

**PERFORMANCE AND FATIGUE CHARACTERISTICS  
OF PARALYMPIC ATHLETES WITH CEREBRAL  
PALSY**

**BY**

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**This thesis is dedicated to my mother, and the memory of my father**

## TABLE OF CONTENTS

DECLARATION .....	vii
ACKNOWLEDGEMENTS .....	viii
PUBLICATIONS ASSOCIATED WITH THIS THESIS .....	x
CONFERENCE PROCEEDINGS ASSOCIATED WITH THIS THESIS .....	xii
ABSTRACT OF THIS THESIS .....	xiii
SUMMARY OF STUDIES INCLUDED IN THIS THESIS .....	xiv
Study 1 .....	xiv
Study 2 .....	xiv
Study 3 .....	xv
Study 4 .....	xvi
LIST OF ABBREVIATIONS USED IN THIS THESIS.....	xvii

<b>CHAPTER 1 - The effects of exercise training on performance and functional capacity in individuals with cerebral palsy .....</b>	<b>1</b>
INTRODUCTION.....	2
Cerebral palsy: origins and definitions.....	2
Cerebral palsy and athletic participation.....	3
Functional classification of cerebral palsy .....	3
Research in cerebral palsy.....	4
METHODS .....	5
RESULTS AND DISCUSSION .....	6
Strength.....	6
Aerobic capacity.....	8
Anaerobic capacity and Agility .....	10
Flexibility .....	12
PROPOSED FACTORS LIMITING PERFORMANCE .....	24
Motor impairments and functional performance.....	24
Central deficit.....	26
Fatigue.....	27
Skeletal muscle morphology .....	28
Physical inactivity.....	28
SUMMARY .....	30

<b>RESEARCH QUESTIONS OF THE CURRENT THESIS</b> .....	31
Question 1 .....	31
Question 2 .....	31
Question 3 .....	31
Question 4 .....	32
<b>CHAPTER 2 - A description of bone mineral density and body composition in elite Paralympic athletes with cerebral palsy</b> .....	33
INTRODUCTION.....	34
METHODS .....	36
Participants .....	36
Dual Energy X-Ray Absorptiometry body scan .....	37
Statistics .....	38
RESULTS.....	38
Participant characteristics .....	38
Age and body composition .....	39
Bone mineral density and bone mineral density Z scores.....	41
Fat mass and fat-free soft tissue mass.....	42
DISCUSSION .....	43
CONCLUSION .....	45
<b>CHAPTER 3 - A description of sprint cycling performance and neuromuscular characteristics in elite Paralympic athletes with cerebral palsy</b> .....	47
INTRODUCTION.....	48
METHODS .....	49
Participants .....	49
General Overview of Testing.....	50
Wingate Anaerobic Sprint Cycle Test.....	51
Electromyography .....	52
Statistical Analysis .....	53
RESULTS.....	54
Participants .....	54
Wingate Anaerobic Sprint Cycle Test.....	55
Electromyography .....	55

DISCUSSION .....	60
CONCLUSION .....	64
<b>CHAPTER 4 - The effects of induced volitional fatigue on sprint and jump performance in elite Paralympic athletes with cerebral palsy .....</b>	<b>65</b>
INTRODUCTION .....	66
METHODS .....	68
Participants .....	68
General Overview of Testing .....	69
40m Sprint Test .....	70
Vertical Jump Test .....	70
Multistage Shuttle Run Test .....	71
Electromyography .....	71
Statistical Analysis .....	73
RESULTS .....	73
Participants .....	73
Multistage shuttle run test .....	74
40m Sprint Test .....	75
Vertical Jump Test .....	75
Electromyography .....	77
DISCUSSION .....	85
CONCLUSION .....	90
<b>CHAPTER 5 - The effect of distance deception on pacing during shuttle running trials in Paralympic athletes with cerebral palsy .....</b>	<b>91</b>
INTRODUCTION .....	92
METHODS .....	94
Participants .....	94
General overview of testing .....	95
Shuttle Run Test .....	95
Heart rate .....	96
Ratings of perceived exertion .....	96
Electromyography .....	96
Statistical analyses .....	97

RESULTS.....	98
Participants.....	98
Performance, heart rate and ratings of perceived exertion .....	99
Shuttle Run Test.....	101
Heart Rate .....	103
Ratings of perceived exertion.....	103
Electromyography .....	104
DISCUSSION .....	106
CONCLUSION .....	113
<b>CHAPTER 6 - Summary, interpretation, conclusions and clinical implications .....</b>	<b>114</b>
SUMMARY OF STUDIES INCLUDED IN THIS THESIS.....	115
INTERPRETATION AND IMPLICATIONS OF THIS THESIS .....	117
Social integration and sport in individuals with disabilities, and the growth of the Paralympic Movement.....	117
Cerebral palsy and the current thesis.....	119
Implications of long term exercise training.....	120
Implications of residual effects of cerebral palsy .....	121
Implications of central deficit and conservative pacing in cerebral palsy .....	124
Application of electromyography as a tool in the rehabilitation and training of individuals with cerebral palsy .....	125
LIMITATIONS OF THIS THESIS .....	126
The use of a small sample size .....	126
The use of T37 and T38 athletes with cerebral palsy .....	127
Selection of the control group.....	127
Study design limitations .....	128
SUGGESTIONS FOR FUTURE RESEARCH.....	130
CONCLUSION .....	131
<b>REFERENCES .....</b>	<b>132</b>
<b>APPENDIX .....</b>	<b>150</b>
APPENDIX I .....	151
APPENDIX II .....	152

## DECLARATION

<b>PhD Thesis title</b>	<b>Performance and fatigue characteristics of Paralympic athletes with cerebral palsy</b>
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### 1. DECLARATIONS:

I PHOEBE ANNE RUNCIMAN (full name) hereby:

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**Signed by candidate**

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## **PUBLICATIONS ASSOCIATED WITH THIS THESIS**

### **Chapter 1**

Runciman, P., Tucker, R., Ferreira, S., Albertus-Kajee, Y. & Derman, W. (2015). The effects of exercise training on performance and function in individuals with cerebral palsy: a critical review. *SA.J.Res.Sport.Phys.Ed.Rec.* Accepted for publication (2015)

### **Chapter 2**

Runciman, P., Micklesfield, L., Ferreira, S., Tucker, R., Albertus-Kajee, Y., & Derman, W. (2015). Sire specific bone mineral density is unaltered despite differences in fat free soft tissue mass between affected and non-affected sides in hemiplegic paralympic athletes with cerebral palsy. *A.J.Phys.Med.Rehab.* In review (2015)

### **Chapter 3**

Runciman, P., Derman, W., Ferreira, S., Albertus-Kajee, Y., & Tucker, R. (2015). A descriptive comparison of sprint cycling performance and neuromuscular characteristics in able-bodied athletes and paralympic athletes with cerebral palsy. *Am.J.Phys.Med.Rehabil.*, 94, 28-37. Published (2015)

### **Chapter 4**

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## **Chapter 5**

Runciman, P., Tucker, R., Ferreira, S., Albertus-Kajee, Y., & Derman, W. Paralympic athletes with cerebral palsy display altered pacing strategies in distance deceived shuttle running trials. *Scand.J.Med.Sci.Sports*. Published online ahead of print (2015)

Permission has been requested and granted for inclusion of all papers published or accepted for publication in this thesis, at the time of submission (August 2015).

## **CONFERENCE PROCEEDINGS ASSOCIATED WITH THIS THESIS**

### **International**

P. Runciman, W. Derman, S. Ferreira, R. Tucker. A description of sprint performance and neuromuscular fatigue in Paralympic athletes with cerebral palsy. IPC VISTA2013 conference. Bonn, Germany (2013).

P. Runciman, Y. Albertus-Kajee, S. Ferreira, W. Derman. Novel uses of electromyography in evaluation of skeletal muscle recruitment during exercise in athletes with disabilities: a key to injury prevention, treatment and future classification? IPC VISTA2013 Conference. Bonn, Germany (2013).

### **Local**

Y. Albertus-Kajee, P. Runciman, J. Fisher, N. Tam, D. Coetzee, G. Milhandri. Clinical electromyography seminar: Neuromuscular characteristics in disability using surface electromyography. 5<sup>th</sup> South African Clinical Sports Medicine Conference. Cape Town, South Africa (2014).

P. Runciman, Y. Albertus-Kajee. Novel uses of electromyography in athletes with disabilities. 5<sup>th</sup> South African Clinical Sports Medicine Conference. Cape Town, South Africa (2012).

## **ABSTRACT OF THIS THESIS**

The studies described in this thesis were the first to investigate in-depth body composition, exercise performance and neuromuscular characteristics of elite Paralympic athletes with cerebral palsy (CP).

In the first study, in-depth whole body and site specific body composition was investigated in six athletes with CP using dual-energy x-ray absorptiometry. There were no differences between non-affected and affected sides with respect to bone mineral density and fat mass. Fat free soft tissue mass was lower on the affected side in both upper and lower limbs of the athletes. The novel findings of this study provided the first insight into anthropometric and bone physiology of elite Paralympic athletes with CP, and the possible residual effect of CP in these individuals.

In the second study, five athletes with CP and 16 able-bodied (AB) age and performance matched controls performed a 30 second Wingate sprint cycle test. Power output was significantly higher in the AB group, although fatigue indices were statistically similar between groups. Muscle activity changed similarly in all muscle groups tested, in both affected and non-affected sides, in both CP and AB groups. However, certain neuromuscular irregularities were identified in the CP group. The similarity in fatigue profile was a novel finding. It was proposed that this similarity in fatigue was the result of long term high level athletic training required for Paralympic competition.

Study three tested the similarity in fatigue between CP and AB athletes (that was described in the second study), using an externally paced fatiguing running trial. Six athletes with CP and 12 AB athletes performed one 40 m sprint test and vertical jump tests off both legs, the affected leg individually and the non-affected leg individually, before and after an adapted multistage shuttle run test to exhaustion. The 40 m sprint test, vertical jump off both legs and vertical jump off the affected leg were significantly compromised in the CP group, while vertical jump off the non-affected leg was similar between groups. Both groups fatigued similarly with regard to performance and muscle activity. The third study's findings generally supported those of the second study. However, it was shown that although athletes with CP may represent a group of individuals who have achieved maximal physiological adaptation toward AB levels, the activity generated by both legs was performed towards the capacity of the affected leg.

Study four attempted to elucidate explanations for the novel findings in studies 2 and 3 through investigation of pacing strategies employed by these athletes. Six athletes with CP and 13 AB athletes performed two trials of eight sets of ten shuttles (totalling 1600 m). One trial was distance deceived and the other was non distance deceived. The CP group ran slower than the AB group in both trials, and differences in pacing were observed in the deceived trial in the CP group. This novel study provided evidence for a possible pacing strategy underlying the exercise performance and fatigue profiles observed in the athletes with CP documented in the previous studies.

The work described in this thesis lends novel insights and understanding to the physiology and physiological adaptations of highly functioning ambulant athletes with CP. The findings might have important implications with respect to the understanding of rehabilitation, coaching and clinical management of individuals with CP.

## **SUMMARY OF STUDIES INCLUDED IN THIS THESIS**

### **Study 1**

The first study in this thesis investigated in-depth whole body and site specific body composition in six elite Paralympic athletes with cerebral palsy (CP) using dual-energy x-ray absorptiometry. Bone mineral density (BMD,  $\text{g/cm}^2$ ), BMD Z scores (standard deviations), fat mass (FM, kg) and fat free soft tissue mass (FFSTM, kg) were measured in specific sites on both affected and non-affected sides. There were no differences between affected and non-affected sides with respect to BMD, BMD Z scores and FM. FFSTM was significantly lower on the affected side in both upper and lower limbs (15% lower,  $P < 0.05$ ). The similarity in BMD between sides as well as FM symmetry provided the first insight into physiological adaptation toward typically-developed individuals in elite athletes with CP, possibly as the result of high level, long term athletic training. Lower whole body BMD and asymmetry in FFSTM between the affected and non-affected sides indicated that there may be a residual effect of CP on the body that limits this adaptation at a certain point.

### **Study 2**

The aim of the second study was to investigate sprint cycling performance and neuromuscular characteristics of elite Paralympic athletes with CP during a fatiguing maximal cycling trial, compared to able-bodied (AB) athletes. Five elite athletes with CP and 16 AB age and performance matched controls performed a 30 second Wingate sprint cycle test. Power output (W/kg) and fatigue index (%) were calculated. Electromyography (EMG) was measured in five bilateral muscles and expressed in mean amplitude (% max) and median frequency (Hz). Power output was significantly higher in the AB group ( $10.4 \pm 0.5$  W/kg for the AB group) vs.  $9.8 \pm 0.5$  W/kg for the CP group,  $P < 0.05$ ). Fatigue index was statistically similar between AB ( $27 \pm 0.1\%$ ) and CP ( $25 \pm 0.1\%$ ). EMG mean amplitude and

frequency changed similarly in all muscle groups tested, in both affected and non-affected sides, in CP and AB groups ( $P < 0.05$ ). However, neuromuscular irregularities were identified in the CP group. The similarity in fatigue between the CP and AB groups indicated that elite athletes with CP may have a different exercise response to others with CP. As proposed in Study 1, it was proposed that these findings may be the result of high level training over many years.

### **Study 3**

Study 3 of the present thesis investigated performance, neuromuscular characteristics and fatigue in elite Paralympic athletes with CP during a maximal explosive performance trial, compared to well trained, sprint specific AB athletes. Six Paralympic athletes with hemiplegic CP and 12 AB athletes performed one 40m sprint test (40m ST, s) and vertical jump tests off both legs (VJ-B, cm), affected leg individually (VJ-A, cm) and non-affected leg individually (VJ-NA, cm) before and after an adapted Multistage Shuttle Run test to exhaustion. Electromyography (EMG) of five bilateral muscles was measured for mean amplitude (% max). Significantly compromised performance was observed in the CP group during the 40m ST (6 – 9%), VJ-B (18%) and VJ-A (33 – 36%), while VJ-NA was similar between groups ( $P < 0.05$ ). Both groups fatigued similarly in performance as well as EMG activity. Affected side EMG was higher than non-affected EMG in VJ-B and VJ-A in both groups. The similarity in fatigue between CP and AB groups confirmed that Paralympic athletes with CP may have overcome the deficits associated with CP, which has been documented in sedentary children. The identified asymmetry may assist with a deeper understanding of performance impairment in individuals with CP, as it was shown that activity generated by both legs was performed towards the capacity of the affected leg.

#### **Study 4**

The fourth study investigated performance and physiological characteristics in order to understand pacing strategies adopted by elite Paralympic athletes with CP. Six Paralympic athletes with CP and 13 AB athletes performed two trials of eight sets of 10 shuttles (total 1600 m). One trial was distance-deceived (DEC, 1000 m + 600 m) and one trial was non-deceived (N-DEC, 1600 m), and were performed in a random order. Time (s), heart rate (HR, bpm), ratings of perceived exertion (RPE, units) and electromyography (EMG) of five bilateral muscles were reported for each set of both trials. The CP group ran 10% slower than the AB group, and pacing differences were seen in the CP DEC trial, presenting as a flattened pacing profile over the trial ( $P < 0.05$ ). HR was higher and RPE was lower in the CP group in both trials ( $P < 0.05$ ). EMG showed small differences between groups, sides and trials. This study provided the first evidence for a possible pacing strategy underlying exercise performance and fatigue in CP. The results of this study show that the athletes with CP underperformed the trials compared to the AB group, and that the CP group utilised a conservative pacing strategy compared to the AB group. It was proposed that even at high levels of performance as well as significant adaptation of elite athletes' physiology toward able-bodied levels, the residual effects of CP may negatively affect performance through the selection of conservative pacing strategies during exercise.

## LIST OF ABBREVIATIONS USED IN THIS THESIS

Able-bodied	AB
Affected	A
Analysis of variance	ANOVA
Body mass index	BMI
Bone mineral content	BMC
Bone mineral density	BMD
Cerebral palsy	CP
Cerebral Palsy International Sports and Recreation Association	CPISRA
Degrees per second	°/sec
Dominant	D
Electromyography	EMG
Fat free soft tissue mass	FFSTM
Fat mass	FM
Forty meter sprint test	40m ST
Gross motor functional classification system	GMFCS
Heart rate	HR
International Paralympic Committee	IPC
Kilograms	kg
Kilogram/kilogram body mass	kg/kg BM
Medical subject heading	MeSH
Meter	m
Microvolt	mV
Millimetres	mm
Milliseconds	ms

Motor axon muscle activation	M-wave
Non-affected	NA
Non-dominant	ND
Not significant	NS
Number of participants	N
Oxygen uptake	VO <sub>2</sub>
Peak oxygen uptake	VO <sub>2</sub> peak
Percentage maximum	% max
Range of movement	ROM
Ratings of perceived exertion	RPE
Seconds	s
Standard deviation	SD
Surface Electromyography for the Non-Invasive Assessment of Muscle	SENIAM
Three dimensional	3D
Track/Field	T/F
Vertical jump off affected leg	VJ-A
Vertical jump off both legs	VJ-B
Vertical jump off non-affected leg	VJ-NA
Watts per kilogram	W/kg

# CHAPTER 1

## CRITICAL REVIEW OF THE LITERATURE

### **The effects of exercise training on performance and functional capacity in individuals with cerebral palsy**

Certain components of the present chapter has been accepted for publication as a critical review article in the *South African Journal for Research in Sport, Physical Education and Recreation* (2015)

## INTRODUCTION

### **Cerebral palsy: origins and definitions**

Cerebral palsy (CP) was first termed Little's Disease after Dr William John Little (1810 - 1894), who was the first to study infantile malformation, and the first to characterise hypertonic diplegic CP, attributing the condition to trauma at birth. Sir William Osler (1849 - 1928) later introduced the term "cerebral palsy" during his extensive clinical research on children with CP. Sigmund Freud (1865-1939), although not recognised for many years, contributed significantly to the area of CP research by identifying pre-natal causes of the condition, as well as grouping the different forms of CP under the umbrella term "infantile cerebral palsies" <sup>1,2</sup>.

Cerebral Palsy, defined by Bax in 1964 as "a disorder of posture and movement due to a defect or lesion of the immature brain" <sup>3</sup> has an estimated incidence of 2.5 per every 1000 live births <sup>4</sup>. This definition has since been expanded to include a group of movement disorders caused by damage to the immature brain before, during, or directly after birth. The damage occurs in one or more of three main areas in the brain controlling movement, namely the motor cortex, cerebellum and basal ganglia <sup>5</sup>. Hypertonic CP is the most common form of CP, with athetoid, ataxic and mixed CP being less common <sup>6</sup>. Limb distribution of the impairment is present in three main categories, namely hemiplegic, diplegic and quadriplegic impairment profiles, where hemiplegic profiles refer to one half (left or right) of the body being affected, diplegic profiles refer to two limbs being affected together (usually both lower limbs) and quadriplegic profiles refer to all four limbs being affected <sup>7</sup>.

### **Cerebral palsy and athletic participation**

Participation in competitive sport by individuals with disabilities is a relatively new phenomenon, with the first organised sports competition for individuals with disabilities held in 1948 for World War II veterans with spinal cord injuries <sup>8</sup>. Between 1948 and 1960, the International Stoke Mandeville Games grew in size and eligible impairment types, until the first Paralympic Games were held in Rome in 1960. The Paralympic movement has grown over the years, with 4176 athletes from 164 countries competing in the 2012 London Paralympic Games, an increase from the 2008 Beijing Paralympic Games which hosted 3951 athletes from 146 countries. One of the most populated categories for athletes with physical disabilities at the Games is that of CP. Athletes with CP compete in many sports, but predominantly track and field, football, swimming and boccia <sup>9</sup>.

### **Functional classification of cerebral palsy**

For clinical purposes within research, the functional abilities of individuals with CP have been classified into five discernible groups within the Gross Motor Function Classification System (GMFCS) <sup>10</sup>. The system is scored from minimally affected (level I, unaided ambulation) to severely affected (level V, permanent wheelchair use) individuals with CP. In Paralympic classification, athletes with CP are similarly characterised according to functional ability or impairment. For example, eight categorical classes of functional ability have been outlined for track and field (T/F) events. These classes range from T/F 31 - 38, where T/F 31 - 34 denote more severely affected athletes using wheelchairs, while T/F 35 - 38 comprise ambulatory athletes <sup>11-13, 197</sup>.

## **Research in cerebral palsy**

With the growth of both clinical and sporting populations with CP over the last century, research in CP has been carried out, predominantly addressing the need for improved quality of life in individuals with the condition. Many interventions have aimed to address quality of life, exercise therapy being an example. The use of exercise in the treatment and management of individuals with CP has yielded positive results from both traditional and alternative methods.

The majority of these studies, however, have focused on quality of life within severely affected paediatric patients, and not exercise performance capacity in athletic adults, due in part to the relative urgency for research within highly affected populations. Although exercise performance measures are often reported in these studies, no studies to date have definitively described exercise and performance capacities of children and adults with CP from a sports performance perspective. Moreover, there is neither consistent nor longitudinal evidence for the use of exercise training in improving sport performance in this patient population.

To provide consistency on this clinical condition, the present review summarises the current literature on the exercise and performance capacities of individuals with CP, describing: 1) the effect of CP on specific aspects of exercise performance, 2) the response to exercise training in individuals with CP and the results of interventions aimed at improving these aspects, and 3) how the physiology underlying the functional or physical deficits present in CP can be better understood.

For the purpose of this review and subsequent studies described in this thesis, only previous studies using individuals in the GMFCS levels I and II were reviewed. This ensured an appropriate comparison of functional performance between individuals with truly ambulant CP and typically developed able-bodied (AB) individuals, as GMFCS classifications beyond level II infer the use of assistive devices or wheelchairs, which would not enable close functional matching. Also, due to the lack of literature in athletic adult samples, studies using paediatric samples have been reviewed. All literature identified in adults or athletes in GMFCS levels I and II were included. For a complete summary of the intervention studies included in this review, refer to Table 1.

## **METHODS**

A literature search was conducted using PubMed, Medline, Embase, Scopus, Web of Science, Science Direct and Google Scholar databases from the earliest possible date to June 2015. The medical subject heading (MeSH) terms included *cerebral palsy*, *brain injury*, *spasticity* and related terms. The search terms, matched with the MeSH included *classification*, *adult*, *athlete*, *exercise training*, *strength*, *aerobic*, *anaerobic*, *agility*, *speed*, *flexibility*, *electromyography*, *muscle*, *physical inactivity* and related terms. Abstracts were screened, and individual articles were selected based on quality and focus of the studies. Furthermore, the reference lists of articles were also searched for further studies. This review primarily investigated individuals with CP, and excluded individuals with traumatic brain injury or other neurological disorders causing movement abnormalities. Only studies published in the English language were included.

## RESULTS AND DISCUSSION

Elite athletic performance is the result of a complex interaction between numerous biomotor abilities that confer a physical performance advantage to an individual. These same biomotor abilities are of interest to those who use exercise training to improve functional quality of life, and include attributes of strength, aerobic capacity, agility, anaerobic capacity and flexibility. As mentioned previously, little research has investigated performance variables in a specifically trained athletic population with CP. Research has primarily been conducted in sedentary children and adults with CP, with the goal of improving quality of life via exercise training.

These variables are described below with reference to, firstly, how CP affects the variable, and secondly, the effect of specific exercise training interventions on the variable.

### **Strength**

Strength is the ability of a musculotendinous unit to produce force, both statically and dynamically, through different types of muscle contractions <sup>14</sup>. There is a longstanding recognition of muscular weakness, or the inability to exert the required force, as an adverse component of CP that affects quality of life in individuals affected by the condition <sup>15</sup>. Indeed, muscular weakness, which was previously thought not to have an effect on functional capacity in CP, is now considered by some to be more harmful to functional ability than spasticity <sup>16</sup>.

Weakness in individuals with CP has been widely demonstrated, with strength deficits ranging from 30 - 73% in children <sup>17-19</sup>, and 12 - 52% in adults <sup>20-22</sup>. Only one of these

studies (De Groot et al, 2012) used an athletic adult sample, of cycling and soccer athletes. They reported an isometric knee extension strength deficit ranging between 31% and 47% in elite soccer players and cyclists with CP, compared to AB soccer players and cyclists <sup>22</sup>.

Because the strength deficits observed in this population are so large, and have negative consequences for normal function, numerous studies have examined how exercise training interventions might improve strength and functional performance. These studies have mainly focused on children, specifically for functional gains, but they offer insight into the potential improvement capacity that may exist for individuals with CP.

The most compelling studies that support the benefits of muscle strength training in individuals with CP have found improvements of between 13% <sup>19</sup> and 69% <sup>23</sup> from six weeks of traditional strength training in hemiplegic and diplegic children in GMFCS levels I and II. This large range is the likely result of variations in methodology, differences between the size of muscle groups trained (smaller upper muscle group and improvement vs. larger lower limb muscle group and improvement), as well as normal inter-individual variations in response to training. Studies conducted on adults show similar results in strength gains, but with longer intervention periods (10 – 12 weeks) <sup>24-26</sup>.

Of particular interest is the effect of strength training on the asymmetry between the affected (A) and non-affected (NA) sides after training. The marked asymmetry between A and NA sides observed in individuals with CP has been found to be reduced, though not eliminated, by strength gains in hemiplegic profiled individuals, suggesting a larger potential for strength gains on the A side. In a study conducted by Damiano (1998), a 24% asymmetry was measured after the six week strength training intervention, and while this was a reduction

from a pre-training asymmetry of 42%, the authors did not speculate whether a longer training intervention would further minimise this difference <sup>23</sup>. Their finding does however invite this possibility.

As expected, detraining was observed to produce the opposite finding, with complete detraining of an adult with CP observed within four weeks of completing a 12 week cycling intervention <sup>24</sup>. This individual had experienced an 19% and 22% improvement in quadriceps and hamstring strength, respectively, during the 12 week training period <sup>24</sup>, however the individual returned to baseline capacity four weeks after complete cessation of exercise. It must be acknowledged that the cessation of functional electrical muscle stimulation may have influenced the rate of detraining in this individual.

In fact, the detraining-induced loss of strength observed in the above mentioned adult individual with CP was greater than that typically observed in well trained and sedentary AB adults <sup>27,28</sup>. This finding suggests that there may be an inability to maintain exercise-induced physiological adaptation in individuals with CP, resulting in heightened detraining effects. No research to date has evaluated the adaptation to long term, high level training in individuals with CP, nor evaluated individuals who have been involved in high volume exercise training for many years, which would allow further understanding of the true effect CP has on the body, without the confounding factors of sedentary participants and short duration intervention durations.

### **Aerobic capacity**

The aerobic capacity of children with CP has long been recognised as limited compared to that of typically developing children <sup>29</sup>. Reductions in  $VO_2$ peak ranging from 15 - 42%

compared to healthy controls indicate a compromised aerobic capacity in paediatric studies<sup>29-32</sup>. These findings have been supported by 23 - 45% and 21 - 61% impairments in  $VO_2$ peak in adult men and women, respectively<sup>33, 34</sup>.

