REHABILITATION IN CEREBRAL PALSY
EVALUATION OF PHYSIOTHERAPY INTERVENTION AFTER MULTI-LEVEL ORTHOPAEDIC SURGERY

A DISSERTATION SUBMITTED TO THE
UNIVERSITY OF OXFORD

IN PARTIAL FULFILLMENT OF THE REQUIREMENTS
FOR THE DEGREE OF
DOCTOR OF PHILOSOPHY

By

MARIA SENIOROU

WOLFSON COLLEGE
OXFORD

and

THE NUFFIELD DEPARTMENT OF ORTHOPAEDIC SURGERY
FACULTY OF CLINICAL MEDICINE

RADCLIFFE SCIENCE LIBRARY OXFORD
DEPOSITED
- 3 DEC 2007
THESIS

Abstract

Rehabilitation in Cerebral Palsy: Evaluation of Physiotherapy Intervention after Multi-Level Orthopaedic Surgery

Maria Seniorou — Faculty of Clinical Medicine

Wolfson College — Trinity Term 2006

Complex orthopaedic surgery is often indicated in the management of deformity in children with spastic diplegic cerebral palsy (CP) and a long rehabilitation programme is important for a successful outcome. However, the frequency and content of physiotherapy treatment following surgery varies between centres. This thesis aimed at providing a scientific basis for post-operative rehabilitation following multi-level surgery in children with CP.

The effect of muscle weakness on function in patients with CP is recognised. However, the short and long-term impact of multi-level orthopaedic surgery on muscle strength is unclear. Strength changes would have implications for both surgical and conservative treatment planning. The objectives of this study were to establish a better understanding of the impact of surgery on muscle strength, gait and function, as well as the natural history of weakness in CP. Furthermore, the study aimed to clarify whether physiotherapy, focused on resistance strengthening techniques, was more effective than routine physiotherapy.

The reliability of a protocol for measuring muscle strength in lower limb muscle groups in this population was firstly established. A pilot study of 10 children with a diagnosis of spastic diplegic CP and healthy counterparts demonstrated a reliable strength testing protocol. The main study of 20 diplegic children who underwent multi-level surgery showed that despite improvements in gait parameters, significant loss of strength in lower limb muscle groups and gross motor function persisted at six months. The value of intensive physiotherapy was assessed in a randomised controlled trial six months after surgery. Advantages of resistance training over active exercise were demonstrated. Assessment at one year showed that surgical patients preserved the post-physiotherapy strength and function gains but these did not reach the pre-operative values. Another group of 10 diplegic children who received routine physiotherapy but no surgical treatment for 12 months showed significant deterioration of their gait.

The rate of deterioration demonstrated in conservatively treated diplegic patients and the degree of weakness caused by surgery, should inform parents and clinicians during decisions regarding surgery. Future research in multi-level surgery should include specific strength assessment protocols. The results from the randomised clinical trial showed that overall intensive strengthening regimes after multi-level surgery are beneficial. This finding would have significant implications on managing resources and designing appropriate rehabilitation programmes after multi-level orthopaedic surgery.

Acknowledgments

I am grateful to a number of people in connection with this thesis. I am indebted to my supervisor, Mr Tim N. Theologis, who has been very kind and supportive throughout. He had sufficient faith to encourage me to continue and complete this work. Particular thanks go to Ms Nicky Thompson, co-supervisor, whose expertise and advice have been very helpful. Financial support for this project was provided by the Oxfordshire Health Services Research Committee and the Wishbone Trust while my studies in Oxford were kindly funded by the Ismene Fitch Scholarship.

I thank all those who initially provided me with the opportunity to start my research degree in Oxford: Professor Dimitrios Glaros, who shared with me his vision for the future of rehabilitation medicine in Greece, Dr John Ashton, Dr Martin Francis, Dr John O'Connor, Professor John Kenwright and all the people in Wolfson College who made my admission to the University possible and my life in Oxford worth remembering.

I am grateful for the help that many people at the Nuffield Orthopaedic Centre have given me over the years. Many thanks to Marian, Nick, Sue, Cindy, Helen, Mike, Kathryn, Suzie, Amy and Ritchie. Special thanks go to Julie whose analytical brain and kind nature made tough times easier to bear.

Most of all I would like to thank my family. They have always let me believe in my dreams and supported me in every way. The love and support of my husband, Paul, and his many helpful suggestions will always be deeply appreciated. This thesis is dedicated to them and to my baby boy Nicholas.

Table of Contents

Abstract i
Acknowledgments ii
Table of Contents iii
List of Tables, Pictures & Graphs vii
SECTION ONE xv
Chapter One 16
1. Introduction 16
1.1 Research Questions 16
1.2 Hypothesis 18
Chapter Two 19
2. Cerebral Palsy 19
2.1 What is Cerebral Palsy? 19
2.1.1 Definition 19
2.1.2 Epidemiology 21
2.2 Aetiology of Cerebral Palsy 22
2.3 Classification 24
2.4 Diagnosis of Cerebral Palsy 26
2.5 Natural history 28
2.6 Principles of management in Cerebral Palsy 30
2.6.1. Treatment modalities 30
2.7 Summary 32
Chapter Three 33
3. Muscle weakness in Cerebral Palsy 33
3.1 The diplegic child 33
3.1.1 Pathogenesis and general characteristics 33
3.1.2 Neurophysiologic basis of spasticity 35
3.1.3 Alterations in muscle function 37
3.1.3.1 Physiology of muscle contraction 37
3.1.3.2 Histopathology of muscle in CP 40
3.2 Muscle weakness and strength profiles in CP 42
3.3 Muscle weakness and surgery in CP 44
3.3.1 Multi-level orthopaedic surgery in spastic diplegia 46
3.4 Physiotherapy management in CP 53
3.4.1 Therapeutic Exercise: evidence of effectiveness and safety of strength training for children and adolescents with spastic cerebral palsy 57
3.5 Summary 61
Chapter Four 62

4. The diplegic gait 62

4.1 The diplegic gait 63
4.1.1 Assessment methods 65
4.1.1.1 Gait Analysis 65
4.1.1.1.1 Normal Gait 65
4.1.1.1.2 Cerebral Palsy gait 68
4.1.1.1.3 Clinical gait analysis 69
4.1.1.1.4 Clinical gait analysis and its role in treatment decision-making 73
4.1.1.2 Muscle strength assessment 74
4.1.1.2.1 Dynamometry 75
4.1.1.2.2 Reliability of strength testing 79
4.1.1.3 Measures of function 82

4.2 Summary 84

4.3 Section Two outline 84
4.3.1 Synopsis of literature review 85
4.3.2 Overview of the study 85

SECTION TWO 87

Chapter Five 88

5. Reliability of strength testing protocol 88

5.1 Design of the study 90
5.1.1 Subjects 90
5.1.2 Method 91
5.1.2.1 Choice of muscle group 92
5.1.2.2 Choice of strength testing positions 93
5.1.2.3 Procedure 95
5.1.2.3.1 Preparation for the muscle test 96
5.1.2.3.2 Dynamometry 97
5.1.2.3.3 Electromyography 104
5.1.3 Analysis 106
5.1.3.1 Processing of data 106
5.1.3.2 Reliability 107
5.1.3.2.1 Within - subject standard deviation 107
5.1.3.2.2 Intraclass Correlation Coefficient 108

5.2 Results 109

5.3 Discussion 114

5.4 Summary 116

Chapter Six 117

6. Changes in strength, function and gait six months after multi-level orthopaedic surgery 117

6.1 Design of the study 119

6.2 Subjects 119

6.3 Operation and therapy details 120
6.3.1 Immediate post-operative physiotherapy 122

6.4 Method 122
6.4.1 Outcome measures 123
6.4.2 Choice of variables 123
6.4.3 Testing session outline 125
6.4.3.1 Gait Analysis and dynamic electromyography 126
6.4.3.2 Digital dynamometry 128

6.4.3.3 Gross motor function assessment 129
6.4.4 Statistical analysis 130
6.5 Results 130
6.5.1 Comparison of muscle strength before and six months after surgery 131
6.5.2 Gross motor function changes after surgery 133
6.5.3 Lower limb kinematics and kinetics after surgery 136
6.6 Discussion 140
6.7 Summary 142
Chapter Seven 143
7. Comparison of strength training techniques after surgery 143
7.1 Current physiotherapy practice 144
7.1.1 Aim 144
7.1.2 Method 145
7.1.3 Analysis 145
7.1.4 Results 146
7.1.5 Summary 148
7.2 Sample size calculation 149
7.2.1 Sample size calculation 149
7.3 Patients 149
7.3.1 Randomisation 150
7.4 Method 152
7.4.1 Outcome measures 152
7.4.2 Strengthening protocol 153
7.4.3 Statistical analysis 157
7.5 Results 158
7.5.1 Strength results 158
7.5.2 Function results 160
7.5.3 Gait results 164
7.5.4 Changes per GMFCS level 165
7.6 Discussion 166
7.7 Summary 170
Chapter Eight 172
8. Changes of strength, gait and function over one year 172
8.1 Changes in strength, function and gait in the non-surgical group over one year 173
8.1.1 Strength changes 175
8.1.2 Gross motor function changes 176
8.1.3 Kinematic and kinetic changes 178
8.2 Changes in strength, function and gait in the surgical groups over one year 180
8.2.1 Overall changes 181
8.2.2 Comparison between surgical groups 187
8.2.3 Surgical versus non-surgical patients 194
8.3 Discussion 196
8.4 Summary 198
Chapter Nine 200

9. Discussion and synopsis 200

9.1 Introduction 200
9.1.1 Aims and hypotheses of the study 200
9.1.2 Study design 202

9.2 Findings and comparison with the literature 203
9.2.1 Pilot study 203
9.2.2 Findings at six months after multi-level surgery 205
9.2.3 Findings at six months after multi-level surgery and physiotherapy programme 206
9.2.4 Findings at one year 210

9.3 Synopsis of the study findings 212

9.4 Study limitations 214

9.5 Significance of findings 215

9.6 Clinical relevance 216

9.7 Future research 217

Bibliography 219

APPENDICES 241

List of Tables, Pictures & Graphs

Picture 3.1 Skeletal muscle architecture (Wright State University 2005 - access date 26/09/2006) 37

Picture 3.2 Muscle length-tension and force-velocity relationship (Huei Ming Chai 2003 - access date 26/09/06) 40

Picture 3.3 The "birthday syndrome" or repeated operations and rehabilitation 47

Picture 3.4 Rectus Femoris transfer 48

Picture 4.1 The normal gait cycle 67

Picture 4.2 Clinical gait analysis (consent obtained) 71

Picture 4.3 Digital dynamometry set-up at the Oxford Gait Analysis Laboratory (consent obtained) 78

Table 5.1 Participants' characteristics (M=male, F=female, CP=cerebral palsy, H=healthy, R=right, L=left) 91

Picture 5.1 Oxford Gait Analysis Laboratory 92

Table 5.2 Subject and dynamometer positions 95

Picture 5.2 Strength measurement equipment 97

(from left to right: wedge for testing knee extensors at 30°, wedge for testing hip flexors, straps to secure stabilisation and attach the transducer of the dynamometer) 97

Picture 5.3 Hip flexors 99

Picture 5.4 Hip extensors 100

Picture 5.5 Hip abductors 101

Picture 5.6 Knee flexors 103

Picture 5.7 Knee extensors at 90° 104

Picture 5.8 Knee extensors at 30 ° 104

Picture 5.9 Electromyography set up..... 105

Graph 5.1 Normalised muscle strength per muscle group and subject group 109

Table 5.3 Mean peak moments (MPM), normalised mean peak moments in CP and healthy children for all muscle groups 110

Table 5.4 Within-subject standard deviations σ_w, limits (repeatability) and coefficient of variation (CV%) for all subjects and muscle groups..... 110

Table 5.5 Intra-class correlation coefficient (ICC) for CP and Healthy children 111

Graph 5.2 Repeated dynamometric signals during knee extension in a child with CP and a Healthy child..... 112

Graph 5.3 Quadriceps & Hamstrings EMG signals during knee extension at 90°..... 113

Graph 5.4 Comparison of strength assessment between raters. Mean absolute difference (SD) for all muscle groups..... 114

Table 6.1 Age in years/months, sex (M=male, F=female), mass (kg) and functional level (GMFCS)..... 120

Table 6.2 Type of operation per subject right (R), left (L), bilateral (B), femoral derotation osteotomy (FDO), tibial derotation osteotomy (TO), calcaneal lengthening osteotomy (CLO), psoas lengthening (PsL), adductors lengthening (AddL), hamstrings lengthening (HL), rectus femoris transfer (RFTr), gastrocnemius lengthening (GL), tibialis posterior lengthening (TPL), tibialis anterior split transfer (TAT) 121

Table 6.3 Strength, function and gait variables tested and analysed..... 125

Table 6.4: Conventional Marker Set..... 127

Table 6.5 Pre and post –surgery muscle strength (mean, sd) and significance of change ($p < 0.05$)..... 131

Table 6.6 Strength changes between pre and six months post operative assessments by subject and limb. ↑ = an increase, ↓ = a decrease in muscle strength * = statistically significant based on change > $2.77\sigma_{w.}$..... 132

Graph 6.1 Gross motor function before and six months after surgery as % score of the GMFM dimension five (walking, running, jumping)..... 133

Table 6.7 Pre and post surgery GMFM scores % and normalised temporal parameters (mean, sd, paired difference) and significance of change ($p < 0.05$)..... 134

Graph 6.2 % of children in sample at each FAQ walking level before and after surgery 135

Table 6.8 Individual change of FAQ walking level per subject 6 months after surgery 135

Table 6.9 Kinematic variables (mean, sd, paired difference) pre and 6 month post surgery and significance of change ($p < 0.05$)..... 136

Table 6.10 Kinetic variables (mean, sd, paired difference) pre and 6 month post surgery and significance of change ($p < 0.05$) HEM = hip extension moment, HFM = hip flexion moment, KEM = knee extension moment, KFM = knee flexion moment, MPM = max plantarflexion moment, MHAM = max hip abduction moment, MHP = max hip power, MAP = max ankle power 137

Graph 6.3 Kinematic data before and 6 months after multi-level surgery (green band = average normal female, blue line = Right side, red line = Left side, thick line = pre-op, thin line = post-op)..... 138

Graph 6.4 Kinetic data before and 6 months after multi-level surgery (green band = average normal female, blue line = Right side, red line = Left side, thick line = pre-op, thin line = post-op) 139

Graph 7.1 % of physiotherapy provided by parent, school assistant and therapist in all participants 146

Graph 7.2 Type of exercise provided (%) in surgical and non-surgical patients 147

Graph 7.3 Duration of physiotherapy session (min) for all subjects 148

Table 7.1 Treatment allocation in groups B & C based on sex, age and GMFCS level 151

Table 7.2 Pre (I) and post (II) muscle strengthening values (Nm/kg), paired mean differences after physiotherapy in both groups B & C (sig. at $p < 0.05$) 159

Graph 7.4 Hip flexors strength pre (hf2) and post-therapy (hf3) in both groups 159

Table 7.3 Group B & C analysis of co-variance (ANCOVA). The parameter estimates show the effect of the predictor (group) on the dependent variable (i.e.hip flexors Nm/kg e.t.c.). ↓ indicates that given two people with similar pre – physio values, it can be expected that the post – physio value of those in group B to be less than of those in group C. The opposite is valid for ↑. 160

Table 7.4 Inter-group comparisons for GMFM total and dimension E %, normalised walking speed, cadence and step length 161

Table 7.5 Group B & C analysis of co-variance (ANCOVA) for motor function. The parameter estimates show the effect of the predictor (group) on the dependent variable (i.e.N velocity e.t.c.). ↓ indicates that given two people with similar pre – physio values, it can be expected that the post – physio value of those in group B to be less than of those in group C. The opposite is valid for ↑. 162

Graph 7.5 % of children in sample at each FAQ walking level before and after the 6-week physiotherapy programme 163

Table 7.6 Individual change of FAQ walking level per subject before and after the 6 weeks strengthening programme 164

Table 7.7 Kinematic variables (mean, sd, mean difference) before and after the physiotherapy programme in groups B & C ($p < 0.05$) RPT = range pelvic tilt, RHF = range hip flexion, RKF = range knee flexion, RAD = range ankle dorsiflexion, MHES = max hip extension stance, MKES = max knee extension stance, MKFS = max knee flexion swing, KFIC = knee flexion

initial contact, RPO = range pelvic obliquity, RPR = range pelvic rotation, MTR = max thigh rotation 164

Table 7.8 Kinetic variables (mean, sd, mean difference) before and after the physiotherapy programme in groups B & C ($p < 0.05$), HEM = hip extension moment, HFM = hip flexion moment, KEM = knee extension moment, KFM = knee flexion moment, MPM = max plantarflexion moment, MHAM = max hip abduction moment, MHP = max hip power, MAP = max ankle power 165

Graph 7.6 Muscle strength pre and post physiotherapy per GMFCS level 166

Table 8.1 Characteristics of non-surgical subjects comprising Group A.... 174

Table 8.2 Normalised muscle strength (Nm/kg) per muscle group at baseline, six months and one year for group A 175

Graph 8.1 % change of muscle strength in group A between visits. Minus shows improvement; (visit 1= baseline, visit 2 = 6 month, visit 3 = 1 year, HF=hip flexors, HE=hip extensors, HABD=hip abductors, KF=knee flexors, KE90=knee exten 90°, KE30=knee exten 30 °)..... 176

Graph 8.2 % GMFM score per subject per visit in group A, group mean and sd (GMFCS I – II, M = Male, F= Female, N/A= not applicable)..... 177

Graph 8.3 % of subjects in group A per FAQ level atbaseline and 1 year . 178

Table 8.3 Individual change of FAQ walking level per subject 178

Table 8.4 Kinematic variables, mean (SD) per visit, paired difference between baseline and one year, significance of difference in group A 179

Table 8.5 Kinetic variables, mean (SD) per visit, paired difference between baseline and one year, significance of difference in group A..... 179

Table 8.6 Temporal parameters, mean (SD) per visit, paired difference between baseline and one year, significance of difference in group A 180

Table 8.7 Muscle strength changes (mean,sd) at pre-surgery, 6 m post-surgery/pre-physio (6mI), 6 m post-surgery.post-physio (6mII) and 1 year with significance of difference for all surgical subjects (groups B&C)..... 181

Graph 8.4 Muscle strength comparison Nm/kg between visits in all surgical patients (visit 1=pre-op, visit 2=6 m pre-physio, visit 3=6 m post-physio, visit4=1 year) 182

Table 8.8 % change of muscle strength Nm/kg in all surgical patients between different assessment sessions (minus denotes decrease) 183

Table 8.9 Kinematic changes (mean,sd) at pre-surgery, 6 m post-surgery/pre-physio (6mI), 6 m post-surgery.post-physio (6mII) and 1 year with significance of difference for all surgical subjects (groups B&C), RPT = range pelvic tilt, RHF = range hip flexion, RKF = range knee flexion, RAD = range ankle dorsiflexion, MHES = max hip extension stance, MKES = max knee extension stance, MKFS = max knee flexion swing, KFIC = knee flexion initial contact, RPO = range pelvic obliquity, RPR = range pelvic rotation, MTR = max thigh rotation 184

Table 8.10 Kinetic changes (mean,sd) at pre-surgery, 6 m post-surgery/pre-physio (6mI), 6 m post-surgery.post-physio (6mII) and 1 year with significance of difference for all surgical subjects (groups B&C), HEM = hip extension moment, HFM = hip flexion moment, KEM = knee extension moment, KFM = knee flexion moment, MPM = max plantarflexion moment, MHAM = max hip abduction moment, MHP = max hip power, MAP = max ankle power..... 185

Table 8.11 Gross motor function and temporal parameters changes (mean,sd) at pre-surgery, 6 m post-surgery/pre-physio (6mI), 6 m post-surgery.post-physio (6mII) and 1 year with significance of difference for all surgical subjects..... 186

Table 8.12 Individual change of FAQ level per surgical subject per visit.. 186

Graph 8.5 % children in sample per FAQ walking level and visit 187

Table 8.13 Muscle strength changes (mean,sd) at pre-surgery, 6 m post-surgery/pre-physio (6mI), 6 m post-surgery.post-physio (6mII) and 1 year with significance of difference for group B (- denotes negative change or decrease, + denotes positive change or increase) 188

Table 8.14 Muscle strength changes (mean,sd) at pre-surgery, 6 m post-surgery/pre-physio (6mI), 6 m post-surgery.post-physio (6mII) and 1 year with significance of difference for group C (- denotes negative change or decrease, + denotes positive change or increase) 188

Table 8.15 Group B & C analysis of co-variance (ANCOVA) at 1 year. The parameter estimates show the effect of the predictor (group) on the dependent variable (i.e.hip flexors Nm/kg e.t.c.). ↓ indicates that given two people with similar pre – physio values, it can be expected that the post – physio value of those in group B to be less than of those in group C. The opposite is valid for ↑. 189

Table 8.16 Function changes (mean,sd) at pre-surgery, 6 m post-surgery/pre-physio (6mI), 6 m post-surgery.post-physio (6mII) and 1 year with significance of difference for group B..... 190

Table 8.17 Function changes (mean,sd) at pre-surgery, 6 m post-surgery/pre-physio (6mI), 6 m post-surgery.post-physio (6mII) and 1 year with significance of difference for group C..... 190

Table 8.18 Kinematic changes (mean,sd) at pre-surgery, 6 m post-surgery/pre-physio (6mI), 6 m post-surgery.post-physio (6mII) and 1 year with significance of difference for group B 191

Table 8.19 Kinematic changes (mean,sd) at pre-surgery, 6 m post-surgery/pre-physio (6mI), 6 m post-surgery.post-physio (6mII) and 1 year with significance of difference for group C 192

Table 8.20 Kinetic changes (mean,sd) at pre-surgery, 6 m post-surgery/pre-physio (6mI), 6 m post-surgery.post-physio (6mII) and 1 year with significance of difference for group B..... 193

Table 8.21 Kinetic changes (mean,sd) at pre-surgery, 6 m post-surgery/pre-physio (6mI), 6 m post-surgery.post-physio (6mII) and 1 year with significance of difference for group C..... 194

Graph 8.6 GMFM % score changes (total and dimension E) for all study groups and visits..... 194

Graph 8.7 Temporal parameters change in all groups and visits (N = normalised) 195

Graph 8.8 Muscle strength change in all muscle and study groups for all visits 195

Graph 8.9 Strength (Nm/kg) per muscle group for 1 healthy (n) and 1 spastic diplegic (p) child, matched for age and sex. 197

SECTION ONE

Chapter One

1. Introduction

1.1 Research Questions

In Cerebral Palsy (CP) a non-progressive central nervous system (CNS) lesion leads to impaired motor control, balance, muscle tone and strength. Muscle weakness has been recognised as a component of CP but its management is controversial. This dissertation deals with muscle weakness in spastic cerebral palsy, its relation to orthopaedic surgery and to different physiotherapy interventions.

Recent evidence suggests that children with CP are weaker than their peers and weakness compromises function. However, it has been shown that strength and function can be improved using short-term strength training programmes. Despite the increasing research interest in this field, there is still little information regarding the distribution of muscle weakness in CP patients, the effect of surgical treatment on strength and on whether weakness changes over time or not.

The most common type of CP is spastic diplegia where musculoskeletal problems are the main cause of disability. During growth, abnormal muscle forces are applied to the immature skeleton and result in deformities, which affect motor function and walking ability. Conservative treatment often fails to prevent deformity during growth and orthopaedic operations may be necessary.

Over the last 30 years staged orthopaedic surgery has largely been superseded by single-stage multi-level surgery and rehabilitation. This is mainly applicable to ambulant children with CP where gait analysis

technology has been a major contributor in the objective assessment of complex gait abnormalities and evaluation of outcomes.

Muscle-tendon lengthening has a positive biomechanical effect on joint alignment through contracture correction. However, it has been shown that lengthening a tendon reduces the force-generating capacity of a muscle. Clinical studies suggest that recovery of strength after surgery can take up to 24 months; however animal studies suggest that recovery of muscle after surgery can be as quick as six weeks. It is unclear to what extend multi-level orthopaedic surgery affects muscle strength in CP patients and what the time frame of recovery is. There is, therefore, a need to critically evaluate multi-level orthopaedic surgery and its relation to muscle strength and motor function in children with CP, in a prospective controlled manner.

For the results of multi-level surgery to be successful, a long and intensive rehabilitation programme is essential. However, the frequency and content of physiotherapy treatment sessions following surgery vary greatly between centres. In the majority of cases, personal experience instead of evidence-based knowledge appears to be the basis on which post-operative physiotherapy treatment for children with CP is provided. Until recently it was thought that strengthening spastic muscles would increase spasticity. Although studies on strengthening programmes in CP patients have shown that this is not the case, little information exists about optimal post-operative rehabilitation programmes.

The overall objective of this study is to provide a scientific basis for post-operative rehabilitation following multi-level orthopaedic surgery in children with CP. It will be clarified whether physiotherapy focused on resisted strengthening techniques is more effective than conventional physiotherapy, in terms of improvement in gait and motor function. The study will aim at establishing a better understanding of the natural history

of muscle weakness in CP and its impact on the outcome of multi-level orthopaedic surgery. The following research questions will be addressed:

1. What are the strength, function and gait changes following multi-level orthopaedic surgery?
2. What is the effect of a progressive resistance exercise programme on muscle strength, function and gait following multi-level orthopaedic surgery?
3. What are the degree and distribution of muscle weakness and strength changes over time in relation to gait and motor function in ambulant children with spastic diplegia?

1.2 Hypothesis

The experimental hypotheses of this research work are:

1. Multi-level orthopaedic surgery will lead to a decrease in muscle strength and gross motor function at six months post-surgery. Strength and function should recover to the pre-operative level approximately 12 months post-surgery approximately.
2. The implementation of a resistance-strengthening programme of five lower-limb muscle groups six months after multi-level orthopaedic surgery will improve gross motor function, muscle strength and ambulation to a greater degree, compared with an active strengthening programme of the same duration, in two randomised groups of spastic diplegic patients.
3. Muscle strength and gross motor function will deteriorate in a group of conservatively treated patients, over a period of 12 months.

Chapter Two

2. Cerebral Palsy

This thesis involves children with cerebral palsy. In order to demonstrate the need for this research work and to enable the reader to follow the text through it was considered important to include a concise description of the condition.

Cerebral Palsy (CP) is a chronic condition resulting from brain damage. In the past, CP was considered the result of brain trauma during birth, but this is now rarely the case. More often, the problem begins before birth with abnormal brain development or damage sustained from infection or hypoxia. Occasionally, the brain injury takes place after birth. The damaged area of the brain does not heal fully, but does not deteriorate over time either. At present, no treatment for the damage to the brain exists, but with the help of a multi-disciplinary team of health professionals a child with CP can achieve more functional goals throughout development.

2.1 What is Cerebral Palsy?

2.1.1 Definition

In the 1860's, an orthopaedic surgeon named William John Little (1810-1894) described a disorder, which affected young children, causing stiff, "spastic" muscles in their legs and, to a lesser degree, their arms. Little described spastic diplegia, one of several types of cerebral palsy and, was the first to suggest that CP may follow birth injury. The term "Little's disease" (Little 1861-1862) was used for many years as a generic term to include all forms of cerebral palsy. The term does not represent a specific diagnosis, but describes children with non-progressive spasticity, ataxia or

involuntary movements who require complex training and therapy to attain their maximum potential.

The variety of descriptions and classifications of these disorders, by authors like W.J. Little (1861-1862), W. Osler (1889) and S. Freud (1893), reflects the heterogeneity of cerebral palsies with regard to cause, severity, and clinical features. Therefore, the need for a simple and inclusive definition is clear.

In 1958, members of the Little Club prepared a Memorandum, which was presented at the *First International Study Group on Child Neurology and Cerebral Palsy*, held at Oxford (Mac Keith 1959). The Memorandum proposed the following definition for cerebral palsy:

"a persisting qualitative motor disorder appearing before the age of three years, due to a non-progressive interference with development of the brain before the growth of the central nervous system is complete."

The authors recognised that the term "cerebral palsy" was not a precise one because of the many different clinical, aetiological and pathological aspects of the disorder. They proposed that the individual names of diseases within the group should be used as more was learnt about each one of them. However, their belief was that "*cerebral palsy as a term was useful for research purposes, exchange of information and common administrative usage*".

A more recent definition reads as follows:

"an umbrella term covering a group of non-progressive, but often changing, motor impairment syndromes secondary to lesions or anomalies of the brain arising in the early stages of its development" (Mutch 1992).

The definition of the word *non-progressive* remains unclear and measuring the severity of motor impairment is still imprecise, as pointed out by Stanley, Blair and Alberman (Stanley 2000a). Despite these problems, the term *cerebral palsy* is accepted worldwide, which justifies its use in this work.

2.1.2 Epidemiology

Prevalence and *incidence* are measures of occurrence and have both been used over the years to describe how common cerebral palsies are. Prevalence is the number of *existing* cases of a certain condition in a given population. Incidence is the number of *new* cases of a condition, symptom, death, or injury that arise during a specific period of time, such as a year. Since CP is, by definition, a permanent condition, the difference between prevalence and incidence is dependent on mortality and migration rates (Stanley 2000c).

CP is the most common cause of motor deficiency in childhood but remains a relatively rare condition with enormous social and financial impact (Cans 2000). Birth prevalence rates in Western countries were in the range of 2 to 2.5 per 1000 births in the 1980s (Paneth 1984). A UK study, which used a population-based register, showed that the overall CP rate for neonatal survivors fell from 2.5 per 1000 between 1984 and 1986 to 1.7 between 1993 and 1995 (Surman 2003). More recent data from the five currently active UK CP registers showed that the mean annual prevalence rate remained around 2 per 1000 live births for birth years 1986-1996 with a 91% of spastic CP (Surman 2006). In the United States the prevalence rate of CP is similar to other industrialised countries (Biether 1993). A review of term and pre-term birth rates in the United States suggested that the rate of cerebral palsy has not decreased in developed countries over the past 30 years (Clark 2003). Moreover, a recent review study from the Netherlands showed that over the last 40 years the prevalence of CP has risen to well above 2 per 1000 live births (Odding 2006).

2.2 Aetiology of Cerebral Palsy

The fundamental question "*why do children have cerebral palsy?*" is still a difficult one to answer. Thirty years ago the literature supported the view that cerebral palsy was usually a consequence of birth-related injury to the brain. This *"birth trauma"* included either physical trauma or asphyxia (Sussman 1992). Although, physical trauma is rare today due to advances in perinatal care, the exact causative role of birth asphyxia in cerebral palsy is unknown. More recently, unidentified prenatal events, which predispose to preterm birth and/or difficult delivery, have been proposed as a cause of cerebral damage. However, the relation of CP to a perinatal insult or prenatal central nervous system abnormality is still unclear and a matter of debate.

Aetiological research concentrates mainly on identifying pre-, peri- and postnatal risk factors, the combination of which could lead to CP. Such risk factors identified in the prenatal period include infertility, maternal medical problems, infection, prior foetal deaths or siblings with motor disorders (Stanley 2000e). Perinatal asphyxia or lack of oxygen during birth has been linked to central nervous system damage. There is evidence to suggest that 10% of CP in developed countries is associated with intrapartum events (Kuban 1994; Roland 1997). CP that is acquired post-natally is consistently quoted around 10% (Paneth 1984; Paneth 1993). Brain-damaging events in a previously neurologically intact child that could lead to postnatal CP include cerebral infection and head injury in the developing and developed countries respectively (Cans 2004).

Additional risk factors linked with CP include multiple pregnancies, prematurity and low-birth weight. Multiple pregnancies have increased since the 1980s, as a result of the development of infertility treatment and are highly associated with CP (Petterson 1998). Prematurity is also recognised as a significant risk factor. The World Health Organization (WHO) defines *very preterm birth (VPTB)* as birth before 32 completed

weeks of gestation (Chiswick 1986). Spastic diplegia and hemiplegia show stronger associations with VPTB than other types of CP (Kuban 1994). However, there is no clear explanation of this association. Prematurity itself makes the brain more vulnerable to postnatal complications, which may later lead to cerebral palsy (Pharoah 1996). Similarly, there is evidence to suggest that there is an increased risk of CP in small for gestational age infants born at term or moderately pre-term (Jarvis 2003).

Irrespective of its aetiology there are some characteristic pathological findings in CP. The most common and strongly related to spastic diplegia are *periventricular leukomalacia (PVL)* and *intraventricular hemorrhage (IVH)* (Sinha 1990; Kuban 1994). The pathological findings in PVL include necrotic areas in the periventricular white matter next to the external angles of the lateral ventricles of the brain (Armstrong 1974; Roscigno 2002). The fragile brain vasculature of the premature neonate may lead to compromised cerebral blood flow (Bozynski 1988) and intra-cerebral bleeding.

Pathological findings help understand the mechanism of injury however, clear causes cannot be inferred. Despite the fact that many risk factors have been associated with the condition, the majority of children with these risk factors do not develop CP. Stanley et al (2000d) suggested that, *"a causal pathway is a sequence of interdependent events culminating in disease".* Every step of this causal pathway is usually dependent on previous steps. Earlier factors may increase the probability of later factors as, for example, very preterm birth may predispose to cerebral haemorrhage. It is therefore possible that the majority of these risk factors are part of a disease process rather that a single cause. The ultimate goal of aetiological research in CP would be to identify the steps that are involved in this process and plan appropriate preventive strategies.

2.3 Classification

The definition of cerebral palsy is descriptive and the term includes motor disorders that can differ greatly. Therefore, the need for a common classification and a widely agreed terminology has been identified long ago (Mac Keith 1959).

Historically the basis of classification in CP included the clinical observation of the type of muscle tone abnormality and the topography of the motor impairment (Mac Keith 1959; Bax 1964; Bleck 1987; Stanley 2000b).

Abnormalities of muscle tone in cerebral palsy include *spasticity, dyskinesia (athetosis, dystonia, chorea), ataxia* and *hypotonia* (appendix one: *terminology*). In *mixed* forms of CP there is not one pattern that clearly predominates and different types of motor impairment coexist. The mixed type of CP is often a combination of spasticity and athetosis affecting the patient globally (Bleck 1987). Patients with spasticity form the biggest group of CP accounting for 75% to 80% of all cases (Krageloh-Mann 1993; Stanley 2000b). Dyskinetic disorders are less common and present mainly as dystonia or athetosis. Hypotonia is also rare and, in most cases, it evolves into spastic or athetoid CP (Bleck 1987).

The topographical classification of CP is often used according to the anatomical distribution of impairment (Bleck 1987). *Right or left hemiplegia* describes involvement of the respective side of the body with the upper limb being usually more affected than the lower limb. *Diplegia* denotes involvement of both lower limbs and, to a lesser degree, of the arms. In *quadriplegia* or *total body involvement* all four limbs are affected together with impaired posture of the trunk, head and neck. When asymmetry of involvement exists, the terms *monoplegia* or *triplegia* may be used. However, careful assessment may often reveal impairments in less obviously affected limbs.

In the past the unclear aetiology of cerebral palsy made classification based on possible causes or sites of cerebral damage difficult (Mac Keith 1959). Recent developments in cerebral imaging technology (Yin 2000) as well as better understanding of the mechanisms that produce the cerebral damage may provide a different basis of classification in the future (Stanley 2000b).

Irrespective of classification of CP, all types are characterised by an inability to a different extent in performing everyday life tasks. The *International Classification of Impairments, Disabilities and Handicaps (ICIDH),* which was first published by the *World Health Organisation* (WHO 1980), provides a framework for describing and classifying patients with special needs as well as planning management. In the ICIDH *impairment* is defined as loss or abnormality of psychological, physiological or anatomical structure or function; *disability* is any restriction or lack of ability to perform an activity in the manner or within the range considered normal for a human being; *handicap* is a social disadvantage for an individual, resulting from an impairment or a disability, that limits or prevents the fulfilment of a role that is normal for that individual.

There is a recent trend to describe patients with special needs in terms of what a person can do or does do, instead of focusing on their individual impairments, disabilities or handicaps. Following systematic field trials and international consensus, the WHO developed a new classification system for the description of health and health-related states. The revised document known as *International Classification of Functioning, Disability and Health (ICF) (WHO 2001),* has moved away from being a "consequences of disease" classification to become a "components of health" classification. *Functioning* is an umbrella term encompassing all body functions, activities and participation; similarly, *disability* serves as an umbrella term for impairments, activity limitation or participation restrictions. In this way, ICF enables the user to record useful profiles of individual functioning, disability and health in various areas of life and plan management.

Despite its usefulness this classification was not used in this work since it was felt that the information it provides would not answer the research questions. All participants in the study were ambulant patients with the spastic diplegic form of CP. Their impairments were similar and there was a need for a specific scale that would examine gross motor function and not overall health. Therefore the gross motor function classification scale (GMFCS) was used instead (section 4.1.1.3).

2.4 Diagnosis of Cerebral Palsy

The presence of CP may be suspected during early infancy but secure diagnosis of CP may be delayed until two years of age or later (Berkow 1992). This delay in diagnosis is often due to the plasticity of the central nervous system and its ability to recover completely or partially after an injury (Johnston 2004). This phenomenon has an effect on the accurate interpretation of clinical signs and symptoms.

Diagnosis of CP requires a complete history (gestation, pre-natal and peri-natal events, family history, developmental milestones), clinical examination (active and passive range of motion, sensation, muscle strength, muscle tone, limb or trunk deformity) and relevant investigations (cranial ultrasonography, MRI, CT, spesialised tests).

The sustained injury to the upper motor neurons affects motor control and decreases the number of effective motor units resulting in weakness. Simultaneously, the descending inhibitory output is lost and increases the excitability of gamma and alpha neurons producing spasticity, a velocity dependent resistance of muscle to stretch (Koman 2004).

A variety of symptoms and signs could indicate central nervous system damage and these become more significant when a relevant history (maternal, obstetric etc) exists. Lethargy, irritability, abnormal posture,

seizures and poor feeding, low or high *muscle tone*, persistence of *primitive reflexes* in response to sensory stimuli are some of these symptoms (Zafeiriou 1995); any delay in reaching *developmental milestones* should raise concern (appendix one: *terminology*). Generally, a child's motor behaviour stabilises by two or three years of age and a clinical picture emerges more clearly, usually consistent with a specific type of CP.

In the spastic type of the condition, when intelligence is normal or near normal, the abnormal neuromotor control and muscle tone in a developing musculoskeletal system would cause the greatest functional limitations. The abnormal tone and impaired central nervous system control affect muscle growth. Muscles remain in an excessively shortened position for most of the time; as a result, the elastic connective tissue is replaced with inelastic fibrous tissue and muscle fibers become permanently short (Booth 2001). Muscle contractures, a permanent tightening of muscle, develop. These limit the range of joint motion and lead to joint contractures and bone deformity. These deformities can deteriorate over time, usually during growth spurts.

The altered muscle alignment and length, limited development and motion contribute to abnormal loading of the skeleton and the gradual development of skeletal deformities. These can interfere with function (e.g. femur internal torsion) or may reduce the action of muscles by changing the lever arm (e.g. ankle varus/valgus) (Hof 2001). The *lever arm disease* seen in CP impairs function and is the result of more than one factors: abnormal muscle forces, loading and weight-bearing, poor posture and absence of the factors that stimulate growth (Robb 1994).

Finally, a variety of associated impairments can be seen in CP. Abnormalities of the oculomotor system and oromotor function, are common. The impaired swallowing mechanism leads to inadequate feeding and aspiration problems. Moreover, the motor or/and cognitive impairment can result in severe malnutrition. Constipation, gastroesophageal reflux and

incontinence are also frequent (McGrath 1983; Agnarsson 1993). Epilepsy, is more active in preschool and early school years and can be controlled with medication in the majority of patients (Stempien 2000). Learning and cognitive difficulties in CP are estimated at around 30% (Kudrjavcec 1985) and the risk increases with the severity of motor disability. In spastic diplegia, cognitive abilities are usually normal, and learning difficulties are only occasionally present.

In conclusion, the diagnosis of cerebral palsy is based on a detailed examination of the child's health status. Delayed motor skills, slow development, abnormal muscle tone and posture are tested and noted. A detailed examination of the musculoskeletal system is also essential. Specific tests such as CT (computed tomography), MRI (magnetic resonance imaging) or intelligence testing, can be helpful. When no injury is identified on brain imaging other causes of movement disorder must be ruled out. It is essential to distinguish CP from progressive hereditary or acquired neurologic, metabolic and muscle disorders or tumours of the nervous system.

2.5 Natural history

Cerebral palsy is a permanent, but not unchanging neuromuscular disorder. In the first years of life the neuromotor function, primitive reflexes and the tone abnormality may improve. However the abnormal tone in conjunction with the associated impairments that exist in different combinations, are never eliminated. These interact with the effects of growth and lead to a number of problems, which are disabling for the child, teenager or adult with CP.

In the ambulatory child, the predominant abnormalities are observed during gait or other gross motor function tasks. Muscle and joint contractures, as well as skeletal deformities progress during childhood and become problematic for the adult. Initially, the muscle contractures are

dynamic and can be corrected passively. Gradually, they progress into fixed or static, uncorrectable contractures. The combination of fixed contractures and bone deformities with muscle weakness and reduced activity produce abnormal gait patterns and limit the child's function in activities of daily living. Most children with hemiplegic or ataxic CP eventually ambulate, the majority by the age of three years. 85% of spastic diplegics and few quadriplegics maintain long-term ambulation (Sala 1995). Children who are able to sit independently by the age of two years become independent ambulators, with or without equipment. Most of those have fewer than three primitive reflexes present at age 18 months (Molnar 1976).

Long-term ambulation is preserved in a few cases of quadriplegia. Hip dislocation and spinal deformity can present early in the life of a quadriplegic patient making hygiene, respiratory function, sitting balance or positioning for sleeping a real challenge for both patient and carer (Bagg 1993).

Osteoporosis, fractures due to falls and pressure sores are not uncommon in the older patient. Common medical problems that can increase with maturation and affect the general health of the patient with CP include dysphagia, gastroesophageal reflux, urinary incontinence or constipation (Bleck 1984). Life expectancy in CP however is very close to the unaffected population with the exception of the more severely involved patients (Hutton 2006).

It is important to remember that the child with CP will become an adult who, in most cases, will rank communication, activities of daily living and mobility in the community as higher goals than ambulation (Bleck 1987). Therefore, health professionals who care for children with CP should be well aware of the natural history of the different types of cerebral palsy and help families and patients set achievable, functional goals.

2.6 Principles of management in Cerebral Palsy

Cerebral palsy is a life-long disability that has different implications at different stages of life and ongoing management is required. Intervention should be evidence based where possible. Optimal management of the child with CP should include the prevention of additional impairments, reduction of disability and integration of the individual into the community, when possible. Effective communication between health professionals, patient and family is essential for this. In my opinion, the ultimate management goal for a child with CP should be to become an adult who achieves maximum independence within the limitations of the environment and his/her own condition, without compromising his/her quality of life.

2.6.1. Treatment modalities

Various forms of treatment may be required for the effective management of patients with cerebral palsy. A brief description of the most important therapeutic interventions available is given below. Those, which are relevant to this work, will be discussed in detail in Chapter Four.

Physiotherapy usually begins in the first few years of life, soon after the diagnosis is made. It uses specific sets of exercises to work towards three important goals: to prevent muscle and joint contractures, to maximise the child's motor development potential and to prevent deterioration of muscles that can follow disuse.

Several therapy systems have been used for the management of children with CP. The most influential ones include: the Neurodevelopmental Treatment developed by Bobath in England in the 1940's (Bobath 1963), the Sensorimotor Approach to Treatment developed by Rood (Rood 1956), the Sensory Integration Approach by Ayres (Ayres 1974), the Vojta Approach (D'Avignon 1981), the Doman-Delacato or Patterning Therapy (American.

1982), the Conductive Education (Bairstow 1993) and Hare approach (Hare 1993). The majority of therapists however, do not strictly follow a single system. They rather prefer an eclectic approach which usually combines several schools of therapy (Harris 1985).

A range of drugs is used to reduce spasticity. The ones commonly used in children with spastic CP include benzodiazepines/diazepam, botulinum type A toxin for intramuscular injection and phenol motor point blocks (Carpenter 1979; Koman 1993; Koman 1999).

In reducing spasticity two neurosurgical procedures hold promise: the insertion of intrathecal baclofen pumps and selective dorsal rhizotomy (SDR) (Wright 1998; Detrembleur 2000).

Musculoskeletal problems around the feet, knees, hips and spine, which are particularly common in spastic CP, can be treated with a variety of orthopaedic surgical procedures. When indicated, orthopaedic surgery may address either bony deformity (e.g. femoral derotation osteotomy) or muscle contracture and imbalance (muscle/tendon lengthening or transfer) and, a period of immobilisation followed by rehabilitation is usually included.

Finally a range of assistive devices and orthotics are used usually in conjunction with other interventions. Orthotics aid function, and prevent deformity by stabilising joints during walking or other motor functions. The early introduction of equipment to improve mobility, function, postural support and communication is important for a child with CP.

According to Molnar (1987), rehabilitation in cerebral palsy aims to (i) decrease complications and (ii) improve the acquisition of new skills. These goals should be supported by a multidisciplinary team, which involves the family and patient as active participants. The paediatric neurologist, orthopaedic surgeon, developmental paediatrician and physiotherapist,

should work as a team in order to design a goal-oriented management plan. The developmental paediatrician's role is to consider all the available treatment modalities and their effect on child's function, taking always into account the family dynamics and social environment.

2.7 Summary

Cerebral palsy is a diverse clinical entity, and the term is commonly used to describe the disability resulting from an, often unspecified, cause rather than the impairment itself. Improved diagnostic methods and treatment modalities have become available in recent years. The opportunities available to people with cerebral palsy have also improved remarkably and many adults can live independently, when appropriately supported. However, my review of the literature suggests that further research is required on the prognosis, early diagnosis, appropriate management, and evaluation of treatment outcome in CP. Orthopaedic management in spastic diplegia and effective rehabilitation following surgery, in particular are specific areas that require further investigation. These will be discussed in the next two Chapters.

Chapter Three

3. Muscle weakness in Cerebral Palsy

In this Chapter, a literature review will be presented in order to demonstrate evidence of muscle weakness in patients with CP as well as evidence on the importance of strengthening and physical activity in children with motor disabilities. There is some evidence to suggest that children with CP are weaker compared with their normal counterparts but the severity and distribution of weakness in CP patients is largely under-investigated. Muscle strength changes over time in patients with CP and the potential effects of physical inactivity in these patients are unknown. The effect of orthopaedic surgery on pre-existing weakness, the time frame of recovery after surgery, the appropriate physiotherapy regimes post-operatively, as well as the benefit from different types of strengthening exercises in patients with CP are issues that attracted interest only recently. These will be discussed in this Chapter.

3.1 The diplegic child

3.1.1 Pathogenesis and general characteristics

The term *diplegia* is used to describe the most common type of cerebral palsy in which, involvement is primarily seen in the lower limbs while minor upper-limb deficits are only present. Common pathological findings include haemorrhage into the third ventricle and associated periventricular leukomalacia while prematurity is the most significant risk factor. If the lesion is confined to that area the injury is limited to the pyramidal tract and the muscle tone is usually "spastic". However, pathologic and imaging investigations suggest the lesions responsible for spastic diplegia do not only

affect fibres originating from the motor cortex; major nuclear components of the extrapyramidal system are often involved in the lesion (Sussman 1992). The type of brain injury in spastic diplegia is sufficient to explain the presence of mixed tone in some cases. However, s*pasticity* "a velocity-dependent increase in tonic stretch reflexes" is usually the predominant tone abnormality, even when mixed tone abnormalities are present.

Upper motor neurons are those in any long descending tract that control or influence movement and muscle tone. The damage in some or all of these pathways produces a combination of clinical symptoms and signs (Sheean 1998). These are broadly divided into two groups. The *negative phenomena* reflect a reduction in motor activity (weakness, acute hypotonia, loss of dexterity, loss of cutaneous reflexes and fatiguability). The *positive phenomena* are characterised by excessive motor activity (increased tendon reflexes, clonus, spasticity, positive babinsky sign, extensor and flexor spasms, mass reflex, co-contraction, associated reactions, flexor withdrawal reflexes and extensor thrust). A combination of these phenomena can be seen in patients with spastic diplegia.

The recognition of all the impairments associated with spastic diplegia is important when planning appropriate management. *Muscle weakness* has recently been given attention since research shows that it has a negative effect on function. Most of the children with spastic diplegia present with *speech and intelligence* that are normal or with minor learning difficulties. *Cognitive and visual spatial deficits* are not by themselves contraindications to surgery but they should be taken into account if they are severe (e.g. blindness, severe learning difficulties). *Poor balance* is the result of multiple sensory system malfunction (somatosensory, visual and vestibular). Although surgery aims to provide a stable basis of support, the patient's balance should be considered. *Dyspraxia* is the impaired ability to plan out an act; it can affect ambulation and should be considered when planning treatment. Impaired *selective motor control* i.e. the ability to isolate

movement at individual joints, is a problem for the majority of spastic diplegics. The decision to operate may be influenced by the severity of this impairment, which may have an effect on the child's ability to co-operate with a post-operative rehabilitation programme. It is also important to assess the patient's psychological potential and the family dynamics. *Social and emotional problems* can affect adversely the patient's outcome. Therefore, they should always be considered along with family's expectations and their cooperation with the long rehabilitation (Sussman 1992). The presence of all these different impairments could affect therapy and surgical decision-making (Sussman 1992).

3.1.2 Neurophysiologic basis of spasticity

Spasticity is defined as "a velocity-dependent increase in tonic stretch reflexes with exaggerated tendon jerks, resulting from hyper-excitability of the stretch reflex as one component of the upper motor neuron syndrome" (Lance 1980). Stretch reflex is a muscle contraction occurring in response to stretch of the muscle (Sheean 1998). Stretch reflexes are of two types according to the duration of the stretch. Phasic stretch reflexes are produced in response to brief stretches, like tendon jerks after quick tendon taps. Tonic stretch reflexes are produced by longer stretches, such as the ones during testing muscle tone clinically. Their synapses are under inhibitory and excitatory supraspinal control.

Spasticity may often be particularly disabling for the individual child with CP. There has been longstanding interest in understanding the underlying mechanisms, measuring the impairment and developing new effective treatment modalities. Spasticity develops after cerebral or spinal lesions, which include upper motor neurons of the corticospinal tract.

Its main characteristic is excessive or inappropriate muscle contraction through an enhanced tonic stretch reflex. The basis of the enhanced stretch reflex is the increased excitability of the interneuronal processing of the Ia

afferent input within the spinal cord. A certain angular velocity of movement is needed to elicit a stretch reflex in upper motor neuron (UMN) lesions, 40° to 100° per second (Powers 1988). This emphasises the fact that spasticity is not present without movement and once the movement ceases there is no spasticity. Moreover, mild degrees of spasticity may not be detected unless the movement is rapid enough.

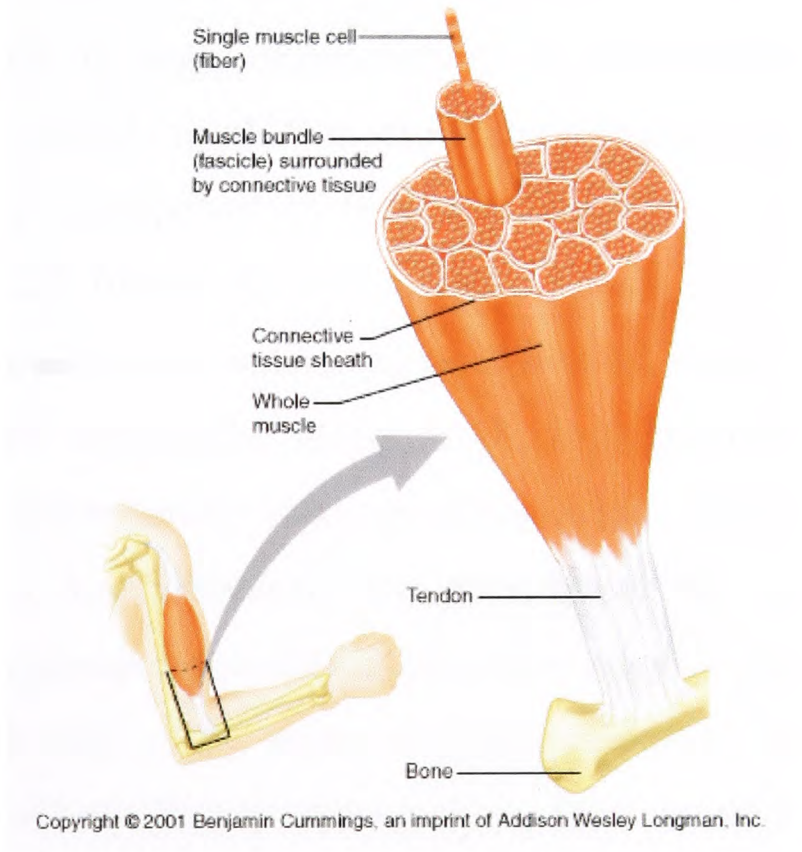
In a survey of health professionals working with neurological patients 94% of responders stated that it was important to measure spasticity, but only 30% attempted to do so in everyday practice (Haas 1994). Clinical measurement of spasticity is difficult mainly because it co-exists with other clinical features (Katz 1992). Several methods for measurement of spasticity have been developed: the Ashworth scale in its original and modified forms (Ashworth 1964; Bohannon 1987), the pendulum test (Wartenburg test) (Bajd 1984), isokinetics (Tardieu 1976; Granata 2000) and myometry (Boiteau 1995). They all assess resistance to passive movement. Whatever the method used, one should bear in mind the highly variable nature of muscle tone in UMN lesions, which make the accurate measurement of spasticity difficult. The assessment of spasticity can be influenced by many factors including the position of the limb against gravity, the degree of co-contraction of the antagonist, the available voluntary control in the limb, the presence of environmental stimuli, the time of day and temperature, fatigue, emotion and many other factors (Katz 1989).

Despite the fact that many questions regarding spasticity and the other clinical features of the UMN syndrome have been answered, many issues are still unresolved. The combination of positive phenomena, such as spasticity with negative phenomena, such as weakness needs further investigation. The optimal way to quantify the neurological contributions of combined weakness and spasticity in the same muscle group in patients with UMN lesions, such as CP, is still an unanswered question (Sussman 1992).

3.1.3 Alterations in muscle function

3.1.3.1 Physiology of muscle contraction

A whole muscle consists of a number of *muscle fibres* arranged in series or in parallel. The muscle fibre is composed of 500 to 10,000 *myofibrils* arranged in parallel and each myofibril contains 1000 to 2,000,000 *sarcomeres* arranged in series. The sarcomere is composed of *protein filaments (thick or myosin and thin or actin filaments)* with other regulatory proteins. The sarcomere is the basic functional unit responsible for the regulation and generation of muscle force. The process by which the myofilaments are activated is known as excitation-contraction coupling (Lieber 1986).



Picture 3.1 Skeletal muscle architecture (Wright State University 2005 - access date 26/09/2006)

The *motor unit* is the basic functional unit of the motor system. It consists of a single *motor neuron*, the *neuromuscular junction* and the *muscle fibres,* which the motor neuron innervates. The *motor endplate* at the neuromuscular junction ensures electrochemical communication between

the neuron and muscle fibre. A chain of biochemical reactions takes place in order to activate the contractile proteins of the fibre (*actin and myosin*) to interact.

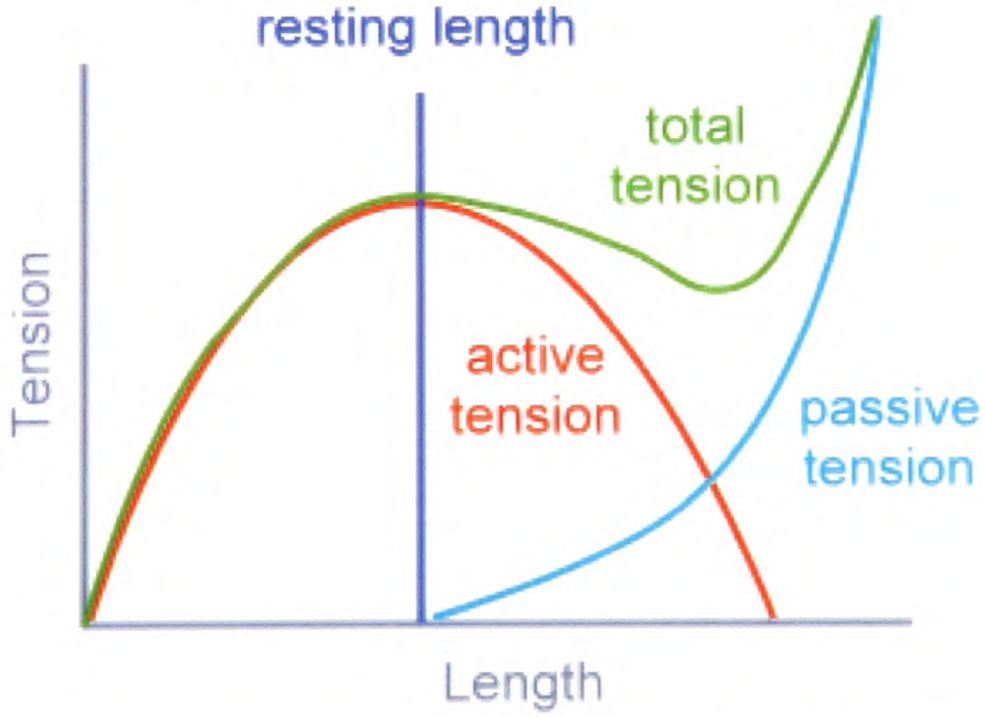
Motor units are classified into three categories: S (slow), FF (fast-fatigable) and FR (fast, fatigue-resistant). The muscle fibres themselves have properties that permit fine-tuning of muscle function. They are similarly divided in: type I (slow muscle fibres), type 2A (fast muscle fibres with oxidative and glycolytic capacity) and type 2B (fast muscle fibres with low oxidative capacity and high glycolytic capacity).

The type of muscle fibre is usually determined histochemically, but the type of motor unit is determined with stimulation of the single alpha-motorneuron. There is high organisation in motorneuron innervation to muscle fibres. In general, small S motor units are composed of type 1 fibres, FR motor units are composed of type 2A fibres and FF motor units are composed of type 2B fibres. At low activity levels, slow muscle fibres are activated first. Type 2A, are activated next and at maximal efforts type 2B fibres are activated. Similarly, motor units that produce small forces are recruited before motor units that produce larger forces. When a motor nucleus begins to be activated by peripheral or descending inputs, individual motor neurons begin firing at a slow regular rate (5 – 10 impulses in humans). Gradually, with more excitatory inputs, larger motor units reach their threshold for firing adding their force as well (Loeb 2000). The overall force of contraction depends on both the size and number of active motor units and their individual firing rates (Deluca 1994). Different muscles, according to their functional demands, use a different balance between recruitment of motor units and firing rates.

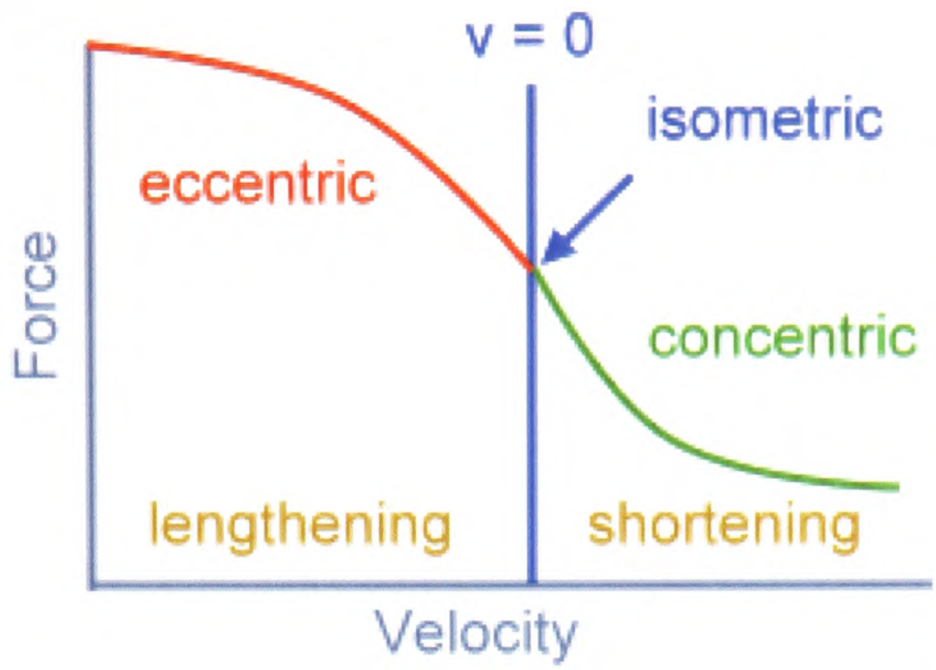
Muscle force is proportional to the number of sarcomeres acting in parallel, while whole-muscle velocity is proportional to the number of sarcomeres acting in series (Lieber 1986). Therefore, the longer the fibres are, the more

sarcomeres in series exist and the greater the contraction velocity is. In addition, the more fibres are in parallel, the greater the muscle force is because of the increased number of parallel sarcomeres. Therefore, muscles are grouped as high-force output (with different pennation angles in order to increase the fibre cross-sectional area), or high-velocity output (long enough in order to increase the sarcomeres in series).

Two relationships characterise a muscle's ability to generate force and/or movement: the length-tension relationship and the force-velocity relationship (Picture 3.2). According to the first, the isometric muscle force (constant muscle length) varies as a function of muscle length. The second states that isotonic velocity (contraction velocity against constant force) varies as a function of muscle force.



Length-Tension Curve of a Muscle



Force-Velocity Curve of a Muscle

Picture 3.2 Muscle length-tension and force-velocity relationship (Huei Ming Chai 2003 - access date 26/09/06)

The properties of skeletal muscles depend on their architecture, fibre-type distribution, motor-unit distribution and characteristics, length-tension properties and force-velocity properties. It would be incorrect to assume that they are static execution organs, which transmit neuronal information. Instead, skeletal muscles are subject to continuous change given the appropriate stimulus.

3.1.3.2 Histopathology of muscle in CP

In upper motor neuron lesions, such as spastic diplegia, muscles may become stiff and less compliant. With time they develop fixed contractures. Normally, a child whose bones are growing stretches the body's muscles through running, walking and other daily activities. This ensures that muscles will grow at the same rate. In children with spastic diplegia, however, spasticity, lack of dexterity and decreased level of activity prevent this stretching. As a result, muscles do not grow fast enough to keep up with lengthening bones and they are placed in shortened positions. This increased muscle stiffness may be due to changes of their connective tissue

(Booth 2001). It has been suggested that muscle fibre stiffness (thixotropy) is increased in the presence of spasticity (Carey 1990). When muscle tone is examined within the range of movement reduced by a contracture, it may be found to be normal; more important is the fact that this kind of resistance, produced by a contracture, is not dependent upon the velocity of the stretch (Sheean 1998).

Although skeletal muscle in children and adults with CP was thought to be structurally intact, with the exception of decreased number of sarcomeres (Tardieu 1982), some studies based on histochemical analysis of spastic muscle in children with CP suggest that spasticity is associated with structural changes (Castle 1979; Rose 1994): type-1 and type-2 muscle fibre atrophy or hypertrophy with varying degrees of predominance, increased fat and connective tissue accumulation and varying patterns of fibre-size and fibre-type distribution. Dietz and Berger (1995) also suggested that changes in the properties of muscle fibre occur in spastic CP. A study from Japan (Ito 1996) on gastrocnemius specimens of spastic cerebral palsy patients showed similar alterations: type-1 fibre predominance and fibre-2B deficiency, with great variation of type-1 fibres. Improved understanding of muscle pathology in CP may lead to better prevention and treatment.

Alterations in motor-unit properties have not been studied in CP. In patients with stroke, motor-unit firing rates are reduced in spastic muscle; further, motor-units are recruited at lower thresholds and fail to increase their firing appropriately as strength of contraction increases (Rose 1998). The impaired modulation of firing rates may contribute to impaired motor control, and similar processes may well take place in CP.

3.2 Muscle weakness and strength profiles in CP

Muscle weakness is one of the deficits in spastic diplegia, which has been surrounded by controversy (Brown 1991; Damiano 1995). Giuliani (1991) suggested that, "*the presence of spasticity only is an insufficient explanation for abnormal movement patterns*", whereas Rose and McGill (1998) reported that "*clinical observations indicate that muscle weakness and spasticity co-exist in patients with CP and present major obstacles*". Despite the fact that Phelps, 50 years ago, advocated resisted exercise for the management of weakness in patients with CP (Scrutton 1984), conventional physiotherapy has consistently argued against strength training not only in children with CP but in all patients with central nervous system damage. Recent research has challenged this approach.

Muscle weakness as defined by Edwards (1977), implies a failure or inability to produce or maintain an anticipated level of force. The maximal force a muscle can produce is highly correlated to its *physiological cross-sectional area* and to a lesser extent to its *anatomical cross-sectional area* (appendix one: *terminology*) (Fukunaga 1996). Specific tension is then produced, which when reduced demonstrates weakness of the muscle.

Understanding the causes of muscle weakness in CP has important implications for the management and appropriate rehabilitation of these patients. There are different mechanisms that may contribute towards weak muscle contractions in CP. Co-activation of antagonist muscles is one potential mechanism (Damiano 2000). Muscle co-contraction is the simultaneous activity of agonist and antagonist muscles crossing the same joint and acting in the same plane and thereby increasing the stiffness at a joint (Olney 1985). Although it is a phenomenon seen during the development of normal movement (Damiano 1993), patients with central nervous system damage demonstrate excessive co-contraction (Ikeda 1998).

This results to increased energy cost and has been shown to contribute to ankle plantarflexion weakness (Brouwer 1998).

It has also been suggested that children with CP may have difficulty in maximally activating their muscles (Sussman 1992), assuming that damage to the pyramidal tract leads to a decrease in upper motor neuronal discharge and weakness. As mentioned in Chapter 3.1.3.2, structural changes of muscles and alterations in motor unit properties could also have a causative role in muscle weakness in patients with CP.

Overall, in developmental lesions of the central nervous system the presence of weakness is probably accentuated by the harmful effects of physical inactivity over the years. Muscles probably fail to develop normally, favouring certain joint positions, bones fail to grow adequately and develop sufficient tensile and compressive strength and the limited amount of movement may cause a lack of development of appropriate neural pathways. There is limited information available on the natural history of weakness in CP and its changes over time. To my knowledge there are no longitudinal trials specifically assessing muscle strength that have followed children with CP to adulthood. Published research, however, has recently recognised the consequence of a motor disability interacting with ageing and physical inactivity (Henderson 2004; Henderson 2005).

Research suggests that children with CP are weaker than normal peers, and a few studies have demonstrated the distribution of weakness in these patients. Muscle weakness can be localised or generalised and it affects some muscles more than others, often showing greater involvement of the plantarflexors and dorsiflexors (Brown 1991; Wiley 1998; Engsberg 2000). Olney et al (1990) reported inadequate power production in the affected lower extremity of hemiplegic children, particularly in the ankle plantarflexor and knee extensor muscles. Furthermore, Bourbonnais (1989) and Wiley (1998) have provided some insight into the severity and

distribution of weakness in spastic hemiparesis and children with CP. However, there is still a need to develop strength profiles based on large age- and type-specific databases of patients with CP.

The recognition of weakness as a component of CP has been longstanding as the term cerebral palsy suggests ("palsy" means weakness originating from the brain). Despite this, management of weakness in CP remains controversial. The use of strengthening techniques has been neglected in neurological patients since there has been concern that excessive strengthening may increase spasticity. The persistence of this concern underlines the need for more research in this area.

3.3 Muscle weakness and surgery in CP

Muscle weakness may affect outcome following surgery and may be unmasked after the reduction of spastic tone. In recent years there has been considerable progress in the pharmacological (Koman 1994) and neurosurgical (Oppenheim 1990) management of spasticity as well as the orthopaedic correction of deformities. However, these types of treatment have revealed the presence of significant underlying muscle weakness in CP.

Selective dorsal rhizotomy (SDR) is a neurosurgical procedure, reserved mainly for selected patients with spastic diplegia. It is used to reduce spasticity and improve function. Because of the loss of inhibition from higher centres in the cerebral palsy lesion, partial division of the posterior nerve rootlets, from L1 to S1 is performed and, encouraging results have been documented with regard to reduction of spasticity (Oppenheim 1990; Engsberg 1998; Engsberg 1999). For this reason, the ideal candidate is a child with spastic diplegia, no severe learning difficulties, no fixed contractures and minimal prior orthopaedic operations (Oppenheim 1990). Rigidity, athetosis, ataxia, hypotonia, and weakness are considered

contraindications. It is performed for non-ambulatory spastic quadriplegics as well to control intractable spasms of the lower limbs since the operation reduces muscle tone (Oppenheim 1990). Gait analysis studies of children undergoing SDR have shown improvement in the range of motion at the knee and hip and reduction of tone as recorded by the Modified Ashworth Scale (McDonald 1991; Boscarino 1993). However, function often remains compromised due to underlying muscle weakness, which becomes evident when the spinal reflex arc is interrupted (Giuliani 1991). Muscle weakness during the post-operative recovery period may be anticipated, and it can reduce the level of independence of children who depend on their spasticity to transfer or stand (Massagli 1991).

Orthopaedic surgery aims to correct deformities and lengthen overactive muscles, which are interfering with function (Gage 1991). The main principles of orthopaedic treatment of spastic diplegic gait are described by Gage (1991). The aim is to restore normal gait priorities, identifying accurately primary and secondary gait abnormalities, correcting deformities at one stage, preserving accelerator muscles, restoring the bony lever arms upon which the muscles or ground reaction forces (GRFs) act and maximising mobility post-operatively. The idea is that when appropriate surgery is performed at the correct time, the improved function will allow enhanced development of motor skills from that time forward.

Common orthopaedic procedures in CP include: muscle and tendon lengthenings or/and releases, tendon transfers and realignment of the skeleton. The overall results of surgery are improved by lengthening all contracted muscles simultaneously rather than in stages, combined or not with bony operations (Bleck 1987; Rang 1990; Gage 1991).

However, the effect of pre-existing muscle weakness on post-operative performance as well as the effect that surgery itself has on muscle strength, is not yet clear. It has been shown that lengthening of the muscle/tendon

unit may lead to an immediate decrease in the effective muscle force (Moseley 1992). Clinical studies have investigated long-term effects of surgery on strength and function suggesting that anything between six to 24 months may be required for complete rehabilitation to be achieved (Reimers 1990a; Reimers 1990b; Damiano 1999). If strength and functional recovery after surgery are that slow then, this form of treatment is potentially detrimental in children who are already weak (Olney 1990) and may explain partially the need for a long rehabilitation programme.

So far, research in orthopaedic surgery in CP has concentrated mainly on the effectiveness of different orthopaedic procedures. Evidence from the earlier studies in this field suggests that surgery overall is an effective way to correct deformities and maintain or improve function (Browne 1987; Nene 1993; Saraph 2000; Saraph 2002). However, there is a need for in depth analysis of these procedures and parameters that affect treatment outcome. Well-controlled randomised studies are few and controversy exists about the time needed for as well as the factors influencing recovery. Standardisation of the rehabilitation programme needed before and after surgery is still lacking. For a cost-effective, evidence-based post-operative management in CP, further research is necessary in this area.

3.3.1 Multi-level orthopaedic surgery in spastic diplegia

Over the past 30 years staged surgery in CP has largely been superseded by single-stage multi-level orthopaedic surgery. With the combination of different orthopaedic procedures in one stage, the "*birthday syndrome*" or repeated operations and rehabilitation every year is avoided (Picture 3.3) (Fabry 1999). Consequently a single rehabilitation programme is required.

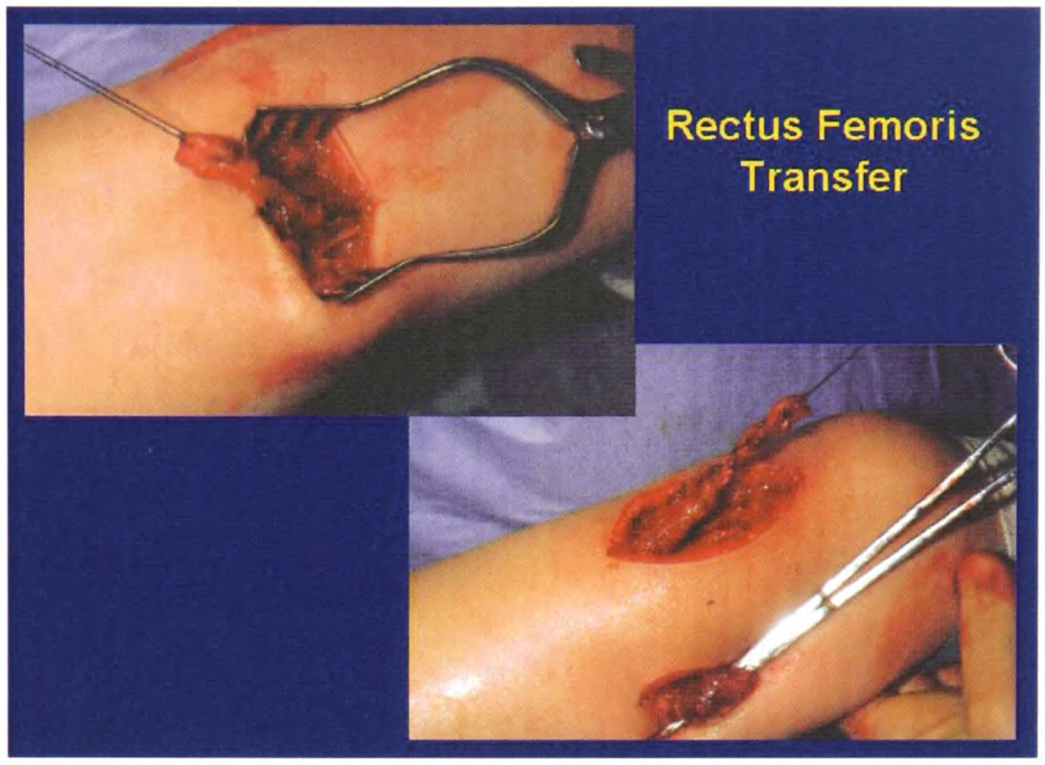


Picture 3.3 The "birthday syndrome" or repeated operations and rehabilitation

Single-stage multi-level orthopaedic surgery is mainly applicable to ambulant children with spastic diplegic CP where gait analysis technology has been a major factor in the objective assessment of complex abnormalities and the evaluation of outcomes (Gage 1991). A long rehabilitation phase that starts from the hospital physiotherapy unit continues with community therapy and intensive home sessions, is required. The family and patient must understand the importance of this phase and participate actively for many months. Clinical experience suggests that a rehabilitation period up to 18 months may be necessary for the full benefits of the operation to be achieved.

A variety of procedures are combined according to the specific neuromuscular and orthopaedic problems of each individual patient. In CP the bi-articular muscles, those crossing two joints, are usually addressed surgically, if contracted. Extensive soft-tissue releases are avoided in order to avoid affecting muscle strength as much as possible and bony operations are used to correct the impaired alignment of long bones and the foot. Lengthening of the iliopsoas, lengthening of the medial hamstrings, aponeurotic lengthening of the gastrocnemius and posterior transfer of the

rectus femoris to the sartorius or gracilis are some of the usual procedures performed (Gage 1987; Tachdjian 1994).



Picture 3.4 Rectus Femoris transfer

The use of gait analysis to inform treatment (De Luca 1997) and to assess outcome following surgery has helped define the indications for individual operative procedures (Rose 1993; Sutherland 1997). "Scissoring" during gait along with demonstrated spasticity of the adductors is the primary indication for adductor lengthening. The main aim of the procedure is to decrease the dynamic deformity and improve standing and gait. Hip flexor lengthening is performed when there is evidence of hip flexion contracture on static testing and increased anterior pelvic tilt and/or decreased hip extension during gait. Hamstring intramuscular lengthening is indicated when there is increased knee flexion in mid to late stance, short stride length and increased popliteal angle greater than 45°. Some patients have overactive rectus femoris, which prevents adequate knee flexion during the swing phase of gait and creates foot clearance problems, especially in the presence of foot deformity and spastic plantarflexors. Circumduction of the limb, or vaulting on the contralateral side are some of the energy

demanding strategies that the patient uses to walk. These problems are managed with transferring of the rectus femoris.

Realignment and correction of lower limb bone deformities are achieved with several procedures. Femoral and tibial derotation osteotomy is planned with the information provided from the transverse plane kinematics combined with clinical and radiographic assessment. Procedures to correct foot deformity include the extra-articular subtalar arthrodesis (Tachdjian 1994) and calcaneal lengthening osteotomy for the planovalgus foot (Mosca 1995). Triple fusion is performed rarely only to correct the rigid and symptomatic foot deformity (Tenuta 1993). Hallux valgus is corrected either by arthrodesis of the first metatarsophalangeal joint (Jenter 1998) or if the deformity is caused by planovalgus foot deformity and not spasticity of the toe tendons, correction of the foot and metatarsal osteotomy is considered.

Several studies have attempted to document the positive results of multi-level surgery in relation to gait performance (Browne 1987; Nene 1993; Ounpuu 1993; Ounpuu 1993; Rose 1993; Abel 1999; Saraph 2000; Saraph 2002; Pirpiris 2003; Gough 2004). Saraph et al (2002) assessed the outcome of multi-level surgery in 25 children with spastic diplegia with a follow up to three years. Their analysis showed significant improvements in the sagittal plane kinematics and the power generation at the hip and ankle as well as at the time-distance parameters. Nene et al (1993) in their review of 18 children with spastic diplegia who underwent multi-level surgery found improvements of hip flexion deformity, hamstring tightness and knee flexion arc in the majority of patients. Similarly, Pirpiris et al (2003) described excellent correction of internal hip rotation and foot progression angles in their prospective study of bilateral femoral derotation osteotomy as part of multi-level surgery in 28 children with spastic diplegia. Although these studies have helped define the indications for specific procedures, most of them were retrospective reviews using small samples (Browne 1987; Nene 1993; Saraph 2002). The majority were uncontrolled designs

comparing pre-operative with post-operative gait and earlier studies did not use gait analysis for the objective assessment of outcome (Browne 1987).

Clinicians and researchers have recently started appreciating the need for in depth analysis of multi-level surgery in a controlled manner. Because multiple deformities are corrected during a single surgical procedure in patients with complicated neuromotor involvement, the problem of evaluating the efficacy of multi-level surgery is complex. Only one study to date has attempted to investigate this complexity. Gough et al (2004) reviewed the outcome in 24 children with spastic diplegia in whom multi-level surgery was recommended. The investigators recognised that, according to the literature, these children in whom, surgical intervention is considered represent a group with deteriorating function. Therefore comparisons of the pre-operative and post-operative gait may underestimate the immediate effect of surgery, as well as ignore the possibility of recurrence of deformity and further loss of mobility following surgery. In their review they compared a group of children who underwent surgery with a control group who, for various reasons, did not undergo surgery. Their results showed that the control group did indeed deteriorate between analyses suggesting that previous studies have underestimated the immediate effect of surgery. At the same time, however, the authors concluded that deterioration is likely to continue following surgery.

Amongst the new areas of research that emerge in the field of multi-level surgery, the question of what determines outcome has not been clarified yet. The effect that pre-existing muscle weakness has on post-operative performance as well as the effect that surgery itself has on muscle strength is unclear. Conflicting evidence exists on the time required for recovery of strength and function after surgery. As a consequence, an optimal rehabilitation regime has not yet been defined.

A few studies have examined the immediate effect of surgery on muscle strength. It has been shown that lengthening of the muscle/tendon unit may lead to an immediate decrease in the effective muscle force (Moseley 1992). Moseley (1992) suggested that the maximal muscle force at an "optimal" joint position does not change postoperatively. The ability to generate force, however, drops off more sharply post-operatively when the joints deviate from the optimal position. Delp and Zajac (1992) using computer modelling of the human lower limb suggested that surgical lengthening could potentially reduce muscle strength and that different parameters related to muscle function have different effects on the force and moment-generating capacity. This effect is potentially detrimental in children who are already weak (Olney 1990) and may partially explain the need for a long rehabilitation programme.

A few clinical studies have investigated the long-term effects of surgery on strength and function. Their results suggest that a period between six and 24 months may be required for rehabilitation to be completed, assuming that no further deterioration of mobility or recurrence of deformity takes place. Damiano et al (1999) examined the changes of hamstrings and quadriceps strength after muscle-tendon surgery. Their study suggested that despite initial post-operative weakening the muscle retained the potential for regaining its original strength, and recovered by nine months after the operation. Function followed the recovery of strength and improvement was not evident until strength values approximated pre-operative values. Reimers (1990a; 1990b) has analysed functional changes in the muscles after lengthening in cerebral palsy and concluded that it often takes six months for gait to improve following surgery. Nene et al (1993) found that the physiologic cost index improved in 14 of 18 patients at one year follow-up and in 10 of 11 patients at two years of follow-up.

However, animal studies examining the long-term effect of muscle surgery on muscle force report a much shorter recovery period than clinical studies.

Brunner et al (2000) showed that intramuscular aponeurotic lengthening of the medial gastrocnemius muscle of six Wistar rats led to acute loss of force, which was recovered in six weeks. Tendon elongation as an alternative technique seems to reduce maximal muscle force more than aponeurotic lengthening for a longer period but still less than clinically observed. Tardieu et al (1979) found a persistent loss of force of minimally 25% after Z-lengthening of Achilles tendon in normal cats. Despite the obvious differences between animal and human studies, the physiologic mechanisms in these species are similar. The discrepancy in recovery time after surgery observed has yet to be analysed in more detail.

Although most researchers recognise the importance of a prolonged rehabilitation programme following multi-level surgery (Nene 1993; Abel 1999; Saraph 2002), very few propose specific post-operative physiotherapy regimes (Saraph 2002). The similarities of programmes observed between centres show that most clinical and research teams appreciate the main factors influencing recovery after multi-level surgery. The differences between these centres, however, underline the need for standardisation of post-multilevel surgery rehabilitation in a way that optimal recovery, preservation of strength and function as well as improvement of mobility are achieved after surgery in the shortest possible time.

Addressing the musculoskeletal problems seen in patients with CP in one stage seems the most appropriate solution. Research, however, has yet to clarify the indications for surgery for specific age groups. The factors influencing outcome require further investigation. More research is required to examine the impact of multi-level surgery on muscle strength in CP especially in cases of pre-existing weakness. An optimal rehabilitation regime following surgery is required once the effects of specific physiotherapy programmes on improving muscle weakness and function are clarified.

3.4 Physiotherapy management in CP

Physiotherapy is essential in the management of children with developmental disabilities such as CP. It starts as soon as the diagnosis is made and in many cases continues through childhood and adolescence. It involves specific techniques, incorporating active treatment, such as motor skill training as well as passive handling such as movements to stretch specific muscle groups (Bower 1992). The aims of physiotherapy for children with CP vary by age and severity (Levitt 1982). Overall it aims to improve motor function and to prevent complications and further deterioration.

Several physiotherapy schools exist for the management of cerebral palsy. Historically, the Crothers approach remains one of the basic principles for involving the patient in a physiotherapy programme. Crothers stressed the need for active movement and stimulation activities to prevent contractures (Gillette 1969). Phelps used an orthopaedic approach and emphasised the inhibition of abnormal movement (Perry 1981). On the other hand, Deaver (1956) favoured functional ability rather than patterns of movement.

Amongst the most influential therapy schemes, the *Bobath* approach is still widely used. It aims at normalising tone, inhibiting primitive reflexes and facilitating automatic reactions and normal movement patterns. Assessment is guided by the patient's reactions to the handling by the therapist. The family is involved in the programme helping with activities of daily living (Bobath 1963).

Conductive education is a systematic approach in which the patient takes part in a group, led by a conductor. Skills introduced and learnt in the group are repeatedly practised as part of the activities of daily living. The method was conceived as a concept, in the 1940s by Dr Adnreas Peto. A detailed evidence report from American Academy of Cerebral Palsy and Developmental Disabilities concluded that the present literature does not provide conclusive evidence either in support of or against conductive

education as an intervention strategy (Darrah 2004). The method for children with CP has been reviewed at the Birmingham Institute (Bairstow 1993).

The *Sensory integration (Ayres)* approach aims at improving efficiency of neural processing and organising better adaptive responses. Work concentrates on the proprioceptive, tactile and vestibular systems and the equipment used relates to these. It is especially applicable to children with learning difficulties but the family cannot administer treatment (Ayres 1974).

The *Hare* approach is based on the hypothesis that neurological signs are unreliable for assessment and treatment planning. Instead, the focus is on the underlying disorder of posture and movement. A system for the analysis of posture and movement is described. Levels of ability are identified. Assessment is made in positions of lying, sitting and standing. Techniques of treatment include the use of arm and leg gaiters, below-knee plaster boots, aids and adapted furniture as appropriate (Hare 1993).

The *Vojta* approach is based on the concept of reflex locomotion and the hypothesis that the "total movement pattern" develops as a result of stimulation of the periphery (body parts) and that the postural disorder observed in the affected newborn is similar to that of the child with CP. Treatment is carried out in supine and prone lying positions, fixed points are identified and stimuli are given to the movements of reflex locomotion, with maximal resistance. It is suitable only for infants and young children and can be administered by the family (D'Avignon 1981).

The *Doman-Delacato patterning therapy* is given to children with neonatal or acquired brain damage. It aims at achieving independent mobility, improving motor coordination, preventing or improving communication problems and enhancing intelligence. It uses sensory and reflex stimulation

and repetition of passive movement patterns. The family administers the treatment several times daily (Pediatrics 1982).

The *Sensorimotor Approach to treatment (Rood)* is applicable to children with CP but cannot be performed by the family. It also uses tactile, proprioceptive, kinaesthetic and sensory stimulation (tapping, brushing, icing etc) and aims at activating postural responses and movement once stability is achieved (Rood 1956).

One of the major issues in relation to physiotherapy for children with CP is the lack of conclusive evidence to support its effectiveness (Butler 2001). Clinical beliefs and opinions based on history, experience and empirical evidence appear to be the primary rationale for the provision of physiotherapy treatment to children with CP. The different schools of therapy that have been developed over the years have a largely unknown efficacy.

