# SEVERE CROUCH GAIT IN THE SAGITTAL GAIT PATTERNS OF SPASTIC DIPLEGIC CEREBRAL PALSY: THE IMPACT OF SINGLE EVENT MULTILEVEL SURGERY

Jillian Maree Rodda

Submitted in total fulfilment of the requirements of the degree of Doctor of Philosophy

November 2005

School of Physiotherapy Faculty of Medicine, Dentistry and Health Sciences The University of Melbourne

### ABSTRACT

The purpose of this thesis was to study the outcome of Single Event Multilevel Surgery (SEMLS) on the gait pattern known as crouch gait in children with spastic diplegic cerebral palsy.

The term "crouch gait" in the literature has been defined by many authors to mean a flexed knee coupled with many different combinations of posture at the ankle. Consequently it was necessary to provide a robust definition of crouch gait before the outcome study could proceed.

Crouch gait was defined in the context of a classification of sagittal gait patterns in spastic diplegia. In the cross-sectional study on the classification of sagittal gait patterns, 187 children with spastic diplegia were categorised according to visual recognition of their gait pattern and sagittal plane kinematic data. Six gait patterns in spastic diplegia were identified, one of which was crouch gait. A pattern of increasing age, severity and biomechanical incompetency in maintaining an extended posture was seen across the gait patterns and crouch gait appeared to be the "end" gait pattern. A longitudinal study documented how the identified gait patterns evolved over time. Thirty-four children were followed for more than one year and the results indicated that the stability of the gait pattern was variable. The reliability of the classification was found to be acceptable.

The definition of crouch gait identified in our classification was distinct from the other gait patterns, but showed large variation in the extent of the crouch gait possible. Consequently a severe crouch gait subgroup was defined so that a homogeneous group of children walking in severe crouch gait was evaluated in the SEMLS outcome study. The children were assessed at baseline, one and five years post SEMLS. At baseline there was a predominance of prior calf surgery and lever arm dysfunction. Post SEMLS, improvements were found at one year at the knee and ankle that were maintained at five years. However improvement at the hip and pelvis was not seen. The anterior pelvic tilt increased and this was attributed to hamstring lengthening without concomitant psoas lengthening at the time of the index surgery.

The results from these studies show that sagittal gait patterns in spastic diplegia can be classified according to a combination of qualitative and quantitative data. The classification was unique as it demonstrated that the patterns were linked to each other biomechanically; they formed a continuum. The classification provided robust definitions that would allow for meaningful research to be undertaken in respect to outcome of gait pathology in spastic diplegia. SEMLS led to an improvement in crouch gait at one year and this improvement was maintained at five years which is important as the natural history of gait pathology in spastic diplegia is for deterioration with time. The surgical prescription did not adequately address the involvement of the hip and pelvis and further study into the effect of psoas lengthening at the hip and pelvis and alternatives to hamstring lengthening for the correction of severe crouch gait should be undertaken.

# DECLARATION

This is to certify that

- (i) the thesis comprises only my original work towards the PhD,
- (ii) due acknowledgement has been made in the text to all other material used,
- (iii) the thesis is less than 100,000 words in length, exclusive of tables, maps, bibliographies and appendices.

Jillian Rodda

November 2005

Declaration

#### ACKNOWLEDGEMENTS

To my beautiful children, Lisa and Angus for keeping me anchored to what is most important and precious in life.

To my supervisors: Professor Mary Galea, Professor Kerr Graham and Dr Richard Baker, for their support and encouragement during my PhD candidature. The wisdom and knowledge that they have shared with me during my candidature and during the years that I have had the good fortune to work clinically with them is greatly appreciated. Mary Galea has provided constant positive support and advice along the way. Kerr Graham has given his amazing clinical insight that has guided me in my thinking and consequent development of this thesis. Richard Baker has challenged me to consider further many different aspects of the thesis.

To Anne McCoy, former Chief Physiotherapist RCH, for her constant support over the many years that I had the honour of working with her, and for her belief in my ability to make a worthwhile contribution to the children and their families, and the physiotherapy profession.

To Dr Rory Wolfe, statistician for his unlimited patience, and ability to explain with immense clarity, such that I could always see the way forward with confidence and for passing on to me an actual enjoyment of statistics.

To Mary Sheedy for all her constant help and support over the years, and for her expert eye for anomalies that helped immensely in the editing of this thesis.

To Pam Thomason, wonderful colleague and great friend who has supported me through the "I cannot do this" times and given sensible and practical advice as needed on so many occasions.

#### Acknowledgements

To Dr Stefania Fatone, dear friend who has always given me immense professional support and her true friendship, and willingly undertook the "journey through crouch gait" by ploughing through this thesis in the later stages of my candidature.

To Fiona Dobson, another wonderful colleague who provided immense support and a sense of humour during this time.

To Dr Jenny McGinley, for her generous support and sharing of the funny and absurd moments that are part of undertaking a PhD! Plus the belief that eating chocolate will make everything appear better.

To Adrienne Fosang, for the tip on a cup of strong expresso (and supply of first bag of coffee) to keep me awake and productive during this whole process.

To Dr Anthony Schache for the practical tips and advice that have helped me move forward.

To Dr Wendy Harding for convincing me to convert to a PhD candidature. The only person who could understand fully what the ramifications of a PhD candidature would mean for me.

To all the children and families, with whom I have had the honour and privilege to work with over the years, for their contribution to my enjoyment of working in paediatrics. There have been happy and sad times, anxious and jubilant times, funny and serious times that I have experienced with many of you. I have learnt so much from you about dignity, bravery, "guts and determination", tenacity, optimism, and unconditional love that only parents have for their children. Having met and worked with you all, I am the one who has been enriched.

To all of you, thank you.

#### **PUBLICATIONS AND CONFERENCE PRESENTATIONS**

#### Publications

**Rodda J**, Graham HK (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.

Pirpiris M, Trivett A, Baker R, **Rodda J**, Nattrass GR, Graham HK (2003): Femoral derotation osteotomy in spastic diplegia. Proximal or distal? *The Journal of Bone and Joint Surgery* 85-B: 265-272.

Pirpiris M, Wilkinson AJ, **Rodda J**, Nguyen TC, Baker RJ, Nattrass GR, Graham HK (2003): Walking speed in children and young adults with neuromuscular disease: comparison between two assessment methods. *Journal of Pediatric Orthopaedics* 23: 302-307.

**Rodda JM**, Graham HK, Carson L, Galea MP, Wolfe R. (2004): Sagittal gait patterns in spastic diplegia. *The Journal of Bone and Joint Surgery* 86-B: 251-258.

Keenan WN, **Rodda J**, Wolfe R, Roberts S, Borton DC, Graham HK (2004): The static examination of children and young adults with cerebral palsy in the gait analysis laboratory: technique and observer agreement. *Journal of Pediatric Orthopaedics Part B* 13: 1-8.

Graham HK, Harvey A, **Rodda J**, Nattrass GR, Pirpiris M (2004): The Functional Mobility Scale (FMS). *Journal of Pediatric Orthopaedics* 24: 514-520.

**Rodda J**, Graham HK, Nattrass GR, Galea MP, Baker RJ, Wolfe R (Submitted): Correction of severe crouch gait in spastic diplegia by multilevel

orthopedic surgery: outcome at one and five years. *The Journal of Bone and Joint Surgery Am.* 

#### **Conference Presentations**

**Rodda J**, Baker R, Galea M, Graham HK (2002): A new biomechanical classification of sagittal gait patterns in spastic diplegia. *Gait and Posture* 16 (Supp 1): S7.

**Rodda J**, Baker R, Wolfe R, Galea M, Graham HK (2002): A new biomechanical classification of sagittal gait patterns in spastic diplegia: a cross-sectional and longitudinal study. *Developmental Medicine and Child Neurology* 44 (Suppl 91): 3.

**Rodda J**, Wolfe R, Galea M, Graham HK. A new biomechanical classification of sagittal gait patterns in spastic diplegia: cross-sectional and longitudinal study. Australasian Academy for Cerebral Palsy & Developmental Medicine 2<sup>nd</sup> Conference. 19-20<sup>th</sup> March 2004.

**Rodda J**, Wolfe R, Galea M, Graham HK. A new biomechanical classification of sagittal gait patterns in spastic diplegia. School of Physiotherapy. University of Melbourne. 26<sup>th</sup> October 2004. Seminar Presentation.

#### Awards

Royal Children's Hospital Brainwave Traveling Scholarship \$4,950. August 2002.

# **TABLE OF CONTENTS**

Title Page	i
Abstract	iii
Declaration	V
Acknowledgements	vii
Publications and Conference Presentations	ix
Table of Contents	xi
List of Tables	xvii
List of Figures	xxi
Abbreviations	xxvii
Glossary	xxix

### **CHAPTER 1 INTRODUCTION**

1.1	PURP	OSE OF THESIS	1
1.2	BACK	GROUND TO THESIS	1
1.3	SYNC	PSIS OF CHAPTERS	5
	1.3.1	Chapter 2	5
	1.3.2	Chapter 3	5
	1.3.3	Chapter 4	5
	1.3.4	Chapter 5	6
	1.3.5	Chapter 6	6
	1.3.6	Chapter 7	6
	1.3.7	Chapter 8	7
1.4	RESE	ARCH QUESTIONS AND HYPOTHESES	7
	1.4.1	Research Questions	7
	1.4.2	Hypotheses	7

### **CHAPTER 2 GAIT IN SPASTIC DIPLEGIA**

2.1	INTRODUCTION	9
2.2	CEREBRAL PALSY	10
	2.2.1 Classification of Cerebral Palsy	11

2.3	SPAS	TIC DIPLEGIA	27
	2.3.1	Gait Classification in Spastic Diplegia	28
2.4	CROU	JCH GAIT	38
	2.4.1	Definition	38
	2.4.2	What is Known about Crouch Gait?	40
2.5	ISSUI	ES RELATING TO CLASSIFICATION IN CEREBRAL	
	PALS	Y RESEARCH	58
	2.5.1	Lack of Homogeneity of the Type of Cerebral Palsy	58
	2.5.2	Lack of Identification of Gait Classification	59
	2.5.3	Lack of Consensus in Gait Classification	60
	2.5.4	Lack of Consensus Regarding Classification of Crouch	
		Gait	60
2.6	SUM	MARY	66
	2.6.1	Classification Consensus	66
	2.6.2	Crouch Gait	66
	2.6.3	Aims of the Research	67

### **CHAPTER 3 MANAGEMENT OF CROUCH GAIT**

3.1	INTR	ODUCTION	69
3.2	MAN	AGEMENT OF CEREBRAL PALSY	69
	3.2.1	Physiotherapy	70
	3.2.2	Spasticity Management	70
	3.2.3	Orthopaedic Surgery	72
	3.2.4	Orthoses	73
3.3	GAIT	ANALYSIS	74
	3.3.1	Visual Gait Assessment	76
	3.3.2	Physical Examination	77
	3.3.3	Three Dimensional Gait Analysis	77
	3.3.4	Electromyography	78
	3.3.5	Energy Expenditure Studies	78
	3.3.6	Interpretation of Data	79
	3.3.7	Issues in Gait Analysis	80
3.4	ORTH	IOPAEDIC SURGERY FOR CROUCH GAIT	89
	3.4.1	Single Event Multilevel Surgery (SEMLS)	89

3.5	ORTHOPAEDIC SURGERY FOR CROUCH GAIT		95
	3.5.1	Possible Causes	95
	3.5.2	Past Surgical Interventions	99
	3.5.3	Current Surgical Interventions	100
3.6	SUM	MARY	102
	3.6.1	Aim of the Research	102

### **CHAPTER 4 GENERAL METHODS**

4.1	INTRODUCTION		105
4.2	ETHI	CAL APPROVAL	105
4.3	SUBJ	ECTS	105
4.4	FUNC	CTIONAL CLASSIFICATION	106
4.5	CLIN	ICAL HISTORY	107
4.6	PHYS	SICAL EXAMINATION	107
	4.6.1	Anthropometric Measures	107
	4.6.2	Joint and Muscle Extensibility	110
	4.6.3	Grading of Rectus Femoris Spasticity	116
	4.6.4	Foot Posture	117
4.7	VIDE	O RECORDING OF GAIT	118
4.8	THRE	EE DIMENSIONAL GAIT ANALYSIS (3DGA)	118
	4.8.1	Equipment and Software	118
	4.8.2	Marker Placement	119
	4.8.3	Hip Joint Centre Calculation	120
	4.8.4	Walking Trials	121
	4.8.5	Terminology Used in Gait Analysis	123
4.9	STAT	'ISTICAL ANALYSIS	130
4.10	FRAN	AEWORK FOR PRESENTATION OF RESULTS	130

### **CHAPTER 5 GAIT PATTERNS IN SPASTIC DIPLEGIA**

INTRODUCTION		133
METH	IODS	134
5.2.1	Subjects	134
5.2.2	Procedure	135
5.2.3	Reliability Study	137
	METH 5.2.1 5.2.2	INTRODUCTION METHODS 5.2.1 Subjects 5.2.2 Procedure 5.2.3 Reliability Study

	5.2.4	Statistical Analysis	138
5.3	RESU	LTS	139
	5.3.1	Cross-sectional Study	139
	5.3.2	Longitudinal Study	145
	5.3.3	Reliability Study	146
5.4	DISCU	JSSION	148
	5.4.1	Cross-sectional Study	148
	5.4.2	Longitudinal Study	155
	5.4.3	Reliability Study	157
	5.4.4	Limitations of the Study	158
5.5	SUMN	ARY	160
5.6	CONC	CLUSION	161

# CHAPTER 6 FUNCTIONAL AND TECHNICAL PARAMETERS OF SEVERE CROUCH GAIT

6.1	INTRODUCTION		163
6.2	METI	HODS	164
	6.2.1	Study Design	164
	6.2.2	Subjects	164
	6.2.3	Procedure	165
	6.2.4	Statistical Analysis	175
6.3	RESU	JLTS	176
	6.3.1	Subjects	176
	6.3.2	Activities	178
	6.3.3	Body Structure and Function	180
6.4	DISCUSSION		193
	6.4.1	Subjects and Surgical History	193
	6.4.2	Activities- Mobility Status	193
	6.4.3	Body Structure and Function	194
	6.4.4	Limitations	207
6.5	SUM	MARY	208
6.6	CONCLUSION		212

#### Table of Contents

-			
7.1	INTRODUCTION		215
7.2	METH	HODS	216
	7.2.1	Subjects	216
	7.2.2	Surgical Intervention	210
	7.2.3	Post-operative Management	217
	7.2.4	Rehabilitation	218
	7.2.5	Study Procedure	219
	7.2.6	Statistical Analysis	219
7.3	RESU	JLTS	220
	7.3.1	SEMLS Intervention	222
	7.3.2	Activities- Mobility Status	225
	7.3.3	Body Structure and Function	230
7.4	DISC	USSION	260
	7.4.1	Positive Changes Post-SEMLS	260
	7.4.2	Maintained Status Post-SEMLS	284
	7.4.3	Negative Changes Post-SEMLS	286
	7.4.4	Crouch Gait: Resistant to SEMLS	300
	7.4.5	Statistical Analysis Considerations	304
	7.4.6	Limitations	305
.5	SUM	MARY	308
.6	CON	CLUSIONS	310

### CHAPTER 7 CAN SEVERE CROUCH GAIT BE CORRECTED?

## **CHAPTER 8 CONCLUSIONS AND CLINICAL IMPLICATIONS**

8.1	CONT	CONTRIBUTION OF THESIS	
8.2	LIMI	TATIONS	314
	8.2.1	Sagittal Gait Patterns in Spastic Diplegia	314
	8.2.2	Severe Crouch Gait	315
8.3	FUTU	JRE DIRECTIONS	316
	8.3.1	Sagittal Gait Patterns in Spastic Diplegia	316
	8.3.2	Severe Crouch Gait	318
8.4	CLIN	ICAL IMPLICATIONS	319
	8.4.1	Sagittal Gait Patterns in Spastic Diplegia	319
	8.4.2	Severe Crouch Gait	321

8.5 CONCL	USION	323
BIBLIOGRAP	HY	325
APPENDIX A	Gross Motor Function Classification System	363

APPENDIX A	Gross Motor Function Classification System
APPENDIX B	Functional Assessment Questionnaires
APPENDIX C	Functional Mobility Scale
APPENDIX D	Ethics Approval

# **LIST OF TABLES**

Table		Page
2.1	Historical timeline of the classification of cerebral palsy.	16
2.2	Classification of gait according to Hoffer et al.	20
2.3	An overview of the ICF.	25
2.4	Summary of criteria for crouch gait reported in the literature.	62
3.1	Summary of studies investigating the reliability of the physical examination measurements.	82
3.2	Summary of possible causes of crouch gait.	97
5.1	Inclusion and exclusion criteria for the cross-sectional study.	135
5.2	Gait pattern definitions: spastic diplegia.	142
5.3	Cross-sectional study of gait patterns: spastic diplegia.	
5.4	Longitudinal study of gait patterns: spastic diplegia.	
5.5	Repeatability of gait patterns: spastic diplegia.	147
6.1	Subject demographics and criteria for severe crouch gait.	177
6.2	Previous surgical intervention for each subject.	178
6.3	Comparison of means $\pm$ standard deviations and difference in means for temporal-spatial parameters at baseline between severe crouch and normal gait.	181
6.4	Comparison of means $\pm$ standard deviations and difference in means for pelvic and hip parameters at baseline between severe crouch and normal gait.	184
6.5	Comparison of pelvic and hip parameters in subjects with severe crouch gait (grouped according to the subject's independence or dependence on assistive devices) and normally developing subjects.	186

- 6.6 Comparison of means ± standard deviations and 188 difference in means for knee parameters at baseline between severe crouch and normal gait.
- 6.7 Comparison of means ± standard deviations and 189 difference in means for ankle parameters at baseline between severe crouch and normal gait.
- 6.8 Comparison of means ± standard deviations and 191 difference in means between severe crouch and normal gait, for the pelvis, hip and foot parameters in the coronal and transverse planes at baseline.
- 6.9 Comparison of means ± standard deviations and 192 difference in means between severe crouch and normal gait, for the timing and rate of change of parameters at baseline.
- 7.1 SEMLS itemized for each subject by code, age at surgery, 223 number of surgical procedures and type of surgery.
- **7.2** Surgery undertaken post-SEMLS by subject code and 224 type of surgery.
- **7.3** Orthotic use pre- and post-SEMLS intervention for the 225 subjects with severe crouch gait.
- **7.4** GMFCS pre, one and five years post-SEMLS for the 226 subjects with severe crouch gait.
- **7.5** FMS 5m pre, one and five years post-SEMLS for the 227 subjects with severe crouch gait.
- **7.6** FMS 50m pre, one and five years post-SEMLS for the 228 subjects with severe crouch gait.
- 7.7 FMS 500m pre, one and five years post-SEMLS for the 229 subjects with severe crouch gait.
- **7.8** FAQ pre, one and five years post-SEMLS for the subjects 230 with severe crouch gait.

xviii

7.9	Data (means $\pm$ standard deviations) for the severe crouch group at pre, one and five years post-SEMLS for	231
7.10	temporal-spatial parameters. Comparison of difference in means for the severe crouch data for temporal-spatial parameters at pre, one and five years post-SEMLS (plus normal data).	232
7.11	Comparison of means $\pm$ standard deviations for severe crouch data for the pelvic and hip parameters at pre, one and five years post-SEMLS.	237
7.12	Comparison of difference in means for the severe crouch data for pelvic and hip parameters at pre, one and five years post-SEMLS (plus normal data).	238
7.13	Comparison of means ± standard deviations for severe crouch data for the pelvic and hip parameters at pre, 1 and 5 years post-SEMLS for subjects without and those with assistive devices.	241
7.14	Comparison of difference in means for the severe crouch data for pelvic and hip parameters at pre, one and five years post-SEMLS for subjects without and those with assistive devices.	242
7.15	Comparison of means $\pm$ standard deviations for severe crouch data for the knee parameters at pre, one and five years post-SEMLS.	244
7.16	Comparison of difference in means for the severe crouch data for knee parameters at pre, one and five years post- SEMLS (plus normal data).	245
7.17	Comparison of means $\pm$ standard deviations for severe crouch data for ankle parameters at pre, one and five years post-SEMLS.	249

7.18	Comparison of difference in means for the severe crouch	250
	data for ankle parameters at pre, one and five years post-	
	SEMLS (plus normal data).	

- **7.19** Comparison of means  $\pm$  standard deviations for severe 253 crouch data for parameters in the coronal and transverse planes at pre, one and five years post-SEMLS.
- 7.20 Comparison of difference in means for the severe crouch 254 data for pelvic and hip parameters in the coronal and transverse planes at pre, one and five years post-SEMLS (plus normal data).
- 7.21 Comparison of means  $\pm$  standard deviations for severe 258 crouch data for the timing and rate of change of parameters at pre, one and five years post-SEMLS.
- 7.22 Comparison of difference in means for the severe crouch 259 data for timing of parameters at pre, one and five years post-SEMLS (plus normal data).
- **7.23** Summary of overview of changes in parameters post-262 SEMLS at five years.

# **LIST OF FIGURES**

Figure		Page
1.1	Concept map outlining the structure of the thesis.	4
2.1	Classification of cerebral palsy as proposed by SCPE.	18
2.2	Interactions between the components of the ICF.	26
2.3	The gait patterns identified by Bleck (1987, p. 306).	28
2.4	Two of the gait patterns identified by Rang et al (1986, p. 353).	29
2.5	The gait patterns in spastic diplegia identified by Miller et al (1995, p. 487).	30
2.6	Common gait patterns at the knee in spastic diplegia (Sutherland and Davids 1993, pp.142-146).	32
2.7	Example of cluster groupings published by O'Byrne et al (1998, pp. 102-105).	37
2.8	Coronal and sagittal views of a child with spastic diplegia who walks in crouch gait from Gage (2004f, p. 384).	39
2.9	Flexed knee with a) equinus and b) calcaneus.	65
3.1	Options for management of spasticity (Graham et al 2000, p. 71).	72
3.2	Orthoses commonly used in the management of cerebral palsy.	74
3.3	"The diving syndrome" as illustrated in Miller et al (1995, p. 488).	75
3.4	A child participating in a 3DGA in the gait laboratory.	78
3.5	A child participating in an energy test in the gait laboratory.	79

3.6	Paediatric orthopaedic surgeons, physiotherapists and biomechanical engineers contributing to a gait analysis interpretation and reporting session within our laboratory.	80
3.7	A child with spastic diplegic cerebral palsy participating in a physiotherapy session as part of her rehabilitation post-SEMLS.	90
3.8	Sagittal view of a child with spastic diplegia, at age 12 years, standing in crouch gait.	96
4.1	Tibial torsion as measured according to the Vicon Clinical Manager protocol (Oxford-Metrics 1995, p. 28).	110
4.2	The procedure for measurement of the Thomas Test.	111
4.3	The procedure for measurement of the popliteal angle.	112
4.4	The procedure for measurement of soleus length.	114
4.5	The procedure for measurement of gastrocnemius length.	114
4.6	The procedure for measurement of internal rotation of the	115
	hips.	
4.7	The procedure for measurement of external rotation of the hips.	116
4.8	Marker placement for subjects in the studies.	121
4.9	Divisions of the gait cycle.	124
4.10	Ankle rockers- showing $1^{st}$ , $2^{nd}$ and $3^{rd}$ rockers in normal gait.	126
4.11	Ground reaction force (seen as line extending from the ground upwards) exerted on the right lower limb in early stance.	128
4.12	The plantarflexion knee extension couple.	129
4.13	A typical sagittal plane kinematic graph of the knee.	130
5.1	Sagittal plane kinematic traces of a subject with spastic diplegia (black trace) for comparison with normal traces $\pm$ one standard deviation band (grey band).	136

### List of Figures

5.2	Algorithm of combinations of permutations and	141
	subsequent classification of gait patterns.	
5.3	Summarised kinematic data of the four basic gait patterns.	143
5.4	Ground reaction vector of gait patterns: spastic diplegia.	148
5.5	Diagram of each sagittal gait pattern, with dominant muscle groups identified for spasticity and/ or contracture management and appropriate orthotic prescription.	152
6.1	The sagittal plane kinematic parameters identified for the pelvis.	167
6.2	The sagittal plane kinematic parameters identified for the hip.	167
6.3	The sagittal plane kinematic parameters identified for the knee.	168
6.4	The sagittal plane kinematic parameters identified for the ankle.	169
6.5	The coronal plane kinematic parameters identified for the pelvis.	170
6.6	The coronal plane kinematic parameters identified for the hip.	170
6.7	The transverse plane kinematic parameters identified for the pelvis.	170
6.8	The sagittal plane moments identified at the hip.	171
6.9	The powers identified at the hip.	172
6.10	The sagittal plane moments identified at the knee.	172
6.11	The powers identified at the knee.	173
6.12	The power identified at the ankle.	173
6.13	Rate of movement of the knee kinematic between maximum knee extension and peak knee flexion in swing.	175
6.14	The rate of movement of the ankle kinematic between maximum dorsiflexion and plantarflexion of third rocker.	175

6.15	Rating of Functional Mobility Scale (FMS) at baseline for	179
	subjects with severe crouch gait.	
6.16	Rating of Functional Assessement Questionnaire (FAQ) at	180
	baseline for subjects with severe crouch gait.	
6.17	Kinematics at baseline.	182
6.18	Kinetics at baseline.	183
6.19	Sagittal plane kinematics for the pelvis and hip for subjects without and with assistive devices.	187
6.20	Radiographs of a) patella alta b) avulsion of patella and c) patellar fracture.	200
6.21	An example of how malalignment in the transverse plane alters the lever arm at the foot.	205
6.22	A schematic diagram of the length of the lever arms of the hamstrings at the hip and knee in a) normal gait and b) crouch gait.	210
7.1	Proximal entry ground reaction ankle foot orthosis (GRAFOs).	218
7.2	Diagram detailing the inclusion of the subjects who participated in the severe crouch SEMLS study.	221
7.3	Rating of GMFCS at pre, one and five years post-SEMLS for the subjects with severe crouch gait.	226
7.4	Rating of 5m FMS at pre, one and five years post-SEMLS for the subjects with severe crouch gait.	227
7.5	Rating of 50m FMS at pre, one and five years post- SEMLS for the subjects with severe crouch gait.	228
7.6	Rating of 500m FMS at pre, one and five years post- SEMLS for the subjects with severe crouch gait.	229
7.7	Rating of FAQ at pre, one and five years post-SEMLS for the subjects with severe crouch gait.	230

7.8	Sagittal plane kinematics pre, one and five years post- SEMLS.	234
7.9	Sagittal plane moments pre, one and five years post-SEMLS.	235
7.10	Kinetics- powers pre, one and five years post-SEMLS.	236
7.11	Pelvic and hip sagittal plane kinematics for subjects with severe crouch gait, walking without assistive devices and those walking with assistive devices pre, one and five years post-SEMLS.	243
7.12	Change in minimum knee flexion for subjects with severe crouch gait at pre, one and five years post-SEMLS.	246
7.13	Change in knee excursion from pre, one and five years post-SEMLS for the subjects with severe crouch gait.	246
7.14	Change in knee flexor moment from pre, one and five years post-SEMLS for the subjects with severe crouch gait.	247
7.15	Change in peak knee flexion in swing from pre, one and five years post-SEMLS for the subjects with severe crouch gait.	248
7.16	Change in maximum dorsiflexion from pre, one and five years post-SEMLS for the subjects with severe crouch gait.	251
7.17	Change in ankle power from pre, one and five years post- SEMLS for the subjects with severe crouch gait.	251
7.18	Coronal plane kinematics pre, one and five years post-SEMLS.	252
7.19	Transverse plane kinematics pre, one and five years post-SEMLS.	255
7.20	Change in mean hip rotation in stance phase from pre, one and five years post-SEMLS for subjects with severe crouch gait.	256

7.21	Subject 2 walking pre,	one and five year	s post-SEMLS.	263
------	------------------------	-------------------	---------------	-----

- 7.22 Subject 8 walking pre, one and five years post-SEMLS. 264
- 7.23 Subject 11 walking pre, one and five years post-SEMLS. 265
- **7.24** The effect on gait of tibial nerve block to the left calf 274 muscles, from Sutherland et al (1980, p. 359).
- **7.25** After sagittal plane soft tissue release surgery, ground 279 reaction ankle foot orthoses (GRAFOs) provide support to stabilize the ankle in mild plantarflexion to force the ground reaction force (GRF) anterior to the knee.
- **7.26** Subject in a) crouch and b) subject post-distal femoral 303 extension osteotomy with the hip/ pelvis unaltered in alignment to the femur.

## **ABBREVIATIONS**

A2	ankle power generation in stance	Gastroc	gastrocnemius
Abd	abduction	Gen	generation of power
Abs	absorption of power	Glut max	gluteus maximus
Add	adduction	GMFCS	Gross Motor Function
ADD-L	adductor longus lengthening		Classification System
AFO	ankle foot orthosis	GMFM	Gross Motor Function
Ank	ankle		Measure
Ant	anterior	GRAFO	ground reaction ankle
AO (ASIF)	"Arbeitsgemeinschaft für		foot orthosis
	Osteosynthesefragen"	GRF	ground reaction force
	Internal Fixation)	H1	hip power generation in
ASIS	anterior superior iliac spine		early stance
BoNT-A	Botulinum toxin A	Н3	hip power generation
CI	confidence interval		late stance
СР	cerebral palsy	HMS	hamstrings
Decr	decrease	ICC	Intraclass Correlation
Deg	degree		Coefficcient
Dega acetab	Dega acetabular osteotomy	ICF	International
DF	dorsiflexion		Classification
Dor	dorsiflexion		of Functioning,
Dorsi	dorsiflexion		Disability and Health
Dorsiflex	dorsiflexion	IGTN	ingrown toe nail surgery
EMG	electromyography	Incr	increase
ER	external rotation	IR	internal rotation
Est. mean diff	Estimated mean difference	ITB	intrathecal baclofen
Ext	extension	KAD	knee alignment device
FAQ	Gillette Functional Assessment	Kext	knee extension
	Questionnaire	Kn	knee
FDO(D)	femoral derotation osteotomy	LAD	lever arm dysfunction
	Distal	LHS	lateral hamstrings
FDO(P)	femoral derotation osteotomy	MHS	medial hamstrings
	Proximal	Mom	moment
FFD	fixed flexion deformity	MRI	magnetic resonance
Flex	flexion		imaging
Flx	flexion	MST	midstance
FMS	Functional Mobility	MTP fusion	metatarsal phalangeal
	Scale		fusion

Nm	Newton metres
Nm/kg	Newton metres/ kilogram
Os calcis Lx	Os calcis lengthening
Peron br lx	peroneus brevis lengthening
Physio	physiotherapy
PIG	Plug in Gait
Pla	plantarflexion
Plantar	plantarflexion
Plantarflex	plantarflexion
PF	plantarflexion
Post	posterior
РОТВ	psoas over the brim
Prox rect	proximal rectus release
PSIS	posterior superior iliac spine
Pst	posterior
РТ	physiotherapist
Quads	quadriceps
RFT-ST	rectus femoris transfer to semitendinosis
SDR	selective dorsal rhizotomy
SEMLS	single event multilevel surgery
SMO	supramalleolar osteotomy
ST	stance phase
STD	standard deviation
STJ fusion	subtalar joint fusion
SW	swing phase
Sx	surgery
TAL	tendo achilles lengthening
Tib ant Tx	tibialis anterior transfer
Tib post divn	tibialis posterior division
Tot	total
VCM	Vicon Clinical Manager
W	Watts
W/kg	Watts/ kilogram
Yr <sup>mm</sup>	years and months of age
3DGA	three dimensional gait analysis

# GLOSSARY

Ankle rockers	motion of foot and ankle during stance phase
1 <sup>st</sup> rocker	initial contact with heel, pre-tibial muscles lower
	forefoot to ground
2 <sup>nd</sup> rocker	forefoot is in contact with ground, fulcrum moves
	forward from ankle to metatarsal heads as tibia
	moves forward over the stationary foot
3 <sup>rd</sup> rocker	fulcrum is located at the metatarsal heads and tibia
	is no longer moving forward in relation to the
	foot, heel lifts from floor
Cadence	number of steps taken per minute (steps/min)
Double support	period of foot contact with ground during which
	contralateral foot is also in contact with ground
External moments	produced by combination of ground reaction
	forces, segmental weight and inertia
Gait cycle	duration of initial contact to next initial contact of
	same foot
Ground reaction force	external force exerted by the supporting surface
	onto the body, applied through the centre of
	pressure
Initial contact	first contact of foot with ground
Initial swing	begins with foot off and finishes when the foot is
	opposite the contralateral stance limb
Internal moments	produced by combination of forces from muscles,
	ligaments, joint capsule and bones
Kinematics	describe movement of joints and body segments:
	linear and angular displacement, velocity and
	accelerations
Kinetics	describe forces that cause movement: moments
	and powers
Loading response	initial contact until toe off of contralateral foot: 1 <sup>st</sup>
	double support

Midstance	toe off of contralateral foot to heel rise of ipsilateral foot
Midswing	begins when the ipsilateral swing foot is opposite the contralateral stance limb and finishes when the ipsilateral limb is brought forward and the tibia is aligned vertically
Moments	product of force applied and distance of the force from the fulcrum on which the force is acting (Newton metres/ kilogram)
<b>Opposite foot contact</b>	start of initial contact of contralateral foot
<b>Opposite foot off</b>	lift off of contralateral foot
Plantarflexion knee	through midstance, the soleus (plantarflexors)
extension couple	slows the forward movement of the tibia over the
	foot and the ground reaction force (GRF) moves
	forward onto the forefoot, which lengthens the
	lever on which the GRF is acting and brings it in
	front of the knee joint and so creates an external
	extension moment at the knee
Power	product of the net joint moment and angular velocity (Watts/ kilogram)
Pre swing	from contralateral foot contact to ipsilateral foot off: 2 <sup>nd</sup> double support
Single support	period of foot contact with ground whilst contralateral foot is in swing phase
Stance phase	duration of single foot contact with ground: initial
Stance phase	contact to toe off
Step length	distance from toe off to intial contact (metres)
Stride length	distance from initial contact to repeat initial
	contact of same foot (metres)
Swing phase	duration of single foot non contact with ground:
	toe off to initial contact
Temporal-spatial data	time and distance parameters
Terminal stance	from ipsilateral heel rise to contralateral foot
	contact with the ground

### Glossary

Terminal swing	from when the tibia is vertical in swing phase until initial
	contact
Toe off	lift off of foot from ground

Glossary

### **CHAPTER 1 INTRODUCTION**

#### **1.1 PURPOSE OF THESIS**

The purpose of this thesis was to evaluate the outcome of Single Event Multilevel Surgery (SEMLS) on severe crouch gait at one and five years in respect to changes from baseline data and normal values, and also at five years in respect to the one year data for deterioration, maintenance or improvement in severe crouch gait.

#### **1.2 BACKGROUND TO THESIS**

One of the defining characteristics of cerebral palsy is that of movement disorder (Bax 2001). This dysfunction in movement is a manifestation of the upper motor neuron lesion that is part of cerebral palsy. There are negative and positive features as a result. The positive features are those of spasticity, hyperreflexia and co-contraction and the negative features are muscle weakness, loss of selective motor control and poor balance (Barnes 2001, Sheean 2002). Both agonist and antagonist muscle groups can be spastic and weak. Over time and with growth of the long bones, the spastic, weak muscles may begin to tighten and develop contractures (Graham and Selber 2003). With long standing contracture, bony torsion develops in the lower limbs. This torsion can be in the femur and/ or tibia and can lead to malalignment in the lever arms of the lower limb (Gage 2004d, Schwartz and Lakin 2003). Eventually this malalignment can lead to the development of joint instability and arthritis especially at the hip and foot (Graham and Selber 2003).

The spastic diplegic form of cerebral palsy, is recognised clinically as bilateral involvement but the lower limbs show greater involvement than the upper limbs (Shevell et al 2003). Crouch gait is a term attributed to a common gait pattern seen in spastic diplegia. Crouch gait has been considered to be excessive knee flexion throughout stance phase (Frost 1971, Sutherland and Cooper 1978,

#### Chapter 1

Sutherland and Davids 1993). This excessive knee flexion throughout stance has been documented as being accompanied by ankle dorsiflexion, hip flexion and in some cases hip internal rotation and adduction (Delp et al 1996, Schutte Other authors have included ankle plantarflexion, not just et al 1997). dorsiflexion in their definitions of crouch gait (Rab 1992b, Steinwender et al 2001). Therefore crouch gait is a nebulous term to imply a flexed knee in stance but with variable hip and ankle posture. This has lead to the term flexed knee gait often being used synonymously clinically and in the literature with the term crouch gait. However due to the lack of a precise definition of the crouch gait pattern, there is a body of research that purports to provide insight into the causes of crouch gait and interventions that are applicable to the crouch gait pattern but this may not necessarily be correct. This lack of classification of gait patterns in spastic diplegia has led to research that has been conducted on children, who were exhibiting many and varied patterns of gait e.g. equinus versus calcaneus at the ankle, and flexed knee versus straight or hyperextended knee gait. It is not possible for research that is being undertaken to look at gait correction to obtain meaningful results if the subjects who have been recruited are not categorised according to their gait pattern. This is because different gait patterns will be responsive to different interventions due to the differing biomechanics of the respective gait patterns. Therefore a rigorous definition of crouch gait was required prior to the commencement of the study on the effect of SEMLS on this gait pattern.

- It was the aim of this thesis to develop a classification system for the sagittal plane gait patterns of children with spastic diplegia and in doing so provide a robust definition of crouch gait.
- It was the aim of this thesis to document the baseline parameters that constitute crouch gait, according to the definition in the classification of sagittal gait patterns in spastic diplegia.

It has been thought that weak, overlong ankle plantarflexor muscles and hamstring contracture are the cause of crouch gait (Sutherland and Cooper 1978,

Sutherland and Davids 1993). The cause of overlong plantarflexor muscles may be iatrogenic from isolated lengthening of these muscles or from inherent muscle weakness (Sutherland and Cooper 1978, Sutherland and Davids 1993). Management of crouch gait in the past has consisted of physiotherapy, orthotic use and orthopaedic surgery. The orthopaedic surgery was undertaken in multiple stages so that the child was subjected to many stays in hospital and subsequently, many periods of rehabilitation (Miller et al 1995). With advances in computer technology, gait analysis studies have become more accessible and have come to represent a part of routine clinical assessment in many clinics, usually indicated when gait begins to exhibit signs of deterioration. The gait analysis provides objective data that the clinicians can use to help in the deciphering of what abnormalities constitute the primary and secondary gait problems and what are compensatory to this (Davids 2003b, Gage et al 1995). Clinicians can now direct management to multiple muscles and bones in the lower limbs with more accuracy and consequently SEMLS has evolved as an SEMLS means for the child one major hospitalisation and intervention. rehabilitation period to address the orthopaedic correction of their gait pathology (Graham and Selber 2003). The effect of SEMLS on the correction of crouch gait and long term ambulatory potential has not been documented in the published literature.

• It was the aim of this thesis to analyse the effect of SEMLS on the correction of crouch gait at one and five years post SEMLS.

A concept map outlining the structure of the thesis is shown in Figure 1.1

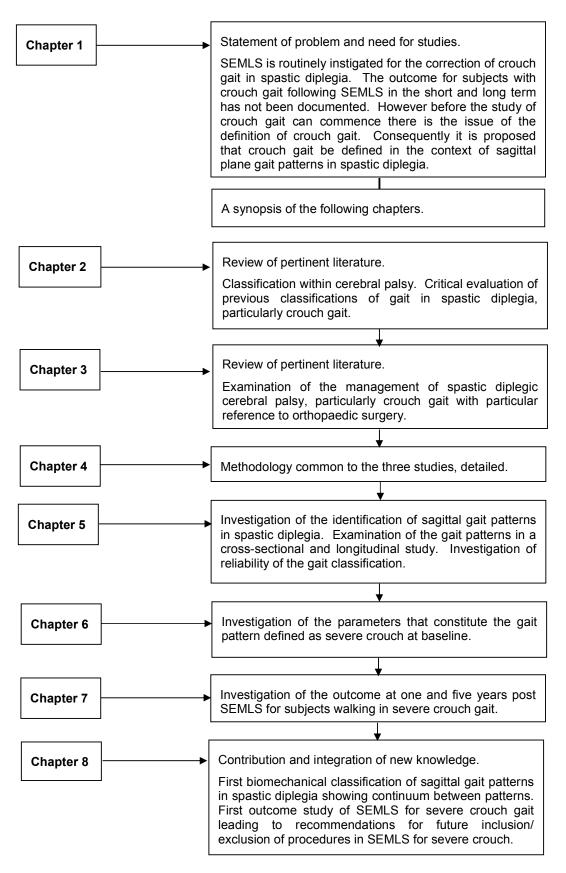


Figure 1.1 Concept map outlining the structure of the thesis.

#### **1.3 SYNOPSIS OF CHAPTERS**

#### 1.3.1 Chapter 2

This chapter provides a summary of the literature that comprises the current knowledge of gait patterns in spastic diplegia, particularly that of crouch gait.

A background to cerebral palsy and its classification is given. Information is provided on spastic diplegia as this is the subgroup of cerebral palsy that constitutes the cohort for the studies. Previous gait classifications in spastic diplegia are examined in respect of whether they were developed from qualitative versus quantitative data and for clinical utility. The literature pertaining to crouch gait is evaluated as to the definition provided and the consequent research findings.

From this examination of the current knowledge of gait patterns in spastic diplegia, and in particular crouch gait, a number of concerns are identified.

#### 1.3.2 Chapter 3

The management of cerebral palsy is outlined as to the variety of management strategies that are available and the indications for their use. Gait analysis as an assessment tool to aid in treatment decision-making is discussed and the limitations associated with it are elaborated on as gait analysis data are used as outcome variables in the studies undertaken. SEMLS as an orthopaedic treatment for children with spastic diplegia is explained and the most common orthopaedic surgeries at the level of the hip, knee and ankle are documented. The diversity in what are purported to be the causative factors in the development of crouch gait is examined and past and current surgery for the correction of crouch gait is detailed.

Unresolved issues in the management of crouch gait are highlighted.

#### 1.3.3 Chapter 4

The general methods that are relevant to the studies in this thesis are outlined here. Any methods that are specific to a particular study are detailed in the relevant chapter under the methods section. Physical examination measures are detailed. The protocol for data collection in a three dimensional gait analysis (3DGA) is given. Common terminology used in gait analysis is provided.

### 1.3.4 Chapter 5

The development of the classification of sagittal gait patterns in spastic diplegia is detailed, and comprises three studies: a cross-sectional, a longitudinal and a repeatability study. The cross-sectional study consists of the process of classifying the gait patterns using video observation and kinematic data. The longitudinal study is undertaken to assess whether the gait patterns are stable or show progression to another gait pattern over time. Intra- and inter-rater repeatability studies are undertaken in order to establish if the classification system will be reliable for use clinically and in research.

# 1.3.5 Chapter 6

The definition of crouch gait from the classification of sagittal gait patterns in Chapter 4 is further refined to designate the subgroup of severe crouch gait. The baseline data for the severe crouch cohort are documented here and referenced to normal data statistically. These baseline data are used in the following outcome study of SEMLS. This provides a comprehensive description of severe crouch gait in respect to physical examination, 3DGA and functional mobility.

#### **1.3.6** Chapter 7

The outcome study of the effect of SEMLS on severe crouch gait at one and five years is discussed in this chapter. This has not been documented previously except by case studies (Gage 2004a), which indicate that at skeletal maturity the correction afforded by SEMLS is maintained. As the literature has shown that the natural history of gait is for deterioration (Bell et al 2002, Gough et al 2004, Johnson et al 1997), the maintenance of correction at skeletal maturity of severe crouch gait post SEMLS would be an important result. Analysis of parameters for improvement, maintenance or deterioration allows for discussion of the individual surgeries and combinations of, within SEMLS that were undertaken and whether these should be continued, modified or discarded in order to obtain

a better correction of some of the parameters. This should lead to new combinations of surgeries being carried out in SEMLS that will be more effective in correcting crouch gait.

# 1.3.7 Chapter 8

The final discussion integrates the results from Chapters 5, 6 and 7 and the implications for clinical practice. The contribution of this thesis to the knowledge in the area of gait pattern classification in spastic diplegia and in particular crouch gait and its causes and effective management, is presented. Methodological issues relating to the studies are discussed and how these might be resolved in future studies are examined. Future directions for research into crouch gait and gait patterns in spastic diplegia, are proposed.

# **1.4 RESEARCH QUESTIONS AND HYPOTHESES**

The aims of the thesis were documented in Section 1.2.

# 1.4.1 Research Questions

- Can the gait patterns in spastic diplegia be inclusively classified in the sagittal plane?
- What are the technical and functional parameters of severe crouch gait if defined according to a robust definition that includes the posture of the pelvis, hip, knee and ankle?
- Can SEMLS correct severe crouch gait? And if correction is obtained, is it maintained at skeletal maturity?

# 1.4.2 Hypotheses

- There are specific sagittal plane gait patterns that can be identified in spastic diplegia.
- Severe crouch gait has specific clinical and biomechanical parameters.
- Biomechanical alignment and functional ability in severe crouch gait can be improved by SEMLS.

• Correction of severe crouch gait post SEMLS can be maintained until skeletal maturity.

# **CHAPTER 2 GAIT IN SPASTIC DIPLEGIA**

# **2.1 INTRODUCTION**

The majority of children who have cerebral palsy are able to walk. However these children exhibit many different gait patterns and great variation in severity. This suggests that gait disorders in cerebral palsy cannot necessarily be managed by the same interventions, as the intervention needs to be tailored to the gait disorder exhibited by the child. Research into the management of gait in cerebral palsy and the resulting implications for clinical management, need to be specific to the different gait patterns that exist. This is reliant on a comprehensive identification of gait patterns within the different subtypes of cerebral palsy.

Crouch gait is a gait pattern, which is seen in bilateral spastic cerebral palsy, such as spastic diplegia. The aim in management of this gait pattern is based on regaining an extended posture at the hip and knee, and plantargrade posture at the ankle. SEMLS is an intervention that aims to achieve this biomechanical alignment. The outcome of SEMLS on the correction of crouch gait has not been followed in the short or long term. However in order for SEMLS for correction of crouch gait to be studied, a sturdy definition of crouch gait needs to be established.

In this chapter, the classification of cerebral palsy has been described, and past descriptions of different walking patterns in the subtype of spastic diplegia have been detailed. In particular, attention has been given to the gait pattern called crouch as to how it has been defined, and what are the known characteristics of crouch gait in the context of the definition used in the particular studies. Any areas of knowledge, which were found to be incomplete in regard to the classification of gait patterns in spastic diplegia and classification of crouch gait were identified. Consequently appropriate research studies, which aim to provide new knowledge in these areas, were proposed.

### 2.2 CEREBRAL PALSY

In 1862 Little, an orthopaedic surgeon, classified cerebral palsy according to clinical findings (Ingram 1984), and Freud later wrote on infantile cerebral palsy (Accardo 1982). There have been many definitions of cerebral palsy published since the time of Little (Accardo 1982, Bax 1964, Ingram 1984, Minear 1956). One of the more recent definitions of cerebral palsy is- "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 et al 1992, p. 549). It is recognised that this definition of cerebral palsy encompasses conditions that are heterogeneous in respect to aetiology, time of onset of lesion to the central nervous system, topography, severity, and neurological symptoms (Flett 2003, Reddihough and Collins 2003, Stanley et al 2000). The key points within the definition are that the lesion is to the immature brain, the lesion is non-progressive and permanent but the resulting movement disorder can be changing (Bax 1964, Flett 2003, Shepherd 1980, Shevell et al 2003, Stanley et al 2000). The management of cerebral palsy therefore aims to address the progression of the motor symptoms over time, not the cerebral lesion that is permanent and non-progressive.

The reported rate for cerebral palsy is around 2 to 2.5 cases per 1000 live births (Stanley et al 2000) and has remained fairly constant in developed countries. This is despite advances in improved obstetric and neonatal care resulting in reduced mortality, challenging the past held notion that birth asphyxia played a major role in the aetiology of cerebral palsy (Nelson 2002, Reddihough and Collins 2003, Stanley et al 2000). Cerebral palsy is also associated with impairments other than motor difficulties. Associated impairments can include epilepsy, reduced intellectual and cognitive function, sensory deficits, communication difficulties, and visual and auditory impairments (Kuban and Leviton 1994, Russman and Gage 1989, Shepherd 1980, Stanley et al 2000, Winders Davis 1997a). There is a new definition of cerebral palsy, currently proposed, which incorporates these associated impairments into the definition of cerebral palsy (Bax et al 2005).

It is important for the aetiologies of cerebral palsy to be identified, so that efforts towards prevention of cerebral palsy can be made. However it is the symptoms resulting from the cerebral palsy such as spasticity, contracture, bony torsion, and muscle weakness that are addressed in treatment (Badawi et al 1998). Identification of the causal factors or aetiology of cerebral palsy as opposed to the risk factors that may lead to the development of cerebral palsy is difficult to decipher (Kuban and Leviton 1994). Aetiological and risk factors for cerebral palsy have been ascribed to antenatal insult(s) (intrauterine bacterial or viral infections, congenital central nervous system anomalies, chromosomal abnormalities, teratogenic drugs, placenta praevia), perinatal difficulties (neonatal encephalopathy with seizures, intrapartum asphyxia, prematurity, low birth weight, multiple gestation) and postnatal insult (infection, anoxia, haemorrhage, trauma, near drowning) (Reddihough and Collins 2003, Winders Davis 1997b). However, there are times when none of these aetiological or risk factors can be ascertained in term infants with cerebral palsy (Rosenbaum 2003, Shepherd 1980, Shevell et al 2003, Yin et al 2000).

### 2.2.1 Classification of Cerebral Palsy

Two children, each with the diagnosis of cerebral palsy, may have different aetiologies, motor disorders, levels of involvement and severity, and functional abilities. The definition of cerebral palsy allows for considerable heterogeneity between children with the diagnosis and "it is unlikely that *any* management intervention is useful across the entire group" (Stanley et al 2000, p. 181). Therefore in order to obtain groups within cerebral palsy at numerous levels have been developed. Classifications within cerebral palsy aims to categorise subjects into a group according to the possession of similar characteristics, qualities or symptoms. Rating scales are useful in providing a measure of difference between subjects that is graded or incremental. A classification system assigns a subject according to delineated categories that may or may not be graduated, and is not developed with the intention that it would be used as an outcome measure. However it cannot be assumed that once assigned to a particular classification, the subject will not require reassigning at a later date due to the

changing nature of the motor impairment symptoms in cerebral palsy. In contrast, a rating scale can be utilised as an outcome measure because it contains ordered and/ or graded descriptions.

Classifications and rating scales can improve communication between clinicians regarding the symptoms or characteristics of a patient, provide homogenous groups of subjects in research, and promote intervention according to appropriate patient groups (Sanger et al 2003).

Cerebral palsy has in the past been classified according to a system that reflects the clinical symptoms in the child presenting for assessment (Stanley et al 2000). This classification has been based on the motor disorder, distribution of involvement, level of function or any combination of these. Classifications based on aetiology or pathology of the brain lesion have been attempted but difficulties with identification of these aspects have limited the use of such classifications in the past (Holling and Leviton 1999, Okumura et al 1997, Rogers et al 1994, Stanley et al 2000).

#### **Classification by Motor Disorder**

#### Spasticity

In 1979 at an international meeting in Arizona, spasticity was defined thus: "Spasticity is a motor disorder characterised by a velocity-dependent increase in tonic stretch reflexes ('muscle tone') with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motor neuron syndrome" (Lance 1980, p. 606, Lance 1990). Spasticity can be expressed more practically "as a velocity-dependent increased resistance to passive muscle stretch" (Albright 1996, p. S2). More recently a dynamic definition of spasticity was proposed: "a movement disorder characterised by increased coupling between the levels of muscle activation and muscle lengthening velocity (either in terms of increased gain or reduced threshold), associated with enhanced mechanical resistance to rotation at the relevant joint(s)" (Crenna 1998, p. 576). This definition recognises that spasticity is not just an entity that is only evoked with passive movement. Spasticity is seen as one component that contributes to the resulting movement, together with recruitment of motor units (this may be deficient and lack selectivity) and altered muscle physiology. All combine to result in the movement dysfunction that the subject experiences and hence treatment should be directed not just to altering spasticity but also the other components of the movement disorder.

#### **Dyskinesia**

The term 'dyskinesia' denotes involuntary movements, which are rarely found in isolation but usually in conjunction with spasticity. Involuntary movements may be characterised as writhing (athetosis), rigid abnormal posturing (dystonia) or fast jerking movements (choreosis) (Stanley et al 2000). Dystonia has been further defined as being induced by active movement in which "involuntary sustained or intermittent muscle contractions cause twisting and repetitive movements, abnormal postures, or both" (Sanger et al 2003, p. 92).

### Ataxia/ Hypotonia

Ataxia is inco-ordination of movement resulting in movement that can be jerky and show dysmetria. Decreased tone affects postural stability around the joints particularly the large, proximal joints (Evans et al 1986). These signs rarely constitute the major neurological motor impairment (Cash 1976, SCPE 2000, Stanley et al 2000).

# **Rigidity**

In children, rigidity is rarely reported (Sanger et al 2003). A recent definition of rigidity states "the resistance to passive movement is independent of posture and speed of movement" (Sanger et al 2003, p. 93). Hence the rigidity is unchanging with variation of the speed of passive movement. There is co-contraction of the agonist and antagonist muscles about the joint being moved and there is no dependence for the joint to return to a certain posture (Sanger et al 2003).

# Mixed

Any of the motor disorders can be found in combination with another and classification is therefore usually based on the predominant motor impairment

found (Stanley et al 2000). A common combination is that of spasticity and dystonia, known as "mixed hypertonia" (Sanger et al 2003, p. 93).

# **Classification by Distribution of Involvement**

The three most common topographies that have been used in cerebral palsy arehemiplegia, diplegia and quadriplegia.

# <u>Hemiplegia</u>

This term is used when there is involvement of only one side of the body (Bobath and Bobath 1978) and the upper limb involvement can be greater than that of the lower limb (Dabney et al 1997).

# <u>Diplegia</u>

In diplegia, the whole body is involved but the lower limbs show greater severity of symptoms and functional involvement than the upper limbs (Bobath and Bobath 1978, Hagberg et al 1975, Shepherd 1980, Shevell et al 2003).

# Quadriplegia

In quadriplegia, again the whole body is involved, but the upper limbs show equal or greater involvement than the lower limbs (Bobath and Bobath 1978, Hagberg et al 1975, Shepherd 1980, Shevell et al 2003).

Monoplegia, paraplegia, tetraplegia and double hemiplegia are terms also used clinically and in the literature (Kuban and Leviton 1994, Minear 1956, Rosenbaum 2003, Russman and Gage 1989)

It has been recommended that the above two methods of classification, motor disorder and distribution of involvement, combined represent the minimum required for classification in cerebral palsy (Scrutton 1992). However classification based on these parameters remains problematic and there is still controversy regarding the correct nomenclature that should be universally adopted. Classifications have been hampered by poor interobserver agreement (Blair and Stanley 1985) and consequently the acceptance of an international classification has been suggested (Blair and Stanley 1985). There have been

many very different classifications of cerebral palsy put forward since the time of Little's original description, which reveal the disparity that has existed in the literature (Bax 1964, Colver and Sethumadhavan 2003, Ingram 1984). Table 2.1 reproduced from Colver and Sethumadhavan (2003, p. 287), very clearly shows how classification of cerebral palsy has developed and changed historically.

Reference	Year	Classification		
Little (1862)	1862	Hemiplegic rigidity Paraplegic rigidity Generalised rigidity Disordered movements without rigidity		
Sachs and Petersen (1890)	1890	Paralysis of intrauterine origin Birth palsies Acute acquired palsies	Diplegia Paraplegia Hemiplegia Diplegia Paraplegia Hemiplegia Diataxia (ataxia) Diplegia Paraplegia Hemiplegia Choreo-athetoid	
Freud (Accardo 1982)	1893	Unilateral disorders— hemiplegia Bilateral disorders—diplegia	Right or left Generalised rigidity Paraplegic rigidity Bilateral hemiplegia Choreo-athetosis Others	
Wylie (1951)	1951	Congenital symmetric diplegia Congenital paraplegia Quadriplegia or bilateral hemiplegia Hemiplegia with additional qualifications referring to all categories	Choreo-athetoid cerebral palsy Mixed forms of cerebral palsy Ataxic cerebral palsy Atonic diplegia	
Ingram (1955)	1955	<i>Neurology</i> Hemiplegia	<i>Extent</i> Right or left	<i>Severity</i> Mild Moderate Severe
		Double hemiplegia Diplegia Hypotonic Dystonic Rigid or spastic Ataxia	Paraplegia Triplegia Tetraplegia	Mild Moderate Severe Mild Moderate Severe
		Cerebellar Vestibular	Unilateral Bilateral	Mild Moderate Severe

Table 2.1Historical timeline of the classification of cerebral palsy. Reproducedfrom Colver and Sethumadhavan (2003, p. 287).

Reference	Year	Classification		
		Ataxic diplegia Hypotonia Spastic Dyskinesia	Paraplegia Triplegia Tetraplegia	Mild Moderate Severe
		Dystonic Choreoid Athetoid Tension Tremor Other	Monoplegia Hemiplegia Triplegia Tetraplegia	Mild Moderate Severe
Minear (1956)	1956	A. Physiological	Spasticity, athetosis, rigidity, ataxia, tremor, atonia, mixed, unclassified	
		B. Topographical	Monoplegia, dipleg paraplegia, hemip triplegia, quadriple	legia,
		C. Aetiological	Prenatal, natal anoxia, postnatal, cause described	
		F. Neuroanatomical G. Functional capacity H. Therapeutic	Class I–IV Class I–IV	
Little Club (MacKeith et al 1959)	1959	Spastic cerebral palsy	Hemiplegia Diplegia Double hemiplegia	a
		Dystonic cerebral palsy Choreo-athetoid cerebral palsy Mixed forms of cerebral palsy Ataxic cerebral palsy Atonic diplegia		
Hagberg et al (1975)	1975	Spastic	Hemiplegia Tetraplegia Diplegia	
		Ataxic	Congenital Diplegia	
		Dyskinetic	Dystonic- mainly Choreo-athetotic	
SCPE(2000)	2000	Spastic	Bilateral spastic Unilateral spastic	
		Ataxic Dyskinetic	Dystonic Choreo-athetotic	

A recent attempt (SCPE 2000) to achieve consensus, involved 14 centres in eight European countries coming together to agree on common definitions, criteria and classification of children with cerebral palsy to facilitate collaborative research, epidemiology and service planning in the future. The terms of spastic quadriplegia, diplegia and hemiplegia were replaced by classifying spastic cerebral palsy as bilateral (limb involvement on both sides) or unilateral (limb involvement only on one side) (SCPE 2000) (Figure 2.1). The term spastic bilateral cerebral palsy therefore encompasses children previously classified as diplegia and quadriplegia differentiated now only by documentation of the number of limbs involved (Gorter et al 2004). This new classification has not been adopted for use in this thesis as it was not published until 2000, and the work contained in this thesis commenced prior to this time. Subject selection was made according to the classification of spastic diplegia at that time. Adoption of the term spastic bilateral cerebral palsy would imply that ambulant children with spastic quadriplegia were included in the studies in this thesis and this was not so.

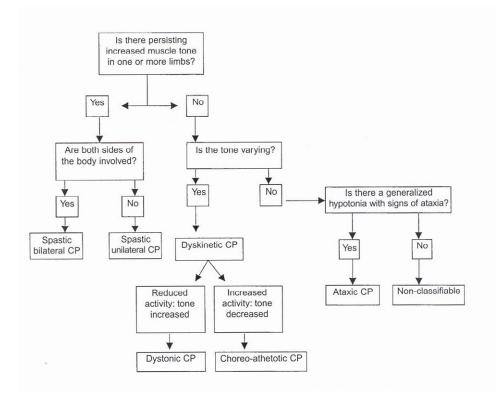


Figure 2.1 Classification of cerebral palsy as proposed by SCPE. Reproduced from SCPE (2000).

#### **Classification by Level of Function**

Functional ability to participate in real everyday situations, such as moving within the home, classroom, workplace, school grounds, and community areas reflects the impact that cerebral palsy has on each individual. The level of function provides a clear indication of the level of severity of the motor impairment. Of course the terms mild, moderate and severe can be used but these terms are inexact without referring to level of function. Below are functional classifications and scales commonly used to describe level of mobility and ability to perform certain ambulatory tasks. Different classifications and scales have placed emphasis on different aspects of mobility.

# Hoffer Classification

The Hoffer Classification was developed to describe ambulation according to four functional levels for patients with myelomeningocele (Hoffer et al 1973). The Hoffer Classification rates the ability to walk according to distance travelled, and assistive devices or mobility aids used (Hoffer et al 1973). Ambulation is classified as community, household, non-functional and nonambulatory (Table 2.2). This classification provides broad categories that can be useful when assessing subjects who show diverse abilities e.g. non ambulant versus function at a community level. The broad categories are a disadvantage if the subjects are all ambulant because the rating is limited to the community and household categories. The category of community ambulator is not discriminative between subjects who are only able to walk short distances versus those who can manage long distances in the community. Ability to manage different surfaces or terrains is also not a factor that is considered. Subjects did show change between levels of ambulation over time in the original study in which it was utilised (Hoffer et al 1973). Therefore the classification was not stable.

Table 2.2	Classification of gait according to Hoffer et al (1	973).

Hoffer Classification				
Term	Description			
Community ambulators	Can walk outside with or without assistive devices or orthoses, with wheelchair use only for long distances out of the community setting			
Household ambulators	Uses assistive devices and does not walk outside of the house. Requires little to no assistance when moving in or out of wheelchair or bed. Wheelchair may be used for some indoor activities at home or school and all activities in the community.			
Non-functional ambulators	Walking only occurs as part of therapy session. Otherwise are dependent on a wheelchair for mobility.			
Non-ambulators	Dependent on a wheelchair. Can transfer from wheelchair to bed.			

# Gross Motor Function Classification System (GMFCS)

The GMFCS has been developed from the information contained in the sections of the Gross Motor Function Measure (GMFM), which is a reliable criterion-referenced measure that is able to measure change in a child's gross motor function (Russell et al 1989). The ongoing scale, GMFM-88 has 88 items that are scored over five dimensions that evaluate: 1) lying and rolling, 2) sitting, 3) crawling and kneeling, 4) standing, 5) walking, running and jumping. These items constitute physical activities that any normally developing five year old should be able to achieve.

The GMFCS classifies a child's level of function according to age specific criteria that range from Level I, reflecting minimal or no restriction in function, to Level V, reflecting minimal or no function at all (Palisano et al 1997). Assignment to a GMFCS level is based on the child's ability to sit with or without assistance, to walk with or without assistive devices and to perform the gross motor skills of running and jumping. Each level is divided into four age bands: one to two years, two to four years, four to six years, and six to twelve

years. These levels are qualified by specific functional descriptions of sitting and walking ability. Although developed initially for assessment of children between 18 months and six years of age, the GMFCS has been shown to be valid and reliable for children between one and 12 years (Palisano et al 1997, Palisano et al 2000, Wood and Rosenbaum 2000). The scale has been recently used with adults and validated through correlation with personal activities of daily living and motor function (Sandström et al 2004). However there is a need for further evaluation of validity and reliability for use in adults (Sandström et al 2004).

The GMFCS has been shown to be generally stable from ages one to two, to six to12 years indicating that a child once rated at a particular level on the GMFCS will most probably stay at that level up until age 12 years (Wood and Rosenbaum 2000). Therefore this scale is used for classifying level of function rather than change with time. However it has been reported that with development a child may be reclassified to another level (Rosenbaum et al 2002).

An advantage of the GMFCS is the inclusion of gross motor activities to differentiate between higher and lower function in children who walk without assistive devices. The ability to sit is used to differentiate between those who walk with assistive devices. Distance walked is not incorporated into the scale and so extent of community ambulation is only inferred from the rating level.

Increasingly, children with cerebral palsy are now being classified according to the Gross Motor Function Classification System. The use of the GMFCS to classify children with cerebral palsy aids the clinician or the researcher to clearly identify and communicate a child's level of function.

# Functional Assessment Questionnaire (FAQ)

The FAQ was developed as a functional outcome measure by the Gillette Children's Specialty Healthcare in Minnesota and was tested on three to 32 year old subjects with cerebral palsy (Novacheck et al 2000). The questionnaire rates the child's walking ability (on a scale of 1 to 10) and ability to complete

specific functional tasks. The use or non use of an assistive device is not considered, and rating is made according to the setting in which ambulation occurs, distance travelled, and ability to cope with obstacles e.g. kerbs, rough ground, stairs, crowds and need for supervision for safety.

Extent of community ambulation is well documented in the higher levels of the FAQ, (levels seven through to ten) (Novacheck et al 2000). However the absence of information regarding the use of assistive devices is a disadvantage of the scale. For example a child may maintain their FAQ rating after an intervention but may have become independent of assistive devices but this improvement in function cannot be ascertained from the FAQ.

The scale has been shown to be valid against standardised functional outcome measures, as well as energy and gait analysis derived data (Novacheck et al 2000). Intra- and inter-rater reliability have been established (Novacheck et al 2000).

#### Functional Mobility Scale (FMS)

The FMS was developed at the Royal Children's Hospital in Melbourne by staff at the Hugh Williamson Gait Laboratory, including this author (Graham et al 2004a). The FMS rates the ability of the child with cerebral palsy to walk over a distance of 5 metres, 50 metres and 500 metres (Graham et al 2004a). The scale ranges from independent with no limitations in function to wheelchair dependent over the particular distance.

The 5 metre distance relates to that usually traversed within the home or classroom; 50 metres is a distance that would be traversed in the backyard, going to a neighbour's home, or in the schoolyard; and 500 metres would represent going to a large shopping centre, sporting complex, or community venue. With the FMS there is scope to rate a child's highest usual level of ability e.g. over 5 metres, but allows the health professional to note ability in different scenarios. For example, walking in a shopping centre would possibly expose the lowest usual level of ability.

The reliability and validity of the FMS has been tested on children with cerebral palsy between the ages of six to 16 years, for validity and reliability. Concurrent and content validity were shown through strong correlations with standardised functional scales and objective data (Graham et al 2004a). Inter-rater reliability was high between the parents, surgeon and the research fellow who participated (Graham et al 2004a). The FMS rates a child's usual level of walking ability and is able to demonstrate change over time or with intervention (Graham et al 2004a).

An advantage of this scale is the ability to demonstrate different walking abilities (independent to wheelchair dependence) over different distances (household to community). A child may be able to walk independently at home (5 metres), use elbow crutches in the playground (50 metres) but use a wheelchair over community distances (500 metres). Change in ability in any of these three scenarios can be recorded. The FMS allows the parent or child to record the highest level of walking ability, which is usually over the 5 metre distance. This is important as it acknowledges the child's best function for the parent and child, whilst the recording of the 50 and 500 metre ratings allows detection of where limitations in walking ability most often exist.

The GMFCS, FAQ and FMS forms can be found in Appendices A, B and C respectively. All of these classifications and scales indicate the child's usual, everyday ability not their best effort ever.

Classification of cerebral palsy by functional mobility does not give information that helps the clinician target the specific physical or neurological limitation that is hindering the function of the child e.g. spasticity of the gastrocnemius or torsional deformity of the femur. Classification of functional mobility does indicate the level of severity of impairment and therefore aids in the classification of cerebral palsy.

23

# Classification by Health and Health Related Issues

The International Classification of Functioning, Disability and Health (ICF)

The International Classification of Functioning, Disability and Health (ICF) (World Health Organization 2001) recognises and emphasises the importance of function in every person's life. It has been developed to "provide a unified and standard language and framework for the description of health and health-related states" (World Health Organization 2001, p. 3) and so has not been developed as a classification exclusively relating to cerebral palsy but to all health conditions. It is not a classification system that categorises people into groups that are distinct from each other but it classifies the health and health-related aspects of the person or group of people. Since publication by the World Health Organisation (WHO) in 2001, the ICF has been adopted widely for use as a model to be utilised in clinical practice and research to provide a common conceptual framework for communication of information re status or outcome relating to a person's health and health related state.

The framework provided by the ICF can be useful for classifying: data that are being collected and recorded, measurement outcomes in research, clinical assessment and rehabilitation outcomes, social policies and educational programmes (World Health Organization 2001).

The ICF consists of two parts, functioning and disability, and contextual factors. Functioning and disability comprise two components, body function and structure, and activities and participation. Contextual factors are indicated by environmental and personal factors (Table 2.3). Table 2.3An overview of the ICF.Reproduced from ICF (World HealthOrganization 2001, p. 18).

	Part 1: Functioning & Disability		Part 2: Contextual Factors	
Components	Body functions and structures	Activities and participation	Environmental factors	Personal factors
Domains	Body functions Body structures	Life areas (tasks, actions)	External influences on functioning and disability	Internal influences on functioning and disability
Constructs	Changes in body functions (physiological) Changes in body structures (anatomical)	Capacity Executing tasks in a standard environment Performance Executing tasks in the current environment	Facilitating or hindering impact of features of the physical, social, and attitudinal world	Impact of attributes of the person
Positive aspects	Functional and structural integrity	Activities Participation	Facilitators	Not applicable
	Functioning			
Negative aspects	Impairment	Activity limitation Participation restriction	Barriers/ hindrances	Not applicable
	Disability			

*Body structure* (anatomical) and *function* (physiological) allow the identification of changes or deviations in the body, which are relevant to intervention by the clinician. *Activities* reflect the ability to do a task and *participation* relates to whether the ability leads to involvement routinely in real life situations. It can be "difficult to distinguish between "Activities" and "Participation" on the basis of domains in the Activities and Participation component" (World Health Organization 2001, p.16). The domain of mobility for Activities and Participation is an example of this difficulty in differentiation. The FAQ and FMS score a child's usual level of walking in the everyday situation, reflecting their ability to walk (*Activities*) but do they reflect *participation* in society? As they reflect the usual, everyday extent of walking the answer could be affirmative but they do not reflect ability to participate in activities with others, so the answer could be negative according to personal interpretation. The ICF also explores *environmental factors* as external influences that impact on the above-mentioned components. *Environmental factors* can help facilitate the ability to do an activity routinely in real life situations e.g. the availability of an assistive device. Conversely *environmental factors* may hinder certain activities e.g. uneven, rough terrain may limit participation out in the schoolyard or park, or prevent access to a building. *Personal factors* may impact on a person's ability to function in society such as sex, age, ethnicity, socio-economic background, educational level, upbringing and any other personal traits that may affect an individual's ability to function with a disability. See Figure 2.2.

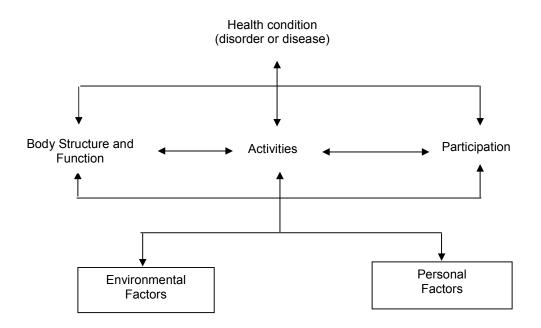


Figure 2.2 Interactions between the components of the ICF. Reproduced from ICF (World Health Organization 2001, p. 18).

#### **2.3 SPASTIC DIPLEGIA**

As stated earlier in Section 2.2.1, spastic diplegia may affect the whole body however the lower limbs are more involved than the upper limbs. The diplegic distribution can be symmetrical or asymmetrical (Miller et al 1995).

A recent study by Shevell et al (Shevell et al 2003) reported that spastic diplegia has been found to be associated with being born preterm (30.2%) and with periventricular leukomalacia (53.9%). Conversely this condition shows the highest incidence of unknown aetiology compared to quadriplegia and hemiplegia (Shevell et al 2003).

Gait in spastic diplegia can be varied and hence described by many different terms and combinations of terms such as equinus, calcaneus, flexed knee, recurvatum, flexed hip, posterior or anterior tilted pelvis, and lordotic. Gait in spastic diplegia has been classified as various different patterns in the past using different methods and all experiencing varying levels of acceptance for clinical usage. A commonly accepted classification of gait in spastic diplegia has not been developed. This is not the case in spastic hemiplegia. One of the most clinically accepted and utilised classification of gait patterns has been in hemiplegia (Winters et al 1987). In this classification, the sagittal plane kinematic data from subjects with hemiplegia were used for analysis and if classification could not be made on this data alone electromyographic (EMG) data were added. This process lead to the classification of four gait patterns in the sagittal plane (Groups I to IV) and across the groups from Group I to Group IV there was evidence of increasing involvement. One of the reasons for the clinical acceptance of this classification of gait patterns for children with hemiplegia was the clinical implication of this progression of involvement that particular muscle groups were responsible for the gait abnormality and hence management was implied. There were indications that a similar classification was being developed for spastic diplegia by the same group, but this classification has not been presented or published (Gage and Ounpuu 1989, Segal et al 1989).

# 2.3.1 Gait Classification in Spastic Diplegia

### **Clinical Observation**

Gait patterns in spastic diplegia have often been based on what the clinician has observed during clinical practice. The labelling of the gait patterns was not necessarily an attempt to classify all gait patterns in spastic diplegia but to aid the teaching of clinical reasoning behind intervention to improve the gait of the child. The identification of such gait patterns represents the beginning of classification of gait in spastic diplegia.

When studying hip flexion deformity, Bleck (1971) identified three gait patterns based on knee posture, found in conjunction with flexion and internal rotation of the hips (Figure 2.3). Ankle posture was not included in the descriptions. These patterns were based on associations between posture in gait, spasticity in a particular muscle and how intervention should be targeted.

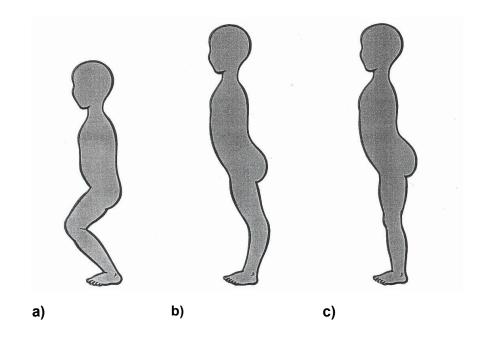


Figure 2.3 The gait patterns identified by Bleck (1987, p. 306). Left to right: a) flexed hips, flexed knees, b) flexed hip, hyperextended knees and c) flexed hips, balance of hamstrings and quadriceps activity but may be abnormal activity. Also internal rotation of hips in most cases.

Rang et al (1986) described gait patterns in spastic diplegia, that were based on visual observation of posture in gait in the sagittal plane and findings on clinical examination (Figure 2.4). The gait patterns were attributed to shortened muscles (either dynamically or statically contracted), and differed depending on the muscles involved. They observed associations between a short psoas and lumbar lordosis; short hamstrings and knee flexion in stance; short adductors and scissoring; short rectus femoris and stiff knee gait; and a short gastrocsoleus and tip toe gait. Linking these observed gait patterns to specific muscle shortening, implied a recommended management strategy (Rang et al 1986). These gait patterns differed from the patterns identified by Bleck (1971) as the posture of the ankle was included. The process of combining patterns at the hip and knee, knee and ankle or all three levels was not made.

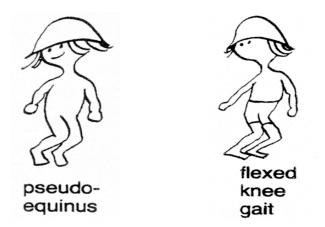


Figure 2.4 Two of the gait patterns identified by Rang et al (1986, p. 353).

Miller et al (1995) further elaborated on the sagittal gait patterns originally described by Rang et al (1986), describing five groups based on the alignment of the hip, knee and ankle in the sagittal plane. The five patterns were described as *jump, crouch, equinus, jump plus equinus, and recurvatum plus equinus* (Figure 2.5). A progression in the patterns from stiff-legged toe walking to a degree of crouch as the child grows, was stated but not explored and did not reflect the ordering of the patterns as they were documented and illustrated. These gait patterns were more extensive than those stated by Bleck (1971) and Rang et al (1986) as they combined together three levels of anatomical involvement.

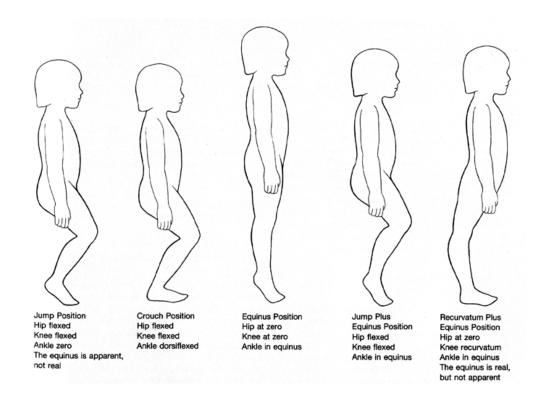


Figure 2.5 The gait patterns in spastic diplegia identified by Miller et al (1995, p. 487).

