

THE EFFECT OF GRADUATED COMPRESSION SOCKS ON CALF MUSCLE OXYGENATION OF ENDURANCE ATHLETES

by
Lara Grobler

*Thesis presented in fulfilment of the requirements for the degree of
Master of Sport Science
in the Department of Sport Science, Faculty of Education
at
Stellenbosch University*



Supervisor: E. Terblanche
Co-supervisor: K.E. Welman

December 2012

DECLARATION

By submitting this thesis electronically, I declare that the entirety of the work contained therein is my own, original work, that I am the owner of the copyright thereof (unless to the extent explicitly otherwise stated) and that I have not previously in its entirety or in part submitted it for obtaining any qualification.

Signature: Lara Grobler

Date: December 2012

Copyright © 2012 Stellenbosch University

All rights reserved

SUMMARY

Compression socks (CS) are used as an ergogenic aid during and after exercise by many athletes of elite and recreational status. The exact mechanism whereby CS affect performance and post-exercise recovery is not yet elucidated. Some research ascribes the beneficial effects to improved lactate removal rates with CS. One hypothesis is that CS improve venous return and thereby remove the lactate from the tissue to other tissues such as the liver, and the second hypothesis is that the CS cause retention of the lactate within the muscle and therefore improve the oxidation of the lactate within the muscle (Berry & McMurray, 1987).

The current study endeavoured to test the hypothesis set by Berry and McMurray (1987) by measuring the effect of CS as well as flight socks (FS) on muscle oxygenation during exercise and recovery in endurance trained runners and triathletes.

Eleven male endurance trained runners and triathletes (age = 34.8 ± 3.8 years, $VO_{2max} = 52.4 \pm 7.1$ mL.kg⁻¹.min⁻¹) participated in the study. They completed an incremental exercise test to exhaustion to determine their maximal aerobic capacity (VO_{2max}) and peak treadmill velocity (PTV). Then they completed two 10 km treadmill running tests at 80 % of their PTV. During these two trials participants wore either CS or FS; the order of treatment was randomly selected. A subset of the study sample (n = 5) also completed a control test wearing only their ankle length sport socks (NS). After these trials, participants completed a 60 minute passive recovery period in the seated position while muscle oxygenation was measured.

Compression under the socks was measured at several anatomically determined measurement points prior to the commencement of the exercise test, along with the determination of blood haemoglobin concentration ([Hb]). During the exercise trials, blood lactate concentration ([BLa]), skin temperature (ST), oxygen consumption (VO_2), carbon dioxide production (VCO_2), heart rate (HR), and muscle oxygenation variables (oxy-haemoglobin (O_2Hb), deoxy-haemoglobin (HHb), tissue oxygenation index (TOI) and total haemoglobin index (nTHI)) was measured. During the 60

minute passive recovery period, [BLa], ST, O₂Hb, HHb, TOI, and nTHI measurements were continued.

The results showed that there were differences in the pressure exerted between the two pressure condition (CS and FS) at the posterior ankle, and under the elastic of the sock as well as on the anterior calf at the level of greatest calf circumference. Differences in ST between the CS and NS and the FS and NS conditions were found between the first four 2 km intervals of the exercise protocol, but not during recovery.

No differences were found in [BLa] between the three different compression conditions during either the exercise ($p = 0.19$) or recovery period ($p = 0.63$), as well as no differences in the cardiorespiratory variables during exercise between the three different compression conditions (VO₂, $p = 0.06$; VCO₂, $p = 0.12$; HR, $p = 0.36$). With regard to the muscle oxygenation variables, no differences were found between the three compression conditions during exercise, however there was a trend for lower oxygen utilization (HHb) during exercise in the NS condition ($p = 0.57$, medium to large practical significance). There were also no differences in these variables (O₂Hb, $p = 0.65$; HHb, $p = 0.57$; TOI, $p = 0.39$; nTHI, $p = 0.22$) during recovery, although oxygen utilization (HHb) showed a faster recovery rate with increasing external pressure.

From the results obtained, it seems that external compression caused a decrease in the blood flow velocity within the muscle, thereby increasing oxygen diffusion rate. During exercise this did not facilitate differences in [BLa], however, after the first 10 minutes of the recovery period, large practical differences were found between the NS and both sock conditions, suggesting that the increase in oxygen diffusion improved lactate clearance. This could support the hypothesis set by Berry and McMurray (1987).

OPSOMMING

Kompressie sokkies (CS) word gereeld deur beide rekreasie- en elite atlete gebruik as 'n ergogeniese hulpmiddel tydens oefening en herstel. Die presiese meganisme waardeur CS prestasie en post-oefening herstel beïnvloed is nog nie volledig verklaar nie. Sommige navorsing skryf die voordelige effekte toe aan die vinniger herstel van laktaat in die sirkulasie. Daar is tans twee hipoteses vir die meganisme waardeur CS laktaat verwydering verbeter. Die eerste hipotese is dat CS die veneuse terugvoer verbeter en daardeur die laktaat van die weefsel verwyder en na ander weefsels soos die lewer vervoer vir verwydering. Die tweede hipotese is dat CS veroorsaak dat die laktaat in die spierweefsel teruggehou word wat dan tot gevolg het dat die laktaat in die spier self deur middel van oksidasie verwyder word (Berry & McMurray, 1987).

Hierdie studie poog om Berry en McMurray (1987) se hipotese te toets deur die effek wat CS sowel as vlugsokkies (FS) op spieroksigenasie het gedurende oefening en herstel in geoefende uithouvermoë hardlopers en driekamp atlete vas te stel.

Elf ingeefende langafstand hardlopers en driekampatlete (mans) (ouderdom = 34.8 ± 3.8 jaar; $VO_{2maks} = 52.4 \pm 7.1$ mL.kg⁻¹.min⁻¹) het aan hierdie studie deel geneem. Die deelnemers het 'n inkrementele toets tot die punt van uitputting voltooi om hul maksimale aërobiese kapasiteit (VO_{2maks}) en piek trapmeul snelheid (PTV) vas te stel. Die elf deelnemers het ook twee 10 km hardlooptoetse teen 80 % van hul PTV voltooi. Gedurende hierdie twee toetse het die deelnemers óf CS óf FS gedra; die volgorde van die intervensie was lukraak aan hulle toegeken. 'n Subgroep van die steekproef (n = 5) het ook 'n kontrole toets voltooi waartydens hulle hul eie enkelhoogte sport sokkies (NS) gedra het. Aan die einde van die hardloop protokol het die deelnemers 'n 60 minuut passiewe herstel periode in die sittende posisie voltooi terwyl spieroksigenasie gemeet is.

Kompressie onder die sokkies is voor die aanvang van die hardloop protokol by verskeie anatomies gedefinieerde punte gemeet. Verder was die bloed hemoglobien konsentrasie ([Hb]) ook gemeet voor die hardloop protokol. Tydens die oefeningtoets is bloedlaktaat konsentrasie

([Hb]), veltemperatuur (ST), suurstof verbruik (VO_2), koolstofdoksied produksie (VCO_2), harttempo (HR), sowel as spieroksigenasie veranderlikes (oksi-hemoglobien (O_2Hb), deoksi-hemoglobien (HHb), weefsel oksigenasie indeks (TOI), en totale hemoglobien indeks (nTHI)) gemeet. Gedurende die 60 minuut passiewe hersteltydperk is [BLa], ST, O_2Hb , HHb, TOI en nTHI metings geneem.

Die resultate toon dat daar 'n verskil is in die druk wat uitgeoefen word in die onderskeie druktoestande (CS en FS) op die been by die posterior enkel en onder die rek van die sokkie, sowel as op die anterior kuit waar die kuit die grootste omtrek het. Verdere verskille tussen die CS en NS en die FS en NS toestande is in ST gevind in the eerste vier 2 km intervalle van die oefeningtoets, alhoewel geen verskille tydens die herstelperiode gevind is nie.

Tydens beide die oefening ($p = 0.19$) en herstel ($p = 0.63$) protokol is geen verskille tussen die drie kompressie toestande met betrekking tot [BLa] gevind nie. En so ook is daar geen verskille tussen die onderskeie kompressie toestande in kardiorespiratoriese veranderlikes (VO_2 , $p = 0.06$; VCO_2 , $p = 0.12$; HR, $p = 0.36$) tydens oefening gevind nie. Met betrekking tot spieroksigenasie veranderlikes was daar geen verskil gevind tussen die drie kompressietoestande gedurende oefening nie, alhoewel daar 'n tendens was vir die NS toestand om tydens oefening minder suurstofverbruik (HHb) ($p = 0.57$, medium tot groot praktiese effek) te lewer. So ook gedurende herstel is daar geen verskil in hierdie veranderlikes (O_2Hb , $p = 0.65$; HHb, $p = 0.57$; TOI, $p = 0.39$; nTHI, $p = 0.22$) gevind nie, alhoewel die suurstofverbruik (HHb) vinniger na die basislyn herstel het met 'n toename in druk.

Die resultate toon dat eksterne kompressie 'n afname in die bloedvloei tempo in die spier veroorsaak wat dan 'n verlengde suurstof diffusie tyd veroorsaak. Hierdie verlengde suurstof diffusie tyd het geen effek op [BLa] tydens oefening gehad nie, alhoewel daar na die eerste 10 minute van die herstelperiode 'n groot praktiese verskil tussen die NS en sokkie toestande gevind was in [BLa]. Hierdie verskil kan daarop dui dat die toename in suurstof diffusie verbeterde laktat verwydering tot gevolg het, wat dan die hipotese van Berry en McMurray (1987) ondersteun.

ACKNOWLEDGMENTS

I would like to thank the following people for helping me in my road to completing this study.

Prof Elmarie Terblanche for all the advice and support with this study. I could not have done it without your guidance.

Dr Karen Welman for all the speculation and nonsense talk sessions.

Mom and Dad for supporting me through all the years of studying. You have always been my greatest supporters and have pushed me to achieve great things.

Zac Mouton for all the support and love especially when things were tough.

Louise Engelbrecht, Pieter Boer, Jana de Villiers and Roy Langheim for all the early mornings and late evenings.

Bradley Fryer and Kurt Schütte, we did it boys.

Hester van Schalkwyk for motivation and technical corrections.

My roommate Arina Cronje for not complaining when I made a racket when having to get up early in the morning.

All glory to God for giving me the abilities and opportunity to endeavour on this road of discovery.

DEDICATION

I dedicate this thesis to my late grandmother Zeta Burger.

I wish this nail had made the wall.

LIST OF ABBREVIATIONS AND ACRONYMS

%	:	Percentage
~	:	About
ε	:	Extinction coefficient
π	:	Pi
μA	:	Microampere
μL	:	Microlitre
μmol	:	Micromole
>	:	Greater than
<	:	Smaller than
\geq	:	Greater or equal to
\leq	:	Smaller or equal to
\pm	:	Plus minus
$^{\circ}$:	Degrees
Δ	:	Delta
%BF	:	Percentage body fat
$^{\circ}\text{C}$:	Degrees Celsius
A	:	Attenuation
A	:	Area

[AU]	:	arbitrary unit concentration
[BLa]	:	Blood lactate concentration
[C]	:	Micromolar concentration
[Hb]	:	Haemoglobin concentration
<i>a</i>	:	Pre-exercise rest period
ANOVA	:	Analysis of variance
ATP	:	Adenosine triphosphate
AU	:	Arbitrary units
a-vO ₂ difference	:	Arterial oxygen extraction
B	:	Point of minimum girth on the ankle
<i>b</i>	:	Average of last 2 km of the running protocol
B1	:	Area at which the Achilles tendon changes into the calf muscle
b.min ⁻¹	:	Beats per minute
BIA	:	Bio-electrical impedance analysis
BMI	:	Body mass index
C	:	Point of maximum calf girth
<i>c</i>	:	Circumference of the limb at a specific point
CG	:	Compression garments
CO	:	Cardiac output
cm	:	Centimetre

CS	:	Compression socks
D	:	Level just below the tibial tuberosity
DOMS	:	Delayed onset muscle soreness
E	:	Centre of the patella and over the back of the knee
eNOS	:	Endothelial nitric oxide synthase
ES	:	Effect size
F	:	Force
F	:	Mid-thigh between the patella and groin
FS	:	Flight socks
G	:	5cm below the center point of the crotch
GCS	:	Graduated compression socks
g.dL ⁻¹	:	Grams per decilitre
h	:	Hours
<i>h</i>	:	Half the increment in muscle oxygenation during recovery
H	:	point of greatest lateral trochanteric projection of the buttocks
Hb	:	Haemoglobin
HHb	:	Deoxy-haemoglobin
HR	:	Heart rate
Hz	:	Hertz
I	:	One

II	:	Two
III	:	Three
<i>I</i>	:	Light out
I_0	:	Light in
ICG	:	Indocyanine green
IV	:	Intravenous
IV	:	Four
K	:	Centre point of the crotch
<i>K</i>	:	Tissue loss
kg	:	Kilogram
kHz	:	KiloHertz
km	:	Kilometre
km.h ⁻¹	:	Kilometre per hour
<i>L</i>	:	Inner dimension of the cuvette
L	:	Litre
L	:	Large practical effect
LIST	:	Loughborough intermittent shuttle test
LSD	:	Least significant difference
LT	:	Lactate threshold
LTV	:	Treadmill velocity at lactate threshold

L-NAME	:	Nitric oxide synthase inhibitor
M	:	Medium practical effect
m	:	Meter
Mb	:	Myoglobin
min	:	Minute
mL	:	Millilitres
mL.b ⁻¹	:	Millilitres per beat
mL.kg ⁻¹ .min ⁻¹	:	Millilitres per kilogram per minute
mL.min ⁻¹	:	Millilitres per minute
mM	:	Millimole
mm	:	Millimetres
mmHg	:	Millimetres of Mercury
mmol.L ⁻¹	:	Millimole per litre
MOE	:	Muscle oxygenation economy
mOxy	:	Mean muscle oxygenation
mRNA	:	Messenger ribonucleic acid
N	:	Newton
n	:	Sample size
NIR	:	Near-infrared
NIRS	:	Near-infrared spectroscopy

nm	:	Nanometre
NO	:	Nitric oxide
NOS	:	Nitric oxide synthase
NS	:	No sock control condition
NSSD	:	No statistically significant difference
nTHI	:	Total haemoglobin index
O ₂	:	Oxygen
O ₂ Hb	:	Oxy-haemoglobin
O ₂ Mb	:	Oxy-myoglobin
P	:	Pressure
p	:	Probability
Pa	:	Pascal
PO	:	Power output
P-MRS	:	Phosphorus magnetic resonance spectroscopy
PTV	:	Peak treadmill velocity
<i>r</i>	:	Radius
<i>r</i>	:	Correlation statistic
R	:	Respiratory exchange ratio
ROM	:	Range of motion
RPE	:	Rating of perceived exertion

R_r	:	Recovery rate
S	:	Small practical effect
s	:	Seconds
SD	:	Standard deviation
SF _{Post}	:	Posterior calf skinfold
SI	:	International System of Units
SRS	:	Spatially resolved spectroscopy
ST	:	Skin temperature
StO ₂	:	Tissue oxygen saturation
SV	:	Stroke volume
T	:	Wall tension
T_R	:	Time taken to reach h
TOI	:	Tissue oxygenation index
Type I	:	Slow oxidative muscle fibres
Type IIa	:	Fast oxidative muscle fibres
Type IIb	:	Fast glycolytic muscle fibres
UV	:	Ultraviolet
VCO ₂	:	Carbon dioxide production
VL	:	Very large practical effect
VO ₂	:	Oxygen consumption

VO_{2max}	:	Maximal aerobic capacity
VT	:	Ventilatory threshold
W	:	Watts
$W \cdot \text{min}^{-1}$:	Watts per minute
$W \cdot \%^{-1}$:	Watts per percentage
x	:	Pathlength factor
x	:	End of exercise
x	:	Mean
y	:	First peak post-exercise
z	:	End of the 60 min recovery period

CONTENT

p.

CHAPTER ONE: INTRODUCTION.....	1
CHAPTER TWO	4
MUSCLE OXYGENATION	4
A. INTRODUCTION.....	4
B. NEAR-INFRARED SPECTROSCOPY.....	5
1. Wavelengths	6
2. Measurement depth.....	7
C. VARIABLES MEASURED	7
1. Validity and reliability	8
D. BLOOD FLOW	9
E. CALCULATION OF DIFFERENT VARIABLES	10
1. Beer-Lambert law	10
2. Spatially resolved spectroscopy	11
3. Other calculations.....	12
G. ADVANTAGES OF NIRS	13
H. FACTORS INFLUENCING NIRS MEASUREMENTS.....	14
1. Skin blood flow	14
2. Adipose tissue thickness	15

3. Other factors	16
I. SKELETAL MUSCLE OXYGENATION	17
1. Capillary blood flow	18
2. Muscle fibre type	19
J. CAUSES OF DESATURATION.....	20
K. MUSCLE OXYGENATION DURING EXERCISE.....	21
1. Incremental exercise	21
2. Continuous sub-maximal exercise.....	23
3. High intensity exercise.....	23
4. Eccentric exercise	24
L. MUSCLE OXYGENATION DURING RECOVERY AFTER EXERCISE	24
M. CONCLUSION	25
CHAPTER THREE: COMPRESSION GARMENTS.....	26
A. DEFINING COMPRESSION GARMENTS.....	26
B. THE APPLICATION OF COMPRESSION GARMENTS	26
C. CLAIMS.....	27
D. COMPRESSION	28
1. Measurement of compression	28
2. Measurement sites	31
3. Graduated compression socks	32
4. Elastic tights	35
E. EFFECT OF COMPRESSION SOCKS DURING EXERCISE	35

1. Physiological effects	35
Cardiovascular function	36
Venous blood flow	39
Blood lactate concentration.....	40
Muscle oxygenation	44
2. Performance effects	47
Endurance performance	47
Maximal oxygen uptake.....	48
Economy of movement	49
Anaerobic threshold	52
Power	52
Sprint performance	53
3. Post-exercise recovery	54
Metabolic end product removal	54
Delayed onset of muscle soreness	54
Muscle oscillations.....	57
4. Perceptions of fatigue.....	58
Fatigue	58
Thermoregulation.....	59
Proprioception	61
F. CONCLUSION.....	61

CHAPTER FOUR: PROBLEM STATEMENT	62
A. SUMMARY OF THE LITERATURE	62
B. RESEARCH LIMITATIONS	63
C. AIMS OF THE CURRENT STUDY	63
CHAPTER FIVE: METHODOLOGY	64
A. STUDY DESIGN.....	64
B. PARTICIPANTS	64
1. Subject selection	64
2. Ethical aspects	65
3. Environmental aspects	65
C. EXPERIMENTAL DESIGN	65
1. Testing sessions.....	66
First session	66
Sessions two to four	66
D. MEASUREMENTS AND TESTS	67
1. Anthropometrical measurements.....	67
Height	67
Body mass.....	67
Percentage body fat.....	68
Lower leg girths	68
Posterior calf skinfold.....	69
2. Maximal aerobic capacity exercise test	69

3. Sock pressure profile.....	70
4. Exercise protocol.....	71
Simulated running protocol	71
Muscle oxygenation	72
Heart rate and Oxygen consumption.....	75
Skin temperature	75
Blood lactate concentration.....	75
Haemoglobin concentration	75
5. Recovery protocol	76
E. STATISTICAL ANALYSIS	76
CHAPTER SIX: RESULTS	79
A. DESCRIPTIVE STATISTICS	79
1. Participants	79
2. Baseline measurements before the simulated run test	81
Haemoglobin	81
Pressure	81
Pressures at greatest circumference of calf.....	83
Pressure ranging from proximal to distal	84
B. COMPARISON IN VARIABLES DURING EXERCISE	85
1. Skin temperature (ST)	86
2. Metabolic data	87
3. Blood lactate concentration	90

4. Muscle oxygenation: O ₂ Hb	92
5. Muscle oxygenation: HHb.....	94
6. Muscle oxygenation: TOI.....	96
7. Muscle oxygenation: nTHI	98
8. Muscle oxygenation: Change from rest to end of exercise.....	100
C. COMPARISON IN VARIABLES DURING RECOVERY	101
1. Skin temperature (ST)	102
2. Blood lactate concentration	103
3. Muscle oxygenation: O ₂ Hb	105
4. Muscle oxygenation: HHb.....	107
5. Muscle oxygenation: TOI.....	109
6. Muscle oxygenation: nTHI	111
7. Muscle oxygenation: Delta calculations post-exercise	113
Delta 2 calculations.....	113
Delta 3 calculations.....	115
Delta 4 calculations.....	117
D. CORRELATIONS	120
CHAPTER SEVEN: DISCUSSION	122
A. INTRODUCTION	122
B. DESCRIPTIVE CHARACTERISTICS	123
1. Haemoglobin	124
C. PRESSURE PROFILES	125

D. THE EFFECT OF COMPRESSION ON MUSCLE PHYSIOLOGY DURING EXERCISE	128
1. Skin temperature	128
2. Metabolic changes	130
3. Blood lactate concentration	132
4. Muscle oxygenation.....	132
E. THE EFFECT OF COMPRESSION ON MUSCLE PHYSIOLOGY DURING POST-EXERCISE RECOVERY.....	137
1. Skin temperature	137
2. Blood lactate concentration	138
3. Muscle oxygenation.....	138
F. CONCLUSION.....	143
1. The effect of CS during exercise	143
2. The effect of CS on post-exercise recovery	145
3. Strengths of the current research	147
G. STUDY LIMITATIONS AND FUTURE RESEARCH	148
REFERENCES	150
APPENDIX A.....	171
APPENDIX B.....	174
APPENDIX C.....	175

LIST OF FIGURES

p.

<i>Figure 2.1.</i>	Factors affecting muscle oxygenation	17
<i>Figure 3.1.</i>	The difference in heart rate (HR) between trained and untrained individuals during exercise (adapted from McArdle, Katch and Katch, 2010).	37
<i>Figure 3.2.</i>	The difference in the stroke volume (SV) of a trained and untrained individual during exercise (adapted from Rowell, 1974 in Powers & Howley, 2007).	37
<i>Figure 3.3.</i>	Factors affecting running economy (with permission from Saunders <i>et al.</i> , 2004).....	51
<i>Figure 5.1.</i>	Classification of anatomical points used for pressure and circumference measurements (<i>Photo by L. Grobler</i>).	71
<i>Figure 5.2.</i>	A representation of the simulated 10 km running protocol, where each column represents a 2 km interval.	72
<i>Figure 5.3.</i>	NIRS probe and temperature sensor placement, with (two photos on left) and without (photo on right) IV plaster. (<i>Photo by L. Grobler</i>).....	73
<i>Figure 5.4.</i>	Simulated running protocol (<i>Photo by L.A. de Villiers</i>).....	74
<i>Figure 5.5.</i>	Graphical explanation of delta calculations shown on raw data as displayed by the NIRO 200NX (Hamamatsu, Japan) software.	78
<i>Figure 6.1.</i>	The number of training sessions per week done by the participants.....	80
<i>Figure 6.2.</i>	Log transformed pressure (P) measurements (AU) at the greatest circumference of the calf.	83

<i>Figure 6.3.</i>	Log transformed pressure measurements (AU) on the posterior calf ($p < 0.05$). Pressure measurements coincide with the indicated level on the calf in the photo (<i>Photo by L. Grobler</i>).....	85
<i>Figure 6.4.</i>	Average oxygen consumption ($\text{mL}\cdot\text{min}^{-1}$) over 2 km intervals during the 10 km running protocol.	88
<i>Figure 6.5.</i>	Average carbon dioxide production ($\text{mL}\cdot\text{min}^{-1}$) over 2 km intervals during the 10 km running protocol.....	89
<i>Figure 6.6.</i>	Average heart rate ($\text{b}\cdot\text{min}^{-1}$) over 2 km intervals during the 10 km running protocol.	90
<i>Figure 6.7.</i>	Log transformed blood lactate concentrations ([BLa]) during the 10 km running protocol.	91
<i>Figure 6.8.</i>	Changes in oxy-haemoglobin (O_2Hb) in the left lateral Gastrocnemius during the 10 km running protocol.....	93
<i>Figure 6.9.</i>	Changes in oxy-haemoglobin (O_2Hb) in the right lateral Gastrocnemius during the 10 km running protocol.....	93
<i>Figure 6.10.</i>	Changes in deoxy-haemoglobin (HHb) in the left lateral Gastrocnemius during the 10 km running protocol.....	95
<i>Figure 6.11.</i>	Changes in deoxy-haemoglobin (HHb) in the right lateral Gastrocnemius during the 10 km running protocol.	95
<i>Figure 6.12.</i>	Changes in tissue oxygenation index (TOI) in the left lateral Gastrocnemius during the 10 km running protocol.	97
<i>Figure 6.13.</i>	Changes in tissue oxygenation index (TOI) in the right lateral Gastrocnemius during the 10 km running protocol.	97

<i>Figure 6.14.</i>	Changes in total haemoglobin index (nTHI) in the left lateral Gastrocnemius during the 10 km running protocol.	99
<i>Figure 6.15.</i>	Changes in total haemoglobin (nTHI) in the right lateral Gastrocnemius during the 10 km running protocol.	99
<i>Figure 6.16.</i>	Blood lactate concentration ([BLa]) (AU) during recovery.	104
<i>Figure 6.17.</i>	Changes in oxy-haemoglobin (O ₂ Hb) during the 60 min recovery period in the left lateral Gastrocnemius.	106
<i>Figure 6.18.</i>	Changes in oxy-haemoglobin (O ₂ Hb) during the 60 min recovery period in the right lateral Gastrocnemius.	106
<i>Figure 6.19.</i>	Change in deoxy-haemoglobin (HHb) during the 60 min recovery period in the left lateral Gastrocnemius.	108
<i>Figure 6.20.</i>	Change in deoxy-haemoglobin (HHb) during the 60 min recovery period in the right lateral Gastrocnemius.	108
<i>Figure 6.21.</i>	Changes in tissue oxygenation index (TOI) during the 60 min recovery period in the left lateral Gastrocnemius.	110
<i>Figure 6.22.</i>	Changes in tissue oxygenation index (TOI) during the 60 min recovery period in the right lateral Gastrocnemius.	110
<i>Figure 6.23.</i>	Changes in total haemoglobin index (nTHI) during the 60 min recovery period in the left lateral Gastrocnemius.	112
<i>Figure 6.24.</i>	Changes in total haemoglobin index (nTHI) during the 60 min recovery period in the right lateral Gastrocnemius.	112
<i>Figure 6.25.</i>	Changes in O ₂ Hb as determined by the Delta 2 calculations.	114
<i>Figure 6.26.</i>	Changes in HHb as determined by the Delta 2 calculations.	114

<i>Figure 6.27.</i>	Changes in O ₂ Hb as determined by Delta 3 calculations.....	116
<i>Figure 6.28.</i>	Changes in HHb as determined by Delta 3 calculations.	116
<i>Figure 6.29.</i>	Changes in O ₂ Hb as determined by Delta 4 calculations.....	118
<i>Figure 6.30.</i>	Changes in HHb as determined by Delta 4 calculations.	119
<i>Figure 7.1.</i>	Proposed model to describe the effect of CS on muscle oxygenation during exercise.....	144
<i>Figure 7.2.</i>	Proposed model to describe the effect of CS on muscle oxygenation during recovery.	146

LIST OF TABLES

p.

<i>Table 3.1.</i>	Specifications of a pressure sensor (Partsch <i>et al.</i> , 2006).....	29
<i>Table 3.2.</i>	Compression data measured in mmHg. Measurements taken at the ankle, calf, knee and thigh on the anterior, lateral, posterior, and medial sides (Adapted from Liu <i>et al.</i> , 2005).....	31
<i>Table 3.3.</i>	Measurement sites for the use of compression garments as prescribed by the European document on normalization (Partsch <i>et al.</i> , 2006).	32
<i>Table 3.4.</i>	Blood lactate concentration ([Bla]) (mMol) of total circulating lactate adjusted for plasma volume shifts (adapted from Berry & McMurray, 1987)	42
<i>Table 3.5.</i>	Summary of research on VO_{2max}	49
<i>Table 6.1.</i>	Physical and anthropometrical characteristics of participants.....	79
<i>Table 6.2.</i>	Physiological characteristics of participants.....	80
<i>Table 6.3.</i>	Baseline [Hb] ($g \cdot dL^{-1}$) measurements before 10km running protocol with different compression conditions ($p > 0.05$).....	81
<i>Table 6.4.</i>	Cohen's effect size results for [Hb] measurements.....	81
<i>Table 6.5.</i>	Raw pressure values (mmHg) for the compression and flight socks.....	82
<i>Table 6.6.</i>	Cohen's effect size results for pressure measurements at the greatest circumference of the calf.	84
<i>Table 6.7.</i>	Cohen's effect size results for posterior calf pressures.....	85
<i>Table 6.8.</i>	Skin temperatures ($^{\circ}C$) in the right and left leg during the 10 km simulated running protocol (mean \pm SD).	86
<i>Table 6.9.</i>	Cohen's effect size results for skin temperature between the different compression conditions during the 10 km run protocol.	87

<i>Table 6.10.</i>	Cohen's effect size results for VO ₂ during the 10 km run protocol between the different compression conditions.	88
<i>Table 6.11.</i>	Results for the Cohen's effect size statistics for VCO ₂ during the 10 km run.	89
<i>Table 6.12.</i>	Cohen's effect size results for heart rate (HR) during the 10 km run protocol between the different compression conditions.	90
<i>Table 6.13.</i>	Absolute blood lactate concentration [BLa] (mmol.L ⁻¹) during the 10 km run (mean ± SD).	91
<i>Table 6.14.</i>	Cohen's effect size results for blood lactate concentrations during the 10 km run protocol.	92
<i>Table 6.15.</i>	Cohen's effect size results for O ₂ Hb during the 10 km run.	94
<i>Table 6.16.</i>	Cohen's effect size results for HHb during the 10 km run.	96
<i>Table 6.17.</i>	Cohen's effect size results for tissue oxygenation index during the 10 km run. .	98
<i>Table 6.18.</i>	Cohen's effect size results for nTHI between the different compression conditions during the 10 km run.	100
<i>Table 6.19.</i>	Change in oxygenation values from rest to end of 10 km run (mean ± SD).	101
<i>Table 6.20.</i>	Cohen's effect size results for the change from rest to end of exercise in NIRS variables.	101
<i>Table 6.21.</i>	Calf skin temperature during the 60 min recovery period.	102
<i>Table 6.22.</i>	Cohens's effect size results for temperature during the 60 min recovery period.	103
<i>Table 6.23.</i>	Absolute blood lactate concentrations ([BLa]) (mmol.L ⁻¹) during the 60 min recovery.	104
<i>Table 6.24.</i>	Cohen's effect sizes for blood lactate concentration values ([BLa]) during the 60 min recovery period.	105
<i>Table 6.25.</i>	Cohen's effect size results for O ₂ Hb during the 60 min recovery period.	107
<i>Table 6.26.</i>	Cohen's effect size results for HHb during the 60 min recovery period.	109

<i>Table 6.27.</i>	Cohen’s effect size results for TOI during the 60 min recovery.....	111
<i>Table 6.28.</i>	Cohen’s effect size results for nTHI during the 60 min recovery period.	113
<i>Table 6.29.</i>	Changes ($x \pm SD$) in TOI and nTHI as determined by the Delta 2 calculations..	114
<i>Table 6.30.</i>	Cohen’s effect size results for the Delta 2 calculations.....	115
<i>Table 6.31.</i>	Changes ($x \pm SD$) in TOI and nTHI as determined by Delta 3 calculations..	117
<i>Table 6.32.</i>	Cohen’s effect size results for Delta 3 calculations.....	117
<i>Table 6.33.</i>	Changes ($x \pm SD$) in TOI and nTHI as determined by Delta 4 calculations..	119
<i>Table 6.34.</i>	Cohen’s effect size results for Delta 4 calculations.....	120
<i>Table 6.35.</i>	Correlations between pressure outcome variables and various anthropometric characteristics.	121
<i>Table 7.1.</i>	Pressure (mmHg) results for the current study compared to previous studies for measurements on the posterior ankle.	131
<i>Table 7.2.</i>	Pressure (mmHg) results for the current study compared to previous studies for measurements on the posterior calf.	131

LIST OF EQUATIONS

p.

<i>Equation 2.1.</i>	Beer-Lambert equation for the determination of concentrations, where A is the attenuation, ϵ is the extinction coefficient, $[C]$ is the micro-molar concentration, and L is the inner dimension of the cuvette in which the solution is measured (Rolfe, 2000).	10
<i>Equation 2.2.</i>	The modified Beer-Lambert equation for use in NIRS, where A is the attenuation, I_0 is the light in, I is the light out, ϵ is the extinction coefficient, $[C]$ is the micro-molar concentration, L is the distance, x is the pathlength factor and K is the tissue loss (Hamamatsu Photonics Deutschland, 2012).	10
<i>Equation 2.3.</i>	Calculation used to determine change in concentration to account for tissue loss, where A_1 and A_2 is the attenuation of light at two different time points, $[C]$ is the micro-molar concentration, ϵ the extinction coefficient, L is the distance, and x is the pathlength factor (Hamamatsu Photonics Deutschland, 2012)...	11
<i>Equation 2.4.</i>	Calculation of tissue oxygenation index, where TOI is the tissue oxygenation index, k is the constant scattering contribution, O_2Hb is the oxy-haemoglobin and HHb is the deoxy-haemoglobin (Hamamatsu Photonics Deutschland, 2012).....	11
<i>Equation 2.5.</i>	Simplified calculation of tissue oxygenation, where TOI is the tissue oxygenation index, O_2Hb is the oxy-haemoglobin, and HHb is the deoxy-haemoglobin (Hamamatsu Photonics Deutschland, 2012).	12
<i>Equation 2.6.</i>	Determination of total haemoglobin index, where k is the constant scattering contribution, O_2Hb is the oxy-haemoglobin concentration, and HHb is the deoxy-haemoglobin concentration (Hamamatsu Photonics Deutschland, 2012).....	12

<i>Equation 2.7.</i>	Determination of the oxygenation index where Hb is the haemoglobin and Mb is myoglobin.....	12
<i>Equation 2.8.</i>	Where MOE is the muscle oxygenation index indicated as $W.\%^{-1}$, (t) is a specified time period, PO is the mean power output during the specified time period, and $mOxy$ is the mean muscle oxygenation during the specified time period (Scanlan <i>et al.</i> , 2008).	13
<i>Equation 2.9.</i>	Where Rr is the recovery rate, h is half the increment in muscle oxygenation during recovery and TR is the time taken to reach h (Ding <i>et al.</i> , 2001).....	24
<i>Equation 3.1.</i>	Determination of pressure, where P is the pressure in Pascal (Pa), F is the force in Newton (N), and A is the area to which the force is applied in square meter (m^2).	29
<i>Equation 3.2.</i>	Laplace equation, where P is the pressure (Pa), T is the wall tension ($N.m^{-1}$) and r is the radius of the cylinder in meter (m).	30
<i>Equation 3.3.</i>	Modified Laplace equation where, P' is the exerted compressive force, T is the circumferential tensile force in the fabric of the CS and C is the circumference of the limb at the specific point (Troynikov <i>et al.</i> , 2010).	33
<i>Equation 3.4.</i>	Determination of cardiac output where, CO is cardiac output in litres per minute ($L.min^{-1}$), HR is the heart rate in beats per minute ($beats.min^{-1}$) and SV is the stroke volume in litres ($L.beat^{-1}$).	36
<i>Equation 5.1.</i>	Conversion calculation to pressure, where x is the force measured (N) by the sensor. _	70
<i>Equation 5.2.</i>	Difference between the average of the pre-exercise rest period (a) and the average of the last 2 km of the running protocol (b).....	77
<i>Equation 5.3.</i>	Difference between end of exercise (x) and the first peak post-exercise (y)..	77
<i>Equation 5.4.</i>	The difference between the first peak post-exercise (y) and the end of the 60 min recovery period (z).....	77

Equation 5.5. The difference between the end of the running protocol (x) and the end of the
60 min recovery period (z)..... 77

CHAPTER ONE

INTRODUCTION

Since the introduction of compression garments (CG) into the sporting world, various claims have been made of the benefits that these garments hold for both performance and recovery. On account of these claims a vast variety of athletes ranging for Olympic to recreational runners, cyclists and triathletes, have started using these garments, although very few claims are sufficiently backed by scientific research.

Compression garments also come in a wide variety of apparel, with compression socks/stockings (CS), calf sleeves, lower body compression garments (LBCG), compression shorts, compression shirts and arm compression sleeves being only a few of the products found on the market. Furthermore, compression garments' pressure profiles differ between manufacturers. This is most likely due to different sizing based on various anthropometrical measurements of the individual. However it has been found that the pressure range, as indicated by the manufacturer, is not always the pressure range exerted by the compression garments due to interpersonal variations in anthropometrical and morphological characteristics (Liu *et al.*, 2005).

Knee-high compression socks were originally designed to treat various diseases of venous insufficiency such as varicose veins, deep vein thrombosis, leg ulcerations as well as lymphoedema (Dascombe *et al.*, 2006b; Rimaud *et al.*, 2007; Creasy, 2008; Liu *et al.*, 2008a; Ali, Creasy and Edge, 2010). Due to the clinical origin of the CS and the benefits it showed with regard to venous return, most research in sport science has focused on these aspects. The most popular hypothesis is that CS exerts a central effect, however, some researchers suggested that compression garments function more at a local level (Bochmann *et al.*, 2005; Macrae *et al.*, 2011).

It has been speculated that CS could, among other things increase cardiac output (Marks *et al.*, 2011), decrease heart rate (Marks *et al.*, 2011), increase sprinting performance (Sear *et al.*, 2010), decrease lactate production (Marks *et al.*, 2011), improve economy of movement (Bringrad, Perrey & Belluye, 2006), decrease muscle damage (Bindemann, 2007) and increase power (Kraemer *et al.*, 1996) during exercise. Similarly, a number of factors that could affect post-exercise recovery have been investigated, such as improved lactate removal (Bindemann, 2007; Lovell *et al.*, 2011), decreased muscle damage markers (Bindemann, 2007; Jakeman, Byrne & Eston, 2010), decreased delayed onset of muscle damage (DOMS) (Jakeman, Byrne & Eston, 2010) and decreased swelling (Kraemer *et al.*, 2000).

An alternative hypothesis to the one that lactate removal post-exercise is improved due to enhanced venous return, was set by Berry and McMurray (1987). Their hypothesis stated that lactate is probably retained within the muscle and then oxidized, thus improving lactate clearance. For the process of lactate oxidation, increased oxygenation of the muscle is assumed.

Near-infrared spectroscopy (NIRS) is a method by which muscle oxygenation can be determined non-invasively during exercise (McCully, Halber & Posner, 1994; Boushel & Piantadosi, 2000; Austin *et al.*, 2005; Thiel *et al.*, 2011). NIRS makes use of the difference in light absorbency of oxy-haemoglobin (O_2Hb) and deoxy-haemoglobin (HHb) at different wave lengths to determine the relative change in each of these in the tissue directly below the probe, with use of the Beer-Lambert equation (McCully, Halber & Posner, 1994; Chance & Bank, 1995; McCully & Hamaoka, 2000). In addition spatially resolved spectroscopy can determine the percentage of saturation experienced by the tissue (tissue oxygenation index, TOI) and the amount of blood (haemoglobin) present in the tissue (total haemoglobin index, nTHI)(Hamamatsu Photonics Deutschland, 2012).

Consequently NIRS can determine the relative change in oxygen delivery (O_2Hb) and oxygen consumption (HHb) in the involved tissue. From this information, certain inferences can be drawn as to which changes are taking place in the tissue to accommodate the applied stimulus, such as exercise, or as in this study, external compression.

Therefore, the application of this measurement technique during exercise and recovery could non-invasively provide information on the changes that are taking place at a cellular level when compression is applied to the muscle, rather than measuring the systemic changes. Therefore, this research endeavours to determine whether external compression has an influence on muscle oxygenation and whether this could then support the hypothesis set by Berry and McMurray (1987).

CHAPTER TWO

MUSCLE OXYGENATION

A. INTRODUCTION

In 1987 Berry and McMurray published the first sports related research on the effects of graduated compression stockings (CS) on blood lactate ([BLa]). In this study, six healthy male college students ($VO_{2max} = 59.9 \pm 6.8 \text{ mL.kg}^{-1}.\text{min}^{-1}$) underwent a three minute bicycle ergometer exercise test at 110 % of their VO_{2max} . The test was performed under three different conditions, namely CS during exercise and recovery, CS only during exercise and no CS during either exercise or recovery. They found markedly lower lactate concentrations when CS were worn during exercise and recovery compared to the control condition. This difference was not seen when comparing the control with wearing CS only during exercise. It was speculated that if compression garments stunted the production of lactate, removal of the CS post exercise would deliver similar lactate concentrations to when completing the exercise without the CS. This was not the case as the highest lactate concentrations five minutes post-exercise were found when participants removed the compression garment after exercise, and furthermore, this was not accompanied by plasma volume shifts. They subsequently hypothesized that the compression garments result in the retention of lactate within the muscle bed, therefore decreasing the concentration measured in the blood. This may then enhance lactate removal through the oxidation of lactate within the skeletal muscle itself. It is this research that has prompted the current study on the oxygenation of the skeletal muscle and the mechanism whereby compression socks may cause improved muscle lactate oxidation.

B. NEAR-INFRARED SPECTROSCOPY

Muscle oxygenation refers to the oxygen saturation level of the muscle. It can be measured in a number of ways of which Near-Infrared Spectroscopy (NIRS) is one. NIRS is a non-invasive method of measuring the tissue oxygenation in real-time and it can be applied to measure the tissue oxygenation of active muscle tissue (McCully, Halber & Posner, 1994; Boushel & Piantadosi, 2000; Austin *et al.*, 2005; Thiel *et al.*, 2011). These measurements give an indication of the change in oxygenation status of the tissue below the measurement probe, especially during exercise, where changes are expected (Bhambhani, Buckley & Susaki, 1997; Ferrari, Binzoni & Quaresima, 1997; Austin *et al.*, 2005). This is more specifically determined by the measurement of the change in oxy-haemoglobin (O₂Hb) and deoxy-haemoglobin (HHb) (McCully, Halber & Posner, 1994) and reflects changes in the arteriolar, capillary and venular beds in the tissue (Chance & Bank, 1995).

NIRS works by emitting near-infrared (NIR) light into the tissue and then measuring the fraction of light that is refracted back to the measuring probe (Boushel & Piantadosi, 2000). There are various techniques to achieve this and it depends on the specific lights, lasers or light-emitting diodes which are used, as well as the modality of light sources, namely continuous wave, continuous wave spatially resolved, pulsed or phase modulation (Ferrari, Binzoni & Quaresima, 1997). The data gained by these different modalities are then used in mathematical equations specific to the measurement modality to estimate the values of the physiological variables.

Since human muscle is too thick for light to pass through and to be detected on the other side as is done in the case of solutions, the refraction of the light must be used to determine the saturation of the muscle (McCully & Hamaoka, 2000). The movement of light photons in tissue is dependent on reflectance, scattering and absorption of the photons caused by the tissue (Jöbsis, 1977). Reflectance is affected by the angle of the light beam with respect to the tissue, whereas scattering is affected by the wavelength of the light and absorption is affected by the properties of the tissue (Jöbsis, 1977). These factors cause the light to move in a “banana-shape” or “U-shape” from the light source to the detector.

1. Wavelengths

As the wavelength of the light is increased, the amount of scattering is decreased. It is because of this characteristic that NIR light is used (Jöbsis, 1977). However, in the NIR light range of 700 to 1300 nm there is a significant amount of radiation that is transferred into the tissue (Jöbsis, 1977). Boushel and Piantadosi (2000) suggested a wavelength range of 700 to 1000 nm as they said that at this wavelength light easily passes through biological tissue such as bone, skin and muscle tissue. McCully and Hamaoka (2000) suggested the use of light in the 700 to 900 nm range as this range delivers good penetration of the tissue. Light is primarily absorbed by the haeme group of haemoglobin (Hb) and myoglobin (Mb) and the amount of light absorbed by these haeme groups is determined by the oxygen (O₂) content, as the peak absorbance of HHb and O₂Hb occur at different wavelengths.

Three factors influence the amount of light that is refracted back from the tissue. These are the O₂ dependent absorption from chromophores, which is the main variable in which change will be measured; the other is absorption from fixed chromophores such as melanin in the skin and light lost due to scattering (Ferrari, Mottola & Quaresima, 2004). When measuring one person, these two factors are constant over the time period of measurement and therefore will not influence the change measured in the variables, as is the case with most NIRS measurement modalities (Boushel *et al.*, 2001).

As stated earlier, the absorption spectra of tissue varies inherently, and is further influenced by the O₂ saturation of Hb. O₂Hb absorbs light at a different wavelength than HHb. It seems that O₂Hb absorbs more light and therefore has a greater absorbance at 850 nm, whereas HHb has greater absorbance at 760 nm (McCully, Halber & Posner, 1994; Chance & Bank, 1995; McCully & Hamaoka, 2000), therefore making it possible to determine the changes in the concentration of both HHb and O₂Hb.

2. Measurement depth

The depth to which the NIRS probe measures the different variables in the tissue is determined by the distance between the light source and the light detector. Generally, the depth of measurement is said to be half the distance between the light source and the detector (McCully & Hamaoka, 2000). This seems to be the case as light is scattered in the tissue. Light is sent into the tissue, where it bounces off different organelles in the tissue such as the mitochondria. Some of these light photons are then scattered back to the surface of the tissue where it can be picked up by the detector. The greater the distance between the source and the detector the greater the initial depth of the photons picked up and the greater the arc with which these photons move (<http://www.lea.de/eng/indexe.html>).

C. VARIABLES MEASURED

Researchers have determined that NIRS is a valuable tool in the measurement of variables related to tissue oxygenation. In a review by Boushel and Piantadosi (2000), it was stated that NIRS can be used to monitor changes in tissue oxygenation as well as blood flow. It is also used in a clinical setting as a tool to monitor early stages of vascular dysfunction by assessing microvascular factors, as well as to determine the blood transport capacity in persons with vascular dysfunction (McCully, Halber & Posner, 1994; Ferreira, Koga & Barstow, 2007).

Some sports-related research has also shown that NIRS can be used to determine the ventilatory threshold (VT) during incremental exercise testing (Bhambhani, Buckley & Susaki, 1997). In this study, VT was determined using both gas exchange analysis as well as NIRS. Subjects (Men, age = 30 ± 6 years, $VO_{2max} = 47.0 \pm 9.0$ mL.kg⁻¹.min⁻¹; Women, age = 27 ± 5 years, $VO_{2max} = 40.6 \pm 8.3$ mL.kg⁻¹.min⁻¹) completed an incremental exercise test to VO_{2max} . The VT was then calculated from the gas exchange data using the V-slope method. This method identifies VT as the VO_2 where VCO_2 increases non-linearly (Beaver, Wasserman & Whipp, 1986). VT was then also determined

from the NIRS data. This was said to occur when the 20 s absorbency measurement crossed the baseline value which was determined directly before exercise commenced. They found a validity coefficient of 0.90 and 0.89 for men and women respectively in the determination of the VT by these two methods. The VT determined by means of gas exchange and NIRS were not statistically significantly different from each other. It was therefore found that NIRS could be used to determine the VT.