When comparing AB controls to aerobically trained athletic adults with CP,  $VO_2$ peak has been found to be 0.3% and 21% lower  $VO_2$ peak for athletes in GMFCS levels I and II, respectively. This is significantly closer to age matched controls compared to other studies conducted on sedentary individuals with CP<sup>22</sup>, and suggests a potential benefit of exercise training.

In terms of performance, compromised aerobic capacity may affect game play in football, with elite football players with CP covering 43 - 50% less distance than position matched AB controls. This performance deficit is similar to that seen in other research which uses sedentary individuals with CP, although this group did present with a similar running economy to the AB control group<sup>35</sup>.

Aerobic exercise interventions have been implemented in children as young as two years old<sup>36</sup>. Although existing intervention studies differ in terms of samples studied, testing methods, intervention structure and outcome measures, the general finding is that exercise training improves aerobic capacity significantly<sup>37</sup>.

Self-selected walking speed, often used as an outcome measure for aerobic capacity in young children with CP, increased after training interventions, along with energy efficiency<sup>38-</sup><sup>43</sup>. A more rigorous testing method using the lactate threshold to determine exercise intensity has reported a 20% increase in aerobic capacity in children and a 12% increase in

aerobic capacity in adults, using a similar protocol<sup>44, 45</sup>. Of interest in these studies was that arm ergometry power output was unchanged as a result of the exercise intervention, whereas leg ergometry power output improved significantly, indicating larger aerobic capacity when training larger muscle groups, as proposed previously<sup>44</sup>.

Larger aerobic capacity effects may also be possible with longer training interventions, since documented improvements were only seen in week eight (final week) of the intervention. This, coupled with the improvements in the paediatric study being almost twice that of the adult study, supports the notion that adaptation at a young age might be easier to achieve, and therefore higher volumes and intensities of training before adulthood should be encouraged.

### **Anaerobic capacity and Agility**

The ability to perform high-intensity activities over a short duration is a function of both anaerobic capacity for the provision of rapidly-available energy, and agility, a function of balance, coordination and movement ability. Both are compromised in individuals with CP

<sup>46-50</sup>

Anaerobic performance of children with CP, assessed using the 30 second Wingate sprint cycle test, has been found to be two to four standard deviations lower than in typically developing children. Other studies report a 27 - 46% deficit in peak power output in children with CP using an adapted 20 second Wingate sprint cycle test<sup>47-50</sup>.

Comparisons of agility between CP and control groups are scarce. From the available literature, it was found that the average time to complete a repeat sprint agility task was 39%

longer in children with CP. Indeed, some participants with CP took over 60 seconds to complete a task that took only 19.5 seconds for the AB participants to complete<sup>48</sup>.

There is also limited research on the potential to increase anaerobic capacity and agility with training in individuals with CP. In the only study available, Verschuren et al. (2007) found a 25% and 15% increase in anaerobic capacity and agility, respectively, following an eight month intervention targeting these variables<sup>51</sup>. This intervention is one of the longest duration interventions in individuals with CP, and also included a four month follow up after completion of the study. This follow-up, during which time no supervised training or encouragement to train was provided, revealed a 9% and 4% reduction in anaerobic capacity and agility, respectively, from peak values achieved at the end of the 8-month programme.

These declines in performance resulted in overall anaerobic capacity and agility that was comparable to performances observed at the four-month mark of the training intervention and are similar to detraining induced declines in performance observed in AB individuals<sup>27</sup>.<sup>28</sup> These declines do not, however, resemble the findings of the strength study conducted by Johnson and Wainwright (2011) on an adult with CP, where a larger detraining effect was seen over a shorter period following the completion of the training programme (see Strength section of this review). This difference in rate of detraining may be explained by individual variation (the strength study was a case study of one individual), a shorter intervention in the strength study, or a consequence of paediatric individuals being used in the anaerobic and agility study. This may provide further evidence that the cerebral palsied body may be unable to maintain adaptation to exercise as an adult due to a lack of childhood exercise

induced adaptation. This would further support early participation in traditional exercise programs by children with CP.

### **Flexibility**

Flexibility is the ability of a muscle to elongate in response to a stretching force, and has functional limits for each particular joint<sup>14</sup>. Flexibility in individuals with CP is purportedly impaired due to spasticity and contracture, although it has also been attributed to a reduction of in-series sarcomeres, muscle atrophy and muscle co-activation<sup>52-58</sup>. A possible benefit of impaired flexibility may be increased stability of the involved joint, but a lack of flexibility may also decrease performance due to improper biomechanics during movement<sup>59</sup>.

Although lack of flexibility and the presence of contractures have been well documented in individuals with CP, it has not yet been concluded whether training interventions can successfully improve flexibility in this population. Some interventions have found improved dynamic flexibility, muscle tone and reduced spasticity with the use of stretching interventions<sup>60, 61</sup>, while other studies found no change in flexibility, despite positive changes in other parameters like spasticity and functional ability at the conclusion of flexibility interventions<sup>62-64</sup>.

The differences in these studies may once again be explained by the age of the participants included in the studies. The only study that reported entirely positive results was conducted by McPherson et al. (1984) in children with CP<sup>60</sup>, while the other studies that reported variable or inconclusive results were conducted on adolescents or adults with CP. Once again, this suggests that interventions may be more successful and have possibly longer term effects if started at a young age<sup>63, 64</sup>.

Table 1 summarises exercise interventions included in the present review of the literature.



					Gross motor function test	Training group: Improved significantly
Andersson et al 2003 <sup>25</sup>	Training group with CP: N=10 (23-44 years) Control group with CP: N=7 (25-47 years)	Strength training	10 weeks	2 days/week	Spasticity	NS
					Range of movement	NS
					Isometric strength	Training group: Significant improvement (hip extensors, hip flexors) Control group: NS
					Concentric strength	Training group: significant improvement at 30°/s and 90°/s in knee extensors Control group: NS
					Eccentric strength	NS
					Gross motor function measure	Training group: Significant difference Control group: NS

					6 minute walk test	Training group: Significant improvement (walking distance, walking velocity) Control group: NS
					Timed up and go	Training group: Significant difference Control group: NS
Damiano & Abel 1998 <sup>23</sup>	CP: N=11 (6-12 years) Diplegic group: N=6 Hemiplegic group: N=5	Strength training	6 weeks	3 days/week	Isometric strength	Diplegic group: ↑ 69% Hemiplegic group: ↑ 20.3% in affected leg Unaffected leg: NS 24% asymmetry across sides maintained
					3D gait analysis	Asymmetry improved
					Gross motor function measure	Improved

Healy et al 1958	CP: N=5 (8-16 years)	Concentric and isometric strength training	8 weeks	3 days/week	Concentric strength	Significant improvement
					Isometric strength	Significant improvement
					Knee ROM	NS
Johnston et al 2011 <sup>24</sup>	CP: N=1 (49 years)	Cycling with superimposed electrical stimulation	12 weeks	3 days/week	Spasticity	NS
					6 minute walk test	NS
					Timed up and go test	Significantly improved by 24.4%
					Concentric strength	Hip flexor ↑ 22.2% Hip extensor ↑ 18.5%
					Gait parameters	NS
					Medical outcomes study 36- item health survey	Significantly improved
					International classification of functioning, disability and	Significantly improved

					health	
MacPhail & Kramer 1995 <sup>65</sup>	CP: N=17 (12-20 years)	Eccentric, isometric, concentric strength training	8 weeks	3 days/week	Peak torque Peak work Spasticity Efficiency	Strength ↑ 21-25% NS NS
Reid et al 2010 <sup>19</sup>	CP: N=14 (9-15 years) Con: N=14 (9-15 years)	Progressive eccentric strength program	6 weeks	3 days/week	Peak torque to body mass (T/BM) Work to body mass (W/BM) Angle at peak torque Curve width EMG activation	Concentric task: ↑ 13% Eccentric task: ↑ 25% NS NS ↓ to normal amplitude
Toner et al 1998 <sup>66</sup>	CP: N=6 (4-7 years)	Strength training with use of biofeedback	6 weeks	7 days/week	Strength Toe tapping ability	NS Improved

<b>Aerobic capacity</b>							
Chan et al 2004 <sup>38</sup>	CP: N=12 (4-11 years)	Treadmill	4 weeks	3 days/week	3D gait analysis	NS	
		training and			Ankle moment quotient	NS	
		electrical			Ankle power quotient		
		stimulation			Gross motor function measure	Significant improvement	
Chrysagis et al 2012 <sup>67</sup>	Training group with CP: N=11	Training group:	12 weeks	3 days/week	Spasticity	NS	
		Treadmill			Self-selected walking speed	Training group: Significant improvement	
		Control group with CP: N=11			training without body weight support	Gross motor function measure	Control group: NS Training group: Significantly improved
	Control group:	Traditional physiotherapy				Control group: NS	
Mattern-Baxter et al	CP: N=6 (2.5-3.9 years)	Treadmill training	4 weeks	3 days/week	Gross motor function measure	Significant improvement	

2009 <sup>36</sup>					Paediatric evaluation of disability inventory	Significant improvement
					10 m walk test	Over-ground walking speed
					6 minute walk test	and distance covered ↑
Phillips et al 2007 <sup>42</sup>	CP: N= (6-14 years)	Body weight supported treadmill training	2 weeks	2 times/day 6 days/week	fMRI activation 10 m walk test 6 minute walk test	fMRI activation increased Over-ground walking speed and distance covered ↑
Provost et al 2007 <sup>43</sup>	CP: N= (6-14 years)	Body weight supported treadmill training	2 weeks	2 times/day 6 days/week	Energy expenditure 10 m walk test 6 minute walk test Single leg balance test Gross motor function measure	Significant improvement in energy expenditure Over-ground walking speed and distance covered ↑ NS Significant difference

Shinohara et al 2002 <sup>44</sup>	CP: N=11 Leg group: N=6 (13.3-15.8 years) Arm group: N=5 (11.8-16.3)	Anaerobic threshold training Leg group: cycle ergometer training Arm group: arm ergometer training	20 weeks	2 days/week	VO <sub>2</sub> at the anaerobic threshold Self reported endurance capacity	Leg group: VO <sub>2</sub> ↑ 20% Arm group: NS Children reported a perception of increased endurance capacity in only the leg group
<b>Anaerobic capacity and Agility</b>						
Verschuren et al 2007 <sup>51</sup>	CP: N=86 (7-18 years)	Aerobic and anaerobic circuit training	8 months	2 days/week	Muscle power sprint test 10 x 5 m sprint test	Anaerobic capacity ↑ 25% Agility ↑ 15%
<b>Flexibility</b>						
Darrah et al	CP: N=23 (11-20 years)	Active	10 weeks	3 days/week	Spasticity of triceps surae	NS

1999 <sup>63</sup>		stretching for lower extremities			Sit and reach (hamstring flexibility)	NS
					Adductor flexibility	NS
Dickin et al 2013 <sup>61</sup>	CP: N=8 (20-51 years)	Individualised whole body vibration therapy	2 sessions	-	Spasticity	NS
					Range of movement	Dynamic ankle range: Significantly improved
					3D gait analysis	Walking speed, stride length: Significantly improved
McPherson et al 1984 <sup>60</sup>	CP: N=4 (10-18 years)	Year 1: static stretching	2 years	Year 1: 5 days/week	Knee extensor range of movement	Knee ROM ↑ 4-9°
		Year 2: standing posture devices		Year 2: 7 days/week	Muscle tone	Year 2: Decreased muscle tone

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O'Dwyer et al	CP: N=15 (6-19 years)	Passive	42 days	3 days/week	Ankle range of movement	NS
1994 <sup>62</sup>		stretching			Contracture of triceps surae	Significant decrease

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## **PROPOSED FACTORS LIMITING PERFORMANCE**

The functional impairments seen in individuals with CP have been attributed to many mechanisms. These mechanisms have been grouped into the categories of motor impairments, central deficits, skeletal muscle morphological changes and physical inactivity. These mechanisms, described below, assist in understanding the mechanisms underlying the functional and physical deficits seen in individuals with CP, and the effect of CP on human physiology, consequently increasing our understanding of the effect of exercise training in individuals with CP.

### **Motor impairments and functional performance**

One of the explanations for decreased strength, speed and endurance in individuals with CP is the combination of motor impairments present as a result of the condition. These include centrally-mediated muscular weakness, inco-ordination, spasticity, contracture and co-activation. Applied to running based events, these motor impairments can cause marked biomechanical limitation and each is described briefly below.

Motor control of movement and posture has been attributed to three primary levels within the central nervous system, and utilises several secondary systems. The first level of central control originates in the supplementary motor cortex, and controls overall movement strategy. The second level lies within the sensorimotor cortex, cerebellum, basal ganglia and brainstem and translates the specific movement strategy into specific motor programs. The third level lies within the spinal cord and translates the motor programs into muscular activity, which is then regulated through stretch reflexes<sup>68, 69</sup>.

Inco-ordination, spasticity, contracture and co-contraction are all movement irregularities observed in individuals where this central nervous system is disturbed or damaged in some way.

Inco-ordination is the inability of the central nervous system to correctly utilise the proposed pathway to recruit motor units in the correct synchronicity to enable fluid movement <sup>70</sup>. In individuals with CP, this lack of synchronicity can result in an ungainly and awkward walking and running stride, due to the inability to correctly create the activation strategies and programs required for the execution of a regular stride.

Spasticity and muscle contractures are among the most prominent features in CP, and result in muscles that have increased resting and dynamic tone, rigidity and decreased range of movement. These muscle irregularities are a result of dysfunction, in part, of the stretch reflex found at the third level of the motor control hierarchy <sup>68, 71</sup>. Movement can be severely affected, as a result of these irregularities. For example, pelvic rotation may occur due to hamstring muscle spasticity or compromised accuracy of foot strike due to gastrocnemius muscle contracture <sup>13</sup>.

Co-activation is the simultaneous contraction of antagonist muscles during contraction of the agonist muscle group, and is usually seen in typically developing individuals only when a need for increased joint stability is required, such as when walking over highly unstable surfaces <sup>72</sup>. However, co-activation during normal tasks in CP has been highlighted as one of the factors causing impaired performance in many tasks <sup>15</sup>. Two studies have shown high levels of co-activation and impaired performance in individuals with CP <sup>17, 73</sup>. This co-activation likely has a beneficial purpose in individuals with CP, since it contributes to increased joint stability as a muscular strategy. However, it also results in negative

outcomes for movement quality and quantity due to greater total muscle mass activation during movement, as well as increasing the risk of injury<sup>54</sup>.

The combination of motor impairments seen in individuals with CP would thus result in abnormal gait patterns, increased energy requirements for the same task conducted by an AB individual, as well as increased time needed to complete the task<sup>54, 74, 75</sup>.

### **Central deficit**

With the development of technology that allows the measurement of central drive via voluntary muscle activation, central mechanisms involved in the deficits observed in CP can now be investigated. Stackhouse et al. (2005) superimposed electrical muscle stimulation during a maximum voluntary contraction in order to examine the possible central contribution to impaired performance, and discovered that voluntary muscle activation was 33% and 49% lower in the quadriceps and triceps surae, respectively, compared to AB individuals<sup>17</sup>.

This demonstrated a substantial impairment in the brain's ability to recruit muscle, which is consistent with other research<sup>18</sup>. However, the number of motor units available to be used was similar, as measured by M-wave maximum muscle activation amplitudes, suggesting under-activation of skeletal muscle due to central inhibition<sup>76, 77</sup>, rather than a deficit at the level of the muscle or motor neuron junction. This occurs along with co-activation of antagonist muscles<sup>17</sup>, leading to the theory that muscular deficits observed in individuals with CP are the result of a combination of muscular under-activation<sup>19</sup>, decreased central input to the muscle due to damage to the supraspinal centres<sup>78</sup> and co-activation of agonist and antagonist muscles<sup>19, 79</sup>.

## **Fatigue**

The development of muscle fatigue during exercise tasks in individuals with CP has been examined by few studies. In these studies, fatigue was defined as the failure to maintain force output over a period of time during a set task<sup>80</sup>. Stackhouse et al. (2005) was the first to investigate fatigue characteristics of children with CP using maximal voluntary isometric contractions. The authors found that the children with CP maintained force output over the trial compared to AB controls (42% and 52% decline in force output over the trial in CP and AB groups, respectively), indicating fatigue resistance in the CP group<sup>17</sup>. The authors attributed this finding to significant muscle weakness in the CP group, which resulted in the inability to create enough force at the beginning of the exercise trial, subsequently resulting in the production of less force over the trial, but for longer periods of time. This pathological fatigue resistance was associated with lower agonist voluntary muscle activation and higher levels of agonist/antagonist co-activation. Moreau et al. (2008), Moreau et al. (2009) and Eken et al. (2013) aimed to further investigate fatigue in individuals with CP using 35 repetition maximum isokinetic dynamometry protocols<sup>81-83</sup>. The authors found fatigue resistance associated with significant muscle weakness in the children with CP compared to AB controls. They further found that the observed fatigue resistance was associated with lower muscle voluntary activation, co-contraction of agonist and antagonist muscle groups and muscle atrophy of working muscle groups in the tested samples. Interestingly, Moreau et al. (2008) found greater fatigue resistance with higher levels of impairment within the CP group, and proposed that higher levels of impairment and muscle weakness resulted in greater fatigue resistance in this population<sup>81</sup>. Thus, fatigue resistance in individuals with CP was closely related to muscle weakness and the adverse consequences of central deficit.

### **Skeletal muscle morphology**

A further contributor to the performance deficits observed in CP is altered skeletal muscle morphology. Force output of a muscle is the combination of neural factors and the quality or nature of the muscle being activated. Thus it stands to reason that changes in morphology as the result of central deficit associated with CP may impair function at the level of the muscle, further compounding neural factors affecting force production. In this regard, several authors have attributed muscular weakness to an identified increase in Type I muscle fibres in lower extremity muscles in children with CP<sup>17, 84, 85</sup>. In addition to Type I muscle fibre predominance, increased intramuscular fat, atrophy and decreased muscle size in the paretic limbs have also been identified as an outcome of CP. These changes have been consistently attributed to sustained low-frequency muscle fibre firing, caused by spasticity and altered central drive in muscle affected by CP<sup>17, 21, 76, 84-87</sup> and these findings indicate changes at both central and peripheral levels in individuals with CP.

### **Physical inactivity**

The attributes and mechanistic factors discussed in this review, including strength, agility, aerobic endurance, central and peripheral factors, contribute to impairment of physical performance and functional capacity in individuals with CP. Unsurprisingly, these factors are associated with physical inactivity in this population, which is perhaps of highest clinical importance, since it is recognised as a crucial limiting factor for the functional prognosis of individuals with CP.

It is widely accepted that children with CP engage in less physical activity than their typically developing peers, with almost exclusively negative consequences<sup>29, 88, 89</sup>. Durstine et al. (2000) proposed a circular mechanism for physical inactivity in groups of individuals with disabilities, whereby individuals with a disability engage in less physical activity, which

results in deconditioning, further decreasing their level of functioning and thus volitional engagement in physical activity<sup>90</sup>.

This cycle is perpetuated further by psychosocial barriers to exercise, including personal and environmental factors such as the fear of injury or complications and the perception that the environment is unfriendly toward those with disabilities<sup>91</sup>. Lack of information about the benefits of physical activity as well as feelings of embarrassment or shame are also among the many factors that play a role in the complex interaction between psychology and exercise participation in individuals with disabilities<sup>91-94</sup>.

Most of the studies in the area of CP, whether in children or adults, attribute some level of poor performance to a relative lack of exercise training when compared to control groups. Engaging in physical activity, however, has been observed as one of the most important factors for the successful maintenance of function in this population<sup>95</sup>. That is, starting and maintaining exercise has been shown by many studies to be a mediating factor in disease progression<sup>96</sup>. Therefore, the participation of those with CP in exercise programs is particularly important, maybe even more so than their typically developing peers, for proper physiological development<sup>41</sup>. Despite this acknowledgement, maintenance of low levels of physical activity in individuals with CP, and only short duration exercise training interventions persist. The improvements seen in these short duration exercise interventions, however, are significant enough to warrant more detailed, longer term investigations into longitudinal, high dose training interventions in individuals with CP from a young age.

## SUMMARY

CP is a group of congenital neuromuscular disorders that result in reduced physical capacity. Research in individuals with CP has focused mainly on improving quality of life of severely affected and sedentary children, with little research on exercise performance capacity in adults and athletes with CP. It has been established that individuals with CP: 1) present with a large range of deficits in exercise performance, and 2) improve significantly with participation in different modalities of exercise training. However, most of the studies have been short in duration and predominantly investigated sedentary individuals.

Through reviewing the existing literature on physiological and exercise performance deficits in individuals with CP, as well as investigating the possible mechanisms responsible for these deficits, it has become clear that, although CP has been extensively researched, there is no comprehensive conclusion regarding the effect of CP on human physiology. Nor is there evidence for the effect of long term exercise training in these individuals. This is mainly due to the use of paediatric samples, the reported physical inactivity in the study samples, and the relatively short interventions administered, which all act as confounding factors to the true effect of CP on the body. It must be acknowledged that the grouping of clinical studies and sporting performance studies can make the reporting of results difficult, as these two groups of research prioritise different aspects of physiology. Clinical studies generally prioritise health-related outcomes whereas sporting performance studies prioritise sporting performance and physiology. However, due to paucity of research in the sporting category, the categories were grouped in an effort to understand the overall effect of CP on human physiology.

To be able to definitively describe the true effect of both CP on the body and long term exercise training on individuals with CP, it would be beneficial to describe individuals where the confounding factors of low activity status and age have been eliminated as much as possible. Examining individuals who have participated in high level training over a long period of time would enable better identification and understanding of the physiological effect of CP on the body. In-depth investigation into an athletic sample would also give insight into the effect of long term exercise training on the impairments observed in CP.

## **RESEARCH QUESTIONS OF THE CURRENT THESIS**

The following specific research questions will be examined in the present thesis:

### **Question 1**

What are the anthropometric, functional and fatigue characteristics of elite Paralympic athletes with cerebral palsy, and how do the affected and non-affected sides in these hemiplegic athletes compare?

### **Question 2**

What are the physiological and neuromuscular performance characteristics of elite Paralympic athletes with CP compared to age matched able-bodied elite athletes?

### **Question 3**

What are the specific fatigue profiles observed in elite Paralympic athletes with CP, and do they compare to the fatigue profiles reported in the literature using sedentary children with CP?

**Question 4**

What are the possible explanations for the fatigue profiles seen in this elite Paralympic athlete cohort?

Finally, this thesis will attempt to address the above-mentioned four questions with respect to interpretation of the findings of this thesis, and suggest clinical and coaching implications from these findings.

## **CHAPTER 2**

# **A description of bone mineral density and body composition in elite Paralympic athletes with cerebral palsy**

The findings of the present chapter are in review for publication as an original research article in the *American Journal of Physical Medicine and Rehabilitation*, with requested revisions submitted to the journal (2015)

## INTRODUCTION

Numerous developmental disorders have been associated with abnormal growth and restricted physical development, due to damage to one or more physiological systems at a young age. Cerebral palsy (CP) affects 2.5 babies per 1000 live births and is one of the most common developmental disorders that affects the neuromuscular system<sup>5</sup>. Damage to the immature brain before, during or directly after birth to one of three areas of the brain that control movement and co-ordination, results in various physiological impairments in individuals with CP<sup>6, 97</sup>.

As the result of this central insult at a young age, individuals with CP are characterised by limb asymmetry between affected (A) and non-affected (NA) sides, shorter stature, lighter body mass and lower levels of bone mineral content and density than able-bodied (AB) controls<sup>98, 99</sup>. Van den Berg (1998) measured basic body composition using four-site skinfold measurements in 20 adolescents with CP, aged 7 - 13 years. Whole body fat percentage was 18% higher in the children with CP compared to the AB controls ( $21.8 \pm 6.1\%$  vs.  $17.9 \pm 5.0\%$ ), despite similar muscle mass between the groups<sup>100</sup>. Chad et al. (2000) compared 17 ambulant and non-ambulant individuals with CP (7.6 - 13.8 years old) to 894 AB individuals in a paediatric database using dual-energy x-ray absorptiometry (DXA) total body scans<sup>99</sup>. Bone mineral content (BMC, a measure used in paediatric populations) and bone mineral density (BMD) were calculated at the proximal femur, femoral neck and in the total body, whilst fat mass (FM) and fat-free soft tissue mass (FFSTM) were calculated in the total body. BMC Z scores, which represent age and gender matched normative values, were found to be 1.8 standard deviations (SD) lower in total body BMD and 3.2 SD lower in femoral neck BMD in individuals with CP, compared to the control group. Interestingly, BMC and BMD were both lower in non-ambulant children than ambulant children with CP.

Further, although FM was similar between CP and control groups, FFSTM was 2 SD below the control group, which is in alignment with the reductions observed in BMC and BMD<sup>99</sup>.

Recent studies on individuals with CP have provided evidence the contribution of non-nutritional factors in the body composition of individuals with this condition, disputing previous research which attributed low BMD in children with CP to vitamin D deficiency<sup>99, 101-103</sup>. These studies focused on the functional weight bearing cortical bones of the hip, which respond to mechanical loading. It was proposed that the lower BMC, BMD and FFSTM reported in these individuals with CP were the result of low volumes of ambulation, and subsequent lower bone and muscle loading in these patients. These findings supported the research discussed above that found that BMD was lower in non-ambulant children with CP. However, both ambulant and non-ambulant CP groups presented with at least 1 SD lower BMD and BMC compared to the control database. This showed a residual effect of CP on the physiology of these individuals, despite levels of ambulation<sup>99</sup>.

The effect of mechanical loading on BMD in cortical bone is well established. Research conducted on astronauts returning from long-term space travel has definitively correlated the lack of mechanical loading whilst in space to significant losses in BMD<sup>104</sup>. When investigating the effect of unilateral loading on BMD in athletic individuals, researchers have shown that BMC and BMD are significantly increased in the dominant arm of squash and tennis players, compared to non-dominant and control group arms<sup>105, 106</sup>. This increase was purportedly not related to isokinetic concentric strength of surrounding muscles in the arm, but rather the high levels of loading over long periods of time associated with racquet sports<sup>105</sup>. In these studies, high BMD was negatively correlated to starting age of high level training, indicating that the younger the athletes started training (pre- vs. post-pubescent), the higher the dominant arm BMD. This has been equated to a 22% higher humeral BMD in

the dominant arm (pre-pubescent starting age) compared to only 9% higher humeral BMD in individuals starting the sport at a post-pubescent age <sup>107</sup>.

