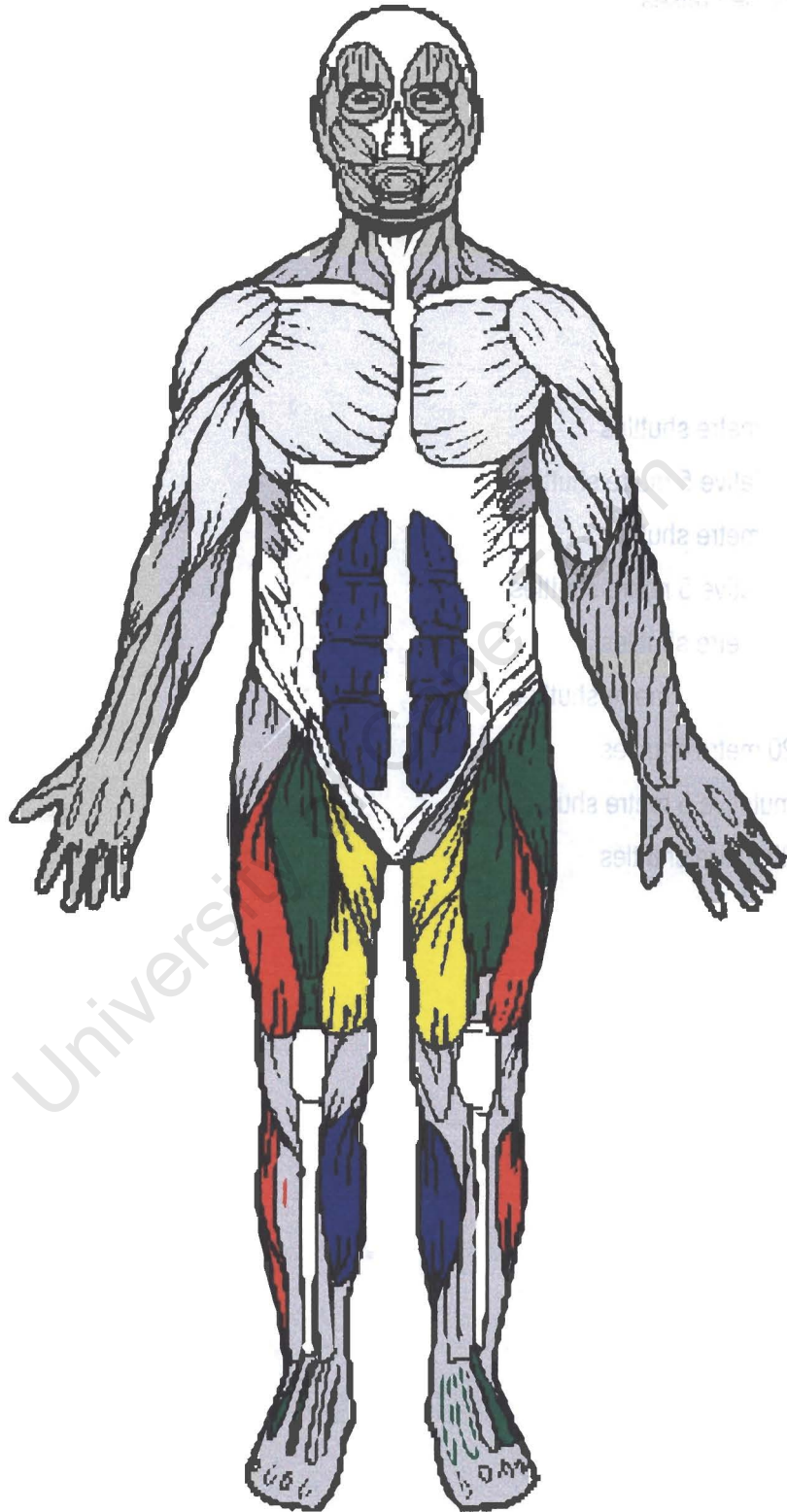
The subjects had to complete the same circuit they had done during the first week of training, now they have to complete the circuit twice.

**Shuttle program for week 3**

1. Stepping
2. Reverse leg raises
3. Skipping
4. Stepping
5. Foot Changes
6. Skipping
7. Stepping
8. Squats
9. Stepping
10. 10 x 20 metre shuttles
11. Accumulative 5 metre shuttles
12. 10 x 20 metre shuttles
13. Accumulative 5 metre shuttles
14. 10 x 20 metre shuttles
15. Accumulative 5 metre shuttles
16. 10 x 20 metre shuttles
17. Accumulative 5 metre shuttles
18. 10 x 20 metre shuttles

University of Cape Town

APPENDIX E MUSCLE SORENESS RATING – ANTERIOR VIEW



## APPENDIX E MUSCLE SORENESS RATING – POSTERIOR VIEW