NIRS measures changes in HHb and O₂Hb concentration and can therefore determine the microvascular extraction of oxygen. However, certain assumptions have to be made as it is an indirect measurement. These assumptions are listed by Ferreira, Koga and Barstow (1999) as:

1. Oxygen extraction is measured by the HHb concentration
2. The Mb signal measured by NIRS does not influence the relationship between the variables
3. Exercise and the intensity thereof does not influence the specific microvascular compartment measured
4. The changes in NIRS measurements when going from rest to exercise are an indication of the changes in oxygen extraction due to the exercise

1. Validity and reliability

Boushel *et al.* (2001) validated NIRS measurements of O₂Hb against arterio-venous O₂ difference, whereas Hyttel-Sorensen *et al.* (2011) validated cerebral oxygenation against jugular bulb saturation. The algorithms used to determine the variables from the light attenuation measured by NIRS have also been validated (Hansen *et al.*, 1996).

Austin *et al.* (2005) determined the reliability of NIRS for the measurement of muscle oxygenation, as well as the determination of a breakpoint in the muscle oxygenation and its correlation with other breakpoints such as the lactate threshold and anaerobic threshold. They tested 25 runners on a treadmill and 21 cyclists on a cycle ergometer with incremental tests to exhaustion. All participants completed two incremental exercise tests, either on the treadmill (runners) or on a

cycle ergometer (cyclists). When these two tests were compared they found NIRS to be a reliable means to measure muscle oxygenation in runners ($r = 0.87$ at lactate threshold and $r = 0.88$ at VO_{2max}) and cyclists ($r = 0.94$ at lactate threshold and $r = 0.99$ at VO_{2max}).

D. BLOOD FLOW

There are several methods of using NIRS equipment to measure blood flow within the tissue. One of these methods is using an exogenous tracer and the Fick principle. Hereby the rate of tracer inflow and accumulation (usually indocyanine green) is used to estimate blood flow. The accumulation of the tracer is the sum of the rate of inflow minus the rate of outflow. Indocyanine green (ICG) is a water soluble tricyanine dye that absorbs NIR light at 800 nm (Boushel & Piantadosi, 2000). Thus NIRS is used to determine the amount of absorption caused by the ICG in the tissue, as this will give an indication of the accumulation of the ICG in the tissue. Other researchers have used changes in O_2Hb to determine arterial blood flow within the tissue (Boushel & Piantadosi, 2000). In essence, O_2Hb indicates the amount of arterial blood present in the tissue, and therefore, increases in this variable could be used as an indication that arterial blood flow has increased.

Another means of measuring blood flow is by making use of laser Doppler techniques. Some NIRS equipment not only makes use of NIR light, but also include laser light. Thus using the Doppler principle, it can determine the flow of blood by determining the velocity by which the erythrocytes move within the microvasculature (<http://www.lea.de/eng/indexe.html>). These measurements of blood flow are valuable as it provides information on the amount of oxygen delivered to the tissue, which in combination with the oxygenation of the tissue will give a better understanding of the physiological processes at play within the tissue.

Other than using the Doppler technique and the Fick principle, NIRS can only be used as an indirect means of estimating blood flow, and more specifically changes thereof.

E. CALCULATION OF DIFFERENT VARIABLES

1. Beer-Lambert law

The Beer-Lambert law (*Equation 2.1.*) is regularly used for the determination of chemical concentrations of solutions:

$$A = \varepsilon C L \quad (2.1)$$

Equation 2.1. Beer-Lambert equation for the determination of concentrations, where A is the attenuation, ε is the extinction coefficient, $[C]$ is the micro-molar concentration, and L is the inner dimension of the cuvette in which the solution is measured (Rolfe, 2000).

With NIRS the Beer-Lambert law is applied to measure the changes in O₂Hb and HHb concentrations. As stated earlier, the amount of light scattered back from the tissue is measured and then applied to the modified Beer-Lambert equation (*Equation 2.2.*) to determine these concentrations.

$$A = \log \frac{I_0}{I} = \varepsilon C Lx + K \quad (2.2)$$

Equation 2.2. The modified Beer-Lambert equation for use in NIRS, where A is the attenuation, I_0 is the light in, I is the light out, ε is the extinction coefficient, $[C]$ is the micro-molar concentration, L is the distance, x is the pathlength factor and K is the tissue loss (Hamamatsu Photonics Deutschland, 2012).

Due to the fact that NIRS is measured *in vivo*, a tissue loss factor (K) has to be added to the Beer-Lambert equation. This then renders it impossible to determine the absolute measurements of O₂Hb and HHb concentrations without the knowledge of this factor. To solve this problem, the change in O₂Hb and HHb concentration is measured (*Equation 2.3.*):

$$\Delta A = A_2 - A_1 = \Delta[C]\varepsilon Lx \quad (2.3)$$

Equation 2.3. Calculation used to determine change in concentration to account for tissue loss, where A_1 and A_2 is the attenuation of light at two different time points, $[C]$ is the micro-molar concentration, ε the extinction coefficient, L is the distance, and x is the pathlength factor (Hamamatsu Photonics Deutschland, 2012).

Therefore with this equation (*Equation 2.3*), and specifically with the system used during this study, the changes in the variables ΔO_2Hb , ΔHHb and ΔcHb , are calculated.

2. Spatially resolved spectroscopy

Tissue oxygenation index (TOI) (*Equation 2.5*) and total haemoglobin index (nTHI) (*Equation 2.6*) is determined by spatially resolved spectroscopy (SRS). These variables are determined by using the difference in attenuation of light at two measuring sites on the detector probe. The distance between the light source and light detector is such that the loss of light on the two measuring sites due to scattering is the same, and therefore the difference between the detected light of these two sites is due to the differences in absorption. The tissue oxygenation index is then calculated with the following equation:

$$TOI = \frac{k_{O_2Hb}}{k_{O_2Hb} + k_{HHb}} \quad (2.4)$$

Equation 2.4. Calculation of tissue oxygenation index, where TOI is the tissue oxygenation index, k is the constant scattering contribution, O_2Hb is the oxy-haemoglobin and HHb is the deoxy-haemoglobin (Hamamatsu Photonics Deutschland, 2012).

This can then be mathematically simplified to exclude the constant scattering contribution variable as follows:

$$TOI = \frac{O_2Hb}{O_2Hb+HHb} \quad (2.5.)$$

Equation 2.5. Simplified calculation of tissue oxygenation, where *TOI* is the tissue oxygenation index, *O₂Hb* is the oxy-haemoglobin, and *HHb* is the deoxy-haemoglobin (Hamamatsu Photonics Deutschland, 2012).

Similarly, *nTHI* is determined by the following equation:

$$nTHI = kO_2Hb + kHHb \quad (2.6.)$$

Equation 2.6. Determination of total haemoglobin index, where *k* is the constant scattering contribution, *O₂Hb* is the oxy-haemoglobin concentration, and *HHb* is the deoxy-haemoglobin concentration (Hamamatsu Photonics Deutschland, 2012).

3. Other calculations

There are also some differences regarding the use of the term oxygenation index and the means by which it is calculated. Grassi *et al.* (2003) determined the oxygenation index by the following equation:

$$Oxygenation\ index = \Delta(oxy\ Hb + Mb - deoxy\ Hb + Mb) \quad (2.7.)$$

Equation 2.7. Determination of the oxygenation index where *Hb* is the haemoglobin and *Mb* is myoglobin.

These different equations are used by different manufacturers. For this particular study the NIRO 200nx (Hamamatsu, Japan) was used and therefore *TOI* as determined by *Equation 2.5.* was used.

Scanlan *et al.* (2008) used *Equation 2.8.* to calculate muscle oxygenation economy during an one hour cycle time trial. This variable indicates how economical the muscle is with regard to oxygen extraction and utilization during exercise relative to the power output.

$$MOE_t = \frac{PO_t}{(100 - mOxy_t)} \quad (2.8.)$$

Equation 2.8. Where *MOE* is the muscle oxygenation index indicated as $W.\%^{-1}$, (*t*) is a specified time period, *PO* is the mean power output during the specified time period, and *mOxy* is the mean muscle oxygenation during the specified time period (Scanlan *et al.*, 2008).

G. ADVANTAGES OF NIRS

When determining the metabolic condition within working muscle, muscle oxygenation is a critical variable that changes under different conditions (Sako *et al.*, 2001). NIRS is a means of measuring these changes. There are a few advantages to NIRS technology. The fact that NIRS is a non-invasive method makes it possible to measure muscle oxygenation of active muscle during exercise, whether in patients with vascular insufficiency or in trained athletes (McCully, Halber & Posner, 1994). Furthermore, when comparing NIRS with other methods, such as phosphorus magnetic resonance spectroscopy (^{31}P -MRS), it was found that NIRS has a higher sensitivity for the changes in metabolism (Sako *et al.*, 2001). NIRS also enables the researcher to determine the oxygenation of very specific areas within the tissue, rather than the tissue as a whole (Boushel *et al.*, 2001).

H. FACTORS INFLUENCING NIRS MEASUREMENTS

1. Skin blood flow

Though there are many advantages in using NIRS, there are also some limitations to this technology. One of the factors that influence the measurement is skin blood flow. Changes in skin blood flow happen as a result of redistribution of blood during exercise and/or increases in skin temperature. These changes in local blood flow affect total blood volume, O₂Hb, as well as TOI in resting conditions (Ferreira, Koga & Barstow, 2007).

Davis *et al.* (2006) found that both local heating of the skin as well as whole body heating caused a significant increase in tissue oxygenation. They tested six healthy individuals with local heating of the skin during 15 minute stages at ~ 37.0, ~ 39.0 and ~ 41.0 °C. They found changes in tissue oxygenation optical density from 0.82 ± 0.89 to 18.21 ± 2.44 and skin blood flow from 26.00 ± 5.00 to 175.00 ± 20.00 (arbitrary units; AU). They also found a correlation between local heating and skin blood flow ($r = 0.95 \pm 0.02$). During a second protocol, whole body heating was induced in eight healthy individuals. Baseline measurements were taken at 34.0 °C where after 46.0 °C was induced until ~ 0.8 °C increase in the internal core temperature was found. This increased tissue oxygenation from -0.31 ± 6.47 to 12.48 ± 1.82 optical density and skin blood flow from 33.60 ± 6.80 to 135.90 ± 13.20 (AU). There was also a correlation found between tissue oxygenation and skin blood flow ($r = 0.89 \pm 0.04$). Thus, skin blood flow could influence NIRS measurements independent of muscle blood flow and muscle oxygenation.

Grassi *et al.* (2003) stated that it is likely that increases in skin blood flow could affect the muscle oxygenation measurement by increasing the amount of oxygenated blood in the skin. To exclude this factor they used $\Delta[\text{deoxy (Hb + Mb)}]$ which only shows changes in capillary oxygenation and therefore the amount of oxygen used by the muscle.

Determining skin temperature allows the researcher to determine whether changes in NIRS variables were solely due to changes in the tissue measured or whether skin blood flow could have had an effect.

2. Adipose tissue thickness

Another factor that influences NIRS measurements is the amount of subcutaneous fat at the measurement site. Adipose tissue below the skin varies between individuals according to the body composition and fat distribution of the person (Van Beekvelt *et al.*, 2001). It seems that the amount of subcutaneous fat influences the absorbance of the light by not absorbing as much light as would other leaner tissues. This then causes a stronger signal that would reflect back to the detector (McCully & Hamaoka, 2000).

Van Beekvelt *et al.* (2001) investigated the effect of adipose tissue thickness on muscle oxygenation measurements and also sought to determine whether there was a gender effect. They found that there were statistically significant decreases in muscle oxygen consumption with the increase in adipose tissue thickness. Thus NIRS derived muscle oxygenation is underestimated in persons with thick subcutaneous fat layers. The researchers also found that the women in the study had lower muscle oxygen consumption levels than the men, which they ascribed to the differences in adipose tissue thickness between genders.

As stated earlier, the measurement depth of the NIRS probes is said to be half the distance between the light source and the light detector (McCully & Hamaoka, 2000). Thus, as the adipose tissue thickness increases, the amount of penetration of light into the muscle tissue below this adipose tissue layer will decrease. Therefore, if the adipose tissue is thicker than the depth at which the NIRS probe measures, the probe will be measuring the oxygenation of the adipose tissue rather than the muscle tissue (Van Beekvelt *et al.*, 2001).

To account for adipose tissue thickness, it should be strived to use participants with small adipose tissue thicknesses. Measuring the adipose tissue thickness will also enable the researchers to determine whether the measurement depth is sufficient to overcome the adipose tissue thickness.

3. Other factors

Though these previously mentioned factors may be the major influences on the measurement of muscle oxygenation, there are a few other factors that also play a role in NIRS measurements. Austin *et al.* (2005) suggested that muscle fibre composition, blood haemoglobin concentration, skin temperature and the ratio of capillary density to cross-sectional area could also influence these measurements. Because of the differences in composition and metabolic demands of different muscle fibre types, these fibre types could be oxygenated to different levels at rest but especially during exercise (Austin *et al.*, 2005). As Type I muscle fibres have high concentrations of mitochondria and high capillary densities, they have high oxidative capacities and therefore utilize more oxygen. Whereas Type II muscle fibres make more use of anaerobic metabolism and therefore rely less on oxygen utilization. This will then have a great effect on the concentration of HHb as the blood leaving the muscle will be more deoxygenated in Type I fibres as more oxygen is utilized in these fibres. This does not influence the credibility of the measurement, but if different values are obtained from different muscles, the muscle fibre type must be taken into account or the data must be reported accordingly.

McCully, Halber and Posner (1994) also saw a difference in the muscle saturation of young (age = 27.5 ± 5.8 years) and old (age = 68.5 ± 6.6 years) individuals during a walking test, though there was no difference in their saturation levels during recovery. In this case the differences were attributed to the fact that the walking exercise was at a lower relative work load for the young group due to their longer leg lengths and training state. Therefore it is not necessarily age that influences the saturation but rather the training state and efficiency of movement. These are all factors that a researcher will have to keep in mind when making use of this technology.

I. SKELETAL MUSCLE OXYGENATION

Thiel *et al.* (2011:279) stated:

“StO₂ represents a changing balance between oxygen consumption and oxygen supply in muscle and other tissues.”

Thus, when monitoring muscle oxygenation, various factors are at play to determine the TOI or StO₂ that is then measured (*Figure 2.1.*). Decreases in muscle oxygenation can therefore be caused either by a decrease in oxygen delivery, such as seen in arterial occlusions, or an increase in oxygen utilization as is evident during exercise. According to Subudhi, Dimmen and Roach (2007) the oxygenation of skeletal muscle beds is not homogenous, as there are certain factors within the body that influence the saturation of tissue, such as capillary blood flow and muscle fibre type.

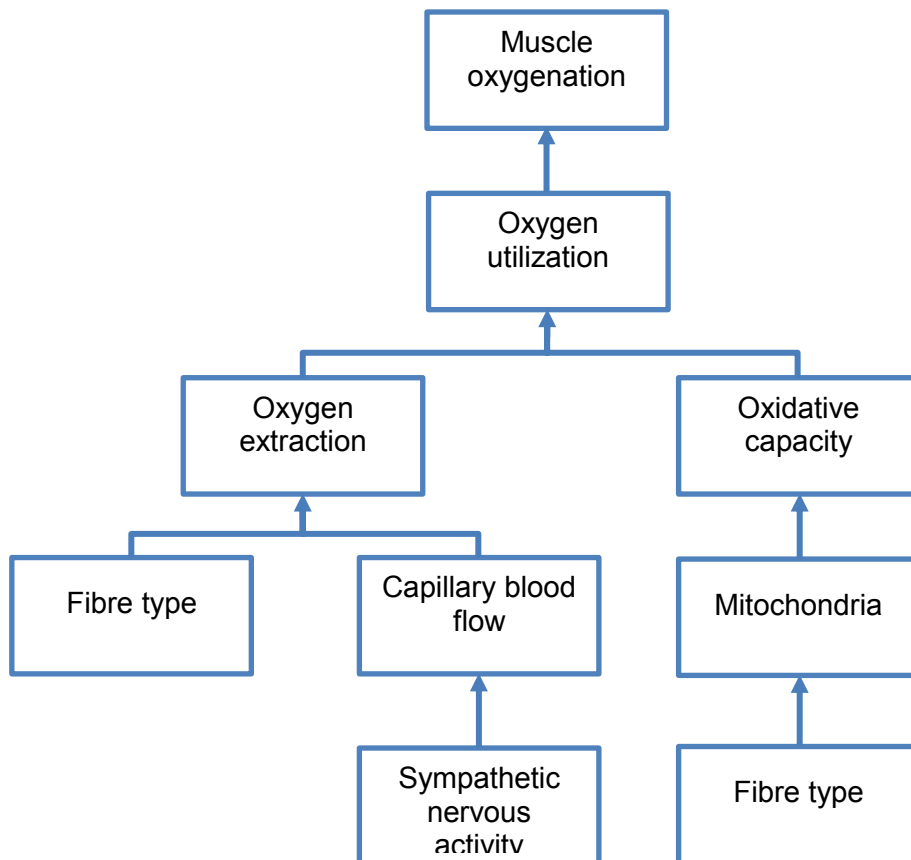


Figure 2.1. Factors affecting muscle oxygenation

1. Capillary blood flow

Capillary blood flow influences oxygen extraction and therefore ultimately muscle oxygenation by influencing the diffusion distance and diffusion time.

Bringard *et al.* (2006) found an increase in muscle oxygenation with the use of compression tights and attributed this to increased capillary blood flow which caused increased volumes of blood and therefore oxygen in the tissue.

The sympathetic nervous system is the major factor regulating the cardiovascular system during exercise. Activation of this system causes the increase in heart rate and blood pressure (Hansen *et al.*, 1996). It also causes vasoconstriction of the vascular beds of most organs and vasodilation of the active skeletal muscle vascular beds, thus redistributing the blood from the gastro-intestinal tract and splenic circulation to that of the active skeletal muscle (Chavoshan *et al.*, 2002). Thus the sympathetic nervous system is primarily responsible for the regulation of skeletal muscle oxygen saturation during exercise.

Hansen *et al.* (1996) tested the hypothesis that the reflex sympathetic activation, that causes a decrease in resting skeletal muscle oxygenation, will not have this effect if the muscle is exercised. They tested the muscle oxygenation of forearm muscles in the resting state as well as during exercise, during lower body negative pressure induction and found that the decrease seen in muscle oxygenation in the resting state was not seen in the exercising forearm muscle. Further tests then also showed that when reflex sympathetic activation was inhibited, there was still a decrease in muscle oxygenation during high intensity exercise. Thus they stated that the reflex sympathetic activation was not a major determining factor in contraction induced muscle deoxygenation.

Chavoshan *et al.* (2002) investigated the effect of nitric oxide (NO) on active muscle oxygenation. By inhibiting NO synthase (NOS) activity with the use of a NOS inhibitor, L-NAME, they were able to determine the vital role that NO production plays in attenuating the exercise induced deoxygenation of the active muscle. They thus concluded that sympathetically mediated decreases

in active muscle oxygenation was less in circumstances where NO was produced, whereas when NO production was inhibited, the amount of deoxygenation in the active muscle was greater.

2. Muscle fibre type

There is variability in the fibre composition of the major skeletal muscle groups. Thus a muscle will not consist of only one fibre type, but will be composed of different fibre types (Behnke *et al.*, 2003). However, a skeletal muscle group consists of a greater percentage of a certain muscle fibre type, therefore giving it the overall characteristics of that specific muscle fibre type. Muscle fibre types differ from each other in terms of oxidative capacities and Mb concentrations (Bladwin, Hooker & Herrick, 1978; Behnke *et al.*, 2003). Slow-twitch muscle fibres have greater oxidative capacities (Peter *et al.*, 1972) as well as greater capillary densities than fast-twitch fibres (Austin *et al.*, 2005).

Behnke *et al.* (2003) found that the Soleus muscle demonstrated greater oxygen consumption than the Peroneal muscle. In these rats the Soleus was composed of 86 % type I muscle fibre, whereas the Peroneal muscle consisted of 84 % type II muscle fibres. This then could indicate that slow-twitch muscle fibres utilize more O₂ as would be expected. Later McDonough *et al.* (2005) found that resting muscle oxygen consumption was higher in the Soleus compared to both the mixed Gastrocnemius and white Gastrocnemius. This occurrence was also seen in low as well as high contraction tempo protocols. In this study the Soleus consisted of type I fibres, the mixed gastrocnemius of type IIa, and the white gastrocnemius of type IIb fibres. Type I muscle fibres have high mitochondrial counts and therefore higher oxidative capacities (Austin *et al.*, 2005). This could therefore be the reason for the differences in oxygen utilization between type I and type II fibres.

It has also been shown that slow-twitch muscle fibres exhibit a greater expression of endothelial nitric oxide synthase (eNOS) mRNA, therefore they have a greater production of eNOS (Behnke *et al.*, 2003; McDonough *et al.*, 2005). eNOS is a vasodilator secreted by the endothelial cells to dilate the arterioles within the muscle bed. Furthermore, slow-twitch fibres are also more sensitive

to stimuli such as shear stress and endothelium-dependent agents, therefore having a greater vasodilatory response to these stressors than fast-twitch muscle fibres (Woodman *et al.*, 2001). This increased vasodilatory response causes an increased delivery of oxygen to the slow-twitch muscles. This was evident in data collected by McDonough *et al.* (2005) who showed that the oxygen delivery in the Soleus (slow-twitch) was higher than in the mixed Gastrocnemius and white Gastrocnemius (fast-twitch) muscle fibres during rest as well as recovery.

Therefore the differences in muscle oxygenation levels of different fibre types can be attributed to the differences in oxidative capacity of the fibre type, oxygen delivery to the muscle, reaction of the mitochondria to the onset of exercise and the matching of blood flow and oxygen extraction (Behnke *et al.*, 2003).

J. CAUSES OF DESATURATION

Various factors influence muscle oxygen consumption and oxygen availability. When using NIRS to measure the oxygenation status of tissue, the assumption is made that the HHb signal displays the oxygen extraction within the tissue investigated (Ferreira, Koga & Barstow, 1999). Because arterial O₂Hb saturation stays relatively constant even with increases in workload, Belardinelli *et al.* (1995) stated that the decrease in oxygenation is due to the desaturation of O₂Hb and oxy-myoglobin (O₂Mb) at the tissue level. Although Wilson *et al.* (1989) is of the opinion that in the NIR range the desaturation in the signal is mainly due to the desaturation of Hb rather than Mb.

Grassi *et al.* (1999) used NIRS to determine whether muscle oxygenation indexes were related to the onset of blood lactate accumulation during incremental exercise. They suggested that muscle deoxygenation occurred when the muscle has reached its maximum vasodilatory capacity, thus when the muscle is fully vasodilated. This indicates that muscle deoxygenation in this situation is due to the increase in muscle oxygen extraction and utilization rather than a decrease in blood flow to the muscle.

Endurance athletes are adapted to make greater use of aerobic metabolism. They thus have greater oxidative capacities as well as greater capillary densities. These adaptations to training enable the athletes to better extract O₂ from the blood. If the haematocrit values in two muscles are the same, then the muscle with the greater capillary density will experience better oxygen extraction as there will be more red blood cells that are adjacent to the muscle fibres for the O₂ to diffuse into the muscle (Behnke *et al.*, 2003; McDonough *et al.*, 2005).

K. MUSCLE OXYGENATION DURING EXERCISE

1. Incremental exercise

Several studies have reported a decrease in muscle oxygenation during incremental exercise and there seems to be a pattern to these changes which can be divided into four phases (Bhambhani, Buckley & Susaki, 1997; Ferreira, Binzoni & Quaresima, 1997; McCully & Hamaoka, 2000; Ding *et al.*, 2001; Kawaguchi *et al.*, 2001; Hiroyuki *et al.*, 2002; Subudhi, Dimmen & Roach, 2007). With the onset of exercise, there is an increase in muscle oxygenation relative to the baseline level (phase 1), but as the workload increases, the muscle starts to desaturate (phase 2). This then levels off until VO_{2max} is reached (phase 3). During recovery (phase 4), there is a large increase in muscle oxygenation above the initial rise in oxygenation seen in the first phase (Bhambhani, Buckley & Susaki, 1997).

Ferreira, Koga and Barstow (2007) attributed this increase in oxygen saturation level of the tissue in the first phase to the faster increase in muscle blood flow than the increase in muscle oxygen use. This increase is due to signalling through the breakdown of adenosine triphosphate (ATP) (Chance & Bank, 1995). With muscle contraction the effect of the skeletal muscle pump causes the facilitation of venous return, therefore increasing the flow of arterial blood into the muscle due to an increased cardiac output (CO); this along with the vasodilation of the vascular bed within the active

muscle causes a rapid increase in the oxygenation of the active muscle during light exercise (Hiroyuki *et al.*, 2002; Ferreira, Koga & Barstow, 2007).

Ferreira, Koga and Barstow (2007) described the desaturation profile of active muscle during incremental exercise as having an S-shaped curve, where the increases in desaturation are less when nearing the VO_{2max} . Belardinelli *et al.* (1995) investigated the effect of incremental cycling exercise with NIRS, where the resistance on the cycle increased by $20 \text{ W}\cdot\text{min}^{-1}$. The researchers found that the decrease in muscle oxygenation was greatest in the first four minutes of the incremental test, up until a resistance of 80 W, where after the decreases in oxygenation was not as pronounced. They also found that the muscle oxygenation did not decrease dramatically from 80 % of VO_{2max} onwards. This was attributed to the fact that a physiological maximum for muscle desaturation was reached as the muscle did not further desaturate even though the VO_2 increased; thus it appears that there is a physiological maximum for oxygen extraction (Spencer, Murias & Paterson, 2012).

Grassi *et al.* (1999) tested five well trained mountain climbers (all men; $VO_{2max} = 51.0 \pm 4.2 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) during an incremental cycle test to exhaustion. Lactate threshold was determined at the inflection point where lactate started to accumulate, thus the lowest point where after the succeeding point was more than 0.5 mM higher than the previous. The muscle oxygenation inflection point was also assessed by determining the combination of linear regressions that resulted in the smallest sum of square residuals. When comparing these inflection points, they found that in four of the five subjects, the lactate and muscle oxygenation inflection points were at the same workload. The lactate inflection point of the other subject was 30 W higher than the muscle oxygenation inflection point.

Yoshikawa *et al.* (2006) found a correlation between lactate threshold and the inflection point of ΔHHb ($r = 0.80$, strong correlation), although the TOI inflection point was not correlated with the lactate threshold. In their study they tested 15 healthy men ($VO_{2max} = 44.9 \pm 6.7 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) completing an incremental cycle test to exhaustion. This ΔHHb is different from the muscle

oxygenation inflection point mentioned above, in that it measures only the muscle oxygen consumption, whereas the muscle oxygenation inflection point measures the saturation of the muscle, which is in effect the TOI measurement. Owing to the fact that similar manners of determining the lactate threshold was used, the difference in these two studies warrants further investigation into which of the variables, oxygen consumption or tissue saturation, will better predict lactate threshold, and whether either of these two have an influence on lactate kinetics.

2. Continuous sub-maximal exercise

During continuous submaximal exercise, it seems that a steady state for muscle oxygenation is reached. Christmass *et al.* (1999) tested muscle oxygenation during sustained submaximal exercise and found small phasic variations in the muscle oxygenation. These variations were attributed to the movement of the muscle during contraction, therefore changing the geometry of the muscle and thus the site measured by the probe which is placed on the surface of the skin.

Kowalchuk *et al.* (2002) studied muscle oxygenation during sustained exercise in six healthy men ($\text{VO}_{2\text{max}} = 4400 \pm 200 \text{ L}\cdot\text{min}^{-1}$). This was done at both high ($244 \pm 20 \text{ W}$) as well as moderate (~ 90 % lactate threshold intensity; $140 \pm 21 \text{ W}$) intensity exercise. At the start of both tests a decrease in oxygenation was found. This was then sustained during moderate intensity exercise, but further deoxygenation took place during the high intensity exercise.

3. High intensity exercise

Christmass *et al.* (1999) also studied the effect of high-intensity intermittent exercise on the muscle oxygenation levels. At the onset of the exercise, there was a decrease in the relative O_2Hb levels and this indicated that the mechanisms used to match O_2 supply with O_2 demand could not keep up with the initial increase in demand of oxygen. As the exercise continued, the muscle oxygenation level increased again and reached a steady state as the supply and demand of oxygen was matched.

4. Eccentric exercise

Davies *et al.* (2008) studied muscle oxygenation with eccentric exercise in nine healthy men. The rationale for this came from the belief that eccentric exercise hinders O₂ delivery by influencing the blood-muscle O₂ flux. They found no relationship between muscle damage markers and the HHb response, but did find a significantly slower HHb (30 % slower) response after the eccentric exercise. They attributed this to a mismatch in O₂ delivery and O₂ consumption due to microcirculation disruption.

L. MUSCLE OXYGENATION DURING RECOVERY AFTER EXERCISE

Ding *et al.* (2001) found that muscle oxygenation after the cessation of exercise was elevated above resting levels prior to exercise. They also defined muscle oxygenation recovery rate as:

$$R_r = \frac{h}{T_R} \quad (2.9)$$

Equation 2.9. Where R_r is the recovery rate, h is half the increment in muscle oxygenation during recovery and T_R is the time taken to reach h (Ding *et al.*, 2001).

Ferrari, Binzoni and Quaresima (1997) stated that O₂ resaturation is faster in endurance trained athletes than in sedentary subjects. Similarly, Belardinelli *et al.* (1997) found that patients with chronic congestive heart failure have slower recovery rates of energy stores after exercise, which they stated could be either due to slow resaturation tempos or slow metabolic recovery of phosphocreatine. Neary, Hall and Bhambhani (2001) hypothesized that the build-up of metabolic by-products as well as the increase in muscle temperature could be the cause for the delay in recovery of muscle oxygenation to resting levels.

M. CONCLUSION

NIRS is a non-invasive means of determining tissue oxygenation and can be applied to a variety of situations. It has been found valid and reliable for determining muscle oxygenation during exercise, though there are a few factors that may influence the accuracy of the measurement such as, adipose tissue thickness and skin blood flow. Further research on the influence of gender and fitness level should be done to determine whether these variables may influence the muscle oxygenation and how this is brought about. Research also has to be done on the effect of compression on the oxygenation of the underlying tissue with regard to the influence on microvascular circulation. Most studies make use of either bandage or plaster in fixing the probes to the body during both rest and exercise, and this could therefore influence these measurements as the pressure applied by these modalities are very variable. It is not known to what extent NIRS measurements are affected by this practice.

CHAPTER THREE

COMPRESSION GARMENTS

A. DEFINING COMPRESSION GARMENTS

Compression garments (CG) are compressive clothing that exert an external force to the underlying parts of the body. This may include full-, upper-, or lower-body garments and are usually manufactured from elastic and other materials. The stretch of the material causes varying degrees of compression, and the magnitude of compression also depends on the shape and form of the body part that the material covers.

B. THE APPLICATION OF COMPRESSION GARMENTS

Originally CG and more specifically compression socks (CS), which are knee length socks, were designed for medical purposes. CG have been successfully implemented in the treatment of an array of conditions such as venous insufficiency, varicose veins, deep vein thrombosis, leg ulcerations as well as lymphoedema (Rimaud *et al.*, 2007; Liu *et al.*, 2008a), due to the increase in venous blood flow and lymph drainage caused by the compression on the limb (Creasy, 2008; Bringard, Perry & Belluye, 2006). Researchers suggested that CG could decrease swelling by increasing hydrostatic pressure and therefore increasing lymph drainage (Kraemer *et al.*, 2001b).

The next step was the development of CS for use during long haul flights, where deep vein thrombosis and excessive swelling of the lower limbs are regularly encountered (Belcaro *et al.*, 2002). Flight socks (FS) (18 – 22 mmHg) were introduced for this purpose, which exerts lower pressures than CS (20 – 30 mmHg) that is typically used in a clinical setting.

In the past decade, the use of CG in sport has been growing in popularity among elite and recreational athletes (Nusser & Senner, 2010; Sperlich *et al.*, 2011). Although it is more popular among endurance athletes, it is also applied in power and sprint activities as well as team sports settings. The socks are worn during training sessions, warm-ups, competition and during cool down and recovery. In endurance sports such as running, cycling and triathlons, athletes make use of CS as both a performance aid as well as a recovery strategy, whereas power-lifters and swimmers make use of full-body CG as a mechanical ergogenic aid (Creasy, 2008). During swimming, compression is said to cause slimming of the body, thereby improving streamline movement of the athlete through the water. During weight- or power-lifting the compression of the joints is said to ease the movement under heavy loads (Harman & Frykman, 1990).

C. CLAIMS

CG for athletes are marketed as having both performance and recovery benefits. Some manufacturers claim that CG enhance strength, power and endurance performance (Bakken, 2011; Dai *et al.*, 2011; Dascombe *et al.*, 2011b). It has been proposed by researchers that CS may assist the skeletal muscle pump in the extremities with venous return of blood (Ali, Creasy & Edge, 2010), which would lead to an increase in stroke volume (SV) and cardiac output (CO) and ultimately enhance oxygen delivery to the active muscles. Manufacturers also claim that CS enhance proprioception (http://revelsports.com/2XU_Compression/2XU_Compression%20_Socks.asp).

It is also claimed that CG enhance recovery after exercise. In some instances, these claims have been supported by research, showing reduced lactate (Bindemann, 2007; Lovell *et al.*, 2011), and reductions in post-exercise muscle fatigue and improved thermoregulation (Duffield *et al.*, 2008; Houghton, Dawson & Maloney, 2009). Some manufacturers also contend that CG support the muscles and reduce muscle fibre damage due to exercise, as well as increase the oxygen delivery to the muscles (www.falke.co.za).

However, most of these claims have not yet been conclusively substantiated by scientific research. Claims such as that lactate kinetics enhance recovery may not be supported by the modern scientific thinking of lactate. Research is needed to fully understand the benefits of compression garments in the sport setting, as well as the mechanism(s) whereby these benefits are achieved.

D. COMPRESSION

Originally CG were designed to apply compression uniformly over the surface of the limbs; therefore the external pressure was the same at all measurement sites. The idea to graduate the compression over lower limbs was developed by Van der Molen (in Choucair & Phillips, 1998), and this idea was further developed into graduated compression stockings (GCS) by Sigg and Ganzoni (in Choucair & Phillips, 1998). In light of the fact that CS were originally used to treat thrombosis, it was reasoned that the greatest compression should be placed over the area where the thrombosis occurs most prominently, which in most cases are the ankles (Sigg, 1963).

1. Measurement of compression

The compression of tissue underneath a CS or CG is measured by means of pneumatic, piezoelectric, fluid-filled, resistive or capacitive measurements (Partsch *et al.*, 2006). Pneumatic sensors make use of a pressure bladder, which is attached to a pressure meter by means of tubing (Ali, Creasy & Edge, 2010). The bladder is then inflated and inserted between the skin and the CS. The CS then compresses the air within the bladder and this is measured by a manometer (Sigg, 1963). Fluid-filled pressure sensors, on the other hand, make use of fluid to fill the bladder and measures the pressure applied to the fluid.

Resistive or capacitive sensors measure pressure by converting force measurements to pressure. Because compression is equal to the force applied over an area, the compression can be calculated if the measurement area is known, as evident in the following equation:

$$P = \frac{F}{A} \quad (3.1.)$$

Equation 3.1. Determination of pressure, where P is the pressure in Pascal (Pa), F is the force in Newton (N), and A is the area to which the force is applied in square meter (m^2).

In this instance, the pressure measurement will be indicated in Pascal ($Pa = N.m^{-2}$) as determined by the International System of Units (SI), though in the medical setting pressure is usually measured in millimetres of mercury (mmHg). For example, blood pressure, where 1 mmHg is equal to 133.322 Pa (Liu *et al.*, 2005). When the force on the sensor is increased and the resistance decreases, the sensor measures the change in capacitance, which is then converted to pressure as seen in *Equation 3.1.* (<http://www.interlinkelectronics.com>).

Partsch *et al.* (2006) proposed a few specifications to which a pressure sensor should comply with when measuring the compression under CG (*Table 3.1.*).

Table 3.1. Specifications of a pressure sensor (Partsch *et al.*, 2006).

Specifications
Thin and flexible to conform to the contours of the body
The size of the sensitive area should be specific to the needs of the measurement
Able to be left in contact with the skin for extended periods of time
Able to measure continuously during active or passive movements
Should be easily calibrated for ease of use
Multiple measurement sites at the same time is preferable

Of these specifications, the thickness of the sensor is likely the most important. If the sensor is too thick, it will influence the pressure applied by the CG and therefore the pressure measured. This is due to the increase in circumference and is explained by the law of Laplace, whereby compression decreases as the circumference increases (Basford, 2002; Liu *et al.*, 2005). This is clearly evident in the simplified Laplace equation for cylinders (*Equation 3.2.*) as indicated by Basford (2002):

$$P = \frac{T}{r} \quad (3.2.)$$

Equation 3.2. Laplace equation, where P is the pressure (Pa), T is the wall tension ($\text{N}\cdot\text{m}^{-1}$) and r is the radius of the cylinder in meter (m).

Thus, the pressure at the specific measurement site will be underestimated if the circumference (i.e. radius of the cylinder) increases. Though this may be the case, Partsch *et al.* (2006) stated that especially on flat surfaces sensor thickness will not influence the accuracy of the measurement. It is suggested that the sensor should not be thicker than one millimetre (mm) and wider than 10 mm (Ferguson-Pell, Hagiwara & Bain, 2000). Ferguson-Pell, Hagiwara & Bain (2000) tested the “FlexiForce” sensor and revealed that smaller curvatures cause inaccuracies in the measurement. The researchers found that for a radius of curvature greater than 32 mm the measurement is accurate, as the problem below this radius of curvature is due to differences in the offset. In the case of most ankles, the radius of curvature is smaller than 32 mm and the output measurement will therefore be overestimated.

The characteristics of CG change during motion. This is due to the changes in the limb girths as the muscles contract (Stolk, Wege van der-Franken & Neumann, 2004). As the CG stretches during movement, sliding of the stitches over each other causes internal friction within the stitching to oppose the stretch of the CS. When the girth size then decreases the elastic properties of the CS causes it to pull together again. This action is also opposed by the internal friction forces between the individual threads of the CS. These forces cause the pressure changes during movement which is suggested to assist the calf muscle pump (Stolk, Wege van der-Franken & Neumann, 2004). Also, the increase and decrease in the radius of the cylinder (i.e. the muscle) as described above influences the compression applied to the limb by the CG or CS. Thus measuring the compression differences during movement can also give information on the elastic properties of the CS (Partsch & Mosti, 2010).

2. Measurement sites

The literature describes many different sites to measure the pressure applied by CG. These are usually dependent on the CG, thus the length of the garment and the goal of the measurement. Liu *et al.* (2005) measured the pressure applied by CG on the lower limb under four compression conditions, namely: light (10 – 14 mmHg and 12 – 16 mmHg), mild (18.4 – 21.2 mmHg and 18 – 25 mmHg), moderate (25.1 – 32.1 mmHg and 20 – 30 mmHg) and strong (36.4 – 46.5 mmHg and 30 – 40 mmHg) compression. The measurements were taken at the ankle, calf, knee and thigh on the posterior, anterior, lateral and medial side of the leg. The researchers found that in all compression conditions, the pressures were significantly lower on the lateral and medial side of the lower limb. It was also clear that the compression on the posterior side of the lower limb was higher than on the other sides (*Table 3.2.*).

Table 3.2. Compression data measured in mmHg. Measurements taken at the ankle, calf, knee and thigh on the anterior, lateral, posterior, and medial sides (Adapted from Liu *et al.*, 2005).

Measurement site	Compression level	Measurement site			
		Anterior (mmHg)	Lateral (mmHg)	Posterior (mmHg)	Medial (mmHg)
Ankle	Mild	14.79	10.01	13.48	7.11
	Strong	24.06	15.09	16.71	9.34
Calf	Mild	12.49	6.42	6.89	7.77
	Strong	22.02	11.81	12.80	13.75
Knee	Mild	7.88	6.57	5.12	5.8
	Strong	11.98	8.23	6.24	9.27
Thigh	Mild	5.40	2.95	3.86	3.58
	Strong	6.74	5.7	5.32	5.32

mmHg, millimetre of mercury

Although the measurement sites are strictly speaking up to the discretion of the researcher, there are recommended sites that may be used. These sites are described by anatomic location in the European document on normalization (Partsch *et al.*, 2006) and are summarized in *Table 3.3.* Of these measurement sites, it has been suggested that B1, the area where the Achilles tendon

changes into the calf muscle, is the most important and should be measured under all circumstances (Partsch, 2006). At this measurement site, the greatest change in circumference of the limb occurs during movement (Stolk, Wege van der-Franken & Neumann, 2004) and therefore the greatest changes in compression occurs at this point.

Table 3.3. Measurement sites for the use of compression garments as prescribed by the European document on normalization (Partsch *et al.*, 2006).

Site name	Site location
B	At the point of minimum girth on the ankle
B1	The area at which the Achilles tendon changes into the calf muscle
C	At the point of maximum calf girth
D	Just below the tibial tuberosity
E	Center of the patella and over the back of the knee
F	At the mid-thigh between the patella and groin (between K and E)
G	5cm below the center point of the crotch
H	At the point of greatest lateral trochanteric projection of the buttock
K	At the center point of the crotch

3. Graduated compression socks

Compression socks are mostly graduated, with the highest compression being distally situated and then decreasing in the proximal direction (Macrae, Cotter & Laing, 2011). The compression applied by the socks is determined by the composition of the materials, the weave and size of the CS as well as the circumference of the limb to which the sock is applied (Creasy, 2008; Liu & Little, 2009; Troynikov *et al.*, 2010; Macrae, Cotter & Laing, 2011).

Due to the variations in the curvatures of the lower limb, the compressive force applied varies over the surface of the limb (Troynikov *et al.*, 2010). Laplace's law states that as the circumference increases, the pressure exerted by the compression sock will decrease (Liu *et al.*, 2005). Troynikov *et al.* (2010) modified the Laplace formula to fit the medical practices in use and described it as follows:

$$P' = 2\pi T \times 133.3/C \quad (3.3.)$$

Equation 3.3. Modified Laplace equation where, P' is the exerted compressive force, T is the circumferential tensile force in the fabric of the CS and C is the circumference of the limb at the specific point (Troynikov *et al.*, 2010).

It is therefore difficult to account for the variations in anthropometry (i.e. calf and ankle circumferences) of different individuals with CS that are not custom made to fit the individual (Wertheim *et al.*, 1999). Liu *et al.* (2005) showed that the compression levels that are claimed by the CS manufacturers are not always the true compression of the CS. This may be partly due to the effect of the variation in anthropometry between individuals and that which is predicted by the manufacturers.

There is no consensus in the literature on what constitutes the optimal compression necessary to attain physiological benefits. Because of the soft and flexible nature of tissue, the application of external pressures may not only have positive effects, but may also negatively affect the venous system below the application site (Dai *et al.*, 2011). Pressure ranges that are too high might occlude the venous system. Lawrence and Kakkar (1980) suggested this tourniquet effect might exist at high compression levels. They found that 25 % of patients experienced a reduction in deep venous blood flow with high compression CG (30 – 12 mmHg) and they also suggested that this high compression may cause decreases in subcutaneous tissue blood flow.

Hafner *et al.* (2000) showed that pressures greater than 50 mmHg caused decreases in venous blood flow of the subcutaneous as well as in the muscle vascular system of healthy individuals. Ali, Creasy and Edge (2010) supported the work of Lawrence and Kakkar (1980) by suggesting that increasing the compression at the ankle to a level equal to or greater than 30 mmHg significantly decreases the deep venous blood flow as well as the subcutaneous blood flow. Partsch and Partsch (2005) determined that a pressure ranging between 50 to 60 mmHg is necessary for occlusion of veins in the sitting position and pressures of 70 mmHg in the standing position. These variations in pressures at which occlusion or decreased venous blood flow occurs may be due to

differences in the measurement protocols, measurement equipment and/or the populations used in different studies.

It is also not known whether these pressures are similar during exercise or in an elite athletic population. In an exercise study by Creasy (2008), subjects complained of pain and numbness of their feet after running on a treadmill with high compression CS (32 – 23 mmHg). It was suggested that blood flow to the feet was impeded due to a tourniquet effect. The researchers suggested that this tourniquet effect caused metabolites to accumulate in the foot and therefore caused pain and numbness.

Lawrence and Kakkar (1980) suggested that the optimal compression to induce increases in venous blood flow in patients with venous thrombosis embolism was 18 mmHg at the ankle, 14 mmHg at the calf, 10 mmHg at the mid lower thigh, and 8 mmHg at the upper thigh. Bochmann *et al.* (2005) also established while experimenting with pressures between 13 and 23 mmHg, that 20 mmHg is the optimal pressure to increase blood flow maximally (two fold) in the forearm during exercise and at rest. Whereas Liu *et al.* (2008a) stated that CG with a mild compression range of 18.4 - 21.1 mmHg has good venous blood flow outcomes in healthy female adults. Thus, there are not only discrepancies in what constitutes an optimal compression range, but it is also not known what the optimal range is to enhance sport performance. There is research indicating that wearing undersized lower body CG, therefore high compression CG have no performance benefit for athletes (Dascombe *et al.*, 2011b), although it was also not specifically claimed that higher compression is detrimental to performance.

For clinical use in patients with venous insufficiencies and other related diseases, CS are classified as follows: < 20 mmHg is classified as mild compression, 20 to < 40 mmHg as medium compression, 40 to < 60 mmHg as high or strong compression and \geq 60 mmHg as very strong compression (Partsch *et al.*, 2008; Keller *et al.*, 2009). The British guidelines for compression hosiery are: Class I (14 – 17 mmHg) light or mild support, Class II (18 – 24 mmHg) moderate support, and Class III (25 – 35 mmHg) strong support, whereas the European Guidelines are:

Class I (18.4 – 21.1 mmHg) light or mild support, Class II (18 -24 mmHg) moderate support, Class III (36.5 – 46.6 mmHg) strong support, and Class IV (\geq 59 mmHg) very strong support (Eagle, 2006).