Attempts to evaluate one treatment against another have met little success. Until 1990 there was little support in the existing literature for the efficacy of physical therapy in the general management of CP (Sussman 1992) and in relation to different interventions. As Levitt (1982) suggests "*it is difficult to decide which treatment system is superior whether on the basis of a scientific study or on theoretical grounds*". A scientific study aiming to compare different therapy methods has to deal with a variety of issues and problems including the matching of children, the therapist's skill, the validity of assessment methods, the child's personality, associated handicaps and home background. Comparison with an untreated control group is also unrealistic. It would not be ethical for example, to subject one tested group to passive muscle stretching, withholding it at the same time from a control group since it is presumed that stretching is beneficial in preventing contractures. Finally, comparison of different treatment systems on theoretical grounds only, would not be possible since there is no single

theory that fully explains the abnormal motor behaviour or the effects of various treatment procedures in CP.

During the last decade studies that attempt to test the empirical knowledge and experience, which dominate in this field, have emerged. Law et al (1991) found an association between younger age and positive treatment outcomes as well as intellectual ability to be unrelated to outcome. Others contradict these results (Sommerfeld 1981; Trahan 1999). Palmer at al (1988) found an association between milder forms of CP and positive treatment outcome, whereas Scherzer et al (1976) found no association with severity. In a randomised controlled trial involving 56 children with CP, Bower et al (2001) studied whether motor function and performance are better enhanced by intensive physiotherapy or collaborative goal-setting. They found no significant difference between intensive and routine physiotherapy or between aim directed and goal directed therapy. In a previous study, the same group (Bower 1996) provided some support for the view that selecting specific goals and intensive therapy accelerate the acquisition of motor skills, for a short period of two weeks.

Giuliani (1991) in a review article suggests that although physical therapy is considered as an important determinant for successful surgical outcome, the duration and intensity vary greatly among clinicians. Three prospective randomised studies (Steinbok 1997; McLaughlin 1998; Wright 1998) compared the results of SDR combined with physiotherapy, against physiotherapy alone. These studies led to contradictory results. Although the Toronto (Wright 1998) and Vancouver (Steinbok 1997) studies showed that the results of SDR combined with physiotherapy were superior to those of physiotherapy alone, the Seattle (McLaughlin 1998) study, which had the longest follow up, showed no difference between these two methods of treatment. Other investigators claimed that, in contrast to previous studies, an increase in strength was observed after SDR and intensive physiotherapy (Engsberg 1998; Engsberg 1999).

At a consensus conference sponsored by the American Physical Therapy Association in 1990 on 'The Efficacy of Physical Therapy Management of Cerebral Palsy' it was established that *'no definitive support for the efficacy of physical therapy, or the lack thereof, in the management of cerebral palsy, exists in the research literature'* (Campbell 1990). Evaluation of the efficacy of physiotherapy treatment interventions in specific chronic neurological disorders remains a priority for physiotherapy research. Despite this uncertainty there is a consensus that physiotherapy should remain a basic management approach for children with CP (Bax 2001).

3.4.1 Therapeutic Exercise: evidence of effectiveness and safety of strength training for children and adolescents with spastic cerebral palsy

Strength training is part of many training programmes in healthy individuals and contributes to improved physical performance (Cusimano 1988). There is a large volume of research on the benefits of physical activity and strengthening on young adults without disabilities. Winett and Carpenelli (2001) in a review article comment that "*resistance training has profound effects on the musculoskeletal system and contributes to the maintenance of functional abilities*". It prevents osteoporosis and low back pain; it may positively affect risk factors such as insulin resistance, high blood pressure and body fat, which are associated with diabetes, heart disease and cancer.

In CP, physiotherapy approaches historically aimed at reducing spasticity to restore optimal motor function. The role of weakness in CP was overlooked for decades during the predominance of *Neurodevelopmental Treatment (NDT)*. Bobath and Bobath (1976) indicated that muscle weakness was not real but relative to opposition by spastic antagonists and it was not considered a primary contributor to the motor dysfunction. The use of strengthening exercises was discouraged because it was felt that excessive

effort would increase spasticity, co-contraction and associated reactions (Bobath 1980). Apart from the assumption that maximum physical effort, associated with weight training, would increase spasticity in these overactive muscles, it was also advocated that children with CP lack sufficient isolated muscle control to increase strength in target muscles. Therefore it was believed that weight training would offer little or no benefit.

Damiano (1995) suggested that these clinical observations lack scientific basis and have led health professionals to underestimate the role of muscle weakness in CP. There has been a recent resurgence of interest in strength testing and training and research has demonstrated the benefits of strengthening exercises in individuals with CP.

Muscles strengthen in response to appropriate stress and any method, provided that the frequency, duration and intensity of the exercises causes enough overload to elicit a training effect, can be effective (Atha 1981). Improvements in muscle performance have been demonstrated using isometric (Healy 1958), isotonic (Healy 1958; Damiano 1995; Damiano 1995; Damiano 1998) and isokinetic exercise (McCubbin 1985; MacPhail 1995) as well as a combination of isotonic exercise and weight machines (Horvat 1987). Strength gains were similar or exceeded those reported in the non-handicapped population (Petersen 1990), and were maintained for three months after the cessation of training (MacPhail 1995). The methodological differences and the different variables measured (force, torque, power) make comparisons of strength gains between these studies difficult, but they have all reported positive results following short periods of training.

A detailed review of the strength training literature by Atha, as early as 1981 (Atha 1981) and a more recent one by Faigenbaum in 2000 (Faigenbaum 2000) showed that isometric training is effective in increasing strength. Most gains are achieved within five weeks of the start of the

programme. Although different combinations of repetitions and sets have proven to be effective, recent data suggest that high repetition (10 to 15) moderate load strength training (13 to 15 RM load) may be more beneficial and better tolerated than low repetition heavy load training. An appropriate warm-up regime is essential before the start of the training and alternate days of training have proven to be beneficial in producing strength gains.

Whatever the method used, progressive resistance training has been shown to produce the greatest gains in strength, endurance, functional and motor performance, as well as increased speed of movement (McCubbin 1985; Horvat 1987; Damiano 1996; Damiano 1998; Dodd 2002). Harris (1978) showed that strength training with resistance improved performance in weak muscles and promoted balance.

The overall aim of strength training is not just to increase strength but to improve gross motor function as well. There is evidence to suggest that a correlation exists between increases in strength and gait and function. Improvements in walking pattern, energy cost and temporal parameters have been documented after strength training using gait analysis (Damiano 1995; MacPhail 1995; Damiano 1998) and the Gross Motor Function Measure (GMFM) (Russell 1989). McPhail and Kramer (1995) found that subjects with larger strength gains were more likely to show functional improvement. Damiano and Abel (1998) found that increases in lower limb strength resulted in improvements in walking, jumping and running. These changes in muscle strength are likely to result in changes in dynamic muscle power, which would be evident in gait kinetics. This however has yet to be explored.

Despite fears of increasing spasticity following strength training, there is no evidence to suggest that strength training of spastic muscle can adversely affect muscle tone. Passive range of motion has been shown to increase rather than diminish as a result of weight training in subjects with CP

(Healy 1958; Horvat 1987). No increase in spasticity has been reported using accepted clinical measurements such as the modified Ashworth Scale (MacPhail 1995), or the pendulum test (Fowler 2001) or measured indirectly by any deterioration in fine and/or gross motor ability (Holland 1990; MacPhail 1995; Damiano 1998). Furthermore, training may reduce the amount of co-contraction, allowing greater activation of agonists and greater force production (MacPhail 1995). Damiano (2000) used surface electrodes to record muscle activity, both for muscle strength-testing and gait analysis, in a study that attempted to determine the relation between co-contraction, muscle strength and motor function in spastic CP children.

Finally, strength and endurance training should be considered important element of fitness and health for patients with CP. In the past, sports were rarely recommended for children with CP. There is no evidence however, for discouraging patients from participating in sporting activities. There is no evidence to preclude even vigorous exercise such as downhill skiing, that were once thought to increase orthopaedic deformity (Stempien 2000). Swimming and horse riding have been shown to be beneficial for children with motor disabilities (Stempien 2000) and their flexibility and endurance may increase through these activities. More importantly, exercise improves self-esteem and helps children to develop interpersonal and social skills.

In the last 10 years, three literature reviews have been published on strength training for patients with CP and all three concluded that this is an accepted technique (Darrah 1997; Haney 1998; Dodd 2002). However, further research is necessary to develop specific treatment regimes. These would aim at training muscles according to individual needs. Specific programmes would also be required when rehabilitation programmes are combined with other interventions, such as surgery.

3.5 Summary

There is evidence in the literature to suggest that muscle weakness in children with spastic diplegic CP contributes to their disability. The natural history of muscle weakness during the growing years of a diplegic child and the effect that multi-level orthopaedic surgery has on weakness are largely unknown. There are no universally accepted treatment techniques for effectively improving muscle strength in these children after single-stage multi-level orthopaedic surgery, although the feasibility to both measure muscle force and to strengthen muscle have been demonstrated.

Chapter Four

4. The diplegic gait

This Chapter includes a review of the literature on the assessment methods used in this thesis.

Spastic diplegia is the most common type of CP and lower limbs are usually more affected than upper limbs. The combination of spasticity, weakness, co-contraction, impaired selective motor control and balance results in characteristic gait patterns. The diplegic gait changes over the years as the nervous system matures, the child increases in height and weight and deformities develop. In most cases, these deformities are amenable to conservative treatment initially, but may require surgical treatment eventually. The severity of spasticity, which affects the flexor muscles more than the extensors, may vary. Spasticity and limitations in selective control of muscles contribute to a less energy-efficient gait. The presence of weakness has recently been recognised as a significant problem and has been attributed to altered muscle properties.

Gait Analysis is used extensively in assessing outcome of treatment in CP. Accurate three dimensional motion data, are crucial for the understanding of complex movements that occur during walking in three planes. During the last decade the contribution of muscle weakness in diplegic gait has been the object of research and instrumented testing of strength has been introduced for this group of patients. Dynamometry is a recognised method for testing strength in healthy individuals but its reliability in CP remains under-investigated. Finally, the central nervous system damage in CP produces a global effect on motor function. For that reason, scales which

measure gross motor function have been developed and their use is widespread both in clinical practice and research.

4.1 The diplegic gait

The effect of abnormal neuromotor control and muscle tone on a developing musculoskeletal system is the main cause of functional limitation, in the majority of spastic diplegic patients (Sussman 1992). The combination of fixed contractures and bone deformities with muscle weakness and decreased activity produce abnormal gait patterns and limit the child's function in activities of daily living.

The prognosis for walking in spastic diplegia has been studied extensively (Watt 1989; Campos da Pas 1994) with the presumption that function remains stable after motor development is complete (Rosenbaum 2002). In the first few years of life the neuromotor function and muscle tone may improve. In most cases, muscle and joint contractures as well as skeletal deformities progress during childhood and become problematic in the adult. However, 85% of spastic diplegics maintain long-term ambulation (Sala 1995). Children who are able to sit independently by the age of two years become independent ambulators, with or without equipment (Molnar 1976). However, there is little information on the natural history of gait after independent or assisted ambulation has been achieved and the limited evidence available suggests that the level of function may not remain stable in all children with spastic diplegia (Johnson 1997; Bottos 2001; Bell 2002).

When examining the gait of a diplegic child it is important to differentiate between primary abnormalities and compensatory mechanisms. For example, spastic rectus femoris and hamstring muscles produce a "*stiff*" knee gait pattern with decreased knee flexion in swing. This creates foot clearance problems, which may be compensated by hip abduction to produce circumduction of the swinging limb. The circumduction is a coping or

compensatory response and is used in order to circumvent the primary problem (Gage 1991). The combination of gait deviations, the interaction between joints, the affected balance and the compensations used make classification of the diplegic gait, the identification of problems and the planning of treatment difficult tasks.

The typical diplegic gait pattern is presented with hip flexion, adduction and internal rotation, and flexion at the knees. Common musculoskeletal deformities include pes valgus, often with a plantar flexed talus, hind foot equinus and forefoot abduction. There is increased femoral anteversion, which is often neutralised by the external tibial torsion, combined with the forefoot abduction. The child usually walks with a toe-toe gait, toe-heel or flat-foot gait. The type of foot contact combined with the torsional deformities and the impaired balance contribute to stance phase instability. The spastic knee flexors and extensors result in restricted range of motion during swing phase. This, combined with deficient pelvic movement and ankle equinus, create foot clearance problems (Bleck 1987; Gage 1991).

The problems responsible for the motor deficits in children with spastic diplegic CP, such as spasticity, out of phase muscle function, development of secondary contractures and bony abnormalities are managed with a variety of treatment modalities. These include physiotherapy, occupational therapy, orthotics, medications, neurosurgery and orthopaedic surgery. Amongst those, orthopaedic surgery forms part of a treatment programme and does not, in itself represent a definitive treatment for CP.

Treatment interventions should be based on evidence for their effectiveness and should result in functional improvement with a low risk of complications. Studies that assess treatment outcome in CP must be based on valid measurements, adequate criteria for patient selection, accurate treatment protocol description and appropriate study design (Sussman 1992). Three-dimensional gait analysis has helped define appropriate

management (DeLuca 1997) and assess outcome following intervention (Abel 1999). Objective assessment of muscle strength has recently been introduced into the management of patients with CP (Damiano 1998). Finally, gross motor function assessment with valid and reliable measures is essential if a holistic approach in the treatment of CP is required (Russell 1989).

4.1.1 Assessment methods

Before treatment in spastic diplegia can be considered effective four primary areas need to be addressed (Sussman 1992) (1) the technical outcome (2) the functional outcome (3) the patient satisfaction with the outcome and (4) the cost of the treatment. The first three areas are addressed routinely with measures, which are used in the management of patients with spastic diplegia; the list, although not exhaustive includes three-dimensional gait analysis, dynamometry, functional assessment measures, and scales which measure patients' satisfaction. Analysis of cost effectiveness of treatment, although important, will not be presented in this work.

4.1.1.1 Gait Analysis

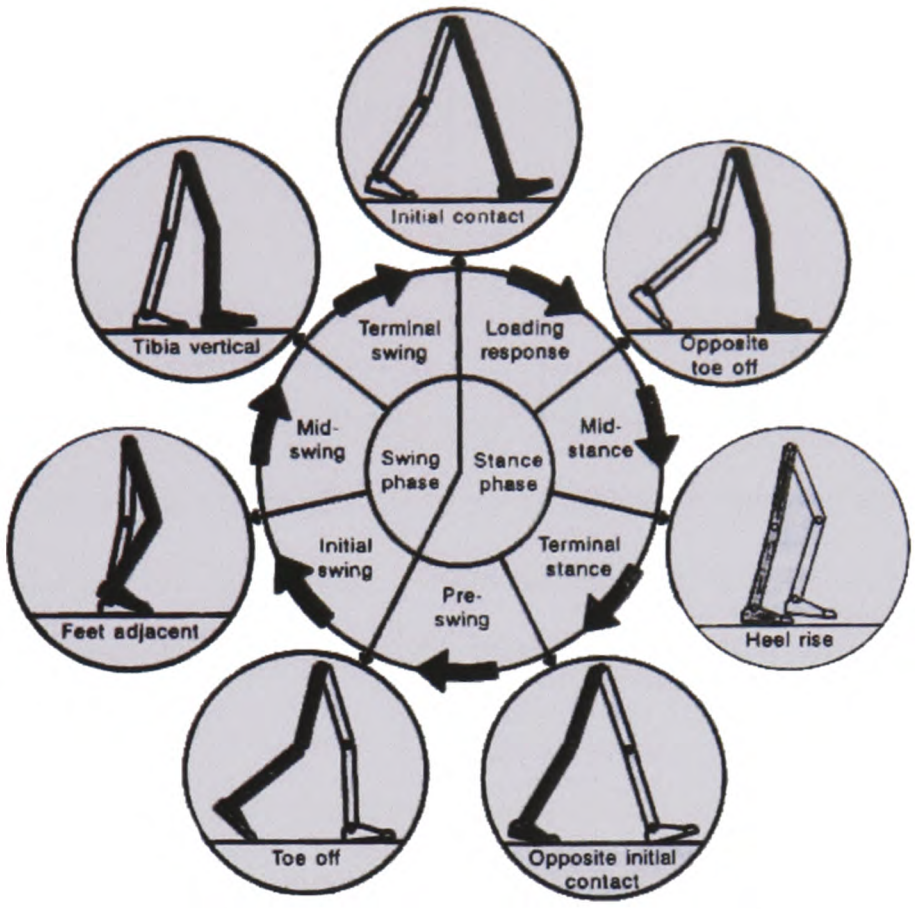
Gait analysis is the systematic study of human locomotion. This information is combined with the clinical information on the subject tested, in order to describe the mechanics of normal and pathologic walking, identify the cause of gait deviations and assess how these alter after intervention (Gage 1991).

4.1.1.1.1 Normal Gait

In order to understand abnormal gait it is necessary to have a detailed understanding of normal gait as a reference frame. It is also important to understand the natural variation of gait amongst healthy individuals of different age groups and sex.

Historically, descriptions of gait were first given by, Leonardo da Vinci, Galileo and Newton. In 1682, Borelli published his work "De Motu Animalum" in which, he calculated the location of the centre of gravity of the body and described how balance is maintained in walking. In 1836, the Weber brothers gave the first description of the gait cycle. The first use of multiple photographic exposures on a single plate of a moving subject (human or animal) was undertaken in the 1870's by Marey in Paris and Muybridge in California. Braune and Fisher in 1895 studied human locomotion and the resulting photographs were used to describe the three-dimensional trajectories, velocities and accelerations of the body segments. Knowing the body masses they were able to estimate the forces involved during the gait cycle. Force plates, which measure the magnitude and direction of the ground reaction force, were first designed by, Amar in 1924 (Kirtley access date 26/09/2006).

Normal gait is a sequence of events that take place over a gait cycle (Gage 1991; Perry 1992). A complete gait cycle or stride begins when one foot strikes the ground and ends when the same foot strikes the ground again (Picture 4.1). Two phases compose a complete gait cycle, *stance* and *swing*. Stance phase begins with *initial contact*, which in normal gait starts with the heel and ends at toe-off when swing phase begins. Swing phase ends when the foot strikes the ground. Initial contact occurs at 0% and 100% of the gait cycle; toe-off occurs at approximately 60% of the gait cycle. Opposite toe-off and heel strike occur at 10% and 50% of the cycle respectively. During a gait cycle there are two periods of "*double support*", where both feet are on the ground (10% of gait cycle). The first double support is called "*loading response*" where the body decelerates as the shock of impact is absorbed. It is followed by a period of *single stance*, during which the opposite limb is in swing phase. These two phases (single stance of standing foot and swing of opposite foot) are equal. In late stance there is the "*pre-swing*" phase, which is equal to the loading response of the opposite foot and during which the foot is prepared for the swing phase.



Picture 4.1 The normal gait cycle

Normal gait requires energy efficiency. To this effect, the prerequisites of normal gait include: stability in stance, clearance of the foot in swing, appropriate pre-positioning of the foot for initial contact and adequate step length. The above are necessary to ensure the very important energy conservation (Gage 1991). Corcoran and Brengelmann (1970) suggested that people select a walking speed that allows minimum energy expenditure.

In normal children, ambulation begins without these prerequisites being fulfilled and gradually evolves to the adult pattern. Sutherland el al (1997), suggested that heel-toe gait in children does not begin until 3 ½ years of age and is depended upon maturation of the CNS. Step length, cadence and walking velocity depend upon central nervous system maturation and growth. Moment and power measurements around the hip, knee and ankle, show that the youngest children use their hip flexor and extensor muscles more than their ankle plantarflexors for power generation. As a child gets older the variability of gait measurements decreases and adult kinematics

are mostly achieved by the age of three years. Temporal parameters take longer to mature and adult values are mostly achieved by age seven years.

4.1.1.1.2 Cerebral Palsy gait

In CP, the characteristics of normal gait are lost. The CNS lesion results in loss of selective muscle control, abnormal muscle tone, imbalance between agonist and antagonist muscles, weakness and impaired balance. These impairments alter the gait of patients with CP.

Furthermore, in CP gait the observed deviations rarely occur in isolation. In fact, there are primary anomalies due to the CNS damage and compensatory mechanisms that the patient uses to overcome the primary problems of gait. It is important to differentiate between primary and secondary mechanisms when analysing CP gait.

Clinical gait analysis has helped in defining characteristic gait patterns in patients with CP (Winters 1987; Sutherland 1993; Hullin 1996; Lin 2000; Rodda 2001). Amongst the different descriptions two classifications are used widely. Winters et al (1987) described four gait patterns in spastic hemiplegia and Sutherland and Davids (1993) described four patterns of sagittal knee motion in spastic diplegia. Coronal and transverse plane abnormalities are usually secondary phenomena attributable to anomalies at other joints. Therefore, the *jump* knee gait, frequent in young spastic diplegic patients, is characterised by increased knee flexion in early stance phase with correction of the knee wave to normal or near-normal extension in mid- and late stance. The *crouch* knee gait presents with increased knee flexion throughout the stance phase, with variable alignment in swing phase and is uncommon before seven years of age. The *stiff* knee gait is characterised by excessive knee extension throughout swing phase with variable alignment in stance phase usually due to inappropriate phasic activity of rectus femoris. The *recurvatum* knee gait describes increased

knee extension in mid- and late stance phase with variable knee motion in the swing phase, usually attributable to triceps surae contracture and overactivity or aggressive lengthening of hamstring muscles.

Since gait problems are complex and, often more than one management method is required to achieve optimal outcomes (e.g. botulinum toxin and ankle foot orthosis in ankle dorsiflexion function), the role of gait analysis in identifying gait patterns in CP is significant.

4.1.1.1.3 Clinical gait analysis

In clinical gait analysis quantitative information is collected in order to understand the aetiology of gait abnormalities and assist in treatment decision-making. Specialised technology such as 3D motion capture systems for measuring joint motion, electrodes placed on skin surface for the study of muscle activity and force platforms embedded in a walkway for measuring forces and moments developed around joints, all produce useful data. Depending on the available resources and organisation of a gait analysis laboratory, a combination of a detailed medical history, physical examination, videotaping, motion and/or muscle assessment and interpretation of data is used.

While quantitative gait analysis uses technology to measure gait parameters, it does not replace the human observer but contributes to the understanding of visual impressions of a patient's gait impairment. In *observational gait analysis* a subject's gait is studied visually. Viewing a videotape of a patient walking, with or without slow-motion, is more consistent than viewing the subject live during repeated walks (Gage 1991). However, because the human eye "frames" only about 16 times a second, gait cycle events lasting as long as 1/16 sec cannot be observed. Moreover, the simultaneous motions that occur at different joints during walking, or/and the compensation mechanisms, cannot be detected by visual

observation. These disadvantages of observational gait analysis led to the development of more sophisticated methods for analysing the human locomotion.

Prior to the modern gait analysis systems, motion of human joints during walking was usually recorded with cinematography and manual digitisation techniques (Sutherland 1984). Because of the time needed for data processing with this method, and with the rapid development of computer systems, new techniques are now used. *Optical tracking systems* identify the displacement of markers, during walking, located at specific anatomical landmarks on body segments (Gage 1991). Active marker systems use active illumination of the markers on the subject's body and passive marker systems employ reflective markers and infra-red cameras. Different number of markers is used in order to define the trunk, upper or lower extremities motion. Their size and the number of cameras also vary. Recent developments in the gait analysis field have produced systems with 12 infra-red cameras and 15 mm diameter reflective markers.

Movement of joints and body segments is recorded in three planes (sagittal, coronal and transverse plane). Infra-red cameras track reflective markers, which are placed by a member of the clinical team on specific anatomic landmarks. Since it is impossible to place markers inside joints at the centre of the axis of rotation, mathematical equations are used to translate the markers from their actual position to the joint centres. The two-dimensional information, together with anthropometric data are programmed into the computer and used to create a three-dimensional biomechanical model through an appropriate interface.



Picture 4.2 Clinical gait analysis (consent obtained)

Processed data provide graphically the motion of joints in all three planes described as *kinematics*. When force plates are used, the ground reaction forces (GRFs) in the vertical, medial-lateral and fore-aft planes, as well as the external moments around the x, y and z axes, are calculated. This information is combined with the calculated joint centres and the mass moment of inertia of the body segments to produce mathematically the internal joint moments and powers or *kinetics*; moments and powers that the muscles produce to counteract the GRFs. Temporal parameters and muscle electrical activity during walking are also recorded. Some laboratories have the additional facilities to measure energy expenditure and pressure distribution under the foot. The graphically displayed data are assessed in combination with video observation of the patient in the sagittal and coronal planes.

Dynamic electromyography (EMG) is widely used as part of gait analysis. The electromyogram, according to Perry (1992), *"is an indirect indicator of*

muscular function". The electrical signals that accompany the chemical stimulation of the muscle fibres can be recorded and analysed, with appropriate instrumentation. Dynamic EMG records these signals during the gait cycle. Although it is not a direct measure of muscle force it is clinically useful. It provides information on muscle phasic activity and neurological control. It also offers some indication of the intensity of individual muscle activity (Rechtien 1996). Electromyography can be performed simultaneously with both gait analysis and dynamometry measurements allowing the recording of co-contraction between agonist and antagonist muscles (Milner-Brown 1979).

The reproducibility and reliability of gait data at the same gait laboratory have been demonstrated (Kirkpatrick 1994). Skaggs (2000) showed that gait analysis interpretation and suggested treatment are as consistent as most orthopaedic classification and outcome tools. The reproducibility of gait analysis between different centres for the same patient with CP however has recently been challenged. Noonan et al (2003) examined the inter-observer variability of raw gait analysis data produced at 4 gait laboratories for 11 patients with spastic CP. They concluded that significant variations exist when the same patient is assessed at different gait centres. More research is necessary to establish the repeatability of gait analysis between different laboratories. The gait data for this study however were collected at the same laboratory where inter-observer variability was assessed and was satisfactory.

The process of gait data collection usually produces the following: video recordings, clinical measures, stride and temporal gait data (step length, cadence, walking speed etc), three-dimensional joint data (kinematics), three-dimensional joint moments and powers (kinetics), electromyographic signals (EMG) and often a measurement of metabolic energy expenditure. These parameters are then evaluated using a database of normal data as baseline.

4.1.1.1.4 Clinical gait analysis and its role in treatment decision-making

The goal of clinical gait analysis is to assist in treatment decision-making in patients with complex walking problems by helping clinicians understand the aetiology of gait deviations. While treatment decision-making is facilitated by clinical gait analysis, these decisions are always made in the context of the clinician's experience in the management of any particular disorder. Therefore, the recommendations proposed are not entirely dictated by gait analysis and "do nothing at present, but continue to follow" is not an uncommon decision.

The quantitative nature of gait analysis facilitates prospective clinical studies and the development of large databases of pre- and post-management information used in retrospective research. This has led to beneficial changes in the approach, for example, to surgical treatment in patients with CP (Ounpuu 1993; Ounpuu 1993; Abel 1999; Damiano 1999; Granata 2000; Saraph 2000). DeLuca (1997) showed that computerised gait analysis led to modifications in surgical treatment recommendations made by experienced physicians in CP. This led to a reduction in the number of procedures and an associated reduction in the cost of surgery. Other studies have helped in the ramification of specific procedures, such as gastrocnemius lengthening (Rose 1993), and the surgical treatment of equinovarus deformities (Waters 1982). Gait analysis techniques have resulted in the development of rectus femoris transfer (Perry 1987), an understanding of the difference in effectiveness of the rectus femoris transfer as compared with the rectus femoris release (Ounpuu 1993), and an appreciation of the significance of the location of rectus femoris transfer (Chung 1997). They have also led clinicians away from specific procedures, such as the hip adductor transfer (Scott 1991) and provided a means to directly assess the effect of similar procedures on function – such as the medial hamstring versus medial and lateral hamstring lengthening (Ounpuu 1997) and hamstring lengthening with (Gage 1987) or without rectus femoris procedure (Bauman 1980).

Gait analysis is not used only when surgery is considered. Questions concerning prescription or evaluation of orthoses, or medication efficacy can also be addressed. The information published however is still limited. Some previous studies have evaluated the effects of physiotherapy treatment including strength training, on strength and function in children with CP using gait analysis. These have demonstrated the need for well-designed clinical trials (Damiano 1995; Damiano 1995; Abel 1999; Damiano 1999). Damiano (1995) designed a study where 14 spastic diplegic children participated in a bilateral isotonic strengthening programme of the quadriceps femoris muscle. Gait analysis was undertaken before and after the strengthening programme to determine whether quadriceps femoris strengthening improved crouch gait. Kinematic data and temporal information were collected and compared with strength measurements. Although the results did not show statistically significant differences after the strengthening programme, there were trends that could be attributed to the strengthening regime. For example, the mean knee flexion at initial contact was decreased by five degrees, a change that is in the range of gait analysis error.

There is little doubt that gait analysis is the most reliable and objective tool currently available for the assessment of gait deviations especially in complex neuromuscular disorders. From the technical aspect, the use of gait analysis in decision making is established, although there is room for improvement in consistency and standardisation (Hailey 2000).

4.1.1.2 Muscle strength assessment

Muscle strength assessment provides information, which aids diagnosis and helps define appropriate management plans. At the research level, strength assessment provides valuable information for the evaluation of treatment efficacy. The recognition of muscle weakness as an important impairment, which contributes to the disability in patients with CP, has made the need for accurate strength measurement essential in this population.

Historically, manual muscle testing has been the clinical standard for strength assessment (Daniels 1972; Brooke 1981) as a quick and inexpensive method. Although studies show that the technique is reliable within one grade (Lilienfeld 1954; Florence 1992), it does depend on a subjective rating and the examiner's experience (Daniels 1972). One major limitation of manual muscle testing is its inability to distinguish sufficiently between different grades of strength, being accurate only when significant strength changes exist (Mendell 1990).

Since manual muscle testing may not be sufficiently sensitive to grade individual muscle groups in some patients, objective measurement of muscle strength has been proposed (Mendell 1990). A few studies have tried to define the relationship between manual muscle testing and objective measurement techniques (Beasley 1956; Beasley 1961; Bohannon 1986; Aitkens 1989; Mendell 1990). They conclude that the inconsistent relationship between the two methods, sometimes significant, makes objective strength testing the preferable research design (Aitkens 1989). It remains to justify its use in the clinical management of patients with CP.

4.1.1.2.1 Dynamometry

Dynamometers provide objective information about muscle strength. This objectivity is what distinguishes instrumented strength testing from manual testing. They have been routinely used in clinical and research settings in adults. However studies involving children remain limited in number, more so in children with CP.

The instrumented systems for testing muscle strength include isokinetic dynamometers, hand-held dynamometers and cable tensiometers. These systems are able to assess muscle function, including strength, endurance and power under different conditions (isokinetic, isotonic, isometric). *Strength* is the maximum tension a muscle can exert during voluntary

action under specified conditions; *power* is the product of the tension created and the velocity of the action; *endurance* is the ability of a muscle to withstand fatigue and perform repetitive sub-maximal activation without significant decline in performance. An *isokinetic* contraction is a dynamic contraction, which takes place at a constant angular velocity. In an *isotonic* contraction, the speed of contraction is variable but the tension is fixed. An *isometric* contraction occurs when the resistance acting on the skeletal lever prevents motion. Finally, in a *concentric* contraction the muscle shortens and in an *eccentric* contraction the muscle lengthens (Jones 1996).

The concept of isokinetics was first described by Hislop and Perrine (1967). Isokinetic dynamometers provide an equal and opposite resistive torque throughout the range of movement at a constant angular velocity. Current systems are capable of measuring strength at an angular velocity of 500°/s. Earlier systems allowed only concentric contractions to be performed; today's systems measure concentric, eccentric and isometric contractions, as well as passive range of motion. Isokinetic testing in children has typically been performed in the range of 0-240°/s since it is considered to be safer. Ayalon et al (2000) reported isokinetic testing was reliable for knee flexors and extensors at 90°/sec, however Van den Berg-Emons (1996) concluded knee extension peak torque cannot be measured reliably at higher velocities (60 °/sec and 120 °/sec) probably due to greater impairment of agonist and antagonist muscle co-ordination. Therefore, an optimal testing velocity has yet to be defined especially for use in children with CP (Jones 2000).

Although, isokinetic dynamometry seems to offer a safe and controlled environment for application in children (Baltopoulos 1989), mainly because no resistance is applied when the movement stops (Osternig 1986), a number of limitations exist. While providing information on strength production in dynamic conditions, fast changes in muscle-tendon length have been shown to evoke a stretch reflex in children with spastic hypertonia during isokinetic dynamometry (Boiteau 1995). Further

limitations include the limited number of muscle groups that can be measured with the existing types of isokinetic machines i.e. quadriceps and hamstrings, their high cost, complexity and inability to be used quickly in a clinical setting since they are not portable. A minimum muscle strength is required since patients who are too weak, like patients with CP, may not be able to operate the machine (Van den Berg-Emons 1996). Finally, testing positions may require modification due to muscle shortening (Damiano 2002).

Hand-held dynamometers, as the name implies, are held, by the examiner. They can be used to assess isometric strength at individual joints. They cannot be used to monitor endurance or power. Available devices use a variety of force-detecting systems, including hydraulics, springs, load cells and strain gauges (Amundsen 1990). The force display can be in kg or Newton, but the lever arm distance (distance between joint centre and site of dynamometer application) has to be measured. Their product is the joint moment or torque (Nm). An examiner holds the dynamometer stationary while the subject exerts a maximal force against it (*make test*), or the examiner applies tension against the subject's maximal force until this is overcome (*break test*).

Make tests are considered safer in measuring strength in children since the stronger examiner does not overcome the child's maximal force and the possibility of injury is avoided (Bohannon 1987; Bohannon 1990; Bohannon 1997; Damiano 2002). This of course can be seen as a compromise since higher forces are associated with break tests because of eccentric contractions (Phillips 2000). On the other hand, "breaking" the resistance of a tested person with near normal strength capacity may be difficult. Although this can be overcome by stabilising the dynamometer to a fixed point, it still makes break tests not entirely appropriate. The subject may experience delayed muscle soreness and the method is contraindicated in the presence of unhealed or sutured tendons.

In a clinical setting, it is important to use a quick assessment method that is objective and provides meaningful results. Hand-held dynamometers have the advantage of portability and quick application to a variety of different muscle groups. Furthermore, muscle strength testing in children young in age, who possibly have learning or behavioural problems, can be a challenging task. Hand-held dynamometers are easier to use with this group of subjects. However, optimal joint angles for tension development remain still undetermined in children (Jones 2000).



Picture 4.3 Digital dynamometry set-up at the Oxford Gait Analysis Laboratory

(consent obtained)

Fixed-load cell systems or cable tensiometers measure isometric muscle strength of the most powerful muscles of healthy young people or athletes. Their advantage over the hand-held dynamometers is that the subject is exerting force against a stable fixed point. Hand-held dynamometers can only be used to assess muscles that generate less force than the examiner can resist. On the other hand, the cable tensiometer, which is suitable for measuring large forces, was not originally sensitive to low forces; this has now changed. Sensitive tensiometers currently exist and can be used for the assessment of patients. They are small in size, and combine hand-held dynamometry properties with the possibility of fixing the transducer to a

stable point for the assessment of strong muscle groups (MIE Medical Research Ltd).

4.1.1.2.2 Reliability of strength testing

Before dynamometry measurements can be considered useful, measurement methods should be shown to be reliable for the population to be measured (Riddle 1989). Reliability of the measurement tools is required in order to interpret accurately the effect of any intervention. The sources of variability and the degree to which they contribute to the overall measurement error must be identified. In tools used in physiotherapy, including dynamometers, the most common sources of error, are the examiner bias, inaccuracies in the instrument and variability of the subject tested (Portney 1993; Bohannon 1997). In CP patients with significant weakness, changes after an intervention are often subtle. Therefore highly reliable measures are required.

Keating and Matyas (1996) reviewed more than 200 articles to evaluate the effects of variation in subject factors and test procedures on clinical dynamometry measurements. They concluded that factors related to subjects that can affect measurements are age, gender, weight, athletic background, disability and, limb dominance. Test conditions that could lead to measurement-variability are range of movement within which values were obtained, type of contraction (concentric, eccentric, isokinetic, isometric, isotonic), pre-test procedures (warm-up, gravity correction, starting position, stabilisation, axes alignment, lever arm length), test conditions (speed, test sequence, rest intervals, feedback), and type of data analysis. According to Bohannon (1997) hand-held dynamometry should be performed only when specific conditions are applied: an appropriate device is used, testers are of adequate strength, make tests are used, gravity is eliminated and adequate stabilisation is secured.

During the last 30 years numerous studies have reported findings based on data from dynamometers. Most of these have used small sample sizes, giving insufficient description of testing protocols, which would be essential for accurate replication of the procedure. Despite this, there is abundant published information on a variety of related subjects: normative values from various types of subjects and muscle groups (Edwards 1977; Aniansson 1980; Backman 1989; Gleeson 1992; Backman 1995; Andrews 1996; Stoll 2000), quantification of exercise (Nelson 1990; Bishop 1991), evaluation of the effects of training or testing modes (Duncan 1989; Bishop 1991), testing or training speed (Bishop 1991) and duration of training (Carpenter 1991), investigation of the relationship between dynamometric and other measurements (Bohannon 1990), investigation of factors that correlate with measurements, and establishment of the relative efficacy of various treatment and training regimes (Carpenter 1991).

The reliability of isokinetic and hand-held dynamometry in healthy adults has been studied extensively (Markhede 1980; Nistor 1982; Bohannon 1987; Emery 1999; Holmback 1999; Phillips 2004), showing that both methods are highly reliable. Publications on the reliability of dynamometric measurements in adults with different neuromuscular disorders vary in their findings. Some claim high intra- and inter-rater reliability of the method used (Bohannon 1986; Bohannon 1987; Riddle 1989; Bohannon 1990; Kilfoil 1993; Holland 1994; Kilmer 1997), whereas others recognise that the reliability can be affected by the quantifying method or testing velocity (Hsu 2002). There are also studies that show relatively large variation of the strength values of patients with neuromuscular disorders (De Boer 1982; Lennon 1993).

Research on reliability of isokinetic measurements in healthy children and children with neuromuscular disorders is limited and results have been variable. Comparison between studies, as with most dynamometric reliability studies, is compromised by differences in design, methodology and

analysis of data (Molnar 1979; Weltman 1988; Burnett 1990; Merlini 1995). Pearson correlation coefficients between test and retest in young children range from 0.78-0.99, but up to 40% of variation is unaccounted (Merlini 1995). Intraclass correlation coefficient has been reported in the range of 0.49-0.84, with lower values in the hip abductors and adductors (Burnett 1990). The review of the literature suggests that the reliability of isokinetic dynamometry in children is not yet proven and requires further study.

Research on the reliability of hand-held dynamometry in children with or without, neuromuscular disorders is mostly limited to small sample sizes. Comparison between studies is similarly a problem (Hosking 1976; Wiles 1983; Stuberg 1988; Brussock 1992; Effgen 1992; Sloan 2002). Correlation coefficients for intra-rater reliability are in the range of 0.72-0.99 (Stuberg 1988). Lower inter-rater reliability is observed. However, hand-held dynamometry in children appears to be reliable, at least when the same examiner tests the same population under standardised procedures.

Evidence of reliability of dynamometry in CP children remains very limited. Only four studies have examined the reliability of isokinetic (Van den Berg-Emons 1996; Ayalon M 2000), hand-held (Taylor 2004) and fixed digital (Van der Linden 2004) dynamometric measurements in CP to date. Sample sizes in all the studies range from 10 to 12 patients, and only intra-rater reliability was assessed. Knee flexor and extensor strength was the strength variable measured in the isokinetic protocols (Van den Berg-Emons 1996; Ayalon M 2000), while Taylor et al (2004) assessed several muscle groups of the lower extremities. The two studies assessing the reliability of isokinetic measurement of knee muscle groups found high reliability as expressed with intraclass correlation coefficient (Ayalon M 2000) and r correlation coefficients (Van den Berg-Emons 1996); however their results were different for different angular velocities. Ayalon et al (2000) found high reliability at 90°/s of angular velocity, whereas Van den Berg-Emons et al (1996) found similar results at 30°/s. Taylor et al (2004) in their study of

test-retest reliability of hand-held dynamometry found that the method could reliably detect strength changes for groups of patients but not for individuals.

The reliability of dynamometry depends on factors such as the population measured and the technique used. Future research in muscle weakness in CP should provide information about the reliability of the specific method used to measure strength in this population, before conclusions about management are made.

4.1.1.3 Measures of function

A number of instruments are available to assess motor function in children with CP. Haley et al (1991) suggested that the development and use of paediatric functional measures were in their infancy and much work was needed to create efficient outcome measures for use in paediatric rehabilitation programmes.

According to the *International Classification of Impairments, Disabilities and Handicaps (ICIDH)* (1980), functional assessment measures are disability – oriented. Thus, they measure the functional capacities of a person (disability) instead of the failure of specific organs (impairment). Examples are: the *Peabody Developmental Motor Scales* (Folio 1983), which provides an indication of motor delay, the *Bleck Scale* (Bleck 1975) predicts ambulation at the age of seven years, and the *Gross Motor Function Measure* (Russell 1989) measures the magnitude of change in function over time or after treatment. Other measures used for children with CP are: the *Barthel Index* (Mahoney 1965), the *Bayley Scales for Infant Development (BSID)* (Bayley 1969), the *Paediatric Evaluation of Disability Inventory (PEDI)* (Haley 1992), and the *Functional Independence Measure for Children (WeeFIM)* (Msall 1994).

A recent review (Ketelaar 1998) of functional assessment measures in CP concluded that the GMFM and PEDI scales fulfill the criteria of reliability and validity with respect to responsiveness to change. The PEDI is a checklist, which should be completed by the parent or carer, while the GMFM is administered by a therapist or trained clinician. On this basis, it was decided to use the GMFM in this work.

The *Gross Motor Function Measure (GMFM)* is an observational measure of "how much" of a function a child can accomplish; it was developed and validated to assess quantitatively gross motor function and its changes in children with cerebral palsy (Russell 1989). The GMFM evaluates 88 items covering five motor domains including (a) lying and rolling, (b) sitting, (c) crawling and kneeling, (d) standing and (f) walking, running and jumping. A total score is calculated by averaging the scores of the five domains, which are expressed as a percentage. Normally, a five-year old healthy child could score 100%, without difficulty. The measure has been used for the documentation of the development of gross motor function in children with CP (Palisano 2000). It is well established for its reliability and validity and its responsiveness to change after intervention (Beckung 2000; Russell 2000).

Prediction of long-term function for a child with CP is difficult because factors other than the performance of specific motor abilities influence it. None of the above-mentioned measures has been developed for the purpose of long-term prediction of function. Another measure has recently been advocated for its predictive value, from the same team who developed the GMFM. The *Gross Motor Function Classification System (GMFCS)* (Wood 2000) is based on the concepts of abilities and limitations in gross motor function. It is designed for children up to 12 years of age and has five levels based on differences particularly related to walking and sitting. The measure has been validated and its prognostic value has been shown (Wood 2000; Rosenbaum 2002).

Another measure, which concentrates specifically on assessment of the ranges of functional walking or mobility skills, is the walking scale of the *Gillette Functional Assessment Questionnaire (FAQ)* (Novacheck 2000). This is a 10-level parent or guardian report, which describes a range of walking abilities from non-ambulatory to ambulatory in different community settings and terrains. Good test-retest reliability between parents and good inter-rater reliability between parents and community caregivers was shown, as well as high validity. The scale was used in this work as a report of the parents' impression of their child walking ability at different stages of the study.

4.2 Summary

The diplegic gait is the result of a complex interaction between the damaged central nervous system and the musculoskeletal one in the growing child. Research on how specific impairments, such as spasticity or weakness, contribute to gross motor function limitations is on-going. Our understanding can be improved by using accurate assessment methods to investigate the natural history and the effects of treatment on gait and function. Gait analysis, dynamometry and functional scales are available and provide useful information. Further research is required to improve the existing evidence, the reliability of the above methods and their applications in order to improve the management of patients with spastic diplegia.

4.3 Section Two outline

In Section One, background information was provided about muscle weakness in cerebral palsy and its debilitating effect on function. The literature was explored in order to assess the effect that multi-level orthopaedic surgery may have on strength and function, as well as the role of resistance strengthening exercise as part of a rehabilitation programme; areas of uncertainty and debate were identified.

4.3.1 Synopsis of literature review

The key points from the literature review included:

1. Cerebral palsy is a diverse clinical entity where further research is required on the prognosis, early diagnosis, appropriate management, and evaluation of treatment outcome.
2. The diplegic gait is the result of a complex interaction between the central nervous system and the musculoskeletal unit in the growing child.
3. Gait analysis, dynamometry and functional scales are available outcome measures and provide useful information. The reliability of these outcome measures, however and their clinical application in patients with spastic diplegia are still subjects of research.
4. There is evidence in the literature to suggest that muscle weakness in children with spastic diplegic CP contributes to their disability.
5. Research on how specific impairments, such as spasticity or weakness, contribute to gross motor function limitations is on going.
6. The natural history of muscle weakness in spastic diplegia during growth is largely unknown.
7. The feasibility to both measure muscle force and to strengthen muscle has been demonstrated.
8. Research in the reliability of measuring muscle strength in children with CP remains limited.
9. The effect of multi-level orthopaedic surgery on muscle weakness is not well documented.
10. There are no universally accepted treatment techniques for effectively improving muscle strength in these children after single-stage multi-level orthopaedic surgery.

4.3.2 Overview of the study

In Section Two, the design, method, analysis and results from different parts of the study are presented in four separate Chapters. Chapter Five reports on the pilot phase of the study. This involved repeated strength measurements of the lower limb muscle groups in healthy children and

children with spastic diplegic CP. Chapter Six presents the results from the six month post-operative assessment of children with CP. The effects of surgery on strength, gait and function are reported. Chapter Seven presents the results following the administration of the six week strengthening programme in two randomly selected groups of surgical patients. The two regimes are compared for their effectiveness in improving strength, gait and function. Chapter Eight summarises the changes in strength, gait and function one year after entering the study. Conservatively and surgically treated patients are analysed separately. Chapter Nine summarises the results of this study and discusses its limitations. It also identifies the clinical significance of the findings and the implications for treatment recommendations. All the relevant documentation is presented in appendices three to six.

SECTION TWO

Chapter Five

5. Reliability of strength testing protocol

Manual muscle testing has been the clinical standard for measuring strength over the years. However, distinction between different grades is not always reliable and minimal strength changes are not always detected. Therefore, it has been suggested that quantitative methods should be used instead (Mendell 1990). These methods should demonstrate sufficient reliability for clinical or research application.

This Chapter describes the pilot study, which was designed to test the reliability of the lower limb isometric strength measurement protocol used in the main research project, which is presented in the following Chapters.

The literature suggests that children with CP are weaker than children with no neuromuscular or other pathology and a few studies have demonstrated the distribution of weakness in these patients (Bourbonnais 1989; Wiley 1998). In order to present these differences between CP and non-CP children as well as to demonstrate whether CP children can reproduce a muscle contraction in the same manner as healthy children, a control group of children with no neuromuscular or other condition was included in the protocol.

The majority of studies assessing muscle strength following strength training programmes in CP have used either isometric or isokinetic measurements. Both methods have been shown to be reliable in healthy children (Weltman 1988; Merlini 1995; Sloan 2002), children and adults with neuromuscular disease (Burnett 1990; Holland 1994; Kilmer 1997) and adults with neurological disorders, mainly stroke (Riddle 1989). However,

there is limited evidence of the reliability of strength testing specifically in children with CP. The small number of studies on this subject used different protocols (Van den Berg-Emons 1996; Ayalon M 2000; Taylor 2004; Van der Linden 2004).

Isokinetic dynamometry allows measurement of muscle performance during limb movement at a constant angular velocity, with variable resistance throughout the range to match the applied force. While providing information on strength production in dynamic conditions, its use in this work was not considered appropriate. The reasons for this were discussed in Chapter Four and they will be explained here in brief.

As discussed in Chapter 4.1.1.2, fast changes in muscle-tendon length have been shown to evoke a stretch reflex in children with spastic hypertonia during isokinetic dynamometry (Boiteau 1995). Indeed, optimal testing velocity has not yet been defined in these patients (Jones 2000). Isokinetic dynamometers measure only a small number of muscle groups; especially in children with CP testing positions may need modifications due to muscle shortening (Damiano 2002). Their cost remains high and they cannot be considered a quick and portable clinical tool. More importantly, a minimum muscle strength is required to perform the test and weak patients with CP, may not be able to operate the machine (Van den Berg-Emons 1996).

The hand-held technique is recommended for measuring weakness rather than near normal strength since, in practice, the counter-force that can be applied by the examiner is likely to be limited to 25-30 kg (Van der Ploeg 1991). When hand-held dynamometers are modified and fixed to a stable point, they can also be used for measuring stronger muscle groups. Although, they test isometric strength at isolated joint positions and cannot be used to monitor endurance or power, their advantages over isokinetics were considered greater. Therefore they were preferred for use in this work.

As discussed in Chapter 3.2 muscle co-contraction is the simultaneous activity of agonist and antagonist muscles crossing the same joint and acting in the same plane and, thereby increasing the stiffness at a joint (Olney 1985). Patients with central nervous system damage, such as children with CP, demonstrate excessive co-contraction (Ikeda 1998) which can increase the energy cost of movement (Damiano 1993) and could potentially affect muscle strength performance. To test this hypothesis, surface EMG activity data from the quadriceps and hamstrings muscle groups were obtained during isometric flexion and extension contractions.

5.1 Design of the study

This was a prospective cohort study where children with spastic diplegia were recruited and children with no neuromuscular disorder were invited to participate. A protocol was developed for testing isometric muscle strength of the hip and knee muscle groups with a modified hand-held dynamometer. The intra- and inter-rater reliability was assessed. Electromyographic signals were also recorded from the knee extensor and flexor muscle groups during isometric contractions to observe differences of co-contraction between CP and normal children. Ultimately the goal was to establish an objective assessment method of muscle strength testing in CP, and an outcome measure for strength training during post-operative rehabilitation.

5.1.1 Subjects

According to the protocol, 10 children aged five to 16 years (mean 10 years 9 months, SD 3 y) participated in the study. Five children were diagnosed with spastic diplegic CP and five children did not have a history of neuromuscular disorder. Their characteristics are presented in table 5.1. All participants with CP were patients, seen at the Nuffield Orthopaedic Centre NHS Trust, and they had not undergone orthopaedic surgery or Botulinum toxin injections to the lower limbs within the year prior to the study.

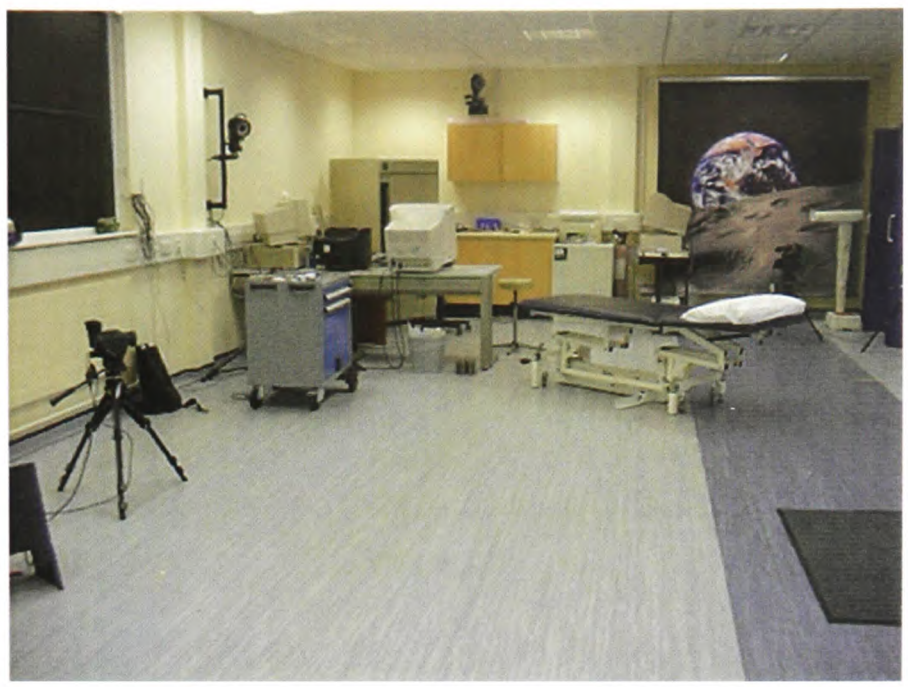
Healthy siblings and children of staff members were invited to the study. These children provided a control group for comparison with the CP children when lower limb muscle strength and performance were assessed. The study was approved by the Oxfordshire Ethics Committee and informed consent was obtained by the parents or guardians.

Participant	*Sex*	*CP/H*	*Age (y/m)*	*Weight (kg)*	*Height (m)*	*GMFCS*	*Hand Dominance*
1	M	CP	11.11	39,5	1.42	II	R
2	F	CP	11.6	28	1.37	II	R
3	F	CP	9.3	45	1.32	I	R
4	F	CP	14.1	32,5	1.42	II	R
5	M	CP	7.4	31	1.25	I	R
6	F	H	15.1	66	1.69	N/A	R
7	M	H	15.1	78	1.62	N/A	R
8	F	H	11.7	42,5	1.50	N/A	L
9	F	H	6	17,3	1.10	N/A	L
10	M	H	8.11	34	1.43	N/A	L
Mean (sd)			**10.9 (3)**	**41.4 (18.2)**	**1.4 (0.2)**		

Table 5.1 Participants' characteristics (M=male, F=female, CP=cerebral palsy, H=healthy, R=right, L=left)

5.1.2 Method

Clinical examination, dynamometric measurements and electomyographic recordings were collected by two clinicians at the Oxford Gait Analysis Laboratory.



Picture 5.1 Oxford Gait Analysis Laboratory

A standard clinical examination was performed to record range of motion, joint deformities, spasticity, selective muscle control, and muscle strength on the Medical Research Council (MRC) scale in the children with spastic diplegia.

Isometric muscle strength was measured in five muscle groups of both lower extremities of all 10 children. These were the hip flexors, extensors, abductors, knee flexors, extensors at 90° and 30° (dynamometer model MIE Medical Research Ltd, Leeds, UK). Standardised positions were used with a set up that was considered suitable for testing the larger forces seen in the knee extensors muscle groups. Calibration trials using known weights were performed at the beginning of the session to ensure validity of the dynamometer readings.

Muscle activity during isometric contractions was recorded using surface electrodes in order to monitor muscle activity in an attempt to minimise the influence of the antagonists (model MA100, Motion Lab Systems, Baton Rouge, USA).

5.1.2.1 Choice of muscle group

The muscle groups tested were the following:

1. Hip flexors
2. Hip extensors
3. Hip abductors
4. Knee flexors
5. Knee extensors at 90° of knee flexion
6. Knee extensors at 30° of knee flexion

These lower limb muscle groups were chosen based on:

(a) Their known positive response to strengthening programmes as cited in the literature (Horvat 1987; Damiano 1995; Damiano 1998)

(b) Clinical experience of muscle groups, which are sufficiently strong to cope with a progressive resistance exercise programme

(c) Published evidence about the effect of surgery on muscle strength. The literature suggests that when the spastic agonist is lengthened, the antagonist should be strengthened (Reimers 1990a; Reimers 1990b). The clinical significance of muscle groups known to be functionally relevant in spastic diplegia was also taken into account: the hip flexors are important power generators during gait; the hamstrings, in addition to being knee flexors, are used to augment hip extensor power. Strengthening of these muscles, even following their lengthening, was considered essential and they were included in the testing protocol.

Considering the lack of evidence on the effects of long term use of orthotics on muscle strength, combined with the fact that gait analysis post-operatively would have been confounded by their use, a decision was made against testing (or strengthening) of the foot plantarflexors and dorsiflexors, despite their importantance in gait (Gage 1991).

5.1.2.2 Choice of strength testing positions

A standard order of testing positions was used as presented in Table 5.2. These positions were based on the strength measurement literature, the specific characteristics of patients with CP, the modifications of the

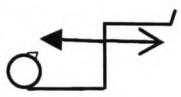
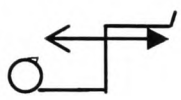
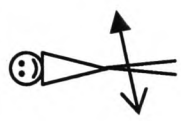
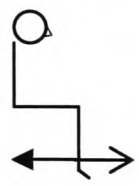
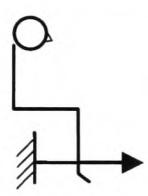
dynamometer used in this protocol and for comparison with other studies. In particular:

(a) According to Hosking (1976) and Bohannon (1990) when a hand held dynamometer is used, the examiner should stabilise it to allow joint angles not to change between measurements and to provide adequate fixation of surrounding joints in order to prevent substitute movements.

(b) Measurements should be taken in gravity-eliminated positions, with the tested muscle group placed in a specific joint position or muscle length (preferably the middle of its length range) and the transducer of the dynamometer applied perpendicularly to the limb segment (lever arm) (Bohannon 1990; Andrews 1996).

(c) Joint angles should allow for existing contractures and limitations of the range of motion (Stuberg 1988) (e.g hip abductors measured at 30° of hip flexion).

(d) The 30° knee extension position was thought to be important since it gave an estimation of force near full knee extension, which would be relevant during crouch gait (Damiano 1995; Damiano 1995).

Figure	*Muscle group*	*Subject position*	*Lever arm*	*Dynamometer position*
	Hip flexors	Supine	Greater trochanter to thigh cuff	Hip and knee flexed to 90°. Cuff around distal thigh proximal to femoral condyle axis. Transducer is held so that hip flexion is resisted.
	Hip extensors	Supine	Greater trochanter to thigh cuff	Hip and knee flexed to 90°. Cuff around distal thigh proximal to femoral condyle axis. Transducer is held so that hip extension is resisted.
	Hip abductors	Supine	Greater trochanter to thigh cuff	Hip flexed to 30° with neutral abduction. Cuff is attached around distal thigh proximal to femoral condyle axis. Transducer is held so that hip abduction is resisted.
	Knee flexors	Sitting	Femoral condyle axis to leg cuff	Hip and knee flexed to 90°. Cuff around distal leg proximal to malleoli. Transducer is held so that knee flexion is resisted.
	Knee extensors 90°	Sitting	Femoral condyle axis to leg cuff	Hip and knee flexed to 90°. Cuff around distal leg proximal to malleoli. **Transducer is fixed to a stable point so that knee extension is resisted.**
	Knee extensors 30°	Sitting	Femoral condyle axis to leg cuff	Hip flexed to 90° and knee to 30°. Cuff around distal leg proximal to malleoli. **Transducer is fixed to a stable point so that knee extension is resisted.**

→ Direction of active muscle contraction, //// fixed point, → direction of resistance

Table 5.2 Subject and dynamometer positions

5.1.2.3 Procedure

Two examiners completed the testing in three visits held at least one week apart. Each examiner tested the child independently and there was a 15 min interval between them. The author tested all children for the duration of the study to allow the intra-rater reliability to be assessed. The second examiner measured a different muscle group at each one of the three visits for the inter-rater reliability to be assessed. The reason for limiting the measurements taken by the second examiner was to keep the duration of the procedure to a minimum and preserve the cooperation and motivation of

the child. A third examiner was introduced for one CP and one healthy child because the second examiner was unable to attend these sessions. All examiners were blind to each other's results. Children continued with their usual activities during this period but resistance strengthening exercises were discouraged.

Prior to testing, the procedure was explained to the child and a few trials were performed to familiarise him/her with the dynamometer and the examiners.

Age, gender, weight, height and limb dominance were recorded. In the CP children, routine clinical examination was performed to measure the range of joint motion in the legs bilaterally. Muscle strength on the MRC scale, spasticity using the modified Ashworth scale and selective control were also recorded.

5.1.2.3.1 Preparation for the muscle test

(a) The child was as free as possible from discomfort or pain for the duration of each test.

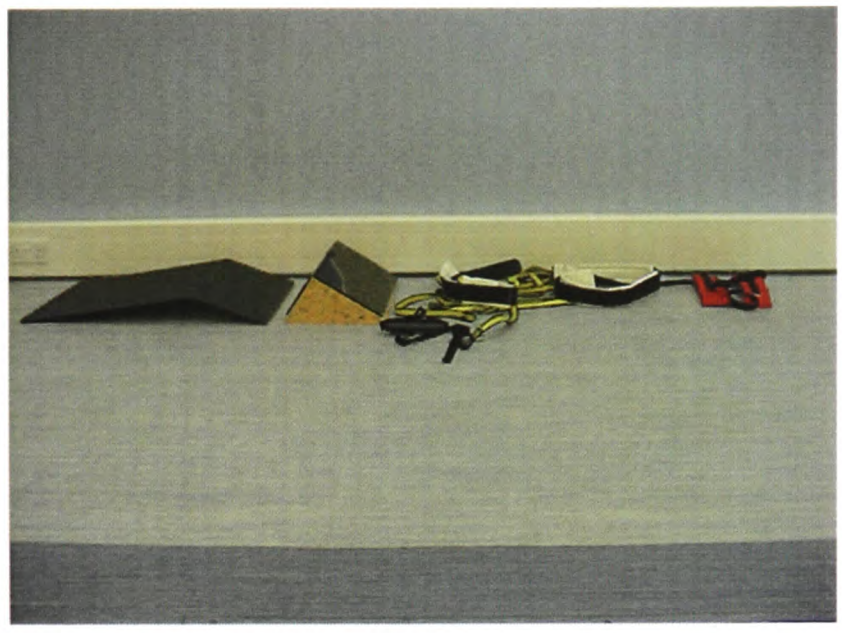
(b) The environment was quite with comfortable temperature.

(c) The table for testing was firm (minimally padded), with minimum friction. The height of the table was adjustable to allow the examiner to use proper leverage and body positioning.

(d) Patient position was carefully planned, so that position changes were few. Every position allowed adequate stabilisation of the part being tested by virtue of body weight, help by other person and straps.

(e) Electrodes were placed at specified landmarks on the lower limbs in order to record the electromyographic signals simultaneously with the isometric contractions.

(e) Materials needed for the test were at hand before the start of the test (documentation forms, pen or computer terminal, measurement equipment, pads, wedges, goniometer, straps, reference material).



Picture 5.2 Strength measurement equipment

(from left to right: wedge for testing knee extensors at 30°, wedge for testing hip flexors, straps to secure stabilisation and attach the transducer of the dynamometer)

Straps of different sizes were made at the Orthotics department of NOC which were used for:

(a) Stabilisation of the pelvis both in supine and sitting positions

(b) Fixation of the transducer of the dynamometer to a stable point under the examining table to test the knee extensors muscle group at 90 ° and 30°

5.1.2.3.2 Dynamometry

Isometric muscle strength of hip flexors, hip extensors, hip abductors, knee flexors and extensors of both lower extremities was tested using a digital dynamometer (MIE Medical Research Ltd, Leeds, UK). The analogue signal was captured on a personal computer at 1000 Hz. The digital unit of the dynamometer displayed the results in Newton (N) up to a maximum of 1000

N. It held and displayed the maximum voluntary contraction (MVC). The device was calibrated before each session using a known weight.

The participants were advised to pull or push as hard as they could. There is no consensus in the literature about the duration of an isometric contraction, the interval between repetitions and the rest time between muscle groups. Duration of contractures between two and seven sec have been reported (Backman 1989; Bohannon 1990; Andrews 1996). It was found in this study that the total duration of each attempt had to last seven to 10 sec to allow children with CP enough time to fully activate the correct muscle group. Isometric make tests were used since they were considered safer, as discussed in Chapter 4.1.1.2. Children were encouraged to built their force gradually to a maximum voluntary effort and then maintain this for five sec. Three consecutive repetitions were recorded with a 15-sec interval between repetitions. The dynamometer was set up to save the maximum voluntary contraction (MVC) provided that both performance and graphical representation showed an adequate attempt. An attempt was considered adequate when the child performed the contraction without a "kick" or a jerky movement at the start and he/she was able to produce a force plateau. This was represented graphically as shown in Graph 5.2. One-minute rest was allowed between different muscle tests, during which subject and dynamometer positions were changed. Consistent verbal instructions and encouragement were given by the examiners.
The protocol of dynamometric measurements was the following:

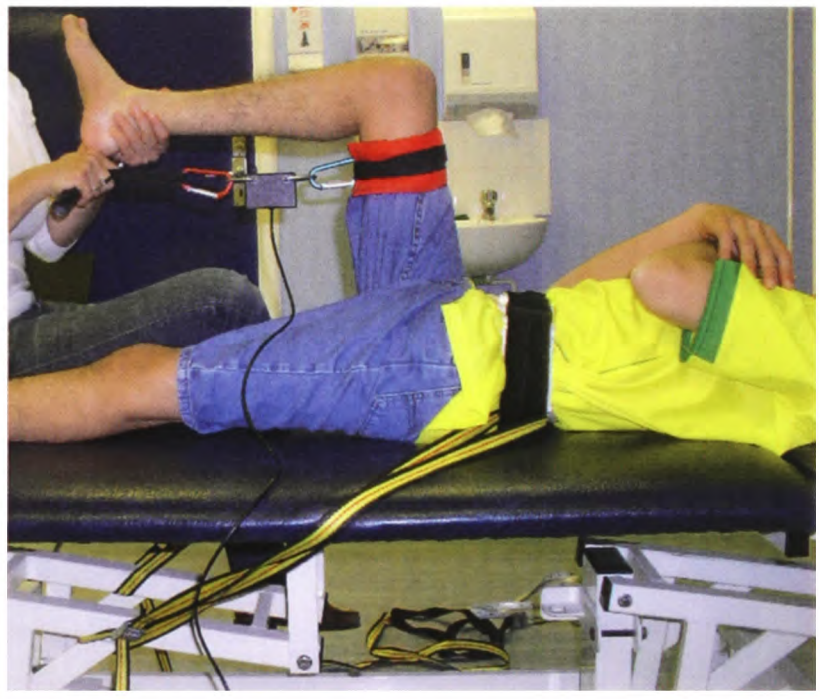
HIP FLEXION (Psoas major & Iliacus)
Position of patient - stabilisation: Supine. Test limb supported by the examiner with hand on the posterior surface of leg. Hip and knee joints flexed at 90°. Pelvis is stabilised on table firmly by waist straps.
Position of examiner and dynamometer: Place the strap around the subject's thigh distally and proximal to femoral condyles. Force is applied to the anterior surface of the distal thigh. The examiner is sitting at the side of the

patient holding the other end of the strap with the transducer vertically to the distal thigh and parallel to the horizontal (ground). Ask the subject to apply a small load so as to tighten the straps. Adjust the other strap to position the joint in 90° of hip flexion. Measure the distance between the centre of the strap and the hip joint (greater trochanter marker).

Test: Patient attempts to flex the hip while maintaining the leg in neutral rotation. Patient exerts maximum force against the dynamometer without flexing the hip (isometric contraction).

Instructions to Participant: "Bring your knee up to your nose. Pull hard. Very well!"

Helpful hints: Be careful of sartorius substitution (external rotation and abduction of hip), tensor fascia latae substitution (internal rotation and abduction of the hip normally, but if the patient is tested in supine gravity will cause the limb to externally rotate), rectus femoris substitution (knee extension). When trunk is weak, supine position is more accurate.



Picture 5.3 Hip flexors

HIP EXTENSION (to isolate Gluteus maximus)

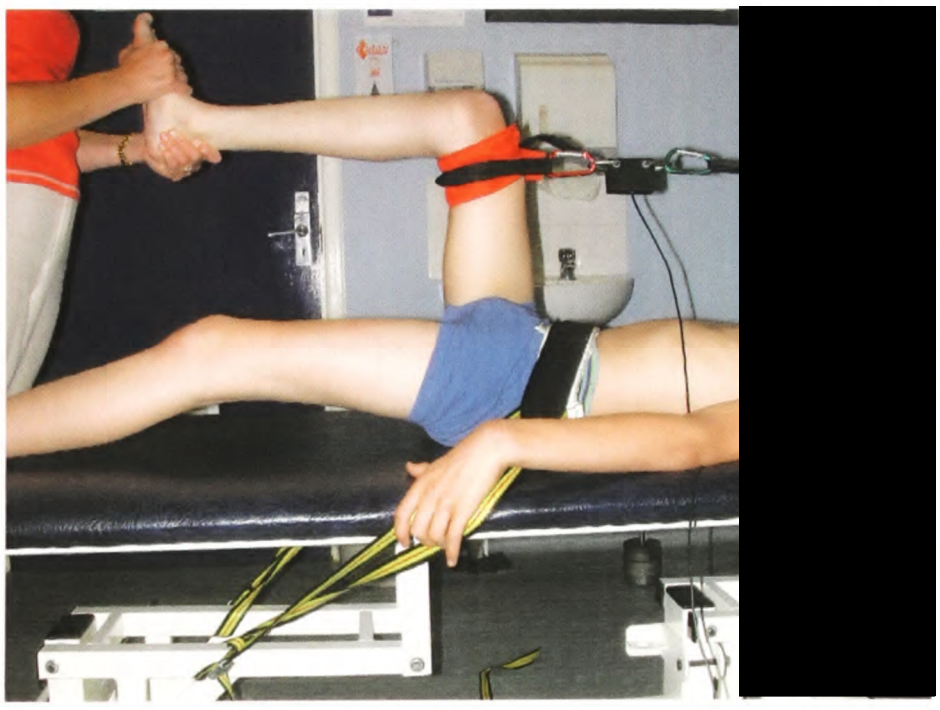
Position of patient - stabilisation: Supine. Test limb supported by the parent/guardian with hand on the posterior surface of leg. Hip and knee joints flexed at 90°. Pelvis is stabilised on table firmly by waist straps.

Position of examiner and dynamometer: Place the strap around the subject's thigh distally and proximal to femoral condyles. Force is applied to the posterior surface of the distal thigh. The examiner is standing at the head of the patient holding the other end of the strap with the transducer vertically to the distal thigh and parallel to the horizontal (ground). Ask the subject to apply a small load so as to tighten the straps. Adjust the other strap to position the joint in 90° of hip flexion. Measure the distance between the centre of the strap and the hip joint (greater trochanter marker).

Test: Patient attempts to extend the hip while maintaining the leg in neutral rotation. Patient exerts maximum force against the dynamometer without extending the hip (isometric contraction).

Instructions to Participant: "Straighten your hip. Pull hard. Very well!"

Helpful hints: Keeping the knee in extension will test all hip extensor muscles; with the knee flexed, the isolated gluteus maximus will be evaluated.



Picture 5.4 Hip extensors

HIP ABDUCTION (Glutei medius and minimus)

Position of patient - stabilisation: Supine. Pelvis is stabilised on the examining table firmly by the waist straps. To minimise friction, a smooth sliding board is placed under the leg. No support of the leg under the ankle

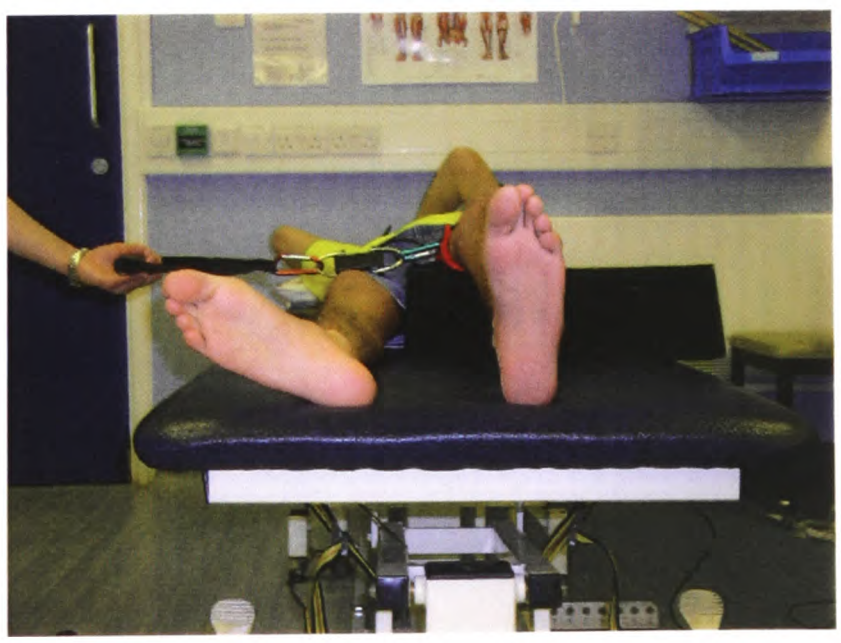
is necessary by the examiner. A foam wedge is placed underneath thighs creating 30° of hip flexion.

Position of examiner and dynamometer: Place the strap around the subject's thigh distally and proximal to femoral condyles. Force is applied to the lateral surface of the distal thigh. The examiner is standing at the bedside holding the other end of the strap vertically to the distal thigh and parallel to the horizontal (ground). Ask the subject to apply a small load so as to tighten the straps. Measure the distance between the centre of the strap and the hip joint (greater trochanter marker).

Test: The child attempts to abduct the hip without flexing it or rotating it in either direction. The child exerts maximum force against the dynamometer without abducting the hip (isometric contraction).

Instructions to participant: "Bring your leg out to the side. Pull hard. Very well!"

Helpful hints: Be careful for substitutions. Tensor fascia latae substitution (when the test is allowed to begin with the hip in flexion or with active hip flexion). Hip flexors oblique action substitution (flexion and external rotation during the motion of abduction). Hip-like substitution (pelvis motion towards thorax using the lateral trunk muscles).



Picture 5.5 Hip abductors

KNEE FLEXION (All hamstring muscles)

Position of patient - stabilisation: Short sitting with the thighs fully supported on the table and the legs hanging over the edge. The femur must be maintained in the horizontal position. This is ensured by aligning the distance between the femoral condyles with the edge of the table. Hands rest on table on either side of the body for stability, or may grasp the table edge. Pelvis is stabilised firmly with straps. A parent or guardian is allowed to hold the trunk of a CP child if balance is lacking.

Position of examiner and dynamometer: Place the strap around the subject's leg distally and above the malleoli. Force is applied to the posterior surface of distal leg. The examiner is sitting in front of the patient holding the other end of the strap vertically to the leg and parallel to the horizontal (ground). Ask the subject to apply a small load so as to tighten the straps. Adjust the other strap to position the knee joint in 90° of flexion. Measure the distance between the centre of the strap and the knee joint (femoral epicondyles marker).

Test: The child attempts to flex the knee while maintaining the leg in neutral rotation (measuring all hamstring muscles). The child exerts maximum force against the dynamometer without flexing the knee (isometric contraction).

Instructions to participant: "Bent your knee, don't let me straighten it. Pull hard, very well!"

Helpful hints: Be careful for hip flexion substitution, sartorius substitution (causes flexion and external rotation of the hip), gracilis substitution (causes hip adduction motion), gastrocnemius substitution (the patient uses strong plantarflexion). If the biceps femoris is stronger than the medial hamstrings, the leg will rotate externally during knee flexion. The opposite happens if the medial hamstrings are stronger.



Picture 5.6 Knee flexors

KNEE EXTENSION (Quadriceps muscles are tested together as a functional group in 90° and 30° of knee flexion)

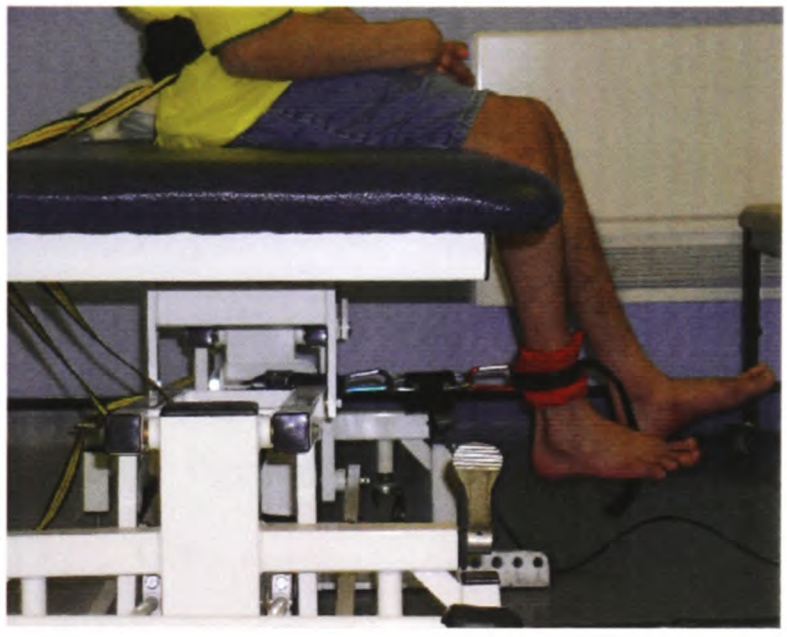
Position of patient - stabilisation: Short sitting with the thighs fully supported on the table and the legs hanging over the edge. The femur must be maintained in the horizontal position. This is ensured by aligning the distance between the femoral condyles with the edge of the table. Hands rest on table on either side of the body for stability, or may grasp the table edge. Pelvis is stabilised firmly with straps. A parent or guardian is allowed to hold the trunk of a CP child if balance is lacking.

Position of examiner and dynamometer: Place the strap around the subject's leg distally and above the malleoli. Force is applied to the anterior surface of distal leg. The transducer is fixed through the strap under the examining table, vertically to the leg and parallel to the horizontal (ground). Ask the subject to apply a small load so as to tighten the straps. Adjust the other strap to position the knee joint in 90° of flexion. Measure the distance between the centre of the strap and the knee joint (femoral epicondyles marker). For the 30° test place the subject on the modified wedge and adjust the dynamometer to the floor. Make sure that it is positioned vertically to the leg. Follow the same procedure as with the 90° test.

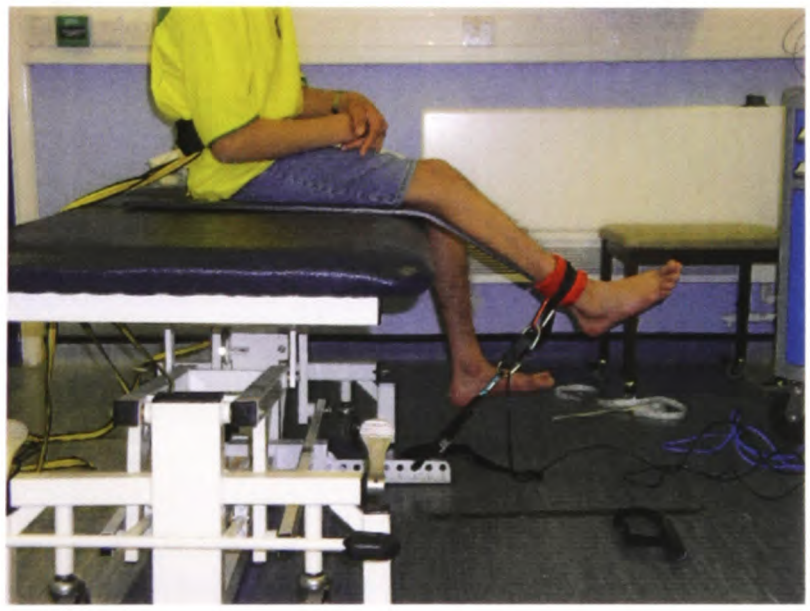
Test: The child attempts to extend the knee while maintaining the leg in neutral rotation. The child exerts maximum force against the dynamometer without extending the knee (isometric contraction).

Instructions to participant: "Straighten your knee. Pull hard! Very well."

Helpful hints: Be careful for activation of hip internal rotators to substitute for the quadriceps femoris.



Picture 5.7 Knee extensors at 90°



Picture 5.8 Knee extensors at 30 °

5.1.2.3.3 Electromyography

In order to monitor muscle activity during isometric contractions and to minimise the influence of the antagonists in children with CP,

electromyographic signals from the knee flexors and knee extensors were recorded. EMG signals were sampled at 1000 Hz (system MA100, Motion Lab Systems, Baton Rouge, USA). The placement of the surface electrodes was undertaken according to the recommendations of SENIAM working group on EMG (Hermens 1997).

	EMG ELECTRODE – placement
Rectus Femoris	Sitting with knees in slight flexion, electrodes placed at 50% on line between ASIS – superior patellar border in the direction of the line.
Vastus Medialis	Sitting with knees slightly flexed, electrodes placed at 80% on line between ASIS – in front of anterior border of medial ligament in knee joint, almost perpendicular to this line.
Medial hamstrings (Semitendinosus)	Prone with knee flexed < 90 degrees and extremity medially rotated. Electrodes placed at 50% on the line between ischial tuberosity and medial epicondyle of the tibia in the direction of the line.

The EMG set is presented in picture 5.9



Picture 5.9 Electromyography set up

5.1.3 Analysis

The purpose of the analysis was two-fold: to determine the reliability of the strength testing protocol and to demonstrate any differences of the effect of co-contraction during isometric testing on performance between CP and healthy children. Relevant data files are presented in appendix five.

5.1.3.1 Processing of data

Muscle strength data were transferred from the data sheets to electronic form and then checked for missing values. The graphical representation of the isometric contractions was studied initially in Polygon (version 2.4, Vicon 612, Motion Systems, Oxford, UK), a commercial type of gait analysis software. Isometric muscle strength was collected in units of force (N) and then expressed in moments (Nm). For their calculation, the distance between the middle of the strap and a specified anatomical landmark was measured with a measure tape. Finally moments were normalised to body mass (Nm/kg).

The literature suggests that factors such as weight, age, sex or height, alone or combined, may affect performance. According to Backman et al (1989), the best predictor of muscle force is the combination of age and weight. There is no consensus on normalisation and different investigators have used different factors (Edwards 1977). Due to the small size of the sample and in order to allow comparison with previous strength testing protocols, it was decided to normalise moments to body weight only. Mean peak moments and normalised mean peak moments for healthy and CP children were calculated using the peak value from every visit for all tested muscle groups for right and left sides.

Similarly, EMG data were transferred to excel spreadsheets and combined with the graphical representation of the signal, they were analysed. A 70% window of maximum moment value (Nm) was chosen in order to quantify a co-contraction index (CI) for the rectus femoris and medial hamstrings.

5.1.3.2 Reliability

"Measurement error is the variation between measurements of the same quantity on the same individual because of natural variation in the subject, variation in the measurement process, or both" (Bland 1996). This error indicates whether the observed / measured change represents a real change in a patient's condition or not. In order to quantify the measurement error, repeated measurements on several subjects are needed.

An index of measurement error that provides a direct clinical interpretation is the within-subject standard deviation or σ_w (Bland 1996).

Another commonly used method is the correlation coefficient between repeated measurements, which is also known as the reliability of the measurement method or test-retest reliability (Bland 1996). The intra-class correlation coefficient or **ICC** has often been used in the strength testing literature.

Both the above indices were used in this work: the within-subject standard deviation was selected for its clinical usefulness and the ICC for comparison with other studies.

5.1.3.2.1 Within - subject standard deviation

The intra-rater reliability was calculated using the method described by Bland and Altman (1996; 1996). The standard deviation of repeated measurements on the same subject indicates the size of the measurement error. If we assume that this standard deviation is the same for all subjects then we obtain the **within-subject standard deviation** or σ_w.

It is important to check whether the σ_w is related to the magnitude of the measurement, if so, a logarithmic transformation of the data is essential. There was indeed such a relationship in the data when values were

expressed as Nm. This was not the case however when the same values were normalised to body mass (Nm/kg). Therefore the mean peak normalised moments (Nm/kg) for each muscle group were used in the calculation of σ_w.

The right and left sides of the body were analysed initially separately and then in combination. The **limits**, which should include the subject's mean for 95% of observations were constructed (Bland 1996). According to the same authors, another useful way of presenting measurement error is the **repeatability** or $2.77*\sigma_w$, where the difference between two measurements for the same subject is expected to be less than this value for 95% of pairs of observations. If not, then the difference should be attributed to factors other than measurement error (e.g. treatment). The variability of the measurements for each muscle group was also expressed as the ratio of standard deviation to the mean [% (σ_w x mean^{-1})], a quantity known as **coefficient of variation (CV)**.

5.1.3.2.2 Intraclass Correlation Coefficient

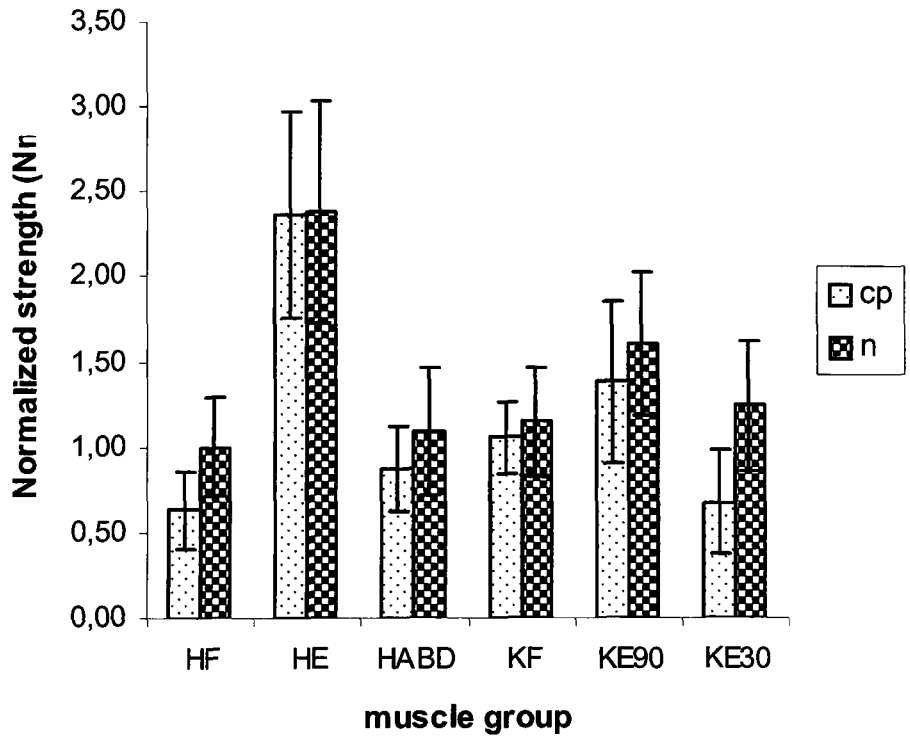
The **intra-class correlation coefficient (ICC)** is a reliability index for measurements subject to measurement error. There are several different types of ICC's in common use, usually referred to as ICC(1,1), ICC(2,1), ICC(3,1), ICC(1,k), ICC(2,k) and ICC(3,k). The definitions were provided by Shrout and Fleiss (1979) and have been used in strength testing studies. Unfortunately these definitions were designed for completely balanced data, and were based on the sums of squares from ANOVA tables. In the presence of severe imbalance in the data, or of covariates affecting the measurement these formulae cannot be used.