# **Clinical Data**

Eventually the identification and classification of gait patterns in spastic diplegia were undertaken using various forms of technology (video recording, 3DGA) as they became more readily available and user friendly. These classifications of gait patterns represent studies of a group of subjects who were all classified according to their gait pattern, not just the dominant patterns seen in the clinical setting.

# Cross-Sectional Studies

Digitised sagittal plane video recording was used to examine the gait patterns of 13 children with spastic diplegia who did not require ambulatory aids and had no surgical intervention (Huk et al 1987). Four main gait patterns emerged based on a simultaneous description of the posture of the hip, knee and ankle throughout the gait cycle:

- "Pattern 1- excess ankle equinus, excess knee flexion and excess hip flexion throughout stride.
- Pattern 2- excess stance ankle dorsiflexion, excess stride knee flexion, excess stride hip flexion.
- Pattern 3- excess stride ankle equinus, knee stance hyperextension, excess stride hip flexion.
- Pattern 4- excess stance ankle dorsiflexion, and limited late stance/ swing plantarflexion, knee stance hyperextension, excess stride hip flexion." (Huk et al 1987, p. 211).

It was proposed that these patterns would enable management strategies to be developed and provide an understanding of the pathomechanics of gait in spastic diplegia. Clinical utilisation of these gait patterns has not been documented, neither has any application to research. This may in part be attributed to the data being published as an abstract, and not followed by a paper, which would have limited reader exposure to the classification.

Sutherland and Davids (1993) described four typical gait abnormalities affecting the knee in 588 children with spastic diplegia. The definitions were based on sagittal plane kinematics and kinetics, with the common findings of clinical examination, electromyographic studies and force plate data described. Knee posture at various events, periods and phases of the gait cycle were highlighted. The four groups were *jump knee, crouch knee, stiff knee and recurvatum knee* (Figure 2.6). The gait patterns here were considered to be a direct consequence of contractures across joints and muscle spasticity and so were relevant to clinical management. It was recognised that not every child with spastic diplegic cerebral palsy would exhibit these patterns were illustrated with sagittal plane kinematic data from individual cases, there was no quantitative collective assessment of any of the data for all the subjects who demonstrated each knee pattern.

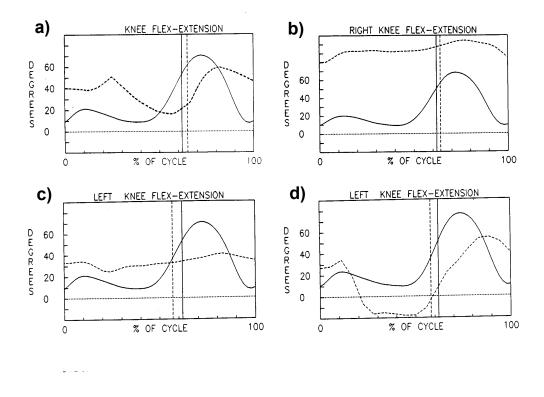


Figure 2.6 Common gait patterns at the knee in spastic diplegia (Sutherland and Davids 1993, pp. 142-146): a) jump, b) crouch. c)stiff and d) recurvatum knee. Vertical lines indicate toe-off. Solid lines indicate normal data and dotted lines, data from children with spastic diplegia.

Further work by Lin et al (2000) explored the common abnormal kinetic patterns of the knee in 19 spastic diplegic subjects. Again on kinematic evidence, four patterns describing the sagittal plane gait abnormalities of the knee were defined. However instead of Sutherland and David's stiff knee pattern (Sutherland and Davids 1993), Lin et al (2000) described a different pattern, mild knee. Kinematic data were linked to the corresponding sagittal plane moments. The internal moments of the knee at opposite foot off and opposite foot contact showed flexor moments for the recurvatum group and extensor moments for the remaining three groups (the crouch group having the largest). The internal moments of the ankle similarly showed differentiation between the groups. The largest sustained plantarflexor moment was in the crouch group with an acute increase seen in loading response for both the crouch and jump knee. However the jump knee showed a decreased plantarflexor moment in midstance but again there was an increase in terminal stance. The corresponding values for the power parameters showed that at opposite toe off, the jump group demonstrated the largest power generation at the knee and at opposite heel strike, the crouch group developed the greatest absorption power. The gait patterns were described as separate entities. This work provided information regarding the power absorbed or generated in each of the gait patterns, which was an addition to the knowledge previously supplied by Sutherland and Davids (1993). By classifying gait by kinematics and kinetics rather than just kinematics, it was proposed that this would lead to an increased understanding for clinical management. Whether this has happened is unknown as research utilising these gait patterns to study intervention strategies, has not been found.

A study of specific gait abnormalities in the lower limbs of children with cerebral palsy, has been recently published (Wren et al 2005a). The authors did not attempt to classify gait patterns but examined the posture of the hip, knee and ankle in the topographical subtypes of cerebral palsy. This was a large retrospective cohort study (n = 492), with almost half of the group having had prior surgery but the type of surgery was not documented. Gait abnormalities were defined prior to data collection and the presence of each gait abnormality noted from the examination of kinematic data for each subject. The most common postures found in spastic diplegia were that of a stiff knee, flexed knee (labelled by the authors as crouch), excess hip flexion, intoeing and equinus. When the data were examined in respect to increasing age of the subjects, the likelihood of increasing knee flexion was found in spastic diplegia and increasing calcaneus (regardless of prior surgery) and rotational malalignment in all subtypes of cerebral palsy. Subjects with prior surgery showed increased odds of having a flexed and stiff knee, calcaneus ankle and external foot progression. This study did not identify and classify gait patterns but it described combinations of gait abnormalities or postures in the lower limb that were found in spastic diplegia. These identified combinations were not seen as constituting general gait patterns in spastic diplegia but their clinical relevance to progression with time and surgical intervention was stated. This publication should alert clinicians to be proactive in working to prevent the development of progressive knee flexion and calcaneus, in older children with cerebral palsy in the long term.

### Longitudinal Studies

There have been only a few longitudinal studies documenting changes in gait patterns in spastic diplegia. Yokochi (2001) investigated the changes in gait patterns in children with spastic diplegia as they grew older. The children had been born premature and magnetic resonance imaging (MRI) results were consistent with periventricular leukomalacia. All twenty children were able to walk and their gait, without braces, was recorded by video in both the sagittal and coronal planes. Assessment was conducted on a yearly basis over a minimum period of five years for each subject. Range of motion of the trunk, hip, knee and ankle was assessed in the sagittal view using slow motion and freeze frame. The author recognised that internal rotation of the hips would confound assessment in the sagittal view, however internal rotation was reported to be mild in most subjects. Contracture of the hip, knee and ankle was also assessed from clinical examination. Initial and final walking patterns were documented according to trunk, pelvic, hip, knee and ankle postures. It was concluded that the dominance of hip flexors, knee flexors and ankle plantarflexors determines the gait pattern. Despite these findings, there was no attempt to use this information as a means of classifying gait in spastic diplegia nor were implications for management of these deficits explored.

Gough et al (2004) studied the effect of surgical intervention on the natural history of the gait pattern in spastic diplegia. Twenty children were assessed using 3DGA and recommended for surgical intervention, however only 10 children had surgery. A second gait analysis was completed more than a year later. There was deterioration in the kinematics in the control group and improvement or no change in the surgical group. The control group showed increased hip and knee flexion in their gait pattern at the second gait analysis. This study is significant as it documents that the natural history of gait in spastic diplegia is for deterioration over time into more hip and knee flexion, and that surgical intervention that stabilises the gait pattern constitutes an improvement.

Another study of children with spastic diplegia documented decreased excursion at the pelvis, knee and ankle over time as well as deleterious changes in temporal-spatial parameters such as decreased velocity and stride length

(Johnson et al 1997). This study involved eighteen subjects and half of the subjects had undergone prior surgery so natural history could not be assessed in those subjects. The authors compared the two groups and found that with surgery, subjects had a greater decreased period of single support and subjects without prior surgery experienced decreased excursion at the pelvis and knee.

Similar results were reported in a study by Bell et al (2002), though the 28 children studied were a heterogenous group comprising those with hemiplegia, diplegia and quadriplegia. No differentiation was made between the topographical diagnoses as they were studied as one group over time except for subgroups that were created according to walking velocity.

These four studies did not aim to classify gait patterns but described how gait patterns change over time and in doing so documented the gait patterns. Longitudinal studies are important as they can provide insight into how gait changes and the implied ramifications for the walking ability of the children who are affected by such changes in gait. This knowledge can be used to proactively implement management strategies that will prevent or delay the development of these deleterious changes in gait.

#### Statistical Analysis

Various statistical analysis techniques have been applied to data to define gait patterns in cerebral palsy. Wong et al (1983) applied cluster analysis to gait parameters to redefine the classification of cerebral palsy. However such a classification was limited as not all children with cerebral palsy have the ability to ambulate and this meant that an incomplete sample was tested. One hundred and twenty-eight children with quadriplegia, diplegia and hemiplegia, who could walk were included in this study. Gait velocity and angular motions for the right leg only were analysed. It was possible that a hemiplegic subject may or may not have had their affected side analysed. Four cluster groups were identified that related to different velocities and, in some cases, different ankle motion.

Kadaba et al (1991) used principal component and cluster analyses to identify ten gait patterns in spastic diplegia. The data were taken from 30 subjects, but only 59 limbs were analysed, which was odd as there should have been 60 limbs available for analysis if the subjects had spastic diplegia. This discrepancy was not explained as the analysis was only described in a conference abstract and a corresponding publication could not be found. Subjects had not undergone previous surgery and did not use assistive devices, which means that subjects with severe diplegia were not included in this study and so the gait patterns identified may not be inclusive of all subjects with spastic diplegia. Four ankle patterns were found which ranged from plantarflexion to dorsiflexion. Using the ankle patterns as part of a stepwise approach to pattern recognition, knee and hip postures of increasing severity were then identified. Identifying gait patterns was stated to be valuable in their application to clinical practice and treatment regimes, but evidence of this has not been documented since publication of the abstract.

Using fuzzy clustering techniques, O'Malley et al (1997) defined five cluster groups based on cadence and stride length that had been normalised by leg length and age, of 88 children with spastic diplegia. These groupings reflected the ambulation of children with and without cerebral palsy. O'Byrne et al (1998) used cluster analysis on the sagittal plane kinematics of the hip, knee and ankle of 237 "plegic" limbs. The aim was to classify quantitative data into clinically relevant descriptive patterns that would allow for treatment planning. The range of movement and the maximum and minimum values of the hip, knee and ankle were identified for the analysis. The cluster analysis found that the creation of eight groups allowed distinction between the groups clinically and a classification that was said not to be cumbersome. The eight clusters were labelled, mobile crouch, stiff crouch with toe walking, drop foot pattern, ankle double bump pattern, proximally flexed ankle walkers, mild recurvatum, severe recurvatum, and severe crouch (Figure 2.7). Kinematic parameters featured in the descriptions of the patterns, but again there was no exploration of how these patterns might be related to each other.

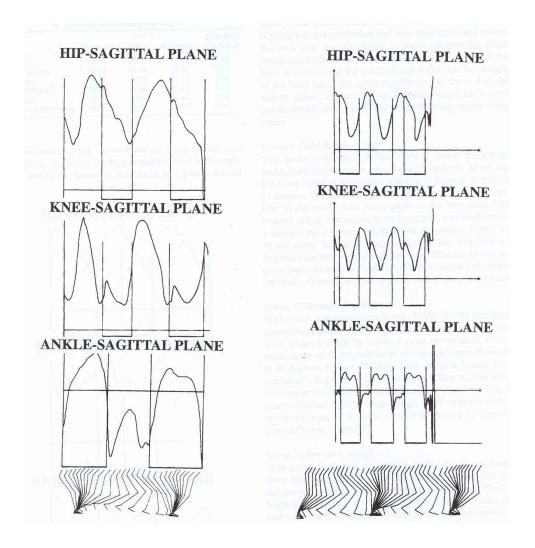


Figure 2.7 Example of cluster groupings published by O'Byrne et al (1998, pp. 102-105): sagittal kinematic graphs for a limb typical of Group 3 on the left and Group 4 on the right.

Classifications based on cluster analysis may be quantitative, objective and statistically sophisticated but as they lack any qualitative differentiation between the groupings, such classifications do not always correlate with what clinicians observe. In contrast, the patterns identified by Rang et al (1986), Miller et al (1995) and Sutherland and Davids (1993) appear to correlate with clinical observations, permit decision-making regarding management strategies, and are therefore more widely accepted. However as these qualitative classifications are not based on quantitative data, except for that by Sutherland and Davids (1993), their validity can be questioned.

37

# 2.4 CROUCH GAIT

Crouch gait is a term that has been used in the literature to describe a particular gait pattern that is often attributed to children with spastic diplegic cerebral palsy (Frost 1971, Rab 1992b, Steinwender et al 2001, Sutherland and Cooper 1978). Steinwender et al (2001) has stated though that "Crouch gait is reported very ambiguously in orthopaedic literature" (p. 80). This is of concern as research conducted into crouch gait in spastic diplegic cerebral palsy is being hampered by this ongoing problem regarding the precise definition of crouch gait.

# 2.4.1 Definition

One of the earliest papers devoted to crouch gait was by Frost (Frost 1971), who described spastic crouch *qualitatively* according to involvement at four anatomical levels- a posture observed when the subject is standing and walking with the trunk in a lordotic posture, hip and knee excessively flexed and the ankle dorsiflexed (Figure 2.8). A *quantitative* definition of crouch was provided by Sutherland and Cooper (1978): more than 30° knee flexion throughout stance with exaggerated ankle dorsiflexion. However the posture of the hips or trunk was not included in this definition, thereby limiting the definition to two anatomical levels. When classifying gait abnormalities of the knee, Sutherland and Davids (1993) later repeated the former definition for crouch and included increased hip flexion posture. However a numeric value was not stated to define increased dorsiflexion and hip flexion.



Figure 2.8 Coronal and sagittal views of a child with spastic diplegia who walks in crouch gait from Gage (2004f, p. 384).

A later definition (Rab 1992b) was much less specific, "the knee lacks normal dynamic extension during stance phase; the ankles may be plantarflexed, neutral, or dorsiflexed; and the hips generally are flexed, and often internally rotated and adducted" (p. 337).

The only common aspect to the definitions of crouch gait appears to be the presence of increased knee flexion in stance phase. However a flexed knee pattern is common to many of the gait patterns identified in Section 2.3.1 e.g. three of the five gait patterns identified by Miller et al (1995) have a flexed knee. In order to clearly differentiate between gait patterns, the posture of more than one lower limb joint needs to be recognised. Within this thesis, crouch gait has been defined as *increased knee flexion and increased dorsiflexion at the ankle in stance phase*. Flexed knees without identification of the alignment at the ankles has been labelled *flexed knee gait*.

#### 2.4.2 What is Known about Crouch Gait?

Knowledge of crouch gait has been evolving with specific research publications on subjects who were identified as walking in crouch gait. The following sections appraise the literature in this area in respect to whether the publications related to crouch gait, not just a flexed knee posture and detail the knowledge that may be attributed to crouch gait.

# **Overview of Crouch Gait**

Frost (1971) described crouch in spastic diplegia and hemiplegia, discussing the spastic dynamic muscular component and the eventual development of a fixed structural component in the muscles. A posture of excessive hip and knee flexion, ankle dorsiflexion and lordosis in standing and during walking, was identified. However there was no elaboration regarding the involved muscles and joints. Increased internal femoral torsion, equinus of the foot, heel varus and forefoot adduction, or rocker bottom feet, were all mentioned in conjunction with crouch gait. Frost noted that the internal femoral torsion was responsible for what was described visually as "scissoring" in gait. Prior to 3DGA, "scissoring" was usually attributed to tight adductor muscles, and not to increased dynamic internal rotation of the femur or static internal femoral torsion. This analysis of "scissoring" gait by Frost was most astute as he did not have access to 3DGA in 1971.

Orthotic intervention was not advocated by Frost (1971) due to the extensiveness of bracing required and the lack of permanent improvement once removed. Surgical intervention at the hip, knee and ankle was recommended, but Frost pointed out that biomechanical improvement was often at the cost of independent ambulation. Fifty percent of his patients required assistive devices postoperatively, whereas they were independent of assistive devices preoperatively. In addition, occasionally families complained that cosmetic improvement did not transfer into functional improvement. Faced with this mixed outcome, Frost decided to accept crouch gait and not to intervene using orthoses or surgery, except for heel cord lengthening, which he saw as improving balance by increasing the anterior-posterior dimension of the base of support (Frost 1971). Many authors (Berghof et al 1997, Borton et al 2001,

Dillin and Samilson 1983, Gage 1990, Rab 1992b, Sutherland and Cooper 1978) since have attributed isolated heel cord lengthening as a possible iatrogenic factor leading to the development of crouch. Frost recognised that the underlying abnormality is in the nervous system and that bracing and surgery do not address this but rather address the resulting alignment changes in the lower limbs. This paper did not detail research into crouch gait but did give a definition and discussion of likely causes and management options based on the clinical experience of the author.

Rab (1992b) cited possible causes of crouch gait as follows:

- static and/or dynamic contracture of the hamstrings and not quadriceps weakness.
- imbalance in muscle strength at the hip leading to static or dynamic hip contracture.
- possible hip extensor muscle weakness.
- iatrogenic over-lengthening of the calf.
- ankle-foot deformities and external tibial torsion that disadvantage the lever arm available to help extend the knee.
- impaired balance and other manifestations of the central nervous system disorder.

The indications for hamstring surgery were stated as knee flexion in stance of 20-30°; inadequate knee extension in swing; popliteal angle of more than 40° and straight leg raise less than 45°. Isolated calf surgery was discouraged and hamstring surgery timed with calf surgery was recommended. Evaluation of the hip was also promoted prior to any surgery. The need for further research in the area of the actual causes of crouch gait was identified.

More recently a book chapter by Dr Jim Gage (2004f) has been devoted to the management of crouch gait. The definition of crouch gait was not stated but the posture of flexed hips and knees and unrestrained forward progression of the tibia over the foot were discussed. The development of crouch gait was said to coincide with the adolescent growth spurt and the presence of lever arm dysfunction and soleus weakness. An increase in height and weight during the

adolescent growth spurt was responsible for the relative decrease in relative strength. Lever arm dysfunction referred to the change in the orientation of bones or muscles in respect to a fulcrum, such that their function was disrupted to varying degrees and hence the moments that they would normally exert about the joints were altered and gait was affected. Soleus weakness was attributed to tendoachilles lengthening or division of S1 and S2 nerve rootlets at the time of selective dorsal rhizotomy. The inability of the knee to extend in stance was attributed to both lever arm dysfunction and soleus weakness and the absence of a knee flexor moment in stance was a consequence of this. The resulting stress on the patella and development of patella alta and eventual avulsion fractures of the patella or tibial tubercle were noted. Patella alta refers to the abnormal positioning of the patella, superior to the femoral trochlea. The cycle of hamstring- rectus femoris co-contraction in order to maintain an upright position in crouch gait was emphasised.

Prevention of crouch gait was advocated by avoiding lengthening the tendoachilles tendon, no division of S2 nerve rootlets in a selective dorsal rhizotomy, correcting lever arm dysfunction prior to the adolescent growth spurt and instigation of wearing ground reaction foot orthoses (GRAFOs) if the soleus did show evidence of weakness. Treatment of crouch gait was directed towards restoring an erect posture by correcting lever arm dysfunction, lengthening tight muscles, correcting fixed flexion deformity at joints and shortening long muscles. The use of GRAFOs in a child with growth remaining was favoured to prevent continuous stretching of the weak soleus whilst bone growth would retension the soleus. In the child who had finished growing, re-tensioning of muscles was obtained by surgical intervention such as patellar tendon advancement. Resistant contracture at the knee was dealt with by distal femoral extension osteotomy.

# **Pathomechanics**

In 1978, Sutherland and Cooper (1978) used three dimensional gait analysis to investigate the pathomechanics of crouch gait in four subjects. Criteria for inclusion into the study were that the subject had more than 30° of knee flexion throughout stance phase with an associated increase in dorsiflexion. All

subjects were independent (no assistive devices or orthoses used), and complained of knee pain, an increasing degree of crouch and fatigue over an unspecified time period.

The general sagittal plane patterns of flexion at the hip and knee, and dorsiflexion of the ankle were described. In one case, a decrease in active and passive knee extension was noted. There was also electromyographic evidence of the prolongation of stance phase activity in hip and knee musculature. In two cases, increased external flexion torque was measured at both hip and knee joints in stance phase, and patella alta was demonstrated in one case.

It was concluded from the above kinematic, kinetic and electromyographic evidence that a large increase in quadriceps tension was harnessed, to provide stability to the flexed knee during stance. The long term effects of such increases in tension in the quadriceps are likely to be patella alta and associated knee pain. As the quadriceps muscle fibres become overstretched with increasing crouch, the patellar tendon becomes elongated and this causes the migration of the patella superiorly.

Of significance in this study was the history of bilateral heel cord lengthenings preceding the onset of crouch gait in all four subjects. The consequences of weakening the plantarflexor muscles are likely to be increased knee flexion and eventual hamstring contracture. When bilateral heel cord lengthenings are undertaken, both the gastrocnemius and soleus muscles are weakened and the ability of the soleus to restrain the forward movement of the tibia over the foot is diminished. This prevents the ground reaction force from moving forward on the foot and exerting an external knee extensor moment and so knee flexion throughout stance results.

The researchers advocated rigid bracing to prevent overstretching of the weakened plantarflexor muscles after heel cord lengthening. The recommended surgical intervention to improve crouch gait was lengthening of the hamstring muscles followed by rehabilitation, including quadriceps strengthening.

43

Sutherland and Davids (1993) further defined crouch gait in their paper describing common gait abnormalities of the knee. Children with crouch gait were documented to have the following striking features on clinical examination: tight hamstrings, quadriceps weakness particularly at the end of range, gastrocsoleus weakness plus or minus contracture, variable hip flexion tightness and variable pes valgus. Analysis of sagittal plane kinematic and kinetic data with electromyography were interpreted as showing that hamstring contracture was probably the primary cause of crouch gait again with isolated gastrocsoleus lengthening as an iatrogenic cause.

The kinetic patterns at the knee in crouch gait have been further documented (Lin et al 2000). The definition of crouch was based on kinematic data showing increased knee flexion in stance. However the degree of increased knee flexion was not stipulated. A graph of the mean sagittal plane knee kinematic for the crouch subjects showed that, the minimum knee flexion in stance was approximately 50°. The range of values and standard deviations were not reported. The mean knee flexion value of 50° indicates that the eight subjects that constituted their crouch group must have had high minimum knee flexion values. The mean sagittal plane ankle kinematic also showed excessive dorsiflexion (maximum approximately 19°) throughout stance. Therefore this group of subjects fits the criteria of the definition for crouch gait by Sutherland and Cooper (1978).

Previous studies (Sutherland and Cooper 1978, Sutherland and Davids 1993), which reported external knee flexion moments in crouch throughout stance, were supported by Lin et al (2000). Lin et al (2000) reported internal knee extensor moments, which are equal to external knee flexor moments. The ankle and hip moments were examined and large plantarflexor ankle moments and large hip extensor moments were reported. Sagittal plane joint powers at the knee demonstrated greater absorption in crouch gait compared to recurvatum gait (Lin et al 2000).

## Muscle Length Modelling

Musculoskeletal simulation of gait provides insight into the biomechanics that are found in abnormal gait. Such information may be helpful to the formulation of appropriate intervention. Usually musculoskeletal models are based on the musculoskeletal parameters of adults with normal anatomy and neuromuscular control and are referred to as generic models (Arnold and Delp 2004). Muscle tendon actuators have their line of action defined by the three dimensional geometry of their origins and insertions into bone (Delp et al 1990). When combined with kinematic data from a gait analysis, muscle length during the gait cycle can be calculated. However the limitations of such modelling cannot be overlooked. The application of the generic model will not always be appropriate as the ability to modify the model to reflect different conditions such as children with fixed deformities is currently limited and needs to be studied further. Spastic muscle displays unique behaviour compared to that of normal muscle and is not accounted for in the generic model. The influence of disordered neuromuscular control is not considered (Arnold and Delp 2004).

There have been numerous studies on the length of the hamstring muscle in children walking in crouch gait using musculoskeletal modelling (Delp et al 1996, Hoffinger et al 1993, Schutte et al 1997, Thompson et al 1998, Thompson et al 2001). However most of these studies have been marred by an omission of the definition of the crouch gait of their subjects (Hoffinger et al 1993, Thompson et al 1998, Thompson et al 2001) or incomplete criteria given for crouch gait (Delp et al 1996, Schutte et al 1997) with respect to the definition by Sutherland and Cooper (1978).

#### Hamstring Length

Hoffinger et al (1993) studied the role of hamstrings in hip extension and the effect of hamstring length on the position of the hip in crouch gait. Sixteen subjects with spastic diplegia were classified clinically as having crouch gait. No further definition of the parameters that constituted clinical crouch gait was given. Data for each subject were collected from 3DGA and electromyography. Results included static examination findings but the protocol for measurement was not documented.

Clinical examination showed no fixed contracture at either the hip or knee. On kinematic analysis in stance phase, maximum hip and knee extension ranged from values within normal limits to those outside normal limits. Subjects who exhibited maximum knee extension values in stance that were in the normal range could not be considered to be in crouch. Ankle kinematics were not documented. If the ankle was in equinus in stance phase, this would not satisfy the definition of crouch gait by Sutherland and Cooper (1978). From the kinematic results of the population studied, it appears that despite being classified as having clinical crouch gait, eleven out of sixteen subjects did not have crouch gait as defined by Sutherland and Cooper (1978).

The discussion focussed on the average values of data and so masked the variability in the data and the diversity of the subjects included. The conclusion was that dynamic hamstring muscle length in stance was longer than resting hamstring length. While these results represent the subjects studied, there were few subjects in crouch gait. As a result this study does not provide a great deal of clarification regarding the length of hamstrings in crouch gait.

In contrast to the study above, dynamic hamstring length has been documented as short in more than 60% of subjects walking in crouch in a recent published abstract (Do et al 2004). The hamstring muscle was considered short if it was greater than two standard deviations below normal. Hamstring surgery within the context of single event multilevel surgery was undertaken and the dynamic hamstring length increased in most subjects, as did their sagittal knee kinematic parameters. The extent of the hamstring surgery, i.e. medial only or medial and lateral hamstrings, was not stated. However the sagittal hip kinematics were relatively unchanged post-surgery. Unfortunately the definition of the crouch cohort was not stated and so the application to subjects walking in crouch according to the definition by Sutherland and Cooper (1978) can not be made with certainty.

## Hamstring and Psoas Length

Delp et al (1996) investigated hamstring and psoas muscle length in normal and crouch gait. Delp was concerned that Hoffinger et al (1993) had modelled the

knee joint as a simple hinge and this would lead to errors in the calculation of muscle length. Another concern was that some of the subjects studied had had previous orthopaedic surgery that would confound the results.

For inclusion in the study undertaken by Delp et al (1996), the subjects had to have spastic cerebral palsy, a minimum of 20° of knee flexion in stance phase in one or both limbs, no previous orthopaedic surgery and not use assistive devices or orthoses. Ankle position was variable with four subjects walking in equinus. This does not satisfy the definition of crouch by Sutherland and Cooper (1978).

Data from subjects with cerebral palsy were compared with those from 10 normally developing subjects. The dynamic muscle length was normalised by the anatomical length of that muscle so that subjects of different sizes could be compared. Muscles were considered short if their length was more than one standard deviation below the mean normative data. Of the twenty-eight limbs in flexed knee gait that were studied, only four showed a shorter than normal maximum muscle length of the hamstrings, whereas the maximum muscle length of the provide the crouch limbs was shorter. This was consistent with the results of Hoffinger et al (1993). The greater than normal muscle length of the hamstrings was explained in the context of the hamstrings being a biarticular muscle. Knee flexion is associated with hamstring shortening, but simultaneous hip flexion due to the large hip extension moment arm of the hamstrings at the hip, will place the hamstrings in a lengthened position.

This study by Delp et al (1996) considered the limitations of the musculoskeletal model but concluded that the errors were minimal. Measurement of static muscle length of the hamstrings based on the popliteal angles gave values that were considered adequate for normal gait. This, like many other studies (Orendurff et al 1998, Orendurff et al 2000, Schutte et al 1997, Thompson et al 1998), brings into question the relevance of static muscle length when it does not correlate with the dynamic changes observed in gait. The implication is that static muscle length should not be considered in isolation when orthopaedic surgical decisions are being made. Extrapolation of these results to subjects walking in crouch gait according to the definition by

Sutherland and Cooper (1978) is not possible due to the lack of information regarding ankle kinematics in the sample studied.

# Femoral Anteversion, Hamstring and Psoas Length

The effect of femoral anteversion on hamstring and psoas muscle length in crouch gait was studied by Schutte et al (1997). Because previous studies (Delp et al 1996, Hoffinger et al 1993) had used musculoskeletal models that were based on normal adult bony architecture to calculate the muscle length, this study incorporated into the musculoskeletal model the femoral anteversion that is commonly found in the spastic diplegic subject.

Criteria for inclusion were similar to the study by Delp et al (1996). Crouch gait was defined as that with knee flexion greater than 20° throughout the gait cycle however again no reference to ankle kinematics was made. Subjects had no previous orthopaedic surgery and could walk without assistive devices or orthoses. There were 21 limbs considered to be in crouch gait. Twenty-nine subjects provided the normative data.

The muscle lengths were calculated in three ways:

- A generic model that did not include femoral anteversion.
- A deformed model that incorporated femoral anteversion and the generic anatomical resting muscle lengths to normalise the data.
- A deformed model that incorporated femoral anteversion and the deformed anatomical resting muscle lengths to normalise the data.

Normalisation of the muscle lengths allowed comparison of subjects of different sizes. If the muscle length was greater than two standard deviations (SD) below the normative data, it was considered short. This differs from the study by Delp et al (1996) in which muscles were classified as short if the muscle length was one standard deviation from normal. A muscle was labelled "long" if the muscle was calculated as having a "maximum length either within 2 SDs of the normal average maximum length or longer" (Schutte et al 1997, p. 617).

More than 50% of the hamstring and psoas muscles were shorter than those in the normative sample by more than two standard deviations. If the classification by Delp et al (1996) were used, there may have been more cases of short muscle length in the hamstrings and the psoas in this study. Despite similar subject populations in both studies, Schutte et al (1997) showed that hamstrings are not consistently long, in contrast to the findings of Delp et al (1996). These results were interpreted as being due to variations in the gait patterns in the two study groups. The deformed model for muscle length calculation only changed the classification of two hamstrings from short to long, but changed the classification of five psoas muscles (normalised by generic anatomical resting muscle length) and 10 psoas muscles (normalised by deformed anatomical resting muscle length) from short to long.

The incorporation of femoral anteversion into the model had minimal effect on the hamstring muscle length but influenced the psoas muscle length, particularly when psoas was normalised using the deformed resting muscle length. Femoral anteversion in the deformed model assumed that the rotation occurred superior to the lesser trochanter. Because the psoas inserts into the lesser trochanter, this rotation will influence the length of psoas and have minimal effect on the hamstrings. Therefore it was concluded that the length of the hamstrings can be calculated using the generic model.

This study showed that, once again, there was poor correlation between static and dynamic muscle length as measured by goniometric measurement in a clinical examination and modelling of dynamic muscle length in gait. The omission of ankle kinematics in the criteria does not permit application of the findings of this study to subjects who walk in crouch gait according to the definition by Sutherland and Cooper (1978).

#### Effect of Different Musculoskeletal Models on Muscle Lengths

Arnold et al (2001a) studied the accuracy of different musculoskeletal models in calculating muscle-tendon lengths of the hamstrings and psoas muscles. The generic model that was originally used in previous studies (Delp et al 1996, Hoffinger et al 1993) is based on the normal musculoskeletal geometry of an

adult (height 1.8 metres, weight 75 kilograms). In the study by Schutte et al (1997), the model was altered to take into account the angle of femoral anteversion. In this study, magnetic resonance images (MRI) were used to obtain three dimensional data from the femurs of four subjects who had crouch gait, and a MRI based model for calculating muscle-tendon lengths was developed. The muscle-tendon lengths of the four patients with crouch gait were calculated from the MRI based model, and estimated using the undeformed generic model, the undeformed generic model scaled to the subject along anatomical axes, the generic model deformed by the subject's femoral neck anteversion, and the generic model deformed to the subject's anteversion, neck-shaft angle, and lesser trochanter torsion.

Psoas length was under estimated in the undeformed generic model and it was recommended that for estimation of psoas length, the deformed generic model using femoral neck anteversion be used. Hamstring length was found to be acceptable either by estimation by the generic or MRI based models. Scaling for the bony dimensions did not improve accuracy. The subject with the most severe crouch gait showed differences in semitendinosus length between the MRI based and generic models and this was attributed to the abnormally posterior route of the hamstrings in relation to the knee found on MRI. Although maximum and minimum knee flexion during stance phase were documented, ankle dorsiflexion was not, and so whether or not the subjects fitted the definition of crouch gait by Sutherland and Cooper (1978) is unknown. The results cannot therefore be confidently extrapolated to all who walk in crouch gait.

#### Hamstring Muscle – Tendon Length and Lengthening Velocity

One study has investigated a large cohort of subjects (n=152 from two different paediatric centres) walking in crouch gait (Arnold et al 2005b). Crouch was defined as greater than  $20^{\circ}$  of knee flexion at either initial contact or terminal swing, without regard to the minimum knee flexion obtained in stance phase. No reference to the ankle or hip alignment was made. The data were analysed retrospectively as the data collection period was over 14 years for one centre and eight years for the other centre.

It was found that only a third of the subjects had short hamstrings on muscle length modelling at baseline. A similar percentage of subjects had slow hamstring lengthening velocities at that time and a third of subjects had neither short nor slow hamstrings. For a majority of the subjects, hamstring surgery was undertaken along with other surgical procedures that were not documented. It was found that those subjects with short and slow hamstrings pre-surgery, who had improved knee extension post-intervention, also had hamstrings that demonstrated longer lengths and faster lengthening velocities. In subjects who did not obtain adequate knee extension, the muscle-tendon length and lengthening velocities of the hamstrings also did not improve.

Of the subjects who had neither short nor slow hamstrings at baseline, improvement in knee extension at terminal swing and initial contact was gained regardless of whether the surgical procedures included hamstring lengthening or not. This implied that the increase in knee extension post-surgery was attributable to other procedures and hence the lack of knee extension at baseline was not due to hamstring pathology but pathology elsewhere. If hamstring lengthening was undertaken on subjects with neither short or slow hamstrings, the pelvic position either did not alter or tilted further anteriorly postintervention. Therefore caution was advised in lengthening hamstrings, which were not short in muscle length nor slow in lengthening velocity. However the pelvic position of only this subgroup of subjects was documented and no information on pelvic position for the other subgroups was provided.

The issue of whether or not to lengthen the hamstrings in crouch gait, is an important one as this study has shown that hamstring lengthening to improve knee extension may result in a deleterious effect at another anatomical level by increasing anterior pelvic tilt. As the definition of crouch gait in this study was based on the knee posture and the posture of the ankle and hip was unspecified, the extent of crouch gait in the cohort according to the definition by Sutherland and Cooper (1978) is unknown. Consequently the results may or may not have relevance to children who walk in crouch gait.

# Contribution of Muscles to Gait

Computer modelling of normal gait has been used to investigate the causes of crouch gait (Arnold et al 2005a). The contribution of the gluteus maximus to promoting hip and knee extension was substantial in early single support. However the vasti contributed slightly more to the acceleration of the knee into extension during this time period. In mid to late single support, hip extension was reliant more on the posterior section of the gluteus medius and knee extension, on the soleus. It was proposed that strengthening of these muscles, particularly the gluteus maximus may be beneficial in cases of crouch gait to promote improved hip and knee extension.

# **Botulinum Toxin Injections**

The effect of botulinum toxin on the dynamic length of the hamstrings in crouch gait has been studied (Thompson et al 1998). However the definition of crouch gait, was not detailed in the methods. In the results, the mean minimum knee flexion in stance was 26° pre-intervention, however the range of values and standard deviations were not presented. Ankle posture was not reported. Consequently, without knowing the variation in the minimum knee flexion values or the ankle posture, the extent of the crouch gait of the subjects is unknown and the impact and relevance of the findings from the study in relation to crouch gait cannot be ascertained.

Prior to injection of botulinum toxin, only four limbs out of 18 limbs showed short medial hamstrings and no shortness in biceps femoris. Post- botulinum toxin injection, muscles lengths were compared with their pre- injection classification. Range of excursion in the short semimembranosus was significantly increased whereas the increase was not significant in the adequate muscle length group. There was no significant difference in the absolute change in muscle length between the two groups. The post-injection data demonstrated the changes at two weeks post-injection. The graphics based musculoskeletal model, SIMMGait was used which bases parameters on those of an average sized adult male. The limitations of doing so have been mentioned previously (Section 2.4.2).

These results are of value in understanding the effect of botulinum toxin on hamstring muscles that are short or of adequate muscle length but not necessarily in crouch gait. Injections should be directed to short hamstring muscles that show the best response. It should be noted that this best response is at two weeks post-injection and further follow-up post-injection is necessary to assess the long term results.

#### **Physical Examination Measures**

Attention has been directed to identifying the factors that cause crouch gait. Two abstracts (Orendurff et al 1998, Orendurff et al 2000) from Gait and Clinical Movement Analysis Society meetings have reported that measures of passive range of motion, strength and bony deformity are not predictive of knee position in stance. Other studies have also found that there is poor correlation between static and dynamic measurement of motion (Delp et al 1996, Schutte et al 1997, Thompson et al 2001). Hence the muscles or bony abnormalities that should be targeted in the correction of crouch gait remain poorly defined. In neither abstract was a definition of crouch gait given. Without this information the results cannot necessarily be applied to children with spastic diplegia walking in crouch gait. In the earlier work (Orendurff et al 1998), children with spastic hemiplegia and diplegia were analysed together.

Thompson (2001) examined dynamic hamstring length and excursion in crouch gait with respect to static measurement via popliteal angle and modified popliteal angle. Again, the definition for crouch gait was not detailed and children with diplegia and quadriplegia were included as subjects.

There were nine observers, who measured the popliteal and modified popliteal angles of the subjects. Results for the observer who had demonstrated the best intra-observer reliability for the measurement of the modified popliteal angle, demonstrated that an increasing modified popliteal angle showed a significant linear relationship with decreasing maximum hamstring musculotendon unit length and hamstring excursion. However the remaining eight observers did not confirm this result.

If the individual maximum musculotendon unit length or excursion was less than the normal average length or excursion, minus one standard deviation, the muscle was classified as short or having reduced excursion. On dynamic muscle length calculation, medial hamstrings were shown to be short in 10 out of 32 limbs and hamstring excursion was decreased for 30 limbs. These results showing medial hamstrings are short in only a small number of subjects in crouch gait, were similar to those found by Delp (Delp et al 1996) but again the result can only be generalised to the subject group in the study and not necessarily to subjects in crouch gait, as crouch gait was not defined.

The results of this study also concur with those of previous studies in that change in static muscle length does not imply that there is change in dynamic muscle length (Delp et al 1996, Orendurff et al 1998, Orendurff et al 2000, Schutte et al 1997). The reason for this finding was attributed to the poor reliability, found in the testing of static muscle length. This was based on the data from one of the nine observers, which showed that if the static muscle testing was repeatable, there was a relationship between static and dynamic muscle length (Thompson et al 2001).

## Muscle Strength

In the last decade attention has been directed to research into muscle strength in cerebral palsy (Damiano and Abel 1998, Damiano et al 1999, Damiano et al 1995b, Dodd et al 2003, Kramer and MacPhail 1994, Ross and Engsberg 2002, van der Linden et al 2003b) and one such study detailed the results of quadriceps femoris strengthening on crouch gait (Damiano et al 1995a). As already mentioned Sutherland and Cooper (1978), suggested quadriceps strengthening as a possible intervention for crouch gait.

The study by Damiano et al (1995a) assessed the changes in muscle strength of the quadriceps at 90°, 60° and 30° of knee flexion using a hand held dynamometer, as well as temporal-spatial measures of gait and knee kinematic variables, after a six week quadriceps strength training programme. Inclusion criteria for the study were children with spastic diplegia, full passive knee extension in supine and crouch gait defined as more than 10° of knee flexion at initial contact. However when the results were reported, crouch gait was redefined as the previously stated knee kinematic with continuous knee flexion in stance phase. Fourteen children participated, however it is questionable whether they had crouch gait according to the definition by Sutherland and Cooper (1978) as ankle position in stance was not documented. It was mentioned that one subject had a kinematic of knee hyperextension in midstance prior to strength training. This calls into question the definition of crouch gait for the cohort in this study. The reported pre-training mean minimum knee flexion in midstance for the cohort was 13° with a standard deviation of 14° indicating that there was considerable variability in the knee extension in stance.

There were significant improvements in strength of the quadriceps, and a significant reduction in knee flexion at initial contact, indicating that strength training in spastic diplegia could be beneficial, but due to the study inclusion criteria, these results do not necessarily reflect the effects of quadriceps strengthening on crouch gait.

# Hamstring Surgery

Evaluation of surgery to correct crouch by proximal hamstring release was undertaken by Drummond et al (1974). Subjects had spastic diplegia or quadriplegia, no fixed flexion deformity of the knee but there was limitation of straight leg raise, and inability to sit with knees extended. Ability to walk was not a criterion. Crouch according to the definition by Sutherland and Cooper (1978) could not be established as this study preceded the publication of that definition.

The surgical procedure consisted of the hamstring origin being released from the ischial tuberosity. Of the 25 subjects evaluated at follow-up, only five showed functional deterioration or same status as pre-operation. Increased lumbar lordosis was reported in eight of the 25 subjects and genu recurvatum in seven subjects. Proximal hamstring release was consequently not recommended and caution recommended whenever hip flexion deformity was in excess of 25° and observed in combination with abdominal muscle weakness.

## **Pelvic Kinematics**

Tylkowski et al (1988) in a conference abstract reported a study of 49 subjects with crouch gait, who were classified into three groups according to their sagittal plane pelvic kinematics. If the pelvis was tilted anteriorly, electromyography of the gluteus maximus was out of phase and occasionally hamstring activity was prolonged, then hip flexor contracture was assigned as the cause of the pattern. If there was a posterior pelvic tilt, and prolonged hamstring activity on electromyography, then hamstring contracture was attributed as the cause of the pattern. When the pelvic tilt was within normal range, hamstring activity was abnormal and gluteus maximus activity occasionally out of phase, both hip flexor and hamstring contracture were said to contribute to the gait pattern.

The study has been reported only in abstract form and a corresponding research paper cannot be located. The criteria for definition of crouch were not documented and so the findings cannot be extrapolated with certainty to subjects with crouch gait. However the study was the first to consider pelvic kinematics combined with electromyography in order to explain the mechanisms underlying crouch gait.

# Hip Parameters

The gait parameters of the hip in subjects with crouch gait, were documented by Steinwender et al (2001). The mechanisms of hip hike, hip extensor muscle involvement in propulsion, and the presence of adduction were described. Children with spastic diplegic cerebral palsy were investigated. A non crouch group (n=29) and a crouch group (n=15) were compared with a normally developing group (n=16). All children could walk for 10 minutes without the use of assistive devices and had no past history of surgery to the lower limbs. The inclusion criteria for the crouch group were according to the definition by Sutherland and Cooper (1978). The averaged sagittal plane knee and ankle kinematic graphs for the crouch group showed a minimum knee flexion of more than  $30^{\circ}$  in stance and excessive ankle dorsiflexion.

The crouch group showed inability to achieve abduction in pre- and early swing phase. The children with cerebral palsy showed a higher power generation at the hip in first double support than the group of normally developing children. This early power generation at the hip was considered to be a compensation for the diminished power generation observed at toe off of the contralateral limb. In the first period of double support, all groups showed a correlation between mean hip power generation and mean hip angular velocity alone. However in the first half of stance, both the normally developing and non crouch group showed correlations between mean hip power generation and both mean hip angular velocity and mean hip moments, whereas in the crouch group only mean hip power generation correlated with the mean hip moments. Kinematic data in the crouch group showed increased hip flexion in the sagittal plane and consequently an increased hip moment and decreased joint angular velocity, so the power generation would be dependent on the large hip moment.

There were no differences between the three groups in the amount of adduction at the hip in the first half of stance. This implied that despite the visual appearance of increased hip adduction in stance in children with cerebral palsy this is an apparent, and not a real increase, in adduction. The mean hip internal rotation during the first half of stance correlated with mean hip flexionadduction and knee flexion for the non crouch group, and with mean hip flexion only in the crouch group which was attributed to spastic medial hamstring involvement.

# Foot Posture

Crouch gait has also been subdivided into two types according to foot alignment (Berghof et al 1997). This is documented in an abstract for a Gait and Clinical Movement Analysis Society Meeting. Crouch was defined according to Sutherland and Cooper (1978) for the knee but not clearly for the ankle. There were four subjects who had had previous bilateral heel cord lengthenings. Calcaneal gait with increased dorsiflexion, and pes valgus with external foot progression were the two postures of the feet identified. Ankle plantarflexion moments were increased in stance for both foot postures but ankle and hip power generation at toe off was decreased more in crouch with pes valgus than calcaneus.

# 2.5 ISSUES RELATING TO CLASSIFICATION IN CEREBRAL PALSY RESEARCH

Research in cerebral palsy has been conducted for decades (Accardo 1982, Bax 1964, Bennet et al 1982, Damron et al 1994, Gugenheim et al 1979, Kay et al 2002, Minear 1956). Often research has been conducted retrospectively using a sample of convenience consisting of subjects who attended an outpatient clinic or gait analysis appointment in a certain time period (Chambers et al 1998, Chung et al 1997, Miller et al 1997, Õunpuu et al 1993a, Õunpuu et al 1993b, Saw et al 2003, Yngve et al 2002). Unless the research is conducted in a large centre, hospital or institution, which specialises in cerebral palsy and services a large geographical area, the number of suitable subjects can be limited (Patrick et al 2001, Stanley et al 2000). This situation has meant that criteria for inclusion into a research project can be generous at times, particularly if the outcome of certain interventions is being studied.

# 2.5.1 Lack of Homogeneity of the Type of Cerebral Palsy

In most research studies, the type of motor disorder is usually well defined and two different motor types (e.g. spasticity and hypotonicity) would not be assessed as the same condition. However in spastic cerebral palsy, so often the criterion for inclusion into an outcome study is merely the diagnosis of spastic cerebral palsy; and so subjects with hemiplegia, diplegia and even quadriplegia may all be included (Bell et al 2002, Chung et al 1997, Gage et al 1987, Norlin and Odenrick 1986, Rethlefsen et al 1999b, Sutherland et al 1990). If the independent variable is the different type by distribution of involvement in spastic cerebral palsy, this is appropriate (Borton et al 2001, Rattey et al 1993, Wren et al 2005a). However if spastic cerebral palsy is the sample for the study and the independent variable is treatment regime, then the sample should be homogeneous. A common combination for studies into treatment outcomes for mobility for spastic cerebral palsy involves subjects with hemiplegia and diplegia as the sample for study (Kay et al 2002, O'Byrne et al 1998, Orendurff et al 2002). Using spastic cerebral palsy as the inclusion criterion ignores the disparity in level of function between the groups and the impact this will have on the dependent variable if it is a gait parameter. Subjects with hemiplegia have a non-involved lower limb that is capable of compensatory movement to counter the disturbance in gait caused by the neurologically involved lower limb. Subjects with diplegia have both lower limbs affected and so their ability to compensate is consequently limited. Hence subjects with hemiplegia have different gait patterns (Winters et al 1987) from those subjects with diplegia (Gage 1993) and differing impairments in function as seen by the GMFCS level for hemiplegia being predominantly from level I-II whereas for spastic diplegia, it is level I-III (Howard et al 2005). If the study is conducted with the type by distribution of involvement as an independent variable and not exclusively as the sample, then inferences can be made regarding the outcome in each of the types, but not across types.

# 2.5.2 Lack of Identification of Gait Classification

Research into gait in spastic diplegia has been hindered by the lack of a recognised, universally accepted and used classification of gait patterns. So often a sample for study will have as inclusion criteria: spastic diplegia, walking with and without assistive devices (Fabry et al 1999, Nene et al 1993, Saraph et al 2002, Zwick et al 2001). The independent variable may be a particular intervention and the dependent variable, a gait parameter. A gait pattern emerges from a combination of the abnormal neurological deficit and biomechanical forces on the musculoskeletal system. An intervention for one specific gait pattern may have a completely different result when used to manage another gait pattern. For example, the use of hinged ankle foot orthoses (AFOs) when some subjects have a gait pattern that includes equinus at the ankle in stance and other subjects within the sample, have calcaneus. If the independent variable was defined according to the attributes of the ankle being in equinus or calcaneus, then the effect on each of these gait deviations could be ascertained (Abel et al 1998). Not taking this into account will lead to less meaningful results as the two gait patterns at the ankle could possibly negate each other's results – using hinged AFOs in equinus may result in gait

improvement but in calcaneus, gait deterioration (Gage and Quanbeck 2004e). This would not offer any evidence as to when the prescription of hinged AFOs is indicated. Often studies into selection of orthoses have been marred by this problem (Buckon et al 2004, Rethlefsen et al 1999a, Smiley et al 2002). Using identification of the type of cerebral palsy as the inclusion criterion does not recognise the impact of the actual movement pattern on outcome measures (Scrutton 1998).

A recent study that had the inclusion criteria of spastic hemiplegic cerebral palsy used the definition of gait patterns in hemiplegia by Winters et al (1987) in order to subtype the gait pattern (Aminian et al 2003). By classifying the gait patterns within the hemiplegic sample, the study was able to show that average internal hip rotation and pelvic retraction during gait increased with increasing severity of hemiplegia as denoted by the gait classification of Winters et al (1987). This is an example of how the use of a gait classification within a topographical group can be utilised in research to provide enhanced knowledge that would not be revealed if the topographical group were studied as a group assumed to be exhibiting a common gait pattern.

## 2.5.3 Lack of Consensus in Gait Classification

As already stated above, various authors have identified gait patterns in spastic diplegia but only a few have been adopted for use in research or clinical communication (Sutherland and Cooper 1978, Sutherland and Davids 1993). The most recognised would be the classification by Sutherland and Davids (1993) based on knee kinematics from gait analysis data - jump, crouch, recurvatum and stiff knee. However research utilising these gait patterns as independent variables, is limited (Goldberg et al 2003).

#### 2.5.4 Lack of Consensus Regarding Classification of Crouch Gait

Crouch gait has been generally recognised as increased knee flexion in stance, but the only biomechanical definition has been provided by Sutherland and Cooper (1978). The 1992 consensus statement from the American Academy of Orthopaedic Surgeons (Rab 1992b) on crouch gait was non specific with regard

to biomechanical parameters at the ankle in particular, so that subjects enrolled in studies of crouch gait may not be homogeneous, making comparison difficult.

Often in the literature, crouch gait is simply stated as the inclusion criterion for a study and no parameters for the degree of maximum knee extension or minimum knee flexion are defined (Drummond et al 1974, Johnston et al 2004a, Orendurff et al 1998, Thompson et al 2001, Tylkowski et al 1988). In some instances, however mean knee extension for the sample and the corresponding standard deviation and/or range of values have been available from the results (Hoffinger et al 1993, Thompson et al 1998). Alternatively crouch gait has been defined as broadly as increased knee flexion in stance (Drummond et al 1974, Gage 1990, Hoffinger et al 1993, McNee et al 2002), or increased knee and hip flexion in stance (Orendurff et al 2000, Rang et al 1986), or increased knee and hip flexion with increased dorsiflexion in stance (Frost 1971, Lin et al 2000, Miller et al 1995, Rab 1992b).

Other studies have defined crouch gait according to the maximum knee extension or minimum knee flexion during the stance phase of the gait cycle, but the position of the other joints, especially the ankle has been omitted (Arnold et al 2001a, Berghof et al 1997, Delp et al 1996, Schutte et al 1997) or can only be ascertained from comparison of the pre- and post-intervention ankle kinematic data (Steinwender et al 2001).

It is not often that the specific value of knee flexion by Sutherland and Cooper (1978) has been utilised to define the inclusion criterion into a study on crouch gait (Berghof et al 1997, Steinwender et al 2001). Different quantitative values of knee flexion have also been documented. Sagittal plane minimum knee flexion of 20° in stance, with increased hip flexion has been used (Delp et al 1996, Schutte et al 1997). Knee flexion of greater than, or equal to 10° at initial contact, is a definition of crouch gait that has not previously been used (Damiano et al 1995a).

Table 2.4 shows a summary of different authors' definitions of crouch gait as reported in the literature.

Table 2.4 Summary of criteria for crouch gait reported in the literature.

							ĺ
Authors	Year	Knee	Ankle	Hip	Pelvis	Kinetics- Moments EMG	
Frost (1971)	1971	flexed in standing and walking	excess dorsiflex	excessive flex lordosis	lordosis		
Drummond et al (1974)	1974	flexed					
Sutherland and Cooper (1978)	1978	flexed >30° throughout ST	exaggerated dorsiflex			incr hip and kn <u>external</u> flexor prolonged quads, moment	nged quads, glut max
Rang et al (1986)	1986	flexed		flexed			
Tylkowski et al (1988)	1988	no definition			anterior, neutral, posterior		
Gage (1990)	1990	flexed ST					
Rab (1992a)	1992	persistent flexion, lacks norm ext ST	plantarflex/ neutral/ dorsiflex	flex, add, IR			
Sutherland and Davids (1993)	1993	sagittal plane incr flex>30° or decr ext late swing	incr dorsifiex ST	incr flex stance		kn flex to ext moment absent prolonged quads, HMS	nged quads,
Hoffinger et al (1993)	1993	persistent and dynamic flexion stance	no mention				

Authors	Year	Knee	Ankle	Hip	Pelvis	Kinetics-Moments EMG	
Miller et al (1995)	1995	flexed	dorsiflex	flexed			
Damiano et al (1995a)	1995	excessive flexion ST, =>10° flex initial contact, mentions maintains flex ST					
Delp et al (1996)	1996	flexed >20° throughout ST in one or both limbs	variable	incr hip flex, add, IR			
Schutte et al (1997) 1997	1997	flexed >20° throughout ST		incr hip flex, add, IR			
<b>Berghof et al (1997)</b> 1997	1997	flexed>=30° ST					
Thompson et al (1998)	1998	no definition - only in results average maximum ext =26°, no range					
Lin et al (2000)	2000	incr flex throughout ST	incr dorsiflex ST	incr flex gait cycle		incr hip ext moment till MST/ prolonged rectus constant kn ext moment/ femoris rapid incr & constant ank plantarflex moment ST	jed rectus
Orendurff et al (2000)	2000	flexed		flexed			
Steinwender et al (2001)	2001	flexed >30° throughout ST	incr dorsifiex ST	incr flex ST(from graph)			

63

Authors	Year Knee	Knee	Ankle	Hip	Pelvis	Kinetics- Moments	EMG
Thompson et al (2001)	2001	no definition and no results to ascertain further					
<b>Arnold et al (2001a)</b> 2001	2001	no definition - only in demographics, admits one is jump knee					
<b>McNee et al (2002)</b> 2002		flex ST					
<b>Arnold et al (2005b)</b> 2005	2005	flexed >20° initial contact (0-4% gait cycle) or terminal swing (96-100% gait cycle)					

Another common term used to describe gait in children with spastic diplegia, is that of a flexed knee gait. Often the terms 'flexed knee' and 'crouch' appear to be used interchangeably. This is an important issue for research into crouch gait in spastic diplegia as such definitions imply that intervention for crouch is undertaken based on the knee position only. In particular, if the ankle position is not taken into consideration then the biomechanics of the gait pattern as a whole are ignored and this can impact on the decision regarding which soft tissue surgery and orthotic intervention is appropriate. A flexed knee with the ankle in equinus is different in biomechanical alignment to a flexed knee with the ankle in calcaneus and the interventions would be different as a consequence of this. Figure 2.9 demonstrates this concern. So by defining inclusion criteria as spastic diplegia, mobility with or without assistive devices, and crouch, will not guarantee homogeneity because of the lack of specification of degree of crouch and the posture of the hip and ankle. Consequently the results from such a study do not necessarily improve our understanding of crouch.

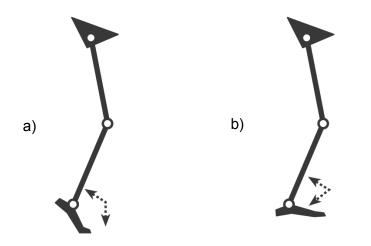


Figure 2.9 Flexed knee with a) equinus and b) calcaneus. A hinged AFO may restrict plantarflexion and promote dorsiflexion in a) which would be beneficial but in b) the increased dorsiflexion will not be alleviated (Gage and Quanbeck 2004e).

As already stated, published research on crouch in spastic diplegia, may be criticised on the basis of the above concerns. The acceptance and utilisation of a gait classification in spastic diplegia by the professionals involved in gait and cerebral palsy research and clinical management, would result in better designed studies that would yield more meaningful results.

## 2.6 SUMMARY

## 2.6.1 Classification Consensus

Classification of cerebral palsy according to the type by distribution of involvement and by neurological disturbance has been used for many years. As technology has developed and improved our understanding and knowledge of cerebral palsy, the research and clinical management has not become more specific in relation to further subtypes of cerebral palsy.

## **Research Implications**

In cerebral palsy, research into management of gait problems is somewhat hindered by poor definition of inclusion criteria according to the type by distribution of involvement, type of gait pattern and specific parameters for gait patterns. This has led to heterogeneity in groups studied, and needs to be addressed so that meaningful research can be conducted and results can be applied to specific groups.

# **Clinical Implications**

Without research, which is specific to classifications of gait in spastic diplegia, clinical management of gait abnormalities will also not be specific enough to tailor interventions (spasticity management, surgery or orthotic prescription) appropriately and effectively to correct the gait pattern of the child with spastic diplegia.

# 2.6.2 Crouch Gait

The research into crouch gait in spastic diplegia has been hampered by the lack of use of a specific definition of crouch gait in many of the studies. Acceptance of a more exact definition of crouch gait would promote meaningful research into this gait pattern and may lead to advances in our knowledge of the predisposing or causative factors that lead to a crouch gait, and how to successfully correct this limiting gait pattern.

# 2.6.3 Aims of the Research

Consequently the aim of the first study was to identify and classify gait patterns in spastic diplegia, including crouch gait. The results of this study are reported in Chapter 5.

# **CHAPTER 3 MANAGEMENT OF CROUCH GAIT**

# **3.1 INTRODUCTION**

Children with spastic diplegia may walk in a gait pattern known as crouch gait. As already discussed in Chapter 2, this gait pattern is poorly defined in the literature and hence knowledge of crouch gait is hampered by this concern, as is the management of crouch gait.

In this chapter, the general management of cerebral palsy is described, with particular attention on the management of gait in spastic diplegia, including the assessments relating to gait that are made prior to surgical intervention. Past and current orthopaedic surgery to correct crouch gait, are detailed. Issues relating to the surgical management of crouch gait are identified and appropriate research studies proposed to provide new knowledge in the surgical intervention for crouch gait.

# **3.2 MANAGEMENT OF CEREBRAL PALSY**

Cerebral palsy is usually managed by a multidisciplinary approach and may incorporate the input of paediatricians, orthopaedic surgeons, neurologists, paediatric rehabilitation specialists, physiotherapists, occupational therapists, speech pathologists, orthotists, social workers, psychologists, dieticians/ nutritionists and educationalists depending on the needs of the child and family (Graham and Selber 2003, Shepherd 1980).

Optimal physical function for the child with cerebral palsy is dictated by the symptoms of the cerebral palsy from the upper motor neurone lesion. There are positive and negative features of the upper motor neurone lesion. Spasticity, hyper-reflexia and co-contraction are the positive features and muscle weakness, loss of selective muscle control and poor balance, constitute the negative

features (Barnes 2001, Carr et al 1995, Mayer 1997, Sheean 2002). These features combined over time and with longitudinal growth of bones, lead to the musculoskeletal changes of loss of muscle extensibility, muscle and joint contracture, bony torsion, joint instability and eventual development of degenerative arthritis (Graham and Selber 2003).

## 3.2.1 Physiotherapy

Physiotherapy management has predominantly been directed towards reducing spasticity (Bobath and Bobath 1978, Shepherd 1980) and thereby cocontraction, but there is now recognition of the need to strengthen weak muscles (Damiano et al 2002, Damiano et al 1995a, Dodd et al 2002, Dodd et al 2003). Physiotherapists have used many treatment methods over the years, such as Bobath, Peto and Motor Control Learning to achieve reduction of spasticity and promote movement (Shepherd 1980), and now Progressive Resistance Strength Training is being added to physiotherapy regimes (Dodd et al 2003, Morton et al 2005). Increased recognition of dynamical systems theory has promoted understanding of functional movement as an integration of many subsystems such as the nervous system, musculoskeletal system and the environment in which a functional task, such as walking takes place (Campbell 2000, Horak 1991, Shumway-Cook and Woollacott 2001). When there is a "critical change" (Shumway-Cook and Woollacott 2001, p.19) in one of the systems, there is potential to effect change in the movement produced.

#### **3.2.2 Spasticity Management**

A variety of different interventions can be used in the management of spasticity. These have included physiotherapy exercises to gain or maintain range of motion and the instigation of orthoses or bracing, oral medications (oral baclofen, diazepam, dantrolene) (Gormley 2001a), neurolytic agents (Botulinum toxin A, phenol), selective dorsal rhizotomy and intrathecal baclofen (Flett 2003, Gormley 2001a).

Physicians and orthopaedic surgeons may use neurolytic agents, such as botulinum toxin A (BoNT-A), that can be injected into the spastic muscle to

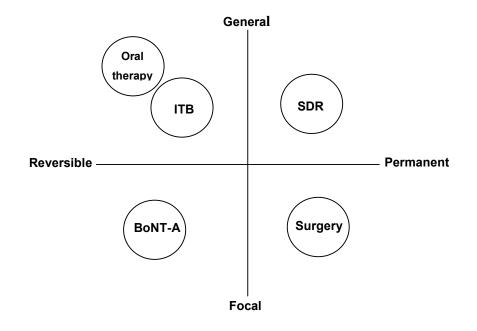
70

achieve chemodenervation of the target muscle and thereby achieve reduction of spasticity.

The effect of BoNT-A is focal and temporary, whereas intrathecal baclofen (ITB) has a generalised effect that is long lasting whilst being administered but is reversible on pump removal (Graham and Selber 2003). BoNT-A is indicated when there is focal spasticity with minimal or no contracture, and this usually is relevant to young children.

ITB is delivered by an implanted pump and has a generalised effect on the muscle tone, so it is useful in cases where spasticity is present in most muscle groups of the body. However complications can be serious (Morton 1999) and can require pump removal (Flett 2003, Graham and Selber 2003).

A surgical option to reduce spasticity permanently, is selective dorsal rhizotomy (SDR). The posterior nerve rootlets from L1 to S1 are tested by electrical stimulation and electromyography detects any abnormal responses (Gormley 2001a). Between 20-40% of the rootlets are sectioned according to intraoperative testing (Graham and Selber 2003). This leads to a reduction in spasticity but also muscle weakness, requiring intensive physiotherapy rehabilitation post-operatively (Graham and Selber 2003). Selective dorsal rhizotomy has also been associated in the longer term with the development of spinal and foot deformities, hip subluxation and weak lower limbs (Graham 1997), and so appropriate selection of children who will benefit from this intervention must be made (Gormley 2001a). Ideally the children should have spasticity affecting most muscle groups of the lower limbs, be young (between four to six years old), strong (minimal underlying muscle weakness and good selective motor control), straight (minimal static contractures), have good cognition and a supportive family (as they need to cooperate and cope with an intense rehabilitation programme) (Bache et al 2003, Gormley 2001a). Figure 3.1 shows the options for the management of spasticity.



# **Management of Spasticity**

Figure 3.1 Options for management of spasticity (Graham et al 2000, p. 71). This diagram shows how management may be generalised to many muscles and either reversible or permanent, or management may be focal to a particular muscle group and again either reversible or permanent.

# 3.2.3 Orthopaedic Surgery

Orthopaedic surgery is usually instigated when muscle and joint contracture, bony torsion and/or joint instability are present and interfering with the ability of the child with cerebral palsy to function adequately or causing deterioration in usual function or mobility. Soft tissue surgery lengthens contracted muscles such as psoas, hamstrings, and gastrocnemius. Tendon transfers augment movement, for example the rectus femoris tendon transfer to aid knee flexion in the swing phase of the gait cycle. Bony surgery re-aligns bony segments, for example the femoral or tibial derotation osteotomy to realign the femur and tibia or os calcis lengthening to re-align the forefoot with the hindfoot (Bache et al 2003, Flett 2003).

#### 3.2.4 Orthoses

Orthoses are often prescribed to augment physiotherapy, spasticity and orthopaedic management. Orthoses are predominantly used to assist with biomechanical alignment (Patrick et al 2001). The orthosis alters the relationship of the foot with the tibia by restricting or preventing movement, and thereby the orthosis can help redirect the ground reaction force so that the lower limbs are better aligned towards a more normal posture.

The most prescribed orthosis for children with cerebral palsy is the ankle-foot orthosis (AFO) and the appropriate use of its variations- leaf spring, hinged, solid and ground reaction, should be understood so that the appropriate prescription is made (Gage 1991, Graham 1997). The leaf spring AFO is prescribed when a drop foot in swing is the main problem and spasticity is absent, as it assists dorsiflexion in swing phase but allows virtually unrestricted ankle movement in stance. The hinged AFO holds the ankle in plantargrade alignment in swing phase, and in stance restricts plantarflexion. First rocker is lost during loading response, but dorsiflexion occurs during second rocker, before blocking third rocker. This type of AFO is prescribed if control of second rocker is adequate but there is a drop foot in swing with dynamic equinus that will interfere with prepositioning of the foot at initial contact. The solid AFO maintains the ankle in plantargrade alignment throughout the gait cycle and so all rockers are prevented. This is indicated if the child in barefoot walking shows poor control of second rocker so that the tibia advances too rapidly over the foot. The solid AFO can also control dynamic pes valgus or varus. The ground reaction AFO (GRAFO) has the ankle positioned in slight plantarflexion and so the ground reaction force is directed anterior to the knee in stance, to assist knee extension.



Figure 3.2 Orthoses commonly used in the management of cerebral palsy. From left to right- hinged AFO, solid AFO, leaf spring AFO and GRAFO.

# **3.3 GAIT ANALYSIS**

Decisions regarding which line of management is most appropriate at a given time, need to be made and these are difficult decisions because the gait pattern that is seen can be influenced by so many different factors. As Rang (Rang 1990) has commented: "The decision is more important than the incision" (p. 483). For example, is it weakness, spasticity, contracture or joint instability that is causing the foot to be in equinus in swing phase? It is important to differentiate between the primary gait deviations that are a result of the central nervous lesion (e.g. spasticity, weakness), the secondary gait deviations that develop over time as a consequence of the primary problems (e.g. muscle contracture, bony torsion) and the tertiary deviations that are used to counteract the effects on the gait pattern by the primary and secondary gait deviations (Davids et al 2003a, Gage et al 1995). The primary and secondary gait abnormalities need to be identified and targeted for treatment and the compensatory gait patterns need to be identified but not treated as they will

diminish or disappear when the primary/ secondary problems have been treated, as they are no longer required (Gage et al 1995, Graham 2004b).

In the past, orthopaedic surgery was undertaken at one level at a time, with the level most involved treated first (Miller et al 1995). This led to multiple admissions for surgical intervention over the years for a child with cerebral palsy- a situation coined the "Birthday Party Syndrome" (Rang et al 1986, p. 365). For example, a child with cerebral palsy walks into the surgeon's office and has a gait pattern of extended hips and knees and feet in equinus. The equinus is visually striking and is dealt with by lengthening of the calf, but the hips and knees become flexed. The surgeon and family become concerned again so hamstring lengthening is undertaken to straighten the knees. However the child now walks with flexed hips and soon the surgeon and family realise that psoas lengthening surgery is required. What about the stiff knees, as all this surgery has not addressed this problem? So back the child goes for more surgery, this time to transfer rectus. So multiple admissions for surgery occur and rehabilitation is never ending. This is also known as "the diving syndrome" (Miller et al 1995, p 488) (Rang et al 1986, p 365) (Figure 3.3).

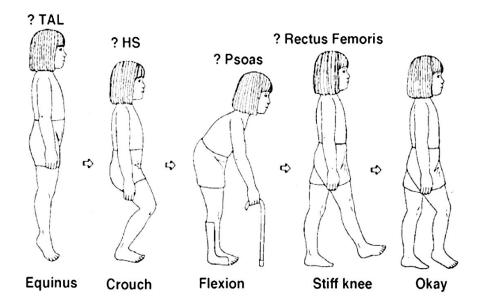


Figure 3.3 "The diving syndrome" as illustrated in Miller et al (1995, p 488). TAL refers to lengthening of the Achilles tendon and HS, to hamstrings lengthening. The diagram shows the progression of gait abnormality according to the relevant muscle group targeted for surgery at the time.

Today, orthopaedic surgery for a child with cerebral palsy is very different, as surgeons aim to do all the procedures in the one surgical session, and so there is only one admission and one rehabilitation period (Browne and McManus 1987, Gormley 2001a, Graham and Selber 2003, Norlin and Tkaczuk 1985, Rang 1990). This type of surgery in Australia is termed Single Event Multilevel Surgery (SEMLS), and will consist of 4-16 orthopaedic procedures in the one surgical session. However debate still remains on the place of multi event single level surgery and it may on occasion still be relevant for less involved children or those from rural settings.

How can the surgeon confidently target the gait abnormalities that need to be dealt with? Gait analysis has been a catalyst for SEMLS as it can help the surgeon distinguish the primary/ secondary gait deviations from the compensatory gait deviations (Gage et al 1995, Gormley 2001a, Miller et al 1995).

Gait analysis typically consists of the following tests: visual gait assessment by using video recording, physical examination, 3DGA; and as required electromyography and/ or energy expenditure studies (Bache et al 2003, Gage et al 1995, Rose et al 1991). Prior to the advent of 3DGA, the assessment of gait consisted of the visual observation of the child walking and the physical examination of the joint range, muscle length and strength, and the degree of spasticity. Nowadays 3DGA provides objective data pertinent to the gait pattern. The visual assessment of gait and physical examination provide additional information that allows a more discerning interpretation of the 3DGA.

# 3.3.1 Visual Gait Assessment

The video recording of gait provides an objective, permanent record and qualitative information of the gait pattern. The visual gait assessment made from the video recording is subjective because it involves interpretation by the clinician. The visual gait assessment highlights the main deficiencies of the gait pattern, such as foot drop in swing, inadequate foot clearance in swing phase, stiff knee gait, lack of heel contact in stance. Once the overall impression of the gait deviations is obtained, then questions as to the causes can be formulated.

## 3.3.2 Physical Examination

Physical examination is the static measurement of joint range and muscle extensibility, bony torsion, muscle strength in the direction of joint movement and the rating of spasticity passively (in the absence of active movement by the child). The information provided relates to the child as they lie at rest on the examination plinth, i.e. it is a horizontal examination. The gait pattern is examined in relation to isolated pieces of information from the physical examination. The information obtained from the physical examination confirms, negates, or at worst is equivocal about a particular joint or muscle contracture, muscle weakness or spasticity being the cause of the gait deviations observed and documented in the visual gait assessment and 3DGA.

#### **3.3.3** Three Dimensional Gait Analysis

The 3DGA is the dynamic biomechanical assessment during walking, of the combined effects of available joint and muscle range, muscle strength and spasticity. It is the objective quantification of the gait deviations seen in the visual gait assessment, i.e. it is a vertical examination.

A 3DGA is conducted using high-level computer technology incorporating specialised cameras, retroreflective markers, forceplates and specially designed software for data collection and processing (Figure 3.4). Temporal-spatial, kinematic and kinetic data are documented. Temporal-spatial data provide information regarding the timing and duration of events in the gait cycle as well as velocity, cadence, step and stride length. Kinematic data describe the orientation of body segments to each other and the related linear and angular displacements, velocities and accelerations. Kinetic data describe the moments and powers acting at the joints. Information is obtained in the sagittal, coronal and transverse planes regarding the excursion of the pelvis, hip, knee and ankle joints throughout the gait cycle (kinematics) and the joint moments and powers at the hip, knee and ankle (kinetics). A description of the process for a 3DGA and the type of data obtained is found in Chapter 4 Methods.



Figure 3.4 A child participating in a 3DGA in the gait laboratory. Two AMTI force plates are incorporated into the grey walkway. In the background there are two Vicon cameras mounted on the walls and a video camera on a tripod on the floor.

### 3.3.4 Electromyography

EMG is a dynamic test of muscle activation. EMG records the electrical signals of the tested muscles as they are activated during the gait cycle, and so provides information regarding the timing of activation (Davids et al 2003a, Miller et al 1995) and whether this is normal, prolonged, premature, delayed, curtailed, and/ or out of phase. This information can be used to ascertain if a muscle is working abnormally or interfering with another muscle's function in gait, and so can assist in identifying the causes of the gait deviation (Gage and Õunpuu 1989, Rose et al 1991).

### 3.3.5 Energy Expenditure Studies

Energy expenditure testing gives objective information of how a child functions over a continuous set period of time, thus simulating real life situations, for example, when a subject goes on an excursion or into a large shopping centre or out into the playground at recess (Figure 3.5). The results of energy expenditure testing reflect the impact of the gait abnormalities on the child's ability to walk

for a predetermined period of time. Energy expenditure testing does not provide any information regarding the causative factors of the gait abnormality.

Interpretation of gait deviations can be made without the data from the energy expenditure test. However energy expenditure does give an indication of how the gait impacts on the child's function. If energy expenditure values are severely elevated, then the question is asked as to whether intervention to improve gait is feasible and in such cases this may influence the recommendations arising from the interpretation of the gait data- the visual gait assessment, 3DGA, physical exam and electromyographic studies.



Figure 3.5 A child participating in an energy test in the gait laboratory. The test is conducted on a 20 metre oval walkway (delineated by the yellow lines) within the gait laboratory. The Cosmed K4 unit is an open spirometry system that is lightweight and telemetric. The child is wearing the face mask which directs the expired air to the processing unit which is worn on the front, and the battery pack is worn on the back.

### 3.3.6 Interpretation of Data

Visual gait assessment is subjective. The physical examination can be objective but results are influenced by the rigor of the protocol, the technique used and the cooperation of the child involved. 3DGA is objective but is only as accurate as the assumptions of the model used and the accuracy of the marker placement employed. Having identified in the visual gait assessment and quantified in the 3DGA the main abnormalities of the gait pattern, then questions regarding the cause of these patterns of gait are asked. Data interpretation involves the assessment of the results of these three tests and then inferences are made as to the primary, secondary and compensatory causes of the gait abnormalities that are seen and appropriate SEMLS can then be planned (Davis 1997). The interpretation of gait data is not objective and is dependent on the experience of the professionals involved in assessing the gait data (Arnold et al 2001a, Davis 1997), reviewing postoperative data to assess the success of interventions, and auditing outcome measures post-intervention (Davis 1997, DeLuca et al 1997, Miller et al 1995) (Figure 3.6). Interpretation of gait data is subject to variability as a consequence (Skaggs et al 2000).



Figure 3.6 Paediatric orthopaedic surgeons, physiotherapists and biomechanical engineers contributing to a gait analysis interpretation and reporting session within our laboratory.

### 3.3.7 Issues in Gait Analysis

There are various issues with respect to the clinical utility of gait analysis. There are limitations in the reliability of the physical examination, the implementation of the 3DGA model used, which determines the type of data obtained, and then in the process of interpretation of the gait analysis data and subsequent recommendations for management.

#### **Physical Examination**

The clinical measurement of static muscle extensibility and joint range must be accurate as these measurements are used in conjunction with the data from the 3DGA to decide on management strategies for the particular gait problem that has been documented.