4. Elastic tights

Some of the earliest forms of compression clothing in sport may be elastic tights. Though they may not possess the same pressure ranges as CG, they still exhibit compressive qualities. Research has indicated that the use of elastic tights is not as effective as CG. For instance, where CG were found to improve lactate kinetics after exercise, elastic tights had no effect (Creasy, 2008; Bringard, Perrey & Belluye, 2006). The former study also found that elastic tights had no effect on running economy or oxygen consumption and heart rate during cycling and post-exercise recovery (Creasy, 2008). Although Millet *et al.* (2006) found that elastic tights decreased muscle fatigue by decreasing the VO_2 slow component, thus suggesting faster oxygen delivery to the muscles, it is generally thought that elastic tights do not produce sufficient compression to achieve physiological benefits (Bringard, Perrey & Belluye, 2006). This may also suggest that CS with low compressive forces may not be sufficient for use by sportsmen.

E. EFFECT OF COMPRESSION SOCKS DURING EXERCISE

Compression garments have been found to enhance an array of physiological responses during exercise and performance. It is said to not only have physiological benefits, but also biomechanical, performance, and psychological effects.

1. Physiological effects

Within sport and exercise science, the major physiological variables that may influence performance are considered those within the muscular, cardiovascular and respiratory systems.

Some of these variables include: VO_2 , Heart rate (HR), lactate kinetics, muscle oxygenation, blood flow, muscle metabolism and muscle damage markers.

Cardiovascular function

Cardiovascular function is dictated by the cardiac output achieved by the heart. Cardiac output (CO) is the volume of blood (litres) pumped by the heart per minute. It is determined by the following equation:

$$CO = HR \times SV \quad (3.4)$$

Equation 3.4. Determination of cardiac output where, CO is cardiac output in litres per minute ($L \cdot min^{-1}$), HR is the heart rate in beats per minute ($beats \cdot min^{-1}$) and SV is the stroke volume in litres ($L \cdot beat^{-1}$).

During exercise, heart rate (HR) and stroke volume (SV) increase to deliver greater amounts of oxygenated blood to the active muscle. As the exercise intensity increases, HR increases proportionally to a maximal value, which is usually related to age, genetics and heart health, and cannot be trained. Thus with training there is seldom any significant change in maximal HR (*Figure 3.1.*), whereas SV is greatly enhanced by exercise training (*Figure 3.2.*). There are a few factors that influence the SV, namely end diastolic volume, ventricular contractility and total peripheral resistance. End diastolic volume is influenced by plasma volume, ventricular filling time and volume, and venous return. Except for total peripheral resistance, an increase in all these factors will increase SV (Rowell, 1974; Power & Howley, 2007).

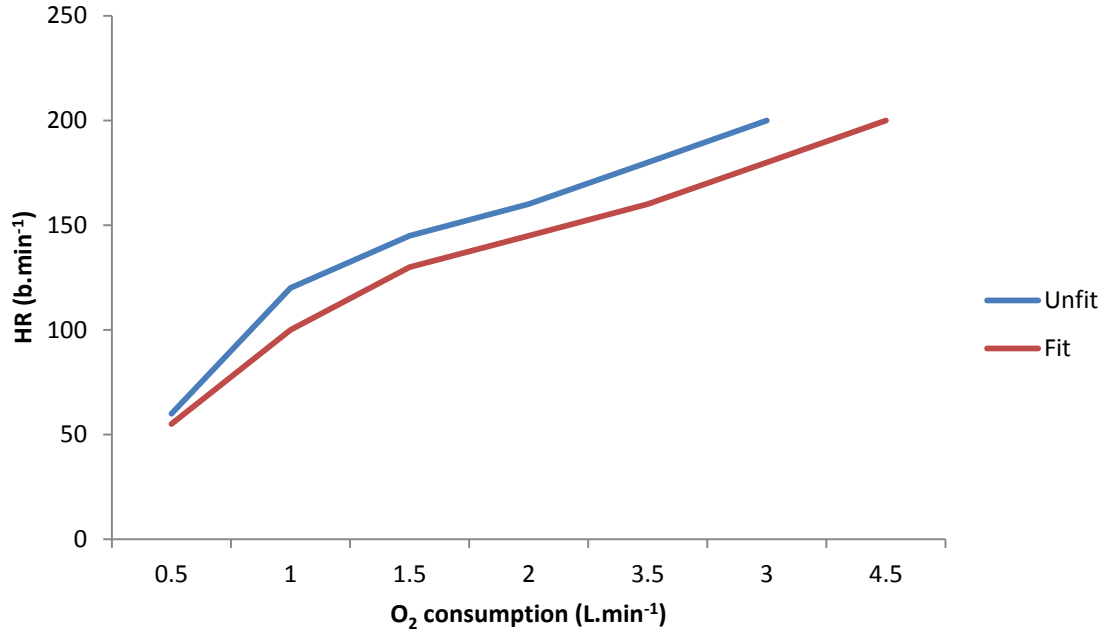


Figure 3.1. The difference in heart rate (HR) between trained and untrained individuals during exercise (adapted from McArdle, Katch and Katch, 2010).

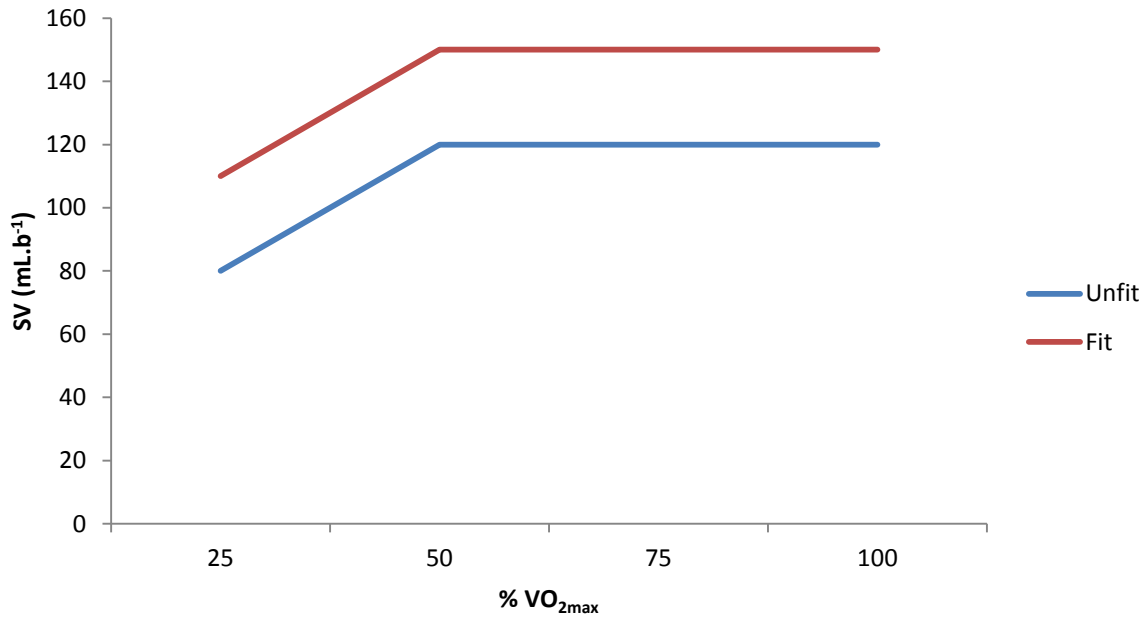


Figure 3.2. The difference in the stroke volume (SV) of a trained and untrained individual during exercise (adapted from Rowell, 1974 in Powers & Howley, 2007).

Various authors have studied the effects of compression socks on the blood flow away from the compressed area and also more specifically in the lower limbs. This is probably because CS were originally designed with the view to improve venous return and therefore SV. Researchers then suggested that this increase in SV will lead to a decrease in HR, specifically during exercise (Sear *et al.*, 2010).

Although this effect may be what is expected in theory, it is not what most researchers have found. During submaximal exercise with CS, most researchers have found no differences in the HR when compared with control conditions (Ali, Caine & Snow, 2007; Ali, Creasy & Edge, 2011; Sperlich *et al.*, 2011). Marks *et al.* (2011) found a significantly lower HR with the use of CS during submaximal exercise. However, one should recognize that this study was done at altitude with healthy college students, whereas the other studies involved well trained athletes completing 10 km running tests.

It has also been shown that CS have no effect on HR during intermittent sprint type exercise (Duffield & Portus, 2007; Duffield *et al.*, 2008; Higgins *et al.*, 2009), standing fatigue (Kraemer *et al.*, 2000), and exercise to maximal exertion (Rimaud *et al.*, 2010). Stenger *et al.* (2010) found increases in SV, CO and systolic blood pressure without a change in HR during a tilt test. On the other hand, Platts *et al.* (2009) found that CG reduces the increased HR seen post-spaceflight, as well as symptoms of hypovolemia during a tilt test. In light of these discrepant results, Ali, Creasy and Edge (2011) proposed that the skeletal muscle pump of athletes is too well developed and therefore CS will have no further effect on venous return and HR.

MacRae *et al.* (2012) found that CG caused significantly greater CO during exercise which was accompanied by higher HR, although this HR difference was not found to be statistically significant. Strangely enough, SV was found to be unaffected by the CG, therefore, in this case the increased CO could be caused by an increase in HR rather than an increase in SV as has been previously suggested.

One of the problems with the current literature is that different exercise modalities, exercise intensities and study populations are used, and it is therefore difficult to determine whether the lack

of an effect should be attributed to the methods employed or whether there is truly no effect of CS on HR. Collectively, however, it seems that CG may improve cardiovascular function through an increase in SV, but without a concomitant decrease in HR.

Venous blood flow

Venous blood flow does not only influence the cardiovascular variables such as SV and HR, but it is hypothesised that it also enhances the removal of metabolic end products, such as lactate. Although venous return is not always measured directly, but is rather estimated using HR, there is research indicating that CS increase venous blood flow from the compressed area. For instance, venous return was increased with the use of compression bandages (Stanton *et al.*, 1949), in healthy individuals (Millet *et al.*, 2006), as a preventative measure against post-operative venous thromboembolism in hospital patients (Lawrence & Kakkar, 1980), as well as during exercise in well trained endurance runners ($VO_{2max} = 59.0 \pm 6.7 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) (Dascombe *et al.*, 2011b). Furthermore, indirect evidence of increased venous return was found in the form of narrowing of the veins (Kraemer *et al.*, 2000).

The effect of CG on the velocity of blood flow in the venous system has also been investigated. This research has shown enhancement in the venous velocity with the use of CG (Liu *et al.*, 2006; Dascombe *et al.*, 2011b), as well as increased deep venous blood flow (Berry & McMurray, 1987; Brown & Brown, 1995; Chatard *et al.*, 2004).

These improvements in venous blood flow are said to decrease blood pooling in the lower extremities (Berry & McMurray, 1987; Kraemer *et al.*, 2000; Millet *et al.*, 2006; Ali, Caine & Snow, 2007; Stenger *et al.*, 2010), as well as improve removal of metabolic products from the muscle (Creasy, 2008; Scanlan *et al.*, 2008). Blood pooling is further decreased through the enhancement of the skeletal muscle pump. Due to the veins having unidirectional valves to prevent the backflow of blood due to gravity, compression of the veins by the skeletal muscles causes blood to be pushed upwards through these valves (Sperlich *et al.*, 2011). With the application of external

compression, the effects of the skeletal muscle pump is said to be enhanced. Dascombe *et al.* (2011b) suggested that the better functioning of the skeletal muscle pump could be explained by clinical findings, such as enhanced valvular cusp function, or decreases in cross-sectional area of the veins.

Though the decrease in blood pooling due to the increased efficiency of the skeletal muscle pump was observed in several research studies, there are some researchers that hypothesize that the skeletal muscle pump, and more specifically the calf muscle pump of athletes, is too well developed and that compression will have no further effect (Creasy, 2008; Ali, Creasy & Edge, 2011). If true, it may explain the lack of performance improvements with CG despite enhanced venous blood flow, as shown by Dascombe *et al.* (2011b).

Blood lactate concentration

One of the possible advantages with the use of CG during exercise is the improvement in the usage of lactate as a fuel source. Lactate is a metabolic end product that is produced within the muscle fibres by the conversion of pyruvic acid to lactic acid (Robergs, 2001). Lactic acid is almost immediately converted to lactate at physiological pH. The lactate is then removed either by oxidation within the muscle mitochondria, converted to pyruvate through the Cori cycle, or gluconeogenesis. It has been found that during exercise approximately 70 % of lactate is removed via oxidation (Thomas *et al.*, 2004).

The effects of CG on lactate kinetics have been studied during exercise as well as during recovery after exercise. The reason being, that if the lactate kinetics is improved during exercise, the athlete will be able to perform at higher intensities and this could relate to performance improvements. Also, if the lactate removal post-exercise is improved, the athlete will recover sooner from exercise, and will therefore be able to perform better during subsequent training sessions or competitions.

Earlier research has shown that active recovery is the most effective means of post-exercise recovery, as it elevates the flow of blood through the muscles, thereby increasing the removal of

by-products of metabolism (Rimaud *et al.*, 2010). Though this may be true, active recovery still makes use of metabolic energy. Therefore, if the same recovery effect can be achieved without work being done, as with CG, the athlete will not have to use energy to recover.

Most studies reported no differences in the lactate concentration at the end of exercise bouts with and without CS (Duffield *et al.*, 2008; Ali, Creasy & Edge, 2010; Ali, Creasy & Edge, 2011). Duffield *et al.* (2008), in simulating team game exercise in under 21 club level rugby players found that CG had no effect on the lactate concentration measured between high intensity training sessions as well as 15 minutes post exercise. They took this to show that CG did not improve metabolic recovery.

With regard to endurance trained athletes, there are also studies indicating no effect of CS on lactate concentration. Ali, Creasy and Edge (2010) studied runners and triathletes ($VO_{2max} = 70.4 \pm 6.1 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) with high (32 – 22 mmHg) and low (15 – 12 mmHg) compression CS. These athletes completed 40 minutes of treadmill running at 90 % of their best 10 km time at a 1 % gradient. No differences were found in [BLa] during the running protocol, but unfortunately lactate recovery was not monitored.

Bakken *et al.* (2011) studied five men ($VO_{2max} = 58.4 \pm 7.2 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) and six women ($VO_{2max} = 51.3 \pm 4.3 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) during 10 km runs at 85 % of the HR at lactate threshold. They also found no difference in [BLa] directly post-exercise, but also did not monitor lactate recovery. Similarly, Ali, Creasy and Edge (2011) also measured pre and post [BLa] in trained men and women ($VO_{2max} = 68.7 \pm 5.8 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) during a 10 km time trial and found no effect of CS. Rimaud *et al.* (2007), however, found lower [BLa] after maximal exercise in participants with spinal cord injuries at the sixth thoracic vertebra. Although it seems that CS do not influence lactate production, there is no direct evidence suggesting this and the necessity for lactate measurement on cellular level exists to investigate this suggestion.

Berry and McMurray (1987) conducted two experiments. The first of these was an exercise test to exhaustion completed by well trained, male, college students ($VO_{2max} = 52.8 \pm 8.0 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$).

When comparing the CS and no CS conditions it was found that [Bla] was lower during recovery in the CS condition than in the no CS condition, but the difference was only statistically significant at 15 minutes post exercise. They then tested healthy male college students ($VO_{2max} = 59.9 \pm 6.8$ mL.kg⁻¹.min⁻¹) for three minutes at 110 % VO_{2max} with three CS conditions. The first being without CS, second was exercising with CS but taking it off during recovery and the third was exercise with CS and keeping it on during recovery. *Table 3.4.* shows the lactate results obtained from this experiment.

Table 3.4. Blood lactate concentration ([Bla]) (mMol) of total circulating lactate adjusted for plasma volume shifts (adapted from Berry & McMurray, 1987)

Minutes post exercise	5	10	15
No CS	69.7	57.6	33.3
CS	59.3	49.7	32.1
CS exercise only	75.0	62.4	35.5

CS, compression socks

These combined findings suggest that CS do not have an influence on lactate production as stated earlier. These results may suggest that the use of CS caused retention of the lactate within the muscle itself, as the lactate concentration was the highest when the CS were removed after exercise. In line with these findings, Rimaud *et al.* (2010) found significantly higher [BLa] after maximal cycling exercise to exhaustion in healthy men wearing reverse pressure gradient CS when compared to control (without socks) conditions. These reverse pressures have increasing pressure in the proximal direction, other than traditional graduated CS which have decreasing pressure in the proximal direction. Reverse pressure gradient socks were used to supply the greatest pressure on the area of the leg where it is said to have its effect. The sock conditions were maintained throughout both exercise and recovery. Lactate recovery was also monitored for 60 minutes post-exercise, but no significant effect of CS was found on lactate kinetics. The researchers suggested that the net lactate release rate was higher in the compression condition.

Chatard *et al.* (2004), on the other hand, studied older trained cyclists (Age = 63 ± 3 years; $VO_{2max} = 49 \pm 6 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$). Each person completed four testing sessions. The sessions comprised of two five minute maximal cycle ergometer tests at a specific brake force and cadence, with 80 minutes recovery between the two sessions. The four tests were spread over two weeks with a two day interval between sessions in week one and two separately. After the completion of the first test, the participants completed the recovery period with a randomly assigned, CG or without CG. After removal of the CG it was found that there was no difference in [Bla] directly after exercise bouts, thus the level from which recovery took place was the same. They found that lactate was removed more effectively with the CG condition. This could indicate that the lactate is held within the muscle during the recovery period, or that lactate removal is enhanced due to improved blood flow. In the case of lactate being retained in the muscle, this can be explained as follows. The lactate that is measured in the blood after exercise is removed as in the case of the no compression condition, but no further lactate enters the blood circulation from the muscles as was the case with the no compression condition.

In contrast, Marks *et al.* (2011) found a decrease in [BLa] after exercise compared to the control condition at altitude (1219.2 – 2438.4 m). This study was done on healthy college students wearing athletic grade CS (experimental group) or hiking socks producing no compression (control group). On the 12th day at altitude the [BLa], after a five minute step test, was significantly lower in the experimental group ($2.7 \pm 1.3 \text{ mmol}\cdot\text{L}^{-1}$) compared to the control group ($5.2 \pm 2.1 \text{ mmol}\cdot\text{L}^{-1}$). Three possible mechanisms for this occurrence are: 1) decreased lactate production with CS, 2) increased cellular retention of lactate due to the compression, or 3) increased lactate removal with CS due to increased venous return (Berry & McMurray, 1987).

The difference in post-exercise lactate kinetics with CG has been attributed to greater oxidation of the lactate within the muscle itself, or by flushing the lactate away from the muscle towards other tissues that remove lactate (Scanlan *et al.*, 2008) such as cardiac muscle, the kidney cortex and the liver (Billat *et al.*, 2003; Hubbard, 1972; Baldwin, Hooker & Herrick, 1978).

It is therefore still not known whether CG improve lactate kinetics, and by what mechanisms this possible improvement is brought about.

Muscle oxygenation

Lewis *et al.* (1976) were the first to suggest that the tourniquet effect could be a mechanism whereby lactate can be retained within the muscle. Later, Berry and McMurray (1987) attributed their findings of lower [BLa] during exercise with CS to the lactate being retained within the muscle. Should this be true, and should CS also improve muscle oxygenation, then lactate may actually be removed via oxidation within the muscle mitochondria (Brooks, 2002) and thus improve post exercise recovery time (Bringard *et al.*, 2006). This may suggest that the hypothesis that lactate is removed through increases in venous blood flow (i.e. flushing the muscles) may be partially incorrect.

Bringard *et al.* (2006) tested 12 experienced male runners. Muscle oxygenation was measured under three conditions namely: CG, elastic tights, and no compression. Baseline measurements were taken for five minutes prior to each measurement condition in the supine position. Participants then completed five minutes of lying down, sitting and then standing. They found that resting oxygenation of the medial Gastrocnemius was significantly increased with CG compared with control conditions for both the standing and supine positions. They speculated that these increases in muscle oxygenation could be due to: elevated blood flow to the muscle, thus increasing the volume of oxygen available to the muscle, more efficient utilization of oxygen, changes in skin blood flow, as well as increases in SV and CO through an increase in venous return. Similarly Ménétrier *et al.* (2011) found increased tissue saturation with the use of CS during rest before exercise, as well as during the recovery period after the exercise. This would suggest that CS have no influence on muscle oxygenation during resting conditions.

Subsequent to the study above, higher muscle oxygenation rates during exercise with CS have been found in various studies. Sear *et al.* (2010) found that the tissue oxygenation index (TOI) was

higher with CG directly after prolonged high intensity intermittent exercise when wearing whole body CG compared to the control condition (soccer kit). Marks *et al.* (2011) studied the effect of CG on muscle oxygenation at moderate altitude in seven men and nine women. They found improvements in muscle oxygenation during the step test in the CS condition, compared to control condition (placebo socks) although these improvements were not found to be statistically significant.

Dascombe *et al.* (2011b) found that high pressure CG (under-sized garments) improved muscle TOI during 8 km.h⁻¹ running exercise compared to low/ moderate pressure (regular sized garments) and control conditions. With an increase in running speed (12 km.h⁻¹) both the compression conditions improved the TOI measurements. This improvement in TOI was accompanied by an increase in total haemoglobin index (nTHI) and a decrease in HR, which Dascombe *et al.* (2011b) suggested could imply increased venous flow and cardiac return, which could be due to increased skeletal muscle pump function. They further suggested that oxygenation could also be enhanced through improved vascular cusp function and/or a reduction in venous cross-sectional area, and thus improved venous blood flow, as previously suggested in clinical studies.

However, there are a few studies that report no changes in muscle oxygenation with CG. Scanlan *et al.* (2008) tested well trained competitive male cyclists ($VO_{2max} = 55.2 \pm 6.8 \text{ mL.kg}^{-1}.\text{min}^{-1}$) during two incremental tests to exhaustion and two one hour time trial tests, with and without CG. Muscle oxygenation of the Vastus lateralis was measured during the exercise tests. Mean muscle oxygenation did not significantly differ between CG or control (normal underwear) condition in either the incremental or one hour time trial tests. In this study muscle oxygenation economy (MOE) was used to determine the percentage of O₂ utilized by the muscle. During the one hour time trial they found no difference between the control and experimental conditions, at any of the 15 minute intervals, although there was a possible practical significant difference (Effect size = 0.6) in the mean MOE values (control = $5.1 \pm 1.2 \text{ W.}\%^{-1}$, CS = $5.8 \pm 1.9 \text{ W.}\%^{-1}$). This suggests greater muscle oxygenation economy rather than increased oxygen utilization. The researchers stated that

these results, along with the similar power outputs achieved during these tests, could suggest that the muscle was more efficient at using oxygen during the exercise when wearing the CG.

Sperlich *et al.* (2011) studied well trained endurance runners ($VO_{2max} = 63.7 \pm 4.9 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) during 45 min treadmill running at 70 % of VO_{2max} . The athletes were tested with five different pressure gradient CS with the following compressions at the area of maximum girth around the calf muscle: 0, 10, 20, 30, and 40 mmHg. No differences in muscle oxygenation were found among the various compression conditions. The fact that there was no improvement in muscle oxygenation with increasing pressures may be an indication that there was no increase in oxygen delivery to the muscle and therefore could indicate that venous return is not improved by CS.

Wahl *et al.* (2011) investigated the effect of external compression applied to exercising muscle on the properties of red blood cells. They tested healthy non-smoking, well trained runners ($VO_{2max} = 57.7 \pm 4.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$). Participants completed an incremental test to exhaustion to determine VO_{2max} . This was used to determine the speed during the next four tests. Each of the next four tests were completed at 70 % VO_{2max} for 30 minutes with different compression conditions (10, 20, 30, and 40 mmHg pressures at the calf), where after time to exhaustion was determined with a ramp protocol. Blood samples were taken from the earlobe and the ektacytometric principle was used to determine red blood cell deformability. The movement of red blood cells through the capillary system is influenced by the diameter of the capillaries, and thus if the compression applied decreases the capillary diameter to a certain extent, the red blood cells will not be able to move through. This may influence oxygen delivery to the active muscles by influencing the transit time of the red blood cells. They found an increase in deformability of the red blood cells during exercise, however, this effect was diminished with an increase in compression gradient. From these findings it can be concluded that the deformability of the red blood cells is not affected by compression to such an extent that oxygen delivery to the active muscles is hampered.

2. Performance effects

Endurance performance

Most sport scientists agree that endurance running performance is mainly determined by the aerobic power (VO_2) at the anaerobic threshold, running speed at VO_{2max} and running economy (Creasy, 2008). Although Tanaka *et al.* (1986) found that VO_2 at the anaerobic threshold had the best correlation to performance. Should CG improve any of these variables, it could be considered an ergogenic aid. Though there are many anecdotal claims that CG improve sport performance, there is actually very limited data to support this notion. In fact, Creasy (2008) claimed that no published research has shown improvements in performance with the use of CG.

Since then, Kemmler *et al.* (2009) has shown improved endurance running performance in moderately trained men ($VO_{2max} = 52.0 \pm 6.1 \text{ mL.kg}^{-1}.\text{min}^{-1}$). They found increases in time to exhaustion during an incremental test with CS, although VO_{2max} was not significantly improved. They also found that running speed at the anaerobic (CS = $14.11 \pm 1.13 \text{ km.h}^{-1}$ vs. control = $13.90 \pm 1.13 \text{ km.h}^{-1}$) and aerobic threshold (CS = $13.02 \pm 1.10 \text{ km.h}^{-1}$ vs. control = $12.74 \pm 1.04 \text{ km.h}^{-1}$) was significantly higher with CS.

On the other hand, Ali, Creasy and Edge (2011) found no effect of four different levels of CS on endurance performance, determined by measuring these variables during a 40 min treadmill run, in well-trained runners ($VO_{2max} = 70.4 \pm 6.1 \text{ mL.kg}^{-1}.\text{min}^{-1}$), which may suggest that the training status of participants could influence the responses. These findings were consistent with those of Dascombe *et al.* (2011b) who also showed that increased compression did not influence the endurance running performance of well-trained men ($VO_{2max} = 59.0 \pm 6.7 \text{ mL.kg}^{-1}.\text{min}^{-1}$). From these studies it is thus unclear whether CS or CG can be considered an ergogenic aid, although CS could have an indirect performance benefit by improving recovery and therefore improving the quality of the subsequent training sessions, or by maintaining performance such as seen in Ali, Creasy and Edge (2011).

Maximal oxygen uptake

For endurance sports, maximal oxygen uptake (VO_{2max}) is a common predictor of performance, although it is not the only factor influencing and therefore determining performance outcomes. VO_{2max} is determined through an incremental exercise test to exhaustion during which time the oxygen uptake and utilisation is measured by breath analysis. VO_{2max} is the point at which the oxygen uptake (VO_2) “levels off” whilst the workload still increases (Saltin & Åstrand, 1967), although VO_2 often does not level off. The criteria used to determine whether a true VO_{2max} has been achieved are as follows: 1) a plateau in the VO_2 with a further increase in exercise intensity, 2) lactate concentrations above 8mM, 3) respiratory exchange ratio above 1.15, and 4) reaching within 10 $b \cdot min^{-1}$ of the age predicted heart rate maximum (Howley, Bassett & Welch, 1995). It is generally accepted that VO_2 is directly related to work rate when using an incremental exercise protocol (Storfer, Davis & Caiozzo, 1989). Therefore, if an athlete has a high VO_{2max} he will be able to exercise at a higher work rate, and therefore at a greater speed which will lead to success in endurance performance.

An individual's VO_{2max} is the product of the CO and arterio-venous oxygen difference ($a-vO_2$ difference). Thus, if CS increases the CO by increasing venous return and SV, it is hypothesized that CS must enhance VO_{2max} and therefore endurance capacity.

Table 3.5. Summary of research on VO_{2max} .

Author	Test type	Statistics	Training status	VO_{2max} (mL.kg ⁻¹ .min ⁻¹)
Berry and McMurray (1989)	Treadmill	NSSD	Trained	CS = 58.1 ± 4.9, Control = 56.9 ± 6.4
Rimaud <i>et al.</i> (2007)	Wheelchair	NSSD	8 well trained, 6 trained	CS = 21.9 ± 7.5, Control = 23.5 ± 7.6
Scanlan <i>et al.</i> (2008)	Cycle	NSSD	Well trained	CG = 53.5 ± 6.5, Control = 55.2 ± 6.8
Kemmler <i>et al.</i> (2009)	Treadmill	NSSD	Moderately trained	CS = 53.3 ± 5.8, Control = 52.2 ± 6.2
Rimaud <i>et al.</i> (2010)	Cycle	NSSD	Trained	CS = 54.3 ± 2.7 Control = 53.3 ± 2.7
Dascombe <i>et al.</i> (2011b)	Treadmill	NSSD	Well trained	Undersized CG = 59.9 ± 6.3, regular sized CG = 60.6 ± 6.6, Control = 59.0 ± 6.7

NSSD, no statistically significant difference; CS, compression socks; CG, compression garments

This indicates that the effect that CG have on venous blood flow (assuming that it does), does not translate into enhanced VO_{2max} performance. On the one hand this may indicate that the improved venous blood flow does not cause an increase in CO, which may be reflected in the studies that have shown no effect of CG on HR during exercise (see section E.1.). On the other hand it is also possible that there may be an improvement in CO, but that this increase is nullified by a decrease in oxygen extraction brought about by the CG; thus the muscles could be less efficient at extracting oxygen when compression is applied. At this point it is not clear what the mechanism is and further research has to be done in this field.

Economy of movement

Movement economy is defined as the amount of work needed to perform a movement at a specific intensity (Bakken, 2011). A more efficient athlete will use less oxygen than a less efficient athlete at the same exercise intensity (Saunders *et al.*, 2004). Furthermore, if two athletes have similar VO_{2max} values, the athlete with the better movement economy will have superior performance due to the efficiency of the movement. Due to the homogeneity in VO_{2max} of trained endurance athletes,

movement economy is considered a better predictor of performance than VO_{2max} (Saunders *et al.*, 2004). Movement economy and more specifically running economy is influenced by physiological, biomechanical, training, environmental, and anthropometrical variables (*Figure 3.3*). Consequently it is not easy to explain the physiological mechanisms behind any changes in movement economy.

Bringard, Perry and Belluye (2006) found that running economy in trained runners ($VO_{2max} = 60.94 \pm 4.4 \text{ mL.kg}^{-1}.\text{min}^{-1}$), was improved with CS at 10, 12 and 14 km.h^{-1} , although this trend was not seen at 16 km.h^{-1} . Researchers have suggested various mechanisms whereby movement economy can be improved with CS. Ali, Creasy and Edge (2011) attributed the improvement to more economical motor recruitment patterns, greater physical support of the contracting muscle, as well as improvements in oxygen delivery due to increases in venous return. Others have also stated that the supportive effect of CS on the skeletal muscle pump may have a positive effect on energy expenditure (Higging, Naughton & Burgess, 2009). Bringard, Perry and Belluye (2006), on the other hand, attributed it to improvements in proprioception, muscle contraction and propulsive forces. By improving proprioception it is possible that muscle recruitment patterns could become more efficient, and therefore cause more economical movement.

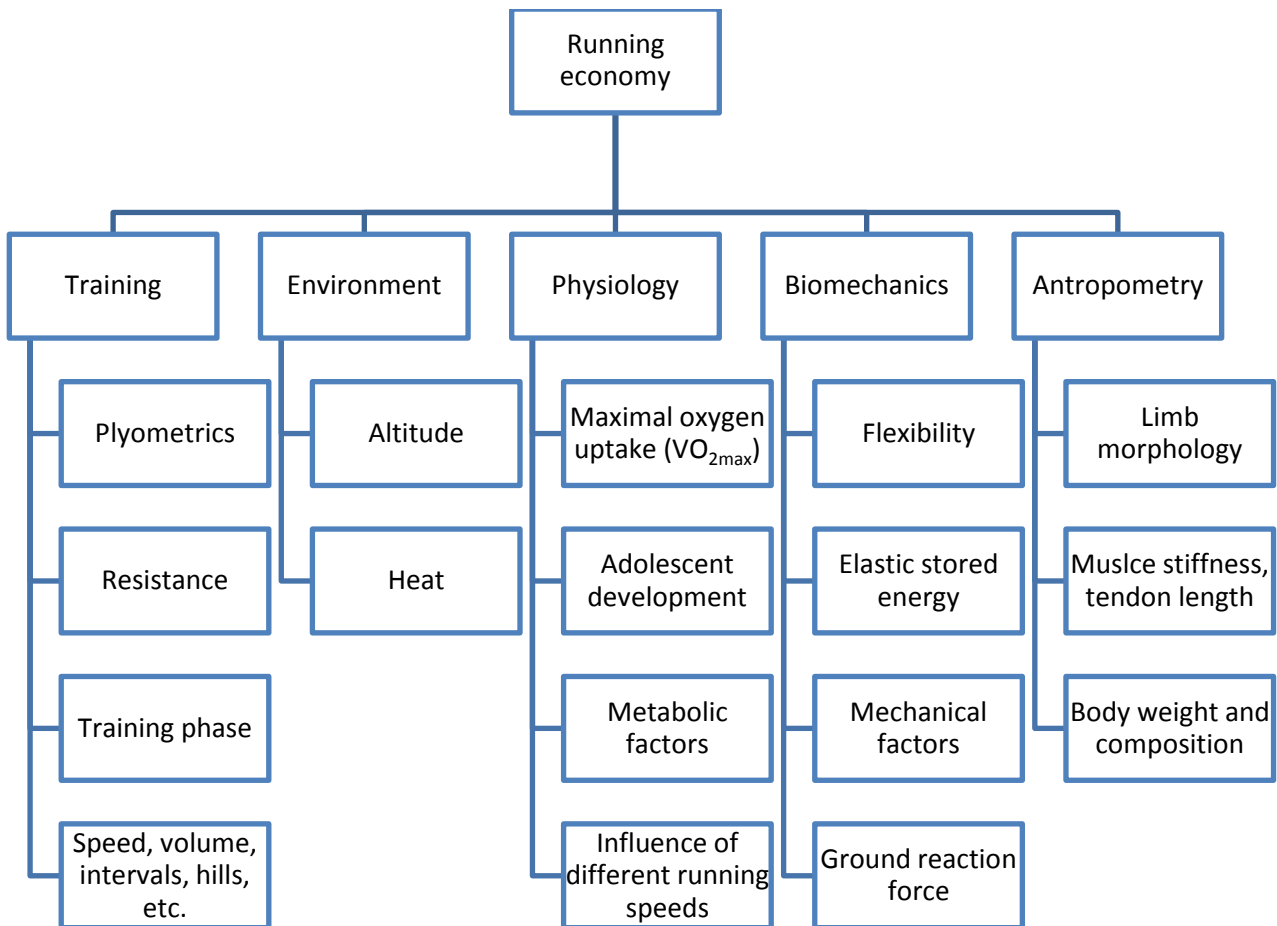


Figure 3.3. Factors affecting running economy (with permission from Saunders *et al.*, 2004).

In a recent study by Varela-Sanz *et al.* (2011), no statistically significant difference was found in running economy between two groups of trained endurance runners (Age = 35 ± 6 years; $VO_{2max} = 62.8 \pm 9.0 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$). Information on the kinematics of running was also determined between the two groups during a time limiting running protocol. This protocol requires that the participant runs on the treadmill at a speed of 105 % of their recent 10 km run time, at a 1 % incline until exhaustion. It was found that CS also did not influence the kinematic variables, further substantiating the results of this study that CS do not have an influence on running economy. Similarly, De Glanville and Hamlin (2012) found that CS did not influence oxygen consumption during a 40 km cycling time trial in multisport athletes (Age = 34 ± 7 years).

Anaerobic threshold

Svedahl and MacIntosh (2003: 300-301) defined the anaerobic threshold as:

“An intensity of exercise, involving a large muscle mass, above which measurement of oxygen uptake cannot account for all the required energy.”

This is evident in the fact that if the energy required for the work to be done is greater than can be obtained from oxygen dependent metabolic reactions, the oxygen uptake for that intensity will be lower than expected, as the energy requirements will be met by oxygen independent metabolic reactions (Wasserman *et al.*, 1973).

Scanlan *et al.* (2008) showed that lower body CG increased power output at the anaerobic threshold in well trained male cyclists ($VO_{2max} = 55.2 \pm 6.8 \text{ mL.kg}^{-1}.\text{min}^{-1}$), therefore, improved the anaerobic threshold. It was suggested that these improvements were due to the circulatory improvements associated with the use of CG leading to greater oxidation of lactate outside the working muscle. As suggested by Kemmler *et al.* (2009) the lactate and other metabolic waste products could also be retained within the muscle leading to improvements in the anaerobic threshold.

Power

As fatigue sets in during competition or training, it influences the ability of the athlete to maintain the work rate or power output as in the beginning of the exercise bout. This not only hampers the performance of endurance athletes but also that of team sport athletes. Thus the maintenance of power output throughout the duration of the competition is an important variable influencing performance.

In research, jump height post-exercise is used to determine the amount of power retained after an exercise bout (Ali, Creasy & Edge, 2011), which can be compared to that achieved pre-exercise.

Ali, Creasy and Edge (2011) showed that CS (pre = 33.6 ± 8.2 cm, post = 33.1 ± 8.4 cm) positively affected jump height post-exercise compared to control (pre = 33.8 ± 9.2 cm, post = 30.9 ± 9.7 cm) conditions. Whether this benefit was transferred to improved performance at the end of an endurance bout is speculative as running speed was not measured.

Kraemer *et al.* (1996) also showed that the power output was better maintained during a maximal repeated jumping fatigue test with compression shorts. They suggested earlier that CG could maintain power output by altering the range of motion of the limbs (Kraemer *et al.*, 1996). Though this may be true, it is not consistent with the findings of Duffield *et al.* (2008). They tested club rugby players during an 80 minute simulated team game and found no effect with lower body CG at three different time intervals. These intervals were at time points when fatigue would be expected, thus before and after half time, as well as before full time. In this study, the participants wore lower body CG, which covers the lower body from the ankles to the hips. It is thus possible that these specific garments may have hindered the biomechanics of the subjects during sprinting, and therefore nullifying the beneficial effect that CG may have had on power maintenance.

Sprint performance

Duffield *et al.* (2008) studied the effect of CG on the speed and power produced during repeated 20 m sprints while wearing CG, as well as performance after 24 h of recovery time in club rugby players. They found no influence of CG on any of these variables.

Due to the compression of the CG, there may be an influence on the biomechanics of running when wearing these garments. However, the literature reports conflicting results in this regard. Doan *et al.* (2003) found no influence of CG on the kinematics of sprinting. They studied the hip and knee range of motion (ROM) in nationally competitive university track athletes. On the other hand, Bernhardt and Anderson (2005) found that compression shorts significantly decreased hip flexion ROM (no compression = 98.25 ± 8.86 °, compression = 88.50 ± 8.80 °).

3. Post-exercise recovery

Metabolic end product removal

Increased metabolic activity during exercise causes the build-up of metabolic end products within the muscle. The removal of these end products is part of the post-exercise recovery process. When exercise is stopped, the main stimulus causing increased blood flow, namely the skeletal muscle pump, is removed (Carter *et al.*, 1999). Thus increasing venous return for instance, through active recovery, is partly responsible for the removal of metabolic end products (Berry & McMurray, 1987).

It has thus been proposed that the effect of CS on venous return may facilitate the removal of end products (Jakeman, Byrne & Eston, 2010). Scanlan *et al.* (2008) tested well trained male cyclists ($VO_{2max} = 55.2 \pm 60.8 \text{ ml.kg}^{-1}.\text{min}^{-1}$) and reported improved [BLa] removal with the use of lower body CG. They suggested that CG improve the removal of metabolic end products by increasing the transport of these products to the non-working muscles.

Delayed onset of muscle soreness

Delayed onset of muscle soreness (DOMS) refers to the pain or discomfort that athletes experience between 24 - 72 h post-exercise, usually with unaccustomed exercise. There are at least six theories proposed to explain the occurrence of DOMS, namely the lactic acid, muscle spasm, connective tissue, muscle damage, inflammation, and the enzyme efflux theories (Cheung, Hume & Maxwell, 2003).

The lactic acid theory dictates that the increased production of lactic acid is continued post-exercise, which stimulates pain receptors, whereas the muscle spasm theory states that the increase in resting muscle activity after DOMS-inducing exercise indicates a tonic spasm in the affected muscle causing the build-up of pain inducing substances due to vasoconstriction caused by the pressure of the muscle on the venous system.

Damage to the connective tissue surrounding the muscle fibres is the essence of the connective tissue damage theory. This damage is said to be caused by stretch and excessive strain, therefore specifically related to eccentric type exercises. Whereas the muscle damage theory states that the damage to the z-lines of muscle fibres causes DOMS. This is visible by microscopic lesions in the muscle histology after DOMS-inducing exercise.

With DOMS-inducing exercise, such as eccentric contractions, there is increased oedema formation and inflammatory marker infiltration in the affected muscle. The inflammation theory therefore states that this build-up and coinciding increase in monocytes and neutrophils cause an osmotic pressure which leads to oedema formation and the stimulation of pain receptors.

The enzyme efflux theory states that the build-up of calcium due to sarcolemmal damage causes a decrease in adenosine triphosphate (ATP) regeneration due to the inhibition of cellular respiration. This weakens the active transport of calcium to the sarcoplasmic reticulum. The increase in calcium also causes further damage to the z-lines of the muscle fibres which causes stimulation of the free nerve endings and thus causing the pain sensations (Cheung, Hume & Maxwell, 2003).

Research has shown that it may not be one individual theory that explains the onset of DOMS, but rather a combination of theories (Cheung, Hume & Maxwell, 2003). Since there is no clarity on the exact mechanism(s) of DOMS, it is difficult to determine by which mechanism CS alleviate DOMS and post-exercise muscle soreness. The lactic acid theory cannot explain DOMS, as lactic acid gets converted to lactate at physiological pH so quickly that it could almost be said that lactic acid does not exist. Further, this theory has been rejected as [BLa] levels return to resting levels within an hour following exercise (Cheung, Hume & Maxwell, 2003).

Nevertheless, a number of mechanisms have been proposed by which CS may alleviate DOMS. These mechanisms all relate to the different theories whereby DOMS is induced. One of these theories is that CS or CG provide mechanical support to the muscle(s) during exercise. Thus it is proposed that the compression exerted by CG keeps the muscle(s) together and reduces the oscillations experienced within the muscle(s) during exercise, especially during weight bearing

exercise such as running (Kraemer *et al.*, 2001a; Kraemer *et al.*, 2001b). In support of this theory, Doan *et al.* (2003) examined the effect of compression shorts on muscle oscillations during vertical jump landings and found that there were significantly less lateral and anterior-posterior movements in the thigh muscles during these vertical jump landings when wearing CG.

According to the muscle damage theory of DOMS, it is the disruption of the contractile components of the muscle, more specifically the z-lines, that causes DOMS (Cheung, Hume & Maxwell, 2003). This disruption is caused by the mechanical trauma within the muscle due to the oscillation of the muscle. Thus if CS or CG provides sufficient mechanical support, it could also minimize muscle fibre damage during exercise.

Another proposed mechanism to decrease DOMS is the effect of CS on inflammation and swelling (Jakeman, Byrne & Eston, 2010). According to the inflammation theory of DOMS, the pain is the result of the formation of oedema and the infiltration of inflammatory cells such as monocytes and neutrophils into the injured site (Cheung, Hume & Maxwell, 2003), although the oedema is the main reason for the pain experienced (Creasy, 2008). Kraemer *et al.* (2000) showed that CS reduced swelling in healthy young women during a standing fatigue test. This study was done after previous research has shown reduced swelling in patients with clinical symptoms of insufficient venous function. These results thus showed that the effect of CS was not only evident in patients but also in the healthy population.

Similar effects were found when researchers tested the effectiveness of CG to alleviate post-spaceflight symptoms. Stenger *et al.* (2010) found that the use of CG decreased the amount of venous pooling and therefore swelling after spaceflight. Kraemer *et al.* (2010), by means of ultrasound investigation, found a significantly decreased amount of swelling after heavy resistance exercise in men and women after 24 hours of recovery in the Vastus lateralis muscle.

Muscle oscillations

During running, the impact upon foot strike causes a shock wave to move through the body, resulting in muscle oscillations of the leg. These oscillations may also cause friction between muscle fibres causing damage to these fibres. The frequency of the shock wave moving through the leg is similar to the natural frequency of soft tissue such as muscles, and could therefore cause resonance within the muscle leading to damage of the tissue (Nigg & Wakeling, 2001). Subsequent research has shown that adaptations by the central nervous system cause muscles to adapt to repetitive foot strikes, a process called muscle tuning, which prepares for the shock wave before impact, thereby dampening its effects (Nigg & Wakeling, 2001). Nevertheless, one can expect that these shock waves would still have a significant effect during prolonged exercise especially when the muscle fatigues.

Doan *et al.* (2003) found decreases in the amount of muscle oscillations experienced in the thigh muscles of both men and women with the use of compression shorts during counter movement jumps. This decrease was in the longitudinal as well as the anterior-posterior direction. Bringard, Perrey and Belluye (2006) suggested that decreased muscle oscillation due to CG could reduce fatigue by supporting the muscle in the direction of contraction.

Similarly, Borràs *et al.* (2011) found reduced amounts of muscular displacement in quadriceps muscle (Rectus femoris, Vastus lateralis, Vastus medialis) during a 40 min running protocol with the use of compression shorts compared to the control (no compression) condition. This was accompanied by a significantly lower albumin level in the compression condition. Albumin is a marker for sarcomere injury. This then indicates that the compression decreased damage to the muscle by decreasing oscillation thereof.

4. Perceptions of fatigue

Fatigue

The feeling of fatigue during exercise could hinder performance, both physiologically and psychologically. Therefore if a strategy can improve not only the physiological effect of exercise but also the perceptual feeling of fatigue experienced during exercise, this could prove beneficial to the athlete. It was suggested by Millet *et al.* (2006) that CS affect the physiological mechanisms underlying fatigue, namely thermoregulation, increased oxygen availability, and mechanical support.

Rating of perceived exertion (RPE) is a scale that is used to quantify the perception an individual has about the intensity of exercise being performed (Buckley & Eston, 2007).

As the effect of maximal exercise is expected to be similar during different sessions, it means that if CG have an influence on the exertion experienced during exercise, this will be more evident during submaximal exercise at set exercise intensities. Bringard, Perrey and Belluye (2006) tested two groups of six trained male runners each. The first group completed incremental exercise tests to exhaustion and the second group completed 15 min of high intensity submaximal running. They showed that there was no significant effect of different clothing conditions (CS, elastic tights, and regular running attire) on the RPE experienced in either of the test groups. Thus RPE was not influenced by compression during either maximal exertion or submaximal exercise.

Houghton, Dawson & Maloney (2009) tested field hockey players (estimated $VO_{2max} = 58.6 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) with the Loughborough Intermittent Shuttle Test (LIST) wearing either, only hockey kit, or compression shorts and short sleeved compression shirts under their hockey kit. They found no difference in the RPE scores between the conditions after any of the stages.