The set of data discussed here consisted of measurements of strength for six muscle groups, on each of the left and right sides, on five patients with

cerebral palsy and five control subjects. The study design was to repeat each measurement on each of three visits, and for some visits to repeat the measurements for two raters. The final data are markedly unbalanced. Therefore, the problem was approached via linear mixed models. It was expected that the strength measurements would be influenced by the size of the subjects, so this needed to be adjusted for in the model. One of the problems was that there was information for only one rater for hip flexors, knee extensors and knee flexors therefore it was not possible to estimate a between-rater variance for these groups.

5.2 Results

Mean peak moments ranged between 22.24 to 81.43 Nm and 51.33 to 119.70 Nm for all muscle groups in CP and healthy children respectively. The normalised mean peak moments were in the range of 0.63 to 2.36 and 1.00 to 2.38 Nm/kg for all muscle groups in CP and healthy children respectively (Table 5.3, Graph 5.1).



Graph 5.1 Normalised muscle strength per muscle group and subject group

Muscle group	**CP**	**Healthy**	**CP**	**Healthy**
	MPM (SD) Nm	**MPM (SD) Nm**	**Normalised MPM Nm/kg (SD)**	**Normalised MPM Nm/kg (SD)**
Hip flexors	22.24(9.45)	51.33(27.74)	0.63(0.22)	1.00(0.29)
Hip extensors	81.43(22.16)	119.70(59.61)	2.36(0.61)	2.38(0.65)
Hip abductors	30.66(10.63)	55.83(31.80)	0.87(0.25)	1.09(0.37)
Knee flexors	37.24(9.81)	59.65(34.81)	1.05(0.21)	1.14(0.32)
Knee extensors	48.65(18.14)	81.19(41.59)	1.38(0.47)	1.60(0.42)
Knee extensors 30°	23.74(13.13)	65.35(38.40)	0.68(0.30)	1.24(0.38)

Table 5.3 Mean peak moments (MPM), normalised mean peak moments in CP and healthy children for all muscle groups

The patient group was generally weaker than the healthy group as expected in all muscle groups apart from hip extensors (Table 5.3).

Muscle group	**CP**	**Healthy**	**CP**	**Healthy**	**CP**	**Healthy**
	σ_w	σ_w	**Repeatability**	**Repeatability**	**CV%**	**CV%**
Hip flexors	0.23	0.29	0.64	0.82	36.54	29.50
Hip extensors	0.63	0.67	1.75	1.86	26.73	28.19
Hip abductors	0.25	0.40	0.69	1.10	28.62	36.58
Knee flexors	0.23	0.33	0.64	0.91	22.13	28.96
Knee extensors	0.51	0.43	1.42	1.20	37.16	27.17
Knee extensors 30°	0.15	0.39	0.41	1.09	21.81	31.65

Table 5.4 Within-subject standard deviations σ_w, limits (repeatability) and coefficient of variation (CV%) for all subjects and muscle groups

The within-subject standard deviations σ_w and limits, which include the subject's mean for 95% of observations, showed that, in comparison, CP and

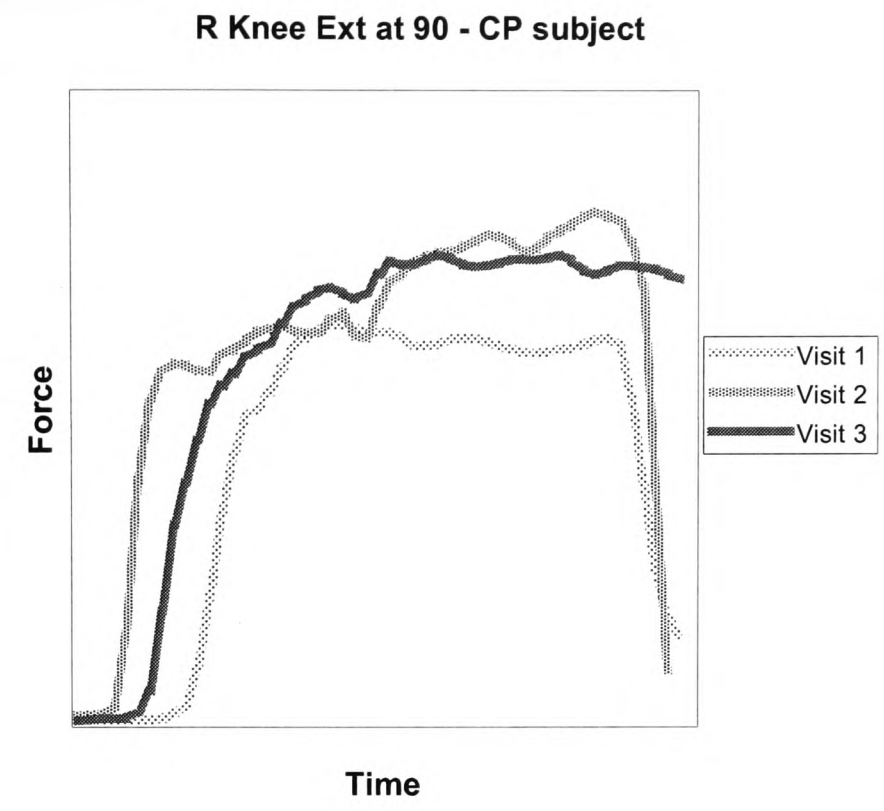
healthy children had similar measurement error for all muscle groups (Table 5.4) Similarly, the repeatability and the within-subject coefficients of variation (CV) were not significantly different ($p < 0.05$) between healthy and CP children (table 5.4).

The linear mixed model analysis was used in order to calculate the ICCs. Due to the unbalanced data of the present study, logarithmic transformation of the normalised mean peak moments was essential (Nm/kg) (Bland 1996). Table 5.5 shows that ICC was satisfactory for most muscle groups apart from knee flexors (Altman 1991). The marginal differences in ICCs between healthy and CP children were not significant ($p <0.05$).

Muscle group	**CP**	**Healthy**
	ICC	**ICC**
Hip flexors	0.68	0.73
Hip extensors	0.78	0.72
Hip abductors	0.70	0.72
Knee flexors	0.43	0.55
Knee extensors	0.77	0.78
Knee extensors 30°	0.81	0.91

Table 5.5 Intra-class correlation coefficient (ICC) for CP and Healthy children

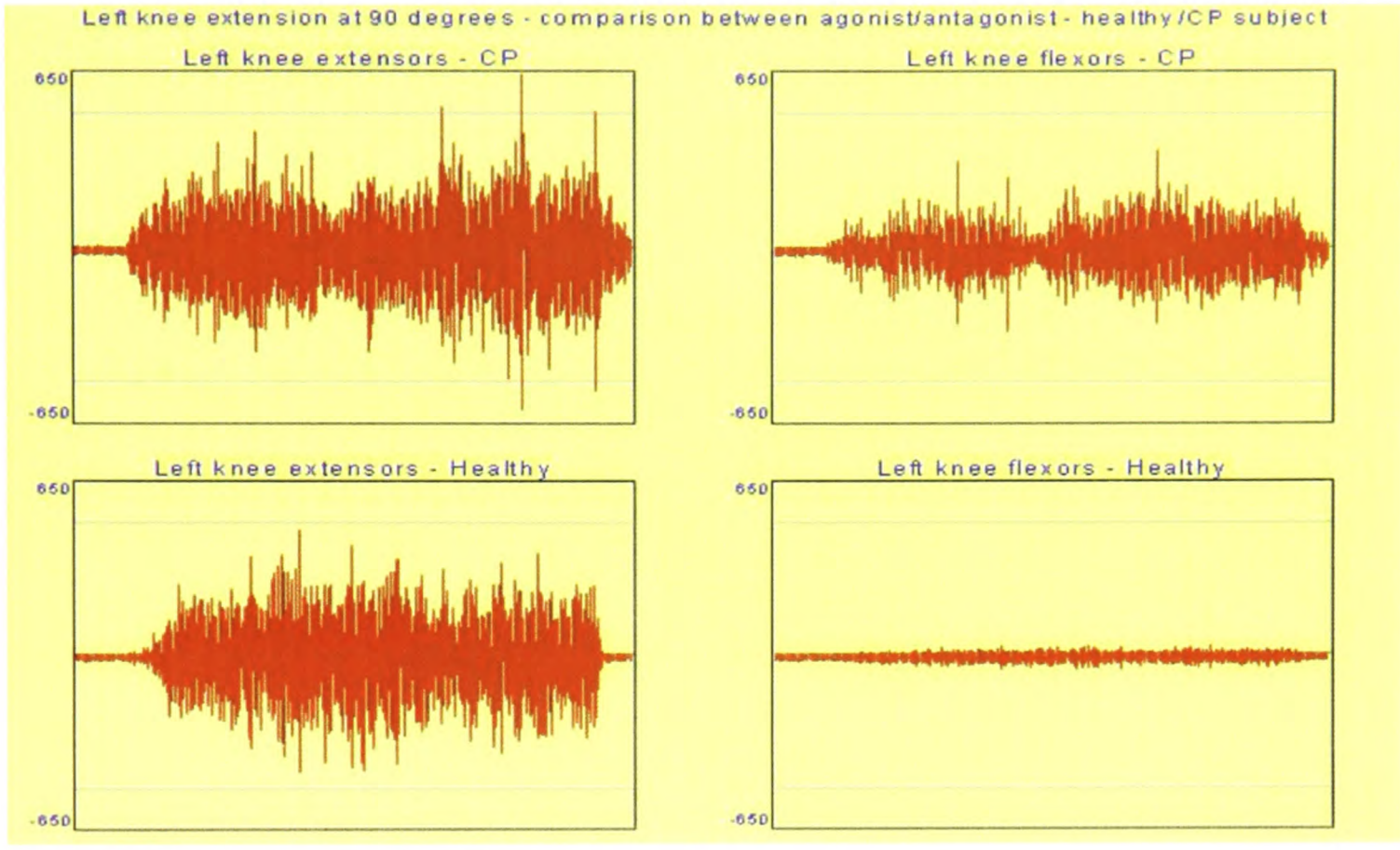
Children with CP needed longer time to achieve their peak strength than healthy children and an example of knee extensors dynamometric signals in a CP and a healthy child during repeated assessments is illustrated in Graph 5.2.



Graph 5.2 Repeated dynamometric signals during knee extension in a child with CP and a Healthy child

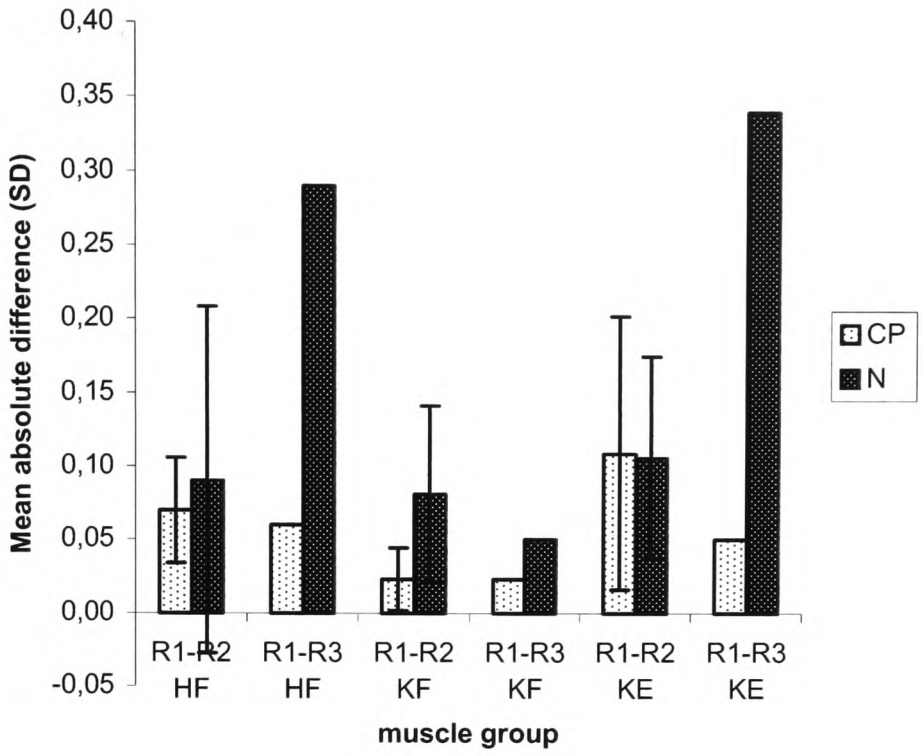
A co-contraction index for rectus femoris and medial hamstrings was calculated. This index is the ratio of the muscle acting as antagonist to the muscle acting as agonist. The raw EMG signal was analysed in order to identify a 70% window of maximum moment value (Nm). The maximum moments were extracted from excel spreadsheets and then used to quantify

a co-contraction index (CI) for rectus femoris and medial hamstrings. There was a significant difference between the two groups when the co-contraction indices of agonist/antagonist were compared ($p < 0.05$) (Graph 5.3)



Graph 5.3 Quadriceps & Hamstrings EMG signals during knee extension at 90°

The measurement error of the method when different raters were used was not possible to estimate due to limited data. The mean and standard error (SE) as well as box-plots describing the measurements are presented in more detail in appendix five. However, assessment of muscle strength did not appear considerably different between raters for the same muscle group ($p < 0.05$). A graphical representation of the differences between raters is given in Graph 5.4. Rater three assessed only one subject (CP & N), therefore no SD was calculated.



Graph 5.4 Comparison of strength assessment between raters. Mean absolute difference (SD) for all muscle groups

5.3 Discussion

This study was designed to estimate the size of measurement error of a strength assessment protocol for children with CP and compare it to that of healthy children. A modified hand-held dynamometer was used as a portable, inexpensive and relatively fast strength assessment tool; when attached to a fixed point strong muscle groups that were difficult to control with a standard hand-held dynamometer could also be measured.

The results of this study are consistent with previous reports and confirm that children with CP are weaker than healthy peers of similar age (Damiano 1995; Engsberg 1998; Wiley 1998; Engsberg 1999).

All participants were able to achieve and sustain a maximum voluntary contraction (MVC). Children with CP however, needed more time to achieve a force plateau. Limitations in selective motor control and the presence of co-contraction may have played a role. Spasticity itself could not affect the

performance of the contractions since during isometric contractions there was no velocity dependent change of the muscle length and therefore no spastic reflex can be elicited.

Previous studies have shown that isokinetic dynamometry, in adults and children with CP, is highly reliable (Holland 1994; Van den Berg-Emons 1996; Ayalon M 2000). One recent study has investigated the reliability of hand-held dynamometry in this population and found it to be reliable for measuring mean change in groups of patients with CP, but not for individuals (Taylor 2004). Unfortunately, and despite similarities in the testing protocol, it was not possible to compare the present results with those from Taylor et al (2004) since theirs were reported in kg. It should be noted that Taylor et al also commented on the unsuitability of ICCs to describe reliability of strength assessment in a clinically meaningful manner. Limitations of the ICC include its dependency on the distribution of data and the variability between subjects; in samples containing subjects who differ greatly the ICC would be considerably inflated (Bland 1996; Madsen 2002).

ICCs in this study were found to be good but still lower than those reported in the literature and this is possibly due to the limited spread of strength values between subjects.

In agreement with Taylor et al (2004), it was found that the assessment of hip extensors was not easy or reliable and this reflected on the wide confidence intervals. The position was chosen both because of its ease to apply in patients with CP and bibliographic reference (Stuberg 1988). However, as the dynamometer was held and not fixed, the strong extensor force often overcame the examiner's resistance. Moreover, and despite stabilisation of the hips and pelvis, children would frequently use their back extensors during their attempt to push. Although the ICC was 0.78 and 0.72 for CP and healthy children respectively, this was probably due to the

greater variability between subjects and it is another indication of the ICC's unsuitability as a measure of reliability in this experimental setting.

The results of this study are reported in terms of within-subject standard deviation (σ_w) and related measures of repeatability. These indices are subject to a more direct interpretation and can be applied to individual patients.

In conclusion, these results show that the measurement error of this strength assessment protocol is similar in CP and healthy children for all muscle groups. The within-subject variability may seem large, but the possible range of strength values is wide and muscle strength assessment is sufficiently precise to distinguish change due to natural history or treatment.

5.4 Summary

1. The strength assessment protocol used in this thesis is reliable for this population and research setting.
2. Children with CP are able to perform isometric contractions and they can achieve and maintain a maximum voluntary contraction (MVC). However, they need more time than healthy children to reach MVC.
3. The results of the pilot study confirm that children with spastic diplegia are weaker than children without neuromuscular or other conditions.
4. Despite modifications of the dynamometer, an optimal position for testing hip extensors was not found. This was reflected on the low reliability of this muscle group.

Chapter Six

6. Changes in strength, function and gait six months after multi-level orthopaedic surgery

The aim of this part of the study was to quantify changes in lower limb muscle strength, as well as overall changes of gross motor function and gait in the children who underwent multi-level orthopaedic surgery. A better understanding of the factors that determine rehabilitation after surgery was sought.

Single stage multi-level orthopaedic surgery is a procedure usually performed on children with spastic diplegia. It aims to correct all deformities and improve ambulation while minimising the child's exposure to repeated surgery and post-operative rehabilitation. Over the last 30 years various studies have defined the indications for this type of surgery, established the efficacy of the multi-level single-stage approach and clarified the use of specific procedures. Clinicians and researchers are now appreciating the need for more in depth analysis in a controlled manner of factors affecting outcomes of this surgical approach in children with a complex neurological condition (Gough 2004).

Amongst these factors muscle weakness is significant in limiting walking ability and motor function (Wiley 1998). In the past, spasticity has been considered the major obstacle to motor function (Bobath 1980). However, advances in the management of spasticity have highlighted compelling clinical evidence that muscle weakness limits functional improvement (Giuliani 1991). Furthermore orthopaedic surgery involving muscle-tendon

lengthening procedures is known to reduce the force-generating capacity of a muscle (Moseley 1992). Since CP principally appears to affect the bi-articular muscles, multi-level surgery, which includes simultaneous lengthening or transfer of these muscles, potentially compounds problems of post-operative weakness, particularly in the presence of pre-existing weakness. Strength gains can be achieved in this population through strength training (Damiano 1995) however the optimal content of a rehabilitation regime following multi-level orthopaedic surgery is largely empirical and the time taken to reach a new functional plateau not known.

Little objective information has been published on post-operative changes in muscle strength in CP. Anecdotally Nene et al (1993) and Sutherland et al (1997) reported no loss of hip flexor strength following multi-level surgery using manual muscle testing. However, while manual muscle testing is the most widely used clinical method of testing strength, it is subjective, reliable to within one grade only, and lacks sensitivity particularly at the upper end of the scale (Bohannon 2000). Quantitative methods of monitoring muscle performance are necessary if we are to accurately evaluate changes in patient status and the outcomes of treatment. Assessment of strength using isometric rather than isokinetic testing is preferable in children with spastic hypertonia since fast changes in muscle-tendon length may evoke a stretch reflex during isokinetic dynamometry (Boiteau 1995). In order to clarify how strength affects function in the presence of complex interactions in CP, objective assessment of strength should be combined with gait and gross motor function testing.

In this Chapter, post-operative changes of strength, gait and function were assessed in order to further contribute towards the development of optimal rehabilitation programmes following multi-level surgery.

6.1 Design of the study

This was a prospective study using a cohort of spastic diplegic patients who underwent multi-level orthopaedic surgery. Patients referred for a paediatric orthopaedic assessment were approached either at the Cerebral Palsy Clinic or the Gait Analysis Laboratory at the Nuffield Orthopaedic Centre NHS Trust. The study aim and overall design was explained to the children and their guardians, and an invitation to participate was given. Families who agreed to take part in the study signed a consent form in the beginning of the pre-operative gait or strength and function testing.

6.2 Subjects

Twenty one children with a diagnosis of cerebral palsy/spastic diplegia ranging in age from seven to 16 years at the time of surgery (mean age 13 years, SD 2 years) participated in this prospective evaluation of muscle strength, gait and functional changes after multi-level orthopaedic surgery. Ambulant children (Gross Motor Functional Classification System levels I-III) were recruited into the study when surgery was indicated. Children were excluded if they had been treated with Botulinum Toxin or orthopaedic surgery in the year prior to admission to the study. Child MS06 was tested pre-operatively but was unable to complete the post-op assessment because of her learning difficulties; with her guardian's agreement she was excluded from the overall study. The participants' characteristics are listed in Table 6.1

Child	Age (y/m)	Sex	Mass (kg)	GMFCS level
MS01 (1)	11.7	F	27	II
MS02 (2)	9.3	M	32	II
MS03 (3)	10.3	F	28	II
MS04 (4)	15.7	M	49	I
MS05 (5)	13.7	F	50	II
MS07 (6)	11.2	M	35	II
MS08 (7)	15.1	M	49	III
MS09 (8)	12.9	F	32	III
MS10 (9)	10.5	F	31	II
MS11 (10)	8.8	M	43	I
MS12 (11)	13.11	M	41	II
MS13 (12)	15.7	F	40	II
MS14 (13)	11.6	F	20	III
MS15 (14)	14.0	M	47	II
MS16 (15)	16.0	M	42	III
MS17 (16)	7.9	F	27	II
MS18 (17)	11.2	M	34	II
MS19 (18)	14.5	F	56	I
MS20 (19)	11.7	M	52	II
MS21 (20)	15.3	F	48	II
Mean (sd)	**13 (2)**		**39 (10)**	

Table 6.1 Age in years/months, sex (M=male, F=female), mass (kg) and functional level (GMFCS)

6.3 Operation and therapy details

Bony and soft tissue operations were combined and performed in each participant by the same Paediatric Orthopaedic Surgeon. Their choice was dictated by the child's impairments as these were assessed prior to surgery by detailed clinical assessment and instrumented 3-dimensional gait analysis. Fractional lengthening at the musculotendinous junction was performed whenever muscle or tendon lengthening was necessary in order to minimise post-operative weakness. Details of the operations that each participant underwent are shown in Table 6.2

Child	FDO	TO	CLO	PsL	AddL	HL	RFTr	GL	TPL	TAT	other
1	B	-	B	B	-	B	B	B	-	-	
2	B	-	B	B	-	B	B	B	-	-	
3	B	-	-	B	-	B	B	B	-	-	
4	B	-	-	-	-	-	-	B		B	
5	B	-	R	L	-	B	B	B	-	L	
6	L	-	-	L	L	L	L	B	-	-	
7	B	-	B	B	-	B	-	B	-	-	Patella adv./distal FDO
8	B	-	-	B	-	B	-	B	B	-	L Acetabuloplasty
9	-	-	B	B	-	B	B	B	-	-	
10	R	-	L	B	-	B	B	B	R	R	
11	B	-	B	-	R	B	-	-	-	-	Distal FDO
12	B	-	B	-	B	B	B	B	-	-	
13	B	-	L	-	L	B	-	B	-	-	
14	R	-	-	-	-	R	-	R	-	-	R Pelvic osteotomy
15	B	B	-	-	B	B	-	-	-	-	Distal FDO
16	R	-	L	-	-	B	B	B	R	-	
17	L	-	-	B	-	B	B	B	-	-	
18	B	-	-	-	-	B	-	B	-	-	
19	B	-	B	-	B	B	-	B	-	-	B Subtalar arthr./distal FDO
20	B	-	-	B	-	B	B	B	-	-	
Mean	**33**	**2**	**18**	**20**	**9**	**36**	**21**	**35**	**4**	**4**	

Table 6.2 Type of operation per subject right (R), left (L), bilateral (B), femoral derotation osteotomy (FDO), tibial derotation osteotomy (TO), calcaneal lengthening osteotomy (CLO), psoas lengthening (PsL), adductors lengthening (AddL), hamstrings lengthening (HL), rectus femoris transfer (RFTr), gastrocnemius lengthening (GL), tibialis posterior lengthening (TPL), tibialis anterior split transfer (TAT)

All children underwent a combination of bony and soft tissue operations. The majority of them had surgery bilaterally with the exception of one child who was operated only on the right side (subject 14). The most frequently performed operations were femoral derotation osteotomy, medial hamstrings and gastrocnemius lengthening. No immediate post-operative complications were reported.

Although the same Paediatric Orthopaedic Surgeon performed the operations in the majority of children, these could not be standardised. Therefore, the surgical intervention itself was a confounding variable in assessing outcome. Although one could attempt to analyse the specific effects of each operation, this was not considered adequate for this study design. The size of the study would bias considerably any statistical method used to assess the effect.

6.3.1 Immediate post-operative physiotherapy

After surgery children underwent routine post-operative physiotherapy as in-patients, which was similar across subjects. This was administered by Paediatric Physiotherapists at the Nuffield Orthopaedic Centre NHS Trust. Following discharge from the hospital, therapy was provided by Community Physiotherapists. Participants were commencing weight-bearing at three to four days after surgery if no femoral/tibial osteotomies were performed. Weight-bearing was delayed for four to six weeks when patients had undergone long bone osteotomies. No weight-training exercises were included in this initial rehabilitation regime in any subject. When the gastrocnemious was lengthened or any foot procedure was undertaken below-knee plaster casts were used for six weeks. Knee immobilisers (gaiters) were also used for three to four days continuously and during the night for up to six months. The physiotherapy protocol followed by the Paediatric Physiotherapists was frequently adjusted to the individual patient needs.

6.4 Method

Muscle strength, gait and gross motor function were measured pre-operatively and at six months post-operatively in all 20 subjects. The average time of testing following surgery was 28 weeks (sd 3 weeks) with

only one child (MS08) tested at 44 weeks due to social issues involved in arranging this visit.

6.4.1 Outcome measures

The outcome measures included:

1. Standard clinical examination to record medical history, ambulatory status, lower limb range of motion, any musculoskeletal deformities, muscle strength, tone and selective control.
2. A digital dynamometer (model MIE Medical Research Ltd, Leeds, UK) was used to quantify isometric strength of the hip flexors, extensors and abductors, knee flexors and extensors at 90° and 30° bilaterally. Standardised testing positions were used.
3. Gait was recorded using a motion analysis system (Polygon 2.4, Workstation 4.6, Vicon 612, Vicon Motion Systems, Oxford, UK), for production of three-dimensional kinematics and kinetics variables, temporal parameters, two-plane video recording, and dynamic electromyography (model MA300, Motion Lab Systems, Baton Rouge, USA).
4. Motor function was recorded using the Gross Motor Function Measure, GMFM-88 (Russell 1989).
5. Function, covering community settings and terrains, as described by the parent or guardian was assessed using the Gillette Functional Assessment Questionnaire (FAQ) (Novacheck 2000). The functional walking scale part of it stratifies subjects across the spectrum of community mobility independently of the need for assistive walking devices.

6.4.2 Choice of variables

The variables tested and analysed in the study were divided in three categories: those assessing muscle strength, gross motor function and gait respectively. These are presented in Table 6.3.

As discussed in Chapter Three, muscle strength is the maximum tension a muscle can exert under specified conditions (Jones 1996). During isometric

contractions the force produced measured in Newton (N) was multiplied with the lever arm distance (m) resulting in moment (Nm). Moments were then normalised to body mass giving a measure of force produced by a muscle group that could be used in the analysis of multiple subjects (Nm/kg).

Gross motor function was measured with a percent score calculated from a validated scale, the gross motor function measure scale (GMFM 88) (Russell 1989). Community ambulation was assessed, by using the walking scale part of the Gillette Functional Assessment Questionnaire (FAQ) (Novacheck 2000).

Parameters describing gait were those measuring angle joints (kinematics), muscle moments and powers (kinetics) as well as temporal parameters during walking. As discussed in Chapter 4.1.1.1, ambulation is the result of a complex interaction between the nervous and musculoskeletal systems. Instrumented gait analysis has the advantage, compared with observation, of studying gait in all three planes of movement. This is particularly important in multi-level surgery, which usually corrects deformities and improves muscle balance at three levels affecting the pelvis/hip, knee and ankle areas. For these reasons, parameters which describe joint motions, moments and power in all three planes (sagittal, transverse and coronal) as well as three levels (pelvis/hip, knee and ankle) were used in the present study. It was thought that these parameters would be affected sufficiently to demonstrate, any changes caused by surgery and/or physiotherapy.

During gait analysis, clinical examination data were also collected. These variables, for example, muscle strength tested manually, selective control or spasticity were not included in the analysis. Although their clinical significance is not an issue for debate in this work, their insufficient precision and repeatability of measurement was the reason for excluding them. Dynamic electromyography data were not considered relevant in this

context since the information provided would not answer any of the questions of the present study.

Strength and Function Variables	**Kinematic and Kinetic Variables**
GMFM total %	Range of pelvic tilt (°) (RPT)
GMFM dimension E %	Range of hip flexion (°) (RHF)
FAQ walking scale	Range of knee flexion (°) (RKF)
Normalized velocity	Range of ankle dor/fl (°) (RAD)
Normalized Cadence	Max hip extension stance (°) (MHES)
Normalized Right step	Max knee extension stance (°) (MKES)
Normalized Left step	Max knee flexion swing (°) (MKFS)
Hip flexors Nm/kg	Knee flexion initial contact (°) (KFIC)
Hip extensors Nm/kg	Range of pelvic obliquity (°) (RPO)
Hip abductors Nm/kg	Range of pelvic rotation (°) (RPR)
Knee flexors Nm/kg	Max thigh rotation (°) (MTR)
Knee extensors at 90° Nm/kg	Hip extension moment (HEM)
Knee extensors at 30° Nm/kg	Hip flexion moment (HFM)
	Knee extension moment (KEM)
	Knee flexion moment (KFM)
	Max plantarflexion moment (MPM)
	Max hip abduction moment (MHAM)
	Max hip power (MHP)
	Max ankle power (MKP)

Table 6.3 Strength, function and gait variables tested and analysed

6.4.3 Testing session outline

On arrival at the gait laboratory, children and their parents/guardians were informed of the protocol and consent was obtained. The first part of the assessment included the subjects' medical history, clinical examination and anthropometric measurements necessary for the gait analysis procedure. Surface electrodes and retro-reflective markers were then attached at specific anatomical landmarks of the lower limbs and three-dimensional gait analysis was undertaken.

When sufficient gait data were collected, subjects were allowed to rest before the second part of the assessment. The strength measurement set-up was prepared and subjects would normally complete this assessment in 45 to 60 min. During that time the parents or guardians were asked to complete the FAQ questionnaire. The last part of the session included the administration of the GMFM by the author.

6.4.3.1 Gait Analysis and dynamic electromyography

After the session plan was discussed with the child and parent, a medical history was taken if this was the first visit or information was given about the child's progress since the previous visit. The medical history included birth history, motor milestones, current ambulatory status and level of physical activity, any pain experienced, and perceived goals from treatment. Clinical examination of strength, range of motion, spasticity and selective control in the lower limbs followed. At this time, anthropometric measurements required for the lower body 3D computer model were also taken.

Surface electrodes were attached to the medial hamstrings, rectus femoris and lateral head of the gastrocnemius muscles. Electrodes were placed over the centre of the muscle belly, which was palpated asking the subject to actively contract the relevant muscle (where possible) and according to SENIAM guidelines (Surface EMG for the Non-Invasive Assessment of Muscles) (Hermens 1997). Electrode leads were plugged into a box, which was carried by the subject, attached with Velcro to their back with a custom-made vest.

Fifteen retro-reflective spherical markers (14 mm in diameter) were attached with double-sided tape to specific anatomical landmarks on the lower limb (Table 6.4), according to the model described by Davis (1996)

Table 6.4: Conventional Marker Set

Marker Name	Position
SACR	Midway between the 2 Posterior Superior Iliac Spines
RASI	directly over the Right Anterior Superior Iliac Spine
LASI	directly over the Left Anterior Superior Iliac Spine
RTHI	Lateral aspect of Right Thigh (wand)
LTHI	Lateral aspect of Left Thigh (wand)
RKNE	Lateral epicondyle of the Right Knee
LKNE	Lateral epicondyle of the Left Knee
RTIB	Lateral aspect of Right lower leg (wand)
LTIB	Lateral aspect of Left lower leg (wand)
RANK	Right lateral malleolus in line with transmalleolar axis
LANK	Left lateral malleolus in line with transmalleolar axis
RTOE	Between 2nd and 3rd metatarsal heads on right foot
LTOE	Between 2nd and 3rd metatarsal heads on left foot
RHEE	Posterior aspect of right calcaneus
LHEE	Posterior aspect of left calcaneus
RMMA	Right medial malleolus in line with transmalleolar axis
LMMA	Left medial malleolus in line with transmalleolar axis

Twelve infra-red cameras (Vicon 612) were used to measure three-dimensional motion of the lower limbs. Cameras sampled at 100Hz. Two AMTI (Advanced Medical Technologies Incorporated) force plates were used to collect ground reaction force data, sampling at 1000Hz. A static trial was initially collected with the subject standing in the anatomical neutral position in the middle of one of the force plates. This was used to reference particular markers, and to measure the subject's mass. Once the static trial was completed, two of the markers were removed (RMMA and LMMA, Table 6.4). Subjects were then taken to the end of the ten meter walkway, and instructed to walk at their normal pace down to the other end of the walkway, over the force plates. This was repeated several times until at least three clean strikes on the force plates were achieved for each foot. This

took an average of 20 trials per session. Subjects were lined up at either end with the help of coloured lines on the floor. Sagittal and coronal video footage of the subject walking was collected following the 3D gait analysis. If the subject customarily wore an orthosis, the same procedure was repeated in order to obtain gait analysis data. Video footage of the subject was taken simultaneously.

It should be noted that gait analysis data at six months post-operatively, both before and after the physiotherapy intervention, were always collected in orthoses. Patients at that point of their recovery were still using orthoses full time, as part of their clinical protocol. A number of subjects were not able to provide a clean strike on the force plates, which made acquisition of kinetic data impossible. Gait analysis data were checked before starting the second part of the assessment. Complete processing of the data was undertaken at a later stage.

6.4.3.2 Digital dynamometry

Following gait analysis and rest, strength testing was performed. Subjects were shown the experimental set-up and familiarised themselves with the transducer. A combination of fixed and hand-held dynamometry was utilised using a digital dynamometer (MIE Medical Research Ltd, Leeds, UK); fixed dynamometry was used for the knee extensors where normal values were expected to exceed the strength of the examiner using a hand-held technique (Stuberg 1988). The transducer was placed at a specific position on the tested limb with adequate padding for comfort. The lever arm distance was then measured. The joint centres for the placement of the transducer and the measurement of the lever arm distance were assessed visually. An appropriate computer interface was used where the data were collected for later processing.

The following muscle groups were tested: hip flexors, hip extensors, hip abductors, knee flexors and the knee extensors at both 90° and 30° of

flexion. Testing was performed in a standard order to minimise changes of position. Standardised testing positions were based on existing literature with modifications to accommodate up to 30° of hip and/or knee flexion contracture for the patients with CP. These are described in detail in Chapter Five. Pelvic stabilisation was used in all positions to minimise compensatory movements. The child was placed supine on the testing table for assessment of hip muscle groups. Then he/she was asked to sit up with hips at 90° of flexion with legs over the edge of the table for assessment of the knee muscle groups.

Maximal isometric contractions were chosen to diminish the effects of spasticity. The "make test" technique was used with the examiner balancing without overcoming the child's strength. Each child performed three maximal isometric contractions for each testing position; the highest force was used after processing in the data analysis.

Despite careful consideration of the testing positions, some limitations existed with the dynamometry set-up. As it was discussed in Chapter Five, the assessment of hip extensors was challenging, especially in more involved patients.

6.4.3.3 Gross motor function assessment

During the last part of the assessment the subject was tested barefoot at the physiotherapy gymnasium or the gait laboratory. The 88 items that comprise the GMFM assess function in five different domains, which span the range of daily activities: (a) lying and rolling, (b) sitting, (c) crawling and kneeling, (d) standing and (e) walking, running and jumping. A separate score for each dimension was calculated and an overall score gave the GMFM result for the particular visit.

6.4.4 Statistical analysis

Isometric muscle strength was expressed in moments (Nm) by multiplying the force measurements by lever arm distance. The peak values for each muscle group and assessment were recorded and normalised to body mass (Nm/kg) for analysis.

Group mean changes in strength for each muscle group were assessed for all limbs that were operated on with valid strength measurements; pre and post operative measures were compared using paired sample t-tests (SPSS v.10.0, SPSS Inc. Chicago, IL. USA).

Three gait cycles taken from representative walking trials were processed using Plug-In-Gait modeling software based on the protocol described by Davis and DeLuca (1996). Kinematic, kinetic and temporal parameters were extracted for each side and averaged for each child. Walking speed, cadence and step length were normalised to account for leg length (Hof 1996). Mean changes in kinematic and kinetic variables were assessed using paired sample t-tests.

Changes in total GMFM and dimension E scores were also evaluated with paired samples t-tests.

A significance level of $p<0.05$ was used in all analyses.

6.5 Results

Twenty subjects were included in the analysis. Strength and gait analysis measurements were analysed based on individual limbs. Data sets and statistic tests are presented in more detail in Appendices Five and Six.

6.5.1 Comparison of muscle strength before and six months after surgery

Statistical comparisons in this group of surgical patients with spastic diplegia indicated that strength did decrease significantly at six months following multi-level orthopaedic surgery in all muscle groups (Table 6.5, $p<0.05$).

Muscle group	No. legs in analysis	Pre-op Mean (SD) (Nm/kg)	6 month post-op Mean (SD) (Nm/kg)	Mean difference (SD) (Nm/kg)	% Reduction	p
Hip Flexors	39	0.48 (0.23)	0.26 (0.14)	-0.22 (0.21)	45.1	.000
Hip extensors	39	2.55 (0.92)	1.99 (0.75)	-0.56 (0.73)	21.9	.000
Hip abductors	37	0.62 (0.22)	0.51 (0.25)	-0.11 (0.20)	17.3	.002
Knee flexors	37	0.98 (0.29)	0.42 (0.21)	-0.56 (0.30)	57.6	.000
Knee extensors 90°	39	1.57 (0.66)	1.06 (0.44)	-0.51 (0.50)	32.5	.000
Knee extensors 30°	21	0.59 (0.39)	0.39 (0.23)	-0.12 (0.19)	18.7	.008

Table 6.5 Pre and post –surgery muscle strength (mean, sd) and significance of change ($p < 0.05$)

The greatest percentage reduction in strength was seen in the knee flexors (57.6%) followed by the hip flexors (45.1%). The hip abductors were the group that showed the least percentage decrease in strength (17.3%), although this was still significant.

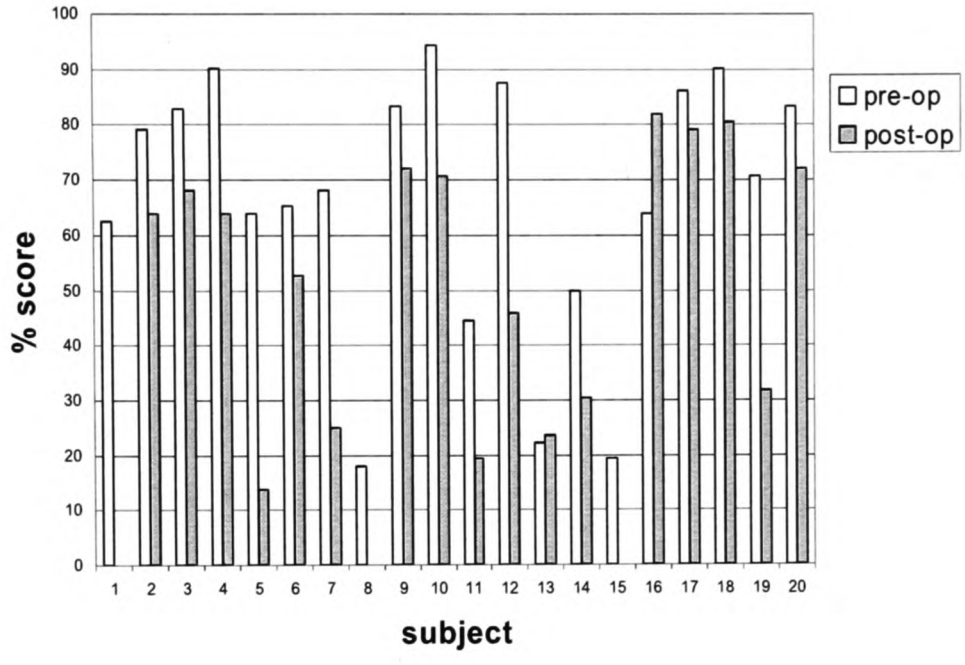
When strength changes were considered by limb (table 6.6), most showed a reduction in muscle strength with the greatest number of significant individual changes seen in the knee flexors (14/37 legs). The hip abductors were the only group, where no individual reduction in strength was found to be statistically significant. While measurements from some individual limbs showed a small increase in muscle strength none of these were significant based on the repeatability protocol as described in Chapter Five.

Child	*Hip flexors*		*Hip extensors*		*Hip abductors*		*Knee flexors*		*Knee extensors 90°*		*Knee extensors 30°*	
	L	*R*	*L*	*R*	*L*	*R*	*L*	*R*	*L*	*R*	*L*	*R*
1	↓	↓	↓	↓	↓	↓	↓*	↓*	↓	↓	↑	↓
2	↓	↓	↓	↓	↓	↓	↓	↓	↓	↓	↓	↓
3	↓	0	↑	↓	↓	↓	↓	↓	↓	↓	↓	↓
4	↓	↓	↓	↓	↓	↓	↓	↓	↓	↓*	X	X
5	↓	↓	↓	↓	↓	↓	↓	↓	↓	↓	↓	↓
6	↓	↑	↓	↑	↓	↑	↓	↑	↓	↑	↓	↓
7	↓	↓*	↓	↓	↑	↑	↓*	↓*	↑	↑	X	X
8	↓	↓	↓	↓	↓	↓	↓	↓*	↓	↑	X	X
9	↓	↓	↑	↑	↓	↓	↓	↓	↓	↓	0	↓
10	↓	↓	↑	↓	↓	↓	↓*	↓*	↓	↓	X	X
11	↓	↓	↑	↓	↓	↑	↓*	↓*	↓	↓	X	X
12	↓	↓	↓	↓	↓	↓	↓*	↓*	↓	↓	↓	↓*
13	↓	↑	↓	↓	↓	↓	↓	↓	↓	↓	X	X
14	↑	↓	↓	↓	↑	↓	↓	↓	↑	↓	↓	X
15	↓	↓	↓	↓	X	X	↓*	↓*	↓	↑	X	X
16	↑	↑	↑	↓	↑	↑	↓	↓*	↑	↑	X	↑
17	↑	↓	↓*	↓*	↓	↓	↓	↓	↓	↓	↓	↑
18	↓	↓	↓	↑	↑	↓	↓	↓	↓	↓	↓*	↓
19	↓	↓	↓	↓	↑	↑	↓	↓	↓	↓	↓	↑
20	↓	↓	↓	↓	↑	↓	X	X	↓	↓	X	X

*Table 6.6 Strength changes between pre and six months post operative assessments by subject and limb. ↑ = an increase, ↓ = a decrease in muscle strength * = statistically significant based on change > 2.77σ_w.*

6.5.2 Gross motor function changes after surgery

Changes in total GMFM and dimension E scores separately were also evaluated with paired samples t-tests (Graph 6.1).



Graph 6.1 Gross motor function before and six months after surgery as % score of the GMFM dimension five (walking, running, jumping)

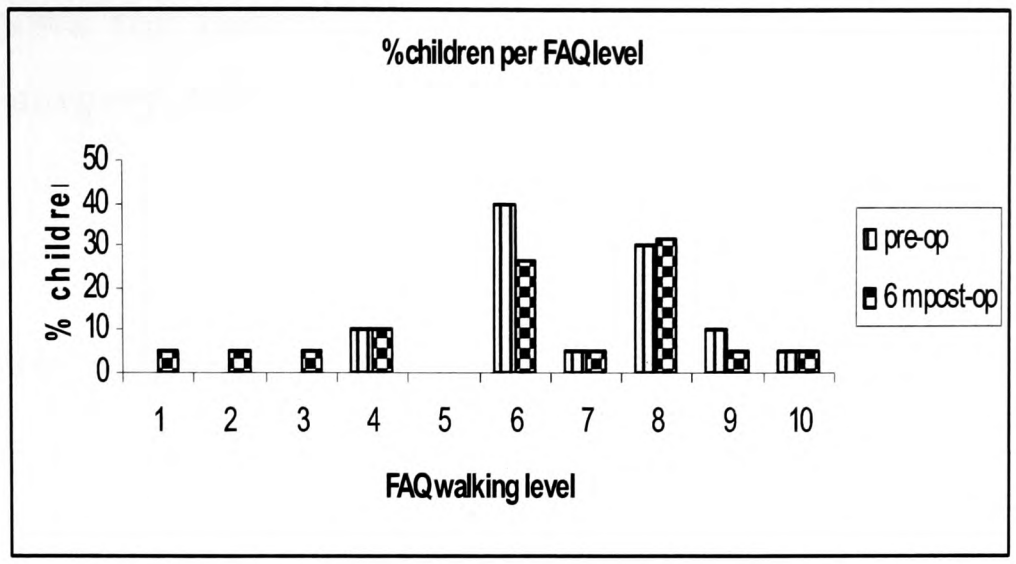
The functional changes relating to walking, running and jumping, as assessed with the GMFM were reduced in 18/20 of the participants and when considered as a group, a significant decrease in the total GMFM and dimension E was seen. Normalised walking speed and cadence were also significantly reduced in the whole group (Table 6.7). Step length showed a small non-significant increase post-operatively, however the use of orthoses and/or crutches during that gait analysis session should be noted since it can affect step length.

GMFM scores & Temporal parameters	Pre-surgery Mean (SD)	Post-surgery mean (SD)	Paired mean diff (SD) pre/ post surgery	p_{ii} pre /post surg
GMFM total %	84.9(12.6)	77.2(14.3)	7.6(9.9)	0.003
GMFM E %	66.3(24.1)	44.8(29.2)	21.5(18.6)	0.000
N walk speed	0.31(0.1)	0.26(0.1)	0.05(0.1)	0.002
N cadence	0.53(0.1)	0.44(0.1)	0.09(0.1)	0.000
N step length Right	0.58(0.1)	0.58(0.2)	-0.01(0.1)	0.754
N step length Left	0.58(0.1)	0.58(0.2)	-0.01(0.1)	0.857

Table 6.7 Pre and post surgery GMFM scores % and normalised temporal parameters (mean, sd, paired difference) and significance of change ($p < 0.05$)

Analysis of the walking scale scores of the FAQ is represented in the Graph 6.2. As discussed in Section One, not taking any steps and not walking on a routine basis are described by levels one and two. Exercise ambulation levels are three-four. Level five is consistent with household or classroom walking and level six is limited community walking. Routine community walking on level surfaces is considered a level seven. Levels eight and nine are more advanced levels of walking on a variety of terrains. Level 10 is consistent with a typical, non compromised level of walking ability.

Interestingly, as it is shown in Graph 6.2, the majority of children before the surgery were rated at least level six on the walking scale of the FAQ with only two children rated as level four. At six months following surgery the parents' impression of the child's walking ability was quite different. Many children were rated at a lower level representing a loss in function as shown by the drop in the GMFM score.



Graph 6.2 % of children in sample at each FAQ walking level before and after surgery

Table 6.8 Individual change of FAQ walking level per subject 6 months after

Subject	FAQ pre-op	FAQ post-op
1	6	6
2	9	9
3	8	8
4	10	10
5	7	6
6	6	7
7	6	6
8	4	4
9	8	6
10	8	6
11	6	8
12	8	8
13	6	4
14	6	3
15	4	1
16	6	2
17	9	8
18	8	8
19	6	,
20	8	8

Table 6.8 shows the individual changes of the FAQ level before and six month after surgery. Only subjects six and 11 improved slightly their FAQ level.

6.5.3 Lower limb kinematics and kinetics after surgery

As a group, a significant improvement was seen in the sagittal and transverse plane kinematic gait variables. Pelvic motion in these planes improved, knee extension increased significantly during stance, knee flexion at initial contact significantly reduced and internal thigh rotation improved dramatically (Table 6.9).

Kinematic Parameters	**Pre – surgery mean (SD)**	**Post-surgery mean (SD)**	**Paired mean dif (SD) Pre-post surgery**	**p_i Pre-post surgery**
RPT (°)	10.2(3.0)	8.1(2.7)	2.1(3.3)	**0.000**
RHF (°)	42.4(7.5)	43.3(9.1)	-0.8(8.3)	0.523
RKF (°)	31.9(12.9)	49.0(16.1)	19.2(16.9)	**0.000**
RAD (°)	31.0(13.9)	11.8(7.5)	2.2(8.3)	0.103
MHES (°)	9.0(11.9)	6.8(13.8)	23.6(24.7)	**0.000**
MKES (°)	32.9(24.7)	9.3(17.4)	6.3(15.3)	0.12
MKFS (°)	64.6(16.4)	58.2(6.8)	23.0(18.8)	**0.000**
KFIC (°)	44.1(19.7)	21.1(14.1)	1.4(4.6)	0.063
RPO (°)	8.9(4.2)	7.5(2.7)	1.6(8.8)	0.256
RPR (°)	18.5(6.8)	16.9(5.1)	17.9(19.3)	**0.000**
MTR (°)	22.7(12.6)	4.8(17.6)	-17.1(17.6)	**0.000**

RPT = range pelvic tilt, RHF = range hip flexion, RKF = range knee flexion, RAD = range ankle dorsiflexion, MHES = max hip extension stance, MKES = max knee extension stance, MKFS = max knee flexion swing, KFIC = knee flexion initial contact, RPO = range pelvic obliquity, RPR = range pelvic rotation, MTR = max thigh rotation

Table 6.9 Kinematic variables (mean, sd, paired difference) pre and 6 month post surgery and significance of change ($p < 0.05$)

Comparisons between pre-op and six month post-op kinetic values also demonstrated significant improvements.

Kinetic Parameters	Pre – surgery mean (SD)	Post-surgery Mean (SD)	Paired mean dif (SD) Pre-post surgery	p_i Pre-post surgery
HEM	1420.7(335.9)	1280.0(497.4)	45.2(489.4)	0.684
HFM	-665.8(226.6)	-584.0(135.9)	-128.8(224.2)	**0.019**
KEM	588.9(346.1)	360.4(147.9)	179.8(290.5)	**0.012**
KFM	-482.6(280.9)	-512.9(170.0)	13.9(290.0)	0.833
MPM	1209.0(318.7)	1100.7(182.6)	91.8(326.4)	0.224
MHAM	624.6(216.7)	370.4(110.9)	266.9(233.3)	**0.000**
MHP	1.6(0.6)	1.3(0.7)	0.2(0.9)	0.252
MAP	1.8(0.7)	1.0(0.6)	0.8(1.1)	**0.004**

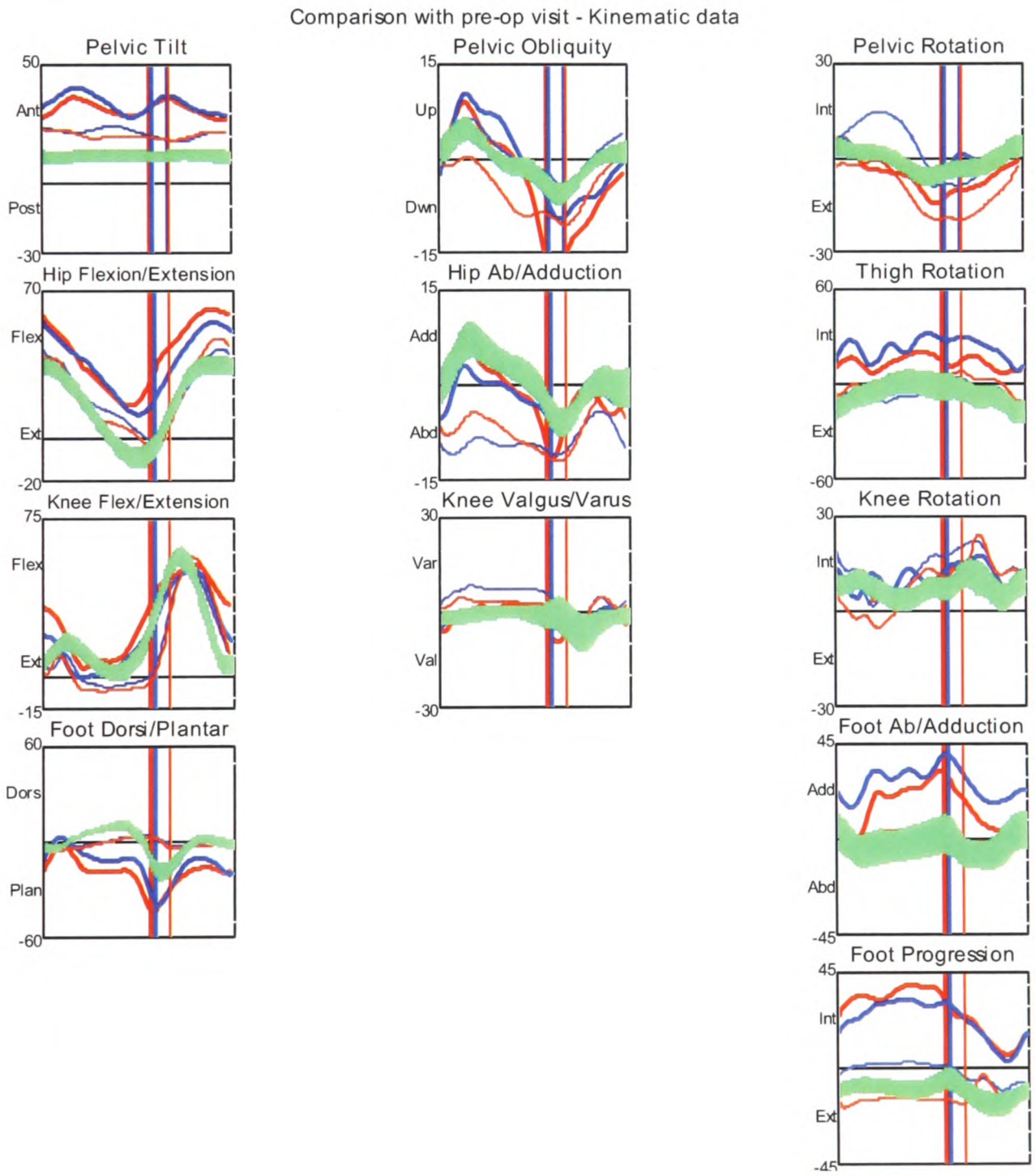
Table 6.10 Kinetic variables (mean, sd, paired difference) pre and 6 month post surgery and significance of change ($p < 0.05$) HEM = hip extension moment, HFM = hip flexion moment, KEM = knee extension moment, KFM = knee flexion moment, MPM = max plantarflexion moment, MHAM = max hip abduction moment, MHP = max hip power, MAP = max ankle power

Table 6.10 shows significantly increased knee extension and hip abduction moments with improved ankle power. Interestingly, hip flexion moment was significantly reduced at six months after surgery.

Case study

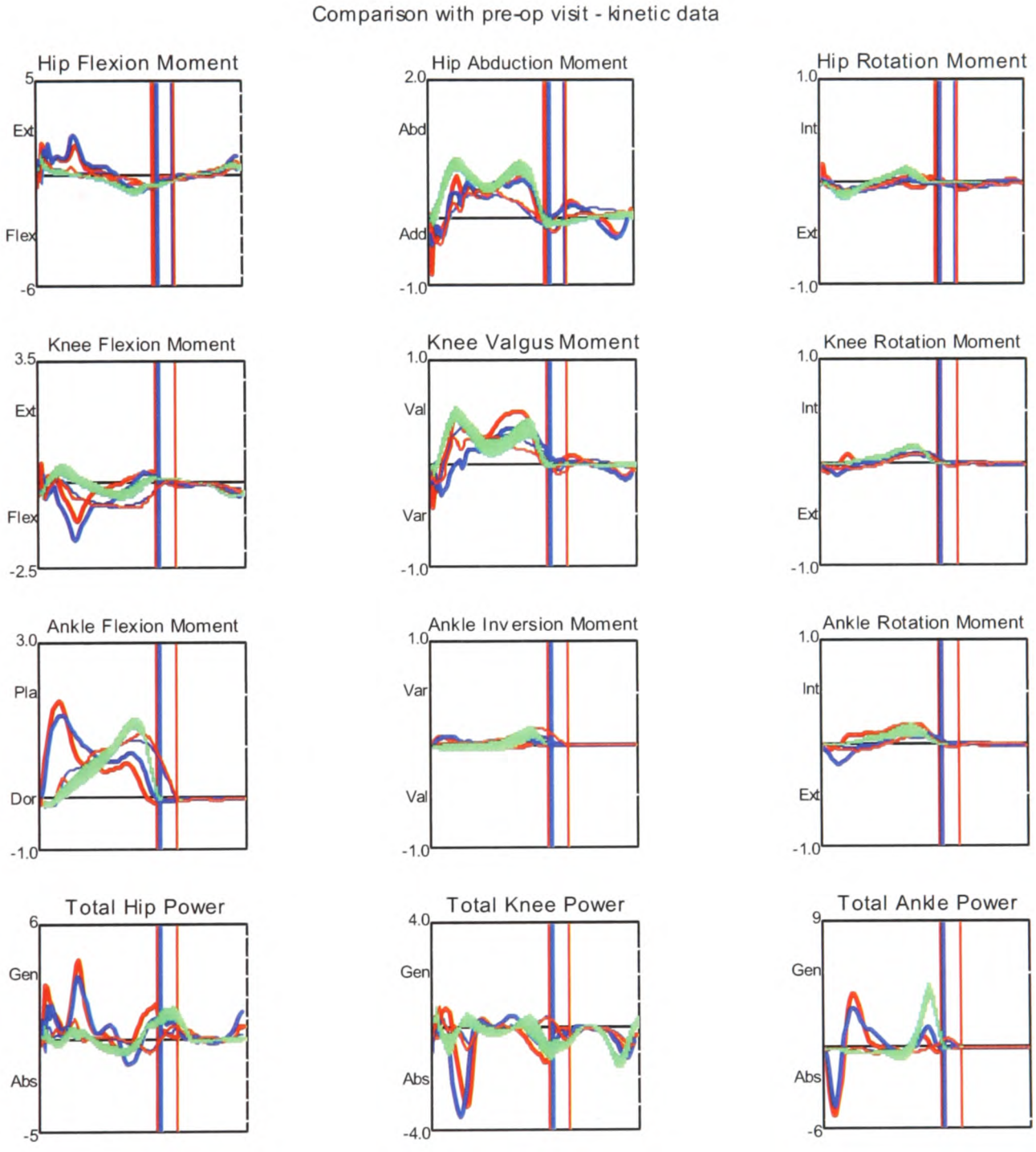
An example of post-operative improvement is presented here. Graphs 6.3 and 6.4 illustrate joint angles (kinematics) and joint moments/powers (kinetics) from a female, who underwent multi-level orthopaedic surgery. Subject MS21 (20), who was 15 y 3 m at the time of surgery, had a combination of bony and soft tissue operations in one stage: bilateral femoral derotation osteotomies, psoas, hamstrings and gastrocnemious lengthening as well as bilateral rectus femoris transfers. She followed the standard rehabilitation regime as in-patient and continued with physiotherapy provided by a Physiotherapist in the community following discharge. No weight training exercises were performed until the six month

testing. In Graph 6.3, a comparison of kinematics before and after the surgery is presented. Improvements are seen in all three planes. Pelvic angles are closer to the average line after surgery with decreased anterior pelvic tilt. Hip joint angles show a dramatic improvement in the sagittal and transverse planes with near normal values. Knee flexion at initial contact is decreased with improved knee flexion in swing phase. The overall progression of gait returns to near normal.



Graph 6.3 Kinematic data before and 6 months after multi-level surgery (green band = average normal female, blue line = Right side, red line = Left side, thick line = pre-op, thin line = post-op)

Similarly, hip, knee and ankle joint moments show improvements following surgery and near normal values (Graph 6.4). Interestingly, powers are not dramatically improved. It should be noted that their assessment, especially that of total ankle power was affected by the use of orthoses (GRAFO's) and crutches at this stage.



Graph 6.4 Kinetic data before and 6 months after multi-level surgery (green band = average normal female, blue line = Right side, red line = Left side, thick line = pre-op, thin line = post-op)

6.6 Discussion

Results presented in this Chapter showed that at six months post multi-level orthopaedic surgery there is an immediate improvement in joint kinematics and kinetics but significant loss of muscle strength and function persists.

The effect of multi-level surgery in five proximal muscle groups (six positions) was quantified. It was found that at six months after surgery there was a significant reduction in muscle strength in all groups measured. The greatest percentage reduction was in the knee flexors (57.6%) followed closely by the hip flexors (45.1%). These muscle groups were surgically lengthened, which may explain the greater loss of strength (Moseley 1992), although studies have suggested that muscle force is preserved after aponeurotic lengthening (Brunner 2000), in contrast to a loss of force after tendon elongation (Delp 1995).

Soft-tissue surgery was performed most commonly to the hamstrings (36/39 legs) and gastrocnemius (35/39 legs) followed by rectus femoris (21/39 legs) and psoas (20/39 legs). The least reduction in strength was seen in the hip abductors (17.3%) followed by the knee extensors at 30° of flexion (18.7%). The hip abductors were the only group where no individual reduction in strength was found to be statistically significant. The hip abductors and the hip extensors were the two muscle groups which showed strength increases in the greatest number of individual limbs (10 and eight respectively). Of the muscles measured, these two groups were not operated on directly. The most common bony procedure performed was femoral derotation osteotomy (33/39 legs), which is thought to correct lever-arm dysfunction and, in doing so, may confer a biomechanical advantage on the hip abductors post-operatively.

Knee extensors at 30° of flexion were not measured in 18/39 limbs due to knee contractures ≥ 30° in 10 limbs. Following surgical correction of knee

contractures it was possible to produce some knee extensor force in this position post-operatively, but these limbs were not included in the statistical analysis. In the only non-operated limb (child 14) muscle strength increased in three muscle groups and reduced in three muscle groups, none of which were statistically significant.

In agreement with published data, these results showed significant early improvements in all planes of lower limb joint angles and moments after surgery. In theory, the improvement in kinematics and kinetics would lead, in time, to a better overall function when rehabilitation is complete. However it was found that at six months after surgery gross motor function and walking velocity were significantly reduced. Indeed Damiano et al (1999) did not show functional improvement following hamstring lengthening, as evidenced by increased stride length, until strength values approximated or exceeded pre-operative values at nine months post operatively. Two subjects in this study (child 13 and 16) did show improvements in the GMFM. Child 16's improvement in the GMFM was the greater of the two and this child also showed a tendency to improve muscle strength in the greatest number of muscle groups (9/12).

It should be noted, however, that rehabilitation should be considered incomplete at this stage. Although animal studies suggest that muscle or tendon surgery produces a temporary loss of strength, which is completely recovered in six weeks (Brunner 2000), clinical experience has shown the rehabilitation period following multi-level orthopaedic surgery to be prolonged with continued improvement for up to two years (Nene 1993). Rehabilitation may be particularly long because the degree of post-operative weakness has not been fully appreciated or adequately addressed in children with central nervous system disorders. Strength gains can be achieved through resistance training at the same rate as in healthy individuals (Damiano 1995) and, if employed post-operatively, may shorten the rehabilitation period following multi-level surgery. Therefore any

evidence based physiotherapy methods, which could potentially shorten this period, would be welcome; these are described in the next Chapter.

6.7 Summary

1. At six months after multi-level surgery significant improvements in joint kinematics and kinetics were demonstrated.
2. Despite improvements in gait analysis parameters, significant loss of strength in lower limb muscle groups persists. Knee and hip flexor muscle groups show the greatest reduction in strength.
3. Gross motor function follows the same trend as muscle strength and remains significantly lower compared with pre-operative values.
4. The value of multi-level surgery in improving deformities is confirmed. However, rehabilitation at six months should be considered incomplete. Further research into ways of shortening and / or improving post-operative physiotherapy management would be beneficial.

Chapter Seven

7. Comparison of strength training techniques after surgery

The aim of this part of the study was to answer the second research question outlined in Chapter One: what is the effect of a progressive resistance exercise programme on muscle strength, function and gait following multi-level orthopaedic surgery?

Patients with spastic disorders, including CP, are known to be weak (Wiley 1998) and their strength is directly related to function (Damiano 1998). These patients can increase muscle strength at a rate similar to that observed in patients with weakness unrelated to central nervous system lesions (Petersen 1990). In particular, strengthening exercises result in a direct improvement of function and in contrast to traditional beliefs, they do not increase spasticity (MacPhail 1995; Fowler 2001).

Sports literature suggests that loading of a muscle is essential in order to increase strength (Atha 1981). This load must be intense (65 to 100% of MVC) and a low number of repetitions with appropriate recovery of muscle is effective (Faigenbaum 2000).

The literature suggests that when the spastic agonist is lengthened, the antagonist should be strengthened e.g lengthening of the hamstrings combined with strengthening of the quadriceps (Reimers 1990b). It is also essential to concentrate on muscles responsible for power production during gait e.g. hip flexors and ankle plantarflexors.

It has been suggested that the choice of strengthening programme should be decided based on the particular problem identified. Isotonic exercise would be appropriate for end range weakness, isokinetic for weakness throughout the range, difficulty in fast movements and eccentric weakness and electrical stimulation for difficulty in activating the muscle or aquatics to minimise joint stress. Research in this area is scarce despite the interest in this field.

A few studies have produced evidence-based results to date but there is still limited information about effective physiotherapy treatment regimes following multi-level orthopaedic surgery. In the UK the type, intensity and duration of treatment varies geographically and no widely accepted guidelines exist on paediatric physiotherapy programmes for rehabilitation following surgery.

For the reasons described above, a randomised clinical trial was designed to determine if a progressive resistance strengthening programme was more effective than active strengthening exercises in improving strength, function and gait following multi-level orthopaedic surgery. In order to examine the role of progressive resistance in improving strength and function after surgery, the two programmes were designed to differ mainly on the presence of load.

7.1 Current physiotherapy practice

In order to design an appropriate exercise regime and ultimately monitor the physiotherapy that the participants were receiving in the community, the local community physiotherapists and/or the patients' parents were approached and interviewed during the study.

7.1.1 Aim

The objective of this investigation was to:

Monitor the content and frequency of physiotherapy that participants were receiving in the community during the study.
Ultimately, it was intended to ensure that any extra physiotherapy received was similar across subjects. At the same time this information would help define the "routine" therapy provided in this population in the study area.

7.1.2 Method

A short questionnaire was developed. The questions are summarised below:
1. Please tick the type or types of physical activity the child participates in (e.g. hydrotherapy, horse riding, school PE, community OT, other)
2. Is the child currently having any physiotherapy?
3. How often does the child receive a physiotherapy programme (by a therapist, by a school assistant, by a parent/ him/herself)?
4. What type of physiotherapy approach would you predominantly follow (you may tick more than one: Bobath, conductive education, eclectic, musculoskeletal, other)?
5. What type of exercises does the child perform during the physiotherapy programme, including use of equipment and orthoses (you may tick more than one: passive stretching, passive corrective manual handling, positioning, muscle strengthening using active movements, muscle strengthening with resistance, gross motor skill training, other)?
6. Any additional comments?

The survey included all participants in this project, surgical and non – surgical. 30 questionnaires were handed over, mailed or completed through a telephone interview with a Senior I Paediatric Physiotherapist or/and the participants' parents.

7.1.3 Analysis

This survey generated nominal data and descriptive statistics were used for analysis.

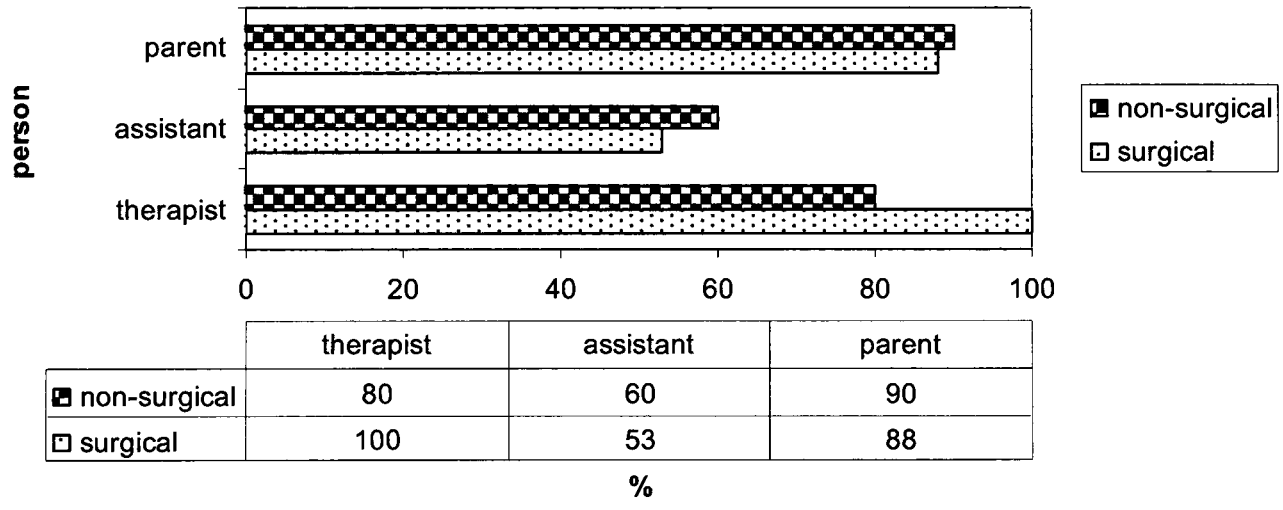
7.1.4 Results

27 questionnaires were completed giving a response rate of 90%.

The survey showed the following findings (Appendix 5 for Graphs & Tables):

1. All participants undertook some form of exercise during the year of their participation to the study. However, the providers differed between the surgical and non-surgical patients. Patients who had surgery all received treatment by a Paediatric Physiotherapist (100%), but only half of them had physiotherapy by a classroom or Physiotherapy Assistant at school (53%). Parents/carers were involved in the rehabilitation programme in the majority of children (88%). In the non-surgical patients, parents were the main providers of physiotherapy (90%). Two children received no treatment by a Physiotherapist, during the year of their participation in the study.

Graph 7.1 % of physiotherapy provided by parent, school assistant and therapist in all participants



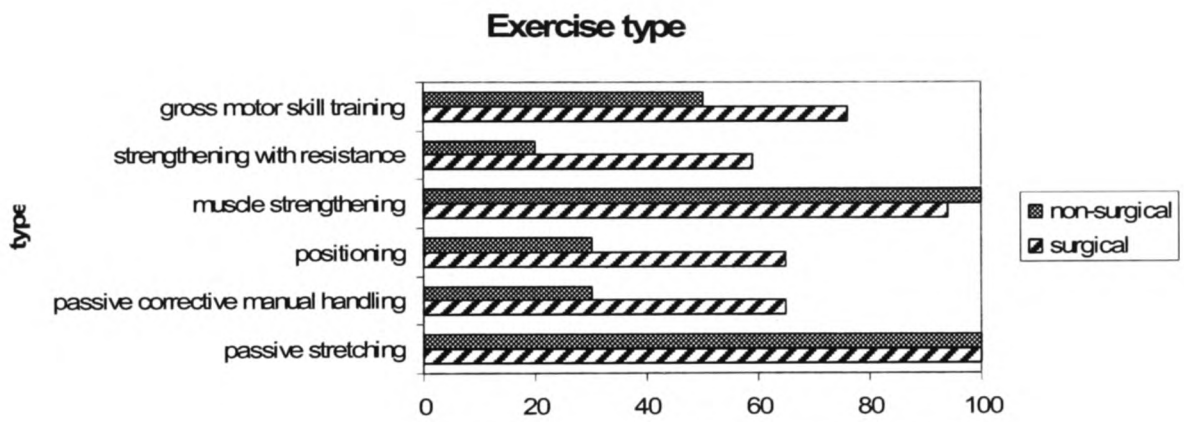
	therapist	assistant	parent
non-surgical	80	60	90
surgical	100	53	88

2. Participation in physical activities other than physiotherapy included mainly physical education at school (PE) (90% non-surgical, 65% surgical). The majority of children in both groups were involved in other physical activities, which included mainly swimming.

3. The type of physiotherapy treatment was different for surgical and non-surgical participants. All surgical patients received musculoskeletal therapy

(100%). In more than half of them (59%) the eclectic approach was used by their Physiotherapists (Chapter 3.4). Only one child from this group undertook Bobath exercises during the study and a further four at a younger age. In the non-surgical group, the majority of children were treated by the eclectic approach a further two underwent Bobath treatment and one conductive education. None of these children had any form of musculoskeletal training.

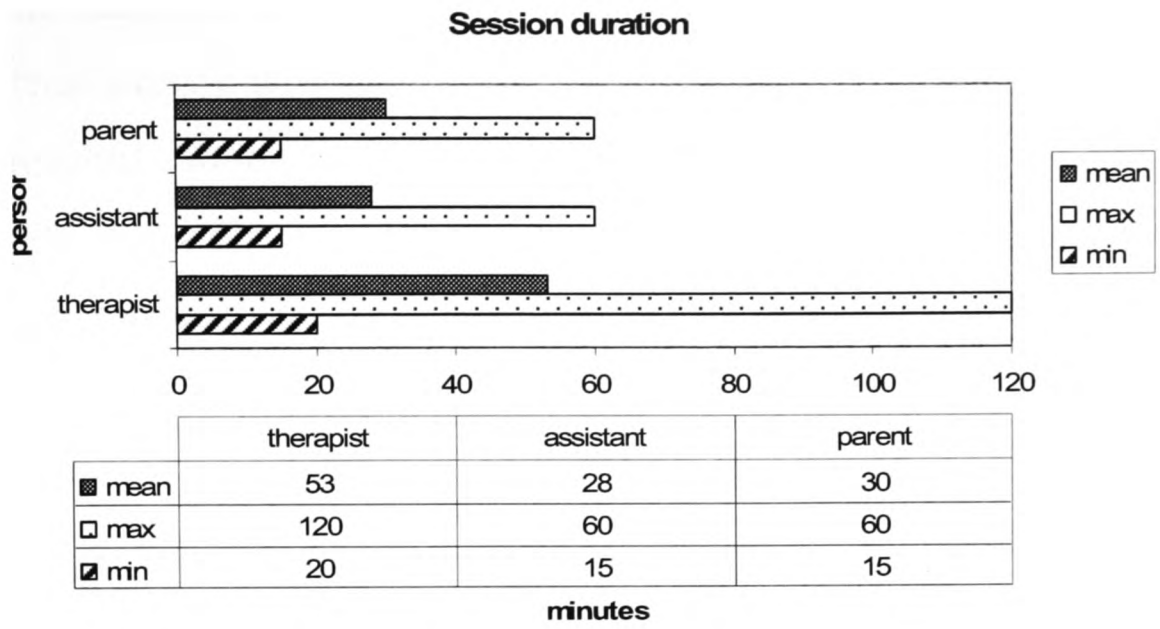
Graph 7.2 Type of exercise provided (%) in surgical and non-surgical patients



	passive stretching	passive corrective	positioning	muscle strengthening	strengthening with	gross motor skill
non-surgical	100	30	30	100	20	50
surgical	100	65	65	94	59	76

%

4. All children in both groups received passive stretching exercises. Strengthening without any form of resistance was undertaken by all non-surgical and the majority of surgical patients (94%). However, resistance training, which was provided manually, was used in only two non-surgical and in half of the surgical patients (59%). Only three older children from the surgical group reported using weights towards the end of the study and after the six weeks physiotherapy intervention.



	therapist	assistant	parent
mean	53	28	30
max	120	60	60
min	20	15	15

Graph 7.3 Duration of physiotherapy session (min) for all subjects

5. The frequency and duration of therapy was variable and dependent on the provider. Overall, children in the non-surgical group received less treatment by a Physiotherapist than the surgical group during their participation to the study with a minimum of two sessions in 24 weeks and maximum one session per week (one child). Two children received no treatment at all by a Physiotherapist. In the surgical group, frequency was also dependent on the progress of rehabilitation. In the three months following surgery (in some cases six months), frequency of therapy was between one session per day (one child) to one session in two weeks with the majority of children receiving one or two sessions weekly. Six months post-operatively the frequency of treatment reduced considerably for some children, especially those approaching adulthood (one/12 weeks for one child, two/year for one child).

7.1.5 Summary

The results of this initial investigation demonstrated that the surgical population of this study received largely similar type of therapy during the year of the study. Interestingly, resistance strengthening using weights was not popular amongst Paediatric Physiotherapists. The frequency and duration of therapy were variable especially in the non-surgical group but

also in the surgical group as the rehabilitation period progressed. The design of this survey was not meant to lead to any definite conclusions but only a general idea on local practice. The interpretation of these observations can only be speculative. These results therefore cannot be regarded as representative of current physiotherapy practice across the country.

7.2 Sample size calculation

The study was a two group randomised controlled clinical trial and the sample size was determined to allow differences in strength variables to be detected.

7.2.1 Sample size calculation

The power calculations were based on published results of a strengthening programme, which examined a number of outcome measures at three different time points (Damiano 1998). These results suggest that the effect of such a programme expressed as an average improvement across all outcomes would be equal to 50 Newton, with a standard deviation of 40 Newton.

This effect was selected as the minimum one that would be important to detect. Any smaller effect would not be of clinical significance. A proposed sample size of 10 pairs of cases, would give this study power of 93% to yield a statistically significant result. Similarly, an observed mean difference of 50 would be reported with a 95.0% confidence interval of 17.52 to 82.68.

7.3 Patients

The group of children, who underwent multi-level surgery, as described in Chapter Six, also participated in this part of the study.
Patients excluded were those who:

1. had undergone Botulinum toxin injections or orthopaedic surgery the year prior to admission to the study
2. were unable to follow commands due to significant learning difficulties.

7.3.1 Randomisation

Twenty-one children, 11 females and 10 males, were recruited initially, at the pre-op stage, ranging in age from seven years nine months to 16 years (mean 12y 5m SD 2y 5m) at the time of entry to the study. At six months post-surgery each child was allocated to one of two strengthening groups B (active strengthening) or C (resistance strengthening). At that point, child MS06 (AS) was excluded from the study due to learning difficulties, which made it impossible to continue with the programme. The remaining 20 children comprised the two groups after randomisation.

The primary objective of randomisation was to ensure that all factors, which could influence outcome, would be equally represented in the two groups, leaving the treatment tested (resistance strengthening versus active strengthening) as the only difference. The factors that were considered as most likely to influence outcome and therefore their equal distribution amongst groups, was necessary included age, sex and functional level (GMFCS). This decision was based on clinical grounds since no published evidence exists, to my knowledge, regarding the influence of sex, age or functional level on muscle strength in this population.

It has been shown that in clinical trials where sample size is small and possible prognostic factors are numerous, a large difference between randomised groups in one or more of the prognostic factors can occur purely by chance and can bias the results of the trial (Treasure 1998). The way to avoid this or, at least, decrease the uncertainty about the validity of the conclusion is by a minimisation technique (Taves 1974). With this technique the group allocation does not rely solely on chance; at the point of recruitment, the treatment allocation is decided by determining which

allocation would minimise any differences in the prognostic factors for the specific patient. For example, if group B (active strengthening) had a higher average age of GMFCS level III patients, then the next older patient with a functional level III would be more likely to be allocated in group C (resistance strengthening). This process was handled entirely by an independent observer, a member of the Statistics department of the University of Oxford, who was given the participants' details as their six-month post-surgery allocation was approaching. She was not a clinician and she was unable to predict treatment outcome based on the information provided. Therefore, introduction of bias during treatment allocation was minimized. Minimisation resulted in two groups of nine and 11 children as shown in Table 7.1. The treatment allocation file is presented in Appendix Five.

Factors					
Sex	**Age group (y)**	**GMFCS**	**Group C (resistance)**	**Group B (active)**	**Grand Total**
Female	<10	II		1	1
	>15	II	2		2
	10 to 14	I	1		1
		II	2	1	3
		III	1	2	3
Male	<10	II		1	1
		III	1		1
	>15	II		1	1
		III	1	1	2
	10 to 14	II	1	2	3
		III	2		2
Grand Total			**11**	**9**	**20**

Table 7.1 Treatment allocation in groups B & C based on sex, age and GMFCS level

7.4 Method

Muscle strength, function and gait were assessed before and after the strengthening programme following multi-level orthopaedic surgery. The average time from pre-op testing to pre and post - physiotherapy assessment was 28 (sd 3) and 37 (sd 4) weeks respectively. Only two children, MS08 (CM) and MS12 (QS), had their pre-physio testing at 44 wks and post-physio testing at 55 wks due to changes in their surgery date. Participant MS20 (DB) did not follow the physiotherapy programme and did not attend the post-physiotherapy assessment.

7.4.1 Outcome measures

The outcome measures used in this part of the study were those described in Chapter Six.

Isometric muscle strength of hip flexors, hip extensors, hip abductors, knee flexors, and knee extensors at 90° and 30° of flexion was measured using the same protocol described in the pilot study (Chapter Five). In a few cases, testing of specific muscles was not possible; a detailed description of missing values and reasons for exclusion is presented in Appendix Five.

Three-dimensional gait analysis was performed using a Vicon 612 motion analysis system with 15 retro-reflective surface markers on the pelvis and lower limbs. This included clinical examination (ROM, muscle strength, tone, selective control and anthropometric measurements), electromyography, kinematic, kinetic and video data. Each child walked along a 10 meter walkway at their self-selected walking speed and a minimum of four walking trials was collected. Data were recorded with the children walking with orthoses and/or assistive devices during the two post-operative assessments before and after physiotherapy.

Motor function, as described in Chapter Six, was assessed using the Gross Motor Function Measure (GMFM 88), which was administered by the author at either the Nuffield Orthopaedic Centre gymnasium or the Gait Laboratory. Testing was performed with the child barefoot in appropriate clothing and in the presence of his/her guardian. Community ambulation was also assessed with the walking scale subset of the Gillette Functional Assessment Questionnaire (FAQ) (Novacheck 2000).

7.4.2 Strengthening protocol

In order to examine the role of progressive resistance exercise in improving strength, gait and function after multi-level surgery, two strengthening regimes were designed. The duration of the programmes, the frequency of the sessions and the type of the isotonic exercises were the same in both groups B and C. The main difference between the two regimes was the presence or absence of resistance (or load).

Groups B (active strengthening) & C (progressive resistance strengthening) received a physiotherapy programme consisting of three sessions per week for six weeks. At six months post-operatively the patient was expected to have achieved reasonable mobility. The aim was to complete 18 sessions with a rest day between sessions. The programme was administered at home or school by the author or another clinician trained in paediatric physiotherapy methods for the purpose of the study.

Group B received physiotherapy based on conventional techniques, which are considered common practice in paediatric physiotherapy. Information collected from the study sample of community therapists helped define the active strengthening protocol. This is presented below:

Group B - Active strengthening programme

Warm – up: 5 to 7 min. If it is easy for the patient to walk, ask him/her to walk around the room for 2 min

Passive stretching exercises (without orthoses)

The passive stretching programme is determined by the findings of the 6 month post-op clinical assessment. Only joints which have residual tightness after surgery are stretched.

Hip flexors: Starting position - prone lying, hip at 0°, knee at 90°. Therapist holds knee and extends hip towards ceiling while stabilising pelvis with other hand. Maintain maximum hip extension for count of 10. Repeat with each leg x 10.

Hamstrings: Starting position - supine lying, hip and knee at 90°. Therapist lifts lower leg towards ceiling to achieve maximum knee extension, while maintaining hip at 90°. Maintain maximum stretch for count of 5. Repeat with each leg x 10.

Hip Adductors: Starting position – supine lying, hip and knee at 0°. Therapist abducts the straight leg, stabilising the pelvis for compensatory movements. Maintain maximum stretch for count of 5. Repeat with each leg x 10.

Quadriceps: Starting position – prone lying, hip and knee at 0°. Therapist flexes the knee slowly, keeping the other leg straight. Maintain maximum stretch for count of 5. Repeat with each leg x 10.

Strengthening exercises (without orthoses, without resistance)

The starting position for each muscle group may vary depending on the strength of the individual patient as this is determined clinically at the 6 month post-op assessment e.g if the hip flexors have antigravity strength then the starting position would be sitting; if the hip flexors have subgravity strength then the starting position would be side lying etc.

Hip flexors: Starting position - sitting with hips and knees at 90°, arms crossed. Patient lifts knee up towards ceiling and holds for count of 5. Perform 3 sets of 10 repetitions with each leg.

Hip extensors (isolate Gluteus maximus): Starting position - side lying, hip at 0° and knee at 90°. Therapist supports leg with one hand and stabilises pelvis with other. Patient actively extends hip and maintains for count of 5.

Perform 3 sets of 10 repetitions with each leg. Aim to progress to prone position and straight leg raise.

Hip abductors: Starting position – supine lying, hip and knee at 0°. Patient abducts leg to side with therapist stabilising pelvis. Maintain neutral hip extension at all times – tendency to use stronger hip flexors). Maintain for count of 5. Perform 3 sets of 10 repetitions with each leg.

Knee flexors: Starting position – prone lying, hip and knee at 0°. Patient flexes knee to 90° and maintains for count of 5. Perform 3 sets of 10 repetitions with each leg.

Knee extensors: Starting position – sitting, hip and knee at 90°. Patient extends knee to neutral and maintains for count of 5. Perform 3 sets of 10 repetitions with each leg. Control last degrees of extension.

Functional exercises (with orthoses if barefoot uncomfortable)

Standing - Step-ups with hand support as necessary. Repeat leading with each leg 10 times.

Crook lying (bridges) - Gluteal contractions and pelvis lifting 10 times.

Standing - Side stepping with hand support as necessary. Repeat leading with each leg 10 times.

Sitting - arms crossed if possible. Controlled sitting to standing & standing to sitting. Repeat 10 times.

Cool down: up to 5 min, as for warm up.