The reliability of measurements of muscle extensibility and joint range for children with cerebral palsy has been reported in a number of studies (Allington et al 2002, Fosang et al 2003, Keenan et al 2004, Kilgour et al 2003, McDowell et al 2000, Thompson et al 2001, Watkins et al 1995). Some of the studies involved only physiotherapists as the raters (Allington et al 2002, Fosang et al 2003, Kilgour et al 2003, McDowell et al 2000, Watkins et al 1995) and others a mixture of physiotherapist and paediatric orthopaedic surgeon (Keenan et al 2004, Thompson et al 2001). Some studies included a pre-training session for standardisation of measurement techniques (Allington et al 2002, Fosang et al 2003, Kilgour et al 2003, McDowell et al 2000, Thompson et al 2001) and others did not (Keenan et al 2004, Watkins et al 1995) so that the procedure reflected what was currently done in the clinical setting (Watkins et al 1995). Standardising the measurement techniques with a protocol and allowing for training prior to data collection ensures that the methods are repeatable. The non standardisation of the techniques reflects more often what would happen in the real life situation (Watkins et al 1995) e.g. doctor's clinic and the physiotherapy session. However this approach means that the method used, is not reproducible and therefore does not necessarily represent what would happen in another setting. This type of study is helpful in assessing reliability of current clinical practice in a particular setting and as such is a quality control exercise. However such a study does not constitute research that will provide findings that are applicable to other clinicians. Details of the studies are summarised in Table 3.1.

ion measurements.
al examinatior
y of the physic
he reliability o
investigating th
nary of studies
Summa
Table 3.1

Author	Year	Tested	Raters	Subjects	Protocol	Intra-rater overall	Inter-rater overall
Keenan, Rodda et al (2004)	2004	Thomas test, Popliteal, Knee ext, Dorsiflex-knee ext/ flex	PT and orthopaedic surgeon	CP n=20, 5-25yrs	2x same day each rater	CCC=0.67- 0.96	CCC=0.34-0.87
Fosang et al (2003)	2003	Popliteal, Dorsiflex- knee ext Abduction in hip ext	6 PT	CP n=18, 2-10yrs	2x over 6 days	SEM= 3-9° ICC=0.62-0.78	SEM=5-8° ICC=0.55-0.97
Kilgour et al (2003)	2003	Thomas test, Popliteal, Knee ext, Dorsiflex-knee ext/ flex	1 PT	CP= 25, Norm=25 6-17yrs	2x same day repeated 7 days later	Mean absolute diff = 1.0-4.2º for CP	Mean absolute diff= max 7º for CP
Allington et al (2002)	2002	Dorsiflex-knee ext/ flex, Plantarflex, Inversion, Eversion	2 PT	CP n=24, 3-14yrs	2x same day 3x within 10 days	MME= 5° SD= 5°	MME= 5° SD= 5°
Thompson et al (2001)	2001	Popliteal	9 raters- PT orthopaedic surgeons	CP n=16, 5-13yrs	2x 2 weeks apart	SD=Range 7- 14°	
McDowell et al (2000)	2000	Abduction, Popliteal, Dorsiflex- knee ext, Hip IR, Foot/ thigh angle	3 PT	CP n=12, 5-18yrs	3x same day repeated 7 days later	10-14°	18-28°
Watkins et al (1995)	1995	Dorsiflex- knee ext	10 PT	N=75 CP, JCA and norm, 2-17 yrs	2x same day	SEM=3° for CP	SEM=7° for CP
CCC- Concord SEM- Standard ICC- Intraclas MME- Mean M SD- Standard	Concordance Correlation Coeffic Standard Error of Measurement In traclass Correlation Coefficient Mean Measurement Error Standard Deviation	cient PT- CP- It JCA- norm- Popliteal-	arthritis pping subject	dorsiflex- dorsiflexion x- ext- extension yrs flex- flexion IR- internal rotation diff- difference	x- times yrs- years		

The Thomas test was found to have good intra-rater agreement but generally poor inter-rater agreement in the study by Keenan, Rodda et al (2004). This study considered  $\pm 10^{\circ}$  as the limit for clinically acceptable agreement for parameters. The popliteal angle and ankle dorsiflexion measurement with the knee flexed did not fulfil the  $\pm 10^{\circ}$  limit for clinically acceptable agreement, despite good intra- and inter-rater agreement. Knee extension and ankle dorsiflexion with the knee extended both demonstrated good intra- and interrater agreement and fulfilled the  $\pm 10^{\circ}$  limit. As all measurements were taken on the same day, there is no inter-session information available, which means the reliability of these measures overtime is unknown.

In the study by Kilgour et al (2003), this question was addressed. Intra-rater agreement was acceptable when measurements were taken in the same session by the same rater, but the level of agreement diminished between sessions for all parameters measured. This was true for both normally developing children and for those with spastic diplegia, so variability because of spasticity could not be blamed. This study showed good intraclass correlation coefficients within session for the Thomas test, popliteal angle, and ankle dorsiflexion with and without the knee flexed. Unfortunately this study did not provide confidence in reliability for measures taken over time.

When hip internal rotation, thigh foot angle, popliteal angle, ankle dorsiflexion with the knee extended and some other parameters were measured,  $\pm 10-14^{\circ}$  of error were found intra- and inter-session, except for the popliteal angle and ankle dorsiflexion which showed a greater inter-session error of  $\pm 18-28^{\circ}$  (McDowell et al 2000). The increased error was considered to be attributable to these muscles being bi-articular and the recommendation was for care to be taken with the technique of assessment of these muscles (McDowell et al 2000).

The popliteal angle and ankle dorsiflexion with the knee extended were also included in the study by Fosang et al (2003). Intra-rater reliability between sessions and inter-rater reliability at each session was acceptable for these two measures of bi-articular muscle length, with intra-rater error of  $\pm 3-10^{\circ}$ . This

study indicated that measures taken by the same rater at different sessions can be reliable if a protocol has been developed and practised.

Ankle dorsiflexion with the knee flexed and extended has been the entire focus of two studies (Allington et al 2002, Watkins et al 1995). The standard error of measurement was  $\pm 6.7^{\circ}$  for the subjects with cerebral palsy for inter-rater and within sessions data in the study by Watkins et al (1995), and excellent intraand inter-rater agreement and acceptable error were found within and between sessions in the study by Allington et al (2002). Both of these studies provide further confidence in the reliability of the technique of measurement over time despite one study having a protocol that was practised (Allington et al 2002) and the other study allowing raters to measure according to their current clinical practice (Watkins et al 1995).

The repeatability of the popliteal and modified popliteal angle was investigated in a study on hamstring length (Thompson et al 2001). The popliteal angle recorded by the nine raters on two occasions, showed a range of between 37° to 66° with a standard deviation range of 7-14° inter-session. Intra- and inter-rater variability was attributed to 74% of the observed variability and the positioning of the pelvis (which is not standardised in this test) was considered to contribute to this. Hence the modified popliteal angle was proposed as a more clinically useful measurement as the pelvic position is standardised in this test. This recommendation does not take into account however that the results relating to the modified popliteal angle in this study were only marginally better from those of the popliteal angle, implying that the modified popliteal angle is not necessarily the more repeatable measure.

The physical examination measures that were used in the studies reported in this thesis were: Thomas test, popliteal angle, knee extension, ankle dorsiflexion with and without knee extension, hip internal and external rotation, and the thigh foot angle. The current literature on the reliability of these measurements for subjects with cerebral palsy shows a great variation in results. Consequently it is important to recognise that variability in these clinical measures will be present to some extent and the rater must exhibit great care and precision in the

techniques utilised and preferably use a specific protocol for the measurement and be trained prior to data collection. When the clinical measurements are considered in relation to the data from the 3DGA in the interpretation process, the variability of the measure should also be taken into account.

### **3DGA Model**

The model used for 3DGA has been known under various names- the Helen Hayes Model, Newington Model, Kadaba Model to name a few (Baker and Rodda 2003) but overall these models are essentially the same. The model has been adopted due to its incorporation in commercially available systems and software and the number of markers used makes for ease and speed in application (Kadaba et al 1990, Kirtley 2002). However the model is very sensitive to marker misplacement and hence so are the data obtained (Baker et al 1999, Baker and Rodda 2003, Kirtley 2002). An example of this problem is the delineation of the knee flexion-extension axis by markers, which impacts on the amplitude of the hip rotation parameter documented (Baker et al 1999, Baker and Rodda 2003, Kadaba et al 1990, Kirtley 2002). However intra- and inter-rater repeatability of placement of this knee marker has been shown to be poor at the same laboratory site (Schwartz et al 2004), between laboratory sites (Gorton et al 2001) and even with the implementation of a specific in-service training programme aimed to remedy the problem between testers and sites (Gorton et al 2002). Inter-trial variability of the data obtained from normally developing subjects has been shown to be minor (Gorton et al 2001, Kadaba et al 1989, Schwartz et al 2004, Steinwender et al 2000) but is greater for children with cerebral palsy (Steinwender et al 2000).

The estimation of the hip joint centre in the 3DGA model is calculated by equations that are based on 25 hip radiographs and it is not clear if these were the normal hips of adult or child subjects (Davis et al 1991). How the hip joint centre is altered if there is pathology at the hip is not considered. As the model is hierarchical, any inaccuracies in data at the pelvic level and hip will automatically affect the distal joints through a flow-on effect, compounding any inaccuracies from marker placement at these lower levels (Baker and Rodda 2003, Schwartz et al 2004).

#### Interpretation of Data

Various authors have looked at how the data obtained from gait analysis influences the decisions made for surgical intervention (Cook et al 2003, DeLuca et al 1997, Kay et al 2000, Lee et al 1992). Agreement regarding whether surgery should or should not occur may be high (89%) but the level or type of surgery recommended following gait analysis may differ from that without 3DGA by 40% (Cook et al 2003). One study (DeLuca et al 1997) explored the relationship between the surgical recommendations made by 1) the referring surgeon prior to gait analysis, 2) the gait laboratory team using information from the physical examination and video observation alone and 3) the gait laboratory team using information from a gait analysis. This study reported changed recommendations for surgery for 52% of the patients when comparing recommendations made from physical examination and video observation alone, to that with the addition of 3DGA data. This increased to a 92% change in surgical recommendations when the recommendations were compared between the referring surgeon and the gait laboratory team using physical examination and video observation information. The difference in surgical recommendations obtained with the addition of 3DGA information for interpretation is clear from these studies but whether the surgery recommended leads to better improvements in gait for the children undergoing the surgical management has not been demonstrated so far (Noonan et al 2003).

The difference between what is recommended prior to gait analysis by the referring surgeon, what is recommended after interpretation of the gait analysis by the team in the gait laboratory and the surgery actually performed by the referring surgeon after receiving the recommendations from the gait analysis, has been studied (Kay et al 2000). Eighty-nine percent of surgical recommendations changed after interpretation of the gait analysis. More interestingly, in only 51% of cases were the surgical recommendations made from the interpretation of the gait analysis actually followed by the referring surgeon. This seriously brings into question the utility of the gait analysis process for surgical decision making if recommendations are not considered robust enough to adhere to in the surgical programme undertaken. A further study (Wren et al 2005b) has shown better agreement between the surgical

recommendations made from gait analysis data and the actual surgery undertaken (93%). Surgeons participating in this study, completed a questionnaire regarding the reasons for changing the surgical prescription from that recommended from the gait analysis report. However the questionnaire was administered retrospectively and hence relied on the accuracy of the surgeons' memories. It was recognised that the gait analysis data alone do not provide a comprehensive assessment of the child and information gained from radiology, physical examination and evaluation under anaesthesia are also important pieces of information that have a bearing on the surgical decision making process (Wren et al 2005b). Also two doctors can evaluate the same gait analysis data but arrive at different conclusions (Wren et al 2005b). With increased length of time (greater than six months) between the collection of the gait analysis data and the surgery being undertaken, there was a decrease in level of agreement between the gait analysis recommendations and the surgical procedures undertaken (Wren et al 2005b).

An early study (Lee et al 1992) compared the outcome of surgery that adhered to the recommendations made from the interpretation of gait analysis data to that of surgery undertaken which was based on clinical assessment by the respective surgeon. Children were assessed approximately one year post-surgery and identified as having improvement or no improvement according to whether their post-operative data showed a shift towards or away from normal values. Of the children who did not improve post-surgery, five out of seven children had not had surgery that was recommended from the gait analysis findings. Of the children who improved, 14 out of 16 children had had surgery according to gait analysis recommendations. These results indicate that the addition of data from a 3DGA can result in surgical decision making that leads to improvement in gait.

The interpretation of gait analysis data has been scrutinised for variability (Noonan et al 2003, Skaggs et al 2000). In the study by Skaggs et al (2000) gait analysis data from seven patients were sent to 12 experienced gait laboratory physicians in six gait laboratories. Identification of gait problems and treatment recommendations were made. Agreement between physicians was slight to

moderate (based on kappa values) for identification of gait problems and for soft tissue surgery; whereas the agreement for bony surgery was even less. Agreement on gait problems and management strategies, within an institution was higher than that between institutions implying that possibly interpretation of gait analysis data may be influenced by treatment philosophies held by mentors and colleagues within an institution (DeLuca et al 1997). A clinician's level of experience with gait analysis data can also influence the interpretation and recommendations that are made (Davis 1997)

Overall variability of gait analysis has been examined (Noonan et al 2003), from physical examination measures, kinematics and kinetics, electromyography and treatment recommendations. This study involved 11 patients attending four gait laboratories for data collection and interpretation of findings. Substantial variability was found in all the data that were collected and in its interpretation (Noonan et al 2003).

Awareness of the need for precision in the collection of the data in gait analysis, from the measures in physical examination to marker placement in the 3DGA, is imperative by the staff responsible for this data collection. Time must be spent on training and ongoing in-service relating to these techniques and devised protocols. The issues relating to what the data mean and which treatment options are indicated from the data, need to be recognised. Staff involved in the interpretation of data from a gait analysis must be aware of bias in evaluating data for identification of problems within particular treatment philosophies. They also need to be receptive to new literature on gait analysis and surgical treatment but simultaneously critically appraise new findings so that the best treatment options are available to patients seeking their help. As Roy Davis (1997) noted, a gait analysis "does not specify a particular treatment direction e.g. an involved surgical intervention" (p. 254). What the gait analysis data do provide is support for identification of gait problems and their possible causes, and the intervention chosen by the treating surgeon is made with respect to their current treatment philosophies, knowledge of current research in the area and, of course, the needs of the child and family seeking their assistance.

#### **3.4 ORTHOPAEDIC SURGERY FOR SPASTIC DIPLEGIA**

Orthopaedic surgery may be indicated for the child with spastic diplegia when gait and/ or mobility begin to show signs of deterioration. Common complaints by the child or parents are of increased abnormal posturing of the lower limbs (legs turning inwards, knees rubbing, walking on toes, knees are always bent); pain (in or around the patellae particularly); poor foot clearance (toe drag, increased tripping or falls, excessive shoe wear in the toe region or on the medial border); increased energy expenditure (cannot walk as far as previously, decreased participation in usual physical activities). Often these complaints occur around the time of rapid physical growth of the child (entering puberty) when muscle contracture and bony torsion become more accentuated. The presence of hip subluxation also prompts scheduling of the child with spastic diplegia for orthopaedic surgery.

#### **3.4.1** Single Event Multilevel Surgery (SEMLS)

SEMLS is often not undertaken in the young child as physical growth over time may see redevelopment of muscle contractures and bony torsion. However leaving SEMLS to be undertaken during the teenage years is not ideal as the teenager with spastic diplegia has increased social and academic pressures at this time in life.

In order to be effective, SEMLS requires meticulous planning and execution to achieve optimal functional results for the child. Careful assessment to identify the primary, secondary and tertiary problems must be made from gait analysis, radiography and evaluation under anaesthesia in order to plan the appropriate surgical interventions (Bache et al 2003). Analgesia post-surgery must be monitored for the child's comfort and to promote the child's cooperation with early rehabilitation during the inpatient stay in hospital (Bache et al 2003). The family needs to comprehend the aims of the surgery and the extent of the intensive rehabilitation required in the year post-surgery to maximise the effects of the surgery in improving the child's function and gait (Figure 3.7). Monitoring of progress post-surgery requires many hospital outpatient visits to the gait laboratory by the child and family, so that orthotic and assistive device

prescription can be altered to promote improved function and gait (Bache et al 2003).







Figure 3.7 A child with spastic diplegic cerebral palsy participating in a physiotherapy session as part of her rehabilitation post-SEMLS.

SEMLS is usually a combination of soft tissue surgery to lengthen contracted muscles or the transfer of muscles to augment function, and bony surgery to correct bony malalignment or joint instability. The sagittal plane on three dimensional gait analysis, shows gait deviations most attributable to muscle weakness and contracture, particularly contracture of the biarticular muscles-hamstrings, rectus femoris and gastrocsoleus. The gait deviations in the transverse plane mainly reflect the influence of bony malalignment and joint instability.

### Hip

Intramuscular psoas lengthening using the techniques of Gage (Novacheck et al 2002) or Sutherland et al (1997) may be indicated if the child walks with increased anterior pelvic tilt and decreased hip extension in stance on visual gait observation and three dimensional gait analysis. However this posture can be attributable not only to a contracted psoas muscle but also to weak muscles such as the abdominals, gluteus maximus and hamstrings (Davids et al 2003a, Reimers 1973). The Thomas or Staheli tests will be positive for fixed flexion deformity of the hip if the psoas is contracted, though both of these tests can give misleading results if not conducted with attention to correct technique (Davids et al 2003a).

The decision to include percutaneous lengthening of the adductor longus can be difficult to make, because despite the child walking with the femurs adducted when viewed in the coronal plane, on three dimensional gait analysis the femurs are often not adducted but internally rotated and flexed at the hip (Bache et al 2003). The physical examination should show tightness in the adductor's available range of passive movement and the three dimensional gait analysis will show the hip to be adducted in stance in the coronal plane.

External derotation osteotomy of the femur may be performed when there is adduction of the femurs in the coronal view, the clinical exam reveals an increased measure of femoral neck anteversion and internal rotation of the femur and corresponding decreased external rotation, and 3DGA shows increased internal rotation of the femur during stance and/ or swing in the

transverse plane (Davids et al 2003a). Alternatively if the radiology of the hip joint shows subluxation, then derotation of the femur proximally at the level of the intertrochanteric region is undertaken (Pirpiris et al 2003). Distal derotation of the femur at the supracondylar level can be performed if there is no hip joint subluxation (Pirpiris et al 2003). The indications for and outcome relating to proximal versus distal femoral derotation oteotomies have been documented at this (Pirpiris et al 2003) and other centres (Kay et al 2003).

## Knee

Whether to lengthen the medial hamstring muscles is a difficult question. The child may walk with the knees flexed in stance and have reduced stride length on three dimensional gait analysis and on visual gait observation, but the flexed knee posture could be due to equinus or calcaneus at the ankle or flexion at the The popliteal angle and the modified popliteal angle can help to hip. differentiate whether the hamstrings are passively short or short due to the influence of the position of the pelvis on testing. Studies of dynamic muscle length have shown that despite the gait pattern of a flexed knee, the hamstrings may have adequate length or even be long (Delp et al 1996, Hoffinger et al 1993). Medial hamstrings should rarely be lengthened in isolation, as lengthening will not only allow increased knee extension but may weaken the hamstrings ability to simultaneously aid hip extension and lead to increased anterior pelvic tilt (Gage 1990, Hsu and Li 1990). This has been found clinically in our hospital (Bache et al 2003) but the literature declares otherwise (DeLuca et al 1998, Thometz et al 1989, van der Linden et al 2003a). Lateral hamstrings lengthening is usually only undertaken when there is fixed flexion at the knee and a posterior tilted pelvis. Excessive and isolated hamstrings surgery can lead to hyperextension of the knee in stance in the presence of equinus due to the consequences of an overactive plantarflexion knee extension couple (Bache et al 2003, Kay et al 2002, Thometz et al 1989).

Poor foot clearance in swing can be due to rectus and hamstring co-contraction that decreases or delays peak knee flexion in swing phase (Bache et al 2003, Gage et al 1987, Õunpuu et al 1993a, Sutherland et al 1990). Rectus transfer can augment knee flexion in swing (Chambers et al 1998, Gage et al 1987, Õunpuu

et al 1993b, Rethlefsen et al 1999b, Saw et al 2003, Sutherland et al 1990). The transfer can be to semitendinosis if the medial hamstrings are to be lengthened during the same operation, or otherwise transfer can be to gracilis, sartorius or tensor fascia lata (Bache et al 2003, Gage et al 1987, Õunpuu et al 1993a, Sutherland et al 1990).

If full passive knee extension is not gained by muscle lengthening, anterior stapling of the distal femoral epiphysiodesis can be used to gradually obtain knee extension in the child who has not reached puberty (Bache et al 2003). In the older child, a supracondylar extension osteotomy can be considered (Gage et al 2000) but this may necessitate simultaneous patellar tendon shortening if the knee flexion deformity has been long standing (Bache et al 2003, Beals 2001).

#### Ankle and Foot

Toe walking is a common complaint by parents of a child with spastic diplegia. This can be attributable to real equinus of the hindfoot or apparent equinus caused by knee and hip flexion that cause the hindfoot not to have contact with the ground. In apparent equinus, the hindfoot can simultaneously have a plantargrade or even calcaneus alignment with the tibia (Graham 1997). It is imperative that this distinction between true and apparent equinus is made so that inappropriate calf lengthening surgery does not occur. The sagittal plane ankle kinematic in three dimensional gait analysis can help make this differentiation (Davids et al 2003a). The Silfverskiold test in the physical examination assesses the length of the soleus and gastrocnemius muscles so that calf lengthening can be specific to the muscle that is causing the true equinus (Saraph et al 2000). The Strayer operation is the favoured technique for calf lengthening for the child with spastic diplegia at the Royal Children's Hospital in Melbourne, as it allows selective lengthening of the gastrocnemius intramuscularly and if found necessary, the soleus can be lengthened with discretion through fascial lengthening (Strayer 1950).

Often if there is increased femoral anteversion present, there is a compensatory out-toeing distally that develops in order to maintain as neutral a foot progression as possible (Bache et al 2003, Stefko et al 1998). External rotation

of the tibia and pronation of the foot attempts to counter the internal rotation of the femur (Dabney et al 1997). If the femur is derotated externally, the tibia may require a supramalleolar internal derotation osteotomy (Selber et al 2004) to realign the tibia with the femur and so restore the alignment of the lever arm so that muscle contraction can produce adequate moments for stability.

Valgus of the hindfoot, planus of the midfoot and abduction of the forefoot can result from spastic equinus. A tight gastrocsoleus can force the hindfoot into valgus and eventual planus at the midfoot (Bache et al 2003, O'Connell et al 1998, Stefko et al 1998). Persistent poor foot clearance in swing due to rectus and hamstring co-contraction and/ or foot drop in swing due to contracture of the calf and/ or weakness of tibialis anterior, plus bony malalignment proximally from the femur and/ or tibia, may all in some way contribute to abduction of the forefoot.

Careful assessment of the orientation of the tibia and the foot to the femur can help assess where the deviation is occurring (Bache et al 2003). Clinical examination of the bimalleolar axis, thigh-foot angle and heel-thigh axis will give the alignment of the tibia with the femur, the whole foot to the femur and the hindfoot to the femur, respectively (Bache et al 2003). Two dimensional video of the femur aligned in the coronal plane when the child is in standing can give appreciation of any malalignment between the femur, tibia, hindfoot and forefoot. Three dimensional gait analysis can help ascertain this also by assessment of the transverse plane hip and ankle rotations and to some extent the foot progression kinematic. However the foot progression can be misleading if the assessor is unaware of whether marker placement is biased towards the hindfoot to give more accurate data for the ankle kinematic in the sagittal plane. If this is the case, it is hindfoot progression that is plotted in the transverse plane and not the progression of the whole foot.

Correction of a planoabductovalgus foot can be obtained by os calcis lengthening (Andreacchio et al 2000, Bache et al 2003, Noritake et al 2005) or by subtalar fusion. If both the tibia and foot are contributing to the malalignment, surgery to both the tibia and foot simultaneously can be undertaken but may be complicated by excess swelling, bleeding and wound breakdown (Bache et al 2003).

Varus of the hindfoot in spastic diplegia is less common (Bennet et al 1982, O'Connell et al 1998) and is managed by surgery to tibialis posterior and/ or tibialis anterior (Bache et al 2003).

# **3.5 ORTHOPAEDIC SURGERY FOR CROUCH GAIT**

Chronic crouch gait can lead to the development of quite disabling symptoms such as knee pain, fatigue and deterioration in gait pattern (Miller et al 1995, Sutherland and Cooper 1978). Knee pain is often found in association with patella alta and in severe cases there can be "high stress pathologies" (Miller et al 1995, p. 494), such as avulsion or fragmentation of the pole of the patella (Lotman 1976, Miller et al 1995, Rosenthal and Levine 1977). It is these troublesome symptoms of crouch gait, that lead the child and family to seek treatment.

The definitive treatment to correct crouch gait in spastic diplegia is dependent on the identification of the factors that lead to the development of crouch gait in the first instance.

### 3.5.1 Possible Causes

The causes of crouch gait remain elusive (Arnold et al 2005a). Is it due to contracture of the hamstrings or psoas muscles or both (Drummond et al 1974, Gage 1990, Rab 1992b, Rang et al 1986, Sutherland and Davids 1993, Tylkowski et al 1988)? What part does muscle weakness play in crouch gait? It has been attributed to weakness in the gastrocsoleus (Delp et al 1996, Gage 1990, Miller et al 1995, Sutherland and Davids 1993), knee extensors (Damiano et al 1995a, McNee et al 2002, Sutherland and Davids 1993) and hip extensors (Delp et al 1996, Rab 1992b). The effect of lever arm dysfunction particularly associated with external tibial torsion or pes valgus has been discussed (Berghof et al 1997, Delp et al 1996, Gage 1990, Gage 2004a, McNee et al 2002, Rab

1992b, Sutherland and Davids 1993). Deficient motor control (Orendurff et al 2000) and balance mechanisms (Delp et al 1996) may also have a role to play in crouch gait (Gage 1990, Rab 1992b). And then there is the possibility that crouch gait results from the interaction of all of the above. Crouch gait has been known to develop after isolated gastrocsoleus lengthening (Berghof et al 1997, Borton et al 2001, Dillin and Samilson 1983, Rab 1992b, Sutherland and Cooper 1978) (Figure 3.8) and it is thought that unrecognised contracture of the knee and hip flexors at the time of the gastrocsoleus lengthening perpetuates the posture of knee and hip flexion. Post-surgery, the gastrocsoleus is unable to restrict the advancement of the tibia over the foot and hence works in a lengthened position and crouch gait results (Gage 1990). Table 3.2 shows a summary of suggested causes of crouch gait.



Figure 3.8 Sagittal view of a child with spastic diplegia, at age 12 years, standing in crouch gait. This child had percutaneous elongation of the tendoachilles at eight years of age.

- ,			<b>)</b>					
		Cause- Contracture	ure		<b>Cause- Weakness</b>	kness		Cause-
1 <sup>st</sup> Author	Year	Knee	Hip	Ankle	Knee	Hip	Ankle	Lever Arm Dysfunction
Drummond et al (1974)	1974	SMH						
Sutherland and Cooper (1978)	1978	HMS	psoas				iatrogenic	
Rang et al (1986)	1986	SMH	psoas				gastrocsoleus ± iatrogenic	
Tylkowski et al (1988)	1988	SMH	psoas					
Gage (1990)	1990	HMS ± FFD kn	psoas				gastrocsoleus	yes
Rab (1992a)	1992	SMH	psoas		not quads	hip ext?	gastrocsoleus ± iatrogenic	pes valgus/ ext tib torsion
Sutherland and Davids (1993)	1993	SMH	psoas	gastrocsoleus (p143)	quads		gastrocsoleus ± iatrogenic	pes valgus/ ext tib torsion
Hoffinger et al (1993)	1993	оц	psoas					
Miller et al (1995)	1995	FFD kn					gastrocsoleus ± iatrogenic	
Damiano et al (1995a)	1995				quads			
Delp et al (1996)	1996	оц	psoas?			hip ext	gastrocsoleus	ext tib torsion

		Cause- Contracture	are		Cause- Weakness	<u>eakness</u>		Cause-
1 <sup>st</sup> Author	Year	Knee	Hip	Ankle	Knee	Hip	Ankle	Lever Arm Dysfunction
Schutte et al (1997)	1997	14/21 short HMS						
Berghof et al (1997)	1997						gastrocsoleus ± iatrogenic	pes valgus
Thompson et al (1998)	1998	4/18 short HMS	psoas					
Orendurff et al (2000)	2000	оп	оц	оц	ou	Q	оц	
Thompson et al (2001)	2001	10/32 short MHS						
McNee et al (2002)	2002				quads			levers at foot and knee
Gage (2004f)	2004	HMS ± FFD kn	psoas				gastrocsoleus	yes
Arnold et al (2005a)	2005				vasti	gluteus maximus	soleus	
Arnold et al (2005b)	2005	54/152 short HMS						

HMS- hamsrings, FFD kn- knee fixed flexion deformity, MHS- medial hamstrings, quads- quadriceps, ext- extension, ext tib torsion- external tibial torsion

Chapter 3

98

#### **3.5.2** Past Surgical Interventions

Improved knowledge over time regarding some of the causes of crouch gait, has led to the abandonment, modification or continued use of specific surgical procedures.

Most authors in the past recognised that the knee flexion could arise from many sources: spastic or contracted hamstrings or psoas or both (Keats and Kambin 1962, Lotman 1976); weak hip or knee extensors or plantarflexors (Keats and Kambin 1962, Lotman 1976); or lever arm dysfunction from bony torsion. Surgery would need to be directed at the cause(s) (Keats and Kambin 1962). Hamstrings surgery was considered to correct not only the knee flexion contracture but also the flexion at the hip with operations favoured that converted the hamstrings to contract as a monoarticular muscle. Many different forms of hamstrings surgery have enjoyed popularity in the past: Egger's procedure which also aimed to improve knee extension through the transfer of the hamstrings to the posterior femoral condyles whilst maintaining hip extension (Eggers 1952, Keats and Kambin 1962, Pollack and English 1967); lengthening of the hamstrings (Keats and Kambin 1962); and proximal hamstring recession (Seymour and Sharrard 1968) which had the undesired result of increasing lumbar lordosis and genu recurvatum (Drummond et al 1974). Elongation of the patellar tendon due to the constant flexed knee posture was recognised and countered by surgery such as the advancement of the patellar tendon to shorten its length and hence gain effective contraction in knee extension (Bosworth and Thompson 1946, Chandler 1933, Cleveland and Bosworth 1936, Keats and Kambin 1962, Roberts and Adams 1953). It was consequently recommended that this procedure be undertaken in conjunction with hamstring surgery (Baker 1956, Keats and Kambin 1962).

It was recognised in early literature specific to crouch gait (Frost 1971) that crouch was the result of a dynamic component that only came into play when movement occurred, and then there was the static component that was always present that is, contracture of muscles and joints and bony torsion. Treatment had consisted of energy expensive bracing that extended from the ankle to mid thorax that gave a straighter alignment whilst the child wore the bracing. However without the brace, the dynamic forces reverted posture and gait back to that of crouch. Surgical procedures that had been tried were mainly directed to the knee, or sometimes directed to the hip such as anterior flexor releases or femoral subtrochanteric osteotomy. However the price for improved alignment from surgical intervention was at the expense of walking function, often requiring more reliance on assistive devices post-surgery. Bracing and surgery were not recommended by Frost (1971) and the only surgery regularly advocated was that of gastrocsoleus lengthening to improve the base of support and surgical releases for flexion contracture.

### 3.5.3 Current Surgical Interventions

More recently, the decision regarding which operations should be performed to correct crouch gait, has been based on the interpretation of gait analysis results which has led to an increased understanding by surgeons of the interaction of the dynamic and static components of abnormal gait patterns. How a muscle traversing two joints, such as the hamstrings or gastrocnemius can affect movement at the individual joints and their interaction with monoarticular muscles, is grappled with when interpreting gait analysis and making the subsequent surgical recommendations. More specific, tailored surgical recommendations are made. Considering knee flexion in the context of the biomechanics of the whole lower limb and trunk, has lead to more combinations of orthopaedic surgical procedures (SEMLS) being used to correct crouch gait (Gage 1990, Rab 1992b). If the child with spastic diplegia is walking in crouch gait, the surgery combines correction of short muscles in the sagittal plane and bony malalignment in the transverse plane using the same principles as stated for SEMLS in spastic diplegia.

The recent dilemma emerging from the literature is whether the hamstrings should be lengthened, as they are most likely adequate or long in length. This focuses attention on the influence of psoas contracture in producing crouch gait. If there is contracture in the psoas, this can be corrected by muscle lengthening. If there is contracture of the hamstrings, this too can be lengthened. If it is due to calcaneus posture of the foot, this is difficult to remedy. Is it a combination of any two or all three of the above (Gage 1990, Renshaw et al 1995)? This

view also ignores the contribution of weakness in the abdominals, gluteus maximus and the hamstrings in flexion at the hip and weakness of the hamstrings and rectus femoris in knee flexion and soleus weakness in calcaneus at the foot. Bony malalignment causes lever arm dysfunction and so the length of the lever arms from which muscles would contract are altered and this too could predispose a child with diplegia to walk in crouch gait.

Hamstrings surgery is recommended with caution now due to concern that these muscles may indeed not be short but actually long (Delp et al 1996, Hoffinger et al 1993, Schutte et al 1997, Thompson et al 1998). The effect of iliopsoas in accentuating hip flexion, if the hamstrings are fractionally lengthened and thereby weakened, is now considered and lengthening of the psoas may also be undertaken in conjunction with hamstrings surgery or in isolation (Novacheck et al 1996). However psoas surgery has also been questioned as this may weaken and hinder flexion of the limb into swing (Delp et al 1996), particularly in the presence of pes valgus, as hip and ankle power generation is diminished (Berghof et al 1997).

When the hamstrings are lengthened, the need for transfer of the rectus femoris tendon to assist the weakened, lengthened hamstrings to achieve knee flexion in swing phase and hence limb clearance, is now promoted (Gage 1990, Rab 1992b).

Bony torsion in the lower limb diminishes the effect of the plantarflexion knee extension couple and so may need to be factored into the surgical decision making (Gage 1990, McNee et al 2002) by including derotation osteotomies or bony stabilisations in the surgical prescription.

Because numerous authors have indicated iatrogenic calf lengthening as a cause of crouch gait, calf lengthening is rarely performed unless in the presence of a hindfoot valgus which, once corrected, reveals the presence of a tight calf (Dabney et al 1997). Then the calf surgery should be accompanied by other surgery that is simultaneously performed proximally to correct hip and knee flexion (Bache et al 2003, Renshaw et al 1995).

SEMLS deals with lengthening muscles that are tight dynamically or structurally, and bones that have torsion or are malaligned. It does not address muscle weakness, particularly of the quadriceps, that may contribute to crouch gait (Sutherland and Cooper 1978). The role of quadriceps strengthening in improving crouch gait as a conservative management option or as part of a management regime has only recently been studied (Damiano et al 1995a).

If plantargrade alignment of the tibia to the foot is restored, will the child resume a more upright posture? The answer is probably not if the knee or hip have dynamic or static flexion contractures and/ or weakness in the knee or hip extensor muscles. If the knee or hip remain flexed but the ankle is plantargrade, the child's ability to stay upright will be compromised as the centre of gravity will not be within their base of support. Surgery will be required that will impact on all levels of the lower limbs so that an upright posture can be feasible. And if an upright posture can be gained, can it be maintained over time? There are many unanswered questions regarding the predisposing or causative factors that lead to crouch gait and then how to gain sustained surgical correction of crouch gait.

# **3.6 SUMMARY**

The exact causes of crouch gait and the combination of surgical procedures in SEMLS, which will effectively correct crouch gait remains undecided. And if correction of crouch gait is obtained in the short term post-SEMLS, can this correction be maintained in the long term?

### 3.6.1 Aim of the Research

Consequently the aims of the second and third studies reported in Chapters 6 and 7 in this thesis were to:

• Identify in subjects with severe crouch gait, common parameters from baseline data obtained from three dimensional analysis with particular emphasis on the biomechanics of crouch gait.

• Evaluate the outcomes of SEMLS on severe crouch gait at one year postoperation and again at five years.

## CHAPTER 4 GENERAL METHODS

#### **4.1 INTRODUCTION**

Many of the testing procedures were common to all studies undertaken. These procedures will be discussed here. Testing procedures specific to a particular study will be detailed in the relevant chapter.

## **4.2 ETHICAL APPROVAL**

Approval for clinical audit was obtained from the Divisional Director of the Division of Surgery at the Royal Children's Hospital, Melbourne. The granting of a clinical audit complied with the ethical requirements of the Ethics in Human Research Committee at the Royal Children's Hospital (RCH). See Appendix D for the form signed by the Divisional Director for clinical audit approval and the letter from the RCH Ethics in Human Research Committee recognising compliance with ethical requirements.

## **4.3 SUBJECTS**

In all of the studies reported in this thesis, children with spastic diplegic type of cerebral palsy were compared with children with normal neuromuscular function. This comparison is reported in Chapter 5 so that the gait patterns of children with spastic diplegia could be referenced against the gait patterns of normally developing children. In Chapter 6 children who walk in crouch gait were compared to normally developing children to show the extent of the gait deviations in crouch gait at baseline. In Chapter 7 the data from the children who had undergone SEMLS to improve their crouch gait were compared to their preoperative data as well as to reference data to ascertain if the improvements in crouch gait approached normal gait.

In order for this comparison to occur, two groups of subjects were required. The subjects with the gait disturbance were required to have:

- A diagnosis of cerebral palsy and spastic diplegia.
- The ability to walk with or without assistive devices but without "hands on" supervision for safety purposes.
- Be within the age range: 4 to 18 years.
- No previous intervention by selective dorsal rhizotomy or intrathecal baclofen, and the absence of any other major health problem.

Reference data were collected on children who were assessed as having normal neurological, musculoskeletal and cardiorespiratory systems, had gestation of 36 weeks or more, and commenced walking before 18 months of age. A reference database was compiled from data from 17 subjects, ten females and seven males with a mean age of 10.2 years (range 4.5 - 14.9 years) for the study of classification of gait patterns in the sagittal plane in spastic diplegia (Chapter 4). For the studies on crouch gait in spastic diplegia (Chapters 5 and 6), a subgroup of 14 normally developing subjects who were in the age range of subjects with spastic diplegia was used (5 males and 9 females, mean age 11.9 years, range 7.5 - 14.9 years).

The normally developing subjects completed the same physical examination and followed the same protocol for the three dimensional gait analysis data collection as the subjects with cerebral palsy.

### 4.4 FUNCTIONAL CLASSIFICATION

The subjects with spastic diplegia were classified according to three systems of classification- the Gross Motor Function Classification System for Cerebral Palsy (GMFCS) (Palisano et al 1997, Wood and Rosenbaum 2000), the Gillette Functional Assessment Questionnaire (FAQ) (Novacheck et al 2000), and the Functional Mobility Scale (FMS) (Graham et al 2004a). The physiotherapist rated the GMFCS, FAQ and FMS in consultation with the subject and their

parents or retrospectively from information obtained from interview. Refer to Appendices A, B and C for these forms.

### **4.5 CLINICAL HISTORY**

For the subjects diagnosed with spastic diplegia, a record of the subject's clinical history was taken detailing in particular:

- Recent changes in gait relating to function and the gait pattern, such as deterioration in gait pattern, increasing number of falls, increased fatigue, presence of pain, inability to participate in activities that were previously possible to undertake.
- Previous interventions (botulinum toxin A injections and surgeries).
- Use of orthoses and assistive gait devices.

### 4.6 PHYSICAL EXAMINATION

The protocol used for the physical examination was the standardised protocol that was being used in the Hugh Williamson Gait Laboratory (RCH) at that time. It consisted of three sections: anthropometric measures, joint and muscle extensibility and grading of spasticity. Routine testing of muscle strength was added later to the assessment protocol and so is not reported in any of the studies in this thesis.

### 4.6.1 Anthropometric Measures

Anthropometric measurements were required as input into the software for the processing of the three dimensional gait analysis data and were dictated by the protocol for the Vicon Clinical Manager (VCM) (Oxford-Metrics 1995). The measurements required were: height, weight, bilateral measures of leg lengths, and knee and ankle widths. The following optional measurements were included: inter anterior superior iliac spine (ASIS) distance, anterior-posterior distance from ASIS to greater trochanter, and tibial torsion.

## Height

Height was measured using a wall mounted height device (Holtain Ltd, Crymycn, Dyfed, Britain) if the subject could stand with straight knees. If the subject had a knee joint contracture, then their height was measured in supine, using the sum of the following three measurements: top of head to ASIS, ASIS to mid point of medial knee joint line, and mid point of medial knee joint line to plantar surface of heel (passing through the medial malleolus). Measurement was in centimetres taken to one decimal place.

# Weight

Weight was measured using electronic scales (Tanita BWB 600, Wedderburn Scales, Melbourne, Australia). Measurement was in kilograms taken to two decimal places.

# Leg Length

Leg length was measured from ASIS to medial malleolus via the knee joint. In cases of knee joint contracture the measurement was the sum of the following distances: ASIS to mid point of medial knee joint line, and mid point of medial knee joint line to the medial malleolus. Measurement was in centimetres taken to one decimal place.

### Knee and Ankle Widths

Knee and ankle widths were taken using vernier callipers (Series 534, Mitutoyo, Japan) with the subject in the standing position. The lateral and medial epicondyles of the knee and the lateral and medial malleoli of the fibula and tibia respectively were palpated and the axes of the knee and ankle joints estimated. The distance between the condyles for the knee width and between malleoli for the ankle width was measured, with care not to indent the skin (the placement of the knee or ankle marker would not compress the skin). Measurement was in centimetres taken to two decimal places.

#### Inter ASIS Measurement

Inter-ASIS measurement was made using the same vernier calliper, with the subject in supine. Measurement was from left ASIS to right ASIS, in centimetres taken to two decimal places.

### ASIS to Greater Trochanter Measurement

The vertical distance of the ASIS to greater trochanter was made using the same vernier calliper, again with the subject in supine. Measurement was made with the lower limb in neutral rotation as judged by palpation of the greater trochanter in its most lateral position. One arm of the calliper was placed on the palpated greater trochanter such that the upper edge of the arm was no longer visible to the examiner and the upper arm was placed on the ASIS. Care was taken to keep the calliper vertical. Measurement was in centimetres taken to two decimal places.

#### **Tibial Torsion**

Tibial torsion as defined by the Vicon Clinical Manager Manual (VCM) (Oxford-Metrics 1995), is the angle of the "bimalleolar axis with the knee flexion/ extension axis" (p. 28). Measurement was in degrees taken in 5° increments.

The subject was in prone with the knee flexed to  $90^{\circ}$ . The physiotherapist knelt on the end of the plinth looking directly over the foot. The heel was gently positioned in plantargrade (the subtalar joint position was not altered). The angle between the bimalleolar axis and the line perpendicular to the line of the thigh was measured. The centre of the goniometer was placed lateral to the heel with one arm of the goniometer on the line between the lateral and medial malleloli and the other arm perpendicular to the line down the centre of the thigh (Figure 4.1).



Figure 4.1 Tibial torsion as measured according to the Vicon Clinical Manager protocol (Oxford-Metrics 1995, p. 28).

### 4.6.2 Joint and Muscle Extensibility

These were measured according to the Hugh Williamson Gait Laboratory Protocol. Studies detailing the reliability of the following measures were detailed in Chapter 3, Section 3.3.7 (Allington et al 2002, Fosang et al 2003, Keenan et al 2004, Kilgour et al 2003, McDowell et al 2000, Thompson et al 2001, Watkins et al 1995).

The following principles for conducting the tests were adhered to:

- As the limb segment was moved, the examiner observed any accessory movements in other body segments.
- The end of the available passive range was reached when any accessory movements of other body segments were observed.
- Once the limb segment had been taken to the end of the available range of motion, the stationary limb segment was rechecked for any movement away from the starting position.
- The relevant bony or soft tissue reference points, from which the measurement was to be taken, were palpated. The arms of the goniometer were aligned in line with these reference points and the angle made was recorded.

The pertinent tests are detailed below:

## Hip Flexor Length

This was measured using the Thomas Test to assess for lack of passive hip extension.

## Thomas Test (Keenan et al 2004)

The subject was in supine and both legs were flexed until the ASIS was positioned vertically over the posterior superior iliac spines (PSIS)- this enabled standardisation of the alignment of the pelvis and amount of lumbar spine flexion. The hip of the leg to be measured was extended. If there was a knee flexion contracture at the knee then the lower leg was taken over the end of the plinth so that the knee contracture did not limit the available hip range to be measured. The pelvis was not allowed to tilt forward or the lumbar spine to extend. The angle of the femur with the horizontal was measured by placing the centre of the goniometer over the greater trochanter of the femur, one arm of the goniometer in line with the lateral condyle of the femur and the other arm, parallel with the horizontal (Figure 4.2).



Figure 4.2 The procedure for measurement of the Thomas Test.

## Hamstring Muscle Length

This was measured using the popliteal and modified popliteal angle. The popliteal angle measures the length of the hamstrings without reference to the pelvic position, whereas the modified popliteal angle standardises the position of the pelvis so that the influence of the position of the pelvis is negated (Thompson et al 2001).

# Popliteal Angle (Keenan et al 2004)

The subject was in supine and the limb to be measured was flexed to 90° at the hip. The limb was extended at the knee, whilst ensuring that the hip remained flexed at 90°. Care was taken so that the contralateral lower limb did not lift from its resting position on the plinth, nor did the measured limb begin to lift from the pelvis or extend from the thigh. The angle of the tibia with the vertical was measured as a positive value. The centre of the goniometer was placed over the lateral epicondyle of the femur, with one arm of the goniometer in line with the greater trochanter of the femur and the other arm in line with the lateral malleolus (Figure 4.3).



Figure 4.3 The procedure for measurement of the popliteal angle.

#### Modified Popliteal Angle (Thompson et al 2001)

This measurement differs from the popliteal angle in the starting position. The subject was in supine and both legs were flexed until the ASIS was vertical over the PSIS on palpation. This enabled standardisation of the position of the pelvis. The leg to be measured was brought back to the point of 90° of hip flexion and the limb was extended at the knee, whilst ensuring that the hip remained flexed at 90°. The measurement was made according to the same guidelines as stated above for the popliteal angle.

### Knee Joint Range of Movement

The range of movement of the knee joint was measured to assess the presence of a knee joint contracture.

#### Knee Extension (Keenan et al 2004)

The subject was in supine and the tibia was extended on the femur. The angle between the femur and the tibia was measured by placing the centre of the goniometer over the lateral epicondyle of the femur and one arm of the goniometer aligned with the greater trochanter of the femur and the other in line with lateral malleolus of the tibia.

### Gastrocsoleus Length

The length of the gastrocsoleus was measured by the Silverskiold test to differentiate between the length of the gastronemius and soleus muscle (Keenan et al 2004).

#### <u>Soleus</u>

The subject was in supine and the limb to be measured was flexed to 90° at the hip and 90° at the knee. The ankle was dorsiflexed with the subtalar joint held in neutral, ensuring that the hip and knee remained flexed at 90°. Care was taken to ensure that the subject did not push against the movement. The angle between the line that was parallel to the inferior border of the heel and the line perpendicular to the line of the fibula head and the lateral malleolus, was measured. The centre of the goniometer was placed over the lateral malleolus with one arm of the goniometer parallel to the head of the fibula and the other arm parallel to the inferior border of the calcaneum (Figure 4.4).



Figure 4.4 The procedure for measurement of soleus length.

## Gastrocnemius

The subject was in supine and the limb to be measured was flexed to  $90^{\circ}$  at the hip and  $90^{\circ}$  at the knee. The ankle was dorsiflexed whilst the subtalar joint was held in neutral, and the hip and knee were slowly extended to  $0^{\circ}$ . Again care was taken to ensure that the subject did not push against the movement. The angle and reference points for the goniometer were the same as those used for measuring the length of the soleus (Figure 4.5).



Figure 4.5 The procedure for measurement of gastrocnemius length.

# Arc of Rotation of the Hips

# Internal Rotation (Õunpuu et al 2002a)

The subject was in prone, thighs together and the pelvis was stabilised. Both knees were flexed to  $90^{\circ}$  and both hips were internally rotated. Care was taken to ensure that the pelvis did not rotate and that the thighs did not abduct. The angle between the line of the tibia and the vertical was measured by placing the centre of the goniometer over the midpoint between the two femoral condyles. One arm of the goniometer was in line with the line of the tibia and the other arm vertical, perpendicular to the table (Figure 4.6).



Figure 4.6 The procedure for measurement of internal rotation of the hips.

## External Rotation (Õunpuu et al 2002a)

The subject was in prone, thighs together and the pelvis was stabilised. The limb to be measured was flexed at the knee to 90°, the other limb remained on the bed with knee extended. The hip was externally rotated from a position of internal rotation (as there may have been a negative external rotation range available). The hip was externally rotated to the point where the pelvis lifted off the bed. The angle and reference points for the goniometer were the same as those used for measuring the internal rotation of the hip (Figure 4.7).



Figure 4.7 The procedure for measurement of external rotation of the hips.

### **Orientation of the Femur to Foot**

### Thigh Foot Angle (McDowell et al 2000)

The thigh foot angle measures the position of whole foot in relation to the thigh. The subject was prone and the knee was flexed to 90°. The physiotherapist knelt on the end of the plinth looking directly over the foot. The heel was gently placed in plantargrade (the subtalar joint position was not corrected). The angle between the line of the thigh and the line through the centre of the heel and  $2^{nd}$  ray of the toes was measured. The centre of the goniometer was placed over the mid point of the posterior heel and one arm of the goniometer was in line with the  $2^{nd}$  toe and the other arm with the line down the centre of the thigh.

### 4.6.3 Grading of Rectus Femoris Spasticity

The spasticity in the rectus femoris muscle was assessed according to the Duncan Ely test. The Duncan Ely test assesses the spasticity of rectus femoris, and whether spasticity is present with fast or slow movements (Marks et al 2003). Only results from the fast test were utilised in this thesis.

The subject was in prone and the knee was flexed rapidly through range. The test was repeated if the subject actively resisted or assisted with the movement. The test was classified as positive or negative depending on the presence of a catch that interrupted temporarily or otherwise, the passive movement of the knee. Lifting of the pelvis from the table was recorded as a rise.

#### 4.6.4 Foot Posture

This was measured in barefoot standing with the child unsupported or supported only by assistive devices, which were usually used for walking.

### Hindfoot

Hindfoot orientation in the sagittal plane was recorded as normal, equinus or calcaneus by assessing the relationship of the hindfoot to the tibia. A plantargrade hindfoot in relation to the tibia was considered normal. A hindfoot that was in plantarflexion in relation to the tibia was recorded as equinus. Calcaneus was recorded if the hindfoot was in dorsiflexion in relation to the tibia.

Hindfoot orientation in the coronal plane was recorded as normal, varus or valgus by assessing the relationship of the hindfoot to the tibia. If the alignment of the heel was in approximately  $5^{\circ}$  ( $\pm 5^{\circ}$ ) of valgus to the tibia, this was recorded as normal. Varus was considered to be an alignment of the heel medial to the line of the tibia (towards the midline of the body). Valgus was noted if the alignment of the heel was > 10° lateral to the line of the tibia (away from the midline of the body).

### Midfoot

Midfoot orientation was assessed in the sagittal plane. This was recorded as normal, planus or cavus by assessing the relationship of the talus with the navicular. A flattened medial arch was recorded as planus and an unusually high medial arch, as cavus.

### Forefoot

Forefoot orientation was assessed in the horizontal plane. This was recorded as normal, abducted or adducted by assessing the relationship of the hindfoot with the forefoot. It was understood that the joints that comprise the midfoot influenced this relationship between the hindfoot and the forefoot. Normal alignment was recorded if the alignment of the forefoot was in direct line to the line of the hindfoot. Abduction was noted if alignment of the forefoot was lateral to the line of the hindfoot (away from the midline of the body). Adduction was an alignment of the forefoot medial to the line of the hindfoot (towards the midline of the body).

### 4.7 VIDEO RECORDING OF GAIT

Video of the subjects' gait was recorded in the sagittal, coronal and split screen views using the same instructions and clinical scenario as detailed in Section 4.8 below.

### 4.8 THREE DIMENSIONAL GAIT ANALYSIS (3DGA)

The purpose of the gait analysis was to record the subjects' usual gait, not the best gait possible. The children were asked to walk using their typical gait pattern, at a self-selected speed along a 10 metre walkway in the gait laboratory. If the usual method of walking required the use of an assistive device, then this device was used during the walking trials for data collection. Subjects wore suitable clothing such as shorts and rolled up T-shirts. All markers were placed directly onto the skin to minimise movement during gait.

#### 4.8.1 Equipment and Software

A Vicon 370 System (Oxford Metrics, Oxford, England) with five infra-red cameras, was used for the initial three dimensional gait analyses. The walkway incorporated two force plates (Advanced Mechanical Technology, Incorporated, Newton, MA, USA) positioned in series, which provided the data for calculation of kinetic data for subjects who did not use an assistive device. The Vicon Clinical Manager (VCM) was the software used to process the data collected from the Vicon 370 system. The system was subsequently upgraded, so that some of the five year data collected for the crouch gait study were collected using the Vicon 512 System (plus eight cameras) with processing undertaken using Plug in Gait (PIG) in Polygon software. Because processing of kinetic data in VCM consistently underestimates the magnitude of the kinetics for data captured at 50 Hz (Baker and Rodda 2003), all of the kinetic data were

processed using PIG so that the data could be compared from pre-surgery to five years post-surgery. This difference in magnitude of kinetics between VCM and PIG processing is due to the filtering of data at the force plate level prior to processing in VCM and the spline filtering in VCM processing that is sensitive to the frequency of data capture (Baker and Rodda 2003). Processing of kinematics in VCM is comparable to that processed in PIG and so kinematic data that had originally been processed in VCM were not reprocessed in PIG.

Issues relating to the accuracy of the data collected in a 3DGA have been reviewed in Chapter 3, Section 3.3.7.

### 4.8.2 Marker Placement

Marker placement was as described in the Vicon Clinical Manager Manual (Oxford-Metrics 1995) with the Knee Alignment Device (KAD) used during the static trial (Figure 4.8). Markers were placed on:

- Each ASIS and midway between the PSIS.
- Thigh wands on the lateral aspect of the thighs (at a level so as not to obstruct arm swing).
- KAD at the knee to delineate the axis of knee flexion-extension at the knee joint.
- Shank wands on the lateral aspect of the tibia.
- Lateral malleolus in line with the ankle joint axis.
- The intermediate cuneiform in line with the 2<sup>nd</sup> metatarsal. This marker was placed proximal to that stated in the Vicon Clinical Manager Manual so that the data obtained would depict the movement of the hindfoot and not the whole foot. This was done because many subjects had mid and forefoot abnormalities, which would lead to inaccurate recording of sagittal plane ankle kinematics if the marker was on the distal portion of the 2<sup>nd</sup> metatarsal.
- The posterior aspect of the heel, such that this marker, together with the aforementioned marker, was in line with the orientation of the hindfoot. The height of these two markers, were equal from the inferior aspect of the line of the foot.

## 4.8.3 Hip Joint Centre Calculation

The hip joint centre location was calculated using the model described by Davis et al (1991). This requires measurement of leg length and the vertical distance between the ASIS and greater trochanter when the greater trochanter is positioned most lateral. The VCM and PIG processing was undertaken with the tibial torsion measurement entered into the program. The kinematic and kinetic data were calculated according to published work (Bell et al 1990, Davis et al 1991, Kadaba et al 1990) as documented in the Vicon Clinical Manger Manual (Oxford-Metrics 1995).



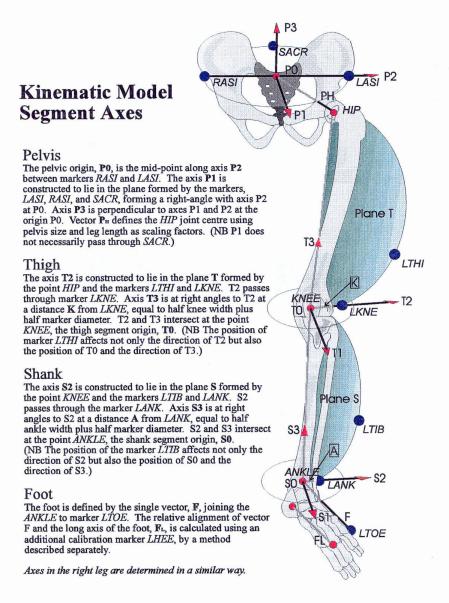


Figure 4.8 Marker placement for subjects in the studies. Reproduced from the Vicon Clinical Manager Manual (Oxford-Metrics 1995, p. 15).

## 4.8.4 Walking Trials

All walking trials for analysis in these studies were undertaken with the subjects barefoot. Subjects who used ankle foot orthoses (AFO) on a daily basis also

had walking trials undertaken with the orthoses. However the data pertaining to the orthoses were not reported in any of the studies within this thesis.

In the study on classification of gait patterns in spastic diplegia, the natural gait pattern of the subject was required for classification and so barefoot trials were considered suitable. Using AFOs would only serve as a confounder in both the cross-sectional and longitudinal parts of the study as the AFOs would alter the natural gait pattern.

In the studies on severe crouch gait, again only barefoot walking trials were analysed as the extent of severe crouch was being evaluated in the baseline study and AFOs would potentially mask and alter these results. In the study on SEMLS correction of severe crouch gait, trials using AFOs were not analysed as the performance of SEMLS on correcting severe crouch gait and long term maintenance of improvement was being evaluated and AFOs would again be a confounder in this study as they impact on the gait pattern.

It was recognised that AFOs may be normally worn pre-operatively and postoperatively for some subjects and improvement in gait may only be seen when the AFOs were worn. However only four subjects wore any orthoses pre-SEMLS and at five years post-SEMLS only two of these subjects were available for re-assessment so data from studies with AFOs were not considered meaningful in this situation.

The use of assistive devices during walking trials was allowed in the studies, as assistive devices were essential for some subjects to be able to walk. Any subject who could functionally walk unaided despite having an assistive device, was evaluated using the unaided walking trials. Functional walking was considered to be walking that was:

- Conducted safely over a distance of 10 metres without "hands on" supervision by another person for safety.
- Fluent- not a step, stop, regain balance and then step again process.

Five walking trials with force plate data (in the cases in which gait assistive devices were not used) were processed using PIG in Polygon software. Three to six trials were then scrutinised from which a typical representative trial for the left and right sides was chosen for analysis as part of each respective study. A typical representative trial was identified according to the following criteria:

- a common velocity with other trials.
- kinematic and kinetic traces similar in pattern and excursion to other trials (no outlying traces).
- stance/ swing ratio similar to other trials.

Parameters chosen for analysis pertinent to each study are detailed in the relevant chapter. All moments reported in this thesis are internal moments in the sagittal plane and all power parameters are the total from the three planes.

Two physiotherapists (JR and RB) were involved in the acquisition of data for the subjects in the study on the classification of sagittal plane gait patterns in spastic diplegia. The same physiotherapist (JR) undertook all of the above testing procedures for all subjects in all instances in the studies on severe crouch gait.

## 4.8.5 Terminology Used in Gait Analysis

There is common terminology that is used in a 3DGA that is found in this thesis.

The gait cycle is taken from the contact of the foot with the ground until repeat contact of the same foot with the ground. The gait cycle is divided by the events of initial contact and toe off, into the periods of stance and swing (Perry 1992).

- Initial contact is the contact of the foot with the ground.
- Toe off is the lift off of the foot from contact with the ground.
- Stance phase is the duration of a foot contact with the ground, beginning with initial foot contact and ending with toe off of the same foot.
- Swing phase is the duration of a single foot non contact with the ground beginning with toe off and ending with initial contact of the same foot with the ground.

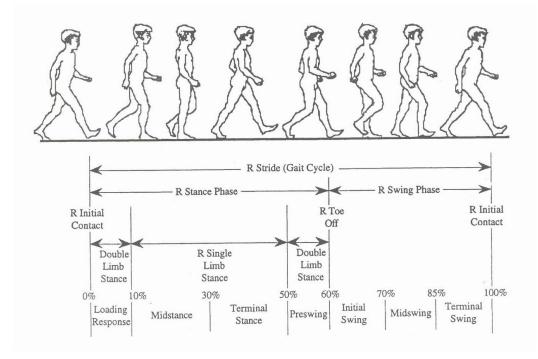


Figure 4.9 Divisions of the gait cycle. Reproduced from Davids (2003b).

The stance phase is divided into three intervals: initial double support, single limb support and second double support (Perry 1992), which are defined by the gait events of opposite foot off and opposite foot contact (Figure 4.9).

- Opposite foot off is the lift off of the contralateral foot from contact with the ground.
- Opposite foot contact is the contact with the ground of the contralateral foot.
- Single support is the period of foot contact with the ground whilst the contralateral foot is in swing phase.
- Double support is the period of foot contact with the ground during which the contralateral foot is also in contact with the ground. There are two periods of double support, one at the beginning of the gait cycle and one prior to toe off.

The stance phase has four sub-phases, which consist of loading response, mid stance, terminal stance and pre-swing (Davids 2003b). Swing phase has three sub-phases known as initial, mid and terminal swing (Davids 2003b). Despite being sub-phases of the gait cycle, these are also commonly referred to as

phases (Perry 1992). Although there is controversy over terminology, the terminology used by Perry (1992) has been adopted in this thesis. These phases contribute to the tasks required by the limbs for gait to occur: weight acceptance, single limb support and limb advancement.

- Loading response occurs from initial contact until the contralateral foot achieves toe off. It comprises 1<sup>st</sup> double support.
- Mid stance is from toe off of the contralateral foot to heel rise of the ipsilateral foot. It comprises the 1<sup>st</sup> half of the interval of single limb support.
- Terminal stance is from ipsilateral heel rise to contralateral foot contact with the ground. It comprises the 2<sup>nd</sup> half of the interval of single limb support.
- Pre-swing is from the time of contralateral foot contact to ipsilateral foot off. It comprises the 2<sup>nd</sup> interval of double support.
- Initial swing begins with foot off and finishes when the foot is opposite the contralateral stance limb.
- Mid swing commences when the ipsilateral swing foot is opposite the contralateral stance limb and finishes when the ipsilateral limb is brought forward and the tibia is aligned vertically.
- Terminal swing occurs from the moment the tibia is vertical in swing phase and ends at initial contact.

# Ankle Rockers

Three rockers have been identified that describe the motion of the foot and ankle during stance phase (Perry 1992) (Figure 4.10). Perry (1992) refers to these as the heel, ankle and forefoot rockers and Gage (2004a) refers to them as the  $1^{\text{st}}$ ,  $2^{\text{nd}}$  and  $3^{\text{rd}}$  rockers. The latter terminology will be used in this thesis.



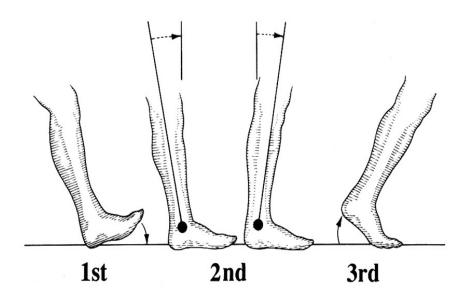


Figure 4.10 Ankle rockers- showing 1<sup>st</sup>, 2<sup>nd</sup> and 3<sup>rd</sup> rockers in normal gait. Reproduced from Gage (2004a, p. 55).

- 1<sup>st</sup> rocker begins at initial contact and in normal gait this contact is made with the heel and the pre-tibial muscles control the lowering of the forefoot to the floor and advance the tibia forward.
- 2<sup>nd</sup> rocker begins when the forefoot is in contact with the floor and in normal gait, the fulcrum is located at the ankle and moves forward over the metatarsal heads as the tibia moves forward over the stationary foot.
- 3<sup>rd</sup> rocker begins with the fulcrum located at the metatarsal heads and the tibia is no longer moving forward in relation to the foot and in normal gait, the heel lifts from the floor.

## **Temporal-Spatial Parameters**

Temporal-spatial data provide information relating to time and distance. Common temporal-spatial parameters measured were:

- Velocity: speed taken over a gait cycle. Recorded in this thesis as metres per second (m/s).
- Cadence: number of steps taken per minute (steps/min).
- Stride length: as for a single gait cycle. Stride length is reported in metres in this thesis.

• Step length: Distance between the same point on each foot during double limb support (Sutherland 1984). Again, length of the step is usually reported in metres.

## Kinematic Data

Kinematics describe the movement of joints and body segments- linear and angular displacement, velocity and accelerations. Kinematic data do not reflect the causes of this movement.

## Kinetics

Kinetics describe the forces that cause movement. Moments and powers are kinetic data.

## Moments

Moments are the product of the force applied and the perpendicular distance of the force from the fulcrum about which the force is acting. Moments are sometimes referred to as torque in the literature (Davis 2003). Moments are usually expressed in Newton-metres and divided by the subject's body weight (Nm/kg) to normalise the measurement. Internal joint moments are produced by the body in response to the application of external loads that have produced external joint moments (Davis 2003).

- Internal moments are produced by a combination of forces from muscle, ligaments and joint capsule.
- External moments are produced by a combination of ground reaction forces, segmental weight and inertia.

In this thesis internal moments have been reported.

## Power

Power is the product of the net joint moment and angular velocity. It is usually expressed as Watts per kilogram (W/kg).

## **Ground Reaction Force**

The ground reaction force (GRF) is the external force exerted by the supporting surface on the body and is applied through the centre of pressure (Figure 4.11).

The GRF is calculated from data from the force plates and is composed of three components: the vertical force, the fore/ aft shear and the medial/ lateral shear (Davis 2004, Sutherland et al 1988).

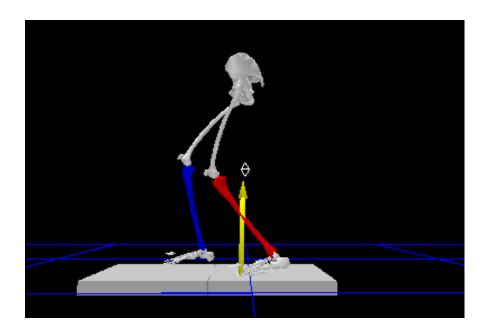


Figure 4.11 Ground reaction force (seen as line extending from the ground upwards) exerted on the right lower limb in early stance.

# Plantarflexion Knee Extension Couple

Through midstance, the soleus (plantarflexor) slows the forward movement of the tibia over the foot and the GRF moves forward onto the forefoot, which lengthens the lever on which the GRF is acting upon and brings it anterior to the knee joint, so creating an external extension moment at the knee (Gage 2004a). This is known as the plantarflexion knee extension couple (Figure 4.12).

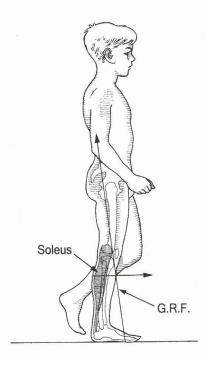


Figure 4.12 The plantarflexion knee extension couple. Reproduced from Gage(2004a, p. 45).

All terminology was developed in reference to what happens in normal gait. When this terminology is applied to gait that deviates from normal, some of the terminology becomes ambiguous particularly in respect to the ankle rockers. For example, if the child does not have heel contact at any time in stance, then  $2^{nd}$  rocker, which is the rotation primarily about the ankle, does not occur. However this terminology remains widely used and accepted in the gait analysis community and provides a common language for describing gait.

## Orientation of a 3DGA Graph

Graphs with 3DGA data are presented in a typical manner (Figure 4.13). The X axis denotes 100% of a gait cycle and along this axis there is a vertical line that depicts the toe off of the limb in question. Therefore the area of the graph to the left of the toe off line represents stance phase and to the right of the toe off line, swing phase. The Y axis represents degrees of movement or moments or powers at a particular joint. The demarcations on the Y axis will change according to the joint and variable being measured, so it is important to check this on each graph before assessing the traces on the graph. There is usually a

horizontal line on the Y axis, denoting zero degrees for the particular joint being assessed.

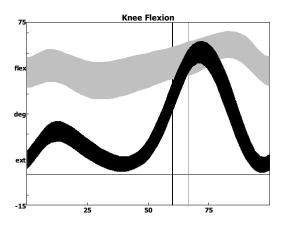


Figure 4.13 A typical sagittal plane kinematic graph of the knee. Data shown represent one standard deviation about the mean for severe crouch data (shaded grey) and normal data (shaded black).

## 4.9 STATISTICAL ANALYSIS

The method of statistical analysis for each study is described under the specific methods section in the pertinent chapter.

## 4.10 FRAMEWORK FOR PRESENTATION OF RESULTS

In the studies of severe crouch gait and outcome post-SEMLS, the framework of the ICF has been used to present results according to two of the three levels of functioning- body level and individual level, and not societal level. It is recognised that the allocation of particular parameters for outcome evaluation to the different components of the ICF, is dependent on the interpretation of the definitions provided under the components in the ICF model. This may lead to differences in opinions as to where evaluated parameters should be allocated under respective components. In this thesis, changes in *body structure* and *function* have been identified and rated as to how they affect activities, so that

intervention aimed at enhancing the ability to do activities could be evaluated. Body structure has been documented by recording the following physical examination measures: muscle length, joint contracture, bony torsion and spasticity. Body function was recorded using three dimensional gait analysis (3DGA) and temporal-spatial parameters. Activities were recorded through the The studies in this thesis did not assess the effect on FAO and FMS. participation as there were no measurements taken of the subjects' level of participation in everyday activities, e.g. play in the schoolyard, ability to keep up with friends when shopping or going to a movie, involvement in community activities. Environmental factors were also not accounted for in this thesis, as there was no documentation regarding access to assistive devices or mobility aids, or limitations in home, school, or community environment that facilitated or hindered the ability to walk, and participate in everyday activities. Personal factors are not classified in the ICF (Dahl 2002, World Health Organization 2001) and therefore are not documented.

# CHAPTER 5 GAIT PATTERNS IN SPASTIC DIPLEGIA

### **5.1 INTRODUCTION**

Children with spastic diplegia are usually able to walk in the home, and some in the community. The majority have an easily recognised gait disorder which may include sagittal gait deviations such as toe-walking, flexed and stiff knees, flexed hips and an anteriorly tilted pelvis with a lumbar lordosis (Miller et al 1995). Many also have other characteristics such as reduced walking speed, increased energy cost of walking and decreased functional abilities, compared to their peers (Butler et al 1984, Nene et al 1993, Novacheck et al 2000).

Management of the gait disorder in spastic diplegia is usually based on a comprehensive evaluation in the gait analysis laboratory (DeLuca 1991). This typically includes a standardised physical examination, three dimensional kinematics and kinetics, dynamic electromyography and energy studies (Gage et al 1995). The amount of information generated by this type of evaluation is very extensive and can be difficult to prioritise. The philosophical basis for instrumented gait analysis is the premise that children with spastic motor disorders and other gait disturbances are unique and individual. Instrumented gait analysis provides detailed information regarding the precise measurements of the movement disorder and through a process of clinical interpretation this may help the clinician understand the gait deviations and plan appropriate intervention (DeLuca 1991). However experienced clinicians have frequently described gait patterns qualitatively, using a combination of clinical examination and clinical observation of gait (Miller et al 1995, Rang et al 1986, Sutherland and Davids 1993). A number of other authors (O'Byrne et al 1998, O'Malley et al 1997, Wong et al 1983) have taken a different approach and have used cluster analysis techniques to classify gait patterns in cerebral palsy on a purely quantitative basis. Whilst the patterns identified by cluster analysis appear to have statistical validity, none of these classifications have become widely recognised or widely used by clinicians. This may be due to the gait patterns, identified in this way, not being necessarily easy to recognise clinically.

The aim of this study was to combine pattern recognition and quantitative kinematic data to devise a classification of sagittal gait patterns in spastic diplegia. The plan was to develop a template for describing sagittal gait patterns in a clinically useful manner, with relevance to spasticity management, muscle tendon surgery and orthotic management. The study was conducted in three inter-related parts. The first was a cross-sectional study of gait patterns of children with spastic diplegia who presented to the gait laboratory between 1995 and 2001. A longitudinal study was conducted for a subset of patients who had more than one instrumented gait analysis during that period. Then the intra- and inter-observer reliability of the classification was investigated.

#### 5.2 METHODS

#### 5.2.1 Subjects

#### **Cross-sectional Study**

The inclusion and exclusion criteria are shown in Table 5.1. It should be noted that while the inclusion criteria for the 3DGA required it to be conducted prior to any surgical intervention, the exception was calf surgery. Previous calf surgery was not an exclusion factor as such surgery had been used widely in our patient population in the past (Borton et al 2001) and was considered at this time, to be part of the historical progression of gait for children with spastic diplegia at this hospital. Children were included only if the calf surgery had been conducted at least 12 months prior to assessment. Spasticity management using botulinum toxin A injections to the lower limb was acceptable provided this had occurred more than six months prior, as the benefits of the treatment in all likelihood would have diminished in that time (Cosgrove et al 1994b, Eames et al 1999, Koman et al 1993). Radiology of the spine was not undertaken on all subjects as it was a retrospective study but if clinical scoliosis was noted at the gait analysis then previous radiographic assessment was consulted.

 Table 5.1
 Inclusion and exclusion criteria for the cross-sectional study.

Inclusion Criteria	Exclusion Criteria
Spastic diplegia	Other movement disorders: ataxia, dystonia, mixed
4-18 years	Previous orthopaedic surgery, ITB, SDR
Independent ambulation with or without assistive devices	Severe asymmetrical fixed deformity/ scoliosis (Cobb angle >20°)
3DGA prior to any surgical intervention, exception calf surgery	Limb length discrepancy > 2.5 cm
3DGA sagittal kinematics barefoot	Frank hip dislocation

## Longitudinal Study

For the longitudinal study, inclusion criteria were almost the same as for the cross-sectional study except that subjects were required to have had two gait analyses at least 12 months apart with no spasticity management or orthopaedic surgery in the interim. Subjects with previous calf surgery were excluded. These two changes to the criteria for the longitudinal study meant that some subjects from the cross-sectional study were ineligible for this study.

#### 5.2.2 Procedure

Subjects in each study participated in a physical examination, video recording of the gait pattern and a 3DGA as detailed in Chapter 4 Methods.

An experienced paediatric orthopaedic surgeon and senior physiotherapist classified the gait pattern for each subject. To classify the gait pattern, for each subject there was:

- Video observation of the gait pattern in the sagittal plane.
- Comparison of the sagittal plane pelvic, hip, knee and ankle kinematic in mid to late stance with the kinematic data from our gait laboratory's normal database, using the one standard deviation band (Figure 5.1).

• Classification of gait patterns was based first on the position of the ankle, followed by the knee, hip and pelvis in late stance.

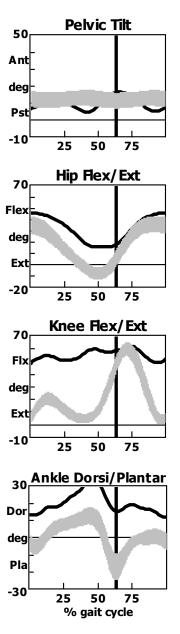


Figure 5.1 Sagittal plane kinematic traces of a subject with spastic diplegia (black trace) for comparison with normal traces ± one standard deviation band (grey band).

The sagittal plane kinematic permutations that were possible were:

- Pelvis: anterior tilt, normal range, posterior tilt
- Hip: flexed, normal range
- Knee: flexed, normal range, recurvatum
- Ankle: equinus, normal range, calcaneus

Therefore a joint was considered in:

- The normal range if the kinematic trace was within the one standard deviation band in late stance.
- Anterior pelvic tilt, flexion at the hip or knee or calcaneus at the ankle if the respective kinematic trace was above the one standard deviation band in late stance.
- Posterior pelvic tilt, knee recurvatum or equinus at the ankle ankle if the respective kinematic trace was below the one standard deviation band in late stance.

The knee kinematic was also classified as stiff if the excursion of the knee throughout the gait cycle was less than 30°, which is 55% of the average range for knee excursion for our laboratory's normal database.

For each subject, age at time of gait analysis, velocity of the representative kinematic trial, previous calf surgery and the presence of stiff knee were also recorded.

### 5.2.3 Reliability Study

The reliability study was undertaken on two separate days, two weeks apart. The sagittal plane kinematic data and video clips from 10 subjects randomly chosen from each gait pattern in the cross-sectional study, were presented to the raters. The raters were three paediatric orthopaedic surgeons and three physiotherapists experienced in 3DGA.

On each occasion, the raters were provided with written definitions and the illustration of the posture of the pelvis, hip, knee and ankle in each gait pattern. They were shown the kinematic traces and video clip for each of the 10 subjects. They were asked to identify the gait pattern that depicted the subject's gait as seen in the video clip and on kinematic data in mid to late stance, comparing against the one standard deviation band for the laboratory's reference database (transparency overlay of this was provided). The data were presented in a random order on both occasions.

#### 5.2.4 Statistical Analysis

Mean age was compared between subjects grouped by the combinations of gait pattern true equinus and jump, and apparent equinus and crouch using a twosample t-test. Velocity (average over right and left limb) was compared between the four gait patterns- true equinus, jump, apparent equinus and crouch, using analysis of variance (ANOVA). The binary characteristics, stiff knee and calf surgery were compared between the four gait patterns using logistic regression. Logistic regression can be used to predict dichotomous clinical outcomes (Portney and Watkins 1993). It was therefore used for statistical analysis as there were two dichotomous dependent variables (stiff knee and calf surgery) and the independent variables of interest were the four gait patterns.

Weighted kappa statistics were used to summarise intra-rater and inter-rater reliability with quadratic weights for the four ordered patterns, true equinus, jump, apparent equinus and crouch. Asymmetric gait was considered to be one category discordant from all four basic patterns. The weighted Kappa statistic looks at the level of observed agreement against the likelihood that the agreement has occurred by chance. The weighting of the kappa statistic allows recognition that the disparity between ratings may not be equal (Portney and Watkins 1993). For example, if the gait pattern of true equinus were rated as jump gait this would not be as serious a discrepancy as if it were rated as crouch gait. The weighted kappa statistic assigns a smaller weight to a disagreement in rating of true equinus as jump gait but a larger one to that of true equinus being rated as crouch gait. As the four ordered patterns are categorical variables, an intraclass correlation coefficient (ICC) could have been used to assess repeatability but the ICC assumes that intervals between measurements are equal and so the ICC was not appropriate to assess reliability in this case (Portney and Watkins 1993).

