



UNIVERSITEIT VAN PRETORIA  
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**THE EFFECT OF SOFT TISSUE MOBILIZATION  
TECHNIQUES ON THE SYMPTOMS OF CHRONIC  
POSTERIOR COMPARTMENT SYNDROME IN RUNNERS: A  
MULTIPLE CASE STUDY APPROACH**

by

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## ABSTRACT

<b>TITLE</b>	<b>The effect of soft tissue mobilization techniques on the symptoms of chronic posterior compartment syndrome in runners: A multiple case study approach.</b>
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Chronic posterior compartment syndrome (CPCS) of the leg is a pathological condition which is often encountered by participants in exercise related activities such as running. To date no successful conservative treatment approach existed for the condition. The mainstay of the management of the condition at present is the surgical release of the involved fascia that surrounds the compartment. The main aim of the research project was thus to develop a successful conservative treatment approach for the symptoms of CPCS. It was identified that the current theoretical base did not incorporate the continuous and relatively inelastic nature of the fascia which plays an important role in the condition. Based on an extended literature review, muscles which are linked to the posterior compartment via the myofascial tissue were identified. Tightness in these *clinically significant* muscles is able to induce stresses in the myofascial chain which could ultimately influence stresses in the posterior compartment of the leg. The release of tightness in these muscles external to the posterior compartment through soft tissue mobilization techniques provides an effective conservative treatment approach for the symptoms of CPCS. A revised model for the pathogenesis of CPCS was developed which formed the basis for treatment interventions. The revised theoretical model for the pathogenesis of CPCS was validated based on a mixed-methodological approach which included a series of exploratory as well as explanatory case studies. This qualitative approach was supplemented by quantitative experiments in which the causal relationships of the condition on certain biomechanical aspects were explored. The treatment



interventions had a hundred percent success rate and the results of the experimental research conducted also supports the new theoretical model for the pathogenesis of CPCS.

***Key words:***

*Chronic Posterior Compartment Syndrome; Pathogenesis; Fascia; Soft tissue myofascial links; Soft tissue mobilization techniques; Conservative interventions; Connective tissue; Running injuries; Qualitative research paradigms; Mixed-methodologies; Alternatives to surgical management.*



## OPSOMMING

<b>TITEL</b>	<b>Die invloed van sagteweefsel mobiliseringstegnieke op die simptome van kroniese posterior kompartementsindroom in hardlopers: ‘n Meervoudige gevallestudie benadering</b>
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Kroniese posterior kompartementsindroom (KPKS) van die onderbeen is ‘n patologiese toestand wat ervaar word deur persone wat aan oefeningsverwante aktiwiteite soos hardloop deelneem. Daar bestaan tans geen suksesvolle konserwatiewe behandeling vir die sindroom nie. Die enigste huidige aanbevole behandeling is die chirurgiese loslating of verwydering van die fascia rondom die simptomatiese kompartement. Die hoofdoel van hierdie studie was dus om ‘n suksesvolle konserwatiewe behandelingsregime vir die behandeling van die simptome van KPKS te ontwikkel. Daar is bevind dat die huidige teoretiese grondslag vir die behandeling van die toestand nie die kontinuïteit en die onelastisiteit van die fascia netwerk, wat ‘n groot rol in die sindroom speel, in ag neem nie. Spiere wat via die fascia netwerk aan mekaar en sodoende aan die posterior kompartement van die onderbeen gekoppel is, is deur middel van ‘n intensiewe literatuursoektoeg geïdentifiseer. Hierdie spiere is die *klinies belangrike spiere* genoem en ‘n styfheid in enige een van hierdie spiere is teoreties dus in staat om kragte in die miofasiale ketting te induseer wat dan weer die kragte op die posterior kompartement oordra. Die loslating van styfheid in hierdie spiere ekstern tot die posterior kompartement deur middel van sagteweefsel mobilisatietegnieke, voorsien ‘n effektiewe konserwatiewe benadering tot die behandeling van die simptome van KPKS. ‘n Hersiene model wat die patologie van KPKS visueel voorstel is ontwikkel en dien as basis vir die konserwatiewe behandeling van die simptome van KPKS. Hierdie model is bevestig deur gebruik te maak van ‘n gemengde metodologiese benadering wat ‘n reeks van ondersoekende sowel as verduidelikende gevallestudies ingesluit het. Die



kwalitatiewe benadering was aangevul met kwantitatiewe eksperimente waartydens oorsaaklike verwantskappe met biomeganiese faktore ondersoek was. Die behandelingsbenadering was 'n honderd persent suksesvol en die resultate van die eksperimentele navorsing wat uitgevoer was, ondersteun dus die nuwe teoretiese model vir die patogenese van KPKS.

***Sleutelwoorde:***

*Kroniese posterior kompartementsindroom; Patogenese; Fasia; Sagteweefsel mobilisasies; Miofasiale ketting; Sagteweefsel mobilisasietegnieke; Konserwatiewe behandeling; Kollageen weefsel; Hardloopbeserings; Kwalitatiewe navorsings paradigmas; Gemengde metodologie; Alternatiewe tot chirurgiese behandeling*



## TABLE OF CONTENTS

ACKNOWLEDGEMENTS	ii
ABSTRACT	iii
OPSOMMING	v
TABLE OF CONTENTS	vii
LIST OF FIGURES	xvi
LIST OF TABLES	xviii
LIST OF ABBREVIATIONS	xxv
GLOSSARY OF TERMS	xxvi

### CHAPTER 1: THE SCOPE OF THE RESEARCH

1.1. INTRODUCTION AND BACKGROUND	1
1.2. THE RESEARCH PROBLEM	2
1.3. THE RESEARCH QUESTION	4
1.4. INVESTIGATIVE QUESTIONS	5
1.5. KEY RESEARCH OBJECTIVES	6
1.6. SIGNIFICANCE OF THE RESEARCH	6
1.7. THE RESEARCH PROCESS	7
1.7.1. Phase 1 – Exploratory research	7
1.7.2. Phase 2 – Explanatory research	9
1.8. THE RESEARCH DESIGN AND METHODOLOGY	11
1.9. RESEARCH ASSUMPTIONS	12
1.10. RESEARCH CONSTRAINTS	13
1.11. CONTEXTUAL BOUNDARIES	13
1.11.1. Limitations	13
1.11.2. Delimitations	14
1.12. CONCLUSION	14

### CHAPTER 2: LITERATURE REVIEW

2.1. INTRODUCTION	16
2.2. RESEARCH METHODOLOGY	16
2.2.1. An introduction to qualitative research	16



2.2.2.	Mixed methodologies	18
2.2.3.	Case study research methodology	19
2.2.3.1.	General approach to case study design	20
2.2.3.2.	Components of research design	21
2.2.3.3.	The role of theory in design	24
2.2.3.4.	Criteria for judging the quality of research designs	24
2.2.3.5.	Case study designs	27
2.2.3.6.	Dimension and classification of case studies	32
2.2.3.7.	Protocol	33
2.2.3.8.	Synthesis and knowledge integration in imbedded case study research	36
2.2.3.9.	Case study protocol as design	41
2.2.4.	Experimental study designs	45
2.2.5.	Conclusion	47
2.3.	<b>CHRONIC POSTERIOR COMPARTMENT SYNDROME</b>	47
2.3.1.	Introduction	47
2.3.2.	Chronic compartment syndrome	48
2.3.2.1.	Anatomy of compartments and their muscles	48
2.3.2.2.	Definition of chronic posterior compartment syndrome (CPCS)	49
2.3.2.3.	Pathogenesis of chronic posterior syndrome (CPCS)	52
2.3.2.4.	Prevalence of compartment syndrome	54
2.3.2.5.	Symptoms of chronic posterior compartment syndrome	55
2.3.2.6.	The differentiation of CPCS from other symptom related conditions	56
2.3.2.7.	Intra-compartmental pressure measurement as confirmation of the diagnosis of CPCS	61
2.3.2.8.	Surgical management of chronic compartment syndrome	63
2.3.2.9.	Conservative management of chronic compartment syndrome	69
2.3.3.	Functional anatomy and biomechanical factors	73
2.3.3.1.	Muscles of the posterior compartments of the lower leg	74
2.3.3.2.	Muscles of the anterior compartments of the lower leg	77
2.3.3.3.	Muscles of the lateral compartments of the lower leg	78





2.3.3.4. Normal running gait	79
2.3.3.5. Dorsi flexion at the ankle joint	84
2.4. KNOWLEDGE OF FASCIA	85
2.4.1. Composition of normal fascia	85
2.4.2. Manual therapy techniques for connective tissue dysfunction	88
2.4.3. Reflection	90
2.5. THE CONTINUITY OF THE SOFT TISSUE LINKS	92
2.5.1. Introduction	92
2.5.2. Description of the myofascial links	93
2.5.3. Posterior myofascial links of the trunk	94
2.5.4. Anterior myofascial links of the trunk	97
2.5.5. Fascia of the leg	100
2.5.6. Conclusion	101
<b>CHAPTER 3: METHODOLOGY</b>	
3.1. INTRODUCTION	103
3.2. EXPLORATORY RESEARCH DESIGN	103
3.2.1. The basis for exploratory research design	104
3.2.2. Design Classification	105
3.2.3. Unit of Analysis	106
3.2.4. Subjects	106
3.2.4.1. Inclusion criteria	106
3.2.4.2. Exclusion criteria	106
3.2.4.3. Recruitment of subjects	107
3.2.4.4. Randomness	108
3.2.5. Database	108
3.2.6. Ethical considerations	108
3.2.7. The research question	109
3.2.8. Investigative questions	109
3.2.9. Theoretical framework	110
3.2.10. Propositions	111
3.2.11. Rival theories	111
3.2.12. Data collection	111



3.2.13. Schedule and reviews	112
3.2.14. Criteria for interpreting results	112
3.2.15. Variables and associated measures	112
3.2.16. Research Process	113
3.2.16.1. Subjective assessment – Interview	113
3.2.16.2. Objective Assessment – Physical examination	114
3.2.17. Intervention	116
3.2.18. Data recording	119
3.2.19. Quality assurance measures	120
3.2.20. Modification of the exploratory research design	121
3.2.21. Conclusion	122
3.3. THE EXPLANATORY RESEARCH DESIGN	122
3.3.1. The research question	123
3.3.2. Theoretical framework	123
3.3.3. Propositions	123
3.3.4. Rival theories	124
3.3.5. The Research Process	125
3.3.6. Conclusion	125
3.4. EXPERIMENTAL RESEARCH	126
3.4.1. Introduction	126
3.4.2. Degree of extension at the metatarsophalangeal joint during terminal stance	126
3.4.2.1. Aim of the study	126
3.4.2.2. Hypothesis	126
3.4.2.3. Research design	127
3.4.2.4. Sample selection	127
3.4.2.5. Inclusion and exclusion criteria for the controls	127
3.4.2.6. Inclusion and exclusion criteria for the subjects	128
3.4.2.7. Outcome measures	128
3.4.2.8. Procedure	128
3.4.3. The effect of soft tissue mobilization on subtalar over pronation in sportsmen	129
3.4.3.1. Introduction	129



3.4.3.2. Aim of the study	129
3.4.3.3. Hypothesis	130
3.4.3.4. Experimental design	131
3.4.3.5. Subjects	131
3.4.3.6. Procedure	132
3.4.3.7. Data management and analysis	133

## **CHAPTER 4: RESEARCH RESULTS**

4.1. INTRODUCTION	135
4.2. EXPLORATORY RESEARCH PHASE	135
4.2.1. Introduction to the exploratory research	135
4.2.2. CASE STUDY 1	135
4.2.2.1. Introduction	135
4.2.2.2. Subject	136
4.2.2.3. The research question	136
4.2.2.4. Theoretical framework	137
4.2.2.5. Propositions	138
4.2.2.6. Rival theories	138
4.2.2.7. Schedule and reviews	138
4.2.2.8. Criteria for interpreting results	138
4.2.2.9. The research procedure	139
4.2.2.10. Interventions and data recording	143
4.2.2.11. Conclusion	154
4.2.2.12. Knowledge integration	154
4.2.3. CASE STUDY 2	156
4.2.3.1. Introduction	156
4.2.3.2. Subject	156
4.2.3.3. The research question	156
4.2.3.4. Theoretical framework	157
4.2.3.5. Propositions	157
4.2.3.6. Rival theories	157
4.2.3.7. Criteria for interpreting results	157
4.2.3.8. The Research Procedure	157



4.2.3.9. Intervention and data recording	163
4.2.4. CASE STUDY 3	167
4.2.4.1. Introduction	167
4.2.4.2. Subject	168
4.2.4.3. The research question	168
4.2.4.4. Theoretical framework	168
4.2.4.5. Propositions	169
4.2.4.6. Rival theories	169
4.2.4.7. The Research Procedure	170
4.2.4.8. Interventions and data recording	174
4.2.4.9. Integration	178
4.2.5. Conclusion	180
4.3. THE DEVELOPMENT OF THE CONCEPT OF CLINICALLY SIGNIFICANT MUSCLES	181
4.3.1. Introduction	181
4.2.2. Myofascial links and clinically significant muscles	182
4.2.3. Significance of the myofascial links	182
4.2.4. Conclusion	185
4.4. THE REVISED THEORETICAL FRAMEWORK	186
4.4.1. Introduction	186
4.4.2. The current theoretical model	186
4.4.3. The theoretical gap	188
4.4.4. A revised model for the pathogenesis of CPCS	189
4.4.5. Rationale for the treatment approach	193
4.4.6. Conclusion	193
4.5. EXPLANATORY RESEARCH RESULTS	194
4.5.1 INTRODUCTION	194
4.5.2 CASE STUDY 4	196
4.5.2.1 The subject	196
4.5.2.2 Research Procedure	196
4.5.2.3 Interventions	205
4.5.2.4 Synthesis/discussion	212



4.5.3	CASE STUDY 5	213
4.5.3.1	The subject	213
4.5.3.2	Research Procedure	213
4.5.3.3	Interventions	220
4.5.3.4	Synthesis/discussion	225
4.5.3.5	Quality assurance measures	226
4.5.4	CASE STUDY 6	226
4.5.4.1	The subject	226
4.5.4.2	Research Procedure	227
4.5.4.3	Interventions	234
4.5.4.4	Synthesis/discussion	239
4.6	CROSS CASE STUDY COMPARISON	241
4.6.1.	Introduction	241
4.6.2.	The symptoms prior and post intervention	244
4.6.3.	Tightness in clinically significant muscles	245
4.6.3.1.	Relative tightness on the left	246
4.6.3.2.	Relative tightness on the right	245
4.6.3.3.	Summary	253
4.6.4.	Assessment of other abnormalities	253
4.6.4.1.	Muscle imbalances	253
4.6.4.2.	Running movement pattern abnormalities	260
4.6.5.	Conclusion	275
4.7.	EXPERIMENTAL RESEARCH	277
4.7.1.	Introduction	277
4.7.2.	Degree of extension at the metatarsophalangeal joint during terminal stance	277
4.7.2.1.	Results	277
4.7.2.2.	Discussion	277
4.7.3.	The effect soft tissue mobilization on subtalar over pronation	282
4.7.3.1.	Results	282
4.7.3.2.	Discussion	283
4.7.4.	Conclusion	284



4.8.	RESEARCH VALIDATION	284
4.8.1	CASE STUDY 7	284
4.8.1.1	The subject	284
4.8.1.2	Research Procedure	285
4.8.1.3	Interventions	294
4.8.1.4	Synthesis/discussion	299
4.8.2	CASE STUDY 8	299
4.8.2.1	The subject	299
4.8.2.2	Research Procedure	300
4.8.2.3	Interventions	305
4.8.2.4	Synthesis/discussion	311
4.8.3	Reflection	311
4.8.3.1	Knowledge from the literature study	312
4.8.3.2	Knowledge gained from experimentation	313
4.8.4	Validation of tightness assessment technique	314