In order to answer the research question outlined in the introduction, the experimental leg of the trial (Group C) received a strengthening programme of progressive resistance isotonic exercises using weights. The protocol was designed based on the strength training literature, as discussed in chapter 4 (Atha 1981; Damiano 1995; Faigenbaum 2000; American Academy of Pediatrics Committee on Sports Medicine and Fitness. 2001); it is presented below:

Group C - Resistance strengthening programme

Warm – up: 5 to 7 min. If it is easy for the patient to walk, ask him/her to walk around the room for 2 min

Passive stretching exercises (without orthoses)

Hip flexors: Starting position - prone lying, hip at 0°, knee at 90°. Therapist holds knee and extends hip towards ceiling while stabilising pelvis with other hand. Maintain maximum hip extension for count of 10. Repeat with each leg x 10.

Hamstrings: Starting position - supine lying, hip and knee at 90°. Therapist lifts lower leg towards ceiling to achieve maximum knee extension, while maintaining hip at 90°. Maintain maximum stretch for count of 5. Repeat with each leg x 10.

Hip Adductors: Starting position – supine lying, hip and knee at 0°. Therapist abducts the straight leg, stabilising the pelvis for compensatory movements. Maintain maximum stretch for count of 5. Repeat with each leg x 10.

Quadriceps: Staring position – prone lying, hip and knee at 0°. Therapist flexes the knee slowly, keeping the other leg straight. Maintain maximum stretch for count of 5. Repeat with each leg x 10.

Progressive resistance strengthening exercises (without orthoses, using weights)

Hip flexors: Starting position - sitting with hips and knees at 90°, arms crossed. Patient lifts knee up towards ceiling and holds for count of 5. Perform 3 sets of 10 repetitions with each leg. Weight placed at distal thigh.

Hip extensors (isolate Gluteus maximus): Starting position - side lying, hip at 0° and knee at 90°. Therapist supports leg with one had and stabilises pelvis with other. Patient actively extends hip and maintains for count of 5. Perform 3 sets of 10 repetitions with each leg. Aim to progress to prone position and straight leg raise. Weight placed at distal thigh.

Hip abductors: Starting position – supine lying, hip and knee at 0°. Patient abducts leg to side with therapist stabilising pelvis. Maintain neutral hip extension at all times – tendency to use stronger hip flexors). Maintain for count of 5. Perform 3 sets of 10 repetitions with each leg. Weight placed at distal thigh.

Knee flexors: Starting position – prone lying, hip and knee at 0°. Patient flexes knee to 90° and maintains for count of 5. Perform 3 sets of 10 repetitions with each leg. Weight placed at ankle.

Knee extensors: Starting position – sitting, hip and knee at 90°. Patient extends knee to neutral and maintains for count of 5. Perform 3 sets of 10 repetitions with each leg. Control last degrees of extension. Weight placed at ankle.

Cool down: up to 5 min, as for warm up.

A variety of exercise weights was used in the study. The child attempted to lift 10 times the maximum weight he or she could. The selected weight was kept for at least three sessions since children progressed at a different rate. Re-assessment and incremental weight increase were dictated by the child's progress.

All children continued with their routine physiotherapy regime for the duration of the study, as dictated by their clinical needs. Routine physiotherapy was controlled as it was similar for both groups and did not include weight training. It represented the clinical practice amongst local Paediatric Physiotherapists, as the interviews demonstrated, and did not pose any ethical problems. A log was kept of the frequency and content of physiotherapy sessions for each participant.

7.4.3 Statistical analysis

The experimental plan was a 'mixed design' with two experimental factors: subject groups (multi-level surgery & non-resistance strengthening B, and multi-level surgery & resistance strengthening C) and time-points (six months post-operatively, before and after physiotherapy programme). For the statistical analysis, parametric paired t tests were performed for intra-group comparisons. For inter-group comparisons a 'factorial repeated-measures analysis of covariance' or ANCOVA was used in order to determine the effects of the intervention (physiotherapy programme) in

modifying the outcome measures described in 'methods' and their changes in time. Significance levels of $p < 0.05$ and confidence intervals CI of 95% were used.

7.5 Results

Nineteen patients were included in the analysis. Eight patients completed programme B and 11 completed programme C. Patient MS20 (DB) did not complete the programme and did not attend the six months post-physiotherapy testing. The target number of physiotherapy sessions per child was 18. On average, participants completed 17 sessions with minimum number 14 (MS04, AH). The programme was administered by the author to all children. Two clinicians familiar with the physiotherapy programme of the study and employed by the Oxford Gait Laboratory assisted with four of the children. The programme was administered at home, school or the hospital depending on individual arrangements. A log of physiotherapy sessions is presented in Appendix Five.

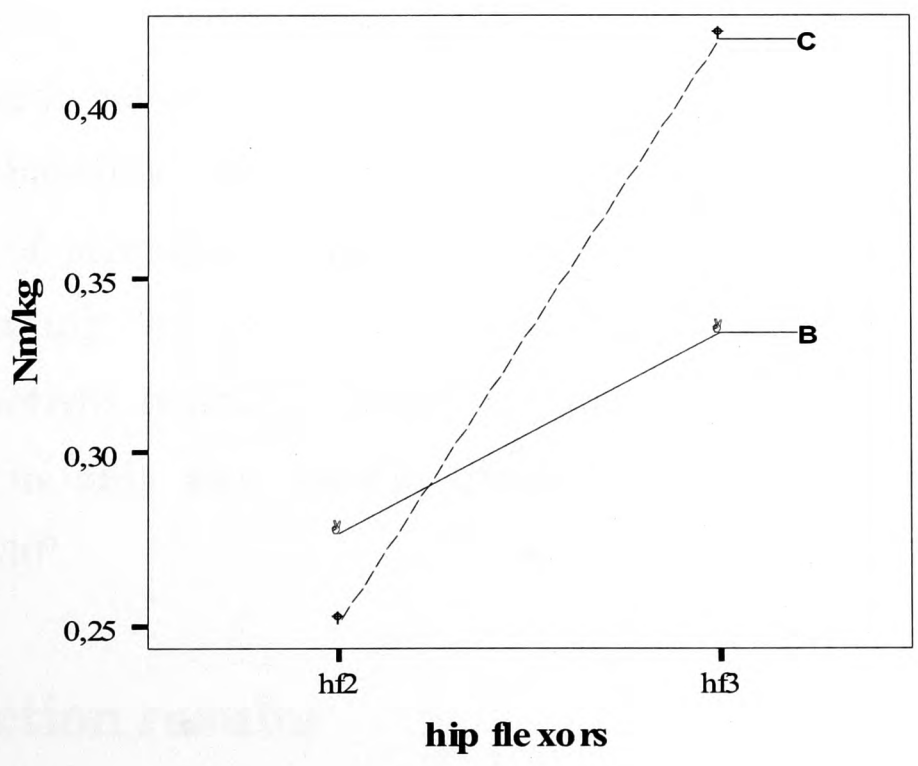
7.5.1 Strength results

Muscle strength values (N) from three contractions were collected. These were converted to moments by multiplying the dynamometer reading by the lever arm (m) of the contraction (the distance of the transducer to the joint centre). Moments were normalised to body mass (Nm/kg) and the highest value was used for statistical analysis. Intra – group comparison results before and after physiotherapy, are presented in Table 7.2.

Muscle Group Nm/kg	6 m I Pre-physio mean (SD)		6 m II Post-physio mean (SD)		Paired mean diff (SD) 6mI / 6mII		p_{ii} 6mI / 6mII	
	B	C	B	C	B	C	B	C
Hip flexors	0.3(0.1)	0.3(0.1)	0.3(0.2)	0.4(0.2)	-0.0(0.1)	-0.2(0.1)	0.122	**0.000**
Hip extensors	1.8(0.7)	2.1(0.8)	2.1(0.8)	2.3(0.6)	-0.1(0.3)	-0.2(0.4)	0.157	0.075
Hip abductors	0.5(0.2)	0.5(0.3)	0.6(0.2)	0.6(0.3)	-0.1(0.1)	-0.1(0.1)	**0.036**	**0.000**
Knee flexors	0.4(0.2)	0.4(0.2)	0.5(0.3)	0.5(0.2)	-0.1(0.1)	-0.1(0.1)	**0.004**	**0.001**
Knee extensors 90°	1.0(0.4)	1.1(0.5)	1.2(0.5)	1.3(0.5)	-0.1(0.2)	-0.2(0.2)	**0.008**	**0.000**
Knee extensors 30°	0.3(0.1)	0.4(0.3)	0.5(0.1)	0.5(0.3)	-0.1(0.1)	-0.1(0.1)	**0.021**	**0.000**

Table 7.2 Pre (I) and post (II) muscle strengthening values (Nm/kg), paired mean differences after physiotherapy in both groups B & C (sig. at $p < 0.05$)

Both groups showed improvement of muscle strength following the six-week programme but group C showed significant strength increase in more muscle groups than group B. Graph 7.4 shows that difference for the hip flexors.



Graph 7.4 Hip flexors strength pre (hf2) and post-therapy (hf3) in both groups

Analysis of co-variance (ANCOVA) was used to identify any existing differences between groups.

Dependent Variable	Parameter estimates – Significance of effect
	Group B vs C
Hip flexors Nm/kg	**0.02** ↓
Hip extensors Nm/kg	0.50 ↓
Hip abductors Nm/kg	0.28 ↓
Knee flexors Nm/kg	0.97 ↓
Knee extensors at 90° Nm/kg	0.75 ↓
Knee extensors at 30° Nm/kg	**0.05** ↓

Table 7.3 Group B & C analysis of co-variance (ANCOVA). The parameter estimates show the effect of the predictor (group) on the dependent variable (i.e.hip flexors Nm/kg e.t.c.). ↓ indicates that given two people with similar pre – physio values, it can be expected that the post – physio value of those in group B to be less than of those in group C. The opposite is valid for ↑.

The model was built with group, GMFCS and sex as fixed factors. Age and muscle strength before physiotherapy were used as covariates in order to adjust for baseline and eliminate any pre-intervention differences. Comparison of strength changes between groups showed that resistance strength training led to higher increase in muscle strength than no-resistance (active) training. However, this difference achieved statistical significance in only two muscle groups, namely hip flexors and knee extensors at 30°.

7.5.2 Function results

Gross motor function was assessed using the GMFM and temporal parameters obtained from gait analysis. The overall GMFM score is presented in Table 7.4, along with the dimension E score of the scale, which

tests walking, running and jumping. Normalised velocity, cadence and step length were also included.

GMFM scores & Temporal parameters	6 m I Pre-physio mean (SD)		6 m II Post-physio Mean (SD)		Paired mean diff (SD) 6mI / 6mII		p_{ii} 6mI / 6mII	
	B	**C**	**B**	**C**	**B**	**C**	**B**	**C**
GMFM total %	75.2(15.9)	78.9(13.4)	83.9(10.4)	83.9(11.4)	-8.0(8.6)	-5.0(4.1)	**0.035**	**0.002**
GMFM E %	44.1(31.3)	45.3(28.8)	60.8(26.5)	55.6(28.0)	-15.1(13.2)	-10.2(9.0)	**0.014**	**0.004**
N walk speed	0.3(0.1)	0.3(0.1)	0.3(0.1)	0.3(0.1)	-0.02(0.1)	-0.04(0.0)	0.346	**0.000**
N cadence	0.5(0.1)	0.4(0.1)	0.5(0.1)	0.4(0.1)	-0.02(0.1)	-0.03(0.0)	0.465	**0.001**
N step length Right	0.6(0.1)	0.6(0.2)	0.6(0.2)	0.7(0.1)	-0.02(0.1)	-0.08(0.1)	0.663	0.069
N step length Left	0.6(0.1)	0.6(0.2)	0.7(0.2)	0.6(0.2)	-0.03(0.1)	-0.08(0.1)	0.442	**0.001**

Table 7.4 Inter-group comparisons for GMFM total and dimension E %, normalised walking speed, cadence and step length

These results show that overall function improved in both groups after the physiotherapy programme. However, only resistance training resulted in statistically significant improvement of the temporal parameters. Although, between groups comparisons did not yield any statistically significant results, a trend towards larger improvement was seen in group C with the exception of GMFM scores (Table 7.5).

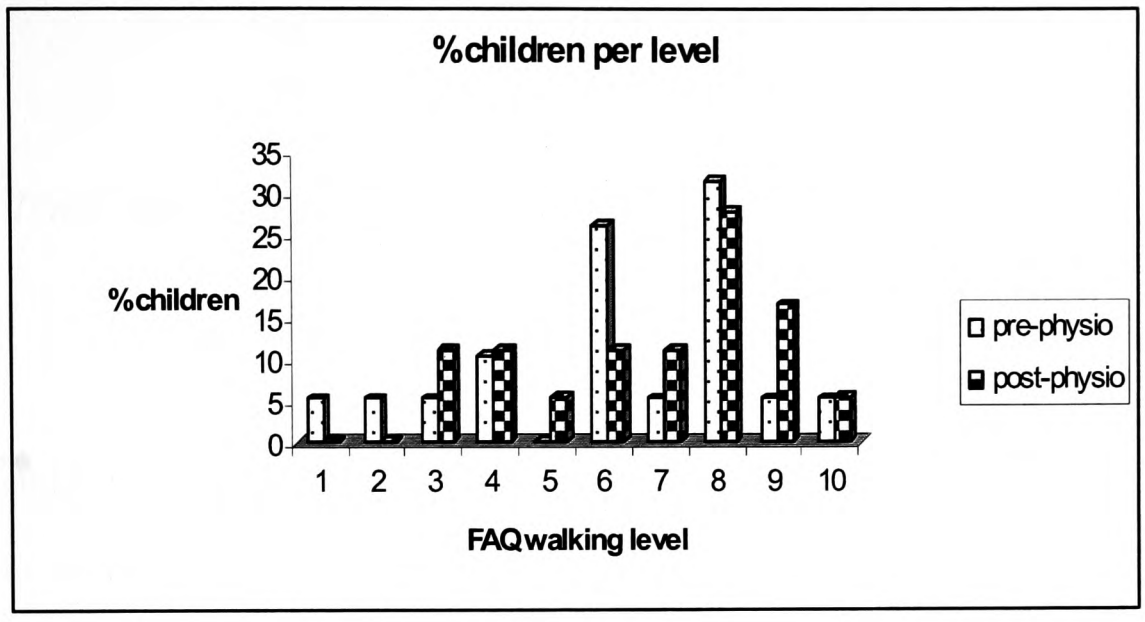
Dependent Variable	**Parameter estimates – Significance of effect**
	Group B vs C
GMFM %	0.75 ↑
GMFM dimension E %	0.88↑
N velocity	0.42 ↓
N cadence	0.76 ↓
N step length Right	0.06 ↓
N step length Left	0.26 ↓

Table 7.5 Group B & C analysis of co-variance (ANCOVA) for motor function. The parameter estimates show the effect of the predictor (group) on the dependent variable (i.e.N velocity e.t.c.). ↓ indicates that given two people with similar pre – physio values, it can be expected that the post – physio value of those in group B to be less than of those in group C. The opposite is valid for ↑.

Analysis of the walking scale scores of the FAQ is presented in the Graph 7.5. As a reminder, not taking any steps and not walking on a routine basis are described by levels one and two. Exercise ambulation levels are three-four. Level five is consistent with household or classroom walking and level six is limited community walking. Routine community walking on level surfaces is considered a level seven. Levels eight and nine are more advanced levels of walking on a variety of terrains. Level 10 is consistent with a typical, non compromised level of walking ability.

As shown in Chapter Six, many children were rated at a lower level of the walking scale at six months following surgery. The majority of them were rated at least level six on the walking scale of the FAQ with only two children rated as level four pre-operatively. At six months following surgery the parents' impression of the child's walking ability was quite different, representing a loss in function as shown by the drop in the GMFM score. Graph 7.5 shows that following a six week strengthening programme (with

or without resistance), children maintained or improved their community walking ability.



Graph 7.5 % of children in sample at each FAQ walking level before and after the 6-week physiotherapy programme

Interestingly, those children whose level of the FAQ scale dropped the most (subjects 15 and 16) showed the greatest improvement after the physiotherapy programme (Table 7.6).

Subject	FAQ pre-physio	FAQ post-physio
1	6	8
2	9	9
3	8	8
4	10	10
5	6	5
6	7	7
7	6	9
8	4	4
9	6	6
10	6	,
11	8	7
12	8	8
13	4	4
14	3	3

15	**1**	**3**
16	**2**	**6**
17	8	8
18	8	9
19	,	,
20	8	8

Table 7.6 Individual change of FAQ walking level per subject before and after the 6 weeks strengthening programme

7.5.3 Gait results

Kinematic and kinetic data were extracted following processing and the average of three trials was used for analysis. Pre-selected gait variables as discussed in Chapter Six were then imported to SPSS statistical programme for further analysis.

Kinematic Variables	**6 m I Pre-physio mean (SD)**		**6 m II Post-physio mean (SD)**		**Paired mean diff (SD) 6mi / 6mII**		**p_{ii} 6mI / 6mII**	
	B	**C**	**B**	**C**	**B**	**C**	**B**	**C**
RPT (°)	7.9(2.4)	8.2(3.0)	7.9(2.3)	8.0(3.9)	0.1(3.1)	0.2(1.8)	0.926	0.619
RHF (°)	43.1(10.1)	43.5(8.4)	45.2(8.5)	44.2(8.2)	0.0(7.2)	-0.7(4.2)	0.979	0.434
RKF (°)	46.4(16.9)	51.2(15.4)	46.8(12.0)	48.5(11.8)	2.7(5.6)	2.7(5.3)	0.078	**0.026**
RAD (°)	13.4(9.2)	10.5(5.6)	13.6(8.4)	11.8(8.0)	0.2(2.9)	-1.3(4.1)	0.733	0.140
MHES (°)	8.8(13.0)	5.2(14.5)	8.1(10.7)	5.7(13.9)	-0.1(8.2)	-0.6(4.9)	0.950	0.600
MKES (°)	12.3(16.5)	6.9(18.2)	13.1(12.8)	8.6(17.2)	-3.0(6.8)	-1.8(3.7)	0.094	**0.033**
MKFS (°)	58.4(6.1)	58.0(7.5)	59.9(9.3)	57.0(9.3)	-0.6(6.7)	1.1(5.1)	0.746	0.323
KFIC (°)	26.1(12.3)	17.0(14.5)	27.1(12.5)	16.8(13.1)	-1.8(5.3)	0.1(4.3)	0.181	0.908
RPO (°)	6.6(2.1)	8.2(3.0)	6.7(2.6)	8.7(3.9)	0.2(2.3)	-0.5(2.4)	0.785	0.389
RPR (°)	15.2(3.9)	18.2(5.6)	16.1(4.8)	17.2(5.7)	-0.9(4.4)	1.0(4.8)	0.446	0.326
MTR (°)	6.4(8.0)	3.5(22.8)	7.0(7.1)	10.1(11.8)	-1.6(6.8)	-6.6(26.9)	0.351	0.261

Table 7.7 Kinematic variables (mean, sd, mean difference) before and after the physiotherapy programme in groups B & C ($p < 0.05$) RPT = range pelvic tilt, RHF = range hip flexion, RKF = range knee flexion, RAD = range ankle dorsiflexion, MHES = max hip extension stance, MKES = max knee extension stance, MKFS = max knee flexion swing, KFIC = knee flexion initial contact, RPO = range pelvic obliquity, RPR = range pelvic rotation, MTR = max thigh rotation

As shown in Tables 7.7 and 7.8 a few kinematic and kinetic parameters, mainly those involving the knee joint and musculature, did show some statistical significance. This could mean that the knee joint and muscle groups were affected more from the intensive strengthening regime. However, interpretation of these results is difficult since they do not present a specific trend.

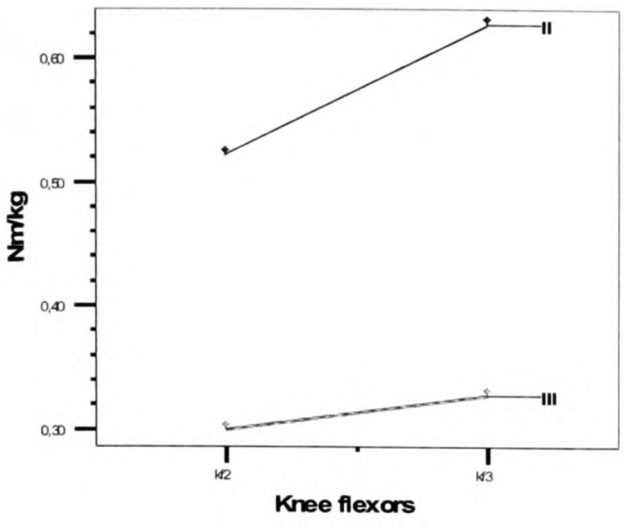
Similarly, inter-group comparison results following ANCOVA were not statistically significant (Appendix Five).

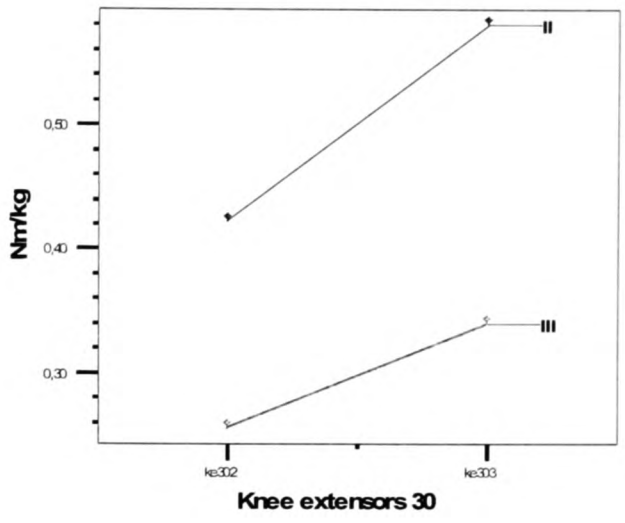
Kinetic Variables	6 m I Pre-physio mean (SD)		6 m II Post-physio mean (SD)		Paired mean diff (SD) 6mi / 6mII		p_{ii} 6mI / 6mII	
	B	C	B	C	B	C	B	C
HEM	1571.2(451.3)	1056.0(419.3)	1500.1(336.4)	1092.9(351.0)	71.1(211.5)	-60.4(223.3)	0.315	0.349
HFM	-538.5(94.7)	-555.4(164.4)	-505.6(130.0)	-557.6(151.5)	-32.9(150.3)	25.5(106.8)	0.506	0.406
KEM	388.5(115.7)	338.8(169.9)	581.7(263.1)	372.9(147.6)	-193.3(161)	-58.8(177.8)	**0.004**	0.256
KFM	-476.4(163.3)	-541.0(176.0)	-415.1(132.6)	-453.4(145.5)	-61.4(84.2)	-91.0(103.5)	**0.047**	**0.008**
MPM	1057.2(232.5)	1134.1(133.4)	1123.3(191.8)	1110.4(126.0)	-66.1(75.4)	42.1(122.2)	**0.022**	0.238
MHAM	395.9(107.9)	350.8(113.3)	417.2(169.1)	344.7(138.6)	-21.3(117.2)	-30.3(82.1)	0.580	0.209
MHP	1.7(0.8)	1.1(0.5)	1.8(0.5)	1.1(0.4)	-0.1(0.6)	-0.1(0.3)	0.438	0.355
MAP	1.2(0.7)	0.8(0.3)	1.3(0.8)	1.0(0.9)	-0.1(0.2)	-0.3(0.8)	0.168	0.133

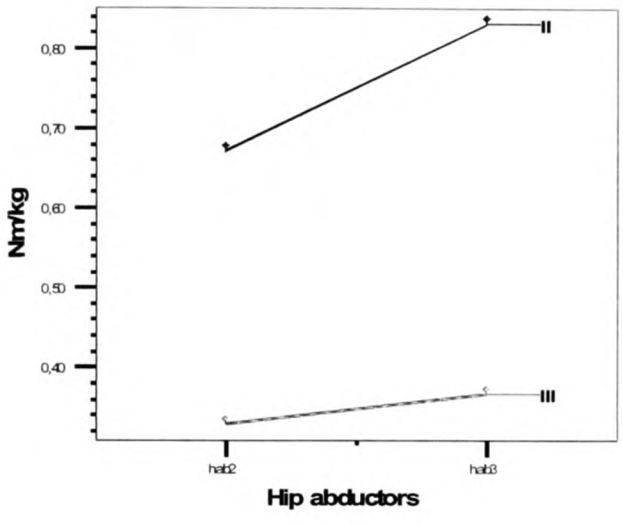
Table 7.8 Kinetic variables (mean, sd, mean difference) before and after the physiotherapy programme in groups B & C ($p < 0.05$), HEM = hip extension moment, HFM = hip flexion moment, KEM = knee extension moment, KFM = knee flexion moment, MPM = max plantarflexion moment, MHAM = max hip abduction moment, MHP = max hip power, MAP = max ankle power

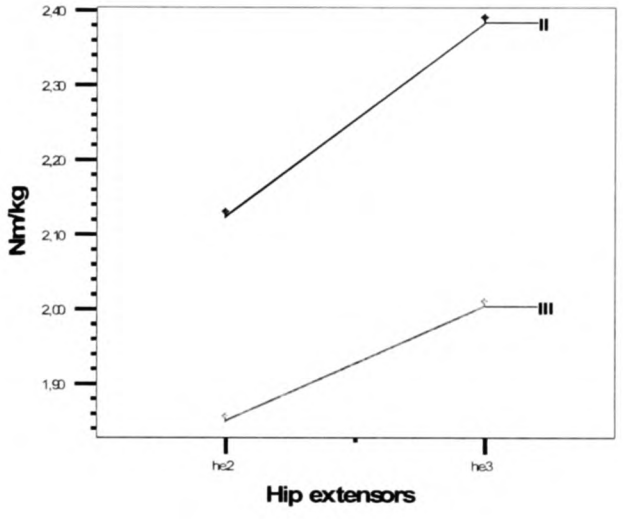
7.5.4 Changes per GMFCS level

Subjects were also compared before and after physiotherapy based on their functional level instead of their group assignment. Analysis of co-variance revealed a consistent finding, across the majority of tested variables. It was shown that children with a higher functional level (II) responded better to strength training than less functional children (III), irrespective of the type of physiotherapy that they had received.









Graph 7.6 Muscle strength pre and post physiotherapy per GMFCS level

7.6 Discussion

In Chapter Six, it was shown that at six months after multi-level surgery there was improvement in gait analysis parameters, illustrated by the changes in kinematics and kinetics. However, there was disproportionate decrease of strength and function, at this, relatively early, stage of rehabilitation. The question on whether intensive physiotherapy regimes could alter this imbalance early in the rehabilitation period and speed up the patients' recovery was dealt with in this Chapter.

Limited evidence exists about the effect of specific physiotherapy regimes following multi-level surgery (Patikas 2006). Therefore, published

guidelines for post multi-level physiotherapy do not exist, to my knowledge. The results presented in this chapter show that using two different intensive physiotherapy regimes is an effective way to improve strength and motor function after surgery. This was the case in both resistance strengthening and active strengthening groups. Furthermore, a progressive resistance strengthening programme showed advantages over active strengthening post-operatively. This is in agreement with previous findings in non surgical patients (Damiano 1995; Damiano 1998; Dodd 2003), but contradicts the recent findings from Patikas et al (2006) in patients following surgery. In this randomised controlled trial of strength training after multi-level surgery, the group that followed a specific strength training programme in addition to regular physiotherapy, failed to show any significant advantages compared with the group that underwent only conventional physiotherapy. Their long-term training programme (40 weeks), lack of supervision and early initiation of the programme after surgery may explain these results.

Although the results presented earlier in this Chapter illustrate a trend towards the resistance group showing better muscle strength recovery following the intensive treatment, statistical significance in the between groups comparison was achieved in two muscle groups only, namely hip flexors and knee extensors at 30°. However, when considering each group separately, in comparing strength before and after treatment, the resistance strengthening group showed improvement in a greater number of muscle groups. The same trend was noticed when function and gait variables were tested. Despite the non-statistically significant differences in between-groups comparisons observed, only resistance training resulted in significant improvement of the temporal parameters when groups were assessed separately. Previous studies have documented well the advantages of strength training in patients with CP. Although results from this study are not conclusive, they certainly show the overall benefits of strength training post-operatively and indicate that resistance training may have

advantages over active exercise. There are several explanations for these conclusions.

Most previous studies that assessed the effectiveness of strength training in CP have used training protocols with a duration of six to eight weeks (Horvat 1987; Damiano 1995; Damiano 1995; MacPhail 1995; Damiano 1998). However, it has been suggested that a training programme longer than six weeks is required to show the benefits of exercise in CP patients (Damiano 1995) and patients with spinal cord injuries (Hicks 2003). The short duration of the programme in this study may be one reason for not revealing significant differences between the two regimes in all the tested parameters. However, this was considered adequate for the time frame and resources available as well as for maintaining the motivation of the participants. This last issue has shown to be a problem in longer training regimes (Patikas 2006).

It could also be argued that earlier initiation of strength training may have led to an earlier improvement in motor function. However, some of these children and particularly the more severely affected ones are unable to co-operate sufficiently in a vigorous strengthening programme at an earlier stage. Patikas et al (2006) started their programme at three to four weeks post-operatively and still no benefits of strength training were shown. On balance, the six months point was chosen in this study as the earliest opportunity where sufficient recovery would allow strength training, while overall rehabilitation is still ongoing and further improvement in function is still expected.

Another factor that may have affected these results is the magnitude of resistance given in the resistance strengthening group. Damiano (1995) used a progressive resistance protocol in their study of quadriceps femoris strengthening in spastic diplegic children. They determined the load used by measuring the MVC of the quadriceps at 90° of knee flexion, doubling this

value and using 65% of this load. This method was found inadequate for the present study design since the set up for measuring muscle strength was a fixed one and required patients to be assessed at the gait analysis laboratory. Furthermore and in accordance with the paediatric strength training literature (Faigenbaum 2000; American Academy of Pediatrics Committee on Sports Medicine and Fitness. 2001) the 10-repetition maximum (RM) was used to determine maximum effort. The child attempted to lift 10 times the maximum weight he or she could achieve and this became the training load for that session, until re-assessment was undertaken. For some, more severely affected, children however this proved to be difficult and a sub-maximal weight was used for a few training sessions (11 or 12 RM). Although, this was a safe and quick method of determining the training load, it may have led to less intensive training in a number of children.

It should also be mentioned that although most parameters that could be controlled were considered, there were still several parameters that could have affected the outcome. All subjects were spastic diplegics with a GMFCS level between I and III. However, there is significant variation between these functional levels that could alter the response to exercise. Interestingly, it was found that children with a higher functional level responded better to strengthening exercises, irrespective of the protocol used. To my knowledge, there is no published evidence to support these findings, but its implications for designing appropriate post multi-level rehabilitation regimes are obvious. Another parameter that was not examined or controlled was the learning effect between assessments. However, it was assumed that following a long interval of six weeks between testing sessions, this effect would be minimal.

All children had multi-level orthopaedic surgery by the same Paediatric Surgeon and received the training programme by the same trained clinician in the majority of cases. However, the operations performed were dictated

by each child's clinical needs and they could not be standardised. In agreement with Patikas et al (2006), the administered strength training programmes may have not been sufficiently long or intensive to overcome the effects of surgery. Although, once could attempt to analyse the combined effects of surgery and physiotherapy on an individual basis this was not considered adequate for this study design. The size of the study would bias considerably any statistical method used to assess this effect.

To my knowledge this is the first objective comparison between different physiotherapy regimes following multi-level surgery in CP. In total, the project of post-operative physiotherapy for patients required covering more than 20.000 miles in 27 months, highlighting the difficulties of conducting tightly controlled physiotherapy trials. This may be responsible for the limited research in this area.

This study demonstrated that following six weeks of intensive physiotherapy treatment focused on strength training, muscle strength and motor function improved significantly. This finding further underlines the deleterious effects of muscle weakness on motor function in CP and emphasises the importance of strength training in the rehabilitation of children with CP following multi-level surgery.

7.7 Summary

1. There is limited evidence based information about the effect of specific physiotherapy programmes following multi-level surgery.
2. No published guidelines exist for post multi-level orthopaedic surgery physiotherapy regimes.
3. Using two different intensive physiotherapy regimes it was shown that muscle strengthening at six months post-surgery is effective in improving strength and motor function.
4. A randomised trial between the two different physiotherapy regimes showed advantages of resistance training over active exercise.

5. Higher functional level may play a significant role in deciding the type and duration of such programmes.
6. Further research with larger samples (multi-centre and longer regimes) is required to identify significant treatment effects.

Chapter Eight

8. Changes of strength, gait and function over one year

In order to answer the final research question outlined in Chapter One (what are the degree and distribution of muscle weakness and strength changes over time in relation to gait and motor function in ambulant children with spastic diplegia?), appropriate data were collected at one year following recruitment. The overall study was designed to allow surgical and non-surgical patients to be followed for a period of 12 months. Candidates for multi-level surgery were recruited and assessed at different stages. Children were examined at baseline, six and 12 months after entering the study. Those who had surgery were assessed twice at six months post-operatively, before and six weeks after physiotherapy.

There is limited information available about the natural history of weakness in CP and how it changes over time. To my knowledge, there is no literature available on the natural history of muscle strength in patients with CP into adulthood. It has been suggested, however, that motor disabilities are affected by ageing and that physical inactivity leads to gradual deterioration (Henderson 2004; Henderson 2005). In the present study, it was attempted to illustrate this deterioration in a group of CP patients who did not receive any form of treatment other than their usual physiotherapy according to their clinical needs.

Multi-level orthopaedic surgery is often used to correct musculoskeletal deformities in CP in order to improve function. Furthermore, it aims to improve muscle balance by tendon lengthening and transfers, which can

potentially weaken the already weak muscles (Wiley 1998). Evidence based post-operative regimes are lacking and only one recent study is published which has attempted to monitor long-term effects of a strengthening programme following surgery (Patikas 2006). The present study followed surgical patients over one year in order to assess the speed of recovery of strength, function and gait after surgery and intensive physiotherapy.

8.1 Changes in strength, function and gait in the non-surgical group over one year

Ten children, five boys and five girls, with a diagnosis of cerebral palsy/spastic diplegia ranging in age from seven years 10 months to 16 years 11 months at the time of recruitment (mean age 11 years, SD 3 years) participated in this prospective evaluation of muscle strength, gait and functional changes over a period of 12 months. Children were excluded if they had been treated with Botulinum Toxin or orthopaedic surgery in the year prior to admission to the study. According to the study protocol, these ambulant children (Gross Motor Functional Classification System levels I-III) should have been candidates for multi-level orthopaedic surgery, who declined surgery at the time of recruitment. However, such patients were difficult to recruit, considering the single-centre design of the study and the time limitations. Therefore, these patients, although potentially suitable for surgery, had actually a slightly younger average age than the surgical group (non surgical mean age 11 years, surgical mean age 13 years) and a higher functional level (GMFCS I or II).

Families were approached at the weekly Cerebral Palsy clinic at the NOC and information regarding the purpose and design of the study was provided. Upon agreement, a consent form was signed by the legal guardian during the child's first attendance for gait analysis, strength and function assessment.

Child	*Age (y/m)*	*Sex*	*Mass (kg)*	*GMFCS Level*
1	14.8	M	57	I
2	11.8	M	31	II
3	7.1	M	24	II
4	9.2	M	22	I
5	8.11	F	31	I
6	8.11	F	32	I
7	9.8	F	27	I
8	14.3	F	49	I
9	11.1	F	56	I
10	16.11	M	49	I
Mean (SD)	11(3)		38(14)	
Total legs	20			

Table 8.1 Characteristics of non-surgical subjects comprising Group A

At six months and one year following recruitment the children were assessed again using gait analysis, strength and function testing. During the last appointment, telephone or mail interviews with the community physiotherapist or the parent were undertaken in order to record the physiotherapy regime that each participant was receiving (Chapter Seven).

The target number of participants, which was achieved, was 10. From the target number of 10, two children did not attend the one-year testing and one child did not attend the six month testing. Their general characteristics are presented in Table 9.1. Initially 15 children in total consented to participate in the study but five were excluded. Subject two was excluded due to learning difficulties that became apparent during the first assessment. Subject nine was excluded because he was treated with botulinum toxin injections according to his clinical needs. Subjects six, 10 and 11 did not attend the six-month testing and were, therefore, excluded.

8.1.1 Strength changes

Isometric muscle strength of hip flexors, hip extensors, hip abductors, knee flexors, and knee extensors at 90° and 30° of flexion was measured bilaterally using the protocol described in the pilot study (Chapter Five). Following processing of data, paired samples t tests were used to compare strength values at different time points.

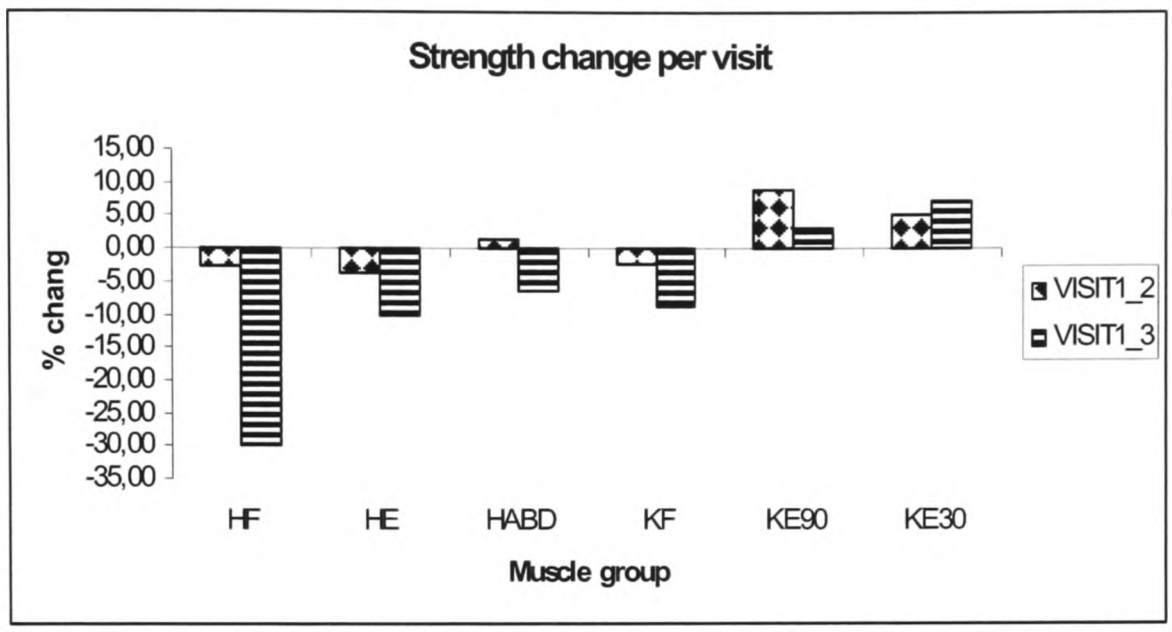
Table 8.2 shows the mean, SD, mean difference and significance of difference of muscle strength for all muscle groups as these were tested at baseline, six months and at one year following recruitment.

Muscle group Group A	**Baseline (1) Mean (SD) (Nm/kg)**	**6 month (2) Mean (SD) (Nm/kg**	**1 year (3) Mean (SD) (Nm/kg)**	**Mean difference (1-2) (SD)(Nm/kg)**	**Mean difference (1-3) (SD) (Nm/kg)**	**p1-2**	**p1-3**
Hip Flexors	0.5 (0.2)	0.4(0.1)	0.6 (0.2)	0.02(0.3)	- 0.1 (0.2)	0.710	**0.037**
Hip extensors	2.6 (0.9)	2.6(0.7)	2.8 (0.8)	0.05(1.0)	- 0.1 (0.8)	0.822	0.515
Hip abductors	0.9 (0.2)	0.8(0.1)	0.9 (0.2)	0.01(0.2)	- 0.1 (0.1)	0.846	0.231
Knee Flexors	0.9 (0.2)	0.9(0.1)	1.0 (0.2)	-0.02(0.3)	- 0.1 (0.2)	0.777	0.062
Knee extensors 90°	1.6 (0.5)	1.4(0.3)	1.6(0.4)	0.14(0.4)	- 0.1 (0.3)	0.180	0.303
Knee extensors 30°	0.6 (0.2)	0.6(0.2)	0.6 (0.2)	0.03(0.2)	0.1 (0.2)	0.571	0.266

Table 8.2 Normalised muscle strength (Nm/kg) per muscle group at baseline, six months and one year for group A

The non-surgical subjects did not deteriorate significantly when their muscle strength was tested during the one year follow up period. Although the hypothesis that children with spastic diplegia get weaker as they get older cannot be supported by these results, the fact that no increases in strength were noted as expected in healthy children may indicate that the follow-up period was not long enough to show a decrease in strength in this

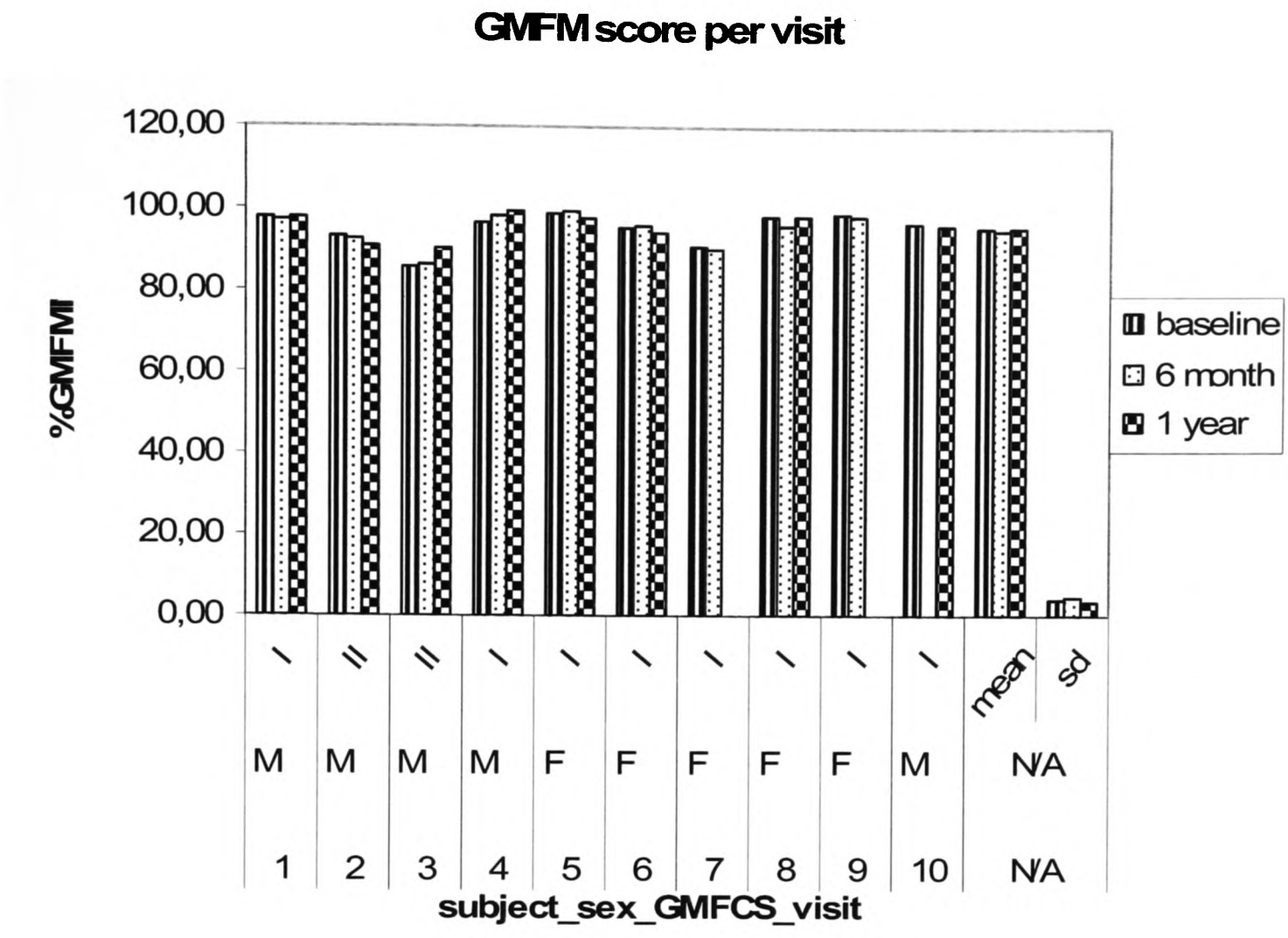
group. In Graph 8.1 the change of muscle strength between visits is shown as percentage. Although statistically non-significant, a trend of deterioration over time is evident for the knee extensors at 90 ° and 30°.



Graph 8.1 % change of muscle strength in group A between visits. Minus shows improvement; (visit 1= baseline, visit 2 = 6 month, visit 3 = 1 year, HF=hip flexors, HE=hip extensors, HABD=hip abductors, KF=knee flexors, KE90=knee exten 90°, KE30=knee exten 30 °)

8.1.2 Gross motor function changes

Motor function was assessed barefoot using the Gross Motor Function Measure (GMFM 88). Graph 8.2 shows the changes of function during the year of the study. Statistical comparisons did not reveal significant changes of the gross motor function after one year of follow up ($p < 0.762$). However, graph 8.2 shows that 8 out of 10 subjects had a tendency towards deterioration as they grew older. Moreover, it should be emphasised that the study was not powered to find a difference in GMFM.



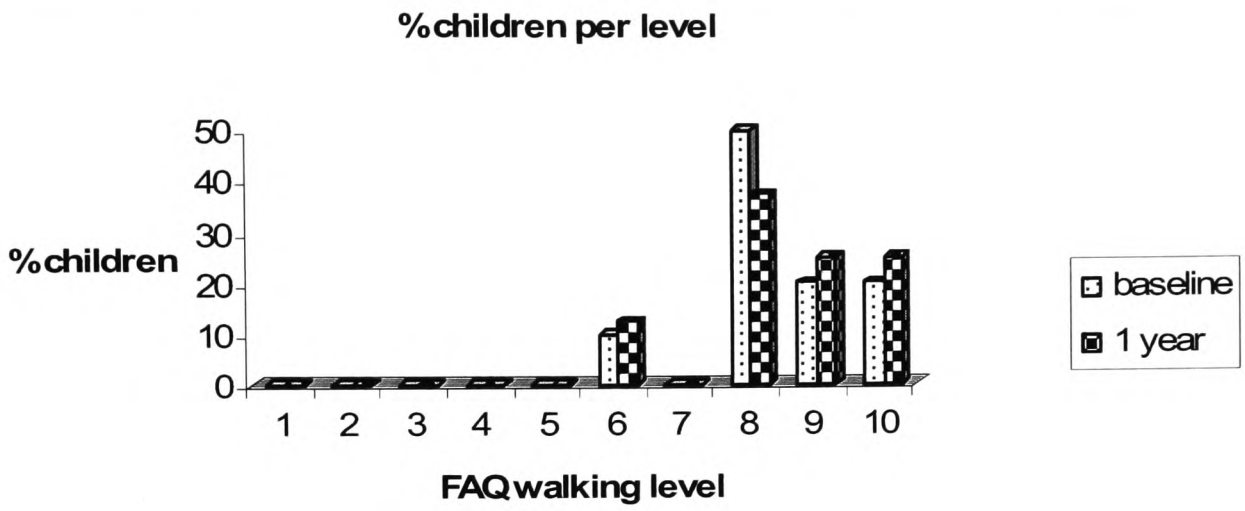
Graph 8.2 % GMFM score per subject per visit in group A, group mean and sd (GMFCS I – II, M = Male, F= Female, N/A= not applicable)

FAQ results

Analysis of the walking scale scores of the FAQ is presented in the Graph 8.3. As a reminder not taking any steps and not walking on a routine basis are described by levels one and two. Exercise ambulation levels are three-four. Level five is consistent with household or classroom walking and level six is limited community walking. Routine community walking on level surfaces is considered a level seven. Levels eight and nine are more advanced levels of walking on a variety of terrains. Level 10 is consistent with a typical, non compromised level of walking ability.

Plotted and raw data from Graph 8.3 and Table 8.3 showed non-consistent changes over one year in parents' perception about their child's walking

ability in the community. For some children walking ability remained unchanged while in others it deteriorated slightly or improved marginally.



Graph 8.3 % of subjects in group A per FAQ level atbaseline and 1 year

Subject	FAQ baseline	FAQ 6 months	FAQ 1 year
1	10	10	10
2	10	9	10
3	6	7	6
4	8	5	9
5	9	9	8
6	9	9	8
7	8	8	,
8	8	8	9
9	8	8	,
10	8	,	8

Table 8.3 Individual change of FAQ walking level per subject

8.1.3 Kinematic and kinetic changes

Three-dimensional gait analysis was performed using a Vicon 612 motion analysis system with 15 retro-reflective surface markers on the pelvis and lower limbs. Each child walked along a 10 meter walkway at their self-selected walking speed and a minimum of four walking trials was collected. Following processing of the data the average of three trials per child was used for further analysis. All children were recorded walking barefoot.

Kinematic variables	**Baseline mean (SD)**	**6 month mean (SD)**	**1 year mean (SD)**	**Paired mean differences (SD) baseline / 1 y**	**p baseline / 1 y**
Range of pelvic tilt (°)	7.4 (3.4)	7.3 (2.5)	6.7 (2.2)	0.6 (1.8)	0.220
Range of hip flexion (°)	48.5 (5.5)	46.3 (5.4)	46.2 (5.2)	1.5 (2.6)	**0.031**
Range of knee flexion (°)	50.8 (10.5)	48.7 (9.8)	46.5 (9.5)	5.1 (4.9)	**0.001**
Range of ankle dor/fl (°)	28.6 (5.8)	30.2 (7.5)	34.0 (10.2)	-4.7 (7.4)	**0.022**
Max hip extension stance (°)	- 0.5 (8.9)	0.5 (6.1)	0.8 (5.9)	-3.7 (3.5)	**0.001**
Max knee extension stance (°)	6.8 (9.7)	9.2 (8.4)	10.8 (10.3)	-4.4 (4.8)	**0.002**
Max knee flexion swing (°)	57.5 (6.7)	57.8 (6.7)	57.3 (6.5)	0.7 (5.1)	0.588
Knee flexion initial contact (°)	23.3 (6.5)	22.9 (7.8)	26.0 (6.7)	-2.9 (4.8)	**0.027**
Range of pelvic obliquity (°)	12.5 (3.9)	11.0 (4.4)	11.8 (4.5)	0.1 (3.0)	0.864
Range of pelvic rotation (°)	17.9 (4.7)	17.7 (4.3)	18.3 (5.3)	-1.2 (4.5)	0.308
Max thigh rotation (°)	17.2 (9.4)	17.6 (9.8)	20.9 (7.2)	-1.7 (5.9)	0.276

Table 8.4 Kinematic variables, mean (SD) per visit, paired difference between baseline and one year, significance of difference in group A

Interestingly, Tables 8.4 and 8.5 demonstrate significant deterioration in the sagittal kinematics during one year with similar changes in the kinetic variables.

Kinetic variables	**Baseline mean (SD)**	**6 month mean (SD)**	**1 year mean (SD)**	**Paired mean differences (SD) baseline / 1 y**	**p baseline / 1 y**
Hip extension moment	1499.8 (403.1)	1408.4 (288.3)	1333.9 (382.4)	68.3 (297.0)	0.388
Hip flexion moment	-704.2 (212.7)	-711.6 (122.6)	-762.2 (151.2)	31.6 (127.8)	0.355
Knee extension moment	453.6 (211.3)	460.4 (193.6)	466.3 (280.6)	-58.2 (151.9)	0.160
Knee flexion moment	-556.7 (302.1)	-484.1 (166.9)	-399.9 (108.0)	-96.2 (118.1)	**0.007**
Max plantarflexion moment	1139.5 (150.7)	1091.4 (142.1)	1231.6 (199.6)	-103.1 (189.5)	0.054
Max hip abduction moment	672.6 (197.1)	667.8 (179.8)	566.7 (130.4)	100.7 (140)	**0.015**
Max hip power	2.2 (1.0)	1.8 (0.7)	1.6 (0.4)	0.3 (0.6)	**0.030**
Max ankle power	2.3 (0.5)	2.4 (0.7)	2.2 (0.5)	-0.0 (0.5)	0.748

Table 8.5 Kinetic variables, mean (SD) per visit, paired difference between baseline and one year, significance of difference in group A

The range of hip and knee flexion decreased, the maximum hip and knee extension in stance decreased, and knee flexion at initial contact was significantly increased at one year. Similarly, a significant decrease was noted in the knee flexion and hip abduction moments.

Temporal parameters followed the same pattern as the gross motor function did. Although no significance decrease was observed, a trend towards deterioration of walking speed, cadence and step length was evident (Table 8.6).

Temporal parameters	**Baseline mean (SD)**	**6 month mean (SD)**	**1 year mean (SD)**	**Paired mean differences (SD) baseline / 1 y**	**p baseline / 1 y**
Normalized barefoot velocity	**0.44(0.1)**	0.42(0.0)	**0.42(0.1)**	0.01(0.1)	0.594
Normalized barefoot cadence	**0.64(0.1)**	0.63(0.0)	**0.62(0.1)**	0.02(0.0)	0.378
Normalized Right barefoot step	**0.69(0.1)**	0.68(0.0)	**0.67(0.1)**	0.01(0.1)	0.786
Normalized Left barefoot step	**0.69(0.1)**	0.66(0.0)	**0.65(0.1)**	0.01(0.1)	0.711

Table 8.6 Temporal parameters, mean (SD) per visit, paired difference between baseline and one year, significance of difference in group A

8.2 Changes in strength, function and gait in the surgical groups over one year

As discussed in Section One, the question of what determines the outcome of multi-level surgery has not yet been clarified. The effect of pre-existing muscle weakness on post-operative performance as well as the effect of surgery itself on muscle strength is still debatable. Conflicting evidence exists about the time needed for recovery of strength and function after surgery.

A few studies have examined the immediate effect of surgery on muscle strength. It has been shown that lengthening of the muscle/tendon unit may

lead to an immediate decrease in the effective muscle force (Moseley 1992). Clinical studies have investigated more long-term effects of surgery on strength and function. Their results suggest that anything between six to 24 months may be required for rehabilitation to be completed assuming that no further deterioration of mobility or recurrence of deformity takes place (Damiano 1999). However, animal studies examining the long-term effect of muscle surgery on muscle force suggest a much shorter recovery period (Brunner 2000).

This part of the analysis aimed to provide some insight to strength, function and gait changes following multi-level surgery at a post-operative stage when clinicians, parents and patients expect to see some of the beneficial effects of the operation and rehabilitation.

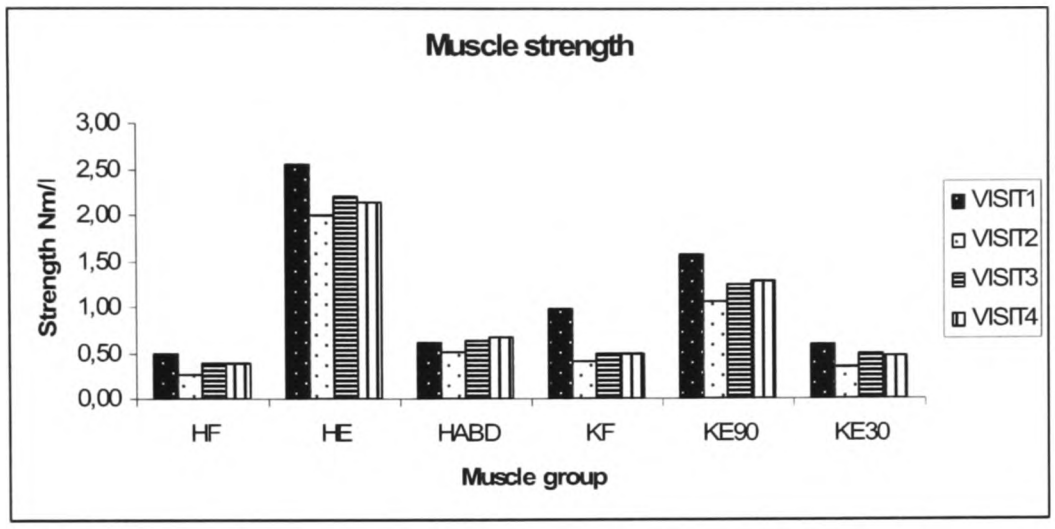
8.2.1 Overall changes

Muscle strength results in all the surgical patients demonstrated a significant decrease of strength six months after surgery. There was noticeable improvement following the 6-week physiotherapy strengthening programme in all patients.

Muscle group Nm/kg	**Pre – op mean (SD)**	**6 m I Pre-physio mean (SD)**	**6 m II Post-physio mean (SD)**	**1 year mean (SD)**	**p_i pre-op/6 m I**	**p_{ii} 6mI/6mII**	**p_{iii} pre-op/1 y**
Hip flexors	0.5(0.2)	0.3(0.1)	0.4(0.2)	0.4(0.2)	**0.000**	**0.000**	**0.008**
Hip extensors	2.5(0.9)	2.0(0.8)	2.2(0.7)	2.2(0.7)	**0.000**	**0.021**	**0.002**
Hip abductors	0.6(0.2)	0.5(0.2)	0.6(0.3)	0.7(0.3)	**0.002**	**0.000**	0.296
Knee flexors	1.0(0.3)	0.4(0.2)	0.5(0.2)	0.5(0.2)	**0.000**	**0.000**	**0.000**
Knee ext at 90°	1.6(0.7)	1.1(0.4)	1.3(0.5)	1.3(0.5)	**0.000**	**0.000**	**0.001**
Knee ext at 30°	0.6(0.4)	0.3(0.2)	0.5(0.2)	0.5(0.2)	**0.008**	**0.000**	0.077

Table 8.7 Muscle strength changes (mean,sd) at pre-surgery, 6 m post-surgery/pre-physio (6mI), 6 m post-surgery.post-physio (6mII) and 1 year with significance of difference for all surgical subjects (groups B&C)

Interestingly, it was noted that the strength achieved through physiotherapy was preserved at one year after surgery. However, with the exception of hip abductors, it did not reach pre-operative values (Table 8.7, Graph 8.4).



Graph 8.4 Muscle strength comparison Nm/kg between visits in all surgical patients (visit 1=pre-op, visit 2=6 m pre-physio, visit 3=6 m post-physio, visit4=1 year)

This is also shown as a percentage change in Table 8.8. It is interesting to notice that a significant improvement was achieved after the physiotherapy programme, mainly in the hip flexors (57.23%) and the knee extensors at 30° (43.28%), when strength was compared to the six-month post-operative values. This was not the case however, when post-physiotherapy results were compared with the pre-operative strength values confirming (a) the positive effect of intensive physiotherapy on strength after surgery and (b) the significant deterioration of strength after surgery. At six months post-operatively, the loss of strength was 45% in the hip flexors and 58% in the knee flexors muscle groups. That loss of strength was significant especially for the knee flexors since even after intensive physiotherapy, it persisted (49% at six months post-physio and 48.65% at one year). On the contrary, hip flexors showed great improvement following the programme, which they maintained at one year.

Muscle group	% change pre-op vs 6 m post-op	% change pre-op vs post-physio	% change pre-physio vs post-physio	% change pre-op vs 1 y post-op
Hip Flexors	**- 45.08**	-13.04	**57.23**	-11.02
Hip extensors	- 21.87	-9.96	10.18	-9.04
Hip abductors	- 17.34	-0.71	26.49	**13.23**
Knee flexors	**- 57.63**	**-49.00**	22.23	**-48.65**
Knee extensors 90°	- 32.51	-17.79	16.16	-14.39
Knee extensors 30°	- 18.7	0.34	**43.28**	**0.91**

Table 8.8 % change of muscle strength Nm/kg in all surgical patients between different assessment sessions (minus denotes decrease)

Kinematic and kinetic results in all surgical patients showed significant improvement in basic parameters e.g. knee flexion at initial contact, maximum internal thigh rotation, maximum knee extension in stance at six months after surgery. There were however, only a few changes following the strengthening programme. Although patients maintained the overall benefit of the operation one year after surgery when compared with the pre-op values, when gait parameters at one year were compared with those obtained from the six month assessments there was some deterioration (Table 8.9).

Kinematic Parameters	Pre – op mean (SD)	6 m I Pre-physio mean (SD)	6 m II Post-physio mean (SD)	1 year mean (SD)	p_i pre-op/6 m I	p_{ii} 6mI/6mII	p_{iii} pre-op/1 y
RPT (°)	10.2(3.0)	8.1(2.7)	7.9(3.3)	7.7(3.4)	**0.000**	0.714	**0.000**
RHF (°)	42.4(7.5)	43.3(9.1)	44.6(8.2)	41.9(9.0)	0.523	0.669	0.715
RKF (°)	31.9(12.9)	49.0(16.1)	**47.8(11.7)**	**43.5(14.1)**	**0.000**	**0.004**	**0.000**
RAD (°)	31.0(13.9)	11.8(7.5)	12.6(8.1)	21.1(9.1)	**0.000**	0.268	**0.000**
MHES (°)	9.0(11.9)	6.8(13.8)	6.7(12.5)	8.9(13.3)	0.103	0.718	0.921
MKES (°)	32.9(24.7)	9.3(17.4)	**10.5(15.5)**	**16.2(16.7)**	**0.000**	**0.009**	**0.000**
MKFS (°)	64.6(16.4)	58.2(6.8)	58.2(9.3)	59.6(8.0)	**0.012**	0.672	**0.036**
KFIC (°)	44.1(19.7)	21.1(14.1)	**21.1(13.7)**	**28.4(11.6)**	**0.000**	0.362	**0.000**
RPO (°)	8.9(4.2)	7.5(2.7)	7.8(3.5)	7.8(3.1)	0.063	0.609	0.175
RPR (°)	18.5(6.8)	16.9(5.1)	16.7(5.3)	16.1(5.8)	0.256	0.762	0.119
MTR (°)	22.7(12.6)	4.8(17.6)	**8.8(10.1)**	**11.4(9.4)**	**0.000**	0.190	**0.000**

Table 8.9 Kinematic changes (mean,sd) at pre-surgery, 6 m post-surgery/pre-physio (6mI), 6 m post-surgery.post-physio (6mII) and 1 year with significance of difference for all surgical subjects (groups B&C), RPT = range pelvic tilt, RHF = range hip flexion, RKF = range knee flexion, RAD = range ankle dorsiflexion, MHES = max hip extension stance, MKES = max knee extension stance, MKFS = max knee flexion swing, KFIC = knee flexion initial contact, RPO = range pelvic obliquity, RPR = range pelvic rotation, MTR = max thigh rotation

Kinetic variables showed the same trend as the kinematic ones but these changes were smaller (Table 8.10).

Gross motor function in all surgical patients followed the strength trend. At six months following surgery there was a significant decrease of all functional variables, while improvements were seen following physiotherapy. However, at one year after surgery function had not reached the pre-op values, being significantly lower at one year when assessed with the dimension E of the GMFM scale.

FAQ results

Parents' perception of their child's walking ability in the community followed quite closely the functional changes that were observed at the different stages of the study. At six months after surgery a significant

number of children dropped FAQ level reflecting the deterioration seen when they were assessed with the GMFM scale. Some improvement was noted following the intensive six week strengthening programme and at one year the majority of children had improved further their community ambulation.

However, a small number of children continued to perform worse than pre-operatively (Graph 8.5, Table 8.12).

Kinetic Parameters	**Pre – op mean (SD)**	**6 m I Pre-physio mean (SD)**	**6 m II Post-physio Mean (SD)**	**1 year mean (SD)**	**p_i pre-op/6 m I**	**p_{ii} 6mI/6mII**	**p_{iii} pre-op/1 y**
HEM	1420.7(335.9)	1280.0(497.4)	1255.8(394.7)	1303.1(375.8)	0.684	0.946	0.408
HFM	-665.8(226.6)	-584.0(135.9)	-536.8(142.8)	-548.6(168.0)	**0.019**	0.996	**0.034**
KEM	588.9(346.1)	360.4(147.9)	456.4(222.6)	524.6(323.1)	**0.012**	**0.005**	0.507
KFM	-482.6(280.9)	-512.9(170.0)	-438.1(138.0)	-447.9(141.5)	0.833	**0.001**	0.168
MPM	1209.0(318.7)	1100.7(182.6)	1115.6(152.0)	1135.0(186.4)	0.224	0.839	0.141
MHAM	624.6(216.7)	370.4(110.9)	373.7(152.4)	409.1(168.3)	**0.000**	0.204	**0.000**
MHP	1.6(0.6)	1.3(0.7)	1.4(0.5)	1.3(0.4)	0.252	0.237	**0.031**
MAP	1.8(0.7)	1.0(0.6)	1.2(0.9)	1.8(0.7)	**0.004**	0.062	0.691

Table 8.10 Kinetic changes (mean,sd) at pre-surgery, 6 m post-surgery/pre-physio (6mI), 6 m post-surgery.post-physio (6mII) and 1 year with significance of difference for all surgical subjects (groups B&C), HEM = hip extension moment, HFM = hip flexion moment, KEM = knee extension moment, KFM = knee flexion moment, MPM = max plantarflexion moment, MHAM = max hip abduction moment, MHP = max hip power, MAP = max ankle power

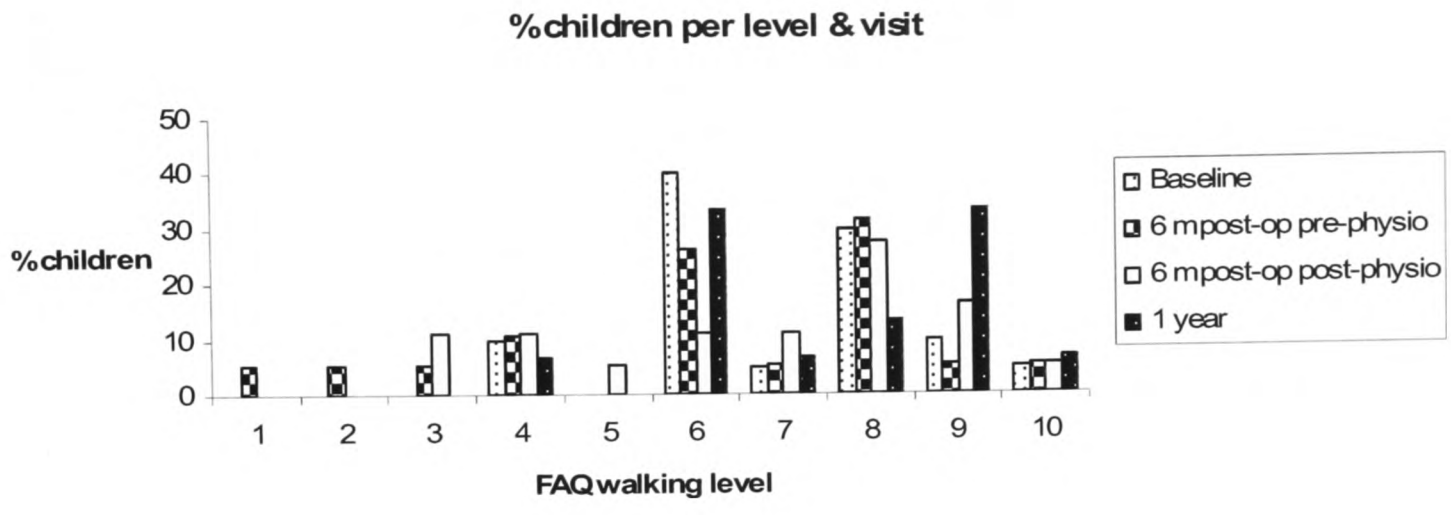
Gross motor function Parameters	Pre – op mean (SD)	6 m I Pre-physio mean (SD)	6 m II Post-physio mean (SD)	1 year mean (SD)	p_i pre-op/6 m I	p_{ii} 6mI/6mII	p_{iii} pre-op/1 y
GMFM total %	**84.9(12.6)**	77.2(14.3)	83.9(10.7)	**82.5(14.0)**	**0.003**	**0.000**	0.191
GMFM E %	**66.3(24.1)**	44.8(29.2)	57.7(26.7)	**56.8(29.3)**	**0.000**	**0.000**	**0.035**
N velocity	**0.31(0.1)**	0.27(0.1)	0.30(0.1)	**0.29(0.1)**	**0.002**	**0.005**	0.233
N cadence	**0.53(0.1)**	0.44(0.1)	0.46(0.1)	**0.51(0.1)**	**0.000**	**0.018**	0.161
N step length R	**0.58(0.1)**	0.60(0.2)	0.66(0.1)	**0.57(0.1)**	0.754	0.085	0.570
N step length L	**0.58(0.1)**	0.59(0.2)	0.65(0.2)	**0.56(0.1)**	0.857	0.007	0.460

Table 8.11 Gross motor function and temporal parameters changes (mean,sd) at pre-surgery, 6 m post-surgery/pre-physio (6mI), 6 m post-surgery.post-physio (6mII) and 1 year with significance of difference for all surgical subjects

Table 8.12 Individual change of FAQ level per surgical subject per visit

Subject	FAQ baseline	FAQ 6 m pre-physio	FAQ 6 m post-physio	FAQ 1 year
1	6	6	8	9
2	9	9	9	9
3	8	8	8	9
4	10	10	10	10
5	**7**	**6**	**5**	**6**
6	6	7	7	9
7	6	6	9	,
8	4	4	4	6
9	8	6	6	8
10	**8**	**6**	**,**	**6**
11	6	8	7	7
12	8	8	8	8
13	**6**	**4**	**4**	**4**
14	6	3	3	,
15	4	1	3	6
16	6	2	6	6
17	9	8	8	,
18	8	8	9	9
19	6	,	,	,
20	8	8	8	,

Graph 8.5 % children in sample per FAQ walking level and visit



8.2.2 Comparison between surgical groups

One of the aims of the study was to compare two different strengthening regimes post-operatively. Therefore the surgical groups B & C were analysed separately and compared.

When muscle strength was examined it was demonstrated that the hip flexors continued to improve between six months and one year for group B but group C retained the already significant gain from the six-month resistance strength training. The hip extensors and knee extensors at 30° remained the same in group B after the programme, but in group C the extra gain from resistance training was lost at one year. Indeed ANCOVA showed that the knee extensors at 30 ° strength was increased significantly more between six months post surgery and one year in group B than group C.

Muscle group Nm/kg Group B	**Pre – op Mean (SD)**	**6 m I Pre-physio mean (SD)**	**6 m II Post-physio mean (SD)**	**1 year mean (SD)**	**p_i pre-op/6 m I**	**p_{ii} 6mI/6mII**	**p_{iii} pre-op/1 y**
Hip flexors	0.4(0.2)	0.3(0.1)	0.3(0.2)	0.4(0.2)	**0.001 (-)**	0.122 (+)	0.403 **(-)**
Hip extensors	2.2(0.9)	1.8(0.7)	2.1(0.8)	2.1(0.8)	**0.014 (-)**	0.157 (+)	0.340 **(-)**
Hip abductors	0.6(0.2)	0.5(0.2)	0.6(0.2)	0.7(0.3)	0.259 **(-)**	**0.036** (+)	0.369 (+)
Knee flexors	1.0(0.3)	0.4(0.2)	0.5(0.3)	0.5(0.3)	**0.000 (-)**	**0.004** (+)	**0.000 (-)**
Knee ext at 90°	1.5(0.7)	1.0(0.4)	1.2(0.5)	1.2(0.5)	**0.001 (-)**	**0.008** (+)	**0.029 (-)**
Knee ext at 30°	0.5(0.4)	0.3(0.1)	0.5(0.1)	0.5(0.2)	0.637 **(-)**	**0.021** (+)	0.941 **(-)**

Table 8.13 Muscle strength changes (mean,sd) at pre-surgery, 6 m post-surgery/pre-physio (6mI), 6 m post-surgery.post-physio (6mII) and 1 year with significance of difference for group B (- denotes negative change or decrease, + denotes positive change or increase)

The hip abductors continued to improve in both groups between six months and one year. The knee flexors retained the strength gained at six months in both groups. The knee extensors at 90 ° remained the same in group B at one year, but continued to improve in group C.

Muscle group Nm/kg Group C	**Pre – op Mean (SD)**	**6 m I Pre-physio mean (SD)**	**6 m II Post-physio mean (SD)**	**1 year mean (SD)**	**p_i pre-op/6 m I**	**p_{ii} 6mI/6mII**	**p_{iii} pre-op/1 y**
Hip flexors	0.5(0.3)	0.3(0.1)	0.4(0.2)	0.4(0.2)	**0.000 (-)**	**0.000 (+)**	**0.006 (-)**
Hip extensors	2.8(0.9)	2.1(0.8)	2.3(0.6)	2.2(0.6)	**0.001 (-)**	0.075 (+)	**0.002 (-)**
Hip abductors	0.6(0.2)	0.5(0.3)	0.6(0.3)	0.7(0.3)	**0.002 (-)**	**0.000 (+)**	0.568
Knee flexors	1.0(0.3)	0.4(0.2)	0.5(0.2)	0.5(0.2)	**0.000 (-)**	**0.001 (+)**	**0.000 (-)**
Knee ext at 90°	1.7(0.6)	1.1(0.5)	1.3(0.5)	1.4(0.6)	**0.000 (-)**	**0.000 (+)**	**0.014 (-)**
Knee ext at 30°	0.7(0.4)	0.4(0.3)	0.5(0.3)	0.4(0.3)	**0.006 (-)**	**0.000 (+)**	**0.024 (-)**

Table 8.14 Muscle strength changes (mean,sd) at pre-surgery, 6 m post-surgery/pre-physio (6mI), 6 m post-surgery.post-physio (6mII) and 1 year with significance of difference for group C (- denotes negative change or decrease, + denotes positive change or increase)

In both groups, the majority of muscle groups did not reach the pre-op strength level at one year from entering the study. It should be noted that although group C was overall stronger at baseline than group B, this was taken into account when the strengthening regimes were compared as presented in Chapter Seven.

Dependent Variable	**Parameter estimates – Significance of effect**
	Group B vs C
Hip flexors Nm/kg	0.84 ↑
Hip extensors Nm/kg	0.54 ↑
Hip abductors Nm/kg	0.9 ↑
Knee flexors Nm/kg	0.17 ↑
Knee extensors at 90° Nm/kg	0.98 ↑
Knee extensors at 30° Nm/kg	**0.04** ↑

Table 8.15 Group B & C analysis of co-variance (ANCOVA) at 1 year. The parameter estimates show the effect of the predictor (group) on the dependent variable (i.e.hip flexors Nm/kg e.t.c.). ↓ indicates that given two people with similar pre – physio values, it can be expected that the post – physio value of those in group B to be less than of those in group C. The opposite is valid for ↑.

The GMFM scores, total and dimension E, as well as the temporal parameters followed a similar trend to muscle strength changes. Following the six-week strengthening programme, group C showed a trend towards greater improvement than group B. However, at one year group B had approached the pre-operative values more than group C. This could be explained by comparing the variables obtained from the six-month assessment. These show that, probably by chance, group B was affected less from the operation than group C. This may explain why in the longer term group B showed a slightly better progress than group C.

Gross motor function Parameters Group B	**Pre – op mean (SD)**	**6 m I Pre-physio mean (SD)**	**6 m II Post-physio mean (SD)**	**1 year Mean (SD)**	**p_i pre-op/6 m I**	**p_{ii} 6mI/6mII**	**p_{iii} pre-op/1 y**
GMFM total %	82.2(13.5)	75.2(15.9)	83.9(10.4)	81.5(16.9)	0.145	**0.035**	0.837
GMFM E %	63.6(26.9)	44.1(31.3)	60.8(26.5)	59.4(35.5)	**0.035**	**0.014**	0.612
N velocity	0.32(0.1)	0.29(0.1)	0.32(0.1)	0.32(0.1)	0.071	0.346	0.945
N cadence	0.55(0.1)	0.48(0.1)	0.49(0.1)	0.56(0.1)	**0.003**	0.465	0.769
N step length R	0.60(0.1)	0.61(0.1)	0.63(0.2)	0.55(0.1)	0.688	0.663	0.207
N step length L	0.55(0.1)	0.63(0.1)	0.66(0.2)	0.58(0.1)	0.378	0.442	0.587

Table 8.16 Function changes (mean,sd) at pre-surgery, 6 m post-surgery/pre-physio (6mI), 6 m post-surgery.post-physio (6mII) and 1 year with significance of difference for group B

Gross motor function Parameters Group C	**Pre – op Mean (SD)**	**6 m I Pre-physio mean (SD)**	**6 m II Post-physio mean (SD)**	**1 year mean (SD)**	**p_i pre-op/6 m I**	**p_{ii} 6mI/6mII**	**p_{iii} pre-op/1 y**
GMFM total %	87.0(11.9)	78.9(13.4)	83.9(11.4)	83.5(12.0)	**0.003**	**0.002**	0.109
GMFM E %	68.5(22.8)	45.3(28.8)	55.6(28.0)	54.5(24.5)	**0.000**	**0.004**	**0.018**
N velocity	0.31(0.1)	0.25(0.1)	0.30(0.1)	0.27(0.1)	**0.021**	**0.000**	**0.038**
N cadence	0.52(0.1)	0.41(0.1)	0.44(0.1)	0.46(0.1)	**0.002**	**0.001**	**0.013**
N step length R	0.57(0.1)	0.60(0.2)	0.67(0.1)	0.58(0.1)	0.351	0.069	0.628
N step length L	0.60(0.1)	0.57(0.2)	0.64(0.2)	0.53(0.2)	0.415	**0.001**	**0.053**

Table 8.17 Function changes (mean,sd) at pre-surgery, 6 m post-surgery/pre-physio (6mI), 6 m post-surgery.post-physio (6mII) and 1 year with significance of difference for group C

When the two groups were compared in order to examine differences in kinematic variables it was shown that both groups followed the same trend.

Kinematic Parameters Group B	**Pre – op mean (SD)**	**6 m I Pre-physio mean (SD)**	**6 m II Post-physio mean (SD)**	**1 year mean (SD)**	**p_i pre-op/6 m I**	**p_{ii} 6mI/6mII**	**p_{iii} pre-op/1 y**
RPT (°)	10.4(1.7)	7.9(2.4)	7.9(2.3)	7.7(3.0)	**0.003**	0.926	**0.004**
RHF (°)	41.4(4.4)	43.1(10.1)	45.2(8.5)	41.7(8.4)	0.489	0.979	0.907
RKF (°)	31.0(12.0)	46.4(16.9)	46.8(12.0)	42.6(12.5)	**0.003**	0.078	**0.003**
RAD (°)	26.9(12.5)	13.4(9.2)	13.6(8.4)	21.7(7.2)	**0.003**	0.733	0.205
MHES (°)	9.7(9.6)	8.8(13.0)	8.1(10.7)	9.9(12.3)	0.684	0.950	0.924
MKES (°)	33.8(24.2)	12.3(16.5)	13.1(12.8)	18.8(14.2)	**0.001**	0.094	**0.008**
MKFS (°)	64.7(17.5)	58.4(6.1)	59.9(9.3)	61.3(7.2)	0.104	0.746	0.349
KFIC (°)	44.7(21.3)	26.1(12.3)	27.1(12.5)	31.3(10.6)	**0.001**	0.181	**0.003**
RPO (°)	7.8(2.1)	6.6(2.1)	6.7(2.6)	6.5(1.9)	0.110	0.785	0.079
RPR (°)	22.1(7.2)	15.2(3.9)	16.1(4.8)	16.2(5.5)	**0.005**	0.446	**0.020**
MTR (°)	23.6(12.8)	6.4(8.0)	7.0(7.1)	11.1(8.2)	**0.000**	0.351	**0.000**

RPT = range pelvic tilt, RHF = range hip flexion, RKF = range knee flexion, RAD = range ankle dorsiflexion, MHES = max hip extension stance, MKES = max knee extension stance, MKFS = max knee flexion swing, KFIC = knee flexion initial contact, RPO = range pelvic obliquity, RPR = range pelvic rotation, MTR = max thigh rotation

Table 8.18 Kinematic changes (mean,sd) at pre-surgery, 6 m post-surgery/pre-physio (6mI), 6 m post-surgery.post-physio (6mII) and 1 year with significance of difference for group B

At six months following surgery a significant improvement was seen in both groups in several kinematic variables with no significant changes observed followed the six week strengthening programme.

Kinematic Parameters Group C	Pre – op mean (SD)	6 m I Pre-physio mean (SD)	6 m II Post-physio mean (SD)	1 year mean (SD)	p_i pre-op/6 m I	p_{ii} 6mI/6mII	p_{iii} pre-op/1 y
RPT (°)	10.0(3.8)	8.2(3.0)	8.0(3.9)	7.7(3.7)	**0.020**	0.619	**0.000**
RHF (°)	43.3(9.4)	43.5(8.4)	44.2(8.2)	42.2(9.6)	0.917	0.434	0.545
RKF (°)	32.7(13.7)	51.2(15.4)	48.5(11.8)	44.3(15.5)	**0.000**	**0.026**	**0.004**
RAD (°)	34.4(14.4)	10.5(5.6)	11.8(8.0)	20.6(10.5)	**0.000**	0.140	**0.000**
MHES (°)	8.4(13.7)	5.2(14.5)	5.7(13.9)	8.0(14.2)	0.061	0.600	0.812
MKES (°)	32.1(25.7)	6.9(18.2)	8.6(17.2)	14.0(18.6)	**0.000**	**0.033**	**0.005**
MKFS (°)	64.5(15.9)	58.0(7.5)	57.0(9.3)	58.3(8.4)	0.066	0.323	0.052
KFIC (°)	43.5(18.7)	17.0(14.5)	16.8(13.1)	26.0(12.1)	**0.000**	0.908	**0.000**
RPO (°)	9.8(5.2)	8.2(3.0)	8.7(3.9)	8.9(3.4)	0.213	0.389	0.514
RPR (°)	15.5(4.9)	18.2(5.6)	17.2(5.7)	16.1(6.1)	**0.031**	0.326	0.727
MTR (°)	21.9(12.6)	3.5(22.8)	10.1(11.8)	11.7(10.4)	**0.001**	0.261	**0.003**

RPT = range pelvic tilt, RHF = range hip flexion, RKF = range knee flexion, RAD = range ankle dorsiflexion, MHES = max hip extension stance, MKES = max knee extension stance, MKFS = max knee flexion swing, KFIC = knee flexion initial contact, RPO = range pelvic obliquity, RPR = range pelvic rotation, MTR = max thigh rotation

Table 8.19 Kinematic changes (mean,sd) at pre-surgery, 6 m post-surgery/pre-physio (6mI), 6 m post-surgery.post-physio (6mII) and 1 year with significance of difference for group C

At one year following surgery examination of the gait variables showed that the benefit of the operation was retained compared with pre-operative values but some deterioration since the six month assessment had occurred.

Similar but less obvious changes were seen in the kinetic variables (Table 8.20 & 8.21).

Kinetic Parameters Group B	Pre – op Mean (SD)	6 m I Pre-physio mean (SD)	6 m II Post-physio mean (SD)	1 year mean (SD)	p_i pre-op/6 m I	p_{ii} 6mI/6mII	p_{iii} pre-op/1 y
HEM	1537.1(255.8)	1571.2(451.3)	1500.1(336.4)	1545.6(358.0)	0.434	0.315	0.818
HFM	-669.7(165.0)	-538.5(94.7)	-505.6(130.0)	-543.4(183.3)	0.089	0.506	0.097
KEM	515.7(167.2)	388.5(115.7)	581.7(263.1)	604.2(415.5)	0.087	**0.004**	0.946
KFM	-453.9(135.7)	-476.4(163.3)	-415.1(132.6)	-443.0(109.5)	0.561	**0.047**	0.391
MPM	1161.6(371.8)	1057.2(232.5)	1123.3(191.8)	1056.0(171.4)	0.747	**0.022**	0.528
MHAM	623.0(280.6)	395.9(107.9)	417.2(169.1)	440.9(180.2)	0.098	0.580	0.081
MHP	1.7(0.4)	1.7(0.8)	1.8(0.5)	1.5(0.4)	0.758	0.438	0.104
MAP	1.5(0.7)	1.2(0.7)	1.3(0.8)	1.7(0.6)	0.810	0.168	0.377

HEM = hip extension moment, HFM = hip flexion moment, KEM = knee extension moment, KFM = knee flexion moment, MPM = max plantarflexion moment, MHAM = max hip abduction moment, MHP = max hip power, MAP = max ankle power

Table 8.20 Kinetic changes (mean,sd) at pre-surgery, 6 m post-surgery/pre-physio (6mI), 6 m post-surgery.post-physio (6mII) and 1 year with significance of difference for group B

Analysis of covariance (ANCOVA) did not reveal any differences between the two groups when kinematic and kinetic variables were compared after the six week strengthening programme and at one year (Appendix Five).