All statistical analysis was undertaken using the Stata 7 (StataCorp) software package (StataCorp. 2001).

## **5.3 RESULTS**

## 5.3.1 Cross-sectional Study

One hundred and eighty-seven subjects fulfilled the study criteria. Six different combinations of permutations in late stance phase were identified and were subsequently classified as the following gait patterns (Table 5.2 and Figure 5.2):

## • Mild Gait

The ankle, knee, hip and pelvis were all within normal range in mid to late stance in the sagittal plane. The pattern was rarely normal though in the other planes.

## • Group I, True Equinus

The ankle was in equinus and the knee extended within normal range or went into mild recurvatum. The hip extended fully and the pelvis was within the normal range or tilted anteriorly.

## • Group II, Jump Gait

The ankle was in equinus, particularly in late stance. The knee and hip were excessively flexed in early stance and then extended to a variable degree in late stance, but never reaching full extension. The pelvis was either within the normal range or tilted anteriorly.

## • Group III, Apparent Equinus

The ankle was within the normal range but the knee and hip were excessively flexed throughout stance. The pelvis was normal or tilted anteriorly.

### • Group IV, Crouch Gait

The ankle was excessively dorsiflexed throughout stance and the knee and hip were excessively flexed. The pelvis was in the normal range or tilted anteriorly or posteriorly.

### • Group V, Asymmetric Gait

The gait pattern was asymmetric to the degree that the subject's right and left lower limbs were classified as different groups e.g. right lower limb Group III, apparent equinus and the left lower limb Group II, jump gait.

Figure 5.1 (p.136) illustrates a comparison of the kinematics of a child with spastic diplegia with the reference data and the resulting classification of crouch gait.

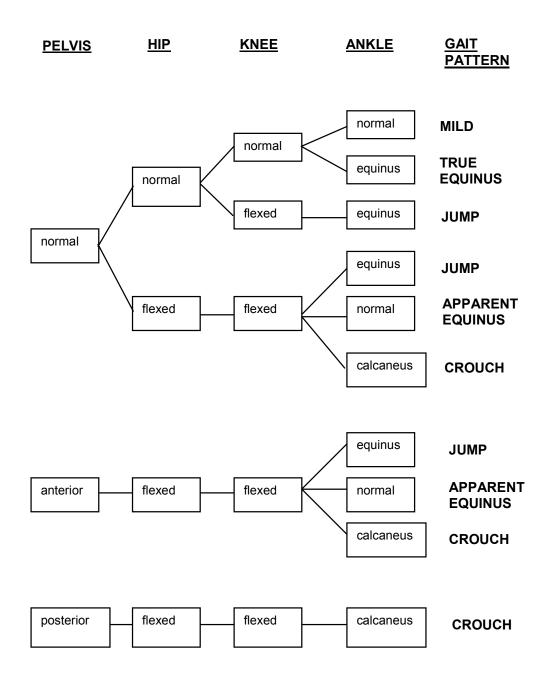


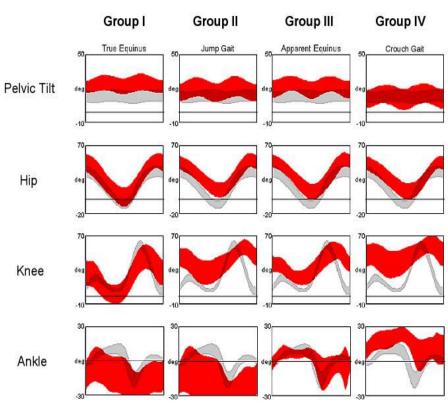
Figure 5.2 Algorithm of combinations of permutations and subsequent classification of gait patterns.

Sagittal Kinematics					
Gait Pattern	No	Pelvis Tilt	Hip Fx/Ext	Knee Fx/Ext	Ankle Df/Pf
Mild Gait	13	normal	normal	normal	normal
Group I True Equinus	47	normal anterior	normal	normal recurvatum	equinus
Group II Jump Gait	38	normal anterior	normal flexed	flexed	equinus
Group III Apparent Equinus	31	normal anterior	flexed	flexed	normal
Group IV Crouch Gait	28	anterior normal posterior	flexed	flexed	calcaneus
Group V Asymmetric Gait	30	com	bination of any tw	wo of the above p	atterns
Total	187				

Table 5.2 Gait pattern definitions: spastic diplegia. Definition of the sagittal plane posture at each level in late stance.

The number of subjects who were classified as having a particular gait pattern is shown in Table 5.2. All of the 187 subjects were able to have their gait classified according to the gait patterns that were labelled as above, indicating that the six gait patterns that had been identified were inclusive of the sagittal plane gait deviations seen in a large cohort of children with spastic diplegia. The gait patterns of true equinus, jump gait, apparent equinus, and crouch gait were considered to be the four basic sagittal plane patterns and because terminology has varied in different reports (Miller et al 1995, Rang et al 1986, Sutherland and Davids 1993), it was decided also to describe them as Groups I to IV. The asymmetric pattern was not considered to be a basic pattern as it represented a combination of any two of the four basic patterns. The mild pattern was also not considered to be a basic pattern. These subjects were usually referred to the gait laboratory for concerns regarding intoeing. A mild group has been previously reported with almost normal kinetic patterns but there were no data detailing sagittal plane kinematic deviations (Lin et al 2000).

The summarised sagittal plane kinematic data for the study population are shown in Figure 5.3. As the procedure for the classification of gait patterns was based first on the position of the ankle, the patterns were ordered according to decreasing equinus from left to right. Across the gait patterns, from true equinus to crouch gait, there were changes across the levels. At the ankle, there was a change from equinus, through plantargrade to calcaneus. At the knee and hip, there was extension, which was followed by increasing flexion. At the pelvis, anterior pelvic tilt reduced with a trend towards a posterior pelvic tilt.



Sagittal Plane Kinematic Data for Gait Patterns: Spastic Diplegia

Figure 5.3 Summarised kinematic data of the four basic gait patterns.

Results of the cross-sectional study for each gait pattern, showing mean age, velocity, and incidence of stiff knees and prior calf surgery are presented in Table 5.3. The youngest subjects walked in the gait patterns of true equinus and jump gait and the oldest walked in apparent equinus and crouch gait. Mean age of children in apparent equinus and crouch patterns was 2.9 (95% CI: 2.1, 3.8) years older than that of children in true equinus and jump gait. Hence we are confident that there is at least a two year difference in mean age between these two combinations of patterns.

The highest incidence of previous calf surgery was in children with crouch gait, then in those with apparent equinus and the least in those with true equinus and jump. The proportion of subjects who had previous calf surgery was not significantly different between true equinus and jump (p = 0.08) but there was a significant difference between apparent equinus and true equinus (p = 0.01), and between crouch gait and true equinus (p<0.001), with apparent equinus and crouch gait having the greater proportion.

The percentage of subjects with stiff knees was greatest in crouch gait, followed by jump and apparent equinus, with zero incidences in true equinus. Mean gait velocity was lower in jump gait than in true equinus (p = 0.001, 95% CI for difference in mean velocities: 0.1, 0.4m/s), lower in apparent equinus than in true equinus (95% CI: 0.0, 0.3m/s) although this could have been a chance finding (p = 0.18), and lower in crouch than in true equinus (p = 0.003, 95% CI: 0.1, 0.4m/s).

Table 5.3	Cross-sectional study of gait patterns: spastic diplegia. Age,	walking
speed, inciden	nce of knee stiffness and prior calf surgery according to gait	pattern.
Mean and one	standard deviation (SD) in parentheses.	

No.	Aae	Velocity		Calf
Subjects	(years)	(m/s)	Stiff Knee	Surgery
47	8 (2.4)	1.0 (0.3)	0%	7%
38	7 (2.1)	0.8 (0.3)	50%	5%
31	10 (2.6)	0.9 (0.4)	39%	29%
28	12 (2.7)	0.8 (0.3)	71%	50%
30	10 (3.1)	0.9 (0.3)	23%	27%
	47 38 31 28	Subjects         (years)           47         8 (2.4)           38         7 (2.1)           31         10 (2.6)           28         12 (2.7)	Subjects         (years)         (m/s)           47         8 (2.4)         1.0 (0.3)           38         7 (2.1)         0.8 (0.3)           31         10 (2.6)         0.9 (0.4)           28         12 (2.7)         0.8 (0.3)	Subjects         (years)         (m/s)         Stiff Knee           47         8 (2.4)         1.0 (0.3)         0%           38         7 (2.1)         0.8 (0.3)         50%           31         10 (2.6)         0.9 (0.4)         39%           28         12 (2.7)         0.8 (0.3)         71%

## 5.3.2 Longitudinal Study

There were 34 subjects (68 limbs) who fulfilled the criteria for the longitudinal study, with a mean time between first and second analyses of 30.2 months (range 13-65). Given that a number of subjects had an asymmetric gait pattern, we have reported results for limbs rather than subjects. Table 5.4 shows the pattern of the limbs at the first gait analysis and then the pattern observed at the second gait analysis. There was no change in gait pattern between gait analyses in 34 limbs; 17 limbs exhibited a shift to the left (towards true equinus) and 17 limbs to the right (towards crouch), suggesting that there was no significant bias in change towards the left or the right.

Table 5.4Longitudinal study of gait patterns: spastic diplegia. There were 34subjects (68 limbs). The gait patterns have been analysed according to *involved limbs*,not by individual patients, therefore Group V asymmetric gait has not been included inthis table. The pattern of each limb at the first and second gait analyses is shown. Nochange in pattern between analyses is shown in the shaded boxes. A change inpattern to the left is in underlined italics and to the right is in normal type.

	1ST	2ND ANALYSIS				
Gait Pattern		Mild Gait	Group I True Equinus	Group II Jump Gait	Group III Apparent Equinus	Group IV Crouch Gait
Mild Gait	5		2	3		
Group I True Equinus	26	<u>2</u>	15	6	3	
Group II Jump Gait	17	<u>2</u>	<u>5</u>	7	3	
Group III Apparent Equinus	12		<u>3</u>	<u>3</u>	6	
Group IV Crouch Gait	8				<u>2</u>	6

# 5.3.3 Reliability Study

Intra-rater reliability ranged from 1.00 to 0.66 (median 0.89) according to weighted kappa statistics. Overall inter-rater reliability across the five patterns for the paediatric orthopaedic surgeons and physiotherapists was 0.74 (95% CI: 0.58, 0.95) on the first occasion and 0.60 (95% CI: 0.36, 0.83) on the second occasion (Table 5.5).

Intra-rater Reliability			
Rater No.	Kappa Statistic (95% CI)		
1	1.00 *		
2	0.77 (0.37, 1)		
3	0.90 (0.67, 1)		
4	0.92 (0.81, 1)		
5	0.88 (0.70, 1)		
6	0.66 (0.26, 0.92)		

Table 5.5Reliability of gait patterns: spastic diplegia.Kappa statistics with 95%confidence intervals (CI) for intra- and inter-rater reliability.

\*A confidence interval could not be calculated because observed agreement was perfect.

Ir	Inter-rater Reliability				
	Time 1	Time2			
Group I True equinus	0.90	0.56			
Group II Jump Gait	0.76	0.31			
Group III Apparent equinus	0.46	0.45			
Group IV Crouch Gait	0.67	1.00			
Group V Asymmetric Gait	0.90	0.76			
Overall (95% Cl)	0.74 (0.58, 0.95)	0.60 (0.36, 0.83)			

## **5.4 DISCUSSION**

## 5.4.1 Cross-sectional Study

Four sagittal plane gait patterns in spastic diplegia have been described, plus two subsidiary groups, which complete the classification. The groups are broad in definition but this limited the number of groups to a clinically relevant number. Previous authors (Miller et al 1995, Rang et al 1986, Sutherland and Davids 1993) have identified some of the patterns, but several different terms and definitions have been used. For this reason, the gait patterns in this study were defined precisely and labelled using descriptive terms combined with roman numerals. The patterns were labelled to reflect the distinct differences between the patterns and thereby be clinically intuitive. The gait patterns have been ordered, based on the clinical observation of changes in biomechanics between the patterns. From left to right across the patterns, there is decreasing equinus, increasing proximal involvement and a change in the direction of the ground reaction force from being anterior to posterior to the knee (Figures 5.3 and 5.4).

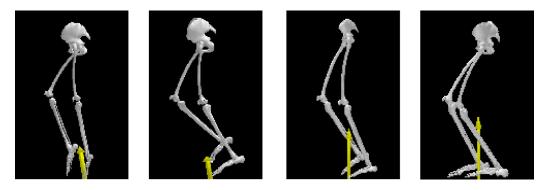


Figure 5.4 Ground reaction vector of gait patterns: spastic diplegia. From left to right, an example of a subject in true equinus, jump gait, apparent equinus, and crouch gait (Group I-IV). The change in the direction of the ground reaction vector from anterior to the knee in true equinus (Group I) progressing to posterior to the knee in crouch gait (Group IV) is seen. N.B. Group V asymmetric gait pattern has been excluded because the ground reaction vector is the same as for the four basic groups illustrated here.

The gait pattern of true equinus in this study encompasses the recurvatum knee (Sutherland and Davids 1993) and the equinus position and the recurvatum plus equinus position (Miller et al 1995) as the clinical management for these is

based on the same principles. It was not considered necessary to create a separate category for recurvatum knee because it was superfluous. Recurvatum in the longitudinal study was rare, mild and temporary. Recurvatum gait in spastic diplegia is probably iatrogenic, usually caused by a contracted or spastic calf and over lengthened hamstrings (Miller et al 1995). In the study conducted by Sutherland and Davids (1993), prior hamstring surgery was not excluded (Sutherland 2002), which would explain the recurvatum knee pattern identified.

In true equinus, the equinus position of the ankle is real.

Jump gait is so called as it involves a decrease in flexion at the hip and knee from initial contact to late stance, though the amount of this decrease can vary. This is coupled with equinus in late stance, which gives the appearance that the subject is jumping up and down. Again the equinus is real.

In apparent equinus, the heel is not in contact with the ground because of flexion contractures at the knee and hip, but the ankle is not in equinus. The equinus is therefore apparent, not real. It is flexion of the hips and knees that gives the illusion of equinus at the ankle. The sagittal kinematic at the ankle is in the normal range in stance though this does not necessarily mean that the ankle-foot complex is structurally normal. A contracted gastrocnemius muscle can pull the hindfoot into equinovalgus and lead to a compensatory dorsiflexion at the midtarsal joint (Miller et al 1995, Rang et al 1986), which can lead to a sagittal ankle kinematic within the normal range. This is important as orthopaedic management in apparent equinus gait that is directed only to the tendo achilles lengthening can result in crouch gait (Borton et al 2001).

Flexed knee gait is not the same as crouch gait unless the ankle kinematic is in the calcaneus range. In this classification, a flexed knee gait encompasses jump gait, apparent equinus or crouch gait, depending on the position of the ankle. This is relevant to research into crouch gait, where in order to ensure homogeneous cohorts both knee and ankle sagittal plane kinematics need to be reported. Without this operational definition, the research could encompass any of the three patterns or a combination of all the three.

The mean age of the subjects walking in true equinus and jump gait was less than that of those in apparent equinus and crouch gait. True equinus and jump gait may be the most common patterns in younger children at the beginning of independent walking. The age gradient from true equinus and jump gait to apparent equinus and crouch gait suggests that these patterns may reflect the natural history of gait in spastic diplegia, as was suggested by Rab (1992a). In the longitudinal study by Yokochi (2001), the initial walking patterns showed flexed knees with plantarflexed ankles or extended knees that were always associated with plantarflexed ankles i.e. our patterns of true equinus and jump gait. The final walking pattern was of flexed knees in all cases and either plantarflexed or dorsiflexed ankles. However in our study, the increased incidence of previous calf surgery in the apparent equinus and crouch groups implicates isolated calf surgery as a major predisposing cause. Moreover apparent equinus and crouch gait are part of the natural history for some children as shown by the study by Yokochi (2001). A recent cross-sectional study that examined the prevalence of gait abnormalities in all types of spastic cerebral palsy, found that with increasing age there was increased likelihood of rotational malalignment between the femur and tibia and calcaneus for all children with cerebral palsy, as well as increased knee flexion for spastic diplegia (Wren et al 2005a). These gait abnormalities if grouped together would be classified as crouch gait according to our classification of sagittal gait patterns in spastic diplegia. Therefore the data from the study by Wren (2005a) support the notion that crouch gait is a gait pattern that develops in the older child. However no previous studies have identified an age gradient between actual gait patterns.

The finding that the two gait patterns with the slowest velocities also had the two largest percentages of stiff knee gait seems to implicate the presence of a stiff knee as a cause of decreased speed in walking. However the converse could be true as it has been shown that normally developing children who were asked to walk slowly had a decrease in the peak amplitude of knee flexion in swing (van der Linden et al 2002).

#### **Biomechanical Continuum**

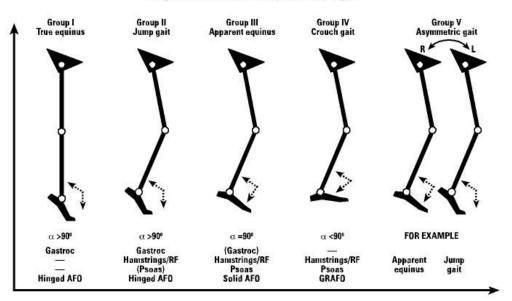
By ordering the gait patterns from true equinus, jump gait, apparent equinus to crouch gait, and by combining the pelvic, hip, knee and ankle sagittal plane kinematics in the pattern definition (Figure 5.3), these gait patterns appear to represent a change from mainly distal to mainly proximal muscle involvement. The gait pattern is the result of sagittal plane muscle imbalance, not at a single joint but between anatomical levels (Silver et al 1985). In true equinus, the calf is spastic or contracted and is dominant. In crouch gait, the calf is long and weak and the hamstrings and iliopsoas are spastic or contracted, and dominant. The issue of muscle weakness is obviously very important in determining the final gait pattern (Damiano et al 2002). Children may fall into crouch gait because of weakness as well as being pulled down into crouch because of contracted hamstrings and hip flexors. In most children, weakness and contracture, both contribute to the gait pattern.

There also appears to be a change in the integrity of the plantarflexion knee extension couple from being overactive in true equinus to being incompetent in crouch gait when the sagittal plane kinematics are ordered as shown in Figure 5.3. This corresponds with the change in direction of the ground reaction force from being anterior to the knee in true equinus to posterior to the knee in crouch gait. This biomechanical continuum between gait patterns has not been documented prior to this study.

### **Management Strategies**

These changes across the gait patterns in level of muscle involvement, competence of the plantarflexion knee extension couple and direction of the ground reaction force can be used to aid clinical decision making in the management of the gait disorder. These gait patterns can be linked to management strategies for spasticity, contracture and weakness such as botulinum toxin injections, orthopaedic surgery and orthotic prescription (Figure 5.5). It is recognised that the management of gait abnormalities in spastic diplegia is complex and cannot be reduced to a simple recipe. However the following suggested broad guidelines are merely a starting point that

clinicians may find helpful to consider when initially planning management of a gait pattern. There will always be exceptions.



#### Sagittal Gait Patterns: Spastic Diplegia

Figure 5.5 Diagram of each sagittal gait pattern, with dominant muscle groups identified for spasticity and/ or contracture management and appropriate orthotic prescription. Group V is a combination of Groups I to IV, with a different group in the right lower limb compared to the left lower limb. In this example, the right lower limb is Group III apparent equinus and the left lower limb is Group II jump gait.

In the mild pattern, the kinematic data of the transverse plane are abnormal. The level or levels causing the gait deviation need to be ascertained and then addressed by surgery to the bony levers e.g. derotation femoral osteotomies, supramalleolar osteotomies and/or foot stabilisation procedures (Gage et al 1995).

In Group I (true equinus), calf spasticity and/ or contracture is dominant over that of the hamstrings and psoas. This gait pattern is usually seen in the younger child, so spasticity, rather than contracture is usually the influencing factor and botulinum toxin injections to the calf muscle may be appropriate (Boyd and Graham 1997, Cosgrove et al 1994b, Graham et al 2000). Older children, walking in true equinus will often have some spasticity and some degree of

contracture at the hip and knee level that is not obvious. Although lengthening of the contracted gastrocsoleus may be the most obvious prescription, careful evaluation is required so that surgical intervention addresses all involved levels (Gage 1991).

In Group II (jump gait), all three levels are involved. In the younger child with spasticity, where minimal or no contracture is present, selective dorsal rhizotomy (SDR) may be considered if the child fulfils the appropriate pre-requisites for this procedure (Gormley et al 2001b). The role of multilevel botulinum toxin injections to the calf, hamstrings and psoas, has been suggested and remains under evaluation (Molenaers et al 2001, Molenaers et al 1999a, Molenaers et al 1999b). Again where contracture is established in jump gait, then SEMLS consisting of surgical lengthening of the calf, hamstrings and psoas may be considered (Gage 1991, Molenaers et al 2001).

Orthotic requirements in true equinus and jump gait are similar since the spasticity or contracture of the calf restricts or curtails second rocker at the ankle. Consequently the ground reaction force is directed anteriorly to the knee, leading to an overactive plantarflexion knee extension couple. A hinged ankle foot orthosis restricts excessive movement into plantarflexion and so aids movement into dorsiflexion during second rocker, thereby realigning the ground reaction force and normalising the plantarflexion knee extension couple (Butler and Nene 1991).

The hamstrings and psoas are the dominant influences in Group III (apparent equinus). As this pattern tends to be associated with the older child (as seen in the cross-sectional study), contracture management is relevant and soft tissue surgery to the hamstrings and psoas needs consideration. Bony surgery may be required to correct lever arm dysfunction in the transverse plane. The gastrocnemius may or may not be contracted and needs careful evaluation but the soleus is not contracted in apparent equinus. The second rocker may be poorly controlled from mid stance onwards, and so the ground reaction force is no longer anterior to the knee. A solid ankle foot orthosis can assist the plantargrade alignment of the tibia with the foot during second rocker and

restore the anterior location of the ground reaction force (Butler and Nene 1991).

In Group IV (crouch gait), again the involvement of the hamstrings and psoas coupled with lever arm dysfunction, directs the surgical intervention undertaken. The ankle is in calcaneus and no surgical intervention to the calf is required unless occult shortening of the gastrocnemius is exposed by foot stabilisation. Second rocker is excessive into the dorsiflexion range with the ground reaction force directed posterior to the knee. The plantarflexion knee extension couple is incompetent. A ground reaction ankle foot orthosis holds the ankle in minimal plantarflexion so that the orthosis directs the ground reaction force anterior to the knee throughout stance so that knee extension is gained and the plantarflexion knee extension couple is restored (Harrington et al 1984).

The position of the ankle joint in apparent equinus gait and crouch gait is important for a number of reasons. The ankle position provides differentiation of these two gait patterns from jump gait, plus from each other. Also the declining competence of the plantarflexion knee extension couple is related to the position of the ankle in these two sagittal gait patterns as already discussed in the section *Biomechanical Continuum* on p151. Therefore the ankle positions in apparent equinus gait and crouch gait have an influence on the principles of management for each of the gait patterns in which maintenance (in the case of apparent equinus gait) or restoration (crouch gait) of the competence of the plantarflexion knee extension couple is of importance.

The presence of stiff knees in three of the four gait patterns suggests that this is not a separate sagittal gait pattern but is a specific knee pattern. Lin and colleagues (Lin et al 2000) did not identify a stiff knee group on kinematic data, when they classified the common abnormal kinetic patterns of the knee in spastic diplegic gait. The definition of stiff knee by Sutherland and Davids (1993), identified variable knee position in stance, excessive extension in swing and limited dynamic range of movement. Knee kinematics showed flexion less than 45° with a delay in peak flexion in swing phase. Our definition of a stiff knee concentrates on the reduced excursion (less than 30°) throughout the gait cycle. A knee excursion of 30° is 55% of the knee excursion recorded in our laboratory's reference database. Management of a stiff knee may involve rectus femoris transfer (Gage et al 1987, Õunpuu et al 1993a, Õunpuu et al 1993b, Perry 1987, Sutherland et al 1990), but caution is advised if the quadriceps are weak and the gait pattern characterised by excessive knee flexion.

#### 5.4.2 Longitudinal Study

In the longitudinal study, the gait pattern was stable for 50% of subjects, but for the other subjects there was a change in gait pattern, some to the left (towards true equinus) and others to the right (towards crouch gait). Yokochi (2001) demonstrated similar findings with some subjects who had an initial walking pattern of flexed knees and dorsiflexed ankles (apparent equinus or crouch gait in our gait patterns), having a final pattern of flexed knees and plantarflexed ankles (our jump gait) and other subjects changing from flexed knees and plantarflexed ankles to flexed knees and dorsiflexed ankles. In the study by Gough et al (2004) the group of subjects with spastic diplegia who did not undergo SEMLS showed the following changes between gait analyses: increased hip and knee flexion but variation in ankle posture with some becoming more plantarflexed (our gait pattern of jump gait) and others more dorsiflexed (our gait patterns of apparent equinus or crouch gait). This change was noted over a range of time intervals between 11 to 23 months.

The direction of change from one gait pattern to another is probably influenced by which muscle group develops contracture and becomes dominant with time (Yokochi 2001). This dominance will be related to the degree of spasticity, contracture and weakness present in muscle groups (Gough et al 2004), at each anatomical level. The change in gait pattern may occur over a short or long period of time. What precipitates the change in gait pattern or what the predisposing factors are that contribute to the change, can only be speculated on.

During a growth spurt a child can gain increased height over a short period of time and spastic muscles may become contracted, as their growth is not in unison with that of the bones (Cosgrove and Graham 1994a). In the presence of

muscle contracture, with adequate muscle strength and ligamentous stability at the joints, this increased muscle tightness may translate to changes in alignment in the sagittal plane, such as plantarflexion at the ankle with increased calf spasticity or contracture. However contracture in the presence of muscle weakness and ligamentous laxity at the joints, may lead to lever arm dysfunction as rotational deformities appear and influence the sagittal and transverse planes. An increase in calf spasticity or contracture coupled with muscle weakness and joint laxity may see the contracted calf producing hindfoot valgus, dorsiflexion at the midtarsal joint, pronation of the midfoot and abduction of the forefoot (Bleck 1987). This rotational malalignment will show as increased dorsiflexion in the sagittal plane. Consequently a child who is walking in apparent equinus gait may change to jump or crouch gait during a growth spurt depending on the degree of spasticity, contracture, muscle weakness and ligamentous joint laxity that is present at the time (Mazur et al 1992).

When there is an increase in weight, weak muscles and joint capsules with ligamentous laxity may be at risk of being passively lengthened due to the associated increase in forces across the joints from the weight gain. This scenario may lead to a long over stretched calf and probable malalignment of the foot, leading to changes in the ankle kinematic in the sagittal and transverse plane.

It has been stated that "as children grow their strength does not keep pace with their mass" (Gage 2004b, p. 46). This is referred to as the "Law of Magnitude" by Gage (2004b, p. 46) and is said to be most influential at the time of the adolescent growth spurt. Hence muscles that were able to support the body in the upright position may become disadvantaged due to growth and no longer be able to support the body in extension. This may be one of the reasons why the ankle sometimes becomes more dorsiflexed with an increase in height and weight during the pubertal growth spurt.

A spastic or contracted calf muscle has been recognised as being associated with other foot deformities such as valgus or varus (Bleck 1987, Mazur et al 1992).

O'Connell et al (1998) found that the dominant foot deformity in children with spastic diplegia was equinovalgus (42%), followed by calcaneus (17%) and equinus (16%). The subjects in that study had had no prior surgical intervention and so the development of foot deformity was part of the natural history. An association was found between the level of mobility (limited and fully mobile) and primary foot deformity but the authors did not analyse foot deformity in relation to the age of the subject or over time. Bennet et al (1982) studied varus and valgus of the hindfoot in cerebral palsy with electromyography and concluded that decreased tibialis posterior activity played an important role in the development of valgus.

The change in classification of 17 limbs towards a gait pattern with a more equinus ankle, and 17 limbs towards a gait pattern with a more calaneus ankle, could also be attributed to some random error associated with the classification of subjects to the different gait patterns. Random errors are unpredictable and involve chance (Portney and Watkins 1993). However the gait patterns for each time period, were classified on the same day, by the same physiotherapist (the author) and senior surgeon using the same criteria for allocation to a gait pattern. Intra- and inter-rater reliability were established on two separate occasions two weeks apart. Therefore classification of gait patterns on the same day could be expected to show similar or higher reliability as there was no time lapse thus suggesting that the results from the longitudinal study may be a true representation of change in gait patterns over time, with minimum random error.

### 5.4.3 Reliability Study

Reliability of the gait classification has been established by the weighted Kappa statistic for both intra- and inter-rater repeatability, which showed moderate to substantial agreement (Portney and Watkins 1993). However the use of quadratic weights in the Kappa statistic may have led to an overestimate of agreement. The use of quadratic weights meant that a score of 0.89 was allotted if the gait pattern was misclassified by one place, 0.56 by two places and 0 for three places. It could be argued that a misclassification by one place signifies a disagreement of more than 0.11. However the classification of sagittal gait did not produce discrete patterns; rather these patterns formed a continuum and so

misclassification by one place could be due to a pattern being close to its boundary. Hence the use of quadratic weights could be acceptable.

There was a low level of agreement regarding the classification of apparent equinus gait on both occasions, which may be due to difficulty by the raters in detecting on the video a plantargrade foot posture in late stance when the heel is not in contact with the ground due to knee flexion. However for apparent equinus, the graph of the ankle sagittal plane kinematic would have been within the one standard deviation band of the normal data and should have aided the raters in their scoring. On the second occasion, the inter-rater reliability decreased for the gait pattern of jump gait. The reasons for this are unclear as again the kinematic traces should have aided the raters in the classification. Whether raters remained aware that the gait classification is made according to the sagittal traces in late stance and not over the whole stance phase, is unknown but written instructions specified this condition in assigning to a classification. In the future, increased emphasis should be placed on the period of stance on which the classification of gait is made, to prevent occurrence of this potential error. The gait patterns of true equinus, crouch gait and asymmetric gait did not show similar problems and demonstrated substantial inter-rater agreement.

The results from the reliability study suggest that with sagittal plane video and kinematic traces, clinicians can classify the gait pattern of a child with spastic diplegia and be reasonably confident that a colleague would classify accordingly.

### 5.4.4 Limitations of the Study

### Continuity of Sagittal Plane Kinematic Data

A criticism of this classification of sagittal gait patterns is that there is not a discrete cut-off point between one posture and the next at each anatomical level as the one standard deviation band of the reference data in mid to late stance serves as the demarcation for classification at each anatomical level. However the gait pattern was classified according to the combination of postures at the four anatomical levels. A consequence of this is that a subject with a pattern of jump gait may have a flexed knee that is more flexed than that of a subject with

a pattern of crouch gait. The distinction between gait patterns for these two subjects is made based on the posture of the ankle in mid to late stance. The important point is that the two gait patterns whilst having similar features at some levels can still be differentiated according to posture at another level, thus leading to a different classification of gait pattern for the two subjects.

### Transverse Plane Deviations

The interaction of gait deviations in the other planes, on the sagittal plane is recognised, for example, hip rotation in the transverse plane and knee flexion in the sagittal plane. However a suitable method to incorporate these interactions into this classification still needs to be devised.

### **Referral Based Sample Versus Population Based Sample**

The subjects did not constitute a population-based sample as the subjects were all children who attended the gait laboratory due to concern regarding their gait. Therefore children with spastic diplegia who did not have concerns regarding their walking ability were not available to study. This suggests that this classification of sagittal gait patterns was predominantly representative of spastic diplegic gait that was troublesome and may not include all the gait patterns that are exhibited by children with spastic diplegia. However the subjects sampled in this study were representative of children who present in the clinical setting seeking the clinician's expertise and assistance. It is the gait patterns of such subjects that the clinician needs to understand and manage. Therefore this sample of children characterised those who are most relevant to the clinician in the everyday setting and so the issue of the gait patterns not being representative of a population based sample was not deemed a major limitation in this study.

### Calf Surgery

The inclusion criteria allowed for prior calf surgery in the cross-sectional study and whilst recognising that this surgery was a frequent intervention for most children with spastic diplegia until the last 10 years, the inclusion meant that the natural history of spastic diplegic gait was confounded by this. Calf lengthening is now recognised as a probable causative factor in the development of crouch gait, and therefore never performed in isolation in our centre. It would be informative to repeat the study excluding subjects with isolated calf lengthening to investigate the natural history of gait in spastic diplegia, in particular noting variation in incidence of crouch gait particularly with respect to age.

### Assistive Devices

One of the limitations of this study is that it does not take into account the effect of the use of assistive devices on the gait pattern. However within each of the gait patterns there were some subjects using assistive devices, so their use did not preclude inclusion in any of the patterns or dictate classification to any particular pattern. Stratification of the data according to GMFCS level would have provided this information. The GMFCS was first published in 1997 and some of the subjects in this study had data collected prior to this time.

# Retrospective Nature of Study

This was a retrospective study and therefore limited by the data that were or were not collected at the time. If strength, ligamentous laxity, transverse plane alignment of the femur and foot and foot posture had been recorded in the longitudinal study, it may be ascertained whether weak muscles, increased ligamentous laxity, lever arm dysfunction and plano abducto valgus foot posture are risk factors for progression to a gait pattern that moves from an equinus ankle posture to apparent equinus or crouch gait.

### 5.5 SUMMARY

This gait classification is unique for many reasons. It considers the sagittal plane as a whole without dependence on the position of any one joint. It is derived from both pattern recognition and objective kinematic data, which combines qualitative and quantitative data. The ordered gait patterns are seen to form a continuum based on biomechanical principles, as there is a gradual failure of the competence of the plantarflexion knee extension couple and from true equinus through to crouch gait, there is a change from calf dominance to hamstring/ iliopsoas dominance. There appears to be an age gradient suggesting that these patterns may be part of the natural history of gait in spastic diplegia. The patterns identified have relevance to the management of spasticity and contracture and provide an intuitive tool for teaching and communication between gait laboratory staff and referring clinicians.

### **5.6 CONCLUSION**

Currently research investigates surgical techniques, orthotic prescription, muscle length simulations in relation to the child with spastic diplegia without regard to their gait patterns, apart from the use of the nebulous terms *equinus* or *crouch*. Given that the level of pathological involvement and biomechanics differs between the gait patterns identified in this study, acceptance and utilisation of this classification of gait patterns will allow future research to be specific in its application and perhaps more definitive answers to research questions will be forthcoming as a result.

This classification was designed for an administrative convenience, like the term *cerebral palsy* and the *GMFCS*. Having defined crouch gait in the context of a biomechanical classification of sagittal plane gait patterns, it was now possible to identify a cohort who walked in crouch gait and investigate further the baseline characteristics of crouch gait and the effect of SEMLS for the correction of this gait pattern.

# CHAPTER 6 FUNCTIONAL AND TECHNICAL PARAMETERS OF SEVERE CROUCH GAIT

### **6.1 INTRODUCTION**

The lack of consistency and precision in the definition of the term crouch gait has been of concern in reviewing past studies that have specifically investigated crouch gait and its treatment. This was discussed in Chapter 2, Section 2.5.4. Hoffinger et al (1993) acknowledged that crouch gait has been "poorly defined biomechanically" (p. 273).

In Chapter 5, a biomechanical definition of crouch gait was proposed. This included a variable pelvic position, increased hip and knee flexion, and increased dorsiflexion at the ankle in late stance phase; compared to the reference range of +/- one standard deviation for our laboratory's database of normal paediatric kinematic and kinetic data. This definition encompasses a spectrum of mild to severe pathology in crouch gait. The mean minimum knee flexion in stance phase for our normal database is 4.9° with a standard deviation of 3.8°. The upper reference range in normally developing subjects for this parameter would be 8.7°. Therefore any value greater than this would fulfil the criterion for the knee to be in crouch provided the hip and ankle values were also greater than the respective upper reference ranges.

The descriptions by Sutherland and Cooper (1978) and Sutherland and Davids (1993) defined the minimum knee flexion in stance as greater than 30° which is considerably more than the 8.7° in our definition of crouch gait. A child with spastic diplegia walking in mild crouch gait (for example with a minimum knee flexion of 10°) will be coping with this gait abnormality differently from a child walking in severe crouch (minimum knee flexion of more than 30°). The pathology will be much more pronounced and obvious in severe crouch gait, and the associated symptoms more disabling for the child. The child with

severe crouch gait is therefore more likely to present to the clinic for management.

As a consequence, it was decided to investigate a cohort of children with spastic diplegia who walked in severe crouch gait. This subgroup of crouch gait was identified according to the following criteria: a minimum knee flexion greater than 30° (Sutherland and Cooper 1978, Sutherland and Davids 1993) and ankle dorsiflexion greater than one standard deviation from our reference data (definition from Chapter 5) in order to obtain a homogeneous group of subjects. The study of a more homogeneous group of subjects would facilitate: 1) characterisation of severe crouch gait according to functional and technical parameters; 2) a comparison between severe crouch and normal gait; and 3) identification of the key parameters that constitute severe crouch. A comprehensive study of severe crouch gait and lead to prevention or at least, effective remediation of this gait pattern in the future.

#### **6.2 METHODS**

#### 6.2.1 Study Design

This was a retrospective cohort study, conducted in a gait analysis laboratory in a children's tertiary level care hospital.

# 6.2.2 Subjects

The subjects were a consecutive sample of children with spastic diplegic cerebral palsy, GMFCS level II-III, walking in severe crouch gait.

### **Inclusion** Criteria

Subjects were included in the study if they demonstrated the following features on barefoot kinematic analysis in the sagittal plane in stance phase:

• Minimum knee flexion of 30° or more. This definition of the knee in crouch gait is from Sutherland and Davids (1993) and is considered the

most complete and precise definition for the knee in severe crouch gait in the literature to date.

- Maximum dorsiflexion of 15° or more. The mean maximum dorsiflexion value in our laboratory's normal data was 14.5°.
- Increased hip flexion and variable pelvis position according to the definition of crouch gait in the classification of sagittal gait patterns previously described in Chapter 5.

Using this new more precise definition would mean that the study could be replicated in the future, and in other gait laboratories.

Other inclusion criteria were:

- Age range between 4 to 18 years of age.
- Ability to walk at least six metres without "hands on" supervision, with or without assistive devices.

### **Exclusion** Criteria

Children were excluded if they had

- Previous intervention by selective dorsal rhizotomy or intrathecal baclofen; or botulinum toxin A injections within 12 months prior to 3DGA.
- Any other major health problems.

### 6.2.3 Procedure

Mobility status and physical examination findings were recorded and 3DGA conducted. The procedures for data collection have all been previously described in Chapter 4 Methods. Comparative data from normally developing subjects were also collected as described in Chapter 4.

### Activities

Mobility status was recorded by using the FMS classification and FAQ as both questionnaires provide information relating to the extent a subject walks in the wider community setting. The FMS details how a subject manages walking in different settings using, if necessary, different assistive devices.

### **Body Structure and Function**

### **Physical Examination**

Physical examination variables considered relevant were fixed flexion deformity of the hip and knee, hamstring length assessed by popliteal angle, gastrocnemius and soleus length assessed by the Silfverskiold test, rotational profile of the hip, orientation of the foot with the femur assessed by the thigh foot angle, the Duncan Ely test for the presence of rectus femoris spasticity, posture of the hindfoot, midfoot and forefoot. Procedures for the assessment of these parameters were described previously in Chapter 4, Section 4.6.

### Temporal-spatial Parameters

Temporal-spatial values considered relevant were normalised velocity and stride length (Hof 1996, Hof and Zijlstra 1997, O'Malley 1996, van der Linden et al 2003a), timing of toe off within the gait cycle, and timing of opposite foot off and opposite foot contact within stance phase. The calculation of normalised velocity and stride length were based on the following equations.

Normalised velocity = 
$$\frac{\text{velocity}}{\sqrt{g \times \text{height}}}$$

Normalised stride length = stride length  $\times \frac{1}{\text{height}}$ 

where, velocity = m/s, stride length = m and g = acceleration due to gravity,  $9.81 \text{m/s}^2$ .

### Kinematic Parameters

Sagittal Plane

The following parameters were measured at the pelvis: maximum and minimum pelvic tilt, mean pelvic tilt, pelvic excursion (Figure 6.1).

Mean pelvic tilt was the average of the data points over 100% of the gait cycle. Pelvic excursion was the difference between the maximum and minimum anterior pelvic tilt over the whole gait cycle.

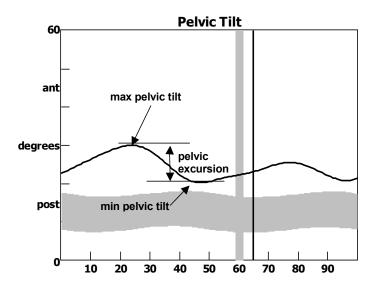
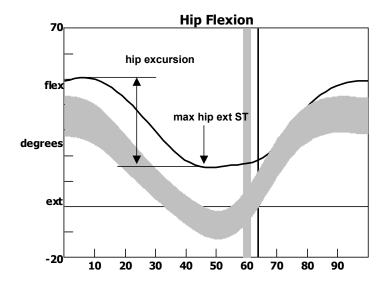


Figure 6.1 The sagittal plane kinematic parameters identified for the pelvis. The grey band denotes the one standard deviation band of the normal data. The black line denotes data from a severe crouch subject.

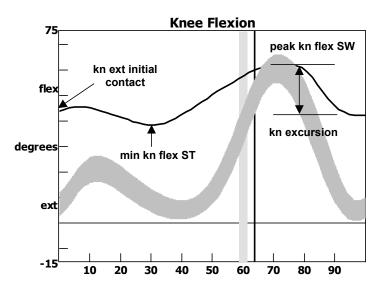


% gait cycle

Figure 6.2 The sagittal plane kinematic parameters identified for the hip. The grey band denotes the one standard deviation band of the normal data. The black line denotes data from a severe crouch subject.

For the hip the following parameters were measured: maximum hip extension in stance, hip excursion (Figure 6.2). Hip excursion was calculated from the maximum hip extension and maximum hip flexion over the whole gait cycle.

The following parameters were measured for the knee: knee extension at initial contact, minimum knee flexion in stance, knee excursion, peak knee flexion in swing (Figure 6.3). Knee flexion at initial contact was taken at 0% of the gait cycle. Knee excursion was calculated as the difference between the minimum knee flexion in stance and the maximum peak knee flexion in swing. This was chosen as the definition as the knee excursion relating to foot clearance obtained during swing, rather than over the whole cycle was the parameter of interest.



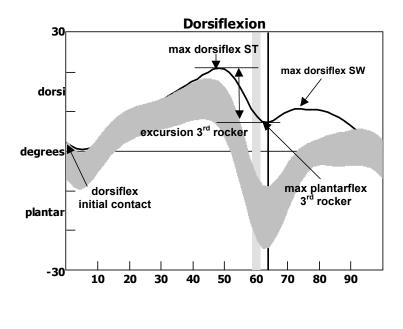
#### % gait cycle

Figure 6.3 The sagittal plane kinematic parameters identified for the knee. The grey band denotes the one standard deviation band of the normal data. The black line denotes data from a severe crouch subject.

At the ankle, the following parameters were measured: dorsiflexion at initial contact, maximum dorsiflexion in stance and swing phase, maximum plantarflexion of third rocker in stance, excursion of third rocker (Figure 6.4).

Maximum plantarflexion of third rocker was taken as the maximum plantarflexion prior to toe off, recognising that maximum plantarflexion may

not be a value in the plantarflexion range but a minimum dorsiflexion value instead if the ankle does not go into plantarflexion. Excursion of third rocker was derived from the difference between stance phase maximum dorsiflexion that was followed by successive decreasing dorsiflexion around the time of toe off, and maximum plantarflexion of third rocker as defined above.



% gait cycle

Figure 6.4 The sagittal plane kinematic parameters identified for the ankle. The grey band denotes the one standard deviation band of the normal data. The black line denotes data from a severe crouch subject.

### <u>Coronal Plane</u>

At the pelvis, excursion in the coronal plane was measured (Figure 6.5). This parameter was derived from the maximum and minimum values of the pelvic obliquity over the whole gait cycle in the coronal plane. Maximum adduction at the hip was measured in stance and swing phases (Figure 6.6).

### Transverse Plane

Excursion in the transverse plane at the pelvis was measured (Figure 6.7). This parameter was derived from the maximum and minimum values of the pelvic rotation over the whole gait cycle in the transverse plane.

Chapter 6

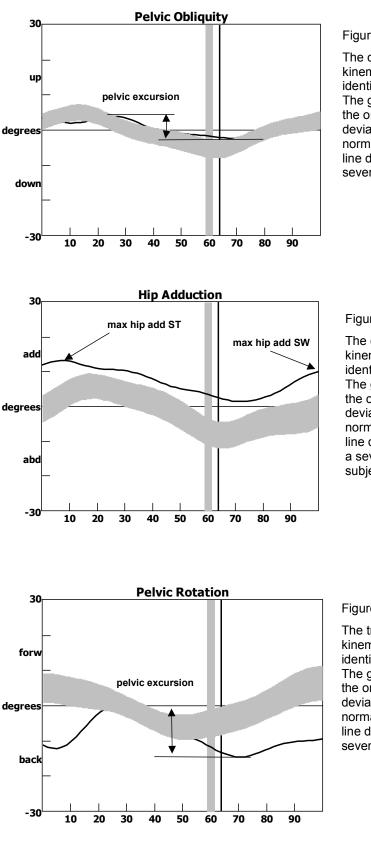


Figure 6.5

The coronal plane kinematic parameters identified for the pelvis. The grey band denotes the one standard deviation band of the normal data. The black line denotes data from a severe crouch subject.

### Figure 6.6

The coronal plane kinematic parameters identified for the hip. The grey band denotes the one standard deviation band of the normal data. The black line denotes data from a severe crouch subject.

% gait cycle

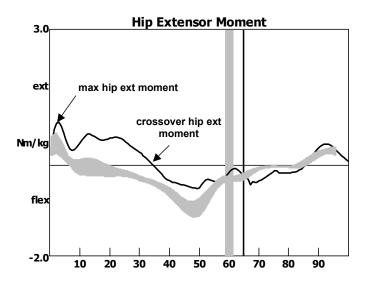
# Figure 6.7

The transverse plane kinematic parameters identified for the pelvis. The grey band denotes the one standard deviation band of the normal data. The black line denotes data from a severe crouch subject.

Mean rotation at the hip and mean foot progression over stance phase, were recorded. These parameters were defined as the mean of all of the values of hip rotation or foot progression prior to toe off. The mean value during stance phase was selected because the alignment of the hip, tibia and foot-ankle complex in the transverse plane has been reported to have an effect on the attainment of knee extension in stance (Fabry et al 1999, Gage 1990, Schwartz and Lakin 2003, Sutherland and Davids 1993). Therefore as crouch gait is part of a flexed knee pattern in the sagittal plane, the assessment of the alignment of these limbs in the transverse plane was relevant.

### Kinetic Parameters

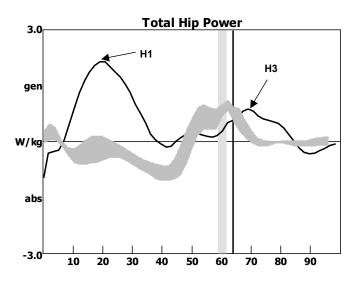
The following parameters were measured at the hip: maximum hip extensor moment in stance (Figure 6.8), maximum hip power in early (H1) and late (H3) stance (Figure 6.9)



% gait cycle

Figure 6.8 The sagittal plane moments identified at the hip. The grey band denotes the one standard deviation band of the normal data. The black line denotes data from a severe crouch subject.

Chapter 6



% gait cycle

Figure 6.9 The powers identified at the hip. The grey band denotes the one standard deviation band of the normal data. The black line denotes data from a severe crouch subject.

At the knee, the following parameters were measured: maximum knee extensor moment in loading response and maximum knee flexor moment in stance phase (Figure 6.10), maximum knee generation power in early stance and absorption power in late stance (Figure 6.11). Maximum ankle power generation (A2) prior to toe off (Figure 6.12) was also measured.

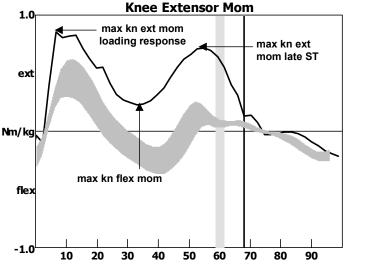


Figure 6.10

The sagittal plane moments identified at the knee. The grey band denotes the one standard deviation band of the normal data. The black line denotes data from a severe crouch subject.

% gait cycle

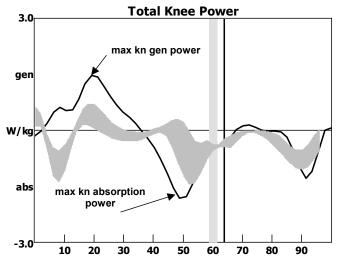


Figure 6.11

The powers identified at the knee. The grey band denotes the one standard deviation band of the normal data. The black line denotes data from a severe crouch subject.



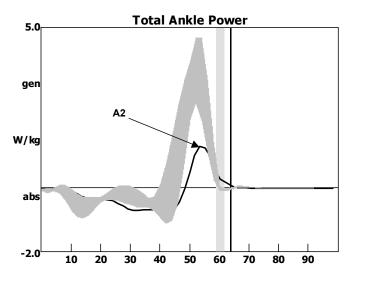


Figure 6.12

The power identified at the ankle. The grey band denotes the one standard deviation band of the normal data. The black line denotes data from a severe crouch subject.

% gait cycle

# **Timing of Parameters**

The timing of selected kinematic and kinetic parameters in relation to percentage of the stance phase or swing phase of the gait cycle was also calculated:

If the parameter occurred during the stance phase, the following calculation was used:

Timing (stance) =  $\frac{\text{timing of parameter in gait cycle}}{\text{duration stance}} \times 100$ 

If the parameter occurred during swing phase, the following calculation was used:

Timing (swing) = 
$$\frac{\text{timing of parameter in gait cycle - duration stance}}{\text{duration swing}} \times 100$$

The timing of maximum hip extension in stance and timing of the hip moment crossing from an extensor to flexor moment were recorded. Timing of the hip moment crossing from an extensor to flexor moment was taken as the percentage of the gait cycle where the first negative value for the hip moment occurs.

At the knee, timing of minimum knee flexion in stance and peak knee flexion in swing, were recorded. Timing of peak plantarflexion in swing and timing of maximum ankle power generation prior to toe off were noted for the ankle.

### Rate of Movement (degrees/sec)

Rate of movement of the knee kinematic between maximum knee extension and peak knee flexion in swing (Figure 6.13) was calculated, as was the rate of movement of the ankle kinematic between maximum dorsiflexion and plantarflexion of third rocker (Figure 6.14).

The rate of movement (degrees/second) was calculated by:

Rate of movement =  $\frac{\text{excursion}}{\text{duration of excursion in gait cycle}}$ 

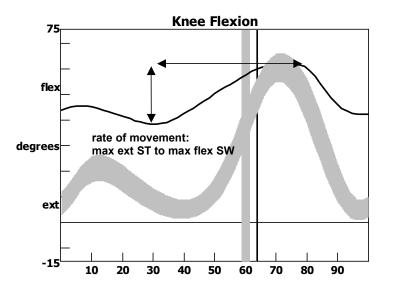


Figure 6.13

Rate of movement of the knee kinematic between maximum knee extension and peak knee flexion in swing. The grey band denotes the one standard deviation band of the normal data. The black line denotes data from a severe crouch subject.



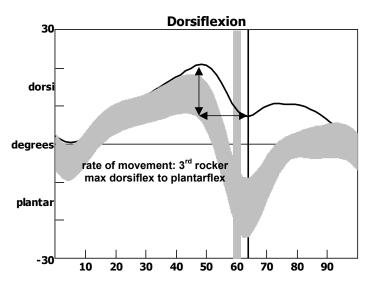


Figure 6.14

The rate of movement of the ankle kinematic between maximum dorsiflexion and plantarflexion of third rocker. The grey band denotes the one standard deviation band of the normal data. The black line denotes data from a severe crouch subject.

### % gait cycle

### 6.2.4 Statistical Analysis

To compare mean outcomes between normally developing subjects and the crouch group at baseline, one year and five years (the latter two time points were involved post-surgical intervention), linear regression models with robust standard errors to allow for the repeated measurements from individual patients over time, were used (Forbes and Wolfe 2001, StataCorp. 2001). Data from both limbs of each of the 14 subjects in both groups were included in the

statistical analysis as the robust standard errors are inflated to take into account any excess correlation in measurements from the two limbs from the same subject (Forbes and Wolfe 2001). P values and 95% confidence intervals of the estimated difference in means were documented. P values of 0.05 were considered to be statistically significant which is standard practice (Norman and Streiner 2000, Portney and Watkins 2000, Sterne and Davey Smith 2001). Parameters that were analysed using this method were temporal-spatial parameters, physical examination and three dimensional gait analysis variables. Statistical analysis was undertaken using the Stata 7 (StataCorp) software package (StataCorp. 2001).

### **6.3 RESULTS**

# 6.3.1 Subjects

### **Demographics**

Fourteen subjects were eligible for inclusion in the severe crouch cohort. Four of the subjects were classified as GMFCS level II and 10 subjects as level III (71.4%). Mean age of the subjects was 12.1 years (range 7.6 - 16.1 years), and there were 10 males (mean age 12.8 years) and four females (mean age 10.3 years) (Table 6.1). Orthoses were normally worn by only four subjects, all of whom required assistive devices to walk.

Crouch Criteria: Kinematics								
Code	age*	GMFCS	min kn flex		max ank	dorsflex		
	yr	level	R	L	R	L		
1	10.0	III	56.0	33.9	48.6	18.1		
2	12.5	III	41.2	49.4	21.5	36.8		
3	13.3	III	41.0	37.0	21.9	25.0		
4	14.1	Ш	50.6	39.1	39.8	35.2		
5	13.7	Ш	54.0	49.1	31.8	22.5		
6	15.7	Ш	54.4	53.2	53.2	27.7		
7	16.1	П	50.4	58.6	34.6	28.5		
8	9.9	Ш	43.4	44.3	47.3	26.8		
9	10.4	Ш	57.8	59.7	23.7	41.8		
10	11.8	П	35.4	42.8 20.4	20.4	37.3		
11	11.1	Ш	40.8	46.1	24.7	26.3		
12	7.6	П	46.1	45.7	27.2	28.1		
13	13.8	П	31.0	31.0	19.4	22.0		
14	8.8	III	38.5	39.4	18.5	16.3		
mean	12.1		45.8	45.0	30.9	28.0		
std dev	2.6		8.3	8.6	11.9	7.5		
min	7.6		31.0	31.0	18.5	16.3		
max	16.1		57.8	59.7	53.2	41.8		

Table 6.1Subject demographics and criteria for severe crouch gait. \*Age at timeof baseline assessment.Mean, standard deviation, and minimum and maximumvalues are shown.

yr GMFCS level min kn flex max ank dorsiflex R, L std dev min max age in years Gross Motor Function Classification System level minimum knee flexion maximum ankle dorsiflexion right, left standard deviation minimum value maximum value

# Surgical History

Two subjects had no prior surgery at baseline. Eleven subjects had previous calf surgery (Table 6.2). Six subjects had isolated calf lengthening, and four subjects had calf and hamstring surgery. Repeat calf lengthening had been undertaken on two occasions for two subjects and one subject had hamstring

lengthening on three occasions. Two subjects, who did not have a history of previous calf lengthening surgery, had been injected with botulinum toxin A into the calves in the past.

	History of Previous Surgery							
Code Type of Surgery								
	Calf	HMS	Other					
1	Bakers							
2	TALx2							
3	Nil							
4	Bakers	MHS						
5	Bakers		foot stabilisation, R hallux valgus s					
6	Gastroc length(mid calf)x2	MHS x3	ADD L, POTB, prox rect release					
7	Bakers	MHS, LHS						
8	TAL							
9	TAL							
10	TAL							
11	TAL	MHS	foot stabilisation					
12			Adductors, Btx HMS & calf x2					
13	TAL							
14			Nil, Btx HMS & calf					

Bakers	Bakers release
TAL	lengthening of the Achilles tendon
gastroc	gastrocnemius
MHS	medial hamstrings lengthening
LHS	lateral hamstrings lengthening
Sx	surgery
ADD L	adductor longus lengthening
prox rect release	proximal rectus release
POTB	psoas over the brim
Btx	botulinum toxin A

# 6.3.2 Activities

### **Functional Mobility Scale**

Over 500 metres, 10 subjects used a wheelchair and all were dependent on assistive devices; over 50 metres, two subjects used a wheelchair and again all required assistive devices; and at 5 metres, none required a wheelchair, 10

required assistive devices and four were independent (Figure 6.15). This shows that this cohort of children with severe crouch gait could all manage to walk either independently or with assistive devices in the home or classroom environment; all required assistive devices to walk outside the home or in the schoolyard and most children needed a wheelchair to cope with community distances. It should be noted that in this cohort, six subjects did not have assistive devices to use but received a rating of 4 because they regularly used another person, walls, furniture and fences in order to walk over the distance that was scored.

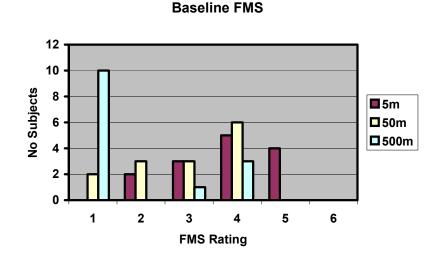


Figure 6.15 Rating of Functional Mobility Scale (FMS) at baseline for subjects with severe crouch gait.

### **Functional Assessment Questionnaire**

The range of scores on the FAQ was between level 5 and 8: three subjects at level 5, seven at level 6, three at level 7 and one at level 8 (Figure 6.16). Household and limited community ambulation are represented by levels 5 and 6 of the FAQ (Novacheck et al 2000). From Figure 6.16 it can be seen that 71.4% of the children were functioning at this level, with 28.6% being rated as community walkers (levels 7-10).



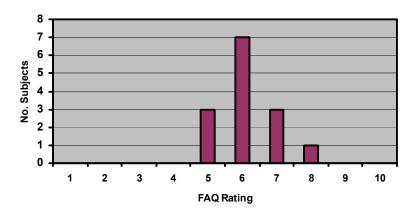


Figure 6.16 Rating of Functional Assessment Questionnaire (FAQ) at baseline for subjects with severe crouch gait.

# 6.3.3 Body Structure and Function

# **Temporal-spatial Parameters**

Temporal-spatial parameters in children with severe crouch gait differed significantly from those of the normally developing subjects. Normalised speed was slower and normalised stride length reduced. There was delayed timing of toe off in the gait cycle. The timing of opposite foot toe off was also delayed but timing of opposite foot contact was premature (Table 6.3)

Table 6.3	Comparison of means ± standard deviations and difference in means
for temporal-s	patial parameters at baseline between severe crouch and normal gait.

Parameters	Severe Crouch	Normal	Seve	e Crouch to Normal			
	mean ±SD	mean ±SD	P value	Est. mean diff.	di	mean iff. ⁄a Cl	
Velocity (m/s)	0.7 ± 0.2	1.3 ± 0.1	<0.001	-0.64	-0.78	-0.50	
Normalised velocity	0.018 ± 0.006	0.034 ± 0.004	<0.001	-0.02	-0.02	-0.01	
Stride length (m)	0.7 ± 0.2	1.2 ± 0.1	<0.001	-0.5	-0.62	-0.39	
Normalised stride length	0.005 ± 0.0001	0.008 ± 0.0006	<0.001	-0.003	-0.004	-0.003	
Toe off %cycle	68 ± 5	60 ± 2	<0.001	8.4	5.7	11.2	
Opposite foot off %ST	26 ± 7	16 ± 3	<0.001	9.5	6.3	12.7	
Opposite foot contact %ST	73 ± 6	84 ± 2	<0.001	-10.9	-13.8	-8.0	

SD

Est. mean diff. Est. mean diff. 95% CI ST SW standard deviation estimated mean difference between severe crouch and normal data 95% confidence intervals of the estimated mean difference between severe crouch and normal data stance phase swing phase

### Kinematics and Kinetics (and Physical Examination)

It is important to note that although kinematic data were collected for all subjects, kinetic data could only be collected for nine subjects in the severe crouch cohort, as five subjects were unable to walk without assistive devices.

Physical examination, kinematic and kinetic parameters were grouped according to anatomical level and the particular plane so that all the information relating to the level and plane could be evaluated together. The particular planes were considered in isolation so that the abnormalities in each plane could be ascertained and the interaction between planes was then considered. The data regarding timing of parameters within the gait cycle and rate of movement were considered separately from the kinematic and kinetic parameters as they were considered to most likely reflect the influence of the central nervous system on the gait abnormality and not the musculoskeletal pathology of bony torsion or muscle contracture.

The kinematic and kinetic data for the subjects with severe crouch gait compared to normally developing subjects are presented in Figures 6.17 and 6.18.

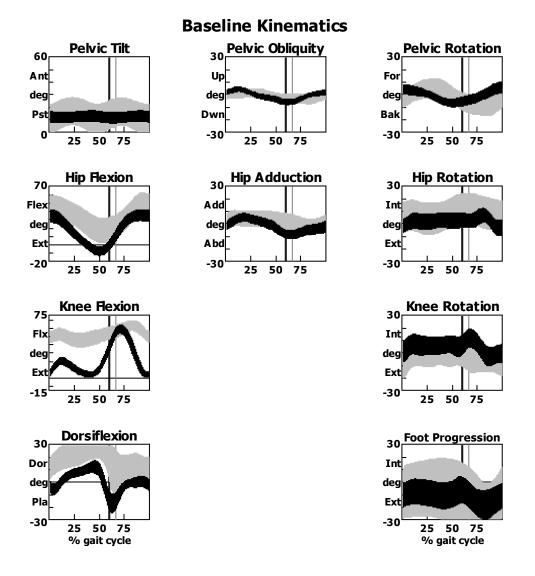
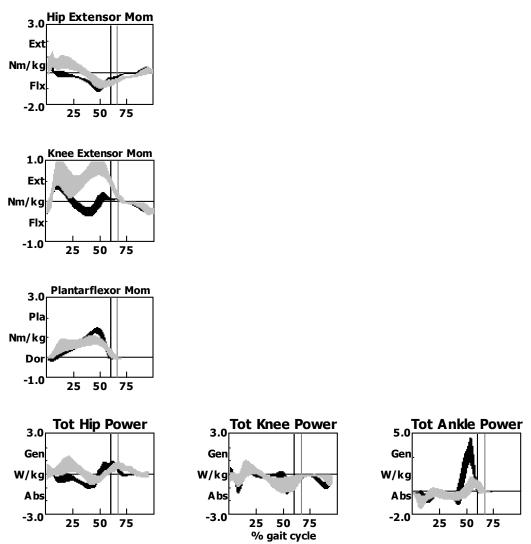


Figure 6.17 Kinematics at baseline. Data shown represent one standard deviation of severe crouch data (shaded grey) and normal data (shaded black). Graphs in the left column represent the sagittal plane; the middle column represents the coronal plane and the right column represents the transverse plane. Note the stiff, flexed knee and the calcaneus ankle.

Chapter 6



# **Moments & Powers Baseline**

Figure 6.18 Kinetics at baseline. Data shown represent one standard deviation of severe crouch data (shaded grey) and normal data (shaded black). Graphs in the left column (top three graphs) represent sagittal moments and the horizontal row at the bottom represents total power produced at each joint. Note the continuous, increased knee extensor moment throughout stance phase.

### Sagittal Plane Parameters

#### Pelvic Parameters (Table 6.4)

The mean pelvic tilt in subjects with severe crouch gait was statistically similar to those of normally developing subjects. However pelvic excursion in the sagittal plane was increased compared to the normal database. The lack of a significant difference between the mean pelvic tilt in crouch gait compared with normal gait was misleading as the standard deviation values for mean pelvic tilt

in crouch gait showed a wide variation when compared to normal data. Inspection of individual pelvic kinematic traces showed that some subjects with severe crouch gait had a posterior tilted pelvis (n = 3 subjects) in respect to normal, whereas some individuals had an anterior tilted pelvis (n = 5) and others had a pelvis that was within normal limits (n = 6). This considerable variation in pelvic position was seen in the sagittal plane pelvic kinematics (Figure 6.17).

Parameters	Severe Crouch	Normal	Sevei	ere Crouch to Normal			
	mean ±SD	mean ±SD	P value	Est. Est. mear mean diff. diff. 95% Cl		ff.	
Max ant pelvic tilt	18 ± 12	14 ± 4	0.26	3.8	-3.0	10.6	
Min ant pelvic tilt	11 ± 12	11 ± 5	0.89	-0.5	-7.5	6.6	
Mean pelvic tilt	14 ± 11	13 ± 4	0.58	1.9	-5.0	8.8	
Pelvic excursion	10 ± 3	4 ± 1	<0.001	6.1	4.5	7.8	
Max hip ext ST	18 ± 15	8 ± 5	<0.001	25.6	16.8	34.3	
Hip excursion	35 ± 7	45 ± 5	<0.001	-9.4	-13.6	-5.2	
Max hip ext moment	1.1 ± 0.5	0.7 ± 0.2	0.017	0.41	0.08	0.75	
Max hip power gen early ST	1.0 ± 0.5	0.5 ± 0.4	0.005	0.52	0.18	0.87	
Max hip power gen late ST	0.8 ± 0.3	1.1 ± 0.4	0.007	-0.34	-0.62	-0.11	
Crossover hip ext moment to flex %ST	56 ± 10	34 ± 13	<0.001	21.9	13.0	30.8	
FFD hip	-21 ± 11	0 ± 0*	<0.001	-21.0	-26.3	-15.8	
SD stand	dard deviation						

Table 6.4Comparison of means ± standard deviations and difference in meansfor pelvic and hip parameters at baseline between severe crouch and normal gait.Statistically significant difference indicated by shaded data.

standard deviation

estimated mean difference between severe crouch and normal data Est. mean diff. Est. mean diff. 95% CI 95% confidence intervals of the estimated mean difference between severe crouch and normal data ST stance phase SW swing phase anterior ant Max maximum Min minimum extension ext generation of power gen

### Hip Parameters (Table 6.4)

Fixed flexion deformity of the hip was present in the severe crouch cohort. Maximum hip extension in stance and hip excursion were reduced in the severe crouch group compared to the normally developing group. The hip extensor moment was increased, crossover to a hip flexor moment was delayed, hip power in loading response (H1) increased and hip power in late stance (H3) decreased in comparison to the normal data.

### The Influence of Assistive Devices on Pelvic and Hip Parameters

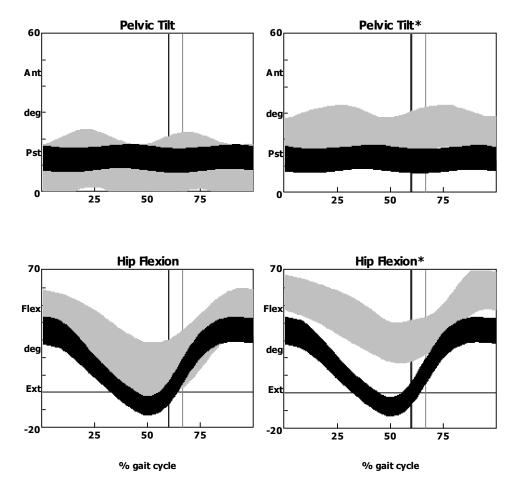
The influence of assistive devices on the kinematic parameters at the pelvis and hip should be considered (Sutherland et al 1997). Clinically, the use of an assistive device tends to promote forward positioning and weight bearing of the upper limbs and in turn, anterior lean of the trunk and tilt of the pelvis, and hence increased hip flexion to some extent. The number of subjects using assistive devices during data capture was small (n = 5), however they had a larger maximum anterior pelvic tilt and a greater decrease in hip extension in stance compared to normal data, than did the subjects who walked independently (Table 6.5).

Table 6.5 Comparison of pelvic and hip parameters in subjects with severe crouch gait (grouped according to the subject's independence or dependence on assistive devices) and normally developing subjects. Shaded data indicate statistically significant difference between the particular severe crouch subgroup and the normally developing subjects.

Parameters	Norm	Severe C	Crouch	No	<u>No Aids to Norm</u>	El	Ā	Aids to Norm	
		No Aids	Aids		Tet moon diff	מיוד מיוד		104 mo	Eet moon diff
	mean	mean	mean	P value	ESL: 1116411 95% CI	cl	P value	5% CI	
Max ant pelvic tilt	14.1	13.8	23.5	0.75	-10.2	7.5	0.026	1.3	17.5
Min ant pelvic tilt	11.3	6.3	15.4	0.27	-14.3	4.2	0.36	-5.1	13.2
<b>Pelvic excursion</b>	3.9	9.7	10.4	<0.001	3.9	7.6	<0.001	3.8	9.2
Max hip ext ST	-7.6	13.5	22.4	0.003	8.3	34.0	<0.001	19.3	40.7
Hip excursion	44.9	34.7	36.3	0.002	-16.3	-4.1	0.002	-13.7	-3.5
FFD hip	0	-20.4	-21.8	<0.001	-28.2	-12.5	<0.001	-29.2	-14.4
Norm Aids Est. mean diff. Est. mean diff. 95% CI ST ant Max Min ext	normally developing subjects assistive devices estimated mean difference between severe crouch and normal data 95% confidence intervals of the estimated mean difference between severe crouch and normal data. stance phase anterior maximum minimum	bjects nce between s als of the estim	evere crouch and	d normal data ence between severe c	rouch and norr	nal data.			

Chapter 6

186



# No aids versus With aids\* Kinematics

Figure 6.19 Sagittal plane kinematics for the pelvis and hip for subjects without and with assistive devices. The subjects without assistive devices are shown in the left column and those with assistive devices in the right column (graph title marked with \*). Data shown represent one standard deviation of crouch data (shaded grey) and normal data (shaded black).

#### Knee Parameters (Table 6.6)

There was fixed flexion contracture at the knee and the popliteal angle confirmed hamstring tightness in the severe crouch group. All crouch subjects had a positive Duncan Ely test. Compared to the normally developing subjects, those with severe crouch gait showed flexed, stiff knees with increased knee flexion at initial contact; increased minimum knee flexion in stance, decreased knee excursion and knee flexor moment in stance; and both peaks of knee extensor moment were increased. In children with severe crouch gait, peak knee flexion in swing phase showed a small estimated difference in means

compared with normal values but this was not statistically significant. No differences were found between normal values and that of severe crouch for knee power generation in early stance and absorption in late stance.

Table 6.6Comparison of means ± standard deviations and difference in meansfor knee parameters at baseline between severe crouch and normal gait.Statisticallysignificant difference indicated by shaded data.

Parameters	Severe Crouch	Normal	Severe Crouch to Normal		rmal	
	mean ±SD	mean ±SD	P value	Est. mean diff.	di	mean ff. % Cl
Kn ext initial contact	51 ± 7	7 ± 5	<0.001	44.0	39.9	48.0
Min kn flex ST	46 ± 9	5 ± 4	<0.001	41.0	36.3	45.8
Kn excursion	20 ± 8	57 ± 5	<0.001	-37.3	-41.6	-33.0
Max peak kn flex SW	66 ± 7	62 ± 6	0.049	4.0	0.01	7.9
Max kn flexor moment	$0.3 \pm 0.3$	-0.2 ± 0.2	<0.001	-0.52	-0.37	-0.68
Max kn ext mom 1 <sup>st</sup> peak	$0.8 \pm 0.3$	0.5 ± 0.2	0.018	0.30	0.06	0.54
Max kn ext mom 2 <sup>nd</sup> peak	$1.0 \pm 0.2$	0.3 ± 0.1	<0.001	0.74	0.57	0.91
Max kn power gen early ST	0.7 ± 0.7	0.8 ± 0.4	0.76	-0.07	-0.53	0.40
Max kn power absorp late ST	-1.2 ± 0.5	-1.2 ± 0.6	0.73	0.07	-0.36	0.50
FFD knee	-18 ± 8	0.7 ± 2	<0.001	-18.8	-23.3	-14.2
Popliteal angle	68 ± 14	31 ± 12	<0.001	29.1	19.3	38.9
Duncan Ely (fast)	all +ve	all -ve	n/a	n/a	n/a	n/a

Est. mean diff.estimated mean difference between severe crouch and normal dataEst. mean diff. 95% CI95% confidence intervals of the estimated mean difference between severe crouch and normal dataSTstance phaseSWswing phaseKnkneeextextensionflexflexion
crouch and normal dataSTstance phaseSWswing phaseKnkneeextextension
SW     swing phase       Kn     knee       ext     extension
Kn knee ext extension
ext extension
flex flexion
mom moment
gen generation of power
absorp absorption of power
FFD fixed flexion deformity
+ve positive test result
-ve negative test result
n/a not applicable

# Ankle Parameters (Table 6.7)

In children with severe crouch gait, static measures of muscle length of the gastrocnemius and soleus by the Silfverskiold test were not different from the normal data. Dorsiflexion at initial contact was increased compared to normal data, as was maximum dorsiflexion in stance and swing phase. Children with severe crouch gait showed reductions in maximum plantarflexion of third rocker, excursion of third rocker and ankle power generation in late stance (A2) compared with normally developing children.

Table 6.7Comparison of means ± standard deviations and difference in meansfor ankle parameters at baseline between severe crouch data and normal data.Statistically significant difference indicated by shaded data.

Parameters	Severe Crouch	Normal	Seve	re Crouc	h to No	rmal
	mean ±SD	mean ±SD	P value	Est. mean diff.	di	mean ff. % Cl
Dorsiflexion initial contact	12 ± 9	-1 ± 3	<0.001	13.4	8.8	18.1
Max dorsiflex ST	29 ± 10	15 ± 4	<0.001	15.0	10.8	19.1
Max plantarflex 3 <sup>rd</sup> rocker	8 ± 13	-17 ± 6	<0.001	-24.8	-17.3	-32.4
Excursion 3 <sup>rd</sup> rocker	21 ± 13	31 ± 5	0.003	-10.1	-16.5	-3.8
Max dorsiflex SW	19 ± 9	3 ± 3	<0.001	16.0	11.4	20.7
Max ankle power gen late ST	1.2 ± 0.6	4.2 ± 0.8	<0.001	-3.0	-3.5	-2.5
Dorsiflexion (kn flexion)	21 ± 12	23 ± 7	0.66	-1.6	-9.0	5.8
Dorsiflexion (kn extension)	2 ± 8	5 ± 4	0.22	-2.9	-7.5	1.8

SD

standard deviation

-		
E	Est. mean diff.	estimated mean difference between severe crouch and normal data
E	Est. mean diff. 95% Cl	95% confidence intervals of the estimated mean difference between severe
		crouch and normal data
S	ST .	stance phase
S	SW	swing phase
Ν	Лах	maximum
d	lorsiflex	dorsiflexion
р	lantarflex	plantarflexion
•	ien	generation of power
	in .	knee

# Coronal Plane Parameters

(Refer to Table 6.8 for parameters in the coronal and transverse plane).

## Pelvic Parameters

The pelvic excursion in the coronal plane was similar to that of normally developing subjects.

## Hip Parameters

In children with severe crouch gait, maximum hip adduction in stance phase was not different from normal data. However in swing phase, maximum hip adduction was different for the severe crouch group as the normally developing group achieved hip abduction in swing.

# Transverse Plane Parameters

Physical examination of the arc of hip rotation for the severe crouch group demonstrated increased internal rotation and decreased external rotation compared to normally developing subjects. The mean thigh foot angle was not different from normal data but the standard deviation showed considerable variability. All 14 subjects had calcaneus of the hindfoot, 11/14 had hindfoot valgus, 14/14 had planus of the midfoot and 7/14 had forefoot abduction.

The pelvic excursion in the transverse plane was not different from normal data. Mean hip rotation in stance phase showed more internal rotation than normal. Mean foot progression in stance however was not different from normal, but the 95% confidence interval for the estimated difference in means was wide (p = 0.5, 95% CI -8.4°, 16.7°) indicating considerable variability in mean foot progression for the severe crouch group. Of the 28 lower limbs in the severe crouch cohort, 11 showed a foot progression that was internally orientated to the one standard deviation band of normal, nine were within normal limits and eight were external.

Table 6.8 Comparison of means ± standard deviations and difference in means between severe crouch and normal gait, for the pelvis, hip and foot parameters in the coronal and transverse planes at baseline. Statistically significant difference indicated by shaded data.