It is therefore interesting to investigate BMD, FM and FFSTM in elite hemiplegic athletes with CP, who compete in Paralympic level athletics. As discussed above, there is evidence of lower BMD and FFSTM in individuals with CP, which is correlated in part to lower levels of ambulation. Furthermore, high levels of BMD have been associated with increased weight bearing load involved in continued exercise participation in AB individuals. Therefore, examining a group of elite Paralympic athletes with CP who have undergone high levels of intensive athletic training over many years may further the understanding of the complex interaction between weight bearing activity, exercise and body composition in individuals with CP.

The present study aimed to describe BMD, FM and FFSTM in six elite male Paralympic athletes with hemiplegic CP.

## **METHODS**

### **Participants**

Six male elite Paralympic athletes with CP were recruited for the study (CP group, five participants with hypertonic CP, one participant with athetoid CP). Ethnic distribution of the CP group included four white ancestry individuals, one mixed ancestry individual and one black ancestry individual. All participants were track and field sprinters with hemiplegic CP, as diagnosed by a physician. They all competed in track athletics at National and International Paralympic level. Two of the participants were classified as T38 and four as T37 athletes, in accordance with the criteria established by the Cerebral Palsy International

Sport and Recreation Association (CPISRA) and International Paralympic Committee (IPC) for Paralympic competition <sup>11, 12, 197</sup>. T38 athletes are distinguished by limitations in running activities in any limb distribution, as a result of hypertonia, ataxia or athetosis while T37 athletes present with ambulant hypertonic hemiplegic profiles, presenting with distinctive unilateral impairment on the affected (A) side, with a fully-functional non-affected (NA) side <sup>11, 12, 197</sup>. Both T38 and T37 athletes fall into levels I to II on the Gross Motor Function Classification System (GMFCS) <sup>108</sup>. Refer to Table 2 for details.

Each participant provided written informed consent prior to the study. The study was approved by the University's Human Research Ethics Committee (Ref: 156/2011).

### **Dual Energy X-Ray Absorptiometry body scan**

Participants' height in meters (m) and weight in kilograms (kg) was recorded on a calibrated scale (Seca, Model 708, Germany) and body mass index calculated as kilograms per meters (BMI, kg/m<sup>2</sup>) was calculated. Both height and weight were measured while the participant was wearing minimal clothing, and without shoes. BMD as well as FM and FFSTM, were measured using DXA total body scans (Hologic Discovery-W, software version 13.4.1, Hologic Bedford Inc., Bedford MA, USA).

### *Bone mineral density*

Whole body (WB), lumbar spine (LS), femoral neck (FN) and total hip (TH) BMD was measured using DXA and expressed as grams per centimeter (g/cm<sup>2</sup>). BMD Z scores, which represent a participants' comparison to age and gender matched individuals in the DXA reference database of AB controls, were calculated by the DXA software and expressed as SD difference from the control database individuals. One measurement was

given for WB and LS, while bilateral measurements were given for FN and TH for A and NA sides.

#### *Fat mass and fat free soft tissue mass*

FM and FFSTM were measured for individual upper limb (arm) and lower limb (leg) as well as unilateral appendicular limb distributions and expressed as kilograms (kg). Measurements were given for A and NA sides in the above groupings of limbs.

#### **Statistics**

All data were analysed using statistical software (Statistica 12, Statsoft Inc. Tulsa, Oklahoma, USA). As this was a descriptive investigation of one group of athletes (N = 6), statistical analyses were not performed on whole-body total values (height, weight, BMI, WB BMD, LS BMD, WB BMD Z score, LS BMD Z score). Independent t-tests were performed to compare differences in FN BMD, TH BMD, FN BMD Z score, TH BMD Z score, upper limb FM, lower limb FM, appendicular limb FM, upper limb FFSTM, lower limb FFSTM and appendicular limb FFSTM between A and NA sides. Significance was accepted at  $P < 0.05$ .

## **RESULTS**

### **Participant characteristics**

Table 2 presents descriptive characteristics of six athletes with CP with regard to class and level of competition, diagnosis, resting spasticity level, hemiplegic involved side and current 100 meter (m) performance time in seconds (s) as a general overview of the current sample.

**Table 2:** International Paralympic Committee competition classification, level of competition, diagnosis, resting Ashworth Scale spasticity level, involved side and current 100m sprint time of six participants with cerebral palsy

Participant	IPC class	Level of competition	Diagnosis	Resting spasticity	Involved side	100 m time (s)
1	T 37	Paralympic	Hypertonic hemiplegia	1 – 2	Right	11.54
2	T 38	Paralympic	Hypertonic hemiplegia	1 – 2	Left	11.29
3	T 37	Paralympic	Hypertonic hemiplegia	1 – 2	Right	12.21
4	T 37	National	Hypertonic hemiplegia	1 – 2	Right	12.49
5	T 37	Paralympic	Hypertonic hemiplegia	1 – 2	Right	12.6
6	T 38	Paralympic	Athetoid hemiplegia	-	Left	11.21
<b>Mean (SD)</b>	-		-		-	<b>11.9 (0.9)</b>

### Age and body composition

Table 3 presents data for body composition, BMD, BMD Z score, FM and FFSTM. Mean age for the participants was  $23.3 \pm 3.8$  years. Height and weight were  $1.76 \pm 0.03$  m and  $68.59 \pm 2.73$  kg. BMI was  $22.14 \pm 1.40$  kg/m<sup>2</sup> in this group of athletes with CP.

**Table 3:** Descriptive data of body composition, bone mineral density (BMD), bone mineral density age matched Z scores (BMD Z score), fat mass (FM) and fat-free soft tissue mass (FFSTM) of six participants with cerebral palsy. Total scores are provided for variables as whole-body measurements, and unilateral values are given for affected (A) and non-affected (NA) sides. \*  $P < 0.05$  side main effect.

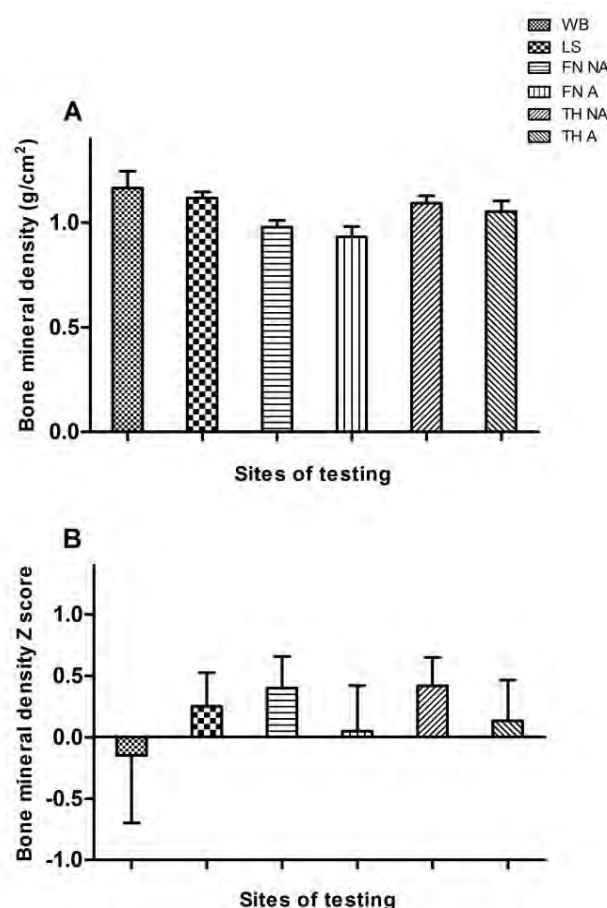
	Total	A	NA
<b>Age (years)</b>	23.3 ± 3.8		
<b>Body Composition</b>			
Height (m)	1.76 ± 0.03		
Weight (kg)	68.59 ± 2.73		
Body Mass Index (kg/m <sup>2</sup> )	22.14 ± 1.40		
<b>Bone Mineral Density</b>			
Whole body BMD (g/cm <sup>2</sup> )	1.164 ± 0.081		
Lumbar spine BMD (g/cm <sup>2</sup> )	1.117 ± 0.072		
Femoral neck BMD (g/cm <sup>2</sup> )		0.932 ± 0.124	0.979 ± 0.080
Total hip BMD (g/cm <sup>2</sup> )		1.053 ± 0.124	1.092 ± 0.087
Whole body BMD Z score	-0.150 ± 0.547		
Lumbar spine BMD Z score	0.250 ± 0.672		
Femoral neck BMD Z score		0.050 ± 0.909	0.400 ± 0.626
Total hip BMD Z score		0.133 ± 0.809	0.417 ± 0.567
<b>Fat Mass</b>			
Upper limb FM (kg)		0.46 ± 0.14	0.42 ± 0.11
Lower limb FM (kg)		1.58 ± 0.51	1.46 ± 0.46
Appendicular FM (kg)		2.04 ± 0.65	1.89 ± 0.56

**Fat-free soft tissue mass**

Upper limb FFSTM (kg)	2.96 ± 0.44	*	3.60 ± 0.39
Lower limb FFSTM (kg)	8.81 ± 0.27	*	10.34 ± 0.82
Appendicular FFSTM (kg)	11.77 ± 0.64	*	13.94 ± 1.18

**Bone mineral density and bone mineral density Z scores**

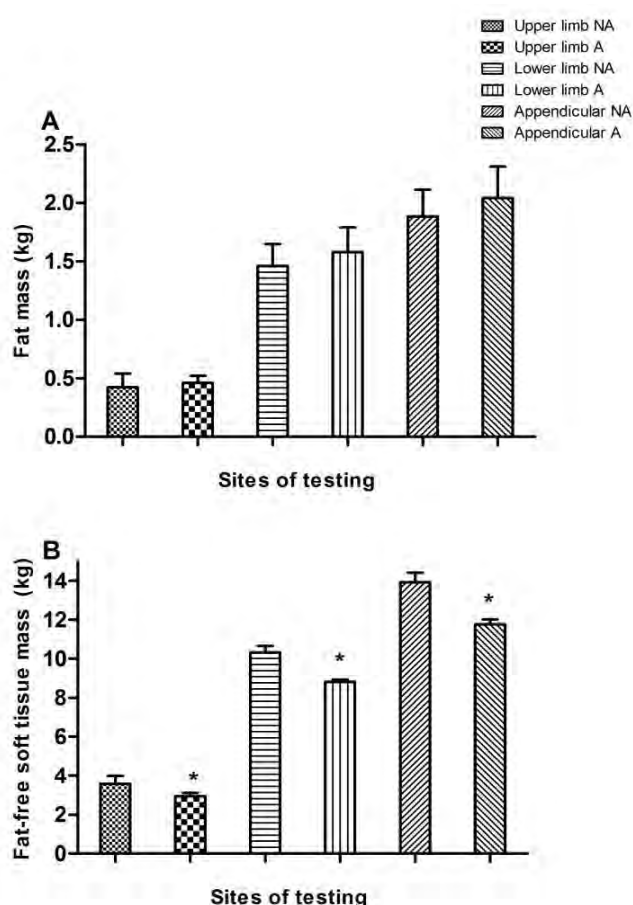
There were no differences between FN and TH BMD or BMD Z scores between A and NA sides (Table 3, Figure 1).

**Figure 1: Bone mineral density**

Bone mineral density (Figure 1 A) and bone mineral density age matched Z scores (Figure 1 B) for specific locations in the body, including whole body (WB), lumbar spine (LS), femoral neck non-affected side (FN NA), femoral neck affected side (FN A), total hip non-affected side (TH NA) and total hip affected side (TH A).

### Fat mass and fat-free soft tissue mass

Upper limb FFSTM was significantly lower in the A side compared to the NA side (Table 3, Figure 2 B,  $2.96 \pm 0.44$  kg vs.  $3.60 \pm 0.39$  kg,  $P < 0.05$ ). Lower limb FFSTM was significantly lower on the A side ( $8.81 \pm 0.27$  kg for A and  $10.34 \pm 0.82$  kg for NA,  $P < 0.01$ ). Appendicular FFSTM was also significantly lower on the A side compared to the NA side ( $11.77 \pm 0.64$  kg vs.  $13.94 \pm 1.18$  kg,  $P < 0.01$ ). There were no differences between A and NA FM in any limb distribution (Table 3, Figure 2 A).



**Figure 2: Fat mass and lean mass**

Fat mass (FM, Figure 2 A) and fat-free soft tissue mass (FFSTM, Figure 2 B) for specific locations in the body, including the upper limb non-affected side (upper limb NA), upper limb affected side (upper limb A), lower limb non-affected side (lower limb NA), lower limb affected side (lower limb A) and appendicular upper and lower limb non-affected and affected sides (appendicular NA, appendicular A), for unilateral FM and FFSTM distributions. \*  $P < 0.05$  side main effect.

## DISCUSSION

The present chapter was the first to describe BMD and body composition in elite Paralympic athletes with CP. BMD, FM and FFSTM were measured at whole body sites, as well as at unilateral sites on the A and NA sides.

The first important finding of this study was the similarity in FN and TH BMD and BMD Z scores between A and NA sides in this group of elite Paralympic athletes with CP (Table 3, Figure 1). There was no difference in absolute BMD between the A and NA sides (Figure 1 A), and although there was a tendency toward differences in BMD Z scores between the A and NA sides, they did not reach significance (Figure 1 B). These findings showed that the BMD of this group of athletes was closely matched to that of AB individuals in the normative database. Of interest and although not significantly different, BMD Z scores of the LS, FN and TH were above that of the age and gender matched AB individuals in the control database. WB BMD was the only testing site where a lower than average Z score was observed.

The second important finding was the difference observed in FFSTM between A and NA sides, despite similarities in FM between the sides (Table 3, Figure 2). These findings were similar to those reported in the literature, described in the introduction of this chapter<sup>99</sup>.

Previous research has demonstrated that individuals with CP show significant reductions in functional capacity and exercise performance compared to AB individuals. These deficits have been documented as a 30 - 73%, 15 - 42% and 27 - 46% reduction in strength, aerobic and anaerobic capacity, respectively<sup>17-19, 30, 31, 31, 32, 48-50, 109, 110</sup>, which emulates the 1.8 – 3.2 SD lower BMD Z scores observed in similar samples of individuals with CP<sup>99</sup>.

However, as discussed in Chapter 1 of this thesis, the previous literature on individuals with CP was conducted using sedentary children. There has been very little research conducted on adults with CP, and even less on adults with CP who have participated in regular physical activity for many years. Thus, sedentary behaviour has been a confounding factor in the research conducted on the physical capacity of individuals with CP, also possibly obscuring the true effects of CP on the body.

As such, the findings of similar BMD, BMD Z scores and FM in the present study represent the first descriptions of bone and body composition of elite Paralympic athletes with CP. The finding of similar BMD and BMD Z scores contradict previous research which proposed that sedentary children with CP present with large differences in bone physiology and functional capacity compared to AB controls. Furthermore, the greater than average BMD Z scores in the FN and TH, despite lower BMD in the rest of the body, indicates that athletes with CP have higher BMD in load bearing sites of the body which have a greater proportion of cortical bone compared to other non-loadbearing areas of the body. As these findings emulate research conducted on BMD and load bearing in athletic individuals, they may provide the first insight into the effect of high levels of exercise training on physiology in this group of athletes.

Similarly, the finding of comparable FM between A and NA sides provides insight into the effects of long term athletic training on health parameters in individuals with CP. However, the finding that FFSTM was lower on the A side in this group of athletes, despite similar FM between sides, and which was in accordance with previous literature, provided evidence that there may be an upper limit of bone adaptation in individuals with CP with regard to muscle physiology. That is, although bone density and FM may be highly adaptable in individuals

with CP, the central inhibition of muscle recruitment and neuromuscular deficits described in Chapter 1 may still have a large effect on muscle size, function and physiology<sup>17, 82, 111</sup>. This would result in the lower FFSTM on the A side observed in this chapter.

The present study provided novel insight into the BMD, FM and FFSTM of elite Paralympic athletes with CP. It is acknowledged that limitations of the research include the small sample size, differences in ethnic selection of the sample, lack of a control group and lack of longitudinal tracking of BMD. This study was conducted in a finite group of elite athletes, where no more than six athletes were available to be tested, which also applies to the combination of four white ancestry participants, one mixed ancestry participant and one black ancestry participant in the sample. However, all participants included in the study were male, and comparisons were made between each individual's A and NA sides, making differences in ethnicity less important to the conclusions drawn from the data. Another limitation of this study was the lack of an AB control group. Further research is required using a longitudinal, case-controlled study to determine the effect of exercise on bone development and body composition in a larger sample of elite athletes with CP.

## **CONCLUSION**

The present chapter offers the first novel insights into in-depth body composition of elite Paralympic athletes with CP. Similar BMD and FM between A and NA sides indicates that high levels of athletic training over many years may have positive physiological consequences, including adaptation of these variables. However, asymmetries in FFSTM indicated that there may be a limit to the adaptation observed in these athletes. This study aimed to describe anthropometry of elite Paralympic athletes with CP. Further studies investigating in-depth exercise performance and functional characteristics in a similar

sample of athletes with CP are required to further describe elite Paralympic athletes with CP, which will be the focus of work described in the next three chapters of this thesis.

## **CHAPTER 3**

# **A description of sprint cycling performance and neuromuscular characteristics in elite Paralympic athletes with cerebral palsy**

The findings of the present chapter have been published as an original research article in the

*American Journal of Physical Medicine and Rehabilitation*, 94: 28-37 (2015)

## INTRODUCTION

As discussed in detail in Chapter 1 of this thesis, several studies using paediatric participants with cerebral palsy (CP) have described marked impairments in exercise performance in this population. Isometric strength<sup>17</sup> and sprint running speed<sup>48</sup> have been found to be 39% to 56% lower in individuals with CP than able-bodied (AB) controls. Deficits ranging between two and four standard deviations have been found in anaerobic performance of children with CP when compared to performance of age and activity matched individuals without CP using the Wingate sprint cycle test<sup>49, 112</sup>. Furthermore, Verschuren et al. (2010) found that aerobic endurance performance ( $VO_{2peak}$ ) was 15% lower in individuals with CP, when using a progressive maximal treadmill test<sup>29</sup>.

There is evidence that regular physical training has functional benefits in children with CP. Exercise intervention studies using sedentary, paediatric samples have found dramatic improvements in strength, aerobic capacity and anaerobic capacity in both diplegic and hemiplegic groups, with as little as six weeks of training<sup>19, 51, 65</sup>. Elite athletes with and without disabilities train at very high volumes for many years in order to compete at Olympic or Paralympic level. As no exercise performance research exists on this athletic calibre of individual with CP, it is intriguing to consider whether elite training status in individuals with CP alters factors that contribute to performance, compared to AB individuals. These factors include fatigue profiles, peak power output capacity and neuromuscular activation. A comparison between athletes with CP and AB athletes would improve understanding of the true effects of CP, whilst eliminating the effects of sedentary behaviour on the results.

Therefore, the aim of this study was to describe sprint cycle performance and neuromuscular characteristics of elite Paralympic athletes with CP during a maximal cycling trial, compared

to well-trained, running sprint performance matched AB athletes. The athletes with CP were internationally competitive track and field athletes, who have trained for between six and 20 hours per week over many years. Power output, muscle activation and fatigue characteristics, as well as the presence of neuromuscular irregularities were investigated between the groups. It was hypothesised that the athletes with CP would perform the Wingate sprint cycle test at a significantly lower power output than the AB athletes, and would show a flatter fatigue profile in power output over the course of the test. It was further hypothesized that the lower power output would be associated with a significantly lower level of muscle activation, as measured by electromyography (EMG). This would be in keeping with the published literature in untrained and paediatric individuals with CP.

## **METHODS**

### **Participants**

Five male elite athletes with hypertonic hemiplegic CP (CP group) and 16 male well trained AB controls (AB group) were recruited for the study. The participants in the CP group were all track sprinters with hemiplegic CP, as diagnosed by a physician. They all competed at International Paralympic level. Three of the participants were classified as T38 and two as T37 athletes, in accordance with the criteria established by the CPISRA and IPC for Paralympic competition<sup>11, 12, 197</sup>. T38 athletes are distinguished by limitations in running activities in any limb distribution, as a result of hypertonia, ataxia or athetosis. T37 athletes are classified as having ambulant hypertonic hemiplegic profiles and present with distinctive hemiplegic limitations, with a fully-functional non-affected side. Both T38 and T37 athletes present with grade 1 to 2 spasticity on the Modified Ashworth Scale, which indicates a mild to moderate level of resting spasticity in the affected limbs<sup>12</sup>. Both T38 and T37 athletes fall into levels I to II on the Gross Motor Function Classification System (GMFCS)<sup>108</sup>. In terms

of athletic performance, T38 and T37 athletes are approximately 14% and 22% slower compared to AB 100m and 200m world record sprint performances (unpublished observations).

AB participants were selected from rugby, cricket and hockey teams, and were additionally selected only if they played in positions where sprint ability was an important attribute for performance. They competed from club to International level.

All participants were unaccustomed to leg cycling exercise, as their main form of exercise was running based sprint exercise. Cycling was used as a modality as the Wingate sprint cycle test is a validated method for assessing sprint performance, and allows the capture of data at a high sampling rate. The use of the Wingate sprint cycle test minimized the effect of experience of cycling on performance in either group.

### **General Overview of Testing**

Participants reported to the laboratory on one occasion. Their height in meters (m) and body mass in kilograms (kg) were measured on calibrated equipment (Seca, Model 708, Germany). Body mass index (BMI) was calculated as kilograms per meter ( $\text{kg}/\text{m}^2$ ). Participants performed a 10 second sprint test, adapted from the standard 30 second Wingate sprint cycle test. Following this, participants rested for 15 minutes, and then performed a traditional 30 second Wingate sprint cycle test (WIN30). The 10 second sprint was used to ensure a maximal effort in WIN30 through a comparison of the first 10 seconds power output of both tests. It was also used for normalisation of the EMG signal recorded during WIN30. Bilateral EMG of five muscles was measured during both tests. The specific details of these measurements are described subsequently.

Each participant provided written informed consent prior to the study. The study was approved by the University's Human Research Ethics Committee (Ref: 156/2011).

### **Wingate Anaerobic Sprint Cycle Test**

The WIN30 was performed using an electromagnetically braked cycle ergometer (Velotron Dynafit Pro, Racermate Inc., Seattle, WA, USA). This test has been validated in many populations, including individuals with CP and has an Interclass Correlation Coefficient of 0.95 in trained and non-trained individuals with CP <sup>113, 114</sup>. A standardised warm up comprised cycling at a constant workload of 1.5 watts per kilogram (W/kg) at a self-selected cadence for five minutes, followed by a two-minute rest period before commencement of the 10 second sprint. Upon completion of the sprint a break of 15 minutes, WIN30 was performed. For standardisation in both the sprint and WIN30, the participants were given 20 seconds to reach 100 revolutions per minute (RPM) and then were instructed to maintain 100 RPM for 10 seconds, after which time a load of 0.075 kilogram/kilogram body mass (kg/kg BM) body mass was applied, commencing the test. Participants were instructed to pedal as hard and as fast as they could against the resistance for the set period of time (10 or 30 seconds). They were instructed to remain in a seated position throughout the test. Power output was expressed relative to body mass (W/kg).

### *Fatigue Index*

The fatigue index (FI) was calculated as the highest power output (W/kg) achieved during the test minus the power output (W/kg) achieved in the final second of the test. This was divided by the peak power. The FI was expressed as a percentage (%).

## Electromyography

### *Amplitude*

Muscle activity (EMG) of five muscles, namely erector spinae (ES), gluteus medius (GM), biceps femoris (BF), vastus lateralis (VLO) and gastrocnemius (GC), was recorded in both legs using a telemetric EMG system (Telemyo GT2400 G2, Noraxon, USA Inc, Arizona, USA), to allow comparison of affected (A) and non-affected (NA) sides in the participants with CP. In the CP group, A and NA sides corresponded to their own non-dominant (ND) and dominant (D) sides. In the AB group, the ND and D sides were similarly compared to, and expressed as, the CP group's A and NA sides. Dominance was self-reported. Prior to the placement of the electrodes, the area was shaved and cleaned with ethanol. Two electrodes with a 20 millimeter inter-electrode distance (Blue Sensor, Medicotest, Denmark) were placed over the muscle belly whilst a reference electrode was placed on the anterior superior iliac spine of the right leg. Placement was in accordance with Surface Electromyography for the Non-Invasive Assessment of Muscle (SENIAM ) recommendations<sup>115</sup>. As with previous research using EMG during cycling, each test was divided into five second epochs for processing and analysis<sup>116-118</sup>. For analysis of the EMG signal, the raw EMG signals were band passed filtered between 20 and 500 Hertz (Hz). All signals were rectified and smoothed using root mean square analysis for a 50 millisecond time window.

The EMG in WIN30 was normalised to the highest mean amplitude five second period of the 10 second sprint. The use of a 10 second sprint for EMG normalisation establishes a constant baseline of EMG activity to which all subsequent activity is compared and expressed as a percentage. This has been shown to be a valid method of normalization for dynamic exercise<sup>119</sup>. To ensure reliable EMG data reporting, EMG was measured in a rested state as well as during manual isolation of every muscle. This ensured that the signals recorded were not artefacts of incorrect placement, movement or noise generated by

external variables. Mean normalised amplitude of the five second periods as well as individual root-mean-squared EMG signals to identify irregularities were analysed.

### *Frequency*

Median frequency spectral analysis of the first and last five second periods of WIN30 was analysed using a Fast Fourier Transform algorithm. EMG frequency is the rate at which action potentials are sent to the working muscles from the central nervous system. Higher median frequencies are a mechanism of muscular force summation (higher force), while a change in frequency indicates a change in the type of muscle fibers activated, and ultimately fatigue<sup>120, 121</sup>. The analysis was performed to compare frequency between non-fatigued states (PRE, in the first five seconds of the test) and fatigued states (POST, in the last five seconds of the test). The frequency analyses were between 5 - 500 Hz, as EMG signal outside this range contains mostly noise and unusable signal<sup>122</sup>. As fatigue was of interest, the change from PRE to POST is reported.

### **Statistical Analysis**

Power output and EMG were averaged over one and five second periods, respectively. All data were analysed using statistical software (Statistica 10, Statsoft Inc. Tulsa, Oklahoma, USA). Significance was accepted at a P value <0.05. Intragroup comparisons between A and NA sides, as well as change over time between the groups were performed using a three way repeated measures analyses of variance ANOVA (Time x Side x Group interaction) for all data sets. Descriptive data of the two groups were compared using independent t-tests.

## RESULTS

### Participants

Table 4 shows descriptive characteristics of the CP, which comprised five elite Paralympic athletes with CP. The groups were matched for age ( $21.6 \pm 4.2$  and  $23.4 \pm 3.0$  years, CP and AB, respectively) and current 100 meter running sprint performance ( $12.2 \pm 0.9$  and  $12.3 \pm 1.1$  seconds, for CP and AB, respectively). However, the AB participants were significantly heavier than the CP group (BMI of  $25.9 \pm 2.4$  kg/m<sup>2</sup> vs.  $21.1 \pm 1.4$  kg/m<sup>2</sup>).