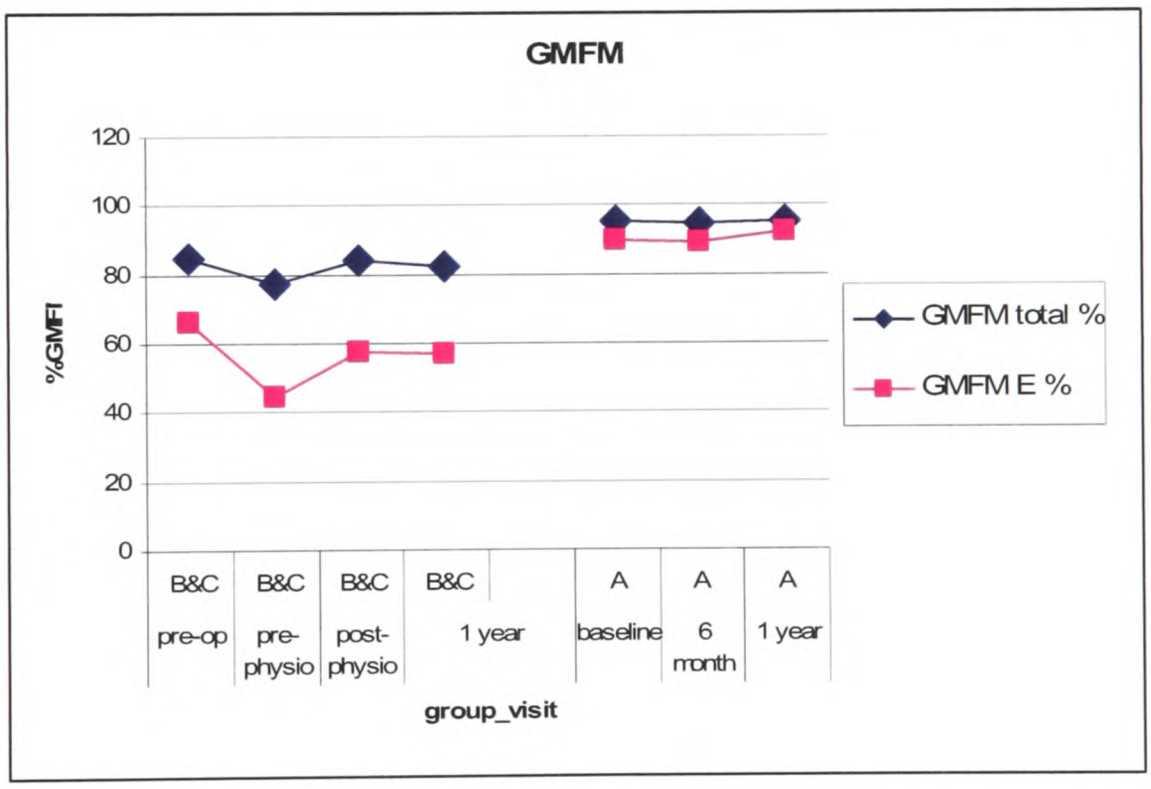
Kinetic Parameters Group C	Pre – op Mean (SD)	6 m I Pre-physio mean (SD)	6 m II Post-physio mean (SD)	1 year mean (SD)	p_i pre-op/6 m I	p_{ii} 6mI/6mII	p_{iii} pre-op/1 y
HEM	1337.6(369.4)	1056.0(419.3)	1092.9(351.0)	1095.2(249.6)	0.197	0.349	0.212
HFM	-663.0(268.3)	-555.4(164.4)	-557.6(151.5)	-553.0(160.7)	0.127	0.406	0.211
KEM	641.3(430.7)	338.8(169.9)	372.9(147.6)	456.3(209.2)	0.058	0.256	0.450
KFM	-503.0(354.7)	-541.0(176.0)	-453.4(145.5)	-452.1(168.3)	0.995	**0.008**	0.279
MPM	1242.9(284.7)	1134.1(133.4)	1110.4(126.0)	1202.7(176.8)	0.225	0.238	0.177
MHAM	625.8(169.1)	350.8(113.3)	344.7(138.6)	381.8(159.0)	**0.000**	0.209	**0.001**
MHP	1.6(0.8)	1.1(0.5)	1.1(0.4)	1.1(0.2)	0.115	0.355	0.075
MAP	2.0(0.7)	0.8(0.3)	1.0(0.9)	1.9(0.8)	**0.000**	0.133	0.635

HEM = hip extension moment, HFM = hip flexion moment, KEM = knee extension moment, KFM = knee flexion moment, MPM = max plantarflexion moment, MHAM = max hip abduction moment, MHP = max hip power, MAP = max ankle power

Table 8.21 Kinetic changes (mean,sd) at pre-surgery, 6 m post-surgery/pre-physio (6mI), 6 m post-surgery.post-physio (6mII) and 1 year with significance of difference for group C

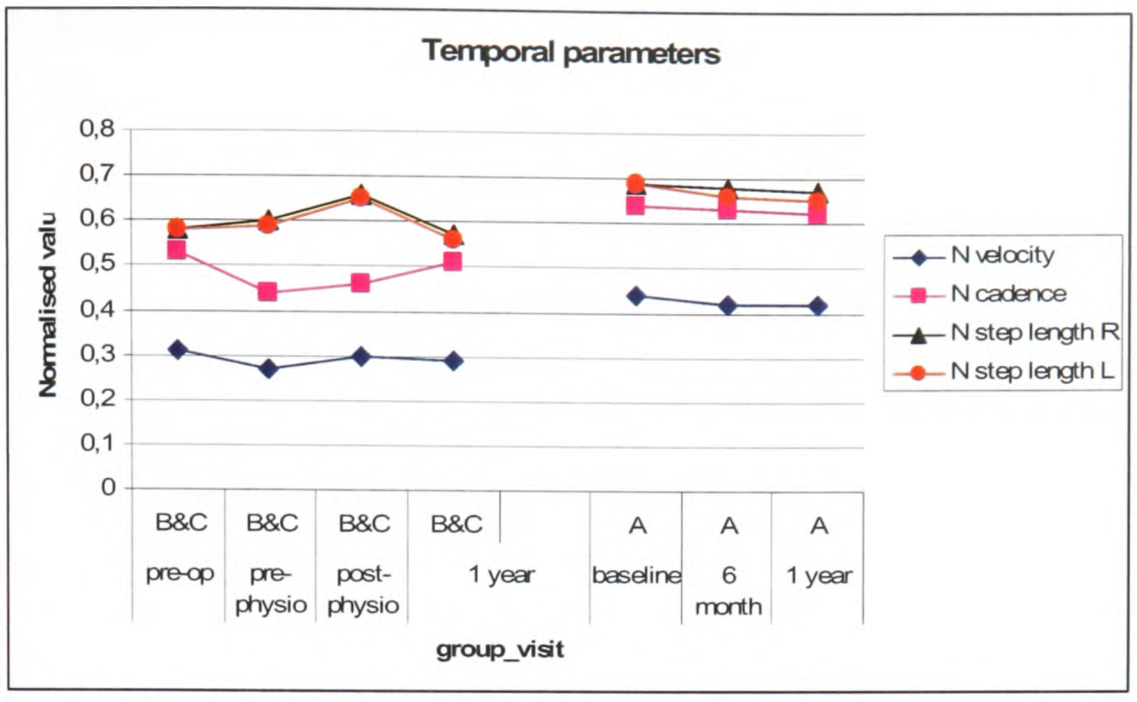
8.2.3 Surgical versus non-surgical patients

Patients who had surgery were compared with those who received only routine physiotherapy for the duration of the study. As it was mentioned previously, group A was not matched with groups B & C since it was self-selected and not randomised. Although, the use of statistical tests for this comparison would be inappropriate, it was thought that a graphical representation of the data from all three groups would provide a visual understanding of the effects of surgery, intensive physiotherapy and natural history on strength and function.



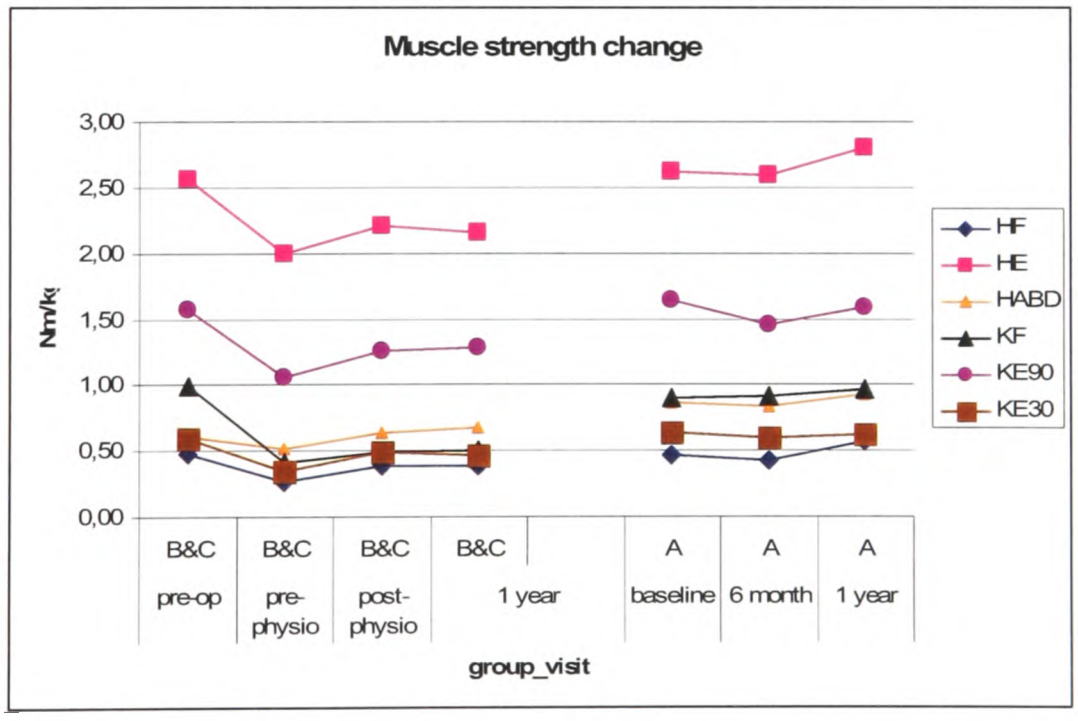
Graph 8.6 GMFM % score changes (total and dimension E) for all study groups and visits

Graph 8.6 shows that, probably due to a slightly younger average age or by chance, group A was more functional than the surgical groups. However, surgery and intensive physiotherapy had a significant impact on function in the surgical patients.



Graph 8.7 Temporal parameters change in all groups and visits (N = normalised)

Interestingly, temporal parameters showed deterioration over time in the conservatively treated group, confirming that even younger and more functional children with spastic diplegia can deteriorate within a year (Graph 8.7). This is supported by the changes in gait analysis parameters presented earlier in this Chapter (Tables 8.4 & 8.5).



Graph 8.8 Muscle strength change in all muscle and study groups for all visits

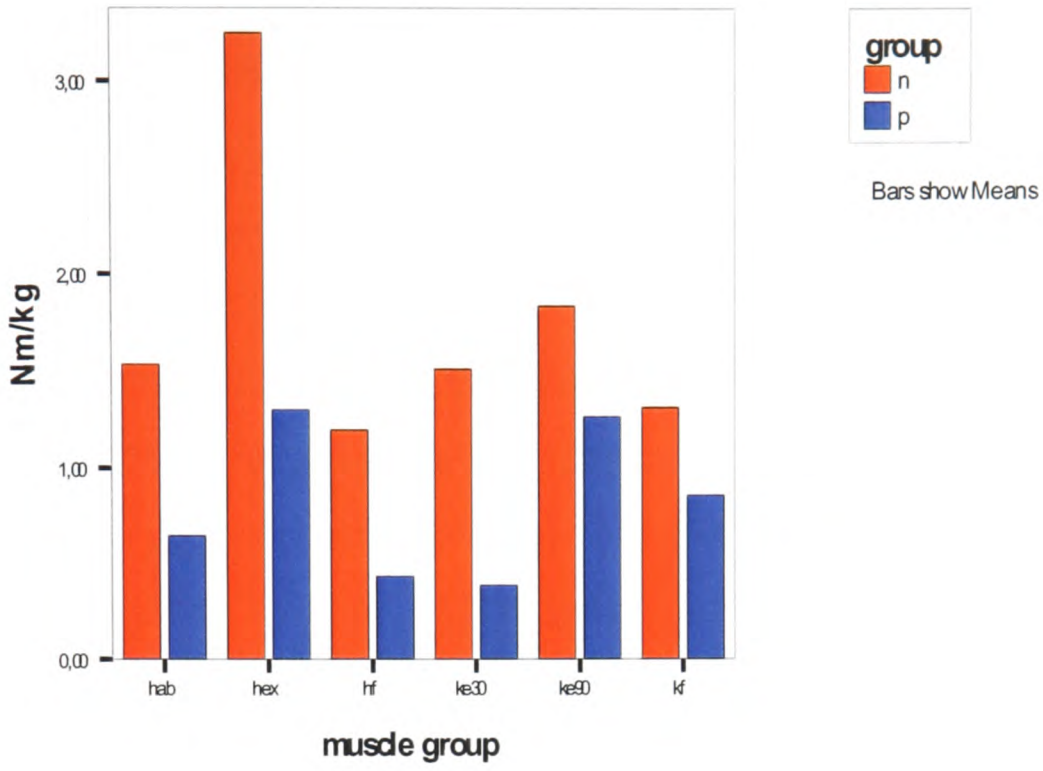
Graph 8.8 shows the change in muscle strength throughout the duration of the study for all muscle and study groups. The significant effect of surgery

and physiotherapy is confirmed, but the natural history of strength in the conservatively treated sample of patients did not show consistent deterioration over time.

8.3 Discussion

Although research suggests that children with CP are weaker than normal counterparts, few studies have demonstrated the distribution of weakness in these patients. Muscle weakness can be localised or generalised and it affects some muscles more than others, often showing greater involvement of the plantarflexors and dorsiflexors (Brown 1991; Wiley 1998; Engsberg 2000). Olney et al (1990) reported inadequate power production in the affected lower extremity of hemiplegic children, particularly in the ankle plantarflexor and knee extensor muscles. Furthermore, Bourbonnais (1989) and Wiley (1998) have provided some insight into the severity and distribution of weakness in spastic hemiparesis and children with CP, respectively. The results of the present study confirmed the findings of the literature and showed that spastic diplegic patients are weaker that their healthy counterparts when lower limb muscle strength is assessed (graph 9.4). However, there is still a need for developing strength profiles based on large age and type specific databases of patients with CP.

There is limited information regarding the natural history of muscle strength in children with spastic diplegia and no long-term studies exist with follow-up of these patients to adulthood. The different interventions performed at different stages throughout these patients' development render the design of such studies difficult. However, the findings of this study highlight the changes of strength and function over time in patients who do not receive any form of treatment other than community physiotherapy for a period of one year.



Graph 8.9 Strength (Nm/kg) per muscle group for 1 healthy (n) and 1 spastic diplegic (p) child, matched for age and sex.

It was shown that although no statistically significant decrease in gross motor function and temporal parameters was observed, a trend towards deterioration in function was evident. More importantly, there was a clear deterioration of the kinematic and kinetic variables of gait during the year of the study. This could have significant implications to the advice given to parents regarding surgery, since there is evidence that even at a younger age and higher functional level children with spastic diplegia might deteriorate faster than previously thought. The non-significant changes in muscle strength did not support the initial hypothesis that children would be weaker over time. Although this finding could be attributed to the small sample size, the limited follow-up period, the younger average age and the better functional level, an important observation could still be made. Muscle strength did not increase in this group, as it would be expected in growing healthy children.

Following the patients who underwent multi-level surgery during the one year of the study revealed some interesting findings. All surgical patients showed a significant decrease of muscle strength at six months following surgery. This decrease however, was probably by chance, greater in group C. This could have affected the group's long-term speed of recovery compared with group B. The six-week intensive strengthening programme improved muscle strength in all surgical patients. This change was greater for the group who received the progressive resistance strengthening programme. However, at one year muscle strength did not reach pre-operative values. Function, as described by the GMFM, and temporal parameters followed the same trend. These results suggest that although intensive physiotherapy following multi-level surgery can improve strength and function significantly, further research into the duration and type of post-op physiotherapy is needed. It may be that a longer strengthening programme is essential to improve the speed of recovery after multi-level surgery, which could have significant implications on the overall management of patients with spastic diplegia.

The changes in gait showed a significant improvement after surgery, as expected. However, there was no observed difference following the six-week physiotherapy programme. More importantly, gait parameters remained improved compared to the pre-operative values but some deterioration was observed between the six and 12-month assessments. Again, this deterioration could have been avoided if a longer or more intensive programme had been implemented.

8.4 Summary

The key points in this Chapter are summarised:

1. No statistically significant deterioration in strength or function was found in the conservatively treated group. There was however a trend towards deterioration over time, seen mainly in the temporal parameters.

2. A significant deterioration in sagittal kinematic parameters was found in the non-surgical group.

3. Surgical patients preserved the post-physiotherapy strength and function gains but these did not reach the pre-op values.

4. At one year, the surgical patients showed some deterioration in gait parameters but these were still significantly improved compared to pre-operative values.

Chapter Nine

9. Discussion and synopsis

9.1 Introduction

This dissertation was based on the recognition of muscle weakness as a significant component of cerebral palsy. Review of the literature strengthened the author's view about the limited information available regarding the effects of pre-existing weakness and multi-level orthopaedic surgery itself on the rehabilitation progress of patients with spastic diplegia. Evidence about the best post-operative physiotherapy management, which would consider the effects of weakness, was sought.

9.1.1 Aims and hypotheses of the study

The overall aim of this study was to provide an evidence-based approach to post-operative rehabilitation following multi-level orthopaedic surgery in children with spastic diplegic cerebral palsy.

The principal hypothesis was that multi-level orthopaedic surgery would lead to a decrease in muscle strength and gross motor function at six months post-surgery, and that those should recover to the pre-operative level approximately 12 months post-surgery. It could be argued that muscle or tendon surgery produces a temporary loss of strength. Animal studies suggest that this loss of strength is completely recovered in six weeks (Brunner 2000). However, clinical experience has shown the rehabilitation period following multi-level orthopaedic surgery to be prolonged with continued improvement for up to two years (Nene 1993). In support of this hypothesis, patients of this study who underwent multi-level surgery

showed improvements in gait analysis parameters but significant loss of strength in lower limb muscle groups and gross motor function at six months after surgery. When strength and function were assessed again one year after surgery, they were found close to but still lower than the pre-operative values.

The literature suggests that patients with CP can increase muscle strength at a rate similar to that observed in patients with weakness unrelated to central nervous system lesions (Petersen 1990). Furthermore, strengthening exercises result to a direct improvement of function and in contrast to traditional beliefs, they do not increase spasticity (MacPhail 1995; Fowler 2001). However, no evidence existed at the time of designing this study about the use of strengthening exercises following multi-level surgery. Therefore, it was hypothesised that the implementation of a weight - resistance strengthening programme of five lower-limb muscle groups six months after multi-level orthopaedic surgery would improve gross motor function, muscle strength and ambulation to a greater degree, compared with an active strengthening programme of the same duration, in two randomised groups of spastic diplegic patients. Overall results of this part of the study support this hypothesis showing advantages of resistance training over active exercise.

Finally, limited information was found on the natural history of weakness in CP as no trials are available with follow up of patients with CP to adulthood. In order to investigate the degree and distribution of muscle weakness and strength changes over time, it was hypothesised that muscle strength and gross motor function would deteriorate in a group of conservatively treated patients, over a period of 12 months. Overall results showed trends supporting this hypothesis, although there was not a statistically significant decrease of the measured strength variables. Interestingly, kinematic and kinetic variables measured one year after entering the study did show a significant deterioration.

9.1.2 Study design

In order to answer the questions of this study, the design included randomised and prospective cohort assessments. In particular, the randomisation was essential in order to compare in an objective way two strengthening regimes at six months post multi-level surgery. Apart from the surgical patients, there was an additional group of conservatively treated patients who were assessed for the duration of the study. This group did not participate in the randomisation process for ethical reasons, but was prospectively monitored.

In order to identify a sample size, which would allow differences in strength variables to be detected, the power calculations were based on published results of a strengthening programme, which examined a number of outcome measures at three different time points (Damiano 1998). If we assumed a difference of 60 Newton between conservative treatment (*n observations*) and interventions (*2n observations to be allocated to two arms of n observations each*) with a common within-group standard deviation of 45 Newton, then a proposed sample size of 10 and 20, would provide the study with power of 91% to yield a statistically significant result. Although, the assumed effects above may not be entirely accurate, the proposed sample sizes of 10, in each one of the three experimental groups, were thought to be sufficient to produce statistical significant results with high power.

Based on these calculations, three study groups of 10 subjects in each group were defined (groups A, B & C). Group A was treated conservatively. Groups B & C were treated with multi-level orthopaedic surgery. All groups were monitored for one year. Surgical candidates were randomly allocated into Group B or C, six months post-operatively, in order to compare the effects of two post-operative rehabilitation regimes. They were matched for age, sex and GMFCS level, based on clinical grounds since no evidence exists to my

knowledge about the effects of these factors on muscle strength in this population.

9.2 Findings and comparison with the literature

9.2.1 Pilot study

The pilot study was designed to assess the size of strength measurement error in children with CP and compare it to that of healthy children. The overall results were consistent with previous reports and confirmed that children with CP are weaker than healthy peers (Damiano 1995; Engsberg 1998; Wiley 1998; Engsberg 1999).

Children with CP were able to achieve and sustain a maximum voluntary contraction (MVC) but needed more time to achieve a force plateau. Limitations in selective motor control and the presence of co-contraction may have played a role. Spasticity itself could not affect the performance of the contractions since during isometric contractions there is no velocity dependent change of the muscle length and therefore no spastic reflex can be elicited.

Previous studies have shown that isokinetic dynamometry, in adults and children with CP, is highly reliable (Holland 1994; Van den Berg-Emons 1996; Ayalon M 2000). One recent study has investigated the reliability of hand-held dynamometry in this population and found it to be reliable for measuring mean change in groups of patients with CP, but not for individuals (Taylor 2004). Despite similarities in the testing protocol, it was not possible to compare these results of the present study with those from Taylor et al (2004) since theirs were reported in kg. However, the present results are in agreement with their view about the unsuitability of ICCs to describe reliability of strength assessment in a clinically meaningful manner. Limitations of the ICC include its dependency on the distribution

of data and the variability between subjects; in samples containing subjects who differ greatly between them, the ICC will be inflated considerably (Bland 1996; Madsen 2002).

ICCs in this study were found to be good but still lower than those reported in the literature and this is possibly due to the limited spread of strength values between subjects.

In agreement with Taylor et al (2004), it was also found that the assessment of hip extensors was not easy or reliable as indicated by the wide confidence intervals. The position was chosen both because of its ease to apply in patients with CP and its support by bibliographic reference (Stuberg 1988). However, as the dynamometer was held and not fixed, the strong extensor force often overcame the examiner's resistance. Despite stabilisation of the hips and pelvis, children would frequently use their back extensors during their attempt to push. Although the ICC was 0.78 and 0.72 for CP and healthy children respectively, this was probably due to the greater variability between subjects and is another indication of its unsuitability as a measure of reliability in this experimental setting.

The results of this study are reported in terms of within-subject standard deviation (σ_w) and related measures of repeatability. These indices can be interpreted more directly and can be applied to individual patients.

In conclusion, the results of the pilot study showed that the measurement error of this strength assessment protocol is similar in CP and healthy children for all muscle groups. The within-subject variability may seem large, but the possible range of strength values is wide and muscle strength assessment is sufficiently precise to distinguish change due to natural history or treatment.

9.2.2 Findings at six months after multi-level surgery

Results at six months post multi-level orthopaedic surgery showed improvement in joint kinematics and kinetics, but also significant loss of muscle strength and function.

The effect of multi-level surgery in five proximal muscle groups (six positions) was quantified. It was found that at six months after surgery there was a significant reduction in muscle strength in all groups measured. The greatest percentage reduction was in the knee flexors (57.6%) followed closely by the hip flexors (45.1%). These muscle groups were surgically lengthened, which may explain the greater loss of strength (Moseley 1992). However, studies have suggested that muscle force is preserved after aponeurotic lengthening (Brunner 2000), in contrast to a loss of force after tendon elongation (Delp 1995).

The hip abductors were the only group where no individual reduction in strength was found to be statistically significant. The hip abductors and the hip extensors were the two muscle groups which showed strength increases in the greatest number of individual limbs (10 and eight respectively). Of the muscles measured, these two groups were not operated on directly. The most common bony procedure performed was femoral derotation osteotomy (33/39 legs), which is thought to correct lever-arm dysfunction and, in doing so, may confer a biomechanical advantage on the hip abductors post-operatively.

Knee extensors at 30° of flexion were not measured in 18/39 limbs due mainly to knee contractures ≥ 30° (10 limbs – see Appendix four for all reasons of excluded/missing values). Following surgical correction of knee contractures it was possible to produce some knee extensor force in this position post-operatively, but these limbs were not included in the statistical analysis.

In agreement with published data, these results showed significant early improvements in all planes of lower limb joint angles and moments after surgery. In theory, the improvement in kinematics and kinetics would lead, in time, to a better overall function when rehabilitation is complete. However it was found that at six months after surgery gross motor function and walking velocity were significantly reduced. Indeed Damiano et al (1999) did not show functional improvement following hamstring lengthening, as evidenced by increased stride length, until strength values approximated or exceeded pre-operative values at nine months post operatively.

It should be noted, however, that rehabilitation would be considered incomplete at this stage. Although animal studies suggest that muscle or tendon surgery produces a short-term loss of strength, which is completely recovered in six weeks (Brunner 2000), clinical experience has shown the rehabilitation period following multi-level orthopaedic surgery to be prolonged with continued improvement for up to two years (Nene 1993). Rehabilitation may be particularly long in children with central nervous system disorders because the degree of post-operative weakness has not been fully appreciated or adequately addressed. Strength gains can be achieved through resistance training at the same rate as in healthy individuals (Damiano 1995) and, if employed post-operatively, may shorten the rehabilitation period following multi-level surgery. It would be desirable to produce further evidence on which physiotherapy regimes would be most effective in reducing the rehabilitation period.

9.2.3 Findings at six months after multi-level surgery and physiotherapy programme

Limited evidence exists about the effect of specific physiotherapy regimes following multi-level surgery (Patikas 2006). Therefore, published guidelines for physiotherapy post multi-level surgery do not exist to my knowledge. The results presented in Chapter Seven show that intensive

physiotherapy regimes were effective in improving strength and motor function after surgery. This was the case in both resistance and active strengthening groups. Furthermore, the progressive resistance strengthening programme showed advantages over active strengthening post-operatively. This is in agreement with previous findings in non surgical patients (Damiano 1995; Damiano 1998; Dodd 2003), but contradicts the recent findings from Patikas et al (2006) in patients following surgery. In this randomised controlled trial of strength training after multi-level surgery, the group that followed a specific strength training programme in addition to regular physiotherapy, failed to show any significant advantages compared with the group that had only conventional physiotherapy. Their long training programme (40 weeks), lack of supervision and early initiation of the programme after surgery may have influenced their results.

The results of the present study indicate a trend towards better muscle strength recovery in the resistance group. Statistical significance in the between groups comparison was achieved in only two muscle groups, namely the hip flexors and knee extensors at 30°. However, when considering each group separately, the resistance strengthening group showed improvement in a greater number of muscle groups following treatment. The same trend was noticed when function and gait variables were tested. Furthermore, resistance training resulted in significant improvement of the temporal parameters when groups were assessed separately. Previous studies have documented the advantages of strength training in patients with CP. The results of the present study showed the overall benefits of strength training post-operatively and indicate that resistance training may have advantages over active exercise. There are several explanations for these findings.

Most previous studies that assessed the effectiveness of strength training in CP have used training protocols with a duration of six to eight weeks (Horvat 1987; Damiano 1995; Damiano 1995; MacPhail 1995; Damiano

1998). However, it has been suggested that a training programme longer than six weeks is required to show the benefits of exercise in CP patients (Damiano 1995) and patients with spinal cord injuries (Hicks 2003). The short duration of the programme in this study may be an explanation for the presence of significant differences between the two regimes in a sub-group of the tested parameters only. However, the duration of the programme was considered adequate for the time frame and resources available as well as for maintaining motivation of the participants. This last issue was shown to be problematic in longer training regimes (Patikas 2006).

It could be argued that an earlier start of strength training may have led to an earlier improvement in motor function. However, some of these children and particularly, the more severely affected ones would have been unable to co-operate sufficiently in a vigorous strengthening programme at an earlier stage. Patikas et al (2006) started their programme at three to four weeks post-operatively and yet no benefits of strength training were shown. On balance, the six months point was chosen in this study as the earliest opportunity where sufficient recovery would have occurred to allow strength training, while overall rehabilitation was still ongoing and further improvement in function was expected.

Another factor that may have affected these results is the intensity of resistance given in the resistance strengthening group. Damiano (1995) used a progressive resistance protocol in their study of quadriceps femoris strengthening in spastic diplegic children. They determined the load used by measuring the MVC of the quadriceps at 90° of knee flexion, doubling this value and using 65% of this load. This method was found inadequate for this study design since the set up for measuring muscle strength was a fixed one and required patients to be assessed at the gait analysis laboratory. Instead, the 10-repetition maximum (RM) was used to determine maximum effort as suggested by the paediatric strength training literature (Faigenbaum 2000; American Academy of Pediatrics Committee on Sports Medicine and

Fitness. 2001). The child attempted to lift 10 times the maximum weight he or she could and this became the training load for that session until re-assessment. For some severely affected children however a sub-maximal weight was used for training (11 or 12 RM), which although proved to be a quick and safe method of determining the training load, it may have led to less intensive exercises.

It should also be mentioned that additional parameters, which were not controlled, could have affected the outcome. All subjects had spastic diplegia with GMFCS level between I and III. However, there was significant variation between these functional levels, which may have influenced the response to exercise. Interestingly, it was found that children with a higher functional level responded better to strengthening exercises irrespective of the protocol type. To my knowledge, there is no previous report of similar findings, and their implications on designing appropriate post multi-level rehabilitation regimes are obvious. Clinicians should expect easier strengthening and better functional recovery from the less severely affected patients.

All children had three-level orthopaedic surgery by the same paediatric surgeon and received the training programme by one trained clinician in the majority of cases. However, the operations performed were dictated by the child's individual clinical needs and some variation existed. As in the study by Patikas et al (2006), the administered strength training programmes, in the present study may have not been sufficiently long or intensive to overcome the muscle weakness caused by surgery. Although, once could attempt to analyse the individual effect of each operation used in the context of multi-level surgery, this was not considered adequate for the present study design. The sample size of this study would be too small for any statistical method used to assess this effect.

To my knowledge this is the first objective comparison between different physiotherapy regimes following multi-level surgery in CP. It demonstrated that following six weeks of intensive physiotherapy treatment focused on strength training, muscle strength and motor function improved significantly. This finding further underlines the deleterious effects of muscle weakness on motor function in CP and emphasises the importance of strength training in the rehabilitation of children with CP following multi-level surgery.

9.2.4 Findings at one year

Although research suggests that children with CP are weaker than their healthy counterparts, a few studies have demonstrated the distribution of weakness in these patients. Muscle weakness can be localised or generalised and it affects some muscles more than others, often showing greater involvement of the plantarflexors and dorsiflexors (Brown 1991; Wiley 1998; Engsberg 2000). Olney et al (1990) reported inadequate power production in the affected lower extremity of hemiplegic children, particularly in the ankle plantarflexor and knee extensor muscles. Furthermore, Bourbonnais (1989) and Wiley (1998) have provided some insight into the severity and distribution of weakness in spastic hemiparesis and children with CP, respectively. The results of the present study confirmed those previously published in the literature and showed that spastic diplegic patients are weaker than their healthy counterparts in lower limb muscle strength. However, the need for developing strength profiles based on large populations of CP patients with variable ages and type of involvement remains.

There is limited information regarding the natural history of muscle strength in children with spastic diplegia and no long term studies exist which follow these patients to adulthood. The different interventions performed at different stages throughout these patients' development do not allow for this type of research to be accomplished. However, the findings of

this study highlighted the changes of strength and function over time in patients who did not receive any form of treatment other than "routine" community physiotherapy for a period of one year.

It was shown that although no statistically significant decrease in gross motor function and temporal parameters was observed, a trend towards deterioration in function was evident. More importantly, there was a clear deterioration of the kinematic and kinetic variables describing gait during the year of the study. This would have significant implications on the advice given to parents regarding surgery: the findings suggest that even at a young age and high functional level, children with spastic diplegia may deteriorate faster than previously thought. The lack of significance in changes of muscle strength did not support the initial hypothesis that diplegic children would be weaker over time. Although this finding could be attributed to the sample size, the limited follow up period, the younger average age and the better functional level, still an important observation could be made. Muscle strength did not increase in this group as would have been expected in healthy growing children.

Following the patients who underwent multi-level surgery during the year of the study revealed some interesting findings. All surgical patients did show a significant decrease of muscle strength at six months following surgery. This decrease however, was greater in group C, probably by chance. This could have affected this group's speed of recovery compared with group B. The six-week intensive strengthening programme did improve muscle strength in all surgical patients. This change was greater for group C who received the progressive resistance strengthening programme.

At one year muscle strength did not reach pre-operative values and function, as described by the GMFM, and temporal parameters followed the same trend. These results suggest that intensive physiotherapy following multi-level surgery can improve strength and function but further research

into the duration and other types of post-op physiotherapy is needed. Maybe a longer strengthening programme is essential to improve the speed of recovery after multi-level surgery, something that could have implications for the overall management of patients with spastic diplegia.

Finally, the changes in gait showed a significant improvement after surgery as expected. However there was no observed difference following the six-week physiotherapy programme. Some deterioration was observed between the six and 12-month assessments but gait parameters still remained significantly improved compared to pre-operative levels. It could be argued that this deterioration could have been avoided if a longer or more intensive programme had been implemented.

9.3 Synopsis of the study findings

1. The strength assessment protocol used in this thesis is reliable for this population and research setting.
2. Children with CP are able to perform isometric contractions and can achieve and maintain a maximum voluntary contraction. However, they need more time than healthy children to reach MVC.
3. The results of the pilot study confirm that children with spastic diplegia are weaker than their healthy counterparts.
4. Despite modifications of the dynamometer, no optimal position for testing hip extensors was found. This resulted in low reliability measurements in this muscle group.
5. At six months after multi-level surgery significant improvements in joint kinematics and kinetics were shown.
6. Despite improvements in gait analysis parameters, significant loss of strength in lower limb muscle groups persisted at six months. Knee and hip flexor muscle groups showed the greatest reduction in strength.
7. Gross motor function followed the same trend as muscle strength and remained significantly lower compared with pre-operative values.

8. Gait analysis confirmed the effectiveness of multi-level surgery in improving deformities. However, rehabilitation at six months should be considered incomplete. Further research into improving post-operative physiotherapy management would be beneficial.
9. There is limited evidence based information about the effect of specific physiotherapy programmes following multi-level surgery.
10. Using two different intensive physiotherapy regimes it was shown that muscle strengthening at six months post-surgery was effective in improving strength and motor function.
11. A randomised trial between two different physiotherapy regimes showed advantages of resistance training over active exercise.
12. The patient's functional level plays a significant role in deciding the type and duration of such programmes and in predicting outcome.
13. Further research with larger samples is required to identify significant treatment effects.
14. At one year, no significant deterioration in strength or function was found in the conservatively treated group. There was however a trend towards deterioration over time. The fact that this group was younger, with a higher functional level may have played a role.
15. At one year, significant deterioration in sagittal kinematic parameters was found in the conservatively treated group. This could mean that changes in strength and function present later in a deteriorating diplegic child.
16. At one year, surgical patients preserved the post-physiotherapy strength and function gains but these did not reach the pre-op values. This would support the clinical observation that prolonged and intensive physiotherapy is required for these patients post-multi level surgery and that rehabilitation exceeds the 12-month point.
17. At one year, the surgical patients showed some deterioration in gait parameters but these were still significantly improved compared to pre-operative values.

9.4 Study limitations

There were several limitations in this study. Firstly, the sample of conservatively treated patients (group A) was self-selected and not randomised. Although this was decided for ethical reasons, it resulted in a younger and more highly functional group of patients than the surgical groups. This may have led to the lack of statistically significant deterioration of the strength and function variables at one year in this group, which did not fully support one of the hypotheses of the study. However, the significant deterioration of the kinematic and kinetic variables provided some insight to the natural history of diplegic CP and supported the clinical observation of deterioration over time. It would be of interest to follow these patients for a longer period of time and see at what stage deterioration of strength and function occurs.

Although it would have been of interest to compare statistically the conservatively treated group with the surgical groups, this was not possible. Patients in group A were not matched with these in groups B & C who were also randomised.

It could be argued than a larger sample would have shown more clearly the different changes especially since different combinations of operative procedures were used. Due to the single centre design of the study the number of patients was limited, which may have compromised the statistical significance of the results.

Some aspects of the assessment methodology used could also be subject of criticism. Direct comparisons of gait parameters were undertaken despite inconsistent use of orthoses by a number of patients throughout the duration of the study. Although, the gait parameters chosen were thought to provide useful information relevant to the questions of the study, it could be argued that a different set of variables could have been used.

It would have been useful to use EMG recordings during the strength assessment sessions of the main study in order to monitor the effect of co-contraction on muscle performance and investigate any changes after the implementation of the intensive strengthening regimes. However, this would have increased considerably the duration of the testing session, something that most patients would have found extremely difficult.

It could be argued that a 6-week strengthening regime is not long enough to produce detectable strength gains. However, based on the strength training literature (Damiano 1995; Damiano 1995) and available resources it was felt that this would be an acceptable intervention period for patients and families.

9.5 Significance of findings

The present study contributed to the relevant area of knowledge in several ways. Firstly, a reliable and objective assessment protocol of lower limb muscle strength was tested for the CP population. This allowed it to be used in further research. In addition, it could be modified to examine different muscle groups.

This is the first study, which attempted to objectively measure strength of lower limb muscle groups in spastic diplegia six months after multi-level surgery. The results of this study confirmed the clinical impression that a significant degree of muscle weakness is present at this point of the rehabilitation. This loss of strength led to a significant deterioration of gross motor function. Although multi-level orthopaedic surgery corrected the musculoskeletal deformities and improved significantly the gait parameters, at this early point of rehabilitation it also caused loss of function and strength.

The overall aim of the study was to provide scientific evidence for post-operative rehabilitation. This objective has been achieved. This is the first

study, which examined two different physiotherapy regimes following multi-level orthopaedic surgery in a controlled prospective manner. It showed that an intensive course of strengthening (resistance or active) after this type of surgery is beneficial to the overall rehabilitation process. Furthermore, it provided evidence that progressive resistance strengthening programmes using isotonic exercises and weights contribute to a greater increase in strength and function than active strengthening exercises.

An unexpected significant finding was the relationship of functional level with the response to exercise. It was found that irrespectively of physiotherapy type provided, patients of higher functional level responded better to exercise. This response was demonstrated with improvements in strength and function.

The results at one year after surgery led to further research questions on the exact content and duration of these programmes. Indeed, assessment at one year after surgery showed that although strength, function and gait continue to improve, the rehabilitation is still incomplete, despite the implementation of such programmes.

9.6 Clinical relevance

This study has emphasised the importance of objective measurement of muscle strength in children with spastic diplegia. It is proposed that future research in this area should include specific strength assessment protocols in the same principle used in gait analysis research. Patients with higher functional level respond better to exercise, as shown in the present study. This indicates the significant contribution of pre-operative strength to the outcome of surgery and rehabilitation. In this context, use of formal quantitative muscle strength measurements in clinical practice when contemplating surgery, should be considered important.

Moreover, the significant deterioration of the gait variables at one year seen in the group of conservatively treated patients could influence the advice given to parents in relation to the indications for multi-level surgery. The rate of deterioration and the presence of weakness could help parents and clinicians make a more informed decision regarding surgery.

This study highlighted the effects of muscle weakness on the rehabilitation process after surgery having first demonstrated that limited evidence existed on appropriate rehabilitation following multi-level orthopaedic surgery, a widely used operative procedure in this population.

For the first time, intensive physiotherapy after multi-level surgery was shown to make a difference in muscle strength as well as in gait and function. The results from this randomised clinical trial showed that overall strengthening regimes after multi-level surgery at a time when patients have recovered from the operation itself are beneficial. In particular, one type of rehabilitation, progressive resistance strengthening isotonic exercises, is not only safe for this population but also more effective that active strengthening exercises. This finding could have significant implications on managing human and financial resources, and designing appropriate rehabilitation programmes after multi-level orthopaedic surgery. It is recommended that resistance strengthening physiotherapy regimes are implemented following multi-level orthopaedic surgery and become part of routine clinical practice.

9.7 Future research

The results of this study have generated more questions and several areas for further study can be suggested.

Quantitative muscle strength measurements in clinical practice should be considered an important part of the decision making process in the conservative or surgical management of children with spastic diplegia.

Therefore, a database of lower limb muscle strength of healthy children and age/sex matched patients, measured with a pre-defined strength protocol, would be necessary and clinically helpful.

This study concentrated on patients with the spastic diplegic form of cerebral palsy. Further research on muscle strength changes over time in less or more severely affected patients is still needed. There is also a need for prospective assessment of muscle strength and function in conservatively treated patients for a longer period of time using larger patient samples in order to understand the natural history of weakness in spastic diplegia and its implications on long-term rehabilitation.

The present study investigated the effectiveness of two specific physiotherapy regimes, which used strengthening with weight resistance or active exercise. Further research could compare additional post multi-level surgery physiotherapy programmes, which would differ in duration, frequency, intensity or type of exercise, in order to create appropriate guidelines for post-operative rehabilitation depending on individual needs.

Finally, this study highlighted the previously underestimated effect of multi-level surgery on muscle strength and function for an extended period of time. Therefore, research into different surgical techniques, which would preserve strength, allow earlier mobilisation and implement a resistance strength training programme at an earlier stage post-operatively, would be considered important for a faster and more optimal recovery of these patients.

Bibliography

Abel, M. F., Damiano, D.L., Pannunzio, M., and Bush, J. (1999). "Muscle-tendon surgery in diplegic Cerebral Palsy: functional and mechanical changes." Journal of Pediatric Orthopaedics **19**(3): 366-375.

Agnarsson, U., Warde, C., McCarthy, G., et al. (1993). "Anorectal function of children with neurological problems. II: Cerebral Palsy." Developmental Medicine & Child Neurology **35**: 903-908.

Aitkens, S., Lord, J., Bernauer, E., Fowler, W.M., Lieberman, J.S., and Berk, P. (1989). "Relationship of manual muscle testing to objective strength measurements." Muscle & Nerve **12**: 173-177.

Altman, D. G. (1991). Practical Statistics for Medical Research. Florida, USA, Chapman & Hall/ CRC.

American Academy of Pediatrics Committee on Sports Medicine and Fitness. (2001). "Srength training by children and adolescents (RE0048), Policy Statement." Pediatrics **107**(6): 1470-1472.

American., A., of., Pediatrics. (1982). "The Doman-Delacato treatment of neurologically handicapped children." Pediatrics **70**: 810-812.

Amundsen, L. R., Ed. (1990). Muscle strength testing. Instrumented and non-instrumented systems. New York, Churchill Livingstone Inc.

Andrews, A. W., Thomas, M.W., and Bohannon, R.W. (1996). "Normative values for isometric muscle force measurements obtained with hand-held dynamometers." Physical Therapy **76**(3): 248-259.

Aniansson, A., Grimby, G., and Rundgren, A. (1980). "Isometric and isokinetic quadriceps muscle strength in 70-year-old men and women." Scandinavian Journal of Rehabilitation Medicine **12**: 161-168.

Armstrong, D., and Norman, MG. (1974). "Periventricular leukomalacia in neonates: complications and sequelae." Archives of Disease in Childhood **49**: 367-375.

Ashworth, B. (1964). "Preliminary trial of carisoprodol in multiple sclerosis." Practitioner **192**: 540-542.

Atha, J. (1981). Strengthening muscle. Exercise and Sport Sciences Reviews. D. J. Miller, USA American College of Sports Medicine. **9**: 1-73.

Ayalon M, B. S. D., Hutzler Y and Gilad T (2000). "Reliability of isokinetic strength measurements of the knee in children with cerebral palsy." Developmental Medicine & Child Neurology **42**: 398-402.

Ayres, A. J. (1974). The development of sensory integration theory and practice. Dubuque, IA, Kendall/Hunt.

Backman, E., Johansson, V., Hager, B., Sjoblom, P., and Henriksson, K.G. (1995). "Isometric muscle strength and muscular endurance in normal persons aged between 17 and 70 years." Scandinavian Journal of Rehabilitation Medicine **27**: 109-117.

Backman, E., Odenrick, P., Henriksson, K.G., and Ledin, T. (1989). "Isometric muscle force and anthropometric values in normal children aged between 3.5 and 15 years." Scandinavian Journal of Rehabilitation Medicine **21**: 105-114.

Bagg, M., Farber, J., and Miller, F. (1993). "Long-term follow-up of hip subluxation in cerebral palsy patients." Journal of Pediatric Orthopaedics **13**: 32-36.

Bairstow, P., Cochrane, R., and Hur, J. (1993). Evaluation of conductive education for children with cerebral palsy. Parts I and II. London, HMSO.

Bajd, T., and Vodovnik, L. (1984). "Pendulum testing of spasticity." Journal of Biomed. Eng. **6**(January): 9-16.

Baltopoulos, V., and Brodie, D.A. (1989). "Isokinetic dynamometry: applications and limitations." Sports Medicine **8**: 101-116.

Bauman, J. U., Ruetsch, H., and Schurmann, K. (1980). "Distal hamstring lengthening in cerebral palsy. An evaluation by gait analysis." International Orthopaedics **3**: 305-309.

Bax, M. (2001). "Does "therapy" have a future?" Developmental Medicine & Child Neurology **43**: 3.

Bax, M. C. O. (1964). "Terminology and classification of Cerebral Pasly." Developmental Medicine & Child Neurology **6**: 295-297.

Bayley, N. (1969). Bayley scales of infant development. New York, The Psychological Corporation.

Beasley, W. C. (1956). "Influence of method on estimates of normal knee extensor force among normal and postpolio children." Physical Therapy Review **36**: 21-41.

Beasley, W. C. (1961). "Quantitative muscle testing: principles and application to research and clinical services." Archives of Physical and Medical Rehabilitation **42**: 398-425.

Beckung, E., and Hagberg, G. (2000). "Correlation between ICIDH handicap code and Gross Motor Function Classification System in children with cerebral palsy." Developmental Medicine and Child Neurology **42**: 669-673.

Bell, K. J., Ounpuu, S., DeLuca, P.A., and Romness, M.J. (2002). "Natural progression of gait in children with cerebral palsy." Journal of Pediatric Orthopaedics **22**: 677-682.

Berkow, R., Ed. (1992). The Merck Manual, Merck Research Laboratories, Merck & Co., INC, Rahway, NJ.

Biether, J., Cummins, SK., and Nelson, KB. (1993). "The California Cerebral Palsy Project." Pediatr Perinatol Epidemiol **6**: 339-351.

Bishop, K. N., Durrant, E., Allesen, P.E., and Merril, G. (1991). "The effect of eccentric strength training at various speeds on concentric strength of the quadriceps and hamstring muscles." Journal of Orthopaedics Sports Physical Therapy **13**: 226-230.

Bland, J. M., and Altman, D.G. (1996). "Measurement error." British Medical Journal **312**: 1654.

Bland, J. M., and Altman, D.G. (1996). "Statistics Notes: measurement error." British Medical Journal **313**: 744.

Bland, J. M., and Altman, D.G. (1996). "Statistics Notes: measurement error and correlation coefficients." British Medical Journal **313**: 41-42.

Bland, J. M., and Altman, D.G. (1996). "Statistics notes: the use of transformation when comparing two means." British Medical Journal **312**: 1153.

Bleck, E. E. (1975). "Locomotor prognosis in cerebral palsy." Developmental Medicine & Child Neurology **17**: 18-25.

Bleck, E. E. (1984). "Where have all the cerebral palsy children gone? The needs of adults." Developmental Medicine & Child Neurology **26**: 674-676.

Bleck, E. E. (1987). Orthopaedic management in cerebral palsy. Oxford, Mac Keith Press.

Bobath, B. (1963). "A neuro-developmental treatment of cerebral palsy." Physiotherapy **49**: 242-244.

Bobath, B. (1976). Adult Hemiplegia: Evaluation and Treatment. London, William Heinemann Medical Books Ltd.

Bobath, K. (1980). Neurophysiological basis for the treatment of cerebral palsy. London, William Heinemann Medical Books Ltd.

Bohannon, R. W. (1986). "Manual muscle test scores and dynamometer test scores of knee extension strength." Archives of Physical Medicine & Rehabilitation **67**: 390-392.

Bohannon, R. W. (1986). "Test-retest reliability of hand-held dynamometry during a single session of strength assessment." Physical Therapy **66**(2): 206-209.

Bohannon, R. W. (1987). "Hand-held dynamometry; stability od muscle strength over multiple measurements." Clinical Biomechanics **2**: 74-77.

Bohannon, R. W. (1987). "Hand-held dynamometry; stability of muscle strength over multiple measurements." Clinical Biomechanics **2**: 74-77.

Bohannon, R. W. (1990). "Hand-held compared with isokinetic dynamometry for measurement of static knee extension torque (parallel reliability of dynamometers)." Clin. Phys. Physiol. Meas. **11**(3): 217-222.

Bohannon, R. W. (1997). "Hand-held dynamometry: factors influencing reliability and validity." Clinical Rehabilitation **11**: 263-264.

Bohannon, R. W., and Andrews, A.W. (1987). "Interrater reliability of hand-held dynamometry." Physical Therapy **67**(6): 931-933.

Bohannon, R. W., and Andrews, A.W. (1990). "Correlation of knee extensor muscle torque and spasticity with gait speed in patients with stroke." Archives of Physical and Medical Rehabilitation **71**: 330-333.

Bohannon, R. W., and Corrigan, D. (2000). "A broad range of forces is encompassed by the maximum manual muscle test grade of five." Percept Mot Skills **90**(3 pT 1): 747-750.

Bohannon, R. W., and Smith, M.B. (1987). "Interrater reliability of a Modified Ashworth scale of muscle spasticity." Physical Therapy **67**(2): 206-207.

Boiteau, M., Malouin, F. and Richards, C.L. (1995). "Use of a hand-held dynamometer and a Kin-Com dynamometer for evaluating spastic hypertonia in children: a reliability study." Physical Therapy **75**(9): 796-802.

Booth, C. M., Cortina-Borjia, M.J.F., Theologis, T.N. (2001). "Collagen accumulation in muscles of children with cerebral palsy and correlation with severity of spasticity." Developmental Medicine & Child Neurology **43**: 314-320.

Boscarino, L. F., Ounpuu, S., Davis, R.V., et al (1993). "Effects of selective dorsal rhizotomy on gait in children with cerebral palsy." Journal of Pediatric Orthopaedics **13**: 174-179.

Bottos, M., Feliciangeli, A., Sciuto, L., Gericke, C., and Vianello, A. (2001). "Functional status of adults with cerebral palsy and implications for treatment of children." Developmental Medicine & Child Neurology **43**: 516-528.

Bourbonnais, D., and Noven, S.V. (1989). "Weakness in patients with hemiparesis." The American Journal of Occupational Therapy **43**(5): 313-319.

Bower, E., and McLellan, D.L. (1992). "Effect of increased exposure to physiotherapy on skill acquisition of children with cerebral palsy." Developmental Medicine & Child Neurology **34**: 25-39.

Bower, E., McLellan, D.L., Arney, J., and Campbell, M.J. (1996). "A randomised controlled trial of different intensities of physiotherapy and different goal-setting procedures in 44 children with cerebral palsy." Developmental Medicine & Child Neurology **38**: 226-237.

Bower, E., Michell, D., Burnett, M., Campbell, M.J., and McLellan, D.L. (2001). "Randomized controlled trial of physiotherapy in 56 children with cerebral

palsy followed for 18 months." Developmental Medicine & Child Neurology **43**: 4-15.

Bozynski, M., Nelson, M., Genaze, D., et al (1988). "Cranial ultrasonography and the production of cerebral palsy in infants weighing 1200 grams at birth." Developmental Medicine & Child Neurology **30**: 342-348.

Brooke, M. H., Griggs, R.C., Mendell, J.R., Fenichel, G.M., Shumate, J.B., and Pellegrino, R.J. (1981). "Clinical trial in Duchenne dystrophy. I. The design of the protocol." Muscle Nerve **4**: 186-197.

Brouwer, B., Wheeldon, R.K., and Stradiotto-Parker, N. (1998). "Reflex excitability and isometric force production in cerebral palsy: the effect of serial casting." Developmental Medicine & Child Neurology **40**: 168-175.

Brown, J. K., Rodda, J., Walsh, E.G., and Wright, G.W. (1991). "Neurophysiology of the lower-limb function in hemiplegic children." Developmental Medicine & Child Neurology **33**: 1037-1047.

Browne, A. O., and McManus, F. (1987). "One-session surgery for bilateral correction of lower limb deformities in spastic diplegia." Journal of Pediatric Orthopaedics **7**(3): 259-261.

Brunner, R., Jaspers, R.T., Pel, J.J.M., and Huijing, P.A. (2000). "Acute and long-term effects on muscle force after intramuscular aponeurotic lengthening." Clinical Orthopaedics and Related Research **378**(September): 264-273.

Brussock, C. M., Haley, S.M., Munsat, T.L., and Bernhardt, D.B. (1992). "Measurement of isometric force in children with and without Duchenne's Muscular Dystrophy." Physical Therapy **72**(2): 105-114.

Burnett, C. N., Betts, E.F., and King, W.M. (1990). "Reliability of isokinetic measurements of hip muscle torque in young boys." Physical Therapy **70**(4): 244-249.

Butler, C., and Darrah, J. (2001). "Effects of neurodevelopmental treatment (NDT) for cerebral palsy: an AACPDM evidence report." Developmental Medicine & Child Neurology **43**: 778-90.

Campbell, S. K. (1990). "Proceedings of the consensus conference on the efficacy of physical therapy in the management of cerebral palsy." Paediatric Phys Ther **2(125)**: 176.

Campos da Pas, A., Burnett, S.M., and Braga, L.W. (1994). "Walking prognosis in cerebral palsy: a 22-year retrospective analysis." Developmental Medicine & Child Neurology **36**: 130-134.

Cans, C., (Surveillance of Cerebral Palsy in Europe) (2000). "Surveillance of cerebral palsy in Europe: a collaboration of cerebral palsy surveys and registers." Developmental Medicine & Child Neurology **42**: 816-824.

Cans, C., McManus, V., Crowley, M., Guillem, P., Platt, M. J., Johnson, A., and Arnaud, C. (2004). "Cerebral palsy of post-neonatal origin: characteristics and risk factors." Paediatr Perinat Epidemiol **18**(3): 214-20.

Carey, J. R. (1990). "Manual stretch: effect on finger movement control in stroke subjects with spastic extrinsic finger flexor muscles." Archives of Physical Medicine & Rehabilitation **71**: 888-894.

Carpenter, D. M., Graves, J.E., Pollock, M.L., et al. (1991). "Effect of 12 and 20 weeks of resistance training on lumbar extension torque production." Physical Therapy **71**: 580-588.

Carpenter, E. B. (1979). "Role of nerve block in the foot and ankle in the therapeutic management of cerebral palsy." Foot Ankle **4**: 164-166.

Castle, M. E., Reyman, T.A., and Schneider, M. (1979). "Pathology of spastic muscle in cerebral palsy." Clinical Orthopaedics **142**: 223-233.

Chiswick, M. L. (1986). "Commentary on current World Health Organization definitions used in perinatal statistics." British Jounal of Obstetrics and Gynaecology **93**: 1236-1238.

Chung, C. Y., Stout, J., and Gage, J.R. (1997). "Rectus femoris transfer - Gracilis versus Sartorius." Gait & Posture **6**: 137-146.

Clark, S. L., and Hankins, G.D. (2003). "Temporal and demographic trends in cerebral palsy - fact and fiction." American Journal of Obstetrics and Gynecology **188**(3): 628-633.

Corcoran, P. J., and Brengelmann, G.L. (1970). "Oxyzen uptake in normal and handicapped subjects, in relation to speed of walking beside velocity-controlled cart." Archives of Physical and Medical Rehabilitation **51**: 78.

Cusimano, S., and Davis, R. (1988). Weight training: The key to individual event success. Training guide to Cerebral Palsy Sports. J. A. Jones, Illinois: Human kinetics books.

Damiano, D. L. (1993). "Reviewing muscle cocontraction: is it a developmental, pathological, or motor control issue?" Physical & Occupational Therapy in Pediatrics **12**(4): 3-20.

Damiano, D. L. (1996). "Strength training to improve gait in cerebral palsy." The Standard (Vicon Motion Systems) **1**: 1-3.

Damiano, D. L., Abel, M.F., Pannunzio, M., and Romano, J.P. (1999). "Interrelationships of strength and gait before and after hamstrings lengthening." Journal of Pediatric Orthopaedics **19**(3): 352-358.

Damiano, D. L., and Abel, M.F. (1998). "Functional outcomes of strength training in spastic cerebral palsy." Archives of Physical Medicine & Rehabilitation **79**(February): 119-125.

Damiano, D. L., Dodd, K., and Taylor, N.F. (2002). "Should we be testing and training muscle strength in cerebral palsy?" Developmental Medicine & Child Neurology **44**: 68-72.

Damiano, D. L., Kelly, L.E., and Vaughan, C.L. (1995). "Effects of quadriceps femoris muscle strengthening on crouch gait in children with spastic diplegia." Physical Therapy **75**(8): 658-671.

Damiano, D. L., Martellotta, T.L., Sullivan, D.J., Granata, K.P., and Abel, M.F. (2000). "Muscle force production and functional performance in spastic cerebral palsy: relationship of cocontraction." Archives of Physical Medicine and Rehabilitation **81**(July): 895-900.

Damiano, D. L., Vaughan, C.L., and Abel, M.F. (1995). "Muscle response to heavy resistance exercise in children with spastic cerebral palsy." Developmental Medicine and Child Neurology **37**: 731-739.

Daniels, L., and Worthingham, C. (1972). Muscle testing techniques of manual examination. Philadelphia, W.B. Saunders.

Darrah, J., Fan, J.S., Chen, L.C., Nunweiler, J., and Watkins, B. (1997). "Review of the effects of progressive resisted muscle strengthening in children with cerebral palsy: a clinical consensus exercise." Pediatric Physical Therapy **9**(1): 12-17.

Darrah, J., Watkins, B., Chen, L., and Bonin C. (2004). "Conductive education intervention for children with cerebral palsy: an AACPDM evidence report." Developmental Medicine & Child Neurology **46**: 187-203.

D'Avignon, M., Noren, L., and Arman, R. (1981). "Early physiotherapyand Vojta or Bobath in infants with suspected neuromotor disturbance." Neuropediatrics **12**: 232-237.

Davis, R. B., and DeLuka, P.A. (1996). Clinical gait analysis. Current methods and future directions. Human motion analysis. G. Harns, and Smith P., IEEE Press.

De Boer, A., Boukes, R.J., and Sterk, J.C. (1982). "Reliability of dynamometry in patients with a neuromuscular disorder." Engineering in Medicine **11**(4): 169-174.

De Luca, P. A., Davis III, R.B., Ounpuu, S., Rose, S., and Sirkin, R. (1997). "Alterations in surgical decision making in patients with cerebral palsy based on three-dimensional gait analysis." Journal of Pediatric Orthopaedics **17**(5): 608-614.

Deaver, G. (1956). "Methods of treating the neuromuscular disabilities." Archives of Physical Medicine & Rehabilitation **37**: 363-367.

Delp, S. L., and Zajac, F.E. (1992). "Force- and moment-generating capacity of lower-extremity muscles before and after tendon lengthening." Clinical Orthopaedics and Related Research **284**(November): 247-259.

Delp, S. L., Statler, K., and Carroll, N.C. (1995). "Preserving plantar flexion strength after surgical treatment for contractures of the triceps surae: a computer simulation study." Journal of Orthopaedic Research **13**(1): 96-104.

Deluca, C., and Erim, Z. (1994). "Common drive of motor units in regulation of muscle force." Trends in Neurosciences **17**: 299-305.

DeLuca, P. A., Davis III, R.B., Ounpuu, S., Rose, S., and Sirkin, R. (1997). "Alterations in surgical decision making in patients with cerebral palsy based on three-dimensional gait analysis." Journal of Pediatric Orthopaedics **17**(5): 608-614.

Detrembleur, C., and Plaghki, L. (2000). "Quantitative assessment of intrathecally administered baclofen in spasticity." Archives of Physical Medicine & Rehabilitation **81**(March): 279-284.

Dietz, V., and Berger, W. (1995). "Cerebral Palsy and Muscle Transformation." Developmental Medicine & Child Neurology **37**: 180-184.

Dodd, K. J., Taylor, N.F., and Graham, H.K. (2003). "A randomized clinical trial of tsrength training in young people with cerebral palsy." Dev Med Child Neurol **45**: 652-657.

Dodd, K. J., Taylor, N.F., Damiano, D.L. (2002). "A systematic review of the effectiveness of strength-training programs for people with cerebral palsy." Archives of Physical and Medical Rehabilitation **83**: 1157-1164.

Duncan, P. W., Chandler, J.M., Cananaugh, D.K., et al. (1989). "Mode and speed specificity of eccentric and concentric exercise training." Journal of Orthopaedics Sports Physical Therapy **11**: 70-75.

Edwards, R. H. T., Young, A., Hosking, G.P., and Jones, D.A. (1977). "Human skeletal muscle function: description of tests and normal values." Clinical Science and Molecular Medicine **52**: 283-290.

Effgen, S. K., and Brown, D.A. (1992). "Long-term stability of hand-held dynamometric measurements in children who have myelomeningocele." Physical Therapy **72**(6): 458-465.

Emery, C. A., Maitland, M.E., and Meeuwisse, W.H. (1999). "Test-retest reliability of isokinetic hip adductor and flexor muscle strength." Clinical Journal of Sport Medicine **9**: 79-85.

Engsberg, J. R., Olree, K.S., Ross, S.A., and Park, T.S. (1998). "Spasticity and strength changes as a function of selective dorsal rhizotomy." Journal of Neurosurgery **88**(June): 1020-1026.

Engsberg, J. R., Ross, S.A., and Park, T.S. (1999). "Changes in ankle spasticity and strength following selective dorsal rhizotomy and physical therapy for spastic cerebral palsy." Journal of Neurosurgery **91**: 727-732.

Engsberg, J. R., Ross, S.A., Oltree, K.S., and Park, T.S. (2000). "Ankle spasticity and strength in children with spastic diplegic cerebral palsy." Developmental Medicine and Child Neurology **42**: 42-47.

Fabry, G., Liu, X.C., and Molenaers, G. (1999). "Gait pattern in patients with spastic diplegic cerebral palsy who underwent staged operations." Journal of Pediatric Orthopaedics **8**: 33-38.

Faigenbaum, A. D. (2000). "Strength training for children and adolescents." Clinics in Sports Medicine **19**(4): 593-619.

Florence, M. J., Pandya, S., King, M.W., Robison, D.J., Baty, J., Miller, J.P., Schierbecker, J. and Signore, C.L. (1992). "Intrarater reliability of Manual Muscle Test (Medical Research Council Scale) grades in Duchenne's Muscular Dystrophy." Physical Therapy **72**(2): 115-126.

Folio, R. M., and Fewell, R.F. (1983). Peabody Developmental Motor Scales. Nashville, George Peabody College for Teachers.

Fowler, E. G., Ho, T.W., Nwigwe, A.I., and Dorey, F.J. (2001). "The effect of quadriceps femoris muscle strengthening exercises on spasticity in children with Cerebral Palsy." Physical Therapy **81**(6): 1215-1223.

Freud, S. (1893). Zur kenntniss der cerebralen diplegien des kindesalters (im anschlusse an die little' sche krankheit). Vienna.

Fukunaga, T., Roy, R.R., Shellock, F.G., Hodgson, J.A., and Edgerton, V.R. (1996). "Specific tension of the human plantar flexors and dorsiflexors." Journal of Applied Physiology **80**: 158-165.

Gage, J. (1991). Gait Analysis in Cerebral Palsy. Clinics in Developmental Medicine No. 121. London, England, Mac Keith Press.

Gage, J., Perry, J., Hicks, R.R., Koop, S., and Werntz, J.R. (1987). "Rectus femoris transfer to improve knee function of children with cerebral palsy." Developmental Medicine & Child Neurology **29**: 159-66.

Gillette, H. (1969). Systems of therapy in cerebral palsy. Springfield IL, Charles C Thomas.

Giuliani, C. A. (1991). "Dorsal Rhizotomy for children with Cerebral Palsy: support for concepts of motor control." Physical Therapy **71**(3): 248-259.

Gleeson, N. P., and Mercer, T.H. (1992). "Reproducibility of isokinetic leg strength and endurance characteristics of adult men and women." European Journal of Applied Physiology and Occupational Physiology **65**: 221-228.

Gough, M., Eve, L.C., Robinson, R.O., and Shortland, A.P. (2004). "Short-term outcome of multi-level surgical intervention in spastic diplegic cerebral palsy compared with natural history." Developmental Medicine & Child Neurology **46**: 91-97.

Granata, K. P., Abel, M.F., and Damiano, D.L. (2000). "Joint angular velocity in spastic gait and the influence of muscle-tendon lengthening." The Journal of Bone and Joint Surgery **82-A**(2): 174-186.

Granata, K. P., Ikeda, A.J., and Abel, M.F., (2000). "Electromechanical delay and reflex response in spastic cerebral palsy." Archives of Physical Medicine & Rehabilitation **81**(July): 888-94.

Haas, B. M. (1994). "Measuring spasticity. A survey of current practice among health care professionals." Br J Ther Rehabil **2**: 90-95.

Hailey, D., and Tomie, J. (2000). "An assessment of gait analysis in the rehabilitation of children with walking difficulties." Disability and Rehabilitation **22**(6): 275-280.

Haley, S. M., Coster, W., and Ludlow, L.H. (1991). "Pediatric functional outcome measures." Phys Med Rehabil Clin North Am **2**: 689-723.

Haley, S. M., Coster, W., Ludlow, L.H., and al. (1992). Pediatric Evaluation of Disability Inventory (PEDI). Boston, New England Medical Center Hospitals.

Haney, N. B. (1998). "Muscle strengthening in children with cerebral palsy." Physical & Occupational Therapy in Pediatrics **18**: 149-157.

Hare, N. (1993). The physical ability scale. The physical ability scale and variants. N. Hare. London, Hare Association for Physical Ability: 7-25.

Harris, F. A. (1978). "Muscle stretch receptor hypersensitization in spasticity." American Journal of Physical Medicine **57**(1): 16-28.

Harris, S. R., Atwater, S., and Crowe, T. (1985). "Accepted and controversial neuromotor therapies for infants at high risk for cerebral palsy." J Perinatol **8**: 3-12.

Healy, A. (1958). "Two methods of weight-training for children with spastic type of cerebral palsy." The Research Quarterly **29**(4): 389-395.

Henderson, R. C., Kairalla, J. A., Barrington, J. W., Abbas, A., and Stevenson, R. D. (2005). "Longitudinal changes in bone density in children and adolescents with moderate to severe cerebral palsy." J Pediatr **146**(6): 769-75.

Henderson, R. C., Kairalla, J., Abbas, A., and Stevenson, R. D. (2004). "Predicting low bone density in children and young adults with quadriplegic cerebral palsy." Dev Med Child Neurol **46**(6): 416-9.

Hermens, H. J., and Freriks, B., Ed. (1997). The state of the art on sensors and sensor placement procedures for surface electromyography: A proposal for sensor placement procedures, Deliverable of the SENIAM project, Roessingh Research and Development.

Hicks, A. L., Martin, K.A., and Ditor, D.S. (2003). "Long-term exercise training in persons with spinal cord injury: effects on strength, arm ergometry performance and psychological well-being." Spinal Cord **41**: 34-43.

Hislop, H. J., and Perrine, J.J. (1967). "The isokinetic concept of exercise." Physical Therapy **47**: 114.

Hof, A. L. (1996). "Scaling gait data to body size." Gait & Posture **4**: 222-223.

Hof, A. L. (2001). "Changes in muscles and tendons due to neural motor disorders: implications for therapeutic intervention." Neural Plasticity **8**(1-2): 71-81.

Holland, L., McCubbin, J., Nelson, E., and Steadward, R. (1994). "Reliability of concentric and eccentric muscle testing of adults with cerebral palsy." Adapted Physical Activity Quarterly **11**: 261-274.

Holland, L. J., and Steadward, R.D. (1990). "Effects of resistance and flexibility training on strength, spasticity/muscle tone, and range of motion of elite athletes with cerebral palsy." PALESTRA: The Forum of Sport, Physical Education and Recreation for the Disabled **Summer 1990**: 27-31.

Holmback, A. M., Porter, M.M., Downham, D., and Lexell, J. (1999). "Reliability of isokinetic ankle dorsiflexor strength measurements in healthy young men and women." Scandinavian Journal of Rehabilitation Medicine **31**: 229-239.

Horvat, M. (1987). "Effects of a progressive resistance training program on an individual with spastic cerebral palsy." Amer. Corr. Ther. J. **41**(1): 7-11.

Hosking, G. P., Bhat, U.S., Dubowitz, V., and Edwards, R.H.T. (1976). "Measurements of muscle strength and performance in children with normal and diseased muscle." Archives of Disease in Childhood **51**: 957-963.

Hsu, A., Tang, P., and Jan, M. (2002). "Test-retest reliability of isokinetic muscle strength of the lower extremities in patients with stroke." Archives of Physical Medicine & Rehabilitation **83**: 1130-1137.

Huei Ming Chai (2003 - access date 26/09/06). Biomechanics of skeletal muscle, http://www.pt.ntu.edu.tw/hmchai/BM03/BMmaterial/Muscle.htm.

Hullin, M. G., Robb, J.E., and Loudon, I.R. (1996). "Gait patterns in children with hemiplegic spastic cerebral palsy." Journal of Pediatric Orthopaedics. Part B **5**(4): 247-251.

Hutton, J. L. (2006). "Cerebral palsy life expectancy." Clin Perinatol **33**(2): 545-55.

Ikeda, A. J., Abel, M.F., Granata, K.P., and Damiano, D.L. (1998). "Quantification of cocontraction in spastic cerebral palsy." Electromyogr. clin. Neurophysiol. **38**: 497-504.

Ito, J., Araki, A., Tanaka, H., Tasaki, T., Cho, K., and Yamazaki, R. (1996). "Muscle histopathology in spastic cerebral palsy." Brain & Development **18**: 299-303.

Jarvis, S., Glinianaia, S.V., Torrioli, M., et al (2003). "Cerebral palsy and intrauterine growth in single births: European collaborative study." The Lancet **362**(4): 1106-1111.

Jenter, M., Lipton, G.E., and Miller, F. (1998). "Operative treatment of hallux valgus in children with cerebral palsy." Foot and Ankle International **19**: 830-835.

Johnson, D. C., Damiano, D.L., and Abel, M.F. (1997). "The evolution of gait in childhood and adolescent Cerebral Palsy." Journal of Pediatric Orthopaedics **17**(3): 392-396.

Johnston, M. V. (2004). "Clinical disorders of brain plasticity." Brain & Development **26**: 73-80.

Jones, K., and Barker, K. (1996). Human Movement Explained. Oxford, Butterworth-Heinemann Ltd.

Jones, M. A., and Stratton, G. (2000). "Muscle function assessment in children." Acta Paediatr **89**: 753-761.

Katz, R. T., and Rymer, W.Z. (1989). "Spastic hypertonia: mechanisms and measurement." Archives of Physical Medicine & Rehabilitation **70**(February): 144-155.

Katz, R. T., Rovai, G.P., Brait, C., and Rymer, W.Z. (1992). "Objective quantification of spastic hypertonia: correlation with clinical findings." Archives of Physical Medicine & Rehabilitation **73**(April): 339-347.

Keating, J. L., and Matyas, T.A. (1996). "The influence of subject and test design on dynamometric measurements of extremity muscles." Physical Therapy **76**(8): 866-889.

Ketelaar, M., Vermeer, A., and Helders, P.J.M. (1998). "Functional motor abilities of children with cerebral palsy: a systematic literature review of assessment measures." Clinical Rehabilitation **12**: 369-380.

Kilfoil, M. R., and Pierre, D.M.M.St. (1993). "Reliability of Cybex II isokinetic evaluations of of torque in post-poliomyelitis syndrome." Archives of Physical Medicine & Rehabilitation **74**: 730-735.

Kilmer, D. D., McCrory, M.A., Wright, N.C., Rosko, R.A., and Kim, H. (1997). "Hand-held dynamometry reliability in persons with neuropathic weakness." Archives of Physical Medicine & Rehabilitation **78**: 1364-1368.

Kirkpatrick, M., Wytch, R., Cole, G., and Helms, P. (1994). "Is the objective assessment of cerebral palsy reproducible?" Journal of Pediatric Orthopaedics **14**(705-708).

Kirtley, C. (access date 26/09/2006). History of the study of locomotion, http://www.univie.ac.at/cga/history/.

Koman, L. A., Mooney III, J.F., and Smith, B.P. (1993). The use of botulinum toxin in the management of cerebral palsy in pediatric patients. Botulinum and Tetanus Neurotoxins: Neurotransimission and Biomedical aspects. B. Dasgupta, R. New York, Plenum Press: 581-587.

Koman, L. A., Mooney III, J.F., Smith, B.P., Goodman, A., and Mulvaney, T. (1994). "Management of spasticity in cerebral palsy with Botulinim-A Toxin: report of a preliminary, randomized, double-blind trial." Journal of Pediatric Orthopaedics **14**(3): 299-303.

Koman, L. A., Smith, B.P. and Shilt, J.S. (2004). "Cerebral Palsy." The Lancet **363**(May 15): 1619-1631.

Koman, L. A., Smith, B.P., Tingey, C.T., Mooney, J.F., Slone, S., and Naughton, M.J. (1999). "The effect of botulinum toxin type A injections on the natural history of equinus foot deformity in paediatric cerebral palsy patients." European Journal of Neurology **6 (suppl 4)**: S19-S22.

Krageloh-Mann, I., Hagberg, G., Meisner, C., et al (1993). "Bilateral spastic cerebral palsy - A comparative study between southwest Germany and western Sweden. I: Clinical patterns and disabilities." Developmental Medicine & Child Neurology **35**: 1031-1047.

Kuban, K. C. K., and Leviton, A. (1994). "Cerebral Palsy." New England Journal of Medicine **330**: 188-195.

Kudrjavcec, T., Schoenberg, BB., Kurland, LT., et al (1985). "Cerebral Palsy: survival rates, associated handicaps, and distribution by clinical subtype - Rochester, MN. 1950-1976." Neurology **35**: 900-903.

Lance, J. W. (1980). Symposium synopsis. Spasticity: disordered motor control. R. G. Feldman, Young, R.R., Koella, W.P. Chicago, Year Book Medical: 485-494.

Law, M., Cadman, D., Rosenbaum, P., Walter, S., Russell, D., and DeMatteo, C. (1991). "Neurodevelopmental therapy and upper extremity inhibitive casting for children with cerebral palsy." Developmental Medicine & Child Neurology **33**: 379-387.

Lennon, S. M., and Ashburn, A. (1993). "Use of myometry in the assessment of neuropathic weakness: testing for reliability in clinical practice." Clinical Rehabilitation **7**: 125-133.

Levitt, S. (1982). Treatment of cerebral palsy and motor delay. Oxford UK, Blackwell Scientific Publications.

Lieber, R. L. (1986). "Skeletal muscle adaptability I: review of basic properties." Developmental Medicine & Child Neurology **28**: 390-397.

Lilienfeld, A. M., Jacobs, M., and Willis, M. (1954). "A study of the reproducibility of muscle testing and certain other aspects of muscle scoring." Physical Therapy Review **34**: 279-289.

Lin, C., Guo, L., Su, F., Chou, Y., and Cherng, R. (2000). "Common abnormal kinetic patterns of the knee in gait in spastic diplegia of cerebral palsy." Gait & Posture **11**: 224-232.

Little, W. J. (1861-1862). "On the influence of abnormal parturition, difficult labours, premature birth and asphyxia neonatorum on the mental and physical condition of the child, especially in relation to deformities." Trans Obstet Soc London **3**: 293-344.

Loeb, G. E., and Ghez, C. (2000). The motor unit and muscle action. Principles of Neural Science. E. R. Kandel, Schwartz, J.H., Jessel, T.M. USA, McGraw-Hill Companies, Inc.

Mac Keith, R. C., Mackenzie, I.C.K., and Polani, P.E. (1959). "The Little Club. Memorandum on terminology and classification of "Cerebral Palsy"." Cerebral Palsy Bulletin **5**: 27-35.

MacPhail, A. H. E., and Kramer, J.F. (1995). "Effect of isokinetic strength training on functional ability and walking efficiency in adolescents with cerebral palsy." Developmental Medicine and Child Neurology **37**: 763-775.

Madsen, O. R. (2002). "Reliability of muscle strength testing quantified by the Intraclass Correlation Coefficient." Archives of Physical Medicine & Rehabilitation **83**(April): 582.

Mahoney, F. I., and Barthel, D.W. (1965). "Functional evaluation: the Barthel Index." Md State Med J **14**: 61-65.

Markhede, G., and Grimby, G. (1980). "Measurement of strength of hip joint muscles." Scandinavian Journal of Rehabilitation Medicine **12**: 169-174.

Massagli, T. L. (1991). "Spasticity and its management in children." Phys Med Rehabil **2**: 867-890.

McCubbin, J. A., and Shasby, G.B. (1985). "Effects of isokinetic exercise on adolescents with Cerebral Palsy." Adapted Physical Activity Quarterly **2**: 56-64.

McDonald, C. (1991). "Selective dorsal rhizotomy: A critical review." Phys Med Rehabil **2**: 891-915.

McGrath, S., Splaingard, M., Alba, H., et al. (1983). "Survival and functional outcome of children with severe cerebral palsy following gastrostomy, abstract." Archives of Physical Medicine & Rehabilitation **73**: 133-137.

McLaughlin, J. F., Bjornson, K.F., Astley, S.J., Graubert, C., Hays, R.M., Roberts, T.S., Price, R., and Temkin, N. (1998). "Selective dorsal rhizotomy: efficacy and safety in an investigator-masked randomized clinical trial." Developmental Medicine & Child Neurology **40**: 220-232.

Mendell, J. R., and Florence, Julaine. (1990). "Manual muscle testing." Muscle & Nerve **Supplement 1990**: S16-S20.

Merlini, L., Dell'Accio, D., and Granata, C. (1995). "Reliability of dynamic strength knee muscle testing in children." Journal of Orthopaedics Sports Physical Therapy **22**(2): 73-76.

Milner-Brown, H. S., and Penn, R.D. (1979). "Pathophysiological mechanisms in cerebral palsy." Journal of Neurology, Neurosurgery and Psychiatry **42**: 606-618.

Molnar, G. (1987). Long-term treatment of spasticity in children with cerebral palsy. International Rehabilitation Medicine Association V Conference.

Molnar, G., and Gordon, SU. (1976). "Cerebral Palsy: predicitve value of selected clinical signs of early prognostication of motor function." Archives of Physical Medicine & Rehabilitation **57**: 153-158.

Molnar, G. E., Alexander, J., Gutfeld, N. (1979). "Reliability of quantitative strength measurements in children." Archives of Physical Medicine & Rehabilitation **60**: 218-221.

Mosca, V. S. (1995). "Calcaneal lengthening for valgus deformity of the hindfoot." Journal of Bone and Joint Surgery **American Volume, 77A**: 500-512.

Moseley, C. F. (1992). Physiologic effects of soft-tissue surgery. The diplegic child, evaluation and management. M. D. Sussman. Rosemont IL, American Academy of Orthopaedic Surgeons: 259-270.

Msall, M. E., Digaudio, K., Rogers, B.T., et al. (1994). "The Functional Independence Measure for Children (WeeFIM): conceptual basis and pilot use in children with developmental disabilities." Clin Pediatr **33**: 421-430.

Mutch, L., Alberman, E., Hangberg, B., Kodama, K, and Perat, M.V. (1992). "Cerebral Palsy Epidemiology: Where are we now and where are we going?" Developmental Medicine & Child Neurology **34**: 547-555.

Nelson, A. G., Arnall, D.A., Loy, S.F. et al (1990). "Consequences of combining strength and endurance training regimens." Physical Therapy **70**: 287-294.

Nene, A. V., Evans, G.A., and Patrick, J.H. (1993). "Simultaneous multiple operations for spastic diplegia. Outcome and functional assessment of walking in 18 patients." Journal of Bone and Joint Surgery **75(B3)**: 488-494.

Nistor, L., Markhede, G., and Grimby, G. (1982). "A technique for measurements of plantar flexion torque with the cybex II dynamometer." Scandinavian Journal of Rehabilitation Medicine **14**: 163-166.

Noonan, K. J., Halliday, S., Browne, R., O'Brien, S., Kayes, K., and Feinberg, J. (2003). "Interobserver variability of gait analysis in patients with cerebral palsy." Journal of Pediatric Orthopaedics **23**: 279-287.

Novacheck, T. F., Stout, J.L., and Tervo, R. (2000). "Reliability and validity of the Gillette Functional Assessment Questionnaire as an outcome measure in children with walking disabilities." Journal of Pediatric Orthopaedics **20**(1): 75-81.

Odding, E., Roebroeck, M. E., and Stam, H. J. (2006). "The epidemiology of cerebral palsy: incidence, impairments and risk factors." Disabil Rehabil **28**(4): 183-91.

Olney, S. J. (1985). Quantitative evaluation of cocontraction of knee and ankle muscles in normal walking. Biomechanics IX-A. D. A. Winter, Norman, R.W., Wells, R.P., Hayes, K.C., and Potla, A.E. Champaign IL, Human Kinetics: 431-435.

Olney, S. J., MacPhail, H.E.A., Hedden, D.M., and Boyce, W.F. (1990). "Work and power in hemiplegic Cerebral Palsy gait." Physical Therapy **70**(7): 431-438.

Oppenheim, W. L. (1990). "Selective posterior rhizotomy for spastic cerebral palsy." Clinical Orthopaedics and Related Research **253**(April): 20-29.

Osler, W. (1889). The cerebral palsies of children. A clinical study from the infirmary for nervous diseases. Philadelphia, PA: Blakiston.

Osternig, L. R. (1986). "Isokinetic dynamometry: implications for muscle testing and rehabilitation." Exercise Sport Sci Rev **14**: 45-80.

Ounpuu, S., DeLuca, P.A., and Davis, R.B. (1997). "The role of hip flexor and hamstring surgery on pelvic and hip motion in persons with cerebral palsy: an examination of the pre and post operative kinematics and kinetics." Gait & Posture **5**: 152.

Ounpuu, S., Muik, E., Davis III, R.B., Gage, J.R., and DeLuca, P.A. (1993). "Rectus femoris surgery in children with Cerebral Palsy. Part I: The effect of rectus femoris transfer location on knee motion." Journal of Pediatric Orthopaedics **13**(3): 325-330.

Ounpuu, S., Muik, E., Davis III, R.B., Gage, J.R., and DeLuca, P.A. (1993). "Rectus Femoris surgery in children with Cerebral Palsy. Part II: A comparison between the effect of transfer and release of the distal rectus femoris on knee motion." Journal of Pediatric Orthopaedics **13**(3): 331-335.

Palisano, R. J., Hanna, S.E., Rosenbaum, P.L., Russell, D.J., Walter, S.D., Wood, E.P., Raina, P.S., and Galuppi, B.E. (2000). "Validation of a model of gross motor function for children with cerebral palsy." Physical Therapy **80**(10): 974-985.

Palmer, F. B., Shapiro, B.K., Wachtel, R.C., Allen, M.C., Hiller, J.E., Harryman, S.E., Mosher, B.S., Meinert, C.L., and Capute, A.J. (1988). "The effects of physical therapy on cerebral palsy." The New England Journal of Medicine **318**(13): 803-808.

Paneth, N. (1993). "The causes of cerebral palsy. Recent evidence." Clinical and Investigative Medicine **16**: 95-102.

Paneth, N., and Kiely, J. (1984). The frequency of cerebral palsy: a review of population studies in industrialised nations since 1950. The epidemiology of the cerebral palsies. Clinics in Developmental Medicine No 87. F. Stanley, and Alberman, E. London, SIMP Blackwell Scientific: 46-56.

Patikas, D., Wolf, S.I., Mund, K., Armbrust, P., Schuster, W., and Doderlein, L. (2006). "Effects of a post-operative strength-training programme on the walking ability of children with cerebral palsy: a randomized controlled trial." Archives of Physical and Medical Rehabilitation **87**: 619-626.

Pediatrics, A. A. o. (1982). "The Doman-Delacato treatment of neurologically handicapped children." Pediatrics **70**: 810-812.

Perry, J. (1987). "Distal rectus femoris transfer." Developmental Medicine & Child Neurology **29**: 153-158.

Perry, J. (1992). Gait Analysis. Normal and Pathological Function. USA, SLACK Incorporated.

Perry, J., Jones, M., and Thomas, L. (1981). "Functional evaluation of Rolfing in cerebral palsy." Developmental Medicine & Child Neurology **23**: 717-729.

Petersen, S., Wessel, J., Bagnall, K., Wilkins, H., Quinney, A., and Wenger, H. (1990). "Influence of concentric resistance training on concentric and eccentric strength." Archives of Physical Medicine & Rehabilitation **71**: 101-105.

Petterson, B., Blair, E., Watson, L., Stanley, F. (1998). "Adverse outcome after multiple pregnancy." Bailliere's Clinical Obstetrics and Gynaecology **12**: 1-17.

Pharoah, P. O. D., and Cooke, T. (1996). "Cerebral palsy and multiple births." Archives of Disease in Childhood **75**: F174-F177.

Phillips, B. A., Lo, S.K., and Mastaglia, F.L. (2000). "Muscle force measured using "break" testing with a hand-held myometer in normal subjects aged 20 to 69 years." Archives of Physical Medicine & Rehabilitation **81**: 653-661.

Phillips, W. T., Batterham, A.M., Valenzuela, J.E., and Burkett, L.N. (2004). "Reliability of maximal strength testing in older adults." Archives of Physical Medicine & Rehabilitation **85**: 329-334.

Pirpiris, M., Trivett, A., Baker, R., Rodda, J., Nattrass, G.R., and Graham, H.K. (2003). "Femoral derotation osteotomy in spastic diplegia." The Journal of Bone and Joint Surgery (Br) **85-B**(2): 265-272.

Portney, L., and Watkins, M. (1993). Foundation of clinical research. USA, Stamford, CT: Appleton & Lange.

Powers, R. K., Marder-Meyer, O.T., and Rymer, W.Z. (1988). "Quantitative relations between hypertonia and stretch reflex threshold in spastic hemiparesis." Annals of Neurology **23**: 115-124.

Rang, M. (1990). Cerebral Palsy. Lovell and Winter's pediatric orthopaedics. R. T. Morissy. Philadelphia, PA, J.B. Lippincott: 465-506.

Rechtien, J. J., Gelblum, J.B., Haig, A., and Gitter, A.J. (1996). "Technology assessment: dynamic electromyography in gait and motion analysis." Muscle & Nerve **19**(March): 396-402.

Reimers, J. (1990a). "Functional changes in the antagonists after lengthening the agonists in Cerebral Palsy. I. Triceps surae lengthening." Clinical Orthopaedics and Related Research **253**(April): 30-37.

Reimers, J. (1990b). "Functional changes in the antagonists after lengthening the agonists in cerebral palsy: II Quadriceps strength before and after distal hamstring lengthening." Clinical Orthopaedics and Related Research **253**: 35-37.