Ali, Creasy and Edge (2010) had male and female runners ($VO_{2max} = 70.4 \pm 6.1 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) complete a 40 min running protocol at 70 % of peak treadmill velocity at a 1 % gradient on a treadmill. No differences were found between clothing conditions regarding RPE scores. Similarly, Sperlich *et al.* (2010) also found no differences in RPE scores in well trained males ($VO_{2max} = 63.7$

$\pm 4.9 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) after a 15 min run at 70 % $\text{VO}_{2\text{max}}$, as well as after a subsequent test at max speed with different clothing conditions. It seems therefore that CS have no influence on exhaustion experienced by the athlete.

Thermoregulation

It is known that body temperature plays a role in the perception of fatigue. González-Alonso *et al.* (1999) found that participants reached fatigue when their core body temperature was elevated to temperatures above 40 °C during moderate exercise in hot conditions. Although this may be true, one has to consider the complexity of this phenomenon and concede that there may be more than one cause of muscle and whole-body fatigue.

Various researchers have investigated the effect of CG on thermoregulation and whether the influence it has on thermoregulation may have deleterious effects on performance. It has been found that lower body CG (Duffield *et al.*, 2008), compression shorts (Doan *et al.*, 2003; Houghton, Dawson & Maloney, 2009) and compression shirts (Houghton, Dawson & Maloney, 2009) increase skin temperature, however, Houghton, Dawson and Maloney (2009) concluded that in moderate temperatures (17 °C), CG do not have a negative influence on performance during simulated team-game activity. Goh *et al.* (2011) tested the effect of CG on running performance in 10 °C and 32 °C conditions. They found no differences in the physiological variables at the two temperatures which would imply that CG should not affect performance negatively. This study also showed that neither skin nor core body temperature was influenced by the CG, although the muscle temperatures of the thigh and calf were significantly higher with the CG.

MacRae *et al.* (2012) on the other hand, found an increase in skin temperature at the covered sites, accompanied by an increase in CO as well as an tendency ($p = 0.06$) for HR drift to be higher in the CG condition. The increase in CO was not accompanied by an increase in SV, and they speculated that this effect could have been masked by an increase in perfusion of other tissues, such as the skin, where increased temperature could be the cause.

Duffield *et al.* (2008) did not find an increase in tympanic temperature (CG = 37.1 ± 0.4 °C vs. control = 37.2 ± 0.6 °C) along with increases in skin temperature in 14 under 21 club standard rugby players during simulated team sport exercise, and Houghton, Dawson and Maloney (2008) also did not find increases in core temperature (~ 38.5 °C) in 12 amateur field hockey players alongside increased skin temperature. This indicates that though CG may increase the skin temperature below the compressed area, this effect does not carry over to increases in core body temperature and therefore CG do not hinder the thermoregulatory system during exercise (Duffield *et al.*, 2008). This may be due to the fact that a large enough surface area is still available for sweat evaporation. It may also be that increased skin blood flow under the compressed area may cause the increased skin temperature.

The main reason for the increase in skin temperatures may be the specific fabric of the CG. Clothing manufacturers have claimed that their products are made to improve heat dissipation and therefore increased skin temperatures are not to be expected. The dissipation of the heat from the surface of the skin is dependent on the heat difference between the skin and the environment, therefore it is suggested that certain materials may create an environment of increased heat and humidity around the area it covers (Millet *et al.*, 2006), therefore not allowing the area to cool. Gavin *et al.* (2001) studied the influence of different fabrics on the thermoregulatory ability of the skin surface under the fabric (synthetic fabric and cotton). They concluded that synthetic materials designed to enhance evaporation and therefore better maintain temperatures, were not more effective at doing this than regular cotton clothing.

Though CG may protect the skin from exposure to the sun and therefore damage caused by UV-rays, there does not seem to be any evidence that supports the hypothesis that CG may be beneficial to the participant by means of improved thermoregulation and evaporation of sweat from the skin.

Proprioception

The placement of body segments in space, as well as the force and velocity with which they move through space is determined by mechanoreceptors and is termed proprioception (Bernardt & Anderson, 2005). Proprioception is important in sport and exercise as it gives the athlete input of his movement performance, as well as enables the athlete to make necessary adaptations to improve performance. Improving proprioception could improve muscle recruitment patterns, thereby improving economy of movement and decreasing the change of obtaining injuries.

Placing external compression on limbs could improve both the joint awareness and the proprioceptive ability of that limb. This is especially evident when the limb is not close to the endpoints of the ROM in the specific movement (MacRae, Cotter & Laing, 2011). Kraemer *et al.* (1996) suggested that it is the interaction between the subcutaneous receptors and the CG that causes these improvements. They further stated that this effect may be important in fatigued muscles.

F. CONCLUSION

The research regarding CG is broad, but due to differences in the exercise modality, compression applied by the garments, garment type and sample population used, it is still difficult to draw conclusions as to the effect that these garments have. It seems that CG improve most variables tested ([Bla] during recovery, running economy, anaerobic threshold, power maintenance, metabolic waste removal, DOMS, muscle oscillation, proprioception), though there are some variables in which no difference is found (HR, [Bla] during exercise, VO_{2max} , RPE, sprint performance). Although some improvements in physiological responses are seen, they do not necessarily translate to improvements in performance of especially endurance type exercise.

CHAPTER FOUR

PROBLEM STATEMENT

A. SUMMARY OF THE LITERATURE

In the clinical setting, compression socks have been used extensively to treat various problems such as venous insufficiency, venous thrombosis, and varicose veins. Over the past few years the use of compression garments has gained popularity in the sport setting.

Researchers have studied the role of CS in sport and exercise with regard to its physiological effects, performance effects, its effectiveness as a recovery strategy as well as athletes' subjective rating in terms of perceived exertion. Only a few studies have attempted to address the physiological mechanism(s) through which CS supposedly exerts an effect on blood flow and blood lactate kinetics.

In 1987 Berry and McMurray proposed two possible mechanisms whereby CS might improve lactate kinetics post-exercise. Firstly it was suggested that lactate is removed from muscle at a quicker rate due to the improvement in blood flow. Secondly it was proposed that lactate was actually retained and oxidized within the muscle. The latter would then suggest that CS improve muscle oxygenation.

Bringard *et al.* (2006) found that CS improved muscle oxygenation in the resting supine position and during standing. Although this may be indicative of what may happen during exercise, the evidence to support this hypothesis, particularly in well trained athletes is scant and inconclusive.

B. RESEARCH LIMITATIONS

In general, research on compression garments are characterised by a number of shortcomings, making it difficult to draw definitive conclusions. One of the main limitations is the fact that the compression used in the various studies is not always indicated and although it may be specified by the manufacturer of the specific compression garment, these pressures may vary significantly among subjects due to variations in leg morphology. In cases where the compression was measured, uniform measurement sites and measurement techniques were not used. The outcomes of these different measurements have not been compared before.

Very little research has focussed on the effect of CS on muscle oxygenation during exercise and especially post-exercise recovery. Researchers have focused more on the role of CS on venous blood flow and oedema in non-athletic populations.

C. AIMS OF THE CURRENT STUDY

The primary aim of this study was to explore a possible mechanism whereby knee high compression socks enhances calf muscle function in endurance trained athletes.

The specific aims of this study were:

1. To determine whether CS improve calf muscle oxygenation of well-trained endurance athletes during and after a 10 km simulated running exercise protocol.
2. To compare the effect of high and low pressure CS on lower leg muscle oxygenation.

CHAPTER FIVE

METHODOLOGY

A. STUDY DESIGN

This study followed a randomized, cross over study design in which several physiological variables were assessed under different compression conditions. Participants completed three exercise tests, of which two tests were completed with high and low compression socks, respectively, and one test without any socks. The physiological variables were also measured during a 60 min recovery period after the exercise tests.

B. PARTICIPANTS

1. Subject selection

Participants who volunteered ($n = 11$) for the study were between the ages of 30 and 40 years. All participants were men and were well trained endurance runners or triathletes. Participants were included if they trained a minimum of three times per week and partook regularly in either running or triathlon events. Participants could only be included if their posterior calf skinfold was equal to or less than 10 millimetres (mm). Participants were excluded from the study if they were suffering from or recovering from an injury, or if they suffered from venous thrombosis. Participants were also excluded if they suffered from any metabolic diseases which could potentially influence lactate kinetics, such as McArdle's disease.

2. Ethical aspects

The study proposal was approved by the Ethics Committee of Research Subcommittee A (Human Research, Non-Health) (Appendix C) at Stellenbosch University (Reference number 511/2011). During the first contact session with the researcher, the study protocol and consent forms were explained verbally to the participants and they were provided opportunity to ask questions. Participants were then requested to sign the consent forms. No invasive procedures were used during this study and none of the tests posed any serious risks for the participants. Participants were reminded that they could withdraw from the study at any time without any penalty.

3. Environmental aspects

Testing was completed in the Sport Physiology laboratory, Stellenbosch University. During all testing sessions, the air conditioner was switched on to try and standardise the conditions during the different testing sessions. Room temperature was measured with the Cosmed Quark CPET metabolic system (Rome, Italy).

C. EXPERIMENTAL DESIGN

Participants were tested on three separate days within a four week period, a minimum of four days between testing sessions. A subset ($n = 5$) of the group completed a fourth testing session. During the course of the study participants continued with their regular exercise regime, except for the day preceding the test day, when the participants were requested to refrain from any physical activity. Participants complied with pre-test requirements before each of the testing sessions. The requirements were: (1) no eating or drinking two to three hours before the test, (2) no consumption of alcohol or caffeine 12 hours before the testing session and (3) no exercise 24 hours before the testing session.

1. Testing sessions

First session

The purpose of the first test session was to determine various descriptive characteristics of the study sample. After the study protocol and consent form (Appendix A) were verbally explained to each participant, time was allowed for questions, where after participants were asked to sign the consent form. Participants then completed a health questionnaire (Appendix B), which was used to screen for any exclusion criteria. A number of anthropometrical measurements were taken which included; height, body mass, percentage body fat, posterior calf skinfold, and calf girth at several specified measurement areas (see section D.1.).

After all baseline measurements were taken, the participants completed an incremental treadmill running test to exhaustion. Measurements taken during this test included maximal aerobic capacity (VO_{2max}), blood lactate concentration ([BLa]), heart rate (HR), and peak treadmill velocity (PTV). Treadmill velocity at LT (LTV) was calculated after the test.

Sessions two to four

During the second and third testing sessions, compression profiles of the leg under the sock, either a sport compression sock (CS) or flight sock (FS) were measured (see section D.2). The probes for the muscle oxygenation measurements were then fixed to the muscle (see section D.4). Thereafter participants completed a simulated run on the treadmill, wearing either the CS or the FS. The order of the tests was randomly assigned. A subset of individuals ($n = 5$) completed a fourth session without socks as a control condition (NS).

The exercise protocol consisted of a simulated 10 km run on the treadmill at 80 % of the athlete's PTV as determined during the first test session. Muscle oxygenation was measured continuously during the run, as well as during the 60 min passive recovery period after the exercise.

D. MEASUREMENTS AND TESTS

The dependent (outcome) variables of the study included: Oxy-haemoglobin (O_2Hb), deoxy-haemoglobin (HHb), tissue oxygenation index (TOI), total haemoglobin index (nTHI), blood lactate concentration ([BLa]), skin temperature (ST), haemoglobin concentration ([Hb]) and sock pressure (mmHg).

1. Anthropometrical measurements

The anthropometrical measurements that were taken included height, body mass, percentage body fat (%BF), posterior calf skin fold (SF_{post}), and several calf girths.

Height

The participant's height was measured using a standing SECA stadiometer (model 220, Hamburg, Germany). The participant stood barefoot on the stadiometer with feet together and heels, buttock and the upper part of the back touching the stadiometer. The participant was placed in the Frankfort plane, with the orbitale in the same horizontal plane as the tragion of the ear. The head board was lowered after the participant was asked to take a deep breath (Norton, 1996). The measurement was taken to the nearest centimetre (cm).

Body mass

Body mass was measured using an electronic scale (UWE BW-150, UK). Participants were weighed barefoot, standing on the centre of the scale with their weight evenly distributed across both feet. Participants were also instructed to look straight ahead while the reading was taken (Norton, 1996). The measurement was taken to the nearest 0.1 kilogram (kg).

Percentage body fat

Percentage body fat (%BF) was measured with a portable Bodystat unit (Quadscan 4000, Isle of Man, United Kingdom) using bio-electrical impedance analysis (BIA). Prior to measurement, participants were asked to empty their bladders. Participants then lay supine on a plinth with their socks and shoes removed and their arms and legs spread apart so as not to touch the centre of the body or, in the case of the legs, not to touch each other. Four electrodes were placed at standard anatomical points on the right side of the body after the area was cleaned with alcohol swabs. One of the electrodes was placed on the dorsal side of the right hand, one centimetre proximal to the knuckle of the middle finger, while the other was placed on the dorsal side of the wrist between the heads of the radius and ulna. The other two electrodes were placed on the dorsal foot, one between the hallux and third phalange, and the other between the medial and lateral malleoli.

The Bodystat unit was connected to the electrodes and a low electrical current (800 μ A at 50 kHz) was sent through the body. Measurements of resistance and reactance were taken and the Bodystat software used this along with anthropometrical data to calculate %BF. This is possible due to the differences in resistance of adipose and non-adipose tissue.

Lower leg girths

Calf girths were measured with a flexible steel anthropometry tape (Rosscraft, Canada). The participants were asked to sit with their knees at 90 degree ($^{\circ}$) angles. Calf circumferences were measured at three different sites, namely the greatest circumference around the calf (C), the smallest circumference of the ankle (B), and at the level just below the head of the fibula (D) (*Figure 5.1.*).

Posterior calf skinfold

The posterior calf skinfold was taken over the area where the NIRS probe would be attached to determine (see section D.4.) whether the sub-cutaneous fat layer was thin enough to ensure measurement of muscle oxygenation. The measurement was taken whilst the participant was in the seated position with the knee bent at a 90° angle and the muscle relaxed, using a Harpenden skinfold calliper (HSK-BI, Baty International, UK). Two measurements were taken to the nearest mm and the average of the two measurements was used as the final score.

2. Maximal aerobic capacity exercise test

Participants completed a maximal aerobic capacity exercise test to determine their VO_{2max} . The test was done on the h/p/cosmos Saturn treadmill (Nussdorf-Traunstein, Germany) and the Cosmed Quark CPET metabolic system (Rome, Italy) was used for continuous monitoring of the metabolic variables.

The participants were strapped into the safety harness of the treadmill and a resting [BLa] was taken by means of a finger prick. The finger was cleaned with an alcohol swab and then pricked using an Accucheck Soft Clix (Roche diagnostics, Mannheim, Germany). Blood was then drawn into the capillary tube of the Lactate Pro lactate analyser (ARKRAY, Inc. Kyoto, Japan). Thereafter, the participant completed a warm-up of ten minutes at a running speed of 8 km.h⁻¹.

After the completion of the warm-up, the participants attached the HR monitor strap and face mask of the metabolic analyser. The participants then completed an incremental test to exhaustion on the treadmill starting at a speed of 10 km.h⁻¹ and an incline of 0 %. The workload was increased every three minutes in 1 km.h⁻¹ increments up until a [BLa] of 4 mmol.L⁻¹, where after the incline also increased with 1 % increments. [BLa] was measured as described above at intervals of three minutes (i.e. at the end of each completed work load) to determine the lactate threshold.

The test was considered maximal if (1) the VO_2 did not increase further despite an increase in workload, (2) the respiratory exchange ratio (R) was at or above 1.15, (3) the HR was within 10

beats per minutes ($\text{b}\cdot\text{min}^{-1}$) of the age predicted maximum, and (4) the final [BLa] was above 8 $\text{mmol}\cdot\text{L}^{-1}$ (Howley, Barrett & Welch, 1995; McArdle, Katch & Katch, 2010). Participants had to reach at least three of these criteria for a test to be classified as maximal.

3. Sock pressure profile

The pressure exerted by both the CS and the FS on the calf was measured using the ELF system (Tekscan, Boston, USA). Calibration of this sensor was done by placing three weights of known weight (9.9 g, 19.7 g, 29.5 g) on the sensor in succession which was placed on a flat surface. The software plotted the given weight to the resistance measured and thereby calculated the calibration curve.

The participants were asked to stand upright with body mass evenly spread over both feet. The force sensor was then inserted between the sock and the skin and measurements were taken at seven separate points. Three of these sites were the posterior points B, B1, and D as described in the European document for normalization (Partsch *et al*, 2006). Measurement site B can be found at the point of minimum ankle girth on the posterior of the lower leg. B1 is the point where the Achilles tendon changes into the gastrocnemius muscle, and D is the posterior point in line with the point just below the tibial tuberosity. Further measurements were taken at the level of the greatest circumference around the calf (C), on the medial, anterior, lateral and posterior side of the calf. The measurement was taken for 10 seconds at a frequency of 2 Hz. The force measurement was taken in Newton (N) and was converted to pressure in millimetre of mercury (mmHg) (*Equation 5.1.*) as is mostly used in a physiological setting.

$$P = \frac{x}{\frac{7.22 \times 10^{-6}}{133.32}} \quad (5.1.)$$

Equation 5.1. Conversion calculation to pressure, where x is the force measured (N) by the sensor.

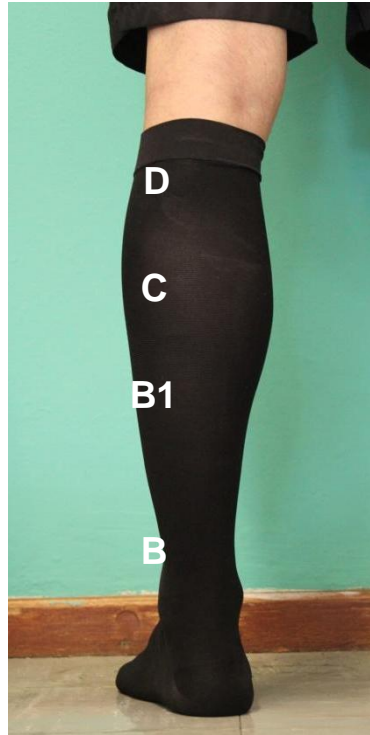


Figure 5.1. Classification of anatomical points used for pressure and circumference measurements (*Photo by L. Grobler*).

4. Exercise protocol

Simulated running protocol

During the three testing sessions participants completed a 5 min warm up at a speed of $8 \text{ km}\cdot\text{h}^{-1}$, where after they ran 10 km at a submaximal exercise intensity. The intensity was set at 80 % of the PTV as determined during the maximal test to exhaustion, however, the speed was adjusted if the participant was unsure whether he would be able to complete the full distance. The incline was set at 1 % for the first and last 2 km, and at 2 % for the distance in between (*Figure 5.2*).

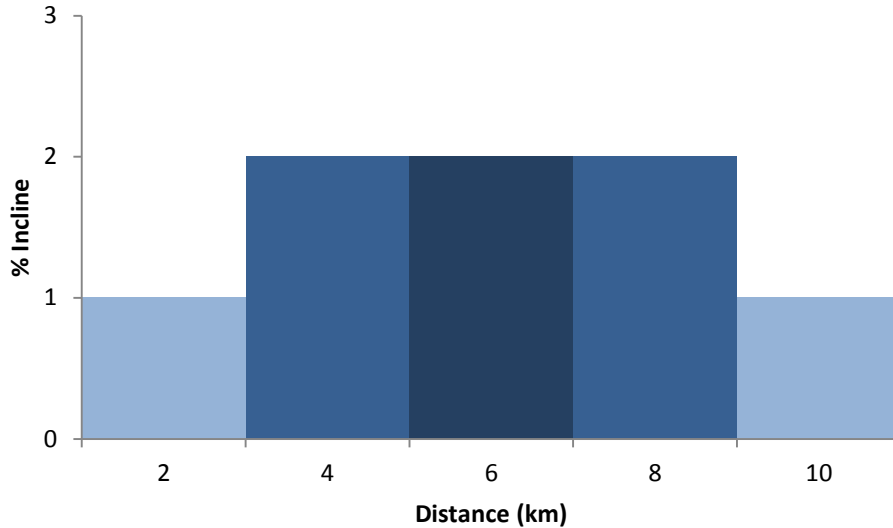


Figure 5.2. A representation of the simulated 10 km running protocol, where each column represents a 2 km interval.

Muscle oxygenation

Muscle oxygenation was measured with the NIRO-200NX monitor (Hamamatsu, Japan). The area to which the probe was attached was prepared by shaving and cleaning it with an alcohol swab. The probe was fixed with clear double-sided adhesive tape at the level of the greatest circumference around the calf on the muscle belly of the lateral Gastrocnemius three centimetres to the lateral side of the point where the line perpendicular to the centre of the bicondylar femur crosses the greatest circumference of the calf (*Figure 5.3*). This site was chosen as the gastrocnemius is one of the major active muscles during running. One probe was attached to each leg. Furthermore, an intravenous (IV) plaster was applied over the probe to keep it in place and ensure that it did not come loose due to sweating. The probe was fixed for the duration of the running and recovery protocol, measuring at a frequency of 1 Hz (*Figure 5.4*). The wavelength was set at 735, 810 and 850 nm as determined by the manufacturers and the distance between the emitter and detector probes were 4 cm.

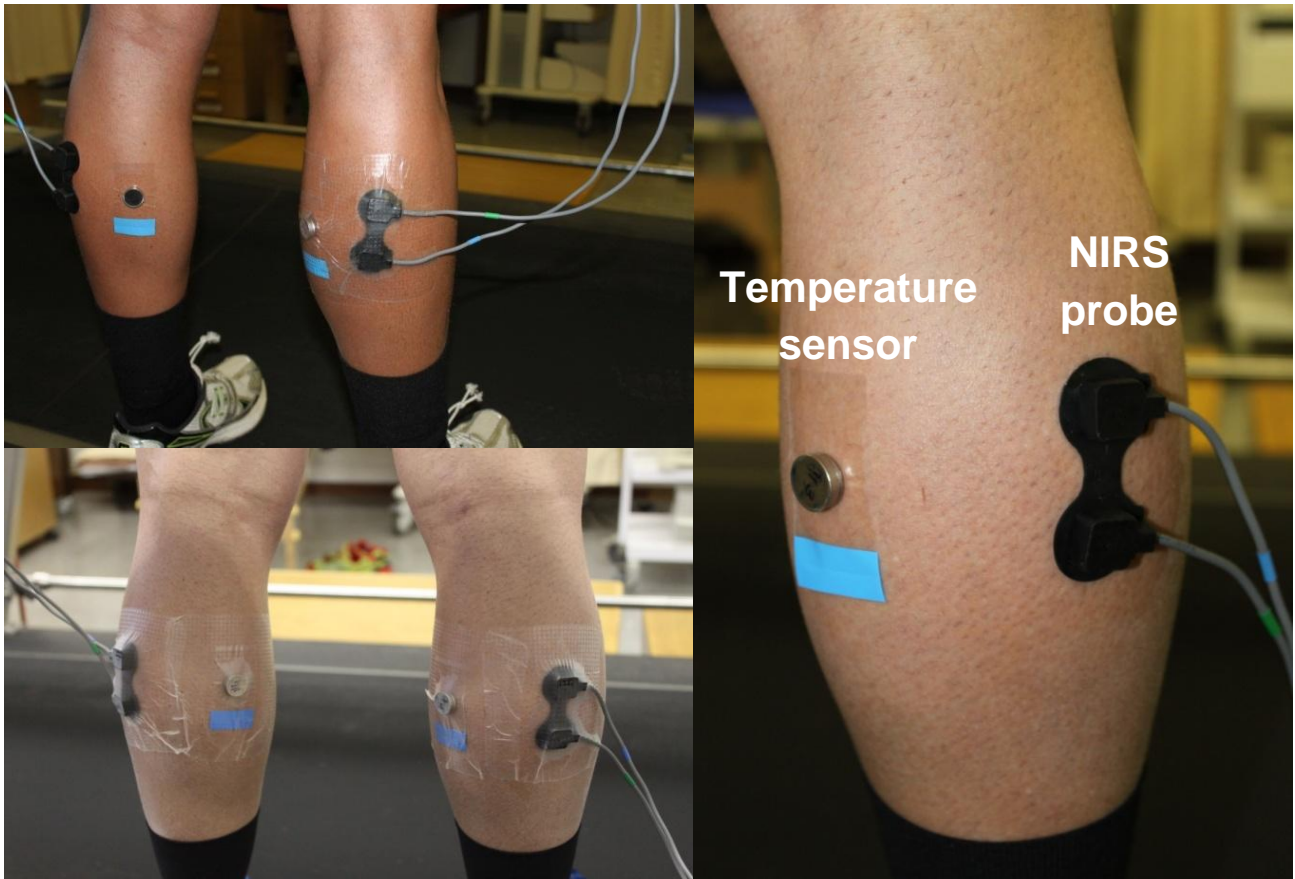


Figure 5.3. NIRS probe and temperature sensor placement, with (two photos on left) and without (photo on right) IV plaster. (Photo by L. Grobler).

The NIRO-200NX (Hamamatsu, Japan) measures oxy-haemoglobin (O_2Hb), deoxy-haemoglobin (HHb), tissue oxygenation index (TOI), and total haemoglobin index (nTHI). O_2Hb and HHb are calculated with the Beer Lambert Law (*Equation 2.3.*), as the change in μmol from baseline and not as absolute values. Spatially resolved spectroscopy is used for the calculation of both TOI (*Equation 2.5.*) and nTHI (*Equation 2.6.*) measurements. TOI is measured in percentage (%) whereas nTHI is measured in arbitrary units (AU).



Figure 5.4. Simulated running protocol (Photo by L.A. de Villiers)

The NIRS measurements were started before warm-up, with the sock already pulled over the probes. When pressure is applied, it changes the vascular bed below the point of pressure. Because the variables O_2Hb and HHb measure the relative change from the initial measurement, measurements were started after the application of pressure, thereby excluding the change due to the pressure. This enables for comparison between the different conditions with regard to the change brought along by the exercise due to the compression condition rather than comparing the combined value for the change due to the compression condition along with the change due to the exercise.

Heart rate and Oxygen consumption

HR, VO_2 and VCO_2 was monitored during the running protocol through breath-by-breath analysis using the Cosmed Quark (CPET) (Rome, Italy). All data was filtered at a frequency of 0.1 Hz.

Skin temperature

Skin temperature was measured using an iButton (Maxim, Sunnyvale, CA, US) temperature probe on each leg. The area where the sensor was attached was shaved clean and the sensor was attached with double sided adhesive tape and covered with an IV plaster to ensure that it did not come loose due to sweating, as well as to simulate the conditions under the NIRS probe. The probe was attached to the posterior medial Gastrocnemius on the muscle belly (*Figure 5.3*). Temperature was measured at a frequency of 0.0167 Hz.

Blood lactate concentration

[BLa] was measured as described earlier. Prior to the start of the 10 km exercise protocol, resting blood lactate was determined, where after measurements were taken after every 2 km interval. For these measurements to be taken, the treadmill was stopped. During recovery, measurements were taken at 0, 10, 20, 30, 40, 50, and 60 min post-exercise.

Haemoglobin concentration

Prior to the start of the 10 km run, [Hb] was assessed using a Hb 201+ (HemoCue, Sweden) Haemoglobin analyser. The area on the finger was cleaned with an alcohol swab and pricked with an Accucheck Soft Clix (Roche diagnostics, Mannheim, Germany). 10 μ L of blood was pulled into the microcuvette by capillary action. The analyser makes use of dual wavelength spectroscopy, at wavelengths 570 nm and 880 nm, along with a modified azidemethemoglobin reaction to determine [Hb].

5. Recovery protocol

After the completion of the two hour simulated running protocol, participants completed 60 min of passive recovery seated in a comfortable chair keeping the sock on. During this recovery time the measurement of muscle oxygenation and skin temperature was continued. [BLa] was also measured by means of finger prick analysis as described earlier.

E. STATISTICAL ANALYSIS

Statistical analysis was performed with the use of Microsoft Office Excel (2010) and STATISTICA 10. Data in tables and figures are indicated as mean and standard deviation (SD). For all analysis the significance level was set at $p < 0.05$.

The pressure measurements were log transformed for normality fit and a single factor analysis of variance (ANOVA) and Fischer's least significant difference (LSD) post hoc test was applied to determine any differences in these variables. Except for the log transformations the data for [Hb] was also analysed in the same manner.

The differences in metabolic variables (VO_2 , VCO_2 , HR, and [BLa]), NIRS variables (O_2Hb , HHb, TOI, and nTHI), and ST over time were determined by a two-way ANOVA (condition x time) and Fischer's LSD post hoc testing. Log transformations were applied to the [BLa] data for an improved normality fit.

Cohen's effect sizes were used to determine practically significant differences between the compression conditions during both exercise and recovery for the various variables. Cohen's effect sizes were determined according to the following criteria: small practical effect, > 0.20 ; medium practical effect, > 0.60 ; large practical effect, > 0.80 ; and very large practical effect, > 1.20 .

Certain calculations for the determination of change in NIRS variables were also applied to the raw data (*Figure 5.5*). The calculations used were as follows:

$$\Delta 1 = a - b \quad (5.2)$$

Equation 5.2. Difference between the average of the pre-exercise rest period (*a*) and the average of the last 2 km of the running protocol (*b*).

$$\Delta 2 = x - y \quad (5.3)$$

Equation 5.3. Difference between end of exercise (*x*) and the first peak post-exercise (*y*).

$$\Delta 3 = y - z \quad (5.4)$$

Equation 5.4. The difference between the first peak post-exercise (*y*) and the end of the 60 min recovery period (*z*).

$$\Delta 4 = x - z \quad (5.5)$$

Equation 5.5. The difference between the end of the running protocol (*x*) and the end of the 60 min recovery period (*z*).

Two-way ANOVA's (condition x time) was used to determine differences between the groups for the results of these calculations. Cohen's effect sizes were also calculated for the muscle oxygenation variables (*Equation 5.3. to Equation 5.5.*). The effect sizes were evaluated according to the criteria above.

Correlations between various anthropometrical variables and pressure variables were determined with Pearson correlation coefficients. The strength of these correlations were determined as follows: $r = 0$, no correlation; $0.00 \leq r \leq 0.24$, weak correlation; $0.25 \leq r \leq 0.49$, moderate correlation; $0.50 \leq r \leq 0.74$, moderate to good correlation; and $0.75 \leq r \leq 1.00$, strong correlation.

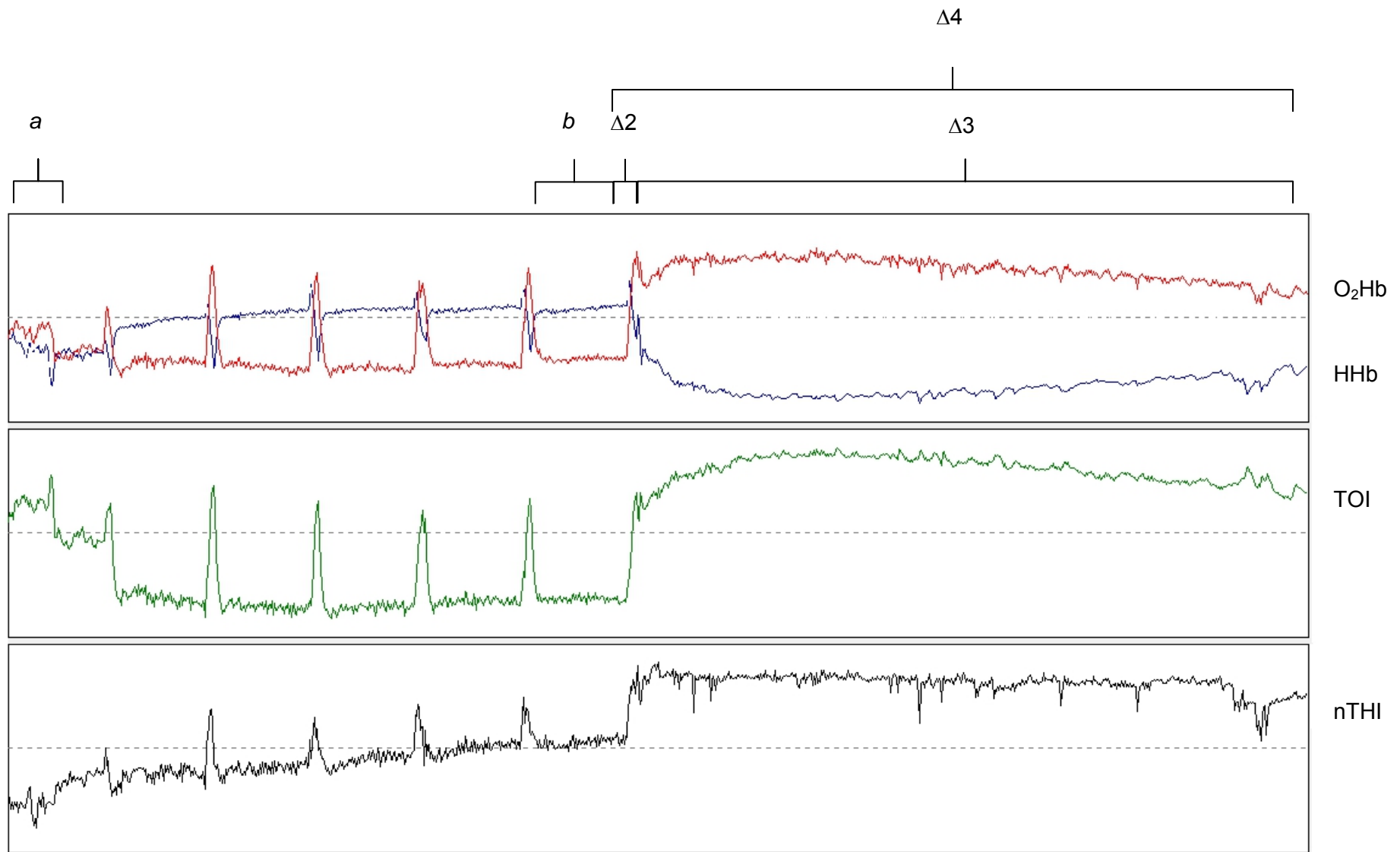


Figure 5.5. Graphical explanation of delta calculations shown on raw data as displayed by the NIRO 200NX (Hamamatsu, Japan) software.

CHAPTER SIX

RESULTS

A. DESCRIPTIVE STATISTICS

1. Participants

Of the 14 individuals who volunteered to partake in the study, two could not complete the testing due to injury, and one participant was excluded as he did not comply with the inclusion criteria. Therefore, 11 men aged between 30 and 40 years completed the baseline testing as well as testing with both the CS and FS condition. Of the 11 participants, five participants also completed a control trial (NS). *Table 6.1.* depicts the physical and anthropometrical characteristics of the participants and *Table 6.2.* the physiological characteristics of the participants.

Table 6.1. Physical and anthropometrical characteristics of participants.

Characteristic	$x \pm SD$	Range
Age (years)	34.8 \pm 3.8	30 - 40
Height (cm)	181.2 \pm 5.6	170.0 - 193.8
Weight (kg)	76.8 \pm 10.6	53.4 - 90.7
BMI (kg.m ⁻²)	23.4 \pm 2.4	18.5 - 27.2
Percentage body fat (%)	14.4 \pm 3.1	8.9 - 19.5
Ankle circumference (cm)	22.1 \pm 0.7	20.9 - 23.4
Calf circumference (cm)	38.6 \pm 1.7	34.9 - 40.8
D circumference (cm)	33.5 \pm 1.2	31 - 35
SF _{Post} (mm)	7.4 \pm 2.2	4.6 - 10.0

BMI, Body Mass Index; D circumference, circumference below the level of the tibial tuberosity; SF_{Post}, posterior calf skinfold;

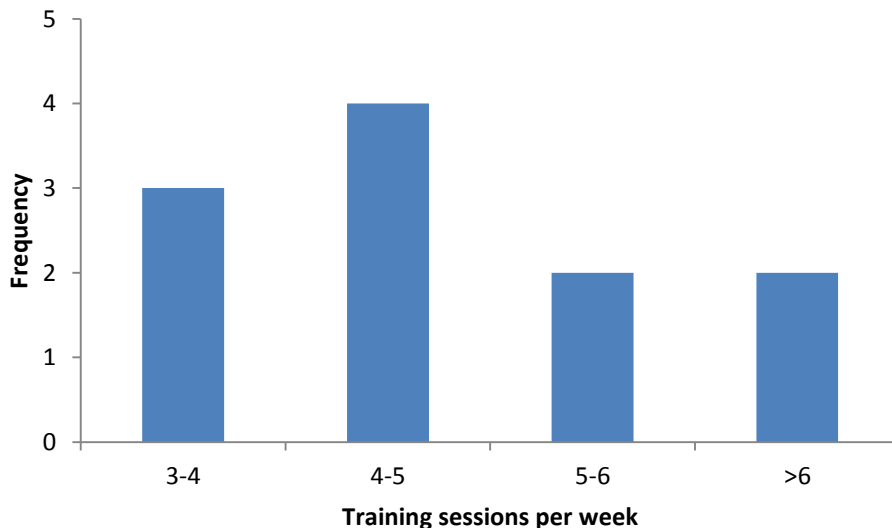
Table 6.2. Physiological characteristics of participants.

Characteristic	$\bar{x} \pm SD$	Range
PTV ($\text{km}\cdot\text{h}^{-1}$)	17.1 ± 1.2	16 - 19
$\text{VO}_{2\text{max}}$ ($\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$)	52.37 ± 7.12	40.50 - 63.10
$\text{VO}_{2\text{max}}$ ($\text{mL}\cdot\text{min}^{-1}$)	4083.09 ± 504.19	3369 - 5020
Percentage of $\text{VO}_{2\text{max}}$ at LT (%)	85.2 ± 4.8	75.2 - 93.4
Percentage HR_{max} at LT (%)	90.8 ± 3.4	83.8 - 97.0

PTV, peak treadmill velocity; $\text{VO}_{2\text{max}}$, maximum aerobic capacity; LT, lactate threshold ($4 \text{ mmol}\cdot\text{L}^{-1}$); HR_{max} , maximum heart rate;

During testing the temperature in the laboratory with the different compression conditions ranged as follows: CS ($20.2 \pm 1.2 \text{ }^\circ\text{C}$, 18 – 22 $^\circ\text{C}$), FS ($20.6 \pm 1.7 \text{ }^\circ\text{C}$, 18 – 23 $^\circ\text{C}$) and NS ($20.8 \pm 2.6 \text{ }^\circ\text{C}$, 17 – 24 $^\circ\text{C}$).

Figure 6.1. displays the distribution of training sessions per week completed by the participants. Of the 11 participants, four trained four to five times per week. Three participants trained three to four times per week, two participants trained five to six times per week and two participants trained more than six times per week.

**Figure 6.1.** The number of training sessions per week done by the participants.

2. Baseline measurements before the simulated run test

Haemoglobin

Before participants commenced with the warm up for the 10 km simulated running protocol, baseline haemoglobin ([Hb]) measurements were taken. *Table 6.3.* summarizes these results, while Cohen's effect size statistics are depicted in *Table 6.4.* Although there were no statistically significant differences in average [Hb] between the different testing days ($p = 0.17$), the runners who completed the control trial had practically significant higher values on the day of the control testing compared to the other two trial days.

Table 6.3. Baseline [Hb] (g.dL^{-1}) measurements before 10km running protocol with different compression conditions ($p > 0.05$).

	$x \pm \text{SD}$	Range
CS (n = 11)	15.0 ± 0.9	13.5 - 16.5
FS (n = 11)	15.3 ± 1.2	13.5 - 17.2
NS (n = 5)	16.2 ± 0.4	15.0 - 16.6

CS, compression socks; FS, flight socks; NS, no socks; x, mean; SD, standard deviation

Table 6.4. Cohen's effect size results for [Hb] measurements

	CS	FS	NS	ES
CS			-1.42	VL
FS	-0.24			S
NS		-0.89		L

CS, compression socks; FS, flight socks; NS, no socks; ES, effect size; S, small practical effect; L, large practical effect; VL, very large practical effect

Pressure

Before completing the simulated running protocol, the pressures exerted by the specific sock (i.e. CS or FS) worn by an individual during the run tests were measured (*Table 6.5.*).

Table 6.5. Raw pressure values (mmHg) for the compression and flight socks.

		Ankle	B1	D	Posterior	Lateral	Anterior	Medial
CS	$(x \pm SD)$	41.38±	31.06±	19.22±	69.09±	59.28±	49.97±	20.74±
		20.97	43.96	12.43	62.76	56.46	30.25	14.27
FS	$(x \pm SD)$	23.39±	25.13±	7.91±	59.92±	44.26±	20.03±	20.33±
		13.01	58.42	3.09	72.50	59.45	8.43	9.89

CS, compression socks; FS, flight socks; B1, pressure on the posterior calf at the point where the Achilles tendon and the Gastrocnemius meet; D, pressure on the posterior calf at the level just below the tibial tuberosity; x , mean; SD, standard deviation

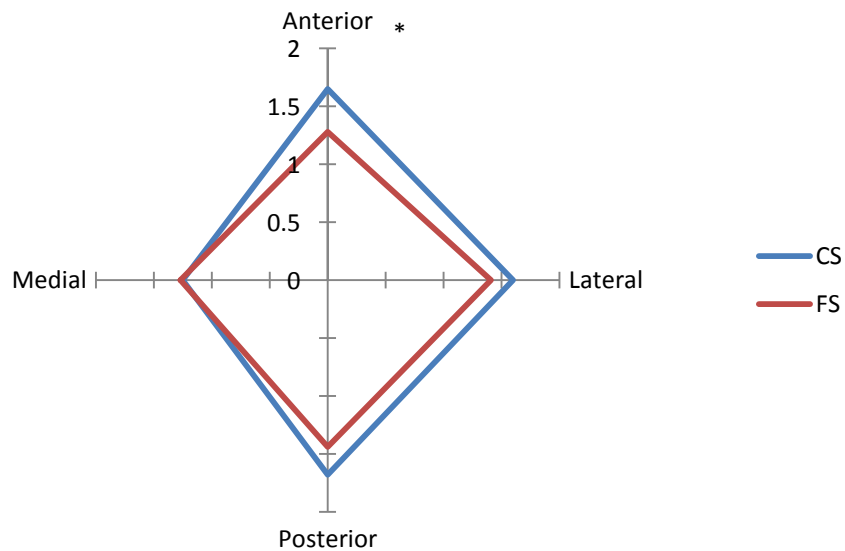
After log transformations, statistically significant differences ($p < 0.05$) were found in the CS condition between ankle and elastic (1.56 ± 0.88 AU vs. 1.22 ± 0.77 AU), ankle and medial (1.56 ± 0.88 vs. 1.25 AU ± 0.69 AU), B1 and posterior (1.24 ± 0.67 AU vs. 1.68 ± 1.03 AU), B1 and lateral (1.24 ± 0.67 AU vs. 1.60 ± 1.00 AU), B1 and anterior (1.24 ± 0.67 AU vs. 1.65 ± 1.32 AU), D and anterior (1.65 ± 1.32 AU vs. 1.22 ± 0.77 AU), D and posterior (1.22 ± 0.77 AU vs. 1.68 ± 1.03 AU), posterior and medial (1.68 ± 1.03 AU vs. 1.25 ± 0.69 AU), and anterior and medial (1.22 ± 0.77 AU vs. 1.25 ± 0.69 AU) measurement positions. No statistically significant differences were found between the other combinations.

With the FS condition, statistically significant differences were found between the ankle and B1 (1.31 ± 0.77 AU vs. 0.75 ± 0.00 AU), ankle and D (1.31 ± 0.77 AU vs. 0.93 ± 0.74 AU), B1 and posterior (0.75 ± 0.00 AU vs. 1.44 ± 0.42 AU), B1 and lateral (0.75 ± 0.00 AU vs. 1.41 ± 1.00 AU), B1 and anterior (0.75 ± 0.00 AU vs. 1.28 ± 0.97 AU), B1 and medial (0.75 ± 0.00 AU vs. 1.27 ± 0.75 AU), D and posterior (0.93 ± 0.74 AU vs. 1.44 ± 0.42 AU), D and lateral (0.93 ± 0.74 AU vs. 1.41 ± 1.00 AU), D and anterior (0.93 ± 0.74 AU vs. 1.28 ± 0.97 AU), and D and medial (0.93 ± 0.74 AU vs. 1.27 ± 0.75 AU) measurement points of the calf. No statistically significant differences were found with the other combinations.

For both CS and FS, the greatest variation in pressures was observed for the B1, posterior and lateral measurements ($> 120\%$). Least variation was observed for the FS pressures at the D, anterior and medial measurement sites, although the variations were still greater than 38%.

Pressures at greatest circumference of calf

The pressure measurements taken at the greatest circumference of the calf is illustrated in *Figure 6.2*. No significant differences were found on the posterior (CS: 69.09 ± 62.76 mmHg vs. FS: 59.92 ± 72.50 mmHg; $p = 0.30$), medial (CS: 20.75 ± 14.27 mmHg vs. FS: 20.33 ± 9.89 mmHg, $p = 0.87$) and lateral side of the calf (CS: 59.28 ± 56.46 mmHg vs. FS: 44.26 ± 59.42 mmHg; $p = 0.34$). A significant difference ($p = 0.002$) was found between the two sock conditions on the anterior calf (CS: 49.97 ± 30.02 mmHg vs. FS: 20.03 ± 8.43 mmHg). *Table 6.6.* represents the effect size results for the pressures measured around the calf at the greatest circumference of the calf. There was a small practical difference between CS and FS at the lateral calf, and a very large practical difference on the anterior calf between CS and FS. In both cases, CS measured higher pressures than FS.

**Figure 6.2.**

Log transformed pressure (P) measurements (AU) at the greatest circumference of the calf.

* Significant difference between CS and FS ($p < 0.05$)

Table 6.6. Cohen's effect size results for pressure measurements at the greatest circumference of the calf.

	CS vs. FS	ES
Posterior	0.14	
Lateral	0.26	S
Anterior	1.35	VL
Medial	0.03	

CS, compression socks; FS, flight socks; ES, effect size; S, small practical effect; VL, very large practical effect

Pressure ranging from proximal to distal

As illustrated in *Figure 6.3.*, statistically significant differences ($p < 0.05$) were found between the compression measurements in the two sock conditions for the ankle (CS: 41.38 ± 20.97 mmHg vs. FS: 23.39 ± 13.01 mmHg), at measurement point D (CS: 19.22 ± 12.43 mmHg vs. FS: 7.91 ± 3.09 mmHg) and at B1 (CS: 31.06 ± 43.96 mmHg vs. FS: 20.56 ± 53.23 mmHg). In all cases CS exerted higher pressures compared to FS. According to Cohen's effect sizes, the differences in pressures at the ankle and at point D were also of large practical significance (*Table 6.7.*).