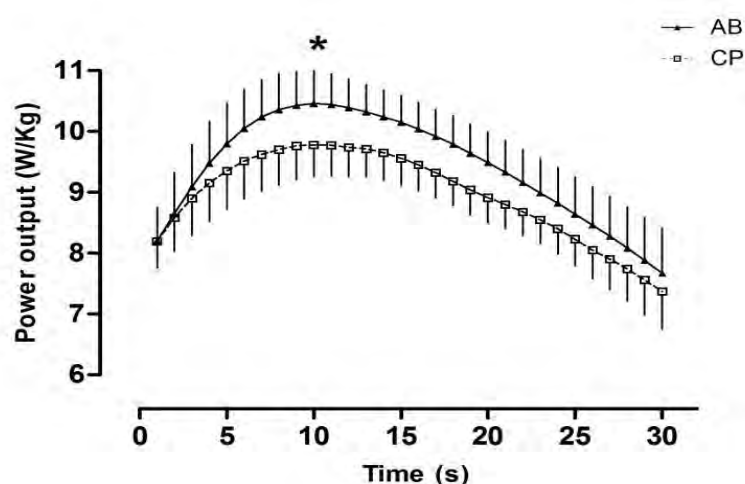
**Table 4:** International Paralympic Committee competition classification, level of competition, diagnosis, resting Ashworth Scale spasticity level, involved side and current 100m sprint time in seconds of six participants with cerebral palsy

Participant	IPC class	Level of competition	Diagnosis	Resting spasticity	Involved side	100m time (s)
1	T 37	Paralympic	Hypertonic hemiplegia	1 – 2	Right	12.3
2	T 37	Paralympic	Hypertonic hemiplegia	1 – 2	Left	11.2
3	T 38	National	Hypertonic hemiplegia	1 – 2	Right	13.5
4	T 38	Paralympic	Hypertonic hemiplegia	1 – 2	Right	11.9
5	T 38	Paralympic	Hypertonic hemiplegia	1 – 2	Left	12.2
<b>Mean (SD)</b>						<b>12.2 (0.9)</b>

### Wingate Anaerobic Sprint Cycle Test

Peak power output during WIN30 was  $10.4 \pm 0.5$  and  $9.8 \pm 0.5$  W/kg for AB and CP groups respectively ( $P < 0.05$ ), and occurred at 10 seconds in both groups (Figure 3). Power output was significantly higher in AB throughout the trial ( $P < 0.05$ ).

Power output declined similarly between groups, with the FI of the AB and CP groups calculated as  $27 \pm 6.9$  % and  $25 \pm 7.3$  %, respectively.



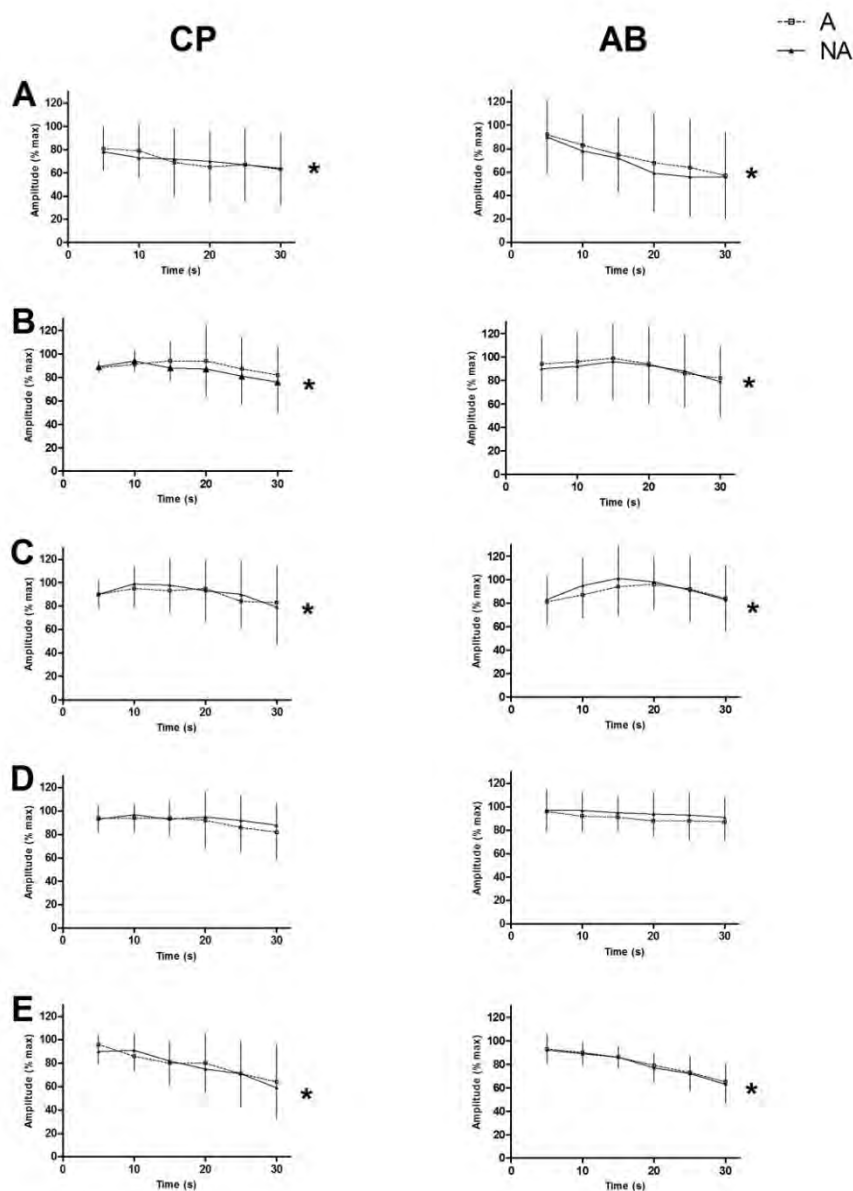
**Figure 3:** Power output (W/kg) of 30 second Wingate sprint cycle test over 30 seconds (s) for the able-bodied group (solid triangle) and cerebral palsy group (open square), with peak in both groups at 10 seconds. \*  $P < 0.05$ , time and group effect.

### Electromyography

#### Amplitude

Figure 4 A - E shows normalized EMG activity as percentage maximum activation (% max) on the A and NA sides of the five measured muscles in the CP group (left panels) and AB group (right panels). There were no differences in mean muscle activity over time between A and NA sides, nor between AB and CP groups. In four muscle groups (ES, Figure 4 A; Glut, Figure 4 B; BF, Figure 4 C; GC, Figure 4 E), EMG amplitude decreased significantly

over the trial in both limbs, and in both CP and AB groups ( $P < 0.001$ ). In both the CP and AB groups, VLO activity remained unchanged over time (Figure 4 D).



**Figure 4:** Normalised muscle activation over a 30 second Wingate sprint cycle test for affected (A) and non-affected (NA) sides of the cerebral palsy group (left panel) and correlating non-dominant and dominant sides of the able-bodied group (right panel) for erector spinae (Figure 4 A), gluteus medius (Figure 4 B), biceps femoris (Figure 4 C), vastus lateralis (Figure 4 D) and gastrocnemius (Figure 4 E). \*  $P < 0.05$  time effect.

### Frequency

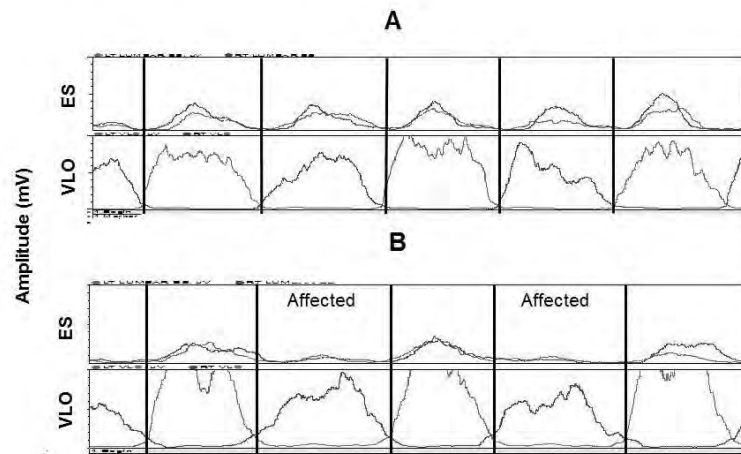
Median frequency decreased significantly in both AB and CP groups, on both the A and NA sides (Table 5,  $P < 0.05$ ). Median frequency decreased significantly more from PRE to POST in VLO on the A side of the CP group compared to the AB group ( $P < 0.05$ ).

**Table 5:** Percentage decrease in median frequency from pre-fatigued (PRE) to post-fatigued (POST) states for affected (A) and non-affected (NA) sides of muscle activation in the cerebral palsy group (left column) and correlating non-dominant (ND) and dominant (D) sides in the able-bodied group (right column). \* $P < 0.05$  time effect; # $P < 0.05$ , side and time and group effect

Muscle	CP		AB	
	A	NA	ND	D
Erector spinae	17.16 % *	10.08 % *	20.99 % *	7.03 % *
Gluteus medius	15.10 % *	11.42 % *	3.08 % *	5.17 % *
Biceps femoris	20.24 % *	15.68 % *	8.54 % *	7.41 % *
Vastus lateralis	13.35 % #	12.68 % *	8.48 % *	13.10 % *
Gastrocnemius	6.91 % *	10.48 % *	14.67 % *	7.45 % *

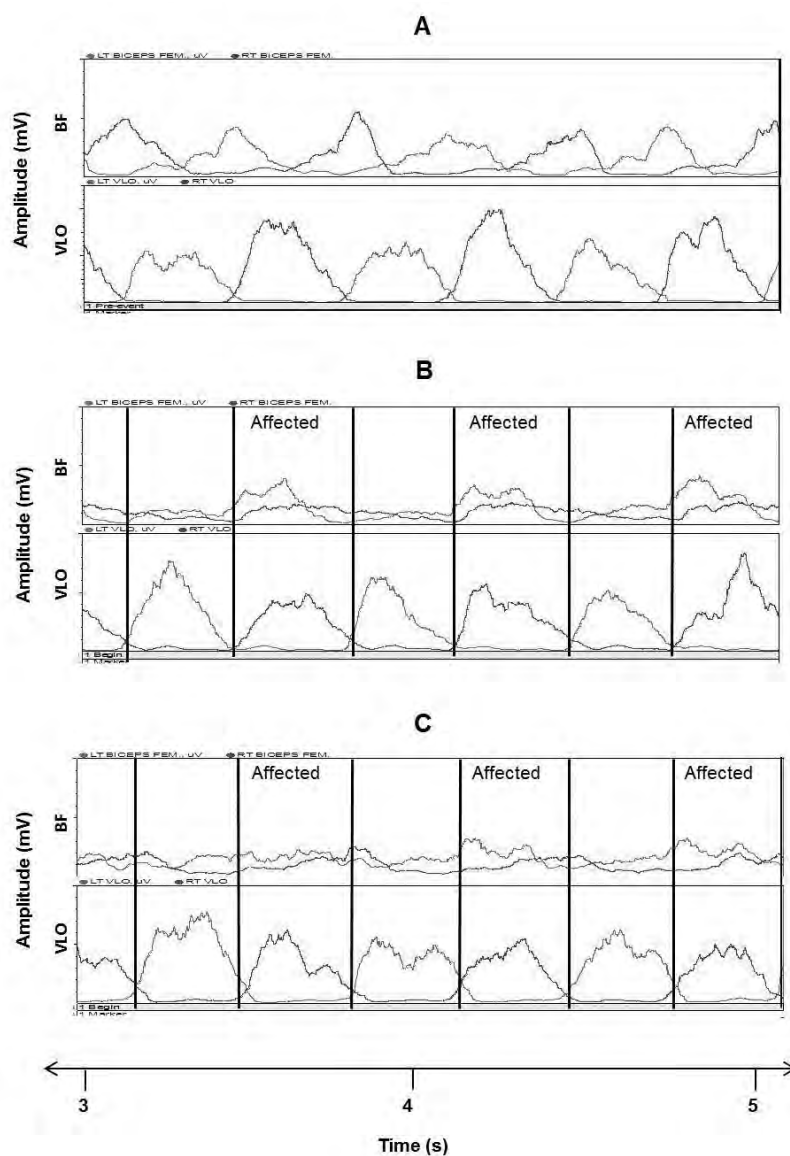
### Individual EMG Trace Patterns

Figure 5 A shows bilateral co-activation of the stabilising ES muscles on each pedal stroke in an AB participant during WIN30. The pedal stroke is identified by VLO activation in the bottom trace of the panel, with the top trace depicting ES activation. Figure 5 B depicts an atypical firing pattern of the ES observed in a participant with CP. ES activity occurs during the pedal stroke on the NA pedal stroke only, with no activity during the A side's pedal stroke.



**Figure 5:** Muscle activation irregularities in stabilising muscles in cerebral palsy. Typical firing pattern of the erector spinae in an able-bodied athlete (Figure 5 A) with a lack of required co-activation of the erector spinae is observed in a participant with cerebral palsy (Figure 5 B).

Figure 6 A shows a typical firing pattern of two power producing muscles (BF and VLO) in an AB participant, which reciprocally activate per pedal stroke. Figure 6 B shows the same muscle traces of a participant with CP. BF activation on both sides increases when the A side pedal stroke occurs. On the NA side, there is significantly less BF activation. Figure 6 C displays continuous activation of BF muscles of both the A and NA sides, with typical firing patterns of the VLO muscle.



**Figure 6:** Muscle activation irregularities in power-producing muscles in cerebral palsy. Typical firing pattern of vastus lateralis (VLO) and biceps femoris (BF) in an able-bodied athlete (Figure 6 A) in the first five second epoch of 30 second Wingate sprint cycle test (non-fatigued state). Co-activation of the BF and VLO (Figure 6 B) and atypical motor drive of the BF (Figure 6 C) observed in two participants with cerebral palsy.

## DISCUSSION

The present study compared elite Paralympic athletes with CP to age and 100m sprint performance matched AB athletes, in a fatiguing maximal cycling task. Based on previous literature, it was initially hypothesised that individuals with CP would present with significantly lower power outputs and muscle activation levels, as well as display less attenuation of these parameters with fatigue during a maximal sprint trial. It was found in the present study that the AB athletes produce higher power output throughout WIN30 compared to the CP group (Figure 3), but that all other aspects of performance, including FI and muscle activation levels, were similar between groups. These findings provide the first exercise performance based evidence showing that athletes with CP may have overcome previously documented deficits in performance and neuromuscular activation.

FI was similar between groups, demonstrating a similar decline in power output from peak value, measured at similar time points in CP and AB (Figure 3). Associated mean EMG changed similarly over the trial in all measured muscle groups (Figure 4). Furthermore, changes in EMG over the trial were similar between A and NA sides in the CP group and corresponding ND and D sides in the AB group.

The existing literature suggests that individuals with similar forms of CP to the current sample (GMFCS levels I and II) exhibit lower levels of neuromuscular fatigue as a result of sub-optimal central drive, and the consequent inability to produce enough isokinetic torque to create fatigue<sup>17, 81, 123</sup>. This muscle weakness has been attributed to multiple factors including spasticity, co-activation, Type I muscle fibre predominance and intramuscular weakness<sup>124-127</sup>. The findings of the current study suggest that despite a relative weakness in the CP group, demonstrated by lower power outputs in the Wingate sprint cycle test (10.4

$\pm 0.5$  W/kg and  $9.8 \pm 0.5$  W/kg for AB and CP respectively,  $P < 0.05$ ), the associated lower neuromuscular fatigue shown by literature was not present in this elite athletic sample. This was demonstrated by the similarity in FI and progressive decrease in EMG activity over the trial between AB and CP groups in all five muscles tested ( $P < 0.05$ ).

These results may be the effect of long term, high level athletic training, as first proposed in Chapter 2 of the current thesis. Research to date which show deficits in performance and flattened fatigue profiles in CP, has been performed on sedentary children<sup>128</sup>. However, marked changes in muscular adaptation have been observed with exercise training in these samples, making it clear that there is capacity to significantly improve exercise performance and functional capacity in individuals with CP<sup>19, 51, 65</sup>. Therefore, with the findings of the present chapter, the current sample of elite Paralympic athletes may represent a group of individuals with CP that have reached near-maximal muscular adaptation.

This adaptation may include the possible movement away from Type I muscle fibre predominance and muscular weakness. This explains the similar fatigue profiles seen between groups (FI of  $27 \pm 6.9$  % and  $25 \pm 7.3$  % for AB and CP groups, respectively, Figure 3) and a greater peak power output achieved in the CP group compared to previously published power values for soccer and cycling non elite athletes with CP (9.8 W/kg in this study vs. 8.3 W/kg reported in previous literature)<sup>114</sup>. Frequency analysis of the EMG signal further support that the CP and AB groups fatigued similarly, as frequency shifts were similar between the groups. The observed changes, a shift from higher to lower frequencies, is typical of a shift from Type II muscle fibre activation to Type I fibre activation, due to fatigue of the fast-twitch power-producing fibres<sup>129</sup>. The similarity in frequency decline in both the CP and AB groups may further support the hypotheses of 1) a higher proportion of and 2) a higher utilisation of Type II muscle fibres in this sample of elite athletes with CP<sup>129, 130</sup>. A

significantly greater reduction in frequency occurred in the CP group's A side VLO (the primary power producing muscle), further suggesting the utilization of Type II muscle fibres.

It may be argued that the similarities in physiology observed between the groups may be the result of the current sample of athletes consisting athletes in classes T37 and T38, representing a group of individuals with milder forms of CP. It is believed that this is not the primary origin of the results of the present chapter, as only reference to previous studies conducted using individuals in GMFCS levels I to II was made, which were comparable to the level of impairment in the current sample of athletes with CP. The possibility of this contributing factor, however, cannot be disregarded.

The second important finding was the presence of CP-associated neuromuscular irregularities identified in the athletes with CP during the WIN30, in both stabilising (ES muscles, Figure 5) and power (VLO and BF muscles, Figure 6) muscles, despite the similarities in fatigue between the CP and AB groups. Figure 5 shows co-activation of both ES muscles on the NA side's pedal stroke and complete lack of activation on the A side's pedal stroke of an athlete with CP (Figure 5 B). The existing theory on the role of the stabilising ES muscles during locomotion is that the ES muscles bilaterally co-activate with heel strike in walking / pedal stroke in cycling to counteract unwanted trunk movement<sup>131</sup>,<sup>132</sup>. These muscles also, in conjunction with other lower back muscles, balance external loads to decrease forces on the spine which, when disrupted, leads to injury<sup>133, 134</sup>. Thus the lack of firing on the CP body's A side may impair the stability required for that side's pedal stroke, and predispose this athlete to injury.

Furthermore, neuromuscular irregularities in power muscles (Figure 6) may have an impact on injury incidence, as well as overall performance<sup>54, 79</sup>. Figure 6 A demonstrates typical BF

and VLO activity in an AB participant. VLO and BF are active in a reciprocal manner, ensuring that the application of downward force to the pedal is timed appropriately during the upstroke. Figure 6 B demonstrates co-activation of the agonist (VLO) and antagonist (BF) muscles on the A side, with a lack of activation on the NA side, where it is also required for correct pedal power production. It has been proposed that bi-articular muscles, including the BF, are involved in the control of the direction of force application on the pedal during cycling<sup>135</sup>, therefore co-activation may produce counter-acting torques on each pedal stroke. That is, firing of the BF on the downward portion of the pedal stroke may have exerted opposite forces to the VLO, which would result in both forces being exerted on the pedal simultaneously with a consequent loss of economy. The observed co-activation of this antagonist pair, however, may also be an activation strategy required for increased joint stability on the A side, by equalising articular movement and pressure during force application<sup>136-138</sup>. Furthermore Figure 6 C displays continuous activation of BF muscles of both the A and NA legs despite typical firing patterns of the VLO muscle, indicating an atypical motor drive to that muscle group<sup>76</sup>. The complete lack of coherent firing patterns may lead to a lack of co-ordinated power production for both the A and NA sides, which would result in a reduction of power output. There may also be an increased risk of injury due to compensation by the antagonist muscle groups, which has been shown to be a major risk factor for muscle injuries in runners<sup>139, 140</sup>.

The present study has provided the first description of elite Paralympic athletes with CP compared to AB athletes. It has identified interesting findings in an as yet unstudied sample, but with certain limitations. Stroke-by-stroke bilateral power output on the cycle ergometer was not measured, a measurement that would have proved very useful in identifying neuromuscular differences between sides. The use of M-wave muscle stimulation would have also allowed for absolute amplitude EMG activation comparisons between sides, and is

advised for future studies in this area. Finally, it would have been ideal to be able to conduct fibre typing via biopsy in the athletes, which would support the initial hypothesis, but the expensive and invasive nature of the test needs to be taken into consideration. Future studies are required during running exercise, which would be applicable to the athletes' training preference.

## **CONCLUSION**

The present chapter described performance and neuromuscular characteristics of elite Paralympic athletes with CP during maximal sprint cycling. The similarity in FI and neuromuscular fatigue between the CP and AB groups indicates that the current sample of elite athletes with CP may have a different physiology to previously studied untrained individuals with CP. It was proposed that this movement toward AB levels of physiology may be a result of high level training over many years. This may have both empirical and clinical application, as it may indicate the upper-most ability of the cerebral-palsied body to adapt toward typically-developed levels of physiology. Further research, however, is required to either support or refute the performance and fatigue findings of the current study. Chapter 4 aims to test the findings of the present chapter, through additional exploration into exercise performance and fatigue in athletes with CP.

## **CHAPTER 4**

# **The effects of induced volitional fatigue on sprint and jump performance in elite Paralympic athletes with cerebral palsy**

The findings of the present chapter have been accepted for publication as an original research article in the *American Journal of Physical Medicine and Rehabilitation* (2015)

## INTRODUCTION

Complex regulation of both central and peripheral physiological factors is important for maximal exercise performance. This complex regulation allows the body to perform at peak capacity, while preventing potentially adverse perturbations in homeostasis<sup>80, 141</sup>. Fatigue, or failure to maintain expected force output, in both clinical and athletic populations, has therefore been a topic of interest for many years.

Studies of fatigue in individuals with cerebral palsy (CP) have been made difficult to interpret by differences in methods used to measure fatigue. One method used to measure fatigue is the determination of decline in force output during repeated maximal contractions. Using this method, individuals with CP have been shown to maintain force output for longer than able-bodied (AB) controls<sup>17, 81, 82</sup>, suggesting greater fatigue resistance. Similarly, when the participants in these studies were graded into the various levels of the Gross Motor Function Classification System (GMFCS), it was found that the participants in the higher levels (lower functional capacity) showed a greater fatigue resistance than those in the lower levels (higher functional capacity). It was thus demonstrated that greater fatigue resistance was observed in participants with greater impairment, at least when this definition of fatigue is used<sup>81</sup>.

However, this method of measurement of the rate of decline of force output may be confounded by the inability of individuals with CP to generate high force output at the onset of exercise, which results in a flatter fatigue profile compared to AB individuals. Fatigue resistance may thus be a misleading term when applied to a population with CP, as fatigue profiles are strongly influenced by the ability of an individual to generate high initial

workrates, and subsequently the observed fatigue resistance was a direct result of muscle weakness in these individuals<sup>142, 143</sup>.

It has been postulated that the muscle weakness observed in individuals with CP is due to the effect of primary central damage which negatively influences motor unit recruitment<sup>17</sup>, and induces secondary changes at the level of the skeletal muscle<sup>82</sup>. This would account for the reduced initial force output produced by individuals with CP, since it would be present even in the absence of fatigue. Furthermore, co-activation of agonist and antagonist muscles, and subsequent incoordination of movement, are also factors to be considered in the reduced force output observed in individuals with CP<sup>82</sup>. It is important to note that the abovementioned findings were described during assessment of isokinetic dynamometry, and therefore functional weakness may be more apparent during more complex tasks involving the greater integration of muscle groups, as would occur during running.

It has been shown in Chapter 3 of this thesis that during sprint cycling exercise, elite Paralympic athletes with CP do not display the fatigue resistance observed in studies using young sedentary individuals. Indeed, similar fatigue profiles during a 30 second Wingate sprint cycle test were observed in athletic adult individuals with CP, compared to AB controls, despite a lower power output throughout the test in the CP group. Certain other central irregularities were documented, including co-activation of agonist and antagonist muscles, lack of correct firing patterns in stabilising and power producing muscles and atypical motor drive. It was proposed that the similar time course changes in performance in athletes with CP was the consequence of their ability to produce a comparable initial power output to AB controls (only 10% lower, compared to 30 - 50% deficit observed in sedentary individuals in previous research), which perhaps could be attributed to long term, elite level athletic training. A limitation of Chapter 3 was that cycling exercise was used to evaluate

fatigue in athletes who are predominantly sprint runners. This study therefore aimed to investigate performance, fatigue and neuromuscular activation during maximal explosive exercise in elite Paralympic athletes with CP, compared to well trained, sprint specific AB athletes. Specifically, sprint performance, maximal jump performance and muscle activation were investigated before and after a bout of exercise to exhaustion.

Therefore, based on the findings of Chapter 3, it was hypothesized that the athletes with CP would show reduced performance in the sprint and jump tests, but would display a similar reduction in performance as a result of the prior fatiguing bout of exercise, compared to controls. It was further hypothesized that a similar reduction in performance would be associated with changes in muscle activation, measured by electromyography (EMG).

## **METHODS**

### **Participants**

Eighteen male athletes were recruited for this study, comprising six athletes with hemiplegic CP (CP group) and 12 well trained AB controls (AB group).

The CP group consisted of Paralympic level track sprinters and had trained between 14 and 20 hours per week for between one and 10 years. All athletes presented with hemiplegic CP, as diagnosed by a physician. Two athletes were classified as T38 athletes and four were classified as T37 athletes. T38 athletes are classified as having functional limitations in function in any limb distribution, resulting from hypertonia, ataxia, or athetosis, as designated by the CPISRA and the IPC<sup>11, 12, 197</sup>. In addition, T37 athletes are classified as having ambulant hemiplegic profiles, usually presenting with a hypertonic affected (A) side, and full-functioning non-affected (NA) side<sup>12</sup>. Both T38 and T37 classes present with grade 1 to 2

on the Modified Ashworth Scale, which indicates a mild to moderate level of resting spasticity in the A limbs<sup>144</sup> and were classified as level I of the Gross Motor Function Classification System (GMFCS)<sup>108</sup>.

The AB participants were selected from National and Olympic men's field hockey squad if they played in positions where sprint ability was an important attribute for performance (midfielders, strikers).