Riddle, D. L., Finucane, S.D., Rothstein, J.M., and Walker, M.L. (1989). "Intasession and intersession reliability of hand-held dynamometer measurements taken on brain-damaged patients." Physical Therapy **69**(3): 182-194.

Robb, J. (1994). Orthopaedic management of cerebral palsy. Children's orthopaedics and fractures. M. Benson, Fixsen, JA., and Macnicol, MF. London, Churchill Livingstone: 255-275.

Rodda, J., and Graham, H.K. (2001). "Classification of gait patterns in spastic hemiplegia and spastic diplegia: a basis for a management algorithm." European Journal of Neurology **8**(Suppl. 5): 98-108.

Roland, E. H., and Hill, A. (1997). "How important is perinatal asphyxia in the causation of brain injury?" Mental Retardation and Developmental Disabilities Research Reviews **3**: 22-27.

Rood, M. (1956). "Neurophysiological mechanisms utilized in the treatment of neuromuscular dysfunction." Am J Occup Ther **10**: 220-224.

Roscigno, C. I. (2002). "Periventricular leukomalacia: pathophysiological concerns due to immature development of the brain." J Neurosci Nurs **34**(6): 296-302.

Rose, J., and McGill, K.C. (1998). "The motor unit in Cerebral Palsy." Developmental Medicine & Child Neurology **40**: 270-277.

Rose, J., Haskell, W.L., Gamble, J.G., et al. (1994). "Muscle pathology and clinical measures of disability in children with cerebral palsy." Journal of Orthopaedic Research **12**: 758-768.

Rose, S. A., DeLuca, P.A., Davis III, R.B., Ounpuu, S., and Gage, J.R (1993). "Kinematic and kinetic evaluation of the ankle after lengthening of the gastrocnemius fascia in children with cerebral palsy." Journal of Pediatric Orthopaedics **13**(6): 727-732.

Rosenbaum, P. L., Walter, S.D., Hanna, S.E., Palisano, R.J., Russell, D.J., Raina, P., Wood, E., Bartlett, D.J., and Galuppi, B.E. (2002). "Prognosis for gross motor function in cerebral palsy. Creation of motor development curves." J.A.M.A. **288**(11): 1357-1363.

Russell, J. D., Avery, L.M., Rosenbaum, P.L., Raina, P.S., Walter, S.D., and Palisano, R.J. (2000). "Improved scaling of the Gross Motor Function Measure for children with Cerebral Palsy: evidence of reliability and validity." Physical Therapy **80**(9): 873-885.

Russell, J. D., Rosenbaum, P., Cadman, D.T.,Gowland, C., Hardy, S., and Jarvis, S. (1989). "The gross motor function measure: a means to evaluate the effects of physical therapy." Developmental Medicine & Child Neurology **31**: 341-352.

Sala, D., and Grant, AD. (1995). "Prognosis for ambulation in cerebral palsy." Developmental Medicine & Child Neurology **37**: 1020-1026.

Saraph, V., Zwick, E.B., Uitz, C., Linhart, W., and Steinwender, G. (2000). "The Baumann procedure for fixed contracture of the gastrosoleus in cerebral palsy. Evaluation of function of the ankle after multilevel surgery." The Journal of Bone and Joint Surgery **82-B**(4): 535-540.

Saraph, V., Zwick, E.B., Zwick, G., Steinwender, G., Steinwender, G., and Linhart, W. (2002). "Multilevel surgery in spastic diplegia: evaluation by physical examination and gait analysis in 25 children." Journal of Pediatric Orthopaedics **22**: 150-157.

Scherzer, A. L., Mike, V., and Ilson, J. (1976). "Physical therapy as a determinant of change in the cerbral palsied infant." Pediatrics **58**: 47-52.

Scott, A., Chambers, C., Cain, T.E., et al (1991). "Adductor transfer in cerebral palsy: A retrospective study." Developmental Medicine & Child Neurology **33**(9): 6-7.

Scrutton, D., Ed. (1984). Management of motor disorders of children with cerebral palsy. London, Spastic International Medical Publications Mac Keith Press p 59-74.

Sheean, G., Ed. (1998). Spasticity Rehabilitation. London, Churchill Communications Europe Ltd.

Shrout, P. E., and Fleiss, J.L. (1979). "Intraclass correlations: uses in assessing rater reliability." Psychological bulletin **86**(2): 420-428.

Sinha, S., D' Souza, SW., Rivlin, E., et al. (1990). "Ischemic brain lesions diagnosed at birth in preterm infants: Clinical events and developmental outcome." Archives of Disease in Childhood **65**: 1017-1020.

Skaggs, D. L., Rethlefsen, S.A., Kay, R.M., Dennis, S.W., Reynolds, R.A.K., and Tolo, V.T. (2000). "Variability in gait analysis interpretation." Journal of Pediatric Orthopaedics **20**: 759-764.

Sloan, C. (2002). "Review of the reliability and validity of myometry with children." Physical & Occupational Therapy in Pediatrics **22**(2): 79-93.

Sommerfeld, D., Fraser, B., Hensinger, R.N., and Beresford, C.V. (1981). "Evaluation of physical therapy service for severely mentally impaired students with cerebral palsy." Physical Therapy **61**: 338-344.

Stanley, F., Blair, E. and Alberman, E. (2000d). Introduction. Cerebral Palsies: Epidemiology and Causal Pathways. Clinics In Developmental Medicine. M. C. O. Bax. London, Mac Keith Press. **No 151**: 1-7.

Stanley, F., Blair, E., and Alberman, E. (2000a). What are the cerebral palsies? Cerebral Palsies: Epidemiology and Causal Pathways. Clinics In Developmental Medicine. M. C. O. Bax. London, Mac Keith Press. **No 151**: 8-13.

Stanley, F., Blair, E., and Alberman, E. (2000b). The classification of the cerebral palsies. Cerebral Palsies: Epidemiology and Causal Pathways. Clinics In Developmental Medicine. M. C. O. Bax. London, Mac Keith Press. **No 151**: 14-21.

Stanley, F., Blair, E., and Alberman, E. (2000c). How common are the cerebral palsies? Cerebral Palsies: Epidemiology and Causal Pathways. Clinics In

Developmental Medicine. M. C. O. Bax. London, Mac keith Press. **No 151**: 22-39.

Stanley, F., Blair, E., and Alberman, E. (2000e). Causal pathways initiated preconceptionally or in early pregnancy. Cerebral Palsies: Epidemiology and Causal Pathways. Clinics In Developmental Medicine. M. C. O. Bax. London, Mac Keith Press. **No 151**: 48-59.

Steinbok, P., Reiner, A.M., Beauchamp, R., Armstrong, R.W., and Cochrane, D.D. (1997). "A randomised clinical trial to compare selective posterior rhizotomy plus physiotherapy with physiotherapy alone in children with spastic diplegic cerebral palsy." Developmental Medicine & Child Neurology **39**: 178-184.

Stempien, L. M., and Gaebler-Spira, D. (2000). Rehabilitation of children and adults with cerebral palsy. Physical Medicine and Rehabilitation. R. L. Braddom, W.B. Saunders Company.

Stoll, T., Huber, E., Seifert, B., Michel, B.A., and Stucki, G. (2000). "Maximal isometric muscle strength:normative values and gender-specific relation to age." Clinical Rheumatology **19**: 105-113.

Stuberg, A. W., Metcalf, K.W. (1988). "Reliability of quantitative muscle testing in healthy children and in children with Duchenne Muscular Dystrophy using a hand-held dynamometer." Physical Therapy **68**(6): 977-982.

Surman, G., Bonellie, S., Chalmers, J., Colver, A., Dolk, H., Hemming, K., King, A., Kurinczuk, J. J., Parkes, J., and Platt, M. J. (2006). "UKCP: a collaborative network of cerebral palsy registers in the United Kingdom." J Public Health (Oxf) **28**(2): 148-56.

Surman, G., Newdick, H., and Johnson, A. (2003). "Cerebral palsy rates among low-birthweight infants fell in the 1990's." Developmental Medicine & Child Neurology **45**(7): 456-462.

Sussman, M. D., Shriners Hospital for Crippled Children, Ed. (1992). The Diplegic Child. Evaluation and Management. "Symposium, Charlottesville, Virginia, Nov. 21-24, 1991". Rosemont, American Academy of Orthopaedic Surgeons.

Sutherland, D. (1997). "The development of mature gait." Gait & Posture **6**: 163-170.

Sutherland, D., Zilberfarb, J.L., Kaufman, K.R., Wyatt, M.P., and Chambers, H.G. (1997). "Psoas release at the pelvic brim in ambulatory patients with cerebral palsy: operative technique and functional outcome." Journal of Pediatric Orthopaedics **17**: 563-570.

Sutherland, D. H. (1984). Gait disorders in childhood and adolescence. Baltimore, Williams & Wilkins.

Sutherland, D. H., and Davids, J.R. (1993). "Common gait abnormalities of the knee in Cerebral Palsy." Clinical Orthopaedics and Related Research **288**(March): 139-147.

Sutherland, D. H., Zilberfarb, J.L., Kaufman, K.R., Wyatt, M.P., and Chambers, H.G. (1997). "Psoas release at the pelvic brim in ambulatory patients with cerebral palsy: operative technique and functional outcome." Journal of Pediatric Orthopaedics **17**: 563-570.

Tachdjian, M. O. (1994). Atlas of pediatric orthopaedic surgery. Philadelphia, PA, W.B. Saunders.

Tardieu, C., Colbeau-Justin, Lespargot, A. et al. (1976). "An apparatus and a method for measuring the relationship of triceps surae torques to tibio-trsal angles in man." European Journal of Applied Physiology **35**: 11-20.

Tardieu, C., Huet, E., Bret, M.D., Tardieu, G. (1982). "Muscle hypoextensibility in children with cerebral palsy: clinical and experimental observations." Archives of Physical Medicine & Rehabilitation **63**: 97-102.

Tardieu, G., Thuilleux, G., Tardieu, C., and Huet de la Tour, E. (1979). "Long-term effects of surgical elongation of the tendo calcaneus in the normal cat." Developmental Medicine & Child Neurology **21**: 83-94.

Taves, D. R. (1974). "Minimization: a new method of assigning patients to treatment and control groups." Clin Pharmacol Therap **15**: 443-453.

Taylor, N. F., Dodd, K.J., and Graham, H.K. (2004). "Test-retest reliability of hand-held dynamometric strength testing in young people with cerebral palsy." Archives of Physical Medicine & Rehabilitation **85**: 77-80.

Tenuta, J., Shelton, Y.A., and Miller, F. (1993). "Long-term follow-up of triple arthrodesis in patients with cerebral palsy." Journal of Pediatric Orthopaedics **13**: 713-716.

Trahan, J., and Malouin, F. (1999). "Changes in the gross motor function measure in children with different types of cerebral palsy: an eight-month follow-up study." Paediatric Phys Ther **2**: 12-17.

Treasure, T. a. M., K.D. (1998). "Minimisation: the platinum standard for trials?" British Medical Jounal **317**: 362-363.

Van den Berg-Emons, R. J. G., Van Baak, M.A., De Barbanson, D.C., Speth, L., and Saris, W.H.M. (1996). "Reliability of tests to determine peak aerobic power, anaerobic power and isokinetic muscle strength in children with spastic cerebral palsy." Developmental Medicine & Child Neurology **38**: 1117-1125.

Van der Linden, M. L., Aitchison, A.M., Hazlewood, M.E., Hillman, S.J., and Robb, J.E. (2004). "Test-retest repeatability of gluteus maximus strength testing using a fixed digital dynamometer in children with cerebral palsy." Archives of Physical Medicine & Rehabilitation **85**: 2058-2063.

Van der Ploeg, R. J. O., Fidler, V., and Oosterhuis, H.J.G.H. (1991). "Hand-held myometry: reference values." Journal of Neurology, Neurosurgery and Psychiatry **54**: 244-247.

Waters, R. L., Frazier, J., Garland, D.E. et al (1982). "Electromyografical gait analysis before and after operative treatment for hemiplegic equinus and

equinovarus deformity." Journal of Bone and Joint Surgery (Am) **64A**: 284-288.

Watt, J. M., Robertson, C.M.T., and Grace, M.G.A. (1989). "Early prognosis for ambulation of neonatal intensive care survivors with cerebral palsy." Developmental Medicine & Child Neurology **31**: 766-773.

Weltman, A., Tippet, S., Janney, C., Strand, K., Rians, C., Cahill, B.R., et al. (1988). "Measurement of isokinetic strength in prepubertal males." Journal of Orthopaedics Sports Physical Therapy **9**: 345-351.

WHO (1980). The International Classification of Impairments, Disabilities and Handicaps (ICIDH). Geneva, World Health Organization (WHO).

WHO (2001). International Classification of Functioning, Disability and Health (ICF). Geneva, World Health Organization.

Wiles, C. M., and Karni, Y. (1983). "The measurement of muscle strength in patients with peripheral neuromuscular disorders." Journal of Neurology, Neurosurgery and Psychiatry **46**: 1006-1013.

Wiley, M. E., and Damiano, D.L. (1998). "Lower-extremity strength profiles in spastic cerebral palsy." Developmental Medicine and Child Neurology **40**: 100-107.

Winett, R. A. and R. N. Carpinelli (2001). "Potential health-related benefits of resistance training." Prev Med **33**(5): 503-13.

Winters, T. F., Gage, J.R., and Hicks, R. (1987). "Gait patterns in spastic hemiplegia in children and young adults." The Journal of Bone and Joint Surgery **69-A**(3): 437-441.

Wood, E., and Rosenbaum, P. (2000). "The Gross Motor Function Classification System for Cerebral Palsy: a study of reliability and stability over time." Developmental Medicine & Child Neurology **42**: 292-96.

Wright, F. V., Sheil, E.M.H., Drake, J.M., Wedge, J.H., and Naumann, S. (1998). "Evaluation of selective dorsal rhizotomy for the reduction of spasticity in cerebral palsy: a randomized controlled trial." Developmental Medicine & Child Neurology **40**: 239-247.

Wright State University (2005 - access date 26/09/2006). Skeletal muscle architecture, http://www.cs.wright.edu/%7edkender/bme422/muscle.htm.

Yin, R., Reddihough, D.S., Ditchfield, M.R., and Collins, k.J. (2000). "Magnetic resonance imaging findings in cerebral palsy." J.Paediatri. Child Health **36**: 139-144.

Zafeiriou, D. I., Tsikoulas, I.G., and Kremenopoulos, G.M. (1995). "Prospective follow-up of primitive reflex profiles in high-risk infants: clues to an early diagnosis of cerebral palsy." Pediatric Neurology **13**(2): 148-152.

APPENDICES

APPENDIX ONE Terminology

Dyskinesia impairment of voluntary movements resulting in fragmented or jerky motions

Athetosis a nervous disorder that is marked by continual slow movements especially of the extremities and is usually due to a brain lesion

Dystonia a state of disordered tonicity of tissues (as of muscle)

Chorea any of various nervous disorders of infectious or organic origin marked by spasmodic movements of the limbs and facial muscles and by in-coordination

Ataxia an inability to coordinate muscle activity during voluntary movement; most often due to disorders of the cerebellum or the posterior columns of the spinal cord

Hypotonia a condition in which there is loss of muscular tonicity

Muscle tone the internal state of muscle fibre tension within individual muscle; degree of muscle tension or resistance during rest or in response to stretching

Primitive reflexes an involuntary reaction in response to a stimulus applied to the periphery and transmitted to the nervous centres of the brain or spinal cord which disappears after the first months of life

Developmental milestones the average age during which children will achieve certain skills

Muscle cross-sectional area product of the size and number of muscle fibres

Physiological muscle cross-sectional area is taken perpendicular to the muscle fibres

Anatomical muscle cross-sectional area is taken perpendicular to the long axis of the muscle

APPENDIX TWO

Related Conference Proceedings and Scientific Presentations

1. "Reliability of strength measurements in healthy children and children with cerebral palsy using a digital myometer"
M. Seniorou, N. Thompson, M.E. Harrington, J.A. Stebbins, T.N. Theologis.
ESMAC 2002 Conference, European Society for Movement Analysis in Adults and Children, Leuven, Belgium, Sept 19-21, Gait & Posture, vol. 16, suppl. 1, pp: S175-176

2. "Reliability of measuring muscle strength in children with spastic cerebral palsy and the influence of co-contraction"
Thompson N, **Seniorou M**, Harrington ME, Stebbins JA, Theologis TN.
14th International World Confederation for Physical Therapy Congress 2003, Barcelona, Spain, June 7-12, Abstract # RR – PL - 1855

3. "Effect of multi-level orthopaedic surgery on lower limb muscle strength in spastic diplegic cerebral palsy: preliminary results"
Maria Seniorou MD, Nicky Thompson MSc MSCP, Marian Harrington DPhil Ceng, Tim Theologis FRCS MSc PhD
The American Academy for Cerebral Palsy & Developmental Medicine Annual Meeting 2004, Los Angeles, CA, Sept 29 - Oct 2, Developmental Medicine and Child Neurology, Suppl No 99, Sept 2004, Vol 46 p 13 **Nominated for the 2004 Gayle G. Arnold Award for Excellence in the care of children with cerebral palsy**

4. "Early Effects of Multi-Level Orthopaedic Surgery on Gait Kinematics, Muscle Strength and Motor Function in Children with Cerebral Palsy" **Seniorou M**, Thompson N, Harrington ME, Theologis TN. ESMAC 2004 Conference, European Society for Movement Analysis in Adults and Children, Warsaw, Poland, Sept 23-25, Gait & Posture 2004, 20, S1, 95

5. "Outcome of strength training following multi-level orthopaedic surgery in children with spastic diplegic cerebral palsy"
Maria Seniorou MD, Nicky Thompson MSc MSCP, Marian Harrington DPhil Ceng, Tim Theologis FRCS MSc PhD
The American Academy for Cerebral Palsy & Developmental Medicine Annual Meeting 2005, Orlando, Florida, Sept 14 – 17, Developmental Medicine and Child Neurology, Suppl No 102, Sept 2005, Vol 47 p 3-4 (abstract) - **Nominated for the 2005 Gayle G. Arnold Award for Excellence in the care of children with cerebral palsy**

6. "The results of two strength training regimes on gait and function in cerebral palsy following multi-level surgery"
Thompson N, **Seniorou M**, Harrington M and Theologis TN
ESMAC 2005 Conference, European Society for Movement Analysis in Adults and Children, Barcelona, Spain, Sept 22-24, Gait & Posture ESMAC Abstracts 22S S2-S3 **Nominated for Best Paper Award**

7. "Recovery of muscle strength following multi-level orthopaedic surgery in diplegic cerebral palsy" **Maria Seniorou**, Nicky Thompson, Tim Theologis ESMAC 2006 Conference, European Society for Movement Analysis in Adults and Children, Amsterdam, The Netherlands, Sept 28-30, Gait & Posture, abstract available on line, **Best Paper Award**

8. "Recovery of muscle strength following multi-level orthopaedic surgery in diplegic cerebral palsy" **Maria Seniorou**, Nicky Thompson, Marian Elizabeth Harrington Tim Theologis. Gait & Posture, accepted 22nd July 2007, article in Press, available online www.sciencedirect.com

APPENDIX THREE

Appendix 3-i Pilot study related forms

1. Poster
2. Information sheet
3. Letter to parents
4. Child's information sheet
5. Consent form
6. Letter to doctor
7. Clinical examination form
8. Dynamometer and EMG data collection form
9. Dynamometer collection form – 2nd examiner

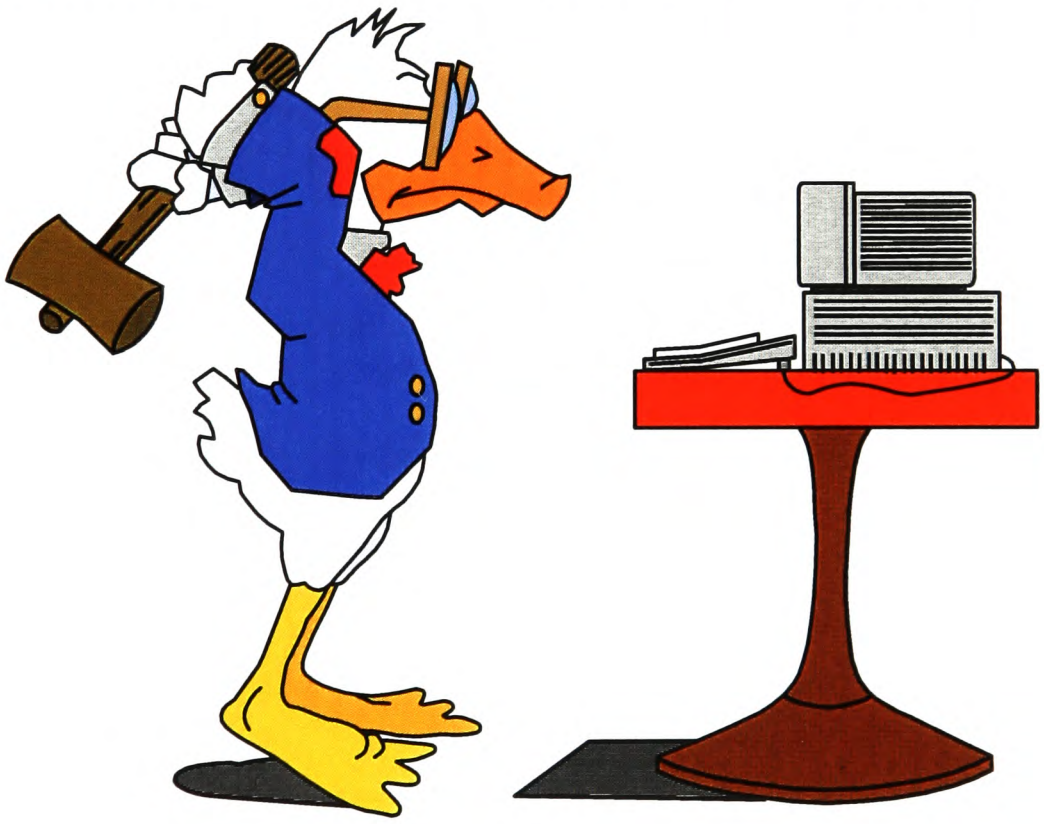
Nuffield Orthopaedic Centre NHS Trust

OxREC No: C01.139

DO YOU WANT TO FIND OUT HOW STRONG YOU ARE?

We are looking for healthy volunteers aged 5-16 years, to help us with research, which will lead to improvements in the management (surgical treatment and physiotherapy) in children who have cerebral palsy.

We would like to measure children's muscle strength using a hand-held device, at the Oxford Gait Laboratory.



If you would like to help us with this research or for more information please contact:

Mrs Nicky Thompson or Dr Marian Harrington
Oxford Gait Laboratory
Nuffield Orthopaedic Centre
Windmill Road
Headington
Oxford OX3 7LD
Tel: 01865 227609
Email: gait.lab@noc.anglox.nhs.uk

Nuffield NHS
Orthopaedic Centre
NHS Trust

Reliability of Strength Measurements in Children with Cerebral Palsy

Parents' Information Sheet

Introduction

We wish to establish whether it is possible to measure reliably muscle strength in children with Cerebral Palsy (CP). In recent years, muscle weakness has been recognised as an important factor which influences overall function in children with CP. At the Oxford Gait Laboratory we are trying to improve the understanding of muscle weakness in CP and to establish a reliable strength measurement protocol.

What is involved in taking part?

The study would involve bringing your child to the hospital on three occasions for strength testing in the Gait Laboratory.

Your child would see a Physiotherapist or a Doctor who would take some measurements of his/her legs. Your child would be asked to wear shorts and a T-shirt so sticky electrodes could be applied to the skin to measure muscle activity during testing. Strength measurements would then be undertaken using a hand-held device. Your child would be asked to push hard against the device for a few seconds; we would measure the strength of five muscles in each leg.

You would be asked to attend for an initial visit followed by two further visits in approximately 2 and 4 weeks time. Each visit would be likely to take about 30 minutes.

What are the possible risks of taking part?

The procedures are not harmful and carry no risks to health; they are used regularly in clinical practice. Your child would be allowed to take rests as necessary between measurements.

Are there any possible benefits?

We anticipate that the measurements your child would provide us with would benefit children with cerebral palsy by providing more detailed information on which to base these patients' management (surgical treatment and physiotherapy).

Does my child have to take part?

No, taking part is entirely voluntary. If you would prefer for your child not to take part or if he/she is not willing to participate, that is fine and you do not have to give any reason; if you initially agree to help us you would be free to change your mind at any time.

We may want to inform your child's GP that you are taking part, with your permission.

What do I do now?

If you have any questions, please contact either Mrs Nicky Thompson or Dr Marian Harrington at the Oxford Gait Laboratory on 01865 227609. Then if you are happy to participate in this study please complete and return the slip below to the Oxford Gait Laboratory and we will arrange a convenient time for you to take part.

Mrs Nicky Thompson MSc MCSP
Clinical Specialist in Gait Analysis

OxREC ref. C01.139

✂.....

Please, send me an appointment for Strength Testing related to the reliability assessment research project. I understand that agreement is subject to further information and my written consent at the time of this appointment.

Signed..... (Parent/Guardian)

Child's name:
Date of birth:
Contacttel:

Nuffield NHS
Orthopaedic Centre
NHS Trust

Oxford Gait Laboratory
Windmill Road
Headington
OXFORD
OX3 7LD

Tel: 01865 227609
Fax: 01865 744277
Email address:gait.lab@noc.anglox.nhs.uk

To the Parent or Guardian of:

Dear 's parent or guardian,

Re. Reliability of Hand-held Dynamometry Measurements in Children with Cerebral Palsy

We are currently undertaking a study on both healthy volunteers and children with cerebral palsy in order to improve the criteria for these children's management (surgical treatment and physiotherapy).

We would like to ask whether you would be willing for your child to take part in this study. This would involve three visits to the Oxford Gait Laboratory at the Nuffield Orthopaedic Centre where your child would have muscle strength measurements with a hand-held device.

Please, find enclosed an information leaflet on the study that explains what would be involved. If you are willing for your child to be involved in the study please return the slip at the bottom of the information sheet and we will arrange a convenient appointment for you. If you have any further questions, please contact me or Dr Marian Harrington at the Oxford Gait Laboratory on 01865 227609.

Yours sincerely,

Mrs Nicky Thompson MSc MCSP
Clinical Specialist in Gait Analysis

INVESTOR IN PEOPLE

Chairman Dr David Evered
Acting Chief Executive Mr. Ed Macalister-Smith

Nuffield NHS
Orthopaedic Centre
NHS Trust

Reliability of Strength Measurements in Children with Cerebral Palsy

CHILD'S INFORMATION SHEET

What do you want me to do?

We are asking you if you would like to help us in a research project to help us make decisions about treatment in children with cerebral palsy.
At this hospital we have a small machine to measure how strong your muscles are. We would ask you to push your legs hard against this machine that can show the force made by your muscles.

What will happen to me?

We would want to see you and your Mum or Dad at the hospital on three occasions.
Firstly, we would take some measurements of your legs when you are sitting or lying down. We would like you to wear shorts and a T-shirt so we can stick small electrodes to your skin with sticky tape. These would show how your muscles work during the strength measurements. We would like to test five muscles of both your legs. The measurements would take about half an hour and you would be able to rest between them.
The machine for testing your strength and the sticky electrodes are harmless and would not hurt in any way.

Can I change my mind?

Yes, you do not need to take part. If you want to help us now but you change your mind later on, it doesn't matter. You can stop joining in at any time you want. If you have any questions about what is happening to you then you can ask your Mum or Dad or the doctors or nurses. They will answer all your questions. It does not matter if you think your questions are silly – if you want to know anything you only have to ask.

OxREC No: C01.139

Nuffield NHS
Orthopaedic Centre
NHS Trust

COREC No: C01.139

RELIABILITY OF HAND-HELD DYNAMOMETRY MEASUREMENTS IN CHILDREN WITH CEREBRAL PALSY

CONSENT FORM

(The Parent/Guardian should complete this sheet)

Re: [ ]

Have you read the Information Leaflet? Yes/No

Have you had the opportunity to ask questions and discuss the study? Yes/No

Have you received satisfactory answers to your questions? Yes/No

Have you received enough information about the study? Yes/No

Who have you spoken to? Dr/Mr/Ms_____

Do you understand that you are free to leave the study?

- At any time
- Without having to give a reason
- Without affecting your child's medical care? Yes/No

Signed_____ Date:_____

Nuffield NHS
Orthopaedic Centre
NHS Trust

Oxford Gait Laboratory
Windmill Road
Headington
OXFORD
OX3 7LD

Tel: 01865 227609
Fax: 01865 744277
Email address:gait.lab@noc.anglox.nhs.uk

date

RELIABILITY OF HAND-HELD DYNAMOMETRY MEASUREMENTS IN CHILDREN WITH CEREBRAL PALSY

OxREC No: C01.139

Dear Dr

Re. Your patient_____

We are currently undertaking a study on both healthy volunteers and children with cerebral palsy which involves taking a clinical examination and muscle strength measurements of the lower limbs, with a hand-held dynamometer. This patient's parent/guardian has agreed to allow his/her child to take part in this study.

Please, find enclosed a copy of the Parent's Information Leaflet. The procedures involved are non-invasive and safe. Travelling costs are covered by the study and there are no expenses attributable to General Practitioners or Specialists.

Please, do not hesitate to contact me if you have any concerns or questions.

Yours sincerely,
Mrs Nicky Thompson MSc MCSP
Clinical Specialist in Gait Analysis
Oxford Gait Laboratory

INVESTOR IN PEOPLE

Chairman Dr David Evered
Acting Chief Executive Mr. Ed Macalister-Smith

Nuffield NHS
Orthopaedic Centre
NHS Trust

Oxford Gait Laboratory
Windmill Road
Headington
OXFORD
OX3 7LD
Tel: 01865 227609
Fax: 01865 227943
Email address:gait.lab@noc.anglox.nhs.uk

December 12th 2001

Dear Mr/Mrs .....,

Re. Reliability of Hand-held Dynamometry Measurements in Children with Cerebral Palsy

Thank you very much for agreeing to participate in this study.

Please, find enclosed an appointment card, which confirms the date and time of your appointments. If you have any further questions, please contact me or Dr Marian Harrington at the Oxford Gait Laboratory on 01865 227609.

Yours sincerely,

Mrs Nicky Thompson MSc MCSP
Clinical Specialist in Gait Analysis

INVESTOR IN PEOPLE

Chairman Dr David Evered
Chief Executive Mr. Ed Macalister-Smith

OXFORD GAIT LABORATORY
CLINICAL EXAMINATION

Reliability of hand – held dynamometry measurements in children with Cerebral Palsy
Ox REC No: C01.139

Name:				**DOB:**		
Gender:				**Assessor:**		
Date of 1st test:		**Date of 2nd test:**			**Date of 3rd test:**	
History – diagnosis – comments:						
	R.O.M			**STRENGTH**		**COMMENTS**
	R	**L**		**R**	**L**	
HIP:						
Flexion						
Extension (Thomas Test)						
Abduction (hip ext/knee flex)						
Abduction (hip ext/knee ext)						
Adduction						
Internal Rotation (prone)						
External Rotation (prone)						
Anteversion						
KNEE:						
Flexion						
Extension						
Popliteal angle						
SLR						
ANKLE:						
Dorsiflexion (knee flexed)						
Dorsiflexion (knee extended)						
Plantarflexion						
HINDFOOT:						
Inversion						
Eversion						
FOREFOOT:						
Supination						
Pronation						
Central balance:						
Handedness:						
Weight (kg):	Height (cm):					
Leg length (cm):			ASIS – Medial malleous, most distal point			
Thigh length (cm):			Greater trochanter – Knee joint line			
Shank length (cm):			Knee joint line – Lateral malleolus, most distal point			

OXFORD GAIT LABORATORY
CLINICAL EXAMINATION
Reliability of hand – held dynamometry measurements in children with Cerebral Palsy

	TONE (Modified Ashworth Scale)	
	R	**L**
Hip adductors (Gracilis test)		
Rectus Femoris (Duncan Ely test)		
Hamstrings		
Ankle plantarflexors - (Clonus)		

	SELECTIVE MOTOR CONTROL test	
HIP	**R**	**L**
Flexors		
KNEE		
Flexors		
Extensors		
ANKLE		
Dorsiflexors		

	DYNAMOMETER results 1st Visit						JOINT-STRAP DISTANCE		HOLE No	
KNEE	R MVC			L MVC			R	L	R	L
Flexors										
Extensors 90°										
Extensors 30°										
HIP										
Flexors										
Extensors										
Abductors										

EMG CHANNELS	AMP	MUSCLE GROUP	STIM
1			
2			
3			
4			
5			
6			
7			
8			
9			
10			
Earth location			

	DYNAMOMETER results 2nd Visit						JOINT-STRAP DISTANCE		HOLE No	
KNEE	R MVC			L MVC			R	L	R	L
Flexors										
Extensors 90°										
Extensors 30°										
HIP										
Flexors										
Extensors										
Abductors										

EMG CHANNELS	AMP	MUSCLE GROUP	STIM
1			
2			
3			
4			
5			
6			
7			
8			
9			
10			
Earth location			

	DYNAMOMETER results 3rd Visit						JOINT-STRAP DISTANCE		HOLE No	
KNEE	R MVC			L MVC			R	L	R	L
Flexors										
Extensors 90°										
Extensors 30°										
HIP										
Flexors										
Extensors										
Abductors										

EMG CHANNELS	AMP	MUSCLE GROUP	STIM
1			
2			
3			
4			
5			
6			
7			
8			
9			
10			
Earth location			

Guidelines to Standardised Clinical Examination

Muscle Strength

Muscle strength is scored in the available range of motion (ROM), using the Medical Research Council (MRC) scale:

0 No contraction
1 Flicker or trace of contraction
2 Active movement with gravity eliminated
3 Active movement against gravity
4 Active movement against gravity & light resistance
5 Normal strength

Use (-) with grades 2 & 3 to indicate motion is not full range

Tone / Spasticity

Tone is scored using the Modified Ashworth Scale:

0 No increase in muscle tone
1 Slight increase in muscle tone (catch and release or minimal resistance at the end of ROM)
2 Slight increase in muscle tone (catch followed by minimal resistance through out the remain of ROM)
3 Marked increase in tone through most of ROM
4 Considerable increase in tone, passive movement difficult
5 Affected part(s) rigid in flexion/extension

Selective Motor Control

0 No selective control, no (or minimal contraction of some of the demanded muscles)
0.5 Minimal contraction of the demanded muscles, almost no motion, and/or a lot of co-contraction
1 Mild selective control, limited range, co-contraction, no smooth motion, with co-contraction occasionally
1.5 Good co-contraction of correct muscles, some co-contraction, no perfect smooth motion, slightly limited range
2 Perfect control

Reliability of hand – held dynamometry measurements in children with Cerebral Palsy

Dynamometer Results
2nd Assessor

Name:		DOB:
CP/Normal:		Hospital No:
Assessor:		
Date of 1st test:	Date of 2nd test:	Date of 3rd test:

	DYNAMOMETER results						**JOINT-STRAP DISTANCE**	
KNEE	**R MVC**			**L MVC**			**R**	**L**
Flexors – 1st visit								
Extensors 90° - 2nd visit								
HIP								
Flexors – 3rd visit								

Comments:

Ox REC No: C01.139

Appendix 3-ii **Main study related forms**

1. Information sheet
2. Child's information sheet
3. Consent form
4. Child's consent form
5. Poster
6. Letter to parents
7. Letter to Doctor
8. Dynamometer data collection form
9. Gross Motor Function Measure form (GMFM)
10. Gillette's Functional Assessment Questionnaire (FAQ)

Nuffield NHS
Orthopaedic Centre
NHS Trust

Rehabilitation in Cerebral Palsy
Parents' Information Sheet

Introduction

You are being invited to take part in a research study. Before you decide, it is important for you to understand why the research is being done and what it would involve. Please take time to read the following information carefully and discuss it with friends, relatives and your GP if you wish. Ask us if there is anything that is not clear or if you would like more information. Take time to decide whether or not you wish to take part.
Thank you for reading this.

What is the purpose of this study

At the Nuffield Orthopaedic Centre we are trying to improve the understanding of muscle weakness in conservatively and surgically treated patients with Cerebral Palsy. We aim to examine the effect of different physiotherapy treatments on muscle strength and function, and whether muscle strength changes over time in CP. Currently there is no objective evidence of the effect of orthopaedic surgery on muscle weakness and the time period of recovery. We hope the results of this study will improve the effectiveness of physiotherapy and orthopaedic surgery in children with Cerebral Palsy.

What is involved in taking part?

The study would involve bringing your child to the hospital on one occasion in addition to the usual clinical appointments. Travel expenses would be reimbursed for this visit. It would also involve a few extra measurements being taken at routine gait analysis appointments.

This would involve your child seeing a Physiotherapist and/or Doctor who would take some measurements of his/her legs. Your child would be asked to perform simple tasks in order for their functional activities to be recorded. He/ She would be asked to wear shorts and a T-shirt so sticky markers could be applied to the skin. Cameras would then track these markers to record the walking pattern while walking up and down the gait lab several times. Strength measurements of the legs would then be undertaken using a hand-held device. Your child would be asked to push hard against the device for a few seconds; we would measure the strength of five muscles in each leg.

In those children undergoing surgery we would like to compare the effectiveness of two different post-operative physiotherapy programmes. If you choose to go ahead with surgery we would like to assign you to one or the other programme in addition to the routine post-operative physiotherapy. **The groups would be determined by chance in a computer, which has no information about the individual.** The additional physiotherapy programme would be at 6 months post-operatively, over a period of 6 weeks.

What are the possible risks of taking part?

The procedures are not harmful and carry no risks to health; they are used regularly in clinical practice.

All participants will continue with their conventional physiotherapy regime for the duration of the study, dictated by their clinical needs. A log will be kept of the frequency and content of physiotherapy sessions throughout the study. In the case of the additional post-operative

physiotherapy programmes, the protocol will be supervised by an experienced paediatric physiotherapist.

Are there any possible benefits?

We anticipate that the outcome of the study would benefit children with cerebral palsy by providing more detailed information on which to base these patients' management.

Does my child have to take part?

No, taking part is entirely voluntary. If you would prefer for your child not to take part or if he/she is not willing to participate, that is fine and you do not have to give any reason; if you initially agree to help us you would be free to change your mind at any time.
We would want to inform your child's GP that you are taking part, with your permission.

What do I do now?

If you have any questions, please contact either Mrs Nicky Thompson or Dr Maria Seniorou at the Oxford Gait Laboratory on 01865 227946 or 227609. Then if you are happy to participate in this study please complete and return the attached response card to the Oxford Gait Laboratory and we will arrange a convenient time for you to take part.

Mrs Nicky Thompson MSc MCSP
Clinical Specialist in Gait Analysis

OxREC No. C01.230

Nuffield NHS
Orthopaedic Centre
NHS Trust

REHABILITATION IN CEREBRAL PALSY

CHILD'S INFORMATION SHEET

What do you want me to do?

We are asking you if you would like to help us in a research project to help us make decisions about treatment in children with cerebral palsy.
At this hospital we have a laboratory with computers that are smart enough to show us how you walk. We would ask you to walk up and down for a few times, while our cameras and computers are watching you. There is also a small machine that measures how strong your muscles are. We would ask you to push your legs hard against this machine that can show the force made by your muscles.

What will happen to me?

We would want to see you and your Mum or Dad at the hospital.
Firstly, we would take some measurements of your legs when you are sitting or lying down. You would also show us how good you are at doing exercises such as rolling over or jumping. We would like you to wear shorts and a T-shirt so we can stick small markers to your skin with sticky tape. Some cameras would track these markers and our computer would show how your bones move during walking. We would also like to test five muscles of both your legs with a small machine, which measures strength. You would spend the morning or afternoon in our lab and you would be able to rest during that time. The machine for testing your strength and the sticky electrodes are harmless and would not hurt in any way.

Can I change my mind?

Yes, you do not need to take part. If you want to help us now but you change your mind later on, it doesn't matter. You can stop joining in at any time you want. If you have any questions about what is happening to you then you can ask your Mum or Dad or the doctors or nurses. They will answer all your questions. It does not matter if you think your questions are silly – if you want to know anything you only have to ask.

OxREC No: C01.230

Nuffield NHS
Orthopaedic Centre
NHS Trust

OxREC No: C01.230

REHABILITATION IN CEREBRAL PALSY

CONSENT FORM

(The Parent/Guardian should complete this sheet)

Re: _____

Have you read the Information Leaflet? Yes/No

Have you had the opportunity to ask questions and discuss the study? Yes/No

Have you received satisfactory answers to your questions? Yes/No

Have you received enough information about the study? Yes/No

Who have you spoken to? Dr/Mr/Ms_____

Do you understand that you are free to leave the study:

- At any time
- Without having to give a reason
- Without affecting your child's medical care? Yes/No

Signed: _____ Date: _____

Nuffield NHS
Orthopaedic Centre
NHS Trust

OxREC No: C01.230

REHABILITATION IN CEREBRAL PALSY

PARTICIPANT'S CONSENT FORM

(The Participant should complete this sheet)

Re: [ ]

Have you read the Information Leaflet? Yes/No

Have you had the opportunity to ask questions and discuss the study? Yes/No

Have you received satisfactory answers to your questions? Yes/No

Have you received enough information about the study? Yes/No

Who have you spoken to? Dr/Mr/Ms_____

Do you understand that you are free to leave the study:

- At any time
- Without having to give a reason
- Without affecting your medical care? Yes/No

Participant's signature: _____ Date: _____

Parent / Guardian's signature: _____

Nuffield NHS
Orthopaedic Centre
NHS Trust

OxREC No: C01.230

DO YOU WANT TO FIND OUT HOW YOUR BONES MOVE WHILE WALKING AND HOW STRONG YOUR MUSCLES ARE?

We are looking for volunteers with cerebral palsy/spastic diplegia aged 5-18 years, to help us with research, which will lead to improvements in their overall management (surgical treatment and physiotherapy).

We would like to perform a complete assessment of children's gait, strength and motor function at the Oxford Gait Laboratory.



If you would like to help us with this research or for more information please contact:

Mrs Nicky Thompson or Dr Marian Harrington
Oxford Gait Laboratory
Nuffield Orthopaedic Centre
Windmill Road
Headington
Oxford OX3 7LD

Tel: 01865 227609 - email: gait.lab@noc.anglox.nhs.uk

6

Nuffield NHS
Orthopaedic Centre
NHS Trust

Oxford Gait Laboratory
Windmill Road
Headington
OXFORD
OX3 7LD

Tel: 01865 227609
Fax: 01865 744277
Email address: gait.lab@noc.anglox.nhs.uk

Date

Address

Dear,

Re. REHABILITATION IN CEREBRAL PALSY - OxREC No C01.230

At the Oxford Gait Laboratory we are currently undertaking a study on children with cerebral palsy in order to improve the criteria for these children's management (surgical treatment and physiotherapy).

We would like to ask you whether you would be willing for …… to take part in this study. This would just involve one additional visit to the Oxford Gait Laboratory at the Nuffield Orthopaedic Centre, 6 months after surgery, where your child would have a complete assessment of her gait, muscle strength and motor function.

Please, find enclosed information leaflets on the study that explain its purpose. If you have any further questions, please contact me or Mrs Nicky Thompson at the Oxford Gait Laboratory on 01865 227946 or 227609.

Yours sincerely,

Dr Maria Seniorou

Enc.

Nuffield NHS
Orthopaedic Centre
NHS Trust

Oxford Gait Laboratory
Windmill Road
Headington
OXFORD
OX3 7LD

Tel: 01865 227609
Fax: 01865 744277
Email address:gait.lab@noc.anglox.nhs.uk

Date

REHABILITATION IN CEREBRAL PALSY: EVALUATION OF PHYSIOTHERAPY INTERVENTION AFTER MULTI-LEVEL ORTHOPAEDIC SURGERY
OxREC No. C01.230

Dear Dr

Re. Your patient

We are currently undertaking a study on children with cerebral palsy which involves taking a clinical examination, muscle strength measurements of the lower limbs, with a hand-held dynamometer, examination of gait with 3D Gait Analysis and assessment of motor function with the Gross Motor Function Measure (GMFM-88). This patient's parent/guardian has agreed to allow his/her child to take part in this study.

Please, find enclosed a copy of the Parent's Information Leaflet. The procedures involved are non-invasive and safe. Travelling costs are covered by the study and there are no expenses attributable to General Practitioners or Specialists.

Please, do not hesitate to contact me if you have any concerns or questions.

Yours sincerely,

Mrs Nicky Thompson MSc MCSP
Clinical Specialist in Gait Analysis
Oxford Gait Laboratory

INVESTOR IN PEOPLE

Chairman Dr David Evered
Acting Chief Executive Mr. Ed Macalister-Smith

OXFORD GAIT LABORATORY
MUSCLE STRENGTH ASSESSMENT
Rehabilitation in Cerebral Palsy

NAME: DOB: HOSP No:

Date:	**DYNAMOMETER results Baseline Assessment**						**JOINT-STRAP DISTANCE**		**HOLE No**	
KNEE	**R MVC**			**L MVC**			**R**	**L**	**R**	**L**
Flexors										
Extensors 90°										
Extensors 30°										
HIP										
Flexors										
Extensors										
Abductors										

Date:	**DYNAMOMETER results 6-month Assessment I**						**JOINT-STRAP DISTANCE**		**HOLE No**	
KNEE	**R MVC**			**L MVC**			**R**	**L**	**R**	**L**
Flexors										
Extensors 90°										
Extensors 30°										
HIP										
Flexors										
Extensors										
Abductors										

OxREC No: C01.230

Date:	DYNAMOMETER results 6-month Assessment II						JOINT-STRAP DISTANCE		HOLE No	
KNEE	R MVC			L MVC			R	L	R	L
Flexors										
Extensors 90°										
Extensors 30°										
HIP										
Flexors										
Extensors										
Abductors										

Date:	DYNAMOMETER results 12-month Assessment						JOINT-STRAP DISTANCE		HOLE No	
KNEE	R MVC			L MVC			R	L	R	L
Flexors										
Extensors 90°										
Extensors 30°										
HIP										
Flexors										
Extensors										
Abductors										

COMMENTS

OxREC No: C01.230

GROSS MOTOR FUNCTION MEASURE
GMFM

SCORE SHEET

Child's Name: _____ I.D. #: _____

Date of Birth: ____/____/____ (yy / mm / dd) Assessment date: ____/____/____ (yy / mm / dd)

Diagnosis: _____ Severity: ☐ ☐ ☐
Mild Moderate Severe

Evaluator's Name _____

Testing Conditions (e.g. room, clothing, time, others present)

_____

_____

_____

_____

The GMFM is a standardized observational instrument designed and validated to measure change in gross motor function over time in children with cerebral palsy.

*SCORING KEY	0	=	does not initiate
	1	=	initiates
	2	=	partially completes
	3	=	completes

*Unless otherwise specified, "initiates" is defined as completion of less than 10% of the item. "Partially completes" is defined as completion of 10% to less than 100%.

The scoring key is meant to be a general guideline. However, most of the items have specific descriptors for each score. It is imperative that the **guidelines be used for scoring each item.**

Contact address:
Dianne Russell, Gross Motor Measure Group, Chedoke-McMaster Hospitals, Chedoke Hospital, Building 74, Room 29, Box 2000, Station "A", Hamilton, Ontario L8N 3Z5

Children's Developmental Rehabilitation Programme at Chedoke-McMaster Hospitals, Hamilton, Ontario, Hugh MacMillan Rehabilitation Centre, Toronto, Ontario, and McMaster University, Hamilton, Ontario

©Gross Motor Measures Group, 1990. Revised September, 1993

(over)

Item C: **CRAWLING AND KNEELING**					**SCORE**	
38.	PR: CREEPS FORWARD 6'	0 ☐	1 ☐	2 ☐	3 ☐	38.
39.	4 POINT: MAINTAINS, WEIGHT ON HANDS AND KNEES, 10 SECONDS	0 ☐	1 ☐	2 ☐	3 ☐	39.
40.	4 POINT: ATTAINS SIT ARMS FREE	0 ☐	1 ☐	2 ☐	3 ☐	40.
41.	PR: ATTAINS 4 POINT, WEIGHT ON HANDS AND KNEES	0 ☐	1 ☐	2 ☐	3 ☐	41.
42.	4 POINT: REACHES FORWARD WITH R ARM, HAND ABOVE SHOULDER LEVEL	0 ☐	1 ☐	2 ☐	3 ☐	42.
43.	4 POINT: REACHES FORWARD WITH L ARM, HAND ABOVE SHOULDER LEVEL	0 ☐	1 ☐	2 ☐	3 ☐	43.
44.	4 POINT: CRAWLS OR HITCHES FORWARD 6'	0 ☐	1 ☐	2 ☐	3 ☐	44.
45.	4 POINT: CRAWLS RECIPROCALLY FORWARD 6'	0 ☐	1 ☐	2 ☐	3 ☐	45.
46.	4 POINT: CRAWLS UP 4 STEPS ON HANDS AND KNEES/FEET	0 ☐	1 ☐	2 ☐	3 ☐	46.
47.	4 POINT: CRAWLS BACKWARDS DOWN 4 STEPS ON HANDS AND KNEES/FEET	0 ☐	1 ☐	2 ☐	3 ☐	47.
48.	SIT ON MAT: ATTAINS HIGH KN USING ARMS, MAINTAINS, ARMS FREE, 10 SECONDS	0 ☐	1 ☐	2 ☐	3 ☐	48.
49.	HIGH KN: ATTAINS HALF KN ON R KNEE USING ARMS, MAINTAINS, ARMS FREE, 10 SECONDS	0 ☐	1 ☐	2 ☐	3 ☐	49.
50.	HIGH KN: ATTAINS HALF KN ON L KNEE USING ARMS, MAINTAINS, ARMS FREE, 10 SECONDS	0 ☐	1 ☐	2 ☐	3 ☐	50.
51.	HIGH KN: KN WALKS FORWARD 10 STEPS, ARMS FREE	0 ☐	1 ☐	2 ☐	3 ☐	51.

TOTAL DIMENSION C ☐

Item D: **STANDING**					**SCORE**	
52.	ON THE FLOOR: PULLS TO STD AT LARGE BENCH	0 ☐	1 ☐	2 ☐	3 ☐	52.
53.	STD: MAINTAINS, ARMS FREE, 3 SECONDS	0 ☐	1 ☐	2 ☐	3 ☐	53.
54.	STD: HOLDING ON TO LARGE BENCH WITH ONE HAND, LIFTS R FOOT, 3 SECONDS	0 ☐	1 ☐	2 ☐	3 ☐	54.
55.	STD: HOLDING ON TO LARGE BENCH WITH ONE HAND, LIFTS L FOOT, 3 SECONDS	0 ☐	1 ☐	2 ☐	3 ☐	55.
56.	STD: MAINTAINS, ARMS FREE, 20 SECONDS	0 ☐	1 ☐	2 ☐	3 ☐	56.
57.	STD: LIFTS L FOOT, ARMS FREE, 10 SECONDS	0 ☐	1 ☐	2 ☐	3 ☐	57.
58.	STD: LIFTS R FOOT, ARMS FREE, 10 SECONDS	0 ☐	1 ☐	2 ☐	3 ☐	58.
59.	SIT ON SMALL BENCH: ATTAINS STD WITHOUT USING ARMS	0 ☐	1 ☐	2 ☐	3 ☐	59.
60.	HIGH KN: ATTAINS STD THROUGH HALF KN ON R KNEE, WITHOUT USING ARMS	0 ☐	1 ☐	2 ☐	3 ☐	60.
61.	HIGH KN: ATTAINS STD THROUGH HALF KN ON L KNEE, WITHOUT USING ARMS	0 ☐	1 ☐	2 ☐	3 ☐	61.
62.	STD: LOWERS TO SIT ON FLOOR WITH CONTROL, ARMS FREE	0 ☐	1 ☐	2 ☐	3 ☐	62.
63.	STD: ATTAINS SQUAT, ARMS FREE	0 ☐	1 ☐	2 ☐	3 ☐	63.
64.	STD: PICKS UP OBJECT FROM FLOOR, ARMS FREE, RETURNS TO STAND	0 ☐	1 ☐	2 ☐	3 ☐	64.

TOTAL DIMENSION D ☐

GMFM

SUMMARY SCORE

DIMENSION	CALCULATION OF DIMENSION % SCORES	GOAL AREA (indicated with ✓ check)
A. Lying & Rolling	$\frac{\text{Total Dimension A}}{51} = \frac{\quad}{51} \times 100 = \quad\%$	A. ☐
B. Sitting	$\frac{\text{Total Dimension B}}{60} = \frac{\quad}{60} \times 100 = \quad\%$	B. ☐
C. Crawling & Kneeling	$\frac{\text{Total Dimension C}}{42} = \frac{\quad}{42} \times 100 = \quad\%$	C. ☐
D. Standing	$\frac{\text{Total Dimension D}}{39} = \frac{\quad}{39} \times 100 = \quad\%$	D. ☐
E. Walking, Running & Jumping	$\frac{\text{Total Dimension E}}{72} = \frac{\quad}{72} \times 100 = \quad\%$	E. ☐

TOTAL SCORE $= \frac{\%\,A + \%\,B + \%\,C + \%\,D + \%\,E}{\text{Total \# of Dimensions}}$

$= \frac{\quad + \quad + \quad + \quad + \quad}{5} = \frac{\quad}{5} = \quad\%$

GOAL TOTAL SCORE $= \frac{\text{Sum of \% scores for each dimension identified as a goal area}}{\text{\# Goal areas}}$

$= \quad = \quad\%$

©Gross Motor Measures Group, 1990. Revised September, 1993

Item	E. WALKING, RUNNING AND JUMPING	SCORE	
65.	STD, 2 HANDS ON LARGE BENCH: CRUISES 5 STEPS TO R	0 1 2 3	65.
66.	STD, 2 HANDS ON LARGE BENCH: CRUISES 5 STEPS TO L	0 1 2 3	66.
67.	STD, 2 HANDS HELD: WALKS FORWARD 10 STEPS	0 1 2 3	67.
68.	STD, 1 HAND HELD: WALKS FORWARD 10 STEPS	0 1 2 3	68.
69.	STD: WALKS FORWARD 10 STEPS	0 1 2 3	69.
70.	STD: WALKS FORWARD 10 STEPS, STOPS, TURNS 180°, RETURNS	0 1 2 3	70.
71.	STD: WALKS BACKWARD 10 STEPS	0 1 2 3	71.
72.	STD: WALKS FORWARD 10 STEPS, CARRYING A LARGE OBJECT WITH 2 HANDS	0 1 2 3	72.
73.	STD: WALKS FORWARD 10 CONSECUTIVE STEPS BETWEEN PARALLEL LINES 8'' APART	0 1 2 3	73.
74.	STD: WALKS FORWARD 10 CONSECUTIVE STEPS ON A STRAIGHT LINE ¾'' WIDE	0 1 2 3	74.
75.	STD: STEPS OVER STICK AT KNEE LEVEL, R FOOT LEADING	0 1 2 3	75.
76.	STD: STEPS OVER STICK AT KNEE LEVEL, L FOOT LEADING	0 1 2 3	76.
77.	STD: RUNS 15 FEET, STOPS & RETURNS	0 1 2 3	77.
78.	STD: KICKS BALL WITH R FOOT	0 1 2 3	78.
79.	STD: KICKS BALL WITH L FOOT	0 1 2 3	79.
80.	STD: JUMPS 12'' HIGH, BOTH FEET SIMULTANEOUSLY	0 1 2 3	80.
81.	STD: JUMPS FORWARD 12'', BOTH FEET SIMULTANEOUSLY	0 1 2 3	81.
82.	STD ON R FOOT: HOPS ON R FOOT 10 TIMES WITHIN A 24'' CIRCLE	0 1 2 3	82.
83.	STD ON L FOOT: HOPS ON L FOOT 10 TIMES WITHIN A 24'' CIRCLE	0 1 2 3	83.
84.	STD, HOLDING 1 RAIL: WALKS UP 4 STEPS, HOLDING 1 RAIL, ALTERNATING FEET	0 1 2 3	84.
85.	STD, HOLDING 1 RAIL: WALKS DOWN 4 STEPS, HOLDING 1 RAIL, ALTERNATING FEET	0 1 2 3	85.
86.	STD: WALKS UP 4 STEPS, ALTERNATING FEET	0 1 2 3	86.
87.	STD: WALKS DOWN 4 STEPS, ALTERNATING FEET	0 1 2 3	87.
88.	STD ON 6'' STEP: JUMPS OFF, BOTH FEET SIMULTANEOUSLY	0 1 2 3	88.

TOTAL DIMENSION E ☐

Was this assessment indicative of this child's ''regular'' performance? YES ☐ NO ☐

COMMENTS:

Nuffield Orthopaedic Centre NHS

NHS Trust

Functional Assessment Questionnaire.

1. Please choose the answer below that best describes your child's usual or typical walking abilities (with the use of assistive devices typically used). Choose only one answer. My child:

__ (1) Cannot take any steps at all.

__ (2) Can do some stepping on his/her own with the help of another person. Does not take full weight on feet; Does not walk on routine basis.

__ (3) Walks for exercise in therapy and/or less than typical household distances.

__ (4) Walks for household distances, but makes slow progress. Does not use walking at home as preferred mobility. (primarily walks in therapy or as exercise).

__ (5) Walks for household distances routinely at home and/or school. Indoor walking only.

__ (6) Walks more than 15-50 ft. outside the home but usually uses a wheelchair or stroller for community distances or in congested areas.

__ (7) Walks outside for community distances, but only on level surfaces (cannot perform curbs, uneven terrain, or stairs without assistance of another person).

__ (8) Walks outside the home for community distances, is able to get around on curbs and uneven terrain in addition to level surfaces, but usually requires minimal assistance or supervision for safety.

__ (9) Walks outside the home for community distances, easily gets around on level ground, curbs, and uneven terrain, but has difficulty or requires minimal assistance or supervision with running climbing and/or stairs.

__ (10) Walks, runs, and climbs on level and uneven terrain and does stairs without difficulty or assistance.

2. In your opinion, which of the following limits the patient's walking ability. Check as many as you think apply.

__ Pain
__ Weakness
__ Safety concerns
__ Balance
__ Mental ability, such as lack of concentration or awareness
__ Endurance (tolerance of activity for long periods)
__ Other reason, please describe:

_____

3. Please check all the things the patient is able to do, in addition to walking using their assistive device(s) if needed:

___ Walk carrying an object
___ Walk carrying a fragile object or glass of liquid
___ Walk up and down stairs using the railing
___ Walk up and down stairs without needing the railing
___ Steps up and down curb independently
___ Runs
___ Runs well including around a corner with good control
___ Can take steps backwards
___ Can maneuver in tight areas
___ Get on and off a bus by him/herself
___ Jump rope
___ Jumps off a single step
___ Hop on right foot
___ Hop on left foot
___ Step over an object, right foot first
___ Step over an object, left foot first
___ Kick a ball with right foot
___ Kick a ball with left foot
___ Ride 2 wheel bike
___ Ride 3 wheel bike
___ Ice skate or roller skate
___ Ride an escalator, can step on/off without physical help from another person

APPENDIX FOUR Study Protocols

1. History
2. Clinical Marker Placement
3. EMG Placement
4. Video Procedure
5. Gait Analysis data collection & processing protocol
6. Dynamometer data collection protocol

Clinical History Taking
Oxford Gait Laboratory Documentation

For a patient with Cerebral Palsy the following should be considered and relevant information summarised succinctly in the report:

History

Perinatal history

Developmental history, in particular age first walked

Medical and surgical history. Relevant/associated medical problems eg learning/speech/hearing problems, epilepsy. Note relevant medications eg anti-spasmodic drugs. List relevant surgical/treatment procedures and dates. Hip status from x-rays

Current ambulatory status. To include distance walked, walking aids required eg limited community ambulatory using a Kaye walker. Indication of functional ability eg participation in sports/PE

Current complaint/concerns. Record concerns of patient/family in relation to mobility, recent changes (improving/deteriorating) and possible reasons for change eg growth spurt/illness

Parental expectations

Orthoses. Current and previous types used and effect on mobility ie do they make a difference, if so, what? Compliance/pressure problems

Physiotherapy. Type/frequency/compliance of home and/or school programmme

Education. Mainstream/special needs plus any support required

'Plug In Gait' Marker Placement
Oxford Gait Laboratory Documentation

Where left side markers only are listed, the positioning is identical for the right side.

Refer to VCM form with link.

Lower Body
Standard clinical marker set markers shown in bold
17 markers in total (4 removed for dynamic trials)

LASI Left ASIS
RASI Right ASIS
Markers are placed directly over the anterior superior iliac spines. In obese patients it can be difficult to place markers accurately on the ASIS and excessive soft tissue can obscure the markers. In this case move each marker laterally by an equal amount along the LASIS-RASIS axis. If it is not possible to move markers laterally without also moving them antero-posteriorally the ant-post ASIS to trochanter distance should be recorded and input into the model. The true inter-ASIS distance must then be recorded and entered on the VCM form. These markers, together with the sacral marker or LPSI and RPSI markers, define the pelvic axes.

LPSI Left PSIS
RPSI Right PSIS

Two skin dimples lie over the sacroiliac joints (at the point where the spine joins the pelvis) A marker is placed on each PSIS which whose bony prominences can be palpated underneath the dimples.

SACR Sacral marker
May be used as an alternative to the LPSI and RPSI markers. Placed on the skin mid-way between the posterior superior iliac spines (PSIS).

If losing visibility of the sacral marker is a problem, a skin marker can be replaced by a 'wand marker' to extend the marker away from the body. In this case it must be positioned to lie in the plane formed by the ASIS and PSIS.

LKNE Left knee
The lateral epicondyle of the femur lies on the knee joint axis to a good approximation. A marker placed here will thus also lie on the axis. Palpate the lateral epicondyle, mark skin with pen and passively flex and extend the knee to confirm position ie the marked point should have minimal motion during passive knee flexion/extension. Place marker at this point.

LTHI Left thigh wand marker
The thigh markers are used to calculate the knee flexion axis location and orientation. Place the marker over the lower 1/3 lateral surface of the thigh, just below the swing of the hand, and at least two marker diameters proximal to the knee marker (the height is not critical).

The marker should be aligned in the plane that contains the hip and knee joint centers and the knee flexion/extension axis ie line the marker up with the knee marker and greater trochanter (this alignment is not critical since it can be adjusted in the software).

LANK Left ankle on malleolus
Placed on the most prominent point of the lateral malleolus along an imaginary line that passes through the transmalleolar axis.

LTIB Left tibial wand marker. This shank marker determines the alignment of the ankle flexion axis and is aligned by the software. Place the marker over the lower 1/3 lateral surface of shank approximately, in the plane that contains the knee and ankle joint centres.

LTOE Left toe
Placed between the distal ends of the second and third metatarsals.

LHEE Left heel (needed for static trial only)
The heel marker should be placed on the posterior aspect of the calcaneous, at the same height above the plantar surface of the foot as the toe marker. It should be aligned such that the line joining the heel and toe marker defines the progression of the foot in the transverse plane.This can be adjusted in the software if the subject is able to stand with their foot flat on the floor. lf the heel is not flat on the floor this distance will need to be carefully measured

LMMA Left medial malleolus (needed for static trial only)
Placed on the most prominent point of the medial malleolus along an imaginary line that passes through the transmalleolar axis.

RTHl, RKNE, RANK, RTlB, RTOE, RHEE & RMMA as for the Left.

Antrhopometric Measurements Required for Lower Body (to be entered on VCM form)

Height in standing (In the presence of significant hip and knee contractures measure 'true' height with tape measure in supine lying).
Weight (taken from force plates)
Leg Length – measure in supine lying the distance between the ASIS and the most prominent point of the medial malleolus, via the medial aspect of the knee joint. This method will take into account any contractures and is NOT therefore the shortest distance between the ASIS and medial malleoli, but rather the measure of the skeletal leg length.
The following width measurements using a width caliper should ideally be taken with the subject in standing:
Inter-ASIS distance - ASIS-ASIS distance, is the distance between the left ASlS and right ASlS.
Knee Width - The medio-lateral width of the knee across the line of the knee axis.
Ankle Width - The medio-lateral distance across the malleoli. Note: if measuring subject in AFOs this should be taken as the external distance including the AFOs.

Optional Subject Measurements for the Lower Body

(if not entered these are calculated by the model)
This measurement is only needed when markers cannot be placed directly on the ASIS, for example, in obese patients.ASIS-Trochanter Distance - ASIS-greater trochanter distance is the vertical distance, in the sagittal plane, between the ASIS and greater trochanter when the subject is lying supine. Measure this distance with the femur rotated such that the greater trochanter is positioned as lateral as possible.
Knee/Ankle Angle (Tibial Torsion) – angle between the knee and ankle axes in standing.

All measurements should be entered in centimetres.

EMG
Oxford Gait Laboratory Documentation

Clinically, in cerebral palsy patients, EMG data is most commonly recorded from the following muscle groups using surface electrodes:
Rectus Femoris
Medial Hamstrings
Gastrocnemius
Tibialis Anterior
We are also able to use fine-wire electrodes and most commonly record the activity of Tibialis Posterior using this method.

Procedure for use of Surface Electrodes

Electrode preparation

Prepare electrodes prior to patient arrival.

Put double-sided tape on amplifiers, and sticky electrode washers over each electrode. Apply the washers with the paper tab opposite the wire to enable the electrodes to be placed with the sticky tape overlapping. This gives an inter-electrode distance of 15mm.. To insert the electrode gel into the centre of the electrodes first put gel in syringe and push down the plunger to eliminate air. Carefully insert the gel into the electrode ensuring there are no air holes and the gel is proud of the edge for good skin contact. Use a fresh syringe of gel for each patient so it does not dry out.

Skin preparation

The skin is prepared prior to electrode application to get good electrode-skin contact and reduce skin impedance. Clean the skin by rubbing the area with a skin prep pad. If the area is covered with hair, shave patch using a razor. (If the patient has used body lotion this will affect the adhesiveness of the electrodes and the area will need to be washed with soap and water and dried thoroughly before using the prep pads).

Positioning the patient

Position of the patient will vary depending on the muscle group to be tested. The patient should be positioned so the muscle can be clearly identified by palpation/ manual muscle testing and the anatomical landmarks which help determine accurate electrode placement (see under individual muscle groups).

Electrode placement

Patient needs to wear appropriate size EMG jacket. Prior to positioning electrodes attach ' back-pack' unit to back of jacket and earth electrode to upper arm - hold in place with Coban self adhesive bandage.

General considerations affecting EMG placement:
Body/muscle size eg tall men may have high muscle belly.
Muscle atrophy/hypertrophy.

Previous surgeries/tendon transfers – check operating notes.

RECTUS FEMORIS	
Origin	Anterior inferior iliac spine and brim of the acetabulum
Insertion	Through the patellar tendon, on the tibial tubercle.
Function	Extension of the knee and flexion of the hip.
Patient position	Sitting on couch with the knee flexed to 90° to minimise other quadriceps muscles.
Electrode placement	Anterior aspect of the thigh, on a line halfway between the anterior superior iliac spine and the superior border of the patella.
Muscle test	Flex the hip and resist hip flexion
MEDIAL HAMSRTRINGS (Semitendinosis)	
Origin	Ischial tuberosity
Insertion	Medial condyle of the tibia
Function	Flexion and medial rotation of the knee. Also extends and assists in medial rotation of the hip.
Patient position	Standing. Ideal position is prone lying (see European recommendations) but this is usually not practical in CP children with motor difficulties.
Electrode placement	Posterior aspect of thigh on a line halfway between the ischial tuberosity and medial epicondyle of the tibia.
Muscle test	Resist knee flexion. (This may be difficult in a CP patient – alternatively palpate muscle during active knee flexion with foot still on floor . If this is not possible then palpate muscle by locating the tendon at the knee and moving proximally).
GASTROCNEMIUS (Lateral head)	
Origin	Lateral femoral condyle
Insertion	Posterior aspect of the calcaneous through the Achille's tendon
Function	Plantarflexion of the ankle and assists in knee flexion.
Patient position	Sitting on couch with knee flexed to 90°
Electrode placement	On the lateral muscle belly on a line a third of the way between the head of the fibula and the heel.
Muscle test	Resist plantaflexion of the foot with knee extended
TIBIALIS ANTERIOR	
Origin	Lateral condyle and proximal half of lateral surface of tibia, interosseus membrane, deep fascia and lateral intermuscular septum.
Insertion	Medial and plantar surface of medial cuneform bone, base of first metatrsal.
Function	Dorsiflexion of ankle and assists inversion of the forefoot
Patient position	Sitting on couch with knee at 90°
Electrode placement	On the anterior aspect of the of the shank on a line one third of the way between the head of the fibula and the medial malleolus, just lateral to the tibial crest.
Muscle test	Ideally resist dorsiflexion and inversion. This may be too difficult if there is poor selective control, in which case resisting dorsiflexion only is usually adequate.

*For placement of surface electrodes for other muscle groups refer to 'European Recommendations for Surface Electromyography', a publication of the SENIAM project, published by Roessingh Research and Development. ISBN 90-75452-15-2.

Connect EMG leads to back-pack unit. Leads are colour coded - carefully record which channels are used for which muscle using this colour code on VCM form: \Forms and Templates\VCM_form_MA300 with muscles.doc

Cleaning electrodes

It is important the electrodes are thoroughly cleaned after each use. Remove all traces of gel using cotton wool bud sticks and then clean with alcohol wipes.

VIDEO PROCEDURE
Oxford Gait Laboratory Documentation

Patient, parent or guardian must sign video consent form which will be filed in medical notes. Video Consent Form

The Gait Laboratory has two video cameras set up to record sagittal and coronal video pictures. Using any walking aids necessary, the usual procedure for most patients is:

BAREFOOT

Marker placement:
Record marker placement in standing after the static trial. Waist down only required - front, back and both side views. Ask patient to cross arms if possible to ensure all markers are visible.

After capturing 3D gait data and markers are removed, then record:

Standing posture:
Record full length standing - front, back and both side views. Also close up of feet, front and back views.

Sagittal plane:
Full length views, two walks each side ie 4 walks in all.
Panned full length view, 1 walk each side ie 2 walks in all.
Panned close up of feet, 1 walk each side ie 2 walks in all.

Coronal plane:
Full length views, two walks each of front and back views ie 4 walks in all.
Close up of feet, one walk front and back view ie 2 walks in all.

WITH ORTHOSES

Standing posture:
Record a/a

Sagittal plane
Full length views, two walks each side ie 4 walks in all.

Coronal plane
Full length views, two walks each of front and back views ie 4 walks in all.

NB
If the patients's walking ability is limited 3D gait data and video can be recorded simultaneously. Split screen video is also available.
If the patient is usually dependent on walking aids but can walk short distances independently this should also be recorded.

Gait Analysis Protocol

1. Prepare the lab:
    a. Lights
    b. Close curtain
    c. Video-camera (1&2)
    d. "Do not enter" sign
    e. Computer (log in: XXXX / go to **temp clinical data** from workstation and either find the patient No under **Cerebral Palsy** or create new patient and new session / the additional research session should always have the format: CxxxMS)
    f. Force pates (1&2 / switch on wall and boxes / **1 hour earlier / during weekdays they stay on**)
    g. Video and TV and mixer (prepare the tape, be careful not to overwrite previous session / prepare the blue screen – 2,4,5,6,8 on small box) / for the tape: find where you are by observing both the video and the sec, play, pause, when you are ready play and record together, just before recording hit again pause (now recording is starting).
    h. EMG (switch on wall and box from behind, check for green light, if red is on check if cable is connected) also it should be 1 = LRF, 2 = RRF, 3 = LH, 4 = RH etc.
    i. VICON 612
    j. Check system configuration for clinical default.car file
    k. Start link (from system)
2. Calibrate:
    a. L-frame on FP1 (with round screws on bottom)
    b. Go to workstation – system – live monitors – check each camera for all 4 markers or/and for reflections – correct them - calibrate cameras – choose Long wand 500 28 Nov 2001 – ok – start – stops automatically – you need to be in the right session for the calibration to begin
    c. Take off the L-frame – take the 2 ball wand and move it in the capture area trying to cover as much space as possible
    d. Camera 8 and 12 are a bit high (1,…) – check for reflections – use the black carpet and the yellow wedge to diminish them – accept the best one – sometimes the reflections are from other cameras – if a calibration is moderate accept it and try again. Only if you accept the next one the previous will be overwritten.
    e. Calibrate force plates – zero both FPs without putting anything on them – go system / calibrate analogue zero cameras and tick FP1 and FP2, but you have to be in the right session - then put L-frame on FP1 and weight on FP2 - go trial, capture, select FP test or Gait capture with movie to prepare for reboot if crashes – check for 4 markers and the weight, it should be < 100 N – if you selected Gait capture with movie check if movie capture works – repeat with L-frame on FP2 and weight on FP1. If a strange vector appears on any of the FPs, clean the edges with a card (small stones may be there and create this vector) – or leave it – FP 1 is tight and this why you have this vector.

You are ready to start

1. Depending on the patient you would need to define the sequence of capturing data (e.g very involved patients, in GRAFO's, barefoot, video first etc)
2. You need smaller trials so the processing can be quicker
3. Before you start check the analogue set-up and define subjects - new – (name).
4. In general:
    a. First go trial –Select Gait static – capture – start - check for all the markers – label them quickly (when you are familiar with the procedure) – Later you can use the static trial to calculate the BW if patient does not use crutches or walker (Fz1 or Fz2 N/ 9.81). If the patient uses crutches or sticks ask him/her to lift them if they can.
    b. Remember to name and keep notes of important trial features in workstation as you collect (there are some drop-down menus) e.g. FP1 test, static, in AFO's etc
    c. Ready for the EMG connection (remember to switch on the EMG box) - System analogue set-up: for EMG MA300 change the frequency to 1000 Hz and mark the relevant channels – check if all the others are on
    d. If you have time enter the subj. measurements – trial - subj measurements, yellow spaces, values from VCM form (select the lower body model) – later you may need to change the knee valgus/varus from Polygon.
    e. Select Gait capture with movie – remember to connect the EMG cable
    f. In order to check the first dynamic trial for markers you need to reconstruct first.
    g. Check the EMG signal and the markers with the clinician
    h. You aim for 6 good R and L strikes
    i. Remember the video – you need saggital R and L – frontal face and back – create appropriate blue screens e.g. for AFO's etc
    j. If walker you cannot capture kinetic data
    k. You disconnect the EMG if you have enough trials and you need some more strikes for kinetics (6 R&L).
    l. If the patient is able you record video after the collection of gait data, otherwise you do it at the same time
    m. In order to show to the patient a skeleton you go to the 1st trial, reconstruct – label – process (see details later)
    n. Check the movie from the beginning if it is the appropriate one; you may need help
    o. If the digital video crushes you need basically to reboot!

You have finished collecting

1. Copy raw data to a CD
2. Switch off everything if you are the last one collecting

Process the data

1. You need to prepare your PC for processing gait data:
    a. Install the latest VICON software (change to VlCON1 the old version)

b. Install the "Oxford Clinical Template MA300" and other files you will use (remember to update regularly the clinical template)
c. Install the baseline and 6-month post-op gait data from lab cds (remember that in archive disks you need to create first in workstation both the session and the session PG report; then copy all the files from both folders in the new folders that you created)- remember that in some cds the PG report doesn't open. Try to open it in another PC or laptop. In any case copy the data that you need.
d. Create appropriate database in workstation.
e. Name the raw data cd (a) and the processed cd (b) for each patient
f. Order cds through Helen NDOS

Ready to process

2. In workstation – batch process (remember space bar) – reconstruct (green P) and save trial (**check the defaults settings through options; for plug-in model with 13 markers these settings should be fine; the min cameras should be 3; tick ignore processed trials and tick unmark trials after processing)**
3. Open static trial – (**you do this only if you have not done already during the collection period, which is actually the best time to do it -** go trial – define subj – create new – trial – options – tick the new subj – trial) - attach marker set (saved under strength template folder) – label (first the sacr then L then R side and just hit down arrow) – **(go to trial – enter subject measurements, if you haven't put them in already)** – trial – pipeline – tick apply woltring filtering routine (check and tick in options for MSE value 20) – tick run static model but change in options 15mm instead of 25mm as marker diameter – tick there R and L if subject is plantigrade – tick save trial – process – check the "trousers" for correct R and L, if they look "strange" you probably labeled them incorrectly – close static trial
4. In workstation again, open dynamic trial (**for the dynamic model it's better to start with a trial that has good R and L strikes – you need first to check your movies)** – go trial – label - create auto-label calibration – tick create subj C3D file – select the subj – then go trial – pipeline – you may not need to fill gaps - tick apply woltring filtering routine (check in options for MSE value 20) – detect events – autocorrelate events – generate events - run dynamic model, but first check options – save trial – the "trousers" are produced – check again for R and L (in case of mistake just reconstruct and label again)– define events (or when you learn the procedure you can use the detect gait cycle events, autocorrelate and generate… for quicker processing)
5. After the first semi-processed dynamic trial there are still things to do!
    a. You need to check the thigh off-set and the shank off-set
    b. Go to Polygon – create a **check graphs report (saved under C:/temp)** and import this semi-processed trial. You can also import the normal template (female or male from the same folder). **Check the knee valgus/varus wave** – you need then to "fiddle" a little with the "thigh rotation off-set" values in subj measurements in workstation, taking into account any true valgus / varus that may exist. Remember that after any changes in workstation you need to run again the dynamic model and of course update in Polygon in order to see the

changes on the check-graphs (remember what Vicon calls tibial torsion is what we call in the PG report knee angle axis).

c. Once you've done this and you are happy with the output, you are ready to correct the shank off-set (this programme substitutes the lack of the knee alignment device).

d. Open the **static** trial again and run the **dynamic** model to apply the settings in your static one. Then perform the static bodylanguage modeling – from options browse for offset – bring the shank offset – and from subject measurements put 1.5 cm in marker diameter (this will appear automatically once you have selected the bodylanguage modeling) – tick also save trial – process now – run static model again. Finally! Run dynamic model in dynamic trials again.

e. How many? Until you are familiar with the procedure, identify from the movies (workstation) the trials with good R and L strikes. Process these first and import them into Polygon. If you need more kinematics process and import some more.

f. If you miss a marker in some frames start by changing the reconstruction parameters (No of cameras, intersection limit etc), reconstruct and check. It may work, so you proceed normally and delete (edit) the unlabeled trajectories (ghosts). Otherwise you may need to fill gaps (when you do that, do not save, first check). If you have many gaps, the precision is diminished so you should avoid this solution. You may need to just use another trial. (Reasons for gaps: calibration, hands, bad markers etc).

g. If you have markers ghosts then the autolabel doesn't work. You can either label manually twice in different frames, or change the reconstruction parameters or fill gaps (last option, be careful).

6. Go to Polygon:

a. Create a new report (for clinical-research purposes Cxxx PG report)

b. Base your report on the main template MA300. This will be modified according to what you want to check.

c. Import the trials that you thing are the best – **do not save anything until you are happy with the trials that you have** – instead of deleting links you can just rename them (e.g barefoot links if you tested in AFO's only) – after deciding the final format you can delete hyperlinks that you do not need – When you enter these 6 trials you go first to the consistency graphs and check them one by one by using the time bar. When you find the most representative cycles, select them. Remember that for the example kinematic and kinetic data you need basically one cycle, but for the EMG you need at least three.

d. In order to compare with previous visits you will need to import the trials that were analyzed previously in the new report – you cannot put together more than one reports

e. For the temporal parameters you go data – average – select the trials you want – hit ok – a new node will be created. Open it and click on the appropriate parameters after you have prepared the table. For temporary parameters from previous visits you just copy and paste (from the relevant trial).

f. For the EMG you need to go back to workstation and create a copy of the trials that you will use for EMG (green P – right click – set status -

adjusted). Then you click this new green P from eclipse in Polygon and you place it to the appropriate EMG placeholder. Then you can modify your graphs – Remember "events" because you may need to use trials that are not processed. You need to go back to workstation and label or auto-label and add events there. If when you go back to the PG report, you miss an event, check where this/these event/s can reasonably go and add it/them.

g. For the preordered angles link you need to go back to workstation. Choose one L and R example trial and open it, pipeline and tick dynamic bodylanguage modeling, import Belfast pelvis model and process. (This change doesn't show in workstation so you need to note the trial that you used). Go back to PG and update; the graphs will be updated too.
h. One of the pre-op or previous links doesn't work
i. Walking speeds will appear automatically when you use the barefoot links
j. For the consistency graphs you need to select a cycle for every trial that is on the graph. Sometimes a good kinematic cycle is not the one that has also kinetics. It's up to you to decide.
k. If the "pre-op" or "previous" links don't work, just go to the relevant trials and "add variables" (with right click) to the graphs – first remember to change from the preferences the thickness (bold is old!) and the colour of the graphs
l. When you finish with kinematics, kinetics (if you have), EMGs, temporal parameters and movies you store the file in a CD
m. For presentation you will need to prepare a report in word and have the cd ready for the laptop or preferably put the file in the network
n. When you finalize the parameters that are going to be analyzed you need to modify further the template MA300 and name it of course differently so it can contain myometer data and GMFM results in word document
o. Remember to store everything under each patient's file in RESEARCH DATABASE and keep printouts of important documents in their paper files. Back up.
p. Import GSD file into Polygon
q. Check for trials that were originally used (check the VCM form or the medical notes)
r. Do not worry about not corresponding events to movie
s. Shift + T, shift + L for looking EMGs in workstation

7. Additional comments
    a. PG report: no decimal places in thigh off-set and knee/ankle angle.
    b. When you import data for comparison state code, date and relevant info (barefoot etc)
    c. Put all the details of processing in Eclipse. In PG output page correct the header (and in graphs), and put details of trials that you used for comparison.
    d. When the report is checked (MH or NMA) possible changes should be given back to MS

e. After you processed the data, wrote the letter and completed the PG output, you should
    - Copy the file to network
    - Change the files from read-only
    - Give to SW consent form, VCM, and clinexam (keep copies)
    - Remember: letter for hemiplegics and past diplegics, full report for new diplegics
    - Remember when processing: EMG scales, delete unused placeholders, make sure one gait cycle is selected, include patients' details in temporal parameter page, the directory structure should be Cxxx (CxxxA for data and CxxxA PG report)
    - After copying the data to network remember to delete name from your database.