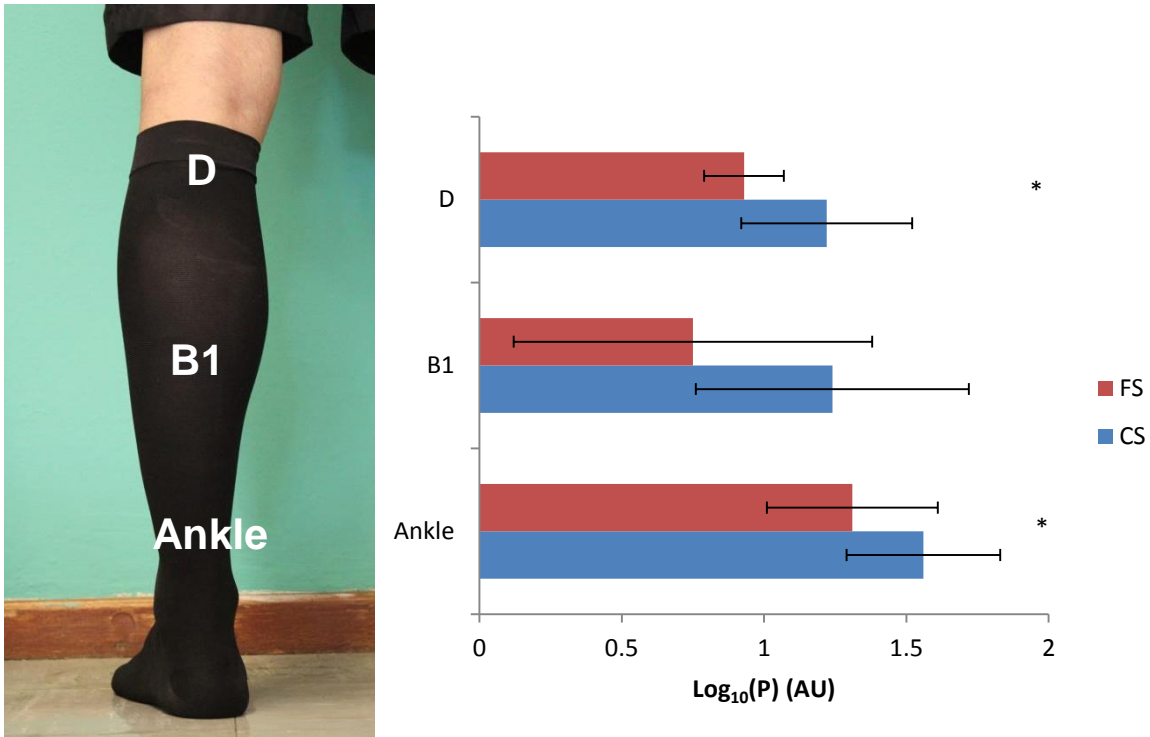


Figure 6.3. Log transformed pressure measurements (AU) on the posterior calf ($p < 0.05$). Pressure measurements coincide with the indicated level on the calf in the photo (Photo by L. Grobler).
 * Statistically significant difference between CS and FS ($p < 0.05$).

Table 6.7. Cohen’s effect size results for posterior calf pressures.

	CS vs FS	ES
Ankle	1.03	L
B1	0.11	
D	1.25	VL

CS, compression socks; FS, flight socks; ES, effect size; B1, pressure on the posterior calf at the point where the Achilles tendon and the Gastrocnemius meet; D, pressure on the posterior calf at the level just below the tibial tuberosity; L, large practical effect; VL, very large practical effect

B. COMPARISON IN VARIABLES DURING EXERCISE

Participants completed a 10 km simulated running protocol, during which a number of physical and physiological variables were measured. Statistically significant changes took place over time in all variables ($p < 0.05$). However, there was no treatment order effect ($p > 0.05$).

1. Skin temperature (ST)

Table 6.8. displays the calf skin temperatures measured during exercise in the three experimental conditions. No statistically significant difference was found between the three compression conditions ($p = 0.17$). No statistically significant differences were found between the CS and FS conditions at any of the time points, or between the left and right leg and the different time points. In both legs, there were statistically significant differences in ST between the CS and NS condition at 2 km (left, $p = 0.001$; right, $p = 0.001$), 4 km (left, $p = 0.001$; right, $p = 0.001$), 6 km (left, $p = 0.01$; right, $p = 0.002$), and 8 km (left, $p = 0.02$; right, $p = 0.02$). Similarly, statistically significant differences were found between FS and NS at 2 km (left, $p = 0.003$; right, $p = 0.001$), 4 km (left, $p = 0.002$; right, $p = 0.001$), 6 km (left, $p = 0.01$; right, $p = 0.01$), and 8 km (left, $p = 0.01$; right, $p = 0.04$). Overall, skin temperatures were lower during the NS run (Left: 30.66 ± 0.61 °C, Right: 30.94 ± 0.57 °C) compared to CS (Left: 32.44 ± 0.20 °C, $p = 0.01$; Right: 32.54 ± 0.16 °C, $p = 0.001$) and FS (Left: 32.14 ± 0.06 °C, $p = 0.01$; Right: 32.45 ± 0.19 °C, $p = 0.001$).

Table 6.8. Skin temperatures (°C) in the right and left leg during the 10 km simulated running protocol (mean \pm SD).

	Distance (km)	CS (°C)	FS (°C)	NS (°C)
Left	2	32.6 ± 1.1	32.2 ± 0.8	$30.1 \pm 0.7^{*}\#$
	4	32.7 ± 1.3	32.2 ± 0.9	$30.2 \pm 0.8^{*}\#$
	6	32.4 ± 1.4	32.1 ± 1.1	$30.5 \pm 0.9^{*}\#$
	8	32.3 ± 1.5	32.1 ± 1.3	$30.9 \pm 0.8^{*}\#$
	10	32.2 ± 1.6	32.2 ± 1.2	31.6 ± 0.6
Right	2	32.7 ± 0.6	32.3 ± 1.0	$30.5 \pm 1.0^{*}\#$
	4	32.6 ± 0.9	32.4 ± 1.0	$30.5 \pm 1.0^{*}\#$
	6	32.5 ± 1.0	32.3 ± 1.3	$30.8 \pm 1.2^{*}\#$
	8	32.3 ± 1.1	32.5 ± 1.4	$31.0 \pm 1.3^{*}\#$
	10	32.6 ± 1.1	32.8 ± 1.3	31.9 ± 1.4

CS, compression socks; FS, flight socks; NS, no socks

* Significant difference between CS and NS conditions ($p < 0.05$)

Significant difference between FS and NS conditions ($p < 0.05$)

Cohen's effect sizes confirm that there were strong practically significant differences in ST during the run trials with socks (CS and FS higher ST) compared to the NS run. Only a small practically significant difference was observed between CS and FS (Table 6.9).

Table 6.9.

Cohen's effect size results for skin temperature between the different compression conditions during the 10 km run protocol.

		Left				Right			
		CS	FS	NS	ES	CS	FS	NS	ES
2 km	CS			2.44	VL			2.84	VL
	FS	0.44			S	0.47			S
	NS		2.62		VL		1.85		VL
4 km	CS			2.12	VL			2.32	VL
	FS	0.41			S	0.23			S
	NS		2.32		VL		1.95		VL
6 km	CS			1.54	VL			1.57	VL
	FS	0.25			S	0.13			S
	NS		1.56		VL		1.24		VL
8 km	CS			1.01	L			1.12	L
	FS	0.16			L	-0.13			L
	NS		0.94		L		1.07		L
10 km	CS			0.47	S			0.61	M
	FS	0.04			S	-0.12			S
	NS		0.52		S		0.67		M

CS, compression socks; FS, flight socks; NS, no socks; ES, effect size; S, small practical effect; M, medium practical effect; L, large practical effect; VL, very large practical effect

2. Metabolic data

Figure 6.4. to Figure 6.6. illustrate the average values in various metabolic variables over 2 km intervals during the 10 km run test. There was no statistically significant difference in the carbon dioxide production (VCO_2) ($p = 0.12$) over the course of the 10 km run between the three conditions, however, oxygen consumption (VO_2) showed a strong tendency ($p = 0.06$) to change significantly over time between the three conditions.

For both VO_2 (Figure 6.4.) and VCO_2 (Figure 6.5.) there was a trend for higher values during the first four km for the CS and FS conditions compared to the NS condition ($p = 0.06$). Overall, only small practically significant differences (lower values) were observed between the VO_2 values for the NS trial compared to the socks trials (Table 6.10.). Similarly, only small practically significant differences (lower values) were observed for VCO_2 over the first 6 km in the NS trial compared to the socks trials (Figure 6.11.).

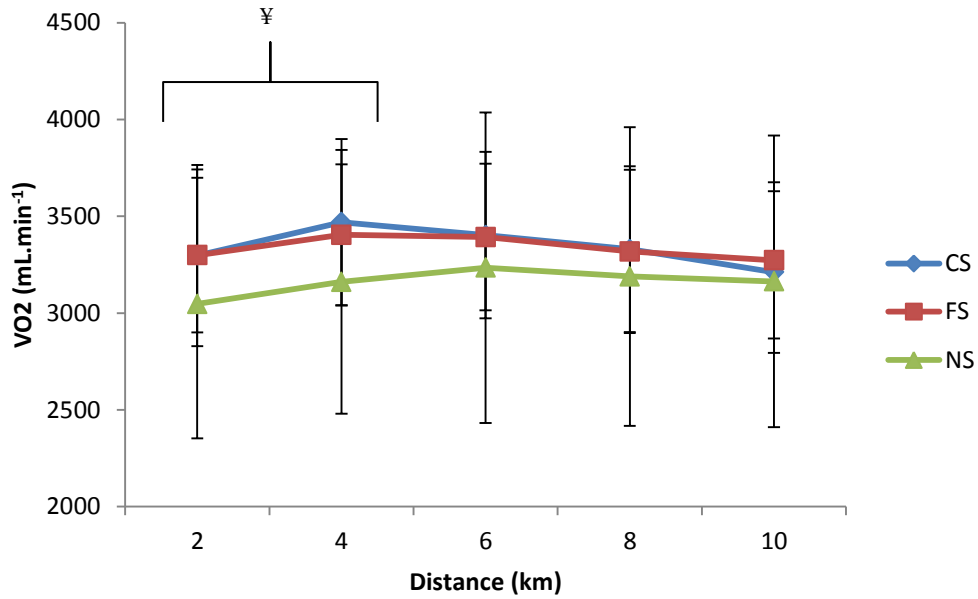


Figure 6.4. Average oxygen consumption (mL.min⁻¹) over 2 km intervals during the 10 km running protocol.
 ¥ Tendency for significant difference between NS and both CS and FS ($p = 0.06$).

Table 6.10. Cohen's effect size results for VO₂ during the 10 km run protocol between the different compression conditions.

		CS	FS	NS	ES
2 km	CS			0.56	S
	FS	0.09			
	NS		0.50		S
4 km	CS			0.68	M
	FS	0.23			S
	NS		0.51		S
6 km	CS			0.32	S
	FS	0.03			
	NS		0.30		S
8 km	CS			0.24	S
	FS	-0.01			
	NS		0.24		S
10 km	CS			0.11	
	FS	-0.14			
	NS		0.21		S

CS, compression socks; FS, flight socks; NS, no socks; ES, effect size; S, small practical effect; M, medium practical effect

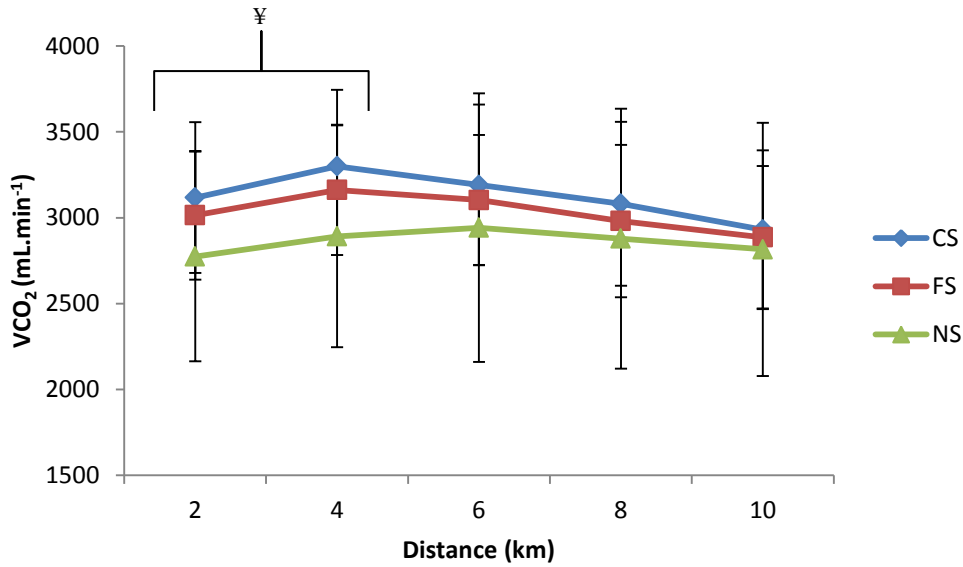


Figure 6.5. Average carbon dioxide production (mL.min⁻¹) over 2 km intervals during the 10 km running protocol.
 ¥ Tendency for significant difference between NS and both CS and FS ($p = 0.06$).

Table 6.11. Results for the Cohen's effect size statistics for VCO₂ during the 10 km run.

		CS	FS	NS	ES
2 km	CS			0.72	M
	FS	0.25			S
	NS		0.53		S
4 km	CS			0.82	L
	FS	0.32			S
	NS		0.57		S
6 km	CS			0.40	S
	FS	0.15			
	NS		0.31		S
8 km	CS			0.29	S
	FS	0.12			
	NS		0.19		
10 km	CS			-0.02	
	FS	-0.02			
	NS		0.13		

CS, compression socks; FS, flight socks; NS, no socks; ES, effect size; S, small practical effect; M, medium practical effect; L, large practical effect

There were no statistically significant differences in the heart rate (*Figure 6.6.*) responses of the participants during the three runs ($p = 0.36$). However, *Table 6.12.* shows that there were some small practically significant differences in HR between the trials, especially during the last 2 km where the HR was higher for the NS trial compared to the socks trials.

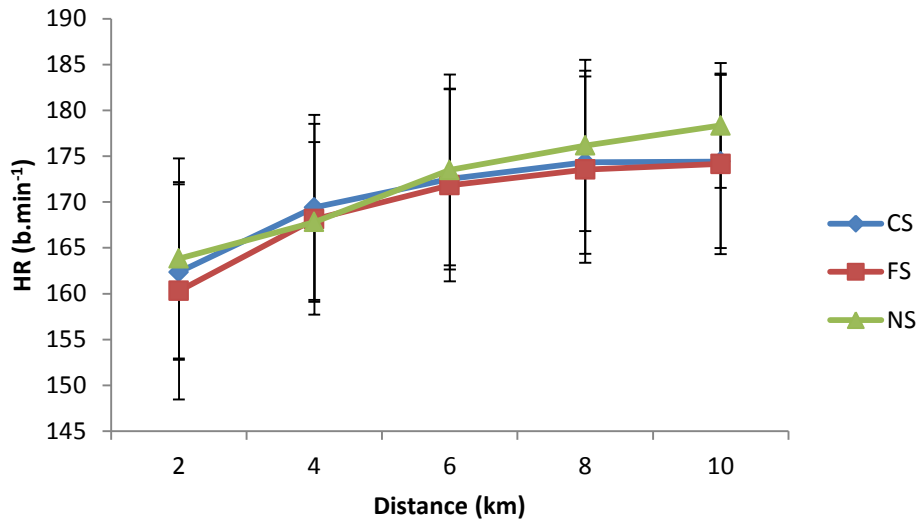


Figure 6.6. Average heart rate (b.min⁻¹) over 2 km intervals during the 10 km running protocol.

Table 6.12. Cohen’s effect size results for heart rate (HR) during the 10 km run protocol between the different compression conditions.

		CS	FS	NS	ES
2 km	CS			-0.15	
	FS	0.21			S
	NS		0.30		S
4 km	CS			0.15	
	FS	0.12			
	NS		0.03		
6 km	CS			-0.27	S
	FS	-0.10			
	NS		0.16		
8 km	CS			-0.33	S
	FS	-0.08			
	NS		-0.26		S
10 km	CS			-0.66	M
	FS	-0.23			S
	NS		0.45		S

CS, compression socks; FS, flight socks; NS, no socks; ES, effect size; S, small practical effect; M, medium practical effect

3. Blood lactate concentration

Figure 6.7. illustrates the changes in blood lactate concentration [BLa] during the 10 km running protocol. Due to the non-Gaussian distribution of the data, log transformations were applied. No statistically significant differences were found between the three trials at any of the time points ($p =$

0.11). The majority of these differences were of small practical significance (*Table 6.14.*). The absolute [BLa] is depicted in *Table 6.13.*

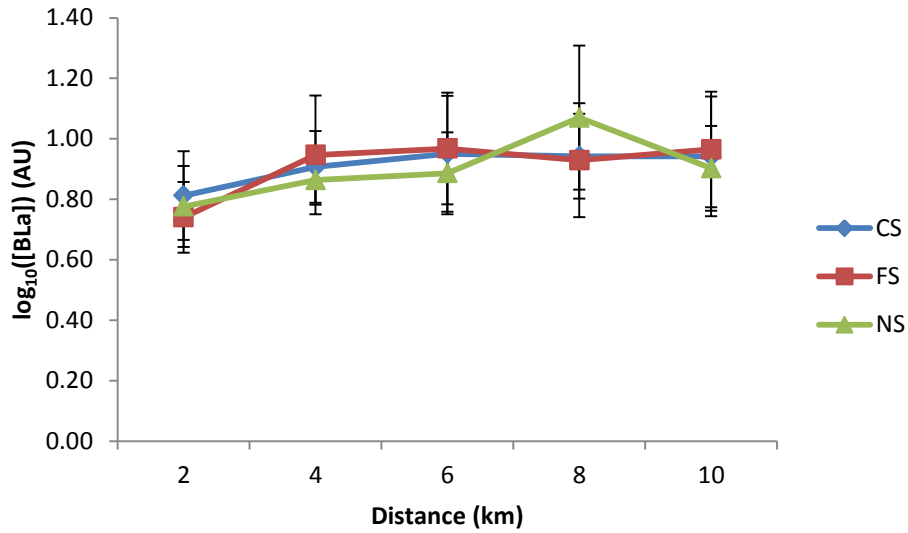


Figure 6.7. Log transformed blood lactate concentrations ([BLa]) during the 10 km running protocol.

Table 6.13. Absolute blood lactate concentration [BLa] (mmol.L⁻¹) during the 10 km run (mean ± SD).

	2 km	4 km	6 km	8 km	10 km
CS	5.9 ± 2.6	7.3 ± 2.2	8.9 ± 5.2	8.1 ± 2.4	8.7 ± 5.1
FS	4.7 ± 1.4	8.8 ± 5.2	9.1 ± 4.9	8.4 ± 5.1	9.2 ± 5.4
NS	5.2 ± 2.1	6.4 ± 1.3	7.0 ± 2.3	12.2 ± 7.3	7.3 ± 2.9

CS, compression socks; FS, flight socks; NS, no socks

Table 6.14. Cohen's effect size results for blood lactate concentrations during the 10 km run protocol.

		CS	FS	NS	ES
2 km	CS			0.25	S
	FS	0.56			S
	NS		-0.33		S
4 km	CS			0.48	S
	FS	-0.36			S
	NS		0.53		S
6 km	CS			0.41	S
	FS	-0.06			S
	NS		0.50		S
8 km	CS			-0.94	L
	FS	-0.06			M
	NS		-0.67		M
10 km	CS			0.29	S
	FS	-0.09			S
	NS		0.38		S

CS, compression socks; FS, flight socks; NS, no socks; ES, effect size; S, small practical effect; M, medium practical effect; L, large practical effect

4. Muscle oxygenation: O₂Hb

Figure 6.8. and *Figure 6.9.* illustrate the changes in O₂Hb measured in the left and right lateral Gastrocnemius. The y-axis depicts the changes in O₂Hb from resting values. In other words, negative values indicate a decrease from the resting value, while positive values indicate an increase from the resting value.

No statistically significant differences were found in O₂Hb between the three running tests in the left and right leg ($p = 0.88$). However, as evident from the figures, there were small practically significant differences in oxygen delivery to the muscles of the left leg between NS and FS (NS better), and medium to large practically significant differences in the muscles of the right leg between NS and CS (NS better) (*Table 6.15.*). No clear differences in O₂Hb were observed between FS and CS in either leg.

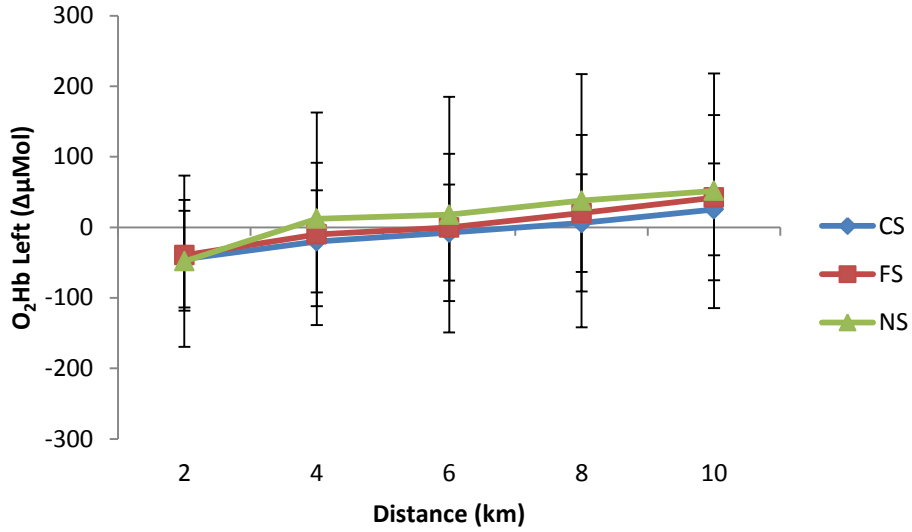


Figure 6.8.

Changes in oxy-haemoglobin (O₂Hb) in the left lateral Gastrocnemius during the 10 km running protocol.

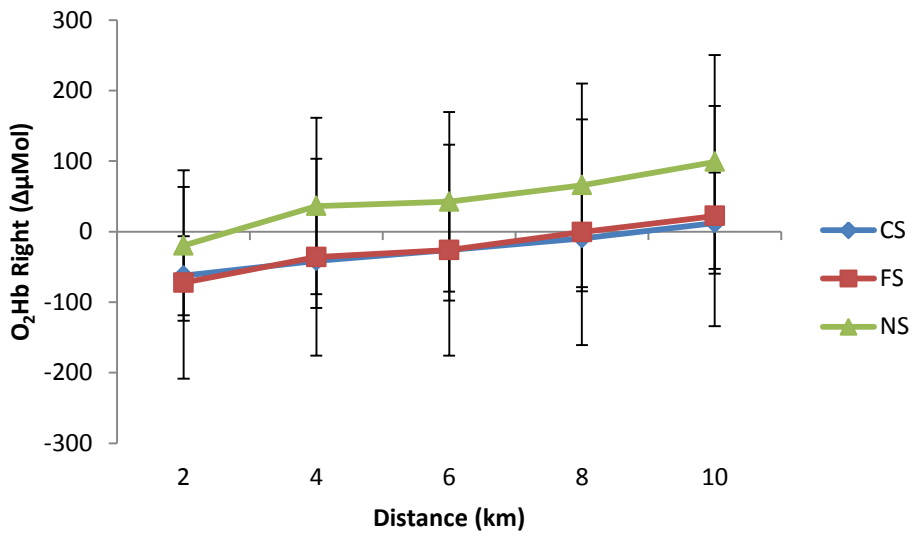


Figure 6.9.

Changes in oxy-haemoglobin (O₂Hb) in the right lateral Gastrocnemius during the 10 km running protocol.

Table 6.15. Cohen's effect size results for O₂Hb during the 10 km run.

		Left				Right			
		CS	FS	NS	ES	CS	FS	NS	ES
2 km	CS			0.04				-0.58	S
	FS	-0.07				0.10			
	NS		0.09				-0.41		S
4 km	CS			-0.19				-0.89	L
	FS	-0.32			S	-0.05			
	NS		0.19				-0.53		S
6 km	CS			-0.24	S			-0.75	M
	FS	-0.08				-0.001			
	NS		-0.14				-0.48		S
8 km	CS			-0.28	S			-0.75	M
	FS	-0.15				-0.09			
	NS		-0.13				-0.43		S
10 km	CS			-0.25	S			-0.86	L
	FS	-0.18				-0.08			
	NS		-0.07				-0.49		S

CS, compression socks; FS, flight socks; NS, no socks; ES, effect size; S, Small practical effect; M, Medium practical effect; L, Large practical effect

5. Muscle oxygenation: HHb

Illustrated in *Figure 6.10.* and *Figure 6.11.* are the changes in HHb for the left and right lateral Gastrocnemius. The y-axis indicates the change in HHb from resting values; negative values indicate a decrease from the resting value, whereas a positive value indicates an increase from the resting value.

Although the HHb values were lower for the NS condition than for the CS and FS conditions, differences between the three trials were not statistically significant ($p = 0.21$). However, Cohen's effect sizes suggest that there were practically significant differences in oxygen consumption levels in the muscles (*Table 6.16.*). For both legs, large practically significant differences were observed between NS and CS, suggesting better oxygen consumption by the muscles with CS. Small to medium practically significant differences were found between NS and FS, also suggesting that the external pressure provided by the FS caused better oxygen consumption. While there were no clear practically significant differences in HHb between FS and CS in the left leg, small effect sizes were observed between FS and CS in the right leg, with higher HHb values associated with the higher pressure socks.

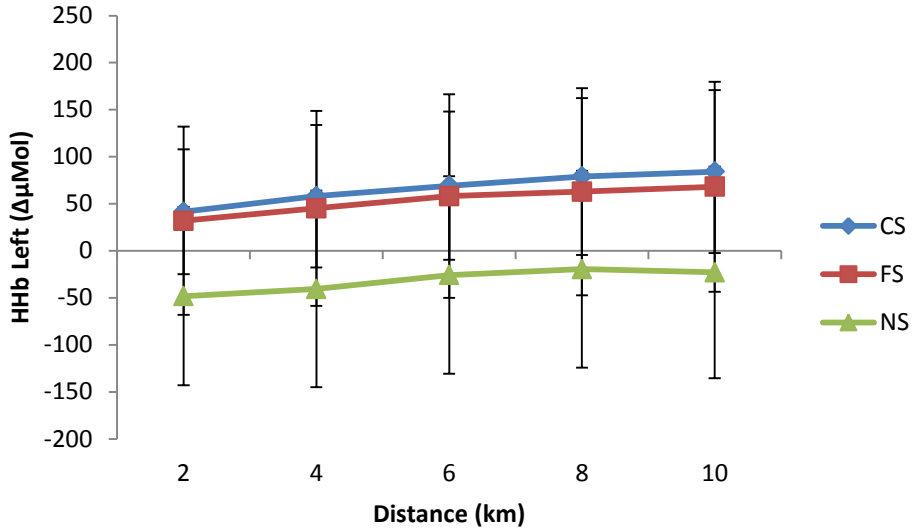


Figure 6.10.

Changes in deoxy-haemoglobin (HHb) in the left lateral Gastrocnemius during the 10 km running protocol.

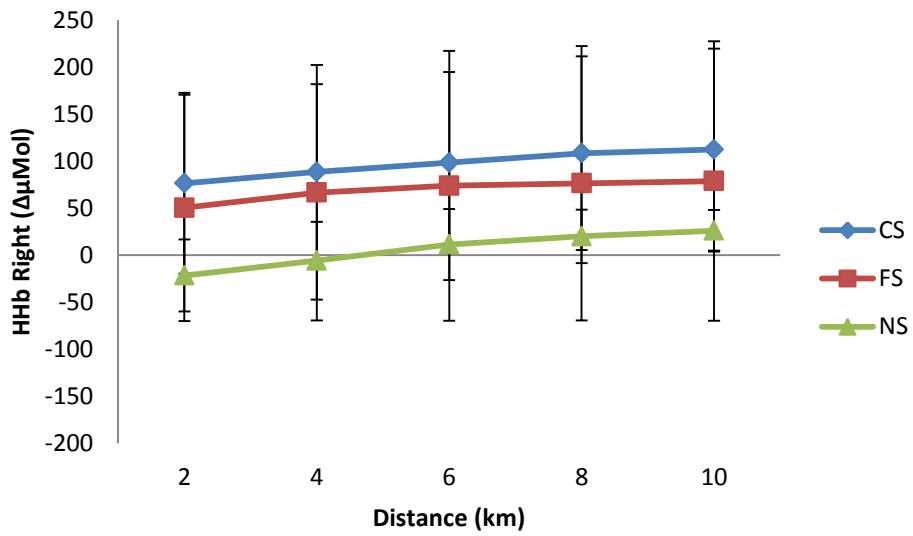


Figure 6.11.

Changes in deoxy-haemoglobin (HHb) in the right lateral Gastrocnemius during the 10 km running protocol.

Table 6.16. Cohen's effect size results for HHb during the 10 km run.

		Left				Right			
		CS	FS	NS	ES	CS	FS	NS	ES
2 km	CS			1.19	L			1.17	L
	FS	0.11				0.24			S
	NS		0.81		L		0.69		M
4 km	CS			1.16	L			1.15	L
	FS	0.14				0.19			
	NS		0.83		L		0.62		M
6 km	CS			1.09	L			1.04	L
	FS	0.12				0.20			S
	NS		0.78		M		0.51		S
8 km	CS			1.10	L			1.00	L
	FS	0.16				0.25			S
	NS		0.76		M		0.45		S
10 km	CS			1.13	L			0.94	L
	FS	0.16				0.28			S
	NS		0.81		M		0.43		S

CS, compression socks; FS, flight socks; NS, no socks; ES, effect size; S, Small practical effect; M, Medium practical effect; L, Large practical effect

6. Muscle oxygenation: TOI

Figure 6.12. and Figure 6.13. illustrates the TOI values (muscle saturation) in the left and right lateral Gastrocnemius during the 10 km running protocol. In both legs the NS condition resulted in higher TOI values, however, these values were not statistically significantly higher than CS and FS ($p = 0.89$). Whereas small practically significant differences were found in the saturation levels of the right leg between FS and NS, these differences tended to be large for the left leg (Table 6.17.) The differences in TOI between CS and NS were generally of medium practical significance for both legs. No clear difference in saturation levels were observed between the two compression conditions.

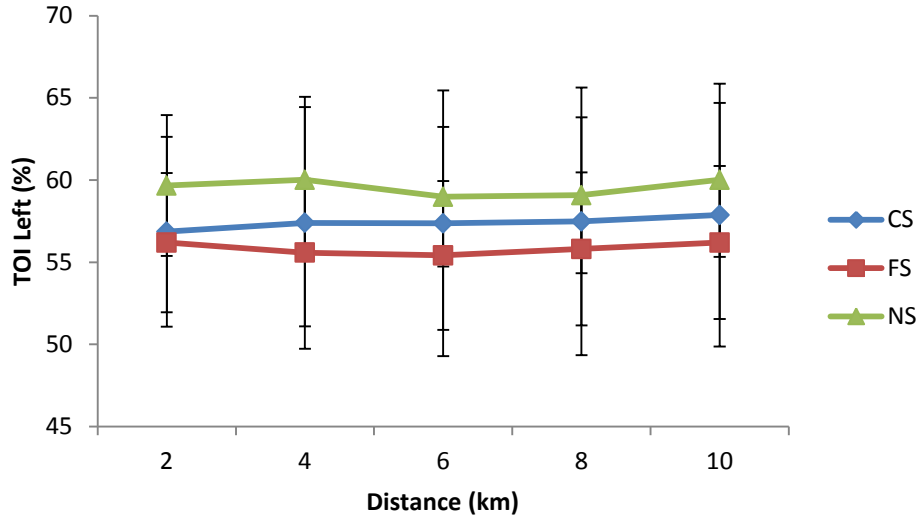


Figure 6.12. Changes in tissue oxygenation index (TOI) in the left lateral Gastrocnemius during the 10 km running protocol.

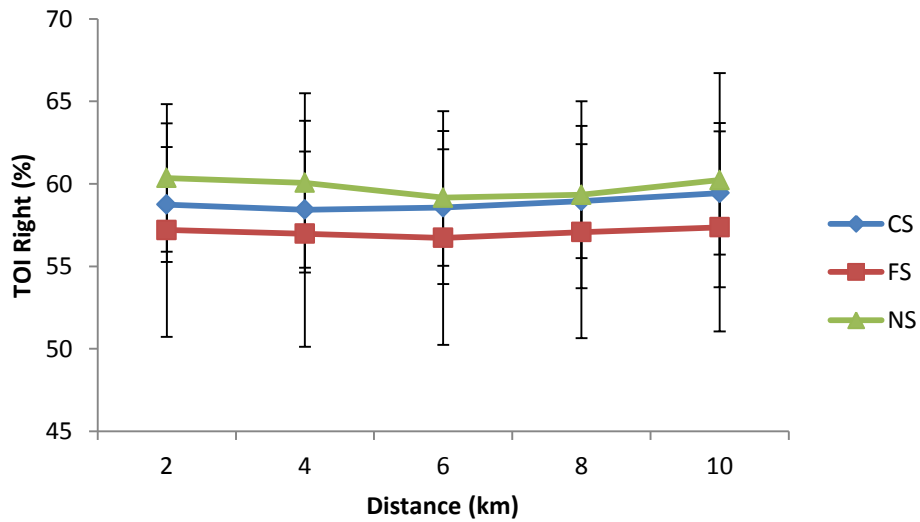


Figure 6.13. Changes in tissue oxygenation index (TOI) in the right lateral Gastrocnemius during the 10 km running protocol.

Table 6.17. Cohen's effect size results for tissue oxygenation index during the 10 km run.

		Left				Right			
		CS	FS	NS	ES	CS	FS	NS	ES
2 km	CS			-0.67	M			-0.75	M
	FS	0.05				0.09			
	NS		-0.81		L		-0.53		S
4 km	CS			-0.59	M			-0.67	M
	FS	0.15				0.07			
	NS		-0.99		L		-0.48		S
6 km	CS			-0.57	M			-0.43	S
	FS	0.16				0.14			
	NS		-1.03		L		-0.40		S
8 km	CS			-0.40	S			-0.38	S
	FS	0.12				0.12			
	NS		-0.70		M		-0.36		S
10 km	CS			-0.47	S			-0.40	S
	FS	0.14				0.20			
	NS		-0.82		L		-0.45		S

CS, compression socks; FS, flight socks; NS, no socks; ES, effect size; S, small practical effect; M, medium practical effect; L, large practical effect

7. Muscle oxygenation: nTHI

Illustrated in *Figure 6.14.* and *Figure 6.15.* are the nTHI measurements in the left and right lateral Gastrocnemius over the 10 km running protocol. These measurements indicate the amount of Hb present in the tissue in arbitrary units (AU). Values for the left leg during the NS condition were lower, but not statistically significantly lower, compared to the CS and FS conditions ($p = 0.99$). These differences were, however, of medium to large practical significance, suggesting that less Hb was present in the muscle of the left leg during the NS trial. On the other hand, nTHI values for the right leg during the CS condition was higher than for the FS and NS condition suggesting more Hb in the muscle with high compression. However, these differences were not statistically significant ($p = 0.99$) and according to *Table 6.18.*, in general, only of small practical significance.

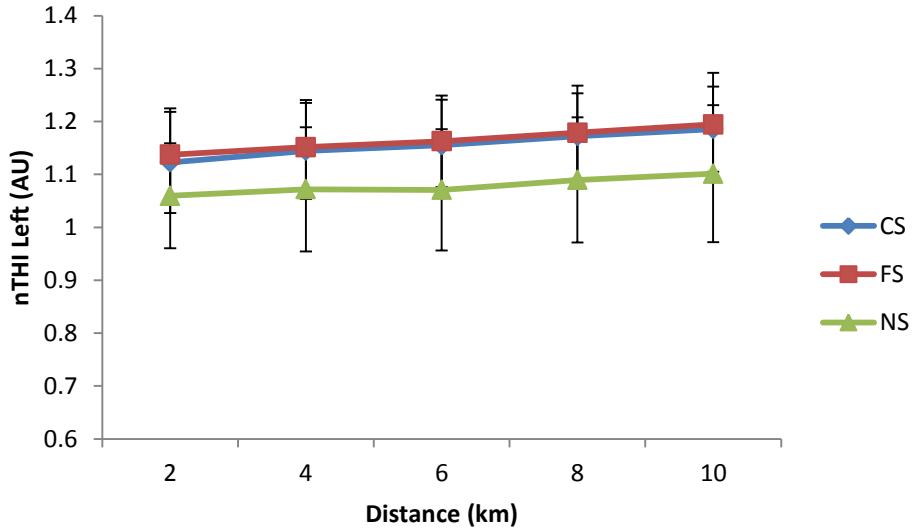


Figure 6.14. Changes in total haemoglobin index (nTHI) in the left lateral Gastrocnemius during the 10 km running protocol.

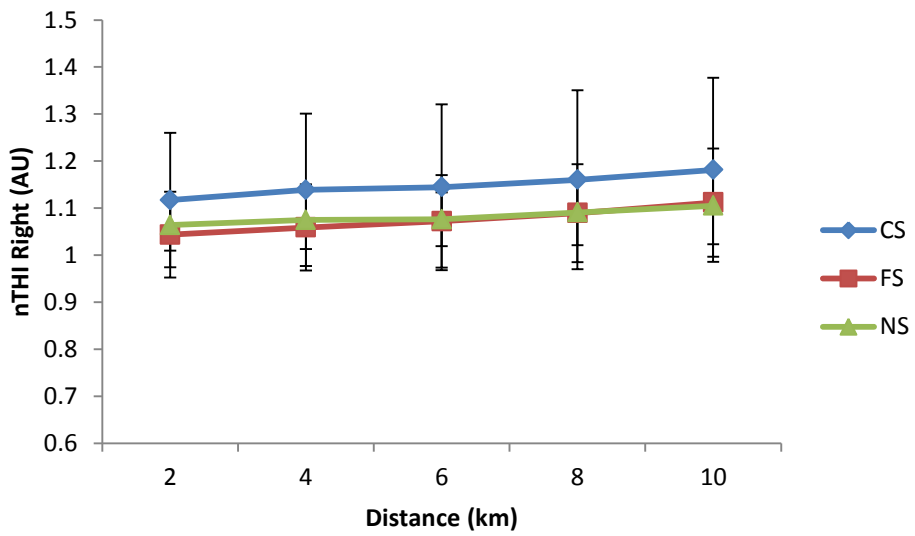


Figure 6.15. Changes in total haemoglobin (nTHI) in the right lateral Gastrocnemius during the 10 km running protocol.

Table 6.18.

Cohen's effect size results for nTHI between the different compression conditions during the 10 km run.

		Left				Right			
		CS	FS	NS	ES	CS	FS	NS	ES
2 km	CS			0.60	M			0.49	S
	FS	-0.21			S	0.68			M
	NS		0.86		L		-0.25		S
4 km	CS			0.70	M			0.50	S
	FS	-0.11				0.85			L
	NS		0.80		L		-0.12		
6 km	CS			0.94	L			0.39	S
	FS	0.00				0.48			S
	NS		0.94		L		-0.12		
8 km	CS			0.86	L			0.42	S
	FS	-0.12				0.46			S
	NS		0.86		L		0.00		
10 km	CS			0.93	L			0.98	L
	FS	0.00				0.42			S
	NS		0.93		L		0.91		L

CS, compression socks; FS, flight socks; NS, no socks; ES, effect size

8. Muscle oxygenation: Change from rest to end of exercise

The general trend in oxygenation for each condition was calculated as the changes between the pre-exercise resting values and the average for the last 2 km of the 10 km run (*Table 6.19*). A positive value indicates that there was a decrease in the variable over 10 km, while a negative value indicates an increase in the variable over 10 km.

For both the left and right leg, there was a greater increase in O₂Hb during the NS condition compared to CS and FS, while HHb tended to decrease during the NS condition compared to the increases during CS and FS. In total, the changes in oxygenation values were not statistically significantly different between any of the conditions ($p > 0.05$). However, moderate to large practical differences were observed between the NS and both sock conditions in all variables except for O₂Hb in the left leg, and nTHI in the right leg.

Table 6.19. Change in oxygenation values from rest to end of 10 km run (mean ± SD).

	Left			Right		
	CS	FS	NS	CS	FS	NS
O ₂ Hb	-6.18 ± 46.29	-20.70 ± 86.67	-54.40 ± 150.80	9.75 ± 76.27	-2.18 ± 139.80	-106.00 ± 178.40
HHb	-74.70 ± 98.79	-74.20 ± 101.50	14.01 ± 52.78	-96.70 ± 88.30	-57.80 ± 128.10	11.54 ± 14.37
TOI	8.49 ± 4.32	7.92 ± 4.37	4.72 ± 4.16	7.17 ± 2.60	7.99 ± 5.28	2.87 ± 6.73
nTHI	-0.19 ± 0.09	-0.20 ± 0.09	-0.12 ± 0.09	-0.14 ± 0.20	-0.1 ± 0.11	-0.08 ± 0.11

CS, compression socks; FS, flight socks; NS, no socks; O₂Hb, oxy-haemoglobin; HHb, deoxy-haemoglobin; TOI, tissue oxygenation index; nTHI, total haemoglobin index

Table 6.20. Cohen’s effect size results for the change from rest to end of exercise in NIRS variables.

		Left				Right			
		CS	FS	NS	ES	CS	FS	NS	ES
O ₂ Hb	CS			0.54	S			1.01	L
	FS	0.21			S	0.11			
	NS		0.31		S		0.68		M
HHb	CS			-1.01	L			-1.44	VL
	FS	-0.01			L	-0.35			S
	NS		-0.98		L		-0.64		M
TOI	CS			0.88	L			1.02	L
	FS	0.13			M	-0.20			S
	NS		0.74		M		0.89		L
nTHI	CS			-0.78	M			-0.34	S
	FS	0.11			L	-0.25			S
	NS		-0.89		L		-0.18		

CS, compression socks; FS, flight socks; NS, no socks; O₂Hb, oxy-haemoglobin; HHb, deoxy-haemoglobin; TOI, tissue oxygenation index; nTHI, total haemoglobin index; S, small practical effect; M, medium practical effect; L, large practical effect; VL, very large practical effect

C. COMPARISON IN VARIABLES DURING RECOVERY

After the completion of the 10 km running protocol, skin temperature, blood lactate concentrations and NIRS measurements were recorded during a 60 minute recovery period, while participants sat comfortably in a chair. The pressure condition during the run test was sustained throughout the recovery period.

1. Skin temperature (ST)

The average ST over 10 min intervals is depicted in *Table 6.21*. No significant differences were found between the conditions at any of the specific time points ($p = 0.75$). However, on average, ST was statistically significantly higher in the left leg over the complete 60 min recovery period after the NS trial, compared to CS (CS, 29.55 ± 1.53 °C vs. NS, 29.92 ± 1.72 °C, $p = 0.04$) and FS (FS, 29.49 ± 1.53 vs. NS, 29.92 ± 1.72 °C, $p = 0.04$). ST was also significantly higher during recovery in the left leg after CS compared to FS (CS, 29.55 ± 1.53 °C vs. FS, 29.49 ± 1.53 °C, $p = 0.04$). In the right leg, statistically significant differences were found between CS and NS (CS, 30.03 ± 1.66 °C vs. NS, 30.28 ± 1.43 °C, $p = 0.04$), and FS and NS (30.03 ± 1.74 °C vs. 30.28 ± 1.43 °C, $p = 0.04$). In general, however, Cohen's effect sizes showed that these differences were either of no practical significance or, at most, a small practical difference (*Table 6.22*).

Table 6.21. Calf skin temperature during the 60 min recovery period.

	Time (min)	CS (°C)	FS (°C)	NS (°C)
Left	10	31.5 ± 1.8	31.6 ± 1.4	31.8 ± 0.9
	20	30.5 ± 1.6	30.6 ± 1.3	30.7 ± 0.9
	30	29.9 ± 1.7	29.8 ± 1.5	30.3 ± 0.9
	40	29.4 ± 2.0	29.3 ± 1.6	29.9 ± 1.0
	50	29.0 ± 2.0	29.0 ± 1.7	29.6 ± 1.0
	60	27.0 ± 2.3	26.5 ± 2.6	27.2 ± 1.0
Right	10	32.6 ± 1.7	32.5 ± 1.6	32.1 ± 2.0
	20	31.3 ± 1.7	31.3 ± 1.7	31.0 ± 1.9
	30	30.6 ± 1.7	30.6 ± 1.7	30.5 ± 1.3
	40	30.0 ± 2.0	30.1 ± 1.7	30.2 ± 1.1
	50	29.7 ± 2.1	30.0 ± 1.8	30.1 ± 0.9
	60	27.7 ± 2.7	27.3 ± 2.7	27.8 ± 0.7

CS, compression socks; FS, flight socks; NS, no socks

Table 6.22. Cohens's effect size results for temperature during the 60 min recovery period.

		Left				Right			
		CS	FS	NS	ES	CS	FS	NS	ES
10 min	CS			-0.15				0.28	S
	FS	-0.06				0.04			
	NS		-0.12				0.25		S
20 min	CS			-0.19				0.16	
	FS	-0.08				-0.02			
	NS		-0.13				0.19		
30 min	CS			-0.26	S			0.07	
	FS	0.02				-0.01			
	NS		-0.26		S		0.08		
40 min	CS			-0.30	S			-0.29	S
	FS	0.04				-0.08			
	NS		-0.30		S		-0.25		S
50 min	CS			-0.34	S			-0.21	S
	FS	-0.02				-0.16			
	NS		-0.34		S		-0.05		
60 min	CS			-0.08				-0.05	
	FS	0.20				0.13			
	NS		-0.08				-0.20		S

CS, compression socks; FS, flight socks; NS, no socks; ES, effect size; S, small practical effect

2. Blood lactate concentration

Figure 6.16. illustrates the log transformed data for [BLa] (AU) over the 60 min recovery period. Absolute [BLa] values are given in Table 6.23. No statistically significant differences were found between the three conditions at any of the time points ($p = 0.29$), and in the majority of cases the differences were only of small practical significance (Figure 6.24.) The [BLa] at the end of the recovery period were within resting ranges, although 90 % of the participants during CS, 54 % during FS, and 60 % during NS had [BLa] greater than their resting values.