Parameters	Severe Crouch	Normal	Severe Crouch to Normal		rmal	
	mean ±SD	mean ±SD	P value	Est. mean diff.	Est. ı di 95%	
<u>Coronal Plane</u>						
Pelvic excursion	10 ± 5	11 ± 3	0.45	-1.1	-4.2	1.9
Max hip adduction ST	8 ± 6	6 ± 4	0.29	1.7	-1.5	5.0
Max hip adduction SW	5 ± 6	-1 ± 4	<0.001	5.6	2.8	8.5
<u>Transverse Plane</u>						
Pelvic excursion	22 ± 14	15 ± 6	0.10	6.6	-1.3	14.6
Mean hip rotation ST	8 ± 11	2 ± 6	0.008	6.3	1.8	10.8
Mean foot progression ST	-6 ± 24	-10 ± 6	0.50	4.2	-8.4	16.7
Hip internal rotation	58 ± 10	49 ± 13	0.04	9.6	0.58	18.7
Hip external rotation	15 ± 16	33 ± 8	<0.001	-18.4	-27.6	-9.2
Thigh/ foot angle	4 ± 15	8 ± 4	0.37	-3.81	-12.4	4.7

SD Est. mean diff. Est. mean diff. 95% Cl	standard deviation estimated mean difference between severe crouch and normal data 95% confidence intervals of the estimated mean difference between severe crouch and normal data
ST	stance phase
SW	swing phase
Max	maximum

# **Timing of Parameters**

(Refer to Table 6.9 for parameters relating to timing in gait and rate of change).

The timing of parameters within stance or swing phase for children with severe crouch gait was significantly different from those in normally developing subjects: the timing in stance of maximum hip and knee extension, and A2 were all premature whilst peak knee flexion in swing phase was delayed.

# Rate of Movement (degrees/sec)

For the severe crouch group, the rate of change from maximum knee extension in stance to peak knee flexion in swing and the rate of change in third rocker from maximum dorsiflexion to peak plantarflexion were slower than those of normally developing children.

Table 6.9Comparison of means ± standard deviations and difference in means<br/>between severe crouch and normal gait, for the timing and rate of change of<br/>parameters at baseline. Statistically significant difference indicated by shaded data.

Parameters	Severe Crouch	Normal	Seve	re Crouc	h to No	rmal
	mean ±SD	mean ±SD	P value	Est. mean diff.	di	mean ff. ⁄6 Cl
<u>Timing (% Stance or</u> <u>Swing)</u>						
Max hip ext %ST	79 ± 6	84 ± 3	0.001	-4.7	-7.3	-2.0
Crossover hip ext moment to flex %ST	56 ± 10	34 ± 13	<0.001	21.9	13.0	30.8
Min kn flex %ST	51 ± 18	65 ± 6	0.003	-14.4	-23.4	-5.4
Max peak kn flex %cycle	82 ± 6	71 ± 2	<0.001	11.0	7.8	14.2
Max peak kn flex %SW	43 ± 17	28 ± 4	0.002	15.0	6.1	23.9
Max plantarflex 3 <sup>rd</sup> rocker %SW	6 ± 12	7 ± 4	0.7	-1.2	-7.5	5.2
Max ankle power gen %ST	84 ± 3	88 ± 2	<0.001	-4.1	-5.9	-2.4
Rate of Change (°/s)						
Max kn ext to peak kn flex	40 ± 19	190 ± 34	<0.001	-150	-169	-131
Max dorsiflex to plantarflex 3 <sup>rd</sup> rocker	86 ± 63	181 ± 32	<0.001	-95	-129	-61

SD	standard deviation				
Est. mean diff.	estimated mean difference	e between crouch ar	id normal data		
Est. mean diff. 95% Cl	95% confidence intervals of the estimated mean difference between crouch and normal data				
ST	stance phase	SW	swing phase		
max	maximum	min	minium		
ext	extension	flex	flexion		
kn	knee	plantarflex	plantarflexion		
gen	generation of power	dorsiflex	dorsiflexion		

## **6.4 DISCUSSION**

#### 6.4.1 Subjects and Surgical History

Crouch gait has been reported to develop around the time of the adolescent growth spurt (Gage 2004f). It is therefore interesting that of the 14 subjects, five subjects were under the age of 11 years and consequently would not yet be considered to be in their adolescent growth spurt. Three of these subjects had had prior isolated calf or TAL lengthening and two subjects had only had prior botulinum toxin A injections to the calf and hamstrings. The effect of surgical lengthening to the calf complex should not be underestimated in respect to its relationship to the development of crouch gait. However growth spurts must also accentuate a developing posture that will become crouch gait, e.g. apparent equinus is probably the precursor to crouch gait prior to an adolescent growth spurt.

The predominance of prior calf lengthening in most of the subjects with severe crouch gait was suggestive of its involvement in the development of crouch gait. In some of the subjects, hamstring surgery had also been undertaken either at a prior date, simultaneously or at a later date to calf surgery. Severe crouch gait still developed in those particular subjects despite the hamstring lengthening being undertaken to decrease knee flexion. Interestingly one subject had had calf, hamstring and psoas surgery as multiple event single level surgery prior to this study and so the effect of not having all the surgeries simultaneously (as in SEMLS) and the influence of lever arm dysfunction (LAD) on crouch gait must be considered in this case. Gage (2004f) believes that the precursors to developing crouch gait "almost always include LAD and /or previous weakening of the soleus" (p. 383).

#### 6.4.2 Activities- Mobility Status

The level of activity can be ascertained by mobility scales providing that the rater records the level according to what the subject usually does and not by their best performance. Limitations in activity were documented in all the scales used.

The results from the FMS and FAQ reflected a similar pattern: that the majority of the children with severe crouch gait did not walk within their community but did walk in the home and school environment. The GMFCS and FMS confirmed that most children required assistive devices within the home and at school. The majority of children who walked in severe crouch gait were not true community ambulators but used wheelchairs over a 500m distance, and tended to use assistive devices in order to walk in the home and at school. This limitation in walking range has also been described previously (Sutherland and Davids 1993). It should be noted that three of the children who were rated as GMFCS level III at the time of the study, had been independent ambulators prior to gait deterioration and accordingly would have been classified previously as GMFCS level II, which reflects that the cohort was not necessarily stable, but was deteriorating in respect to level of ambulation (Chambers 2001). These three children could still walk unaided on the walkway in the laboratory for 5m so that kinetic data were collected from them, but all other ambulation was assisted by assistive devices and hence the rating of GMFCS level III. These findings were consistent with the symptoms often complained of by children walking in crouch gait: deteriorating gait, increasing energy required to participate, and poor stability.

## 6.4.3 Body Structure and Function

# **Temporal-spatial Parameters**

Temporal-spatial measures were collected during the 3DGA, which is a clinical situation, and the data collected may represent a child's best effort or conversely the level of indifference to a test! Velocity and stride length were considered appropriate variables to examine as they represent a person's ability to "keep up" with family and peers and ability to get to a destination within a reasonable time frame. Normalisation of the two parameters was undertaken to diminish the effect of different rates of growth on their values by conversion to dimensionless units (Hof 1996) and subject height was used in the calculations (Õunpuu et al 2002a, van der Linden et al 2003a). There are differing arguments as to whether height (O'Malley 1996) or leg length (Hof and Zijlstra 1997) should be used in the calculations with no resolution currently in the

literature. In children, the relationship between step length and height has been shown to have a high correlation coefficient (0.91), the same as for step length and leg length (Sutherland et al 1988). As children grow their body proportions vary (Sutherland et al 1988) and so leg length will not necessarily have a consistent relationship with the length of the torso above. It was therefore decided to use height in this study as it is the sum of the growth between body proportions and it eliminated the need to average between two leg lengths or choose between differing leg lengths for a subject. Recent studies in the area of gait analysis with children, who are normally developing or have cerebral palsy, have used height to normalise data (Õunpuu et al 2002a, Stansfield et al 2001a, Stansfield et al 2001b, van der Linden et al 2003a, van der Linden et al 2002). Timing of toe off within the gait cycle, opposite foot off and opposite foot contact in stance were chosen, because duration of stance and swing and the period of single limb support could be ascertained from these parameters. These parameters would indicate the level of stability and balance whilst walking for the severe crouch cohort (Sutherland et al 1988).

Temporal-spatial measures indicated that severe crouch gait was hindered by decreased speed and stride length, with prolonged stance phase and decreased single limb stance, suggesting that stability was possibly challenged in single limb stance. These changes in temporal-spatial measures in crouch gait have been noted previously (Segal et al 1989, Steinwender et al 2001, Sutherland and Davids 1993) and in particular in a study on plantarflexor muscle function in gait (Sutherland et al 1980).

# Pelvic Parameters in the Sagittal Plane

Pelvic parameters for crouch gait have only been reported in the form of a published abstract by Tylkowski et al (1988). The pelvis has been ignored in other studies (Lin et al 2000, Sutherland and Cooper 1978, Sutherland and Davids 1993) on crouch gait even though the position of the pelvis will influence hip extension. This study is the first to report the position of the pelvis in children with severe crouch gait. The lack of significant difference between the mean pelvic tilt in severe crouch gait compared to that of normal gait, did not reflect the level of variability of the pelvic position in the severe

crouch cohort (Figures 6.17). This variability in pelvic position agreed with the previous findings, which showed that pelvic tilt may be anterior, neutral or posterior (Tylkowski et al 1988). The 95% confidence interval showed this variability and this was a convincing example of why mean kinematic parameters and statistical significance should not be assessed in isolation.

When there is a neutral pelvic position, this possibly reflects a balance (albeit precarious) between muscles that are contracted, those that are spastic and those that are weak, that all combine to influence the position of the pelvis. The hamstrings or psoas muscles may be contracted, or spastic and the glutei, abdominals and hamstrings may be weak. Various combinations may lead to a mean pelvic tilt that is within normal range. Contracted or spastic hamstrings and psoas muscles could balance each other (Tylkowski et al 1988); contracted hamstrings may counter weak abdominals in the absence of a contracted or spastic psoas. A posteriorly tilted pelvis has been attributed to hamstring dominance and prolonged activity, and the anteriorly tilted pelvis to hip flexor contracture and poor function of gluteus maximus (Tylkowski et al 1988). Fixed flexion contracture of the hip and an increased popliteal angle were found in this study but the strength of the abdominal muscles and gluteus maximus were not recorded and so the influence of weak muscles combined with contracted muscles cannot be ascertained but only surmised here. Tylkowski et al (1988) also did not measure muscle strength but through EMG recording found that the gluteus maximus muscle contracted out of phase if the pelvis was anteriorly tilted, or contracted on occasion if the pelvis was neutral. This implies that the gluteus maximus is not working optimally in both cases, whether from weakness, biomechanical disadvantage, or disruption by central nervous system involvement, is not possible to ascertain.

The increased excursion of the pelvis in severe crouch gait may have been due to spasticity in the psoas and hamstrings that forces the pelvis into increased excursion. The increased pelvic excursion may have been a compensatory action to gain as long a stride length as possible by increasing hip extension in stance and hip flexion in swing to assist foot clearance. The variability of the pelvic position in the sagittal plane and the increased pelvic excursion provide evidence of the involvement of the pelvis in severe crouch gait and the need to consider the pelvis when planning intervention to rectify the gait pattern.

# Hip Parameters in the Sagittal Plane

The decreased hip extension in stance and reduced hip excursion in severe crouch gait may have been indicative of muscle weakness in the hip extensors (four subjects had had prior hamstring surgery), or hip flexor contracture or spasticity (fixed hip flexion deformity was observed and only one subject had had prior psoas surgery) or again a combination of both influences. Accordingly, if hip extension was lacking then the hip extensor moment would be increased and prolonged in the effort to gain as much hip extension as possible by trying to direct the ground reaction force posterior to the hip joint. The decreased hip extension in stance and increased internal hip extensor moments have been previously noted but not the reduction in hip excursion nor the delay in crossover of the hip extensor moment to a flexor moment (Lin et al 2000, Sutherland and Cooper 1978, Sutherland and Davids 1993). The decrease in hip power generation in late stance coupled with the decrease in ankle power generation also in late stance, leads to the question of the source of the power generation in the trailing limb to aid forward movement into swing. Alternately is movement into swing reliant on the increased hip power generation in loading response of the forward limb as demonstrated in this study and the study by Steinwender et al (2001)? According to Gage (1991), if the power for propulsion is reduced from the hip and ankle in late stance, then 58% of the power source for propulsion has been affected.

The decrease in hip extension and excursion would have contributed to the decrease in stride length for the severe crouch group.

The use of assistive devices appeared to influence the pelvic and hip kinematic parameters but because of the small number of subjects in the subsets, the results should be viewed with some caution.

## Knee Parameters in the Sagittal Plane

Because the definition of severe crouch gait stipulated a minimum knee flexion of greater than 30°, it was not surprising that knee kinematics and kinetics (except peak knee flexion in swing) were significantly different from those of normally developing children. Previous studies had noted the increased knee flexion in stance and the increased knee extensor moment (Lin et al 2000, Sutherland and Cooper 1978, Sutherland and Davids 1993), and tightness of the popliteal angle, which has been thought to indicate hamstring contracture (Sutherland and Davids 1993). Increased power absorption at the knee had been found in crouch gait in late stance (Lin et al 2000). However this was not the case in this study as values for severe crouch gait were similar to normal values implying that the knee was unable to provide compensatory power to counter the severe crouch gait (Figure 6.18). From inspection of the total knee power graph in late stance phase, the percentage of the gait cycle over which the power absorption occurred for the severe crouch group was longer than that for the normally developing subjects. The premature and prolonged knee power absorption was also seen in the knee power graph in the study by Lin et al (2000, p 228) but was not commented on in the text. This increased duration of knee absorption power implies that the knee was attempting to counter the severe crouch gait over an increased duration of the gait cycle. The number of subjects in this study who had kinetic data collected (n = 9) was similar to the number in the study by Lin et al (2000) (n = 8). The small number in both studies limits generalisation of the findings to the wider population of children walking in severe crouch gait although they provide important insights.

The presence of fixed flexion at the knee, rectus spasticity and the absence of cross over from a knee extensor moment to a knee flexor moment in stance, had also been noted prior to this study of severe crouch gait (Gage 2004d, Lin et al 2000, Sutherland and Cooper 1978). The lack of a trough between peaks in the vertical ground reaction force had been described (Lin et al 2000) and was linked to the absence of the knee flexor moment in mid stance in crouch gait. The absence in knee flexor moment in severe crouch gait implied that at no point was the ground reaction vector directed anterior to the knee joint and the quadriceps mechanism therefore had been activated continuously throughout the

stance phase in the effort to maintain knee extension. Electromyography has confirmed the prolonged activation of the rectus femoris (Lin et al 2000, Sutherland and Cooper 1978, Sutherland and Davids 1993), vastus medialis, medial hamstrings (Sutherland and Cooper 1978, Sutherland and Davids 1993) and gluteus maximus in stance (Sutherland and Cooper 1978) that would be inferred due to the increased hip and knee extensor moments. All of this co-contraction at the knee and hip is understandable, as it is needed to maintain an upright posture, and prevent further sinking down into flexion.

Standing in 30° or more of knee flexion has been shown to increase the forces on the quadriceps, proximal tibia and patella, and the quadriceps are required to work at 51% of their maximum force in order to stabilise the knee (Perry et al 1975). Constant walking in crouch gait has been reported to place great stress on the patello-femoral joint (Perry et al 1975) and lead to anatomical and functional changes at this joint such as patella alta, and in some cases avulsion or fracture of the inferior pole of the patella. Walking with a flexed knee gait can therefore be problematic and lead to not just biomechanical (Haxton 1945, Lieb and Perry 1968, Lotman 1976, Perry et al 1975) and physiological changes but anatomical complications (Lloyd-Roberts et al 1985, Rosenthal and Levine 1977, Topoleski et al 2000) (Figure 6.20) resulting in knee pain and curtailment of ambulation as a consequence.



a) patella alta



b) patellar avulsion



c) patellar fracture

Figure 6.20 Radiographs of a) patella alta b) avulsion of patella and c) patellar fracture. These radiographs are from three of the subjects with severe crouch gait.

The decrease in knee excursion in severe crouch gait was not related to a decrease in the peak knee flexion in swing phase as it was close to within normal limits, but was attributable to the decrease in knee extension in stance.

## Ankle Parameters in the Sagittal Plane

The majority of subjects in the severe crouch group (11/14 subjects) had previous calf lengthening and it has been shown that calf lengthening can lead to iatrogenic calcaneus (Borton et al 2001, Delp et al 1995, Dillin and Samilson 1983, Segal et al 1989, Truscelli et al 1979). In one study, which examined the long term effects of isolated calf lengthening surgery, it was found that the risk of calcaneus in spastic diplegia was 44% if the surgery was undertaken when the child was less than eight years old, and 19% if the child was older than eight years (Borton et al 2001). This study was undertaken at our centre and the follow-up ranged from 5-10 years and 134 children were assessed for calcaneus gait and calcaneus deformity. Being a female and having a past history of percutaneous lengthening of the tendo achilles were shown to be risk factors for the development of calcaneus, more so than being a male or having undergone other isolated calf lengthening procedures (Borton et al 2001). Another study showed an incidence of calcaneus in spastic diplegia post calf surgery of 30%. However the sample size was only 20 subjects and follow-up was between 1-11 years (Segal et al 1989). The lower incidence may therefore be attributable to some of the subjects only having a short period between surgery and follow-up (Segal et al 1989).

The effect of different types of calf lengthening on the passive range of dorsiflexion and strength of the plantarflexors has been studied using computer simulations (Delp et al 1995). If only the gastrocnemius was contracted, lengthening of the gastrocnemius aponeurosis in isolation improved the passive range of dorsiflexion and maintained the moment generating potential of the plantarflexors, whereas lengthening of the Achilles tendon reduced the latter. This weakness of the plantarflexors post-Achilles tendon lengthening occurred because the soleus was also lengthened by the surgery resulting in inadvertent weakness of the soleus. When the Achilles tendon is lengthened, the tension on the muscle fibres of both the gastrocnemius and soleus is decreased and because

the architecture of the soleus is different to that of the gastrocnemius, the weakening is more pronounced in the soleus (Delp et al 1995). If both the gastronemius and soleus were contracted, the lengthening of the Achilles tendon was able to improve passive dorsiflexion range but again the capacity to generate moments by the plantarflexors was decreased. However, if only a lengthening of the gastrocnemius aponeurosis was undertaken the soleus contracture was not altered and dorsiflexion range of motion reflected this. This study recommended that if a calf lengthening was considered necessary, the type of calf surgery undertaken should be carefully chosen in order to maintain the moment generating capacity of the calf whilst increasing passive range of motion. It was suggested that aponeurotic lengthenings of the gastrocnemius and soleus be performed to improve passive range of motion into dorsiflexion, and that these surgeries would preserve the strength of the plantarflexors (Delp et al 1995). Most of the calf lengthenings in the subjects in the severe crouch study were either lengthenings of the Achilles tendon or Bakers calf lengthenings. Neither of these calf lengthenings is selective in lengthening only the gastrocnemius. Therefore both the gastrocnemius and soleus were lengthened and the decrease in ability to produce an adequate plantarflexor moment post calf lengthening was not surprising, considering the results from the above study of computer simulation of calf surgery (Delp et al 1995).

The injection of botulinum toxin A into the calf as an intervention for dynamic equinus, perhaps should be used carefully as two of the severe crouch subjects had no prior history of calf surgery, but a history of botulinum toxin A injections into the calves. The development of crouch gait post-botulinum toxin A injections to the calf, has never been reported but is clinically recognised as a potential complication. A possible logical effect of botulinum toxin A injections into the calf, may be to weaken the muscle and this could lead to the development of crouch gait (Gough et al 2005). It should therefore be recommended that post calf injection with botulinum toxin A, the child's gait is monitored regularly for signs of the development of crouch gait and solid AFOs prescribed if detected.

The excess dorsiflexion at initial contact and maximum dorsiflexion in swing, both emphasised the extent of the calcaneus gait found in the subjects with severe crouch gait (Figure 6.17). The decrease in maximum plantarflexion, excursion and ankle power generation in third rocker may have been attributable to the constant excessive dorsiflexion in stance (regardless of cause- natural history or iatrogenic) that would cause the soleus to repeatedly contract in its outer range and lead to weakness in its inner range, therefore preventing development of a normal third rocker (Delp et al 1995, Fabry et al 1999).

In the physical examination, the static range of dorsiflexion obtained was limited by the physiotherapist's ability (i.e. strength) to counter any plantarflexor resistance that was present. Therefore the static range of dorsiflexion recorded in weight bearing and hence walking does not necessarily equate with that from the physical examination. Hence it was not surprising that although the static range of dorsiflexion for the severe crouch group was similar to that from normally developing children, in walking the maximum dorsiflexion in stance was significantly greater. It has been shown previously that measures of static range of motion do not correlate with the dynamic measures found in 3DGA for subjects with cerebral palsy (Boyd et al 1998, Orendurff et al 1998, Ounpuu et al 2004, Pliatsios et al 1998, Selber et al 2000) or subjects acting as controls who do not have neuromuscular pathology (McMulkin et al 2000). This difference between static and dynamic measures has already been discussed with regard to predicting crouch gait in Chapter 2 Section 2.4.2. This information has relevance to the use of botulinum toxin A injections for a tight calf. A calf may feel tight to the examiner during the physical examination but when the child walks the ankle is neutral or calcaneus in gait. The indication for botulinum toxin A injections into the calf in this case would be questionable.

The increase in dorsiflexion in stance (Lin et al 2000, Sutherland and Cooper 1978, Sutherland and Davids 1993) and increased plantarflexor moment (Lin et al 2000) accompanied by the decrease in ankle power generation prior to toe off, associated with crouch gait are already known, but the decrease in the third ankle rocker of plantarflexion and excursion, has not been previously reported

in crouch gait. Calf weakness (as a consequence of previous calf lengthening or from calf contracture that has led to the development of pes valgus) has been considered as a cause of the increased stance phase dorsiflexion and resulting increased plantarflexor moment (Sutherland and Cooper 1978, Sutherland and Davids 1993).

# **Coronal Plane Parameters**

The pelvic excursion in the coronal plane in severe crouch gait was similar to that of normal gait. This implies that there was no compensatory movement of the pelvis to assist clearance of the limbs in swing phase (Table 6.17). A previous study that used different coronal plane pelvic parameters to evaluate hip hike to assist limb clearance in swing phase in crouch gait, came to the same conclusion (Steinwender et al 2001).

Although the maximum adduction in stance phase in severe crouch gait was not greater than normal (Steinwender et al 2001), Figure 6.17 shows that adduction in stance was prolonged in stance phase for the crouch subjects compared to the normally developing subjects. The increased maximum adduction in swing suggests that abduction was not easily achieved for the subjects with severe crouch gait.

# **Transverse Plane Parameters**

Transverse plane malalignment of the femur, tibia and/ or foot is an example of what is referred to as lever arm deficiency (Gage 1991) or dysfunction (Schwartz and Lakin 2003). This malalignment in the transverse plane is known to impact on the ability of the knee to extend in stance (Fabry et al 1999, Gage 1990, Schwartz and Lakin 2003, Sutherland and Davids 1993). In the children with severe crouch gait, the mean hip rotation showed a tendency to be clinically internally rotated and there was a large variability in the foot progression angle (nine feet normally aligned, eight feet in internal and 11 feet in external progression). This malalignment of the segments of the lower limbs (hip internally rotated to a normally positioned or externally rotated foot progression) has the effect of inhibiting the generation of an adequate moment at the knee to maintain knee extension, by shortening the lever arm at the ankle

due to the rotated foot posture (Gage 1990, Schwartz and Lakin 2003) (Figure 6.21). Dominance of hip internal rotation over external rotation was found in the physical examination for the severe crouch group and the foot progression may be a compensation for this or may be the primary deviation. However on physical examination the thigh foot angle was not significantly different to normal values but there was a large range in values as indicated by the large standard deviation. Pes valgus and external tibial torsion have been reported as features of crouch gait contributing to the lack of knee extension (Sutherland and Davids 1993). The high incidence of the foot posture of calcaneus, valgus, and planus in weight bearing in the severe crouch group suggests that pes valgus was a characteristic feature of the group. This together with the internal rotation of the femur in stance, shows that lever arm dysfunction was present in this cohort. The excursion of the pelvis in the transverse plane was not increased, which indicates that it was not used as a compensatory mechanism to increase stride or step length or help propel the limb forward in swing phase.

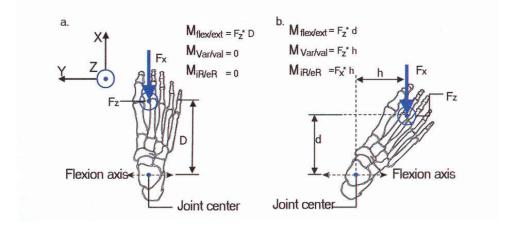


Figure 6.21 An example of how malalignment in the transverse plane alters the lever arm at the foot. This is reproduced from Gage and Schwartz (2004c, p. 199). The normal alignment of the foot/ tibia with the femur is shown in a) with  $Fz^*D$  representing the length of the extension moment in respect to the foot. Note the varus/ valgus and rotation moments are zero. In the presence of malalignment of the femur, tibia or foot in respect to each other b) the length of the extension moment  $Fz^*d$  is shortened (compare length D in the normal foot to length d in the malaligned foot). Also the varus/ valgus and rotation moment acting at the knee and introduces deforming forces at the foot, ankle and knee (Gage and Schwartz 2004c).

# Timing of Parameters

The abnormal timing of key kinematic and kinetic parameters, plus the diminished rate of change between parameters in severe crouch gait reflect the influence of abnormal central motor control on the lower limb as spasticity, combined with fixed and/ or dynamic contracture, and muscle weakness, all result in abnormal biomechanics (Mayer 1997) of the lower limbs in crouch gait. The premature maximum hip extension and minumum knee flexion in stance in the severe crouch group may have been due to the inability of the lower limb to continue to extend in stance once single support began. This may have been due to a combination of muscle weakness of the glutei and hamstrings at the hip, quadriceps at the knee and soleus at the ankle; and contacture and/ or spasticity of the psoas and hamstrings at the hip and hamstrings at the knee, all combining to prevent further extension of the limb during stance. The delayed peak knee flexion in swing has been attributed to rectus spasticity (Õunpuu et al 1993a, Õunpuu et al 1993b, Perry 1987, Piazza and Delp 1996, Sutherland et al 1990) and rectus/ hamstring co-contraction (Gage et al 1987).

Similarly the decreased change in rate of maximum knee extension to peak knee flexion and from maximum dorsiflexion to plantarflexion in third rocker for the severe crouch group, could be multifactorial. Weakness of the hamstrings and hip flexors, rectus/ hamstring co-contraction, gastrocsoleus weakness, contracture or spasticity of the tibialis anterior or peroneus brevis, could all contribute. The slow rate of change at the knee with the delayed peak knee flexion in swing and the decreased knee excursion would all affect the ability of the limb in swing phase to gain adequate foot clearance, for a foot that was likely to be already in a position of external foot progression in stance and if dragged along the floor in swing, would only help to perpetuate that posture and possible hallux valgus (Davids et al 2001). The slow rate of change in third rocker at the ankle in combination with decreased maximum plantarflexion and excursion of third rocker, would decrease the joint angular velocity at the ankle and this would have a deleterious effect on ankle power generation (Gage 1991). A criticism of the parameter of rate of change of third rocker is that it is influenced by gravity as the knee flexes and the foot is unloaded. Plantarflexion

can be active through concentric muscle contraction as well as passive due to gravity as the leg is lifted into swing and so rate of change in third rocker will not necessarily have an influence on the amount of ankle power generation at toe off.

# 6.4.4 Limitations

One of the limitations of this study of severe crouch gait was the small number of subjects involved. However previous studies characterising crouch gait have all been affected by the same concern. There were four subjects in Sutherland and Cooper's study (Sutherland and Cooper 1978) and eight subjects in the study by Lin et al (Lin et al 2000). In both studies, subjects did not use assistive devices (assumed in the study by Lin et al (2000) as kinetic data were collected), whereas in our study kinetic data could only be collected for nine subjects and only six of these subjects would have usually walked without assistive devices outside the home or in the schoolyard. To have excluded the subjects who used assistive devices would have restricted the cohort to subjects with a rating of GMFCS level II and ignored the fact that this would not be representative of those who walk in severe crouch gait. The use of assistive devices had an influence on pelvic and hip posture, which confounded the results. This was the reason for examining the pelvis and hip results according to those subjects who did and did not use assistive devices. Statistical analysis was inappropriate because the numbers in each group were too small.

The collection of data relating to strength and electromyography of the muscles in the lower limbs would have given further insight into the characteristics of severe crouch gait. Unfortunately at the time of data collection, strength testing was not routinely undertaken and electromyography was not available in the gait laboratory where the data were collected. The information provided by strength testing would have assisted our understanding of which muscles are routinely weak in crouch gait e.g. possibly abdominals and gluteus maximus at the pelvic-hip level, quadriceps at the knee and soleus at the ankle. If weak, then these muscles could be targeted for strengthening within physiotherapy treatment regimes and community gym programmes. Electromyographic studies would have provided additional information to that of Sutherland and

207

colleagues (1978, 1993) and Lin et al (2000), on muscle activation patterns in severe crouch gait.

Trunk data were not included in routine data collection at the pre-SEMLS period. Inclusion of trunk data would have been helpful in understanding the complete picture of the biomechanics of severe crouch gait and whether subjects attempt to use forward trunk lean to bring the knee extensor moment anterior to the knee in severe crouch gait, or whether this compensation is unavailable to them due to the severity of the crouch gait. A recent conference abstract documented normally developing subjects who assumed a crouch gait with and without a forward trunk lean (Westwell et al 2005). Subjects who walked in crouch gait with a normal trunk tilt demonstrated a substantial increase in the mean knee extensor moment in stance, whereas if they walked in crouch with a forward trunk lean, this moment was decreased (Westwell et al 2005). If trunk data had been collected for the severe crouch cohort, then trunk posture as a compensation to minimise knee stress and pain could have been investigated.

# 6.5 SUMMARY

Dr Jim Gage (2004a), in his book pertaining to the treatment of gait abnormalities in cerebral palsy, points out the vicious cycle that occurs in crouch gait particularly in respect to the hamstrings and rectus femoris. As noted above, the hamstrings are powerful hip extensors as the lever arm that their origin makes with the hip joint is much longer and therefore more powerful than the shorter lever arm that their insertion makes with the knee joint (Delp et al 1996, Gage and Schwartz 2004c, Hoffinger et al 1993). When the plantarflexion knee extension couple begins to fail due to soleus weakness or malalignment of lever arms particularly at the foot and/ or tibia, the external knee moment is no longer extensor but becomes flexor as the GRF is behind the knee and the knee is not held in extension, but begins to flex. As the knee flexes, the insertion of the hamstrings is moved further away from the knee joint and so the hamstrings lever arm at the knee increases in length and therefore power increases. With increasing knee flexion, the lever arm at the knee will

eventually become longer than that at the hip, and so the hamstrings become a stronger knee flexor than a hip extensor and their activity forces the knee into more and more flexion with time (Figure 6.22). The rectus femoris together with the vasti will be forced to try and counter the external knee flexor moment. As the rectus is also a hip flexor, its increased activity may also force the hip to flex further if the lever arm at the hip is longer than that at the knee due to the increased knee flexion, which occurs when the GRF is posterior to the knee and anterior to the hip. With the GRF in front of the hip, the external hip moment will be flexor, so the internal moment at the hip will be extensor and the hamstrings will be recruited with the other hip extensor muscles to counter the external hip flexor moment. However their extensor activity will be inhibited at the hip because they are now more powerful as knee flexors. A vicious cycle between the hamstrings and rectus is created whereby the rectus will try and counter the increased knee flexion but also cause increased hip flexion. This then requires increased hip extension to remain upright but the hamstrings, in countering the increased hip flexion, will cause increased knee flexion (Gage and Schwartz 2004c). It is not suprising that crouch gait deteriorates with time, and that the overloaded knee extensor mechanism fails.

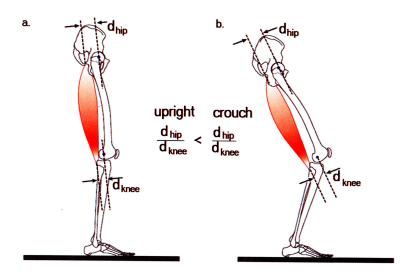


Figure 6.22 A schematic diagram of the length of the lever arms of the hamstrings at the hip and knee in a) normal gait and b) crouch gait. This is reproduced from Gage (2004c, p. 202). In normal gait a) with the body upright, the lever arm of the hamstrings at the hip is greater than that at the knee. In crouch gait b) with increasing knee flexion, eventually the lever arm of the hamstrings at the knee becomes greater than that at the hip. (I believe the < sign in the diagram should therefore be >). Consequently in crouch gait, the hamstrings can eventually become more powerful knee flexors than hip extensors. As the rectus femoris attempts to maintain knee extension but simultaneously flexes the hip, the hamstrings must counter this by extending the hip. But because of the change in lever arm length in the presence of crouch gait, this leads to increased knee flexion. And so a vicious cycle is formed.

Could a different scenario produce the same cycle? The abdominal muscles have been found to work in a feed-forward activation sequence prior to lower limb movement that suggests that they act to "stiffen" the spine in anticipation of forces that will result from lower limb movement (Hodges and Richardson 1997). If the abdominal muscles are weak or impaired in activation then their ability to stabilise the spine, compress the abdominal contents and oppose gravity will be impaired and the effect will be to allow anterior displacement of the abdominal wall and may increase anterior tilt of the pelvis (Baker 1956, DeLuca et al 1998, Evans 1975a, Trost 2004) possibly enough to bring the GRF anterior to the hip. This will require the hip extensor muscles to increase their activity but the gluteus maximus is often weak (Reimers 1973, Waters et al 1974). The gluteus maximus has been shown to be a powerful contributor to the development of hip and knee extension in early single support and therefore if weak will predispose the hip and knee to a flexed posture (Arnold et al 2005a).

hip may already be diminished due to increased hip flexion and the hamstrings will pull the knee into flexion, which will begin the vicious cycle with the rectus femoris. In order to maintain biomechanical alignment the ankle goes into equinus (Bleck 1987, Bottos et al 2001). If surgical intervention to remedy the calf length for lacking heel contact with the ground is undertaken in isolation, the calf will eventually become long and weak post-surgery due to the continued posture of the trunk, pelvis, hips and knees described above. If surgical intervention to the calf is wisely avoided, the calf will eventually become tight and, depending on the integrity of the ligaments and strength of the tibial muscles, may lead to pes valgus and the setting for lever arm dysfunction at the foot and tibia will now be in place. The overlong calf or the presence of lever arm dysfunction at the foot will lead to decreased competence of the plantarflexion knee extension couple and the GRF will be eventually directed posterior to the knee and the vicious cycle between the hamstrings and the rectus femoris will be reinforced. Could it be that proximal weakness has a role in the development of the abnormal biomechanics that initially leads to the child rising onto their toes and with time, the development of crouch gait as postulated above?

The predisposing factor(s) that leads to crouch gait is/ are still not entirely clear, even though most of the severe cohort could be assigned a label of iatrogenic calf lengthening as the reason for their development of crouch gait. Nevertheless two subjects in this group did not have calf lengthenings. What precipitates a decline into crouch gait in the absence of calf surgery? In two of the three subjects who had not had prior calf lengthening, botulinum toxin A injections to the calf had been part of the subjects' management, and possibly excessive or recurrent weakness of the calf post-injection may have led to the development of crouch gait in those subjects. The tight calf may lead to pes valgus with an increase in weight and or height (bone growth), in the presence of ligamentous laxity (Bleck 1987) and/or muscle weakness (Mazur et al 1992). The tight calf will prevent dorsiflexion at the ankle and by pulling the hindfoot into valgus, dorsiflexion will be promoted at the subtalar joint (Bennet et al 1982, Duffy and Cosgrove 2002, Mazur et al 1992, Mosca 1995) and the foot will no longer be a rigid lever on which the GRF can act, leading to

incompetency of the plantarflexion knee extension couple (Duffy and Cosgrove 2002). This is not calcaneus initially, but with time due to longstanding abnormal forces, the calf becomes ineffectual in this position and calcaneus results. However the mechanism of why the calf becomes tight in the first place may be multifactorial as postulated.

# 6.6 CONCLUSION

There was a high incidence of prior calf weakening by either surgery (11/14 subjects) or botulinum toxin A calf injections (2/14) in the cohort with severe crouch gait and this strongly implies their involvement in predisposing a child to the development of a crouch gait.

At the ankle in severe crouch gait, excessive dorsiflexion dominated over the whole gait cycle and plantarflexion was limited at third rocker and consequently excursion of third rocker was limited also. The rate of change from dorsi to plantarflexion in third rocker was also slow and the reduction in power generation at the ankle in pre-swing reflected the influence of these abnormal kinematics. The variability of the foot progression angle and presence of calcaneus, planus and valgus postures of the feet contributed to lever arm dysfunction caused by malalignment in the transverse plane.

At the knee, flexion dominated the gait cycle with increased and premature minimum knee flexion accompanied by limited excursion. Knee kinetics revealed large continuous extensor moments in loading response and pre-swing and the lack of a flexor moment in midstance and so the presence of patella alta and associated pathology as an end product of this scenario would not be surprising due to the inability to off load the knee extensor mechanism. The delay in peak knee flexion indicated that rectus spasticity was a concern but the amplitude of peak flexion was deceptive, as it could be within normal limits. However as the knee extension was severely compromised in stance, the limb was only required to flex a small amount to achieve a value within normal range. The calculation of rate of change between maximum knee extension and

peak knee flexion indicated the reduced ability of the limb in crouch gait to achieve adequate movement into swing phase.

At the hip, the pattern was of increased flexion, with decreased and premature maximum extension and reduced excursion. Kinetic data supported this pattern showing increased and delayed crossover of the extensor moment. Hip power generation was increased in loading response as a possible compensation to aid foot clearance of the contralateral limb in swing. Hip power generation was then decreased in late stance and was associated with an inability to bring the limb into swing. The tendency for the hip to be internally rotated in stance phase, coupled with the variable foot progression angle, contributed to lever arm dysfunction and perpetuated the difficulty in achieving adequate knee extension in stance.

At the pelvis, the excursion of pelvic tilt was increased, possibly as a compensation for lack of hip extension, to help increase stride length and facilitate clearance of the limb in swing. Excursion of the pelvis was not different from normal in the other planes and so limb progression was not compensated for in the transverse plane or limb clearance in swing in the coronal plane.

Having documented baseline functional and technical characteristics of severe crouch gait, it was now possible to investigate the outcome of SEMLS on these parameters at one and five years for children walking in severe crouch gait.

# CHAPTER 7 CAN SEVERE CROUCH GAIT BE CORRECTED?

# 7.1 INTRODUCTION

A lack of knowledge of the causes of crouch gait, makes the decisions regarding intervention to correct crouch gait so difficult. The possible causes of crouch gait were reviewed in Chapter 3 Section 3.5.1 and Chapter 6 Section 6.5. Prevention of the development of crouch gait is the ideal scenario as then correction will not be required, for example avoidance of iatrogenic causes is an obvious target. As a consequence the popularity of isolated calf lengthening in spastic diplegia has waned over the years as more published literature highlights the high incidence of crouch gait in studies detailing long term follow-up to such surgery (Berghof et al 1997, Borton et al 2001, Dillin and Samilson 1983, Rab 1992b, Sutherland and Cooper 1978). The introduction of botulinum toxin A injections was aimed at avoiding early calf lengthening surgery and so preventing the development of crouch gait (Graham et al 2000). The practice of dividing a high percentage of the dorsal nerve rootlets at sacral levels S1 and S2 during an SDR is no longer occurring in most centres, as these nerve rootlets innervate the calf muscle and division of these nerve rootlets will weaken the calf and may lead to crouch gait (Gage 2004f, Molenaers et al 2004).

Does SEMLS that has been tailored to each child who presents walking in severe crouch gait lead to an improvement in crouch gait? Although several studies have examined the effects of SEMLS on heterogenous groups of children with cerebral palsy (Browne and McManus 1987, Nene et al 1993, Norlin and Tkaczuk 1985, Norlin and Tkaczuk 1992, Saraph et al 2005, Saraph et al 2002, Zwick et al 2001), there are no reported studies which have assessed the long term outcome of SEMLS for the correction of severe crouch gait. Therefore the aims of the study were to 1) evaluate the effect of SEMLS on severe crouch gait at one year and five years post-surgery; 2) ascertain whether

the effects at one year post-surgery were maintained at five years; and 3) ascertain whether the surgery resulted in a more normal gait pattern.

# 7.2 METHODS

# 7.2.1 Subjects

This study presents the post-surgical follow-up of the children whose baseline data were reported in Chapter 5. Inclusion criteria were as previously stated.

## 7.2.2 Surgical Intervention

SEMLS was based on the interpretation of the pre-operative gait analysis by the same two consultant paediatric orthopaedic surgeons (HKG and GRN), who subsequently made the decisions regarding the resulting surgical prescription for each subject. The principles of correction for severe crouch gait were to correct "short" contracted muscles and fixed flexion deformities, correct lever arm dysfunction, and retension "long" muscles. A team of four paediatric orthopaedic surgeons undertook all procedures: the same two consultant surgeons, assisted by various paediatric orthopaedic fellows and registrars. Two surgical teams worked simultaneously on one lower limb each of the subject.

The surgical procedures usually consisted of lengthening of contracted muscletendon units, particularly fractional lengthening of the hamstrings (Herring 2002), intramuscular lengthening of the psoas muscle at the pelvic brim (Sutherland et al 1997), transfer of rectus femoris to semitendinosus (Chambers et al 1998), percutaneous lengthening of the adductor longus (Reimers 1980) as well as rotational osteotomies and bony stabilisation procedures to correct lever arm dysfunction. Proximal femoral derotation osteotomies were performed in the intertrochanteric region, with the patient in the prone position, as previously described (Beauchesne et al 1992, Hau et al 2000, Root and Siegal 1980). Stable internal fixation was achieved by using an AO (ASIF) 90° fixed angle blade plate (Hau et al 2000). Distal femoral derotation osteotomies were performed in the supracondylar area, using a tourniquet, with the child in the supine position, as previously described (Cooke et al 1989). Stable internal fixation was achieved by using an AO (ASIF) large fragment six-hole dynamic compression plate. Supramalleolar osteotomy of the tibia was performed as previously described for the correction of excessive lateral tibial torsion (Bache et al 2003, Dodgin et al 1998, Selber et al 2004). Pes valgus was corrected by either os calcis lengthening (Bache et al 2003, Mosca 1995) or subtalar fusion (Bache et al 2003, Dennyson and Fulford 1976).

A first generation cephalosporin was commenced at the induction of anaesthesia and continued for the first twenty-four hours post-operatively. All patients had analgesia administered by continuous epidural infusion of bupivacaine and fentanyl, for three to five days after surgery. Post-operative analgesia was jointly supervised by the surgical and pain management teams.

#### 7.2.3 Post-operative Management

After foot and ankle surgery, the lower limbs were immobilised in below-knee plaster slabs and knee immobilisers were used to maintain knee extension. Plaster slabs were removed to permit wound inspection and radiographs of osteotomies at three weeks after surgery, followed by application of fibreglass below knee casts. Subject to satisfactory wound healing and radiographic appearances, weight bearing as tolerated was encouraged at that time. At six weeks after surgery, casts were removed and proximal entry ground reaction ankle foot orthoses (GRAFOs) were fitted (Figure 7.1). Radiographs of all osteotomies were obtained and full weight bearing encouraged. Knee immobilisers were used continuously for the first six weeks, and at night only for another six months, to reduce the risk of recurrent knee contractures. They were removed for therapy and replaced at the end of therapy sessions.



Figure 7.1 Proximal entry ground reaction ankle foot orthosis (GRAFOs).

# 7.2.4 Rehabilitation

Physiotherapy commenced whilst the epidural analgesia was in place, in order to provide the children with the confidence to move without the knee immobilisers. The children were commenced on passive and active range of motion exercises. If femoral osteotomies were included in the surgical programme, children were discharged when they could stand for transfers. They were permitted to weight-bear as tolerated when the incisions had healed. If SEMLS included os calcis lengthening and supramalleolar tibial osteotomies, children were discharged when they could transfer from the bed to a wheelchair and were not permitted to weight-bear for a period of three weeks.

Children received individually tailored, community based rehabilitation programmes initially incorporating three to four sessions of physiotherapy and one or two sessions of hydrotherapy per week, from six weeks after surgery (at the time of cast removal) for the next 12 weeks. The children were gradually advanced from a passive to an assisted range of motion protocol and finally to a resistance program. Physiotherapy sessions were reduced to weekly after six months with encouragement given to the subjects to participate also in physical recreational activities such as bicycle riding, swimming, horse riding, local gym programmes that they enjoyed. Post-operatively as per the protocol for the Hugh Williamson Gait Laboratory, each subject was reviewed by video observation in the gait laboratory at three monthly intervals so that appropriate changes could be made to orthoses, assistive devices and the physiotherapy regime to promote each subject's rehabilitation process.

# 7.2.5 Study Procedure

Data collected were the same as those reported in Chapter 6. Post-operative changes were evaluated at one and five years using the same parameters described in Chapter 6 Section 6.2.3.

## 7.2.6 Statistical Analysis

Statistical analysis was conducted to compare performance between changes from:

- baseline to one year post-surgery.
- baseline to five years post-surgery.
- one year to five years post-surgery.
- five years post-surgery to normal values.

Linear regression models with robust standard errors were used, to allow for the repeated measurements from individual patients over time and to account for the correlation between limbs by the inclusion of both limbs from each subject (Forbes and Wolfe 2001). P values and 95% confidence intervals of the estimated difference in means were calculated. P values of 0.05 were considered to be statistically significant, which is standard practice (Norman and Streiner 2000, Portney and Watkins 2000, Sterne and Davey Smith 2001). Variables that were analysed using this method were temporal-spatial parameters, physical examination and three dimensional gait analysis variables.

Comparisons across time in mobility status were described by odds ratios calculated from ordered logistic regression with robust standard errors. Logistic regression can analyse categorical dependent variables that are dichotomous or polytomous and the independent variable can have one or more values (Anagnoson and DeLeon 1997). In the data here the interest was in the

comparison across time in mobility at baseline, one and five years post-SEMLS according to specified mobility scales and the rating accorded at each time point.

Statistical analysis was undertaken using the Stata 7 (StataCorp) software package (StataCorp. 2001).

The issues relating to missing data and to limited subject numbers for some data points were considered in consultation with a statistician and the following was decided. All of the four subjects who had missing data (two subjects at each time period post-surgery) were considered to be missing due to random chance rather than bias in the data. The two subjects with missing data at one year only, were unavailable due to administrative error, which meant that these two subjects were not approached to attend a one year follow-up appointment. Of the two subjects with missing data at the five year time period, one lives interstate and was not able to be contacted and the other subject had not reached the five year mark at the time of this analysis. The small number of subjects able to provide kinetic data (nine subjects at pre-SEMLS, four at one year and seven at five years post-SEMLS) raised questions regarding the suitability of these data for statistical analysis. As this group of patients were unique due to the precise definition of severe crouch gait, and the limited ability to collect prospective data from such a group again in the future, it was decided to analyse kinetic data to examine changes from baseline to five years and at this point to compare cerebral palsy children with normally developing children, and ignore the one year kinetic data. Any inferences made from the results of the kinetic data would be tempered by recognition that there were a limited number of subjects contributing to the data set.

# 7.3 RESULTS

The demographics for the normal and severe crouch subjects were the same as those reported in Chapter 6. Data collection at baseline was conducted between 1995 and 1999, and SEMLS was undertaken between 1995 and 2000. Two

subjects were not available to participate in the one year follow-up, but they participated in the five year follow-up study. Therefore there were 12 complete data sets at the one year follow-up study and 12 complete sets at the five year follow-up, and 10 complete sets comprising pre-operative, one and five year data (Figure 7.2).

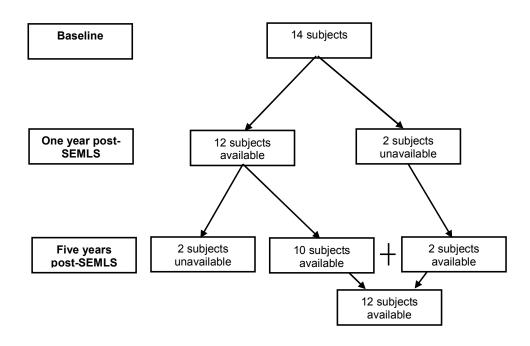


Figure 7.2 Diagram detailing the inclusion of the subjects who participated in the severe crouch SEMLS study.

Mean time from baseline data collection to SEMLS was 0.7 years (range 0.2-2.5 years), from SEMLS to follow-up at one year was 1.1 years (range 0.6-1.4 years) and at five years was 5.2 years (range 4.3- 5.8 years).

Mean age of subjects at baseline data collection was 12.1 years (range 7.6-16.1 years), at SEMLS 12.7 years (range 7.9-16.3 years), at the one year follow-up 13.3 years (range 8.9-17.2 years) and at five years, 17.7 years (range 12.9-21.3 years).

The majority of subjects were peri-pubertal and increased in height by a mean of 8.1 cm (SD 5.3 cm) from baseline to one year post-SEMLS and 19.2 cm (SD

10.8 cm) from baseline to five years post-SEMLS, with corresponding mean increases in weight of 5.8 kg (SD 4.0 kg) and 15.0 kg (SD 5.3 kg) respectively.

# 7.3.1 SEMLS Intervention

Surgery prior to SEMLS was detailed in Table 6.2 in Chapter 6. The surgical procedures for SEMLS for each subject are shown in Table 7.1. Overall, SEMLS consisted of a mean of seven procedures per subject (range 5-10). There were 21 bony surgical procedures and 64 soft tissue surgical procedures. Any additional surgery that was undertaken between six months after SEMLS and prior to the five year assessment is documented in Table 7.2. The most frequent procedure was for ingrown toenails (15 procedures).

						SEMLS SURGERY	GERY					
Code	Age at Sx	No Sx	SEMLS									
~	10.3	2	POTB		MHS	RFT-ST		R SMO				
5	12.8	~ ~	POTB	ADD L	SHM		R FDO(D)					
ю	13.3	10	POTB		SHM		~		Os calcis Lx	Strayer	Peron br length	_
4	14.5	9	POTB	ADD L			FDO(P)varus	S		•	Peron br length	
S	14.2	2		ADD L	SHM		L FDO(D)				•	
9	15.9	5		Repeat MHS	_	LHS-post kn release		R SMO				
7	16.3	თ	POTB		MHS LHS	6	L FDO(D)					IGTN
œ	11.0	7			MHS LHS	S	R FDO(D)	R SMO	L STJ fusion			
6	10.7	ω			MHS LHS,	ù,	FDO(D)		STJ fusion			
10	12.4	ω			MHS LHS	S RFT-ST	FDO(D)					
77	11.4	8	POTB		MHS LHS,	ù,		SMO				
12	7.9	9			MHS LHS	S RFT-ST/ gracilis	gracilis					
13	16.2	8			MHS LHS		FDO(P)					
14	10.6	9		ADD L	SHM	Prox rect						
mean/ median	12.7	7										
min	7.9	5										
max	16.3	10										
No. limbs			12	8	26 16	3 10	12	5	ъ	7	4	2
No. subjects			9	4	13 8	S	ø	4	ო	<del>.                                    </del>	2	~

Chapter 7

223

Surgery undertaken post-SEMLS by subject code and type of surgery. Table 7.2

		Surg	Surgery Post-SEMLS		
	Type of Surgery:				
Code	-		Bony Surgery	Toe Surgery	
~			L Dega acetab osteot, STJ fusion		
7	L prox rect		L FDO(D)	L IGTN	
e	MHS, LHS				
4				1st MTP fusion, IGTN x3	x3
S	Prox rect release, L tib post divn x	o post divn x2 , L tib ant Tx		multiple metatarsal and toe Sx	d toe Sx
9	Btx HMS, infrapatellar bursa injection	tion			
7				MTP fusion IGTN x2, L IGTN x2	. IGTN x2
œ	Prox rect		R prox tibial epiphysiodesis		
6	Prox rect				
10	POTB			R IGTN	
1	Prox rect		FDO(P)		
12	Nil				
13				B IGTN	
4	RFT-ST				
יבו פר. 19	16 ICTN procedures (5 subjects) -2 subjects had 6 surgeries each -3/5 had MTD fusion also	ote had 6 euro	arias aach 2/5 had MTD fusion als	c	
		יטנט וומט ט טטוא		0	
6 M I F	6 MTP tusion (3 subjects)				
9 prox	9 prox rectus release (5 subjects) 2 RFT (1 subjects)	(1 subjects)			
e.,	psoas over the brim lateral hamstrings lengthening	ADD-L RFT-ST	adductor longus lengthening rectus femoris transfer to semitendinosus		medial hamstrings lengther proximal rectus release
ant Tx ja acetab	tibialis anterior transfer Dega acetabular osteotomy	Tib post divn FDO(P)	tibialis posterior division femoral derotation osteotomy proximal	Peron br Lx FDO(D)	Peroneus brevis lengthenin femoral derotation osteoton
O P fusion	supramalleolar osteotomy metatarsal phalangeal fusion	STJ fusion IGTN	subtalar joint fusion ingrown toe nail surgery	Os calcis Lx Btx HMS	os calcis lengthening botulinum toxin A to hamstr
	•		. ,		

224

medial hamstrings lengthening proximal rectus release Peroneus brevis lengthening femoral derotation osteotomy distal os calcis lengthening botulinum toxin A to hamstrings

Tib ant Tx Dega acetab SMO MTP fusion

POTB LHS

### 7.3.2 Activities- Mobility Status

## **Orthotic Use**

All severe crouch gait subjects wore orthoses at one year post-SEMLS, with the majority wearing GRAFOs; whilst at five years post-SEMLS only five subjects continued to wear orthoses and only one of these subjects was wearing GRAFOs (Table 7.3).

Table 7.3Orthotic use pre- and post-SEMLS intervention for the subjects withsevere crouch gait.

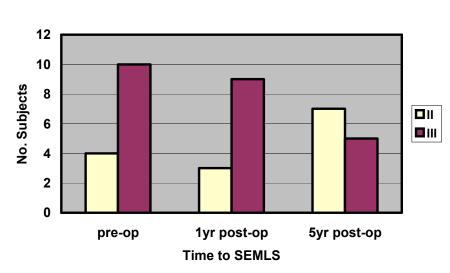
		Orthotic Use	
Code	Pre-op	1yr post-op	5yr post-op
1	Solid AFOs	GRAFOs	Solid AFOs
2	nil	GRAFOs	nil
3	nil	GRAFOs	nil
4	nil	-	nil
5	nil	GRAFOs	Solid AFOs
6	GRAFOs	GRAFOs	-
7	nil	-	nil
8	nil	GRAFOs	nil
9	nil	GRAFOs	Hinged AFOs
10	nil	HingedAFOs	nil
11	Hinged AFOs	GRAFOs	GRAFOs
12	nil	Solid AFOs	Solid AFOs
13	nil	GRAFOs	nil
14	Solid AFOs	Solid AFOs	-

#### **GMFCS**

Statistically there was no significant change in GMFCS level from baseline to one or five years post-SEMLS. However when the GMFCS level at five years post-SEMLS was compared to one year, there was a 7-8 times greater odds that a subject would be at a level of II (rather than III) at five years. (Table 7.4 & Figure 7.3).

	GMF	CS	
Time Comparison	Odds Ratio	P value	95% CI
1yr - pre	1.2	0.78	0.3, 4.4
5yr - pre	0.3	0.10	0.1, 1.3
5yr - 1yr	0.2	0.018	0.1, 0.8

Table 7.4 GMFCS pre, one and five years post-SEMLS for the subjects with severe crouch gait.



# **GMFCS Status**

Figure 7.3 Rating of GMFCS at pre, one and five years post-SEMLS for the subjects with severe crouch.

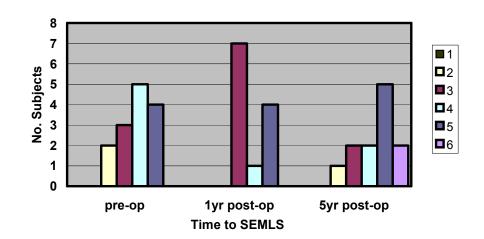
# Functional Mobility Scale

The three distances of 5, 50 and 500m on the FMS were individually analysed for change post-SEMLS.

• 5 metres: At one year there was no significant change from baseline but by five years there was approximately a threefold greater odds that a subject had improved in ambulatory status towards independent walking over 5 metres from the baseline rating, for example: a change in rating to a FMS of 5 (rather than 4) (Table 7.5 & Figure 7.4).

Functional Mot	oility Scale 5m	
Odds Ratio	P value	95% CI
0.9	0.68	0.5, 1.6
3.1	0.02	1.2, 8.0
3.6	0.04	1.0, 12.2
	Odds Ratio 0.9 3.1	0.9 0.68 3.1 0.02

Table 7.5 FMS 5m pre, one and five years post-SEMLS for the subjects with severe crouch gait.



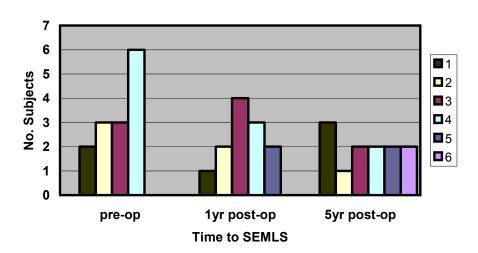
**5m FMS Status** 

Figure 7.4 Rating of 5m FMS at pre, one and five years post-SEMLS for the subjects with severe crouch gait.

• 50 metres: There was no significant change in performance over time. However four subjects became independent in walking at five years (none had been pre-SEMLS) but three subjects used wheelchairs over this distance at five years, whereas prior to SEMLS only two subjects were using wheelchairs (Table 7.6 & Figure 7.5).

	Functional Mob	ility Scale 50m	
Time Comparison	Odds Ratio	P value	95% CI
1yr - pre	1.4	0.3	0.7, 2.6
5yr - pre	1.8	0.3	0.5, 6.0
5yr - 1yr	1.3	0.7	0.4, 4.7

Table 7.6FMS 50m pre, one and five years post-SEMLS for subjects with severecrouch gait.



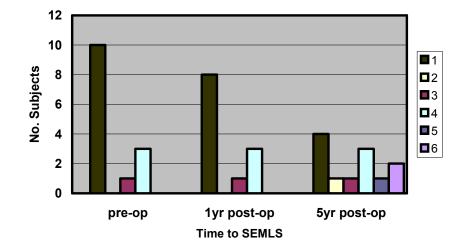
# **50 FMS Status**

Figure 7.5 Rating of 50m FMS at pre, one and five years post-SEMLS for the subjects with severe crouch gait.

• 500m: Significant improvement in ambulatory status was found at five years compared to baseline and one year post-SEMLS but not between baseline and one year. The 500m FMS showed that compared to baseline, there was a five times greater odds that a subject would have a rating of 5 or 6 (rather than 4 or lower) at five years. At five years there was more independent ambulation (none prior to SEMLS and three subjects at five years) and less use of wheelchairs (10m at baseline and four at five years) (Table 7.7 & Figure 7.6).

	Functional Mobi	lity Scale 500m	
Time Comparison	Odds Ratio	P value	95% CI
1yr - pre	1.2	0.2	0.9, 1.7
5yr - pre	5.3	0.002	1.8, 15.8
5yr - 1yr	4.3	0.01	1.4, 13.2

Table 7.7 FMS 500m pre, one and five years post-SEMLS for subjects with severe crouch gait.



## 500m FMS Status

Figure 7.6 Rating of 500m FMS at pre, one and five years post-SEMLS for subjects with severe crouch gait.

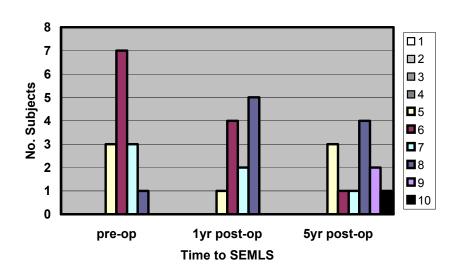
# Functional Assessment Questionnaire

The FAQ did not show significant change at five years from baseline and one year post-SEMLS but there was significant change between baseline and one year. This was attributable to the increased spread of data at five years across the rating scores; at level 5, the subject numbers did not change but the number of subjects at levels 9 and 10 increased. This spread of data is also seen in the 95% confidence intervals for pre-SEMLS and one year post- with five years post-SEMLS. Whereas at one year post-SEMLS subject numbers dropped at levels 5, 6 and 7 and increased at level 8. Compared to baseline, this

represented a threefold greater odds of a rating 8 (or higher) rather than 7 (or lower) (Table 7.8 & Figure 7.7).

	Functional Assessm	nent Questionnai	re
Time Comparison	Odds Ratio	P value	95% CI
1yr - pre	2.9	0.007	1.3, 6.3
5yr - pre	5.7	0.07	0.9, 35.9
5yr - 1yr	2.0	0.42	0.4, 10.1

Table 7.8FAQ pre, one and five years post-SEMLS for subjects with severecrouch gait.



**FAQ Status** 

Figure 7.7 Rating of FAQ at pre, one and five years post-SEMLS for the subjects with severe crouch gait.

# 7.3.3 Body Structure and Function

#### **Physical Examination**

The physical examination parameters are reported in the following section 7.3.3 in conjunction with kinematic and kinetic data. This was done to facilitate the complete reporting of parameters relating to different anatomical levels in the different planes.

## **Temporal-spatial Parameters**

Normalised speed remained decreased and did not change from baseline to one and five years post-SEMLS. Normalised stride length remained reduced, timing of toe off in the gait cycle remained delayed, as was opposite foot off, and opposite foot contact continued to be premature (Tables 7.9 & 7.10).

Table 7.9 Data (means ± standard deviations) for the severe crouch group at pre, one and five years post-SEMLS for temporal-spatial parameters. Includes normal data for comparison. Statistically significant changes indicated by shaded data.

		SEMLS status		
Parameters	Pre	1 year	5 year	Normals
	mean ±SD	mean ±SD	mean ±SD	mean ±SD
Velocity (m/s)	0.7 ± 0.2	0.7 ± 0.3	0.8 ± 0.3^	1.3 ± 0.1
Normalised velocity	0.018 ± 0.006	0.019 ± 0.007	0.019 ± 0.007^	0.034 ± 0.004
Stride length (m)	0.7 ± 0.2	$0.8 \pm 0.2^{*}$	0.9 ± 0.2^	1.2 ± 0.1
Normalised stride length	0.005 ± 0.0001	0.006 ± 0.0010	0.005 ± 0.0014^	0.008 ± 0.0006
Toe off %cycle	68 ± 5	68 ± 4	67 ± 5^	60 ± 2
Opposite foot off %ST	26 ± 7	27 ± 6	26 ± 7^	16 ± 3
Opposite foot contact %ST	73 ± 6	75 ± 5	76 ± 6^	84 ± 2

 significance at P value<0.05 between pre- and 1 yr post-SEMLS</li>
 significance at P value<0.05 between 1 and 5 yr post-SEMLS</li>
 significance at P value<0.05 between 1 and 5 yr post-SEMLS</li> significance at P value<0.05 between pre- and 1 yr post-SEMLS

significance at P value<0.05 between 5 yr post-SEMLS and normal data

SD standard deviation

ST SW stance phase

swing phase

Table 7.10 Comparison of difference in means for severe crouch data for temporal-spatial parameters at pre, one and five years post-SEMLS (plus normal data). Statistically significant changes indicated by shaded data.

	Diffe	erence in	Difference in mean change between pre, 1 and 5 years post-SEMLS and to normal data	en pre, 1	and 5 years post-SE	MLS and	<u>to normal data.</u>	
	Pre – 1yr		Pre – 5yr		<u> 1yr – 5yr</u>		<u>5yr – normal</u>	
Parameters	Est mean diff (95% Cl)	P value	Est mean diff (95% Cl)	Р value	Est mean diff (95% Cl)	Р value	Est mean diff (95% Cl)	Р value
Velocity (m/s)	0.05 (-0.11, 0.21)	0.54	0.09 (-0.08, 0.26)	0.29	0.04 (-0.11, 0.20)	0.59	-0.54 (-0.72, -0.37)	<0.001
Normalised velocity	0.001 (-0.004, 0.006)	0.64	0.001 (-0.003, 0.006)	0.59	0.0001 (-0.004, 0.004)	0.94	-0.015 (-0.020, -0.011)	<0.001
Stride length (m)	0.10 (0.01, 0.20)	0.03	0.14 (0.00, 0.27)	0.04	0.03 (-0.08, 0.15)	0.54	-0.36(-0.51, -0.22)	<0.001
Normalised stride length	0.0005 (-0.0002, 0.0012)	0.16	0.0003 (–0.0006, 0.0012)	0.56	-0.0002 (-0.0010, 0.0005)	0.48	-0.0030 (-0.0039, -0.0021)	<0.001
Toe off %cycle	-0.6 (-3.4, 2.2)	0.67	-1.5 (-5.2, 2.2)	0.41	-0.9 (-4.6, 2.8)	0.62	6.9 (3.8, 10.0)	<0.001
Opposite foot off %ST	1.2 (-2.6, 5.1)	0.52	0.14 (-3.9, 4.2)	0.94	-1.1 (-5.7, 3.5)	0.63	9.7 (6.1, 13.3)	<0.001
<b>Opposite foot contact %ST</b>	2.4 (-0.6, 5.3)	0.11	2.8 (-1.3, 7.0)	0.18	0.5 (-3.3, 4.3)	0.80	-8.1 (-11.1, -5.1)	<0.001
SD standard deviation								

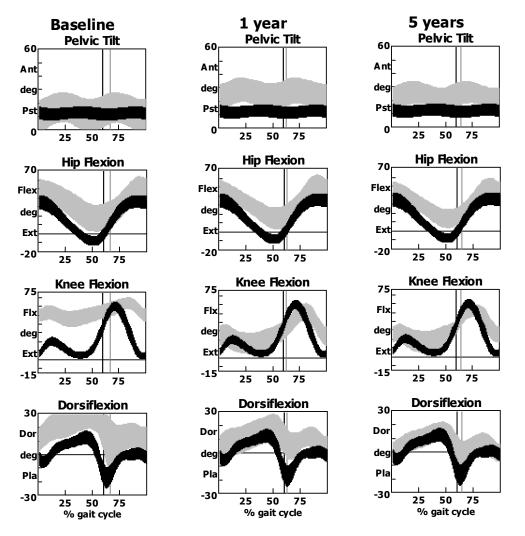
estimated mean difference between crouch and normal data 95% confidence intervals of the estimated mean difference between crouch and normal data stance phase swing phase Est. mean diff. 95% Cl ST SW

## Kinematics and Kinetics (and Physical Examination)

Kinematic data were collected at the two time periods post-SEMLS for the crouch subjects who presented for data collection (n=12 each time), however kinetic data could only be collected for four subjects at the one year mark and seven subjects at five years post-SEMLS. Therefore due to the small number of subjects at the one year mark, kinetic data were not analysed for that time point.

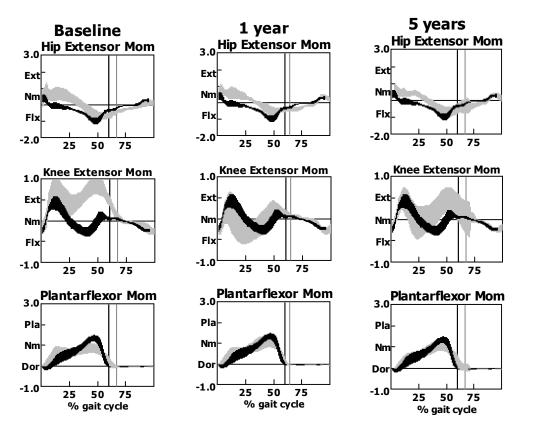
# Sagittal Plane

Figures 7.8, 7.9 and 7.10 illustrate sagittal plane kinematics, sagittal plane moments and total power at baseline, one and five years post-SEMLS.



# **Sagittal Kinematics**

Figure 7.8 Sagittal plane kinematics pre, one and five years post-SEMLS. Grey band represents the severe crouch data and the black band, normal data. Note the improvement at the ankle (dorsiflexion reduced) and knee (extension increased) with no change at the hip and deterioration at the pelvis (anterior tilt increased).



# **Sagittal Moments**

Figure 7.9 Sagittal plane moments pre, one and five years post-SEMLS. Grey band represents the severe crouch data and the black band, normal data. Note the reduction in the knee extensor moment.

# **Total Powers**

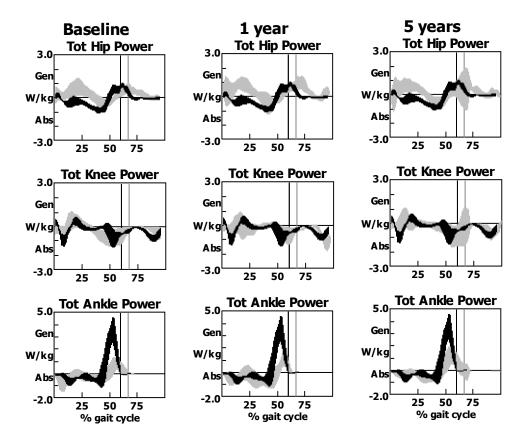


Figure 7.10 Kinetics- powers pre, one and five years post-SEMLS. Grey band represents the severe crouch data and the black band, normal data. Note the small increase in ankle power (A2).

#### Pelvis

Maximum and minimum pelvic tilt increased in anterior tilt at one and five years post-SEMLS compared to pre-operative values (Tables 7.11 & 7.12). Maximum pelvic tilt was not different at five years compared to one year, however there was a trend to a reduction in anterior tilt at five years. Pelvic excursion remained increased post-SEMLS at each time point. These pelvic parameters hence remained significantly different from normal and, in the case of anterior tilt, showed further deterioration from pre-SEMLS values.

Table 7.11 Comparison of means ± standard deviations for severe crouch data for the pelvic and hip parameters at pre, one and five years post-SEMLS. Includes normal data for comparison. Statistically significant changes indicated by shaded data.

		SEMLS status		
Parameters	Pre	1 year	5 year	Normals
	mean ±SD	mean ±SD	mean ±SD	mean ±SD
Max ant pelvic tilt	18 ± 12	$30 \pm 9^*$	26 ± 9†^	14 ± 4
Min ant pelvic tilt	11 ± 12	23 ± 10*	20 ± 8†^	11 ± 5
Mean pelvic tilt	14 ± 11	27 ± 9*	23 ± 8†^	13 ± 4
Pelvic excursion	10 ± 3	9 ±4	9 ± 3^	4 ± 1
Max hip ext ST	18 ± 15	15 ± 11	15 ± 10^	-8 ± 5
Hip excursion	35 ± 7	39 ± 7	37 ± 8^	45 ± 5
Max hip ext moment	1.1 ± 0.5	UA	0.9 ± 0.2^	0.7 ± 0.2
Max hip power gen early ST	1.0 ± 0.5	UA	1.0 ± 0.5^	$0.5 \pm 0.4$
Max hip power gen late ST	0.8 ± 0.3	UA	1.1 ± 0.3	1.1 ± 0.4
Crossover hip ext moment to flex %ST	56 ± 10	UA	54 ± 6^	34 ± 13
FFD hip	-21 ± 11	-13 ± 8*	-10 ± 5†^	0 ± 0*

Statistical analysis by linear regression models with robust standard errors

- significance at P value<0.05 between pre- and 1 yr post-SEMLS</li>
  significance at P value<0.05 between 1 and 5 yr post-SEMLS</li>
  significance at P value<0.05 between 1 and 5 yr post-SEMLS</li>

significance at P value<0.05 between 5 yr post-SEMLS and normal data

SD standard deviation

significance at P value<0.05 between pre- and 1 yr post-SEMLS

ST stance phase

SW swing phase

UA unable to assess due to n=4 subjects for kinetic data at 1 year

	_,	Difference	Difference in mean change between pre, 1 and 5 years post-SEMLS and to normal data	en pre, 1 a	nd 5 years post-SEML	S and to r	iormal data.	
	<u>1yr to pre-op</u>		5yr to pre-op		5yr to 1yr		5yr to normal	
Parameters	Est mean diff (95% CI)	<i>P</i> value	Est mean diff (95% CI)	<i>P</i> value	Est mean diff (95% CI)	<i>P</i> value	Est mean diff (95% Cl)	P value
Max ant pelvic tilt	12 (7, 16)	<0.001	8 (0.3, 15)	0.04	-4 (-9, 1)	0.11	12 (6, 18)	<0.001
Min ant pelvic tilt	12 (8, 16)	<0.001	9 (2, 16)	0.01	-3 (-8, 2)	0.21	9 (3, 14)	0.004
Mean pelvic tilt	12 (8, 16)	<0.001	9 (1, 16)	0.02	-4 (-8, 1)	0.13	11 (5, 16)	0.001
Pelvic excursion	-1.2 (-3.1, 0.8)	0.23	-1.3 (-3.1, 0.4)	0.13	-0.2 (-1.8, 1.5)	0.84	4.8 (2.7, 6.9)	<0.001
Max hip ext ST	-3 (-9, 3)	0.34	-3 (-11, 4)	0.40	-0.4 (-5, 4)	0.85	22 (16, 29)	,0.001
Hip excursion	3 (-0.9, 8)	0.12	2 (-4, 7)	0.58	-2 (-6, 2)	0.40	-8 (-13, -3)	0.002
Max hip ext moment	UA	NA	-0.23 (-0.50, 0.04)	0.09	ЛА	NA	0.18 (0.02, 0.34)	0.029
Max hip power gen early ST	UA	NA	-0.00 (-0.36, 0.35)	0.99	ЛА	NA	0.52 (0.20, 0.85)	0.003
Max hip power gen late ST	UA	NA	0.35 (-0.04, 0.74)	0.07	ЛА	NA	-0.01 (-0.33, 0.31)	0.93
Crossover hip ext moment to flex %ST	NA	N	-2 (-7, 3)	0.47	ПA	NA	20 (13, 28)	<0.001
FFD hip	8 (4, 12)	<0.001	11 (5, 18)	0.001	3 (-1, 8)	0.15	-10 (-13, -7)	<0.001
SD standard deviation Est. mean diff. estimated mean dif 95% Cl 95% confidence int	ference between crouch ervals of the estimated n	and normal data nean difference b	ata e between crouch and norm:	UA al data	unable to assess due t	o n=4 subje	unable to assess due to n=4 subjects for kinetic data at 1 year	

238

<u>Hip</u>

Maximum hip extension in stance did not improve after SEMLS but remained increased compared to normal values. The hip extensor moment after SEMLS was not altered from pre-operative values and remained increased with respect to normal values. The timing of crossover of the extensor moment to a flexor moment was also not altered post-SEMLS and thereby remained delayed in respect to normal parameters. The hip power generation in loading response was similar to the pre-operative level at five years but this was still increased compared to normal. Pre-intervention, the hip power generation in late stance was decreased compared to normal and although there was no statistically significant difference in mean values found between pre-SEMLS and five years after SEMLS, the values of the 95% confidence interval indicated an increase in power generation at five years that was similar to normal values, indicating improvement towards normal. Fixed flexion deformity assessed by Thomas test showed improvement post-SEMLS with reduction in hip contracture present but this was not sufficient to reach normal range. (See Tables 7.11 & 7.12).

Examination of the differences in pelvic and hip variables for subjects with assistive devices and those without assistive devices, was undertaken for clinical reasons as explained previously in Chapter 6 Section 6.3.3. The statistical analysis was used to assess trends in the data but it was recognised that there was not a large enough sample size due to the small number of subjects in the two groups. There were seven subjects in each group at baseline and due to missing data at five years post-SEMLS, there were only five subjects in the group using assistive devices at that time. A subject was designated to the group using assistive devices if they could only walk with an assistive device at either time period. Tables 7.13 and 7.14 and Figure 7.11 show results for these two groups of subjects. In both groups, maximum pelvic tilt showed a trend towards more anterior tilt at one year post-SEMLS. Those who were independent of assistive devices continued to show more anterior pelvic tilt at five years after surgery, while those who used assistive devices did show slight return towards pre-operative range (Table 7.13). While both groups of subjects did not show any improvement in maximum hip extension in stance, fixed flexion contracture of the hip was reduced. The unaided subjects showed a

239

greater clinical improvement in the hip fixed flexion contracture at one year that appeared to be maintained at five years, whereas the group using assistive devices did not show as much improvement at one year as the unaided group. However by five years post-surgery this group continued to improve and had achieved degrees of improvement similar to the unaided subjects.

Table 7.13 Comparison of means ± standard deviations for severe crouch data for the pelvic and hip parameters at pre, one and five years post-SEMLS for subjects without and those with assistive devices. Includes normal data for comparison. Shaded data indicate statistically significant changes.