## **CHAPTER 5: CONCLUSIONS AND RECOMMENDATIONS**

5.1	INTRODUCTION	318
5.2	KNOWLEDGE INTEGRATION	319
5.2.1.	Replication of results	319
5.2.2.	Muscle tightness	320
5.2.3.	Muscle imbalances	320
5.2.4.	Gait analysis	321
5.2.5.	Biomechanical measures	321
5.3.	THE RESEARCH PROBLEM	322
5.4.	THE RESEARCH AND INVESTIGATIVE QUESTIONS	323
5.5.	KEY RESEARCH OBJECTIVES	325
5.6.	SIGNIFICANCE OF THE RESEARCH	326
5.7.	THE RESEARCH PROCESS	326
5.8.	THE RESEARCH DESIGN AND METHODOLOGY	328
5.9.	CONTEXTUAL BOUNDARIES AND SHORTCOMINGS	328
5.10.	RECOMMENDATIONS	330
	REFERENCES	331



APPENDICES	344
APPENDIX 1: STUDENT PROTOCOL AND THE ETHICAL COMMITTEE	345
APPENDIX 2: PATIENT INFORMED CONSENT	346
APPENDIX 3: E-MAIL FOR THE RECRUITMENT OF SUBJECTS	351



## LIST OF FIGURES

Figure 1.1	The research process – Phase 1	8
Figure 1.2	The research process – Phase 2	10
Figure 2.1	Theory of knowledge	36
Figure 2.2	Brunswikian Lens Model	40
Figure 2.3	Proposed pathogenesis of CPCS	54
Figure 2.4	Rotation of the Achilles tendon	77
Figure 2.5	The thoraco-dorsal fascia and its links	95
Figure 2.6	The gluteus maximus muscle and its fascia links	96
Figure 2.7	The continuity of the myofascial links through the sacroiliac ligament	96
Figure 2.8	The myofascial links connecting to the sacrotuberous ligament	97
Figure 2.9	An illustration of some of the anterior myofascial links	97
Figure 2.10	Examples of anterior and posterior trunk fascia links to one another	99
Figure 3.1	Pathogenesis of CPCS	110
Figure 3.2	Anatomy of the compartments of the lower leg	116
Figure 3.3	Mobilization of the posterior inter-muscular septum	117
Figure 3.4	Soft tissue mobilization of the deep transverse fascia	117
Figure 3.5	Mobilization of the fascia between the soleus- and gastrocnemius muscles	118
Figure 3.6	Modification of exploratory designs	121
Figure 3.7	Proposed modified model for the pathogenesis of CPCS	124
Figure 3.8	Fascial stress induced pronation	130
Figure 3.9	Measurement by means of Coral Draw software	132
Figure 4.1	Pathogenesis of CPCS	137
Figure 4.2	The chronological development of Case Study 1	154





Figure 4.3	Modified theoretical framework (Case Study 1)	155
Figure 4.4	The evolved theoretical framework	169
Figure 4.5	A summary of the different posterior myofascial links	183
Figure 4.6	A summary of the different anterior myofascial links	184
Figure 4.7	Posterior compartments	188
Figure 4.8	Induced stresses in posterior compartments	191
Figure 4.9	Modified model for the pathogenesis of CPCS	192
Figure 4.10	Progressive change from <i>Exploration</i> to <i>Explanation</i>	242
Figure 4.11	Correlations between peak torque performance pre- and post intervention	258
Figure 4.12	Correlation between work performed per repetition pre- and post intervention	260
Figure 4.13	Metatarsophalangeal extension	278
Figure 4.14	Improvement of the metatarsophalangeal extension: left leg	280
Figure 4.15	Improvement of the metatarsophalangeal extension: right leg	281
Figure 4.16	Normalisation of hind foot pronation	283
Figure 5.1	Towards a new conception of the pathogenesis of CPCS	318



## LIST OF TABLES

Table 2.1	Tactics for ensuring quality research designs	24
Table 2.2	Different kinds of rival explanations	26
Table 2.3	Basic types of designs for case studies	30
Table 2.4	Dimensions and classifications of case studies	32
Table 2.5	Case study protocol conformance matrix	43
Table 2.6	Summary of the anatomy of the compartments of the lower leg	50
Table 2.7	Summary of the occurrence of chronic compartment syndrome	55
Table 2.8(a)	Outcomes of surgical interventions during 1983 to 1998	67
Table 2.8(b)	Outcomes of surgical interventions during 1998 to 2002	68
Table 2.9	Sequence of movement at the pelvis, hips, knees, ankles and feet during running	80
Table 3.1	Design classification of Case Study 1	105
Table 3.2	Case study questions	109
Table 3.3	Case study variables and associated measures	113
Table 3.4	Quality assurance measures	120
Table 3.5	Dimensions and classifications of the experiment	127
Table 4.1	Outcomes of running gait analysis for Case Study 1 prior to intervention	142
Table 4.2	Muscle strength results prior to intervention: Case Study 2	158
Table 4.3	Outcomes of running gait analysis for Case Study 2 prior to intervention	161
Table 4.4	Biomechanical angles: Case Study 2 during intervention	162
Table 4.5	Muscle strength results prior to intervention: Case Study 3	171
Table 4.6	Outcomes of the running gait analysis for Case Study 3 prior to intervention	172
Table 4.7	Biomechanical angles for Case Study 3 during intervention	173
Table 4.8	The intensity of pain/ discomfort before and after intervention	179
Table 4.9	The distance run before commencement of symptoms	179
Table 4.10	The total weekly-distances run before and after intervention.	179



Table 4.11	Muscle imbalances in peak torque performance prior to interventions	180
Table 4.12	A summary of the chronological progression of case studies 1 to 3	181
Table 4.13	Clinically significant muscles	185
Table 4.14	Isokinetic dynamometer test results* prior to intervention: Case Study 4	200
Table 4.15 (a)	Running gait analysis: Case Study 4 prior to intervention (Upper body)	201
Table 4.15 (b)	Running gait analysis: Case Study 4 prior to intervention (Lower body)	202
Table 4.16	Biomechanical angles: Case Study 4	203
Table 4.17	Tightness of clinically significant muscles: Case Study 4 prior to intervention:	204
Table 4.18	Isokinetic dynamometer test results after intervention: Case Study 4	208
Table 4.19	Running gait analysis: Case Study 4 after intervention	209
Table 4.20	Biomechanical angles: Case Study 4	210
Table 4.21	Tightness of clinically significant muscles: Case Study 4 after intervention	211
Table 4.22	Isokinetic dynamometer test results* prior to intervention: Case Study 5	215
Table 4.23(a)	Running gait analysis: Case Study 5 prior to intervention (Upper body)	216
Table 4.23(b)	Running gait analysis: Case Study 5 prior to intervention (Lower body)	217
Table 4.24	Biomechanical angles: Case Study 5 during intervention	218
Table 4.25	Tightness of clinically significant muscles: Case Study 5 prior to intervention	219
Table 4.26	Running gait analysis: Case Study 5 after intervention	222
Table 4.27	Biomechanical angles: Case Study 5	223
Table 4.28	Tightness of clinically significant muscles: Case Study 5 (Final)	224



Table 4.29	Isokinetic dynamometer test results prior to intervention: Case Study 6	229
Table 4.30 (a)	Running gait analysis: Case Study 6 prior to intervention (Upper body)	230
Table 4.30 (b)	Running gait analysis: Case Study 6 prior to intervention (Lower body)	231
Table 4.31	Biomechanical angles: Case Study 6 prior to intervention	232
Table 4.32	Tightness of clinically significant muscles: Case Study 6 prior to intervention	233
Table 4.33	Running gait analysis: Case Study 6 after intervention	236
Table 4.34	Biomechanical angles: Case Study 6 after intervention	237
Table 4.35	Tightness of clinically significant muscles: Case Study 6 after intervention (Date: 25/10/2003) (Interim)	238
Table 4.36	Tightness of clinically significant muscles: Case Study 6 (Final) (Date: 24/01/2004)	240
Table 4.37	A summary of the chronological progression of case studies 1 to 6	243
Table 4.38	Intensity of pain and discomfort before and after intervention	244
Table 4.39	Commencement of symptoms before and after intervention	244
Table 4.40	The total weekly-distances run before and after intervention	245
Table 4.41 (a)	Relative tightness of posterior links prior to intervention (left)	246
Table 4.41 (b)	Relative tightness of anterior links prior to intervention (left)	247
Table 4.42 (a)	Relative tightness of posterior links post intervention (left)	248
Table 4.42 (b)	Relative tightness of the anterior links post intervention (left)	249
Table 4.43 (a)	Relative tightness of posterior links prior to intervention (right)	250
Table 4.43 (b)	Relative tightness of anterior links prior to intervention (right)	251
Table 4.44 (a)	Relative tightness of the posterior links post intervention (right)	252
Table 4.44 (b)	Relative tightness of the anterior links post intervention (right)	253



Table 4.45	Muscle imbalances in peak torque performance (Prior to treatment)	254
Table 4.46	Muscle imbalances: work performance per repetition (Prior to treatment)	255
Table 4.47	The effect of the treatments on peak torque performance (Nm)	257
Table 4.48	Analysis of the effect of treatment on work performance (Nm/sec)	259
Table 4.49(a)	A comparative summary of running gait analysis prior and post intervention: weight acceptance phase – initial contact (Upper Body - Shoulder)	261
Table 4.49(b)	A comparative summary of running gait analysis prior and post intervention: weight acceptance phase – initial contact (Upper Body - Thoracic region)	262
Table 4.49(c)	A comparative summary of running gait analysis prior and post intervention: weight acceptance phase – initial contact (Upper Body – Pelvis)	262
Table 4.50(a)	A comparative summary of running gait analysis prior and post intervention: weight acceptance phase – initial contact (Lower Body -Hip)	263
Table 4.50(b)	A comparative summary of running gait analysis prior and post intervention: weight acceptance phase – initial contact (Lower Body -Knee)	263
Table 4.50(c)	A comparative summary of running gait analysis prior and post intervention: weight acceptance phase – initial contact (Lower Body -Ankle)	264
Table 4.50(d)	A comparative summary of running gait analysis prior and post intervention: weight acceptance phase – initial contact (Lower Body -Toes)	264
Table 4.51(a)	A comparative summary of running gait analysis prior and post intervention: weight acceptance phase – loading response (Pelvis)	265
Table 4.51(b)	A comparative summary of running gait analysis prior and post intervention: weight acceptance phase – loading response (Hip)	265
Table 4.51(c)	A comparative summary of running gait analysis prior and post intervention: weight acceptance phase – loading response (Ankle)	266



Table 4.52(a)	A comparative summary of running gait analysis prior and post intervention: Single leg support – mid stance phase (Pelvis)	266
Table 4.52(b)	A comparative summary of running gait analysis prior and post intervention: Single leg support – mid stance phase (Hip)	267
Table 4.52(c)	A comparative summary of running gait analysis prior and post intervention: Single leg support – mid stance phase (Ankle)	267
Table 4.53(a)	A comparative summary of running gait analysis prior and post intervention: Single leg support – terminal stance phase (Pelvis)	268
Table 4.53(b)	A comparative summary of running gait analysis prior and post intervention: Single leg support – terminal stance phase (Hip)	268
Table 4.53(c)	A comparative summary of running gait analysis prior and post intervention: Single leg support – terminal stance phase (Ankle)	269
Table 4.53(d)	A comparative summary of running gait analysis prior and post intervention: Single leg support – terminal stance phase (Toes)	269
Table 4.54(a)	A comparative summary of running gait analysis prior and post intervention: Single leg support – pre swing phase (Pelvis)	270
Table 4.54(b)	A comparative summary of running gait analysis prior and post intervention: Single leg support – pre swing phase (Hip)	270
Table 4.54(c)	A comparative summary of running gait analysis prior and post intervention: Single leg support – pre swing phase (Ankle)	271
Table 4.55(a)	A comparative summary of running gait analysis prior and post intervention: swing leg advancement – initial swing phase (Knee)	271
Table 4.55(b)	A comparative summary of running gait analysis prior and post intervention: swing leg advancement – initial swing phase (Ankle)	272
Table 4.56(a)	A comparative summary of running gait analysis prior and post intervention: swing leg advancement – mid swing phase (Hip)	272
Table 4.56(b)	A comparative summary of running gait analysis prior and post intervention: swing leg advancement – mid swing phase (Knee)	273



Table 4.57(a)	A comparative summary of running gait analysis prior and post intervention: swing leg advancement – terminal swing (Pelvis)	273
Table 4.57(b)	A comparative summary of running gait analysis prior and post intervention: swing leg advancement – terminal swing (Hip)	274
Table 4.57(c)	A comparative summary of running gait analysis prior and post intervention: swing leg advancement – terminal swing (Ankle)	274
Table 4.57(d)	A comparative summary of running gait analysis prior and post intervention: swing leg advancement – terminal swing (Toe)	275
Table 4.58	Metatarsophalangeal extension at the first toe	277
Table 4.59	Biomechanical measures before and after intervention	280
Table 4.60	Change in left hind foot pronation before and after intervention	282
Table 4.61	Change in right hind foot pronation before and after intervention	282
Table 4.62	Isokinetic dynamometer test results* prior to intervention: Case Study 7	287
Table 4.63(a)	Running gait analysis: Case Study 7 prior to intervention (Upper body)	288
Table 4.63(b)	Running gait analysis: Case Study 7 prior to intervention (Lower body)	289
Table 4.64	Biomechanical angles: Case Study 7 before intervention	292
Table 4.65	Tightness of clinically significant muscles: Case Study 7 prior to intervention	293
Table 4.66	Isokinetic dynamometer test results* after intervention: Case Study 7	295
Table 4.67	Running gait analysis: Case Study 7 after intervention	296
Table 4.68	Biomechanical angles: Case Study 7 after intervention	297
Table 4.69	Tightness of clinically significant muscles: Case Study 7 after intervention	298
Table 4.70	Isokinetic dynamometer test results* prior to intervention: Case Study 8	302
Table 4.71(a)	Running gait analysis: Case Study 8 prior to intervention (Upper body)	303



Table 4.71(b)	Running gait analysis: Case Study 8 prior to intervention (Lower body)	304
Table 4.72	Biomechanical angles: Case Study 8 before intervention	305
Table 4.73	Tightness of clinically significant muscles: Case Study 8 prior to intervention	306
Table 4.74	Running gait analysis: Case Study 8 after intervention	309
Table 4.75	Tightness of clinically significant muscles: Case Study 8 after intervention	310
Table 4.76	Summary of biomechanical angles: Case Study 8	311
Table 4.77	Validation of soft tissue rating process (Posterior fascia links)	315
Table 4.78	Validation of soft tissue rating process (Anterior fascia links)	316
Table 4.79	Analysis of validation data on tightness	317





## LIST OF ABBREVIATIONS

AMA	American Medical Association
CACS	Chronic Anterior Compartment Syndrome
CCS	Chronic Compartment Syndrome
CECS	Chronic Exertional Compartment Syndrome
CPCS	Chronic Posterior Compartment Syndrome
CT	Connective Tissue
ECM	Extracellular Matrix
EDL	Extensor Digitorum Longus
ERLP	Exercise Related Leg Pain
FDL	Flexor Digitorum Longus
FHL	Flexor Hallucis Longus
Inv	Inversion
km	Kilometre
km/h	Kilometre per hour
mm	Millimetre
MTP	Metatarsophalangeal
Nm	Newton-metre
Nm/s	Newton-metre per second
TA	Tibialis Anterior
TDF	Thoraco-Dorsal Fascia
PB	Peroneus Brevis
PCS	Posterior Compartment Syndrome
PF	Plantar Flexion
PG	Protoglycans
PT	Peroneus tertius
VAS	Visual Analogue Scale
S.v.	Sub verbo



## GLOSSARY OF TERMS

### *Chronic posterior compartment syndrome*

Chronic posterior compartment syndrome is a pathological condition of skeletal muscle characterized by increased interstitial pressure within an anatomically confined muscle compartment, specifically the posterior compartment, which interferes with the circulation and function of the muscle and neurovascular components of the compartment (Nicholas & Herschman, 1995a).