### **General Overview of Testing**

Participants reported to the laboratory on two occasions. The first visit was used as a familiarization trial. On the second visit, their height in meters (m) and body mass in kilograms (kg) were measured using calibrated equipment (Seca, Model 708, Germany). After an adequate self-selected warm up, participants performed one 40m Sprint test (40m ST), followed by two Vertical Jump tests off both legs (VJ-B), and two Vertical Jump tests off the (A, VJ-A) and (NA, VJ-NA) legs respectively. The participants were then required to complete a modified Multistage Shuttle Run Test (MSRT) to exhaustion. This was used to induce maximal fatigue. Within a minute of completion of the MSRT, participants repeated the 40m ST, VJ-B, VJ-A and VJ-NA test protocol, to assess changes from a pre-fatigued (PRE) state to a post-fatigued (POST) state. Bilateral muscle activity (EMG) of five muscles was measured during the 40m ST, VJ-B, VJ-A and VJ-NA. The specific details of these methodologies are described subsequently.

Each participant provided written informed consent prior to the study. The study was approved by the University's Human Research Ethics Committee (Ref: 156/2011).

### **40m Sprint Test**

The 40m ST was performed using an indoor track and SmartSpeed timing gates (FusionSport, Grabba International Pty Ltd, Queensland, AUS). The 40m ST is a validated test used to measure running performance over short distances <sup>145</sup>. Starting from a crouched position, participants were instructed to run as fast as possible down a straight 40m track, only decelerating after passing the last timing gate. Total 40m time was recorded in seconds (s).

### **Vertical Jump Test**

#### *Vertical Jump (both legs)*

The Vertical Jump Test, a validated test for the assessment of lower limb muscle power <sup>146</sup>, was performed using a Vertec Vertical Jump Stand (Sports Imports, Hilliard, Ohio, USA). Before testing commenced, participants were required to touch the stand as high as possible with their dominant arm stretching vertically above their head, but with their heels on the ground, eliminating the effect of participant maximum height on final results. Participants were instructed to jump as high as possible off both legs (VJ-B), from a standing position, hitting the highest point on the stand with their dominant hand. No arm swinging was permitted and only one knee bend was permitted. The final result was height jumped in centimeters (cm).

#### *Vertical Jump (affected and non-affected legs)*

Vertical Jump Tests were performed on the affected (VJ-A) and non-affected (VJ-NA) legs individually. Both the A and NA legs in the CP group were matched to non-dominant (ND) and dominant (D) legs in the AB group, respectively. The CP group's A and NA sides also corresponded to their own ND and D sides. Dominance was self-reported. Using the same

equipment as described above, participants were instructed to jump as high as possible off a single leg (either A or NA) after a two step walk in onto the jumping leg.

Two attempts at each jump were taken (VJ-B, VJ-A, VJ-NA), with the highest jump in cm recorded.

### **Multistage Shuttle Run Test**

An adapted MSRT was completed to exhaustion. Based on the standard MSRT, participants ran between two lines 20m apart, to an externally paced audible signal, until they were unable to continue at the pace of the test. The initial running speed corresponded to shuttle 82 (level 10) on the standard validated MSRT, as sprint endurance was the main outcome of the test<sup>147</sup>. Since the ability to stop suddenly and change direction is limited in athletes with CP, the standard test was adapted. Thus, participants ran one shuttle, followed by a short rest period (corresponding to the duration of the following shuttle), before resuming running. Ratings of perceived exertion (RPE, scale units) and heart rate in beats per minute (HR, bpm) were recorded at the end of every level reached. Total number of shuttles was recorded at termination of the test as a measure of volitional fatigue between the groups.

### **Electromyography**

#### *Amplitude*

EMG activity of five muscles, namely erector spinae (ES), gluteus medius (GM), biceps femoris (BF), vastus lateralis (VLO) and gastrocnemius (GC) was recorded from both legs using a telemetric EMG system (GT2400 G2, Noraxon, USA Inc, Arizona, USA). This allowed comparison between A and NA sides in the participants with CP (which

corresponded to their own ND and D sides, respectively). In the AB group, the ND and D legs were similarly compared.

Prior to the placement of the electrodes, the area was shaved and cleaned with ethanol. Two electrodes with 20 millimeter inter-electrode distance (Blue Sensor, Medicotest, Denmark) were placed over the muscle belly whilst a reference electrode was placed on the anterior superior iliac spine of the right leg. Placement was in accordance with Surface Electromyography for the Non-Invasive Assessment of Muscle (SENIAM) recommendations<sup>115</sup>, and the electrodes were not removed at any stage of the testing protocol. To ensure reliable EMG data reporting, EMG was measured in a rested state as well as during isolated contraction of every muscle. This ensured that the signals recorded were not artefacts of incorrect placement, movement or noise generated by external variables. For analysis of the EMG signal, the raw EMG signals were band passed filtered between 20 and 500 Hertz (Hz). All signals were rectified and smoothed using root mean square analysis for a 50 millisecond window<sup>148</sup>.

For the 40m ST, EMG was analysed as mean muscle activity per second. The sampling period began after four strides, and was measured for a period of three seconds thereafter. For the VJ-B, VJ-A and VJ-NA, the sampling period began at the visible onset of muscle activation of the jumping leg's VLO muscle, and was measured in all muscles for the entire period of that VLO's muscle activation. All EMG in the 40m ST, VJ-B, VJ-A and VJ-NA was normalised to maximum muscle activation achieved the first three seconds of the first 40m ST. This was performed as the first three seconds represented the highest measured muscle activation. EMG was expressed as percentage maximum activation (% max).

EMG frequency was not reported in this chapter, as frequency is not a reliable measure of fatigue during dynamic exercise, and is used mainly in static or cycling exercise.

### **Statistical Analysis**

All data were analysed using statistical software (Statistica 12, Statsoft Inc. Tulsa, Oklahoma, USA). Significance was accepted at a P value < 0.05. Comparisons of sprint performance and jump height between A and NA sides, as well as fatigue-induced changes between groups and sides were performed using a three way repeated measures analyses of variance ANOVA (Time x Side x Group interaction). Magnitude-based inferences of change (effect size) were calculated between PRE and POST 40m ST in both groups, according to Cohen's *d*. A Cohen's *d* of zero denotes no effect, whereas 0.2, 0.5 and 0.8 represent small, medium and large effects, respectively<sup>149, 150</sup>. Descriptive data of the two groups were compared using independent t-tests.

## **RESULTS**

### **Participants**

Table 6 shows the participant characteristics of the CP group. The groups were matched for age ( $22.7 \pm 3.6$  and  $26.1 \pm 3.7$  years, CP and AB respectively). Body Mass Index (BMI) calculated as kilograms per meters was significantly lower in the CP group ( $22.1 \pm 1.4$  and  $24.4 \pm 2.1$  kg/m<sup>2</sup>, CP and AB respectively,  $P < 0.05$ ) but bilateral skinfold measurements were similar in both groups.

**Table 6:** International Paralympic Committee competition classification, level of competition, diagnosis, resting Ashworth Scale spasticity level, involved side and current 100m sprint time of six participants with cerebral palsy

Participant	IPC class	Level of competition	Diagnosis	Resting spasticity	Involved side	100m time (s)
1	T 37	Paralympic	Hypertonic hemiplegia	1 – 2	Right	11.54
2	T 38	Paralympic	Hypertonic hemiplegia	1 – 2	Left	11.29
3	T 37	Paralympic	Hypertonic hemiplegia	1 – 2	Right	12.21
4	T 37	National	Hypertonic hemiplegia	1 – 2	Right	12.49
5	T 37	Paralympic	Hypertonic hemiplegia	1 – 2	Right	12.6
6	T 38	Paralympic	Athetoid hemiplegia	-	Left	11.21
<b>Mean (SD)</b>	-		-		-	<b>11.9 (0.9)</b>

### Multistage shuttle run test

Performance during the adapted MSRT was different between AB and CP ( $244.1 \pm 56.1$  vs.  $164.8 \pm 17.9$  shuttles completed in AB and CP, respectively,  $P < 0.001$ ). However, volitional exhaustion (fatigue) was marked by similar maximal HR ( $186 \pm 9$  bpm in AB vs.  $190 \pm 8$  bpm in CP) and RPE ( $19.8 \pm 0.6$  units in AB and  $18.1 \pm 1.9$  units in CP).

### 40m Sprint Test

40m ST was  $5.3 \pm 0.2$  s (PRE) and  $5.4 \pm 0.2$  s (POST) for the AB group and  $5.6 \pm 0.3$  s (PRE) and  $5.9 \pm 0.4$  s (POST) for the CP group (Table 7, Figure 7 A). The AB group was faster than the CP group in both the PRE and POST 40m ST ( $P < 0.01$ , Group main effect). The CP group was significantly slower in the POST 40m ST ( $P < 0.001$ , Interaction effect), whereas the AB group showed a strong tendency to be slower POST 40m ST ( $P = 0.054$ ). Cohen's  $d$  was 0.76 and 0.60 for CP and AB groups, respectively, representing a medium effect PRE to POST fatigue.

### Vertical Jump Test

#### *Vertical Jump (both legs)*

VJ-B jump height was greater in the AB group compared to the CP group ( $54.5 \pm 4.6$  cm (AB) vs.  $44.9 \pm 4.5$  cm (CP) for PRE and  $55.7 \pm 4.9$  cm (AB) vs.  $45.5 \pm 4.1$  cm (CP) for POST, Table 7, Figure 7 B,  $P < 0.0005$ , Group main effect). Neither the AB nor CP group jump heights changed significantly after fatigue.

#### *Vertical Jump (affected leg)*

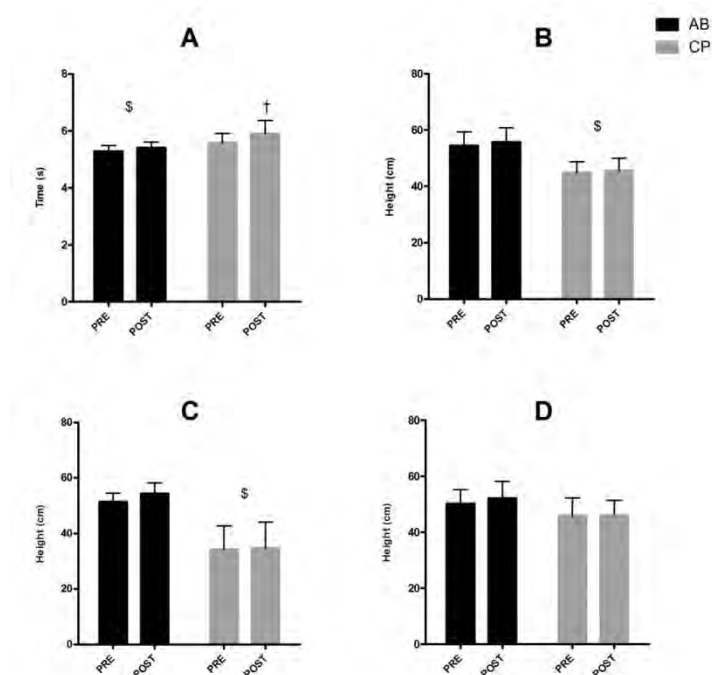
Jump height was higher in the AB group at both PRE and POST MSRT ( $51.4 \pm 2.9$  cm (PRE) and  $54.5 \pm 3.6$  cm (POST) for AB, vs.  $34.3 \pm 7.8$  cm (PRE) and  $34.7 \pm 8.6$  cm (POST) for CP, Table 7, Figure 7 C,  $P < 0.00000$ ). Fatigue did not affect jump height on the A leg in either group.

#### *Vertical Jump (non-affected leg)*

No significant differences were found in VJ-NA between AB and CP ( $50.2 \pm 4.9$  cm (PRE) and  $53.3 \pm 5.7$  cm (POST) for AB and  $45.9 \pm 5.8$  cm (PRE) and  $46.1 \pm 4.9$  cm (POST) for CP, Table 7, Figure 7 D). There was no effect of fatigue on jump height during VJ-NA.

**Table 7:** Performance in seconds in the able-bodied and cerebral palsy groups in 40m Sprint Test (40m ST, s) and vertical jump performances in centimetres on both legs (VJ-B, cm), affected leg (VJ-A, cm) and non-affected leg (VJ-NA) from PRE to POST fatigue. \$  $P < 0.001$  group main effect, †  $P < 0.05$  time x group interaction.

	AB		CP		
	PRE	POST	PRE	POST	
<b>40m ST (s)</b>	5.3 ± 0.2	5.4 ± 0.2	5.6 ± 0.3	5.9 ± 0.4	\$ †
<b>VJ-B (cm)</b>	54.5 ± 4.6	55.7 ± 4.9	44.9 ± 4.5	45.5 ± 4.1	\$
<b>VJ-A (cm)</b>	51.4 ± 2.9	54.5 ± 3.6	34.3 ± 7.8	34.7 ± 8.6	\$
<b>VJ-NA (cm)</b>	50.2 ± 4.9	53.3 ± 5.7	45.9 ± 5.8	46.1 ± 4.9	



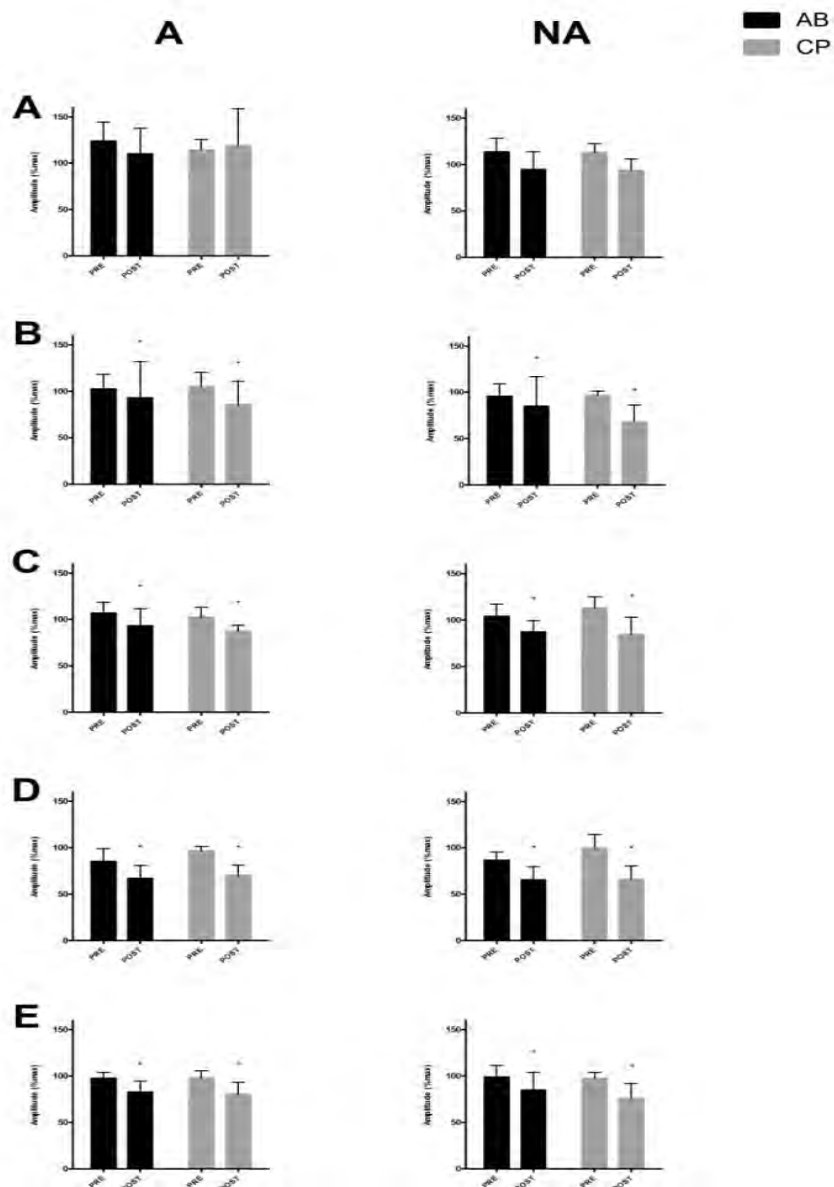
**Figure 7:** Time in seconds (s) for 40m sprint test (Figure 7 A), vertical jump height on both legs (Figure 7 B), vertical jump height on affected leg (Figure 7 C) and vertical jump height on non-affected leg (Figure 7 D) for able-bodied group (AB, black bars) and cerebral palsy group (CP, grey bars) from PRE and POST fatigue. \$  $P < 0.001$  group main effect, †  $P < 0.05$  time x group interaction.

## **Electromyography**

Figures 8 A - E, 9 A - E, 10 A - E and 11 A - E depict normalised EMG amplitude expressed as percentage maximum activation (% max) on the A and NA sides of the five measured muscles in the AB group (black bars) and the CP group (grey bars).

### *40m Sprint Test*

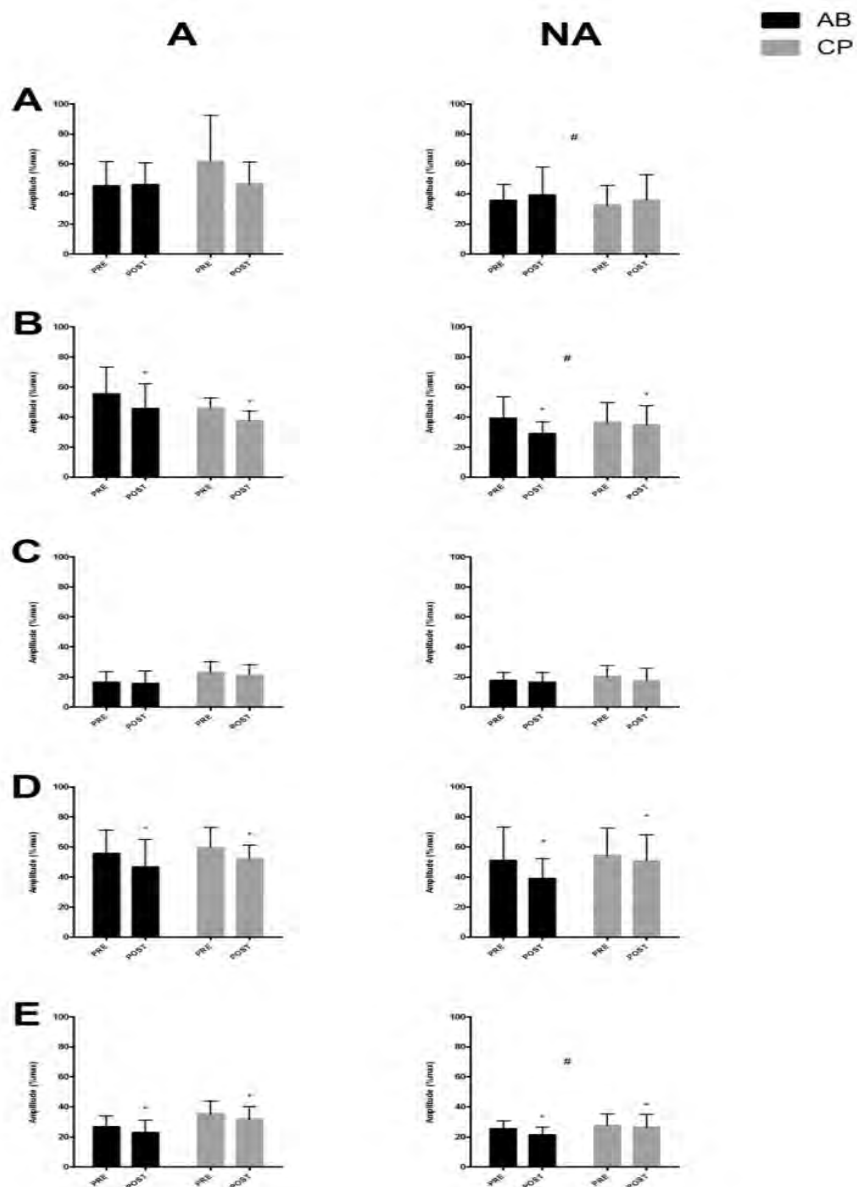
EMG of all tested muscles during the 40m ST changed similarly following the MSRT in the CP and AB groups. A significant reduction in EMG activity was observed in four of the five muscles after fatigue in both groups (Figure 8 B, GM; Figure 8 C, BF; Figure 8 D, VLO; Figure 8 E, GC; Time main effect,  $P < 0.05$ ) while no difference was found in the ES (Figure 8 A).



**Figure 8:** 40m sprint test EMG: Normalised muscle activation of the 40m sprint for affected/non-dominant side (A, left panel) and non-affected/dominant side (NA, right panel) for the able-bodied group (AB, black bars) and cerebral palsy group (CP, grey bars) from PRE to POST fatigue, for erector spinae (Figure 8 A), gluteus medius (Figure 8 B), biceps femoris (Figure 8 C), vastus lateralis (Figure 8 D) and gastrocnemius (Figure 8 E). \*  $P < 0.05$  time main effect.

*Vertical Jump (both legs)*

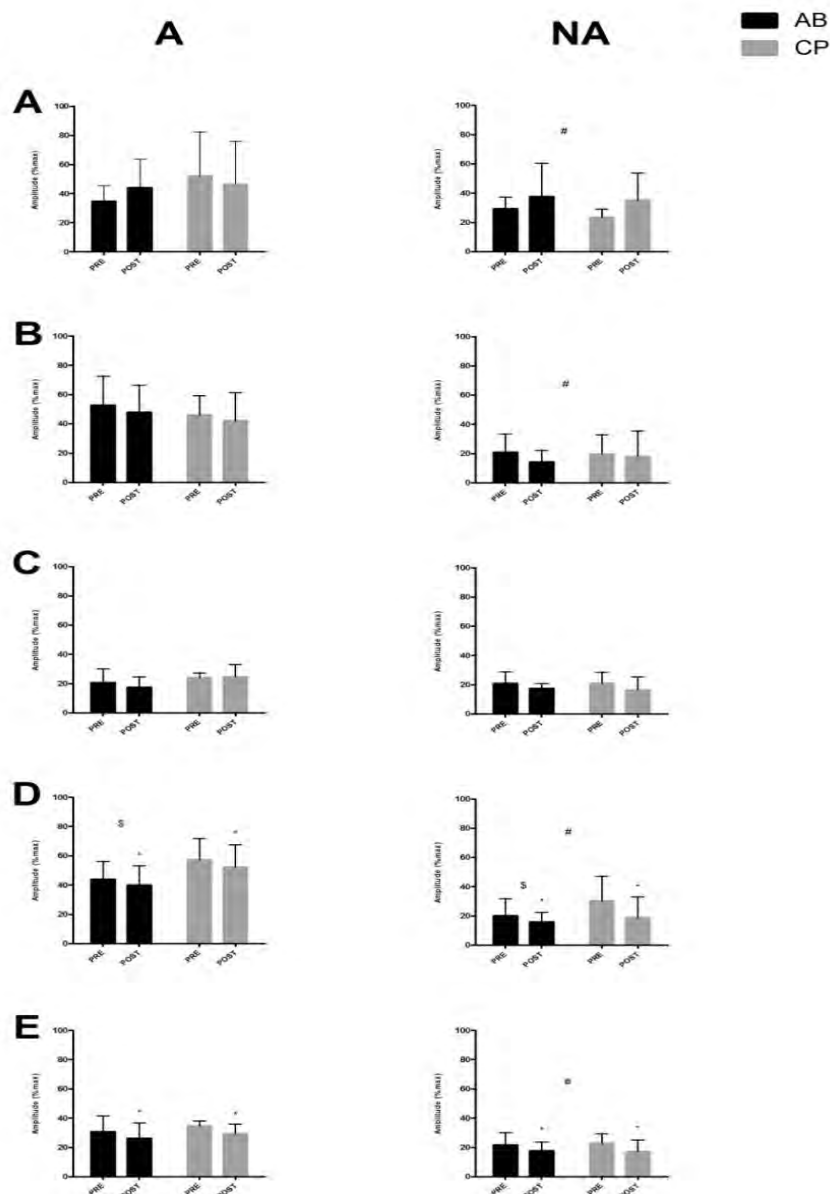
There was a time main effect for GM, VLO and GC, which decreased significantly after fatigue in both groups (Figure 9,  $P < 0.05$ ). EMG activity was lower on the NA side of both the CP and AB groups in three of the five measured muscles (Figure 9 A, ES; Figure 9 B, GM; Figure 9 E, GC; Side main effect,  $P < 0.05$ ).



**Figure 9:** VJ-B EMG: Normalised muscle activation of the vertical jump on both legs (VJ-B) for affected/non-dominant side (A, left panel) and non-affected/dominant side (NA, right panel) for the able-bodied group (AB, black bars) and cerebral palsy group (CP, grey bars) from PRE to POST fatigue, for erector spinae (Figure 9 A), gluteus medius (Figure 9 B), biceps femoris (Figure 9 C), vastus lateralis (Figure 9 D) and gastrocnemius (Figure 9 E). \*  $P < 0.05$  time main effect, #  $P < 0.05$  group main effect.

*Vertical jump (affected leg)*

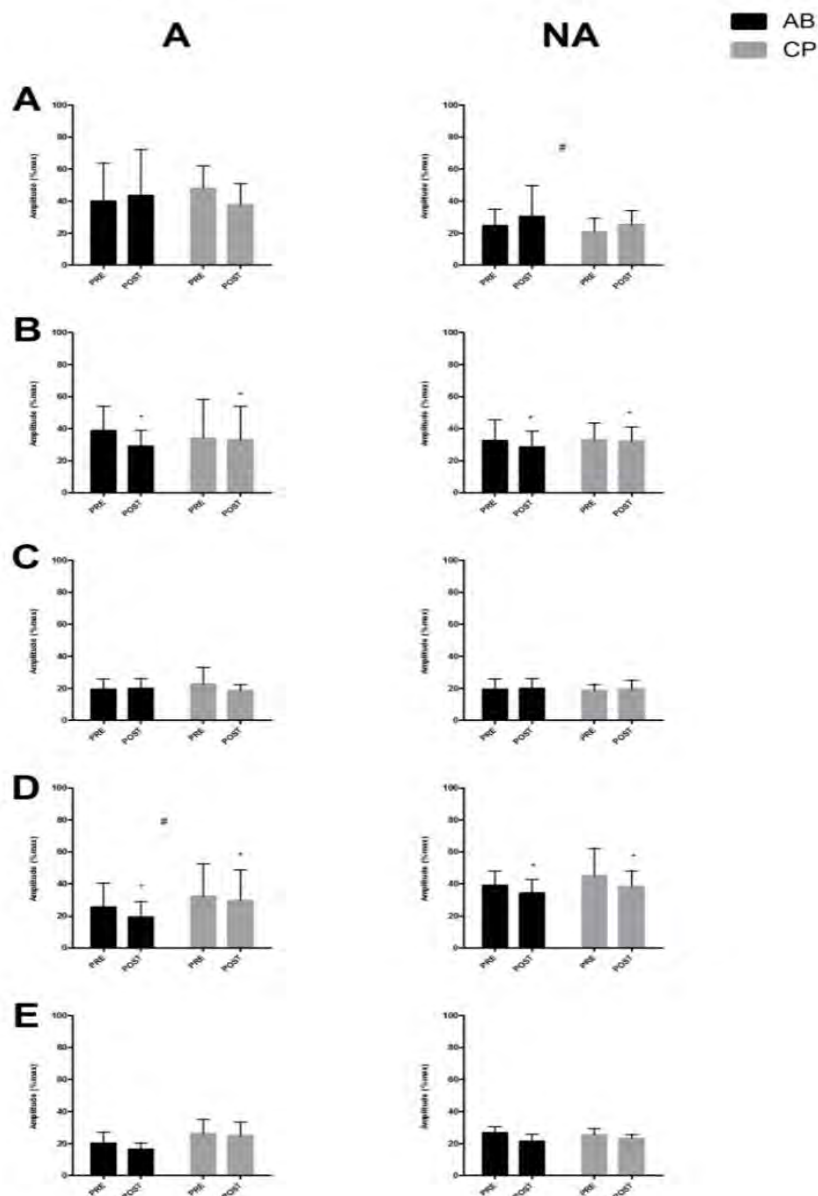
In the VJ-A, EMG activity was lower on the NA side in both groups, in four of the five measured muscles (Figure 10 A, ES; Figure 10 B, GM; Figure 10 D, VLO; Figure 10E, GC; Side main effect,  $P < 001$ ). EMG activity decreased after fatigue on both sides in GC and VLO in both groups (Figure 10 D, VLO; Figure 10 E, GC; Time main effect,  $P < 0.05$ ) and EMG activity was lower in VLO in the AB group compared to the CP group (Figure 10 D, VLO; Group main effect,  $P < 0.05$ ).