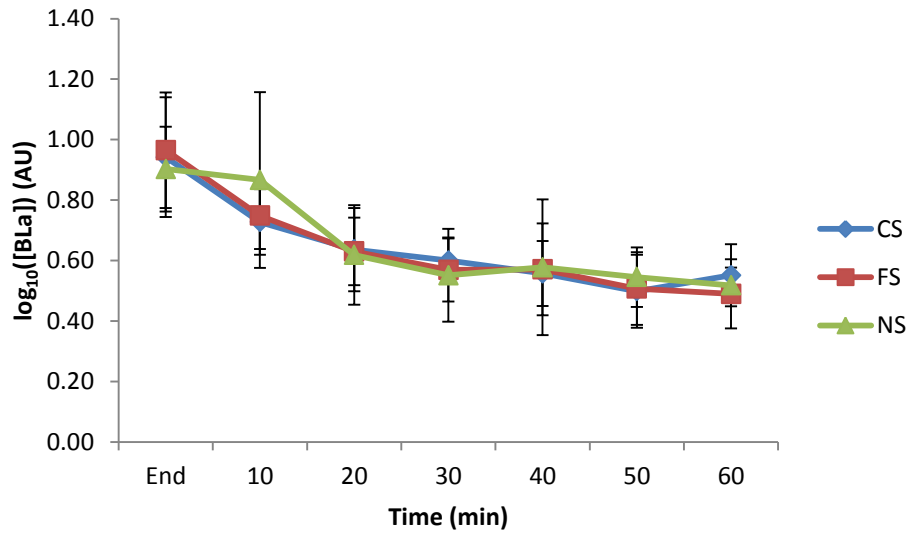


Figure 6.16. Blood lactate concentration ([BLa]) (AU) during recovery.

Table 6.23. Absolute blood lactate concentrations ([BLa]) (mmol.L⁻¹) during the 60 min recovery.

	10 min	20 min	30 min	40 min	50 min	60 min
CS	4.5 ± 1.4	3.5 ± 1.5	3.0 ± 0.7	2.7 ± 0.9	2.2 ± 0.9	2.7 ± 1.0
FS	4.8 ± 1.4	3.4 ± 1.0	2.8 ± 1.0	2.9 ± 1.2	2.5 ± 0.9	2.2 ± 0.8
NS	7.8 ± 5.8	3.2 ± 1.3	2.8 ± 1.5	3.3 ± 2.9	2.6 ± 0.8	2.3 ± 0.5

CS, compression socks; FS, flight socks; NS, no socks

Table 6.24. Cohen's effect sizes for blood lactate concentration values ([BLa]) during the 60 min recovery period.

		CS	FS	NS	ES
10 min	CS			-1.00	L
	FS	-0.20			S
	NS		-0.92		L
20 min	CS			0.23	S
	FS	0.12			
	NS		0.17		
30 min	CS			0.27	S
	FS	0.27			S
	NS		0.04		
40 min	CS			-0.34	S
	FS	-0.19			
	NS		-0.21		S
50 min	CS			-0.37	S
	FS	-0.11			
	NS		-0.26		S
60 min	CS			0.39	S
	FS	0.54			S
	NS		-0.20		S

CS, compression socks; FS, flight socks; NS, no socks; ES, effect size; S, small practical effect; L, large practical effect

3. Muscle oxygenation: O₂Hb

Illustrated in *Figure 6.17.* and *Figure 6.18.* are the changes in O₂Hb during the recovery period relative to the baseline measurement. The y-axis indicates the changes in O₂Hb from resting values, in other words, negative values indicate a decrease from the resting value, while positive values indicate an increase from the resting value. No statistically significant differences were found between the different conditions at any of the time points ($p = 0.92$). Similarly, Cohen's effect sizes varied from no clear practically significant difference to small practically significant differences in O₂Hb between NS, CS and FS (*Table 6.25.*).

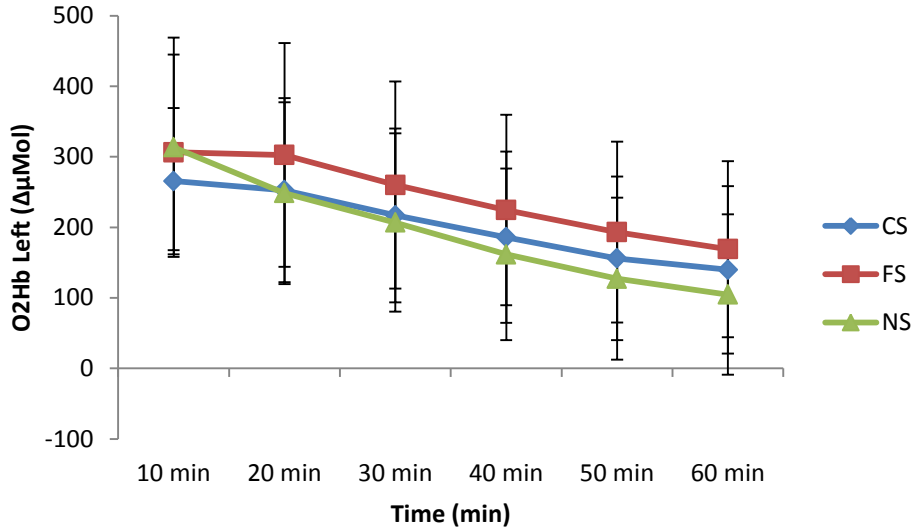


Figure 6.17. Changes in oxy-haemoglobin (O₂Hb) during the 60 min recovery period in the left lateral Gastrocnemius.

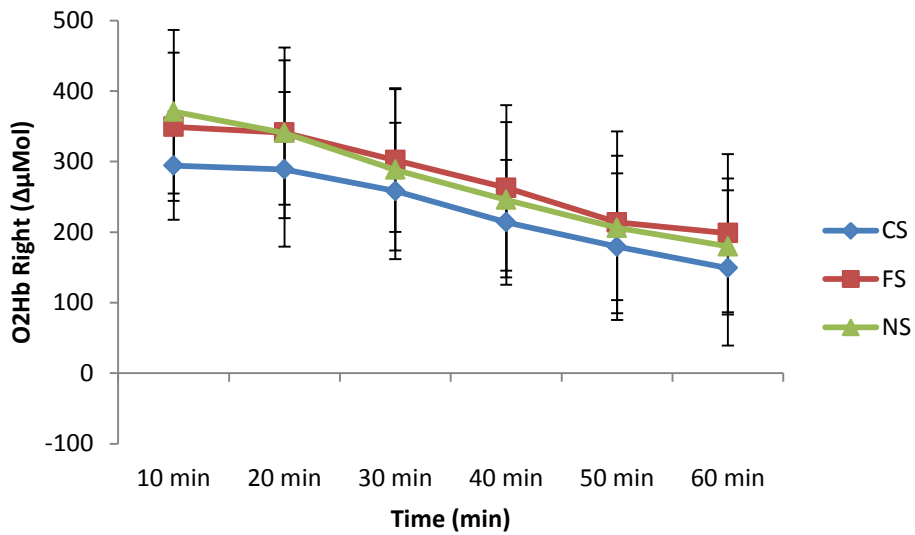


Figure 6.18. Changes in oxy-haemoglobin (O₂Hb) during the 60 min recovery period in the right lateral Gastrocnemius.

Table 6.25. Cohen's effect size results for O₂Hb during the 60 min recovery period.

		Left				Right			
		CS	FS	NS	ES	CS	FS	NS	ES
10 min	CS			-0.40	S			-0.85	L
	FS	-0.33			S	-0.60			M
	NS		-0.05				-0.20		S
20 min	CS			0.03				0.46	S
	FS	-0.34			S	-0.49			S
	NS		0.36		S		0.003		
30 min	CS			0.08				-0.30	S
	FS	-0.32			S	-0.44			S
	NS		0.38		S		0.13		
40 min	CS			0.20	S			-0.33	S
	FS	-0.30			S	-0.47			S
	NS		0.48		S		0.15		
50 min	CS			0.25	S			-0.26	S
	FS	-0.31			S	-0.29			S
	NS		0.53		S		0.04		
60 min	CS			0.30	S			-0.29	S
	FS	-0.24			S	-0.45			S
	NS		0.53		S		0.18		

CS, compression socks; FS, flight socks; NS, no socks; ES, effect size; S, Small practical effect; M, Medium practical effect; L, Large practical effect

4. Muscle oxygenation: HHb

Figure 6.19. and Figure 6.20. illustrates the changes in HHb over the 60 min recovery period relative to baseline. The y-axis indicates the changes in HHb from resting values. Thus, negative values indicate a decrease from the resting value, while positive values indicate an increase from the resting value. No statistically significant differences were found between the three conditions at any of the time points ($p = 0.22$). In the left leg, the differences between the trials were mainly of small practical significance (Table 6.26.). Similarly, in the right leg, differences between CS and FS, and FS and NS were of small practical significance. However, larger Cohen's effect sizes were observed for the differences between CS and NS in the right leg, suggesting greater deoxygenation effects after the CS trial.

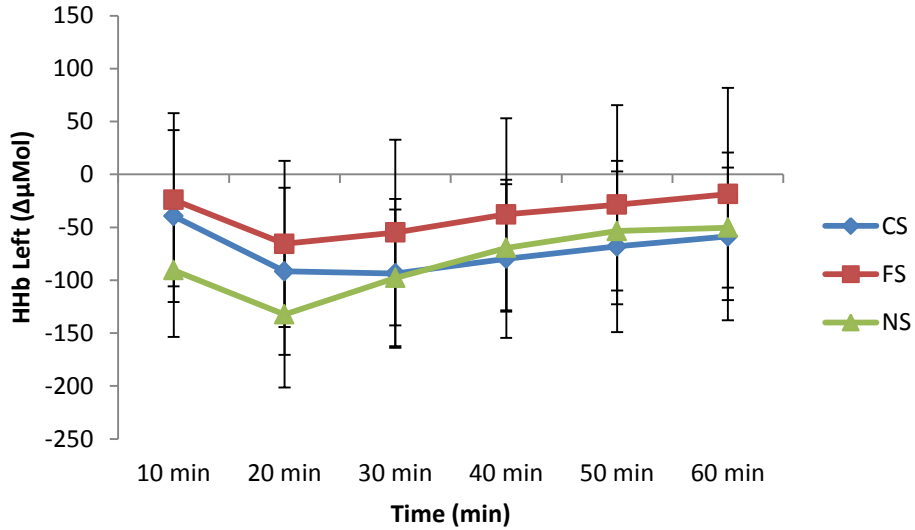


Figure 6.19. Change in deoxy-haemoglobin (HHb) during the 60 min recovery period in the left lateral Gastrocnemius.

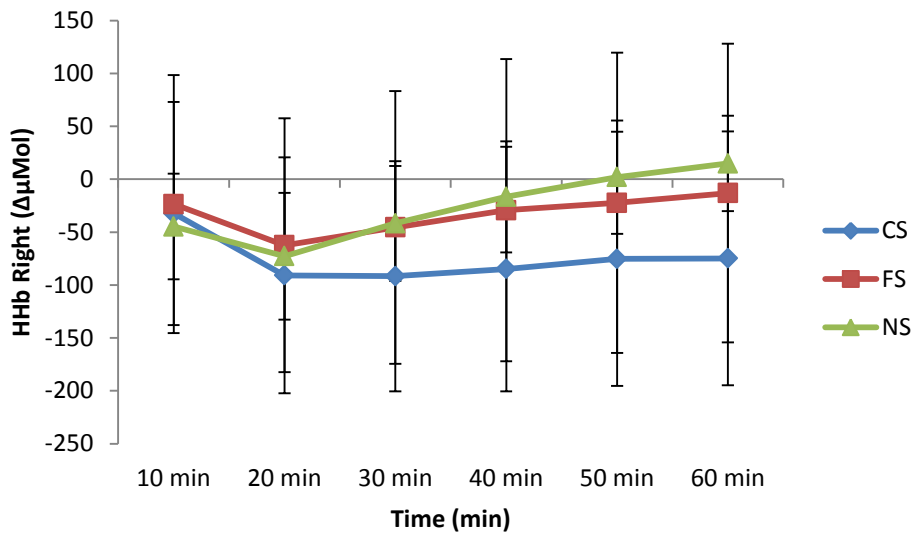


Figure 6.20. Change in deoxy-haemoglobin (HHb) during the 60 min recovery period in the right lateral Gastrocnemius.

Table 6.26. Cohen's effect size results for HHb during the 60 min recovery period.

		Left				Right			
		CS	FS	NS	ES	CS	FS	NS	ES
10 min	CS			0.67	M			0.13	
	FS	-0.19				-0.80			L
	NS		0.87		L		0.20		S
20 min	CS			0.54	S			-0.18	
	FS	-0.33			S	-0.25			S
	NS		0.88		L		0.10		
30 min	CS			0.06				-0.52	S
	FS	-0.49			S	-0.39			S
	NS		0.53		S		-0.03		
40 min	CS			-0.15				-0.67	M
	FS	0.50			S	-0.43			S
	NS		0.38		S		-0.10		
50 min	CS			-0.19				-0.73	M
	FS	-0.45			S	-0.40			S
	NS		0.29		S		-0.20		S
60 min	CS			-0.11				-0.86	L
	FS	-0.44			S	-0.47			S
	NS		0.35		S		-0.23		S

CS, compression socks; FS, flight socks; NS, no socks; ES, effect size; S, Small practical effect; M, Medium practical effect; L, Large practical effect

5. Muscle oxygenation: TOI

As indicated in *Figure 6.21.* and *Figure 6.22.* no statistically significant differences were found in TOI between the different conditions ($p = 0.99$) during the recovery period. In the majority of cases, there were only small practically significant differences in saturation levels in both legs during recovery after exercise (*Table 6.27.*).

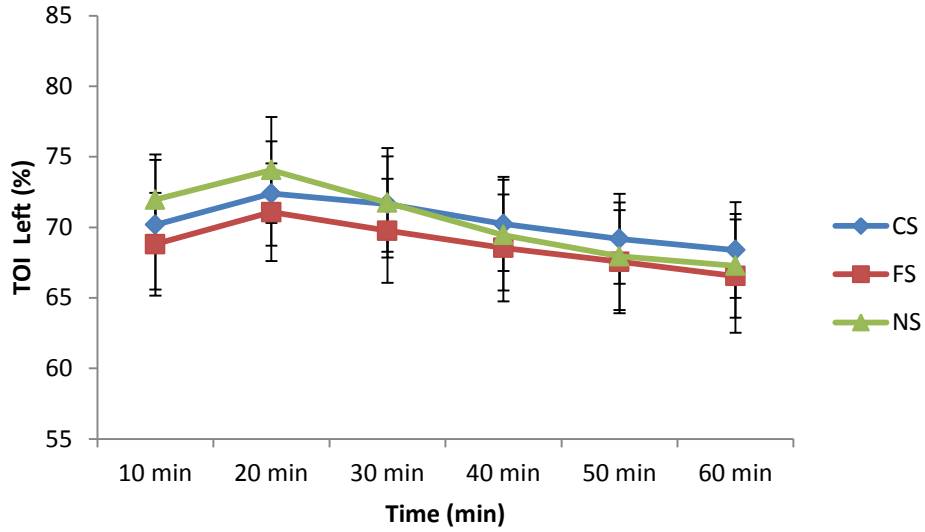


Figure 6.21. Changes in tissue oxygenation index (TOI) during the 60 min recovery period in the left lateral Gastrocnemius.

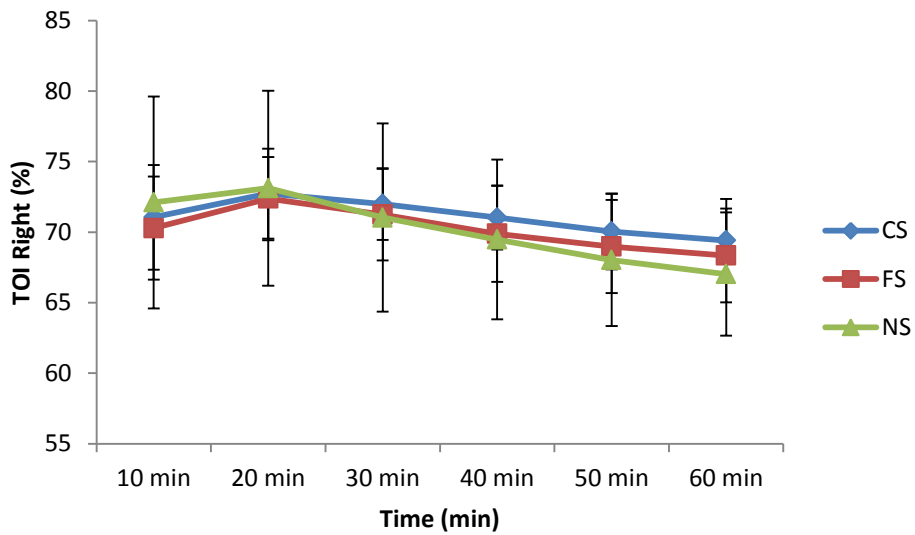


Figure 6.22. Changes in tissue oxygenation index (TOI) during the 60 min recovery period in the right lateral Gastrocnemius.

Table 6.27. Cohen's effect size results for TOI during the 60 min recovery.

		Left				Right			
		CS	FS	NS	ES	CS	FS	NS	ES
10 min	CS			-0.45	S			-0.21	S
	FS	0.32			S	0.21			S
	NS		-0.96		L		-0.36		S
20 min	CS			-0.45	S			-0.08	
	FS	0.37			S	0.11			
	NS		-0.84		L		-0.15		
30 min	CS			-0.03				0.23	S
	FS	0.54			S	0.26			S
	NS		-0.65		M		0.05		
40 min	CS			0.23	S			0.44	S
	FS	0.48			S	0.40			S
	NS		-0.24		S		0.10		
50 min	CS			0.37	S			0.60	M
	FS	0.47			S	0.36			S
	NS		-0.10				0.25		S
60 min	CS			0.32	S			0.70	M
	FS	0.50			S	0.34			S
	NS		-0.18				0.35		S

CS, compression socks; FS, flight socks; NS, no socks; ES, effect size; S, Small practical effect; M, Medium practical effect; L, large practical effect

6. Muscle oxygenation: nTHI

There were no significant differences ($p = 0.66$) in the nTHI values between the different compression conditions during the recovery period (see *Figure 6.23.* and *Figure 6.24.*). Cohen's effect sizes (*Table 6.28.*) also indicated no clear difference between CS and FS for both left and right legs. However, between CS and NS, and between FS and NS there were large practically significant differences in nTHI in the left leg (NS with less Hb in the muscle tissue), but only small practically significant differences in the right leg (NS with less Hb in the muscle tissue).

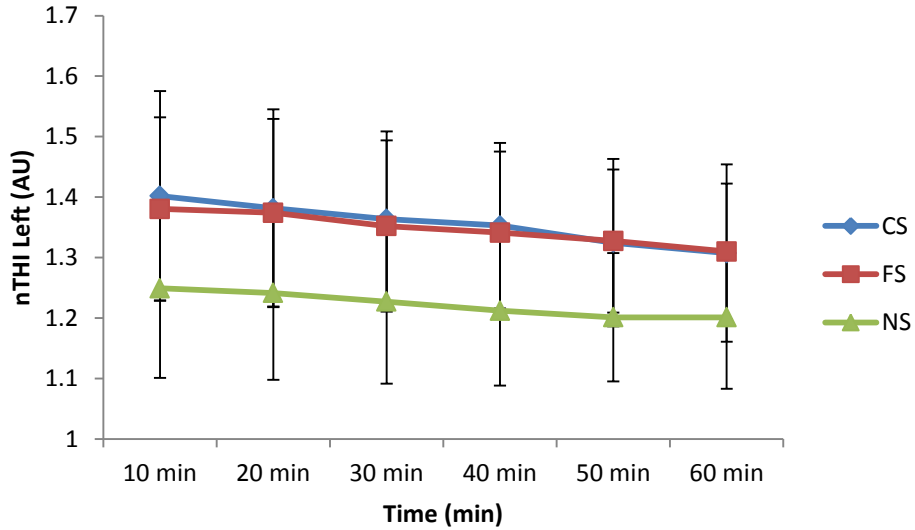


Figure 6.23.

Changes in total haemoglobin index (nTHI) during the 60 min recovery period in the left lateral Gastrocnemius.

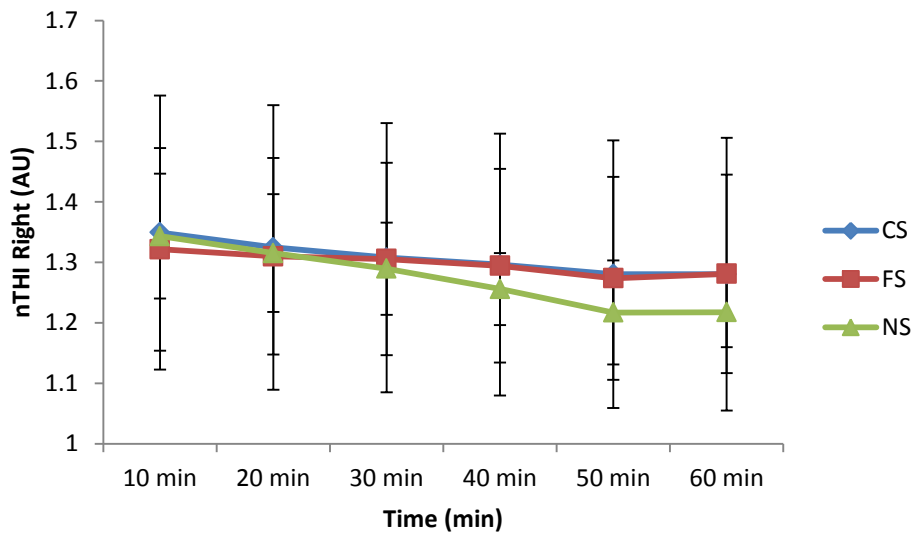


Figure 6.24.

Changes in total haemoglobin index (nTHI) during the 60 min recovery period in the right lateral Gastrocnemius.

Table 6.28. Cohen's effect size results for nTHI during the 60 min recovery period.

		Left				Right			
		CS	FS	NS	ES	CS	FS	NS	ES
10 min	CS			0.91	L			0.05	
	FS	0.12				0.15			
	NS		0.87		L		-0.13		
20 min	CS			0.91	L			0.00	
	FS	0.06				0.05			
	NS		0.88		L		-0.07		
30 min	CS			0.07				0.10	
	FS	0.07				0.00			
	NS		0.00				0.00		
40 min	CS			1.04	L			0.21	S
	FS	0.07				0.05			
	NS		1.02		L		0.22		S
50 min	CS			0.91	L			0.31	S
	FS	-0.08				0.05			
	NS		1.11		L		0.33		S
60 min	CS			0.77	M			0.30	S
	FS	0.00				0.00			
	NS		0.97		L		0.43		S

CS, compression socks; FS, flight socks; NS, no socks; ES, effect size; S, small practical effect; M, medium practical effect; L, large practical effect

7. Muscle oxygenation: Delta calculations post-exercise

The general trend in oxygenation values during the recovery period was also analysed and compared between the three conditions. These changes include: (a) the change in NIRS measurements from the end of exercise to the peak reached after stopping exercise (Delta 2), (b) the change in NIRS measurements from the peak after exercise to the end of the 60 min recovery period (Delta 3), and (c) the change in the NIRS measurements from the end of exercise to the end of the 60 min recovery period (Delta 4) (see *Figure 5.5.*, Chapter Five).

Delta 2 calculations

The means and standard deviations for the Delta 2 calculations are given in *Figure 6.25.*, *Figure 6.26.*, and *Table 6.29.* In these depictions, positive values indicate a decrease in the variable from the end of exercise, whereas negative values indicate an increase in the variable from the end of exercise.

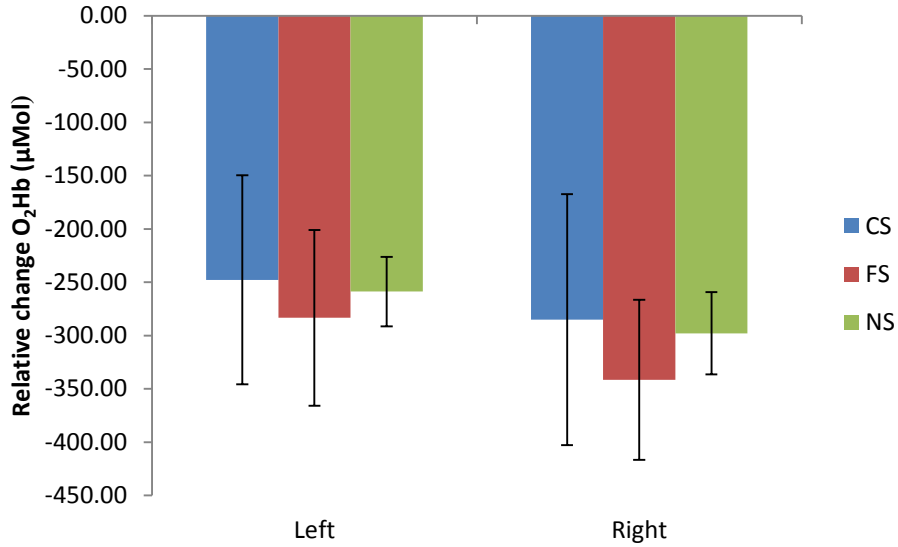


Figure 6.25. Changes in O₂Hb as determined by the Delta 2 calculations.

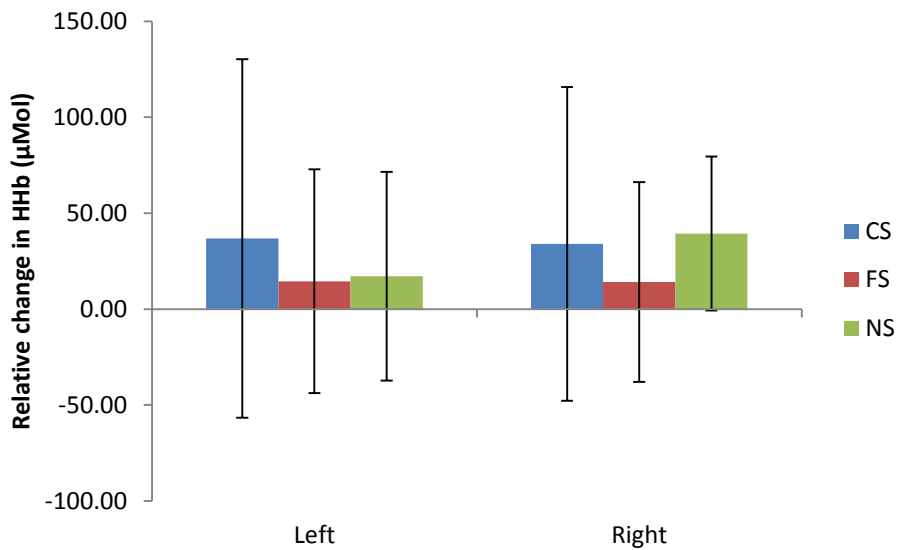


Figure 6.26. Changes in HHb as determined by the Delta 2 calculations.

Table 6.29. Changes ($x \pm SD$) in TOI and nTHI as determined by the Delta 2 calculations.

	CS Left	FS Left	NS Left	CS Right	FS Right	NS Right
TOI	-10.04 ± 5.20	-9.92 ± 5.26	-10.48 ± 2.15	-3.94 ± 17.77	-10.49 ± 4.5	-11.80 ± 2.30
nTHI	-0.14 ± 0.11	-0.09 ± 0.06	-0.12 ± 0.05	-0.13 ± 0.12	-0.15 ± 0.09	-0.18 ± 0.07

CS, compression socks; FS, flight socks; NS, no socks; TOI, tissue oxygenation index; nTHI, total haemoglobin index

No statistically significant differences were found between the different compression conditions for the various variables, although small effect sizes were observed (*Table 6.30.*).

Table 6.30. Cohen's effect size results for the Delta 2 calculations.

		Left				Right			
		CS	FS	NS	ES	CS	FS	NS	ES
O ₂ Hb	CS			0.13				0.13	
	FS	0.39			S	0.57			S
	NS		-0.34		S		-0.65		M
HHb	CS			0.23	S			-0.07	
	FS	0.29			S	0.29			S
	NS		-0.05				-0.52		S
TOI	CS			0.10				0.52	S
	FS	-0.02				0.51			S
	NS		0.1				0.33		S
nTHI	CS			-0.21	S			0.46	S
	FS	-0.56			S	0.19			
	NS		0.52		S		0.35		S

CS, compression socks; FS, flight socks; NS, no socks; O₂Hb, oxy-haemoglobin; HHb, deoxy-haemoglobin; TOI, tissue oxygenation index; nTHI, total haemoglobin index; S, small practical effect; M, medium practical effect

Delta 3 calculations

In *Table 6.30.*, *Figure 6.27.*, and *Figure 6.28.* positive values indicate a decrease in the variable from the peak to the end of exercise, whereas negative values indicate an increase in the variable from the peak to the end of exercise.

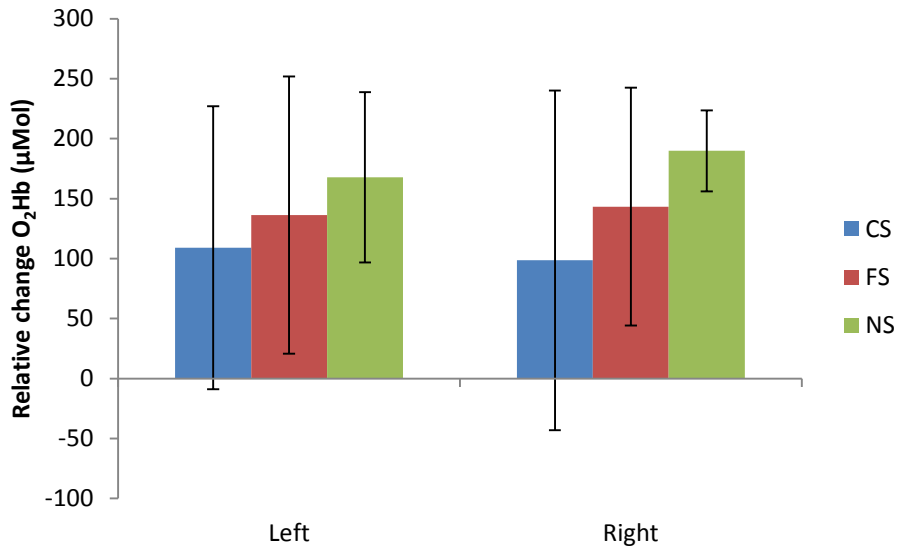


Figure 6.27. Changes in O₂Hb as determined by Delta 3 calculations.

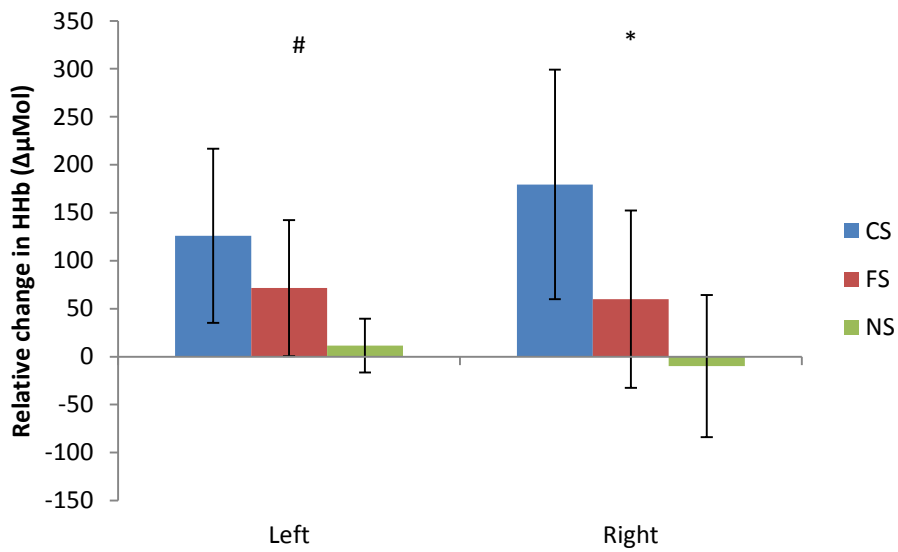


Figure 6.28. Changes in HHb as determined by Delta 3 calculations.
 * Statistically significant difference between the three compression conditions ($p < 0.05$)
 # Statistically significant difference between NS and both CS and FS ($p < 0.05$)

Statistically significant differences were found for the changes in HHb between the different pressure conditions with the Delta 3 calculation. The post hoc tests revealed that the change in NS was statistically significantly smaller than both the CS ($p = 0.04$) and FS ($p = 0.04$) in the left leg. In

the right leg there were statistically significant differences in the changes of all three compression conditions (CS > FS > NS). Effect size results show medium to very large practically significant differences with regard to HHb and small to medium practically significant effects in all other variables (O₂Hb, TOI, nTHI). *Figure 6.27.* and *Figure 6.28.* a clear trend with regard to the pressure applied to the limb.

Table 6.31. Changes ($x \pm SD$) in TOI and nTHI as determined by Delta 3 calculations.

	CS Left	FS Left	NS Left	CS Right	FS Right	NS Right
TOI	-2.43 ± 6.63	-0.54 ± 5.71	1.66 ± 2.60	-8.3 ± 18.84	-0.66 ± 3.68	3.96 ± 40.08
nTHI	0.05 ± 0.15	-0.01 ± 0.01	0.002 ± 0.07	-0.003 ± 0.14	-0.01 ± 0.11	0.06 ± 0.08

CS, compression socks; FS, flight socks; NS, no socks; TOI, tissue oxygenation index; nTHI, total haemoglobin index

Table 6.32. Cohen's effect size results for Delta 3 calculations.

		Left				Right			
		CS	FS	NS	ES	CS	FS	NS	ES
O ₂ Hb	CS			-0.55	S			-0.75	M
	FS	-0.23			S	-0.37			S
	NS		-0.3		S		-0.54		S
HHb	CS			1.46	VL			1.74	VL
	FS	0.67			M	1.12			L
	NS		0.97		L		0.8		L
TOI	CS			-0.71	M			-0.46	S
	FS	-0.31			S	-0.56			S
	NS		-0.44		S		-0.21		S
nTHI	CS			0.36	S			-0.50	S
	FS	0.56			S	0.06			
	NS		-0.31		S		-0.68		M

CS, compression socks; FS, flight socks; NS, no socks; O₂Hb, oxy-haemoglobin; HHb, deoxy-haemoglobin; TOI, tissue oxygenation index; nTHI, total

Delta 4 calculations

In Table 6.33., *Figure 6.29.*, and *Figure 6.30.* positive values indicate a decrease in the variable from the end of exercise to the end of the recovery period, whereas negative values indicate an increase in the variable from the end of exercise to the end of the recovery period. As illustrated in

Figure 6.30. there was a statistically significant difference in the change in HHb between all three of the compression conditions (CS > FS > NS) with the left leg. In the right leg, there was a statistically significant difference in the change calculated with the Delta 4 calculations in HHb between the CS and FS condition ($p = 0.01$), as well as between the CS and NS condition ($p = 0.04$). Large to very large practically significant differences were found for HHb in both the left and right leg. With regard to TOI, a trend with regard to pressure applied to the limb is visible. The strongest effect sizes were observed between CS and NS in both legs.

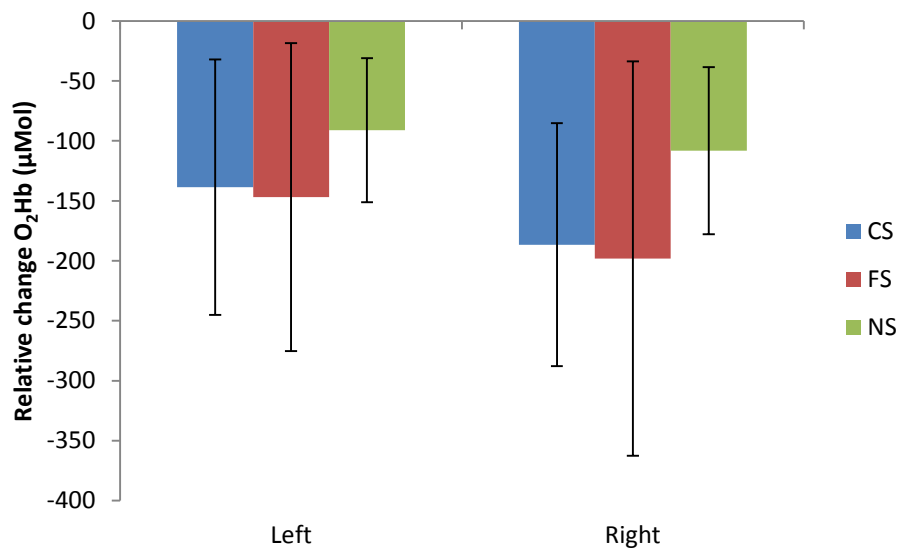


Figure 6.29. Changes in O₂Hb as determined by Delta 4 calculations.

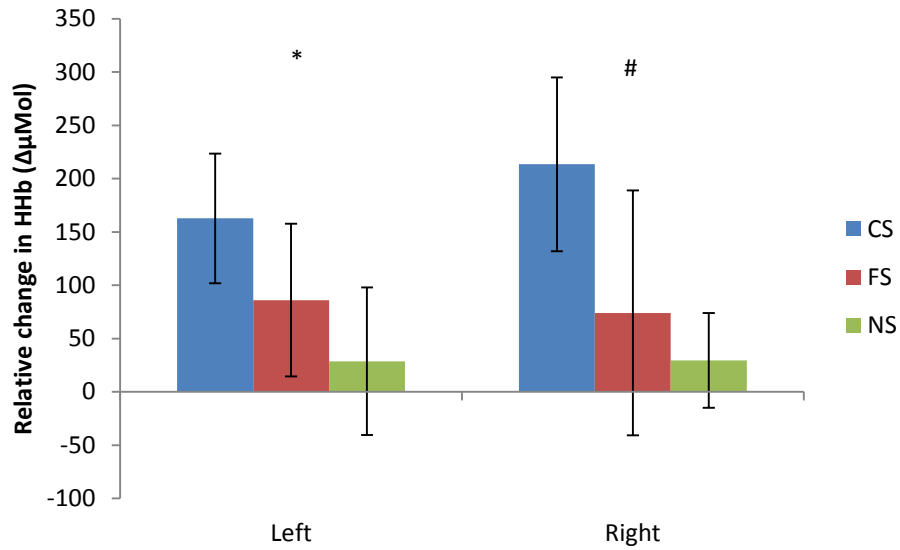


Figure 6.30. Changes in HHb as determined by Delta 4 calculations.
 * Statistically significant difference between all three compression conditions ($p < 0.05$)
 # Statistically significant difference between CS and both the FS and NS ($p < 0.05$)

Table 6.33. Changes ($x \pm SD$) in TOI and nTHI as determined by Delta 4 calculations.

	CS Left	FS Left	NS Left	CS Right	FS Right	NS Right
TOI	-12.46 ± 6.06	-10.45 ± 3.12	-8.82 ± 3.92	-12.24 ± 4.23	-11.15 ± 5.43	-7.84 ± 2.11
nTHI	-0.09 ± 0.08	-0.11 ± 0.06	-0.12 ± 0.04	-0.13 ± 0.12	-0.16 ± 0.14	-0.12 ± 0.04

CS, compression socks; FS, flight socks; NS, no socks; TOI, tissue oxygenation index; nTHI, total haemoglobin index

Table 6.34. Cohen's effect size results for Delta 4 calculations.

		Left				Right			
		CS	FS	NS	ES	CS	FS	NS	ES
O ₂ Hb	CS			-0.50	S			-0.84	L
	FS	0.07				0.08			
	NS		-0.49		S		-0.63		M
HHb	CS			0.97	L			2.52	VL
	FS	1.15			L	1.4			L
	NS		0.81		L		0.45		S
TOI	CS			-0.66	M			-1.17	L
	FS	-0.42			S	-0.22			S
	NS		-0.46		S		-0.70		M
nTHI	CS			0.40	S			-0.10	
	FS	0.28			S	0.23			S
	NS		0.17				-0.33		S

CS, compression socks; FS, flight socks; NS, no socks; O₂Hb, oxy-haemoglobin; HHb, deoxy-haemoglobin; TOI, tissue oxygenation index; nTHI, total haemoglobin index; S, small practical effect; M, medium practical effect; L, large practical effect; VL, very large practical effect

D. CORRELATIONS

Correlations between the anthropometric characteristics and the pressure outcome variables are illustrated in *Table 6.35*. Correlations between ankle girth and ankle pressure for both CS ($r = 0.05$) and FS ($r = -0.24$) displayed weak relationships, with FS showing a negative relationship. With regard to calf girth and posterior pressure in CS and FS, weak relationships were found with CS ($r = 0.06$), with FS ($r = -0.35$) showing a moderate negative relationship. In the case of correlations drawn between the D position and the pressure under the elastic of CS ($r = 0.47$) and FS ($r = -0.36$), moderate relationships were found, with the FS condition once again displaying a negative relationship.

Table 6.35. Correlations between pressure outcome variables and various anthropometric characteristics.

	P Ankle CS	P Ankle FS	P Post CS	P Post FS	P D CS	P D FS
Ankle girth	0.05	-0.24				
Calf girth			0.06	-0.35		
D girth					0.47	-0.36
SF _{Post}	-0.17	-0.09	0.02	-0.58	0.06	-0.21

SF_{Post}, posterior calf skinfold; D girth, girth at the level just below the tibial tuberosity; P Ankle CS, ankle pressure CS; P Ankle FS, ankle pressure FS; P Post CS, posterior calf pressure CS; P Post FS, Posterior calf pressure FS; P D CS, pressure on the posterior calf at the level just below the tibial tuberosity with CS; P D FS, pressure on the posterior calf at the level just below the tibial tuberosity with FS

The correlations between posterior skin fold and the ankle pressure in CS ($r = -0.17$) and FS ($r = -0.09$), as well as the pressure under the elastic in CS ($r = 0.06$) and FS ($r = -0.21$) showed weak correlations. When the posterior calf skinfold was correlated with the posterior calf pressure, a weak relationship was found in CS ($r = 0.02$), whereas FS ($r = -0.58$) displayed a moderate to good negative relationship.

CHAPTER SEVEN

DISCUSSION

A. INTRODUCTION

The purpose of this study was to explore a possible mechanism whereby knee high compression socks (CS) enhance calf muscle function, specifically muscle oxygenation, in endurance trained athletes. This stems from the hypothesis proposed by Berry and McMurray (1987) that CS could enhance lactate removal through increased oxidation within the muscle. A further aim was to determine whether a high pressure gradient sock would elicit a more pronounced effect on muscle oxygenation than a low pressure gradient sock. This study can be subdivided into two parts, namely measurements during exercise and measurements during recovery. For the purpose of this discussion, the results were therefore also divided into these two parts.

The main findings of the exercise part of the study were that both CS (high pressure gradient) and FS (low pressure gradient) cause similarly higher skin temperatures during the first 8 km of a 10 km treadmill run compared to NS. Furthermore, neither high nor low compression influenced systemic [BLa] or tissue oxygenation levels (TOI) during exercise, although greater O₂ utilization (HHb) was observed for both compression conditions.

The main finding of the study for the recovery period was that covering the calf in material did not have an influence on skin temperature. Furthermore, as during exercise, compression did not influence systemic [BLa] or TOI, although a large practical difference was found between the NS and both sock conditions in [BLa] after 10 minutes of recovery.

B. DESCRIPTIVE CHARACTERISTICS

CS were originally designed for patients with venous pathologies, however, in sport, these socks are popular among endurance runners and triathletes.

Participants in the current study were aged between 30 and 40 years, thus representing the population of endurance athletes who, according to the literature, account for the majority of runners in competitive events and perform best in endurance events (Williams et al., 2012). Lepers *et al.* (2010) found that men aged between 30 – 34 years have the best average 10 km run times during a triathlon, followed by younger runners between 18 and 29 years and then older runners in the age range of 35 – 40 years. Conversely, in longer distances, i.e. marathons, older runners between 35 to 40 years perform better than younger runners in the age range of 30 – 34 years. Rüst *et al.* (2012) also found that in a 30 km run during a triathlon, men up to the age of 44 years were still competitive.

Another reason for including this age group (30 – 40 years) in the study is due to the apparent effect of age on the lactate distribution during post-exercise recovery. Tzankoff and Norris (1979) determined that the speed of diffusion and distribution of lactate during recovery decreases with increasing age. They showed that from the age group 30 – 39 years, the appearance of lactate in the blood is delayed, therefore the peak lactate measurement after exercise appears later in the circulation. No clear conclusion could be drawn from the results to explain the possible mechanism for this finding, although it was suggested that differences in blood flow distribution, diffusion of lactate from the muscle or an extended period of increased lactate production post-exercise could possibly explain these findings. Since the aim of the current study was to determine if external compression affects muscle oxygenation and thus lactate removal, it was argued that it would be more likely to see the effects of compression on lactate recovery in this age group, as the lactate would not be redistributed as rapidly as in younger runners.

According to the literature, the runners in this study, which included well trained endurance runners and triathletes, had BMI values within the usual ranges for this population. Hoffman (2008)

reported an average BMI of 23.2 ± 2.3 for men between 30 and 39 years, whereas the average BMI in male triathletes was 24.3 ± 1.7 (Knechtle, Knechtle & Rosemann, 2009). The leanness of the participants was especially important in this study, as subcutaneous tissue thickness affects the NIRS measurements. It has been suggested that the interoptode distance determines the depth of measurement (McCully & Hamaoka, 2000). Therefore, with a four centimetre interoptode distance, as in the current study, measurement depth is two centimetres. Thus if the subcutaneous fat layer is two centimetres thick, the NIRS measurement will be limited to the subcutaneous fat layer. Therefore the criterion was set very strict so as to measure as deeply as possible into the muscle and also to minimise the influence of subcutaneous fat on the measurement. Therefore, one of the inclusion criteria for the study was that participants had a posterior skinfold of ≤ 10 mm.

1. Haemoglobin

Austin *et al.* (2005) suggested that NIRS measurements could possibly be influenced by haemoglobin concentration ([Hb]). The researchers suggested this as a possible factor after finding that adipose tissue thickness is not a confounding factor in the determination of a breakpoint in tissue oxygenation during a maximal exercise test. For this reason, resting pre-exercise [Hb] was measured. The ACSM guidelines (2010) for normative [Hb] in men is between the range of $13.5 - 17.5 \text{ g.dL}^{-1}$. The [Hb] during the three trials all fell within these guidelines with no statistical significance between trials. However, a very large practical effect was found between CS and NS, and a large practical effect was found between FS and NS, with the NS condition measuring greater concentrations in both cases.

This difference was not however shown in the nTHI measurement. However, it should be kept in mind that there is a possible effect of [Hb] differences when analysing the muscle oxygenation data. Higher [Hb], such as in the NS condition, could lead to higher muscle oxygenation levels.