### *Dysfunctional fascia*

Fascia, in the normal healthy state is relaxed and wavy in configuration. Due to its visco-elastic biomechanical properties it has a limited ability to “stretch” and move without restriction. When connective tissue experiences physical trauma, scarring or inflammation, the fascia loses its pliability (Culav *et al.*, 1999). It becomes tight, restricted and a source of tension to the rest of the body (dysfunctional). Trauma, such as a repetitive strain injury, has cumulative effects. The changes they cause in the fascial system influence comfort and the functioning of the body.

Micro-structurally, the end results of the healing / reorganizing process in connective tissue are that the tissue a) has a more irregular arrangement (the arrangement and the alignment are a result of the mechanical stresses applied to the tissue); b) has a lower water content and c) contains more random cross-links between fibres, fibre bundles and adjacent tissues. As the collagen fibres are more randomly aligned with respect to forces applied to the tissue, the fibres must resist forces that are not parallel to their longitudinal axes. This is a task for which collagen is not structurally designed. In addition the loss of water diminishes the ease with which the collagen bundles might slide past one another (Threkeld, 1992). In other words, the fascia does not function the way it was designed to. Fascia that has been injured and has undergone structural changes affecting its function, as mentioned above, will therefore be called dysfunctional fascia by the researcher.



### ***Effective functional length of myofascial chain***

The researcher has defined the *effective functional length of myofascial chain* as that length of the myofascial web that is available for the execution of a normal range of body movements. Restrictions in the myofascial web could thus compromise the range of normal movement.

### ***Muscle imbalances***

Muscle imbalances can be described as a deviation from a theoretical optimal posture or movement by a disproportional effort from muscles working around a joint or joint series. In relation to gait, this can result in abnormal stress through the kinetic chain causing deformities, pathology and symptoms. (Harradine *et al.*, 2006)

### ***Myofascia***

The word “myofascial” connotes the bundled together, inseparable nature of muscle tissue (myo-) and its accompanying web of connective tissue (fascia) (Comerford, 2000; Myers, 2001).

### ***Myofascial chain***

The word “chain” indicates the continuous nature of the myofascia throughout the body (Robertson, 2001).

### ***Myofascial links***

The word “links” implies that the muscles are linked via the fascia to one another (Myers, 2003).

### ***Myofascial release techniques according to Manheim (1994) and Barnes (1990)***

Myofascial procedures vary significantly, ranging from prolonged stretching and soft tissue mobilizing techniques to subtle indirect techniques (Manheim, 1994). Barnes (1990) has defined myofascial release techniques as the three-dimensional application of sustained pressure and movement into the fascial system in order to eliminate fascial restrictions.



### ***Pronation***

Pronation is classically defined as abduction and eversion of the foot along with hind foot eversion (Dugan & Bhat, 2005).

### ***Release***

Release means to set free from restraint (Concise English Dictionary, 1982, S.v. 'release'); to allow moving or flowing freely (The Concise Oxford English Dictionary, 2002, S.v. 'release').

### ***Restrictions in the myofascial chain***

Restrictions in the myofascial chain have been defined by the researcher as anything that can lead to a decrease in the *effective functional length of the myofascial chain*, such as trigger points, scar tissue and inflammation.

### ***Runner***

A person who runs a minimum distance of between 20 to 30 km per week on a regular basis and has been running consistently for a minimum period of time exceeding one year (Hreljac, 2005).

### ***Soft tissue mobilization techniques***

For the purpose of this specific research, soft tissue mobilizing techniques will refer to a variety of soft tissue mobilizing techniques aimed at the release of the tightness of tight myofascial tissue. The following soft tissue approaches were used:

- trigger point release techniques according to Travell and Simons (1999);
- myofascial release techniques according to Barnes (1990) and Manheim (1994);  
and
- specific soft tissue mobilizations according to Hunter (1998).

### ***Specific soft tissue mobilizing techniques***

This approach relies on the use of specific soft tissue mobilization techniques which are applied to a specific area of tightness with the aim of restoring normal movement (Hunter, 1998).



***Trigger point release techniques***

Active trigger points are deactivated through ischemic compression. Ischemic compression applies sustained pressure to the trigger point with sufficient force and for a long enough time to deactivate it (Travell & Simons, 1999).



## CHAPTER 1

### THE SCOPE OF THE RESEARCH

#### 1.1 INTRODUCTION AND BACKGROUND

During October 2007 a landmark event occurred with the first ever international conference held at the Harvard Medical School in Boston, USA, dedicated to fascia research. The First International Fascia Research Congress was used as a catalyst for all scientific researchers and practicing healthcare professionals to learn from each other and to gain new insights into the human body's soft tissue web. As demonstrated by this research study, the exploration of this new frontier in medicine is bound to lead to the solution of many sport related injuries that to date have been considered as unsolvable. The integration of this new knowledge with regard to the continuous myofascial web together with the existing knowledge base is bound to fill many gaps that exist in current theoretical perspectives.

In a recent article by Harden (2007), he concludes, based on an extensive MEDLINE literature review, that “no successful, comprehensive, consensus- or empirical based effort to determine clinically valid diagnostic criteria for myofascial pain syndrome” has been done since the groundbreaking work of Travell & Simons (1999). Of the 135 references in his article, only five have been published during the last five years (Desmeules *et al.*, 2003; Harden *et al.*, 2003; Staud, 2003; Gerwin *et al.*, 2004 and Shah *et al.*, 2005). Of these five references, one (Gerwin *et al.*, 2004) deals with an extension of the research by Travel & Simons (1999) while the other four address fibromyalgia pain syndrome. Limited publications are encountered in the alternative medicine publications, such as the work by Gemmell *et al.* (2005) which deals with a single case study on myofascial distortion treatment of a tennis player.

The contribution of this research study is groundbreaking in the sense that it provides empirically based evidence of the fundamental role that the continuity of the myofascial web plays in fascia related injuries such as Chronic Posterior Compartment Syndrome (CPCS). The success of the research could be largely



contributed to the qualitative approach that had been followed coupled with the exploration of the characteristics and role that fascia plays in CPCS.

In addition to groundbreaking theoretical concepts developed by the research study, a qualitative research methodology has been followed which is a unique approach to a field normally dominated by quantitative methodologies. This approach enabled the progressive development of the theory basis for the causal relationships which leads to the development of CPCS. The initial apprehension of the researcher about the use of a *qualitative* paradigm was progressively eliminated. The approach proved to be as powerful as the identification of the theoretical gaps which existed in the current theory about the pathogenesis of CPCS. The deductive logic and systematic elimination of rival theories generated profound results that would not have been possible with a research approach based on the inference from observation and statistics.

The objective of the first chapter is to provide the reader with a high-level overview of the thesis. It provides the contextual background to the reasons and motivation for the research project. It deals chronologically with a high-level overview of the research problem; the research question; the associated investigative questions; key research objectives; significance of the research; the research process that was followed; research design and methodology; research assumptions; research constraints; and the contextual boundaries to the research.

The chapter concludes with an overview of the thesis structure as well as a section on chapter and content analysis. Although the first chapter provides a picture of the progressive development of the research study, the structure of the thesis does not reflect this chronology. The section on chapter and content analysis thus provides a framework for the interpretation of the functional structure of the thesis.

## **1.2 THE RESEARCH PROBLEM**

Since the mid-1980's, a much greater awareness existed in terms of the benefits of regular exercise (Lee *et al.*, 2000; Oguma *et al.*, 2002; Taunton *et al.*, 2002;



Cornelissen & Fagard, 2005; Williams, 2007). This awareness has led to an exercise boom, and for many, running have become the exercise of choice as result of its convenience, health benefits and economical considerations. Running as the most popular leisure sports activity is widely recognised (Taunton *et al.*, 2002; Van Sluijs *et al.*, 2004).

It is however also important to realise the negative side of running (van Mechelen, 1992; Plastaras *et al.*, 2005; van Gent *et al.*, 2007). The annual sport injury rate amongst runners according to van Gent *et al.* (2007) for lower extremity running injuries in long distance runners ranges from 19.4% to 79.3%. These findings support the work of Taunton *et al.* (2002) who analysed 2002 running injuries. In accordance with their findings the highest incidence of injuries was associated with the lower leg. With recreational runners, the highest incidence rate was that of knee injuries, whereas lower leg and foot injuries were more prevalent in the elite middle distance runners. Taunton *et al.* (2002) classified patients as having a running injury if:

- “they had pain or symptoms during or immediately after a run;
- they had pain or symptoms within the approximate time span of beginning a running programme;
- the injury was felt to be related to running; and
- the injury was significant enough to force them to stop running or significantly reduce their running mileage and seek medical assistance.

Not all of these injuries respond well to conservative treatment (Swain & Ross, 1999; Fraipont & Adams, 2003). Some of these injuries only respond to surgical therapy (Martens *et al.*, 1984; Gerow *et al.*, 1993). The effect of surgical intervention on the longer term is however not always successful (Verleisdonk *et al.*, 2004), and also not very attractive for the elite athletes. As a result many promising athletes are forced to take-up a less demanding sport. One of these injuries that do not respond to conservative treatment is *chronic posterior compartment syndrome*. Martens *et al.* (1984) is even of the opinion that no place exists for the conservative treatment in the management of this pathology.





### ***The research problem***

*No known successful conservative treatment exists for the symptoms of chronic posterior compartmental syndrome (CPCS) in runners.*

### **1.3 THE RESEARCH QUESTION**

*Compartment syndrome* is a pathological condition of skeletal muscle characterized by increased interstitial pressure within an anatomically confined muscle compartment that interferes with the circulation and function of the muscle and neurovascular components of the compartment (Nicholas & Herschman, 1995a). The initial complaint of the patient is usually one of pain or a deep ache over the involved compartment. If this condition occurs in the posterior compartment of the lower leg, it is referred to as “*posterior compartment syndrome* (PCS)”. The condition is considered to be chronic when the symptoms persist for longer than three months (Klenerman *et al.*, 1995; Von Korff & Saunders, 1996; Grabois, 2005).

In addition to the lack of response to conservative treatment, it is also not easy to diagnose “*chronic posterior compartment syndrome*”. A thorough history, a careful physical examination as well as the testing of intra-compartmental pressures are considered by some as essential to establish the diagnosis (Fraipont & Adamson, 2003). The healthcare practitioner thus often has to deal with a situation where the symptoms are clearly manifested, but the diagnosis of the underlying cause not that obvious. The objective of the research is to develop soft tissue mobilization techniques that will provide a relief of the symptoms of posterior compartment syndrome, irrespective of whether the underlying cause is CPCS or for example “*medial stress syndrome*” which is often confused with the former. Both of these conditions present with the same symptoms do not respond to conservative treatment and both do respond favourably to surgical treatment. This difficulty of a proper diagnosis has a direct bearing on the title of this research study. The title is “*The effect of soft tissue mobilization techniques on the symptoms of chronic posterior compartment syndrome in runners*”. The objective is thus to find a conservative treatment approach for practitioner who do not normally have access to the facilities for a differential diagnosis of the underlying cause of these symptoms.



The current physiotherapy treatment that is prescribed for PCS consists mainly of the treatment of the involved calf muscles. Treatment techniques include cross frictions, massage, ultrasound- and interferential treatment, calf muscle stretches and eccentric strengthening exercises of the calf muscles (Davey *et al.*, 1984; Martens *et al.*, 1984; Detmer *et al.*, 1985; Melberg & Styf, 1989; Biedert & Marti, 1997; Blackman *et al.*, 1998; Schepsis & Lynch, 1998; Micheli *et al.*, 1999; Garcia-Mata *et al.*, 2001). None of these interventions provided any significant lasting effects on the symptoms of CPCS (Davey *et al.*, 1984; Biedert & Marti, 1997; Fraipont & Adamson, 2003). Elite runners are often sceptical of the surgical option. The first consideration is the effect of the treatment option on their ability to train, since they are unable to train for a period of time post- operatively. Secondly, there is also a great deal of uncertainty with regard to the success rates of the surgical option. Another alternative available to the runner is a decrease in his training programmes to the extent that he no longer experience symptoms (Ross, 1996). None of these options are acceptable to dedicated long distance runners, and this has resulted in many promising athletes changing from running to a physically less demanding sport.

### ***The research question***

*Can a successful conservative treatment regime/approach be developed for runners with symptoms of CPCS?*

## **1.4 INVESTIGATIVE QUESTIONS**

It is the intent to answer the following investigative questions in this thesis:

- *Why are the current known conservative approaches to the treatment of CPCS unsuccessful?*
- *Are there any theoretical gaps in the treatment of CPCS?*
- *Can an improved theoretical framework for the pathogenesis of CPCS be developed?*
- *Can such a model be used to develop a successful conservative treatment regime for the condition?*



- *Would the application of such a conservative treatment approach in a multiple case study experiment provide sufficient replication of results to validate such a model?*
- *Do other biomechanical deviations/abnormalities play a role in the perpetuation of the condition?*
- *Would the successful treatment of the conditions resolve such abnormalities?*
- *Would the envisaged theoretical framework for CPCS be able to explain and clarify the underlying cause-effect relationships involved?*

## **1.5 KEY RESEARCH OBJECTIVES**

The key research objectives are:

- *To develop an enhanced theoretical framework as basis for the treatment of CPCS in runners.*
- *To develop a successful conservative methodology for the treatment of CPCS in runners.*
- *That theoretical contributions formulated within the ambit of this thesis in addition to academic contributions also provide for practical application in solving the frustrating effects of CPCS in both runners and the therapists involved with the treatment of the condition.*
- *To make a significant contribution to the existing body of knowledge with specific reference to a more holistic approach towards the clinical treatment of myofascial system related dysfunctions in the field of physiotherapy.*
- *That such a holistic approach to the treatment of soft tissue dysfunctions would lead to paradigm shift in the treatment of sport injuries in general.*

## **1.6 SIGNIFICANCE OF THE RESEARCH**

A successful conservative treatment for the symptoms of CPCS would be a major breakthrough. The current physiotherapy approaches, reported on in the literature,



provide no lasting success, whilst the documented surgical option also has a limited success rate, especially with regard to the posterior compartment. A successful conservative approach, alleviating the symptoms of CPCS, could form the basis for the successful treatment of other chronic fascia-related injuries such as iliotibial band syndrome, plantar fasciitis and chronic compartment syndromes (CCS) of the other compartments of the lower leg.

Such an approach for the treatment of the symptoms of CPCS could have positive economic and psychological implications. The consequence of a successful treatment through manual, conservative means, would be that costs will be saved on surgery, hospitalization, post-operative rehabilitation and many promising athletes would be able to continue their sport careers.