		No Aids			Aids		
Parameters	Pre	1 year	5 year	Pre	1 year	5 year	Normals
	mean ±SD	mean ±SD	mean ±SD	mean ±SD	mean ±SD	mean ±SD	mean ±SD
Max ant pelvic tilt	13.8	25.6*	23.3 <sup>^</sup>	23.5	33.1*	29.6 <sup>^</sup>	14.1
Min ant pelvic tilt	6.3	18.2	16.5 <sup>^</sup>	15.4	26.4*	24.8 <sup>^</sup>	11.3
Pelvic excursion	9.7	9.2	9.5v	10.4	8.7*	7.6†^	3.9
Max hip ext ST	13.5	8.1	10.2 <sup>^</sup>	22.4	20.3	21.2 <sup>^</sup>	-7.6
Hip excursion	34.7	41.2*	39.8	36.3	37.1	33.1^	44.9
FFD hip	-20.4	-8.5*	-9.7†^	-21.8	-16.1*	-9.7†^	0

Statistical analysis by linear regression models with robust standard errors \* significance at P value<0.05 between pre- and 1 yr post-SEMLS † significance at P value<0.05 between pre- and 5 yr post-SEMLS ‡ significance at P value<0.05 between 1 and 5 yr post-SEMLS ^ significance at P value<0.05 between 5 yr post-SEMLS and normal data

standard deviation stance phase swing phase st sd

		Differe	Difference in mean change between pre, 1 and 5 years post-SEMLS and to normal data	een pre, 1	and 5 years post-SEMLS	s and to no	<u>rmal data</u>	
Parameters <u>No Aids</u>	<u>1yr to pre-op</u> Est mean diff (95% Cl)	<i>P</i> value	<u>5vr to pre-op</u> Est mean diff (95% Cl)	P value	<u>5yr to 1yr</u> Est mean diff (95% Cl)	P value	<u>5yr to normal</u> Est mean diff (95% CI)	P value
Max ant pelvic tilt	13 (4, 21)	0.005	11 (-1, 22)	0.06	-2 (-6, 2)	0.24	9 (3, 15)	0.005
Min ant pelvic tilt	12 (4, 20)	0.008	10 (-1, 21)	0.07	-2 (-7, 3)	0.49	5 (0, 10)	0.05
Pelvic excursion	0 (-5, 4)	0.82	0 (-2, 2)	0.89	0 (-3, 4)	0.85	6 (2, 9)	0.002
Max hip ext ST	-6 (-18, 7)	0.37	-3 (-16, 9)	0.57	2 (-5, 9)	0.52	18 (10, 25)	<0.001
Hip excursion	6 (1, 12)	0.02	5 (-2, 12)	0.14	-1 (-6, 4)	0.58	-5 (-11, 1)	0.09
FFD hip	12 (5, 18)	0.001	11 (2, 20)	0.02	-1 (-6, 3)	0.57	-10 (-13, -6)	<0.001
Aids								
Max ant pelvic tilt	10 (6, 13)	<0.001	6 (-5, 17)	0.27	-4 (-13, 6)	0.43	15 (6, 25)	0.003
Min ant pelvic tilt	11 (7, 15)	<0.001	9 (-1, 20)	0.07	-2 (-10, 7)	0.72	14 (4, 23)	0.006
Pelvic excursion	-2 (-4, 0)	0.05	-3 (-5, -1)	0.003	-1 (-3, 1)	0.22	4 (2, 6)	0.001
Max hip ext ST	-2 (-7, 3)	0.41	-1 (-12, 9)	0.82	1 (-8, 10)	0.84	29 (21, 37)	<0.001
Hip excursion	1 (-5, 6)	0.76	-3 (-12, 6)	0.46	-4 (-11, 3)	0.28	-12 (-17, -6)	<0.001
FFD hip	6 (2, 9)	0.004	12 (3, 21)	0.01	6 (-1, 13)	0.07	-10 (-15, -4)	0.001

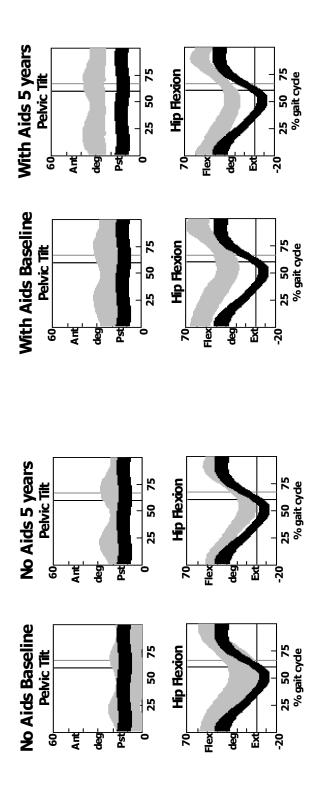
Comparison of difference in means for severe crouch data for pelvic and hip parameters at pre, one and five years post-SEMLS for subjects Table 7.14

Chapter 7

242

Figure 7.11 Pelvic and hip sagittal plane kinematics for subjects with severe crouch gait, walking without assistive devices and those walking with assistive devices pre, one and five years post-SEMLS. Grey band represents the severe crouch data and the black band, normal data.





### Knee

Knee extension at initial contact and minimum flexion in stance phase showed substantial improvement post-SEMLS, but not sufficient to reach the range of normal values (Tables 7.15 & 7.16, Figure 7.12). There was a deterioration between one and five years post-SEMLS, implying that the minimum knee flexion at one year was possibly not being maintained at five years. However this change was not statistically significant. Knee excursion increased post-SEMLS but did not reach normal range (Figure 7.13).

Table 7.15 Comparison of means ± standard deviations for severe crouch data for the knee parameters at pre, one and five years post-SEMLS. Includes normal data for comparison. Statistically significant changes indicated by shaded data.

		SEMLS status		
Parameters	Pre	1 year	5 year	Normals
	mean ±SD	mean ±SD	mean ±SD	mean ±SD
Kn ext initial contact	51 ± 7	26 ± 10*	28 ± 11†^	7 ± 5
Min kn flex ST	46 ± 9	14 ± 11*	19 ± 13†^	5 ± 4
Kn excursion	20 ± 8	38 ± 10*	35 ± 11†^	57 ± 5
Max peak kn flex SW	66 ± 7	52 ± 10*	55 ± 7†^	62 ± 6
Max kn flexor moment	$0.3 \pm 0.3$	UA	-0.1 ± 0.3†	-0.2 ± 0.2
Max kn ext mom 1 <sup>st</sup> peak	$0.8 \pm 0.3$	UA	$0.6 \pm 0.3$	0.5 ± 0.2
Max kn ext mom 2 <sup>nd</sup> peak	1.0 ± 0.2	UA	0.6 ± 0.2†^	0.3 ± 0.1
Max kn power gen early ST	0.7 ± 0.7	UA	$0.9 \pm 0.6$	0.8 ± 0.4
Max kn power absorp late ST	-1.2 ± 0.5	UA	-1.2 ± 0.6	-1.2 ± 0.6
FFD knee	-18 ± 8	-7 ± 7*	-7 ± 8†^	0.7 ± 2
Popliteal angle	68 ± 14	58 ± 17	56 ± 12^	39 ± 12
Duncan Ely (fast)	all +ve	all +ve	all +ve	all -ve

significance at P value<0.05 between pre- and 1 yr post-SEMLS

significance at P value<0.05 between pre- and 5 yr post-SEMLS</li>
 significance at P value<0.05 between 1 and 5 yr post-SEMLS</li>
 significance at P value<0.05 between 5 yr post-SEMLS and norm</li>

significance at P value<0.05 between 5 yr post-SEMLS and normal data

SD ST standard deviation

stance phase

SW swing phase

UA unable to assess due to n=4 subjects for kinetic data at 1 year

	-	Difference	Difference in mean change between pre, 1 and 5 years post-SEMLS and to normal data.	sii pre, i o	alla o yeals post-ocivit			
	1yr to pre-op		5yr to pre-op		5yr to 1yr		5yr to normal	
Parameters	Est mean diff (95% CI)	P value	Est mean diff (95% CI)	<i>P</i> value	Est mean diff (95% CI)	<i>P</i> value	Est mean diff (95% Cl)	<i>P</i> value
Kn ext initial contact	-26 (-31, -20)	<0.001	-23 (-29, -17)	<0.001	2 (-3, 7)	0.35	21 (14, 27)	<0.001
Min kn flex ST	-32 (-39, -24)	<0.001	-26 (-35, -18)	<0.001	5 (-2, 12)	0.13	15 (7, 22)	<0.001
Kn excursion	18 (13, 23)	<0.001	15 (8, 22)	<0.001	-2 (-9, 4)	0.48	-22 (-28, -16)	<0.001
Max peak kn flex SW	-14 (-19, -8)	<0.001	-11 (-14, -8)	<0.001	3 (-2, 7)	0.23	-7 (-11, -3)	0.001
Max kn flexor moment	NA	NA	-0.4 (-0.7, -0.2)	0.002	UA	NA	0.1 (-0.2, 0.3)	0.50
Max kn ext mom 1 <sup>st</sup> peak	NA	NA	-0.2 (-0.6, 0.1)	0.14	NA	NA	0.1 (-0.2, 0.3)	0.66
Max kn ext mom 2 <sup>nd</sup> peak	NA	NA	-0.4 (-0.7, -0.2)	<0.001	UA	NA	0.3 (0.1, 0.5)	0.002
Max kn power gen early ST	UA	NA	0.1 (-0.6, 0.8)	0.69	ПА	NA	0.1 (-0.5, 0.6)	0.80
Max kn power absorp late ST	NA	NA	-0.1 (-0.6, 0.5)	0.79	ПА	NA	0.0 (-0.5, 0.5)	0.99
FFD knee	11 (7, 16)	<0.001	11 (5, 17)	0.001	-0.2 (-5, 4)	0.94	-8 (-13, -3)	0.003
Popliteal angle	-10 (-23, 3)	0.11	-12 (-24, 1)	0.06	-2 (-11, 8)	0.71	17 (8, 27)	0.001
SD standard deviation Est. mean diff. estimated mean di 95% Cl 95% confidence in UA	standard deviation estimated mean difference between crouch and normal data 95% confidence intervals of the estimated mean difference be unable to assess due to n=4 subjects for kinetic data at 1 year	nd normal da ean differenc tic data at 1	standard deviation estimated mean difference between crouch and normal data 95% confidence intervals of the estimated mean difference between crouch and normal data. unable to assess due to n=4 subjects for kinetic data at 1 year	ST SW al data.	stance phase swing phase			

Table 7.16 Comparison of difference in means for severe crouch data for knee parameters at pre, one and five years post-SEMLS (plus normal data). Shaded data indicate statistically significant changes.

Chapter 7

245



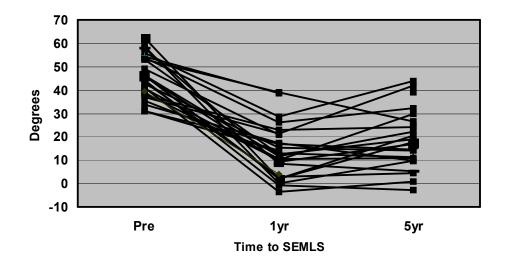


Figure 7.12 Change in minimum knee flexion for subjects with severe crouch gait at pre, one and five years post-SEMLS.

**Knee Excursion** 

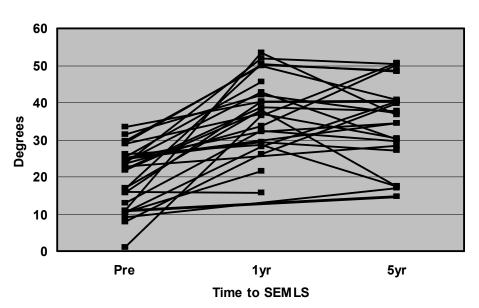
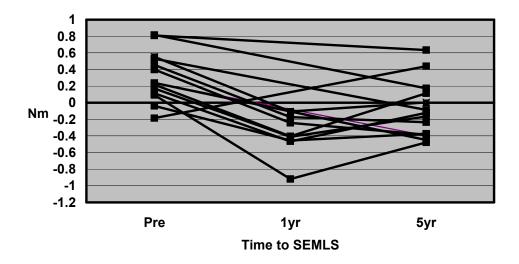


Figure 7.13 Change in knee excursion from pre, one and five years post-SEMLS

Figure 7.13 Change in knee excursion from pre, one and five years post-SEM for the subjects with severe crouch gait.

From the above changes in knee kinematic parameters, corresponding changes in kinetics would be expected. The knee extensor moment in loading response decreased to within normal limits at the five year period post-SEMLS, but the knee extensor moment in late stance did not show a similar degree of change, despite statistically significant improvements at five years post-SEMLS compared to pre-SEMLS. The knee flexor moment post-SEMLS showed improvement (that is an increase towards a flexor moment) compared to pre-SEMLS and normal data (Figure 7.14). At five years, the knee flexor moment was not statistically greater than normal values. Knee power generation in loading response was unaffected by SEMLS and remained similar to the normal values, as was the case for knee absorption power in late stance.



# **Knee Flexor Moment**

Figure 7.14 Change in knee flexor moment from pre, one and five years post-SEMLS for the subjects with severe crouch gait. Negative value indicates a flexor moment.

Knee extension on physical examination increased after SEMLS although not to normal values. The popliteal angle showed a trend towards improvement after SEMLS but remained increased compared to normal values. The peak knee flexion in swing diminished at one year post-SEMLS and remained so at the five year mark (Figure 7.15). The Duncan Ely test for rectus spasticity was positive pre- and post-surgery for all severe crouch subjects.



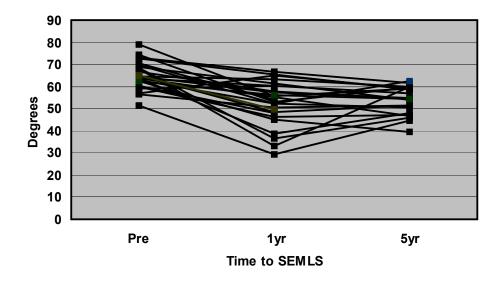


Figure 7.15 Change in peak knee flexion in swing from pre, one and five years post-SEMLS for the subjects with severe crouch gait.

#### <u>Ankle</u>

There was a reduction in the degree of dorsiflexion at initial contact post-SEMLS, with the five year data within the normal range (Tables 7.17 & 7.18). Maximum dorsiflexion in stance post-SEMLS reduced to within normal range at five years post-SEMLS (Figure 7.16). Conversely maximum dorsiflexion in swing was reduced post-SEMLS but this improvement was only statistically significant at five years from baseline, and this was not within the normal range.

After SEMLS, maximum plantarflexion in third rocker increased at five years though not to normal levels. Excursion of third rocker did not increase with SEMLS and remained outside normal values. Although the diminished power generation in late stance at the ankle observed prior to SEMLS improved at five years post-surgery, it was still decreased with respect to normal values (Figure 7.17). Gastrocnemius length measured by physical examination was not significantly different from normal values pre- or post-SEMLS though the values of the 95% confidence interval (Table 7.18) suggest the gastrocnemius was clinically shorter post-SEMLS. The static length of soleus was not changed

after SEMLS, however there was a trend toward decreased length compared to pre-surgery at one year more so than at five years.

-		SEMLS status		
Parameters	Pre	1 year	5 year	Normals
	mean ±SD	mean ±SD	mean ±SD	mean ±SD
Dorsiflexion initial contact	12 ± 9	$4 \pm 9^{*}$	2 ± 7†	-1 ± 3
Max dorsiflex ST	29 ± 10	17 ± 8*	16 ± 7†	15 ± 4
Max plantarflex 3 <sup>rd</sup> rocker	8 ± 13	0.4 ± 7*	-1 ± 8†^	-17 ± 6
Excursion 3 <sup>rd</sup> rocker	21 ± 13	16 ± 6	17 ± 6^	31 ± 5
Max dorsiflex SW	19 ± 9	14 ± 8	9 ± 8†^	3 ± 3
Max ankle power gen late ST	1.2 ± 0.6	UA	1.8 ± 0.4†^	$4.2 \pm 0.8$
Dorsiflexion (kn flexion)	21 ± 12	14 ± 15	17 ± 13	23 ± 7
Dorsiflexion (kn extension)	2 ± 8	0 ± 9	2 ± 7	5 ± 4

Table 7.17 Comparison of means ± standard deviations for severe crouch data for the ankle parameters at pre, one and five years post-SEMLS. Includes normal data for comparison. Statistically significant changes indicated by shaded data.

significance at P value<0.05 between pre- and 1 yr post-SEMLS

† significance at P value<0.05 between pre- and 5 yr post-SEMLS

significance at P value<0.05 between 1 and 5 yr post-SEMLS</li>
 significance at P value<0.05 between 5 yr post-SEMLS and normal data</li>

SD standard deviation

- ST stance phase
- SW swing phase
- -ve plantarflexion

UA unable to assess due to n=4 subjects for kinetic data at 1 year

Table 7.18 Comparison of difference in means for severe crouch data for ankle parameters at pre, one and five years post-SEMLS (plus normal data). Shaded data indicate statistically significant changes.

	E	ference in	Difference in mean change between pre, 1 and 5 years post-SEMLS and to normal data	en pre, 1 a	ind 5 years post-SI	EMLS and	to normal data.	
	<u>1yr to pre-op</u>		5yr to pre-op		5yr to 1yr		5yr to normal	
Parameters	Est mean diff (95% CI)	f <i>P</i> value	Est mean diff (95% Cl)	f <i>P</i> value	Est mean diff (95% Cl)	P value	Est mean diff (95% Cl)	ff <i>P</i> value
Dorsiflexion initial contact	-8 (-15, -2)	0.01	-10 (-14, -6)	<0.001	-2 (-7, 3)	0.47	3 (-1, 8)	0.12
Max dorsiflex ST	-13 (-17, -8)	<0.001	-13 (-17, -9)	<0.001	-1 (-6, 5)	0.85	2 (-2, 6)	0.34
Max plantarflex 3 <sup>rd</sup> rocker	-8 (-14, -1)	0.03	-9 (-16, -2)	0.01	-2 (-6, 3)	0.51	16 (10, 21)	<0.001
Excursion 3 <sup>rd</sup> rocker	-5 (-10, 0 <u>)</u>	0.07	-4 (-10, 3)	0.26	1 (-3, 4 <u>)</u>	0.58	-14 (-18, -10)	<0.001
Max dorsiflex SW	-5 (-11, 1)	0.09	-10 (-14, -6)	<0.001	-5 (-10, 1)	0.09	6 (2, 11)	0.007
Max ankle power gen late ST	NA	NA	0.7 (0.3, 1.1)	0.002	UA	NA	-2.4 (-2.8, -1.9)	<0.001
Dorsiflexion (kn flexion)	-7 (-14, 1)	0.09	-4 (-10, 3)	0.25	3 (-5, 10)	0.44	-5 (-13, 3)	0.18
Dorsiflexion (kn extension)	-1 (-6, 3)	0.62	0 (-6, 5)	0.97	1 (-5, 7)	0.74	-3 (-8, 2)	0.21

standard deviation estimated mean difference between crouch and normal data 95% confidence intervals of the estimated mean difference between crouch and normal data stance phase swing phase unable to assess due to n=4 subjects for kinetic data at 1 year

SD Est. mean diff. 95% CI SV UA

250



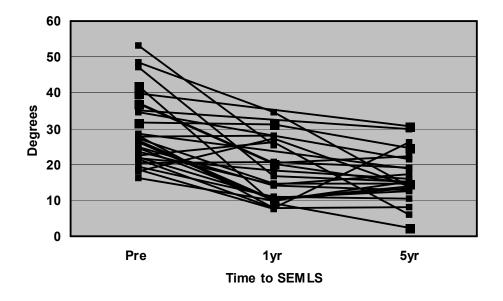


Figure 7.16 Change in maximum dorsiflexion from pre, one and five years post-SEMLS for the subjects with severe crouch gait.

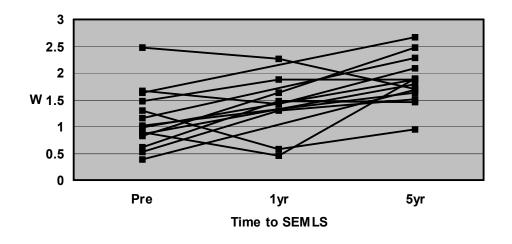
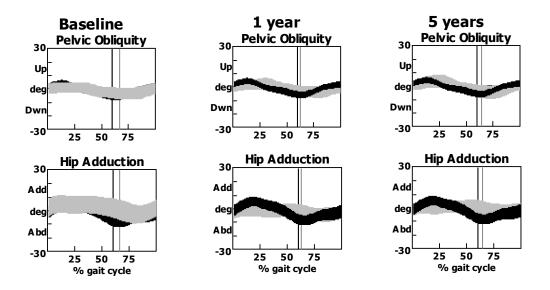




Figure 7.17 Change in ankle power from pre, one and five years post-SEMLS for the subjects with severe crouch gait.

# Coronal Plane

Figure 7.18 illustrates the coronal plane kinematics at baseline, one and five years post-SEMLS.



# **Coronal Kinematics**

Figure 7.18 Coronal plane kinematics pre, one and five years post-SEMLS. Grey band represents the severe crouch data and the black band, normal data.

#### Pelvis

Excursion of the pelvis in the coronal plane was not altered by SEMLS and stayed within normal range (Tables 7.19 & 7.20).

### <u> Hip</u>

Maximum hip adduction in stance decreased after SEMLS. Maximum adduction in swing was also decreased after surgery.

Table 7.19 Comparison of means ± standard deviations for severe crouch data for parameters in the coronal and transverse planes at pre, one and five years post-SEMLS. Includes normal data for comparison. Statistically significant changes indicated by shaded data.

		SEMLS status		
Parameters	Pre	1 year	5 year	Normals
	mean ±SD	mean ±SD	mean ±SD	mean ±SD
<u>Coronal Plane</u>				
Pelvic excursion	10 ± 5	10 ± 5	10 ± 5	11 ± 3
Max hip adduction ST	8 ± 6	2 ± 4*	4 ± 4†^	6 ± 4
Max hip adduction SW	5 ± 6	1 ± 5*	2 ± 5†^	-1 ± 4
<u>Transverse Plane</u>				
Pelvic excursion	22 ± 14	16 ± 11	17 ± 6	15 ± 6
Mean hip rotation ST	8 ± 11	0 ± 11*	7 ± 9‡^	2 ± 6
Mean foot progression ST	-6 ± 24	-6 ± 12	-5 ± 11	-10 ± 6
Hip internal rotation	58 ± 10	55 ± 13	52 ± 13	49 ± 13
Hip external rotation	15 ± 16	16 ± 11	14 ± 11^	33 ± 8
Thigh/ foot angle	4 ± 15	3 ± 10	5 ± 10	8 ± 4

- ST SW stance phase swing phase

 <sup>\*</sup> significance at P value<0.05 between pre- and 1 yr post-SEMLS</li>
 + significance at P value<0.05 between pre- and 5 yr post-SEMLS</li>
 + significance at P value<0.05 between 1 and 5 yr post-SEMLS</li>
 ^ significance at P value<0.05 between 5 yr post-SEMLS and normal data</li>
 SD standard deviation.

		Difference	Difference in mean change between pre, 1 and 5 years post-SEMLS and to normal data.	en pre, 1 a	nd 5 years post-SEMI	-S and to I	<u>normal data.</u>	
	1yr to pre-op		5yr to pre-op		5yr to 1yr		5yr to normal	
Parameters	Est mean diff (95% CI)	<i>P</i> value	Est mean diff (95% CI)	<i>P</i> value	Est mean diff (95% CI)	<i>P</i> value	Est mean diff (95% CI)	<i>P</i> value
<u>Coronal Plane</u>								
Pelvic excursion	0 (-2, 2)	0.96	1 (-3, 5)	0.62	1 (-3, 5)	0.62	0 (-4, 3)	0.87
Max hip adduction ST	-6 (-8, -3)	<0.001	-4 (-8, 0)	0.04	2 (0, 4)	0.10	-2 (-4, 0)	0.02
Max hip adduction SW	-4 (-6, -2)	0.002	-3 (-5, -1)	0.005	1 (-1, 3)	0.35	3 (1, 5)	0.01
Transverse Plane								
Pelvic excursion	-5 (-12, 1)	0.09	-5 (-12, 3)	0.21	1 (-6, 8)	0.77	2 (-3, 7)	0.36
Mean hip rotation ST	-8 (-15, -2)	0.02	-1 (-6, 3)	0.56	7 (2, 12)	0.009	5 (1, 9)	0.01
Mean foot progression ST	0 (-12, 11)	0.94	1 (-11, 13)	0.88	1 (-6, 9)	0.73	5 (-1, 11)	0.11
Hip internal rotation	-3 (-10, 4)	0.36	-6 (-13, 1)	0.09	-3 (-8, 3)	0.29	4 (-6, 14]	0.48
Hip external rotation	1 (-8, 11)	0.76	-1 (-10, 8)	0.86	-2 (-9, 4)	0.50	-19 (-26, -13)	<0.001
Thigh/ foot angle	-1 (-9, 6.9)	0.78	1 (-8, 9)	0.87	2 (-3, 7)	0.46	-3 (-8, 2)	0.22
SD standard deviation Est. mean diff. estimated mean di 95% CI 95% confidence in	standard deviation estimated mean difference between crouch and normal data 95% confidence intervals of the estimated mean difference b	nd normal da an differenc	standard deviation setimated mean difference between crouch and normal data 95% confidence intervals of the estimated mean difference between crouch and normal data	al data	ST stance phase SW swing phase	8.0		

Comparison of difference in means for severe crouch data for pelvic and hip parameters in the coronal and transverse planes at pre, one and Table 7.20

Chapter 7

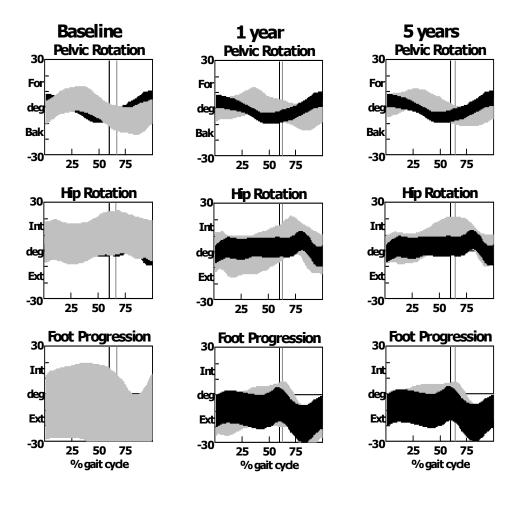
254

### Transverse Plane

Figure 7.19 shows transverse plane kinematics at baseline, one and five years post-SEMLS.

## <u>Pelvis</u>

The excursion of the pelvis in the transverse plane decreased post-SEMLS but this was not statistically significant compared to baseline values (Tables 7.19 & 7.20).

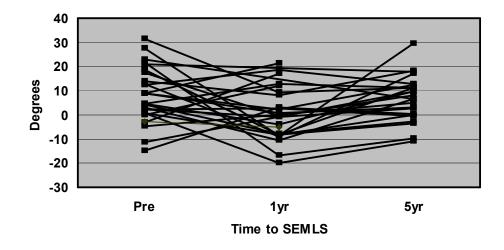


# **Transverse Kinematics**

Figure 7.19 Transverse plane kinematics pre, one and five years post-SEMLS. Grey band represents the severe crouch data and the black band, normal data.

### <u>Hip</u>

The mean position of the hip during stance showed reduced internal rotation at one year post-SEMLS compared with baseline, placing the internal rotation within the normal range (p=0.4, 95% CI –7.1°, 3.2°). This change was not maintained at five years, with a return to increased hip internal rotation similar to pre-SEMLS levels (Figure 7.20). Internal rotation of the hip as measured on physical examination, although reduced, was not statistically significantly altered post-SEMLS. The external rotation of the hip continued to be decreased in available range post-SEMLS.



### **Hip Rotation Stance**

Figure 7.20 Change in mean hip rotation in stance phase from pre, one and five years post-SEMLS for subjects with severe crouch gait.

### <u>Ankle</u>

The average foot progression remained similar to normal values post-SEMLS. The foot/ thigh angle was unaltered by SEMLS and remained similar to normal values post-SEMLS. However the large standard deviations of both variables indicated considerable variability.

### Timing of Parameters Within Stance or Swing Phase

The timing of maximum hip extension in stance phase was premature pre-SEMLS and did not improve post-surgery (Tables 7.21 & 7.22). The minimum knee flexion also occurred prematurely pre-SEMLS and did not change afterwards, although the timing seemed to be even earlier. The delay in timing of the peak knee flexion in swing observed pre-SEMLS, continued at one year post-SEMLS. However there was an improvement at five years with timing similar to normal values. If the timing of peak knee flexion is considered in relation to the gait cycle, there was again less delay at five years post-surgery but not to within normal values. No change in timing occurred in A2 post-SEMLS.

## Rate of Movement (degrees/sec)

The rate of change between maximum knee extension and peak knee flexion in swing phase increased post-SEMLS but remained different from normal values. In third rocker, the rate of change from dorsiflexion to plantarflexion was not changed from pre-SEMLS data and therefore remained considerably less than normal values (Tables 7.21 & 7.22).

Table 7.21 Comparison of means ± standard deviations for severe crouch data for the timing and rate of change of parameters at pre, one and five years post-SEMLS. Includes normal data for comparison. Statistically significant changes indicated by the shaded data.

	SEMLS status			
Parameters	Pre	1 year	5 year	Normals
	Mean ±SD	Mean ±SD	Mean ±SD	Mean ±SD
Timing (% Stance or Swing)				
Max hip ext %ST	79 ± 6	80 ± 6	79 ± 6^	84 ± 3
Crossover hip ext moment to flex %ST	56 ± 10	UA	54 ± 6^	34 ± 13
Min kn flex %ST	51 ± 18	42 ±12	45 ± 10^	65 ± 6
Max peak kn flex %cycle	82 ± 6	80 ± 4	77 ± 6†‡^	71 ± 2
Max peak kn flex %SW	43 ± 17	39 ± 10	32 ± 15†	28 ± 4
Max plantarflex 3 <sup>rd</sup> rocker % SW	6 ± 12	5 ± 12	2 ± 7^	7 ± 4
Max ankle power gen %ST	84 ± 3	UA	84 ± 5^	88 ± 2
<u>Rate of Change (°/s)</u>				
Max kn ext to peak kn flex	40 ± 19	67 ± 42*	65 ± 26†^	190 ± 34
Max dorsi to plantarflex 3 <sup>rd</sup> rocker	86 ± 63	72 ± 32	76 ± 29^	181 ± 32

significance at P value<0.05 between pre- and 1 yr post-SEMLS</li>
significance at P value<0.05 between pre- and 5 yr post-SEMLS</li>
significance at P value<0.05 between 1 and 5 yr post-SEMLS</li>
significance at P value<0.05 between 5 yr post-SEMLS</li> significance at P value<0.05 between 5 yr post-SEMLS and normal data

SD standard deviation

ST stance phase

SW

swing phase unable to assess due to n=4 subjects for kinetic data at 1 year UA

Parameters	<u>Pre – 1yr</u> Est mean diff (95% Cl)	Difference P value	Difference in mean change between pre, 1 and 5 years post-SEMLS and to hormal data. <u>Pre - 5yr</u> P value Est mean diff (95% P value Est mean CI) CI	een pre, 1 a P value	nd 5 years post-semLS and to <u>1yr – 5yr</u> Est mean diff (95% <i>P</i> value CI)	P value	<u>iormal data.</u> <u>5yr – normal</u> Est mean diff (95% Cl)	<i>P</i> value
Timing (% Stance or Swing)								
Max hip ext %ST	0.6 (-3.1, 4.3)	0.75	-0.3 (-4.5, 3.9)	0.89	-0.9 (-5.1, 3.3)	0.67	-5.0 (-8.8, -1.1)	0.01
Crossover hip ext moment to flex %ST	3.8 (-3.2, 10.7)	0.28	-1.8 (-6.9, 3.3)	0.47	-5.6 (-12.7, 1.6)	0.12	20 (13, 28)	<0.001
Min kn flex %ST	-9.0 (-21.2, 3.2)	0.14	-6.3 (-17.0, 4.5)	0.24	2.7 (-2.1, 7.5)	0.26	-21(-26, -16)	<0.001
Max peak kn flex %cycle	-1.8 (-4.2, 0.6)	0.13	-4.8 (-7.3, -2.3)	0.001	-3.0 (-5.0, -1.0)	0.006	6.2 (3.0, 9.4)	<0.001
Max peak kn flex %SW	-4.1 (-12.4, 4.2)	0.32	-11.5 (-19.1, -4.0)	0.004	-7.4 (-15.9, 1.0)	0.08	3.4 (-5.2, 12.1)	0.42
Max plantarflex 3 <sup>rd</sup> rocker % SW	-0.6 (-9.5, 8.4)	06.0	-4.2 (-10.2, 1.8)	0.16	-3.6 (-11.1, 3.9)	0.33	-5.3 (-8.9, -1.8)	0.005
Max ankle power gen %ST	UA	Ν	0.2 (-1.8, 2.2)	0.83	UA	Ν	-3.9 (-6.3, -1.5)	0.003
Rate of Change (°/s)								

Table 7.22 Comparison of difference in means for severe crouch data for timing of parameters at pre, one and five years post-SEMLS (plus normal data). Shaded data indicate statistically significant changes.

SD-standard deviation, Est. mean diff. -estimated mean difference between crouch & normal data, 95% CI -95% confidence intervals of the estimated mean difference between crouch & normal data, UA -unable to assess due to n=4 subjects for kinetic data at 1 year, ST -stance phase, SW -swing phase

259

Chapter 7

0.003 0.59

-10 (-47, 27)

-14 (-42, 14)

Max dorsi to plantarflex 3<sup>rd</sup> rocker

25 (9, 41)

0.03 0.31

27 (4, 51)

Max kn ext to peak kn flex

<0.001<br/><0.001

-125 (-147, -104) -105 (-125, -85)

0.84 0.71

-2 (-24, 20) 4 (-19, 28)

# 7.4 DISCUSSION

When orthopaedic surgical intervention is undertaken for children with severe crouch gait it is important to know not only whether the children change in respect to their baseline status but also whether they improve in the direction of the normal range. A change from pre- to post-intervention does not necessarily signify an improvement unless the direction of change in respect to normal values is taken into consideration. The aim of surgical intervention is to improve on the natural history or maintain the subject's ability to walk and thereby improve or stabilise their ability to participate in everyday activities. The intervention therefore, is not expected to have the subject walk in the same way as a normally developing subject as this is not possible. Orthopaedic surgery can cause change at the musculoskeletal level but cannot change abnormalities at the neurological level e.g. timing of contraction, speed of recruitment of muscle fibres (Graham and Fixsen 1988, Norlin and Tkaczuk 1985, Perry et al 1974). As Mercer Rang succinctly wrote "after you operate, patients still have cerebral palsy" (Rang 1990, p. 483). According to dynamic systems theory the implementation of SEMLS, which addressed pathology in the musculoskeletal system, provided the opportunity for a critical change to occur in the child's function of walking (Shumway-Cook and Woollacott 2001). Rehabilitation aimed to provide an environment that optimised this change in one subsystem (musculoskeletal) to alter and improve the dynamics of the other subsystems that contribute to gait (Larin 2000). At one year post-SEMLS, whether the children improved, deteriorated or maintained status quo compared to baseline, was assessed. At five years post-operation, the question of maintenance of any improvements found at one year post-surgery was investigated, as well as whether the children deteriorated towards baseline levels. There was also the question of whether, if there was improvement from pre-op status, was the improvement within the range of normal values for any of the parameters.

This is the first study describing the short and long term outcome of SEMLS for severe crouch gait. This is also the first study of severe crouch gait that has been based on a precise definition of sagittal plane kinematics at four

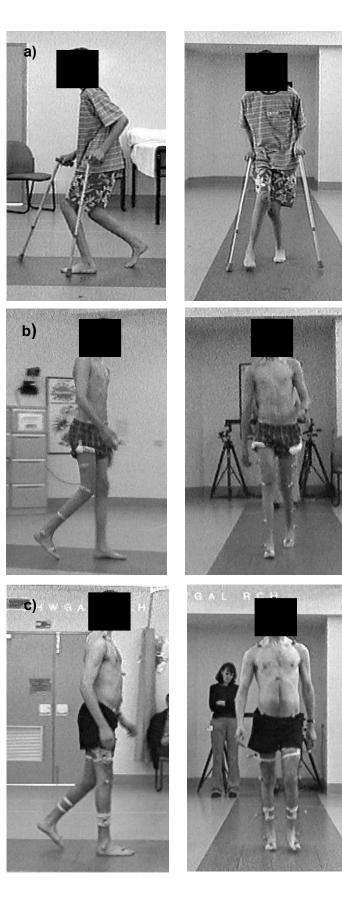
anatomical levels, so that there is no misunderstanding regarding the actual gait pattern that is being investigated.

The SEMLS consisted of mostly soft tissue surgery that concentrated on the lengthening or transferring of muscles in the sagittal plane around the knee and then the hip. The transverse plane was dealt with by correcting lever arm dysfunction by bony surgery at the femur, tibia and foot. The purpose of the SEMLS was to rectify the pattern of crouch gait. By gaining increased extension of the lower limbs in the sagittal plane and appropriate biomechanical alignment of the bony levers in the transverse plane, improved functional mobility was presumed to follow.

A general summary of the status of parameters post-SEMLS is documented in Table 7.23. Improvement in status was observed predominantly in the knee and ankle parameters in the sagittal plane, and the hip in the coronal and transverse planes (Figures 7.21 to 7.23). Status was unchanged for temporal-spatial parameters, the hip in the sagittal plane, pelvis and foot in the transverse plane and the timing of parameters. Deterioration in status was found at the pelvis and the knee (in swing phase) in the sagittal plane.

Table 7.23Summary of overview of changes in parameters post-SEMLS at five<br/>years.

		Changes post-SEMLS		
Parameter	Improvement	Unchanged	Deterioration	
<u>Mobility</u>	GMFCS FMS 5, 50, 500 FAQ			
Temporal-		Normalised velocity		
<u>spatial</u>		Normalised stride length Toe off %cycle Opposite foot off %ST Opposite foot contact %ST		
<u>Pelvis</u>		Pelvic excursion	Ant pelvic tilt	
<u>Hip</u>	Max hip power gen late ST Max hip adduction ST Max hip adduction SW Mean hip rotation ST	Max hip ext ST Hip excursion Max hip ext moment Max hip power gen early ST Crossover hip ext moment to flex %ST Hip internal rotation Hip external rotation Max hip ext %ST Crossover hip ext moment to flex %ST		
<u>Knee</u>	Kn ext initial contact Min kn flex ST Kn excursion Max kn flexor moment Max kn ext mom 1 <sup>st</sup> peak FFD knee Max peak kn flex % cycle Max peak kn flex %SW Rate of max kn ext to peak kn flex	Max kn ext mom 2 <sup>nd</sup> peak Max kn power gen early ST Max kn power absorp late ST Popliteal angle Duncan Ely (fast) Min kn flex %ST	Max peak kn flex SV	
<u>Ankle</u>	Dorsiflexion initial contact Max dorsiflex ST Max plantarflex 3 <sup>rd</sup> rocker Max dorsiflex SW Max ankle power gen late ST	Excursion 3 <sup>rd</sup> rocker Dorsiflexion (kn flexion) Dorsiflexion (kn extension) Mean foot progression ST Thigh/ foot angle Max plantarflex 3 <sup>rd</sup> rocker %SW Max ankle power gen %ST Rate of max dorsi to plantarflex 3 <sup>rd</sup> rocker		



#### Figure 7.21

a) Subject 2 (12 years old) GMFCS level III, walking in severe crouch gait at baseline. Past history included lengthening of the Achilles tendon on two occasions and now bilateral patellar fractures. In the sagittal plane there is excessive knee flexion and ankle dorsiflexion. The coronal plane shows increased internal rotation of the femurs but normal foot progression.

b) Same boy at one year post-SEMLS, which consisted of medial hamstrings, psoas and adductor longus lengthenings, and L femoral derotation osteotomy. Full surgical recommendations had recommended bilateral femoral derotation osteotomies and rectus femoris transfers but these were declined by parents.

c) Gait pattern at five years post-SEMLS. At one year post-SEMLS, he underwent a right femoral derotation osteotomy and proximal rectus releases. He was now classified as a GMFCS level II.





# Figure 7.22

a) Subject 8 (10 years old) GMFCS level II, walking in severe crouch gait at baseline. Past surgical history was of Achilles tendon lengthenings. Note posterior tilted pelvis.





b) Same boy, GMFCS level III, walking at one year post-SEMLS which included medial and lateral hamstrings lengthening, R femoral derotation osteotomy and supramalleolar osteotomy and L subtalar fusion. Needed crutches to walk at this time. Further surgery after this time consisted of proximal rectus releases and right proximal tibial epiphysiodesis.





c) Gait pattern at five years post-SEMLS. Now classified GMFCS level II.





Figure 7.23

a) Subject 11 (11 years old) GMFCS level III, walking in severe crouch gait at baseline. Past surgical history included calf, hamstring and foot surgery.





b) Same girl walking at one year post-SEMLS which consisted of psoas, medial and lateral hamstrings, supramalleolar osteotomies. Proximal rectus releases and femoral derotation osteotmies were undertaken after this time.





c) Gait at five years post-SEMLS. GMFCS level III remains constant.

# 7.4.1 Positive Changes Post-SEMLS

# Activities- Mobility Status

Mobility status improved after SEMLS as the majority of subjects were classified as GMFCS level III pre-SEMLS and at five years post-SEMLS the majority of subjects were now classified as GMFCS level II. The change in GMFCS level was not expected as the GMFCS is not an outcome measure and has been shown to be stable with age (Morris et al 2004, Wood and Rosenbaum 2000). The statistics showed that this improvement in level occurred between one and five years and possibly reflects the fact that functional improvements do not cease at one year but can still be made in the following years post-SEMLS. This has implications for rehabilitation post-SEMLS, as this finding suggests that continued efforts in rehabilitation may afford benefits for the child post-SEMLS for severe crouch gait.

The FMS showed that in the community there was less reliance on wheelchair mobility and some subjects could now mobilise independently without assistive devices, whereas none could prior to SEMLS. In the immediate neighbourhood, schoolyard or work environment, at five years after surgery, subjects were not all dependent on assistive devices as they had previously been. Some subjects were now able to walk without aids. In the home, classroom or office the majority of subjects now were ambulating independently instead of using aids as most subjects had prior to SEMLS. This improvement in FMS was not seen at one year post-SEMLS but as with the rating of the GMFCS, the change was significant between one and five years post-SEMLS.

It is worth noting that the FMS over 50 metres showed an increase in wheelchair use over this distance at five years despite a decrease at one year post-surgery. The decline in wheelchair use at one year was attributable to the missing data of one subject at that time point who was using a wheelchair at baseline over this distance. At five years, data for this subject were able to be collected and accounts for the increase of one subject at this rating. The subject who was newly rated a wheelchair user over this distance at five years, began using a wheelchair over 50 metres, in this case at school, despite being capable of walking this distance. The change to wheelchair use occurred because the

subject was now in his senior years at secondary school and he needed to carry lots of books to class (which interfered with his balance and stability) and be there on time (which meant that he had to rush between classes). The use of a single stick over 50 and 500 metres at one year was recorded for three subjects although they could walk both distances without a stick. When questioned as to why they continued to use a stick over these distances, the subjects indicated that without the stick they did not feel confident walking in crowds, such as in school corridors or shopping centres, and preferred to take the stick with them just in case they were jostled. At five years, two subjects were again independent over these distances and the third subject had reverted to elbow crutches (not over 5 metres) on recommendation of their surgeon for greater stability over these longer distances. These scenarios highlight how performance is altered for a person according to their needs to function effectively and confidently in society, and it is difficult for scales that rate function to reflect how these factors impact on the actual rating of function (Palisano et al 2003).

Prior to surgery the majority of the severe crouch subjects were rated as limited community ambulators on the FAQ, but by five years post-SEMLS the majority were rated as community ambulators. This change was statistically significant between pre- and one year post-SEMLS, unlike the GMFCS and FMS which both showed statistically significant change after the one year time period. This difference is mostly likely due to the FAQ being rated without regard to use or non-use of assistive devices. The FAQ showed that the subjects were able to walk further one year post-SEMLS compared with pre-SEMLS. The lack of change in the FAQ after this time does not mean that mobility remained the same, as subjects may have improved further and discarded assistive devices or deteriorated and required increased use of assistive devices in order to maintain status quo on the FAQ.

The overall improvement in mobility was not due to increased speed or stride length as these were unchanged after surgery, nor stability as measured by duration of stance and double support. What factors led to this improvement in functional status? The degree of knee flexion and ankle dorsiflexion in stance

decreased post-surgery, and so posture in the sagittal plane was more erect. Being more upright would alleviate the stressful forces at the knee when subjects walk with a flexed knee gait, as documented by Perry et al (1975). Other studies have shown that varying amounts of increased knee flexion increase energy requirements (Duffy et al 1997, Sienko Thomas et al 1996).

It may be inferred that the energy cost of walking improved for the subjects walking in severe crouch gait, as the study by Johnston et al (2004b) showed that there was a correlation (0.87) between energy cost and GMFCS level. This study found that there were statistically significant differences in energy expenditure between GMFCS levels so that the lower the GMFCS level, the lower the energy cost recorded (Johnston et al 2004b). In our study, the GMFCS levels decreased post-SEMLS implying that energy cost also decreased for these subjects. Although there were positive changes in the FMS and FAQ post-SEMLS, neither rating of function is strongly correlated with the energy measure: correlation coefficient of 0.51-0.55 for the FMS (Graham et al 2004a) and -0.424 for the FAQ (Novacheck et al 2000). The energy requirements of a subject walking in crouch gait should be the subject of future research in order to confirm the findings of previous studies and to test whether SEMLS for severe crouch gait can lead to a reduction in energy requirements.

# **Body Structure and Function**

#### Knee Parameters in the Sagittal Plane

In stance phase knee parameters were all improved after SEMLS (Figures 7.8, 7.9 and 7.10). Both knee extension at initial contact and the minimum flexion in stance were improved between 20 to 31° and 24 to 39° respectively according to the 95% confidence intervals at one year post-surgery. At five years, this improvement appeared to reduce to between 17 to 29° at initial contact and 18 to 35° for minimum flexion. A similar finding has been reported in another long term study post-SEMLS with subjects with spastic diplegia (Saraph et al 2005). Despite the initial improvement seen in knee extension, this degree of improvement was not maintained. However, again the knee did not relapse to pre-operative levels at the final assessment at a mean of four years in that study (Saraph et al 2005).

The corresponding reduction in knee extensor moment at loading response and the change to a flexor moment in midstance indicate that the knee was taking much less stress through the patella and the rectus femoris tendon. A change to a flexor moment signifies that the ground reaction force is no longer constantly posterior to the knee and the rectus femoris can effectively 'switch off' as the knee should be stable through passive forces at that point in the gait cycle. The presence of a flexor moment in stance signifies that adequate knee extension has been obtained in stance. This reduces the demand on the extensor mechanism, which should lead to reduction in knee pain and allow patellar fractures to heal. Many of the subjects in the severe crouch cohort complained of knee pain and had patella alta or patellar fractures pre-SEMLS but the data relating to pre- and post-SEMLS knee pain and knee radiology were not collected by the author and so not presented within this thesis.

Pre- and post-surgery the knee powers of generation in loading response and absorption in late stance were within normal values. This may be due to the reduced knee movement over the gait cycle, implying low angular velocities that would not give large values for power. The lack of increase in knee power at all three time periods suggests that the power for propulsion and maintenance of the upright posture is coming from other levels, not the knee. This result is in contrast to that found by Lin et al (2000). In that study the power absorption in late stance was increased compared to other gait patterns but this result was not compared to normative data. Inspection of the mean joint power reported by Lin et al (Lin et al 2000), shows that the maximum knee power absorption in late stance for the crouch group was approximately -0.7and -0.8 watts/kg and our value was larger, between -1.2 to -0.9 watts/kg.

In our study the pre-SEMLS knee excursion was 33° to 42° less than normal and at five years despite some improvement, remained 16° to 28° less than normal values. It would appear that the improvements in knee parameters in stance were a positive consequence of the high incidence of lengthening of the hamstrings in this cohort and is similar to the findings of other studies (Baumann et al 1980, Chang et al 2004, Kay et al 2004a, Kay et al 2002, Rethlefsen et al 1999b, Thometz et al 1989, van der Linden et al 2003a, Yngve

et al 2002). Knee recurvatum was not a problem post-SEMLS as only one limb recorded on gait analysis a knee hyperextension of 4° at one year and only one limb from a different subject had 3° of hyperextension at five years.

The physical examination of the knee showed a reduced fixed flexion contracture, but the improvement was not enough to consider it within the normal range. The mean popliteal angle was only clinically improved compared to values pre-SEMLS by 11° and 12° at one and five years respectively. This measure did not show a deterioration as has been reported in one long term study (Chang et al 2004). The dynamic improvement observed in gait parameters at the knee, seemed to surpass the static improvement found on physical examination of the knee, which concurs with results from other studies (Orendurff et al 1998, Orendurff et al 2000, Õunpuu et al 2004).

# Ankle Parameters in the Sagittal Plane

Prior to SEMLS, 11/14 subjects had had some form of calf surgery, six subjects having had the calf surgery in isolation from other lower limb procedures and four subjects having the calf surgery in combination with hamstrings surgery. In total there were 26 surgeries to the calf with two subjects undergoing the surgery on two occasions. Only one subject at the time of SEMLS had a Strayer procedure. The Strayer was done in conjunction with an os calcis lengthening and peroneus brevis tendon lengthening, in order to improve the range of dorsiflexion, which can be diminished after an os calcis lengthening has been performed (Duffy and Cosgrove 2002, Evans 1975b).

There was no direct corrective surgery to the calf at the time of SEMLS or thereafter, but it was recognised that for crouch gait to be corrected, the excessive dorsiflexion at the ankle during gait needed to be decreased. In order to achieve an improvement in biomechanical alignment at the ankle and knee post-surgery so that gait rehabilitation could be optimised, all subjects wore GRAFOs until three months post-SEMLS. After this, if the subject could show on barefoot walking that the knee could be maintained in active extension within the normal range during stance, the GRAFOs were changed to solid AFOs (Zwick et al 2001). The solid AFOs were continued until control of 2<sup>nd</sup>

rocker at the ankle on barefoot walking was demonstrated, and then the AFOs were hinged. Control of 2<sup>nd</sup> rocker was considered important as this signified that the soleus was able to appropriately restrain the forward progression of the tibia over the foot. Hinged AFO use was often continued to provide assistance with foot drop in swing phase if this was a concern (e.g. causing tripping and falls) otherwise the AFOs were discarded overtime as teenagers do not like wearing them!

Improvements were observed in the ankle kinematics and kinetics from pre- to five years post-SEMLS, and in stance both dorsiflexion at initial contact and maximum dorsiflexion were not significantly different from normal values at five years (Figure 7.8, 7.9 and 7.10). This is an extremely important change as it implies restoration of the competence of the plantarflexion knee extension couple. Initially post-SEMLS the wearing of GRAFOs provided passive knee extension and ankle stability, by producing a plantarflexion knee extension couple that brought the GRF anterior to the knee (Chung et al 1997). There was a decrease in muscle extensibility of the soleus on the Silfverskiold test at one and five years post-SEMLS, which indicates that the soleus muscle was no longer overlong and consequently could contract effectively. This decrease in soleus length may be due in part to orthotic use (GRAFOs and solid AFOS) and in part to the subject's growth. The orthoses held the ankle in slight plantarflexion or plantargrade in the first year of rehabilitation and so restricted continued, unrestrained dorsiflexion in stance, which would prevent the soleus muscle from being in a constant lengthened position (Chung et al 1997, Gage 2004f). Post-SEMLS, our cohort grew on average 8 cm and 19 cm, at one and five years post-SEMLS respectively. It has been postulated that if the ankle is held in plantargrade during a growth spurt, an overlong soleus muscle has the opportunity to retension (Gage 2004f). The results from our study of severe crouch gait support this. To date a study on GRAFO use in crouch gait has not been published.

Power generation at the ankle in terminal stance at five years increased from pre-SEMLS and may be related to the increased maximum plantarflexion in 3<sup>rd</sup> rocker and decreased passive length of soleus, despite the excursion and rate of

271

change of 3<sup>rd</sup> rocker essentially being unchanged. However despite this improvement, A2 continued to be considerably decreased compared to that generated by normally developing subjects. Although there was an increase in maximum plantarflexion in 3<sup>rd</sup> rocker, this was countered by a decrease in maximum dorsiflexion in stance and so excursion of the ankle was unchanged.

There have been various theories put forward regarding the role of the plantarflexors in gait, as to whether they provide active push off or a rollover in terminal stance and possible acceleration into swing phase (Neptune et al 2001). The function of the plantarflexors, particularly soleus, in restraining the forward progression of the tibia in stance and the coupling of this with stabilisation of the knee into extension through the gastrocnemius (plantarflexion knee extension couple) has been well documented (Gage 2004b, Murray et al 1978, Sutherland 1966, Sutherland et al 1980). In terminal stance, it has been reported that the plantarflexors do not propel the body forward because the forward velocity of the centre of mass increased despite paralysis of the plantarflexors (Sutherland et al 1980) and the body was considered to be undergoing the process of a controlled fall (Perry 1992). In contrast another study has shown that with absent plantarflexor muscles there was a delay in heel rise and decreased plantarflexion in late stance (Murray et al 1978). Both of these studies used subjects with normal neuromuscular control, and plantarflexor function was ascertained from paralysis from a tibial nerve block (Sutherland et al 1980) and surgical removal of the gastrocsoleus due to a tumour (Murray et al 1978).

Documented changes with the removal of gastrocsoleus function in the study by Murray (Murray et al 1978) were: increased dorsiflexion in stance, decreased plantarflexion, prolonged activity of the quadriceps in stance, inability to alter velocity, decreased stance phase, decreased excursion into plantarflexion, increased dorsiflexion in swing. In the study by Sutherland (Sutherland et al 1980), the findings were: increased ankle dorsiflexion and knee flexion in stance, decreased single support, centre of pressure located predominately under the ankle joint, decreased vertical force in late single stance, premature reversal and decrease in the fore-aft force, absence of external extensor torque in stance,

decreased velocity, increased energy output, decreased forward velocity in the first half of stance and increased in the second half of stance.

The observation by Sutherland et al (1980) regarding the difficulty in moving the weight forward over the left foot (plantarflexors paralysed) in single support "without the right foot on the floor to preserve balance" (p. 358), possibly deserves further consideration. This difficulty in weight shift led to a decrease in single support on the paralysed side and a corresponding decrease in the swing phase of the contralateral limb. This reduced duration of swing on the contralateral side would translate to the foot being more readily available to provide stability in double support to the paralysed limb. Velocity has already been noted to be reduced post-paralysis of the plantarflexors (Sutherland et al 1980). The knee flexion/ extension kinematic curves in this publication show that following tibial nerve paralysis, the peak knee flexion in swing was decreased in both limbs compared to pre-paralysis (Sutherland et al 1980) (Figure 7.24). Decreased peak knee flexion has been associated with decreased velocity in normally developing subjects (van der Linden et al 2002), and this could be another example. But considering the statement regarding the difficulty in weight transfer forwards on the foot, perhaps the reduced peak knee flexion is a part of a compensation for the instability found in single support with plantarflexor insufficiency. If this is so, then improved function of the plantarflexors in crouch gait would lead to an increase in time in single support for the weight-bearing limb and hence swing phase for the contralateral limb, possibly an increase in velocity and peak knee flexion in swing. In this severe crouch cohort, ankle kinematics improved in stance phase post-SEMLS but single support and normalised velocity were unchanged and amplitude of peak knee flexion diminished.

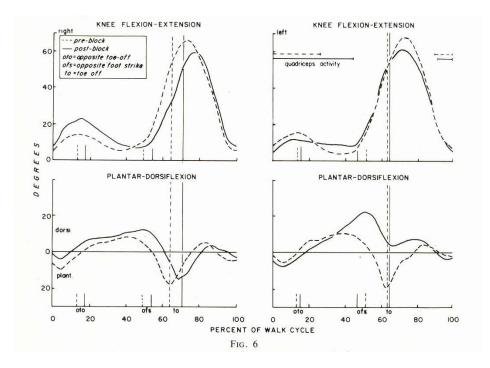


Figure 7.24 The effect on gait of tibial nerve block to the left calf muscles, from Sutherland et al (1980, p. 359). In both knee graphs, the peak knee flexion has decreased post-block (solid black lines).

In the last decade, there have been a number of studies that have explored the support (Anderson and Pandy 2003, Arnold et al 2005a, Jonkers et al 2003, Kepple et al 1997, Neptune et al 2001) and forward progression of the body during gait (Kepple et al 1997, Neptune et al 2001), and swing initiation (Neptune et al 2001). These studies have used various computer programmes to simulate normal walking and then study components of normal walking using musculoskeletal modelling, optimisation frameworks, and forward dynamics simulation of normal walking. A study of the relative contributions from net joint moments to support and forward progression, found that support in gait was mainly from the plantarflexors in single support and then in combination with knee and hip extensors in double support. Forward progression was mostly from the plantarflexors with considerable aid from the knee extensors (Kepple et al 1997). Neptune et al (2001) found that support and forward progression in gait stemmed largely from the contributions of the soleus and gastrocnemius. In late single leg support most of the contribution of the gastrocnemius is directed to the leg and the soleus, to the trunk. Knee stability was provided by the soleus in single support, which has implications if the soleus has decreased function as

knee stability will be threatened. The gastrocnemius was the only muscle to contribute to swing initiation in pre-swing.

Another study looked at the contributions of muscles to providing stability in mid stance in the sagittal plane (Jonkers et al 2003). The effect of removing the contribution of the gluteus maximus, led to increased activity of the hamstrings, and simultaneous activation of gastrocnemius for stability in knee extension. So if the plantarflexors were dysfunctional as they can be in pathological gait, then the hamstrings would not able to compensate for the knee flexion according to this model. When soleus activity was removed, the model compensated with increased contributions by the gastrocnemius, hamstrings and knee extensors. If both the plantarflexors are compromised then the ability of the hamstrings and knee extensors to compensate was reduced. Without hamstring activity, gluteus maximus and gastrocnemius contributions increased.

The individual muscle contributions to stability over the whole of the stance phase have now been documented (Anderson and Pandy 2003). The dorsiflexors supplied the support from just after heel strike to before foot flat. From then until opposite foot off, the gluteus maximus, vasti and posterior gluteus medius/ minimus were dominant in their contribution to the vertical ground reaction force. Until midstance, the posterior and then the anterior gluteus medius/ minimus were consecutively active, with the soleus and gastrocnemius providing support in late stance. However the contribution from the soleus was almost twice that of the gastrocnemius. Biarticular muscles such as the hamstrings or rectus femoris were found to contribute little to the vertical ground reaction force at any time in stance.

Many of these studies concentrated on muscles or moments acting in the sagittal plane and the effects in the other planes were not considered (Jonkers et al 2003, Kepple et al 1997, Neptune et al 2001).

Hip and knee extension in the single limb support phase has been modelled (Arnold et al 2005a). The gluteus maximus was found to contribute substantially to the hip extension moment and the vasti to the knee extension

275

moment in early single limb stance. It was noted that the gluteus maximus rivalled the vasti in promoting knee extension in the simulation. Mid to late single support, the hip and knee extension were more reliant on the contributions of the posterior section of the gluteus medius and the soleus respectively. This research indicates the importance of the soleus in providing stability in knee extension and its contribution to the development of crouch when weakened.

Propulsion for walking was studied by having normal adults walk on a treadmill with a horizontal force applied at waist level that impeded or aided forward progression (Gottschall and Kram 2003). Energy cost and EMG recordings were taken. It was found that the impeding force increased energy cost much more than the aiding force decreased energy cost. Individual muscle EMG showed different responses. The medial gastrocnemius showed a decrease in magnitude of EMG activity when the force applied aided forward progression (59%) and increased 190% with an impeding force. The soleus activity did not alter greatly in response to the aiding force but increased by 159% with impedance to movement. Tibialis anterior activity was unaltered. From these results the authors inferred that the medial gastrocnemius was important for propulsion in gait, not soleus. However as the authors pointed out, the EMG in this study only provides information regarding the timing and magnitude of the muscle's activation and not the force developed by the muscle. Even with this limitation, this study does support the finding by Neptune et al (2001) that the gastrocnemius contributes to propulsion in gait. Clinically this study implies that in severe crouch gait, with compromised plantarflexors, propulsion will be decreased as the gastrocnemius is impaired and the soleus will not be able to be recruited to aid gastrocnemius as it did when an impeding force was applied in the study. If propulsion is diminished in pathological gait then energy cost will increase as a consequence, as this will be similar to applying an impeding force. This would apply to the severe crouch cohort, as plantarflexor function was compromised pre-SEMLS and with the improvements found at the ankle post-SEMLS, particularly A2, forward progression in gait should be less hindered.

From this study of severe crouch subjects, the long term use (at least to 12 months post-SEMLS) of GRAFOs or solid AFOs in severe crouch gait, particularly when a growth spurt was occurring, seems to be justified by the improvements found at the ankle post-SEMLS. No direct surgery to the calf in the sagittal plane was undertaken at the time of SEMLS or thereafter, and so to obtain plantargrade biomechanical alignment of the tibia and foot the wearing of GRAFOs or solid AFOs was instituted. This must have had some role in improvement and maintenance of the ankle parameters in gait found at five years post-SEMLS.

### Coronal Plane

This plane has only been analysed in crouch gait in the study by Steinwender et al (2001). In that study, mean abduction in the first half of stance, and in single support was not different to that exhibited by subjects who did not walk in crouch or normally developing subjects. Conversely in early swing, mean adduction for their crouch cohort was significantly different to the other groups according to statistical analysis and the graphs shown, although this was not commented on in the paper.

At the time of SEMLS four subjects from the severe crouch cohort underwent adductor longus lengthening. Prior to SEMLS, maximum adduction in stance and swing were increased with respect to normal values. This adduction decreased post-SEMLS but was not maintained at five years, in the swing phase. The mechanism for this tendency for deterioration is not clear. The adductors have been documented as developing forces during stance but do not have a significant role in support of the body during this phase, and it was considered that their force would probably be directed at controlling the pelvis in the coronal plane (Anderson and Pandy 2003). Increased medial and lateral ground reaction forces have been recorded in subjects who had undergone staged orthopaedic surgery and were tending to walk in crouch gait (Fabry et al 1999). Again this was attributed to the need to gain pelvic stability in the coronal plane in stance phase. However this does not explain why the adduction increased again during swing phase at five years post-SEMLS.

# Transverse Plane

There has been great emphasis placed on the correction of lever arm dysfunction in pathological gait, particularly that of crouch gait (Gage 2004f). Lever arm dysfunction is found in all three planes. In crouch gait, the lever arm dysfunction in the sagittal plane is derived from the position of the segments of the lower limb i.e. the flexed hips, knees and ankles. Excessive flexion at these joints leads to the biarticular muscles experiencing a change in length of the moment arm at the joints on which they act. For example, the moment arm of the hamstrings at the knee is increased in crouch gait due to the excess flexion in the sagittal plane, and its ability to provide force at the knee is enhanced by this flexion. This means that its action at the hip as an extensor is compromised. The coronal plane may have an unstable fulcrum if there is hip subluxation present or a short lever arm for the abductor muscles if femoral neck anteversion is present. Both scenarios will decrease the force production available at the hip. In the transverse plane, dysfunction can result from malalignment of the femur and tibia due to femoral anteversion and external tibial torsion. This type of lever arm dysfunction has been coined "malignant malalignment syndrome" (Gage and Schwartz 2004c, p. 198). The presence of femoral neck anteversion will decrease the effective length of the lever arm of the gluteus medius and will be partially compensated for by an increase in internal rotation of the hip in an attempt to lengthen this lever and restore mechanical efficiency to the gluteus medius (Arnold et al 1997). External tibial torsion shortens the lever arm of the foot by maldirection due to the external rotation of the foot as a consequence. Thereby this diminishes the ground reaction force generated and reduces the competency of the plantarflexion knee extension couple generated (Schwartz and Lakin 2003). The foot in the transverse plane can also contribute to lever arm dysfunction, if the posture of the foot is flexible due to pes valgus, midfoot planus and forefoot abduction. Such a foot posture is an example of a flexible lever as the foot is pliable due to the midfoot break and does not provide a rigid lever on which the moment can act (Gage and Schwartz 2004c). In order for muscles to produce adequate force the levers need to be restored (Figure 7.25). In the transverse plane, the femoral neck anteversion can be corrected by external derotation osteotomy of the femur either proximally or distally. The tibial torsion can be rectified by a

supramalleolar osteotomy of the tibia and fibula. The foot posture can be corrected by foot stabilisation surgery such as os calcis lengthening to align the lateral column of the foot, or subtalar fusion to stabilise not just the forefoot and midfoot but the hindfoot also.

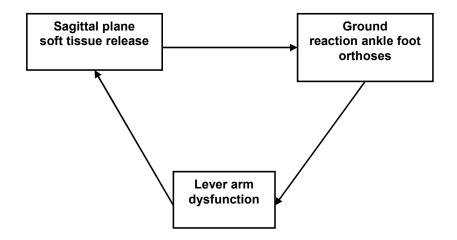


Figure 7.25 After sagittal plane soft tissue release surgery, ground reaction ankle foot orthoses (GRAFOs) provide support to stabilise the ankle in mild plantarflexion to force the ground reaction force (GRF) anterior to the knee. This artificially restores the plantarflexion knee extension couple. However if the bony levers consisting of the femur, tibia and/ or foot are malaligned the GRAFO is unable to provide effective redirection of the GRF. If the lever arm dysfunction is not corrected by bony derotation surgery, the knee will tend to remain in flexion and eventually redevelop contracture requiring further soft tissue surgery.

Only two subjects did not have bony surgery directed to correction of transverse plane lever arm dysfunction. These children were the youngest subjects of the cohort. Overall, 5/14 subjects had only femoral derotation osteotomy, 1/14 had only supramalleolar osteotomy, 1/14 had only foot stabilisation, 1/14 had supramalleolar osteotomy and foot stabilisation, 2/14 had femoral derotation osteotomy and foot stabilisation, and 2/14 had all three levels. The internal rotation of the hip over stance phase was improved post-SEMLS but the long term result at five years was less convincing as there was the tendency towards internal rotation although not near pre-SEMLS levels. Follow-up studies at one year post-femoral derotation surgery have found that the hip is less internally rotated on 3DGA parameters (Kay et al 2004b, Kay et al 2003, Murray-Weir et al 2003, Pirpiris et al 2003). However long term results have not been conclusive as one study showed maintenance of this improvement on 3DGA but not on physical examination (Õunpuu et al 2002a), and another study

(documented in an abstract) reported deterioration on 3DGA (Kim et al 2004). The long term deterioration found in that study was predominantly in children less than 10 years old at time of operation (Kim et al 2004). In the study by Brunner and Baumann (1997), which retrospectively assessed recurrence of increased femoral neck anteversion at follow-up between 11 and 18 years post-derotation osteotomy of the femur, recurrence was found mainly in children under the age of eight years at the time of the surgery. It was also noted that these children were more severely involved (Brunner and Baumann 1997).

There has been debate over whether better results are afforded by the proximal versus the distal derotation osteotomy of the femur and so far the results have not shown convincing evidence in support towards either osteotomy site (Kay et al 2003, Õunpuu et al 2002b, Pirpiris et al 2003). Interestingly it has been commented that despite obtaining considerable external rotation intraoperatively, on gait analysis the decrease in hip internal rotation is always less than that at time of the surgery (Kay et al 2003, Pirpiris et al 2003). The femoral osteotomies in our severe crouch gait study were undertaken at either osteotomy site. Of the studies cited above it is worth noting that only one study had a cohort of only children with spastic diplegia (Pirpiris et al 2003). The other studies included children with spastic diplegia, hemiplegia and quadriplegia as a group (Brunner and Baumann 1997, Kay et al 2004b, Kay et al 2003, Murray-Weir et al 2003, Õunpuu et al 2002a).

In the past, the cause of internal rotation during gait has been assigned to spastic muscles such as the hamstrings (Sutherland et al 1969) and adductors. Using three dimensional computer modelling, a more recent study indicated that an internal rotation gait is an attempt at compensation for a decreased lever arm of the gluteus medius and minimus caused by increased femoral anteversion (Arnold et al 1997). The increased femoral anteversion is believed to be the result of persistent fetal malalignment caused by failure to remodel the proximal femur (Bache et al 2003, Gage and Schwartz 2004c). Remodelling normally results from pressure on the femoral neck by Bigelow's ligament when the hip is extended in gait (Bache et al 2003). However the child with cerebral palsy, is invariably delayed in the acquisition of walking and when able to walk, hip

extension is usually incomplete (Gage and Schwartz 2004c). Bone remodelling is greatest in the first year of life when rate of growth is greatest and so delayed walking in children with cerebral palsy leads to remodelling at a time of slower rate of growth (Gage and Schwartz 2004c). Hence increased femoral anteversion in cerebral palsy is a very common finding.

With increasing hip flexion, the internal rotation moment arm of the hip has been found to increase, and consequently part of the gluteus medius and maximus muscles display a change in their respective moment arms at the hip from external to internal (Delp et al 1999). This is relevant to subjects who are walking in crouch gait as the increased hip flexion may well be a reason for an increase in hip internal rotation in conjunction with an increased femoral neck anteversion. In the study by Delp at al (1999), the iliopsoas was shown to have an internal rotation moment arm at the hip that was small and was therefore not considered to be involved directly in increasing hip internal rotation, although it may be indirectly involved due to its ability to flex the hip. In simulations of crouch and internally rotated gait, the moment arms of the hamstrings and adductors at the hip have been shown to be negligible or external in rotation (Arnold et al 2000, Arnold and Delp 2001b), and that of the gluteus medius to be internally rotated even more with increasing hip flexion (Arnold and Delp 2001b). So the involvement of the hamstrings and adductor muscles in causing internal rotation gait is now seriously challenged and targeting of these muscles for lengthening to alleviate internal rotation gait has been questioned (Arnold et al 2000).

The effect of external derotation osteotomy of the femur on the length of muscles around the hip has been of concern as it has been suggested in the past that lateral hamstrings may have a decreased length following this type of surgery (Gage 1991). In addition the adductor longus tends to become tighter with femoral derotation osteotomy as shown by muscle length modelling (Rab 1991). The concern arose from the notion that if the muscles become tighter post-femoral derotation, this could lead to recurrence of the deformity. The effect of external derotation osteotomy of the femur on the hamstrings and adductor muscles for varying osteotomy sites, degrees of derotation of the

osteotomy, femoral neck anteversion angles and rotation angles of the hip, has been shown to be minimal and consequently the need for concomitant lengthening of these muscles unnecessary (Schmidt et al 1999). This implies that the inclusion or exclusion of hamstrings or adductor muscle lengthenings at the time of SEMLS is probably not the reason for the mild recurrence of internally rotated gait seen five years post-SEMLS in the severe crouch group.

Despite the consistency of the foot progression on kinematic data, the variability as observed in the transverse plane graphs (Figure 7.19), standard deviations (Table 7.19) and 95% confidence intervals (Table 7.20) at baseline decreased at the one and five year time periods. Six subjects underwent supramalleolar osteotomies, foot stabilisation surgery or both at the time of SEMLS. The decrease in variability of the foot progression indicates that the interpretation of the level at which the malalignment was occurring (i.e. the femur, tibia and/ or foot) was astutely made, as the transverse plane surgery decreased the variability and resulted in a foot progression that was close to normal values.

Increased external tibial torsion has been shown on computer modelling to decrease the acceleration of the knee into extension, to increase knee acceleration into valgus, to decrease the support function of the soleus in stance phase and to decrease the forward acceleration of the centre of pressure (Schwartz and Lakin 2003). Supramalleolar (Selber et al 2004) or distal tibial osteotomy (Dodgin et al 1998) are accepted as appropriate surgical interventions for correction of tibial torsion (Dodgin et al 1998, Selber et al 2004, Stefko et al 1998).

Also of importance is the integrity of the structure of the foot. As stated earlier in this section, a planus, abducted, valgus foot will not provide a rigid lever on which the ground reaction force can act (Gage and Schwartz 2004c) and the lever will also be malaligned and shortened by the abduction of the forefoot (Duffy and Cosgrove 2002). The lateral column of the foot is shortened in such a foot and calcaneal lengthening has been shown to restore the lateral column of the foot (Andreacchio et al 2000, Duffy and Cosgrove 2002, Evans 1975b, Mosca 1995, Noritake et al 2005). This operation was originally thought to be unsuitable for patients with spasticity due to concern regarding overcorrection of the deformity (Evans 1975b), however since then this surgery has been shown to be safe and effective in children with cerebral palsy (Andreacchio et al 2000, Mosca 1995, Noritake et al 2005). Lateral column lengthening is thought to be indicated for flexible, mild to moderate planovalgus feet in the ambulatory child (Andreacchio et al 2000). Its advantage over a subtalar fusion is that motion is maintained at the subtalar joint and is thought to protect against degenerative arthritis that has been associated in the long term with subtalar fusions and triple arthodeses (Andreacchio et al 2000, Mosca 1995). Subtalar fusion may still be used to correct alignment of the foot if the deformity is severe and the foot is not flexible i.e. not corrigible to correct the foot alignment issues in the severe crouch cohort and at five years the results according to foot progression parameters were satisfactory.

Between one and five years post-SEMLS, the most common operation was for ingrown toenails (five subjects-16 operations). Ingrown toenails can be one of the symptoms associated with hallux valgus (Duffy and Cosgrove 2002). There were also metatarso-phalangeal joint arthrodeses performed to correct hallux valgus in the severe crouch group but to a lesser extent than surgery for ingrown toenails. The development of hallux valgus is thought to be a consequence of muscle imbalance and transverse plane malalignment (Davids et al 2001). The effect of muscle imbalance is supported by the fact that children who are non ambulatory develop hallux valgus (Davids et al 2001). However malalignment and consequent abnormal loading on the foot is also a feasible explanation. During 3<sup>rd</sup> rocker in terminal stance, if the foot is externally rotated in relation to the line of progression, as the centre of pressure moves towards the metarsophalangeal joint it will be directed medially in respect to the foot and force the hallux into valgus and the foot and tibia into more external rotation. If this is coupled with reduced peak knee flexion or a foot drop in swing phase then the medial aspect of the hallux and foot will experience further forces to their medial aspects. This will also promote external rotation of the tibia (Selber et al 2004). To correct the hallux valgus successfully, any abnormal alignment in the transverse plane of the femur, tibia and foot must first be dealt with so that the

deforming medial forces are removed and arthrodesis of the metarso- phalangeal joint is successful. If this is achieved then the incidence of ingrown toenails should also lessen.

# 7.4.2 Maintained Status Post-SEMLS

# **Body Structure and Function**

# Temporal-spatial Parameters

Improvement in temporal-spatial measures was not observed despite the subjects' considerable growth in height between baseline and five years post-SEMLS, and improvement in overall knee extension and rectification of excessive dorsiflexion in stance phase. Normalisation of the velocity and stride length by height diminished the influence of having longer limbs due to growth on the two parameters, at the different time points. However increased extension in the sagittal plane did not have the effect of increasing velocity and stride length. Lengthening the hamstrings has been purported to aid in achieving increased knee extension at initial contact, which would contribute to increased stride length. However this was not seen, even though extension of the knee at initial contact did increase.

The timing of events in the gait cycle such as toe off, opposite foot off and opposite foot contact were resistant to change, perhaps because timing of movement is more dependent on the subjects' difficulty with selective motor control, which is a function of the central nervous system. Orthopaedic surgery is unable to effect change in this area of dysfunction. Prolonged stance and double support phases suggest that balance and stability present difficulties for these subjects in their efforts to mobilise (Sutherland et al 1988). Similar lack of improvement in temporal-spatial parameters has been reported post-surgery (Rethlefsen et al 1999b, Saw et al 2003, Thometz et al 1989). However a lack of improvement is not necessarily a negative outcome, as there is growing evidence in the literature that the natural history of gait in cerebral palsy is one of deterioration (Bell et al 2002, Gough et al 2004, Johnson et al 1997). Maintenance of gait parameters is therefore still a positive outcome from surgery.

# Hip Parameters in the Sagittal Plane

Despite published reports of predominantly improved hip extension with hamstring and/ or psoas lengthenings (Chambers et al 1992, DeLuca et al 1998, Novacheck et al 2002, Sutherland et al 1997), mean hip extension was not altered post-SEMLS in our cohort which is similar to findings of other studies (Kay et al 2002, Zwick et al 2002). The only hip parameter to show change was the hip power generation in late stance and that was an increase to within normal values. This is in contrast to the hip power generation in loading response remaining elevated compared to normal values, implying that hip power in loading response of the forward limb in stance is still being utilised to supplement the power generation of the trailing limb at toe off (Steinwender et al 2001, Zwick et al 2001) (Figure 7.8, 7.9 and 7.10). The ankle power generation in late stance did not increase to normal values although it was improved compared with pre-SEMLS values, hence the need for hip power generation in loading response to compensate for this (Zwick et al 2001). Other studies reporting unaltered maximum hip extension in stance post-SEMLS, showed unchanged hip power generation in loading response and pre-swing (Zwick et al 2002).