Collection of Dynamometer data

- Oxford Gait Laboratory laptop
- Use individual password
- Click "Matlab" icon on desktop (or go to "programs")
- Check for directory C:Matlab6p1/work
- All data is stored there. Transfer to MS computer
- Check that you are in the right folder:
    - dir
    - pwd
    - ans

To run the programme

- testdaq
- hit enter to collect
- save (yes/no)
- return / up arrow

Remember to watch the examiner and co-ordinate

Zero transducer only if examiner takes the weight of it

Press hold

Wait for examiner to say "1 2 3 go…"

Press enter with 1

APPENDIX FIVE

Word files

1.Tables of missing/excluded strength values

Excel files

Pilot study

2. Maximum moments for all CP subjects/visits/muscle groups/examiners

3.Maximum moments for all Normal subjects/visits/muscle groups/examiners

4. Normalised mean peak moments for all subjects and muscle groups

5. Normalised EMG data for CP subjects

6. Normalised EMG data for Normal subjects

7. % co-contraction index for all subjects (Right and Left side)

8. Within-subject SD, repeatability and coefficient of variation for all subjects. Data from all raters

Main study

9. Normalised strength data from all visits for groups B & C. % differences between visits

10. Normalised temporal parameters for all visits and all groups (A,B&C)

11. Overall physiotherapy questionnaire results

12. Log of completed physiotherapy sessions

13. Minimisation file

14. Group A GMFM results between visits

15.Graphical representation of strength, function and temporal overall data for all visits and study groups (A,B&C)

FILE 1

Table 1. Excluded strength values per muscle group, side and visit for all subjects (for all reasons)

subj/mg	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	count
RHF1																					20
LHF1														*							19
RHE1																					20
LHE1														*							19
RHA1															*						19
LHA1														*	*						18
RKF1																				*	19
LKF1														*						*	18
RKE1																					20
LKE1														*							19
RKE301							*	*		*	*		*	*	*	*				*	11
LKE301							*	*		*	*		*	*	*					*	12
RHF2																					20
LHF2														*							19
RHE2																					20
LHE2														*							19
RHA2															*						19
LHA2														*	*						18
RKF2																					20
LKF2														*							19
RKE2																					20
LKE2														*							19
RKE302				*									*	*	*						16
LKE302				*									*	*	*						16
RHF3																			*		19
LHF3														*					*		18

OP 24

RHE3																			*		19
LHE3														*					*		18
RHA3															*				*		18
LHA3														*	*				*		17
RKF3																			*		19
LKF3														*					*		18
RKE3																			*		19
LKE3														*					*		18
s/m	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**	**12**	**13**	**14**	**15**	**16**	**17**	**18**	**19**	**20**	**count**
RKE303													*	*	*				*		16
LKE303													*	*	*				*		16
RHF4																					20
LHF4														*							19
RHE4																					20
LHE4														*							19
RHA4														*	*						18
LHA4														*	*						18
RKF4																					20
LKF4														*							19
RKE4																					20
LKE4														*							19
RKE304													*	*	*	*					16
LKE304													*	*	*						17

Table 2. Reasons for excluded / missing strength values

Subject	Missing/excluded strength values	Reasons
1 LF		
2 AW		
3 HM		
4 AH	R&LKE30 (2)	Had to leave for Germany
5 HA		
6 CM		
7 CM	R&LKE30 (1)	Knee flexors contractures > 30
8 ML	R&LKE30 (1)	Knee flexors contractures > 30
9 YJ		
10 OL	R&LKE30 (1)	Technical problem (dynamometer)
11 QS	R&LKE30 (1)	Knee flexors contractures > 30
12 SC		
13 KM	R&LKE30 (1)(2)(3)(4)	Limited selective motor control
14 OS	RKE30(1)(2)(3)(4), RHA(4), All (L) values	L values *excluded* since R side only was operated on. RKE30 & RHA *missing* due to contractures, quads lag or limited motor control
15 BB	R&LKE30 (1)(2)(3)(4), R&LHA (1)(2)(3)(4)	Knee flexors contr >30, residual contr. & quads lag (KE30), limited selective control (HABD)
16 SVB	RKE30 (1)(4)	Limited motor control (1), Contracture (4)
17 ES		
18 EH		
19 DB	All for visit 3	DNA
20 EC	R&LKF(1), R&LKE30(1)	Technical problem (dynamometer)

Number in parenthesis denotes visit

FILE 2

MAX MOMENTS FOR ALL CP SUBJECTS/MUSCLE GROUPS/EXAMINERS

TABLE 1						
HF MAX MOMENT	PSCP01	PSCP02	PSCP03	PSCP04	PSCP05	
RHF 1	38.22	13.75	12.6	19.58	18.24	
RHF 2	46.02	14.25	15	18.48	21.78	
RHF 3	33	17.43	21.06	18.7	25.3	
LHF 1	52.75	16	19.14	20.46	15.96	
LHF 2	30.52	15	18	17.82	19.8	
LHF 3	28.25	15.75	18.4	20.93	25.08	
TABLE 2						
HE MAX MOMENT	PSCP01	PSCP02	PSCP03	PSCP04	PSCP05	
RHE 1	135.72	66	77.84	81.88	81.51	
RHE 2	117	79	91	76.56	68.86	
RHE 3	107.5	72.03	90	71.94	61.32	
LHE 1	106.12	58.25	95.12	61.6	56.07	
LHE 2	108.08	69	88.08	43.34	65.78	
LHE 3	119.52	73.92	97.52	43.93	78.32	
TABLE 3						
HA MAX MOMENT	PSCP01	PSCP02	PSCP03	PSCP04	PSCP05	
RHA 1	37.18	31	22.96	27.14	26.98	
RHA 2	59.7	26.75	27	28.6	23.1	
RHA 3	51.25	21.21	20.5	27.72	24.36	
LHA 1	41.44	19.25	29.58	30.36	29.4	
LHA 2	59.36	22	24.96	28.82	32.56	
LHA 3	43.92	19.95	22.31	31.05	29.48	
TABLE 4						
KF MAX MOMENT	PSCP01	PSCP02	PSCP03	PSCP04	PSCP05	
RKF 1		30.51	46.72	34.56	35.5	
RKF 2	43.96	27.81	38.34	41.31	33.75	
RKF 3	46.76	29.7	43.68	48.88		
LKF 1	55.5	17.55	31.2	37.2	25.44	
LKF 2	51.33	20.8	32.19	43.21	42.75	
LKF 3	48.16	22.14	32.76	43.68		
TABLE 5						
KE 90 MAX MNT	PSCP01	PSCP02	PSCP03	PSCP04	PSCP05	
RKE 1		28.89	68.8	42.25	38.75	
RKE 2	76.44	36.4	71.92	47.58	26	
RKE 3	76.5	31.05	57.96	54.6		
LKE 1	79.5	22.14	64.2	32.4	37.44	
LKE 2	80.33	36.96	49.01	34.8	42.75	
LKE 3	63	27.27	49	37.7		
TABLE 6						
KE 30 MAX MNT	PSCP01	PSCP02	PSCP03	PSCP04	PSCP05	
RKE 1		15.66	13.44		21	
RKE 2	42.84	15.96	12.04	35	17.5	
RKE 3	63.6	22.96	11.76	25.22		
LKE 1		16.2	13.5		23.75	
LKE 2	45.82	14.3	18.27	24.65	23.75	
LKE 3	43.12	18.63	12.88	17.94		

TABLE 7	Examiners	NT & TA			
HF MAX MOMENT	PSCP01	PSCP02	PSCP03	PSCP04	PSCP05
RHF 1					
RHF 2	49.25				
RHF 3				20.24	29.89
LHF 1					
LHF 2	30.82				
LHF 3				24.72	26.4
TABLE 8		COMBINED VALUES	FROM ALL	EXAMINERS	
HF MAX MOMENT	PSCP01	PSCP02	PSCP03	PSCP04	PSCP05
RHF MS	46.02			18.7	25.3
RHF NT	49.25				29.89
RHF TA				20.24	
LHF MS	30.52			20.93	25.08
LHF NT	30.82				26.4
LHF TA				24.72	

TABLE 9	Examiners	NT & TA			
KF MAX MNT	PSCP01	PSCP02	PSCP03	PSCP04	PSCP05
RKF 1		30.24	46.08		
RKF 2					
RKF 3				43.68	31.44
LKF 1	53.7	17.82	34.2		
LKF 2					
LKF 3				40.56	28.32

TABLE 10	Examiners	NT & TA			
KE 90 MAX MNT	PSCP01	PSCP02	PSCP03	PSCP04	PSCP05
RKE 1					
RKE 2		33.75	77.72		
RKE 3				55.16	
LKE 1					
LKE 2	72.8	32.48	59.74		
LKE 3				41.31	39.01

TABLE 11	COMBINED VALUES	FROM ALL	EXAMINERS		
KF Max mnt	PSCP01	PSCP02	PSCP03	PSCP04	PSCP05
RKF MS		30.51	46.72	48.88	30
RKF NT		30.24	46.08		31.44
RKF TA				43.68	
LKF MS	55.5	17.55	31.2	43.68	
LKF NT	53.7	17.82	34.2		28.32
LKF TA				40.56	

TABLE 12	COMBINED VALUES	FROM ALL	EXAMINERS		
KE 90 Max MNT	PSCP01	PSCP02	PSCP03	PSCP04	PSCP05
RKE MS		36.4	71.92	54.6	
RKE NT		33.75	77.72		
RKE TA				55.16	
LKE MS	80.33	36.96	49.01	37.7	35.52
LKE NT	72.8	32.48	59.74		39.01
LKE TA				41.31	

FILE 3

MAX MOMENTS FOR ALL N SUBJECTS/MUSCLE GROUPS/EXAMINERS

TABLE 1						
HF MAX MOMENT	PSN01	PSN02	PSN03	PSN04	PSN05	
RHF 1	68.68	71.3	50.16	14	35.5	
RHF 2	85.75	116.87	52.11	10.62	32.16	
RHF 3	65.1	78.03	49.88	11	43.79	
LHF 1	76.5	72.85	50.82	11.55	32.5	
LHF 2	75.6	84.32	59.1	8.6	27.6	
LHF 3	77.7	67.2	59.92	12.2	38.61	
TABLE 2						
HE MAX MOMENT	PSN01	PSN02	PSN03	PSN04	PSN05	
RHE 1	185.3	143.84	140.8	35.8	92.25	
RHE 2	187.95	198.09	130.68	16.56	84.92	
RHE 3	201.25	171.18	117.74	28	96.57	
LHE 1	153.34	150.04	135.3	28.14	82.94	
LHE 2	158.55	190.03	133.2	31.4	75.12	
LHE 3	201.25	187.04	115.36	28	90.45	
TABLE 3						
HA MAX MOMENT	PSN01	PSN02	PSN03	PSN04	PSN05	
RHA 1	57.12	95.48	60.8	13.2	33.75	
RHA 2	88.2	106.95	63.99	9.36	26.88	
RHA 3	82.25	89.1	62.93	11	39.15	
LHA 1	41.82	97.03	69.225	10.5		
LHA 2		107.26	70.8	10.6	34.32	
LHA 3	74.2	86.52	56.28	14.4	50.17	
TABLE 4						
KF MAX MOMENT	PSN01	PSN02	PSN03	PSN04	PSN05	
RKF 1	70.92	99.62	57.33	18.48	35.67	
RKF 2	68.95	126.72	50.75	9.87	46.11	
RKF 3	86.45	125.28	50.88	13.23	46.4	
LKF 1	100.08	87.12	54.25	15.96	39.76	
LKF 2	79.45	122.5	57.9	7.2	40.31	
LKF 3	82.25	90.75	55.04	12.6	37.7	
TABLE 5						
KE 90 MAX MNT	PSN01	PSN02	PSN03	PSN04	PSN05	
RKE 1	109.44	125.12	78.435	19.11	64.96	
RKE 2	110.6	136.44	82.07	13.44	63.8	
RKE 3	147.7	144.72	83.39	14.52	67.5	
LKE 1	130.68	106.92	77.5	20.58	53.76	
LKE 2	105.35	113.75	86.8	19.2	56.55	
LKE 3	116.2	128.7	80.32	20.37	57.68	
TABLE 6						
KE 30 MAX MNT	PSN01	PSN02	PSN03	PSN04	PSN05	
RKE 1	95.4	105.4	64.575	11.96	36.83	
RKE 2	119.35	99.72	55.39	16.33	35.38	
RKE 3	119.35	108	62.31	11.88	36.9	
LKE 1	99.36	111.54	63.55	13.44	43.96	
LKE 2	102.55	113.05	69.44	10.8	37.12	
LKE 3	114.1	92.73	62.08	13.11	35	

TABLE 7	Examiners	NT & TA			
HF MAX MOMENT	PSN01	PSN02	PSN03	PSN04	PSN05
RHF 1					
RHF 2					
RHF 3	72.1	88.56	42.93	10.725	31.05
LHF 1					
LHF 2					
LHF 3	73.15	63.56	44.24	11.1	31.5
TABLE 8	COMBINED VALUES		FROM ALL	EXAMINERS	
HF MAX MOMENT	PSN01	PSN02	PSN03	PSN04	PSN05
RHF MS	65.1	78.03	49.88	11	43.79
RHF NT	72.1	88.56	42.93	10.725	
RHF TA					31.05
LHF MS	77.7	67.2	59.92	12.2	38.61
LHF NT	73.15	63.56	44.24	11.1	
LHF TA					31.5

TABLE 9	Examiners	NT & TA			
KF MAX MOMENT	PSN01	PSN02	PSN03	PSN04	PSN05
RKF 1	80.15	108.46	63.63	20.425	39.44
RKF 2					
RKF 3					
LKF 1		97.02	48.67	16.34	39.44
LKF 2					
LKF 3					

TABLE 10	Examiners	NT & TA			
KE 90 MAX MNT	PSN01	PSN02	PSN03	PSN04	PSN05
RKE 1					
RKE 2	120.75	145.86	82.2	17.6	50.4
RKE 3					
LKE 1					
LKE 2		122.5	74.1	15.265	46.98
LKE 3					

TABLE 11	COMBINED VALUES	FROM ALL	EXAMINERS		
KF Max mnt	PSN01	PSN02	PSN03	PSN04	PSN05
RKF MS	70.92	99.62	57.33	18.48	35.67
RKF NT	80.15	108.46	63.63	20.425	
RKF TA					39.44
LKF MS		87.12	54.25	15.96	39.76
LKF NT		97.02	48.67	16.34	
LKF TA					39.44

TABLE 12	COMBINED VALUES	FROM ALL	EXAMINERS		
KE 90 Max MNT	PSN01	PSN02	PSN03	PSN04	PSN05
RKE MS	110.6	136.44	82.07	13.44	63.8
RKE NT	120.75	145.86	82.2	17.6	
RKE TA					50.4
LKE MS		113.75	86.8	19.2	56.55
LKE NT		122.5	74.1	15.265	
LKE TA					46.98

FILE 4

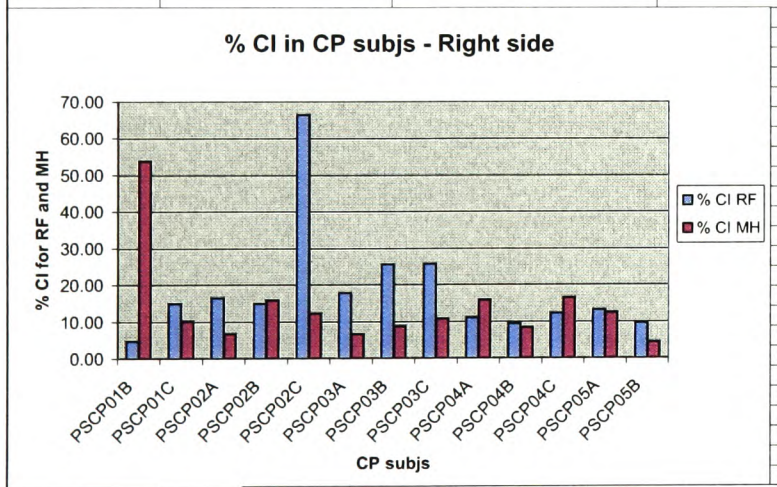
NORMALISED MEAN PEAK MOMENTS FOR ALL SUBJECTS AND MUSCLE GROUPS

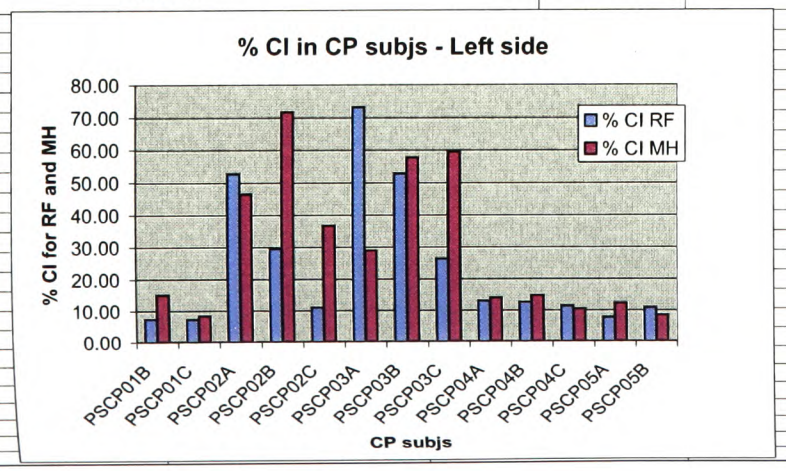
	Spastic Diplegia	Healthy	Spastic Diplegia	Healthy
Muscle group (R side)	**Mean peak moment Nm (SD)**	**Mean peak moment Nm (SD)**	**Relative mean peak moment Nm/kg (SD)**	**Relative mean peak moment Nm/kg (SD)**
Hip flexors	22.23 (9.64)	52.33 (29.79)	0.63 (0.23)	1.02 (0.28)
Hip extensors	85.21 (20.47)	122.06 (62.14)	2.45 (0.51)	2.42 (0.71)
Hip abductors	30.36 (11.10)	56.01 (32.48)	0.87 (0.26)	1.08 (0.35)
Knee flexors	38.58 (7.13)	60.44 (36.60)	1.11 (0.17)	1.16 (0.32)
Knee extensors	50.55 (18.43)	84.08 (45.23)	1.44 (0.48)	1.64 (0.49)
Knee extensors 30	24.75 (15.46)	65.25 (39.71)	0.70 (0.35)	1.23 (0.38)
Muscle group (L side)				
Hip flexors	22.26 (9.60)	50.34 (26.54)	0.63 (0.23)	0.99 (0.30)
Hip extensors	77.64 (23.82)	117.34 (59.06)	2.27 (0.70)	2.34 (0.60)
Hip abductors	30.96 (10.51)	55.63 (32.32)	0.88 (0.23)	1.10 (0.41)
Knee flexors	35.99 (11.91)	58.86 (34.20)	1.00 (0.24)	1.13 (0.33)
Knee extensors	46.89 (18.38)	78.29 (38.99)	1.33 (0.47)	1.56 (0.36)
Knee extensors 30	22.73 (10.91)	65.46 (38.43)	0.65 (0.25)	1.24 (0.39)
Muscle group				
Hip flexors	22.24(9.45)	51.33(27.74)	0.63(0.22)	1.00(0.29)
Hip extensors	81.43(22.16)	119.70(59.61)	2.36(0.61)	2.38(0.65)
Hip abductors	30.66(10.63)	55.83(31.80)	0.87(0.25)	1.09(0.37)
Knee flexors	37.24(9.81)	59.65(34.81)	1.05(0.21)	1.14(0.32)
Knee extensors	48.65(18.14)	81.19(41.59)	1.38(0.47)	1.60(0.42)
Knee extensors 30	23.74(13.13)	65.35(38.40)	0.68(0.30)	1.24(0.38)

FILE 5

NORMALISED EMG DATA FOR CP SUBJECTS

RIGHT SIDE						RIGHT SIDE					
SUBJECT	RECTUS AS AGONIST-MVC	RECTUS AS ANTAGONIST-MAX	Coactivation Index CI Rectus F	% CI RF		SUBJECT	HAMSTRINGS AS AGONIST - MVC	HAMSTRINGS AS ANTAGONIST-MAX	Coactivation Index CI Medial H	% CI MH	
PSCP01B	1276.00	60.70	0.048	4.76	average	PSCP01B	911.00	490.00	0.538	53.79	average
PSCP01C	2035.00	303.00	0.149	14.89	18.63	PSCP01C	1816.00	182.00	0.100	10.02	14.01
PSCP02A	754.00	124.00	0.164	16.45	max	PSCP02A	1026.00	69.20	0.067	6.74	max
PSCP02B	408.00	61.00	0.150	14.95	66.43	PSCP02B	532.00	84.20	0.158	15.83	53.79
PSCP02C	1278.00	849.00	0.664	66.43	min	PSCP02C	1680.00	206.00	0.123	12.26	min
PSCP03A	1657.00	296.00	0.179	17.86	4.76	PSCP03A	1425.00	93.80	0.066	6.58	4.38
PSCP03B	2048.00	524.00	0.256	25.59	SD	PSCP03B	1759.00	153.00	0.087	8.70	SD
PSCP03C	2048.00	527.00	0.257	25.73	15.56	PSCP03C	1949.00	210.00	0.108	10.77	12.54
PSCP04A	651.00	71.40	0.110	10.97		PSCP04A	325.00	51.40	0.158	15.82	
PSCP04B	1098.00	103.00	0.094	9.38		PSCP04B	1093.00	91.50	0.084	8.37	
PSCP04C	1990.00	245.00	0.123	12.31		PSCP04C	766.00	126.00	0.164	16.45	
PSCP05A	379.00	50.20	0.132	13.25		PSCP05A	1193.00	148.00	0.124	12.41	
PSCP05B	649.00	62.30	0.096	9.60		PSCP05B	1282.00	56.10	0.044	4.38	
LEFT SIDE						LEFT SIDE					
SUBJECT	RECTUS AS AGONIST-MVC	RECTUS AS ANTAGONIST-MAX	Coactivation Index CI Rectus F	% CI RF		SUBJECT	HAMSTRINGS AS AGONIST - MVC	HAMSTRINGS AS ANTAGONIST-MAX	Coactivation Index CI Medial H	% CI MH	
PSCP01B	661.00	49.60	0.075	7.50	average	PSCP01B	1016.00	152.00	0.150	14.96	average
PSCP01C	442.00	32.30	0.073	7.31	24.30	PSCP01C	1167.00	97.90	0.084	8.39	29.52
PSCP02A	500.00	263.00	0.526	52.60	max	PSCP02A	552.00	256.00	0.464	46.38	max
PSCP02B	521.00	154.00	0.296	29.56	73.09	PSCP02B	417.00	299.00	0.717	71.70	71.70
PSCP02C	432.00	48.10	0.111	11.13	min	PSCP02C	977.00	358.00	0.366	36.64	min
PSCP03A	2044.00	1494.00	0.731	73.09	7.31	PSCP03A	785.00	227.00	0.289	28.92	8.39
PSCP03B	2048.00	1081.00	0.528	52.78	SD	PSCP03B	388.00	224.00	0.577	57.73	SD
PSCP03C	1648.00	434.00	0.263	26.33	21.71	PSCP03C	979.00	581.00	0.593	59.35	22.42
PSCP04A	551.00	72.10	0.131	13.09		PSCP04A	869.00	121.00	0.139	13.92	
PSCP04B	753.00	95.60	0.127	12.70		PSCP04B	179.00	26.30	0.147	14.69	
PSCP04C	745.00	84.30	0.113	11.32		PSCP04C	880.00	91.90	0.104	10.44	
PSCP05A	767.00	59.00	0.077	7.69		PSCP05A	769.00	93.90	0.122	12.21	
PSCP05B	851.00	92.30	0.108	10.85		PSCP05B	777.00	65.50	0.084	8.43	



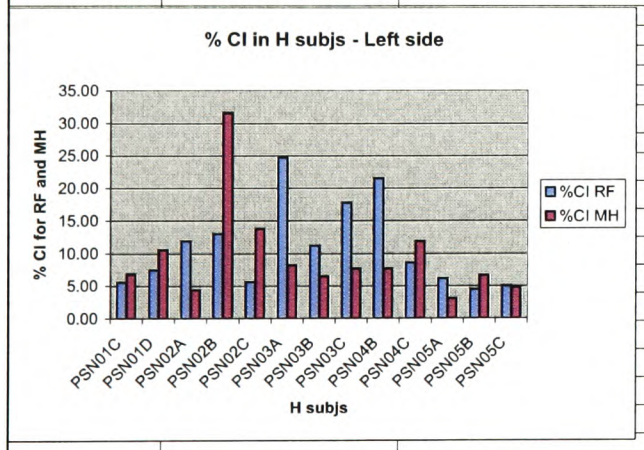


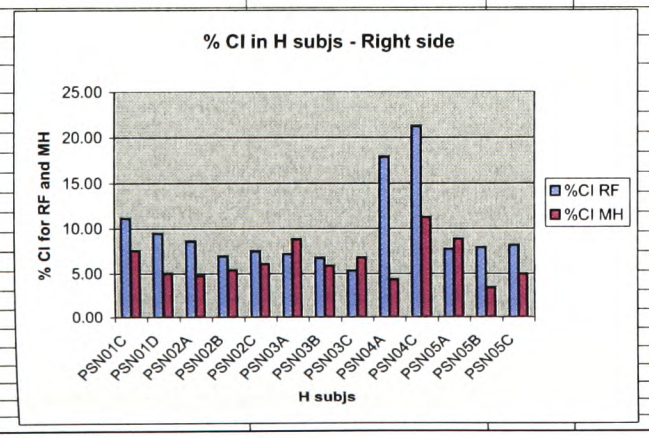
FILE 6

NORMALISED EMG DATA FOR NORMAL SUBJECTS

RIGHT SIDE						RIGHT SIDE						
SUBJECT	RECTUS AS AGONIST- MVC	RECTUS AS ANTAGONIST -MAX	Coactivation Index CI for RF	%CI RF		SUBJECT	HAMSTRINGS AS AGONIST - MVC	HAMSTRINGS AS ANTAGONIST - MAX	Coactivation Index CI for MH	%CI MH		
PSN01C	879.0	98.0	0.111	11.15	average	PSN01C	1007.0	76.0	0.075	7.55	average	
PSN01D	1309.0	124.0	0.095	9.47	9.67	PSN01D	1682.0	84.0	0.050	4.99	6.36	
PSN02A	560.0	48.0	0.086	8.57	max	PSN02A	636.0	30.0	0.047	4.72	max	
PSN02B	1974.0	136.0	0.069	6.89	21.22	PSN02B	1367.0	73.0	0.053	5.34	11.25	
PSN02C	1178.0	88.0	0.075	7.47	min	PSN02C	1191.0	72.0	0.060	6.05	min	
PSN03A	2048.0	147.0	0.072	7.18	5.31	PSN03A	1332.0	117.0	0.088	8.78	3.36	
PSN03B	1992.0	134.0	0.067	6.73	SD	PSN03B	2030.0	118.0	0.058	5.81	SD	
PSN03C	1844.0	98.0	0.053	5.31	4.65	PSN03C	1261.0	85.0	0.067	6.74	2.22	
PSN04A	1292.0	231.0	0.179	17.88		PSN04A	1333.0	57.0	0.043	4.28		
PSN04C	754.0	160.0	0.212	21.22		PSN04C	1271.0	143.0	0.113	11.25		
PSN05A	542.0	42.0	0.077	7.75		PSN05A	406.0	36.0	0.089	8.87		
PSN05B	1584.0	125.0	0.079	7.89		PSN05B	2023.0	68.0	0.034	3.36		
PSN05C	959.0	78.0	0.081	8.13		PSN05C	1789.0	88.0	0.049	4.92		

LEFT SIDE						LEFT SIDE						
SUBJECT	RECTUS AS AGONIST- MVC	RECTUS AS ANTAGONIST -MAX	Coactivation Index CI for RF	%CI RF	average	SUBJECT	HAMSTRINGS AS AGONIST - MVC	HAMSTRINGS AS ANTAGONIST - MAX	Coactivation Index CI for MH	%CI MH		
PSN01C	1121.0	62.0	0.055	5.53	10.96	PSN01C	1368.0	93.0	0.068	6.80	average	16.26
PSN01D	795.0	59.0	0.074	7.42	max	PSN01D	927.0	97.0	0.105	10.46	max	31.56
PSN02A	321.0	38.0	0.118	11.84	24.72	PSN02A	933.0	41.0	0.044	4.39	min	3.09
PSN02B	693.0	90.0	0.130	12.99	min	PSN02B	469.0	148.0	0.316	31.56	SD	7.27
PSN02C	1444.0	81.0	0.056	5.61	4.42	PSN02C	2048.0	282.0	0.138	13.77		
PSN03A	534.0	132.0	0.247	24.72	SD	PSN03A	1935.0	158.0	0.082	8.17		
PSN03B	2048.0	228.0	0.111	11.13	6.64	PSN03B	1767.0	114.0	0.065	6.45		
PSN03C	400.0	71.0	0.178	17.75		PSN03C	693.0	53.0	0.076	7.65		
PSN04B	149.0	32.0	0.215	21.48		PSN04B	747.0	57.0	0.076	7.63		
PSN04C	514.0	44.0	0.086	8.56		PSN04C	1156.0	136.0	0.118	11.76		
PSN05A	882.0	54.0	0.061	6.12		PSN05A	1746.0	54.0	0.031	3.09		
PSN05B	1855.0	82.0	0.044	4.42		PSN05B	2048.0	135.0	0.066	6.59		
PSN05C	1066.0	53.0	0.050	4.97		PSN05C	2048.0	97.0	0.047	4.74		





FILE 7 % CO-CONTRACTION INDEX FOR ALL SUBJECTS BILATERALLY

Subject	% CI RF	% CI MH		Subject	% CI RF	% CI MH
CP	4.76	53.79		N	11.15	7.55
CP	14.89	10.02		N	9.47	4.99
CP	16.45	6.74		N	8.57	4.72
CP	14.95	15.83		N	6.89	5.34
CP	66.43	12.26		N	7.47	6.05
CP	17.86	6.58		N	7.18	8.78
CP	25.59	8.70		N	6.73	5.81
CP	25.73	10.77		N	5.31	6.74
CP	10.97	15.82		N	17.88	4.28
CP	9.38	8.37		N	21.22	11.25
CP	12.31	16.45		N	7.75	8.87
CP	13.25	12.41		N	7.89	3.36
CP	9.60	4.38		N	8.13	4.92
CP	7.50	14.96		N	5.53	6.80
CP	7.31	8.39		N	7.42	10.46
CP	52.60	46.38		N	11.84	4.39
CP	29.56	71.70		N	12.99	31.56
CP	11.13	36.64		N	5.61	13.77
CP	73.09	28.92		N	24.72	8.17
CP	52.78	57.73		N	11.13	6.45
CP	26.33	59.35		N	17.75	7.65
CP	13.09	13.92		N	21.48	7.63
CP	12.70	14.69		N	8.56	11.76
CP	11.32	10.44		N	6.12	3.09
CP	7.69	12.21		N	4.42	6.59
CP	10.85	8.43		N	4.97	4.74
Mean (SD)	21.47(18.73)	21.76(19.48)		Mean (SD)	10.31(5.66)	7.91(5.50)

FILE 7

T-Test %CI EMG

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	CIRECFEM	21,4662	26	18,72653	3,67258
	NCIRECFE	10,3146	26	5,65824	1,10967
Pair 2	CIMEDHAM	21,7646	26	19,47507	3,81937
	NCIMEDHA	7,9123	26	5,50021	1,07868

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	CIRECFEM & NCIRECFE	26	,325	,105
Pair 2	CIMEDHAM & NCIMEDHA	26	,514	,007

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	CIRECFEM - NCIRECFE	11,1515	17,71414	3,47403	3,9966	18,3064	3,210	25	,004
Pair 2	CIMEDHAM - NCIMEDHA	13,8523	17,30477	3,39374	6,8628	20,8419	4,082	25	,000

FILE 8

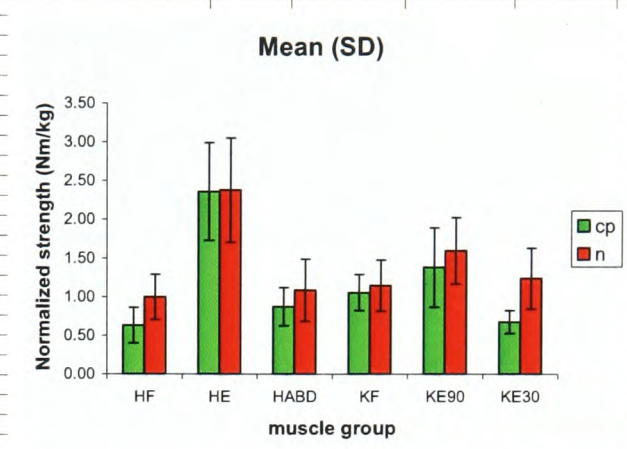
WITHIN-SUBJECT SD, REPEATABILITY AND COEFFICIENT OF VARIATION FOR ALL SUBJECTS

Subj	muscgr	mean	mean within-subj variance	within-subj SD	limits	coefficient of variation	CV %	upper limit	lower limit	CI range	or repeatability	CV	CV%
n	1	1.00	0.09	0.29	0.58	0.30	29.91	1.21	0.05	0.82		0.29	29.50
n	2	2.38	0.45	0.67	1.31	0.68	68.03	3.67	1.05	1.86		0.28	28.19
n	3	1.09	0.16	0.40	0.78	0.40	40.44	1.65	0.09	1.10		0.37	36.58
n	4	1.14	0.11	0.33	0.65	0.33	33.48	1.70	0.40	0.91		0.29	28.96
n	5	1.60	0.19	0.43	0.85	0.44	44.09	2.23	0.53	1.20		0.27	27.17
n	6	1.24	0.15	0.39	0.77	0.40	39.80	1.45	-0.09	1.09		0.32	31.65
cp	1	0.63	0.05	0.23	0.45	0.23	23.35	1.45	0.55	0.64		0.37	36.54
cp	2	2.36	0.40	0.63	1.24	0.64	63.98	3.62	1.14	1.75		0.27	26.73
cp	3	0.87	0.06	0.25	0.49	0.25	25.25	1.58	0.60	0.69		0.29	28.62
cp	4	1.05	0.05	0.23	0.46	0.24	23.57	1.60	0.68	0.64		0.22	22.13
cp	5	1.38	0.26	0.51	1.01	0.52	52.01	2.61	0.59	1.42		0.37	37.16
cp	6	0.68	0.02	0.15	0.29	0.15	15.04	1.53	0.95	0.41		0.22	21.81

	cp	n	sdCP	sdN
HF	**0.63**	**1.00**	**0.23**	**0.29**
HE	**2.36**	**2.38**	**0.63**	**0.67**
HABD	**0.87**	**1.09**	**0.25**	**0.40**
KF	**1.05**	**1.14**	**0.23**	**0.33**
KE90	**1.38**	**1.60**	**0.51**	**0.43**
KE30	**0.68**	**1.24**	**0.15**	**0.39**

scale factor 1

	n		cp	
	L	R	L	R
	Mean	Mean	Mean	Mean
Hip flexors	0.99	1.02	0.63	0.63
Hip ext	2.34	2.42	2.27	2.45
Hip abd	1.10	1.08	0.88	0.87
knee flexor	1.13	1.16	1.00	1.11
knee ext	1.56	1.64	1.33	1.44
knee ext 3(	1.24	1.23	0.65	0.70

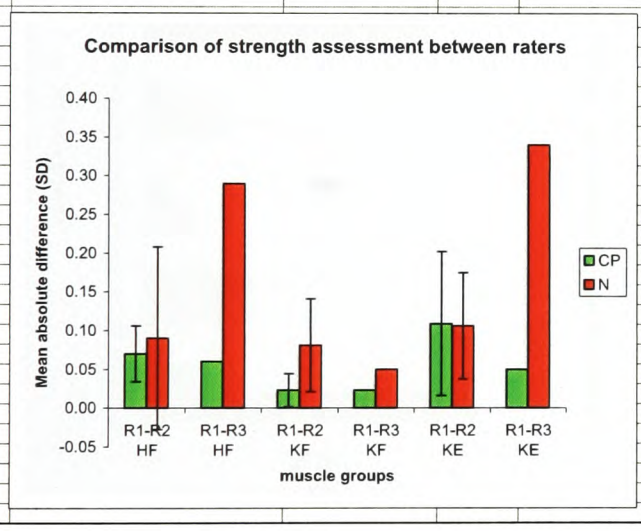


MEAN OF ABSOLUTE DIFFERENCE

	R1-R2 HF	R1-R3 HF	R1-R2 KF	R1-R3 KF	R1-R2 KE	R1-R3 KE
CP	0.07	0.06	0.02	0.02	0.11	0.05
N	0.09	0.29	0.08	0.05	0.11	0.34

sd

	R1-R2 HF	R1-R3 HF	R1-R2 KF	R1-R3 KF	R1-R2 KE	R1-R3 KE
CP	0.04		0.02		0.09	
N	0.12		0.06		0.07	



FILE 8

DATA FROM ALL RATERS

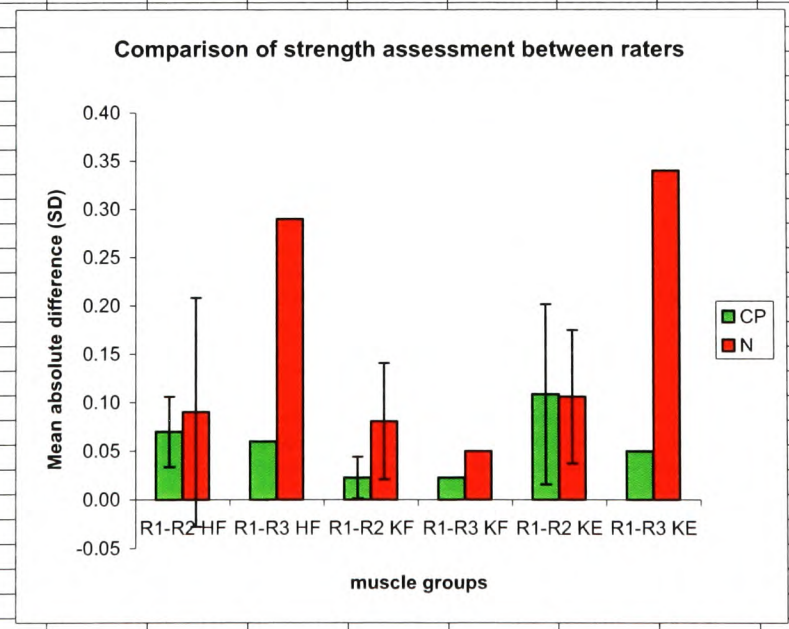
	HF=1													KF=4								
	r1			r2					r3					r1			r2					r3
	R	L	AVE	R	L	AVE	R1-R2	ABSR1-R2	R	L	AVE	R1-R3	ABSR1-R3	R	L	AVE	R	L	AVE	R1-R2	ABSR1-R2	R
cp1	1.17	0.77	0.97	1.25	0.78	1.01	**-0.04**	**0.04**							1.41	1.41		1.36	1.36	**0.05**	**0.05**	
cp2														1.09	0.63	0.86	1.08	0.64	0.86	**0.00**	**0.00**	
cp3														1.44	0.96	1.20	1.42	1.05	1.24	**-0.04**	**0.04**	
cp4	0.42	0.47	0.44						0.45	0.55	0.50	**-0.06**	**0.06**	1.09	0.97	1.03						0.97
cp5	0.82	0.81	0.81	0.96	0.85	0.91	**-0.10**	**0.10**						1.09	0.82	0.96	1.01	0.91	0.96	**-0.01**	**0.01**	
n1	0.99	1.18	1.08	1.09	1.11	1.10	**-0.02**	**0.02**						1.08		1.08	1.21		1.21	**-0.14**	**0.14**	
n2	1.00	0.86	0.93	1.14	0.82	0.98	**-0.04**	**0.04**						1.28	1.12	1.20	1.39	1.24	1.32	**-0.12**	**0.12**	
n3	1.17	1.41	1.29	1.01	1.04	1.03	**0.27**	**0.27**						1.35	1.28	1.31	1.50	1.15	1.32	**-0.01**	**0.01**	
n4	0.52	0.58	0.55	0.51	0.53	0.52	**0.03**	**0.03**						0.88	0.76	0.82	0.97	0.78	0.88	**-0.06**	**0.06**	
n5	1.29	1.14	1.21						0.91	0.93	0.92	**0.29**	**0.29**	1.05	1.17	1.11						1.16

MEAN OF ABSOLUTE DIFFERENCE

	R1-R2 HF	R1-R3 HF	R1-R2 KF	R1-R3 KF	R1-R2 KE	R1-R3 KE
CP	0.07	0.06	0.02	0.02	0.11	0.05
N	0.09	0.29	0.08	0.05	0.11	0.34

sd

	R1-R2 HF	R1-R3 HF	R1-R2 KF	R1-R3 KF	R1-R2 KE	R1-R3 KE
CP	0.04		0.02		0.09	
N	0.12		0.06		0.07	



FILE 8

DATA FROM ALL RATERS

				KE=5												
				r1			r2					r3				
L	AVE	R1-R3	ABSR1-R3	R	L	AVE	R	L	AVE	R1-R2	ABSR1-R2	R	L	AVE	R1-R3	ABSR1-R3
					2.03	2.03		1.84	1.84	**0.19**	**0.19**					
				1.30	1.32	1.31	1.21	1.16	1.18	**0.13**	**0.13**					
0.90	0.94	**0.09**	**0.09**	1.21	0.84	1.03						1.23	0.92	1.07	**-0.05**	**0.05**
				1.25		1.25	1.26		1.26	**-0.01**	**0.01**					
				1.68		1.68	1.83		1.83	**-0.15**	**0.15**					
				1.75	1.46	1.60	1.87	1.57	1.72	**-0.12**	**0.12**					
				1.93	2.04	1.99	1.93	1.74	1.84	**0.15**	**0.15**					
				0.64	0.91	0.78	0.84	0.73	0.78	**-0.01**	**0.01**					
1.16	1.16	**-0.05**	**0.05**	1.88	1.66	1.77						1.48	1.38	1.43	**0.34**	**0.34**

FILE 9

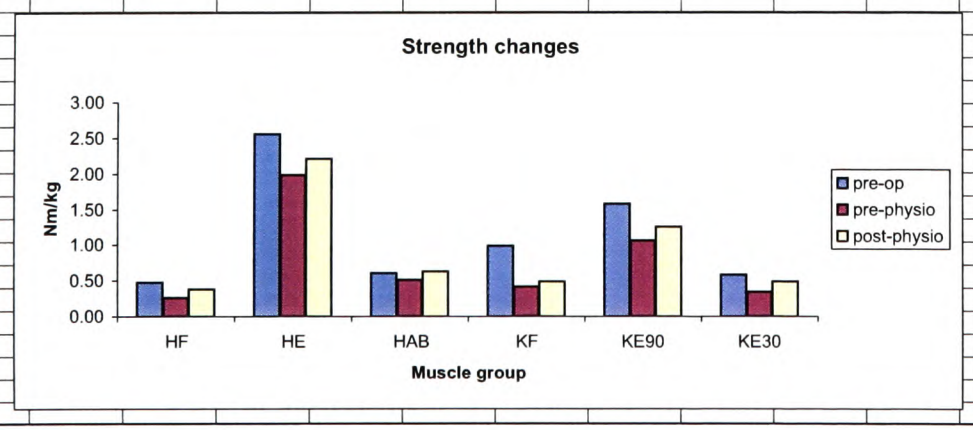
NORMALISED STRENGTH DATA FROM ALL VISITS FOR GROUPS B AND C. % DIFFERENCE BETWEEN VISITS

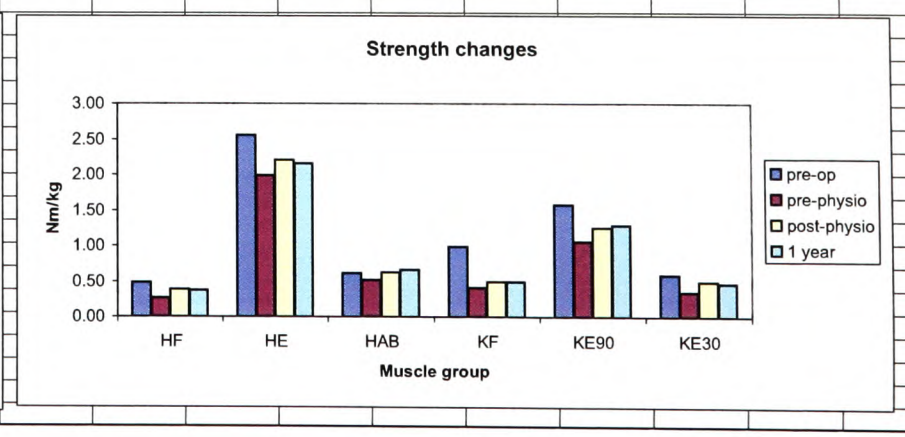
subject	hf1	hf2	hf3	hf4	he1	he2	he3	he4	ha1	ha2	ha3	ha4	kf1	kf2	kf3	kf4	ke90-1	ke90-2	ke90-3	ke90-4	ke30-1	ke30-2	ke30-3	ke30-4		%HF2-3
1.00	0.51	0.32	0.30	0.40	1.93	1.14	0.75	1.53	0.41	0.17	0.34	0.53	0.99	0.22	0.32	0.49	1.14	0.27	0.51	0.51	0.33	0.31	0.38	0.45		6.25
2.00	0.44	0.20	0.23	0.11	2.63	1.44	1.71	1.67	0.63	0.32	0.27	0.18	0.84	0.29	0.18	0.25	1.24	0.61	0.57	0.57	0.57	0.41	0.45	0.25		-15.00
3.00	0.25	0.25	0.35	0.30	1.47	1.41	1.97	1.86	0.69	0.47	0.76	0.75	0.76	0.55	0.60	0.43	1.30	0.75	0.89	0.94	0.28	0.26	0.44	0.42		-40.00
4.00	0.79	0.50	0.31	0.59	3.72	2.74	3.54	3.76	0.97	0.78	0.85	0.89	1.32	0.70	0.84	1.27	3.62	1.66	2.30	2.19	1.24	,	0.82	0.88		38.00
5.00	0.28	0.15	0.30	0.34	3.00	2.12	2.09	2.40	0.46	0.21	0.51	0.59	0.38	0.21	0.43	0.36	2.25	0.88	1.04	1.09	0.68	0.50	0.49	0.47		-100.00
6.00	0.53	0.57	0.63	0.72	2.33	2.51	2.89	2.78	0.89	0.90	1.05	0.96	0.90	1.04	0.99	0.97	0.92	0.96	1.15	1.26	0.58	0.40	0.43	0.56		-10.53
7.00	1.07	0.17	0.53	0.58	3.54	2.49	2.78	2.89	0.48	0.50	0.65	0.80	1.63	0.36	0.35	0.28	1.01	1.23	1.28	1.14	,	0.19	0.28	0.32		-211.76
8.00	0.24	0.16	0.21	0.15	1.76	1.08	1.17	1.76	0.33	0.14	0.20	0.22	1.06	0.19	0.13	0.13	0.61	0.76	0.73	0.57	,	0.11	0.22	0.11		-31.25
9.00	0.27	0.17	0.27	0.22	1.65	2.28	2.38	2.17	0.71	0.62	0.73	0.73	0.75	0.39	0.42	0.48	1.66	1.18	1.30	1.76	0.56	0.36	0.36	0.49		-58.82
10.00	0.32	0.17	0.18	0.26	1.92	1.48	1.35	1.54	0.58	0.43	0.48	0.51	1.59	0.29	0.35	0.39	1.11	0.72	0.69	1.16	,	0.19	0.43	0.40		-5.88
11.00	0.79	0.25	0.74	0.32	3.67	2.96	2.93	3.37	0.34	0.53	0.55	0.86	1.07	0.32	0.42	0.42	2.02	1.28	1.41	1.64	,	0.15	0.27	0.43		-196.00
12.00	0.52	0.16	0.42	0.36	3.24	2.38	2.95	2.55	0.90	0.53	0.92	0.72	1.40	0.69	0.87	0.84	2.54	1.17	1.84	1.68	0.78	0.23	0.41	0.29		-162.50
13.00	0.28	0.36	0.33	0.36	2.08	1.72	1.65	1.99	0.55	0.44	0.29	0.41	0.54	0.22	0.20	0.22	1.71	0.99	1.27	1.31	,	,	,	,		8.33
14.00	1.02	0.51	0.67	0.69	2.81	1.53	1.84	1.36	0.60	0.19	0.19	,	0.74	0.28	0.46	0.28	1.12	0.66	0.90	1.04	,	,	,	,		-31.37
15.00	0.39	0.15	0.24	0.38	3.39	2.18	2.60	1.75	,	,	,	,	1.27	0.36	0.44	0.47	0.96	1.06	0.90	0.70	,	,	,	,		-60.00
16.00	0.46	0.47	0.51	0.39	2.01	2.14	2.34	1.65	0.47	0.97	0.82	0.76	1.13	0.43	0.65	0.41	0.97	1.19	1.38	1.18	,	0.17	0.35	,		-8.51
17.00	0.47	0.17	0.66	0.42	4.53	2.27	2.64	2.04	0.82	0.69	0.77	0.75	0.96	0.55	0.57	0.67	1.85	1.59	1.88	2.06	0.38	0.57	0.81	0.62		-288.24
18.00	0.47	0.42	0.61	0.52	2.15	2.28	2.14	2.32	0.97	0.95	1.13	1.12	1.19	0.64	0.74	0.68	2.64	2.46	2.34	1.88	1.41	1.14	1.14	1.00		-45.24
19.00	0.35	0.12	,	0.34	1.41	0.87	,	1.33	0.27	0.37	,	0.39	0.88	0.50	,	0.32	1.11	0.61	,	0.83	0.13	0.20	,	0.29		
20.00	0.60	0.33	0.36	0.32	3.51	2.50	2.57	1.93	0.97	0.93	1.04	0.94	,	0.34	0.59	0.58	1.47	1.17	1.31	1.31	,	0.32	0.62	0.48		-9.09
1l	0.27	0.08	0.19	0.21	1.39	0.77	0.93	1.78	0.39	0.24	0.40	0.58	0.84	0.18	0.32	0.52	1.03	0.69	0.70	0.71	0.25	0.31	0.39	0.32		-137.50
2l	0.36	0.25	0.16	0.12	2.81	1.54	1.83	1.56	0.67	0.26	0.16	0.18	0.90	0.35	0.47	0.44	1.20	0.71	0.82	0.60	0.51	0.27	0.31	0.10		36.00
3l	0.62	0.17	0.23	0.15	1.14	1.55	2.08	1.76	0.60	0.39	0.53	0.45	0.94	0.57	0.59	0.67	1.24	0.73	0.89	0.87	0.50	0.34	0.49	0.37		-35.29
4l	0.88	0.35	0.33	0.31	4.36	3.08	3.22	4.00	1.05	0.70	0.69	0.79	1.33	0.84	0.90	0.88	2.75	1.74	2.09	2.16	1.32	,	0.62	0.80		5.71
5l	0.27	0.12	0.20	0.20	2.78	2.50	2.12	2.46	0.58	0.22	0.30	0.71	0.61	0.47	0.51	0.48	1.62	0.99	1.07	1.15	0.38	0.15	0.22	0.16		-66.67
6l	0.50	0.29	0.36	0.38	3.00	2.24	2.03	2.17	0.74	0.55	0.86	0.65	0.53	0.21	0.36	0.28	1.48	0.88	0.90	1.16	0.33	0.28	0.34	0.37		-24.14
7l	0.79	0.18	0.31	0.62	3.55	2.63	3.26	2.90	0.59	0.63	0.58	0.48	1.29	0.22	0.18	0.19	1.18	1.65	1.76	2.62	,	0.45	0.63	0.67		-72.22
8l	0.13	0.11	0.18	0.16	1.54	0.85	1.27	1.89	0.14	0.09	0.11	0.13	0.54	0.15	0.12	0.18	1.02	0.65	0.85	0.95	,	0.09	0.13	0.09		-63.64
9l	0.25	0.14	0.23	0.18	1.53	1.59	2.37	2.84	0.63	0.52	0.63	0.90	0.85	0.23	0.26	0.42	1.70	0.97	1.21	1.68	0.43	0.44	0.48	0.73		-64.29
10l	0.47	0.11	0.17	0.14	1.12	1.50	1.26	1.24	0.72	0.42	0.61	0.52	1.29	0.39	0.38	0.48	1.08	0.74	0.75	1.19	,	0.61	0.59	0.80		-54.55
11l	0.30	0.19	0.49	0.54	3.17	4.66	3.34	3.28	0.69	0.49	0.63	0.85	0.97	0.31	0.45	0.35	2.50	1.20	1.57	1.91	,	0.21	0.31	0.41		-157.89
12l	0.79	0.41	0.67	0.38	3.17	1.84	2.71	2.63	0.74	0.52	0.75	0.67	1.07	0.40	0.58	0.51	1.83	1.15	1.45	1.23	0.47	0.31	0.53	0.42		-63.41
13l	0.51	0.35	0.35	0.38	2.29	1.38	1.63	1.67	0.52	0.31	0.33	0.28	0.69	0.29	0.23	0.29	2.00	0.92	1.06	1.14	,	,	,	,		0.00
14l	0.65	,	,	,	2.97	,	,	,	0.22	,	,	,	1.29	,	,	,	1.57	,	,	,	0.63	,	,	,		
15l	0.30	0.23	0.22	0.41	2.60	2.35	2.17	1.82	,	,	,	,	1.12	0.26	0.36	0.38	1.14	0.86	0.80	0.67	,	,	,	,		4.35
16l	0.38	0.43	0.72	0.85	1.44	2.00	2.04	1.75	0.51	0.77	0.84	1.25	1.12	0.50	0.75	0.50	1.02	1.47	1.60	1.47	0.26	0.42	0.45	0.34		-67.44
17l	0.24	0.27	0.36	0.44	3.77	1.88	1.97	1.96	0.70	0.65	0.89	0.79	1.00	0.48	0.62	0.60	1.78	1.30	1.54	1.59	0.45	0.36	0.58	0.52		-33.33
18l	0.67	0.53	0.66	0.69	2.55	2.49	2.77	2.38	0.88	0.92	1.14	1.20	1.12	0.75	0.85	0.90	2.59	1.98	2.32	2.38	1.40	0.85	1.17	1.04		-24.53
19l	0.27	0.17	,	0.42	1.50	0.92	,	1.21	0.31	0.48	,	0.51	0.69	0.51	,	0.51	0.94	0.43	,	0.62	0.25	0.23	,	0.39		
20l	0.59	0.26	0.46	0.40	2.94	2.32	2.45	1.93	0.53	0.73	1.13	1.00	,	0.45	0.62	0.84	2.00	1.17	1.33	1.27	,	0.36	0.54	0.49		-76.92

	hf1	hf2	hf3	hf4	he1	he2	he3	he4	ha1	ha2	ha3	ha4														
Nm/kg	0.48	0.26	0.38	0.38	2.56	1.99	2.21	2.15	0.61	0.51	0.63	0.67	0.99	0.41	0.49	0.50	1.57	1.06	1.25	1.29	0.59	0.35	0.49	0.47		

	pre-op	pre-physio	post-physio	1 year
HF	0.48	0.26	0.38	0.38
HE	2.56	1.99	2.21	2.15
HAB	0.61	0.51	0.63	0.67
KF	0.99	0.41	0.49	0.50
KE90	1.57	1.06	1.25	1.29
KE30	0.59	0.35	0.49	0.47

	pre-op	post-op
HF	0.48	0.26
HE	2.56	1.99
HAB	0.61	0.51
KF	0.99	0.41
KE90	1.57	1.06
KE30	0.59	0.35

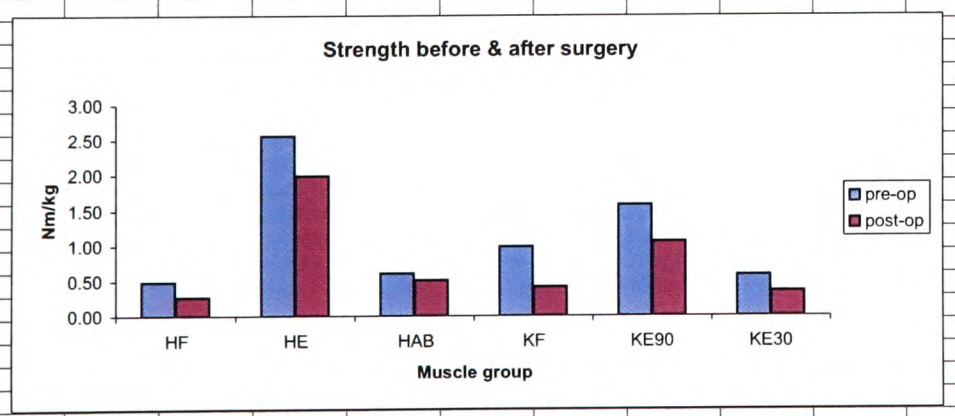




FILE 9

NORMALISED STRENGTH DATA FROM ALL VISITS FOR GROUPS B AND C. % DIFFERENCE BETWEEN VISITS

%HE2-3	%HAB2-3	%KF2-3	%KE902-3	%KE302-3		Mean % change 2-3						%HF1-4	%HE1-4	%HABD1-4	%KF1-4	%KE901-4	%KE301-4		%HF1-3	%HE1-3	%HAB1-3	%KF1-3	%KE901-3	%KE301-3
34.21	-100.00	-45.45	-88.89	-22.58								21.57	20.73	-29.27	50.51	55.26	-36.36		41.18	61.14	17.07	67.68	55.26	-15.15
-18.75	15.63	37.93	6.56	-9.76		HF	HE	HAB	KF	KE90	KE30	75.00	36.50	71.43	70.24	54.03	56.14		47.73	34.98	57.14	78.57	54.03	21.05
-39.72	-61.70	-9.09	-18.67	-69.23		-57.23	-10.18	-26.49	-22.23	-16.16	-43.28	-20.00	-26.53	-8.70	43.42	27.69	-50.00		-40.00	-34.01	-10.14	21.05	31.54	-57.14
-29.20	-8.97	-20.00	-38.55									25.32	-1.08	8.25	3.79	39.50	29.03		60.76	4.84	12.37	36.36	36.46	33.87
1.42	-142.86	-104.76	-18.18	2.00								-21.43	20.00	-28.26	5.26	51.56	30.88		-7.14	30.33	-10.87	-13.16	53.78	27.94
-15.14	-16.67	4.81	-19.79	-7.50		Mean % change 1-4						-35.85	-19.31	-7.87	-7.78	-36.96	3.45		-18.87	-24.03	-17.98	-10.00	-25.00	25.86
-11.65	-30.00	2.78	-4.07	-47.37								45.79	18.36	-66.67	82.82	-12.87			50.47	21.47	-35.42	78.53	-26.73	
-8.33	-42.86	31.58	3.95	-100.00		HF	HE	HAB	KF	KE90	KE30	37.50	0.00	33.33	87.74	6.56			12.50	33.52	39.39	87.74	-19.67	
-4.39	-17.74	-7.69	-10.17	0.00		11.02	9.04	-13.23	48.65	14.39	-0.91	18.52	-31.52	-2.82	36.00	-6.02	12.50		0.00	-44.24	-2.82	44.00	21.69	35.71
8.78	-11.63	-20.69	4.17	-126.32								18.75	19.79	12.07	75.47	-4.50			43.75	29.69	17.24	77.99	37.84	
1.01	-3.77	-31.25	-10.16	-80.00								59.49	8.17	-152.94	60.75	18.81			6.33	20.16	-61.76	60.75	30.20	
-23.95	-73.58	-26.09	-57.26	-78.26								30.77	21.30	20.00	40.00	33.86	62.82		19.23	8.95	-2.22	37.86	27.56	47.44
4.07	34.09	9.09	-28.28			Minus denotes improvement						-28.57	4.33	25.45	59.26	23.39			-17.86	20.67	47.27	62.96	25.73	
-20.26	0.00	-64.29	-36.36									32.35	51.60		62.16	7.14			34.31	34.52	68.33	37.84	19.64	
-19.27		-22.22	15.09			Mean % change 1-3						2.56	48.38		62.99	27.08			38.46	23.30		65.35	6.25	
-9.35	15.46	-51.16	-15.97	-105.88		HF	HE	HAB	KF	KE90	KE30	15.22	17.91	-61.70	63.72	-21.65			-10.87	-16.42	-74.47	42.48	-42.27	
-16.30	-11.59	-3.64	-18.24	-42.11		13.04	9.96	0.71	49.00	17.79	-0.34	10.64	54.97	8.54	30.21	-11.35	-63.16		-40.43	41.72	6.10	40.63	-1.62	-113.16
6.14	-18.95	-15.63	4.88	0.00								-10.64	-7.91	-15.46	42.86	28.79	29.08		-29.79	0.47	-16.49	37.82	11.36	19.15
												2.86	5.67	-44.44	63.64	25.23	-123.08							
-2.80	-11.83	-73.53	-11.97	-93.75								46.67	45.01	3.09		10.88			40.00	26.78	-7.22		10.88	
-20.78	-66.67	-77.78	-1.45	-25.81								22.22	-28.06	-48.72	38.10	31.07	-28.00		29.63	33.09	-2.56	61.90	32.04	-56.00
-18.83	38.46	-34.29	-15.49	-14.81								66.67	44.48	73.13	51.11	50.00	80.39		55.56	34.88	76.12	47.78	31.67	39.22
-34.19	-35.90	-3.51	-21.92	-44.12								75.81	-54.39	25.00	28.72	29.84	26.00		62.90	-82.46	11.67	37.23	28.23	2.00
-4.55	1.43	-7.14	-20.11									64.77	8.26	24.76	33.83	21.45	39.39		62.50	26.15	34.29	32.33	24.00	53.03
15.20	-36.36	-8.51	-8.08	-46.67								25.93	11.51	-22.41	21.31	29.01	57.89		25.93	23.74	48.28	16.39	33.95	42.11
9.38	-56.36	-71.43	-2.27	-21.43								24.00	27.67	12.16	47.17	21.62	-12.12		28.00	32.33	-16.22	32.08	39.19	-3.03
-23.95	7.94	18.18	-6.67	-40.00								21.52	18.31	18.64	85.27	-122.03			60.76	8.17	1.69	86.05	-49.15	
-49.41	-22.22	20.00	-30.77	-44.44								-23.08	-22.73	7.14	66.67	6.86			-38.46	17.53	21.43	77.78	16.67	
-49.06	-21.15	-13.04	-24.74	-9.09								28.00	-85.62	-42.86	50.59	1.18	-69.77		8.00	-54.90	0.00	69.41	28.82	-11.63
16.00	-45.24	2.56	-1.35	3.28								70.21	-10.71	27.78	62.79	-10.19			63.83	-12.50	15.28	70.54	30.56	
28.33	-28.57	-45.16	-30.83	-47.62								-80.00	-3.47	-23.19	63.92	23.60			-63.33	-5.36	8.70	53.61	37.20	
-47.28	-44.23	-45.00	-26.09	-70.97								51.90	17.03	9.46	52.34	32.79	10.64		15.19	14.51	-1.35	45.79	20.77	-12.77
-18.12	-6.45	20.69	-15.22									25.49	27.07	46.15	57.97	43.00			31.37	28.82	36.54	66.67	47.00	
7.66		-38.46	6.98									-36.67	30.00		66.07	41.23			26.67	16.54		67.86	29.82	
-2.00	-9.09	-50.00	-8.84	-7.14								-123.68	-21.53	-145.10	55.36	-44.12	-30.77		-89.47	-41.67	-64.71	33.04	-56.86	-73.08
-4.79	-36.92	-29.17	-18.46	-61.11								-83.33	48.01	-12.86	40.00	10.67	-15.56		-50.00	47.75	-27.14	38.00	13.48	-28.89
-11.24	-23.91	-13.33	-17.17	-37.65								-2.99	6.67	-36.36	19.64	8.11	25.71		1.49	-8.63	-29.55	24.11	10.42	16.43
												-55.56	19.33	-64.52	26.09	34.04	-56.00							
-5.60	-54.79	-37.78	-13.68	-50.00								32.20	34.35	-88.68		36.50			22.03	16.67	-113.21		33.50	



FILE 10

NORMALISED TEMPORAL PARAMETERS FOR ALL VISITS AND ALL GROUPS (A, B,C)

subject	No	Rleg1	Lleg1	bvel1	bcad1	brstep1	blstep1	**nbvel1**	**nbcad1**	**nbrstep1**	**nblstep1**	Rleg2	Lleg2	bvel2	bcad2	brstep2	blstep2	**nbvel2**	**nbcad2**	**nbrstep2**	**nblstep2**
MSA01	1.00	84.00	85.00	1.04	115.33	0.54	0.54	**0.36**	**0.56**	**0.64**	**0.64**	86.00	86.00	1.05	112.83	0.56	0.55	**0.36**	**0.56**	**0.65**	**0.64**
MSA03	2.00	70.00	69.00	1.08	139.17	0.44	0.49	**0.41**	**0.62**	**0.63**	**0.71**	70.00	71.00	1.02	140.17	0.43	0.45	**0.39**	**0.63**	**0.61**	**0.64**
MSA04	3.00	64.00	64.00	1.20	145.00	0.49	0.50	**0.48**	**0.62**	**0.77**	**0.78**	63.00	63.00	1.00	128.67	0.46	0.45	**0.40**	**0.54**	**0.73**	**0.71**
MSA05	4.00	65.00	65.00	1.11	151.33	0.43	0.45	**0.44**	**0.65**	**0.66**	**0.69**	65.00	65.00	1.13	149.50	0.45	0.47	**0.45**	**0.64**	**0.69**	**0.72**
MSA07	5.00	67.00	67.00	1.25	157.67	0.52	0.46	**0.49**	**0.69**	**0.78**	**0.69**	71.00	70.00	1.13	144.33	0.47	0.47	**0.43**	**0.64**	**0.67**	**0.66**
MSA08	6.00	69.00	68.00	0.96	141.83	0.40	0.39	**0.37**	**0.62**	**0.58**	**0.57**	68.00	68.00	1.01	148.00	0.41	0.42	**0.39**	**0.65**	**0.60**	**0.61**
MSA12	7.00	69.00	69.00	1.49	157.83	0.58	0.58	**0.57**	**0.70**	**0.84**	**0.84**	72.00	72.00	1.30	153.00	0.54	0.49	**0.49**	**0.69**	**0.75**	**0.69**
MSA13	8.00	87.00	86.00	1.19	126.50	0.58	0.54	**0.41**	**0.63**	**0.67**	**0.62**	88.00	87.00	1.23	128.00	0.59	0.55	**0.42**	**0.64**	**0.68**	**0.63**
MSA14	9.00	76.00	76.00	1.16	126.33	0.52	0.57	**0.43**	**0.59**	**0.69**	**0.75**	77.00	77.00	1.25	139.67	0.55	0.52	**0.46**	**0.65**	**0.71**	**0.68**
MSA15	10.00	89.00	89.00	1.32	139.83	0.59	0.56	**0.45**	**0.70**	**0.66**	**0.63**										
MS01	1.00	73.00	73.00	0.68	114.67	0.43	0.34	**0.25**	**0.52**	**0.59**	**0.46**	73.00	73.00	0.72	87.77	0.56	0.43	**0.27**	**0.40**	**0.76**	**0.58**
MS02	2.00	70.00	70.00	1.05	110.50	0.56	0.57	**0.40**	**0.49**	**0.80**	**0.81**	73.00	73.00	0.99	96.20	0.63	0.62	**0.37**	**0.44**	**0.87**	**0.84**
MS03	3.00	70.00	70.00	1.06	138.67	0.44	0.48	**0.40**	**0.62**	**0.63**	**0.69**	75.00	75.00	1.03	122.83	0.50	0.47	**0.38**	**0.57**	**0.67**	**0.63**
MS04	4.00	94.00	94.00	1.11	118.00	0.58	0.54	**0.36**	**0.61**	**0.62**	**0.57**	96.00	96.00	0.89	116.17	0.44	0.50	**0.29**	**0.61**	**0.46**	**0.52**
MS05	5.00	86.00	86.00	0.64	94.15	0.34	0.48	**0.22**	**0.46**	**0.40**	**0.56**	83.00	83.00	0.39	59.50	0.46	0.32	**0.14**	**0.29**	**0.56**	**0.39**
MS07	6.00	68.00	67.00	1.09	145.17	0.43	0.46	**0.42**	**0.63**	**0.63**	**0.69**	70.00	70.00	0.86	129.33	0.34	0.44	**0.33**	**0.58**	**0.49**	**0.62**
MS08	7.00	85.00	85.00	0.66	94.35	0.43	0.40	**0.23**	**0.46**	**0.51**	**0.47**	81.00	81.00	0.47	73.80	0.41	0.39	**0.17**	**0.35**	**0.51**	**0.48**
MS09	8.00	74.00	75.00	0.52	97.25	0.30	0.37	**0.19**	**0.45**	**0.41**	**0.49**	73.00	74.00	0.16	46.83	0.24	0.18	**0.06**	**0.21**	**0.33**	**0.24**
MS10	9.00	70.00	71.00	1.07	138.33	0.45	0.49	**0.41**	**0.62**	**0.64**	**0.70**	74.00	74.00	0.98	108.60	0.56	0.56	**0.36**	**0.50**	**0.76**	**0.75**
MS11	10.00	75.00	75.00	1.02	121.17	0.51	0.51	**0.37**	**0.56**	**0.68**	**0.68**	77.00	78.00	1.04	115.50	0.54	0.53	**0.38**	**0.54**	**0.69**	**0.68**
MS12	11.00	87.00	87.00	0.46	69.15	0.42	0.36	**0.16**	**0.34**	**0.48**	**0.41**	88.00	88.00	0.58	70.82	0.52	0.47	**0.20**	**0.35**	**0.59**	**0.53**
MS13	12.00	83.00	82.00	1.00	118.17	0.46	0.55	**0.35**	**0.57**	**0.56**	**0.67**	83.00	82.00	0.76	106.67	0.38	0.46	**0.27**	**0.52**	**0.46**	**0.56**
MS14	13.00	65.00	65.00	0.67	112.67	0.40	0.28	**0.26**	**0.48**	**0.62**	**0.44**	67.00	67.00	0.46	89.02	0.37	0.24	**0.18**	**0.39**	**0.55**	**0.35**
MS15	14.00	87.00	87.00	0.82	113.50	0.44	0.40	**0.28**	**0.56**	**0.51**	**0.46**	90.00	91.00	0.41	63.72	0.36	0.42	**0.14**	**0.32**	**0.40**	**0.46**
MS16	15.00	85.00	85.00	0.69	102.40	0.42	0.33	**0.24**	**0.50**	**0.50**	**0.39**	86.00	86.00	0.47	60.62	0.39	0.62	**0.16**	**0.30**	**0.45**	**0.72**
MS17	16.00	66.00	66.00	0.74	121.83	0.38	0.36	**0.29**	**0.53**	**0.57**	**0.55**	68.00	68.00	0.92	108.70	0.48	0.54	**0.36**	**0.48**	**0.71**	**0.79**
MS18	17.00	65.00	65.00	0.98	135.00	0.41	0.43	**0.39**	**0.58**	**0.63**	**0.66**	71.00	71.00	1.04	113.17	0.56	0.53	**0.39**	**0.51**	**0.79**	**0.74**
MS19	18.00	82.00	83.00	0.99	111.50	0.53	0.55	**0.35**	**0.54**	**0.64**	**0.66**	82.00	83.00	1.11	107.17	0.64	0.61	**0.39**	**0.52**	**0.78**	**0.74**
MS20	19.00	79.00	79.00	0.74	109.17	0.41	0.39	**0.27**	**0.52**	**0.51**	**0.50**	80.00	80.00	0.43	89.25	0.26	0.32	**0.15**	**0.42**	**0.32**	**0.40**
MS21	20.00	83.00	83.00	1.20	127.33	0.56	0.58	**0.42**	**0.62**	**0.67**	**0.70**	84.00	84.00	0.84	93.33	0.52	0.55	**0.29**	**0.46**	**0.62**	**0.65**

NORMALISED TEMPORAL PARAMETERS FOR ALL VISITS AND ALL GROUPS (A, B,C)

Rleg3	Lleg3	bvel3	bcad3	brstep3	blstep3	**nbvel3**	**nbcad3**	**nbrstep3**	**nblstep3**	Rleg4	Lleg4	bvel4	bcad4	brstep4	blstep4	**nbvel4**	**nbcad4**	**nbrstep4**	**nblstep4**
86.00	86.00	1.23	126.83	0.56	0.59	**0.42**	**0.63**	**0.65**	**0.69**										
73.00	73.00	0.98	136.67	0.43	0.40	**0.36**	**0.62**	**0.58**	**0.55**										
66.00	66.00	1.04	133.17	0.47	0.45	**0.41**	**0.58**	**0.72**	**0.69**										
66.00	66.00	1.26	149.67	0.49	0.51	**0.50**	**0.65**	**0.74**	**0.77**										
74.00	73.00	1.32	147.33	0.55	0.49	**0.49**	**0.67**	**0.75**	**0.67**										
71.00	71.00	0.87	117.67	0.43	0.44	**0.33**	**0.53**	**0.61**	**0.62**										
87.00	87.00	1.22	124.00	0.61	0.57	**0.42**	**0.62**	**0.70**	**0.66**										
89.00	88.00	1.17	134.00	0.53	0.52	**0.40**	**0.67**	**0.60**	**0.59**										
73.00	73.00	0.43	65.18	0.44	0.37	**0.16**	**0.30**	**0.61**	**0.50**	77.00	77.00	0.85	102.08	0.46	0.54	**0.31**	**0.48**	**0.59**	**0.70**
73.00	73.00	1.13	102.65	0.67	0.65	**0.42**	**0.47**	**0.92**	**0.89**	73.00	73.00	0.80	97.40	0.50	0.48	**0.30**	**0.44**	**0.69**	**0.66**
72.00	72.00	1.10	134.33	0.52	0.49	**0.41**	**0.61**	**0.72**	**0.69**	75.00	75.00	1.11	141.67	0.47	0.46	**0.41**	**0.65**	**0.63**	**0.61**
96.00	96.00	0.98	112.50	0.53	0.51	**0.32**	**0.59**	**0.56**	**0.53**	93.00	93.00	0.85	108.50	0.45	0.50	**0.28**	**0.56**	**0.48**	**0.54**
83.00	83.00	0.52	68.47	0.51	0.42	**0.18**	**0.33**	**0.62**	**0.50**	87.00	87.00	0.47	76.10	0.44	0.32	**0.16**	**0.38**	**0.51**	**0.36**
70.00	70.00	1.06	146.17	0.45	0.40	**0.40**	**0.65**	**0.64**	**0.57**	74.00	74.00	1.16	145.83	0.51	0.45	**0.43**	**0.67**	**0.68**	**0.60**
82.00	82.00	0.70	87.47	0.52	0.44	**0.25**	**0.42**	**0.63**	**0.54**	82.00	82.00	0.37	72.10	0.35	0.26	**0.13**	**0.35**	**0.42**	**0.32**
73.00	73.00	0.29	53.93	0.54	0.18	**0.11**	**0.25**	**0.74**	**0.25**	73.00	73.00	0.33	66.77	0.38	0.26	**0.12**	**0.30**	**0.52**	**0.36**
74.00	74.00	1.06	115.83	0.54	0.64	**0.39**	**0.53**	**0.73**	**0.87**	76.00	76.00	1.04	127.67	0.47	0.52	**0.38**	**0.59**	**0.62**	**0.68**
77.00	78.00	0.98	114.00	0.51	0.56	**0.35**	**0.53**	**0.66**	**0.72**	82.00	82.00	0.90	121.50	0.46	0.41	**0.32**	**0.59**	**0.57**	**0.50**
88.00	88.00	0.75	76.80	0.59	0.59	**0.25**	**0.38**	**0.67**	**0.67**	88.00	88.00	0.49	71.17	0.39	0.44	**0.17**	**0.36**	**0.44**	**0.50**
83.00	82.00	0.94	107.17	0.47	0.59	**0.33**	**0.52**	**0.57**	**0.71**	84.00	82.00	0.98	107.50	0.51	0.55	**0.34**	**0.52**	**0.62**	**0.66**
67.00	67.00	0.64	95.60	0.42	0.36	**0.25**	**0.42**	**0.63**	**0.54**	68.00	68.00	0.84	131.17	0.42	0.35	**0.32**	**0.58**	**0.62**	**0.51**
90.00	91.00	0.52	69.40	0.40	0.52	**0.18**	**0.35**	**0.44**	**0.57**	88.00	92.00	0.58	92.33	0.42	0.32	**0.20**	**0.47**	**0.47**	**0.36**
86.00	86.00	0.55	79.68	0.26	0.52	**0.19**	**0.39**	**0.30**	**0.61**	87.00	87.00	0.57	79.77	0.42	0.52	**0.20**	**0.40**	**0.48**	**0.60**
69.00	69.00	1.19	117.13	0.63	0.63	**0.46**	**0.52**	**0.91**	**0.92**	72.00	72.00	1.32	157.83	0.46	0.52	**0.49**	**0.71**	**0.64**	**0.73**
71.00	71.00	1.04	115.83	0.57	0.51	**0.39**	**0.52**	**0.81**	**0.72**	72.00	72.00	0.98	121.17	0.50	0.47	**0.37**	**0.55**	**0.70**	**0.65**
82.00	83.00	1.26	115.17	0.63	0.68	**0.44**	**0.56**	**0.76**	**0.82**	83.00	83.00	1.20	116.00	0.63	0.62	**0.42**	**0.56**	**0.76**	**0.75**
										83.00	83.00	0.45	97.85	0.25	0.31	**0.16**	**0.47**	**0.31**	**0.38**
84.00	84.00	0.82	93.45	0.46	0.62	**0.29**	**0.46**	**0.54**	**0.73**	84.00	84.00	0.93	105.83	0.52	0.55	**0.33**	**0.52**	**0.62**	**0.65**

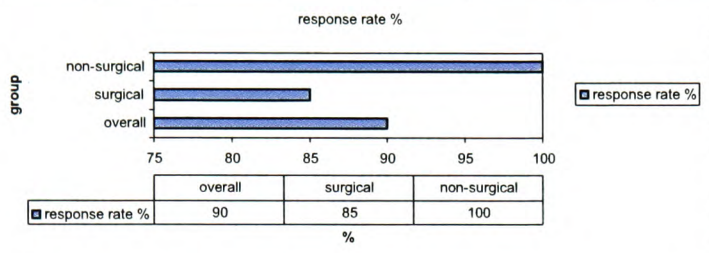
FILE 11

OVERALL PHYSIOTHERAPY QUESTIONNAIRE RESULTS

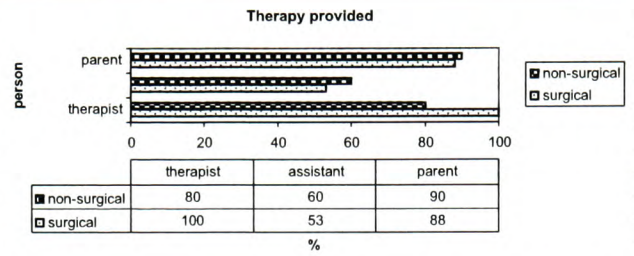
RESULTS		Q given	Q returned	% response														
response rate		30	27	90														
surgical		20	17	85														
no-surgical		10	10	100														
physio YES		27	27	100	by therapist	%	by assistant	%	by parent	%								
surgical		17	17	100	17	100	9	53	15	88								
no-surgical		10	10	100	8	80	6	60	9	90								
frequency		therapist	assistant	parent	Frequency for surgical depends on recovery: weekly sessions until 6 m post-op with decreasing frequency to									down even to 1 session per term				
surgical	min	1/12w	1/w	1/d	for participants approaching adulthood physiotherapy is reduced down to none or 2/yearly for monitoring													
	max	1/d	1/d	2/d														
no-surgical	min	2/24w	3/w	2/w	with 2 children having no physio for the duration of the study													
	max	1/w	1/d	1/d														
duration		therapist	assistant	parent														
surgical	min	20 min	15 min	15 min														
no-surgical	max	120 min	60 min	60 min														
	mean	53	28	30														
	therapist						**assistant**						**parent**					
the above	frequency	surgical	no-surgical	duration	surgical	no-surgical	frequency	surgical	no-surgical	duration	surgical	no-surgical	frequency	surgical	no-surgical	duration	surgical	no-surgical
in detail	1/d			15-20 min			1/d			15-20 min			1/d			15-20 min		
	>1/w			15-30 min			>1/w			15-30 min			>1/w			15-30 min		
	1/w			30-45 min			1/w			30-45 min			1/w			30-45 min		
	>1/4w			>45			>1/4w			>45			>1/4w			>45		
	<1/4w						<1/4w						<1/4w					
activity		hydrother	%	horse	%	PE	%	OT	%	other	%							
surgical	17	5	29	0	0	11	65	3	18	12	71		swimming, football, cycling, golf etc					
no-surgical	10	0	0	4	40	9	90	3	30	9	90							
activ freq	min	3/m		1/w		1/w		1/year										
	max	1/w				2/w		3>/12w										
type		bobath	%	CE	%	eclectic	%	musclul	%	other	%							
surgical	17	1	6	0	0	10	59	17	100	1	6	PNF						
no-surgical	10	2	20	1	10	8	80	0	0	0	0							
		4 surg pre-op		1 surg pre-op		3 surg pre-op												
exercises		stretching	%	MH	%	positioning	%	Strengthen	%	Strength resist	%	gross mot	%	other				
surgical	17	17	100	11	65	11	65	16	94	10	59	13	76					
no-surgical	10	10	100	3	30	3	30	10	100	2	20	5	50					
										no weights used								
										3 surgical used weights but towards the end of the sudy								

OVERALL PHYSIOTHERAPY QUESTIONNAIRE RESULTS

	response rate %
overall	90
surgical	85
non-surgical	100



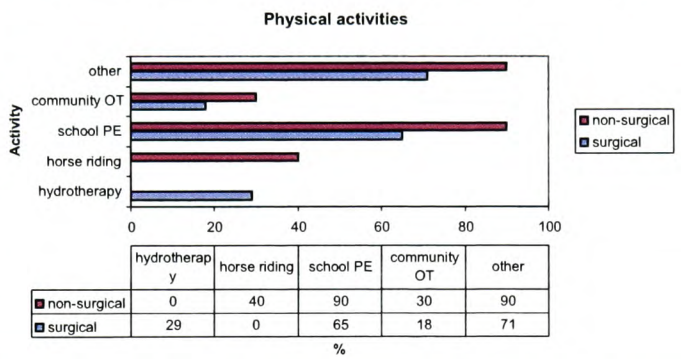
	overall	surgical	non-surgical
response rate %	90	85	100



	therapist	assistant	parent
non-surgical	80	60	90
surgical	100	53	88

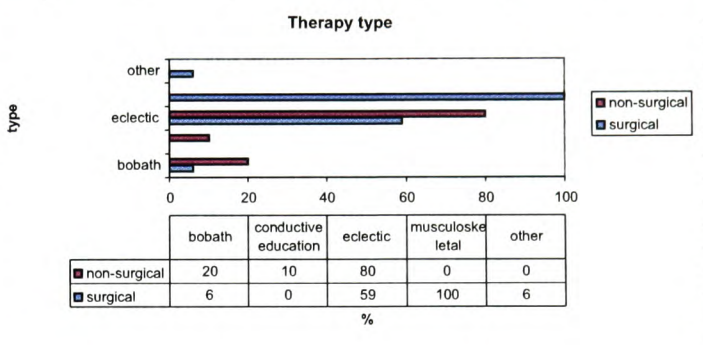
	therapist	assistant	parent
surgical	100	53	88
non-surgical	80	60	90

	hydrotherapy	horse riding	school PE	community	other
surgical	29	0	65	18	71
non-surgical	0	40	90	30	90



	hydrotherapy	horse riding	school PE	community OT	other
non-surgical	0	40	90	30	90
surgical	29	0	65	18	71

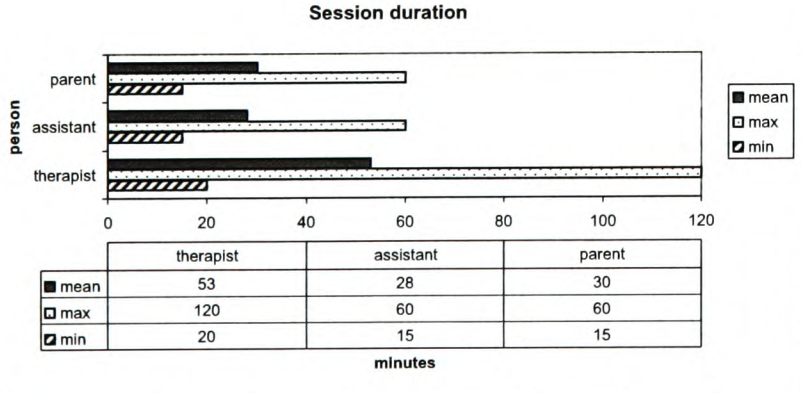
	bobath	conductive	eclectic	musculoske	other
surgical	6	0	59	100	6
non-surgical	20	10	80	0	0



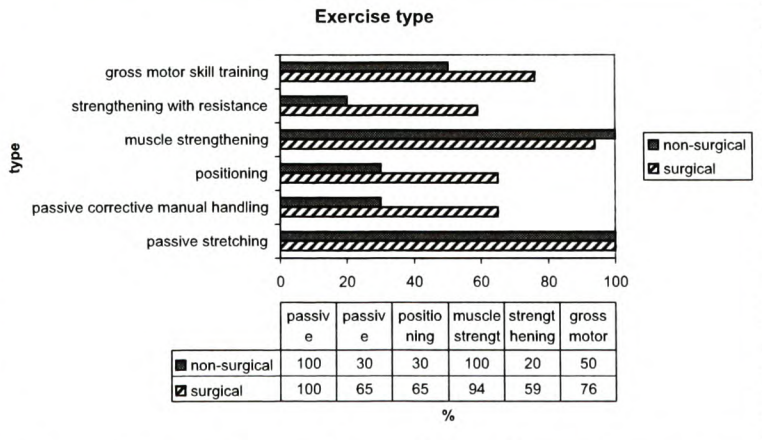
	bobath	conductive education	eclectic	musculoskeletal	other
non-surgical	20	10	80	0	0
surgical	6	0	59	100	6

	passive stretching	passive cor	positioning	muscle stre	strengtheni	gross motor skill training
surgical	100	65	65	94	59	76
non-surgical	100	30	30	100	20	50

duration	therapist	assistant	parent
min	20	15	15
max	120	60	60
mean	53	28	30



	therapist	assistant	parent
mean	53	28	30
max	120	60	60
min	20	15	15



	passive	passive	positioning	muscle strengt	strengthening	gross motor
non-surgical	100	30	30	100	20	50
surgical	100	65	65	94	59	76

FILE 12

LOG OF COMPLETED PHYSIOTHERAPY SESSIONS

Code	Initials	DOB	SURGERY DATE	No sessions	Start date	End date	Physio										
MS01	LF	31/05/90	10/01/2002	16	19/06/2002	24/07/2002	maria										
MS02	AW	29/10/92	31/01/2002	18	21/08/2002	11/10/2002	maria										
MS03	HM	09/01/92	25/04/2002	18	14/11/2002	23/12/2002	maria										
MS04	AH	22/11/86	13/06/2002	14	22/01/2003	21/02/2003	LSA	supervised by maria twice			reason = very far						
MS05	HA	28/06/89	23/01/2003	18	26/08/2003	09/10/2003	maria										
MS07	ChrisM	12/11/91	02/01/2003	18	27/08/2003	03/10/2003	maria										
MS08	CM	30/12/87	06/02/2003	17	05/01/2004	21/02/2004	cindy and maria										
MS09	ML	01/06/90	13/03/2003	17	03/10/2003	06/12/2003	maria										
MS10	YJ	02/10/92	27/03/2003	18	18/11/2003	23/12/2003	maria										
MS11	OL	16/09/94	01/05/2003	15	11/11/2003	18/11/2003	helen and maria										
MS12	QS	11/11/89	13/10/2003	18	26/04/2004	09/06/2004	maria										
MS13	SC	30/12/87	12/06/2003	16	11/11/2003	20/12/2003	helen and maria										
MS14	KM	10/01/92	19/06/2003	15	12/01/2004	20/02/2004	maria										
MS15	OS	28/07/89	24/07/2003	15	12/01/2004	26/02/2004	maria										
MS16	BB	24/09/87	04/09/2003	17	22/03/2004	04/05/2004	maria										
MS17	SVB	01/12/95	18/09/2003	17	12/05/2004	20/06/2004	maria										
MS18	ES	25/07/92	25/09/2003	16	11/05/2004	23/06/2004	maria										
MS19	EH	13/07/89	11/12/2003	18	23/07/2004	03/09/2004	cindy										
MS20	DB	10/06/92	08/01/2004		19/08/2004		maria supervised one, patient did not return log sheet and DNA 6mll testing = assumed not received physio										very far
MS21	EC	10/11/88	12/02/2004	18	16/08/2004	26/09/2004	maria										
ave				**17**													
min				**14**													
max				**18**													

FILE 13

MINIMISATION FILE

	date rand	dob	age	trt	agegp	sex	approx age	gmfcs	balance or randc	coded trt
MS01	23/05/2002	31/05/1990	12	normal	10 to 14	f	12.00	iii	r	b
MS02	23/05/2002	29/10/1992	9	intensive	<10	m	10.00	iii	r(added gfm late	a
MS03	03/09/2002	09/01/1992	10	intensive	10 to 14	f	10.00	ii	b	a
MS04	03/09/2002	22/11/1986	15	normal	>15	m	16.00	ii	b	b
MS05	17/03/2003	25/01/1991	12	normal	10 to 14	f	12.00	iii	r	b
MS06	17/03/2003	28/06/1989	13	intensive	10 to 14	f	14.00	ii	b	a
MS07	17/03/2003	12/11/1991	11	normal	10 to 14	m	12.00	ii	b	b
MS08	17/03/2003	30/12/1987	15	intensive	>15	m	16.00	iii	b	a
MS09	05/09/2003	01/06/1990	13	intensive	10 to 14	f	13.00	iii	b	a
MS10	05/09/2003	02/10/1992	10	normal	10 to 14	f	11.00	ii	b	b
MS11	23/10/2003	16/09/1994	9	normal	<10	m	9.00	ii	b	b
MS13	23/10/2003	30/12/1987	15	intensive	>15	f	16.00	ii	b	a
MS14	23/10/2003	10/01/1992	11	normal	10 to 14	f	11.00	iii	b	b
MS15	06/01/2004	28/07/1989	14	intensive	10 to 14	m	15.00	iii	b	a
MS16	18/02/2004	24/09/1987	16	normal	>15	m	17.00	iii	b	b
MS12	29/03/2004	11/11/1989	14	intensive	10 to 14	m	15.00	iii	b	a
MS18	29/04/2004	25/07/1992	11	intensive	10 to 14	m	12.00	ii	b	a
MS17	03/05/2004	01/12/1995	8	normal	<10	f	9.00	ii	r	b
MS19	11/06/2004	13/07/1989	14	intensive	10 to 14	f	15.00	i	r	a
MS20	11/06/2004	10/06/1992	12	normal	10 to 14	m	12.00	ii	b	b
MS21	11/06/2004	10/11/1988	15	intensive	>15	f	16.00	ii	b	a