**Figure 10:** VJ-A EMG: Normalised muscle activation of the vertical jump on the affected leg (VJ-A) for affected/non-dominant side (A, left panel) and non-affected/dominant side (NA, right panel) for the able-bodied group (AB, black bars) and cerebral palsy group (CP, grey bars) from PRE to POST fatigue, for erector spinae (Figure 10 A), gluteus medius (Figure 10 B), biceps femoris (Figure 10 C), vastus lateralis (Figure 10 D) and gastrocnemius (Figure 10 E). \*  $P < 0.05$  time main effect, #  $P < 0.05$  sides main effect, \$  $P < 0.05$  time x group interaction.

*Vertical jump (non-affected leg)*

During VJ-NA, there was no difference between the CP and AB groups. EMG activity was lower in the NA ES than the A ES (Figure 11 A,  $P < 0.01$ ), and was significantly higher in the NA VLO compared to the A VLO (Figure 11 D; Side main effect,  $P < 0.01$ ). There was a similar reduction in activity with fatigue in two muscles (Figure 11 B, GM; Figure 11 D, VLO; Time main effect,  $P < 0.05$ ).



**Figure 11:** VJ-NA EMG: Normalised muscle activation of the vertical jump on the non-affected leg (VJ-NA) for affected/non-dominant side (A, left panel) and non-affected/dominant side (NA, right panel) for the able-bodied group (AB, black bars) and cerebral palsy group (CP, grey bars) from PRE to POST fatigue, for erector spinae (Figure 11 A), gluteus medius (Figure 11 B), biceps femoris (Figure 11 C), vastus lateralis (Figure 11 D) and gastrocnemius (Figure 11 E). \*  $P < 0.05$  time main effect, #  $P < 0.05$  sides main effect.

## DISCUSSION

The present study investigated the effect of fatiguing exercise on subsequent explosive sprint running performance and neuromuscular characteristics in elite Paralympic athletes with CP. There were two important findings in the present chapter, namely: 1) the similarity in the magnitude of fatigue between athletes with CP and AB athletes, and 2) the jump performance and associated neuromuscular asymmetries that were identified in athletes with CP.

The first important finding of this study was the magnitude-based similarity in fatigue in both groups. 40m ST performance was affected by fatigue from PRE to POST similarly in both groups, while VJ performance was not affected by fatiguing exercise in either group. These similarities in response to fatigue between the groups were supported by the finding of similar EMG measurements in the trials. Previous research evaluating fatigue as a decline in force output over repeated muscle contractions has suggested that individuals with CP display greater fatigue resistance compared to AB individuals<sup>17, 81, 82, 151</sup>. However, the apparent ability to resist fatigue in individuals with CP is likely the consequence of an inability to generate high workloads (and the resultant physiological and metabolic changes that cause fatigue) from the onset of exercise<sup>152</sup>. In individuals with CP, muscle weakness exists as a result of numerous central deficits, which impairs the ability to generate higher force outputs. These central deficits include impaired central drive resulting in lower voluntary muscle activation, impaired motor unit firing rate modulation with altered firing sequencing, which can manifest as spasticity and co-activation<sup>17, 81, 82</sup>. Furthermore, peripheral changes as a consequence of central insult include muscular weakness, stiffness and increased collagen within the muscle. Importantly, a predominance of Type I slow-twitch muscle fibres and atrophy of Type II fast-twitch muscle fibres has been identified in

individuals with CP, purportedly as a result of constant low-level muscle activation associated with spasticity<sup>17, 18, 76, 81, 82</sup>. However, the studies reporting greater fatigue resistance in individuals with CP were conducted in populations of sedentary children with CP, where confounding factors of physical inactivity and muscular immaturity may further obscure the paretic muscle's true response to fatiguing exercise.

The present study has shown that sprint and explosive jump performance is similarly affected by a fatiguing bout of exercise in a group of elite Paralympic athletes with CP, compared to AB athletes. Thus, the magnitude of fatigue resistance was not different between the groups. In fact, there was statistically more fatigue in the CP group than the AB group in the 40m ST, which further denounces the hypothesis that fatigue resistance is present in individuals with CP. This similarity in response to fatigue could be attributed to similarities in initial performance between the groups, which whilst significant, were considerably smaller than those documented in previous research.

In Chapter 3 of this thesis, the performance deficit observed between CP and AB groups was 10%, compared to the 6% and 9% deficits in 40m ST performance PRE- and POST fatigue found in the present chapter, respectively. Furthermore, the present study described a deficit of 18% was observed during vertical jumps off both legs and a 33-36% deficit in performance in the A leg (Figure 7). This was considerably smaller than the power and strength deficits observed in sedentary individuals with CP<sup>17, 18, 49, 50</sup>.

These relative similarities with respect to performance may explain the comparable response to a fatiguing bout of exercise between groups. In both CP and AB groups, 40m ST was compromised by fatigue (5% decrease in the CP group (Medium effect, 0.76) and 2% decrease in the AB group (Medium effect, 0.60, Figure 7 A). However, it was found that

jump performances (VJ-B, VJ-A, VJ-NA, Figure 7 B-D) were not affected by fatigue in either the CP or AB groups. It is possible the finding of no change from PRE to POST in VJ performance in either group resulted from cumulative rest periods, and short term muscle recovery, over the testing protocol<sup>153, 154</sup>, whilst 40m ST performance reflected a true response to the MSRT.

During the 40m ST, all associated normalised EMG measures showed similar changes from PRE to POST fatigue in both groups ( $P < 0.05$ , Figure 8). This finding further supports the findings of Chapter 3 that highly trained individuals with CP fatigue similarly to AB individuals measured in this chapter. Changes in EMG activity were characterized by a reduction in muscle activation in four of the five measured muscles. This supports previous research showing that the central drive to recruit motor units is reduced with the onset of fatigue in AB individuals, and that this is associated with decreased power output or exercise intensity over the course of an exercise trial<sup>117, 155-159</sup>.

Furthermore, EMG activity measured during all vertical jumps also changed in a similar manner in both groups from PRE to POST fatigue (Figures 9 - 11), with the exception of increased normalised VLO activity during VJ-A in the CP group (Figure 10 D). Other EMG measurements reflected the expected reductions in muscle activity after fatigue, including reduced EMG activity during the 40m ST POST fatigue (Figure 8) and during VJ in selected muscles, despite maintenance of performance from PRE to POST (Figures 9 - 11), but were similar between AB and CP groups.

The second important finding was related to the differences observed in the VJ between A and NA sides in the athletes with CP. The study design in this study enabled assessment of each leg individually, as well as the combined leg performance (VJ-B). Significantly reduced

jumping performance was observed during VJ-A and VJ-B, but not in VJ-NA, in the CP group (0% deficit in VJ-NA, compared to 18% in VJ-B and 33 - 36% in VJ-A, Figure 7 B - D). This finding demonstrated that when both legs are used together (VJ-B), performance deficits result from the large influence of the A leg's performance capacity, but that the high levels of training undertaken by the CP group, resulted in identical performance measured on the NA side of athletes with CP, and D side of the AB athletes.

The asymmetry between A and NA legs is similar to levels of asymmetry observed in sedentary individuals with CP<sup>23</sup>. However, previous literature has clearly stated that there is an "affected" and "less-affected" side in hemiplegic individuals with CP due to the brain's adaptation to central damage which produces bilateral impairment after unilateral brain injury<sup>160</sup>. That is, the brain's adaptation to unilateral upper motor neuron lesions, as well as subsequent functional effects of these lesions, result in secondary changes, including weakness, on the NA side of individuals with CP. Indeed, these secondary neuroplastic changes have been previously improved in children with CP, using constraint-induced movement therapy, a method where the use of the A side is increased by constraining the non-affected limb<sup>161, 162</sup>.

The current group of athletes with CP had the same absolute performance on their NA leg as National and International field hockey athletes, supporting Chapters 2 and 3 of this thesis that suggested that long term training may result in an adaptation toward AB physiology, rather than toward impairment. This finding also suggests that the individual with CP is highly trainable through high levels of physical activity. However, this possible maximum adaptation toward typically developed levels did not eliminate the asymmetry between A and NA limbs, as seen in VJ-A. Assuming the asymmetry was to remain constant in all activities, it may be the primary explanation for deficits seen in the VJ-B

(18%), 40m ST (6 – 9%) and indeed, Paralympic track performance (13 – 20% for T38 and T37 100 m world record performance compared to AB world record performance), where both limbs contribute to performance outcomes.

The finding that athletes perform towards their A leg's capacity while performing activities involving both legs was supported by the finding of significantly lower EMG in the NA leg than the A leg, in most muscle groups, for both VJ-B and VJ-A (Figure 9 - 10), but not VJ-NA (Figure 11). This finding suggested that compensation may occur by the central nervous system to counteract the underperformance associated with the asymmetry in muscle power. This compensation has previously been explained as the result of injury <sup>163</sup>. Interestingly, the same compensation strategies were observed in the AB group, indicating that all athletes perform at the level of their A (or ND) leg. However, the asymmetries present in AB athletes are not as marked as those in athletes with CP.

The findings in this study support the findings of Chapter 3, as well as provide further insight into performance and neuromuscular capacities of Paralympic athletes with CP, but had some limitations. Small sample size, the lack of force measurements during the VJ tests, the lack of inclusion of sedentary groups of CP and AB individuals, as well as the lack of onsite spasticity testing were limitations in this study. These factors need to be addressed by further research in a larger sample in order to investigate differences between A and NA sides, the role of spasticity in performance results of athletes with CP, and the real effect of long term athletic training on physiology. Further research is required in a similar group of elite Paralympic athletes with CP to investigate possible explanations for the findings in this chapter, and Chapter 3 of the present thesis.

## CONCLUSION

This chapter described explosive performance, neuromuscular characteristics and fatigue in elite Paralympic athletes with CP and AB athletes. In a sample of elite Paralympic athletes with CP in classes T37 and T38 (corresponding to GMFCS Level I), sprint running and explosive jumping performance was impaired. Performance was particularly impaired whilst using the A leg, but showed similar levels of fatigue in all tests, compared to the AB group. It was proposed, in accordance with the findings of Chapters 2 and 3, that Paralympic athletes with CP may represent a group of individuals who have achieved the highest possible adaptation toward normal physiology, as a result of elite level training over many years, providing evidence that individuals with CP are highly trainable. Despite this, a marked asymmetry between A and NA legs was found in the present study, indicating that activity generated by both legs is performed towards the capacity of the A leg. Chapter 5 investigates possible explanations for the findings of the current chapter and the findings of Chapter 3.

## **CHAPTER 5**

# **The effect of distance deception on pacing during shuttle running trials in Paralympic athletes with cerebral palsy**

The findings of the present chapter have been accepted for publication in the *Scandinavian Journal of Medicine and Science in Sports* (2015)

## INTRODUCTION

The relationship between exercise performance and fatigue development has been studied in humans for many years. There are various physiological models for fatigue and cessation of exercise<sup>164</sup>. The integrated central model of exercise-induced fatigue postulates that exercise performance is not limited, but rather regulated by the complex interaction of central and peripheral factors that allow the body to perform at near-maximum capacity without harmful physiological consequences. This model proposes that through both efferent motor command and afferent feedback, the brain is able to regulate both exercise intensity and the perturbations it causes. This is measurable as a pacing strategy, controlled consciously and subconsciously, that results in appropriate distribution of resources throughout an exercise bout to achieve the desired outcome<sup>165-168</sup>. Cerebral palsy (CP) is a movement disorder caused by central insult to various areas of the brain at a young age, which results in altered neuromuscular physiology and diminished exercise capacity.

Interestingly, studies examining muscle fatigue (the inability to maintain force output over time) in individuals with CP have shown that they exhibit fatigue resistance. That is, the studies showed that individuals with CP display a reduced decline in force output over time during repeated maximal isokinetic muscle contractions, but total force output throughout the trial was lower than able-bodied (AB) controls<sup>17, 81, 82</sup>. However, the fatigue resistance reported in these studies was actually the effect of muscle weakness in the CP group. However, in Chapters 3 and 4 of this thesis, using elite Paralympic athletes with CP, it was shown that fatigue resistance may not be present in individuals with CP. Indeed, it was demonstrated that athletes with CP fatigue at a similar rate to AB athletes in both supramaximal cycling trials (Chapter 3) and running trials following the induction of fatigue (Chapter 4). It was proposed in those chapters that the findings were the result of high

intensity training over many years in the elite athlete cohort studied. Furthermore, it was proposed that this training enabled them to produce similar performance characteristics to AB controls, including a similar measured decline in power output over time. Therefore, the difference in fatigue resistance between individuals with CP and AB individuals was partly eliminated as a result of more similar absolute performance capacities.

The findings of the research raised questions regarding whether there were other factors involved in the fatigue resistance observed in CP. The finding of similar performance profiles during cycling exercise in Chapter 3 occurred despite neuromuscular irregularities including bilateral co-activation, atypical firing patterns, and continuous irregular muscle activation in the 30 second sprint. The atypical changes observed support anecdotal observations of elite athletes with CP falling over the finish line and “tying up” with apparent spasticity in the affected limbs at the end of the 100, 200, and 400 meter (m) running events in competition. This raised questions about whether athletes with CP fatigue more readily or rather pace themselves differently to AB athletes. Therefore, the current study aimed to investigate whether athletes with CP utilise different pacing strategies during prolonged running exercise, compared to AB athletes, in an attempt to understand possible explanations underlying the findings of Chapters 3 and 4.

Researchers have used deception trials to challenge pacing strategy utilisation, as it is understood that the brain paces exercise according to knowledge of the endpoint of the exercise<sup>156, 169</sup>. In this chapter, pacing strategy was examined using shuttle running sets totalling 1600 m in two trials (one deceived, DEC; one non-deceived, N-DEC). It was hypothesised that the athletes with CP would adopt a relatively more aggressive pacing strategy during the deception trial, when the expectation was for a shorter exercise bout, compared to the AB athletes. This would be observed as a significantly faster performance

during the first five sets before deception, and a significant reduction in running speed after disclosure of deception.

## **METHODS**

### **Participants**

Six elite athletes with CP (CP group) and 13 AB athletes (AB group) were recruited for the study. The athletes with CP are all Paralympic track sprinters with hemiplegic CP, including athletes who participate in the 100, 200 and 400 m events. They were all diagnosed by a physician, as well as classified as either T37 or T38 athletes, in accordance with criteria established by the IPC and the CPISRA classification systems<sup>11, 12, 197</sup>. Athletes classified as T37 are classified as having ambulant hemiplegic profiles (as a result of hypertonia, athetosis or ataxia) with marked impairment on the affected (A) side and full function on the non-affected (NA) side. T38 athletes are classified as presenting with minimal, yet significant, impairment in any limb distribution as a result of hypertonia, athetosis or ataxia. Classification of both T37 and T38 athletes includes a score of 1 to 2 on the Modified Ashworth Scale, denoting mild to moderate resting spasticity in the affected limbs<sup>12</sup>. All athletes with CP fell into Level I or II of the Gross Motor Function Classification System (GMFCS)<sup>108, 170</sup>.

The AB athletes included in this study were International and National level field hockey players. Only athletes who competed in sprint-specific positions (striker, midfielder) were recruited.

Each participant in both AB and CP groups provided written informed consent prior to the study. The study was approved by the University's Human Research Ethics Committee (Ref: 156/2011).

### **General overview of testing**

Participants reported to the laboratory on two occasions, with at least 48 hours between visits. On the first visit, height in meters (m) and body mass in kilograms (kg) were measured using calibrated equipment (Seca, Model 708, Germany). On the two visits, after a self-selected warm-up, participants performed a N-DEC trial (1600 m) or a DEC trial (1000 m + 600 m), in a random order. Heart rate (HR), ratings of perceived exertion (RPE) and bilateral electromyography (EMG) of five muscles was recorded every other shuttle in each set. Specific details of testing methods are described subsequently.

### **Shuttle Run Test**

Participants were instructed to run 10 shuttles between two lines 20 m apart, which constituted one 200 m set. Each trial consisted of eight such sets, separated by one minute rest periods. Participants were instructed to complete the entire trial in the shortest possible time, which required each set to be completed as fast as possible, given the known total exercise demand. Participants were further instructed to touch the 20 m line with their non-affected/dominant foot. This was designed to eliminate the effect of turning on performance in the groups as much as possible. In the N-DEC trial, participants were instructed to run eight sets of 10 shuttles (1600 m). In the D trial, participants were informed that they were running a shorter distance and were instructed to run five sets of 10 shuttles (1000 m). Upon completion of the fifth set however, they were informed that they had been deceived and that three additional sets were required (an additional 600 m), thus matching distance in both trials to eight sets of 10 shuttles. The trials were randomised to negate the effect of

awareness of deception on subsequent performance, although it must be recognised that this can never fully be achieved. Split times were recorded after every second shuttle, as well as for each set, and the entire trial, and expressed in seconds (s).

An additional analysis was performed to determine pacing profiles of the groups in the N-DEC and DEC trials. Performance times were averaged for the first five sets, the sixth set, and the last three sets, and compared to the mean of the total N-DEC trial, in each athlete. Data are given for N-DEC and DEC trials in both AB and CP groups.

### **Heart rate**

HR was measured continuously throughout all the sets (Polar Electro Oy, Kempele, Finland). Measurements were recorded at the completion of each set and expressed as beats per minute (bpm).

### **Ratings of perceived exertion**

RPE was recorded at the completion of every set using the 6 – 20 point Borg Scale <sup>171</sup>. This scale represents the participant's perception of effort required to conduct the exercise and is rated from "very very light" to "maximal exertion". The scale was explained to all participants prior to the start of the trial, using a standardized set of instructions. Scores were recorded at the completion of each set, and reported as absolute score units (6 – 20).

### **Electromyography**

EMG of five bilateral muscles was measured using a telemetric EMG system (GT2400 G2, Noraxon, USA Inc., Arizona, USA). Muscles tested included erector spinae (ES), gluteus medius (GM), biceps femoris (BF), vastus lateralis (VLO) and gastrocnemius medius (GC). EMG was measured on both the A and NA sides of both groups, where the A and NA sides

in the CP group corresponded with both their own and the AB group's dominant (D) and non-dominant (ND) sides, respectively. Dominance was self-reported. A specified area over the belly of each muscle was shaved and cleaned with ethanol, and two electrodes (Blue Sensor, Medicotest, Denmark) were placed 2 centimetres (cm) apart, with the reference electrode being placed in the anterior superior iliac spine of the right leg. Electrode placement was in accordance with Surface Electromyography for the Non-Invasive Assessment of Muscle (SENIAM) recommendations<sup>115</sup>. To ensure reliable EMG data collection, EMG was measured in a rested state as well as during individual manual isolation of each muscle. This ensured that the signals recorded were not artefacts of incorrect placement, movement or noise generated by external variables. For analysis of the EMG signal, the raw EMG signals were band passed filtered between 20 and 500 Hz. All signals were rectified and smoothed using root mean square analysis for a 50 millisecond (ms) window.

EMG activity was recorded continuously for each set. A three second epoch of EMG data was analysed every alternate shuttle, and averaged for each set. The EMG was then normalised to the highest mean amplitude in an epoch in the shuttles of the first set of either DEC and N-DEC trials. This maximum amplitude represented the self-paced maximum muscle activation registered in all trials. The use of normalising all activity to the first set establishes a constant baseline to which all activity is compared, and expressed as a percentage maximum activation (% max).

### **Statistical analyses**

All data were analysed and expressed for the eight sets in the N-DEC and DEC trials in both AB and CP groups. Variables included total time (s), HR (bpm) and RPE (scale units) per set, and percentage maximum amplitude for A/ND and NA/D sides mean normalised EMG

(% max). All data were analysed using statistical software (Statistica 12; Statsoft Inc, Tulsa, OK). All data was distributed normally and significance was accepted at  $P < 0.05$ . Comparisons were made between AB and CP groups, between N-DEC and DEC trials and between A and NA sides, using a four way repeated measures analysis of variance ANOVA (group x trial x time x side interaction) for all measured outcomes (shuttle performance, in-depth pacing analysis, HR, RPE and EMG). Descriptive data (age and BMI) of the two groups were compared using independent t-tests.

## RESULTS

### Participants

Table 8 shows the participant characteristics of the CP group. The groups were matched for age ( $26.1 \pm 3.5$  vs.  $22.7 \pm 3.6$  years for AB and CP, respectively). Body mass index (BMI) calculated as kilograms per meter was lower in the CP group ( $24.4 \pm 2.1$  vs.  $22.1 \pm 1.4$   $\text{kg/m}^2$ , AB and CP respectively,  $P < 0.05$ ).

**Table 8:** International Paralympic Committee competition classification, level of competition, diagnosis, resting Ashworth Scale spasticity level, involved side and current 100m sprint time in seconds of six participants with CP

Participant	IPC class	Level of competition	Diagnosis	Resting spasticity	Involved side	100m time (s)
1	T 37	Paralympic	Hypertonic hemiplegia	1 – 2	Right	11.54
2	T 38	Paralympic	Hypertonic hemiplegia	1 – 2	Left	11.29
3	T 37	Paralympic	Hypertonic hemiplegia	1 – 2	Right	12.21
4	T 37	National	Hypertonic hemiplegia	1 – 2	Right	12.49
5	T 37	Paralympic	Hypertonic hemiplegia	1 – 2	Right	12.6
6	T 38	Paralympic	Athetoid hemiplegia	-	Left	11.21
<b>Mean (SD)</b>	-		-		-	<b>11.9 (0.9)</b>

### Performance, heart rate and ratings of perceived exertion

Table 9 presents raw data of shuttle running performance (s), HR (bpm) and RPE (scale units) for eight sets of the N-DEC and DEC trials, of the AB and CP groups. Statistical significance is not shown.

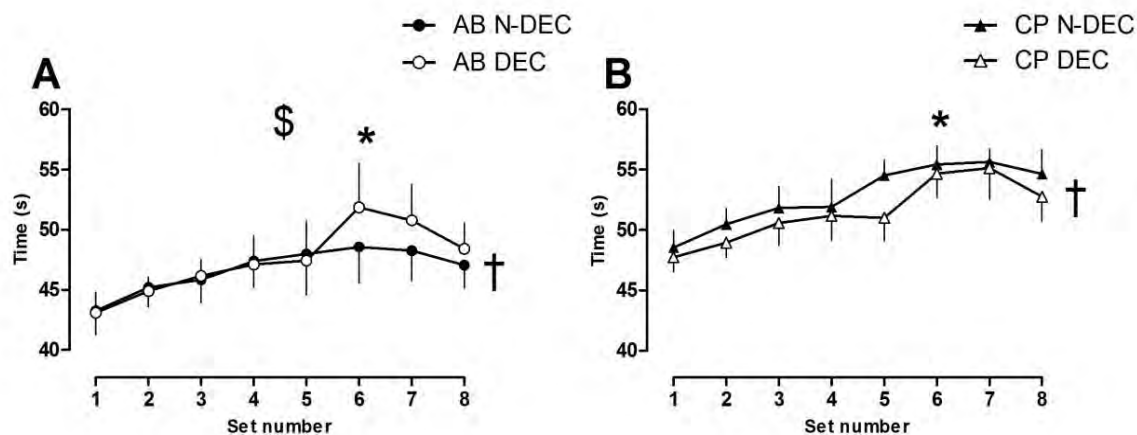
**Table 9:** Shuttle run performance time (seconds), heart rate (beats per minute) and ratings of perceived exertion (RPE, scale units) for able-body group non-deceived (AB N-DEC) and deceived (AB DEC) trials and cerebral palsy group non-deceived (CP N-DEC) and deceived (CP DEC) trials. Statistical significance not shown.