# Timing of Parameters within Stance or Swing Phase

There was no change in the timing of parameters in the relevant phases of the gait cycle except for that of peak knee flexion in swing phase and in the gait cycle. Although rectus femoris transfers were only undertaken in a few subjects and normalised speed was not increased, timing of this parameter was earlier and within normal limits. A change in timing of parameters was not necessarily expected as the timing is related to the upper motor neurone symptoms, which reflect the balance between the positive and negative features (Mayer 1997). Pathological gait is the result of a complex interaction between the upper motor neurone symptoms and their influence on the muscles that control the body, and the biomechanical circumstances that result initially from this, and then the influences of developing contracture and growth on the body over time. The negative symptoms of muscle weakness, loss of selective muscle control and poor balance, coupled with the positive symptoms of spasticity, hyper-reflexia and co-contraction can lead to change in muscle physiology that will impact on

timing, sustainability and duration of a muscle contraction (Mayer 1997, Rose and McGill 2005). As stated earlier in Section 7.4, the SEMLS will alter the musculoskeletal pathology but will not directly alter the upper motor neurone symptoms. However there may be an indirect effect by permitting retraining of movement or reorganisation of the subsystems involved in producing walking as proposed by the dynamic systems theory (Giuliani 1991, Shumway-Cook and Woollacott 2001). In the case of the change in timing of peak knee flexion, the SEMLS may have indirectly altered the dominance of an agonist/ antagonist such that the biomechanical circumstances were altered and timing was subsequently also changed. An example of this relates to the hamstrings lengthening. Surgical lengthening further weakened their ability to stabilise the pelvis and the action of the psoas muscle may thereby have been promoted. As the iliopsoas has the potential to increase knee flexion velocity during double support in normal gait simulations (Goldberg et al 2004), this may have led to the change in timing of the peak knee flexion in swing. The timing of peak angles and peak velocities as a percentage of the gait cycle have been found to be relatively unchanged post muscle-tendon lengthening surgeries, indicating the limited ability of such surgery to alter the underlying neurological input to gait (Granata et al 2000).

### 7.4.3 Negative Changes Post-SEMLS

#### **Body Structure and Function**

#### Pelvic Parameters in the Sagittal Plane

There was an increase in anterior tilt of the pelvis after SEMLS. There was only one subject at one year post-SEMLS and three subjects at five years who had a pelvic tilt within normal range, no subjects had a posterior tilt and the remaining subjects had an anterior tilt. The subjects who at baseline had a posterior pelvic tilt, changed to an anterior pelvic tilt at both time periods post-SEMLS. As the reliance on assistive devices was similar at five years after SEMLS to pre-SEMLS, the increased tilt cannot be attributed to their use. The pelvic excursion was unchanged, so the increased excursion at baseline was still needed for walking. There was a dominance of hamstrings lengthening undertaken in the SEMLS prescription and not all were accompanied by simultaneous psoas lengthening over the pelvic brim (five subjects had combined hamstrings and psoas lengthenings and eight subjects had hamstring surgery without psoas surgery). There was also a high incidence of medial and lateral hamstrings lengthening for the cohort (eight subjects), and of these only two subjects had psoas lengthening over the brim as part of the surgical prescription. Four subjects had had prior hamstring surgery; one subject had recorded three occasions of hamstring lengthening. The emphasis on hamstring surgery may have had consequences for the position of the pelvis.

The increased knee flexion seen in crouch has been predominantly attributed, in the past, to contracture of the hamstrings (Baumann et al 1980, Chang et al 2004, Damron et al 1993, Dhawlikar et al 1992, Eggers 1952, Hsu and Li 1990, Kay et al 2002, Keats and Kambin 1962, Reimers 1974). However tightness in the hip flexors can lead to knee flexion due to postural adjustments in order to maintain the upright position (Reimers 1973, Roosth 1971, Zwick et al 2002). If the hamstrings are targeted for surgery and the psoas is not, increased lordosis can occur as the knee and hip are coupled by the biarticular hamstrings and imbalance between the lengthened hamstrings and the tight psoas leads to tilting of the pelvis even further forwards (Hoffinger et al 1993, Reimers 1973). Increased anterior pelvic tilt has been attributed to lengthening of hamstrings, which according to muscle-tendon length calculations were not short prior to the surgical intervention (Arnold et al 2005b). Improvements in knee extension at initial contact and terminal swing have been gained from surgical prescriptions that did not include hamstrings lengthening (Arnold et al 2005b, Arnold et al 2005c). The implication therefore is that the hamstrings are not necessarily involved in every case of flexed knee posture in gait. Calculation of muscletendon length or lengthening velocity of the hamstrings prior to intervention may help delineate when hamstrings lengthening should or should not be undertaken and therefore prevent deterioration in anterior pelvic tilt posthamstrings surgery.

Lengthening of the hamstrings will lead to some muscle weakness (Kay et al 2002, Zwick et al 2002) and, as it has been shown that the hamstrings contribute about a third of the total hip extensor torque (Waters et al 1974), hip flexion and anterior tilt of the pelvis are likely consequences (Hoffinger et al 1993). If the

psoas is targeted for surgery instead of the hamstrings, the pelvis may revert to a more neutral position and as a result the hamstrings gain length as their origin and insertion are brought closer together (Delp et al 1996, Reimers 1973). If the hamstrings are also deemed to be contracted, then combining the lengthening of the hamstrings and psoas in the one surgical session may combat an increase in lordosis (Hoffinger et al 1993). However the weakening of the hamstrings will reduce their ability to act as hip extensors leading to a reliance on the gluteal muscles to extend the hip. The gluteals are rarely found to be strong enough to be effective hip extensors (Reimers 1973, Waters et al 1974). It should be mentioned that not all studies have shown that the hamstrings are permanently weakened by muscle lengthening (Damiano et al 1999) but this is contrary to the findings of other studies which showed weakness of the hamstrings post-surgery (Kay et al 2002, Zwick et al 2002).

The strength of the abdominals in helping to combat lordosis and maintain a normal pelvic tilt should perhaps also be considered (Baker 1956, DeLuca et al 1998, Evans 1975a, Trost 2004). Increased lordosis was found after proximal hamstring releases if on pre-operative examination there was a fixed flexion deformity at the hip of 25° or more, already increased lordosis and weak abdominals (Drummond et al 1974). In addition weakening of the psoas is not without its consequences as it is needed to propel the hip into swing phase and any lengthening must aim to preserve this function (Delp et al 1996). So far most studies have found that post-psoas lengthening, hip flexor strength has been preserved (DeLuca et al 1998, Novacheck et al 2002, Sutherland et al 1997). This may be explained by computer simulation of tendon lengthening, which has indicated that the hip flexor capacity of the iliopsoas is relatively undiminished with tendon lengthening (Delp and Zajac 1992). Muscle length estimation has shown that the hamstrings are not necessarily short in crouch gait (Arnold et al 2005b, Delp et al 1996, Hoffinger et al 1993, Schutte et al 1997) and the psoas is more likely to be the short muscle (Delp et al 1996), however this is also dependent on the method of modelling used to calculate the length of the psoas muscle (Schutte et al 1997). In contrast, short dynamic hamstring length in crouch gait has been documented in a conference abstract (Do et al 2004). Care with interpreting muscle lengths is needed, as a short muscle length

during the gait cycle does not necessarily mean that a muscle is contracted or spastic, as it may be a compensatory response to abnormal biomechanics at another joint (Schutte et al 1997).

It has been proposed that examination of the tilt of the pelvis, together with EMG activity of the gluteus maximus and hamstrings can help define whether the hip flexors are tight, or the hamstrings or both (Tylkowski et al 1988). However no further research on this possible association has been found. It is certainly known that changes seen in physical examination do not necessarily correspond with changes in 3DGA parameters: e.g. the popliteal angle does not show correlation with knee kinematics (Chang et al 2004, Õunpuu et al 2004) or hamstring length (Delp et al 1996, Do et al 2004, Hoffinger et al 1993, Schutte et al 1997), the straight leg raise shows no association with gait parameters (Thometz et al 1989), hip flexion contracture shows no correlation with maximum hip extension in stance or mean pelvic tilt (DeLuca et al 1998), neither does hip extensor strength or popliteal angle (Õunpuu et al 1995).

Many different surgical procedures for hamstring contracture have been documented over the years: transplantation of hamstrings to femoral condyles (Eggers 1952, Eggers and Evans 1963, Evans 1975a, Gage 1991, Metaxiotis et al 2004, Pollack and English 1967), proximal hamstring recession (Drummond et al 1974, Seymour and Sharrard 1968), proximal semimembranosus release combined with distal semitendinosus and biceps femoris release (Damron et al 1991), distal elongation of medial and lateral hamstrings (Hsu and Li 1990), and distal fractional lengthening (Thometz et al 1989). A number of unwanted complications keep occurring post-hamstring surgery: recurrence of contracture (Chang et al 2004, Dhawlikar et al 1992, Roosth 1971), lack of knee flexion in swing (Baumann et al 1980, Chang et al 2004, Damron et al 1993, Dhawlikar et al 1992, Hsu and Li 1990, Roosth 1971, Thometz et al 1989), increased anterior pelvic tilt/ lumbar lordosis (Chang et al 2004, DeLuca et al 1998, Drummond et al 1974, Hsu and Li 1990, Zwick et al 2002) and genu recurvatum (Chang et al 2004, Damron et al 1991, Dhawlikar et al 1992, Drummond et al 1974, Hsu and Li 1990, Kay et al 2002, Simon et al 1978, Thometz et al 1989, Zwick et al 2002). It has been proposed that distal hamstrings lengthening should be

"carefully graduated" (Baumann et al 1980, p. 308) to take into consideration the function of the individual hamstrings and the goals of the surgical intervention by replacing tendon elongations with fractional lengthenings of the aponeurosis. It was recommended that the semitendinosus and short head of biceps be targeted for lengthening as they were considered to be predominantly contracting in swing phase and loading response and the semimembranosus and long head of biceps were thought to provide the extension of the hip and knee stability in stance (Baumann et al 1980). If increased anterior pelvic tilt or knee hyperextension occurred post-surgery it was probably due to impairment of the latter muscles (Baumann et al 1980). Contrary to this, electromyography conducted in another laboratory (Perry 1992) indicated that the semitendinosus normally shows the greatest activity in stance phase, rather than semimembranosus. Computer modelling of the effect of tendon lengthening surgery has shown that semimembranosus is responsible for most of the contribution to hip extensor force from the hamstrings but is much more sensitive to tendon lengthening than the semitendinosus (Delp and Zajac 1992). This implies that in order to retain hip extensor force, surgical lengthening should be directed towards the semitendinosus rather than the semimembranosus, in accordance with recommendations by Baumann et al (1980).

Research on the effect of psoas surgery with or without hamstring lengthening, or the effect of hamstring surgery with or without psoas lengthening, has been marred by concomitant procedures being undertaken, which muddy the interpretation of the results (Chang et al 2004, Damron et al 1993, DeLuca et al 1998, Dhawlikar et al 1992, Hsu and Li 1990, Kay et al 2002, Novacheck et al 2002, Reimers 1974, Sutherland et al 1997, Thometz et al 1989). In the study by Sutherland et al (1997) on psoas release at the brim, 15 of the 17 subjects had hamstrings lengthening. Improved maximum hip extension was found, as was pelvic tilt in the younger subjects but the pelvic tilt remained unchanged for the whole cohort (Sutherland et al 1997). The majority of the subjects who had psoas lengthenings also had combined hamstring lengthenings in the study by Novacheck et al (2002). Again improvement in maximum hip extension in stance,

was also noted in the study by DeLuca et al (1998). Conversely, combined psoas and hamstring lengthenings have been reported to lead to increased anterior pelvic tilt, and a further decrease in maximum hip extension in stance (Zwick et al 2002). This was attributed to over-lengthening of the hamstrings due to a reliance on measurement obtained from a popliteal angle that did not control for pelvic position and therefore an underestimation of the length of the hamstrings. Pelvic position pre-surgery has been identified as a main determinant of post-surgery pelvic tilt (Chambers et al 1992, DeLuca et al 1998) together with the extent of hamstring surgery undertaken (DeLuca et al 1998). Increased anterior pelvic tilt pre-surgery remains increased after surgery regardless of the type of surgery (Chambers et al 1992, DeLuca et al 1998). If the pelvic position is within normal limits prior to surgery, then it remains the same post-surgery if only the medial hamstrings are lengthened (DeLuca et al 1998). There is an increased anterior tilt if the medial and lateral hamstrings are included (DeLuca et al 1998). Posterior pelvic tilt pre-operatively was reported to change to an increased anterior tilt after surgery (Chambers et al 1992) but in that study the extent of hamstring surgery (medial or lateral or both) was not documented. The pre-operative pelvic position of the severe crouch cohort in this study had a maximum and minimum pelvic tilt that was within normal values and all except one subject had hamstrings lengthening (the majority had medial and lateral hamstrings surgery). It would be expected from the published research findings that the tilt would move anteriorly post-SEMLS and that was confirmed in this study.

A recent abstract reported that with repeat hamstrings lengthening incorporating lengthening of both the medial and lateral hamstrings, pelvic tilt shifted anteriorly (Westwell et al 2004). This was not the case if repeat hamstring lengthening was confined to the medial hamstrings. Four subjects in the severe crouch cohort had had prior hamstrings lengthening, and three of these subjects then had repeat medial and lateral hamstrings lengthening at the time of SEMLS. The increased anterior pelvic tilt in the severe crouch group may therefore have been confounded by the repeat hamstring lengthenings.

Preserving the strength of the hamstrings as hip extensors but decreasing their influence at the level of the knee (Gage 1991), would be an ideal scenario. The transfer of the hamstrings from their tibial insertion to the distal femur in order to achieve this scenario has been reported in the past (Eggers 1952, Eggers and Evans 1963, Evans 1975a, Gage 1991, Metaxiotis et al 2004, Pollack and English 1967). Originally the semitendinosus, semimembranosus, biceps femoris and gracilis were transferred (Eggers 1952) but with time it was realised that the extent of transfer needed be modified to take into account the "degree and pattern of involvement" (Eggers and Evans 1963, p. 1296). Achieving full knee extension was no longer the aim, and the need to obtain a balance between the knee flexors and extensors was recognised (Eggers and Evans 1963, Evans 1975a). The transfer of all the hamstring muscles led to knee flexion weakness, and so retaining some of the hamstrings at their original insertion to augment this was considered wise (Pollack and English 1967). A case study by Gage et al (1991) advocated the transfer of only the semitendinosus to the distal lateral femur for retaining hip extensor strength, decreasing knee flexion and to enhance external rotation at the hip. A recent variation involved only the transfer of the semitendinosus from the tibia to the stump left by the medial head of the gastrocnemius, which in turn had been transferred to the proximal tibia (Metaxiotis et al 2004). The effect of this semitendinosus transfer was difficult to ascertain as simultaneous transfers of the gastrocnemius and rectus femoris were conducted. Genu recurvatum was reported in four cases (out of 40 limbs) but the pelvic anterior tilt was not increased (Metaxiotis et al 2004).

In retrospect, it could be considered that perhaps too many hamstring lengthenings were undertaken, and the extent of hamstrings lengthening was excessive in our cohort. In the cases in which both the medial and lateral hamstring lengthenings were deemed necessary, the subjects may have been better served by medial hamstrings lengthening with semitendinosus transfer to the distal femur, in order to minimise the effect on the pelvis but still gain effect at the knee. Hamstring lengthening for fixed flexion deformity at the knee is not sufficiently efficient due to its inadvertent effect at the pelvis.

292

# Knee Parameters in the Sagittal Plane

Ten subjects had some form of rectus femoris surgery either at the time of the SEMLS surgery (four subjects had rectus transfers to semitendinosus- seven limbs, and to gracilis- one limb; and one subject had proximal release) or in the intervening years (five subjects had proximal release and one subject who had had the previous proximal release with SEMLS had a rectus transfer to tensor fascia lata). However the peak knee flexion in swing was reduced after SEMLS at both time periods, and is most likely due to the high incidence of hamstring lengthenings (13/14 subjects). Only four subjects had rectus femoris transfers at the time of SEMLS and a total of five subjects had rectus transfers before five years. Decreased knee flexion in swing after hamstring lengthening without rectus femoris transfer has been well documented in the literature (Baumann et al 1980, Chang et al 2004, Damron et al 1993, Dhawlikar et al 1992, Hsu and Li 1990, Roosth 1971, Thometz et al 1989, van der Linden et al 2003a, Wren et al 2005a). Rectus femoris transfer, when compared to proximal or distal rectus release has been shown to be more effective in achieving a maintenance of, or an increase in peak knee flexion in swing (Chambers et al 1998, Ounpuu et al 1993b, Sutherland et al 1990). This probably explains why the peak knee flexion in swing decreased in this study because at five years, only five subjects had had rectus transfers and six subjects had had proximal rectus releases. Long term follow-up of rectus femoris transfer in comparison to no rectus femoris surgery has shown deterioration in peak knee flexion and range of motion in swing with time, similar to the natural progression of gait that shows deterioration in these parameters (Moreau et al 2005). This would explain the data for four subjects who did not have any rectus femoris surgery at the time of SEMLS or in the intervening period prior to five years afterwards. The site to which the rectus femoris is transferred, has been found to be inconsequential (Chung et al 1997, Gage et al 1987, Ounpuu et al 1993a) and thereby does not influence our results.

It seems to be uniformly reported that peak knee flexion increases following rectus femoris transfer (Chambers et al 1998, Gage et al 1987, Kay et al 2004a, Miller et al 1997, Moreau et al 2005, Saw et al 2003, Sutherland et al 1990) or is maintained at a pre-surgery level (Chung et al 1997, Hadley et al 1992, Õunpuu

et al 1993a, Õunpuu et al 1993b, Rethlefsen et al 1999b, Yngve et al 2002) and knee range of movement is increased (Chambers et al 1998, Chung et al 1997, Gage et al 1987, Hadley et al 1992, Kay et al 2004a, Moreau et al 2005, Õunpuu et al 1993a, Õunpuu et al 1993b, Rethlefsen et al 1999b, Sutherland et al 1990, Yngve et al 2002). Subjects dependent on assistive devices, have shown a decrease in peak knee flexion in swing following rectus transfer (Yngve et al 2002) and this was the case in five subjects in our study, who were aid dependent prior to SEMLS. Knee excursion was increased post-SEMLS in this study but this improvement was due to decreased minimum knee flexion in stance as the peak knee flexion had decreased. This is similar to other studies that have reported improvement in knee excursion attributable to the improved knee extension rather than an increase in peak knee flexion, which was often unchanged post-surgery (Hadley et al 1992, Kay et al 2004a, Rethlefsen et al 1999b, Yngve et al 2002) or decreased (Thometz et al 1989, van der Linden et al 2003a).

Activity during swing phase only or constant activity over the whole gait cycle, are the most common abnormal findings on electromyography for rectus in children with cerebral palsy (Chambers et al 1998, Miller et al 1997, Perry 1987) with activity in swing and early stance, or activity in stance only also recorded (Perry 1987). Rectus release has been advised only if rectus was active in stance in order to help relieve increased hip flexion (Perry 1987). Proximal rectus release was undertaken in many subjects in this cohort for a variety of reasons that included parental choice and simpler rehabilitation and in an effort to decrease hip flexor moments, improve pelvic tilt and hip extension. Rectus transfer was indicated if there was inadequate knee flexion in swing phase (Perry 1987). Vastus lateralis activity in swing, as well as that of the rectus has been thought to be a contraindication to the rectus transfer being effective but this has not been substantiated (Chambers et al 1998) and is not considered by other groups (Sutherland et al 1990). The children who had constant rectus activity throughout the gait cycle showed less improvement than the children with only swing phase abnormality, and therefore it was hypothesised that children with constant rectus activity were more severely

affected and may require the constant rectus activity to maintain stability in gait (Miller et al 1997).

At five years post-surgery, there was improved timing of the peak of knee flexion within swing phase which was similar to normal values. However timing of peak knee flexion within the gait cycle was only significantly earlier at five years but still outside the normal range. The rate of change (degrees/ second) from maximum knee extension in stance to peak knee flexion in swing improved compared with pre-SEMLS values but was still significantly outside the range of normative values. As noted earlier the normalised speed remained unchanged at one and five years. With rectus transfer, many authors have noted an improved timing of peak knee flexion in swing phase (Kay et al 2004a, Õunpuu et al 1993a, Õunpuu et al 1993b, Rethlefsen et al 1999b, Sutherland et al 1990, Yngve et al 2002) or in the gait cycle (Moreau et al 2005), whilst others have not (Chambers et al 1998, Hadley et al 1992). Conversely, earlier timing of the peak knee flexion in swing has been found without rectus surgery having been performed and has been attributed to a decrease in velocity post-surgery (van der Linden et al 2003a). It has been shown in one study (van der Linden et al 2002) that normally developing children walking at slow velocities, have an earlier peak in respect to swing phase but the timing of the peak is constant in respect to the gait cycle (74%) despite changes in velocity. However as normalised velocity remained unchanged post-intervention in our study, the earlier peak in swing and in the gait cycle were considered positive improvements.

The amplitude of the peak knee flexion in swing decreases with diminution of velocity in normally developing subjects (van der Linden et al 2002) and so the question has to be asked as to how much of the decrease in peak knee flexion is due to diminished velocity and how much to primary pathology relating to the rectus femoris (Goldberg et al 2005). Activity of the rectus on electromyography has been shown to be influenced by gait velocity in normally developing subjects; with increased velocity there is increased activity of rectus and decreased velocity leads to decreased rectus activity (Annaswamy et al 1999) (den Otter et al 2004, Nene et al 2004, Nene et al 1999). With speeds less

than 0.28m/s, there has been detection of additional rectus activity occurring in late swing and this was attributed to the swing leg having a reduction in its pendulum action and the extra rectus activity may be a compensation to counter this by increasing hip flexion (den Otter et al 2004). The relevance of this to children with spastic diplegia, who walk at such clinically slow speeds, needs to be considered as increased rectus activity in late swing may not be entirely pathological.

There is inconsistency in the results obtained from rectus transfer (Asakawa et al 2002, Goldberg et al 2003, Riewald and Delp 1997). Why this is so remains uncertain. The use of computer modelling and simulation has been employed to help investigate the effect of rectus transfer on peak knee flexion in swing phase (Asakawa et al 2002, Delp et al 1994, Goldberg et al 2003, Piazza and Delp 1996, Riewald and Delp 1997). It had been presumed that the transfer of the rectus to a site posterior to the knee joint axis would lead to the rectus developing a flexor moment at the knee in swing. However computer modelling has shown that despite the transfer sites providing knee flexor moment arms (the greatest moment arm being that of the semitendinosus) (Delp et al 1994), the knee continues to develop a knee extensor moment in the swing phase of gait (Riewald and Delp 1997). Post-transfer, the capacity of the rectus to provide knee extension and hence a knee extensor moment has been shown to be lessened, and this may contribute to improvement, as following distal transfer of the rectus, the muscle maintains its ability to generate a hip flexion moment, which in turn promotes knee flexion (Asakawa et al 2002). This has been supported by research that has shown that post-transfer, the rectus femoris muscle-tendon path is angulated and there is scar tissue at the site of angular deviation (Asakawa et al 2004). Post-tendon transfer, the presence of both the angulated path and scar tissue are thought to contribute to the result that the muscle is not able to produce a knee flexion moment as anticipated (Asakawa et al 2004).

The knee flexion velocity at toe off of subjects walking with a stiff knee gait, has been found to be reduced (Goldberg et al 2005, Goldberg et al 2003). With computer simulation, when the knee flexion velocity at toe off was increased to

within normal range, then peak knee flexion likewise was found to increase to within normal range (Goldberg et al 2003). Again it seems that velocity has a role in determining the peak knee flexion obtained, whether by decreased gait velocity (van der Linden et al 2002), decreased knee flexion velocity at toe off (Granata et al 2000, Piazza and Delp 1996), increased hip flexion velocity at toe off (Piazza and Delp 1996) or changes in angular acceleration of the shank (Nene et al 1999). An increase in the knee extensor moment or a decrease in the hip flexor moment prior to and at initial swing also led to a decrease in the peak knee flexion according to one study (Piazza and Delp 1996). Poor outcome post-rectus femoris transfer has been associated with the continued production of an increased knee extensor moment in double support prior to toe off (Goldberg et al 2005). One of the documented prerequisites for rectus femoris transfer is that " adequate acceleration forces must be present to flex the hip and propel the limb through swing" (Chung et al 1997, p. 138). The largest contributor to peak knee flexion in swing phase in normal gait is the velocity of the knee at the time of toe off, and whilst individual muscle contributions may be large in the swing limb, they tend to cancel the effect of each other, and their overall influence on the peak knee flexion is small in amplitude (Anderson et al 2004). The muscles that have an impact on increasing knee angular velocity prior to toe off are the iliopsoas and gastrocnemius in double support, whilst the vasti, rectus and soleus can decrease this velocity (Goldberg et al 2004). It is now thought that the transferred rectus femoris does not become an active knee flexor in swing phase, but however following transfer it has a diminished knee extensor moment and at the same time is able to maintain its capacity to generate a hip flexor moment (Anderson et al 2004, Asakawa et al 2004). In addition by transferring the rectus femoris, its ability to decrease the angular velocity of the knee is diminished (Goldberg et al 2004) and this leads to improvements in a stiff knee gait. The slope of the knee flexion curve has been shown to improve following multiple soft tissue releases that included hamstring lengthenings and distal rectus transfers. However the slope was also influenced by the number of prior orthopaedic surgeries (Hadley et al 1992). The impact of surgery in terms of contributing to a stiff knee gait needs to be considered when planning interventions. If the iliopsoas and hamstrings are targeted for lengthening, knee flexion velocity in double support may possibly

be affected, as in a simulation study both muscles have shown that they have the potential to increase knee flexion angular velocity in double support (Goldberg et al 2004). Although velocity of knee flexion at toe off was not measured in our study, the rate of change from maximum knee extension to peak knee flexion was calculated, and this showed an increase post-SEMLS, although not to within normal values.

In crouch gait, the stance limb in the single support period is precariously held in some degree of increased flexion at all joints, whilst the contralateral limb attempts swing phase. The instability of the flexed limb in the single support period probably determines the decreased duration of the single support period and consequently the short time spent in swing for the contralateral limb (Sutherland et al 1988). Is it possible that the contralateral swing limb has a diminished peak knee flexion in order to minimise the height of clearance of the foot in swing so that stability of the stance limb is less threatened and if the stance limb is unable to maintain stability, the swing limb is 'ready' to begin double support and assist stability? The lack of foot clearance from decreased peak knee flexion in swing has always been presumed to be a consequence of rectus spasticity although velocity seems to be implicated in this reduction, as discussed above, and perhaps also balance, which is neurologically controlled and a confounder in the decrease of peak knee flexion in swing phase. It is usually the more severely involved child who has more balance problems and it has been reported that the more involved children do not benefit from rectus transfer as well as the children who are less involved (Goldberg et al 2005, Miller et al 1997, Yngve et al 2002).

The use of the Duncan Ely test to indicate the presence or otherwise of rectus spasticity that would interfere with knee parameters in swing phase in the sagittal plane has been shown to be useful if the test records positive, as it is likely that decreased and delayed peak knee flexion in swing on kinematic data will be observed along with abnormal activity of the rectus on electromyography (Marks et al 2003). A negative Duncan Ely test does not necessarily imply an absence of rectus abnormality but this finding by Marks et al (2003) was limited by the lack of diversity in the results of the subjects in the

study as most had a positive test. Electromyography has shown however, that the Duncan Ely test is not specific for the rectus femoris, as iliopsoas is also activated during the test (Perry et al 1976). All Duncan Ely tests pre- and post-SEMLS were positive in this study, and interestingly the peak knee flexion was within normal range but delayed in timing pre-surgery. Post-surgery, peak knee flexion was decreased but the timing was earlier. Electromyography data were not collected so the extent of rectus activity could not be ascertained.

There is some consistency amongst authors as to the indications for rectus transfer but adherence to these criteria does not guarantee an improved peak or timing of peak knee flexion in swing post-transfer. Cited criteria for rectus transfer are: decreased maximum knee flexion in swing phase (Chambers et al 1998, Kay et al 2004a, Moreau et al 2005, Sutherland et al 1990), decreased dynamic knee motion (Chambers et al 1998, Hadley et al 1992, Moreau et al 2005, Sutherland et al 1990), delayed peak knee flexion in swing phase (Chambers et al 1998, Kay et al 2004a, Moreau et al 2005, Sutherland et al 1990), impaired foot clearance (Miller et al 1997, Saw et al 2003, Sutherland et al 1990), prolonged or inappropriate activity of the rectus in swing phase on EMG (Chung et al 1997, Gage 1990, Hadley et al 1992, Kay et al 2004a, Moreau et al 2005, Saw et al 2003, Sutherland et al 1990), recording of a positive Duncan Ely test (Chung et al 1997, Sutherland et al 1990), reduction of sagittal plane knee motion of twenty percent or more (Chung et al 1997, Gage 1990), peak swing phase knee flexion less than 50° (Miller et al 1997), peak knee flexion delayed greater than thirty percent of swing phase (Miller et al 1997), EMG activity of rectus in the middle three fifths of swing (Miller et al 1997). Pre-operatively in our study, the severe crouch cohort displayed the following indications for rectus transfer: decreased dynamic knee motion, delayed peak knee flexion in swing phase, and a positive Duncan Ely test.

The incidence of rectus femoris transfers at the time of SEMLS in the severe crouch group should have been higher and this may have averted the postoperative decrease in peak knee flexion in swing that was found. The results from the severe crouch group are similar to those of a study that reported limited incidence of rectus transfers (4%) with a higher incidence of hamstring lengthenings (47%) (Wren et al 2005a). That particular study revealed an increase in odds (an almost four-fold increase) of exhibiting a stiff knee gait and concurrent rectus femoris transfer at the time of hamstrings lengthening was proposed (Wren et al 2005a).

## 7.4.4 Crouch Gait: Resistant to SEMLS

There were a number of subjects in this cohort for whom the results post-SEMLS were not optimal, as they continued to walk in some degree of knee flexion. At five years post-SEMLS, four subjects continued to have knee fixed flexion deformity of more than 10°. On barefoot sagittal knee kinematics, eight subjects had a minimum knee flexion in stance of more than 15°. These subjects may have been better served by distal femoral extension osteotomy (Gage 2004f, Gage et al 2000, Stout et al 2004) to improve the fixed flexion deformity at the knee and a patellar tendon shortening (Beals 2001, Gage 2004f, Gage et al 2000, Normand and Dubousset 1985, Stout et al 2004) to help regain active extension of the knee in stance phase.

There is recognition that the patellar tendon is elongated when patella alta is present and whilst patella alta is associated with pain that leads to impaired function of gait, elongation of the patellar tendon results in reduced active knee extension in the available range of passive movement at the knee: i.e. a quadriceps lag. There may or may not be a fixed flexion deformity at the knee present in such cases. Surgery to gain this range of active knee extension has been performed by many researchers in the past (Baker 1956, Beals 2001, Bosworth and Thompson 1946, Chandler 1933, Cleveland and Bosworth 1936, Gage 2004f, Gage et al 2000, Keats and Kambin 1962, Normand and Dubousset 1985, Roberts and Adams 1953, Stout et al 2004, Zimmerman et al 1982).

Patellar tendon shortening combined with hamstrings lengthening or hamstring transplantation to the femoral condyles, has been undertaken for knee flexion contracture (Keats and Kambin 1962). The shortening of the patellar tendon in this case was achieved by releasing it from its insertion and fixing it distally with an "osteo-periosteal flap" (Keats and Kambin 1962, p. 1147) and screw on the tibial shaft. Good functional results were recorded for the group having this

procedure compared to the groups having only hamstring lengthenings or combined hamstring lengthenings and transplantations (Keats and Kambin 1962). Variations of this technique for patellar advancement have been utilised in the past, and in conjunction with other orthopaedic surgery (Bosworth and Thompson 1946, Chandler 1933, Cleveland and Bosworth 1936, Roberts and Adams 1953).

Hamstrings lengthening and posterior knee capsulotomy have been used to help regain passive knee extension, combined with patellar tendon shortening (Beals 2001). Two types of shortening technique were used. For the skeletally immature, the shortening was achieved by removing the infrapatellar tendon from its attachment to the distal pole of the patellar and then reattaching it there after shortening. If the child was skeletally mature, distal transfer of the infrapatellar tendon and its attached bone block achieved the shortening. This latter technique had some problems with fixation being lost and this was a caution raised by the author (Beals 2001). This surgery was not carried out in the context of SEMLS but the previous surgical history was not tabled except for the incidence of prior calf lengthenings. Follow-up was between 3 to 16 years.

Hamstrings lengthening combined with tenotomy of the rectus, and then patellar tendon shortening have also been used (Normand and Dubousset 1985). The patellar tendon shortening was again achieved by two different techniques. One method was to advance the tibial tubercle distally along the tibia, and the other involved plication of the infrapatellar tendon and stabilisation of this by a metal cerclage that encompassed the superior pole of the patella and onto the tibia. Functional results indicating ability to walk without or with different level of assistive devices were impressive over a follow-up period of four years. The reason for the rectus femoris tenotomy seemed to have been to achieve an enhanced lengthening of the infrapatellar tendon. Again past surgical history was not mentioned and so the surgery cannot be reviewed in that context.

Distal extension osteotomies of the femur have been undertaken in the past on patients with knee flexion deformity that had been resistant to other forms of

301

surgery (Asirvatham et al 1993, Osgood 1913, Zimmerman et al 1982). These patients had diagnoses of polio (Asirvatham et al 1993, Osgood 1913, Zimmerman et al 1982) or myelomeningocele (Zimmerman et al 1982) and relapse of the deformity was recorded when bracing was discarded and ambulation curtailed (Zimmerman et al 1982). Particular emphasis was placed on the removal of the wedge of bone in order to prevent the vessels and nerves being placed under undue tension when full knee extension was obtained (Osgood 1913).

More recently, distal extension osteotomies of the femur have been used to correct the persistent knee flexion in crouch gait, which was resistive to correction despite prior SEMLS, which had included hamstring lengthenings and rectus femoris transfers. It was found that if the extension osteotomy was undertaken without a patellar tendon shortening procedure, the range of passive knee extension improved but crouch gait was not improved (Gage 2004f, Gage et al 2000, Stout et al 2004). The patellar tendon shortening was achieved by tibial tubercle advancement and only done in skeletally mature subjects. When combined with patellar tendon shortening, not only did the passive knee extension improve but sagittal knee kinematics did also. However, despite this improvement, there was deterioration at the pelvic and hip level in the form of an increase in anterior pelvic tilt and decrease in maximum hip extension in stance. This decline has been assumed to be due to psoas contracture and intramuscular lengthening of psoas has been undertaken to remedy this problem (Gage 2004f). Whether this operation has been successful in alleviating the increase in anterior pelvic tilt has not been reported as yet. The realignment of the femoral shaft by the extension osteotomy would automatically bring the pelvis forward unless there was provision to increase hip extension range to prevent this and whether psoas lengthening can adequately address this will be interesting (see Figure 7.26). Weakness may contribute to the problem. In the past a rectus femoris release has been recommended to be undertaken concurrently with patellar tendon advancement distally to "avoid accentuation of hip flexion and restriction of knee flexion" (Evans 1975a, p. 189). This implies that the anterior pelvic tilt may be caused not by psoas but by the rectus femoris becoming taut as a consequence of the patellar tendon advancement

procedure. Normand and Dubousset (1985) incorporated the rectus femoris release in their patellar tendon shortening surgery but pelvic and hip measurement pre- and post- the procedure were not reported. So whether incorporation of a release of the rectus at the time of patellar tendon advancement avoids deterioration in hip and pelvic parameters cannot be assessed at this stage.

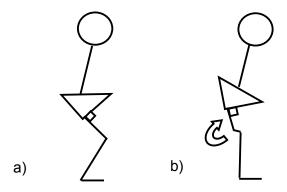


Figure 7.26 Subject in a) crouch and b) subject post-distal femoral extension osteotomy with the hip/ pelvis unaltered in alignment to the femur. It can be seen that unless the hip is extended, the pelvis is tilted more anteriorly post-operatively.

In the skeletally immature child with fixed flexion deformity at the knee, anterior femoral stapling has been used to gradually correct the deformity in preference to a distal femoral extension osteotomy (Kramer and Stevens 2001). Medial physeal stapling of the femur has been used successfully in idiopathic genu valgum with few complications (Stevens et al 2004, Stevens et al 1999). Results from the anterior femoral stapling showed elimination or a reduction in the knee deformity on physical examination for children with cerebral palsy (Kramer and Stevens 2001). It was recommended that this surgery was applicable if the distal femoral physis was open and more than 12 months of growth was predicted to be left (Kramer and Stevens 2001). Staples remained in situ until skeletal maturity was reached or the deformity had been corrected (Kramer and Stevens 2001).

Severe crouch gait may be better managed by a combination of soft tissue lengthenings and bony surgery that incorporates patellar tendon shortening/

303

advancement and distal femoral extension osteotomies if the child is skeletally mature, or anterior femoral stapling if there is skeletal immaturity. Whether the eight subjects who had greater than 15° of minimum knee flexion in stance at five years post-SEMLS would have had a better outcome if the abovementioned types of surgery had been undertaken is unknown, however it is worth considering that inclusion of such surgery may have been advantageous.

## 7.4.5 Statistical Analysis Considerations

The statistical analysis of the data from the 3DGA in this study of severe crouch gait and surgical intervention has been undertaken using univariate analysis. Such analysis has been questioned because of its "specificity and interdependence" (Novacheck and Schwartz 2004, p. 408) and the use of multivariate analysis has been recommended (Novacheck and Schwartz 2004, Schutte et al 2000, Schwartz et al 2000, Schwartz et al 2004). The univariate analysis examines a specific point during the gait cycle at a particular level and in a particular plane. By examining parameters this way, there is criticism that an overview of the data as a whole is missing as parameters are considered in isolation. Concern regarding the interdependence between parameters is valid. If the pelvis is tilted anteriorly in the sagittal plane then the hip will record some decrease in hip extension as the orientation of the hip is reported with respect to the pelvic position. Due to the body segments in the lower limb being interconnected, if there is an increase in knee extension in midstance and the foot remains in contact with the ground at this point in the gait cycle, the tibia must change alignment with the foot as it will be more vertical and so dorsiflexion will decrease in conjunction with an increase in knee extension. A relationship between a kinematic parameter and a temporal-spatial parameter can also exist. If a subject walks more slowly at one time period to another, this can change the kinematic data (see Section 7.4.3 Knee Parameters in the Sagittal Plane) (van der Linden et al 2002)).

Multivariate analysis, if undertaken as a principal component analysis, is able to uncouple parameters so that they are no longer correlated/ interdependent to each other (Novacheck and Schwartz 2004). The Normalcy Index (Schutte et al 2000) and the Hip Flexor Index (Schwartz et al 2000) are examples of principal component analysis. Both indices comprise parameters from temporal-spatial, kinematic and kinetic data that, using principal component analysis, are no longer correlated to each other. The single number that is derived from these data indicates how much a gait pattern deviates from that of a normally developing subject. The indices may constitute an objective number, but the identification of parameters for inclusion in the formulation of each index is subjective, though made on clinical grounds (Schutte et al 2000). However who is to say that these parameters, that currently comprise the Normalcy Index are the most appropriate for depicting the outcome of gait (Schutte et al 2000)?

In this chapter, although parameters were analysed separately when reviewed, the intent was to group results in a clinically coherent manner to give an overview of the data as a whole. The interdependence of parameters chosen for analysis was recognised and considered in the discussion and the implications for clinical decision making. The Normalcy Index could have been used as an outcome measure to indicate improvement or deterioration in gait parameters post-SEMLS. However the Normalcy Index does not indicate which gait parameters have shown change and if there was a change, whether the change represented an improvement or deterioration. Without this information, an intervention such as SEMLS cannot be appraised at a clinically meaningful level. The univariate analysis for the severe crouch group post-SEMLS, has shown that SEMLS has been effective in improving knee and ankle alignment but the hip and pelvis have shown deterioration. Hence efforts to identify more appropriate surgery to address the hip and pelvis posture in severe crouch gait must now be made. The Normalcy Index on its own would not have yielded this information. This is important information that will alter future decision making in the surgical prescription for severe crouch gait. Univariate analysis permits derivation of appropriate clinical information, which can be applied in the hospital or clinic setting for the betterment of the children who seek care.

## 7.4.6 Limitations

The same limitations apply in this study as detailed in Chapter 6, Section 6.4.4. The number of participants decreased to 12 subjects at one and five years post-

SEMLS. Kinetic data were also limited at the one year time period and the issues relating to this were discussed earlier in Section 7.3.3.

Strength testing would have provided insight into the effects of SEMLS on muscle strength. Where deficiencies exist then strength training programmes could target weak muscles during the rehabilitation period post-SEMLS. Elimination or reduction of knee pain would have provided further clinical evidence of the efficacy of the SEMLS as a practical intervention for children with severe crouch gait. There was no evaluation of participation for the subjects according to the ICF. The inclusion of a quality of life questionnaire would have added this perspective and helped assess more fully the impact of SEMLS on the cohort.

Each subject had SEMLS for correction of severe crouch gait but the number and type of procedures that constituted the SEMLS was tailored to each subject. Hence SEMLS was not the same for all the subjects. Also, on occasion the SEMLS prescription was altered by parental choice. There was a common treatment philosophy of lengthening the short muscles, correcting bony malalignment and providing support to long muscles. As a consequence there was a dominance of hamstrings surgery, variation in frequency of bony surgery and other soft tissue lengthenings and instigation of GRAFO support. Therefore the exact surgery cannot be replicated in another study. Moreover the isolated effects of individual surgeries on crouch gait cannot be ascertained as they are confounded by the effect of the other surgeries that were performed at that time, and in some cases, years prior to SEMLS. This is a common problem in research today in spastic diplegia. The effects of individual surgeries can only be surmised from the resulting biomechanics at the anatomical level that the individual surgery should have altered. An example of this would be that of hamstrings lengthening, which should improve extension at the knee but because their function is biarticular, an effect can also occur at the hip and pelvis, such as decreased hip extension and an increase in anterior pelvic tilt. To only perform one procedure at operation would take paediatric orthopaedic surgery back to the times of the "birthday party syndrome" (Rang et al 1986, p. 365) which was not deemed physically, emotionally or financially cost effective

for all concerned (Gage 1993, Patrick et al 2001). This is a quandary, as new technology has allowed objective assessment of outcome of surgical intervention, which allowed the implementation of SEMLS in the process. However this type of surgery now prevents assessment of the outcome of individual surgeries. It is also important to recognise that although the biomechanics of a gait pattern may be the same for two subjects, the causes that lead to this gait pattern may be different. For instance, a pelvic tilt may be increased anteriorly due to weak hamstrings, dynamic or static contracture in psoas, gluteus maximus weakness or a combination of any of these. Depending on the assessment of the causative factor of the increased anterior pelvic tilt, intervention will vary from hamstring and/ or gluteus maximus strengthening, botulinum toxin-A injection to psoas, or psoas lengthening. As no two subjects are exactly the same despite having the same gait pattern, it is the general principles of the surgical intervention that are assessed in this study and the effects of the individual procedures surmised from the outcome.

There were no control subjects with spastic diplegia in this study as the study was retrospective and no comparable group was available as patients referred to the gait laboratory for analysis, were referred because of their deterioration in gait and therefore for planning of surgical intervention. Unlike the study by Gough et al (2004) none of these patients declined to undergo SEMLS or had their SEMLS delayed for other reasons. Hence there was no natural comparative group for the study. In the severe crouch cohort, to have delayed surgery in the face of increasing crouch, decreasing function and knee pain would have been unethical and there were too few subjects to randomise.

If this had been a prospective study, it would have been possible to randomise subjects to intervention now or intervention postponed. Withholding intervention over a five year period would not have been acceptable to the children, families or staff if the child was showing a deterioration in walking ability, which would have been the reason for referral in the first place. In the longitudinal studies previously mentioned (Bell et al 2002, Gough et al 2004, Johnson et al 1997, Wren et al 2005a, Yokochi 2001), it has been established that the natural history of gait is not static, and does not improve but deteriorates

with time. Therefore it can be inferred from these studies that a control group of subjects with severe crouch gait would have shown deterioration over the five years period without SEMLS intervention.

## 7.5 SUMMARY

A very astute senior paediatric orthopaedic surgeon (Mr P Williams, RCH, Melbourne) has been known to ask whether the child is "falling down or being pulled down" i.e. falling down due to muscle weakness, or being pulled down due to spasticity or a combination of both. There are so many possibilities. Regaining an upright, straight posture to correct crouch gait is the goal of surgery and an effective plantarflexion knee extension couple must be restored to achieve this (Gage 2004f). Intervention needs to address lever arm dysfunction in the three planes. In the sagittal plane, muscles may need to be lengthened (hamstrings and/ or psoas) or protected (soleus). In the coronal plane, hip pathology must be corrected if required. In the transverse plane, bony surgery may be required to correct alignment of the femur and/ or tibia; and/ or correct and stabilise the foot. The function of the muscle to be lengthened needs to be taken into account before intervention is undertaken. If the hamstrings are lengthened, the risk of recurvatum at the knee and increased anterior pelvic tilt must be considered. Concurrent lengthening of iliopsoas with the hamstrings may be thought to be appropriate, but if they are weakened, the knee flexion angular velocity required for knee flexion in swing may be decreased (Goldberg et al 2004) and inadequate limb clearance in swing a consequence.

In this study of SEMLS in the severe crouch cohort, the extent of hamstrings lengthening perhaps should have been confined to the medial hamstrings with associated psoas lengthening to protect against an increase in anterior pelvic tilt. Or alternatively medial hamstrings lengthening with transfer of the semitendinosus to the distal femur may be a superior option in decreasing knee flexion but safeguarding the pelvic position. The incidence of rectus femoris transfers to maintain the peak knee flexion in swing could have been higher. The rectus femoris transfer should decrease the knee extensor velocity in double support. This would have assisted in negating the effect of the lengthening of the hamstrings and psoas that would have supposedly decreased their potential to increase knee flexion velocity in double support, and predisposed the peak knee flexion in swing to being decreased. If the semitendinosus transfer to the distal femur is used as part of the hamstrings lengthening procedure, then the rectus could be transferred to the gracilis. The use of GRAFOs or solid AFOs in the first year post-SEMLS appears to have been successful in supporting and promoting re-tensioning of the long soleus muscle and should continue to be part of the rehabilitation programme. The derotation osteotomies to the femur and/ or tibia and the foot stabilisation surgeries seem to have been appropriate in regaining lever arm alignment in this plane.

The improvement in the knee and ankle parameters, and the stability of the hip parameters are positive outcomes, as longitudinal studies have shown that the natural history of gait in cerebral palsy is for deterioration with time (Bell et al 2002, Gough et al 2004, Johnson et al 1997). The increase in the anterior pelvic tilt post-SEMLS is of concern as back pain has been consistently documented in a number of studies on the functional status of adults with cerebral palsy (Andersson and Mattsson 2001, Jahnsen et al 2004a, Murphy et al 1995, Sandström et al 2004, Schwartz et al 1999). In the studies on adults, the focus has been on health (Cathels and Reddihough 1993, Murphy et al 1995), functional level (Ando and Ueda 2000, Sandström et al 2004), ambulatory status (Andersson and Mattsson 2001, Bottos et al 2001, Jahnsen et al 2004b), pain (Jahnsen et al 2004a, Schwartz et al 1999) and fatigue (Jahnsen et al 2003) without reference to previous surgical intervention which may impact on the reported findings.

Walking ability in adults has been shown to deteriorate from the level in childhood or adolescence (Andersson and Mattsson 2001, Ando and Ueda 2000, Bottos et al 2001, Jahnsen et al 2004b, Murphy et al 1995, Sandström et al 2004). Cessation of walking has been associated with fatigue, inefficiency and the realisation that wheelchair use can lead to greater accessibility of activities in young adults. With adults in their mid forties, the presence of joint pain is a factor in cessation of walking (Murphy et al 1995). Joint pain has been

associated with decreased range of movement in the particular joints (Jahnsen et al 2004a, Sandström et al 2004) and the pain is often present in more than one joint (Andersson and Mattsson 2001, Jahnsen et al 2004a, Murphy et al 1995, Sandström et al 2004). Clinical examination has found arthritis in the joints of subjects aged between 15 and 25 years, and the use of non steroidal anti-inflammatory medication to relieve the joint pain has been associated with the finding of gastric ulcers in a number of these subjects (Cathels and Reddihough 1993). Such findings do not propose an optimistic outlook for the adult with cerebral palsy and highlight just how important the positive results are from the SEMLS intervention for severe crouch gait, particularly lasting over a five year period.

The improvements from the SEMLS intervention may help change the outlook for these subjects in their future as adults because biomechanical alignment has been improved. It has been postulated that the mild to moderately impaired adult with cerebral palsy has spent his life using a combination of movements that are dictated by the biomechanics resulting from the neurological lesion and those of the necessary compensatory movements to achieve walking (Bottos et al 2001). It is these movement patterns that lead to joint degeneration, fatigue and pain and an eventual deterioration or loss of walking in later life (Bottos et al 2001). The SEMLS in the severe crouch group has led to an improvement in the biomechanics imposed by the neurological lesion, and reduced the need for compensatory motion as shown by the results of this study. The resultant improved biomechanics hopefully will result in a positive alteration in the natural history of gait for these subjects as adults with cerebral palsy, that will be more optimistic than that previously reported.

## 7.6 CONCLUSIONS

This group of subjects walking in severe crouch gait comprised a homogeneous group defined in the inclusion criteria by a precise biomechanical definition of severe crouch gait. SEMLS for severe crouch gait in spastic diplegia resulted in

consistently marked improvements in dynamic knee and ankle function but not at the hip and pelvic levels.

To improve the results at the pelvis and hip, the SEMLS programme in the future needs to reconsider the incidence and extent of hamstrings lengthening by predominantly lengthening the medial and not the lateral hamstring muscles. Consideration should be given to transfer of the semitendinosus to the distal femur, and not repeating hamstrings surgery in those subjects who have had prior hamstrings surgery. The frequency of psoas lengthening could be increased, as well as rectus femoris transfers. A rehabilitation programme that also targets strengthening of muscles, in particular the abdominals and gluteus medius may, in conjunction with the psoas lengthenings, avoid the increase in anterior pelvic tilt found in this severe crouch group post-SEMLS. Continuation of the transverse plane surgery is proposed as there was only a mild relapse in the femoral alignment in the transverse plane at five years. The wearing of GRAFOs or solid AFOs in the first year post-SEMLS is recommended as the ankle kinematics showed improvement on barefoot walking at both time periods post-SEMLS.

When severe crouch gait has been resistive to SEMLS correction, consideration should be given to distal femoral extension osteotomies and patellar tendon shortening to remediate this situation, as published results of these surgeries in such instances are promising (Beals 2001, Gage 2004f, Gage et al 2000, Normand and Dubousset 1985, Stout et al 2004).

# CHAPTER 8 CONCLUSIONS AND CLINICAL IMPLICATIONS

## **8.1 CONTRIBUTION OF THESIS**

This thesis has identified a new classification of sagittal gait patterns in spastic diplegia that is inclusive of all sagittal gait in the cohort studied. This is the first classification of gait patterns that has been identified, which does not comprise a collection of gait patterns that are discrete entities. It is a classification of gait patterns that reveals a continuum with respect to progression with age, and in terms of biomechanics and involvement of anatomical levels. This is a unique feature of this classification of gait patterns and enhances its utility in the clinical and research settings.

Because the gait patterns show a progressive change in biomechanics and anatomical involvement, interventions need to be tailored to deal with these differences between the gait patterns so that the correct anatomical level(s) is/ are targeted. Consequently this classification has an application to clinical teaching, which is consistent with rationale for the early labelling of gait patterns (Bleck 1971, Miller et al 1995, Rang et al 1986). However our classification is the first to show this continuum of change in terms of biomechanics and anatomical level.

Even more importantly, these gait patterns have a role in research into gait pathology and its management. The differences between the identified gait patterns reinforce the need for research that explores the differences in intervention that are required for each gait pattern to be managed for optimal outcome. The benefits of utilising a classification of gait patterns in spastic diplegia for research into management have not been documented prior to this study.

This classification of gait patterns has provided the basis for the robust definition that was used as the inclusion criterion for the subjects in the studies on severe crouch gait.

This is also the first study of severe crouch gait that has inclusion criteria specifying quantitatively the degree of crouch at the knee, ankle, hip and pelvis in stance using data from 3DGA. Therefore it is feasible that this study can be reproduced as the criteria for subject inclusion has been specific. The extent of gait deviation in severe crouch gait has been quantified through functional mobility levels, kinematic and kinetic data, with adjunct information provided from the physical examination. This has established a comprehensive baseline for severe crouch gait prior to the SEMLS intervention.

The outcome of SEMLS on severe crouch gait has not been documented previously. Consequently this study is the first to confirm that improvement post-SEMLS at one year can be maintained in the long term at five years post-intervention. The study demonstrated that the knee and ankle in severe crouch gait showed a favourable outcome post-SEMLS but the hip was unchanged and the pelvis actually showed deterioration. This insight into the outcome of SEMLS at the different anatomical levels prompts further investigation into the surgical procedures that may have influenced the outcome at the hip and pelvis. The high incidence of hamstrings lengthening and limited psoas lengthening as part of the SEMLS intervention may need to be revised in the future management of severe crouch gait. This research has demonstrated the important link between systematic research and dynamic improvements in clinical practice.

## **8.2 LIMITATIONS**

## 8.2.1 Sagittal Gait Patterns in Spastic Diplegia

Although not population based, this study on sagittal gait patterns in spastic diplegia does represent the children who present with gait problems in the clinical setting. The absence of stringent quantitative boundaries between

postures at each anatomical level does not prevent discrimination between patterns. This is because the overall pattern is dependent on discrimination between postures at a minimum of one anatomical level. This method of defining gait patterns allows for the biomechanical continuum between patterns to be appreciated. The inclusion of subjects with prior calf lengthening surgery is a confounder to the natural history of the progress of gait patterns. As calf surgery is no longer a prevalent surgery in children now presenting to clinics, the study could be repeated without this inclusion criterion, in order to ascertain its impact on gait patterns in the cross-sectional study and progression of gait patterns in the longitudinal study. Because this was a retrospective study, the available data were confined to that collected at the time of presentation of the child and this did limit investigation into the risk factors that may lead to progression between patterns.

## 8.2.2 Severe Crouch Gait

The number of subjects in the severe crouch cohort was small and this may predispose to a type II statistical error due to lack of statistical power (Portney and Watkins 1993). If the sample size is small then the statistical power is diminished, as a small sample may not necessarily be a good representation of the population in question (Portney and Watkins 1993). However obtaining large numbers of subjects for study in the area of cerebral palsy is not easy. As noted previously, the rate of cerebral palsy is around 2 to 2.5 cases per 1000 live births (Stanley et al 2000) and so obtaining large numbers of subjects with a particular type of neurological motor impairment, distribution of involvement, and gait pattern in a particular clinical setting, can be difficult. This is a constant problem that needs to be addressed, however until there is consensus worldwide on classification by type of neurological motor impairment, distribution of involvement and gait pattern (if able to walk), then the possibility of multi-centre trials with a large number of subjects in a study will be hindered. Even if the above concerns were resolved, then the issues of consensus in management between centres would need to be addressed, as different treatment philosophies often exist between centres. Therefore to obtain large numbers of subjects in a severe crouch cohort is currently difficult.

As a retrospective study without a control group of subjects, this study is not as favourable as one that has prospective randomisation and a control group, as the level of evidence provided by the results will not be regarded as highly. The difficulty of obtaining a control group has already been discussed in Chapter 7, Section 7.4.6. However a prospective study would have provided the opportunity to ensure that all relevant data were collected at all time points.

This study does provide a measure of outcome post-SEMLS at one and five years. It is recognised that the outcome may be due to factors other than the SEMLS intervention (Herbert et al 2005) such as co-operation with, accessibility and intensity of the rehabilitation programme, extent of SEMLS (e.g. six versus 12 surgeries), and expertise of the surgical teams and physiotherapist providing rehabilitation. So the outcome is not wholly dependent on the effect of the surgical intervention. The measured outcome does have value as it demonstrated that the overall programme of SEMLS, including for each subject a rehabilitation programme that was accessible and regularly monitored, and the same surgical team ensuring consistency of treatment philosophy, showed a positive outcome. Some of the variables that could not be controlled were personal motivation and co-operation, support of family and school, and expertise of community physiotherapist. Any of these factors could have altered outcomes for the individual and hence the study group (Stanley et al 2000).

## **8.3 FUTURE DIRECTIONS**

## 8.3.1 Sagittal gait patterns in spastic diplegia

This classification of sagittal gait patterns has clinical and research application and this should be promoted so that the classification is integrated into everyday practice for all who work in the field of cerebral palsy. If sagittal gait patterns are to be adopted clinically and in research then they need to be promoted. A DVD or pamphlet, or preferably both are to be developed to promote its use.

The use of the Normalcy Index to explore whether it can provide numerical boundaries between the sagittal gait patterns may be one method of providing further quantification to the classification. The index has been used to ascertain differences between the four subtypes in hemiplegia (Schutte et al 2000) with the mean for each subtype showing increases from Type I hemiplegia to Type IV hemiplegia, but the range of values was large for all subtypes and there was considerable overlap. A wide range of values for the Normalcy Index in spastic diplegia has also been documented (Romei et al 2004, Schutte et al 2000), and if assistive devices were used the range increased even further (Romei et al 2004). Whether this index would be helpful in differentiating numerically between the sagittal gait patterns in spastic diplegia can only be determined by implementing such a study.

The longitudinal study showed changes in gait patterns over time. It is proposed that a longitudinal study that documents the gait patterns of children with spastic diplegia every two years from age four years until SEMLS, be undertaken. This would provide a more detailed documentation (due to more observation times) of how gait patterns change with time. The change in gait patterns may be associated with changes in rating according to the FAQ or FMS. The inclusion of certain physical examination measures at each time period would also add to the knowledge gained from such a study as already stated in Section 8.2.1.

This new classification in spastic diplegia does not incorporate the transverse plane. Incorporating the transverse plane with the current sagittal plane classification of gait would allow any association between lever arm dysfunction in the transverse plane and progression to crouch gait to be explored. The hypothesis would be that lever arm dysfunction in the transverse plane would lead to progression in the sagittal gait patterns from true equinus through to crouch gait. Due to the potential inaccuracies in 3DGA in marker placement in defining the knee flexion/ extension axis and the consequent inaccuracies in femoral rotation in the transverse plane (discussed in Chapter 3, Section 3.3.7), classification of gait patterns in spastic diplegia that incorporates the transverse plane will be difficult. The resulting classification would be

confounded unless impeccable quality assurance of marker placement in respect to these potential errors was ensured.

Reliability has been established for video observation of gait patterns in the sagittal plane combined with sagittal plane kinematics from the pelvis to the ankle, in raters experienced in gait analysis and working in the field of cerebral palsy. It would be of benefit to establish the reliability of recognising the gait patterns with inexperienced raters and also when the data have been limited to video observation in the sagittal plane only. Both scenarios would be worth investigating, as the utility of the classification would be enhanced if reliability was found to be acceptable since these scenarios represent situations that arise clinically on a daily basis.

## 8.3.2 Severe crouch gait

The SEMLS in the severe crouch study was dominated by hamstring lengthenings, medial and often lateral, without concomitant psoas lengthenings. A study that assessed the outcome of SEMLS, comprising fewer hamstring lengthenings and when undertaken, was consistently accompanied by psoas lengthening, would provide better insight into the outcome on the pelvic position in severe crouch gait.

Severe crouch gait has now been studied. Comparison of severe crouch gait with data from subjects walking in crouch gait to a lesser degree could be investigated. Differences in data at baseline and outcome from SEMLS would provide insights for future modification of interventions that would only serve to improve the outcome further for children walking in crouch gait.

Studies that investigate baseline characteristics in each of the sagittal gait patterns and then outcome of intervention could also be instigated. Such studies would provide practical information that could be used in the clinical setting.

This cohort of subjects has a specific kinematic definition provided for severe crouch gait. Therefore the study of the muscle lengths in this cohort would be of interest, as it would provide a cohort similar in size to that of Hoffinger et al (1993) but with the advantage of inclusion criteria quantitatively defining the kinematic parameters of the crouch gait that is studied. Attention would need to be directed to the limitations of the computer simulation programme that would be utilised (Arnold and Delp 2004) so that responsible conclusions were made from the results. Such information would be helpful in the decision making process when individual surgeries are being considered for inclusion as part of a SEMLS programme.

It has been shown in recent studies that the abdominals have a role in preparing the trunk for upper and lower limb movement (Hodges and Richardson 1997, Urquhart et al 2005). Due to deficient neuromuscular control and/ or weakness, this role of the abdominal muscles is probably compromised in CP. How this deficiency impacts on the position of the trunk and gait is unknown. If there is a lack of stabilisation of the trunk prior to and during walking in cerebral palsy, then strengthening of the abdominals may improve this imbalance. With all the attention on Progressive Resistance Strength Training in cerebral palsy as a priority in treatment, it is surprising that the abdominal muscles have not been targeted along with gluteus maximus. A study that examined the role of the abdominals in preparation for lower limb movement in cerebral palsy might confirm the level of abdominal muscle activity. If the abdominals are found to be deficient, a Progressive Resistance Strength Training programme for the abdominals could be examined for its impact on the results of future surgery and compared with the results of this study.

# **8.4 CLINICAL IMPLICATIONS**

## 8.4.1 Sagittal gait patterns in spastic diplegia

This study of sagittal gait patterns in spastic diplegia has shown that gait patterns change with time. The exact mechanism for this change was not identified. However certain reasons were put forward. The integrity of the foot ankle complex was considered to be implicated in this change, for example, ligamentous laxity and a tight heel cord can lead to planovalgus and rotational malalignment of the foot. In contrast if there is no ligamentous laxity, the effect

will be to possibly force the ankle into further equinus. A planovalgus foot with malalignment may predispose a gait pattern towards that of crouch gait whereas an ankle in more equinus may predispose to true equinus or jump gait. The clinical implication would be that observation of the integrity of the foot posture might provide the first indication as to which way a gait pattern may progress.

This classification of sagittal gait patterns is important for clinical teaching, clinical intervention and future research. If this classification is adopted as a means to further categorise spastic diplegia, then this will lead to more detailed understanding of the diversity that is present in the gait of a child with spastic diplegia. This classification will aid the newcomer to the field of cerebral palsy to comprehend the differences in the gait patterns and therefore the differences in the principles of management that are instigated. The clinician will find that this classification can be used as a practical communication tool that will aid discussion of gait in spastic diplegia with colleagues. This classification will also provide a basis on which the clinician can begin to tailor intervention strategies in reference to the gait pattern demonstrated by the child with spastic diplegia.

The most important potential application of this classification of sagittal gait patterns is in research. Use of this classification in research on the outcome of specific interventions on gait in spastic diplegia will allow meaningful results to be documented. The classification of the sagittal gait patterns reveals the extent of the diversity of the patterns in respect to biomechanics and pathological anatomical involvement. If future research chooses to ignore this heterogeneity and study gait in spastic diplegia as an indistinct pattern then outcomes will remain nebulous. As a consequence of this, improved understanding of gait in spastic diplegia will be hindered and hence improvements in clinical management will be delayed in the future. This classification of sagittal plane gait patterns, through adoption as independent variables in research, will provide insight into which surgery will promote the best outcomes according to the particular gait pattern and this can only help advance our knowledge in the effective management of gait in spastic diplegia. The use of this classification for research would mean that obtaining substantial subject numbers would not be easy, and the implementation of studies involving collaboration between laboratories with similar patient demographics would be one solution to this problem (Patrick et al 2001). Prior to laboratories collaborating, reliability and repeatability of data collection methods would have to be documented and found to be high in order for a study to proceed. Such data would need to be included in publications resulting from such a study in order for the results to be viewed as meaningful.

## 8.4.2 Severe crouch gait

The importance of studying severe crouch gait could be questioned, as the incidence of crouch gait should be declining due to the body of knowledge that exists that implicates isolated calf surgery in the development of this gait pattern (Berghof et al 1997, Borton et al 2001, Dillin and Samilson 1983, Gage 1990, Rab 1992b, Sutherland and Cooper 1978). However crouch gait does occur as part of the natural progression of gait (Bell et al 2002, Gough et al 2004, Johnson et al 1997, Yokochi 2001) and so will not necessarily disappear clinically unless clinicians are able to prevent progression to this gait pattern.

Gage (2004c) has emphasised the importance of correcting lever arm dysfunction in all planes in crouch gait, which means that short muscles are lengthened, bony malalignment corrected, long muscles are supported. This study of subjects walking in severe crouch gait has shown that SEMLS can lead to long-term correction at the knee and ankle. The pelvis and hip remained resistant to improvement and the surgical composition that was used in the SEMLS in this study therefore needs to be altered in view of these findings. It is proposed that hamstring lengthenings should be restricted to that of the medial hamstrings and most often done in conjunction with psoas lengthenings in order to diminish the effect of the hamstrings surgery at the level of the pelvis. The incidence of rectus transfers within SEMLS for severe crouch gait should be increased, as peak knee flexion in swing phase decreased post-SEMLS and despite proximal rectus releases at one year post-SEMLS, this did not rectify this gait deviation. Transferring rectus femoris at the time of SEMLS

may be more beneficial as rehabilitation post-SEMLS is intense and may help capitalise on the effect of transferring rectus femoris.

This study has shown that improvement at the knee and ankle post-SEMLS in severe crouch gait can be maintained for five years and in this study, this time lapse coincided with a number of subjects reaching skeletal maturity as they were at the end of their adolescence. As discussed in Chapter 7 (Section 7.5) the literature pertaining to adult function, particularly walking, has not been promising with many adults ceasing to walk due to the high level of effort required and/ or pain (Andersson and Mattsson 2001, Ando and Ueda 2000, Bottos et al 2001, Jahnsen et al 2004b, Murphy et al 1995, Sandström et al 2004). The subjects of these previous studies were most likely recipients of multiple event single level surgery during their childhoods (prior to 1980). Advances in technology and research have meant that the children of the last decade have had the benefit of SEMLS and by the maintenance in improvements in the gait pattern observed in this study, hopefully the ability of these subjects to continue walking during their adult years may be viewed more optimistically. It is therefore important that children with spastic diplegia are given the chance to enter adulthood with the most optimal gait pattern for them. The optimal gait pattern is one that has favourable alignment in order to prevent or delay the onset of joint pain and arthritis, and requires the least possible energy expenditure so that as adults, they have the opportunity to participate in the everyday activities that they choose. Even maintenance of gait over time is a favourable outcome. It is proposed that continued evaluation of this cohort of severe crouch subjects as adults would provide valuable insight into the long term outcome (10 and 20 years) of SEMLS for severe crouch gait, and if there is gait deterioration, to determine at which time points this occurs. Use of quality of life questionnaires in such a study to document prospectively the impact of being an adult with spastic diplegia on gait and function, may be useful in campaigning for government policy to provide support services for these adults which currently tend to be fragmented or non existent (Bleck 1984, Cathels and Reddihough 1993, Murphy et al 1995).

## **8.5 CONCLUSION**

This new classification of sagittal gait patterns in spastic diplegia has the potential to advance knowledge in this area if applied clinically and in particular in the research arena.

The outcome of severe crouch gait post-SEMLS showed that SEMLS is a successful intervention for the correction of severe crouch gait, according to the criteria by Mosca (1995). The correction of severe crouch gait was adequate and maintained on 3DGA and physical examination parameters, function was restored according to the FAQ and FMS and the long term prognosis was favourable as seen at five years post-SEMLS.

# BIBLIOGRAPHY

- Abel MF, Juhl GA, Vaughan CL and Damiano DL (1998): Gait assessment of fixed ankle-foot orthoses in children with spastic diplegia. *Archives of Physical Medicine and Rehabilitation* 79: 126-133.
- Accardo PJ (1982): Freud on diplegia. American Journal of Disease in Childhood 136: 452-456.
- Albright AL (1996): Spasticity and movement disorders in cerebral palsy. Journal of Child Neurology 11, Suppl. 1: S1-4.
- Allington NJ, Leroy N and Doneux C (2002): Ankle joint range of motion measurements in spastic cerebral palsy children: intraobserver and interobserver reliability and reproducibility of goniometry and visual estimation. *Journal of Pediatric Orthopaedics Part B* 11: 236-239.
- Aminian A, Vankoski SJ, Dias L and Novak RA (2003): Spastic hemiplegic cerebral palsy and the femoral derotation osteotomy: effect at the pelvis and hip in the transverse plane during gait. *Journal of Pediatric Orthopaedics* 23: 314-320.
- Anagnoson JT and DeLeon RE (1997): StataQuest 4. Pacific Grove: Duxbury Press at Brooks/ Cole Publishing Company.
- Anderson FC, Goldberg SR, Pandy MG and Delp SL (2004): Contributions of muscle forces and toe-off kinematics to peak knee flexion during the swing phase of normal gait: an induced position analysis. *Journal of Biomechanics* 37: 731-737.
- Anderson FC and Pandy MG (2003): Individual muscle contributions to support in normal walking. *Gait and Posture* 17: 159-169.
- Andersson C and Mattsson E (2001): Adults with cerebral palsy: a survey describing problems, needs, and resources, with special emphasis on locomotion. *Developmental Medicine and Child Neurology* 43: 76-82.
- Ando N and Ueda S (2000): Functional deterioration in adults with cerebral palsy. *Clinical Rehabilitation* 14: 300-306.

- Andreacchio A, Orellana CA, Miller F and Bowen TR (2000): Lateral column lenthening as treatment for planovalgus foot deformity in ambulatory children with spastic cerebral palsy. *Journal of Pediatric Orthopaedics* 20: 501-505.
- Annaswamy TM, Giddings CJ, Croce UD and Kerrigan DC (1999): Rectus femoris: its role in normal gait. *Archives of Physical Medicine and Rehabilitation* 80: 930-934.
- Arnold AS, Anderson FC, Pandy MG and Delp SL (2005a): Muscular contributions to hip and knee extension during the single limb stance phase of normal gait: a framework for investigating the causes of crouch gait. *Journal of Biomechanics* 38: 2181-2189.
- Arnold AS, Asakawa DS and Delp SL (2000): Do the hanstrings and adductors contribute to excessive internal rotation of the hip in persons with cerebral palsy? *Gait and Posture* 11: 181-190.
- Arnold AS, Blemker SS and Delp SL (2001a): Evaluation of a deformable musculoskeletal model for estimating muscle-tendon lengths during crouch gait. *Annals of Biomedical Engineering* 29: 263-274.
- Arnold AS and Delp SL (2001b): Rotational moment arms of the medial hamstrings and adductors vary with femoral geometry and limb position: implications for the treatment of internally rotated gait. *Journal of Biomechanics* 34: 437-447.
- Arnold AS and Delp SL (2004): The role of musculoskeletal models in patient assessment and treatment. In Gage JR (Ed.): The Treatment of Gait Problems in Cerebral Palsy. London: Mac Keith Press, pp. 165-179.
- Arnold AS, Komattu AV and Delp SL (1997): Internal rotation gait: a compensatory mechanism to restore abduction capacity decreased by bone deformity. *Developmental Medicine and Child Neurology* 39: 40-44.
- Arnold AS, Liu MQ, Schwartz MH, Õunpuu S and Delp SL (2005b): The role of estimating muscle-tendon lengths and velocities of the hamstrings in the evaluation and treatment of crouch gait. *Gait and Posture* In Press.

- Arnold AS, Liu MQ, Schwartz MH, Õunpuu S, Dias LS and Delp SL (2005c):Do the hamstrings operate at increased muscle-tendon lengths and velocities after surgical lengthening? *Journal of Biomechanics* In Press.
- Asakawa DS, Blemker SS, Gold GE and Delp SL (2002): In vivo motion of the rectus femoris muscle after tendon transfer surgery. *Journal of Biomechanics* 35: 1029-1037.
- Asakawa DS, Blemker SS, Rab GT, Bagley A and Delp SL (2004): Threedimensional muscle-tendon geometry after rectus femoris tendon transfer. *The Journal of Bone and Joint Surgery* 86-A: 348-354.
- Asirvatham R, Mukherjee A, Agarwal S, Rooney RJ, Ellis RD and Watts HG (1993): Supracondylar femoral extension osteotomy: its complications. *Journal of Pediatric Orthopaedics* 13: 642-645.
- Bache CE, Selber P and Graham HK (2003): Mini-symposium: cerebral pasly(ii) the management of spastic diplegia. *Current Orthopaedics* 17: 88-104.
- Badawi N, Watson L, Petterson B, Blair E, Slee J, Haan E and Stanley F (1998):
  What constitutes cerebral palsy. *Developmental Medicine and Child Neurology* 40: 520-527.
- Baker LD (1956): A rational approach to the surgical needs of the cerebral palsy patient. *The Journal of Bone and Joint Surgery* 38-A: 313-323.
- Baker R, Finney L and Orr J (1999): A new approach to determine the hip rotation profile from clinical gait analysis data. *Human Movement Science* 18: 655-667.
- Baker R and Rodda J (2003). All you ever wanted to know about the conventional gait model but were afraid to ask. Melbourne, Women's & Children's Health.
- Barnes MP (2001): An overview of the clinical management of spasticity. In Barnes MP and Johnson GR (Eds): Upper Motor Neurone Syndrome and Spasticity. Cambridge: Cambridge University Press, pp. 1-11.

- Baumann JU, Ruetsch H and Schürmann K (1980): Distal hamstring lengthening in cerebral palsy. *International Orthopaedics (SICOT)* 3: 305-309.
- Bax M (2001): What's in a name? *Developmental Medicine and Child Neurology* 43: 75.
- Bax M, Goldstein M, Rosenbaum P, Leviton A, Paneth N, Dan B, Jacobsson B and Damiano D (2005): Proposed definition and classification of cerebral palsy, April 2005. *Developmental Medicine and Child Neurology* 47: 571-576.
- Bax MCO (1964): Terminology and classification of cerebral palsy. Developmental Medicine and Child Neurology 6: 295-307.
- Beals RK (2001): Treatment of knee contracture in cerebral palsy by hamstring lengthening, posterior capsulotomy, and quadriceps mechanism shortening. *Developmental Medicine and Child Neurology* 43: 802-805.
- Beauchesne R, Miller F and Moseley C (1992): Proximal femoral osteotomy using the AO fixed- angle blade plate. *Journal of Pediatric Orthopaedics* 12: 735-740.
- Bell AL, Pedersen DR and Brand RA (1990): A comparison of the accuracy of several hip center location prediction methods. *Journal of Biomechanics* 23: 617-621.
- Bell KJ, Õunpuu S, DeLuca PA and Romness MJ (2002): Natural progression of gait in children with cerebral palsy. *Journal of Pediatric Orthopaedics* 22: 677-682.
- Bennet GC, Rang M and Jones D (1982): Varus and valgus deformities of the foot in cerebral palsy. *Developmental Medicine and Child Neurology* 24: 499-503.
- Berghof R, Zwick EB and Döderlein L (1997): Crouch gait in spastic diplegia after heel cord lengthening. *Gait and Posture* 5: 80.
- Blair E and Stanley F (1985): Interobserver agreement in the classification of cerebral palsy. *Developmental Medicine and Child Neurology* 27: 615-622.

- Bleck EE (1971): Postural and gait abnormalities caused by hip-flexion deformity in spastic cerebral palsy. *The Journal of Bone and Joint Surgery* 53-A: 1468-1488.
- Bleck EE (1984): Where have all the CP children gone? the needs of adults. Developmental Medicine and Child Neurology 26: 674-676.
- Bleck EE (1987): Orthopaedic Management in Cerebral Palsy. (1st ed.) London: Mac Keith Press.
- Bobath B and Bobath K (1978): Motor Development in the Different Types of Cerebral Palsy. London: William Heinemann Medical Books Limited.
- Borton DC, Walker K, Pirpiris M, Nattrass GR and Graham HK (2001): Isolated calf lengthening in cerebral palsy. *The Journal of Bone and Joint Surgery* 83-B: 364-370.
- Bosworth DM and Thompson FR (1946): Fixation of the transplanted tibial tubercle. *The Journal of Bone and Joint Surgery* 28-A: 285-287.
- 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 and Child Neurology* 43: 516-528.
- Boyd RN and Graham HK (1997): Botulinum toxin A in the management of children with cerebral palsy: indications and outcome. *European Journal of Neurology* 4: S15-S22.
- Boyd RN, Pliatsios V and Graham HK (1998): Use of objective clinical measures in predicting response to botulinum toxin A in children with cerebral palsy. *Developmental Medicine and Child Neurology* 40: 28-29.
- Browne AO and McManus F (1987): One-session surgery for bilateral correction of lower limb deformities in spastic diplegia. *Journal of Pediatric Orthopaedics* 7: 259-261.
- Brunner R and Baumann JU (1997): Long-term effects of intertrochanteric varus-derotation osteotomy on femur and acetabulum in spastic cerebral palsy: an 11- to 18-year follow-up study. *Journal of Pediatric Orthopaedics* 17: 585-591.