## **1.7 THE RESEARCH PROCESS**

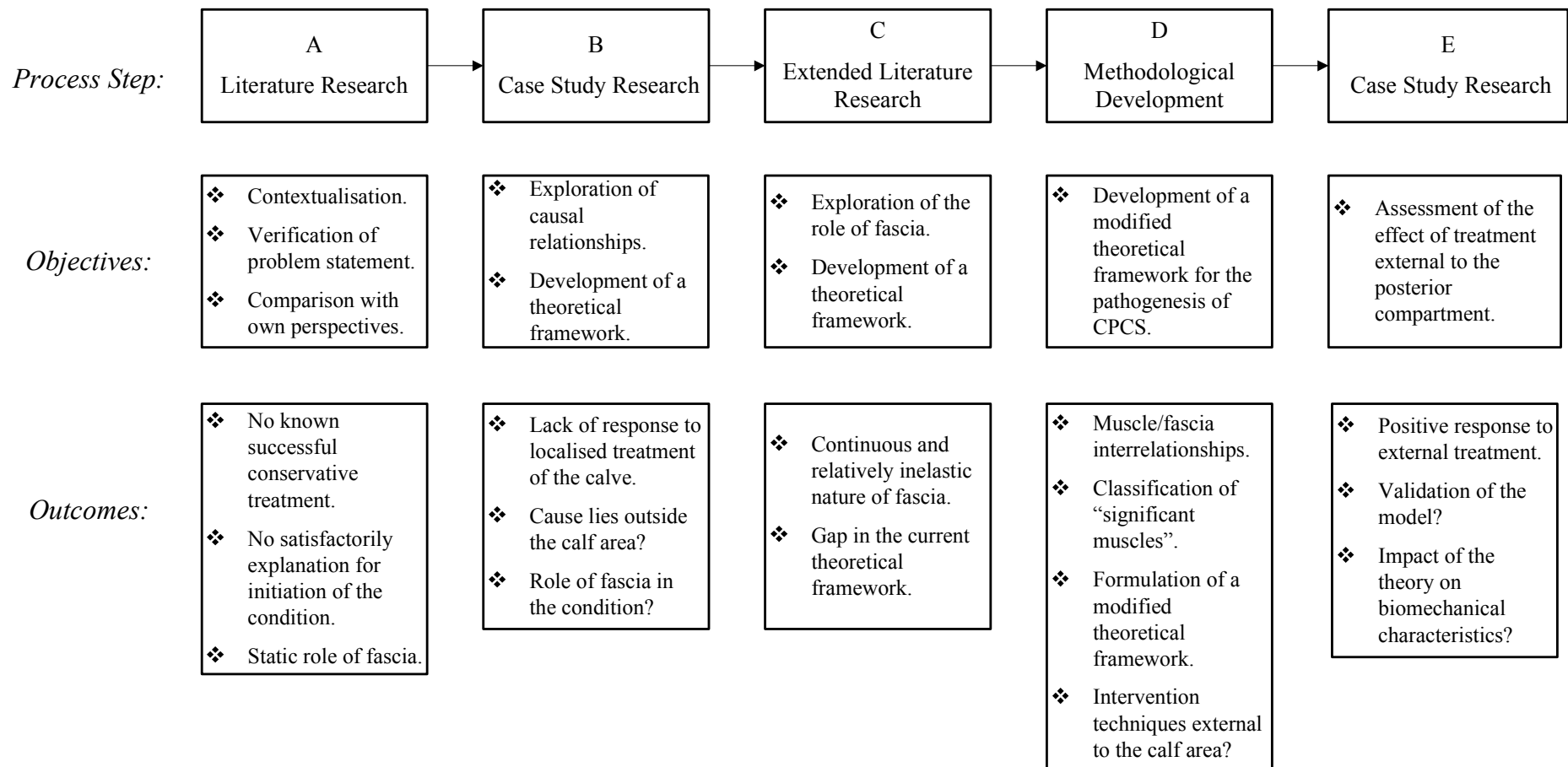
The research process consisted of two major phases. Phase 1 consisted in essence of exploratory research, aimed at the development of a methodological framework for CPCS. In phase 2, the focus was on the verification and validation of both the theoretical model and treatment methodology developed for the symptoms of CPCS.

### **1.7.1 Phase 1 – Exploratory research**

This phase consisted of an initial literature research aimed at CPCS, as well as at research methodologies. The following sections provide a brief overview of this phase which is also reflected in Figure 1.1.

#### **○ Literature research**

The literature study was aimed at two aspects. The first dealt with research methodology aimed at the development of a sound framework for the execution of the research. The second dealt with the current theoretical status of CPCS in order to establish a framework as basis for the development of a treatment regime as well as the basis for predicting the effect of such treatments.



**Figure 1.1: The explanatory research process – Phase 1**



- **Exploratory research**

Initial exploratory work aimed at the validation of the existing theoretical framework for the pathogenesis of CPCS followed. This phase identified the shortcomings in the current theoretical model. Progressive exploratory work indicated that the cause of the symptoms lies outside the calf muscles. These findings coupled with the continuous nature of the myofascial web led to the development of a revised or modified model for the pathogenesis of CPCS. This revised model formed the theoretical base for the development of the treatment interventions.

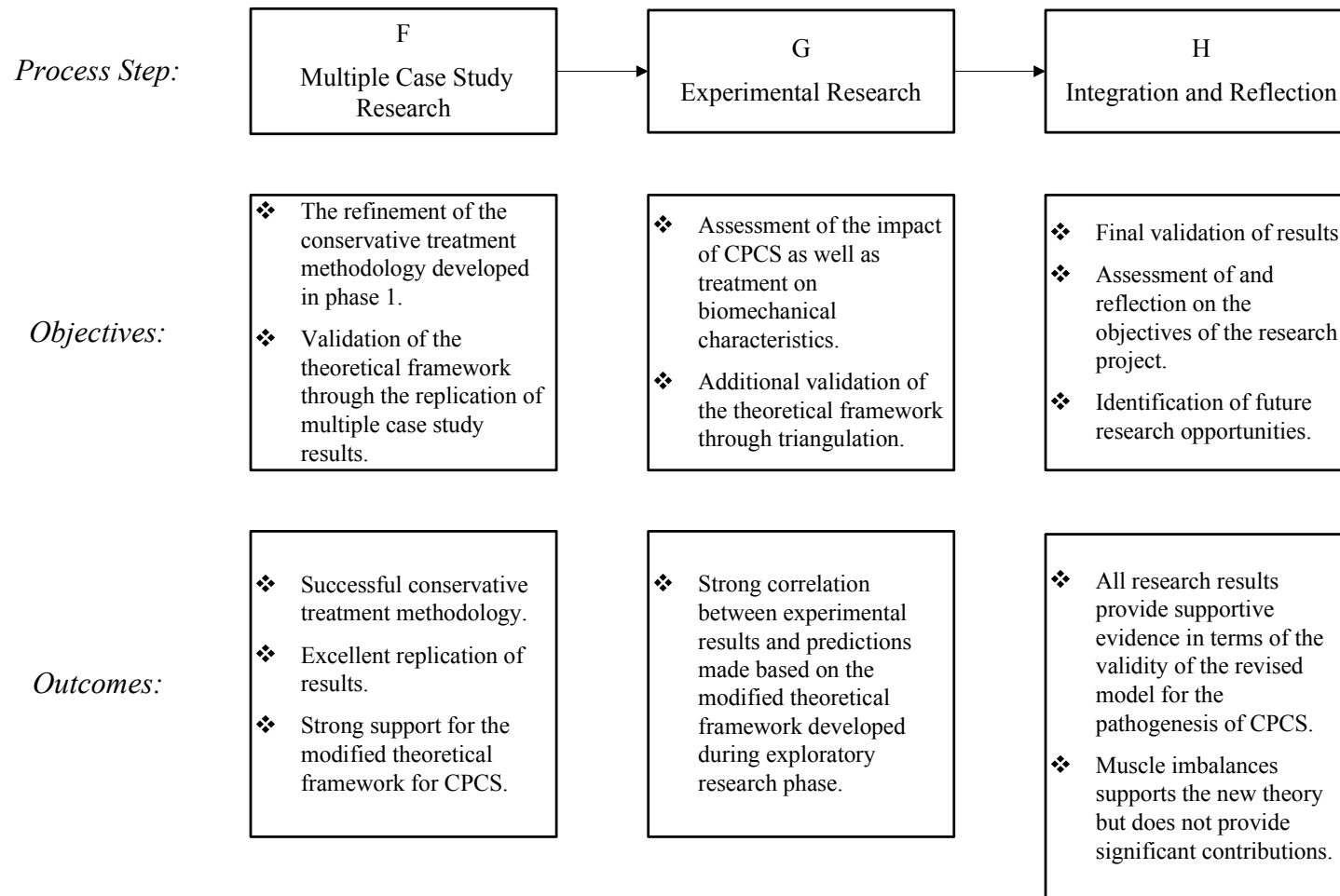
- **The identification of the clinically significant muscles and myofascial links**

In this phase the focus was on the identification of muscles that are linked via the myofascial web to the muscles of the posterior compartment. This research also led to the realisation that the continuous nature and relatively non-elasticity of fascia have been ignored in the theoretical treatment of the condition. This theoretical gap strongly supported the deductions made from the exploratory research in terms of the pathogenesis of CPCS. The newly modified theoretical model postulates that the cause of the symptoms lies outside the calf area. This led to the development of the concept of *clinically significant muscles*. These muscles were identified based on literature and validated during clinical trials.

### **1.7.2 Phase 2 – Explanatory research**

- **Case study research**

During the explanatory research phase the validity of the newly developed theoretical framework as well as the treatment methodology were assessed. Phase 2 consisted of both case study research as well as experimental research. Phase 2 is also reflected in Figure 1.2.



**Figure 1.2: The explanatory research process – Phase 2**



- **Multiple case study research**

The focus of the case study research was on the validation of both the theory and techniques through *replication logic*.

- **Experimental research**

The aim of the experimental research was to investigate the effect of CPCS on selected biomechanical properties. It also provided an opportunity for experiment aimed at eliminating the influence of the researcher. This was achieved by means of a team of under-graduate students which performed the experiment. The results of the experiment also provided statistically significant results in spite of the relatively small sample size involved.

- **Integration and reflection**

The last element in phase 2 consisted of the final integration of results. The question was whether sufficient evidence could be provided through the triangulation of results for generalisation.

## **1.8 THE RESEARCH DESIGN AND METHODOLOGY**

By the very nature of the research question, the research is imbedded in the physical and natural world. Although research in this domain is normally based on the classical scientific method with well-established experimental approaches (Remenyi *et al.*, 2002), this research approach deviated from the norm in the sense that it involved both a qualitative, theory-building component as well as a quantitative component.

The focus of the first part of the research is on exploratory research with the key objective of developing an enhanced understanding of the condition of CPCS. The focus is thus on creating understanding of causal relationships that exist with the condition.





In the second part of the research, the focus is on the application of the theory-building component. The objective with the second part is to develop and validate a treatment intervention based on the theoretical base that was developed. This phase thus deals primarily with deductive reasoning while the first primarily relies on inductive reasoning.

The research thus entails an overlap between qualitative and quantitative methodologies. Although one can make a clear distinction between qualitative and quantitative research, it does not imply identification with either at the exclusion of the other (Babbie, 2005). The use of both often forms the basis of a complete understanding of the subject at hand.

## 1.9 RESEARCH ASSUMPTIONS

Watkins (2006: 53) provides the following quotation from Leedy & Ormrod (2001: 62-63) which eloquently describes the importance of assumptions in research.

*“Assumptions are what the reader takes for granted. But taking things for granted may cause misunderstanding. What we may tacitly assume, others may never have considered. If we act on our assumptions, and if in the final result, such actions make big differences in the outcome, we may face a situation we are totally unprepared to accept. In research we try to leave nothing to chance in the hope of preventing any misunderstanding. All assumptions that have material bearing on the problem should be openly and unreservedly set forth. If others know the assumptions a researcher makes, they are better prepared to evaluate the conclusions that result from such assumptions. To discover your own assumptions, ask yourself, what am I taking for granted with respect to the problem? The answer will bring your assumptions into clear view”.*

At the outset of the research project a number of assumptions were made. These included the following:

- The condition of CPCS was not caused solely due to muscle exertion.



- The condition has to be triggered by some variable or variables that were not considered in the current theoretical treatment of the condition.
- The lack of success with conservative treatment interventions was due to the fact that the current theoretical framework employed as basis for such interventions was incomplete.
- It is possible to develop a successful conservative treatment for the condition.
- It will in all probability be possible to validate the envisaged theoretical base as well as the treatment approach by means of replication in a multiple case study design due to the nature of the biomechanical characteristics associated with the condition.

## **1.10 RESEARCH CONSTRAINTS**

A number of constraints existed that influenced the execution of the research. These constraints are briefly reflected on in the following section.

- The theoretical framework that existed for CPCS had obvious deficiencies that prohibited the use thereof as a basis for the prediction of replication in a multi case study approach.
- The lack of an adequate theoretical framework prohibited the use of classical laboratory experimentation.
- This lack of an adequate theoretical framework also called for a less structured approach with regard to research methodology due to the progressive nature of the research (Olivier, 2004).

## **1.11 CONTEXTUAL BOUNDARIES**

### **1.11.1 Limitations**

- The verification of the success of the treatment interventions is limited to the subjective assessments of the subjects. It excludes sophisticated diagnostics such as the measurement of pressures within the involved posterior compartment.



- The small sample sizes of subjects added a limitation to the use of statistical analysis as a basis for the verification of results.
- From an ethical perspective it was imperative that the research was continuously focussed on the improvement of the subject's performance. This excluded intermittent strategies such as controlled periods of no intervention.
- Intervention techniques will be limited to acknowledged "best practices" as applied by the practising sport physiotherapy community in order to enhance the general applicability of treatment interventions resulting from the research.

### **1.11.2 Delimitations**

- The research excludes the anatomical analysis of the myofascial structures and is limited to published research results as contained in the literature.
- The research excludes the measurements of the actual pressures within the posterior compartment.
- The measurements of biomechanical effects are limited to aspects that are affected by the condition and which are measurable.

## **1.12 CONCLUSION**

The salient features of this chapter are that it provides an overview of the research problem, the research question as well as investigative questions, constraints, limitations and delimitations of the research. It provided a contextual background to the research, as well as a perspective on the contributions made by the research study. It provides the reader with an insight into the structure and the methodological approach followed with the research project. In the next chapter the literature review will be covered. The review consists of four major sections, namely a review of the research methodology; CPCS; fascia (collagen tissue) and myofascial links; as well as a review of biomechanical factors linked to CPCS by this research.

It provides a systematic, critical and integrated discussion of the literature research findings and also provides a clear and explicit description of the key concepts as covered in this thesis.



In reviewing the research methodology special attention will be given to the use of multiple case study research in exploratory research; the importance of a sound theoretical framework for explanatory research; as well as the concept of “replication logic”.

The research makes use of the triangulation of results as encountered in mixed methodological paradigms and these concepts are thus also explored. In this regard some of the biomechanical aspects are investigated by means of classical experimentation and some of these requirements are reflected on in the concluding part of Chapter 2 dealing with research methodology.



## **CHAPTER 2**

### **LITERATURE REVIEW**

#### **2.1. INTRODUCTION**

In the previous chapter an overview of the broader contextual background to the research study was given. This included the research problem, research and investigative questions, the process that was followed, an overview of the structure of this document, and a chapter and content analysis. In this chapter an overview of the literature research will be given. The first section of the chapter deals with the research methodology. This is followed with a section on CPCS. In the third section fascia (collagen tissue) and myofascial links are covered, as well as a review of biomechanical factors linked to CPCS by this research. Section four deals with an extended literature research which was done towards the end of the exploratory phase of the research project.

#### **2.2. RESEARCH METHODOLOGY**

##### **2.2.1. An introduction to qualitative research**

In this section an overview of the literature research on research methodology will be given, with specific reference to qualitative research methodologies.