C. PRESSURE PROFILES

Manufacturers claim that the pressure gradients for sport CS range between 8 – 12 mmHg, 12 -16 mmHg, 15 – 20 mmHg, 20 – 30 mmHg and 30 – 40 mmHg (www.compression-socks.com, 2012). However, Lui *et al.* (2005) have disputed these claims, explaining that the morphology of the leg, specifically the subcutaneous fat layer, bone position and muscle size all have an influence on the pressures at different positions around the leg. For instance, at the anterior side of the lower leg, the prominence of the tibia and the amount of subcutaneous fat covering it significantly affects the pressure measurements, as the tibia is an inelastic structure, whereas muscle and fat is elastic. This explains the findings of Maton *et al.* (2006) that anterior pressures in the lower leg are statistically significantly higher than posterior pressures.

In this study, for both CS and FS, the highest pressures were measured at the posterior ankle (circumference was 22.08 ± 0.72 cm) and it decreased in the proximal direction (circumference at the level of the elastic of the sock was 33.48 ± 1.22 cm). This is in accordance with the modified law of Laplace which states that pressure decreases as circumference increases (Troynikov *et al.*, 2010). Both CS and FS can thus be regarded as graduated compression socks.

It was also found that the pressure measurements for CS around the greatest circumference of the calf were not statistically significant from each other, although the lateral and anterior measurements were significantly higher than the measurement at B1 in the CS condition. With the FS, it was found that the posterior, lateral, anterior, and medial sides measured greater pressures than the B1 measurement site. In both cases (CS and FS) it is expected that the measurement at B1 should be greater than the lateral and anterior measurements because of the fact that these are advertised as graduated compression socks, therefore the greater pressure should be exerted distally. However, taking from the research by Liu *et al.* (2005), it could be that the prominence of the tibia on the anterior side and possibly low levels of subcutaneous fat on the lateral side of the calf could lead to higher pressure measurements at these points. Another reason for this discrepancy is that the pressure may be related to the radius of curvature, rather than the absolute circumference of the calf. Support for this argument was found in the weak correlations that were

observed in this study between ankle circumference and posterior ankle pressure, as well as calf girth and posterior calf pressure. It should be remembered that the human leg is not a perfectly round object, but rather, as in the case of the ankle, has an elliptical form (Liu *et al.*, 2005; Maton *et al.*, 2006). Therefore it is suggested that the curvature of the leg at the specific measurement is a more important determinant of the pressure than the circumference of the leg at that point. This will also explain the large intra-individual variations in pressure measurements, especially at site B1, as no two individuals will have exactly the same leg morphology. Since the circumference of the calf is greater than that of the ankle, there is a downward slope from the calf to the ankle. It is within this slope that the B1 measurement site lies. Therefore, in some cases where the ratio between calf and ankle circumference is very large, it is possible that the sock does not make full contact with the skin and therefore not exerting its full pressure.

At all of the measurement sites, CS induced higher pressures than FS. The only exceptions were significant differences at the ankle, point D, and on the anterior calf. All socks were individually fitted for the participants according to the manufacturers' specifications, and therefore significant differences between CS and FS were expected. However, in both cases the highest average pressure measured for the socks was well above the specifications of the manufacturers. Pressures in the CS should have been 20 mmHg at the ankle and 30 mmHg at the calf, however, an average pressure of 69.09 ± 62.76 mmHg was measured. Similarly the FS should have had pressures in the range of 18 to 22 mmHg, but the average pressure was 59.92 ± 72.50 mmHg on the posterior calf. Considering that these measurements were made during quiet standing, it can only be speculated how high the pressures will increase with muscular contraction (i.e. during exercise), as this will cause the tissue to become more rigid.

Hafner *et al.* (2000) found that pressures in excess of 50 mmHg causes occlusions within the underlying tissue. Similarly, Partsch and Partsch (2005) found occlusion to take place at 50 to 60 mmHg. It is therefore possible that the sport CS used and commercially sold could lead to occlusion within the calf in certain individuals, though this cannot be confirmed by the current study. Furthermore, the large inter-individual variation in pressures at the various measurement points is

a major concern, as this could lead to blood flow improvements in one individual, while causing impairment in the other. The safe prescription of CS to athletes is thus problematic, as there is such a wide variety of variables that seem to influence the compression.

Various authors have studied the pressures exerted by a range of CS. These findings are reported in *Table 7.1.* and *Table 7.2.* It is clear that the pressures that were measured in this study at both the ankle and posterior calf are high in comparison to those in the literature. As stated earlier, a possible explanation for the relatively high pressure measurements in this study may be attributed to the small radius of calf curvature of the individuals.

Ferguson-Pell, Hagusawa and Bain (2000) used the same sensor as in the current study, namely the FlexiForce (Tekscan, USA) force sensor, and found that at a radius of curvature smaller than 32 mm, the sensor overestimates the force applied, due to the great level of bending experienced by the sensor. The FlexiForce sensor is a 0.1mm thick laminated polyester sensor, using pressure sensitive ink to determine the force that is exerted on the sensor. The pneumatic sensors used in previous research consist of an inflated balloon connected to a pressure transducer. In this case, the thickness of the sensor will contribute to the circumference of the calf (thus larger circumferences) and this may lead to the underestimation of pressures (Law of Laplace). Thus, further research should be done on the effect of pressure sensor thickness on pressure measurements to enable researchers to compare measurements between different sensors.

Table 7.1. Pressure (mmHg) results for the current study compared to previous studies for measurements on the posterior ankle.

Authors	Pressure range (mmHg)	Comments
Current study CS	41.37 ± 20.96	
Current study FS	23.38 ± 13.01	
Wertheim <i>et al.</i> (1999)	28.5	
Partsch (2004)	5.9 - 21.8	Different CS ranging from low to high compression
Maton (2006)	17.2 ± 3.0	
Liu <i>et al.</i> (2008)	8.17 - 16.3	Different CS ranging from low to high compression
Sperlich <i>et al.</i> (2011)	21.3 - 45.9	Different CS ranging from low to high compression

CS, Compression socks; FS, Flight socks; mmHg, Millimetre Mercury

Table 7.2. Pressure (mmHg) results for the current study compared to previous studies for measurements on the posterior calf.

Authors	Pressure range (mmHg)	Comments
Current study CS	69.09 ± 62.76	
Current study FS	59.92 ± 72.50	
Current study B1 CS	31.06 ± 43.96	
Current study B1 FS	25.12 ± 58.42	
Wertheim <i>et al.</i> (1999)	42	
Maton <i>et al.</i> (2006)	13.9 ± 2.1	
Liu <i>et al.</i> (2008)	6.37 - 15.2	Different CS ranging from low to high compression
Dascombe <i>et al.</i> (2011)	19.2 ± 3.2 21.7 ± 4.3	Regular sized CG Undersized CG
Goh <i>et al.</i> (2011)	13.6 ± 3.4	
Sperlich <i>et al.</i> (2011)	13.6 - 38.8	Different CS ranging from low to high compression

CS, compression socks; FS, Flight socks; CG, Compression garments; mmHg, Millimetre Mercury

D. THE EFFECT OF COMPRESSION ON MUSCLE PHYSIOLOGY DURING EXERCISE

1. Skin temperature

Indications are that local heating of the skin may influence tissue oxygenation (TOI) due to a possible increase in blood flow. Davis *et al.* (2006) found a positive correlation ($r = 0.89$) between tissue oxygenation (TOI) and skin blood flow in five men and three women (Age = 33 ± 3 years) with whole body heating. Along with this a correlation between local heating and skin blood flow

was also found ($r = 0.92$). For this reason skin temperatures (ST) were measured in the current study. Large practical differences in skin temperatures were observed between the runs with socks (both CS and FS) and without socks (NS). These results are in agreement with those of Houghton, Dawson and Maloney (2009) in their study with compression shorts and a short sleeve compression shirt during the Loughborough Intermittent Shuttle Test (LIST).

These higher ST with compression clothing can be explained by its possible insulating effect or the enhancement of wicking. Purvis and Tunstall (2004) suggested that clothing, such as CS or FS, creates a microenvironment where a higher temperature and humidity is present, and therefore insulates the covered area. They also suggested that in high moisture wicking clothing it is possible that the clothing enhances wicking to such an extent that it inhibits the cooling that would normally take place due to sweat evaporation on the skin. This is substantiated by Gavin *et al.* (2001) who showed that clothing specifically made for moisture wicking does not improve thermoregulation more than traditional clothing, or even minimal clothing. Though the socks were not specifically identified as moisture wicking socks (both the CS and FS), it is probable that any of these two hypotheses could serve as an explanation for the greater ST in the CS and FS condition.

Koga *et al.* (1997) determined that when temperature is increased, isolated mitochondria consume more oxygen. They also found a rightward shift in the O_2 Hb dissociation curve, therefore enhancing the dissociation of O_2 from Hb. It could therefore be these changes that facilitate the increase in TOI with elevated ST. However, it is not necessarily true that elevated ST transfers to elevated intramuscular temperature, as Jutte *et al.* (2001) stated that the relationship between skin temperature and intramuscular temperature is not known. They found that there are various factors that influence both skin and intramuscular temperatures, such as subcutaneous adipose tissue thickness, room temperature and blood flow. Merrick *et al.* (1993) found that in cases where compression reduced blood flow to the area, that it did not have an influence on the muscular temperature in that area. It is therefore difficult to determine how skin and intramuscular temperatures affect one another.

Keeping in mind that with the NIRS technology used in this study, where the emitter-detector distance only allows measurement two centimetres into the tissue under the probe, it follows that when trying to exclude external factors influencing the true muscle oxygenation variables, it is not the core muscle temperature, but rather the temperature of the skin, subcutaneous tissue and the superficial muscle layers that are of importance. Therefore, in an experimental setting, it would be advantageous to keep the conditions on the skin as constant as possible, so that any changes that are observed in NIRS measurements indicate the changes that are taking place in the muscle.

Although both CS and FS in this study caused higher ST, there is no evidence that the higher local temperature affected either blood flow to the muscle or tissue oxygenation. Both O₂Hb and TOI were practically significantly lower during the CS and FS trials, suggesting that the compression may have inhibited blood flow to the muscles slightly (hence lower O₂Hb levels compared to NS) and may have resulted in lower tissue oxygenation levels in the muscles (hence lower TOI levels compared to NS).

2. Metabolic changes

The cardiorespiratory variables measured in this study were VO₂, VCO₂ and HR. Overall, differences in VO₂ and VCO₂ between the socks trials and the control were small to negligible, while differences in HR were practically negligible. This is in accordance with previous research for both incremental exercise to exhaustion (Bringard, Perrey & Belluye, 2006; Rimaud *et al.*, 2007; Kemmler *et al.*, 2009; Ali, Creasy & Edge, 2010; Ali, Creasy & Edge, 2011; Wahl *et al.*, 2011) as well as submaximal exercise (Bringard, Perrey & Belluye, 2006; Rimaud *et al.*, 2007; Kemmler *et al.*, 2009; Ali, Creasy & Edge, 2010; Ali, Creasy & Edge, 2011; Lovell *et al.*, 2011; Wahl *et al.*, 2011).

Ali, Creasy and Edge (2011) suggested that it is possible that the calf muscle pump of well-trained athletes are too well developed, therefore the added effect of the CS does not increase venous return and thereby the other metabolic variables. Rimaud *et al.* (2007) on the other hand stated

that the limiting factor was O₂ utilization rather than O₂ delivery. Therefore if the O₂ delivery is increased with the use of CS, the ability to utilize the O₂ is limited by the oxidative capacity of the muscle. With regard to oxidative capacity, CS could not increase the amount of mitochondria or oxidative enzymes within the muscle, therefore, it is unlikely that CS could improve O₂ utilization in this manner.

The findings of this study therefore support most research which challenges the claim of manufacturers that CS enhance venous blood flow and therefore cardiac output. There are limited evidence in the literature that external compression increases venous blood flow and venous blood flow velocity (Stanton, Freis & Wilkins, 1949; Lawrence & Kakkar, 1980; Kraemer *et al.*, 2000). However, most studies have been done on non-athletes and it is therefore not known whether these findings can be extrapolated to well-trained athletes with well-developed calf muscles. If this was the case, most research would also report that CS enhances sport performance, and in particular endurance performance. However, very few studies have actually observed any significant ergogenic effects with CS. The possibility exists that with well-trained endurance athletes, the physiological adaptation to training is such that the added effect of compression does not cause further improvement in the cardiorespiratory function. On the other hand, it is possible that the external compression provided by most commercially available garments is just not adequate enough, or perhaps too high to enhance calf muscle function and hence stroke volume and cardiac output.

Previous research suggests that certain minimum pressures are required to affect cardiovascular function (MacRae *et al.*, 2012). For instance, Stenger *et al.* (2010) reported increases in SV with an applied pressure of ≈55 mmHg at the ankle and 6 mmHg at the thigh, while MacRae *et al.* (2012) found no effect with a lower body CG which exerted lower pressures (15 and 11 mmHg at the calf and thigh, respectively). Watanaki and Murata (1994) proposed that the minimum pressures needed to affect CO while standing quietly is approximately 17 and 15 mmHg for the leg and thigh, respectively. With exercise in the upright position, these minimum pressures would obviously be much higher.

Although the pressures that were measured in this study were relatively high (>40 mmHg at the calf), there is no evidence that this enhanced venous return. In fact, it is rather unlikely that this would be the case, as the average pressure that was measured at the posterior calf in this study was actually higher than the average pressure at the ankle. The lack of a significant pressure gradient would have hindered any possibility of increased venous blood flow. Thus, although the hypothesis that CS may enhance SV and CO cannot be refuted, further research with well-trained athletes is needed to investigate this matter further.

3. Blood lactate concentration

Differences in systemic [BLa] were largely of small practical significance for the different compression conditions and the control. These results are in agreement with those of Ali, Creasy and Edge (2010) and they concluded that CG had no effect on lactate clearance. Their study was similar to the current study with regard to the exercise (type and intensity), study population, as well as the fact that various compression ranges were used. Other authors have also found no difference in systemic [BLa] in both lower body (Scanlan *et al.*, 2008; Ali, Creasy & Edge, 2011) and upper body (Dascombe *et al.*, 2011) CG. These results are in contrast to the earlier report of Berry and McMurray (1987) that [BLa] is decreased after exercise with CS. They speculated that this improved lactate recovery could be due to lower production of lactate and/or due to increased lactate removal through greater blood flow and oxidation of lactate in the active muscle. The effects of compression on lactate kinetics are further discussed in section D.4.

4. Muscle oxygenation

As alluded to earlier, one of the hypotheses regarding the physiological mechanisms behind CS, is that it may increase blood flow to the muscles and enhance lactate oxidation. Evidence in this regard, however, is not conclusive and was therefore the motivation behind this study.

An increase in O₂Hb during exercise should provide evidence for increased O₂ delivery to the muscles and this may be indirect evidence of enhanced blood flow through the muscle. Simultaneously, an increase in blood flow to the muscle should be accompanied by an increase in TOI levels. However, there was no indication in this study that external compression improved either O₂ delivery to the muscles or O₂ saturation in the muscle. For both the left and right leg, slightly higher values in O₂Hb and TOI were observed for the NS condition compared to the compression conditions, which may be of practical significance. Thus, as discussed in section D.1. there is a slight possibility that the compression socks that were used in this study may have marginally inhibited blood flow to the muscle (perhaps due to too high pressures on the calf), but that it almost certainly did not improve O₂ delivery to the muscle. Another possibility is that the lower TOI levels with compression are related to the practically lower [Hb] that was measured in the subjects during the compression trials.

If O₂ delivery to the muscles is not enhanced by compression, the possibility still exist that it may improve O₂ utilization by the muscle. It has been suggested that HHb could be used as an indication of fractional O₂ extraction (Delorey, Kowalchuk & Paterson, 2003; Ferreira, Koga & Barstow, 2007). Davies et al. (2008) suggested that HHb is to a great extent insensitive to changes in plasma volume and can therefore be used to indicate O₂ utilization. The results of this study showed that there were moderately to large practical significant differences in HHb between the compression trials and NS, with higher HHb levels recorded for both compression conditions. This would suggest greater O₂ extraction by the muscles and thus enhanced O₂ consumption. However, this conclusion is in contrast to the findings of Coza et al (2012).

Coza *et al.* (2012) tested 16 men (age = 26.3 ± 5.1 years) with and without compression sleeves over their calf. Participants completed two minutes of heel raises at a tempo of 40 heel raises per minute, where after they recovered for 10 minutes and then completed the same protocol with the other experimental condition (with CS or without). During these tests, arterial occlusion was also induced on one of the legs of the participants. This enabled the researchers to more directly measure the O₂ utilization rate during the exercise due to the fact that a compartment is created by

the arterial occlusion and thereby eliminating the effect that increased arterial blood flow may have on these measurements. They found O₂ utilization to be similar in the compression and no compression condition. Furthermore, in the leg with no arterial occlusion, they found the increased rate of TOI to be higher in the compression condition. This led them to believe, that in the absence of differences in O₂ utilization, the difference with the compression is due to blood flow changes.

This could show that the increase in HHb seen in the current study cannot entirely be attributed to an increase in O₂ utilization, but that the CS also caused a decrease in blood flow velocity. This decrease in blood flow velocity along with the increase in temperature experienced in the sock conditions, could lead to greater O₂Hb dissociation and therefore greater O₂ uptake by the muscle (Neary, Hall & Bhambhani, 2001). This would be in agreement with Koga *et al.* (1997) who proposed that an increased temperature enhances mitochondrial activity, and therefore increases O₂ utilization.

Further evidence that CS may enhance O₂ diffusion in the muscle relates to the measurement of nTHI levels. It is suggested that nTHI is related to tissue haemoglobin concentration and is therefore directly related to tissue blood flow (Coza *et al.*, 2012). For instance, with arterial occlusion one would expect a constant nTHI, while venous occlusion would cause an increase in nTHI. In this study, small to large practical significant differences were found between the compression conditions and NS, with higher nTHI levels with CS and FS. These findings may confirm, as previously discussed, that arterial blood flow was not enhanced by the external compression. The alternative explanation would then be that CS caused a decrease in venous blood flow, however, this will contradict many previous studies that compression enhances venous blood flow. It could also be argued that if the compression in this study inhibited venous return to the heart, there would have been a cardiovascular compensation in the form of an increased heart rate to make up for the decrease in SV and thus CO. HR was, however, not higher during the compression trials compared to the control trial.

It is therefore proposed that the higher nTHI levels during the compression conditions suggest that the transit time of Hb through the capillary bed was decreased due to the compression of the vasculature and that this allowed for greater unloading of O₂ and thus greater extraction of O₂ by the muscle cells. This explanation would be supported by the fact that O₂ unloading and diffusion was further enhanced by the higher ST with compression. Thus the conclusion is that CS, and to a slightly lesser degree FS, did not improve O₂ delivery to the muscle, but rather O₂ utilization by the muscles.

The fact that no differences could be found between the different compression conditions with regard to [BLa] could be attributed to the intensity of the exercise. It is possible that the participants made near full use of their oxidative capacity during the exercise, and that the added O₂ availability therefore could not be utilized by the mitochondria for oxidative metabolism.

The finding that there was no difference in HR could be indicative that CS did not influence either arterial blood flow or venous return. This is in agreement with the findings of Dascombe *et al.* (2011a), who found upper body CG did not influence NIRS derived muscle blood flow measures. However, with the use of lower body CG, Dascombe *et al.* (2011b) found increased O₂ utilization in conjunction with increased blood flow and a decrease in HR. The first of these two studies looked at upper body CG in elite kayakers, whereas the second made use of lower body CG in well trained distance runners. Various other authors have also found that CS improve deep venous blood flow (Brown & Brown, 1995; Chatard *et al.*, 2004), although this was not determined by the use of nTHI and nTHI can also not determine the blood flow velocity.

As to the differences between the two legs, it must be kept in mind that the nTHI measurement is an absolute value, while O₂Hb and HHb are relative to baseline. Therefore, differences in vascular structure could influence this measurement, as this is not necessarily symmetrical between the two legs. Furthermore, the overall muscle fibre type composition of the two legs may be similar, but on a tissue level as with NIRS measurements, there could be differences in the location of these different fibres, leading to variations between the two legs. This will not be seen in the O₂Hb or

HHb measurements as they are relative to the baseline. As TOI is dependent on changes in O₂Hb and HHb, it will not be as sensitive to small variations in muscle fibre type. However, the exact reasons for these differences are still unclear and remain a physiological anomaly.

Although these findings and explanations are plausible and in agreement with previous research (Austin *et al.*, 2005), it is important to note that Menetrier *et al.* (2011) suggested that changes in NIRS variables could be due to the effect of the pressure applied through the probe on the vascular system, as well as the light signal received from the tissue. Although this possibility cannot be excluded, special care was taken in this study with regards to probe placement and minimizing any additional pressure that may affect the measurements. For instance, NIRS measurements were only started after the sock was pulled over the measurement probe and thus the first measurements (i.e. baseline values) were taken under the same conditions as during exercise and recovery, and therefore expressed relative to the baseline value with the added pressure of the probe. Thus the changes measured can be considered purely the effect of the exercise during these conditions. Participants also acted as their own control, thereby eliminating the effect that variations such as subcutaneous fat layer and muscle fibre type could have on these variables.

With minimal differences in O₂Hb ($p = 0.65$; ES = small to large practical effects) and clear trends for differences in HHb ($p = 0.57$; ES = large practical effects) with the different pressure conditions, it seems that the CS and FS improve O₂ diffusion within the tissue to which pressure is applied. Although, these trends for increased O₂ diffusion were apparent at a local level, systemically there seems to be no difference between the different pressure conditions, except for the first two increments where VO₂ tended to be lower in the NS condition.

The improvement in muscle O₂ utilization does also not relate to improved systemic La kinetics. Lactate was measured systemically, and it is therefore possible that on a muscular level, lactate kinetics within the muscle is improved by the CS, but that the intensity of the exercise was so high as to elicit similar systemic lactate responses with the different compression conditions. Whether

pressure should be applied to a greater muscle mass, or whether lower exercise intensity is necessary for systemic lactate improvements is something that should be addressed in future research.

E. THE EFFECT OF COMPRESSION ON MUSCLE PHYSIOLOGY DURING POST-EXERCISE RECOVERY

1. Skin temperature

Purvis and Tunstall (2004) stated that certain materials can create a microenvironment that is hotter and more humid than the surroundings. This would then cause an increase in ST. A good example would be the foot in a shoe where movement of air is minimal. They also found that with certain materials, sweat evaporation was increased to such an extent that it actually causes an increase in skin temperature. Thus, it is possible that if CS absorbs the sweat and becomes soaked, ST could drop significantly (Otomasu *et al.*, 1997); this could cause the skin temperature to drop significantly.

There were only small to negligible differences in ST between the compression trials and NS. Very small differences were measured between CS and NS of the left leg (0.37 °C) and between FS and NS of the left leg (0.43 °C). During the recovery period, all participants sat quietly in the laboratory, with an air conditioner that was slightly more directed towards their right legs. It is therefore possible that the lower temperatures of the right leg could possibly be attributed to more air movement on this side of the body. However, it is debateable whether such a small difference will have an effect on skin blood flow. Therefore it is unlikely that the slight differences in ST during the different running trials could have significantly affected the measurement of the muscle oxygenation variables.

2. Blood lactate concentration

A number of researchers reported decreased [BLa] with CS during post-exercise recovery (Berry & McMurray, 1987; Bindemann, 2007; Chatard *et al.*, 2004; Rimaud *et al.*, 2007), which contributed to the interest of athletes in the use of CS as a potential recovery strategy. These results were found in trained men following maximal cycle ergometer exercise tests (Berry and McMurray, 1987; Chatard *et al.*, 2004), trained men during a two hour long treadmill running protocol (Bindemann, 2007) and men with spinal cord injuries during maximal wheelchair exercise (Rimaud *et al.*, 2007). The findings in this study, however, are in contrast to previous reports, as there were no clear differences in [BLa] between the compression trials and NS during recovery.

A few methodological differences between the studies may perhaps explain the discrepancies in results. For instance, the study population in the current study was younger (34.8 ± 3.8 years vs. 43.7 ± 5.5 years) and had a higher VO_{2max} (52.4 ± 7.1 mL.kg⁻¹.min⁻¹ vs 45.7 ± 5.0 mL.kg⁻¹.min⁻¹) than the participants in the Bindemann (2007) study. As mentioned earlier, lactate diffusion from the muscle is faster in younger populations (Tzankoff & Norris, 1979) and therefore it is possible that the age difference in the participants can account for the dissimilar results. Furthermore, the participants in the current study were higher level athletes, competing up to international level, whereas the runners in the previous study (Bindemann, 2007) could be classified as “weekend warriors”. Other differences between the two studies relate to the duration (35 to 50 min vs 90 min) and the intensity of exercise (80 % of maximum treadmill speed vs 70 % of VO_{2max}). The lactate kinetics (production and removal) are therefore likely to be very different. This matter will be further discussed in section E.3.

3. Muscle oxygenation

Most muscle oxygenation research has been targeted at the changes during exercise, with very few studies describing the changes during recovery, especially prolonged recovery such as the 60 min recovery period in the current study. Bhambani, Maikala and Buckley (1998) described the

changes during recovery from incremental arm and leg exercise as a rapid initial increase in muscle oxygenation immediately after exercise, and a slow return to baseline within the last two minutes of the six minute recovery period. Ding *et al.* (2001) termed this phenomenon the overshoot recovery of muscle oxygenation. In the current study NIRS variables were measured for 60 min of recovery, and during this time a similar pattern was found, namely an initial rapid increase in muscle oxygenation followed by a slow recovery to baseline. It was only within the last 10 min interval that the TOI measurements came close to resting baseline values. A simultaneous increase in TOI and nTHI could indicate that the overshoot in TOI directly after the end of exercise could be due to a sudden increase in blood flow to the muscle (hyperaemia). This hyperaemia could be the result of sustained higher HR and cardiac output immediately after exercise. This effect is even evident when systemic changes in HR and blood flow are not present as in the case of electronically stimulated muscle contraction (Rendell *et al.*, 1997).

Along with this increase in blood flow to the muscle post-exercise, the overshoot in the muscle after exercise could be linked to excess post-exercise oxygen consumption (EPOC). Danduran, Dixon and Rao (2012) studied cerebral, renal, and muscle (Deltoid and Vastus lateralis) oxygenation during exercise and recovery in children. They found that with exercise (Bruce protocol on a treadmill) the oxygenation of all the tissues measured decreased in varying amounts as the intensity increased. At the end of the exercise, they found that the cerebral, renal and deltoid muscle oxygenation recovered to baseline almost immediately, whereas an overshoot in oxygenation in the Vastus lateralis, as previously described, were evident. It therefore seems that the EPOC phenomenon during the first phase of recovery is due to excessive oxygen consumption by the exercised muscle rather than a full systemic increase in oxygen consumption.

No consistent differences were found in this study between the different pressure conditions with regard to TOI and these differences varied from small to negligible. Closer analysis also showed that the overshoot recovery of muscle oxygenation ($\Delta 2$) (*Table 6.29.*) was not different between the different conditions. There was also no difference in the overshoot recovery of muscle oxygenation to the end of the recovery period ($\Delta 3$) (*Table 6.31.*), or the change from the end of

exercise to the end of the recovery period (Delta 4) (*Table 6.33*). It can be speculated that the Delta 3 and Delta 4 calculations are estimates of the rate of TOI recovery after exercise; the rate of TOI recovery is therefore directly proportional to the change in the TOI in this calculation. Accordingly, the change in TOI was greatest in the CS condition and smallest in the NS condition and these differences were of large practical significance. These calculations can be considered similar to the muscle oxygenation recovery rate that Ding *et al.* (2001) used in their study (*Equation 2.9*). These researchers showed that elite athletes show a greater recovery rate post-exercise in comparison to the control group. A popular hypothesis to explain increased lactate removal during recovery is that CS would aid blood flow and venous return post-exercise. The effect of CS on the muscle pump is therefore proposed to be similar to active recovery, but without the additional metabolic cost. However, results in this regard are varied, with both no effects (Bindemann, 2007) and positive effects (Berry and McMurray, 1987; Rimaud *et al.*, 2010) reported.

It seems as though the CS might cause a decrease in the velocity of blood flow within the capillary bed. This decreased velocity has an impact on various factors regarding recovery. It can be seen in the left leg that the NS condition had a lower nTHI in comparison to the two sock conditions. This indicates that in the sock conditions there was a greater amount of Hb present in the muscle. This increase is related to both an increase in the available O₂Hb and the amount of HHb. As there was no pressure in the NS condition, there is no external factor limiting the arterial blood flow velocity as in the CS and FS conditions. This then could cause lower levels of O₂Hb in the muscle accompanied by a decrease in the amount of HHb in the muscle due to the flushing effect caused by the increased arterial blood flow; the latter would be related to the (temporary) high SV and CO post-exercise. Similarly, the CS cause slower blood flow velocities, therefore, there is more O₂Hb present in the muscle. The decrease in blood flow velocity also cause a slower arterial blood transit time, therefore the diffusion rate is enhanced. This increased diffusion rate facilitates the O₂ uptake of the muscle, thereby increasing the amount of HHb present in the muscle.

Lactate measurements after the first 10 minute interval of recovery showed a large practical difference between the NS and both sock conditions. Therefore, both the sock conditions improved

lactate removal during the first 10 minutes of recovery. This along with the improved overall muscle oxygenation as determined above could suggest that the hypothesis set by Berry and McMurray (1987) is possibly the mechanism whereby CS improve lactate kinetics post-exercise. With regard to the alternative hypothesis for this theory, the current results do not support the alternative hypothesis stating that CS improve venous return and thereby lactate clearance. There were higher nTHI values with CS suggesting either venous pooling or decreased capillary bed blood flow velocity as discussed above.

These findings are backed by research completed by Hafner *et al.* (2000) who found that pressures in excess of 50 mmHg caused decreases in venous blood flow in both the subcutaneous as well as muscle vascular system. In the current study, the average pressure for both the CS (69.1 ± 62.8 mmHg) and FS (59.9 ± 72.5 mmHg) on the posterior calf was higher than the proposed 50 mmHg. This decrease could be due to the compression causing a narrowing of the blood vessels, making it more difficult for red blood cells to move through these vessels (Wahl *et al.*, 2012). This will then cause a decrease in the blood flow velocity.

With the start of the recovery period, an increase in HHb is present (Chapter five, *Figure 5.5*). This increase only lasted a short while and then declined at a fast rate. This initial increase could be attributed to either EPOC, as noted earlier in the discussion of TOI, or venous blood pooling within the muscle due to the abrupt end in exercise and therefore the facilitation of the calf muscle pump. Even quiet standing is known to cause increases in venous pooling (Bringard *et al.*, 2006), therefore the effect will be far greater after exercise is stopped, while heart rate and cardiac output are still elevated. At the end of the run protocol, participants stopped, a lactate measurement was taken and then the participant was seated for the remainder of the 60 minute recovery period. What effect this postural change has on muscle blood flow is not known.

A varied response and no practical meaningful differences were also observed in the nTHI results to the different compression conditions and the two legs. As stated earlier, this variation could be due to the fact that the nTHI measurement is an absolute measurement and therefore not relative

to the first measurement as is the case with O₂Hb and HHb. Therefore, variations in vascular structure, in other words the capillary bed, or even the location of the main veins and arteries in the two legs could account for these variations.

At the end of exercise, nTHI values increased (*Table 6.29.*), and then did not decrease much over the remainder of the recovery period (*Table 6.31.*). Small practical effects were found between the different compression conditions with the Delta 2 calculation, with the right leg showing a trend for increases in change ranging from CS to NS. The nTHI measurement is an indication of the amount of Hb present in the tissue. Therefore, greater change in the Delta 2 calculation suggests greater build-up of Hb in the muscle. What this therefore suggests is that there could possibly be facilitation by the CS (less so by the FS) to improve venous return after the exercise. Increased arterial blood flow is expected in all three conditions at the end of exercise, as the HR and cardiac output is elevated. Therefore, if the venous return is not high enough venous pooling could cause this increase in Hb at the end of exercise, when the effect of the calf muscle pump is absent.

The Delta 3 and Delta 4 calculations (*Table 6.31.* and *Table 6.33.*) suggest that the increased nTHI is sustained over the 60 min recovery period. Koizumi *et al.* (2011) studied the effect of active versus passive recovery on muscle oxygenation between two high intensity cycling bouts. They found that the total haemoglobin was higher in the active recovery group during recovery than in the passive recovery group. They also found that performance in the second bout of cycling exercise was better after active recovery than passive recovery. It is therefore possible that higher Hb levels in the muscle during recovery could improve recovery of metabolic by products such as lactate.

It seems that during recovery, the decreased blood flow velocity brought forth by the external compression causes increases in oxygen diffusion and causes prolonged increased levels of O₂Hb present in the muscle. It is possible that these factors facilitate the improvement in lactate clearance visible after 10 minutes of recovery, although lactate measurements at a muscular level will be necessary to determine whether this is truly the case.

F. CONCLUSION

1. The effect of CS during exercise

When studying exercise, improvements in performance is the major effect sought after, whether it is improvements in cardiorespiratory, metabolic, or biomechanical variables. Therefore, when studying the effects of compression on the exercise response, the determination of variables that could lead to performance improvements is vital. *Figure 7.1.* illustrates the proposed mechanism whereby CS may affect the physiological responses to exercise.

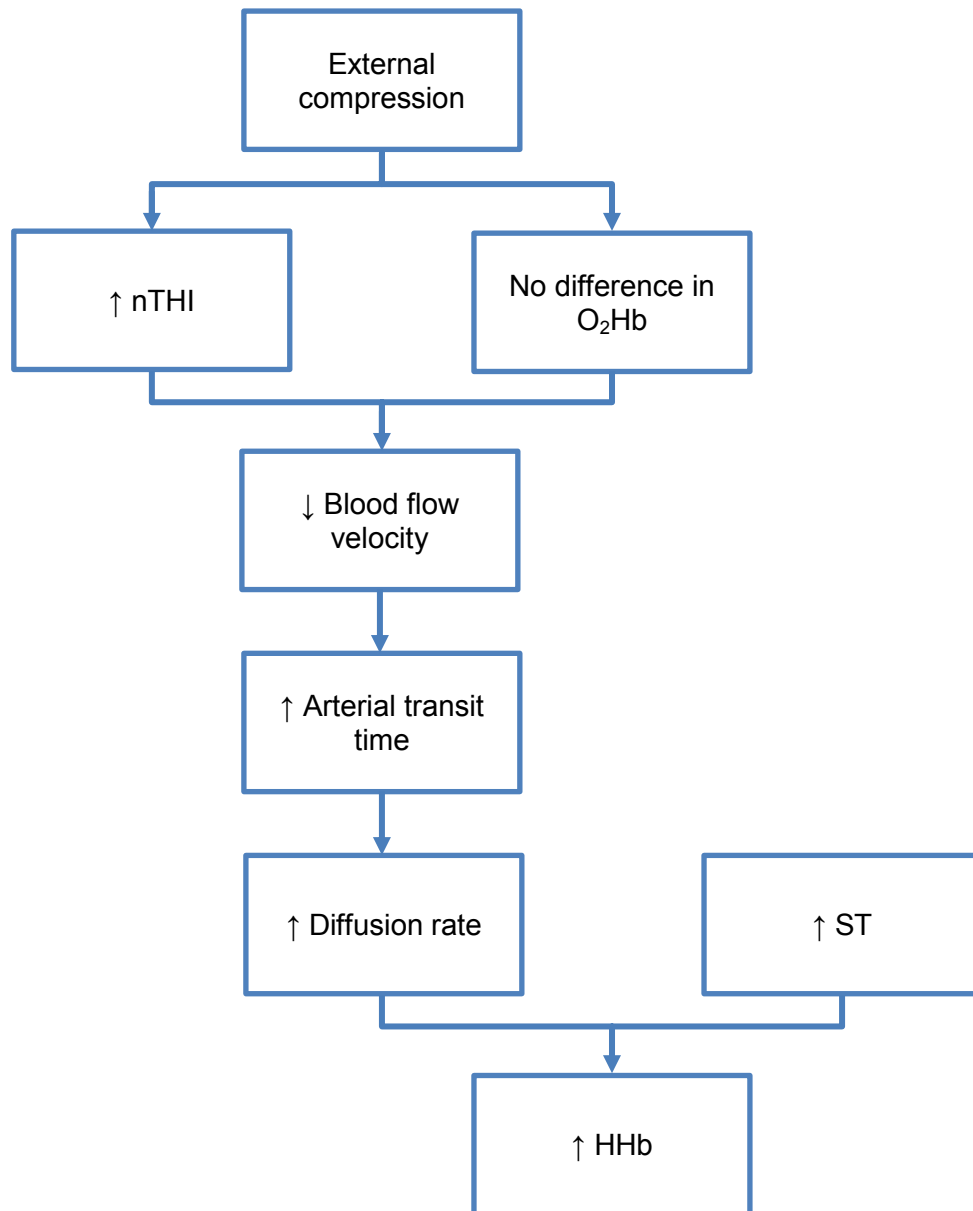


Figure 7.1. Proposed model to describe the effect of CS on muscle oxygenation during exercise. nTHI, Total haemoglobin index; O₂Hb, Oxy-haemoglobin; ST, Skin temperature; HHb, Deoxy-haemoglobin

As illustrated in *Figure 7.1*, it seems that external compression causes a decrease in the blood flow velocity, which ultimately leads to increased O₂ diffusion rate. This along with the increase in skin temperature then facilitates the diffusion of O₂ into the muscle. The oxidative capacity of the muscle then becomes the limiting factor; i.e. limiting the oxidative metabolism and therefore causing similar [BLa] in the different conditions.

It seems that the effect of different pressure gradients is visible in the HHb. This is possibly due to the greater pressure causing more pronounced decreases in blood flow velocity and thereby increased O₂ diffusion rates.

It is possible that the large variation in pressure exerted by the socks could lead to large variations in the physiological effects experienced by different individuals with the use of these socks. It is therefore possible that occlusions could occur in some people due to very high pressures as measured in the current study. In a case like this, the occlusion could have a negative effect on the physiological functioning of the muscle.

2. The effect of CS on post-exercise recovery

Understanding the physiological mechanisms responsible for differences that are seen with the application of different aids, such as CS, is of utmost importance. It enables informed decisions and prescriptions to be made with regard to the use of the specific aid. *Figure 7.2.* illustrates the proposed mechanism whereby CS may affect the physiological responses during post-exercise recovery.

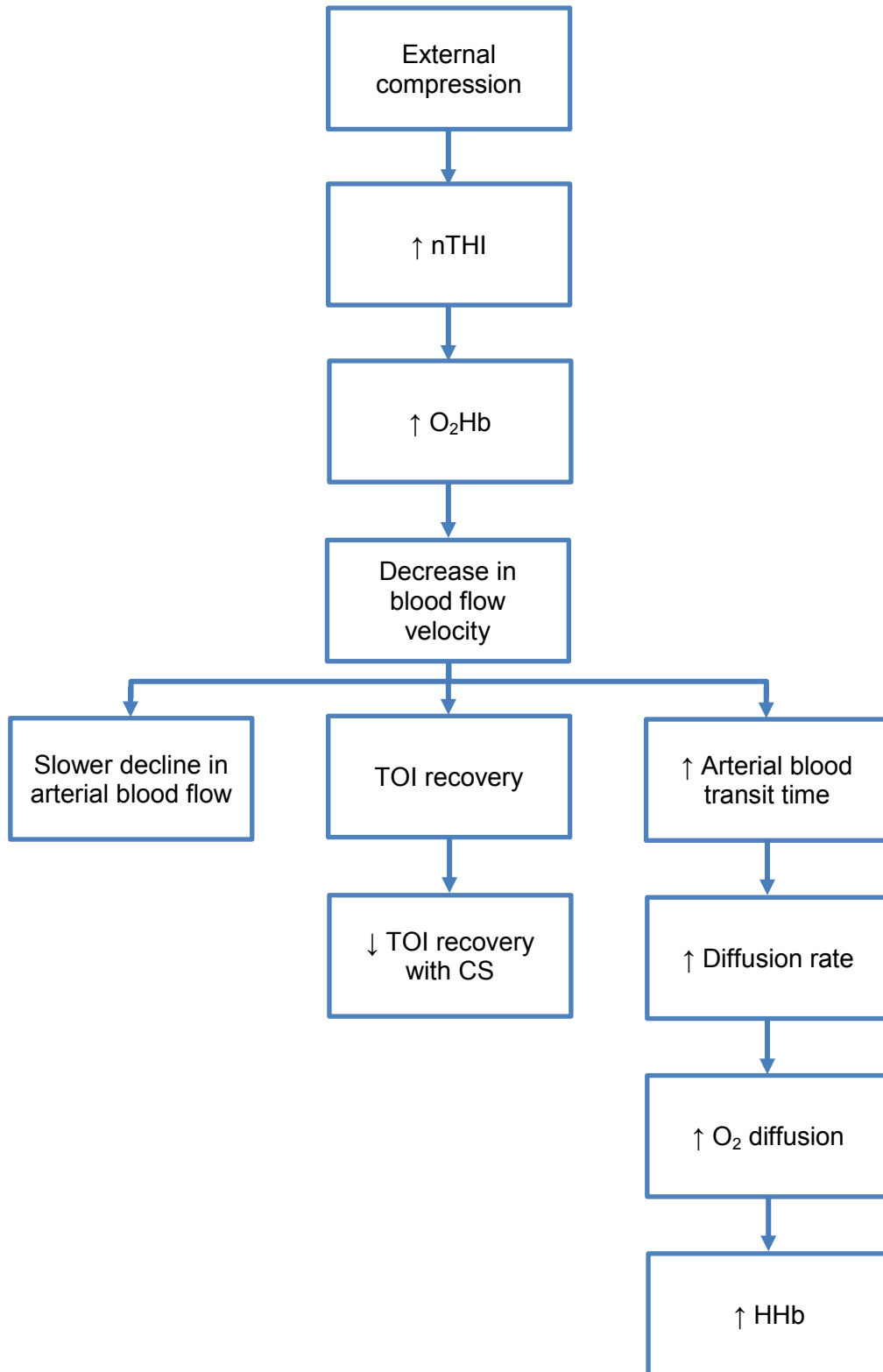


Figure 7.2.

Proposed model to describe the effect of CS on muscle oxygenation during recovery. CS, Compression socks; nTHI, Total haemoglobin index; O₂Hb, Oxy-haemoglobin; TOI, Tissue oxygenation index; O₂, Oxygen; HHb, Deoxy-haemoglobin

It seems that the main effect of external compression on the calf muscle during recovery is a decrease in blood flow velocity. This plays a critical role in the changes seen in the muscle oxygenation variables, causing increased O₂ availability and improved O₂ diffusion into the muscle.

3. Strengths of the current research

The current study holds a few strengths. Firstly it was a crossover study, therefore each participant acted as his own control. This eliminated differences in muscle fibre type, training history, subcutaneous fat thickness and vascular structure which could affect many variables that were measured in this study. Furthermore, an important shortcoming in the literature is the lack of description of the pressure profiles used during the testing. In the current study, large inter-individual variations were found with the same pressure condition due to morphological differences in the legs and muscles of the individuals. Therefore, although the researchers may report the pressure profiles as claimed by manufacturers, this may not be the true profiles. This could lead to differences in the effect the socks have on the variables measured.

This study also measured skin temperature. As skin temperature has an influence on NIRS variables as well as the O₂Hb dissociation curve and mitochondrial activity (Koga *et al.*, 1997) this variable is of importance in the better understanding of the physiological mechanism at play during testing. Furthermore, most studies measure muscle oxygenation variables only in the one leg or arm. This study measured these changes in both legs simultaneously and found differences in the extent to which change occurred as well as the actual change that occurred.

Lastly the current study used trained endurance runners and triathletes ($VO_{2max} = 52.4 \pm 7.1 \text{ mL.kg}^{-1}.\text{min}^{-1}$), therefore the findings in the current study can be translated to an athletic population, rather than the clinical setting in which a lot of compression research is completed.

G. STUDY LIMITATIONS AND FUTURE RESEARCH

This study was limited by a small sample size, especially with regard to the size of the control (NS) condition testing. Some of the variables may not have reached significance due to this small sample size. A further limitation is the inability to measure absolute O₂Hb and HHb values. The NIRO 200nx (Hamamatsu, Japan) used in this study uses the adjusted Beer-Lambert law to calculate these values relative to the first measurement taken. The technology to measure absolute O₂Hb and HHb is developed, although it is not yet freely available and also not as convenient for use in the sport science setting.

Future research should aim at determining the difference between various pressure sensors and the effect that differences in sensor thickness as well as the method of measurement has on the value obtained. Also manufacturers should strive to improve the manner in which fitting of CS is completed. At present most CS as well as FS are fitted in relation to shoe size and ankle circumference, although even in this study, where the socks were fitted strictly to the manufacturer's prescription, very high pressures, in the range of venous occlusion pressures, were exerted by the socks in some cases.

The effect of the added pressure due to insertion of the NIRS probe under both the CS and FS on the vascular bed under the probe is also a limitation in the current study. It was attempted to eliminate this by pulling the sock over the probe before the measurement was started, thereby setting the baseline measurement according to which all the O₂Hb and HHb measurement to follow are obtained. Although this was done, the true effect of the increased pressure on the vascular bed is still not known and this point should be investigated in future.

In future research, markers for muscle damage should be measured in conjunction with the muscle oxygenation measurements. This could give an indication of the level of damage induced and changes in the muscle tissue due to the exercise, as it has been stated by various authors (Kraemer *et al.*, 1998b; Doan *et al.*, 2003; Borrás *et al.*, 2011) that CS decrease the level of muscle damage experienced during exercise.

Furthermore, standardization in the definitions of what the various muscle oxygenation variables are and what they are actually measuring is necessary, as these variables are reported differently in the literature and the conclusions drawn from these variables differ dramatically in some cases due to differences in the interpretation of what they are actually measuring.

REFERENCES

- AHMADI, S., SINCLAIR, P. J., & DAVIS, G. M. (2008). Muscle oxygenation after downhill walking-induced muscle damage. *Clinical Physiology and Functional Imaging* 28(1):55-63.
- ALI, A., CAINE, M.P., & SNOW, B. (2007). Graduated compression stockings: Physiological and perceptual responses during and after exercise. *Journal of Sports Sciences* 25(4):413-419.
- ALI, A., CREASY, R. H., & EDGE, J. A. (2010). Physiological effects of wearing graduated compression stockings during running. *European Journal of Applied Physiology* 109(6):1017-1025.
- ALI, A., CREASY, R. H., & EDGE, J. A. (2011). The effect of graduated compression stockings on running performance. *The Journal of Strength & Conditioning Research* 25(5):1385.
- AUSTIN, K. G., DAIGLE, K. A., PATTERSON, P., COWMAN, J., CHELLAND, S., & HAYMES, E. M. (2005). Reliability of near-infrared spectroscopy for determining muscle oxygen saturation during exercise. *Research Quarterly for Exercise and Sport* 76(4):440-449.
- BAKKEN, B. (2011). The Influence of Lower Body Compression Clothing on Markers of Running Economy during Submaximal Treadmill Running. (Unpublished Thesis, 2011)
- BALDWIN, K., HOOKER, A., & HERRICK, R. (1978). Lactate oxidative capacity in different types of muscle. *Biochemical and Biophysical Research Communications* 83(1):151-157.
- BASFORD, J. R. (2002). The law of Laplace and its relevance to contemporary medicine and rehabilitation. *Archives of Physical Medicine and Rehabilitation* 83(8):1165-1170.
- BEAVER, W. L., WASSERMAN, K., & WHIPP, B. J. (1986). A new method for detecting anaerobic threshold by gas exchange. *Journal of Applied Physiology* 60(6):2020-2027.