		Set 1	Set 2	Set 3	Set 4	Set 5	Set 6	Set 7	Set 8
<b>Time (s)</b>	<b>AB N-DEC</b>	43.2 ± 1.5	45.2 ± 0.8	45.9 ± 1.9	47.4 ± 2.1	48.0 ± 2.7	48.6 ± 3.0	48.3 ± 2.5	47.0 ± 1.9
	<b>AB DEC</b>	43.1 ± 1.8	44.9 ± 1.3	46.2 ± 1.4	47.1 ± 1.9	47.4 ± 2.8	51.9 ± 3.6	50.8 ± 3.0	48.4 ± 2.2
	<b>CP N-DEC</b>	48.5 ± 1.4	50.5 ± 1.3	51.8 ± 1.8	51.9 ± 2.3	54.5 ± 1.3	55.4 ± 1.6	55.7 ± 1.1	54.7 ± 2.0
	<b>CP DEC</b>	47.8 ± 1.2	49.0 ± 1.2	50.6 ± 1.9	51.2 ± 2.1	51.0 ± 1.9	54.7 ± 2.0	55.1 ± 2.6	52.8 ± 2.1
<b>HR (bpm)</b>	<b>AB N-DEC</b>	163.3 ± 13.3	167.1 ± 15.0	171.5 ± 13.0	175.3 ± 8.0	177.5 ± 6.1	178.6 ± 6.0	178.9 ± 5.9	181.6 ± 5.9
	<b>AB DEC</b>	162.3 ± 11.7	170.7 ± 8.6	173.8 ± 6.4	175.3 ± 6.9	176.4 ± 10.0	173.5 ± 10.6	174.9 ± 9.5	177.8 ± 8.8
	<b>CP N-DEC</b>	171.4 ± 7.4	178.0 ± 4.4	180.2 ± 6.4	180.4 ± 5.3	180.8 ± 4.3	181.8 ± 5.3	181.6 ± 6.3	185.4 ± 8.0
	<b>CP DEC</b>	172.3 ± 9.9	181.7 ± 7.2	180.8 ± 2.5	183.7 ± 3.5	187.5 ± 4.7	185.5 ± 2.8	187.5 ± 4.4	190.2 ± 4.6
<b>RPE (units)</b>	<b>AB N-DEC</b>	12.2 ± 1.4	13.7 ± 1.1	15.1 ± 1.1	16.5 ± 1.2	17.6 ± 1.3	18.4 ± 1.0	18.9 ± 1.0	19.4 ± 0.7
	<b>AB DEC</b>	12.2 ± 1.6	14.1 ± 1.4	16.0 ± 1.5	17.9 ± 0.1	19.1 ± 1.0	19.3 ± 0.8	19. ± 0.7	19.8 ± 0.4
	<b>CP N-DEC</b>	10.8 ± 2.2	11.8 ± 2.3	13.1 ± 2.3	14.2 ± 2.0	15.2 ± 2.3	16.0 ± 1.8	16.5 ± 2.0	17.7 ± 1.4
	<b>CP DEC</b>	9.3 ± 1.8	13.2 ± 2.6	14.5 ± 2.8	16.2 ± 2.5	17.8 ± 1.8	18.2 ± 1.6	18.3 ± 1.4	19.0 ± 1.0

### Shuttle Run Test

Figure 12 shows total time for eight sets in the AB group N-DEC and DEC trials (Figure 12 A) and CP group N-DEC and DEC trials (Figure 12 B). The CP group was significantly slower in all eight sets of both trials compared to the AB group (11% difference in N-DEC at first set, and 10% difference in DEC at first set, between AB and CP groups,  $P < 0.001$ ). Comparisons were conducted to examine the effect of testing order of DEC and N-DEC on performance in the groups, and no difference in order effect was found between the groups.

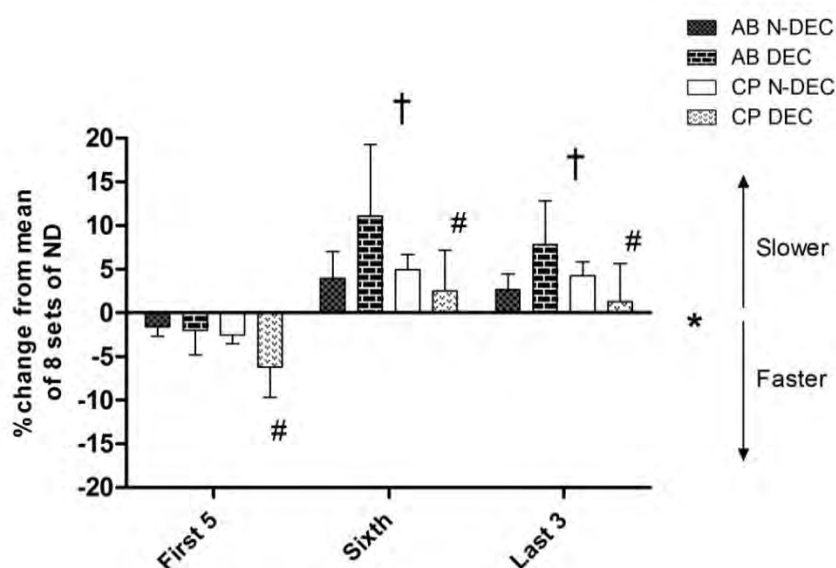
The global pacing strategy employed was similar between groups, with set time increasing progressively over time before a decrease during the eighth set ( $P < 0.0001$ ). In DEC trials, there was a significant increase in set time at set six after disclosure of deception ( $P < 0.001$ ).



**Figure 12: Performance of AB and CP groups during N-DEC and DEC trials**

Time for eight sets of 10 shuttles, separated by one minute rest periods for able-bodied group non-deceived (AB N-DEC, solid circle) and deceived (AB DEC, open circle) trials (Figure 12 A); cerebral palsy group non-deceived (CP N-DEC, solid triangle) and deceived (CP DEC, open triangle) trials (Figure 12 B). \*  $P < 0.001$  time main effect, \$  $P < 0.001$  group main effect, †  $P < 0.05$  trials x group interaction, †  $P < 0.05$  trials x time interaction.

Figure 13 shows the performance of N-DEC and DEC trials in the AB and CP groups, expressed as a percentage change from the N-DEC mean. This was done to compare the effects of deception on the relative pacing strategies utilised when using the N-DEC trials as an anchor. Performance in both groups changed significantly over time ( $P < 0.001$ ). All trials of both groups were faster in the first five sets, followed by a significantly slower running speed for the sixth set and the last three sets compared to the first five sets ( $P < 0.05$ ). The CP DEC trial showed significant differences from the AB DEC trial for all set groupings (first five, sixth, and last three). The CP group began the DEC trial significantly faster relative to N-DEC mean compared to the AB group ( $P < 0.05$ ), then slowed down relatively less after deception (sixth set and last three sets,  $P < 0.05$ ).

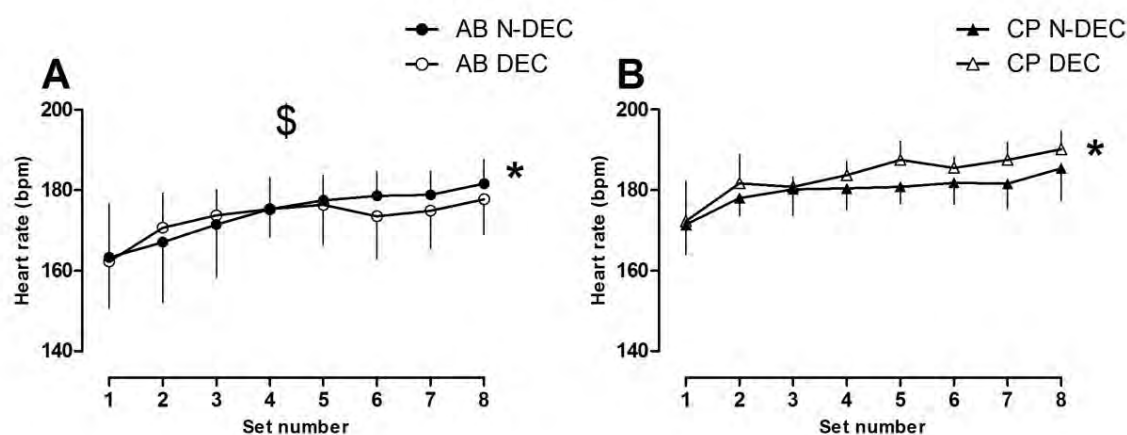


**Figure 13: Relative pacing of N-DEC and DEC trials in the AB and CP groups**

Percent change in performance of the first five, sixth, and last three sets (%) for able-bodied group non-deceived (AB N-DEC, dark chequered) and deceived (AB DEC, dark brick) trials and cerebral palsy group non-deceived (CP N-DEC, solid white) and deceived (CP DEC, white arrows) trials. \*  $P < 0.001$  time main effect, \$  $P < 0.01$  group main effect, #  $P < 0.05$  trials x group interaction, †  $P < 0.05$  trials x time interaction.

## Heart Rate

Both AB (Figure 14 A) and CP (Figure 14 B) groups' HR increased significantly over the eight sets of shuttle runs in both trials (Figure 14,  $P < 0.001$ ). The CP group had a significantly higher HR than the AB group throughout the trial ( $P < 0.05$ ).

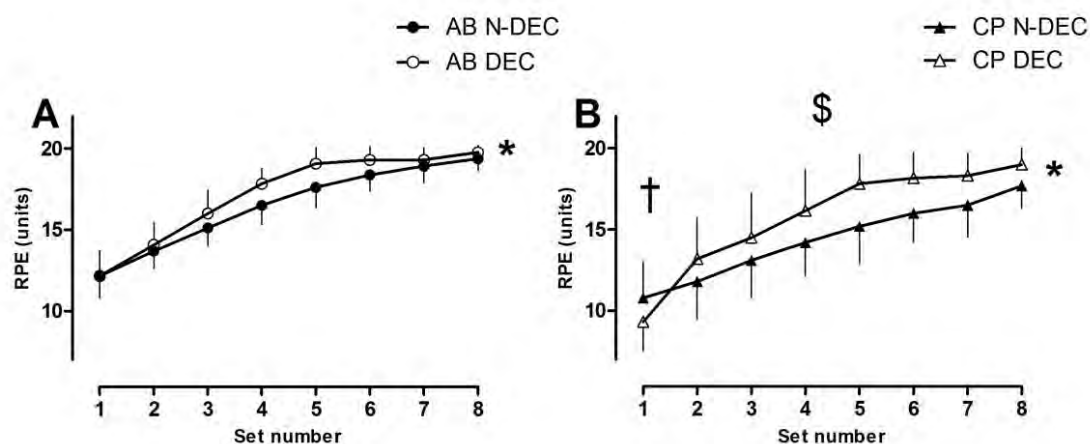


**Figure 14: Heart Rate during N-DEC and DEC trials in the AB and CP groups**

Heart rate for eight sets of 10 shuttles, for able-bodied group non-deceived (AB N-DEC, solid circle) and deceived (AB DEC, open circle) trials (Figure 14 A); cerebral palsy group non-deceived (CP N-DEC, solid triangle) and deceived (CP DEC, open triangle) trials (Figure 14 B). \*  $P < 0.001$  time main effect, \$  $P < 0.05$  group main effect.

## Ratings of perceived exertion

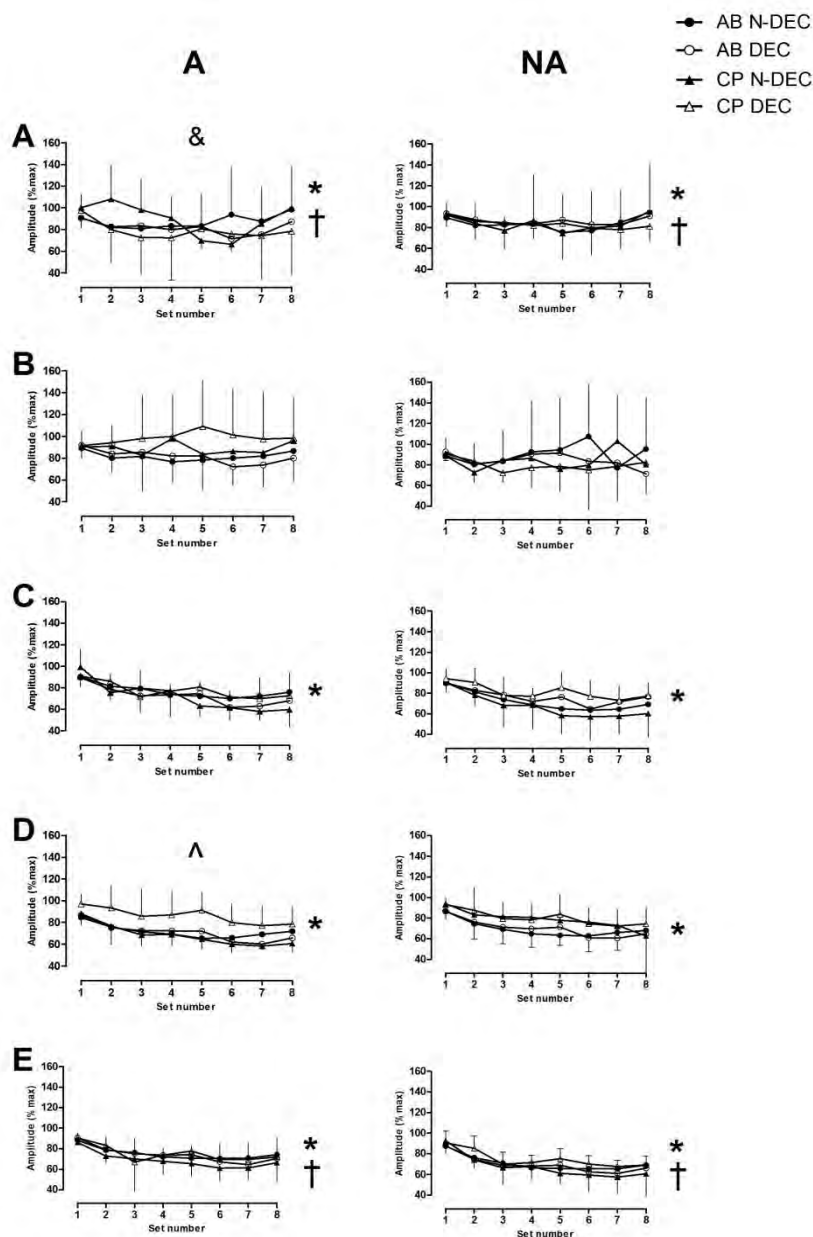
Figure 15 displays the RPE for AB (Figure 15 A) and CP (Figure 15 B) groups. There was a significant increase in RPE scores over time in both groups ( $P < 0.001$ ) as well as a significantly lower scores in the CP group compared to the AB group ( $P < 0.01$ ). RPE during the DEC trial in the CP group was different from all other trials in the first set, with a lower RPE value reported than any other trial ( $P < 0.001$ ).



**Figure 15: Ratings of perceived exertion during N-DEC and DEC trials in the AB and CP groups**  
 Ratings of perceived exertion for eight sets of 10 shuttles, for able-bodied group non-deceived (AB N-DEC, solid circle) and deceived (AB DEC, open circle) trials (Figure 15 A); cerebral palsy group non-deceived (CP N-DEC, solid triangle) and deceived (CP DEC, open triangle) trials (Figure 15 B). \*  $P < 0.001$  time main effect, \$  $P < 0.01$  group main effect, †  $P < 0.001$  trials  $\times$  time  $\times$  group interaction.

### Electromyography

Figure 16 shows EMG activity for A (left panel) and NA (right panel) muscles in ES (Figure 16 A), GM (Figure 16 B), BF (Figure 16 C), VLO (Figure 16 D) and GC (Figure 16 E) muscles for AB and CP groups for both N-DEC and DEC trials. Both NA (D) and A (ND) sides of four muscles (Figure 16 A, ES; Figure 16 C, BF; Figure 16 D, VLO; Figure 16 E, GC) displayed a significant decrease in skeletal muscle activity over time in both N-DEC and DEC trials ( $P < 0.05$ ). The A ES had significantly lower activity than the NA ES in the CP group, as well as ES activity in the AB group in both trials (Figure 16 A;  $P < 0.01$ ). Skeletal muscle EMG activity was significantly lower in both A and NA sides of the CP DEC trial at set eight compared to all other trials in both CP and AB groups (Figure 16 A, ES; Figure 16 E, GC;  $P < 0.05$ ). Skeletal muscle activity of the VLO was significantly higher on the A side in the DEC trial, than all other trials or sides (Figure 16 D, VLO;  $P < 0.01$ ).



**Figure 16: Muscle activity of the A and NA sides in the CP and AB groups during N-DEC and DEC trials**

Normalised muscle activity of eight sets of 10 shuttles, for affected/non-dominant side (left panel) and non-affected/dominant side (right panel) for the able-bodied group non-deceived (AB N-DEC, solid circle) and deceived (AB DEC, open circle) trials and cerebral palsy group non-deceived (CP N-DEC, solid triangle) and deceived (CP DEC, open triangle) trials in five bilateral muscles: erector spinae (Figure 16 A), gluteus medius (Figure 16 B), biceps femoris (Figure 16 C), vastus lateralis (Figure 16 D), gastrocnemius medius (Figure 16 E). \*  $P < 0.05$  time main effect, †  $P < 0.05$  trials x time x group interaction, &  $P < 0.05$  sides x time x group interaction, ^  $P < 0.01$  trials x sides x group interaction.

## DISCUSSION

The current study aimed to describe performance and pacing during repeated shuttle running in AB athletes and elite Paralympic athletes with CP. The first finding of the study was the difference in performance between the groups. The AB group ran faster than the CP group in both N-DEC (10% faster) and DEC (11% faster) trials (Figure 12), which is similar to the 10% difference in supramaximal cycling performance reported in Chapter 3 of this thesis. However, this difference in performance during shuttle running was higher than the 6% difference found in maximum sprint performance reported in the same groups of athletes in Chapter 4 of this thesis. This finding demonstrates that the AB athletes were running at a higher percentage of maximum than the athletes with CP, and a relative underperformance during running in the CP group throughout both D and N-DEC trials.

The difference in performance could be attributed to the effects of CP on performance, or perhaps the difference in familiarity with the type of exercise performed in this chapter. That is, the AB group comprised hockey players, for whom a repeated shuttle run consisting of approximately 50 seconds at high intensity and one minute recovery is more similar to competition than in a group of track sprinters (CP group). The CP group participated in repeated 200 m running drills with one minute recovery during regular training. Most importantly, the primary purpose of this study was to measure fatigue and thus this protocol was utilized for its known fatiguing effects.

Analysis of absolute data (Figure 12) did not provide sufficient clarity on pacing between the groups, therefore a more detailed analysis of performance over time was performed to isolate specific pacing patterns in the groups (Figure 13). Performance data were analysed for both the AB and CP groups for the mean of the first five, sixth and last three sets, relative

to their own N-DEC mean, where a negative change indicated relatively faster performance in comparison to their N-DEC mean and a positive change indicated poorer performance.

Two important findings revealed in the in-depth pacing analysis (Figure 13) are of particular interest. Firstly, both groups were faster in their first five sets than the mean of the whole N-DEC trial. As such, both groups displayed poorer performance in the last three sets of the trial (particularly the sixth set). This observation was expected and has been documented as part of a typical pacing strategy during both time-trial performances and in distance deceived running trials in AB individuals. In these studies, it has typically been found that performance decreases significantly, immediately following disclosure of deception, before a certain level of recovery and an endspurt in performance is observed, which is repeated in this study <sup>164-167, 167, 168</sup>.

In a deception trial, the central regulator, or brain, is deceived into believing it has a certain workload to complete, and regulates the allocation of resources accordingly, as pacing is highly dependent on knowledge of the exercise endpoint. Upon disclosure of deception however, the anticipatory calculations must be adjusted for a new endpoint, often with minimal reserve remaining. In the present study, the deception was revealed at the completion of the expected exercise bout, which would, according to the previous research discussed above, coincide with the depletion of all physiological resources and which would render any further exercise impossible until some recovery is achieved. This was not the case in the present chapter, as both AB and CP groups were able to continue exercising for a sixth set, albeit at a reduced intensity, and so a reserve was clearly maintained even with the known endpoint of exercise approaching. This reserve during maximal exercise of a relatively short duration confirms previous work in AB individuals showing that even sprint exercise is regulated with the maintenance of reserve capacity <sup>156 168, 172, 173</sup>.

The most important novel finding in this study was revealed in the analysis of the relative pacing strategies, and lends insight into the pacing strategies employed by the CP group. Even though similar global pacing strategy was observed in all trials, the CP group appeared to approach the DEC trial differently compared to both the CP N-DEC trial and both the AB trials (Figure 13). In the DEC trial, the CP group ran significantly faster in the first five sets compared to their N-DEC mean, whereas the AB group did not show this difference. It was expected that both groups would start the DEC trial significantly faster, since the total bout was expected to be shorter. That this was only true in the CP group shows that they performed more aggressively in a trial when the anticipated trial duration was reduced. Furthermore, the decline in performance in the CP group following disclosure of deception (set six) was significantly smaller than the decline in performance observed in the AB group, showing that the CP group was relatively less affected by deception than the AB group, and were able to maintain more stable performance over all eight sets of the deception trial better than the AB group.

This observation in the CP group was unexpected, and was not typical of the pacing strategy usually observed in distance-deception running trials<sup>167, 168</sup>, providing evidence for the utilisation of a conservative pacing strategy in this group of athletes. However, it must be borne in mind that the changes shown in Figure 13 are expressed relative to the N-DEC mean, and so this atypical pacing finding in the CP group would also be possible if the N-DEC trial was a relative underperformance. This would be possible if the comparison of the sets in the DEC trial were compared to a slower benchmark, artificially exaggerating them and creating a false impression of a flatter trajectory even after fatigue.

Identifying the possibility of conservative pacing strategy utilisation is difficult. The observation of similar HR measurements in the DEC and N-DEC trials showed that the CP

group performed both trials at the same intensity from the outset. This was also the case for the AB group (Figure 14), showing that the N-DEC trials were maximally performed, if HR is used as an indicator of physiological stress.

With respect to RPE, it was observed that both groups reported RPE increased over the duration of the N-DEC and DEC trials, but the CP group displayed a greater difference between the N-DEC and DEC trials (DEC was non-significantly higher, Fig 15). This finding supports the finding of underperformance of the CP group in the N-DEC trial. Further, it provides evidence that the CP group may not necessarily start DEC more aggressively, rather that they completed the N-DEC trial with a conservative pacing strategy than the AB group. The AB group also presented with consistently higher RPE scores in both N-DEC and DEC trials than the CP group ( $P < 0.01$ ), further supporting a relative underperformance by the CP group in the N-DEC trial.

Previous research conducted using isokinetic strength testing showed that sedentary children with CP produce a flattened fatigue profile over the duration of an exercise trial<sup>17, 81, 82</sup>. This has typically been interpreted as fatigue resistance, though it too is potentially confounded by the possibility that measuring fatigue relative to a lower “anchor” may be misleading. Indeed, the authors of these studies proposed that these findings were the result of muscular weakness that limited the children’s initial force output during the repeated knee flexion-extension strength testing protocol. Both central and peripheral factors were proposed as mechanisms for the muscular weakness observed in the studies<sup>17, 81-83, 111, 174,</sup>

<sup>175</sup>.

However, the study described in Chapter 4 which used externally paced maximal trials, showed that adult athletes with CP display similar levels of fatigue to AB athletes. The

athlete sample used in that study was the same group of athletes used in the present chapter, and comprised elite Paralympic athletes who were more closely matched in sprint performance to elite field hockey players. It was proposed that the observed similarity in fatigue may have been the effect of long term exercise training required by athletes for participation at Paralympic level competition, innate athletic ability, as well as the study of athletes with minimal CP.

The finding of an altered pacing strategy in the CP group in the present study may provide insight into the residual effect of CP, and secondary physiological changes as a result of CP, on the body. It has been well documented that individuals with CP are impaired in the regulation of exercise, due to brain damage in certain key areas related to movement, from a young age. These areas are the motor cortex, cerebellum and basal ganglia, which control and regulate movement, and have also been identified as key areas of the brain involved with implementing movement strategies<sup>176</sup>. Further observed in individuals with CP, damage to one of these areas of the brain results in dysfunction of that area in terms of movement strategy and planning. Compensation strategies employed by the brain affected by CP have been observed as a subconscious underuse of the affected limb during functional activities in individuals with CP<sup>177, 178</sup>. Subsequently, these compensation strategies have been identified and improved using constraint-induced movement therapy. This therapy involves the casting and binding of the NA limb of hemiplegic patients with CP, forcing the increased use and improved function of the A limb during fine motor tasks. Indeed, this intervention has prevented neglect of the A limb, and subsequent sole reliance on the NA limb in mildly to severely affected children with hemiplegic CP<sup>177-179</sup>.

During exercise trials, it has been shown that volitional muscle activation is significantly decreased in individuals with CP, despite the availability of abundant motor units. This

provides further evidence that a brain affected by CP does not utilise the full resources available. This supports the finding of conservative pacing strategy utilisation in athletes with CP in the present chapter. It is hypothesised that the findings in the present study are characteristic of heightened conservative regulation in the brain affected by CP, as a protective mechanism. This hypothesis is supported by the research described previously which highlights the compensation of the brain affected by CP<sup>177, 178</sup>.

The EMG findings described in this study (Figure 16) indicate few differences between groups or trials, despite overall performance and pacing differences between the groups. In both AB and CP groups, EMG decreased significantly over time in A and NA ES (Figure 16 A), BF (Figure 16 C), VLO (Figure 16 D) and GC (Figure 16 E) muscles. These findings were expected, as muscle activity has shown to decrease over time in prolonged exercise trials in AB individuals<sup>155, 156, 158, 159</sup>. The CP group displayed higher mean activation of the A VLO during the DEC trial, indicating a possible increased load on the A leg's power generating muscles, in order to maintain performance during the DEC trial. This was possibly also the result of higher running speeds achieved by the CP group in the DEC trial compared to the N-DEC trial.

The findings of this study provide the first novel insight into pacing strategy and exercise performance in individuals with CP. Chapters 3 and 4 showed that athletes with CP do not present with the fatigue resistance shown in studies performed on sedentary children with CP, when externally-paced fatigue was measured. The current study has provided a plausible explanation for the results found in both Chapters 3 and 4. The altered pacing strategy identified in the CP group as a result of deception provides the first evidence that these individuals employ a conservative pacing strategy. This pacing mechanism may have

presented as fatigue resistance in previous research, as both fatigue resistance and conservative pacing strategies present as lower power output over a longer period of time.

The findings of this study might have clinical implications for the use of different training and pacing strategies by coaches and exercise physiologists in order to maximise exercise performance in elite athletes with CP. Furthermore, evaluation of coaching interventions that utilise pacing manipulation are required to identify whether deception during training may influence performance during competition.

The findings of the present study need to be verified by further research on pacing and maximal performance in sedentary and athletic individuals with CP. The small sample size in the CP group, the use of a 20 m shuttle running test instead of a continuous 400 m track, and the use of AB hockey athletes instead of AB track and field athletes are limitations of the current study. Specifically, the use of a 400 m continuous track would allow for a better real-life comparison of pacing between the groups, but 20 m shuttles were used due to availability of equipment, the ability to control for environmental factors (wind, heat) and the wish to test both groups of athletes in a relatively unfamiliar task, as repeated 200m bouts on a track would be highly familiar to the track and field trained CP group. The results should be viewed as an initial insight into possible mechanisms underlying exercise performance in a sample of minimally-affected athletes. Further research using larger samples is required to provide additional evidence for the utilisation of subconscious pacing strategies in individuals with CP.

## CONCLUSION

The present chapter provides evidence for the use of a conservative pacing strategy as an underlying factor in exercise performance and fatigue profiles in individuals with CP. This study has identified altered pacing strategies during maximal self-paced shuttle running exercise using elite Paralympic athletes with CP (classes T37 and T38). Chapter 2 showed similar bone density and fat mass levels between A and NA sides in the CP group, whilst Chapters 3 and 4 identified similar fatigue profiles in athletes with CP and AB athletes. In the individual chapters, it was proposed that athletes with CP represent a group of individuals who have achieved maximum adaptation toward AB levels. The current chapter supported this proposal, but in alignment with the previous chapters, proposed that there may be a ceiling above which adaptation can no longer occur in the athletes with CP. This chapter proposed that individuals with CP may pace exercise conservatively as a heightened conservative regulation response. The present chapter also indicated that conservative pacing strategies may be present in elite, highly functional Paralympic athletes with CP, and showed that there is a residual effect of CP on the body that cannot be physically surpassed, despite the level of physical training or severity of impairment.