The major divide in the research world is between research in the physical and natural world and research in the social world (Watkins, 2006). Although research approaches are not unique to any of these, one does find that research in the first category is normally dominated by quantitative approaches. This is largely due to the rich wealth of theoretical knowledge that has been developed and underpins research in this area. Research in the natural and physical world is thus normally based on quantitative experimentation and research that does not conform to this framework is often frowned upon.



One however encounters situations where the current theoretical frameworks prove to be inadequate in terms of its ability to provide basic causal relationships between the variables under investigation (Smart & Doody, 2003; Yin, 2003). In such situations the researcher is thus forced to revert to exploratory research in order to develop new or modified theoretical concepts to predict the behavioural characteristics of variables. In such situations, the researcher is forced to the application of qualitative research methodological approaches and associated research techniques to create the necessary fundamental understanding of the variables under consideration.

The focus of this section of the literature study is thus largely on qualitative research techniques and approaches, as well as the transition from relatively unstructured exploratory research, to the validation of new theory by means of explanatory research techniques. It is assumed that the reader is fairly conversant with quantitative research paradigms, and these aspects will thus not be covered in any depth in the literature review. Where appropriate, references will be provided with the actual designs of such quantitative research applications.

As stated in the introductory chapter, the research is in certain respects unique due to a number of considerations. These include:

- The fact that the research is dealing with a subject topic from the medical sciences where research are normally subject to classical experimentation.
- The research project deals with both explorative as well as explanatory research, as the current theoretical framework fails to provide adequate explanation to the initiation as well as behavioural issues associated with the condition.
- The research project deals with both qualitative as well as quantitative research paradigms.

The objectives of the section on research methodology were thus:

- To provide the researcher with the necessary contextual background for the selection of an appropriate research methodological approach for the research project.
- To provide the researcher with an overview of the key concepts associated with the selected research methodology.



- To provide the researcher with the necessary contextual background in order to generate the necessary research designs overview of the research design.
- To provide the researcher with an overview of the criteria for the assessment of the quality of the design.

As stated, the research deals with both exploratory as well as explanatory issues that falls in the domain of qualitative research. It also deals with selective experimental research on biomechanical measures which is used to explore the validity of certain conclusions drawn from new theoretical model developed as a result of the research. The utilization of such diverse research paradigms are from an epistemological perspective, classified as “mixed methodologies”. The literature review will be introduced by the discussion of the concept of “Mixed Methodologies” (Watkins, 2006) as embodied in this thesis.

### **2.2.2. Mixed methodologies**

The utilisation of mixed methods research designs have grown considerably since the late 1970's (Hanson *et al.*, 2005; Christ, 2007). The obvious advantage of a multi-method approach developed by Campbell & Fiske during 1959, according to Hanson *et al.* (2005) is that it eliminates method bias in the research which one would assume increases the generalisation of research findings.

With regard to the philosophical base of the research paradigm general consensus is that it is based on pragmatism (Tashakkori & Teddlie, 2003). In addition to the argument that the *position* as established by triangulation would be more exact, these authors argue that the research question is the prime objective. With this pragmatic perspective, any method which could reveal answers to this question could and should be used.

According to Watkins (2006:44) who cites Easterby-Smith *et al.* (1996:133-134), the use of multiple, but independent measures is known as triangulation, of which four categories exists, namely, *theoretical triangulation*, *data triangulation*, *investigator triangulation* and *methodological triangulation*. Within the ambit of this research only the latter applies. According to Yin (1994) the concept of triangulation in research



methodology originated from the natural sciences where triangulation refers to the definition of a point in a geometric space by means of the intersection of three vectors. One or two such vectors are insufficient whilst a fourth would be redundant. This concept is incorrectly attributed by some (Hanson *et al.*, 2005) to military naval sciences who also used the principle to establish the exact location of an object at sea.

The key question is thus to direct the same question to different sources of evidence.

### ***Methodological triangulation***

Methodological triangulation refers to research where both quantitative as well as qualitative research approaches are used for data collection. This culminates in diverse data collection techniques which can be juxtaposed with regard to the answering of research questions (Watkins, 2006). Within the context of this research both multiple case studies and experimentation are used for data collection. In the following section pertinent aspects associated with case study research will be reviewed.

### **2.2.3. Case study research methodology**

Watkins (2006: 38) drawing descriptions from Hussey & Hussey (1997), Leedy and Ormrod (2001) and Remenyi *et al.* (2002); define case study research as follows:

#### **o Case study research**

*“Primarily falling within the phenomenological (qualitative) paradigm, case study research can equally be applied within the context of the positivistic (quantitative) paradigm. Case study research represents an empirical enquiry that investigates a contemporary phenomenon within a real life context, when the boundaries between phenomenon and context are not clearly evident, and in which multiple sources of evidence are used. It is particularly valuable in answering who, why and how questions in research.”*





Case study research is often used in exploratory research (Boos & Brownie, 1992; Hussey & Hussey, 1997 as cited by Scholz & Tietje, 2002). The latter identify classes of case studies, namely “*Descriptive case studies*”, “*Illustrative case studies*”, “*Experimental or Exploratory case studies*” and “*Explanatory case studies*”. Within the ambit of this thesis only “*Experimental or Exploratory*” and “*Explanatory*” case studies are used. The term “*Exploratory Case Study*” is preferred and will be used throughout this thesis.

- **Exploratory case studies**

Exploratory case studies help to gain insight into the structure of a phenomenon in order to develop hypothesis, models or theory (Scholz & Tietje, 2002). According to them the research design and data collection methods are usually not specified in advance due to the exploratory nature of the research process.

- **Explanatory case studies**

Explanatory case studies can also serve to test cause-and-effect relationships (Scholz & Tietje, 2002; Payne & Williams, 2005). Henning *et al.* (2004) citing Stake (1995) highlights that in a case study the main assumption is that the phenomenon is investigated as a “*bounded system*”. This concept of a “*bounded system*”, proved to be significant in terms of the definition of CPCS during the latter phases of the research.

### **2.2.3.1. General approach to case study design**

According to Yin (2003:19-20), a research design can be defined as:

*“... the logical sequence that connects the empirical data to a study’s initial research question and ultimately, to its conclusions. Colloquially, a research design is an action plan from getting from here to there, where here may be defined as the initial set of questions to be answered, and there is some set of conclusions about these questions”.*



Yin (2003: 20) also quotes Nachimias & Nachimias (1992:77-78) who defines it as “... a *logical model of proof that allows the researcher to draw inference concerning causal relations among variables under investigation*”.

### 2.2.3.2. Components of research design

Yin (2003) identifies five components of research designs that are especially important, namely: a *study’s questions*; its *propositions*, if any; its *units of analysis*; the *logic linking the data to the propositions*; and the *criteria for interpreting the findings*. These components will be briefly reviewed.

#### ○ **The study’s question**

The study’s questions provide guidance as to what the most appropriate research strategy would be. The case study strategy is most likely to be appropriate for *how* and *why* questions.

#### ○ **The study’s proposition**

The proposition directs attention to something that should be addressed. “... *only if you are forced to (able to) state some proposition will you move into the right direction*” (Yin, 2003: 22).

#### ○ **The unit of analysis**

*“The main unit of analysis is likely to be at the level being addressed by the main study questions. ... As a general guide, your tentative definition of the unit of analysis (and therefore the case) is related to the way you have defined your initial research question”* (Yin, 2003: 22 - 23).

According to Yin (2003) the selection of the appropriate unit of analysis occurs when one accurately specifies the primary research question. If the question does not favour one unit of analysis above another, the research question is in all probability too vague. Typical units of analysis according to Hussey & Hussey (1997) could include:



- ***An individual***

A person is a popular unit of analysis.

- ***An event***

An event is a reference to a particular incident that could trigger a certain response.

- ***An object***

An object is a commodity such as a product, a service or a process.

- ***A body of individuals***

A body of individuals includes groups of people.

- ***A relationship***

A relationship refers to the relationship between two or more individuals or bodies.

According to Yin (2003), the design should also tell you what is to be done after the data have been collected – as indicated by the *logic that links the data to proposition* and the *criteria for interpreting findings*. These are discussed in the following paragraphs.

- **Linking data to propositions**

One of the most precise examples quoted by Yin (2003) is that of linking treatments with patients in psychological experiments. This process has direct bearing on the research in question.

- **Criteria for interpreting a study's findings**

Yin (2003) suggests that different patterns are sufficiently contrasted in terms of rival propositions. Hussy & Hussey (1997) identify a further refinement to the above by addressing the attributes of the unit of analysis.



○ **Identification of variables**

For a “*qualitative phenomenon*” they refer to a non-numerical attribute of an object such as hair colour, job grades, or gender. A “*quantitative variable*” refers to a numerical attribute of an individual or an object, such as age, height or weight. A positivistic study would in addition call for the classification of such variables into dependent and independent variables. In this respect an independent variable is the variable that can be manipulated in order to predict behaviour of the dependent variable.

According to Hussey & Hussey (1997) it is important to note that irrespective of the research paradigm (*positivistic* or *phenomenological*), one will always find a combination of qualitative and quantitative inputs into the data collection process. The following data collection techniques are adapted from their work.

○ **Data collection method**

The following methods were considered relevant to the research project:

▪ ***Critical incident technique***

This technique focuses on an observable activity, where the intended purpose is clear and the effect appears to be logical.

▪ ***Interviews***

Represents a method of collecting data where the subjects are asked questions in order to establish their experience base. A positivistic approach suggests structured questions where the respondent answers from a number of presentiment outcomes. With a phenomenological approach unstructured “*open-ended questions*” where the subject is enticed to give his own opinion, would be more appropriate.

▪ ***Participant Observation***

Is a method of collecting data where the researcher is fully involved with the participants and the phenomena being investigated.

### 2.2.3.3. *The role of theory in design*

Yin (2003) proclaims that theory development as part of the design phase is essential. The complete research design embodies a “theory” of what is being studied. The goal is to have some theoretical basis for formulating theoretical propositions, i.e. “*a story about why acts, events, structure, and thoughts occur*”.

### 2.2.3.4. *Criteria for judging the quality of research designs*

According to Yin (2003) four tests are commonly used to establish the quality of any empirical social research. Due to the commonality of the research approaches one could argue the relevance of these for any qualitative research. The tactics associated with each of these are represented in Table 2.1 (Yin, 2003: 34).

**Table 2.1: Tactics for ensuring quality research designs**

Test	Case Study Tactic	Research Phase
<b>Construct Validity</b>	<ul style="list-style-type: none"> <li>➤ Use multiple sources of evidence</li> <li>➤ Establish a chain of evidence</li> <li>➤ Key informant reviewing draft case study report</li> </ul>	<ul style="list-style-type: none"> <li>➤ Data collection</li> <li>➤ Data collection</li> <li>➤ Composition</li> </ul>
<b>Internal Validity</b>	<ul style="list-style-type: none"> <li>➤ Pattern matching</li> <li>➤ Explanation building</li> <li>➤ Addressing rival explanations</li> <li>➤ Arguments based on logic models</li> </ul>	<ul style="list-style-type: none"> <li>➤ Data analysis</li> <li>➤ Data analysis</li> <li>➤ Data analysis</li> <li>➤ Data analysis</li> </ul>
<b>External Validity</b>	<ul style="list-style-type: none"> <li>➤ Use of theory</li> <li>➤ Use of replication logic</li> </ul>	<ul style="list-style-type: none"> <li>➤ Research design</li> <li>➤ Research design</li> </ul>
<b>Reliability</b>	<ul style="list-style-type: none"> <li>➤ Use of case study protocol</li> <li>➤ Develop a case study database</li> </ul>	<ul style="list-style-type: none"> <li>➤ Research design*</li> <li>➤ Data collection</li> </ul>

(\* Yin (2003) reflects this issue as occurring in the data collection phase. Olivier (2004:75) presents a persuasive argument for addressing it during the design phase)



- **Reliability**

Reliability demonstrates that the operation of a study, such as the data collection procedure, can be repeated with the same results.

- **Construct validity**

Construct validity establishes correct operational measures for the concepts under investigation.

- **Internal validity (explanatory or causal studies only)**

Internal validity refers to establishing causal relationships whereby certain conditions are shown to lead to other conditions as distinguished from spurious relationships.

- **Pattern matching or relying on theoretical propositions**

A promising approach is that of Campbell & Fiske (1959) whereby several pieces of information from the same case may be related to some theoretical proposition. According to Yin (2003) the preferred strategy is to follow the theoretical propositions that led to the case study. Theoretical propositions about causal relations can be very useful in guiding case study analysis.

- **Explanation building**

Explanation building is considered by Yin (2003:120) as a special case of pattern matching. The goal is to analyse the case data and to build an explanation about the case. *“To explain a phenomenon is to stipulate a presumed set of causal links about it”*. In most case studies explanation occurs in a narrative form and according to Yin the better case studies are those where the explanations have reflected some *“significant theoretical propositions”*.

- **Addressing rival explanations**

Rival explanations could be based on for example current existing theoretical frameworks against which the new theoretical concepts could be compared (Yin, 2003). Yin reflects on a brief description of the different rival explanations from one

of his earlier works (Yin, 2003). An adapted table of examples are provided in Table 2.2.

**Table 2.2: Different kinds of rival explanations**

Type of Rival	Description or example of cause
<b><i>Craft rivals:</i></b> The Null Hypothesis Threats to Validity Investigator Bias	The observation is the result of chance observation Instability; testing Experimenter effect
<b><i>Real-life rivals:</i></b> Direct rival Commingled rival Implementation rival Rival theory	Another intervention than the target intervention Other interventions plus the target intervention Implementation process rather than targeted intervention A different theory explains results

- **Using logic models**

“The use of logic models as an analytic technique consists of matching empirical observed events to theoretical predicted events” (Yin, 2003: 127). The logic models are distinguished from pattern matching in the sense that it consists of sequential stages where outcomes are differentiated in terms of *immediate*, *intermediate* and *ultimate* outcomes. The logic models thus have a “*programme logic*” that could predict these progressive developments to the eventual *ultimate* outcome.

- **External validity**

External validity refers to establishing the domain to which a study’s findings can be generalised. With regard to the previous two issues Olivier (2004), is of the opinion that this represents the major concerns with any experimentation. Firstly, that the observations are indeed caused by experimental inputs alone and nothing else (*internal validity*), and secondly that the results of the experimentation can be generalised (*external validity*). Most of these problems associated with validity can be addressed during the design phase in the compilation of the research protocol. The protocol defines the research procedure and rationale thereof, and which could be



scrutinised prior to experimentation. Validity in exploratory work according to Olivier (2004) is less of an issue due to the nature of the research, but it remains the product of careful deliberation.

#### ***2.2.3.5. Case study designs***

Yin (2003) proposes four basic types of designs for case studies that are a combination of choices in terms of whether a single or a multiple cases study research approach will be followed; as well as the choice in terms of the unit of analysis that will be selected. In a situation where multiple units of analysis are selected, the research design is referred to as an embedded design. These choices will be briefly reviewed below:

- **Single or multiple designs:**

A number of considerations would influence one's choice in terms of a single or multiple case study design. The following rationale is presented for the use of single case study research:

- **Single case study designs**

##### ***The critical case***

The critical case is used to test a well-formulated theory. The theory has a clear set of propositions which are believed to be true given a specified contextual environment. One may wish to use a single case study to confirm, challenge or to extend the theory.

##### ***An extreme or unique case***

This occurs when a very rare phenomenon is investigated such as a specific injury or disorder where it is considered to be worthwhile to document and analyse the case.

##### ***The representative or typical case***

The objective here is to record what is believed to be a typical representative case that could be used for informative purposes.



### *The revelatory case*

The revelatory case is sited as a situation where an investigator has the unique opportunity to investigate a phenomenon that was previously inaccessible to scientific research.

### *Longitudinal case*

The longitudinal case deals with the same case over an extended period of time where the objective is to observe how conditions change over time.