- BEHNKE, B. J., MCDONOUGH, P., PADILLA, D. J., MUSCH, T. I., & POOLE, D. C. (2003). Oxygen exchange profile in rat muscles of contrasting fibre types. *The Journal of Physiology* 549(2):597-605.
- BELARDINELLI, R., BARSTOW, T. J., PORZASZ, J., & WASSERMAN, K. (1995). Changes in skeletal muscle oxygenation during incremental exercise measured with near infrared spectroscopy. *European Journal of Applied Physiology and Occupational Physiology* 70(6):487-492.
- BELCARO, G., CESARONE, M. R., SHAH, S. S. G., NICOLAIDES, A. N., GEROULAKOS, G., IPPOLITO, E., WINFORD, M., LENNOX, A., PELLEGRINI, L., BRANDOLINI, R., MYERS, K.A., SIMEONE, E., BAVERA, P., DUGALL, M., DI RENZO, A., & MOIA, M. (2002). Prevention of edema, flight microangiopathy and venous thrombosis in long flights with elastic stockings. A randomized trial. *Angiology* 53(6):635-645.
- BERNHARDT, T., & ANDERSON, G. S. (2005). Influence of moderate prophylactic compression on sport performance. *Journal of Strength Conditioning Research* 19(2):292-297.
- BERRY, M. J., & MCMURRAY, R. G. (1987). Effects of graduated compression stockings on blood lactate following an exhaustive bout of exercise. *American Journal of Physical Medicine & Rehabilitation* 66(3):121.
- BHAMBHANI, Y., MAIKALA, R., & BUCKLEY, S. (1998). Muscle oxygenation during incremental arm and leg exercise in men and women. *European Journal of Applied Physiology and Occupational Physiology* 78(5):422-431.
- BHAMBHANI, Y. N., BUCKLEY, S.M., & SUSAKI, T. (1997). Detection of ventilator threshold using near infrared spectroscopy in men and women. *Medicine and Science in Sports and Exercise* 29(3):402-409.

- BILLAT, V. L., SIRVENT, P., PY, G., KORALSZTEIN, J. P., & MERCIER, J. (2003). The concept of maximal lactate steady state: A bridge between biochemistry, physiology and sport science. *Sports Medicine* 33(6):407-426.
- BINDEMANN, K.E. (2007). Compression garments and recovery. (Unpublished Thesis)
- BOCHMANN, R. P., SEIBEL, W., HAASE, E., HIETSCHOLD, V., RÖDEL, H. & DEUSSEN, A. (2005). External compression increases forearm perfusion. *Journal of Applied Physiology* 99(6):2337-2344.
- BORRAS, X., BALIUS, X., DROBNIC, F., TIL, L., TURMO, A. & VALLE, S. (2011). Effects of lower body compression garment in muscle oscillation and tissular injury during intense exercise. *Portoguese Journal of Sport Sciences* 11(2):685-688.
- BOUSHEL, R., LANGBERG, H., OLESEN, J., GONZALES-ALONZO, J., BÜLOW, J., & KJAER, M. (2001). Monitoring tissue oxygen availability with near infrared spectroscopy (NIRS) in health and disease. *Scandinavian Journal of Medicine & Science in Sports* 11(4):213-222.
- BOUSHEL, R., & PIANTADOSI, C. (2000). Near-infrared spectroscopy for monitoring muscle oxygenation. *Acta Physiologica Scandinavica* 168(4):615-622.
- BRINGARD, A.; DENIS, R.; BELLUYE, N., & PERREY, S. (2006). Effects of compression tights on calf muscle oxygenation and venous pooling during quiet resting in supine and standing positions. *Journal of Sports Medicine and Physical Fitness*, 46(4):548.
- BRINGARD, A.; PERREY, S., & BELLUYE, N. (2006). Aerobic energy cost and sensation responses during submaximal running exercise-positive effects of wearing compression tights. *International Journal of Sports Medicine*, 27(5):373-378.
- BROOKS, G. (2002). Lactate shuttles in nature. *Biochemical Society Transactions* 30(2):258.

- BROWN, J.R. & BROWN, A.M. (1995). Non-prescription, padded, lightweight support socks in treatment of mild to moderate lower extremity venous insufficiency. *Journal of American Osteopaths Association* 95(3):173-181
- BUCKLEY, J., & ESTON, R. (2007). Ratings of perceived exertion. In E. M. WINTER, A. M. JONES, R. C. R. DAVISON, P. D. BROMLEY & T. H. MERCER (Eds.), *Sport and exercise physiology testing guidelines. volume one* (pp. 120-129) The British Association of Sport and Exercise Sciences Guide.
- CARTER III, R.; WATENPAUGH, D. E.; WASMUND, W. L.; WASMUND, S. L. & SMITH, M. L. (1999). Muscle pump and central command during recovery from exercise in humans. *Journal of Applied Physiology* 87(4):1463-1469.
- CHANCE, B. & BANK, W. (1995). Genetic disease of mitochondrial function evaluated by NMR and NIR spectroscopy of skeletal tissue. *Biochimica Et Biophysica Acta (BBA)-Molecular Basis of Disease* 1271(1):7-14.
- CHATARD, J. C.; ATLAOUI, D.; FARJANEL, J.; LOUISY, F.; RASTEL, D. & GUEZENNEC, C. Y. (2004). Elastic stockings, performance and leg pain recovery in 63-year-old sportsmen. *European Journal of Applied Physiology* 93(3):347-352.
- CHAVOSHAN, B., SANDER, M., SYBERT, T. E., HANSEN, J., VICTOR, R. G., & THOMAS, G. D. (2002). Nitric oxide-dependent modulation of sympathetic neural control of oxygenation in exercising human skeletal muscle. *The Journal of Physiology* 540(1):377-386.
- CHEUNG, K., HUME, P. A., & MAXWELL, L. (2003). Delayed onset muscle soreness: Treatment strategies and performance factors. *Sports Medicine* 33(2):145-164.
- CHOUCAIR, M., & PHILLIPS, T.J. (1998). Compression therapy. *American Society for Dermatologic Surgery* 24:141-148.

- CHRISTMASS, M. A., DAWSON, B., PASSERETTO, P., & ARTHUR, P. G. (1999). A comparison of skeletal muscle oxygenation and fuel use in sustained continuous and intermittent exercise. *European Journal of Applied Physiology and Occupational Physiology* 80(5):423-435.
- COMPRESSION SOCKS (2012). *Athletic compression socks*. Retrieved 26/08, 2012, from <http://www.compression-socks.com/athletic-compression-socks-c-24.html?osCsid=rgr50ej8ls7bcvsl4hq37t9vo7>
- COZA, A., DUNN, J. F., ANDERSON, B., & NIGG, B. M. (2012). Effects of compression on muscle tissue oxygenation at the onset of exercise. *The Journal of Strength & Conditioning Research* 26(6):1631.
- CREASY, R. (2008). *Performance, physiological, and perceptual effects of wearing graduated compression stockings during running*. (Unpublished Thesis, 2008)
- DAI, X. Q., LU, Y. H., LIN, H., & BAI, L. (2011). Mechanisms of control of human skin blood flow under external pressure. *Biological Rhythm Research* :1-12
- DANDURAN, M. J., DIXON, J. E., & RAO, R. P. (2011). Near infrared spectroscopy describes physiologic payback associated with excess postexercise oxygen consumption in healthy controls and children with complex congenital heart disease. *Pediatric Cardiology* :1-8.
- DASCOMBE, B., LAURSEN, P., NOSAKA, K., & POLGLAZE, T. (2011a). No effect of upper body compression garments in elite flat-water kayakers. *European Journal of Sport Science* :1-9
- DASCOMBE, B. J., HOARE, T. K., SEAR, J. A., REABURN, P. R., & SCANLAN, A. T. (2011b). The effects of wearing undersized lower-body compression garments on endurance running performance. *International Journal of Sports Physiology and Performance* 6(2):160-173.

- DAVIES, R. C., ESTON, R. G., POOLE, D. C., ROWLANDS, A. V., DIMENNA, F., WILKERSON, D. P., TWIST, C., & JONES, A. M. (2008). Effect of eccentric exercise-induced muscle damage on the dynamics of muscle oxygenation and pulmonary oxygen uptake. *Journal of Applied Physiology* 105(5): 1413-1421.
- DAVIS, S. L., FADEL, P. J., CUI, J., THOMAS, G. D., & CRANDALL, C. G. (2006). Skin blood flow influences near-infrared spectroscopy-derived measurements of tissue oxygenation during heat stress. *Journal of Applied Physiology* 100(1):221-224.
- DE GLANVILLE, K. M., & HAMLIN, M. J. (2012). Positive effect of lower body compression garments on subsequent 40-kM cycling time trial performance. *The Journal of Strength & Conditioning Research* 26(2):480.
- DELOREY, D. S., KOWALCHUK, J. M., & PATERSON, D. H. (2003). Relationship between pulmonary O₂ uptake kinetics and muscle deoxygenation during moderate-intensity exercise. *Journal of Applied Physiology* 95(1):113-120.
- DING, H., WANG, G., LEI, W., WANG, R., HUANG, L., XIA, Q., & WU, J. (2001). Non-invasive quantitative assessment of oxidative metabolism in quadriceps muscles by near infrared spectroscopy. *British Journal of Sports Medicine* 35(6):441-444.
- DOAN, B. K., KWON, Y. -H., NEWTON, R. U., SHIM, J., POPPER, E. M., ROGERS, R. A., BOLT, L.R., ROBERTSON, M. & KRAEMER, W.J. (2003). Evaluation of a lower-body compression garment. *Journal of Sports Sciences* 21:601-610.
- DUFFIELD, R., & PORTUS, M. (2007). Comparison of three types of full-body compression garments on throwing and repeat-sprint performance in cricket players. *British Journal of Sports Medicine* 41(7):409-414.

- DUFFIELD, R., EDGE, J., MERRELLS, R., HAWKE, E., BARNES, M., SIMCOCK, D., & GILL, N. (2008). The effects of compression garments on intermittent exercise performance and recovery on consecutive days. *International Journal of Sports Physiology and Performance* 3(4):454-468.
- EAGLE, M. (2006). Selection, measurement and application of graduated compression hosiery. *Wound Essentials* 1:44 - 52.
- FALKE, (2012). *Compression energizing plus*. Retrieved 27/08, 2012, from http://www.falke.co.za/sportswear/sportsocks/iframes/sock_running.html
- FERGUSON-PELL, M., HAGISAWA, S., & BAIN, D. (2000). Evaluation of a sensor for low interface pressure applications. *Medical Engineering & Physics* 22(9):657-663.
- FERRARI, M., BINZONI, T., & QUARESIMA, V. (1997). Oxidative metabolism in muscle. *Philosophical Transactions of the Royal Society of London. Series B: Biological Sciences* 352(1354):677-683.
- FERRARI, M., MOTTOLA, L., & QUARESIMA, V. (2004). Principles, techniques, and limitations of near infrared spectroscopy. *Canadian Journal of Applied Physiology* 29(4):463-487.
- FERREIRA, L. F., KOGA, S., & BARSTOW, T. J. (2007). Dynamics of noninvasively estimated microvascular O₂ extraction during ramp exercise. *Journal of Applied Physiology* 103(6):1999-2004.
- GAVIN, T. P., BABINGTON, J. P., HARMS, C. A., ARDELT, M. E., TANNER, D. A., & STAGER, J. M. (2001). Clothing fabric does not affect thermoregulation during exercise in moderate heat. *Medicine & Science in Sports & Exercise* 33(12):2124.

- GOH, S. S., LAURSEN, P. B., DASCOTBE, B., & NOSAKA, K. (2011). Effect of lower body compression garments on submaximal and maximal running performance in cold (10 C) and hot (32 C) environments. *European Journal of Applied Physiology* 111(5):819-826.
- GONZÁLEZ-ALONSO, J., TELLER, C., ANDERSEN, S. L., JENSEN, F. B., HYLDIG, T., & NIELSEN, B. (1999). Influence of body temperature on the development of fatigue during prolonged exercise in the heat. *Journal of Applied Physiology* 86(3):1032-1039.
- GRASSI, B., POGGIAGHI, S., RAMPICHINI, S., QUARESIMA, V., FERRARI, M., MARCONI, C., & CERRETELLI, P. (2003). Muscle oxygenation and pulmonary gas exchange kinetics during cycling exercise on-transitions in humans. *Journal of Applied Physiology* 95(1):149-158.
- HAFNER, J., LÜTHI, W., HÄNSSLE, H., KAMMERLANDER, G., & BURG, G. (2000). Instruction of compression therapy by means of interface pressure measurement. *Dermatologic Surgery* 26(5):481-488.
- HAMAMATSU PHOTONICS DEUTSCHLAND GMBH (2012). Cerebral oxygenation measuring system, NIRO-200NX.
- HANSEN, J., THOMAS, G. D., HARRIS, S. A., PARSONS, W. J., & VICTOR, R. G. (1996). Differential sympathetic neural control of oxygenation in resting and exercising human skeletal muscle. *Journal of Clinical Investigation* 98(2):584.
- HARMAN, E. & FRYKMAN, P. (1990). The effect of knee wraps on weightlifting performance and injury. *Journal of Strength and Conditioning Research* 12(5):30-35.
- HIGGINS, T., NAUGHTON, G. A., & BURGESS, D. (2009). Effects of wearing compression garments on physiological and performance measures in a simulated game-specific circuit for netball. *Journal of Science and Medicine in Sport* 12(1):223-226.

- HIROYUKI, H., HAMAOKA, T., SAKO, T., NISHIO, S., KIME, R., MURAKAMI, M., & KATSUMURA, T. (2002). Oxygenation in vastus lateralis and lateral head of gastrocnemius during treadmill walking and running in humans. *European Journal of Applied Physiology* 87(4):343-349.
- HOFFMAN, M. (2008). Anthropometric characteristics of ultramarathoners. *International Journal of Sports Medicine* 29(10):808.
- HOUGHTON, L. A., DAWSON, B., & MALONEY, S. K. (2009). Effects of wearing compression garments on thermoregulation during simulated team sport activity in temperate environmental conditions. *Journal of Science and Medicine in Sport* 12(2):303-309.
- HOWLEY, E. T., BASSETT, D. R., & WELCH, H. G. (1995). Criteria for maximal oxygen uptake: Review and commentary. *Medicine and Science in Sports and Exercise* 27:1292-1292.
- HUBBARD, J. L. (1973). The effect of exercise on lactate metabolism. *The Journal of Physiology* 231(1):1-18.
- HYTTEL-SORENSEN, S., SORENSEN, L. C., RIERA, J., & GREISEN, G. (2011). Tissue oximetry: A comparison of mean values of regional tissue saturation, reproducibility and dynamic range of four NIRS-instruments on the human forearm. *Biomedical Optics Express* 2(11):3047-3057.
- INTERLINK ELECTRONICS. (2012). *Force sensors*. Retrieved 02/16, 2012, from <http://www.interlinkelectronics.com/products.php>
- JAKEMAN, J. R., BYRNE, C., & ESTON, R. G. (2010). Lower limb compression garment improves recovery from exercise-induced muscle damage in young, active females. *European Journal of Applied Physiology* 109(6):1137-1144.
- JÖBSIS, F. F. (1977). Noninvasive, infrared monitoring of vertebral and myocardial oxygen sufficiency and circulatory parameters. *Science* 198(4323):1264-1267.

- JUTTE, L. S., MERRICK, M. A., INGERSOLL, C. D., & EDWARDS, J. E. (2001). The relationship between intramuscular temperature, skin temperature, and adipose thickness during cryotherapy and rewarming. *Archives of Physical Medicine and Rehabilitation* 82(6):845-850.
- KAWAGUCHI, K., TABUSADANI, M., SEKIKAWA, K., HAYASHI, Y., & ONARI, K. (2001). Do the kinetics of peripheral muscle oxygenation reflect systemic oxygen intake? *European Journal of Applied Physiology* 84(1):158-161.
- KELLER, A., MÜLLER, M. L., CALOW, T., KERN, I. K., & SCHUMANN, H. (2009). Bandage pressure measurement and training: Simple interventions to improve efficacy in compression bandaging. *International Wound Journal* 6(5):324-330.
- KEMMLER, W., STENGEL, S., KÖCKRITZ, C., MAYHEW, J., WASSERMANN, A., & ZAPF, J. (2009). Effect of compression stockings on running performance in men runners. *The Journal of Strength & Conditioning Research*, 23(1):101-105.
- KNECHTLE, B., KNECHTLE, P., & ROSEMANN, T. (2009). Skin-fold thickness and training volume in ultra-triathletes. *International Journal of Sports Medicine* 30(5):343-347.
- KOGA, S., SHIOJIRI, T., KONDO, N., & BARSTOW, T. J. (1997). Effect of increased muscle temperature on oxygen uptake kinetics during exercise. *Journal of Applied Physiology* 83(4):1333-1338.
- KOIZUMI, K., FUJITA, Y., MURAMATSU, S., MANABE, M., ITO, M., & NOMURA, J. (2011). Active recovery effects on local oxygenation level during intensive cycling bouts. *Journal of Sports Sciences* 29(9):919-926.
- KOWALCHUK, J. M., ROSSITER, H. B., WARD, S. A., & WHIPP, B. J. (2002). The effect of resistive breathing on leg muscle oxygenation using near-infrared spectroscopy during exercise in men. *Experimental Physiology* 87(5):601-611.

- KRAEMER, W. J., BUSH, J. A., BAUER, J. A., TRIPLETT-MCBRIDE, N., PAXTON, N. J., CLEMSON, A., KOZIRIS, P., MANGINO, L.C., FRY, A.C., & NEWTON, R. U. (1996). Influence of compression garments on vertical jump performance in NCAA division I volleyball players. *Journal of Strength and Conditioning Research* 10:180-183.
- KRAEMER, W. J., BUSH, J. A., NEWTON, R. U., DUNCAN, N. D., VOLEK, J. S., DENEGAR, C. R., CANAVAN, P., JOHNSTON, J., PUTUKIAN, M. & SEBASTIANELLI, W. J. (1998a). Influence of a compression garment on repetitive power output production before and after different types of muscle fatigue. *Research in Sports Medicine: An International Journal* 8(2):163-184.
- KRAEMER, W.J., BUSH, J.A., TRIPLETT-MCBRIDE, N.T., KOZIRIS, P., MANGINO, L.C., FRY, A.C., MCBRIDE, J.M., JOHNSTON, J., VOLEK, J.S., YOUNG, C.A., GOMÉZ, A.L., & NEWTON, R.U. (1998b). Compression garments: influence on muscle fatigue. *Journal of Strength and Conditioning Research* 12(4):211-215
- KRAEMER, W. J., BUSH, J. A., WICKHAM, R. B., DENEGAR, C. R., GOMEZ, A. L., GOTSHALK, L. A., DUNCA, N.D., VOLEK, J.S., NEWTON, R.U., PUTUKIAN, M. & SEBASTIANELLI, W.J. (2001a). Continuous compression as an effective therapeutic intervention in treating eccentric exercise induced muscle soreness. *Journal of Sports Rehabilitation* 5(3):200-208.
- KRAEMER, W. J., BUSH, J. A., WICKHAM, R. B., DENEGAR, C. R., GOMEZ, A. L., GOTSHALK, L. A., DUNCAN, N.D., VOLEK, J.S., PUTUKIAN, M. & SEBASTIANELLI, W.J. (2001b). Influence of compression therapy on symptoms following soft tissue injury from maximal eccentric exercise. *Journal of Orthopaedic and Sports Physical Therapy* 31(6):282-290.

- KRAEMER, W. J., FLANAGAN, S. D., COMSTOCK, B. A., FRAGALA, M. S., EARP, J. E., DUNN-LEWIS, C., HO, J.Y., THOMAS, G.A., SOLOMON-HILL, G., PENWELL, Z.R., POWELL, M.D., WOLF, M.R., VOLEK, J.S., DENEGAR, C.R. & MARESH, C.M. (2010). Effects of a whole body compression garment on markers of recovery after a heavy resistance workout in men and women. *Journal of Strength and Conditioning Research* 24(3):804-814.
- KRAEMER, W. J., VOLEK, J. S., BUSH, J. A., GOTSHALK, L. A., WAGNER, P. R., GOMEZ, A. L., ZATSIORSKY, V.M., DUZTRE, M., RATAMESS, N.A., MAZZETTI, S. A. & SELLE, B.J. (2000). Influence of compression hosiery on physiological responses to standing fatigue in women. *Medicine & Science in Sports & Exercise* 32(11):1849-1858.
- LAWRENCE, D., & KAKKAR, V. (1980). Graduated, static, external compression of the lower limb: A physiological assessment. *British Journal of Surgery* 67(2):119-121.
- LEA MEDIZINTECHNIK GMBH.O2C (*oxygen to see*). Retrieved 02/16, 2012, from <http://www.lea.de/eng/indexe.html>
- LEPERS, R., SULTANA, F., BERNARD, T., HAUSSWIRTH, C., & BRISSWALTER, J. (2010). Age-related changes in triathlon performances. *International Journal of Sports Medicine* 31(4):251-256.
- LEWIS JR, C. E., ANTOINE, J., MUELLER, C., TALBOT, W. A., SWAROOP, R., & EDWARDS, W. S. (1976). Elastic compression in the prevention of venous stasis:: A critical reevaluation. *The American Journal of Surgery* 132(6):739-743.
- LIU, R., KWOK, Y. L., LI, Y., LAO, T. T. H., ZHANG, X., & DAI, X. Q. (2005). Objective evaluation of skin pressure distribution of graduated elastic compression stockings. *Dermatologic Surgery* 31(6):615-624.

- LIU, R., KWOK, Y., LI, Y., LAO, T., & ZHANG, X. & DAI, X.Q. (2006). A three-dimensional biomechanical model for numerical simulation of dynamic pressure functional performance of graduated compression stockings (GCS). *Fibers and Polymers* 7(4):389-397.
- LIU, R., LAO, T. T., KWOK, Y. L., LI, Y., & YING, M. T. C. (2008a). Effects of graduated compression stockings with different pressure profiles on lower-limb venous structures and haemodynamics. *Advances in Therapy* 25(5):465-478.
- LIU, R., LAO, T., LI, Y., KWOK, Y. L., & YING, M. (2008b). Physiological response and comfort sensory perception towards physical-mechanical performance of compression hosiery textiles. *Journal Fiber Bioengineering and Informatics* 1:55-64.
- LIU, R., & LITTLE, T. (2009). The 5Ps model to optimize compression athletic wear comfort in sports. *Journal of Fiber Bioengineering and Informatics* 2(1):44-55
- LOVELL, D. I., MASON, D. G., DELPHINUS, E. M., & MCLELLAN, C. P. (2011). Do compression garments enhance the active recovery process after high-intensity running? *The Journal of Strength & Conditioning Research* 25(12):3264-3268.
- MACRAE, B. A., COTTER, J. D., & LAING, R. M. (2011). Compression garments and exercise: Garment considerations, physiology and performance. *Sports Medicine* 41(10):815-843.
- MACRAE, B. A., LAING, R. M., NIVEN, B. E., & COTTER, J. D. (2012). Pressure and coverage effects of sporting compression garments on cardiovascular function, thermoregulatory function, and exercise performance. *European Journal of Applied Physiology* 112(5):1783-1795.
- MARKS, D., COLEMAN, A., EBERHART, S., VALENZUELA, D., SCHIENBEIN, A., SMITH, B., KING, K., & MERMIER, C. (2011). Effects of wearing compression socks on submaximal exercise during prolonged exposure to moderate altitude: A pilot study.

- MATON, B., THINEY, G., DANG, S., TRA, S., BASSEZ, S., WICART, P., & OUCHENE, A. (2006). Human muscle fatigue and elastic compressive stockings. *European Journal of Applied Physiology* 97(4):432-442.
- MCARDLE, W.D., KATCH, F.I., & KATCH, V.L. (2010). *Exercise physiology: Nutrition, energy and human performance*. (Seventh edition). Lippincott Williams & Wilkins
- MCCULLY, K. K., HALBER, C., & POSNER, J. D. (1994). Exercise-induced changes in oxygen saturation in the calf muscles of elderly subjects with peripheral vascular disease. *Journal of Gerontology* 49(3):128-134.
- MCCULLY, K. K., & HAMAOKA, T. (2000). Near-infrared spectroscopy: What can it tell us about oxygen saturation in skeletal muscle. *Exercise and Sport Science Reviews* 28(3):123-127.
- MCDONOUGH, P., BEHNKE, B. J., PADILLA, D. J., MUSCH, T. I., & POOLE, D. C. (2005). Control of microvascular oxygen pressures in rat muscles comprised of different fibre types. *The Journal of Physiology* 563(3):903-913.
- MÉNÉTRIER, A., MOUROT, L., BOUHADDI, M., REGNARD, J., & TORDI, N. (2011). Compression sleeves increase tissue oxygen saturation but not running performance. *International Journal of Sports Medicine* 32(11):864-868.
- MERRICK, M. A., KNIGHT, K. L., INGERSOLL, C. D., & POTTEIGER, J. A. (1993). The effects of ice and compression wraps on intramuscular temperatures at various depths. *Journal of Athletic Training* 28(3):236-245.
- MILLET, G., PERREY, S., DIVERT, C., & FOISSAC, M. (2006). The role of engineering in fatigue reduction during human locomotion—a review. *Sports Engineering* 9(4):209-220.

- NEARY, J., HALL, K., & BHAMBHANI, Y. (2001). Vastus medialis muscle oxygenation trends during a simulated 20-km cycle time trial. *European Journal of Applied Physiology* 85(5):427-433.
- NIGG, B., & WAKELING, J. (2001). Impact forces and muscle tuning: A new paradigm. *Exercise and Sport Sciences Reviews* 29(1):37-41.
- NORTON, K., WHITTINGHAM, N., CARTER, L., KERR, D., GORE, C., & MARFELL-JONES, M. (1996). Measurement techniques in anthropometry. In K. NORTON, & T. OLDS (Eds.), *Anthropometrica : A textbook of body measurement for sports and health courses* (pp. 25-73). Sydney, Australia: UNSW Press.
- NUSSER, M., & SENNER, V. (2010). High-tech-textiles in competition sports. *Procedia Engineering* 2(2):2845-2850.
- OTOMASU, K., YAMAUCHI, M., OHWATARI, N., MATSUMOTO, T., TSUCHIYA, K., & KOSAKA, M. (1997). Analysis of sweat evaporation from clothing materials by the ventilated sweat capsule method. *European Journal of Applied Physiology and Occupational Physiology* 76(1):1-7.
- POWERS, S.C., & HOWLEY, E.T. (2007). *Exercise physiology: Theory and application to fitness and performance*. (sixth edition) McGrawHill
- PARTSCH, B., & PARTSCH, H. (2005). Calf compression pressure required to achieve venous closure from supine to standing positions. *Journal of Vascular Surgery* 42(4):734-738.
- PARTSCH, H., CLARK, M., BASSEZ, S., BENIGNI, J. P., BECKER, F., BLAZEK, V., CAPRINI, J., CORNU-THÉNARD, A., HAFNER, J., FLOUR, M., JÜNGER, M., MOFFATT, C., & NEUMANN, M. (2006). Measurement of lower leg compression in vivo: Recommendations for the performance of measurements of interface pressure and stiffness. *Dermatologic Surgery* 32(2):224-233.

- PARTSCH, H., CLARK, M., MOSTI, G., STEINLECHNER, E., SCHUREN, E., ABEL, J., ABEL, M., BENIGNI, J. -P., COLERIDGE-SMITH, P., CORNU-THÉNARD, A., FLOUR, M., HUTCHINSON, J., GAMBLE, J., ISSBERNER, K., JUENGER, M., MOFFATT, C., NEUMANN, H.A.M., RABE, E., UHL, J.F. & ZIMMET, S. (2008). Classification of compression bandages: Practical aspects. *Dermatologic Surgery* 34:600-609.
- PARTSCH, H., & MOSTI, G. (2010). Comparison of three portable instruments to measure compression pressure. *International Angiology* 29(5):426-430.
- PETER, J. B., BARNARD, R. J., EDGERTON, V. R., GILLESPIE, C. A., & STEMPEL, K. E. (1972). Metabolic profiles of three fiber types of skeletal muscle in guinea pigs and rabbits. *Biochemistry* 11(14):2627-2633.
- PLATTS, S. H., TUXHORN, J. A., RIBEIRO, L. C., STENGER, M. B., LEE, S., & MECK, J. V. (2009). Compression garments as countermeasures to orthostatic intolerance. *Aviation, Space, and Environmental Medicine* 80(5):437-442.
- PURVIS, A., & TUNSTALL, H. (2004). Effects of sock type on foot skin temperature and thermal demand during exercise. *Ergonomics* 47(15):1657-1668.
- RENDELL, M., GREEN, S., CATANIA, A., OLIVETO, J., WELLS, J., BANSET, E., & WANG, H. (1997). Post-exercise cutaneous hyperaemia resulting from local exercise of an extremity. *Clinical Physiology* 17(3):213-224.
- REVEL SPORTS (2012). *2XU compression socks*. Retrieved 27/08, 2012, from [http://revelsports.com/2XU Compression/2XU Compression%20 Socks.asp](http://revelsports.com/2XU%20Compression/2XU%20Compression%20Socks.asp)
- RIMAUD, D., CALMELS, P., ROCHE, F., MONGOLD, J. J., TRUDEAU, F., & DEVILLARD, X. (2007). Effects of graduated compression stockings on cardiovascular and metabolic responses to exercise and exercise recovery in persons with spinal cord injury. *Archives of Physical Medicine and Rehabilitation* 88(6):703-709.

- RIMAUD, D., MESSONNIER, L., CASTELLS, J., DEVILLARD, X., & CALMELS, P. (2010). Effects of compression stockings during exercise and recovery on blood lactate kinetics. *European Journal of Applied Physiology* 110(2):425-433.
- ROBERGS, R. A. (2001). Exercise-induced metabolic acidosis: Where do the protons come from. *Sportscience* 5(2):1-20.
- ROLFE, P. (2000). In vivo near-infrared spectroscopy. *Annual Review of Biomedical Engineering* 2(1):715-754.
- ROWELL, L. (1974). Human cardiovascular adjustments to exercise and thermal stress. *Physiological Reviews* 54(1):75-159.
- RÜST, C. A., KNECHTLE, B., KNECHTLE, P., PFEIFER, S., ROSEMANN, T., LEPERS, R., & SENN, O. (2012). Gender difference and age-related changes in performance at the long distance duathlon world championships. *The Journal of Strength & Conditioning Research* (Published ahead of print)
- SAKO, T., HAMAOKA, T., HIGUCHI, H., KUROSAWA, Y., & KATSUMURA, T. (2001). Validity of NIR spectroscopy for quantitatively measuring muscle oxidative metabolic rate in exercise. *Journal of Applied Physiology* 90(1):338-344.
- SALTIN, B., & ÅSTRAND, P. -O. (1967). Maximal oxygen uptake in athletes. *Journal of Applied Physiology* 23(3):353-358.
- SAUNDERS, P. U., PYNE, D. B., TELFORD, R. D., & HAWLEY, J. A. (2004). Factors affecting running economy in trained distance runners. *Sports Medicine* 34(7):465-485.
- SCANLAN, A. T., DASCOMBE, B. J., REABURN, P., & OSBORNE, M. (2008). The effects of wearing lower-body compression garments during endurance cycling. *International Journal of Sports Physiology and Performance* 3(4):424-438.

- SEAR, J. A., HOARE, T. K., SCANLAN, A. T., ABT, G. A., & DASCOMBE, B. J. (2010). The effects of whole-body compression garments on prolonged high-intensity intermittent exercise. *The Journal of Strength & Conditioning Research* 24(7):1901-1910.
- SIGG, K. (1963). Die kompression mit verbänden und gummistrümpfen zur prophylaxe und therapie venöser beinleiden. *Fortschr Med* 81(15):601-606.
- SIPES, D., GRAYBILL, D., HAAS, D., & CAWLEY, J. (2011). Adidas® TechFit shorts and their effect on anaerobic power output and sports enhancement. *Keystone Journal of Undergraduate Research* 1(1):8-12.
- SPENCER, M. D., MURIAS, J. M., & PATERSON, D. H. (2012). Characterizing the profile of muscle deoxygenation during ramp incremental exercise in young men. *European Journal of Applied Physiology* :1-12.
- SPERLICH, B., HAEGELE, M., ACHTZEHN, S., LINVILLE, J., HOLMBERG, H. C., & MESTER, J. (2010). Different types of compression clothing do not increase sub-maximal and maximal endurance performance in well-trained athletes. *Journal of Sports Sciences* 28(6):609-614.
- SPERLICH, B., HAEGELE, M., KRÜGER, M., SCHIFFER, T., HOLMBERG, H., & MESTER, J. (2011). Cardio-respiratory and metabolic responses to different levels of compression during submaximal exercise. *Phlebology* 26(3):102-106.
- STANTON, J. R., FREIS, E. D., & WILKINS, R. W. (1949). The acceleration of linear flow in the deep veins of the lower extremity of man by local compression. *Journal of Clinical Investigation* 28(3):553-558.
- STENGER, M. B., BROWN, A. K., LEE, S., LOCKE, J. P., & PLATTS, S. H. (2010). Gradient compression garments as a countermeasure to post-spaceflight orthostatic intolerance. *Aviation, Space, and Environmental Medicine* 81(9):883-887.

- STOLK, R., & WEGEN VAN DER-FRANKEN, C.P.M. & NEUMANN, H.A.M. (2004). A method for measuring the dynamic behaviour of medical compression hosiery during walking. *Dermatologic Surgery* 30:729-736.
- STORER, T. W., DAVIS, J. A., & CAIOZZO, V. J. (1990). Accurate prediction of VO₂max in cycle ergometry. *Medicine and Science in Sports and Exercise* 22(5):704-712.
- SUBUDHI, A. W., DIMMEN, A. C., & ROACH, R. C. (2007). Effects of acute hypoxia on cerebral and muscle oxygenation during incremental exercise. *Journal of Applied Physiology* 103(1):177-183.
- SVEDAHL, K., & MACINTOSH, B. R. (2003). Anaerobic threshold: The concept and methods of measurement. *Canadian Journal of Applied Physiology* 28(2):299-323.
- TANAKA, K.;WATANABE, H.;KONISHI, Y.;MITSUZONO, R.;SUMIDA, S.;TANAKA, S.;FUKUDA, T.;NAKADOMO, F. (1986). Longitudinal associations between anaerobic threshold and distance running performance. *European Journal of Applied Physiology and Occupational Physiology* 55(3): 248-252.
- THEDON, T., BELLUYE, N. & PERREY, S. (2008). Compression sleeves significantly counteracts muscular fatigue during strenuous arm exercise (P124). *Engineering of Sport* 7(1):641-648.
- THIEL, C., VOGT, L., HIMMELREICH, H., HÜBSCHER, M., & BANZER, W. (2011). Reproducibility of muscle oxygen saturation. *International Journal of Sports Medicine* 32(4):277-280.
- THOMAS, C., SIRVENT, P., PERREY, S., RAYNAUD, E., & MERCIER, J. (2004). Relationships between maximal muscle oxidative capacity and blood lactate removal after supramaximal exercise and fatigue indexes in humans. *Journal of Applied Physiology* 97(6):2132-2138.

- THOMPSON, W. R., & GORDON, N.F. & PESCATELLO, L.S. (Eds.). (2010). *ACSM's guidelines for exercise testing and prescription* (Eighth Edition ed.). Baltimore: Lippincott Williams & Wilkins.
- TROYNIKOV, O., ASHAYERI, E., BURTON, M., SUBIC, A., ALAM, F., & MARTEAU, S. (2010). Factors influencing the effectiveness of compression garments used in sports. *Procedia Engineering* 2(2):2823-2829.
- TZANKOFF, S. P., & NORRIS, A. H. (1979). Age-related differences in lactate distribution kinetics following maximal exercise. *European Journal of Applied Physiology and Occupational Physiology* 42(1):35-40.
- VAN BEEKVELT, M.C.P., BORGHUIS, M.S., VAN ENGELEN, B.G.M., WEVERS, R.A., & COLIER, W.N.J.M. (2001). Adipose tissue thickness affects in vivo quantitative near-IR spectroscopy in human skeletal muscle. *Clinical Science* 101:21-28.
- VARELA-SANZ, A., ESPAÑA, J., CARR, N., BOULLOSA, D. A., & ESTEVE-LANAO, J. (2011). Effects of gradual-elastic compression stockings on running economy, kinematics, and performance in runners. *The Journal of Strength & Conditioning Research* 25(10):2902-2910.
- WAHL, P., BLOCH, W., MESTER, J., BORN, D. P., & SPERLICH, B. (2011). Effects of different levels of compression during sub-maximal and high-intensity exercise on erythrocyte deformability. *European Journal of Applied Physiology* 112(6):2163-2169.
- WASSERMAN, K., WHIPP, B. J., & KOYAL, S.N. & BEAVER, W.L. (1973). Anaerobic threshold and respiratory gas exchange during exercise. *Journal of Applied Physiology* 35(2):236-243.
- WATANUKI, S., & MURATA, H. (1994). Effects of wearing compression stockings on cardiovascular responses. *The Annals of Physiological Anthropology* 13(3):121-127.

- WERTHEIM, D., MELHUIISH, J., WILLIAMS, R., LANE, I., & HARDING, K. (1999). Movement-related variation in forces under compression stockings. *European Journal of Vascular and Endovascular Surgery* 17(4):334-337.
- WILLIAMS, J., TZORTZIOUBROWN, V., MALLIARAS, P., PERRY, M., & KIPPS, C. (2012). Hydration strategies of runners in the London marathon. *Clinical Journal of Sport Medicine* 20:59–69
- WILSON, J. R., MANCINI, D., MCCULLY, K., FERRARO, N., LANOCE, V., & CHANCE, B. (1989). Noninvasive detection of skeletal muscle underperfusion with near-infrared spectroscopy in patients with heart failure. *Circulation* 80(6):1668-1674.
- WOODMAN, C. R., SCHRAGE, W. G., RUSH, J. W. E., RAY, C. A., PRICE, E. M., HASSER, E. M., & LAUGHLIN, M. H. (2001). Hindlimb unweighting decreases endothelium-dependent dilation and eNOS expression in soleus not gastrocnemius. *Journal of Applied Physiology* 91(3):1091-1098.
- YOSHIKAWA, T., HARA, T., NAKAO, H., SUZUKI, T., FUJIMOTO, S., & WANG, L. (2006). Which common NIRS variable reflects muscle estimated lactate threshold most closely? *Applied Physiology, Nutrition, and Metabolism* 31(5):612-620.

APPENDIX A

STELLENBOSCH UNIVERSITY CONSENT TO PARTICIPATE IN RESEARCH

The effect of graduated compression socks on lower leg muscle oxygenation

You are asked to participate in a research study conducted by Lara Grobler (HonsB Sport Science), from the Department of Sport Science at Stellenbosch University. The results of this study will contribute to a Masters thesis in Sport Science. You were selected as a possible participant in this study because you are a well-trained male endurance athlete.

1. PURPOSE OF THE STUDY

The purpose of the study is to determine whether compression socks have an influence on lower leg muscle oxygenation of endurance athletes. The level of muscle oxygenation is an indication of lactate oxidation within the muscles and will thus be used to assess whether compression socks cause increased lactate oxidation within the active muscle. If the latter is the case, it would show that athletes will recover faster after prolonged exercise when they wear compression socks during exercise.

2. PROCEDURES

If you volunteer to participate in this study, we would ask you to do the following things:

During the first session you will be asked to complete a health questionnaire to determine whether you comply with the requirements of the study. A number of body composition measurements will be done and then you will complete a maximal exercise test on the treadmill to determine your VO_{2max} .

During the second session, you will be fitted with compression socks and you will complete 10km of running on the treadmill at 90% of your VO_{2max} . Muscle oxygenation, that is the amount of oxygen used by the muscle, will be measured using NIRS (Near-Infrared spectroscopy). Two electrodes will be placed on your calf muscles and measurements will be taken during the treadmill run, as well as 60 minutes after the run.

The third session will be the same as the second session, you will just be fitted with a different pressure gradient compression sock.

All testing will be done in the Sport Physiology laboratory at the Department of Sport Science, University of Stellenbosch.

3. POTENTIAL RISKS AND DISCOMFORTS

The study does not carry any serious risk for you as participant. You may experience discomfort with the finger prick for lactate measurements, or during the exercise tests. However, these will be no more than the usual discomfort you feel while training.

4. POTENTIAL BENEFITS TO SUBJECTS AND/OR TO SOCIETY

You will receive a summary of your body composition analysis, as well as VO_{2max} results. This is valuable information which you may use when planning your training program.

The results of this research will contribute to science as it will give us a better understanding of the physiological mechanisms involved in the improved recovery of athletes which has been observed in previous studies.

5. PAYMENT FOR PARTICIPATION

You will not receive payment for your participation in this study.

CONFIDENTIALITY

Any information that is obtained in connection with this study and that can be identified with you will remain confidential and will be disclosed only with your permission or as required by law. Confidentiality will be maintained by means of assigning each participant a code. This code will be used rather than your name. Data will be kept on a personal computer that is password protected and will only be accessible to the researcher.

Confidentiality with publication of results will be kept by not publishing the raw data as well as making use of the codes assigned to participants.

6. PARTICIPATION AND WITHDRAWAL

You can choose whether to be in this study or not. If you volunteer to be in this study, you may withdraw at any time without consequences of any kind. You may also refuse to answer any questions you don't want to answer and still remain in the study. The investigator may withdraw you from this research if circumstances arise which warrant doing so.

7. IDENTIFICATION OF INVESTIGATORS

If you have any questions or concerns about the research, please feel free to contact

Lara Grobler
Phone: 021 808 2818
Email: 15028151@sun.ac.za

Prof E Terblanche
Phone: 021 808 2742
Email: et2@sun.ac.za

8. RIGHTS OF RESEARCH SUBJECTS

You may withdraw your consent at any time and discontinue participation without penalty. You are not waiving any legal claims, rights or remedies because of your participation in this research study. If you have questions regarding your rights as a research subject, contact Ms Maléne Fouché [mfouche@sun.ac.za; 021 808 4622] at the Division for Research Development.

SIGNATURE OF RESEARCH SUBJECT OR LEGAL REPRESENTATIVE

The information above was described to the participant by Lara Grobler in English and the participant is in command of this language or it was satisfactorily translated to him/her. The participant was given the opportunity to ask questions and these questions were answered to his/her satisfaction.

I hereby consent voluntarily to participate in this study. I have been given a copy of this form.

Name of Subject/Participant

Name of Legal Representative (if applicable)

Signature of Subject/Participant or Legal Representative Date _____

SIGNATURE OF INVESTIGATOR

I declare that I explained the information given in this document to _____ [*name of the subject/participant*] and/or [his/her] representative _____ [*name of the representative*]. [He/she] was encouraged and given ample time to ask me any questions. This conversation was conducted in English and no translator was used.

Signature of Investigator Date _____

APPENDIX B

HEALTH QUESTIONNAIRE

Researcher contact info:
Lara Grobler
15028151@sun.ac.za
021 808 2818



Name:
Email address:
Tel:
Participant number (official use):

HEALTH HISTORY

Please complete the following questions.

Contact number of general physician/ doctor		
Has your doctor ever said that you may not do any physical activity?	No <input type="checkbox"/> Yes <input type="checkbox"/>	
Do you feel pain in your chest when you do physical exercise?	No <input type="checkbox"/> Yes <input type="checkbox"/>	
Do you smoke?	No <input type="checkbox"/> Yes <input type="checkbox"/>	
Have you had any chest pains in the past month?	No <input type="checkbox"/> Yes <input type="checkbox"/>	
Do you lose your balance because of dizziness?	No <input type="checkbox"/> Yes <input type="checkbox"/>	
Do you experience the loss of consciousness?	No <input type="checkbox"/> Yes <input type="checkbox"/>	
Do you have a bone or joint problem that could be aggravated with exercise?	No <input type="checkbox"/> Yes <input type="checkbox"/>	
If yes, please specify:		
Are you using any medication?	No <input type="checkbox"/> Yes <input type="checkbox"/>	
If yes, please specify: Name and indicate if chronic		
Do you know of any reason why you should not participate in this study?	No <input type="checkbox"/> Yes <input type="checkbox"/>	
Have you recently had an injury which limited your activity levels?	No <input type="checkbox"/> Yes <input type="checkbox"/>	
Do you suffer from any of the following conditions? Please specify if necessary.		
Musculo- skeletal problems	No <input type="checkbox"/> Yes <input type="checkbox"/>	
Metabolic- and endocrine disorders	No <input type="checkbox"/> Yes <input type="checkbox"/>	
Immune deficiencies	No <input type="checkbox"/> Yes <input type="checkbox"/>	
Cardiorespiratory disorders	No <input type="checkbox"/> Yes <input type="checkbox"/>	
Cardiovascular disorders	No <input type="checkbox"/> Yes <input type="checkbox"/>	
Haematological problems	No <input type="checkbox"/> Yes <input type="checkbox"/>	
Previous performance		
Partake in competition during the past 3 months?	No <input type="checkbox"/> Yes <input type="checkbox"/>	
Last best performance		
How many times per week do you train?		
For how many years have you been competing?		

APPENDIX C

ETHICAL CLEARANCE

Researcher: Ms Lara Grobler

Research Project: The effect of graduated compression socks on calf muscle oxygenation in endurance athletes.

Nature of the Research Project: M degree, Department of Sport Science, Stellenbosch University

Reference number: 511/2011

Date: 31 May 2011

Ethical clearance for the project, *The effect of graduated compression socks on calf muscle oxygenation in endurance athletes*, has been obtained from the Ethics Committee on 31 May 2011 on condition that:

1. The researcher remains within the procedures and protocols indicated in the proposal,
2. The researcher stays within the boundaries of applicable national legislation, institutional guidelines, and applicable standards of scientific rigor that are followed within the field of study,
3. Any substantive changes in the research project should be brought to the attention of the Ethics Committee with a view to obtain ethical clearance for it.