## **CHAPTER 6**

### **SUMMARY OF THE THESIS**

**Summary, interpretation, conclusions and clinical  
implications**

## **SUMMARY OF STUDIES INCLUDED IN THIS THESIS**

The present thesis is the first to describe body composition, performance, fatigue and pacing characteristics of elite Paralympic athletes with cerebral palsy (CP). The findings of the thesis are described briefly below. The strengths of this thesis include the first description of bone mineral density, in-depth physiology and maximal exercise performance of elite Paralympic athletes with CP. Furthermore, this thesis was the first to investigate neuromuscular characteristics and pacing strategies employed by this group of elite athletes.

Chapter 2 investigated in-depth body composition of six elite Paralympic athletes with CP. It was found that there was no difference in bone mineral density and fat mass between affected (A) and non-affected (NA) sides of these athletes. However, there was significantly less fat free soft tissue mass on the A side. This was the first study to describe in-depth body composition of elite athletes with CP, and contradicted previous research which showed large deficits in bone mineral density in sedentary children with CP compared to able-bodied (AB) controls. It was proposed that the high levels of athletic training undertaken by the current sample of athletes may have resulted in the observations documented in this study. It was deemed important to investigate physical function and exercise performance in a similar sample of athletes to lend further understanding to these observations.

Consequently, Chapter 3 described performance and neuromuscular characteristics of five elite Paralympic athletes with CP. Thirty second Wingate sprint cycling performance was 10% lower in five athletes with CP, compared to a group of 16 age and gender matched elite field hockey, rugby and soccer AB athletes. However, fatigue indices and muscle activity over the trials were similar between the groups. These findings were in contrast to those

documented in the existing scientific literature conducted in young sedentary populations, which stated that children with CP present with fatigue resistance as a result of muscle weakness. The similarity in fatigue as well as the closer matching in performance was attributed to long term high level athletic training undertaken by these athletes, which allowed them to achieve maximal adaptation toward the physiology of AB athletes. However, atypical neuromuscular patterns were documented in the athletes with CP, indicating that there was still a marked effect of the condition on the neuromuscular physiology of these individuals.

Chapter 4 provided further evidence for similar fatigability between elite Paralympic athletes with CP and 13 elite AB field hockey athletes, by evaluating explosive sprinting and jumping performance before and after an externally paced adapted multistage shuttle run test to exhaustion. The CP group presented with compromised performance in the sprint test (6%) and vertical jumps off the A leg (33%) and off both legs (18%), while vertical jump performance off the NA leg was equal to those achieved by the AB control group. Indeed, the groups displayed similar fatigue profiles over the course of the testing protocol, which supported the findings in Chapter 3. However, asymmetry with regard to power production and neuromuscular activation between A and NA legs indicated that there may be a ceiling toward which athletes with CP can adapt.

Chapter 5 evaluated pacing strategies employed by the athletes with CP compared to the AB control group, during a distance deceived shuttle running trial. The CP group of six athletes ran 10% slower in all trials compared to 12 elite AB field hockey athletes, which was similar to the self-paced deficit in performance observed in Chapter 3. With the use of distance deception, however, pacing strategies employed by both groups were able to be assessed. The CP group presented with altered pacing strategies in the deceived trial,

indicating a conservative pacing strategy. The findings described in Chapter 5 provide a possible explanation for the differences in performance despite the similarity in fatigue observed between the athletes with CP and AB athletes in Chapters 3 and 4. It was proposed that individuals with CP may pace exercise conservatively as a result of the residual effects of CP on the body.

The findings of Chapters 2 - 5 of the current thesis as well as the findings of previous research discussed in Chapter 1 lend insight into: 1) the physiological and functional capacity of elite Paralympic athletes with CP, and 2) the effect of elite level exercise training on this physiology and function in individuals with CP. The main implications of the current thesis will be discussed subsequently.

## **INTERPRETATION AND IMPLICATIONS OF THIS THESIS**

### **Social integration and sport in individuals with disabilities, and the growth of the Paralympic Movement**

It is widely acknowledged that individuals with a disability have, for the most part, suffered prejudice and segregation within society for thousands of years. There is evidence that ancient Greek and Roman societies viewed individuals with disabilities as inferior humans, resulting in the hiding of adults and children with disabilities away from society in unknown locations <sup>180</sup>. During the Middle Ages, individuals with disabilities were tortured or killed to rid them of what was believed to be possession by evil spirits <sup>181</sup>. Furthermore, supporters of Social Darwinism in the 19<sup>th</sup> century believed that individuals with disabilities should not be state-supported, and argued that preservation of “the unfit” would impede human evolution, deeming even sterilisation appropriate as a form of halting heredity of certain disabilities <sup>182</sup>.

Social isolation and sporting exclusion of individuals with disabilities continued until the 20<sup>th</sup> century. Indeed, public attitudes and sports policies regarding sport and disability only saw progress from the mid-20<sup>th</sup> century, when a sports competition was held alongside the 1948 Olympic Games for individuals with spinal cord injuries<sup>8</sup>. Since then, sport and social integration for individuals with disabilities has made significant progress through the Paralympic Movement and affiliated movements. As an indicator of the Movement's success, the Paralympic Games have grown over the years, culminating in the 2012 London Paralympic Games, which boasted 4176 athletes from 164 countries competing in many sports across many classes of disability<sup>183</sup>. Although the Paralympic Movement continues to grow in terms of performance (witnessed by the still growing number of athletes involved in the sports, as well as continuous breaking of world records) professionalization of athletes with disabilities is becoming more common, allowing for comparable commercial athlete status between Olympic and Paralympic athletes (as seen by equal medal winnings and endorsement deals). Thus, individuals with disabilities have, in a relatively short period of time, gained elite status in the world of international sports participation.

Similarly, classification of impairment in Paralympic sport has seen significant change since the inception of the Paralympic Movement. Individuals with disabilities were initially classified medically, before the commencement of functional and ability-specific testing for classification. Some impairments, such as visual impairment, are still based on medical classification, whereas most physical impairments are classified into classes of competition either categorically (depending on specific factors affecting function and performance) or functionally (depending on functional ability of the individual)<sup>11</sup>.

### **Cerebral palsy and the current thesis**

With the growth of the Paralympic Movement and progression of sport participation in individuals with disabilities, participation in elite sporting events by individuals with CP has also become increasingly prevalent. Athletes with CP participate in mainstream sporting events including athletics, swimming and cycling as well as specific sports including boccia and 7-a-side football<sup>9</sup>.

With the rapid growth of the movement however, especially in areas that do not require technological advancement (such as lower limb running prostheses or sports wheelchairs<sup>184</sup>), the progression of science is sometimes slower than that of the practice. In particular, the majority of research in individuals with CP still focuses on quality of life in sedentary children<sup>37, 185, 186</sup>, with little research on functional capacities of either adults or athletes with CP<sup>25, 114, 187, 188</sup>. The physical inactivity in these earlier studies has to be acknowledged as a major contributing factor to the significant deficits reported in these studies, which document up to a 70% deficit in whole body functional performance in a variety of exercise physiology systems<sup>17, 29, 48, 49, 112</sup>.

The studies described in this thesis are the first of their kind to describe in-depth physiology and functional performance in elite Paralympic athletes with CP. The investigation of such a group of individuals eliminates the confounding effect of sedentary behaviour on the results observed in the studies. As such, the findings of the present thesis are of particular importance in furthering the understanding of the true effects of CP on the body, as well as the effects of long term exercise training in individuals with CP.

The findings of the present thesis can be interpreted in many ways. This thesis, having been drawn from anecdotal, clinical and empirical areas, would hold varying levels of importance,

and different primary findings, depending on the background of the reader. Therefore, the findings of this thesis could hold different implications for a sports scientist compared to a sports physician or a coach. Some of these areas of interest may include: 1) athlete development, 2) brain regulation in CP, 3) high performance sports physiology and biomechanics in athletes with disabilities, or 4) muscle activity in individuals with CP. However, the recurring hypothesis throughout the thesis which suggests that the findings of this thesis occurred as a result of long term exercise training holds significant potential for the lives of individuals with CP, more than any one finding in isolation.

### **Implications of long term exercise training**

The use of elite Paralympic athletes in the current thesis provides a valuable insight into the effect of high level, long term exercise training from a young age on the individual with CP. The athletes used in the studies described in Chapters 2 - 5 of this thesis were all highly physically active in AB sports from a young age, before specialising in track and field athletics in their mid-teenage years. They have been specialised in athletics from between four and 12 years, and complete high volumes of training, ranging from six to 20 hours per week.

In Chapter 1 of this thesis, the effect of short term exercise interventions in children with CP was discussed in detail <sup>19, 51, 65</sup>. The very large changes as the result of these interventions indicate that the body with CP is highly adaptable. In fact, it was reported that children with CP had approximately twice the improvement in functional performance compared to adult studies, further indicating that adaptation at a young age is easier to achieve <sup>44, 189</sup>. Conversely, it was found that detraining after an exercise intervention was significantly marked after only four weeks of inactivity, compared to the effects of detraining in AB

individuals<sup>190, 191</sup>. This finding indicates that although high levels of adaptation are possible, the positive effects of exercise are lost after a short while if the exercise is not continued.

Chapters 2 - 5 provide the first evidence that long term exercise training interventions may provide more benefit to individuals with CP than previously believed. The similarity in fat mass and bone mineral density between A and NA sides reported in Chapter 2; the performance deficits of only 6 – 10% reported in Chapters 3 – 5; and the similarity in fatigue profiles between athletes with CP and AB athletes reported in Chapter 3 and 4 represent novel findings. These findings indicate that long term high level exercise training may positively influence the physiology of the individual with CP toward maximum level of adaptation possible. That is, through exercise, it may be possible for individuals with CP to restrict the secondary effects of physical inactivity and CP on the body.

The acceptance of this hypothesis is arguably the most important finding of this thesis, and holds the potential to radically change the lives of individuals with CP. Clinically, the change in attitude in the treatment of children affected by CP should shift from minimal play-based functional therapy toward higher levels of exercise training from a young age. This new perspective of exercise training would allow these children the opportunity to achieve the full potential of their body. With further research in this area, including longitudinal research on high dose exercise training in children with CP, it is possible that this thesis may have provided the first evidence to change standard exercise prescription and treatment of children and adolescents within this group of individuals with disabilities.

### **Implications of residual effects of cerebral palsy**

In writing this thesis, two main themes emerged in each chapter. Firstly, and as discussed above, there seemed to be a significant effect of long term athletic training, which could be

assessed by using elite Paralympic athletes. Secondly, and sometimes requiring in-depth investigation, a residual effect of CP was observed in these individuals, which high levels of elite athletic training could not surpass. That is, there appears to be a ceiling of impairment towards which an individual can adapt, but no further. The lower fat free soft tissue mass on the A side reported in Chapter 2; the muscle activation irregularities observed in Chapter 3; the asymmetrical impairment in performance on the A side reported in Chapter 4; as well as the altered pacing strategy utilisation reported in Chapter 5 indicate that the effects of CP on the body are present even in highly trained individuals.

These findings represent the “weakest link” in the chain of the elite athlete. And as such, to improve these areas of deficit is to improve the whole body. In the clearest example of residual impairment affecting function, Chapter 4 described the vertical jump performance impairment as 33% whilst jumping off the A leg, 18% whilst jumping off both legs, whilst the NA leg was observed as displaying no performance deficit compared to vertical jump performances of elite AB field hockey athletes. Thus, although there were differences in the jumping protocols of both legs and single legs, the effect of the A leg’s impairment on the activity generated by both legs was clear. It is therefore logical to assume that should one increase the capacity of the A leg, the capacity of the activity generated by both legs would improve.

In musculoskeletal rehabilitation, it is commonly taught that progression of rehabilitation is dictated by the progress of the affected/injured limb<sup>14</sup>. For example, weight added onto a leg press machine, is added at the A leg’s strength threshold, and increased as the A leg improves in strength. The problem with this approach in individuals with CP is that the A leg will be permanently impaired to a certain degree, whilst the NA leg, as shown in this thesis, can reach the same performance capacity of elite National and International field hockey

players. To train both legs at the progress of the A leg would therefore impede progress of the whole body.

The athletes tested in the current thesis already train A and NA legs individually, thus the result of comparable power capacities between their NA leg and AB athletes' legs was possible. However, the athletes involved in this thesis include elite Paralympic athletes who receive evidence-based training interventions by their coach. Standard rehabilitation practise in athletes with CP and more particularly, individuals with CP in the general population is to always perform activity using both limbs, as described above. Should a movement away from this training approach occur, significantly more improvement might be observed in the A limbs, and subsequently the whole body might improve.

It must be noted that to train both legs individually would ultimately increase the asymmetry between A and NA legs as observed in the elite cohort of athletes with CP used in this thesis, if they are working at their own maximal capacity. This needs to be considered and factored into rehabilitation programs devised by the clinician, to avoid increasing compensatory mechanisms in these individuals.

Another implication of these findings, however, is the acceptance of a residual impairment in individuals with CP. The athletes used in this thesis train both legs individually, at very high levels, and yet these asymmetries are maintained. Empirically, it could be said that the impairment witnessed in this group of athletes (such as the atypical muscle activation findings observed in Chapter 3 and asymmetries observed in Chapters 2 and 4) represents the true effect of CP on their bodies. Had this group not been elite athletes, it is reasonable to suggest that the impairments observed would have been significantly more marked, much like the impairments reported in the literature using sedentary adults with CP. The

observation of the actual effect of CP on the body, without confounding factors, is of great importance in the understanding of the true impairment associated with CP.

Interestingly, these observations may also have implications for the Paralympic classification process, where regular classification (as opposed to confirmed classification status of athletes with CP) might be able to identify the true effect of CP on the body, and classify athletes accordingly, over many years of elite level athletic training. The relationship between the findings of this thesis and possible implications for classification require further research.

### **Implications of central deficit and conservative pacing in cerebral palsy**

The current thesis provides insight into the relationship between fatigue resistance and conservative pacing strategies in individuals with CP. Previous research in children proposed that individuals with CP are fatigue resistant as a result of significant muscle strength deficits<sup>17, 81-83</sup>. This fatigue resistance was reported as lower force output in the CP group, although the lower force was maintained over a longer period of time during exercise trials. In the present thesis, athletes with CP represent a group of individuals who have possibly avoided these strength deficits through athletic training, and thus present with similar levels of fatigue to elite AB field hockey athletes (Chapter 3 and 4). However, in Chapter 5, pacing strategies employed by athletes with CP and AB athletes were investigated in isolation with the use of distance deception. It was observed that the athletes with CP paced the exercise conservatively compared to AB athletes. Interestingly, this conservative pacing strategy presents as maintenance of lower power output over the trial, much like the fatigue resistance reported in the earlier studies. Therefore, it is tempting to speculate that the maintenance of lower power output observed in individuals with CP may not be the previously proposed fatigue resistance, but rather the utilisation of a subconscious

conservative pacing strategy, as a result of the residual effect of CP on the bodies of these individuals.

This hypothesis has practical implications for athletes with CP wishing to improve athletic performance. The use of distance and time deception as a coaching technique improves performance by forcing the athlete to push beyond comfortable boundaries with respect to exertion and encourages them to work into their physiological reserve<sup>156, 192</sup>. This is an important aspect of training, as training intensity, although perceived to be maximal by the athlete during training, can never fully recreate the unique conditions and intensity of elite sports competition. In athletes with CP, this reserve may be harder to access than AB athletes, requiring higher threshold training in these individuals. However, access to this reserve in athletes with CP may significantly influence their competitive edge, and even possibly limit the adverse effects of CP experienced toward the end of a race, which is often observed as “tying up” with spasticity in the legs. Indeed, “tying up” toward the finish line in athletes with CP may be the result of a conservative pacing strategy limiting access to the reserve capacity in these athletes. Thus, the use of deception by the coach during training may have significant effects on outcome with respect to competition. This is particularly true for events longer than 100 m, as the 100 m event is performed as an all-out maximal event (despite subconscious pacing during the event), whereas the 200 m or 400 m events require conscious pacing strategies during performance, which can be altered during training of these athletes.

### **Application of electromyography as a tool in the rehabilitation and training of individuals with cerebral palsy**

The inclusion of muscle activity (EMG) activity in the present thesis was initially designed to measure central output indirectly through volitional muscle activation. However, the findings

of irregularities in specific muscle activation in Chapter 3 provided in-depth insight into the dysfunctional muscle activation in these individuals. The identification of these irregularities using EMG has clinical implications for the use of this technique in the identification and rehabilitation of clinical neuromuscular presentations of individuals with CP.

EMG biofeedback has gained popularity in recent years as a rehabilitative tool<sup>62, 66, 193</sup>. In biofeedback, the patient is able to monitor real time muscle activation during specific rehabilitation in order to optimise therapy. In individuals with neuromuscular impairment, including those with CP, biofeedback may provide important information during rehabilitation which cannot be gleaned from traditional rehabilitation alone. Indeed, the irregularities in co-activation timing of both stabilising erector spinae muscles and power producing vastus lateralis and biceps femoris muscles, as well as the irregular atypical motor drive (or spasticity) of the biceps femoris muscles observed in three participants with CP (Figure 5-6 in Chapter 3) provide clear examples of areas requiring attention during clinical management of specific athletes.

## **LIMITATIONS OF THIS THESIS**

### **The use of a small sample size**

An important limitation of the current thesis was the use of a small sample of athletes with CP. The use of a small sample size increases the possibility of a Type 2 statistical error<sup>194</sup>, as well as limits the extrapolation of findings in each study to the greater population of individuals with CP. However, this thesis required a group of elite Paralympic athletes with CP in South Africa, which represents a finite pool from which to draw participants. This thesis recruited every athlete possible in the country that met the requirements for inclusion.

### **The use of T37 and T38 athletes with cerebral palsy**

It must be acknowledged that the use of athletes who fall into classes T37 and T38 of the current athletics classification represent athletes who are minimally affected with CP<sup>12, 197</sup>. The decision to investigate this selection of athletes was twofold: 1) this thesis aimed to examine function in highly performing ambulant individuals with CP, and 2) T37 and T38 sprinting athletes were available for the studies of this thesis through their coach. It may be suggested that athletes who fit the hemiplegic profiles associated with neuromuscular conditions like CP (classes T35 and T36) be used in the CP sample. However, the inclusion of T35 or T36 athletes would have created a heterogeneous group of athletes with CP, as the impairment observed in these groups is significantly higher than in T37 and T38. Thus the decision to select athletes from T37 and T38 classes was also made on the homogeneity of the two classes in levels of impairment.

Additionally, athletes who are classified as T37 and T38 fall into Level I – II in the Gross Motor Function Classification System, representing ambulant individuals who are self-reliant and able to perform physically demanding tasks as well as activities of daily living<sup>7, 10, 170</sup>. With this in mind, only studies using individuals classified as Level I or II in the gross motor function classification system were used as references in this thesis. This is discussed in detail in Chapter 1 and declared in every chapter of this thesis. Therefore, although the findings of the present thesis may be characteristic of using minimally affected athletes with CP, it was controlled for as much as possible by only including research conducted on similarly affected individuals, upon which conclusions were made.

### **Selection of the control group**

The current thesis could have been improved by using an AB control group comprising track and field athletes. As discussed above, the CP group was drawn from a finite number of

athletes with CP. Similarly, the number of AB elite sprinters in South Africa is very small, with only National athletes available, who reside in a different province to the group of Paralympic athletes described in this thesis. Furthermore, in an effort to match performance as closely as possible between the CP and AB groups, sprint specific athletes in running sports were used instead of track and field athletes. The available AB track and field athletes in South Africa were difficult to compare in terms of training volume, performance times or level of performance to the current sample of athletes with CP. Further research is required using an AB track and field athletics control group.

### **Study design limitations**

#### *Equipment specific testing limitations*

Certain limitations of the current thesis were noted with regard to equipment. In Chapter 3, the use of bilateral stroke-by-stroke power output on the cycle ergometer would have provided greater understanding of the role of A and NA legs in power production. Similarly, the use of floor imbedded force plates in Chapter 4 would have allowed for further investigation into the force production/muscle activation ratio in the vertical jump tests on the A and NA legs. In addition, the use of a 400 m continuous track instead of 20 m running field in Chapter 5 would have allowed for a better comparison to real life track running performances. These variables were not investigated due to unavailability of equipment required for the testing, but would be important to use in the planning of future studies.

#### *Cerebral palsy specific testing limitations*

The use of surface EMG in this thesis was both an innovation and a limitation. Surface electromyography is an indirect measure of volitional muscle activation, and as such, is unable to provide actual central output from the brain<sup>195, 196</sup>. Central output would have been beneficial to examine, as individuals with CP are characterised by a decrease in

central output, and the direct study of this would have provided important insight into the brain's muscle activation capacity in this group of athletes. Similarly, volitional muscle activation is limited in individuals with CP, resulting in lower maximum muscle activation on the A side<sup>17, 76, 86</sup>. Therefore, the use of transcranial magnetic stimulation (maximum central activation) or M-wave muscle stimulation (maximum muscle activation) would have provided more insight into neuromuscular aspects of CP. These methods were not used in the present thesis as the equipment was not available. However, in the planning of future studies, it is important to note that: 1) M-wave stimulation is an aversive procedure, particularly to elite athletes, and 2) both transcranial magnetic stimulation and M-wave stimulation require a specific stationary testing protocol.

Another limitation in the current thesis was that we chose not to include muscle biopsies. This thesis suggests that one of the possible reasons for near matching in performance and similarities in fatigue indices between athletes with CP and AB athletes is the preferred utilisation of Type II muscle fibres over Type I muscle fibre recruitment, as a result of athletic training. Muscle biopsies of the A and NA sides in the athletes with CP would be able to provide evidence for this hypothesis. However, due to the aversive nature of the procedure, it was not performed on this group of elite athletes.

The lack of on-site testing of spasticity on the A and NA sides in the athletes with CP using the modified Ashworth Spasticity Scale is a further limitation in this thesis. Measurement of spasticity would have provided insight into the effect of spasticity on the results of the present thesis, especially those of altered pacing after deception in Chapter 5. Spasticity was not measured for two main reasons: 1) a qualified professional experienced enough to provide reliable results on the highly variable Ashworth scale was not available for testing,

and 2) much like M-wave stimulation, the testing protocols of the studies included did not allow for the testing of spasticity testing immediately prior and post exercise testing.

## **SUGGESTIONS FOR FUTURE RESEARCH**

The main areas of future research are related to the limitations discussed in the previous section. For example, studies using testing methods not included in the present thesis would constitute the next step in providing further insight into the findings documented in the present thesis. Particularly, given the similarity in fatigue profiles between elite athletes with CP and AB athletes in this thesis, M-wave stimulation and muscle biopsies would provide further valuable insight into the influence of elite athletic training on muscle development and activation in this group of athletes.

The most important area of future research required is the use of longitudinal studies in larger athlete numbers and additional control groups. Furthermore, it is suggested that future research include the testing of three additional sample groups, namely sedentary adults with CP, sedentary AB individuals and a track and field elite athlete control group. In addition, longitudinal research conducted in young individuals with CP is required. These areas of future research would provide new insights into the effect of elite exercise training from a young age on physiology and function in individuals with CP, by controlling for the possible confounding effects of mild CP and matching to a control group not involved in track and field athletics in the current thesis. Finally, the investigation of training regimens utilised by elite athletes with CP may provide insight into specific high performance exercise training in this population, and its effect on physiology, function and injury prevention from a young age.

## CONCLUSION

The present thesis aimed to describe body composition, exercise performance, fatigue and pacing of elite Paralympic athletes with CP. This thesis provides novel findings of: 1) body composition and bone mineral density similarities between A and NA sides in athletes with CP compared to AB athletes, which contrasts the existing literature which reports a significant deficit on the A side of individuals with CP, 2) less deficit in performance in athletes with CP compared to age and gender matched AB athletes, than stated in research conducted using sedentary children with CP, 3) a similar fatigue response between athletes with CP and AB athletes, 4) the adoption of a conservative pacing strategy in athletes with CP, as the result of deception and 5) the residual effects of the condition in this group of elite athletes with CP.

This thesis provides the first evidence for the considerable effect of long term high load exercise training from a young age on physiology and functional performance in a group of elite Paralympic athletes with CP. The novel findings of the present thesis have significant implications for professionals working in the areas of clinical management, coaching and scientific research in individuals with CP.

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# APPENDIX

## APPENDIX I

### Human Research Ethics Committee Ethical Approval Letter



UNIVERSITY OF CAPE TOWN

Health Sciences Faculty  
Faculty of Health Sciences Research Ethics Committee  
Room E52-24 Groote Schuur Hospital Old Main Building  
Observatory 7925  
Telephone [021] 406 6338 • Facsimile [021] 406 6411  
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11 May 2011

HREC REF: 156/2011

Ms P Runciman  
c/o Dr R Tucker  
Sports Science Institute  
Boundry Road  
Newlands

Dear Ms Runciman

**PROJECT TITLE: PERFORMANCE AND FATIGUE CHARACTERISTICS OF ATHLETES WITH CEREBRAL PALSY.**

Thank you for your comprehensive response.

It is a pleasure to inform you that the Ethics Committee has formally approved the above-mentioned study.

**Approval is granted for one year till the 15 May 2012.**

Please submit a progress form, using the standardised Annual Report Form (FHS016), if the study continues beyond the approval period. Please submit a Standard Closure form (FHS010) if the study is completed within the approval period.

Please note that the ongoing ethical conduct of the study remains the responsibility of the principal investigator.

**Please quote the REC. REF in all your correspondence.**

Yours sincerely

**PROFESSOR M BLOCKMAN**  
**CHAIRPERSON, HSF HUMAN ETHICS**

Federal Wide Assurance Number: FWA00001637.  
Institutional Review Board (IRB) number: IRB00001938  
sAriefdien

## APPENDIX II

### Participant Informed Consent

**Performance and fatigue characteristics of athletes with cerebral palsy**  
***INFORMED CONSENT***

I have read and understood the information sheet provided to me by the investigator. The procedures and risks have also been explained verbally to me and I have had the opportunity to ask questions about the study. The exact procedures used in the trial have been thoroughly explained to me. I understand the nature, procedures and purpose of this study. This study has received Ethics Approval from UCT (Ref: 156/2011).

I understand that I am free to ask questions and withdraw from the study at any time, and without reason, at any time during the testing process. I agree to the information gathered in this study to be used for research, in a statistical format only. None of my personal information may be shared and I am aware that I will be receiving a gift voucher for my participation in this study.

I agree to participate in this research study of the UCT/MRC Research Unit for Exercise Science and Sports Medicine.

Name of participant: .....

Signature: .....

Name of investigator: .....

Signature: .....

**Principal investigator:**

Phoebe Runciman  
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**Chairman of Ethics Committee:**

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