#### ▪ **Multiple case study designs**

As far as Yin (2003) is concerned, there are no methodological differences between a multiple and single case studies, as found in anthropology, and political sciences. In this regard he cites Eckstein (1975), Lijphart (1975) and George (1979).

Multiple case studies however holds distinct advantages from the perspective that the evidence from such studies are often more compelling and the study thus regarded as more robust. The underlying logic for selecting a multiple case design is the same. According to Yin (2003) each case must be carefully selected in order to achieve one of the following objectives. It must either predict similar results (i.e. *literal replication* of results) or predicts contrasting results but for predictable reasons (*theoretical replication*). The importance of a rich theoretical framework to enable such replication is thus strongly emphasised by him. The framework should thus state under which conditions the particular phenomenon is likely to occur (*literal replication*) and under which conditions not (*theoretical replication*).

It is important to realise the methodological differences between “*replication logic*” in multiple case study designs and “*sampling logic*” as encountered in multiple subjects in an experiment. A major insight is to consider multiple cases as one would consider multiple experiments. Any application of sampling logic would be misplaced. The following reasons cited by Yin (2003) are briefly listed:

- i. *Case studies are not the best method for assessing the prevalence of a phenomenon.*



- ii. *If used for such purposes the case study will have to cover both phenomenon and contextual variables which would require very large sample sizes to enable any statistical consideration.*
- iii. *If sampling logic had to be applied to all types of research, many important topics could not be empirically investigated.*

It is also important to note that each case study is a “*whole study*” in which converging evidence is sought regarding facts and conclusions. A last comment on this issue is that it is not uncommon to adapt or modify the design of subsequent cases in order to accommodate new insights. Without such “*redesigns*”, according to Yin (2003), one risks being accused of distorting the enquiry in order to accommodate the original design.

- o **Holistic or imbedded designs:**

Holistic versus imbedded designs deals with the unit of analysis. Holistic designs deals with only one unit of analysis where the multiple case study design deals with multiple units of analysis, i.e. subunits within the unit.

- **Holistic designs**

Holistic case studies are shaped by a thorough qualitative approach that relies on narrative, phenomenological descriptions (Scholz & Tietje, 2002). Scholz & Tietje cite Stake (1995) who claims that themes and hypotheses may be important but should remain subordinate to the understanding of the case.

- **Imbedded designs**

Embedded case studies according to Scholz & Tietje (2002) involve more than one unit, or object, of analysis and are not limited to qualitative analysis. It allows for the analysis of a multiplicity of evidence in “*subunits, which focus on different salient aspects of the case*”.

The combinations of alternative designs that one can create based on the selection of the aforementioned alternatives, are reflected as a two-by-two matrix which is shown

in Table 2.3. These alternatives are referred to as *Type 1*, *Type 2*, *Type 3*, and *Type 4* designs respectively.

**Table 2.3: Basic types of designs for case studies**

Type	Single-case design	Multiple-case design
<b>Holistic</b>	<b>Type 1</b> Single-case; single unit of analysis	<b>Type 3</b> Multi-case; single unit of analysis
<b>Embedded</b>	<b>Type 2</b> Single-case: multiple units of analysis	<b>Type 4</b> Multi-case: multiple units of analysis

○ **The design as a process perspective**

Yin (1994) describes case study design as a process consisting of the following steps:

▪ **Step 1: Develop a hypothesised understanding of the case study**

Yin (1994) stresses the importance of having a thorough understanding of the intended operation and the expected outcomes with explicit attention to the contextual conditions. He also highlights the importance of being open to the potential need for modification later on. According to him the programme should at least reflect:

- The “*programme logic model*” tracing the causal flows of the programme, and
- An emerging taxonomy of contextual conditions within which the programme operates.

▪ **Step 2: Emerge this understanding within previous research**

By emerging this understanding within previous research one should be able to identify rival theories and hypothesis for the programme (*phenomenon under investigation*). This holds the following potential benefits:

- The hypothesised understanding of the programme may be clarified further.
- Rival theories will lead to potent strategies for analysing data.
- The broader range of theory and practice will be the main vehicle for generalising the results of the evaluation.



▪ **Step 3: Tentatively define the unit(s) of analysis**

Define the main units and sub-units of analysis.

▪ **Step 4: Establish a schedule and procedure for interim reports**

Although Yin (2003) highlights the need for a formal schedule to review interim and final results of the study, it is not clear from the text to exactly what the benefits involved are. Two of the potential benefits could include:

- Formal feedback on the progressive development of the research project provides for the opportunity to reflect on the progress made and allows for redirection of efforts in resolving the research questions involved.
- Such a schedule will pace the execution of the research.

▪ **Step 5: Define and test instruments, protocols and field procedures**

The testing and validation of the aforementioned are crucial to the validity of results.

▪ **Step 6: Collect, analyse and synthesise data**

It is important to realise that the data collection and analysis are likely to occur in an intermingled way and not chronologically. It is essential to document the methodological steps thoroughly in order to ensure an unbiased data collection process despite variation in individual cases.

▪ **Step 7: Create a data base**

Organise both quantitative and qualitative data systematically to permit an efficient access process.

▪ **Step 8: Analyse the evidence**

The data analysis process could evolve a wide range of techniques and it is also important to realise that the analysis process might identify the need for additional data collecting – again with the necessary methodological caution.

- **Step 9: Compose the case study report**

Essential characteristics of the report are that it is separate from the database and that it contains explicit presentations of the key evidence used to draw conclusions.

Although not explicitly stated, it is assumed that the case study design must address all of the above.

#### ***2.2.3.6. Dimension and classification of case studies***

Scholz & Tietje (2002) provide the following framework for the classification of case studies which is reflected in Table 2.4.

**Table 2.4: Dimensions and classifications of case studies**

<b>Dimension</b>	<b>Classification</b>
<b>Design</b>	Holistic or imbedded Single case or multiple case
<b>Motivation</b>	Intrinsic or instrumental
<b>Epistemological status</b>	Exploratory, descriptive, or explanatory
<b>Purpose</b>	Research, teaching or action/application
<b>Data</b>	Qualitative or/and quantitative
<b>Format</b>	Highly structured, short vignettes Unstructured or ground braking
<b>Synthesis</b>	Informal, emphatic, or intuitive Formative or method driven

Although most of the aspects reflected in Table 2.4 have been covered in the previous sections, it is worth while to review some of the dimensions listed.

#### ***Intrinsic and instrumental***

Intrinsic motivation refers to research undertaken purely as a result of the interest of the researcher and is aimed solely at understanding the particular case. If the objective



differs, it becomes instrumental. Case studies that are aimed at furthering scientific knowledge thus become instrumental.

### ***Epistemological status***

Epistemological status refers to the classification as either exploratory, teaching, or action/application which has been discussed in the introduction to case study research.

### ***Format***

The format of the case study would vary according to the nature of the research with a lesser degree of structure with exploratory and groundbreaking research.

### ***Synthesis***

The classification in terms of synthesis deals with the manner in which the case study deals with knowledge integration. Holistic case studies tend to be narrative by nature whilst the embedded case study normally deals with both quantitative and qualitative data, and associated strategies for integration (Scholz & Tietje, 2002). The aforementioned researchers make use of the so-called “Brunswikian lens model” for synthesis and knowledge integration. These powerful concepts are discussed in 2.2.4.

#### ***2.2.3.7. Protocol***

According to Yin (2003) the protocol contains instruments as well as the procedures and general rules that must be used in the application of the protocol. He also states that protocol is directed at the investigator or researcher and not the entity that the research is dealing with. He also concludes that a protocol is advisable in all circumstances, but essential for multiple case studies.

### ***Protocol structure***

The structure of the protocol can be briefly summarised as follows:



○ **Overview**

- Background
- Substantive issues being investigated
- Relevant readings about the issue

○ **Procedure**

- The procedure to be followed
- The consent form

○ **Case study questions**

- General orientation: - the questions are directed at the researcher as a reminder of what information is required, and not at the interviewee. Each question should be accompanied by a list of likely sources of evidence.

*Note: It is important to note that the questions form the structure of the enquiry and are not intended as literal questions to be asked of the interviewee. It thus provides guidance to the researcher.*

- Level of questions:
  - i. Level 1: Questions asked of a specific interviewee.
  - ii. Level 2: Questions to be answered by the investigator for every individual case.
  - iii. Level 3: Questions asked of the pattern of findings across multiple cases.
  - iv. Level 4: Questions asked of an entire study, e.g. calling on information beyond the case study evidence, including literature or other published data.
  - v. Level 5: Normative questions about recommendations and conclusions beyond the narrow scope of the study.

○ **Collecting data – sources of information**

- Documentation: - for case studies the use of documents are corroborative and augments evidence from other sources.



- Archival records: - Other formal records to support evidence.
- Interviews: - It is worthy to note the open-ended nature of questions.
- Direct observations, inclusive of participant observations by the researcher.

- **Reporting Case studies**

Yin (2003) proposes the following criteria for exemplary case studies.

- **Case studies must be significant**

Exemplary case studies anticipate obvious alternatives and even advocate their positions as forcefully as possible, and then show empirically on what basis these should be rejected.

- **Case studies must display sufficient evidence**

Critical pieces of evidence must be contained within the case study report. An exemplary case study represents judiciously and effectively presents the most relevant evidence, so that the reader can reach an independent judgement regarding the merits of the analysis. With multiple case studies the researcher should convince the reader that the individual case studies have been treated fairly and that cross-case conclusions have not been biased by undue attention to one or a few of the array of cases.

Finally evidence should be provided about the validity of the evidence. It does not imply that all the cases must be burdened with the full methodological treatises. A few judicious footnotes or words in the preface of the case study can cover the critical validation steps.

- **Case studies must be composed in an engaging manner:**

The report should be engaging. For written reports it implies a clear writing style, which entices the reader to continue reading.



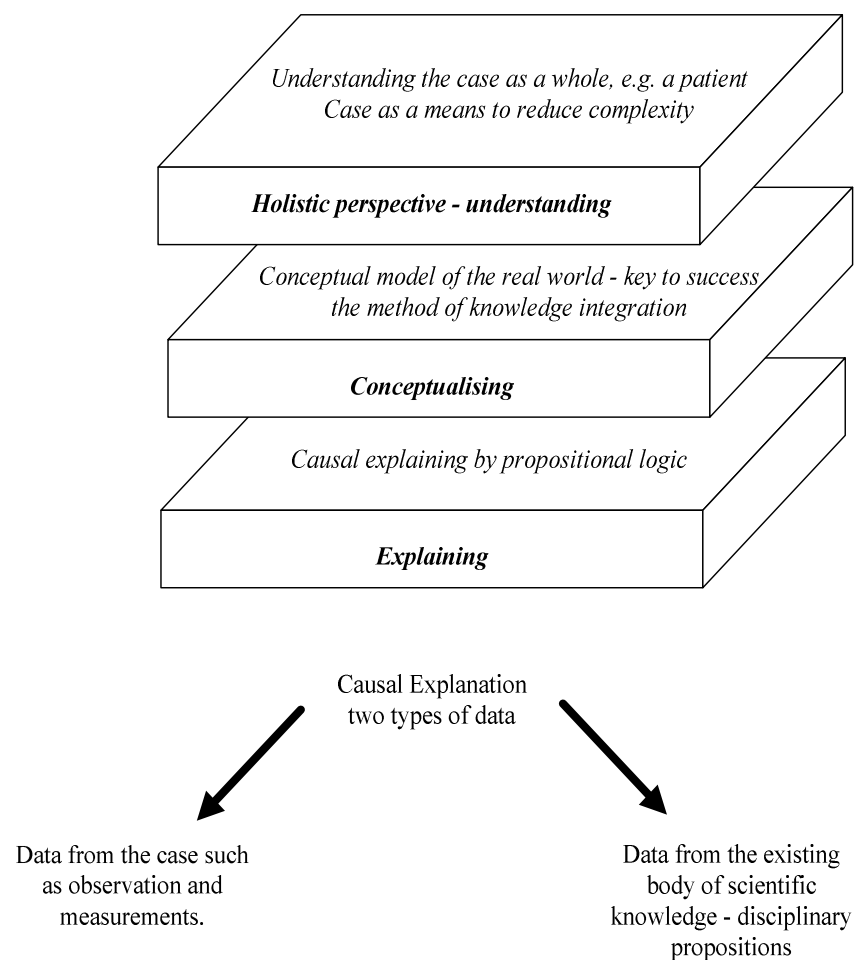
In the next section the synthesis and integration of knowledge will be discussed. It will also include the so-called “Brunswikian lens model” as the basis for alternative models for synthesis and knowledge integration.

### 2.2.3.8. *Synthesis and knowledge integration in imbedded case study research*

This section deals with the architecture of knowledge integration in embedded case studies as well as an overview of methods that could be used in this regard.

- **Levels of knowledge**

Scholz & Tietje (2002) postulate that case studies should be organised and structured on three levels which are represented in Figure 2.1.



**Figure 2.1: Theory of knowledge**

On the first level the objective is to reduce the complexity associated with the discourse of general problems, to one individual state of affairs as encapsulated by the case.

On the second level the case is generalised through the creation of a conceptual model. According to Scholz & Tietje (2002) the key to success on this level lies in the methods used for knowledge integration. These methods aid in the creation of valid understanding and become the vehicles for knowledge integration. This researcher equates this second level with the theoretical model as advocated by Yin (2003).

On the third level are scattered data and results from subprojects. Two types of data exist. The first is the outputs from the case study research, such as data, observations, measurements, surveys, documents and expertise. The second is data from the existing body of scientific knowledge in the form of disciplinary propositions. With regard to the second data element, namely an existing theoretical model, one could argue that if it forms the basis of the replication logic as advocated by Yin (2003) and that it could be considered as level-two knowledge. This argument is based on the fact that Scholz & Tietje (2002) see level two as the vehicle for knowledge integration, and thus for the generalisation of findings.

- **Strategies for synthesis**

Scholz & Tietje (2002) see synthesis as the scientific combination of often-varied data, information and ideas into a consistent whole. Knowledge integration according to them can be seen as a kind of synthesis designed specifically for each case study type. A number of approaches to synthesis exist and a few will be briefly reviewed (Scholz & Tietje, 2002).

- **Synthesis based on an epistemological perspective**

The authors cite four types of synthesis based on an epistemological perspective, namely: synthesis as a philosophical strategy of contemplation, synthesis through a pure case model, synthesis as a pre-stage of higher conceptual knowledge and synthesis as a method for complex problem solving.



### ***Synthesis as a philosophical strategy of contemplation***

Synthesis as a philosophical strategy of contemplation reverts back to a pre-scientific, contemplative approach where case results are the basis for reflection in order to gain understanding and insight into the meaning, true nature and essence of the case study

### ***Synthesis through a pure case model***

The pure case model is based on the so-called Leibnizian model. Leibniz (1664-1716) proposed that one could descend from the general rules of truth to the composed. A critical element in this argument is that the rules of truth had to be found by analytical methods. The most general truths are the natural laws. The authors cite Churchman (1971) who noted that the Leibnizian model relied on the assumption that the truth was provided in the model.

The authors conclude that the Leibnizian model as a strategy for synthesis is appropriate at least as a partial synthesis, particularly for natural sciences.

### ***Synthesis as a pre-stage of higher conceptual knowledge***

Scholz and Tietje (2002) compare analytic and synthetic reasoning at the hand of the comparison between concept and perception as proposed by Immanuel Kant (1724-1804), in the sense that both are regarded as prerequisites for analytical reasoning and theoretical concepts. The following quote from Scholz and Tietje (2002:34) encapsulates the essence of this line of reasoning.