- Buckon CE, Sienko Thomas S, Jakobson-Hutson S, Moor M, Sussman M and Aiona M (2004): Comparison of three ankle-foot orthosis configurations for children with spastic diplegia. *Developmental Medicine and Child Neurology* 46: 590-598.
- Butler P, Engelbrecht M, Major RE, Tait JH, Stallard J and Patrick JH (1984): Physiological cost index of walking for normal children and its use as an indicator of physical handicap. *Developmental Medicine and Child Neurology* 26: 607-612.
- Butler PB and Nene AV (1991): The biomechanics of fixed ankle foot orthoses and their potential in the management of cerebral palsied children. *Physiotherapy* 77: 81-88.
- Campbell SK (2000): The child's development of functional movement. In Campbell SK (Ed.): Physical Therapy for Children. (2nd ed.). Philadelphia: W.B. Saunders Company, pp. 3-44.
- Carr JH, Shepherd RB and Ada L (1995): Spasticity: research findings and implications for intervention. *Physiotherapy* 81: 421-429.
- Cash J (1976): Neurology for Physiotherapists. (1st ed.) London: Faber & Faber.
- Cathels BA and Reddihough DS (1993): The health care of young adults with cerebral palsy. *The Medical Journal of Australia* 159: 444-446.
- Chambers C, Scott A and Cain TE (1992): Does iliopsoas surgery change pelvic tilt? *Developmental Medicine and Child Neurology* 34 (Suppl. 66): 10.
- Chambers H, Lauer A, Kaufman K, Cardelia JM and Sutherland D (1998): Prediction of outcome after rectus femoris surgery in cerebral palsy: the role of cocontraction of the rectus femoris and vastus lateralis. *Journal* of Pediatric Orthopaedics 18: 703-711.
- Chambers HG (2001): Treatment of functional limitations at the knee in ambulatory children with cerebral palsy. *European Journal of Neurology* 8 (Suppl. 5): 59-74.

- Chandler FA (1933): Re-establishment of normal leverage of the patella in knee flexion deformity in spastic paralysis. *Surgery, Gynecology and Obstetrics* 57: 523-527.
- Chang W, Tsirikos AI, Miller F, Lennon N, Schuyler J, Kerstetter L and Glutting J (2004): Distal hamstring lengthening in ambulatory children with cerebral palsy: primary versus revision procedures. *Gait and Posture* 19: 298-304.
- Chung CY, Stout J and Gage JR (1997): Rectus femoris transfer-gracilis versus sartorius. *Gait and Posture* 6: 137-146.
- Cleveland M and Bosworth DM (1936): Surgical correction of flexion deformity of the knees due to spastic paralysis. *Surgery, Gynecology and Obstetrics* 63: 659-664.
- Colver AF and Sethumadhavan T (2003): The term diplegia should be abandoned. *Archives of Disease in Childhood* 88: 286-290.
- Cook RE, Schneider I, Hazlewood ME, Hillman SJ and Robb JE (2003): Gait analysis alters decision-making in cerebral palsy. *Journal of Pediatric Orthopaedics* 23: 292-295.
- Cooke PH, Carey RPL and Williams PF (1989): Lower femoral osteotomy in cerebral palsy: brief report. *The Journal of Bone and Joint Surgery* 71-B: 146-147.
- Cosgrove AP, Corry IS and Graham HK (1994b): Botulinum toxin in the management of the lower limb in cerebral palsy. *Developmental Medicine and Child Neurology* 36: 386-396.
- Cosgrove AP and Graham HK (1994a): Botulinum toxin A prevents the development of contractures in the hereditary spastic mouse. *Developmental Medicine and Child Neurology* 36: 379-385.
- Crenna P (1998): Spasticity and 'spastic' gait in children with cerebral palsy. *Neuroscience and Biobehavioural Review* 22: 571-578.
- Dabney KW, Lipton GE and Miller F (1997): Cerebral palsy. *Current Opinion in Pediatrics* 9: 81-88.

- Dahl TH (2002): International classification of functioning, disability and health: an introduction and discussion of its potential impact on rehabilitation services and research. *Journal of Rehabilitation Medicine* 34: 201-204.
- Damiano DL and Abel MF (1998): Functional outcomes of strength training in spastic cerebral palsy. Archives of Physical Medicine and Rehabilitation 79: 119-125.
- Damiano DL, Abel MF, Pannunzio M and Romano J (1999): Interrelationships of strength and gait before and after hamstrings lengthening. *Journal of Pediatric Orthopaedics* 19: 352-358.
- Damiano DL, Dodd KJ and Taylor NF (2002): Should we be testing and training muscle strength in cerebral palsy? *Developmental Medicine and Child Neurology* 44: 68-72.
- Damiano DL, Kelly LE and Vaughan CL (1995a): Effects of quadriceps femoris muscle strengthening on crouch gait in children with spastic diplegia. *Physical Therapy* 75: 658-667.
- Damiano DL, Vaughan CL and Abel MF (1995b): Muscle response to heavy resistance exercise in children with spastic cerebral palsy. *Developmental Medicine and Child Neurology* 37: 731-739.
- Damron TA, Breed AL and Cook T (1993): Diminished knee flexion after hamstring surgery in cerebral palsy patients: prevalence and severity. *Journal of Pediatric Orthopaedics* 13: 188-191.
- Damron TA, Breed AL and Roecker E (1991): Hamstring tenotomies in cerebral palsy: long term retrospective analysis. *Journal of Pediatric Orthopaedics* 11: 514-519.
- Damron TA, Greenwald TA and Breed AL (1994): Chronologic outcome of surgical tendoachilles lengthening and natural history of gastroc-soleus contracture in cerebral palsy. *Clinical Orthopaedics and Related Research* 301: 249-255.
- Davids JR (2003b): Qualitative description of normal gait. In The Melbourne Gait Course 2003, Melbourne, Australia, pp. 1-11.

- Davids JR, Mason TA, Danko A, Banks D and Blackhurst D (2001): Surgical management of hallux valgus deformity in children with cerebral palsy. *Journal of Pediatric Orthopaedics* 21: 89-94.
- Davids JR, Õunpuu S, DeLuca PA and Davis RB (2003a): Optimization of walking ability of children with cerebral palsy. *The Journal of Bone and Joint Surgery* 85-A: 2224-2234.
- Davis RB (1997): Reflections on clinical gait analysis. *Journal of Electromyography and Kinesiology* 7: 251-257.
- Davis RB (2003): Instructional Course Notes-Normal Kinetics: Concepts & Patterns. In The Melbourne Gait Course, Melbourne, pp. 6.1-6.4.
- Davis RB (2004): The Motion Analysis Laboratory. In Gage JR (Ed.): The Treatment of Gait Problems in Cerebral Palsy. London: Mac Keith Press, pp. 90-98.
- Davis RB, Õunpuu S, Tyburski D and Gage JR (1991): A gait analysis data collection and reduction technique. *Human Movement Science* 10: 575-587.
- Delp SL, Arnold AS, Speers RA and Moore CA (1996): Hamstrings and psoas lengths during normal and crouch gait: implications for muscle-tendon surgery. *Journal of Orthopaedic Research* 14: 144-151.
- Delp SL, Hess WE, Hungerford DS and Jones LC (1999): Variation of rotation moment arms with hip flexion. *Journal of Biomechanics* 32: 493-501.
- Delp SL, Loan JP, Hoy MG, Zajac FE, Topp EL and Rosen JM (1990): An interactive graphics-based model of the lower extremity to study orthopaedic surgical procedures. *IEEE Transactions on Biomedical Engineering* 37: 757-767.
- Delp SL, Ringwelski DA and Carroll NC (1994): Transfer of the rectus femoris: effects of transfer site on moment arms about the knee and hip. *Journal* of Biomechanics 27: 1201-1211.
- Delp SL, Statler K and Carroll NC (1995): Preserving plantar flexion strength after surgical treatment for contracture of the triceps surae: a computer simulation study. *Journal of Orthopaedic Research* 13: 96-104.

- Delp SL and Zajac FE (1992): Force- and moment-generating capacity of lower-extremity muscles before and after tendon lengthening. *Clinical Orthopaedics and Related Research* 284: 247-259.
- DeLuca PA (1991): Gait analysis in the treatment of the ambulatory child with cerebral palsy. *Clinical Orthopaedics and Related Research* 264: 65-75.
- DeLuca PA, Davis RB, Õunpuu S, Rose S and Sirkin R (1997): Alterations in surgical decision making in patients with cerebral palsy based on threedimensional gait analysis. *Journal of Pediatric Orthopaedics* 17: 608-614.
- DeLuca PA, Ounpuu S, Davis RB and Walsh JHP (1998): Effect of hamstring and psoas lengthening on pelvic tilt in patients with spastic diplegic cerebral palsy. *Journal of Pediatric Orthopaedics* 18: 712-718.
- den Otter AR, Geurts ACH, Mulder T and Duysens J (2004): Speed related changes in muscle activity from normal to very slow walking speeds. *Gait and Posture* 19: 270-278.
- Dennyson WG and Fulford GE (1976): Subtalar athrodesis by cancellous grafts and metallic internal fixation. *The Journal of Bone and Joint Surgery* 58-B: 507-510.
- Dhawlikar SH, Root L and Mann RL (1992): Distal lengthening of the hamstrings in patients who have cerebral palsy. *The Journal of Bone and Joint Surgery* 74-A: 1385-1391.
- Dillin W and Samilson RL (1983): Calcaneus deformity in cerebral palsy. *Foot* and Ankle 4: 167-170.
- Do KP, Wren TAL and Kay RM (2004): Dynamic hamstring length in children with cerebral palsy and crouch gait before and after surgery. *Developmental Medicine and Child Neurology* 46 (Suppl. 99): 32-33.
- Dodd KJ, Taylor NF and Damiano DL (2002): A systematic review of the effectiveness of strength-training programs for people with cerebral palsy. *Archives of Physical Medicine and Rehabilitation* 83: 1157-1164.

- Dodd KJ, Taylor NF and Graham HK (2003): A randomized clinical trial of strength training in young people with cerebral palsy. *Developmental Medicine and Child Neurology* 45: 652-657.
- Dodgin DA, De Swart RJ, Stefko RM, Wenger DR and Ko J (1998): Distal tibial/ fibular derotation osteotomy for correction of tibial torsion: review of technique and results in 63 cases. *Journal of Pediatric Orthopaedics* 18: 95-101.
- Drummond DS, Rogala E, Templeton J and Cruess R (1974): Proximal hamstring release for knee flexion and crouched posture in cerebral palsy. *The Journal of Bone and Joint Surgery* 56-A: 1598-1602.
- Duffy CM and Cosgrove AP (2002): Mini-symposium: the paediatric foot (iii) The foot in cerebral palsy. *Current Orthopaedics* 16: 104-113.
- Duffy CM, Hill AE and Graham HK (1997): The influence of flexed-knee gait on the energy cost of walking in children. *Developmental Medicine and Child Neurology* 39: 234-238.
- Eames NWA, Baker R, Hill N, Graham HK, Taylor TC and Cosgrove AP (1999): The effect of botulinum toxin A on gastrocnemius length: magnitude and duration of response. *Developmental Medicine and Child Neurology* 41: 226-232.
- Eggers GWN (1952): Transplantation of hamstring tendons to femoral condyles in order to improve hip extension and to decrease knee flexion in cerebral spastic paralysis. *The Journal of Bone and Joint Surgery* 34-A: 827-830.
- Eggers GWN and Evans EB (1963): Surgery in cerebral palsy. *The Journal of Bone and Joint Surgery* 45-A: 1275-1305.
- Evans D (1975b): Calcaneo-valgus deformity. *The Journal of Bone and Joint Surgery* 57-B: 270-278.
- Evans EB (1975a): The Knee in Cerebral Palsy. In Samilson RL (Ed.): Orthopeadic Aspects of Cerebral Palsy. London: Willaim Heinemann Medical Books Ltd, pp. 173-194.

- Evans P, Johnson A, Mutch L and Alberman E (1986): Report of a Meeting on the Standardisation of the Recording and Reporting of Cerebral Palsy. *Developmental Medicine and Child Neurology* 28: 547-548.
- Fabry G, Liu XC and Molenaers G (1999): Gait pattern in patients with spastic diplegic cerebral palsy who underwent staged operations. *Journal of Pediatric Orthopaedics Part B* 8: 33-38.
- Flett PJ (2003): Rehabilitation of spasticity and related problems in childhood cerebral palsy. *Journal of Paediatric Child Health* 39: 6-14.
- Forbes A and Wolfe R (2001): Analysis of studies with correlated data: a simple approach using robust standard errors. *Australasian Epidemiologist* 8: 13-16.
- Fosang AL, Galea MP, McCoy AT, Reddihough DS and Story I (2003): Measures of muscle and joint performance in the lower limb of children with cerebral palsy. *Developmental Medicine and Child Neurology* 45: 664-670.
- Frost HM (1971): Cerebral palsy. The spastic crouch. *Clinical Orthopaedics* 80: 2-8.
- Gage JR (1990): Surgical treatment of knee dysfunction in cerebral palsy. *Clinical Orthopaedics and Related Research* 253: 45-54.
- Gage JR (1991): Gait Analysis in Cerebral Palsy. London: Mac Keith Press.
- Gage JR (1993): Gait analysis. Clinical Orthopaedics and Related Research 288: 126-134.
- Gage JR (2004a): The Treatment of Gait Problems in Cerebral Palsy. London: Mac Keith Press.
- Gage JR (2004b): A qualitative description of normal gait. In Gage JR (Ed.): The Treatment of Gait Problems in Cerebral Palsy. London: Mac Keith Press, pp. 42-70.
- Gage JR (2004d): Evaluation and treatment of crouch gait in cerebral palsy. Instructional Course No 28. In American Academy for Cerebral Palsy

and Developmental Medicine 58th Annual Meeting, Los Angeles, California, pp. 1-13.

- Gage JR (2004f): Treatment principles for crouch gait. In Gage JR (Ed.): The Treatment of Gait Problems in Cerebral Palsy. London: Mac Keith Press, pp. 382-397.
- Gage JR, DeLuca PA and Renshaw TS (1995): Gait analysis: principles and applications. *The Journal of Bone and Joint Surgery* 77-A: 1607-1623.
- Gage JR and Õunpuu S (1989): Gait analysis in clinical practice. *Seminars in Orthopaedics* 4: 72-87.
- Gage JR, Perry J, Hicks RR, Koop S and Werntz JR (1987): Rectus femoris transfer to improve knee function of children with cerebral palsy. *Developmental Medicine and Child Neurology* 29: 159-166.
- Gage JR and Quanbeck DS (2004e): Orthotics and mobility aids in cerebral palsy. In Gage JR (Ed.): The Treatment of Gait Problems in Cerebral Palsy. London: Mac Keith Press, pp. 273-285.
- Gage JR and Schwartz M (2004c): Pathological gait and lever-arm dysfunction.In Gage JR (Ed.): The Treatment of Gait Problems in Cerebral Palsy.London: Mac Keith Press, pp. 180-204.
- Gage JR, Stout JL, Matsuo K and Novacheck TF (2000): Distal femoral extension osteotomy for treatment of persistent crouch gait. *Gait and Posture* 11: 115.
- Giuliani CA (1991): Theories of motor control: new concepts for physical therapy. In Lister MJ (Ed.): Contempory Management of Motor Control Problems: Proceedings of the II STEP Conference. Alexandria: Foundation for Physical Therapy, pp. 29-35.
- Goldberg SR, Anderson FC, Pandy MG and Delp SL (2004): Muscles that influence knee flexion velocity in double support: implications for stiff-knee gait. *Journal of Biomechanics* 37: 1189-1196.
- Goldberg SR, Õunpuu S, Arnold AS, Gage JR and Delp SL (2005): Kinematic and kinetic factors that correlate with improved knee flexion following treatment for stiff-knee gait. *Journal of Biomechanics* In Press.

- Goldberg SR, Õunpuu S and Delp SL (2003): The importance of swing-phase initial conditions in stiff-knee gait. *Journal of Biomechanics* 36: 1111-1116.
- Gormley ME (2001a): Treatment of neuromuscular and musculoskeletal problems in cerebral palsy. *Pediatric Rehabilitation* 4: 5-16.
- Gormley ME, Krach LE and Piccini L (2001b): Spasticity management in the child with spastic quadriplegia. *European Journal of Neurology* 8 (Suppl. 5): 127-135.
- Gorter JW, Rosenbaum PL, Hanna SE, Palisano RJ, Bartlett DJ, Russell DJ, Walter SD, Raina P, Galuppi BE and Wood E (2004): Limb distribution, motor impairment, and functional classification of cerebral palsy. *Developmental Medicine and Child Neurology* 46: 461-467.
- Gorton G, Hebert D and Goode B (2001): Assessment of the kinematic variability between 12 shriners motion analysis laboratories. *Gait and Posture* 13: 247.
- Gorton G, Hebert D and Goode B (2002): Assessment of the kinematic variability between 12 shriners motion analysis laboratories part 2: Short term follow up. *Gait and Posture* 16 (Suppl. 1): 65-66.
- Gottschall JS and Kram R (2003): Energy cost and muscular activity required for propulsion during walking. *Journal of Applied Physiology* 94: 1766-1772.
- Gough M, Eve LC, Robinson RO and Shortland AP (2004): Short-term outcome of multilevel surgical intervention in spastic diplegic cerebral palsy compared with the natural history. *Developmental Medicine and Child Neurology* 46: 91-97.
- Gough M, Fairhurst C and Shortland AP (2005): Botulinum toxin and cerebral palsy: time for reflection? *Developmental Medicine and Child Neurology* 47: 709-712.
- Graham HK (1997): The orthopaedic management of cerebral palsy. In Broughton NS (Ed.): A Textbook of Paediatric Orthopaedics. London: W.B.Saunders, pp. 101-113.

- Graham HK (2004b): Mechanisms of Deformity. In Scrutton D, Damiano D and Mayston M (Eds): Management of the Motor Disorders of Children with Cerebral Palsy. (2nd ed.). London: Mac Keith Press, pp. 105-129.
- Graham HK, Aoki KR, Autti-Ramo I, Boyd RN, Delgado MR, Gaebler-Spira DJ, Gormley Jr ME, Guyer BM, Heinen F, Holton AF, Matthews D, Molenaers G, Motta F, Garcia Ruiz PJ and Wissel J (2000): Recommendations for the use of botulinum toxin type A in the management of cerebral palsy. *Gait and Posture* 11: 67-79.
- Graham HK and Fixsen JA (1988): Lengthening of the calcaneal tendon in spastic hemiplegia by the White Slide technique. *The Journal of Bone and Joint Surgery* 70-B: 472-475.
- Graham HK, Harvey A, Rodda J, Nattrass GR and Pirpiris M (2004a): The Functional Mobility Scale (FMS). *Journal of Pediatric Orthopaedics* 24: 514-520.
- Graham HK and Selber P (2003): Musculoskeletal aspects of cerebral palsy. *The Journal of Bone and Joint Surgery* 85-B: 157-166.
- Granata KP, Abel MF and Damiano DL (2000): Joint angular velocity in spastic gait and the influence of muscle-tendon lengthening. *The Journal of Bone and Joint Surgery* 82-A: 174-186.
- Gugenheim JJ, Rosenthal RK and Simon SR (1979): Knee flexion deformities and genu recurvatum in cerebral palsy: roentgenographic findings. *Developmental Medicine and Child Neurology* 21: 563-571.
- Hadley N, Chambers C, Scarborough N, Cain T and Rossi D (1992): Knee motion following multiple soft-tissue releases in ambulatory patients with cerebral palsy. *Journal of Pediatric Orthopaedics* 12: 324-328.
- Hagberg B, Hagberg G and Olow I (1975): The changing panorama of cerebral palsy in Sweden 1954-1970. *Acta Paediatrica Scandinavica* 64: 187-192.
- Harrington ED, Lin RS and Gage JR (1984): Use of the anterior floor reaction orthosis in patients with cerebral palsy. *Bulletin of Orthotics and Prosthetics* 37: 34-42.

- Hau R, Dickens DRV, Nattrass GR, O'Sullivan M, Torode IP and Graham HK (2000): Which implant for proximal femoral osteotomy in children? A comparison of the AO (ASIF) 90<sup>o</sup> fixed-angle blade plate and the Richards intermediate hip screw. *Journal of Pediatric Orthopaedics* 20: 336-343.
- Haxton H (1945): The function of the patella and the effects of its excision. Surgery, Gynecology and Obstetrics 8: 389-395.
- Herbert R, Jamtvedt G, Mead J and Hagen KB (2005): Outcome measures measure outcomes, not effects of intervention. *Australian Journal of Physiotherapy* 51: 3-4.
- Herring JA (2002): Disorders of the Brain. In Herring JA (Ed.): Tachdjian's Pediatric Orthopeadics. (3rd ed.), vol 2. Philadelphia: WB Saunders, pp. 1159-1173.
- Hodges PW and Richardson CA (1997): Contraction of the abdominal muscles associated with movement of the lower limb. *Physical Therapy* 77: 132-142.
- Hof AL (1996): Scaling gait data to body size. Gait and Posture 4: 222-223.
- Hof AL and Zijlstra W (1997): Letter to the editor. *Journal of Biomechanics* 30: 299.
- Hoffer MM, Feiwell E, Perry R, Perry J and Bonnett C (1973): Functional ambulation in patients with myelomeningocele. *The Journal of Bone and Joint Surgery* 55-A: 137-148.
- Hoffinger SA, Rab GT and Abou-Ghaida H (1993): Hamstrings in cerebral palsy crouch gait. *Journal of Pediatric Orthopaedics* 13: 722-726.
- Holling EE and Leviton A (1999): Characteristics of cranial ultrasound whitematter echolucencies that predict disability: a review. *Developmental Medicine and Child Neurology* 41: 136-139.
- Horak FB (1991): Assumptions underlying motor control for neurologic rehabilitation. In Lister MJ (Ed.): Contempory Management of Motor Control Problems: Proceedings of the II STEP Conference. Alexandria: Foundation for Physical Therapy, pp. 11-27.

- Howard J, Soo B, Graham HK, Boyd RN, Reid S, Lanigan A, Wolfe R and Reddihough DS (2005): Cererbal palsy in Victoria: Motor types, topography and gross motor function. *Journal of Paediatric Child Health* 41: 479-483.
- Hsu LCS and Li HSY (1990): Distal hamstring elongation in the management of spastic cerebral palsy. *Journal of Pediatric Orthopaedics* 10: 378-381.
- Huk O, Duhaime M and Allard P (1987): Kinematic analysis of gait patterns in unoperated spastic diplegic children. *Orthopaedic Transactions* 11: 211.
- Ingram TTS (1955): A study of cerebral palsy in the childhood population of Edinburgh. *Archives of Diseases in Childhood* 30: 85-98.
- Ingram TTS (1984): A historical review of the definition and classification of the cerebral palsies. In Stanley F and Alberman E (Eds): The Epidemiology of the Cerebral Palsies. London: Spastics International Medical Publications, pp. 1-11.
- Jahnsen R, Villien L, Aamodt G, Stanghelle JK and Holm I (2004a): Musculoskeletal pain in adults with cerebral palsy compared with the general population. *Journal of Rehabilitation Medicine* 36: 78-84.
- Jahnsen R, Villien L, Egeland T, Stanghelle JK and Holm I (2004b): Locomotion skills in adults with cerebral palsy. *Clinical Rehabilitation* 18: 309-316.
- Jahnsen R, Villien L, Stanghelle JK and Holm I (2003): Fatigue in adults with cerebral palsy in Norway compared with the general population. *Developmental Medicine and Child Neurology* 45: 296-303.
- Johnson DC, Damiano DL and Abel MF (1997): The evolution of gait in childhood and adolescent cerebral palsy. *Journal of Pediatric Orthopaedics* 17: 392-396.
- Johnston TE, Finson RL, McCarthy JJ, Smith BT, Betz RR and Mulcahey MJ (2004a): Use of functional electrical stimulation to augment traditional orthopaedic surgery in children with cerebral palsy. *Journal of Pediatric Orthopaedics* 24: 283-291.

- Johnston TE, Moore SE, Quinn LT and Smith BT (2004b): Energy cost of walking in children with cerebral palsy: relation to the Gross Motor Function Classification System. Developmental Medicine and Child Neurology 46: 34-38.
- Jonkers I, Stewart C and Spaepen A (2003): The complementary role of the plantarflexors, hamstrings and gluteus maximus in the control of stance limb stability during gait. *Gait and Posture* 17: 264-272.
- Kadaba MP, Ramakrishnan HK, Jacobs D, Chambers C, Scarborough N and Goode B (1991): Gait pattern recognition in spastic diplegia. *Developmental Medicine and Child Neurology* 33 (Suppl. 64): 28.
- Kadaba MP, Ramakrishnan HK and Wootten ME (1990): Measurement of lower extremity kinematics during level walking. *Journal of Orthopaedic Research* 8: 383-392.
- Kadaba MP, Ramakrishnan HK, Wootten ME, Gainey J, Gorton G and Cochran GVB (1989): Repeatability of kinematic, kinetic, and electromyographic data in normal adult gait. *Journal of Orthopaedic Research* 7: 849-860.
- Kay RM, Dennis S, Rethlefsen S, Reynolds RAK, Skaggs DL and Tolo VT (2000): The effect of preoperative gait analysis on orthopaedic decision making. *Clinical Orthopaedics and Related Research* 372: 217-222.
- Kay RM, Rethlefsen S, Reed M, Do KP, Skaggs DL and Wren TAL (2004b): Changes in pelvic rotation after soft tissue and bony surgery in ambulatory children with cerebral palsy. *Journal of Pediatric Orthopaedics* 24: 278-282.
- Kay RM, Rethlefsen SA, Hale JM, Skaggs DL and Tolo VT (2003): Comparison of proximal and distal rotational femoral osteotomy in children with cerebral palsy. *Journal of Pediatric Orthopaedics* 23: 150-154.
- Kay RM, Rethlefsen SA, Kelly JP and Wren TAL (2004a): Predictive value of the Duncan-Ely test in distal rectus femoris transfer. *Journal of Pediatric Orthopaedics* 24: 59-62.

- Kay RM, Rethlefsen SA, Skaggs D and Leet A (2002): Outcome of medial versus combined medial and lateral hamstring lengthening surgery in cerebral palsy. *Journal of Pediatric Orthopaedics* 22: 169-172.
- Keats S and Kambin P (1962): An evaluation of surgery for the correction of knee-flexion contracture in children with cerebral spastic paralysis. *The Journal of Bone and Joint Surgery* 44-A: 1146-1154.
- Keenan WN, Rodda J, Wolfe R, Roberts S, Borton DC and Graham HK (2004): The static examination of children and young adults with cerebral palsy in the gait analysis laboratory: technique and observer agreement. *Journal of Pediatric Orthopaedics Part B* 13: 1-8.
- Kepple TM, Lohmann Siegel K and Stanhope SJ (1997): Relative contributions of the lower extremity joint moments to forward progression and support during gait. *Gait and Posture* 6: 1-8.
- Kilgour G, McNair P and Stott NS (2003): Intrarater reliability of lower limb sagittal range-of-motion measures in children with spastic diplegia. *Developmental Medicine and Child Neurology* 45: 391-399.
- Kim H, Aiona MD and Sussman MD (2004): Recurrence after femoral derotational osteotomy in cerebral palsy. *Developmental Medicine and Child Neurology* 46 (Suppl. 99): 14.
- Kirtley C (2002): Sensitivity of the modified Helen Hayes Model to marker placement errors. In Three Dimensional Human Movement Analysis Symposium, Newcastle, pp. 68-71.
- Koman LA, Mooney JF, Smith B, Goodman A and Mulvaney T (1993): Management of cerebral palsy with botulinum-A toxin. Preliminary investigation. *Journal of Pediatric Orthopaedics* 13: 489-495.
- Kramer A and Stevens PM (2001): Anterior femoral stapling. *Journal of Pediatric Orthopaedics* 21: 804- 807.
- Kramer JF and MacPhail HEA (1994): Relationships among measures of walking efficiency, gross motor ability, and isokinetic strength in adolescents with cerebral palsy. *Pediatric Physical Therapy* 6: 3-8.

- Kuban KCK and Leviton A (1994): Cerebral Palsy. *The New England Journal* of Medicine 330: 188-195.
- Lance JW (1980): Symposium synopsis. In Feldman RG, Young RR and Koella WP (Eds): Spasticity: Disordered Motor Control. Chicago: Year Book Medical Publishers, pp. 485-494.
- Lance JW (1990): What is spasticity? The Lancet 335: 606.
- Larin HM (2000): Motor learning: theories and strategies for the practitioner. InCampbell SK (Ed.): Physical Therapy for Children. (2nd ed.).Philadelphia: W.B. Saunders Company, pp. 170-197.
- Lee EH, Goh JCH and Bose K (1992): Value of gait analysis in the assessment of surgery in cerebral palsy. *Archives of Physical Medicine and Rehabilitation* 73: 642-646.
- Lieb FJ and Perry J (1968): Quadriceps function. *The Journal of Bone and Joint Surgery* 50-A: 1535-1548.
- Lin CJ, Guo LY, Su FC, Chou YL and Cherng RJ (2000): Common abnormal kinetic patterns of the knee in gait in spastic diplegia of cerebral palsy. *Gait and Posture* 11: 224-232.
- Little WJ (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. *Transactions of the Obstetrical Society of London* 3: 293-344.
- Lloyd-Roberts GC, Jackson AM and Albert JS (1985): Avulsion of the distal pole of the patella in cerebral palsy. *The Journal of Bone and Joint Surgery* 67-B: 252-254.
- Lotman DB (1976): Knee flexion deformity and patella alta in spastic cerebral palsy. *Developmental Medicine and Child Neurology* 18: 315-319.
- MacKeith RC, Mackenzie ICK and Polani PE (1959): The Little Club: memorandum on terminology and classification of cerebral palsy. *Cerebral Palsy Bulletin* 5: 27-35.

- Marks MC, Alexander J, Sutherland DH and Chambers HG (2003): Clinical utility of the Duncan-Ely test for rectus femoris dysfunction during the swing phase of gait. *Developmental Medicine and Child Neurology* 45: 763-768.
- Mayer NH (1997): Clinicophysiologic concepts of spasticity and motor dysfunction in adults with an upper motoneuron lesion. *Muscle and Nerve* 20 (Suppl. 6): S1-13.
- Mazur JM, Shanks DE, Cummings RJ, McCluskey WP, Federico L and Goins M (1992): Nonsurgical treatment of tight achilles tendon. In Sussman MD (Ed.): The Diplegic Child: Evaluation and Management. Rosemont: American Academy of Orthopaedic Surgeons, pp. 343-354.
- McDowell BC, Hewitt V, Nurse A, Weston T and Baker R (2000): The variability of goniometric measurements in ambulatory children with spastic cerebral palsy. *Gait and Posture* 12: 114-121.
- McMulkin ML, Gulliford JJ, Williamson RV and Ferguson RL (2000): Correlation of static to dynamic measures of lower extremity range of motion in cerebral palsy and control populations. *Journal of Pediatric Orthopaedics* 20: 366-369.
- McNee AE, Shortland AP, Gough M and Robinson RO (2002): What holds the child in crouch up? *Gait and Posture* 16 (Suppl. 1): S120.
- Metaxiotis D, Wolf S and Doederlein L (2004): Conversion of biarticular to monoarticular muscles as a component of multilevel surgery in spastic diplegia. *The Journal of Bone and Joint Surgery* 86-B: 102-109.
- Miller F, Cardoso Dias R, Lipton GE, Albarracin JP, Dabney KW and Castagno P (1997): The effect of rectus EMG patterns on the outcome of rectus femoris transfers. *Journal of Pediatric Orthopaedics* 17: 603-607.
- Miller F, Dabney KW and Rang M (1995): Complications in cerebral palsy treatment. In Epps CH and Bowen JR (Eds): Complications in Pediatric Orthopaedic Surgery. Philadelphia: J.B. Lippincott Company, pp. 477-544.

Minear WL (1956): A classification of cerebral palsy. Pediatrics 18: 841-852.

- Molenaers G, Desloovere K, De Cat J, Jonkers I, De Borre L, Pauwels P, Nijs J, Fabry G and De Cock P (2001): Single event multilevel botulinum toxin type A treatment and surgery: similarities and differences. *European Journal of Neurology* 8: 88-97.
- Molenaers G, Desloovere K, Eyssen M, De Cat J, Jonkers I and De Cock P (1999a): Botulinum toxin type A treatment of cerebral palsy: an integrated approach. *European Journal of Neurology* 6: S51-S57.
- Molenaers G, Desloovere K, Pauwels P, De Cat J, Eyssen M, De Borre L, Nuttin B, Timmermans I, Nijs J and Dunn MB (2004): Effect of selective dorsal rhizotomy on gait in children with cerebral palsy: risk of including S2 roots in selective dorsal rhizotomy. *Developmental Medicine and Child Neurology* 46 (Suppl. 99): 8.
- Molenaers G, Eyssen M, Desloovere K and Jonkers I (1999b): A multilevel approach to botulinum toxin type A treatment of the (Ilio)psoas in spasticity in cerebral palsy. *European Journal of Neurology* 6 (Suppl. 4): S59-S62.
- Moreau N, Tinsley S and Li L (2005): Progression of knee joint kinematics in children with cerebral palsy with and without rectus femoris transfers: a long-term follow up. *Gait and Posture* 22: 132-137.
- Morris C, Galuppi BE and Rosenbaum PL (2004): Reliability of family report for the Gross Motor Function Classification System. *Developmental Medicine and Child Neurology* 46: 455-460.
- Morton JF, Brownlee M and McFadyen AK (2005): The effects of progressive resistance training for children with cerebral palsy. *Clinical Rehabilitation* 19: 283-289.
- Morton R (1999): New surgical interventions for cerebral palsy and the place of gait analysis. *Developmental Medicine and Child Neurology* 41: 424-428.
- Mosca VS (1995): Calcaneal lengthening for valgus deformity of the hindfoot. *The Journal of Bone and Joint Surgery* 77-A: 500-512.

- Murphy KP, Molnar GE and Lankasky K (1995): Medical and functional status of adults with cerebral palsy. *Developmental Medicine and Child Neurology* 37: 1075-1084.
- Murray MP, Guten GN, Sepic SB, Gardner GM and Baldwin JM (1978): Function of the triceps surae during gait. *The Journal of Bone and Joint Surgery* 60-A: 473-476.
- Murray-Weir M, Root L, Peterson M, Lenhoff M, Wagner C and Marcus P (2003): Proximal femoral varus rotation osteotomy in cerebral palsy: a prospective gait study. *Journal of Pediatric Orthopaedics* 23: 321-329.
- Mutch L, Alberman E, Hagberg B, Kodama K and Perat MV (1992): Cerebral palsy epidemiology: where are we now and where are we going? *Developmental Medicine and Child Neurology* 34: 547-555.
- Nelson KB (2002): The epidemiology of cerebral palsy in term infants. *Mental Retardation and Developmental Disabilities Research Reviews* 8: 146-150.
- Nene A, Byrne C and Hermens H (2004): Is rectus femoris really a part of quadriceps? Assessment of rectus femoris function during gait in ablebodied adults. *Gait and Posture* 20: 1-13.
- Nene A, Mayagoitia R and Veltink P (1999): Assessment of rectus femoris function during initial swing phase. *Gait and Posture* 9: 1-9.
- Nene AV, Evans GA and Patrick JH (1993): Simultaneous multiple operations for spastic diplegia. *The Journal of Bone and Joint Surgery* 75-B: 488-494.
- Neptune RR, Kautz SA and Zajac FE (2001): Contributions of the individual ankle plantar flexors to support, forward progression and swing initiation during walking. *Journal of Biomechanics* 34: 1387-1398.
- Noonan KJ, 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.

- Noritake K, Yoshihashi Y and Miyata T (2005): Calcaneal lengthening for planovalgus foot deformity in children with spastic cerebral palsy. *Journal of Pediatric Orthopaedics Part B* 14: 274-279.
- Norlin R and Odenrick P (1986): Development of gait in spastic children with cerebral palsy. *Journal of Pediatric Orthopaedics* 6: 674-680.
- Norlin R and Tkaczuk H (1985): One-session surgery for correction of lower extremity deformities in children with cerebral palsy. *Journal of Pediatric Orthopaedics* 5: 208-211.
- Norlin R and Tkaczuk H (1992): One session surgery on the lower limb in children with cerebral palsy. A five year follow-up. *International Orthopaedics (SICOT)* 16: 291-293.
- Norman GR and Streiner DL (2000): Biostatistics. (2nd ed.) Hamilton: BC Decker Inc.
- Normand X and Dubousset J (1985): Remise en tension de l'appareil extenseur du genou dans la démarche en triple flexion chez l'enfant infirme moteur. *Revue de Chirurgie Orthopédique* 71: 301-310.
- Novacheck TF, Chung CY, Trost JP and Gage JR (1996): Crouch gait in cerebral palsy- the effects of psoas lengthening. *Gait and Posture* 4: 184.
- Novacheck TF and Schwartz M (2004): Functional assessment of outcomes. In Gage JR (Ed.): The Treatment of Gait Problems in Cerebral Palsy. London: Mac Keith Press, pp. 406-422.
- Novacheck TF, Stout JL 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: 75-81.
- Novacheck TF, Trost JP and Schwartz MH (2002): Intramuscular psoas lengthening improves dynamic hip function in children with cerebral palsy. *Journal of Pediatric Orthopaedics* 22: 158-164.
- O'Byrne JM, Jenkinson A and O'Brien TM (1998): Quantitative analysis and classification of gait patterns in cerebral palsy using a three-dimensional motion analyzer. *Journal of Child Neurology* 13: 101-108.

- O'Connell PA, D'Souza L, Dudeney S and Stephens M (1998): Foot deformities in children with cerebral palsy. *Journal of Pediatric Orthopaedics* 18: 743-747.
- Okumura A, Kato T, Kuno K, Hayakawa F and Watanabe K (1997): MRI findings in patients with spastic cerebral palsy. II: correlation with type of cerebral palsy. *Developmental Medicine and Child Neurology* 39: 369-372.
- O'Malley M (1996): Normalization of temporal-distance parameters in pediatric gait. *Journal of Biomechanics* 29: 619-625.
- O'Malley MJ, Abel MF, Damiano DL and Vaughan CL (1997): Fuzzy clustering of children with cerebral palsy based on temporal-distance parameters. *IEEE Transactions on Rehabilitation Engineering* 5: 300-309.
- Orendurff MS, Aiona MD, Dorociak RD and Pierce RA (2002): Length and force of the gastrocnemius and soleus during gait following tendo Achilles lengthenings in children with equinus. *Gait and Posture* 15: 130-135.
- Orendurff MS, Chung JS and Pierce RA (1998): Limits to passive range of joint motion and the effect on crouch gait in children with cerebral palsy. *Gait and Posture* 7: 165.
- Orendurff MS, Nichols MP, Kinzey SJ, Dorociak R, Pierce RA and Aiona MD (2000): Strength, joint range, and bony deformity: determining the causes of crouch gait in children with cerebral palsy. *Gait and Posture* 11: 135.
- Osgood RB (1913): A method of osteotomy of the lower end of the femur in cases of permanent flexion of the knee-joint. *American Journal of Orthopaedic Surgery* 11: 336-346.
- Õunpuu S, Davis RB, Walsh HPJ and DeLuca PA (1995): Sagittal plane pelvic motion: relationship to standing pelvic position and clinical measures. *Developmental Medicine and Child Neurology* 37 (Suppl. 73): 25.

- Ounpuu S, DeLuca PA, Davis R and Romness M (2002a): Long-term effects of femoral derotation osteotomies: an evaluation using three-dimensional gait analysis. *Journal of Pediatric Orthopaedics* 22: 139-145.
- Õunpuu S, Lamdan R, Bell K and DeLuca PA (2004): Long-term outcomes of surgical intervention: a comparison of clinical examination measures and related motion data during gait in children with cerebral palsy. *Developmental Medicine and Child Neurology* 46 (Suppl. 99): 43.
- Õunpuu S, Michalak RE, Romness MJ, Bell KJ and DeLuca PA (2002b): The effects of the femoral derotation osteotomy on pelvic motion in children with cerebral palsy spastic hemiplegia. *Gait and Posture* 16: S34-35.
- Õunpuu S, Muik E, Davis RB, Gage JR and DeLuca PA (1993a): 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: 325-330.
- Õunpuu S, Muik E, Davis RB, Gage JR and DeLuca PA (1993b): 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: 331-335.

Oxford-Metrics (1995). Vicon Clinical Manager User's Manual. Oxford: 1-238.

- Palisano R, Rosenbaum P, Walter S, Russell D, Wood E and Galuppi B (1997): Development and reliability of a system to classify gross motor function in children with cerebral palsy. *Developmental Medicine and Child Neurology* 39: 214-223.
- Palisano RJ, Hanna SE, Rosenbaum PL, Russell DJ, Walter SD, Wood EP, Raina PS and Galuppi BE (2000): Validation of a model of gross motor function for children with cerebral palsy. *Physical Therapy* 80: 974-983.
- Palisano RJ, Tieman BL, Walter SD, Bartlett DJ, Rosenbaum PL, Russell D and Hanna SE (2003): Effect of environmental setting on mobility methods of children with cerebral palsy. *Developmental Medicine and Child Neurology* 45: 113-120.

- Patrick JH, Roberts AP and Cole GF (2001): Therapeutic choices in the locomotor management of the child with cerebral palsy- more luck than judgement? *Archives of Disease in Childhood* 85: 275-279.
- Perry J (1987): Distal rectus femoris transfer. *Developmental Medicine and Child Neurology* 29: 153-158.
- Perry J (1992): Gait Analysis Normal and Pathological Function. New Jersey: SLACK Incorporated.
- Perry J, Antonelli D and Ford W (1975): Analysis of knee-joint forces during flexed-knee stance. *The Journal of Bone and Joint Surgery* 57-A: 961-967.
- Perry J, Hoffer MM, Antonelli D, Plut J, Lewis G and Greenberg R (1976): Electromyography before and after surgery for hip deformity in children with cerebral palsy. *The Journal of Bone and Joint Surgery* 58-A: 201-208.
- Perry J, Hoffer MM, Giovan P, Antonelli D and Greenberg R (1974): Gait analysis of the triceps surae in cerebral palsy. *The Journal of Bone and Joint Surgery* 56-A: 511-520.
- Piazza SJ and Delp SL (1996): The influence of muscles on knee flexion during the swing phase of gait. *Journal of Biomechanics* 29: 723-733.
- Pirpiris M, Trivett A, Baker R, Rodda J, Nattrass GR and Graham HK (2003): Femoral derotation osteotomy in spastic diplegia. *The Journal of Bone* and Joint Surgery 85-B: 265-272.
- Pliatsios V, Rodda J, Boyd RN, Starr R, Nattrass GR and Graham HK (1998):
  Validation of the role of clinical gait analysis in children with cerebral palsy: the horizontal and vertical examination are poorly correlated. *Gait and Posture* 8: 78.
- Pollack GA and English TA (1967): Transplantation of the hamstring muscles in cerebral palsy. *The Journal of Bone and Joint Surgery* 49-B: 80-86.
- Portney LG and Watkins MP (1993): Foundations of Clinical Research: Applications to Pratice. Norwalk: Appleton & Lange.

- Portney LG and Watkins MP (2000): Foundations of Clinical Research: Applications to Practice. (2nd ed.) Upper Saddle River: Prentice Hall, Inc.
- Rab GT (1991): External rotation osteotomies for crouch/ internal rotation gait: effect on muscle tension on results. *Developmental Medicine and Child Neurology* 33 (Suppl. 64): 27-28.
- Rab GT (1992a): Diplegic gait: is there more than spasticity? In Sussman MD (Ed.): The Diplegic Child: Evaluation and Management. Rosemont: American Academy of Orthopaedic Surgeons, pp. 99-110.
- Rab GT (1992b): Consensus. In Sussman MD (Ed.): The Diplegic Child: Evaluation and Management. Rosemont: American Academy of Orthopaedic Surgeons, pp. 337-339.
- Rang M (1990): Cerebral Palsy. In Lovell WW and Winter RB (Eds): Pediatric Orthopaedics. (3rd ed.), vol 1. Philadelphia: J.B. Lippincott, pp. 465-506.
- Rang M, Silver R and de la Garza J (1986): Cerebral Palsy. In Lovell WW and Winter RB (Eds): Pediatric Orthopaedics. (2nd ed.), vol 1. Philadelphia: J.B. Lippincott Company, pp. 345-396.
- Rattey TE, Leahey L, Hyndman J, Brown DCS and Gross M (1993): Recurrence after achilles tendon lengthening in cerebral palsy. *Journal* of Pediatric Orthopaedics 13: 184-187.
- Reddihough DS and Collins KJ (2003): The epidemiology and causes of cerebral palsy. *Australian Journal of Physiotherapy* 49: 7-12.
- Reimers J (1973): Static and dynamic problems in spastic cerebral palsy. *The Journal of Bone and Joint Surgery* 55-B: 822-827.
- Reimers J (1974): Contracture of the hamstrings in spastic cerebral palsy. *The Journal of Bone and Joint Surgery* 56-B: 102-109.
- Reimers J (1980): The stability of the hip in children. A radiological study of the results of muscle surgery in cerebral palsy. *Acta Orthopaedica Scandinavica Suppl* 184: 1-100.

- Renshaw TS, Green NE, Griffin PP and Root L (1995): Cerebral palsy: orthopaedic management. *The Journal of Bone and Joint Surgery* 77-A: 1590-1606.
- Rethlefsen S, Kay R, Dennis S, Forstein M and Tolo V (1999a): The effects of fixed and articulated ankle-foot orthoses on gait patterns in subjects with cerebral palsy. *Journal of Pediatric Orthopaedics* 19: 470-474.
- Rethlefsen S, Tolo VT, Reynolds RAK and Kay R (1999b): Outcome of hamstring lengthening and distal rectus femoris transfer surgery. *Journal of Pediatric Orthopaedics Part B* 8: 75-79.
- Riewald SA and Delp SL (1997): The action of the rectus femoris muscle following distal tendon transfer: does it generate knee flexion moment? *Developmental Medicine and Child Neurology* 39: 99-105.
- Roberts WM and Adams JP (1953): The patellar-advancement operation in cerebral palsy. *Journal of Bone and Joint Surgery* 35-A: 958-966.
- Rogers B, Msall M, Owens T, Guernsey K, Brody A, Buck G and Hudak M (1994): Cystic periventricular leukomalacia and type of cerebral palsy in preterm infants. *The Journal of Pediatrics* 125: S1-S8.
- Romei M, Galli M, Motta F, Schwartz M and Crivellini M (2004): Use of the normalcy index for the evaluation of gait pathology. *Gait and Posture* 19: 85-90.
- Roosth HP (1971): Flexion deformity of the hip and knee in spastic cerebral palsy: treatment by early release of the spastic hip-flexor muscles. *The Journal of Bone and Joint Surgery* 53-A: 1489-1510.
- Root L and Siegal T (1980): Osteotomy of the hip in children: posterior approach. *The Journal of Bone and Joint Surgery* 62-A: 571-575.
- Rose J and McGill KC (2005): Neuromuscular activation and motor-unit firing characteristics in cerebral palsy. *Developmental Medicine and Child Neurology* 47: 329-336.
- Rose SA, Õunpuu S and DeLuca PA (1991): Strategies for the assessment of pediatric gait in the clinical setting. *Physical Therapy* 71: 961-980.

- Rosenbaum P (2003): Cerebral palsy: what parents and doctors want to know. *British Medical Journal* 326: 970-974.
- Rosenbaum PL, Walter SD, Hanna SE, Palisano RJ, Russell DJ, Raina P, Wood E, Bartlett DJ and Galuppi BE (2002): Prognosis for gross motor function in cerebral palsy. *The Journal of the American Medical Association* 288: 1357-1363.
- Rosenthal RK and Levine DB (1977): Fragmentation of the distal pole of the patella in spastic cerebral palsy. *The Journal of Bone and Joint Surgery* 59-A: 934-939.
- Ross SA and Engsberg JR (2002): Relation between spasticity and strength in individuals with spastic diplegic cerebral palsy. *Developmental Medicine and Child Neurology* 44: 148-157.
- Russell DJ, Rosenbaum PL, Cadman DT, Gowland C, Hardy S and Jarvis S (1989): The Gross Motor Function Measure: a means to evaluate the effects of physical therapy. *Developmental Medicine and Child Neurology* 31: 341-352.
- Russman BS and Gage JR (1989): Cerebral Palsy. Current Problems in Pediatrics. 19: 69-111.
- Sachs B and Petersen F (1890): A study of cerebral palsies of early life. *J Nerv Ment Dis* 17: 295-332.
- Sandström K, Alinder J and Öberg B (2004): Descriptions of functioning and health and relations to a gross motor classification in adults with cerebral palsy. *Disability and Rehabilitation* 26: 1023-1031.
- Sanger TD, Delgado MR, Gaebler-Spira D, Hallett M and Mink JW (2003): Classification and definition of disorders causing hypertonia in childhood. *Pediatrics* 111: 89-97.
- Saraph V, Zwick EB, Auner C, Schneider F, Steinwender G and Linhart W (2005): Gait improvement surgery in diplegic children. How long do the improvements last? *Journal of Pediatric Orthopaedics* 25: 263-267.

- Saraph V, Zwick EB, Uitz C, Linhart W and Steinwender G (2000): The Baumann procedure for fixed contracture of the gastrosoleus in cerebral palsy. *The Journal of Bone and Joint Surgery* 82-B: 535-540.
- Saraph V, Zwick EB, Zwick G, Steinwender C, 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.
- Saw A, Smith PA, Sirirungruangsarn Y, Chen S, Hassani S, Harris G and Kuo KN (2003): Rectus femoris transfer for children with cerebral palsy: long-term outcome. *Journal of Pediatric Orthopaedics* 23: 672-678.
- Schmidt DJ, Arnold AS, Carroll NC and Delp SL (1999): Length changes of the hamstrings and adductors resulting from derotational osteotomies of the femur. *Journal of Orthopaedic Research* 17: 279-285.
- Schutte LM, Hayden SW and Gage JR (1997): Lengths of hamstrings and psoas muscles during crouch gait: effects of femoral anteversion. *Journal of Orthopaedic Research* 15: 615-621.
- Schutte LM, Narayanan U, Stout JL, Selber P, Gage JR and Schwartz MH (2000): An index for quantifying deviations from normal gait. *Gait and Posture* 11: 25-31.
- Schwartz L, Engel JM and Jensen MP (1999): Pain in persons with cerebral palsy. *Archives of Physical Medicine and Rehabilitation* 80: 1243-1246.
- Schwartz M and Lakin G (2003): The effect of tibial torsion on the dynamic function of the soleus during gait. *Gait and Posture* 17: 113-118.
- Schwartz MH, Novacheck TF and Trost J (2000): A tool for quantifying hip flexor function during gait. *Gait and Posture* 12: 122-127.
- Schwartz MH, Trost JP and Wervey RA (2004): Measurement and management of errors in quantitative gait data. *Gait and Posture* 20: 196-203.
- SCPE (2000): Surveillance of cerebral palsy in Europe: a collaboration of cerebral palsy surveys and registers. *Developmental Medicine and Child Neurology* 42: 816-824.

- Scrutton D (1992): The classification of the cerebral palsies. *Developmental Medicine and Child Neurology* 34: 833.
- Scrutton D (1998): Imprecision? Precisely. *Developmental Medicine and Child Neurology* 40: 75.
- Segal LS, Sienko Thomas SE, Mazur JM and Mauterer M (1989): Calcaneal gait in spastic diplegia after heel cord lengthening: A study with gait analysis. *Journal of Pediatric Orthopaedics* 9: 697-701.
- Selber P, Barbosa A, Kawamura C, Sposati L, Ferraretto I and Dias LS (2000): Physical exam and kinematics of patients with spastic diplegia: Are there any correlation between these sets of data? *Gait and Posture* 11: 136.
- Selber P, Filho ER, Dallalana R, Pirpiris M, Nattrass GR and Graham HK (2004): Supramalleolar derotation osteotomy of the tibia, with T plate fixation. *The Journal of Bone and Joint Surgery* 86-B: 1170-1175.
- Seymour N and Sharrard WJW (1968): Bilateral proximal release of the hamstrings in cerebral palsy. *The Journal of Bone and Joint Surgery* 50-B: 274-277.
- Sheean G (2002): The pathophysiology of spasticity. *European Journal of Neurology* 9 (Suppl. 1): 3-9.
- Shepherd RB (1980): Physiotherapy in Paediatrics. (2nd ed.) London: William Heinemann Medical Books Limited.
- Shevell MI, Majnemer A and Morin I (2003): Etiologic yield of cerebral palsy: a contemporary case series. *Pediatric Neurology* 28: 352-359.
- Shumway-Cook A and Woollacott MH (2001): Motor Control: Theory and Practical Applications. (2nd ed.) Philadelphia: Lippincott Williams & Wilkins.
- Sienko Thomas S, Moore C, Kelp-Lenane C and Norris C (1996): Simulated gait patterns: the resulting effects on gait parameters, dynamic electromyography, joint moments. and physiological cost index. *Gait and Posture* 4: 100-107.

- Silver RL, de la Garza J and Rang M (1985): The myth of muscle balance. *The Journal of Bone and Joint Surgery* 67-B: 432-437.
- Simon SR, Deutsch SD, Nuzzo RM, Mansour MJ, Jackson JL, Koskinen M and Rosenthal RK (1978): Genu recurvatum in spastic cerebral palsy. *The Journal of Bone and Joint Surgery* 60-A: 882-894.
- Skaggs DL, Rethlefsen SA, Kay RM, Dennis SW, Reynolds RAK and Tolo VT (2000): Variability in gait analysis interpretation. *Journal of Pediatric Orthopaedics* 20: 759-764.
- Smiley SJ, Jacobsen FS, Mielke C, Johnston R, Park C and Ovaska GJ (2002): A comparison of the effects of solid, articulated, and posterior leafspring ankle-foot orthoses and shoes alone on gait and energy expenditure in children with spastic diplegic cerebral palsy. *Orthopedics* 25: 411-415.
- Stanley F, Blair E and Alberman E (2000): Cerebral Palsies: Epidemiology and Causal Pathways. London: Mac Keith Press.
- Stansfield BW, Hillman SJ, Hazlewood ME, Lawson AA, Mann AM, Loudon IR and Robb JE (2001a): Normalized speed, not age, characterizes ground reaction force patterns in 5- to 12- year-old children walking at self-selected speeds. *Journal of Pediatric Orthopaedics* 21: 395-402.
- Stansfield BW, Hillman SJ, Hazlewood ME, Lawson AA, Mann AM, Loudon IR and Robb JE (2001b): Sagittal joint kinematics, moments, and powers are predominantly characterized by speed of progression, not age, in normal children. *Journal of Pediatric Orthopaedics* 21: 403-411.
- StataCorp. (2001). Stata Statistical Software: Release 7.0. College Station, TX, Stata Corporation.
- Stefko RM, de Swart RJ, Dodgin DA, Wyatt MP, Kaufman KR, Sutherland DH and Chambers HG (1998): Kinematic and kinetic analysis of distal derotational osteotomy of the leg in children with cerebral palsy. *Journal* of Pediatric Orthopaedics 18: 81-87.

- Steinwender G, Saraph V, Scheiber S, Zwick EB, Uitz C and Hackl K (2000): Intrasubject repeatability of gait analysis data in normal and spastic children. *Clinical Biomechanics* 15: 134-139.
- Steinwender G, Saraph V, Zwick EB, Steinwender C and Linhart W (2001): Hip locomotion mechanisms in cerebral palsy crouch gait. *Gait and Posture* 13: 78-85.
- Sterne JAC and Davey Smith G (2001): Sifting the evidence- what's wrong with significance tests? *British Medical Journal* 322: 226-231.
- Stevens PM, MacWilliams B and Mohr RA (2004): Gait analysis of stapling for genu valgum. *Journal of Pediatric Orthopaedics* 24: 70-74.
- Stevens PM, Maguire M, Dales MD and Robins AJ (1999): Physeal stapling for idiopathic genu valgum. *Journal of Pediatric Orthopaedics* 19: 645-649.
- Stout J, Gage JR, Novacheck TF and Schwartz M (2004): Distal femoral extension osteotomy and patellar tendon advancement for treatment of persistent crouch gait in individuals with cerebral palsy. *Developmental Medicine and Child Neurology* 46 (Suppl. 99): 14.
- Strayer LM (1950): Recession of the gastrocnemius. *The Journal of Bone and Joint Surgery* 32-A: 671-676.
- Sutherland DH (1966): An electromyographic study of the plantar flexors of the ankle in normal walking on the level. *The Journal of Bone and Joint Surgery* 48-A: 66-71.
- Sutherland DH (1984): Gait Disorders in Childhood and Adolescence. Baltimore: Williams & Wilkins.
- Sutherland DH (2002). Personal communication.
- Sutherland DH and Cooper L (1978): The pathomechanics of progressive crouch gait in spastic diplegia. *Orthopedic Clinics of North America* 9: 143-153.
- Sutherland DH, Cooper L and Daniel D (1980): The role of the ankle plantar flexors in normal walking. *The Journal of Bone and Joint Surgery* 62-A: 354-363.

- Sutherland DH and Davids JR (1993): Common gait abnormalities of the knee in cerebral palsy. *Clinical Orthopaedics* 288: 139-147.
- Sutherland DH, Olshen RA, Biden EN and Wyatt MP (1988): The Development of Mature Walking. London: Mac Keith Press.
- Sutherland DH, Santi M and Abel MF (1990): Treatment of stiff-knee gait in cerebral palsy: a comparison by gait analysis of distal rectus femoris transfer versus proximal rectus release. *Journal of Pediatric Orthopaedics* 10: 433-441.
- Sutherland DH, Schottstaedt ER, Larsen LJ, Ashley RK, Callander JN and James PM (1969): Clinical and electromyographic study of seven spastic children with internal rotation gait. *The Journal of Bone and Joint Surgery* 51-A: 1070-1082.
- Sutherland DH, Zilberfarb JL, Kaufman KR, Wyatt MP and Chambers HG (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.
- Thometz J, Simon S and Rosenthal R (1989): The effect on gait of lengthening of the medial hamstrings in cerebral palsy. *The Journal of Bone and Joint Surgery* 71-A: 345-353.
- Thompson NS, Baker RJ, Cosgrove AP, Corry IS and Graham HK (1998): Musculoskeletal modelling in determining the effect of botulinum toxin on the hamstrings of patients with crouch gait. *Developmental Medicine and Child Neurology* 40: 622-625.
- Thompson NS, Baker RJ, Cosgrove AP, Saunders JL and Taylor TC (2001): Relevance of the popliteal angle to hamstring length in cerebral palsy crouch gait. *Journal of Pediatric Orthopaedics* 21: 383-387.
- Topoleski TA, Kurtz CA and Grogan DP (2000): Radiographic abnormalities and clinical symptoms associated with patella alta in ambulatory children with cerebral palsy. *Journal of Pediatric Orthopaedics* 20: 636-639.

- Trost J (2004): Physical assessment and observational gait analysis. In Gage JR (Ed.): The Treatment of Gait Problems in Cerebral Palsy. London: MacKeith Press, pp. 71-89.
- Truscelli D, Lespargot A and Tardieu G (1979): Variation in the long-term results of elongation of the tendo achillis in children with cerebral palsy. *The Journal of Bone and Joint Surgery* 61-B: 466-469.
- Tylkowski CM, Howell-Garvey V and Miller G (1988): The influence of hamstring and hip-flexor musculature on crouch gait in spastic cerebral palsy as determined by gait analysis. *Developmental Medicine and Child Neurology* 30 (Suppl. 57): 14-15.
- Urquhart DM, Hodges PW and Story IH (2005): Postural activity of the abdominal muscles varies between regions of these muscles and between body positions. *Gait and Posture* 22: 295-301.
- van der Linden ML, Aitchison AM, Hazlewood ME, Hillman SJ and Robb JE (2003a): Effects of surgical lengthening of the hamstrings without a concomitant distal rectus femoris transfer in ambulant patients with cerebral palsy. *Journal of Pediatric Orthopaedics* 23: 308-313.
- van der Linden ML, Hazlewood ME, Aitchison AM, Hillman SJ and Robb JE (2003b): Electrical stimulation of gluteus maximus in children with cerebral palsy: effects on gait characteristics and muscle strength. *Developmental Medicine and Child Neurology* 45: 385-390.
- van der Linden ML, Kerr AM, Hazlewood ME, Hillman SJ and Robb JE (2002): Kinematic and kinetic gait characteristics of normal children walking at a range of clinically relevant speeds. *Journal of Pediatric Orthopaedics* 22: 800-806.
- Waters RL, Perry J, McDaniels JM and House K (1974): The relative strength of the hamstrings during hip extension. *The Journal of Bone and Joint Surgery* 56-A: 1592-1597.
- Watkins B, Darrah J and Pain K (1995): Reliability of passive ankle dorsiflexion measurements in children: comparison of universal and biplane goniometers. *Pediatric Physical Therapy* 7: 3-8.

- Westwell M, DeLuca P and Õunpuu S (2004): Effect of repeat hamstring lengthenings in individuals with cerebral palsy. *Developmental Medicine and Child Neurology* 46 (Suppl. 99): 14-15.
- Westwell M, Õunpuu S and Bell K (2005): Effect of upper motion on lower extremity kinetics. In Gait and Clinical Movement Analysis Society Conference Proceedings, Portland, Oregon, pp. 149-150.
- Winders Davis D (1997a): Review of cerebral palsy, Part I: description, incidence, and etiology. *Neonatal Network* 16: 7-11.
- Winders Davis D (1997b): Review of cerebral palsy, Part II: identification and intervention. *Neonatal Network* 16: 19-25.
- Winters TF, Gage JR and Hicks R (1987): Gait patterns in spastic hemiplegia in children and young adults. *The Journal of Bone and Joint Surgery* 69-A: 437-441.
- Wong MA, Simon S and Olshen RA (1983): Statistical analysis of gait patterns of persons with cerebral palsy. *Statistics in Medicine* 2: 345-354.
- 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 and Child Neurology 42: 292-296.
- World Health Organization (2001). International Classification of Functioning, Disability and Health (Short Version). Geneva, World Health Organization: 121-160.
- Wren TAL, Rethlefsen S and Kay RM (2005a): Prevalence of specific gait abnormalities in children with cerebral palsy: influence of cerebral palsy subtype, age and previous surgery. *Journal of Pediatric Orthopaedics* 25: 79-83.
- Wren TAL, Woolf K and Kay RM (2005b): How closely do surgeons follow gait analysis recommendations and why? *Journal of Pediatric Orthopaedics Part B* 14: 202-205.
- Wylie WG (1951): The cerebral palsies in infancy. In Feilin A (Ed.): Modern Trends in Neurology. London: Butterworth, pp. 125-48.

- Yin R, Reddihough DS, Ditchfield MR and Collins KJ (2000): Magnetic resonance imaging findings in cerebral palsy. *Journal of Pediatrics and Child Health* 36: 139-144.
- Yngve DA, Scarborough N, Goode B and Haynes R (2002): Rectus and hamstring surgery in cerebral palsy: a gait analysis study of results by functional ambulation level. *Journal of Pediatric Orthopaedics* 22: 672-676.
- Yokochi K (2001): Gait patterns in children with spastic diplegia and periventricular leukomalacia. *Brain and Development* 23: 34-37.
- Zimmerman MH, Smith CF and Oppenheim WL (1982): Supracondylar femoral extension osteotomies in the treatment of fixed flexion deformity of the knee. *Clinical Orthopaedics and Related Research* 171: 87-93.
- Zwick EB, Saraph V, Linhart W and Steinwender G (2001): Propulsive function during gait in diplegic children: evaluation after surgery for gait improvement. *Journal of Pediatric Orthopaedics Part B* 10: 226-233.
- Zwick EB, Saraph V, Zwick G, Steinwender C, Linhart W and Steinwender G (2002): Medial hamstring lengthening in the presence of hip flexor tightness in spastic diplegia. *Gait and Posture* 16: 288-296.

## **APPENDIX A**

## Gross Motor Function Classification System for Cerebral Palsy

# Robert Palisano, Peter Rosenbaum, Stephen Walter, Dianne Russell, Ellen Wood, Barbara Galuppi

## Introduction & User Instructions

The Gross Motor Function Classification System for cerebral palsy is based on selfinitiated movement with particular emphasis on sitting (truncal control) and walking. When defining a 5 level Classification System, our primary criterion was that the distinctions in motor function between levels must be clinically meaningful. Distinctions between levels of motor function are based on functional limitations, the need for assistive technology, including mobility devices (such as walkers, crutches, and canes) and wheeled mobility, and to much lesser extent quality of movement. Level I includes children with neuromotor impairments whose functional limitations are less than what is typically associated with cerebral palsy, and children who have traditionally been diagnosed as having "minimal brain dysfunction" or "cerebral palsy of minimal severity". The distinctions between Levels I and II therefore are not as pronounced as the distinctions between the other Levels, particularly for infants less than 2 years of age.

The focus is on determining which level best represents the child's present abilities and limitations in motor function. Emphasis is on the child's usual performance in home, school, and community settings. It is therefore important to classify on ordinary performance (not best capacity), and not to include judgments about prognosis. Remember the purpose is to classify a child's present gross motor function, not to judge quality of movement or potential for improvement.

The descriptions of the 5 levels are broad and are not intended to describe all aspects of the function of individual children. For example, an infant with hemiplegia who is unable to crawl on hands and knees, but otherwise fits the description of Level I, would be classified in Level I. The scale is ordinal, with no intent that the distances between levels be considered equal or that children with cerebral palsy are equally distributed among the 5 levels. A summary of the distinctions between each pair of levels is provided to assist in determining the level that most closely resembles a child's current gross motor function.

The title for each level represents the highest level of mobility that a child is expected to achieve between 6-12 years of age. We recognize that classification of motor function is dependent on age, especially during infancy and early childhood. For each level, therefore, separate descriptions are provided for children in several age bands. The functional abilities and limitations for each age interval are intended to serve as guidelines, are not comprehensive, and are not norms. Children below age 2 should be considered at their corrected age if they were premature.

An effort has been made to emphasize children's function rather than their limitations. Thus as a general principle, the gross motor function of children who are able to perform the functions described in any particular level will probably be classified at or above that level; in contrast the gross motor functions of children who cannot perform the functions of a particular level will likely be classified below that level.

Reference: Dev Med Child Neurol 1997;39:214-223 © 1997 *CanChild* Centre for Childhood Disability Research (formerly NCRU)

## Gross Motor Function Classification System for Cerebral Palsy (GMFCS)

#### Before 2nd Birthday

- Level I Infants move in and out of sitting and floor sit with both hands free to manipulate objects. Infants crawl on hands and knees, pull to stand and take steps holding on to furniture. Infants walk between 18 months and 2 years of age without the need for any assistive mobility device.
- Level II Infants maintain floor sitting but may need to use their hands for support to maintain balance. Infants creep on their stomach or crawl on hands and knees. Infants may pull to stand and take steps holding on to furniture.
- Level III Infants maintain floor sitting when the low back is supported. Infants roll and creep forward on their stomachs.
- Level IV Infants have head control but trunk support is required for floor sitting. Infants can roll to supine and may roll to prone.
- Level V Physical impairments limit voluntary control of movement. Infants are unable to maintain antigravity head and trunk postures in prone and sitting. Infants require adult assistance to roll.

#### Between 2nd and 4th Birthday

- Level I Children floor sit with both hands free to manipulate objects. Movements in and out of floor sitting and standing are performed without adult assistance. Children walk as the preferred method of mobility without the need for any assistive mobility device.
- Level II Children floor sit but may have difficulty with balance when both hands are free to manipulate objects. Movements in and out of sitting are performed without adult assistance. Children pull to stand on a stable surface. Children crawl on hands and knees with a reciprocal pattern, cruise holding onto furniture and walk using an assistive mobility device as preferred methods of mobility.
- Level III Children maintain floor sitting often by "W-sitting" (sitting between flexed and internally rotated hips and knees) and may require adult assistance to assume sitting. Children creep on their stomach or crawl on hands and knees (often without reciprocal leg movements) as their primary methods of selfmobility. Children may pull to stand on a stable surface and cruise short distances. Children may walk short distances indoors using an assistive mobility device and adult assistance for steering and turning.
- Level IV Children sit on a chair but need adaptive seating for trunk control and to maximize hand function. Children move in and out of chair sitting with assistance from an adult or a stable surface to push or pull up on with their arms. Children may at best walk short distances with a walker and adult supervision but have difficulty turning and maintaining balance on uneven surfaces. Children are transported in the community. Children may achieve self-mobility using a power wheelchair.
- Level V Physical impairments restrict voluntary control of movement and the ability to maintain antigravity head and trunk postures. All areas of motor function are limited. Functional limitations in sitting and standing are not fully compensated for through the use of adaptive equipment and assistive technology. At Level V, children have no means of independent mobility and are transported. Some children achieve self-mobility using a power wheelchair with extensive adaptations.

#### Between 4th and 6th Birthday

- Level I Children get into and out of, and sit in, a chair without the need for hand support. Children move from the floor and from chair sitting to standing without the need for objects for support. Children walk indoors and outdoors, and climb stairs. Emerging ability to run and jump.
- Level II Children sit in a chair with both hands free to manipulate objects. Children move from the floor to standing and from chair sitting to standing but often require a stable surface to push or pull up on with their arms. Children walk without the need for any assistive mobility device indoors and for short distances on level surfaces outdoors. Children climb stairs holding onto a railing but are unable to run or jump.
- Level III Children sit on a regular chair but may require pelvic or trunk support to maximize hand function.Children move in and out of chair sitting using a stable surface to push on or pull up with their arms. Children walk with an assistive mobility device on level surfaces and climb stairs with assistance from an adult. Children frequently are transported when travelling for long distances or outdoors on uneven terrain.
- Level IV Children sit on a chair but need adaptive seating for trunk control and to maximize hand

function. Children move in and out of chair sitting with assistance from an adult or a stable surface to push or pull up on with their arms. Children may at best walk short distances with a walker and adult supervision but have difficulty turning and maintaining balance on uneven surfaces. Children are transported in the community. Children may achieve self-mobility using a power wheelchair.

Level V Physical impairments restrict voluntary control of movement and the ability to maintain antigravity head and trunk postures. All areas of motor function are limited. Functional limitations in sitting and standing are not fully compensated for through the use of adaptive equipment and assistive technology. At Level V, children have no means of independent mobility and are transported. Some children achieve self-mobility using a power wheelchair with extensive adaptations.

#### Between 6th and 12th Birthday

- Level I Children walk indoors and outdoors, and climb stairs without limitations. Children perform gross motor skills including running and jumping but speed, balance, and coordination are reduced.
- Level II Children walk indoors and outdoors, and climb stairs holding onto a railing but experience limitations walking on uneven surfaces and inclines, and walking in crowds or confined spaces. Children have at best only minimal ability to perform gross motor skills such as running and jumping.
- Level III Children walk indoors or outdoors on a level surface with an assistive mobility device. Children may climb stairs holding onto a railing. Depending on upper limb function, children propel a wheelchair manually or are transported when travelling for long distances or outdoors on uneven terrain.
- Level IV Children may maintain levels of function achieved before age 6 or rely more on wheeled mobility at home, school, and in the community. Children may achieve self-mobility using a power wheelchair.
- Level V Physical impairments restrict voluntary control of movement and the ability to maintain antigravity head and trunk postures. All areas of motor function are limited. Functional limitations in sitting and standing are not fully compensated for through the use of adaptive equipment and assistive technology. At level V, children have no means of independent mobility and are transported. Some children achieve self-mobility using a power wheelchair with extensive adaptations.

#### **Distinctions Between Levels I and II**

Compared with children in Level I, children in Level II have limitations in the ease of performing movement transitions; walking outdoors and in the community; the need for assistive mobility devices when beginning to walk; quality of movement; and the ability to perform gross motor skills such as running and jumping.

#### **Distinctions Between Levels II and III**

Differences are seen in the degree of achievement of functional mobility. Children in Level III need assistive mobility devices and frequently orthoses to walk, while children in Level II do not require assistive mobility devices after age 4.

#### **Distinctions Between Level III and IV**

Differences in sitting ability and mobility exist, even allowing for extensive use of assistive technology. Children in Level III sit independently, have independent floor mobility, and walk with assistive mobility devices. Children in Level IV function in sitting (usually supported) but independent mobility is very limited. Children in Level IV are more likely to be transported or use power mobility.

#### Distinctions Between Levels IV and V

Children in Level V lack independence even in basic antigravity postural control. Selfmobility is achieved only if the child can learn how to operate an electrically powered wheelchair. This work has been supported in part by the Easter Seal Research Institute and the National Health Research and Development Program.

Distribution of the Gross Motor Function Classification System for Cerebral Palsy has been made possible by a grant from the United Cerebral Palsy Research and Educational Foundation, USA.

Want to know more? Contact: Institute for Applied Health Sciences, McMaster University 1400 Main Street West, Rm. 408, Hamilton, ON, Canada L8S 1C7 Tel: 905-525-9140 Ext. 27850 Fax: 905-522-6095 E-mail: canchild@mcmaster.ca Website: www.fhs.mcmaster.ca/canchild

## **APPENDIX B**

#### Gillette Functional Assessment Questionnaire: Functional Walking Scale

Choose the **one** answer below that best describes your child's typical walking ability (with the use of any needed assistive devices).

- 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 a routine basis
- Walks for exercise in therapy and less than typical household distances. Usually requires assistance from another person
- 4. Walks for household distances, but makes slow progress. Does not use walking at home as preferred mobility (primarily walks in therapy)
- 5. Walks more than15-50 feet but only inside at home or school (walks for household distances)
- 6. Walks more than 15-50 feet outside the home, but usually uses a wheelchair or stroller for community distances or in congested areas
- 7. Walks outside the home 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 perform 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 with running, climbing, and/or stairs
- \_\_\_\_ 10. Walks, runs, and climbs on level and uneven terrain without difficulty or assistance

Appendix B

## **APPENDIX C**

#### The Functional Mobility Scale (FMS)

Please rate the child's usual walking ability for each of the distances listed below. Please write, in the space provided, the number that best describes the child's ability or need for assistance, at each of the distances listed.

- 1. Uses wheelchair, stroller or buggy: May stand for transfers and may do some stepping supported by another person or using a walker/ frame
- 2. Uses K-Walker or other walking frame: without help from another person
- 3. Uses two crutches: without help from another person
- 4. Uses one crutch or two sticks: without help from another person
- 5. Independent on level surfaces: does not use walking aids or need help from another person. If uses furniture, walls, fences, shop fronts for support please use 4 as the appropriate description
- **6.** Independent on all surfaces: does not use any walking aids or need help from another person when walking, running, climbing and climbing stairs

Walking Distance	Rating (1-6)
Walking 5 metres (eg, in bedroom or other room)	
Walking 50 metres (eg, at school, in the classroom and playground)	
Walking 500 metres (eg, in shopping mall or street)	

Appendix C



Flemington Road, Parkville Victoria, Australia, 3052

Telephone (03) 9345 5522 ISD (+613) 9345 5522 Facsimile (03) 9345 5789

876.50

17 November 2004

To whom it may concern,

Concerning the research submitted by Jill Rodda as an applicant for her Ph.D thesis entitled "Severe crouch gait in spastic diplegia: impact of single event multilevel surgery on sagittal plane biomechanics and functional ability".

This research was approved as audit by the appropriate divisional director at the Royal Children's Hospital and therefore is recognised as having been conducted with the appropriate compliance with ethical requirements.

Arnold Smith Chair, RCH EHRC

Appendix D



### Royal Children's Hospital, Melbourne

#### CLINICAL AUDIT APPROVAL

a fill a start a start was the start of the st

REF NO: AUD/2001-

PROJECT TITLE: Severe crouch gait in spastic diplegia: impact of single event multilevel surgery on sagittal plane biomechanics and functional ability.

INVESTIGATOR(S):

Jillian Rodda BappSc(PT) Professor Kerr Graham MD, FRCS Ed., FRACS Dr Richard Baker PhD Dr Mary Galéa PhD

DATE OF APPROVAL: 

In my opinion, this project involves audit only, and does not require consideration by the RCH Ethics in Human Research Committee.

SURGER DIVISION: .....

DIRECTOR: J. PALMINGTON (Name)

SIGNED:

Please send copy of Audit form to Ethics and Training Office 1st Floor Main Building, RCH.

A Member of Women's & Children's Health Flemington Road Parkville Victoria 3052 Australia Telephone 03) 9345 5522 Facsimile 03) 9345 5789 http://www.rch.org.au

> Revised 11/10/04 Stock No. 002378

## **University Library**



## A gateway to Melbourne's research publications

Minerva Access is the Institutional Repository of The University of Melbourne

Author/s: Rodda, Jillian Maree

## Title:

Severe crouch gait in the sagittal gait patterns of spastic diplegic cerebral palsy: the impact of single event multilevel surgery

## Date:

2005

## Citation:

Rodda, J. M. (2006). Severe crouch gait in the sagittal gait patterns of spastic diplegic cerebral palsy: the impact of single event multilevel surgery. PhD thesis, Physiotherapy, The University of Melbourne.

Publication Status:

Published

Persistent Link: http://hdl.handle.net/11343/39191

## File Description:

Severe crouch gait in the sagittal gait patterns of spastic diplegic cerebral palsy:the impact of single event multilevel surgery

## Terms and Conditions:

Terms and Conditions: Copyright in works deposited in Minerva Access is retained by the copyright owner. The work may not be altered without permission from the copyright owner. Readers may only download, print and save electronic copies of whole works for their own personal non-commercial use. Any use that exceeds these limits requires permission from the copyright owner. Attribution is essential when quoting or paraphrasing from these works.