Count of gmfcs			trt
sex	agegp	gmfcs	intensive	normal
f	<10	ii		1
	10 to 14	ii	2	1
		iii	1	3
		i	1	
	>15	ii	2	
m	<10	ii		1
		iii	1	
	10 to 14	ii	1	2
		iii	2	
	>15	ii		1
		iii	1	1
Grand Total			11	10

Count of gmfcs	trt		
agegp	intensive	normal	Grand Total
<10	1	2	3
10 to 14	7	6	13
>15	3	2	5
Grand Total	11	10	21

Count of gmfcs	trt		
sex	intensive	normal	Grand Total
m	5	5	10
f	6	5	11
Grand Total	11	10	21

Count of sex	trt		
gmfcs	intensive	normal	Grand Total
i	1		1
ii	5	6	11
iii	5	4	9
Grand Total	11	10	21

FILE 13

MINIMISATION FILE

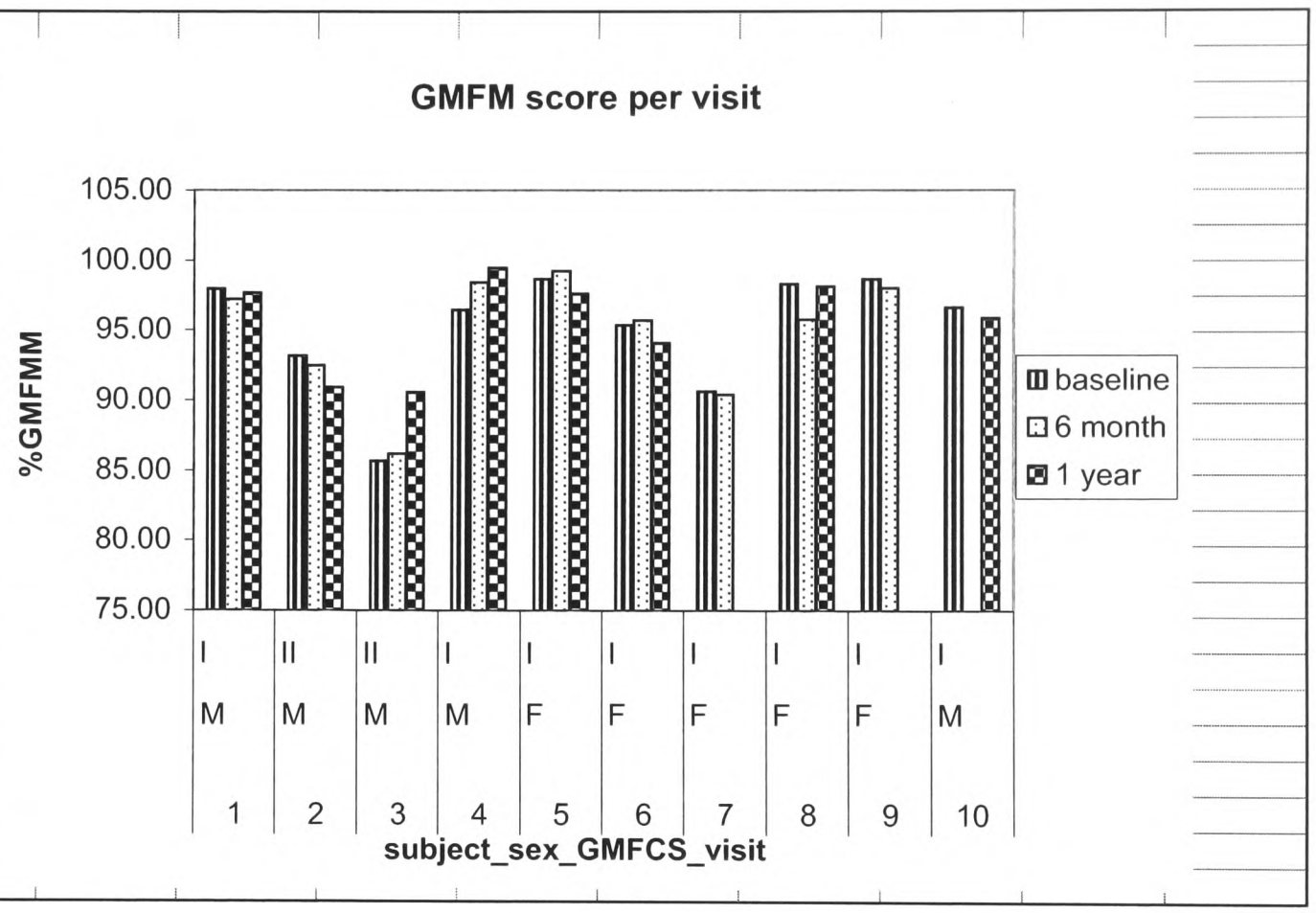
	date rand	dob	age	trt	agegp	sex	age crud	gmfcs	balance or ran	coded trt
MS01	23/05/2002	31/05/1990	12	normal	10 to 14	f	12.00	iii	r	b
MS02	23/05/2002	29/10/1992	9	intensive	<10	m	10.00	iii	r(added gfm la	a
MS03	03/09/2002	09/01/1992	10	intensive	10 to 14	f	10.00	ii	b	a
MS04	03/09/2002	22/11/1986	15	normal	>15	m	16.00	ii	b	b
MS06	17/03/2003	28/06/1989	13	intensive	10 to 14	f	14.00	ii	b	a
MS07	17/03/2003	12/11/1991	11	normal	10 to 14	m	12.00	ii	b	b
MS08	17/03/2003	30/12/1987	15	intensive	>15	m	16.00	iii	b	a
MS09	05/09/2003	01/06/1990	13	intensive	10 to 14	f	13.00	iii	b	a
MS10	05/09/2003	02/10/1992	10	normal	10 to 14	f	11.00	ii	b	b
MS11	23/10/2003	16/09/1994	9	normal	<10	m	9.00	ii	b	b
MS13	23/10/2003	30/12/1987	15	intensive	>15	f	16.00	ii	b	a
MS14	23/10/2003	10/01/1992	11	normal	10 to 14	f	11.00	iii	b	b
MS15	06/01/2004	28/07/1989	14	intensive	10 to 14	m	15.00	iii	b	a
MS16	18/02/2004	24/09/1987	16	normal	>15	m	17.00	iii	b	b
MS12	29/03/2004	11/11/1989	14	intensive	10 to 14	m	15.00	iii	b	a
MS18	29/04/2004	25/07/1992	11	intensive	10 to 14	m	12.00	ii	b	a
MS17	03/05/2004	01/12/1995	8	normal	<10	f	9.00	ii	r	b
MS19	11/06/2004	13/07/1989	14	intensive	10 to 14	f	15.00	i	r	a
MS20	11/06/2004	10/06/1992	12	normal	10 to 14	m	12.00	ii	b	b
MS21	11/06/2004	10/11/1988	15	intensive	>15	f	16.00	ii	b	a

Count of gmfcs			trt		
sex	agegp	gmfcs	intensive	normal	Grand Total
f	<10	ii		1	1
	>15	ii	2		2
	10 to 14	i	1		1
		ii	2	1	3
		iii	1	2	3
m	<10	ii		1	1
		iii	1		1
	>15	ii		1	1
		iii	1	1	2
	10 to 14	ii	1	2	3
		iii	2		2
Grand Total			11	9	20

FILE 14

GROUP A GMFM RESULTS FOR ALL VISITS

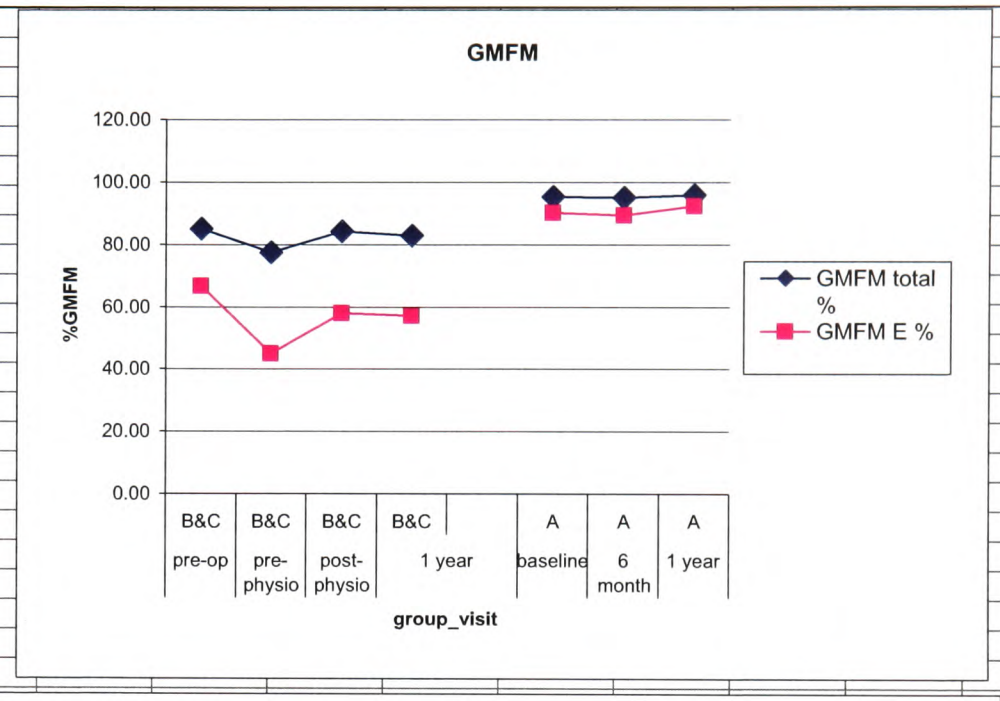
subject	SEX	GMFCS1	baseline	6 month	1 year
1	M	I	97.98	97.20	97.68
2	M	II	93.17	92.48	90.92
3	M	II	85.67	86.17	90.56
4	M	I	96.44	98.45	99.44
5	F	I	98.65	99.21	97.63
6	F	I	95.36	95.72	94.11
7	F	I	90.63	90.38	
8	F	I	98.31	95.77	98.14
9	F	I	98.66	98.03	
10	M	I	96.64		95.89
MEAN			**95.15**	**94.82**	**95.55**
			gmfm1-5	gmfm2-5	gmfm3-5
			100.00	100.00	100.00
			84.72	79.17	80.55
			66.66	75.00	83.33
			95.83	97.22	97.22
			95.83	98.61	97.22
			81.94	88.88	84.72
			84.72	73.61	,
			96.66	91.67	95.83
			97.22	97.22	,
			95.83	,	97.22
MEAN			**89.94**	**89.04**	**92.01**

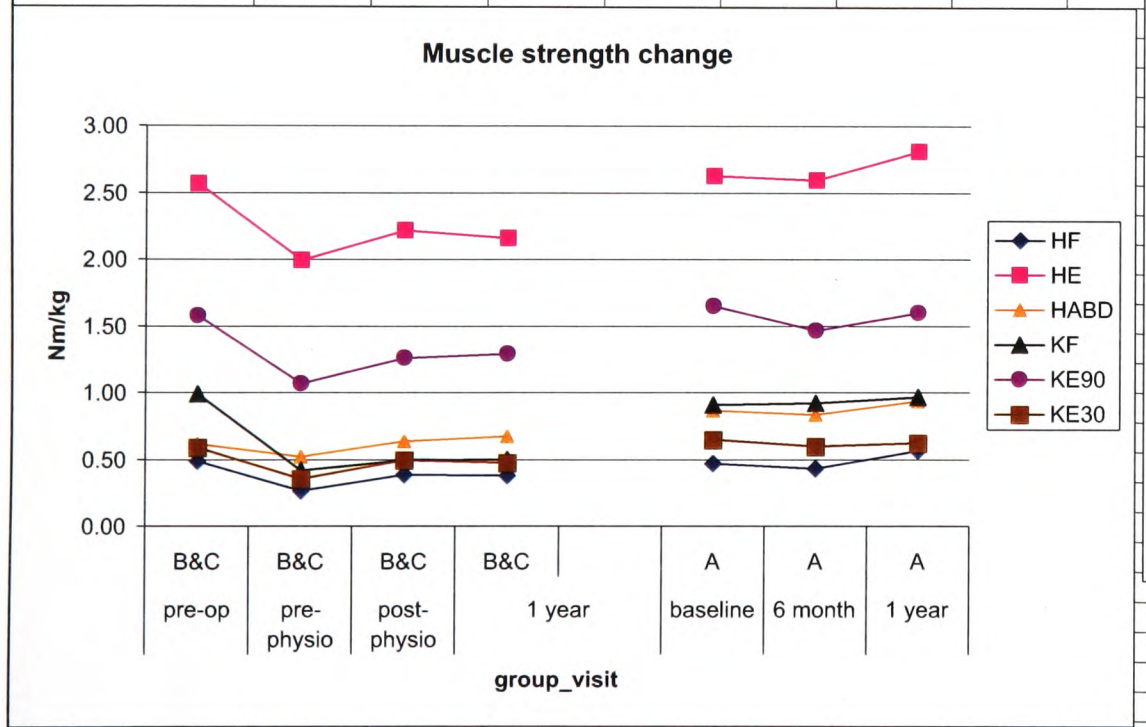


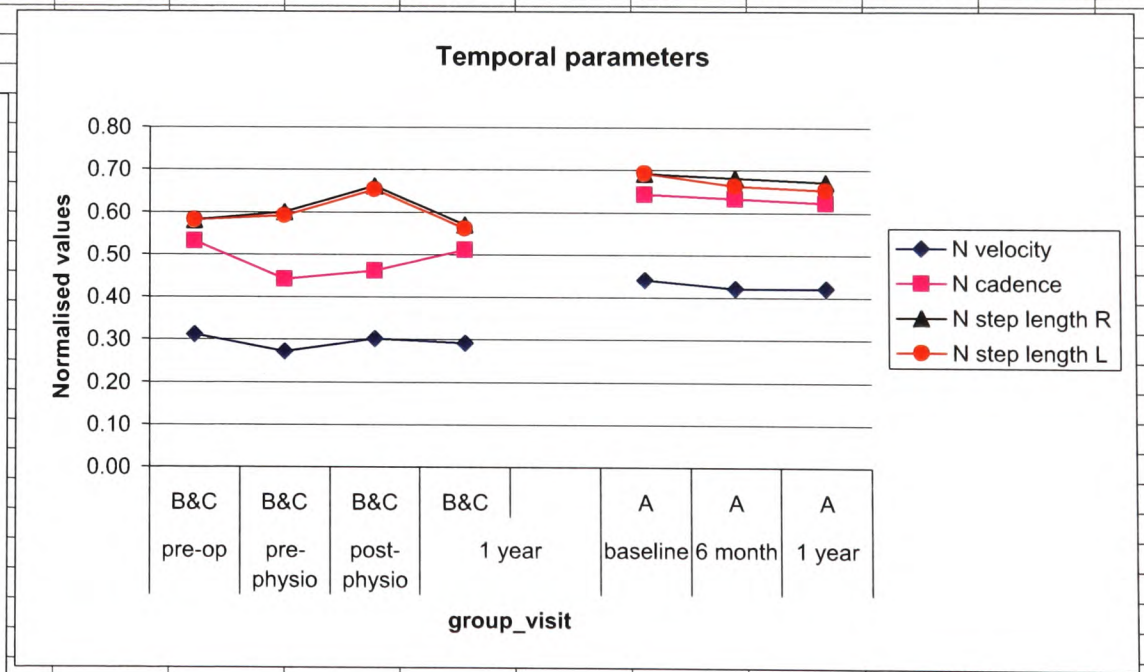
FILE 15

MEAN OVERALL STRENGTH, FUNCTION AND TEMPORAL DATA FOR ALL VISITS AND STUDY GROUPS (A,B,C)

Gross motor function	pre-op	pre-physio	post-physio	1 year		baseline	6 month	1 year
	B&C	B&C	B&C	B&C		A	A	A
GMFM total %	84.90	77.20	83.90	82.50		95.15	94.82	95.55
GMFM E %	66.30	44.80	57.70	56.80		89.94	89.04	92.01
Temporal parameters	pre-op	pre-physio	post-physio	1 year		baseline	6 month	1 year
	B&C	B&C	B&C	B&C		A	A	A
N velocity	0.31	0.27	0.30	0.29		0.44	0.42	0.42
N cadence	0.53	0.44	0.46	0.51		0.64	0.63	0.62
N step length R	0.58	0.60	0.66	0.57		0.69	0.68	0.67
N step length L	0.58	0.59	0.65	0.56		0.69	0.66	0.65
mgroup	pre-op	pre-physio	post-physio	1 year		baseline	6 month	1 year
	B&C	B&C	B&C	B&C		A	A	A
HF	0.48	0.26	0.38	0.38		0.46	0.42	0.56
HE	2.56	1.99	2.21	2.15		2.62	2.59	2.80
HABD	0.61	0.51	0.63	0.67		0.86	0.83	0.93
KF	0.99	0.41	0.49	0.50		0.90	0.92	0.96
KE90	1.57	1.06	1.25	1.29		1.64	1.46	1.59
KE30	0.59	0.35	0.49	0.47		0.64	0.59	0.62







APPENDIX SIX

SPSS files

1. Analysis of Covariance

    a. Strength and GMFM data between groups B&C for visits pre-physiotherapy versus post-physiotherapy

    b. Temporal and GMFM data between groups B&C for visits pre-physiotherapy versus post-physiotherapy

    c. Gait analysis data between groups B&C for visits pre-physiotherapy versus post-physiotherapy

    d. Strength and GMFM data between groups B&C for visits pre-physiotherapy versus 1 year

2. Paired t tests

    a. Group A (strength, function, gait and temporal parameters between visits)

    b. Group B & C (strength, function, gait and temporal parameters between visits)

FILE 1a

Univariate Analysis of Variance of strength and GMFM data between groups B and C for visits pre-physio vs post-physio

Parameter Estimates

Dependent Variable: GMFM3

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	37,924	16,382	2,315	,038	2,533	73,314
[SEX=f]	-,136	2,343	-,058	,954	-5,197	4,925
[SEX=m]	0[a]	,	,	,	,	,
[GMFCS=II]	3,984	4,358	,914	,377	-5,431	13,399
[GMFCS=III]	0[a]	,	,	,	,	,
[GROUP=B]	,906	2,763	,328	,748	-5,062	6,874
[GROUP=C]	0[a]	,	,	,	,	,
GMFM2	,566	,159	3,551	,004	,221	,910
AGE	-2,59E-02	,540	-,048	,962	-1,192	1,140

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: GMFM35

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	29,236	19,540	1,496	,158	-12,977	71,449
[SEX=f]	-3,082	4,735	-,651	,526	-13,310	7,147
[SEX=m]	0[a]	,	,	,	,	,
[GMFCS=II]	20,555	9,997	2,056	,060	-1,041	42,152
[GMFCS=III]	0[a]	,	,	,	,	,
[GROUP=B]	,817	5,310	,154	,880	-10,654	12,288
[GROUP=C]	0[a]	,	,	,	,	,
AGE	-,381	1,150	-,331	,746	-2,866	2,105
GMFM25	,526	,178	2,951	,011	,141	,911

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: HF3

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	,140	,144	,973	,338	-,154	,435
[GROUP=B]	-,123	,048	-2,546	,016	-,221	-2,444E-02
[GROUP=C]	0[a]	,	,	,	,	,
[GMFCS=II]	4,437E-02	,048	,916	,367	-5,442E-02	,143
[GMFCS=III]	0[a]	,	,	,	,	,
[SEX=f]	-2,39E-02	,045	-,530	,600	-,116	6,824E-02
[SEX=m]	0[a]	,	,	,	,	,
HF2	,841	,181	4,642	,000	,472	1,211
AGE	4,437E-03	,010	,446	,659	-1,587E-02	2,474E-02

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: HE3

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	-,129	,351	-,366	,717	-,845	,588
[GROUP=B]	-7,92E-02	,116	-,685	,499	-,315	,157
[GROUP=C]	0[a]	,	,	,	,	,
[GMFCS=II]	,374	,116	3,223	,003	,137	,611
[GMFCS=III]	0[a]	,	,	,	,	,
[SEX=f]	-,137	,120	-1,141	,263	-,382	,108
[SEX=m]	0[a]	,	,	,	,	,
AGE	7,875E-02	,027	2,881	,007	2,300E-02	,134
HE2	,596	,096	6,191	,000	,400	,792

a. This parameter is set to zero because it is redundant.

Tests of Between-Subjects Effects

Dependent Variable: HAB3

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	2,675[a]	5	,535	46,382	,000
Intercept	1,651E-03	1	1,651E-03	,143	,708
GROUP	1,430E-02	1	1,430E-02	1,240	,275
GMFCS	,205	1	,205	17,773	,000
SEX	9,590E-03	1	9,590E-03	,831	,369
AGE	7,082E-02	1	7,082E-02	6,139	,019
HAB2	,422	1	,422	36,605	,000
Error	,335	29	1,154E-02		
Total	17,002	35			
Corrected Total	3,010	34			

a. R Squared = ,889 (Adjusted R Squared = ,870)

Parameter Estimates

Dependent Variable: HAB3

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	-,153	,121	-1,269	,215	-,400	9,364E-02
[GROUP=B]	-4,69E-02	,042	-1,113	,275	-,133	3,929E-02
[GROUP=C]	0[a]	,	,	,	,	,
[GMFCS=II]	,237	,056	4,216	,000	,122	,352
[GMFCS=III]	0[a]	,	,	,	,	,
[SEX=f]	3,398E-02	,037	,912	,369	-4,224E-02	,110
[SEX=m]	0[a]	,	,	,	,	,
AGE	2,270E-02	,009	2,478	,019	3,962E-03	4,143E-02
HAB2	,690	,114	6,050	,000	,457	,924

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: KF3

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	-8,56E-02	,096	-,889	,381	-,282	,111
[GROUP=B]	-1,32E-03	,032	-,041	,967	-6,612E-02	6,349E-02
[GROUP=C]	0[a]	,	,	,	,	,
[GMFCS=II]	9,409E-02	,041	2,308	,028	1,095E-02	,177
[GMFCS=III]	0[a]	,	,	,	,	,
[SEX=f]	2,040E-02	,031	,662	,513	-4,244E-02	8,323E-02
[SEX=m]	0[a]	,	,	,	,	,
AGE	1,248E-02	,007	1,845	,075	-1,319E-03	2,628E-02
KF2	,860	,099	8,649	,000	,657	1,063

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: KE903

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	-,178	,184	-,964	,342	-,554	,198
[GROUP=B]	-1,94E-02	,061	-,318	,752	-,144	,105
[GROUP=C]	0[a]	,	,	,	,	,
[GMFCS=II]	,131	,069	1,894	,068	-1,011E-02	,273
[GMFCS=III]	0[a]	,	,	,	,	,
[SEX=f]	2,215E-02	,058	,383	,705	-9,591E-02	,140
[SEX=m]	0[a]	,	,	,	,	,
AGE	2,332E-02	,015	1,602	,119	-6,361E-03	5,299E-02
KE902	,966	,088	10,984	,000	,787	1,145

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: KE303

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	1,362E-02	,103	,132	,896	-,199	,226
[GROUP=B]	-7,78E-02	,038	-2,030	,054	-,157	1,315E-03
[GROUP=C]	0[a]	,	,	,	,	,
[GMFCS=II]	,119	,034	3,556	,002	5,001E-02	,188
[GMFCS=III]	0[a]	,	,	,	,	,
[SEX=f]	-1,51E-02	,030	-,503	,620	-7,694E-02	4,679E-02
[SEX=m]	0[a]	,	,	,	,	,
AGE	9,194E-03	,008	1,224	,233	-6,304E-03	2,469E-02
KE302	,860	,072	11,887	,000	,711	1,010

a. This parameter is set to zero because it is redundant.

FILE 1b

Univariate Analysis of Variance of temporal and GMFM data, between groups B and C for visits pre-physio vs post-physio

Parameter Estimates

Dependent Variable: NVELOC3

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval		Partial Eta Squared
					Lower Bound	Upper Bound	
Intercept	,144	,125	1,155	,269	-,126	,414	,093
[GROUP=B]	-2,10E-02	,025	-,835	,419	-7,543E-02	3,336E-02	,051
[GROUP=C]	0[a]	,	,	,	,	,	,
[SEX=f]	-1,30E-02	,025	-,527	,607	-6,611E-02	4,020E-02	,021
[SEX=m]	0[a]	,	,	,	,	,	,
[GMFCS=II]	4,467E-02	,041	1,077	,301	-4,492E-02	,134	,082
[GMFCS=III]	0[a]	,	,	,	,	,	,
AGE	-3,58E-03	,006	-,556	,588	-1,752E-02	1,036E-02	,023
NVELOC2	,735	,226	3,246	,006	,246	1,224	,448

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: NCAD3

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval		Partial Eta Squared
					Lower Bound	Upper Bound	
Intercept	,149	,104	1,430	,176	-7,596E-02	,374	,136
[GROUP=B]	-6,80E-03	,022	-,314	,758	-5,351E-02	3,992E-02	,008
[GROUP=C]	0[a]	,	,	,	,	,	,
[SEX=f]	-2,87E-02	,022	-1,327	,207	-7,548E-02	1,803E-02	,119
[SEX=m]	0[a]	,	,	,	,	,	,
[GMFCS=II]	5,048E-02	,043	1,161	,266	-4,344E-02	,144	,094
[GMFCS=III]	0[a]	,	,	,	,	,	,
AGE	-6,04E-04	,004	-,136	,894	-1,020E-02	8,994E-03	,001
NCAD2	,715	,203	3,523	,004	,276	1,153	,488

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: NSTEPR3

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval		Partial Eta Squared
					Lower Bound	Upper Bound	
Intercept	,954	,254	3,760	,002	,406	1,502	,521
[GROUP=B]	-,105	,051	-2,069	,059	-,214	4,657E-03	,248
[GROUP=C]	0[a]	,	,	,	,	,	,
[SEX=f]	2,239E-02	,047	,476	,642	-7,916E-02	,124	,017
[SEX=m]	0[a]	,	,	,	,	,	,
[GMFCS=II]	3,315E-02	,049	,672	,513	-7,339E-02	,140	,034
[GMFCS=III]	0[a]	,	,	,	,	,	,
AGE	-3,50E-02	,012	-2,834	,014	-6,161E-02	-8,309E-03	,382
NSTEPR2	,285	,196	1,453	,170	-,138	,708	,140

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: NSTEPL3

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval		Partial Eta Squared
					Lower Bound	Upper Bound	
Intercept	,185	,175	1,056	,310	-,193	,563	,079
[GROUP=B]	-4,80E-02	,041	-1,178	,260	-,136	4,008E-02	,096
[GROUP=C]	0[a]	,	,	,	,	,	,
[SEX=f]	4,572E-02	,041	1,121	,283	-4,242E-02	,134	,088
[SEX=m]	0[a]	,	,	,	,	,	,
[GMFCS=II]	2,039E-02	,046	,442	,666	-7,928E-02	,120	,015
[GMFCS=III]	0[a]	,	,	,	,	,	,
AGE	-5,85E-03	,009	-,658	,522	-2,506E-02	1,335E-02	,032
NSTEPL2	,888	,156	5,706	,000	,552	1,225	,715

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: GMFM3

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval		Partial Eta Squared
					Lower Bound	Upper Bound	
Intercept	37,924	16,382	2,315	,038	2,533	73,314	,292
[GROUP=B]	,906	2,763	,328	,748	-5,062	6,874	,008
[GROUP=C]	0[a]	,	,	,	,	,	,
[SEX=f]	-,136	2,343	-,058	,954	-5,197	4,925	,000
[SEX=m]	0[a]	,	,	,	,	,	,
[GMFCS=II]	3,984	4,358	,914	,377	-5,431	13,399	,060
[GMFCS=III]	0[a]	,	,	,	,	,	,
AGE	-2,59E-02	,540	-,048	,962	-1,192	1,140	,000
GMFM2	,566	,159	3,551	,004	,221	,910	,492

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: GMFM53

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval		Partial Eta Squared
					Lower Bound	Upper Bound	
Intercept	29,236	19,540	1,496	,158	-12,977	71,449	,147
[GROUP=B]	,817	5,310	,154	,880	-10,654	12,288	,002
[GROUP=C]	0[a]	,	,	,	,	,	,
[SEX=f]	-3,082	4,735	-,651	,526	-13,310	7,147	,032
[SEX=m]	0[a]	,	,	,	,	,	,
[GMFCS=II]	20,555	9,997	2,056	,060	-1,041	42,152	,245
[GMFCS=III]	0[a]	,	,	,	,	,	,
AGE	-,381	1,150	-,331	,746	-2,866	2,105	,008
GMFM52	,526	,178	2,951	,011	,141	,911	,401

a. This parameter is set to zero because it is redundant.

FILE 1c

Univariate Analysis of Variance of gait analysis data for groups B & C for visits pre-physio vs post-physio

Parameter Estimates

Dependent Variable: RPT3

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	4,359	2,565	1,700	,099	-,865	9,584
[GROUP=B]	7,185E-02	,765	,094	,926	-1,486	1,630
[GROUP=C]	0[a]	,	,	,	,	,
[GMFCS=II]	-1,516	,759	-1,998	,054	-3,061	2,929E-02
[GMFCS=III]	0[a]	,	,	,	,	,
[SEX=f]	1,866	,723	2,579	,015	,392	3,339
[SEX=m]	0[a]	,	,	,	,	,
RPT2	,801	,136	5,889	,000	,524	1,077
AGE	-,239	,158	-1,509	,141	-,561	8,340E-02

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: RHF3

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	18,334	8,445	2,171	,037	1,131	35,536
[GROUP=B]	-,955	1,851	-,516	,609	-4,726	2,815
[GROUP=C]	0[a]	,	,	,	,	,
[GMFCS=II]	1,209	1,791	,675	,505	-2,439	4,857
[GMFCS=III]	0[a]	,	,	,	,	,
[SEX=f]	1,985	1,762	1,126	,268	-1,605	5,575
[SEX=m]	0[a]	,	,	,	,	,
AGE	-,419	,401	-1,046	,304	-1,237	,398
RHF2	,689	,111	6,177	,000	,461	,916

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: RKF3

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	11,090	4,428	2,505	,018	2,071	20,109
[GROUP=B]	-,381	1,350	-,282	,780	-3,131	2,370
[GROUP=C]	0[a]	,	,	,	,	,
[GMFCS=II]	-,263	1,320	-,199	,843	-2,952	2,426
[GMFCS=III]	0[a]	,	,	,	,	,
[SEX=f]	-,117	1,295	-,090	,929	-2,755	2,521
[SEX=m]	0[a]	,	,	,	,	,
AGE	-1,45E-02	,279	-,052	,959	-,583	,554
RKF2	,738	,044	16,956	,000	,649	,826

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: RAD3

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	1,207	4,199	,287	,776	-7,345	9,759
[GROUP=B]	-1,713	1,333	-1,285	,208	-4,428	1,002
[GROUP=C]	0[a]	,	,	,	,	,
[GMFCS=II]	,940	1,352	,695	,492	-1,814	3,695
[GMFCS=III]	0[a]	,	,	,	,	,
[SEX=f]	,928	1,434	,647	,522	-1,993	3,850
[SEX=m]	0[a]	,	,	,	,	,
AGE	-4,54E-02	,272	-,167	,868	-,599	,508
RAD2	,982	,099	9,949	,000	,781	1,183

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: MHES3

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	1,218	6,426	,190	,851	-11,871	14,306
[GROUP=B]	,888	2,175	,408	,686	-3,543	5,319
[GROUP=C]	0[a]	,	,	,	,	,
[GMFCS=II]	-2,562	2,275	-1,126	,268	-7,195	2,071
[GMFCS=III]	0[a]	,	,	,	,	,
[SEX=f]	,507	2,015	,251	,803	-3,599	4,612
[SEX=m]	0[a]	,	,	,	,	,
AGE	,111	,442	,252	,803	-,789	1,011
MHES2	,751	,081	9,253	,000	,585	,916

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: MKES3

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	3,233	5,242	,617	,542	-7,445	13,912
[GROUP=B]	1,720	1,686	1,020	,316	-1,716	5,155
[GROUP=C]	0[a]	,	,	,	,	,
[GMFCS=II]	-,126	1,651	-,077	,939	-3,489	3,236
[GMFCS=III]	0[a]	,	,	,	,	,
[SEX=f]	-,677	1,620	-,418	,679	-3,978	2,623
[SEX=m]	0[a]	,	,	,	,	,
AGE	1,849E-03	,349	,005	,996	-,709	,713
MKES2	,848	,048	17,553	,000	,750	,947

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: MKFS3

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	-3,057	11,462	-,267	,791	-26,405	20,290
[GROUP=B]	1,645	2,129	,773	,445	-2,692	5,983
[GROUP=C]	0[a]	,	,	,	,	,
[GMFCS=II]	-,881	2,049	-,430	,670	-5,054	3,292
[GMFCS=III]	0[a]	,	,	,	,	,
[SEX=f]	-,991	2,021	-,490	,627	-5,107	3,125
[SEX=m]	0[a]	,	,	,	,	,
AGE	-2,12E-02	,441	-,048	,962	-,919	,876
MKFS2	1,055	,150	7,040	,000	,750	1,360

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: KFIC3

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	-7,50E-02	5,338	-,014	,989	-10,948	10,798
[GROUP=B]	2,622	1,732	1,514	,140	-,906	6,150
[GROUP=C]	0[a]	,	,	,	,	,
[GMFCS=II]	1,330	1,751	,760	,453	-2,236	4,896
[GMFCS=III]	0[a]	,	,	,	,	,
[SEX=f]	,737	1,541	,478	,636	-2,403	3,876
[SEX=m]	0[a]	,	,	,	,	,
AGE	4,321E-02	,338	,128	,899	-,645	,732
MKFIC2	,904	,064	14,224	,000	,774	1,033

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: RPO3

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	,183	2,625	,070	,945	-5,165	5,530
[GROUP=B]	-,480	,850	-,564	,577	-2,211	1,252
[GROUP=C]	0[a]	,	,	,	,	,
[GMFCS=II]	-,799	,812	-,984	,333	-2,453	,855
[GMFCS=III]	0[a]	,	,	,	,	,
[SEX=f]	1,129	,804	1,404	,170	-,509	2,768
[SEX=m]	0[a]	,	,	,	,	,
AGE	7,085E-02	,180	,393	,697	-,296	,438
RPO2	,885	,158	5,609	,000	,564	1,206

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: RPR3

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	3,514	3,817	,920	,364	-4,262	11,290
[GROUP=B]	1,179	1,199	,983	,333	-1,263	3,620
[GROUP=C]	0[a]	,	,	,	,	,
[GMFCS=II]	-1,529	1,120	-1,365	,182	-3,810	,752
[GMFCS=III]	0[a]	,	,	,	,	,
[SEX=f]	5,864	1,122	5,226	,000	3,578	8,150
[SEX=m]	0[a]	,	,	,	,	,
AGE	,117	,243	,480	,634	-,378	,612
RPR2	,526	,113	4,644	,000	,295	,756

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: MTR3

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	25,290	10,928	2,314	,027	3,029	47,550
[GROUP=B]	-3,847	3,597	-1,069	,293	-11,173	3,480
[GROUP=C]	0[a]	,	,	,	,	,
[GMFCS=II]	-4,300	3,509	-1,226	,229	-11,447	2,847
[GMFCS=III]	0[a]	,	,	,	,	,
[SEX=f]	-1,080	3,470	-,311	,758	-8,147	5,988
[SEX=m]	0[a]	,	,	,	,	,
AGE	-,926	,743	-1,247	,221	-2,439	,587
MTR2	-3,11E-03	,097	-,032	,975	-,202	,195

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: HEM3

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	360,286	243,153	1,482	,157	-152,723	873,295
[GROUP=B]	-90,068	100,357	-,897	,382	-301,802	121,666
[GROUP=C]	0[a]	,	,	,	,	,
[GMFCS=II]	163,648	125,052	1,309	,208	-100,189	427,484
[GMFCS=III]	0[a]	,	,	,	,	,
[SEX=f]	132,344	100,158	1,321	,204	-78,970	343,658
[SEX=m]	0[a]	,	,	,	,	,
AGE	-25,561	15,098	-1,693	,109	-57,416	6,293
HEM2	,831	,111	7,479	,000	,596	1,065

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: HFM3

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	-232,012	172,794	-1,343	,197	-596,575	132,551
[GROUP=B]	26,962	64,720	,417	,682	-109,585	163,509
[GROUP=C]	0[a]	,	,	,	,	,
[GMFCS=II]	85,048	91,943	,925	,368	-108,934	279,030
[GMFCS=III]	0[a]	,	,	,	,	,
[SEX=f]	-16,114	56,805	-,284	,780	-135,961	103,734
[SEX=m]	0[a]	,	,	,	,	,
AGE	-8,224	10,930	-,752	,462	-31,285	14,836
HFM2	,530	,202	2,619	,018	,103	,956

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: KEM3

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	-136,993	249,728	-,549	,590	-663,874	389,887
[GROUP=B]	151,692	77,114	1,967	,066	-11,005	314,389
[GROUP=C]	0[a]	,	,	,	,	,
[GMFCS=II]	154,753	108,859	1,422	,173	-74,918	384,425
[GMFCS=III]	0[a]	,	,	,	,	,
[SEX=f]	153,398	68,126	2,252	,038	9,665	297,131
[SEX=m]	0[a]	,	,	,	,	,
AGE	1,006	14,189	,071	,944	-28,930	30,942
KEM2	,874	,255	3,423	,003	,335	1,412

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: KFM3

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	-1,666	85,498	-,019	,985	-182,050	178,719
[GROUP=B]	19,050	38,646	,493	,628	-62,487	100,587
[GROUP=C]	0[a]	,	,	,	,	,
[GMFCS=II]	-133,028	53,266	-2,497	,023	-245,410	-20,646
[GMFCS=III]	0[a]	,	,	,	,	,
[SEX=f]	-29,250	34,059	-,859	,402	-101,107	42,608
[SEX=m]	0[a]	,	,	,	,	,
AGE	3,556	7,238	,491	,630	-11,715	18,827
KFM2	,689	,108	6,380	,000	,461	,917

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: MPFM3

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	100,645	111,530	,902	,379	-134,662	335,953
[GROUP=B]	36,902	35,508	1,039	,313	-38,013	111,817
[GROUP=C]	0[a]	,	,	,	,	,
[GMFCS=II]	201,643	49,856	4,045	,001	96,456	306,829
[GMFCS=III]	0[a]	,	,	,	,	,
[SEX=f]	-27,158	31,631	-,859	,403	-93,892	39,577
[SEX=m]	0[a]	,	,	,	,	,
AGE	2,262	6,318	,358	,725	-11,068	15,593
MPFM2	,728	,088	8,241	,000	,541	,914

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: MHAM3

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	18,888	150,215	,126	,901	-298,039	335,815
[GROUP=B]	13,880	56,824	,244	,810	-106,009	133,769
[GROUP=C]	0[a]	,	,	,	,	,
[GMFCS=II]	-26,124	89,563	-,292	,774	-215,086	162,838
[GMFCS=III]	0[a]	,	,	,	,	,
[SEX=f]	22,861	51,414	,445	,662	-85,613	131,335
[SEX=m]	0[a]	,	,	,	,	,
AGE	3,447	9,538	,361	,722	-16,676	23,570
MHAM2	,914	,244	3,745	,002	,399	1,430

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: MHP3

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	1,121	,415	2,699	,015	,245	1,998
[GROUP=B]	,230	,171	1,345	,196	-,131	,590
[GROUP=C]	0[a]	,	,	,	,	,
[GMFCS=II]	,115	,226	,510	,617	-,361	,592
[GMFCS=III]	0[a]	,	,	,	,	,
[SEX=f]	,119	,180	,663	,516	-,260	,498
[SEX=m]	0[a]	,	,	,	,	,
AGE	-5,74E-02	,027	-2,121	,049	-,114	-2,927E-04
MHP2	,576	,140	4,114	,001	,281	,872

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: MAP3

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	,563	,444	1,268	,222	-,374	1,499
[GROUP=B]	-,811	,207	-3,916	,001	-1,248	-,374
[GROUP=C]	0[a]	,	,	,	,	,
[GMFCS=II]	,585	,275	2,124	,049	3,994E-03	1,166
[GMFCS=III]	0[a]	,	,	,	,	,
[SEX=f]	,948	,243	3,895	,001	,435	1,462
[SEX=m]	0[a]	,	,	,	,	,
AGE	-,156	,035	-4,503	,000	-,229	-8,281E-02
MAP2	1,967	,222	8,862	,000	1,499	2,435

a. This parameter is set to zero because it is redundant.

FILE 1d

Univariate Analysis of Variance of strength and GMFM data between groups B and C for visits pre-physio vs 1 year

Parameter Estimates

Dependent Variable: HF4

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	-3,95E-02	,142	-,279	,782	-,328	,249
[GROUP=B]	9,617E-03	,046	,208	,837	-8,460E-02	,104
[GROUP=C]	0[a]	,	,	,	,	,
[GMFCS=II]	-1,31E-02	,047	-,278	,783	-,109	8,298E-02
[GMFCS=III]	0[a]	,	,	,	,	,
[SEX=f]	-6,64E-02	,044	-1,508	,141	-,156	2,319E-02
[SEX=m]	0[a]	,	,	,	,	,
AGE	1,740E-02	,010	1,765	,087	-2,659E-03	3,746E-02
HF2	,872	,177	4,926	,000	,512	1,233

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: HE4

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	-9,76E-02	,488	-,200	,843	-1,091	,896
[GROUP=B]	9,994E-02	,159	,627	,535	-,225	,424
[GROUP=C]	0[a]	,	,	,	,	,
[GMFCS=II]	,123	,162	,759	,453	-,207	,453
[GMFCS=III]	0[a]	,	,	,	,	,
[SEX=f]	2,142E-02	,161	,133	,895	-,306	,349
[SEX=m]	0[a]	,	,	,	,	,
AGE	8,166E-02	,038	2,146	,039	4,243E-03	,159
HE2	,539	,126	4,282	,000	,283	,795

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: HAB4

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	-7,69E-02	,197	-,390	,699	-,479	,326
[GROUP=B]	8,600E-03	,066	,130	,897	-,127	,144
[GROUP=C]	0[a]	,	,	,	,	,
[GMFCS=II]	7,150E-02	,088	,811	,424	-,109	,252
[GMFCS=III]	0[a]	,	,	,	,	,
[SEX=f]	8,407E-02	,061	1,377	,179	-4,060E-02	,209
[SEX=m]	0[a]	,	,	,	,	,
AGE	1,944E-02	,015	1,293	,206	-1,127E-02	5,015E-02
HAB2	,778	,189	4,126	,000	,393	1,163

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: KF4

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	-,289	,135	-2,144	,039	-,564	-1,478E-02
[GROUP=B]	6,091E-02	,044	1,399	,171	-2,765E-02	,149
[GROUP=C]	0[a]	,	,	,	,	,
[GMFCS=II]	,135	,053	2,548	,016	2,729E-02	,243
[GMFCS=III]	0[a]	,	,	,	,	,
[SEX=f]	2,204E-02	,043	,511	,613	-6,575E-02	,110
[SEX=m]	0[a]	,	,	,	,	,
AGE	2,809E-02	,009	2,980	,005	8,911E-03	4,727E-02
KF2	,758	,133	5,704	,000	,488	1,029

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: KE904

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	,217	,333	,653	,519	-,460	,894
[GROUP=B]	2,421E-03	,108	,022	,982	-,217	,222
[GROUP=C]	0[a]	,	,	,	,	,
[GMFCS=II]	,142	,125	1,135	,265	-,112	,396
[GMFCS=III]	0[a]	,	,	,	,	,
[SEX=f]	-,120	,103	-1,166	,252	-,330	8,955E-02
[SEX=m]	0[a]	,	,	,	,	,
AGE	4,062E-03	,026	,154	,878	-4,957E-02	5,769E-02
KE902	,946	,157	6,022	,000	,627	1,266

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: KE304

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	-,240	,139	-1,733	,095	-,526	4,535E-02
[GROUP=B]	9,899E-02	,047	2,117	,044	2,690E-03	,195
[GROUP=C]	0[a]	,	,	,	,	,
[GMFCS=II]	,101	,043	2,318	,029	1,120E-02	,190
[GMFCS=III]	0[a]	,	,	,	,	,
[SEX=f]	-7,02E-02	,040	-1,769	,089	-,152	1,152E-02
[SEX=m]	0[a]	,	,	,	,	,
AGE	2,708E-02	,010	2,703	,012	6,447E-03	4,772E-02
KE302	,816	,098	8,292	,000	,613	1,019

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: GMFM45

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	61,237	34,623	1,769	,105	-14,968	137,442
[GROUP=B]	-6,290	9,732	-,646	,531	-27,709	15,129
[GROUP=C]	0[a]	,	,	,	,	,
[GMFCS=II]	30,936	17,180	1,801	,099	-6,876	68,748
[GMFCS=III]	0[a]	,	,	,	,	,
[SEX=f]	-10,075	8,858	-1,137	,280	-29,572	9,422
[SEX=m]	0[a]	,	,	,	,	,
AGE	-2,121	1,977	-1,073	,306	-6,472	2,229
GMFM25	,348	,303	1,148	,275	-,319	1,014

a. This parameter is set to zero because it is redundant.

Parameter Estimates

Dependent Variable: GMFM4

Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	51,916	32,465	1,599	,138	-19,540	123,372
[GROUP=B]	-4,198	5,667	-,741	,474	-16,672	8,276
[GROUP=C]	0[a]	,	,	,	,	,
[GMFCS=II]	8,215	8,892	,924	,375	-11,356	27,786
[GMFCS=III]	0[a]	,	,	,	,	,
[SEX=f]	-3,317	4,874	-,680	,510	-14,045	7,412
[SEX=m]	0[a]	,	,	,	,	,
AGE	-,764	1,049	-,728	,482	-3,072	1,544
GMFM2	,521	,309	1,682	,121	-,160	1,202

a. This parameter is set to zero because it is redundant.

FILE 2a

Paired t-test for group A (strength, function, gait and temporal parameters between visits)

Strength baseline versus 6-month

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	HF1 - HF2	,0228	,25532	,06018	-,1042	,1497	,379	17	,710
Pair 2	HE1 - HE2	,0528	,97921	,23080	-,4342	,5397	,229	17	,822
Pair 3	HABD1 - HABD2	,0094	,19038	,04760	-,0921	,1108	,197	15	,846
Pair 4	KF1 - KF2	-,0189	,27827	,06559	-,1573	,1195	-,288	17	,777
Pair 5	KEX901 - KEX902	,1422	,43153	,10171	-,0724	,3568	1,398	17	,180
Pair 6	KEXT301 - KE302	,0267	,19575	,04614	-,0707	,1240	,578	17	,571

Strength baseline versus 1 year

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	HF1 - HF3	-,0900	,15680	,03920	-,1736	-,0064	-2,296	15	,037
Pair 2	HE1 - HE3	-,1400	,84032	,21008	-,5878	,3078	-,666	15	,515
Pair 3	HABD1 - HABD3	-,0500	,14884	,03978	-,1359	,0359	-1,257	13	,231

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	KF1 - KF3	-,0744	,14756	,03689	-,1530	,0043	-2,016	15	,062
Pair 2	KEX901 - KEX903	,0788	,29543	,07386	-,0787	,2362	1,066	15	,303
Pair 3	KEXT301 - KE303	,0463	,16029	,04007	-,0392	,1317	1,154	15	,266

Function baseline versus 1 year

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	GMFM1 - GMFM3	-,2688	2,41072	,85232	-2,2842	1,7467	-,315	7	,762
Pair 2	GMFM15 - GMFM35	-2,3275	6,16619	2,18008	-7,4826	2,8276	-1,068	7	,321

Kinematic and kinetic baseline versus 1 year

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	RRPT - RRPT3	,5669	1,77236	,44309	-,3775	1,5113	1,279	15	,220
Pair 2	RRHF - RRHF3	1,5450	2,59023	,64756	,1648	2,9252	2,386	15	,031
Pair 3	RRKF - RRKF3	5,1069	4,90722	1,22680	2,4920	7,7217	4,163	15	,001
Pair 4	RRAD - RRAD3	-4,7469	7,44822	1,86206	-8,7158	-,7780	-2,549	15	,022
Pair 5	RMHES - RMHES3	-3,6869	3,51866	,87967	-5,5618	-1,8119	-4,191	15	,001
Pair 6	RMKES - RMKES3	-4,4056	4,78796	1,19699	-6,9569	-1,8543	-3,681	15	,002
Pair 7	RMKFS - RMKFS3	,7031	5,07343	1,26836	-2,0003	3,4066	,554	15	,588
Pair 8	RKFIC - RKFIC3	-2,9262	4,77053	1,19263	-5,4683	-,3842	-2,454	15	,027
Pair 9	RRPO - RRPO3	,1288	2,95341	,73835	-1,4450	1,7025	,174	15	,864
Pair 10	RRPR - RRPR3	-1,1956	4,53647	1,13412	-3,6129	1,2217	-1,054	15	,308
Pair 11	RMTR - RMTR3	-1,6675	5,89818	1,47454	-4,8104	1,4754	-1,131	15	,276

20
8

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	RHEM - RHEM3	68,3333	297,03707	76,69464	-96,1603	232,8270	,891	14	,388
Pair 2	RHFM - RHFM3	31,5547	127,82004	33,00299	-39,2297	102,3390	,956	14	,355
Pair 3	RKEM - RKEM3	-58,1733	151,93309	39,22896	-142,3111	25,9644	-1,483	14	,160
Pair 4	RKFM - RKFM3	-96,1673	118,14236	30,50423	-161,5924	-30,7423	-3,153	14	,007
Pair 5	RMPM - RMPM3	-103,1440	189,53983	48,93897	-208,1077	1,8197	-2,108	14	,054
Pair 6	RMHAM - RMHAM3	100,6893	140,00573	36,14932	23,1567	178,2219	2,785	14	,015
Pair 7	RMHP - RMHP3	,3467	,55637	,14366	,0386	,6548	2,413	14	,030
Pair 8	RMAP - RMAP3	-,0393	,46500	,12006	-,2968	,2182	-,328	14	,748

Temporal baseline versus 1 year

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	VELOCIT1 - VELOCIT3	,0100	,05071	,01793	-,0324	,0524	,558	7	,594
Pair 2	CAD1 - CAD3	,0150	,04504	,01592	-,0227	,0527	,942	7	,378
Pair 3	RSTEP1 - RSTEP3	,0050	,05014	,01773	-,0369	,0469	,282	7	,786
Pair 4	LSTEP1 - LSTEP3	,0112	,08254	,02918	-,0578	,0803	,386	7	,711

FILE 2b

Paired t-test for groups B & C (strength, function, gait and temporal parameters between visits)

Strength and function for all subjects and for groups B & C separately

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	GMFM1 - GMFM2	7,6250	9,86334	2,20551	3,0088	12,2412	3,457	19	,003
Pair 2	GMFM2 - GMFM3	-6,2653	6,33647	1,45369	-9,3193	-3,2112	-4,310	18	,000
Pair 3	GMFM1 - GMFM4	1,9176	5,78360	1,40273	-1,0560	4,8913	1,367	16	,191
Pair 4	GMFM15 - GMFM25	21,5105	18,56130	4,15043	12,8235	30,1975	5,183	19	,000
Pair 5	GMFM25 - GMFM35	-12,2795	10,89830	2,50024	-17,5323	-7,0267	-4,911	18	,000
Pair 6	GMFM15 - GMFM45	9,3747	16,77690	4,06900	,7488	18,0006	2,304	16	,035

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	HF1 - HF2	,2154	,20781	,03328	,1480	,2827	6,473	38	,000
Pair 2	HE1 - HE2	,5587	,72803	,11658	,3227	,7947	4,793	38	,000
Pair 3	HAB1 - HAB2	,1081	,20206	,03322	,0407	,1755	3,255	36	,002
Pair 4	KF1 - KF2	,5665	,30453	,05006	,4650	,6680	11,315	36	,000
Pair 5	KE901 - KE902	,5108	,50415	,08073	,3473	,6742	6,327	38	,000
Pair 6	KE301 - KE302	,1233	,19140	,04177	,0362	,2105	2,953	20	,008

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	HF2 - HF3	-,1132	,14318	,02354	-,1610	-,0655	-4,811	36	,000
Pair 2	HE2 - HE3	-,1600	,40269	,06620	-,2943	-,0257	-2,417	36	,021
Pair 3	HAB2 - HAB3	-,1129	,13601	,02299	-,1596	-,0661	-4,909	34	,000
Pair 4	KF2 - KF3	-,0805	,09404	,01546	-,1119	-,0492	-5,209	36	,000
Pair 5	KE902 - KE903	-,1624	,17614	,02896	-,2212	-,1037	-5,609	36	,000
Pair 6	KE302 - KE303	-,1160	,09438	,01723	-,1512	-,0808	-6,732	29	,000

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	HF1 - HF4	,0997	,22150	,03547	,0279	,1715	2,812	38	,008
Pair 2	HE1 - HE4	,3979	,75714	,12124	,1525	,6434	3,282	38	,002
Pair 3	HAB1 - HAB4	-,0450	,25458	,04243	-,1311	,0411	-1,061	35	,296
Pair 4	KF1 - KF4	,4962	,31407	,05163	,3915	,6009	9,610	36	,000
Pair 5	KE901 - KE904	,2862	,49084	,07860	,1270	,4453	3,641	38	,001
Pair 6	KE301 - KE304	,0961	,24843	,05180	-,0113	,2035	1,855	22	,077

Paired Samples Test[a]

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	GMFM2 - GMFM3	-7,9400	8,58686	3,03591	-15,1188	-,7612	-2,615	7	,035
Pair 2	GMFM25 - GMFM35	-15,1037	13,22543	4,67590	-26,1605	-4,0470	-3,230	7	,014
Pair 3	HF2 - HF3	-,0406	,09923	,02481	-,0935	,0123	-1,638	15	,122
Pair 4	HE2 - HE3	-,1281	,34388	,08597	-,3114	,0551	-1,490	15	,157
Pair 5	HAB2 - HAB3	-,0786	,12551	,03354	-,1510	-,0061	-2,342	13	,036
Pair 6	KF2 - KF3	-,0763	,09003	,02251	-,1242	-,0283	-3,388	15	,004
Pair 7	KE902 - KE903	-,1444	,19079	,04770	-,2460	-,0427	-3,027	15	,008
Pair 8	KE302 - KE303	-,0710	,08048	,02545	-,1286	-,0134	-2,790	9	,021
Pair 9	HF1 - HF4	,0439	,21696	,05114	-,0640	,1518	,858	17	,403
Pair 10	HE1 - HE4	,1494	,64556	,15216	-,1716	,4705	,982	17	,340
Pair 11	HAB1 - HAB4	-,0587	,25348	,06337	-,1938	,0763	-,927	15	,369
Pair 12	KF1 - KF4	,4750	,30834	,07268	,3217	,6283	6,536	17	,000
Pair 13	KE901 - KE904	,2578	,45728	,10778	,0304	,4852	2,392	17	,029
Pair 14	KE301 - KE304	,0055	,23822	,07183	-,1546	,1655	,076	10	,941

a. GROUP = B

Paired Samples Test[a]

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	GMFM2 - GMFM3	-5,0473	4,09658	1,23516	-7,7994	-2,2952	-4,086	10	,002
Pair 2	GMFM25 - GMFM35	-10,2255	8,96274	2,70237	-16,2467	-4,2042	-3,784	10	,004
Pair 3	HF2 - HF3	-,1686	,14860	,03243	-,2362	-,1009	-5,198	20	,000
Pair 4	HE2 - HE3	-,1843	,44917	,09802	-,3887	,0202	-1,880	20	,075
Pair 5	HAB2 - HAB3	-,1357	,14084	,03073	-,1998	-,0716	-4,416	20	,000
Pair 6	KF2 - KF3	-,0838	,09907	,02162	-,1289	-,0387	-3,877	20	,001
Pair 7	KE902 - KE903	-,1762	,16759	,03657	-,2525	-,0999	-4,818	20	,000
Pair 8	KE302 - KE303	-,1385	,09449	,02113	-,1827	-,0943	-6,555	19	,000
Pair 9	HF1 - HF4	,1476	,21906	,04780	,0479	,2473	3,088	20	,006
Pair 10	HE1 - HE4	,6110	,79482	,17344	,2492	,9728	3,522	20	,002
Pair 11	HAB1 - HAB4	-,0340	,26148	,05847	-,1564	,0884	-,582	19	,568
Pair 12	KF1 - KF4	,5163	,32652	,07491	,3589	,6737	6,893	18	,000
Pair 13	KE901 - KE904	,3105	,52790	,11520	,0702	,5508	2,695	20	,014
Pair 14	KE301 - KE304	,1792	,23685	,06837	,0287	,3297	2,620	11	,024

a. GROUP = C

Kinematic and kinetic parameters

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	RPT1 - RPT2	2,1227	3,25975	,51541	1,0802	3,1653	4,119	39	,000
Pair 2	RHF1 - RHF2	-,8468	8,31417	1,31459	-3,5058	1,8123	-,644	39	,523
Pair 3	RAD1 - RAD2	19,2475	16,92919	2,67674	13,8333	24,6617	7,191	39	,000
Pair 4	MHES1 - MHES2	2,1895	8,29267	1,31119	-,4626	4,8416	1,670	39	,103
Pair 5	MKES1 - MKES2	23,5797	24,66281	3,89953	15,6922	31,4673	6,047	39	,000
Pair 6	MKFS1 - MKFS2	6,3450	15,26714	2,41395	1,4623	11,2277	2,628	39	,012
Pair 7	MKFIC1 - MKFIC2	23,0248	18,76317	2,96672	17,0240	29,0255	7,761	39	,000
Pair 8	RPO1 - RPO2	1,3845	4,57489	,72335	-,0786	2,8476	1,914	39	,063
Pair 9	RPR1 - RPR2	1,5967	8,75705	1,38461	-1,2039	4,3974	1,153	39	,256
Pair 10	MTR1 - MTR2	17,8757	19,31455	3,05390	11,6987	24,0528	5,853	39	,000
Pair 11	RKF1 - RKF2	-17,1327	17,56489	2,77725	-22,7503	-11,5152	-6,169	39	,000

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	RPT1 - RPT4	2,5045	2,82224	,44624	1,6019	3,4071	5,613	39	,000
Pair 2	RHF1 - RHF4	,5030	8,64148	1,36634	-2,2607	3,2667	,368	39	,715
Pair 3	RKF1 - RKF4	-11,6212	15,60450	2,46729	-16,6118	-6,6307	-4,710	39	,000
Pair 4	RAD1 - RAD4	9,9227	14,74420	2,33126	5,2073	14,6382	4,256	39	,000
Pair 5	MHES1 - MHES4	,1418	8,93090	1,41210	-2,7145	2,9980	,100	39	,921
Pair 6	MKES1 - MKES4	16,6807	24,46023	3,86750	8,8580	24,5035	4,313	39	,000
Pair 7	MKFS1 - MKFS4	4,9328	14,38119	2,27387	,3334	9,5321	2,169	39	,036
Pair 8	MKFIC1 - KFIC4	15,6618	16,18221	2,55863	10,4864	20,8371	6,121	39	,000
Pair 9	RPO1 - RPO4	1,0648	4,86898	,76985	-,4924	2,6219	1,383	39	,175
Pair 10	RPR1 - RPR4	2,3425	9,29169	1,46915	-,6291	5,3141	1,594	39	,119
Pair 11	MTR1 - MTR4	11,2257	13,08739	2,06930	7,0402	15,4113	5,425	39	,000

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	RPT2 - RPT3	,1450	2,42387	,39320	-,6517	,9417	,369	37	,714
Pair 2	RHF2 - RHF3	-,3897	5,56721	,90312	-2,2196	1,4402	-,432	37	,669
Pair 3	RKF2 - RKF3	2,6963	5,38291	,87322	,9270	4,4656	3,088	37	,004
Pair 4	RAD2 - RAD3	-,6692	3,66629	,59475	-1,8743	,5359	-1,125	37	,268
Pair 5	MHES2 - MHES3	-,3787	6,40814	1,03954	-2,4850	1,7276	-,364	37	,718
Pair 6	MKES2 - MKES3	-2,3126	5,17169	,83896	-4,0125	-,6127	-2,757	37	,009
Pair 7	MKFS2 - MKFS3	,3995	5,76697	,93553	-1,4961	2,2950	,427	37	,672
Pair 8	MKFIC2 - KFIC3	-,7161	4,78450	,77615	-2,2887	,8566	-,923	37	,362
Pair 9	RPO2 - RPO3	-,1963	2,34812	,38091	-,9681	,5755	-,515	37	,609
Pair 10	RPR2 - RPR3	,2326	4,69964	,76238	-1,3121	1,7774	,305	37	,762
Pair 11	MTR2 - MTR3	-4,5266	20,88864	3,38858	-11,3925	2,3393	-1,336	37	,190

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	HEM1 - HEM2	45,2255	489,36899	109,42623	-183,8062	274,2572	,413	19	,684
Pair 2	HFM1 - HFM2	-128,7835	224,16426	50,12465	-233,6956	-23,8714	-2,569	19	,019
Pair 3	KEM1 - KEM2	179,8280	290,48899	64,95531	43,8750	315,7810	2,768	19	,012
Pair 4	KFM1 - KFM2	13,8755	289,98544	64,84272	-121,8419	149,5929	,214	19	,833
Pair 5	MPFM1 - MPFM2	91,7580	326,40683	72,98679	-61,0051	244,5211	1,257	19	,224
Pair 6	MHAM1 - MHAM2	266,9355	233,25184	52,15670	157,7703	376,1007	5,118	19	,000
Pair 7	MHP1 - MHP2	,2485	,93979	,21014	-,1913	,6883	1,183	19	,252
Pair 8	MAP1 - MAP2	,8335	1,13290	,25332	,3033	1,3637	3,290	19	,004

30

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	HEM1 - HEM4	84,6930	447,35254	100,03107	-124,6744	294,0604	,847	19	,408
Pair 2	HFM1 - HFM4	-130,8665	256,41313	57,33572	-250,8715	-10,8615	-2,282	19	,034
Pair 3	KEM1 - KEM4	47,4600	313,73737	70,15381	-99,3736	194,2936	,677	19	,507
Pair 4	KFM1 - KFM4	-79,8410	249,05927	55,69135	-196,4043	36,7223	-1,434	19	,168
Pair 5	MPFM1 - MPFM4	102,6080	298,73731	66,79969	-37,2054	242,4214	1,536	19	,141
Pair 6	MHAM1 - MHAM4	212,7905	207,57765	46,41577	115,6412	309,9398	4,584	19	,000
Pair 7	MHP1 - MHP4	,3510	,67164	,15018	,0367	,6653	2,337	19	,031
Pair 8	MAP1 - MAP4	-,0880	,97562	,21815	-,5446	,3686	-,403	19	,691

P 38

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	HEM2 - HEM3	-3,2174	223,45314	46,59320	-99,8458	93,4110	-,069	22	,946
Pair 2	HFM2 - HFM3	,1217	127,82858	26,65410	-55,1555	55,3990	,005	22	,996
Pair 3	KEM2 - KEM3	-117,2700	180,27261	37,58944	-195,2257	-39,3143	-3,120	22	,005
Pair 4	KFM2 - KFM3	-78,1230	94,70888	19,74817	-119,0782	-37,1679	-3,956	22	,001
Pair 5	MPFM2 - MPFM3	-4,9639	116,07941	24,20423	-55,1604	45,2326	-,205	22	,839
Pair 6	MHAM2 - MHAM3	-26,3639	96,51638	20,12506	-68,1007	15,3729	-1,310	22	,204
Pair 7	MHP2 - MHP3	-,1070	,42154	,08790	-,2892	,0753	-1,217	22	,237
Pair 8	MAP2 - MAP3	-,2448	,59797	,12469	-,5034	,0138	-1,963	22	,062

Paired Samples Test[a]

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	RPT2 - RPT3	,0744	3,13341	,78335	-1,5953	1,7441	,095	15	,926
Pair 2	RHF2 - RHF3	,0481	7,19749	1,79937	-3,7871	3,8834	,027	15	,979
Pair 3	RKF2 - RKF3	2,6525	5,61857	1,40464	-,3414	5,6464	1,888	15	,078
Pair 4	RAD2 - RAD3	,2494	2,87127	,71782	-1,2806	1,7794	,347	15	,733
Pair 5	MHES2 - MHES3	-,1300	8,19603	2,04901	-4,4974	4,2374	-,063	15	,950
Pair 6	MKES2 - MKES3	-3,0262	6,77962	1,69491	-6,6389	,5864	-1,785	15	,094
Pair 7	MKFIC2 - KFIC3	-1,8494	5,27920	1,31980	-4,6625	,9637	-1,401	15	,181
Pair 8	RPO2 - RPO3	,1581	2,27574	,56893	-1,0545	1,3708	,278	15	,785
Pair 9	RPR2 - RPR3	-,8687	4,44345	1,11086	-3,2365	1,4990	-,782	15	,446
Pair 10	MTR2 - MTR3	-1,6419	6,81817	1,70454	-5,2750	1,9913	-,963	15	,351

a. GROUP = B

Paired Samples Test[a]

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	RPT2 - RPT3	,1964	1,82532	,38916	-,6129	1,0057	,505	21	,619
Pair 2	RHF2 - RHF3	-,7082	4,16569	,88813	-2,5551	1,1388	-,797	21	,434
Pair 3	RKF2 - RKF3	2,7282	5,33864	1,13820	,3612	5,0952	2,397	21	,026
Pair 4	RAD2 - RAD3	-1,3373	4,08459	,87084	-3,1483	,4737	-1,536	21	,140
Pair 5	MHES2 - MHES3	-,5595	4,92827	1,05071	-2,7446	1,6255	-,533	21	,600
Pair 6	MKES2 - MKES3	-1,7936	3,69100	,78692	-3,4301	-,1571	-2,279	21	,033
Pair 7	MKFIC2 - KFIC3	,1082	4,32841	,92282	-1,8109	2,0273	,117	21	,908
Pair 8	RPO2 - RPO3	-,4541	2,41866	,51566	-1,5265	,6183	-,881	21	,389
Pair 9	RPR2 - RPR3	1,0336	4,81821	1,02725	-1,1026	3,1699	1,006	21	,326
Pair 10	MTR2 - MTR3	-6,6245	26,91885	5,73912	-18,5597	5,3106	-1,154	21	,261

a. GROUP = C

Paired Samples Test[a]

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	HEM2 - HEM3	71,1330	211,50168	66,88270	-80,1662	222,4322	1,064	9	,315
Pair 2	HFM2 - HFM3	-32,8850	150,28110	47,52306	-140,3896	74,6196	-,692	9	,506
Pair 3	KEM2 - KEM3	-193,2670	161,09420	50,94246	-308,5068	-78,0272	-3,794	9	,004
Pair 4	KFM2 - KFM3	-61,3500	84,19819	26,62581	-121,5818	-1,1182	-2,304	9	,047
Pair 5	MPFM2 - MPFM3	-66,1190	75,35203	23,82840	-120,0226	-12,2154	-2,775	9	,022
Pair 6	MHAM2 - MHAM3	-21,2850	117,15007	37,04610	-105,0891	62,5191	-,575	9	,580
Pair 7	MHP2 - MHP3	-,1450	,56455	,17853	-,5489	,2589	-,812	9	,438
Pair 8	MAP2 - MAP3	-,1180	,24903	,07875	-,2961	,0601	-1,498	9	,168

a. GROUP = B

Paired Samples Test[a]

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	HEM2 - HEM3	-60,4100	223,25048	61,91854	-195,3189	74,4989	-,976	12	,349
Pair 2	HFM2 - HFM3	25,5115	106,82881	29,62898	-39,0445	90,0675	,861	12	,406
Pair 3	KEM2 - KEM3	-58,8108	177,76762	49,30387	-166,2347	48,6131	-1,193	12	,256
Pair 4	KFM2 - KFM3	-91,0254	103,50262	28,70646	-153,5714	-28,4794	-3,171	12	,008
Pair 5	MPFM2 - MPFM3	42,0785	122,19105	33,88970	-31,7609	115,9178	1,242	12	,238
Pair 6	MHAM2 - MHAM3	-30,2708	82,14086	22,78178	-79,9080	19,3665	-1,329	12	,209
Pair 7	MHP2 - MHP3	-,0777	,29087	,08067	-,2535	,0981	-,963	12	,355
Pair 8	MAP2 - MAP3	-,3423	,76507	,21219	-,8046	,1200	-1,613	12	,133

a. GROUP = C

Temporal parameters between visits

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	NVELOC1 - NVELOC2	,0490	,06257	,01399	,0197	,0783	3,502	19	,002
Pair 2	NVELOC2 - NVELOC3	-,0337	,04597	,01055	-,0558	-,0115	-3,194	18	,005
Pair 3	NVELOC1 - NVELOC4	,0210	,07629	,01706	-,0147	,0567	1,231	19	,233
Pair 4	NCAD1 - NCAD2	,0950	,07487	,01674	,0600	,1300	5,675	19	,000
Pair 5	NCAD2 - NCAD3	-,0242	,04073	,00934	-,0438	-,0046	-2,591	18	,018
Pair 6	NCAD1 - NCAD4	,0255	,07817	,01748	-,0111	,0621	1,459	19	,161
Pair 7	NSTEPR1 - NSTEPR2	-,0085	,11935	,02669	-,0644	,0474	-,318	19	,754
Pair 8	NSTEPR2 - NSTEPR3	-,0532	,12702	,02914	-,1144	,0081	-1,824	18	,085
Pair 9	NSTEPR1 - NSTEPR4	,0115	,08893	,01988	-,0301	,0531	,578	19	,570
Pair 10	NSTEPL1 - NSTEPL2	-,0055	,13496	,03018	-,0687	,0577	-,182	19	,857
Pair 11	NSTEPL2 - NSTEPL3	-,0568	,08097	,01858	-,0959	-,0178	-3,060	18	,007
Pair 12	NSTEPL1 - NSTEPL4	,0220	,13033	,02914	-,0390	,0830	,755	19	,460

Paired Samples Test[a]

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	NVELOC1 - NVELOC2	,0433	,06245	,02082	-,0047	,0913	2,082	8	,071
Pair 2	NVELOC2 - NVELOC3	-,0238	,06653	,02352	-,0794	,0319	-1,010	7	,346
Pair 3	NVELOC1 - NVELOC4	-,0022	,09404	,03135	-,0745	,0701	-,071	8	,945
Pair 4	NCAD1 - NCAD2	,0833	,06103	,02034	,0364	,1302	4,096	8	,003
Pair 5	NCAD2 - NCAD3	-,0163	,05951	,02104	-,0660	,0335	-,772	7	,465
Pair 6	NCAD1 - NCAD4	-,0089	,08781	,02927	-,0764	,0586	-,304	8	,769
Pair 7	NSTEPR1 - NSTEPR2	,0189	,13624	,04541	-,0858	,1236	,416	8	,688
Pair 8	NSTEPR2 - NSTEPR3	-,0212	,13206	,04669	-,1317	,0892	-,455	7	,663
Pair 9	NSTEPR1 - NSTEPR4	,0411	,08992	,02997	-,0280	,1102	1,372	8	,207
Pair 10	NSTEPL1 - NSTEPL2	-,0478	,15344	,05115	-,1657	,0702	-,934	8	,378
Pair 11	NSTEPL2 - NSTEPL3	-,0313	,10842	,03833	-,1219	,0594	-,815	7	,442
Pair 12	NSTEPL1 - NSTEPL4	-,0289	,15317	,05106	-,1466	,0888	-,566	8	,587

a. GROUP = B

Paired Samples Test[a]

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	NVELOC1 - NVELOC2	,0536	,06531	,01969	,0098	,0975	2,724	10	,021
Pair 2	NVELOC2 - NVELOC3	-,0409	,02386	,00719	-,0569	-,0249	-5,688	10	,000
Pair 3	NVELOC1 - NVELOC4	,0400	,05568	,01679	,0026	,0774	2,383	10	,038
Pair 4	NCAD1 - NCAD2	,1045	,08630	,02602	,0466	,1625	4,018	10	,002
Pair 5	NCAD2 - NCAD3	-,0300	,02049	,00618	-,0438	-,0162	-4,855	10	,001
Pair 6	NCAD1 - NCAD4	,0536	,05921	,01785	,0139	,0934	3,005	10	,013
Pair 7	NSTEPR1 - NSTEPR2	-,0309	,10483	,03161	-,1013	,0395	-,978	10	,351
Pair 8	NSTEPR2 - NSTEPR3	-,0764	,12420	,03745	-,1598	,0071	-2,039	10	,069
Pair 9	NSTEPR1 - NSTEPR4	-,0127	,08439	,02544	-,0694	,0440	-,500	10	,628
Pair 10	NSTEPL1 - NSTEPL2	,0291	,11335	,03418	-,0471	,1052	,851	10	,415
Pair 11	NSTEPL2 - NSTEPL3	-,0755	,05165	,01557	-,1102	-,0408	-4,846	10	,001
Pair 12	NSTEPL1 - NSTEPL4	,0636	,09626	,02902	-,0010	,1283	2,193	10	,053

a. GROUP = C

Check (✓) the appropriate score:

Item	A: **LYING AND ROLLING**	SCORE				
1.	SUP, HEAD IN MIDLINE: TURNS HEAD WITH EXTREMITIES SYMMETRICAL	0 ☐	1 ☐	2 ☐	3 ☐	1.
2.	SUP: BRINGS HANDS TO MIDLINE, FINGERS ONE WITH THE OTHER	0 ☐	1 ☐	2 ☐	3 ☐	2.
3.	SUP: LIFTS HEAD 45°	0 ☐	1 ☐	2 ☐	3 ☐	3.
4.	SUP: FLEXES R HIP & KNEE THROUGH FULL RANGE	0 ☐	1 ☐	2 ☐	3 ☐	4.
5.	SUP: FLEXES L HIP AND KNEE THROUGH FULL RANGE	0 ☐	1 ☐	2 ☐	3 ☐	5.
6.	SUP: REACHES OUT WITH R ARM, HAND CROSSES MIDLINE TOWARD TOY	0 ☐	1 ☐	2 ☐	3 ☐	6.
7.	SUP: REACHES OUT WITH L ARM, HAND CROSSES MIDLINE TOWARD TOY	0 ☐	1 ☐	2 ☐	3 ☐	7.
8.	SUP: ROLLS TO PR OVER R SIDE	0 ☐	1 ☐	2 ☐	3 ☐	8.
9.	SUP: ROLLS TO PR OVER L SIDE	0 ☐	1 ☐	2 ☐	3 ☐	9.
10.	PR: LIFTS HEAD UPRIGHT	0 ☐	1 ☐	2 ☐	3 ☐	10.
11.	PR ON FOREARMS: LIFTS HEAD UPRIGHT, ELBOWS EXT., CHEST RAISED	0 ☐	1 ☐	2 ☐	3 ☐	11.
12.	PR ON FOREARMS: WEIGHT ON R FOREARM, FULLY EXTENDS OPPOSITE ARM FORWARD	0 ☐	1 ☐	2 ☐	3 ☐	12.
13.	PR ON FOREARMS: WEIGHT ON L FOREARM, FULLY EXTENDS OPPOSITE ARM FORWARD	0 ☐	1 ☐	2 ☐	3 ☐	13.
14.	PR: ROLLS TO SUP OVER R SIDE	0 ☐	1 ☐	2 ☐	3 ☐	14.
15.	PR: ROLLS TO SUP OVER L SIDE	0 ☐	1 ☐	2 ☐	3 ☐	15.
16.	PR: PIVOTS TO R 90° USING EXTREMITIES	0 ☐	1 ☐	2 ☐	3 ☐	16.
17.	PR: PIVOTS TO L 90° USING EXTREMITIES	0 ☐	1 ☐	2 ☐	3 ☐	17.

TOTAL DIMENSION A ☐

Item	B: **SITTING**	SCORE				
18.	SUP, HANDS GRASPED BY EXAMINER: PULLS SELF TO SITTING WITH HEAD CONTROL	0 ☐	1 ☐	2 ☐	3 ☐	18.
19.	SUP: ROLLS TO R SIDE, ATTAINS SITTING	0 ☐	1 ☐	2 ☐	3 ☐	19.
20.	SUP: ROLLS TO L SIDE, ATTAINS SITTING	0 ☐	1 ☐	2 ☐	3 ☐	20.
21.	SIT ON MAT, SUPPORTED AT THORAX BY THERAPIST: LIFTS HEAD UPRIGHT, MAINTAINS 3 SECONDS	0 ☐	1 ☐	2 ☐	3 ☐	21.
22.	SIT ON MAT, SUPPORTED AT THORAX BY THERAPIST: LIFTS HEAD TO MIDLINE, MAINTAINS 10 SECONDS	0 ☐	1 ☐	2 ☐	3 ☐	22.
23.	SIT ON MAT, ARM(S) PROPPING: MAINTAINS, 5 SECONDS	0 ☐	1 ☐	2 ☐	3 ☐	23.
24.	SIT ON MAT: MAINTAINS, ARMS FREE, 3 SECONDS	0 ☐	1 ☐	2 ☐	3 ☐	24.
25.	SIT ON MAT WITH SMALL TOY IN FRONT: LEANS FORWARD, TOUCHES TOY, RE-ERECTS WITHOUT ARM PROPPING	0 ☐	1 ☐	2 ☐	3 ☐	25.
26.	SIT ON MAT: TOUCHES TOY PLACED 45° BEHIND CHILD'S R SIDE, RETURNS TO START	0 ☐	1 ☐	2 ☐	3 ☐	26.
27.	SIT ON MAT: TOUCHES TOY PLACED 45° BEHIND CHILD'S L SIDE, RETURNS TO START	0 ☐	1 ☐	2 ☐	3 ☐	27.
28.	R SIDE SIT: MAINTAINS, ARMS FREE, 5 SECONDS	0 ☐	1 ☐	2 ☐	3 ☐	28.
29.	L SIDE SIT: MAINTAINS, ARMS FREE, 5 SECONDS	0 ☐	1 ☐	2 ☐	3 ☐	29.
30.	SIT ON MAT: LOWERS TO PR WITH CONTROL	0 ☐	1 ☐	2 ☐	3 ☐	30.
31.	SIT ON MAT WITH FEET IN FRONT: ATTAINS 4 POINT OVER R SIDE	0 ☐	1 ☐	2 ☐	3 ☐	31.
32.	SIT ON MAT WITH FEET IN FRONT: ATTAINS 4 POINT OVER L SIDE	0 ☐	1 ☐	2 ☐	3 ☐	32.
33.	SIT ON MAT: PIVOTS 90°, WITHOUT ARMS ASSISTING	0 ☐	1 ☐	2 ☐	3 ☐	33.
34.	SIT ON BENCH: MAINTAINS, ARMS AND FEET FREE, 10 SECONDS	0 ☐	1 ☐	2 ☐	3 ☐	34.
35.	STD: ATTAINS SIT ON SMALL BENCH	0 ☐	1 ☐	2 ☐	3 ☐	35.
36.	ON THE FLOOR: ATTAINS SIT ON SMALL BENCH	0 ☐	1 ☐	2 ☐	3 ☐	36.
37.	ON THE FLOOR: ATTAINS SIT ON LARGE BENCH	0 ☐	1 ☐	2 ☐	3 ☐	37.

TOTAL DIMENSION B ☐

Item	E. WALKING, RUNNING AND JUMPING	SCORE	
65.	STD, 2 HANDS ON LARGE BENCH: CRUISES 5 STEPS TO R	0 ☐ 1 ☐ 2 ☐ 3 ☐	65.
66.	STD, 2 HANDS ON LARGE BENCH: CRUISES 5 STEPS TO L	0 ☐ 1 ☐ 2 ☐ 3 ☐	66.
67.	STD, 2 HANDS HELD: WALKS FORWARD 10 STEPS	0 ☐ 1 ☐ 2 ☐ 3 ☐	67.
68.	STD, 1 HAND HELD: WALKS FORWARD 10 STEPS	0 ☐ 1 ☐ 2 ☐ 3 ☐	68.
69.	STD: WALKS FORWARD 10 STEPS	0 ☐ 1 ☐ 2 ☐ 3 ☐	69.
70.	STD: WALKS FORWARD 10 STEPS, STOPS, TURNS 180°, RETURNS	0 ☐ 1 ☐ 2 ☐ 3 ☐	70.
71.	STD: WALKS BACKWARD 10 STEPS	0 ☐ 1 ☐ 2 ☐ 3 ☐	71.
72.	STD: WALKS FORWARD 10 STEPS, CARRYING A LARGE OBJECT WITH 2 HANDS	0 ☐ 1 ☐ 2 ☐ 3 ☐	72.
73.	STD: WALKS FORWARD 10 CONSECUTIVE STEPS BETWEEN PARALLEL LINES 8" APART	0 ☐ 1 ☐ 2 ☐ 3 ☐	73.
74.	STD: WALKS FORWARD 10 CONSECUTIVE STEPS ON A STRAIGHT LINE ¾" WIDE	0 ☐ 1 ☐ 2 ☐ 3 ☐	74.
75.	STD: STEPS OVER STICK AT KNEE LEVEL, R FOOT LEADING	0 ☐ 1 ☐ 2 ☐ 3 ☐	75.
76.	STD: STEPS OVER STICK AT KNEE LEVEL, L FOOT LEADING	0 ☐ 1 ☐ 2 ☐ 3 ☐	76.
77.	STD: RUNS 15 FEET, STOPS & RETURNS	0 ☐ 1 ☐ 2 ☐ 3 ☐	77.
78.	STD: KICKS BALL WITH R FOOT	0 ☐ 1 ☐ 2 ☐ 3 ☐	78.
79.	STD: KICKS BALL WITH L FOOT	0 ☐ 1 ☐ 2 ☐ 3 ☐	79.
80.	STD: JUMPS 12" HIGH, BOTH FEET SIMULTANEOUSLY	0 ☐ 1 ☐ 2 ☐ 3 ☐	80.
81.	STD: JUMPS FORWARD 12", BOTH FEET SIMULTANEOUSLY	0 ☐ 1 ☐ 2 ☐ 3 ☐	81.
82.	STD ON R FOOT: HOPS ON R FOOT 10 TIMES WITHIN A 24" CIRCLE	0 ☐ 1 ☐ 2 ☐ 3 ☐	82.
83.	STD ON L FOOT: HOPS ON L FOOT 10 TIMES WITHIN A 24" CIRCLE	0 ☐ 1 ☐ 2 ☐ 3 ☐	83.
84.	STD, HOLDING 1 RAIL: WALKS UP 4 STEPS, HOLDING 1 RAIL, ALTERNATING FEET	0 ☐ 1 ☐ 2 ☐ 3 ☐	84.
85.	STD, HOLDING 1 RAIL: WALKS DOWN 4 STEPS, HOLDING 1 RAIL, ALTERNATING FEET	0 ☐ 1 ☐ 2 ☐ 3 ☐	85.
86.	STD: WALKS UP 4 STEPS, ALTERNATING FEET	0 ☐ 1 ☐ 2 ☐ 3 ☐	86.
87.	STD: WALKS DOWN 4 STEPS, ALTERNATING FEET	0 ☐ 1 ☐ 2 ☐ 3 ☐	87.
88.	STD ON 6" STEP: JUMPS OFF, BOTH FEET SIMULTANEOUSLY	0 ☐ 1 ☐ 2 ☐ 3 ☐	88.

TOTAL DIMENSION E ☐

Was this assessment indicative of this child's "regular" performance? YES ☐ NO ☐

COMMENTS:

TESTING WITH AIDS/ORTHOSES

Indicate below with a check (✓) which aid/orthosis was used and what dimension it was first applied. (There may be more than one).

Aid		Dimension	Orthosis		Dimension
Rollator/pusher	☐	_____	Hip Control	☐	_____
Walker	☐	_____	Knee Control	☐	_____
H Frame crutches	☐	_____	Ankle-foot Control	☐	_____
Crutches	☐	_____	Foot Control	☐	_____
Quad Cane	☐	_____	Shoes	☐	_____
Cane	☐	_____	None	☐	_____
None	☐	_____	Other _____ (please specify)	☐	_____
Other _____ (please specify)	☐	_____			

SUMMARY SCORE USING AIDS/ORTHOSES

DIMENSION	CALCULATION OF DIMENSION % SCORES	GOAL AREA (indicated with ✓ check)
A. Lying & Rolling	$\frac{\text{Total Dimension A}}{51} = \frac{____}{51} \times 100 = ____\ \%$	A. ☐
B. Sitting	$\frac{\text{Total Dimension B}}{60} = \frac{____}{60} \times 100 = ____\ \%$	B. ☐
C. Crawling & Kneeling	$\frac{\text{Total Dimension C}}{42} = \frac{____}{42} \times 100 = ____\ \%$	C. ☐
D. Standing	$\frac{\text{Total Dimension D}}{39} = \frac{____}{39} \times 100 = ____\ \%$	D. ☐
E. Walking, Running & Jumping	$\frac{\text{Total Dimension E}}{72} = \frac{____}{72} \times 100 = ____\ \%$	E. ☐

TOTAL SCORE $= \frac{\%\,A + \%\,B + \%\,C + \%\,D + \%\,E}{\text{Total \# of Dimensions}}$

$= \frac{___ + ___ + ___ + ___ + ___}{5} = \frac{____}{5} ____\ \%$

GOAL TOTAL SCORE $= \frac{\text{Sum of \% scores for each dimension identified as a goal area}}{\text{\# Goal areas}}$

$= \frac{_____}{_____} = ____\ \%$