*“The validation of hypothesis-based theory testing thus shows some reference to the Kantian concept and perception. The perceptual side represented by the theories and their hypotheses, and the analytical side is represented by the observed data. Empirical data substantiates, differentiates, and refutes the theories. Clearly, in the Kantian type of inquiry, the theory, not the case, is the point of reference”.*

### *Synthesis as a method for complex problem solving*

This strategy is based on the Hegelian model of inquiry (Schulz & Tietje, 2002). This approach follows an intermediate state of analysis that consists of different and even contradictory models of the case. According to this perspective the truth is approached in a dialectic way. The synthesis incorporates certain aspects from different world models which result in a new, larger model. This approach is particularly appropriate for ill-structured problems such as management, education, politics, urban planning and environmental sciences according to the authors.

#### ○ **Types of knowledge integration**

Schulz and Tietje (2002) distinguish between four types of knowledge integration. These are briefly listed as an additional element of contextualisation of the research.

##### ▪ **Disciplines**

This method of integration looks at the integration of knowledge from a disciplinary context, such as natural sciences and the arts.

##### ▪ **Systems**

The systems approach differentiates based on the subsystems that exist within the larger system boundaries. According to the authors the synthesis of subsystem can often be considered as a partial synthesis that are then subject to further knowledge integration and synthesis.

##### ▪ **Interests**

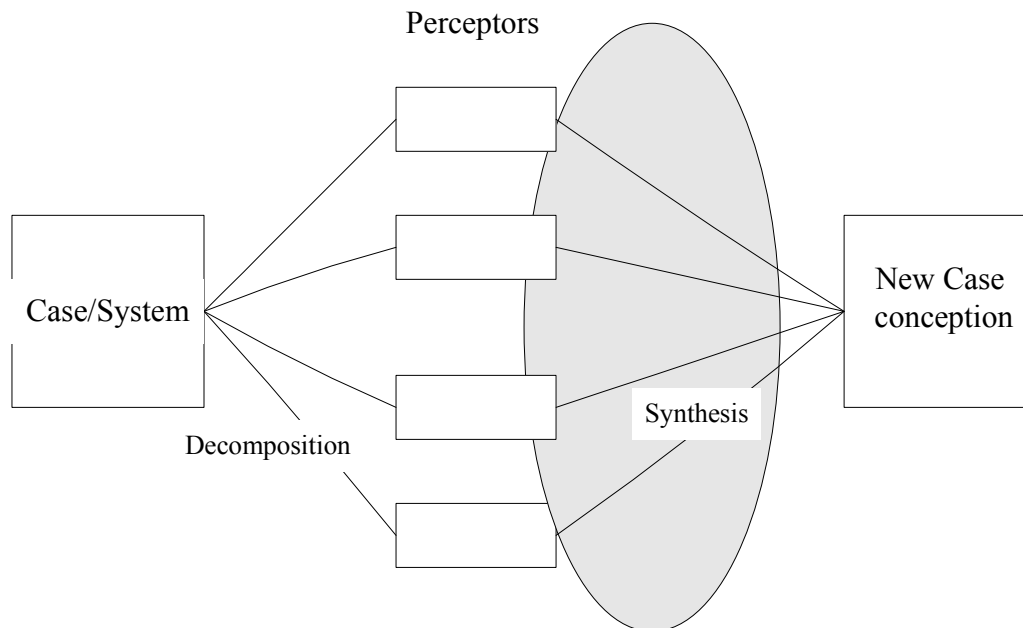
The use of interest as the basis for integration and synthesis could for example include the mediation of values between the various interest groups involved in the case.

##### ▪ **Modes of thought**

This aspect refers to the way in which the participants view the case from a cogitative or conceptual perspective, e.g. left-brain versus right-brain thinking. It refers to the way in which the case mentally or externally represented.

○ **The Brunswikian Lens Model**

Schulz and Tietje (2002) have adapted the so called Brunswikian Lens Model as a generic framework that they use as an integrating framework for a variety of methods for knowledge integration. The model was initially developed by an experimental psychologist who worked in the field of perception (Brunswik & Tolman, 1935).



**Figure 2.2: Brunswikian Lens Model**

The model as developed by Brunswik and Tolman (1935) is fairly complex and technical and has no direct bearing on this research. The basic model however provides a useful conceptual framework for dealing with knowledge integration which is used in this research. In essence the process model deals with the decomposition of the case under consideration into subunits or functions. An assessment and analysis of these subunits are performed through a process of perception performed by so called *preceptors*. The integration of these different perceptions leads to a new conception of the case under consideration. The basic model is reflected in Figure 2.2.

Although a wide variety of models are referred to in the work of Schulz & Tietje (2002), only three that are considered to be relevant in terms of this research will be briefly listed in the following section.

- **Different types of knowledge integration**

- **Formative Scenario Analysis**

Formative Scenario Analysis generates hypothetical future states of a system/case which are referred to as scenarios that assist in the process of gaining insight into the system and its dynamics. The scenarios are based on a sufficient set of system variables and are judged according to possibility and consistency.

- **System Dynamics**

System Dynamics deals with a family of mathematical models that provide insight into the dynamic characteristics of the system. It thus not only provides insight into the eventual steady state of the system, but also address the transient behaviour, i.e. how the characteristics of the system, or variables, change with time.

- **Multi-Attribute Utility Theory**

Multi-Attribute Utility Theory represents a group of methods that describes and models integral evaluations based on different attributes. The criteria may represent different interests, subsystems or disciplinary perspectives.

Although none of these models as presented in the work of Schulz & Tietje (2002), is applicable in the form as presented, the conceptual frameworks in conjunction with the Brunswikian Lens Model is considered useful for handling knowledge integration in the context of this research project. This is especially applicable projects of a mixed-methodological nature which followed with this research.

#### ***2.2.3.9. Case study protocol as design***

In the previous sections a number of perspectives on the design of case studies have been presented. None of these perspectives provide a clear concise definition of exactly what such a design entails. A strong common ground exists between a number of authors such as Yin (2003), Olivier (2004) and Payne and Williams (2005) in that they see the protocol as a significant mechanism to define the scope and extent of the



research. It also serves as a crucial role in defining the approach and ground rules and thus provides a reflection of the validity of the research.

In this research the *design* of the case study will be referred to as the *protocol* of the research. The objectives of the protocol remain the same as what is propagated by the aforementioned authors in terms of the design, i.e. to provide direction, methodology, structure, instruments, procedures, validation requirements, to name a few, as a guide to the researcher for getting from the beginning to the end of the research project.

In an attempt to ensure that the collective perspectives of the authors covered in the literature research, are embodied in the protocol, use has been made of a conformance matrix which is represented in Table 2.5. The objective with the matrix was to ensure that all the significant issues referred to in the literature research (i.e. those issues reflected on the left hand side of the table) were reflected in the subheadings that were created for the protocol, and that all these issues are incorporated in the case study design.

This section provides a brief overview and background to the particular section of the research. It also reflects the classification of the case study research approach.

The case study protocol used in this thesis consists of the following sections.

- **Overview**

A brief overview of the objectives and classification of the research is provided.

- **Study's questions**

The case study's questions provide the researcher with a general orientation to the answers that are required with reference to the likely sources of evidence. It will also classify the questions into the various levels as discussed earlier.

- **Theoretical framework**

This section will cover the underpinning of the research questions in the form of the theoretical model or hypothesis that has been developed in support of the research objective.

**Table 2.5: Case study protocol conformance matrix**

	Overview	Study's Questions	Theoretical framework	Propositions	Rival theories	Units of analysis	Schedule	Procedures	Data collection	Data analysis	Synthesis	Database	Knowledge integration	Consent	Quality assessment
Design classification	x														
<i>Design components</i>															
Study's questions		x													
Theoretical framework			x												
Propositions				x											
Unit of analysis						x									
Variables								x							
Data collection method									x						
Role of theory			x		x										
Quality assessment															x
<i>Protocol</i>															
Overview	x														
Procedure								x							
Consent														x	
Case study's questions		x													
Data collection									x						
Synthesis											x				
<i>Process perspective</i>															
Theoretical base (1)			x												
Rival theories (2)					x										
Units of analysis (3)						x									
Schedule & procedure (4)							x								
Define instruments, etc (5)								x							
Test instruments (5)								x							
Collect data (6)									x						
Analyse data (6)										x					
Synthesis (6)											x				
Create database (7)												x			
Knowledge integration (8)													x		





- **Propositions**

The propositions or expectations in terms of research outcomes based on the theoretical framework.

- **Rival theories**

This section covers the rival theories to the postulated theoretical framework. This includes the Null hypothesis, i.e. that the observations is a result of chance; and that a different theoretical model exists that will explain the observations, or deductions made.

- **Unit(s) of analysis**

The units of analysis are defined in relation to the level of research questions that are being posed.

- **Schedule and reviews**

The schedule reflects the planned feedback on progress made as well as the time frame for the research.

- **Instruments and procedures**

The section on instruments and procedures addresses two aspects, namely the definition as well as the validation of the instruments and procedures that are to be used in the research project.

- **Data collection**

This section defines the data collection methods to be used, as well as how the data collection process will be handled.

- **Data analysis**

The data analysis section will define how the data will be analysed.



- **Synthesis**

This section deals with the synthesis of data and observation by integrating it with the theoretical propositions to establish new concepts of causal relationships. Within this thesis a distinction is made between synthesis and knowledge integration. The last concept is reserved for the final integration of the results from embedded case studies.

- **Database**

This section deals with the structure and format of the database for the recording and retrieving of data.

- **Knowledge integration**

This section deals with the higher levels of knowledge integration of embedded case studies as covered in 2.3. It deals with the final conceptualisation of the case based on the triangulation of results and adapting the holistic understanding of the research case.

- **Informed consent**

This section contains the informed consents from the subjects involved in the case studies.

- **Quality assessment**

This section incorporates the aspects discussed in section 2.2.3.4 on the criteria for the quality of research designs. It deals with the measures for construct, internal, external validity and the reliability of the case study process. In this thesis the protocol will be defined for a group of similar case studies, with annotation of individual deviations as they occur.

#### **2.2.4. Experimental study designs**

According to Olivier (2004) the researcher is confronted with two major concerns. The first is that the observations are indeed caused by the experimental inputs and nothing else. The second is whether the results observed during the experiment can be



generalised. The reader will be able to relate these concerns to that which was discussed earlier in the section on case study design. The first concern thus deals with *internal validity* and the second as *external validity*. The use of control groups is a common technique to ensure internal validity for experimental research or comparisons of techniques. The discussion that follows is based on extracts from the work by Olivier (2004).

- **Two-group experimental designs**

This design selects the subjects for the experimentation, and then splits the group randomly into two. The one group becomes the focus of the intended experimentation whilst the second group fulfils the role of a control group. A fundamental assumption of the technique is that since the groups were randomly composed, that they will be equal in all respects. The potential problem with two-group experiments is that the two groups are not the same. In such a situation one-group experiments are attractive.

- **Single groups**

Single group experiments are experiments where the subjects are subjected to observation of their behaviour with the *unmodified* system, and then subsequently subjected to the modified variables. The group thus become their *own control*.

- **Blind experiments (application by others)**

The aim of blind experiments is in essence to eliminate the psychological effects that the knowledge of the experimentation have on both the participants (subjects) and the researcher. Blind experimentation in general refers to the situation where the participant is not aware whether he is being subjected to the control treatment or the actual experimentation. Double blind refers to the situation where neither the subject nor the researcher is aware of what the treatment entails, i.e. the control treatment or the actual experimental treatment. In certain circumstances blind experimentation could also imply a situation where the application of experimental activities is performed by people other than the researcher.



The research was primarily based on multiple case study designs although blind experimentation was employed during some of the experimental research projects. The decision to use case studies as the primary research approach was based on the fact that the literature research revealed no information on conservative treatment approaches aimed specifically at the fascia. This forced the research to address the problem from an exploratory perspective for which a series of case studies as an exploratory device is considered as the most appropriate (Holloway, 1997).

The assessment, management and outcomes of the different subjects were described as separate case studies. Each case study was analysed individually and the knowledge gained was progressively integrated in the approaches followed with subsequent case studies. Thereafter the results of the separate case studies were compared (cross case) to one another (Yin, 1994; McDonnell *et al.*, 2000).

### **2.2.5. Conclusion**

Case study research as a research methodology has been reviewed in a fair amount of detail. This has been done due to the fact that qualitative research methodologies are not that common in the research field. It is also important that the concept of replication is thoroughly understood due to the vital role that the replication of results plays in the research project. Results are predicted based on the causal relationships of an underlying theoretical model that the researcher tries to prove by means of the replication of predicted results derived from the model.

## **2.3. POSTERIOR COMPARTMENT SYNDROME**

### **2.3.1. Introduction**

Exercise related injuries of the lower leg are often encountered in athletes (Mouhsine *et al.*, 2006). Although it is generally accepted that the chronic compartment syndrome is as a result of exertion and/or overuse (Allen & Barnes, 1986; Balduini *et al.*, 1993), experimental results suggest that other factors are responsible for the condition (Reinking, 2006). Reinking (2006) found little evidence to support the



claims that extrinsic factors such as training volume, training surface, shoes and sport activities are the origin of the problem.

Chronic compartment syndrome is however also difficult to diagnose (Edwards *et al.*, 2005) and is as a result a problem not only to the athletes but also to the health care professionals. Even in situations where the condition is correctly diagnosed, the treatment interventions are not always successful (Edwards *et al.*, 2005; Mouhsine *et al.*, 2006). It is generally accepted that the condition is as a result of non-compliant fascia that encapsulates the compartment (Qvarfordt *et al.*, 1983; Detmer *et al.*, 1985). The use of fasciotomy as intervention is often selected but also often without the required successes (Mouhsine *et al.*, 2006). The athletes are understandably also not keen on a surgical intervention, while the conservative option has not produced any long term solutions to the problem. Cases are encountered where subjects were subjected to prolonged treatment and examination where the physicians fail to successfully diagnose and treat the subject (Mirabelli & Dimeff, 2006). In these cases the subject often forfeit their sport activities as a result of the pain induced during sport activity. The success rate of fasciotomy with the surgical decompression of the posterior compartment varies between 50% and 75% (Slimmon *et al.*, 2002).

This portion of the literature review focuses on the investigation of CPCS. The objective is to provide a comprehensive overview of the current theoretical perspective of the condition, inclusive of the pathogenesis of CPCS. This also includes the identification of factors that might precipitate or perpetuate the symptoms as well as factors that could contribute to the development of a successful conservative treatment approach to the treatment of the symptoms of CPCS in runners.

### **2.3.2. Chronic Compartment Syndrome**

#### **2.3.2.1. *Anatomy of compartments and their muscles***

According to anatomical texts, the leg is divided into four separate osseofascial muscle compartments, namely the anterior compartment, the lateral compartment, the deep posterior compartment and the superficial posterior compartment. All of the



compartments have both osseous and fascial borders, with the exception of the superior posterior compartment, which has only fascial borders. Each compartment contains specific muscle groups and associated tendons, blood vessels and nerves. The localization and diagnosis of the impaired compartment are based on a sound knowledge of the different anatomical structures in the lower leg (Ross, 1996). Davey *et al.* (1984) described a fifth compartment, namely that of the tibialis posterior muscle. According to them, the tibialis posterior muscle's compartment is contained in a separate osseofascial muscle compartment in which an isolated exertional compartment syndrome can occur. Detmer *et al.* (1985) take this view one step further by saying that their experience suggests that the arrangement of muscles within the four compartments is such that chronic compartment syndrome can develop within one subdivision of a compartment without involving other muscles within the same compartment. They conceptualize the leg as having seven functional compartments, namely the anterior-, the lateral-, the posterior superficial medial-, the posterior superficial lateral-, the posterior deep proximal-, the posterior deep distal (flexor digitorum longus, flexor hallucis longus and tibialis posterior) and the posterior superficial distal (distal soleus) compartment. The various compartments of the lower leg and the muscles they contain are reflected in Table 2.6.

#### **2.3.2.2. Definition of chronic posterior compartment syndrome)**

Chronic compartment syndrome is a pathological condition of skeletal muscle characterized by increased interstitial pressure within an anatomically confined muscle compartment which interferes with the circulation and function of the muscle and neurovascular components of the compartment (Nicholas & Herschman, 1995a). When this occurs in the posterior compartment of the lower leg, it is called a posterior compartment syndrome. From Table 2.6, it can be seen that the muscles that could be involved with a posterior compartment syndrome are the tibialis posterior-, the flexor digitorum longus-, the flexor hallucis longus-, the gastrocnemius-, the soleus- and the plantaris muscles. The neurovascular components that can possibly be involved are the posterior tibial- and the sural nerves. A condition is usually defined as chronic when it has been present for a period of longer than three months (Klenerman *et al.*, 1995; Von Korff & Saunders, 1996; Grabis, 2005).

**Table 2.6: Summary of the anatomy of the compartments of the lower leg (Bouche, 1990)**

<i>Compartment</i>	<i>Muscles</i>	<i>Vessels</i>	<i>Nerves</i>	<i>Weakness</i>	<i>Paraesthesia</i>	<i>Pain</i>	<i>Tenderness and tenseness</i>
Anterior	1.Tibialis anterior 2.Extensor hallucis longus 3.Extensor digitorum longus 4.Peroneus tertius	Anterior tibial artery and vein.	Deep peroneal nerve.	Ankle dorsi flexion and toe extension.	1 <sup>st</sup> interspace between big and 2 <sup>nd</sup> toes.	Ankle plantar flexion and toe flexion.	Anterior leg.
Lateral	1.Peroneus longus 2.Peroneus brevis	None.	Superficial peroneal nerve.	Ankle dorsi flexion and eversion.	Dorsum of foot.	Ankle plantar flexion and foot eversion.	Lateral leg.
Deep posterior	1.Tibialis posterior 2.Flexor digitorum longus 3.Flexor hallucis longus	Peroneal artery and vein Posterior tibial artery and vein.	Posterior tibial nerve.	Ankle plantar flexion; eversion and toe flexion.	Sole of foot.	Ankle dorsi flexion; foot eversion and toe flexion .	Medial leg.
Superficial posterior	1.Gastrocnemius 2.Soleus 3.Plantaris	None.	Sural nerve.	Ankle plantar flexion.	Dorsum and lateral foot.	Ankle dorsi flexion.	Calf.



Terms sometimes used as synonyms for chronic compartment syndrome (CCS) are *shin splints*, *medial tibial stress syndrome* and *chronic exertional compartment syndrome*. These terms will be described briefly.

The American Medical Association (AMA) has defined shin splint syndrome as “*pain and discomfort in the leg from repetitive activity on hard surfaces, or due to forceful, excessive use of foot flexors*” (AMA, 1966, pp 126). The AMA states that the diagnosis should be limited to musculo-skeletal inflammatory conditions; excluding stress fractures and ischemic disorders. Batt *et al.* (1998) agrees with the definition of the American Medical Association and feels that the term shin splints should be applied to any exertional lower leg pain not caused by stress fractures, compartment syndrome or muscle hernia. From this description it is clear that the phrase “shin splints” does not qualify as a synonym for compartment syndrome.

The term *medial tibial stress syndrome* seems to be closer in pathogenesis to compartment syndrome. Puranen (1974) believes the medial tibial stress syndrome is a deep posterior compartment syndrome caused by exercise-induced ischemia of the muscle in the medial fascial compartment of the leg. Mubarak *et al.* (1982) on the other hand have been unable to demonstrate an elevated pressure in the deep posterior compartment in patients diagnosed with medial tibial stress syndrome and have questioned the statement of Puranen (1974) that this condition is a compartment syndrome. A further opinion is that of Touliopolous and Herschman (1999) who state that medial stress syndrome is a result of injury involving the fascial origin of the soleus muscle or the periosteum beneath the origin of the tibialis posterior muscle.

In their view subjects diagnosed with a medial stress syndrome, experience pain over the middle and distal one third of the posterior-medial part of the tibia. The pain increases with activity and decreases with rest. According to Touliopolous & Herschman (1999), only two conditions can lead to the diagnosis of medial stress syndrome, namely a stress fracture or CPCS. If X-rays or a bone scan do not confirm a diagnosis of a stress fracture, the cause of medial tibial syndrome is CPCS. Detmer *et al.* (1985) has classified medial tibial stress syndrome into three categories, depending on the symptoms present. In the third category, which is a progression of the first two





categories, Detmer *et al.* (1985) equates the medial tibial stress syndrome to a CPCS. This indicates the existence of a grey area between the CPCS and the medial stress syndrome in which it is not always possible to differentiate between the two conditions.

According to Wallensten (1983) there are two clinical conditions that can become chronic in spite of adequate non-surgical treatment, namely the medial tibial syndrome which he sometimes refers to as *shin splints* or *periostitis* and the *compartment syndrome*. Both these entities have, according to him, responded better to surgical than non-surgical treatment. As seen from the above discussion, it is very difficult to differentiate between a medial stress syndrome and a CPCS since the symptoms are similar and their response to both non-surgical and surgical management is very similar as well.

The term *chronic exertional compartment syndrome (CECS)* on the other hand may be a true synonym for CCS. Most authors agree on a definition for CCS that indicates a clinical condition that arises when increased pressure within a closed anatomical space compromises the function and blood flow within the area. The pressure usually rise transiently following repetitive motion or exercise, thereby producing temporary, reversible ischemia, pain, weakness and occasionally neurological deficits (Bouche, 1990; Hutchinson & Ireland, 1994; Howard *et al.*, 2000). The term *exertional compartment syndrome* indicates that exertion is the cause of the raised interstitial pressure in the compartment.

The definition of CPCS which will be accepted for the discussion of the literature review and the application of the research methodology of this study is the following: “Chronic Posterior Compartment Syndrome is a pathological condition of skeletal muscle that has been present for a period of longer than three to six months. It is characterized by an increased interstitial pressure within the posterior muscle compartment of the lower leg which interferes with the circulation and function of the muscle and neurovascular components of the posterior compartment”.

#### **2.3.2.3. Pathogenesis of chronic posterior compartment syndrome.**

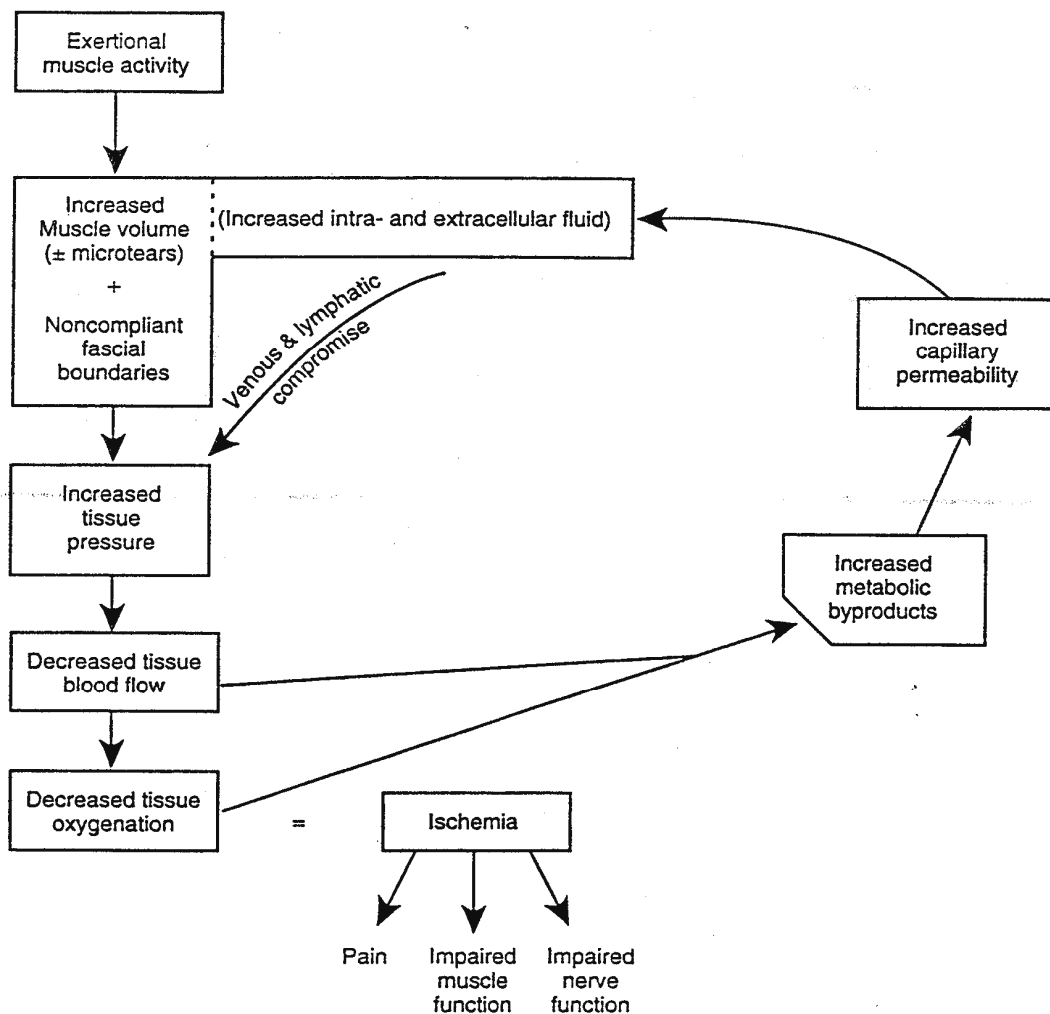
There has been much speculation as to the pathogenesis of the chronic compartment syndrome. It is thought that with repeated contraction, an exercising muscle can



increase its volume by 20% (Bourne & Rorabeck, 1989; Eisele & Sammarco, 1993). If this event occurs in a compartment surrounded by a non-compliant fascia envelope (Clanton & Solcher, 1994; Nicholas & Herschman, 1995b), compartment pressures are expected to increase. This increase in pressure then impedes blood flow and produces ischemic pain (Nicholas & Herschman, 1995b). The elevated compartment pressure increases pressure both on, and within intra-compartmental veins. The elevated venous pressure decreases the arterio-venous gradient, which reduces blood flow within the compartment. A disturbance of micro vascular flow leads to ischemia, depletion of high-energy phosphate stores, and finally cellular acidosis. The stage of ischemia is not necessarily reached in patients with chronic compartment syndrome. The symptoms of CCS, which will be discussed later, seem to appear with the rise in intra-compartmental pressure and decrease in blood flow even before ischemia sets in (Hutchinson & Ireland, 1994). These authors also describe the anatomic components contributing to compartment syndrome which may include a limited compartment size, increased intra-compartmental volume (increased capillary permeability or capillary pressure), and constricted fascia (loss of the elasticity of the compartment's fascia) or increased muscle bulk (hypertrophy). Mouhsine *et al.* (2006) also agreed with this pathogenesis.

Litwiller *et al.* (2007) wrote an article on a new proposed method of diagnosing chronic exertional compartment syndrome (CECS) in which they have stated that many theories existed to explain the etiology of CECS, but none has definitely been proven. They state, that in the case of CECS, there is a delay or barrier to the egress of fluid within the muscle compartment, possibly due to alterations in the basement membranes of vessels or in the fascia surrounding the muscles.

In summary, CCS or CECS is a condition in which the circulation and function of tissues within a closed anatomical space are compromised by increased pressure within the space. The symptoms that are produced because of the above-mentioned pathogenesis abate with periods of rest and return when exercise is resumed. In the case of CPCS, the circulation and function of the muscles and the neurovascular components are compromised in the closed anatomical space of the posterior compartment of the lower leg. According to Reneman (1975), bilateral leg involvement is common and occurs in 95% of patients. The following flow diagram illustrates the proposed pathogenesis of CPCS:



**Figure 2.3: Proposed pathogenesis of CPCS (Clanton & Solcher, 1994)**

#### 2.3.2.4. Prevalence of compartment syndrome

Allen & Barnes (1986) as well as Mouhsine *et al.* (2006) concluded from research that CPCS is clinically seen less often than chronic anterior compartment syndrome (CACS). Allen & Barnes's sample group included 110 subjects (133 abnormal legs) presenting with exercise related pain in the lower leg.

No statistics were found in the literature with regard to the prevalence of CCS and specifically CPCS. The only statistics available were applicable to the incidence of stress fractures. Stress fractures have been reported to occur in 1.9% of college athletes (Goldberg & Pecora, 1994) and 31% of military recruits (Milgrom *et al.*, 1985).

Exercise-induced pain in the lower leg is said to be most common amongst runners, followed by ballet dancers. Detmer *et al.* (1985) have found that seven out of eight people presenting with chronic compartment syndrome were athletes, and predominantly runners. According to Davey *et al.* (1984); Detmer *et al.* (1985) and Brukner (2000) chronic compartment syndrome is more common in the anterior (70%) and the deep posterior compartments (10%) than in the lateral or superficial posterior compartment. The occurrence of chronic compartment syndrome, in general, does not seem prevalent at all. See Table 2.7 for a summary.

**Table 2.7: Summary of the occurrence of chronic compartment syndrome**

<i>Author</i>	<i>Subjects referred</i>	<i>Subjects diagnosed</i>
Martens, <i>et al.</i> , 1984		29
Pedowitz <i>et al.</i> , 1990.	150	45
Rettig <i>et al.</i> , 1991		21*
Clanton & Solcher, 1994	150	33
Styf, 1998	98	26**
Blackman <i>et al.</i> , 1998		7

\* Over a period of three years

\*\* Over a period of five years

#### **2.3.2.5. Symptoms of chronic posterior compartment syndrome**

Clinically patients with symptoms of chronic posterior compartment syndrome complain of activity-related pain and a feeling of tightness that begins within a predicted period of time after the commencement of the exercise or after reaching a certain level of activity. Typically, the patient will report pain over the posterior compartment. The pain usually begins as a dull or cramp-like ache and increases in intensity if training persists. The pain will usually continue after the exercise has stopped, but is fully relieved by rest, usually about 20 minutes after completion of exercise. Stretching of the involved muscles (gastrocnemius-, soleus-, tibialis posterior- and flexor digitorum longus muscles) may also elicit pain. Patients occasionally report paraesthesia / anaesthesia in the foot (sole or dorsum and lateral aspect of the foot, depending on whether the superficial or deep compartment is involved (see Table 2.6)).



The pain can progress into weakness and instability of the ankle if recurrent ankle sprains occur due to the experienced paraesthesia or anaesthesia. Tenderness and tenseness can be palpated over the muscles (gastrocnemius- or soleus muscles) of the involved compartment (Martens *et al.*, 1984; Edwards & Myerson, 1996; Ross, 1996; Hutchinson *et al.*, 1998; Touliopolous & Herschman, 1999; Garcia-Mata *et al.*, 2001; Litwiller *et al.*, 2007).

Exercise related pain in the lower leg can be caused by a variety of conditions. This makes differentiation essential in order to determine whether the patient's symptoms are caused by CPCS or whether the symptoms may be caused by another condition.

#### ***2.3.2.6. The differentiation of CPCS from other symptom related conditions***

Conditions that can mimic symptoms of chronic compartment syndromes (also described as chronic exertional compartment syndrome or medial tibial stress syndrome) in the lower leg, are stress fractures, tendonitis (for example that of flexor hallucis longus), gastrocnemius strain, periostitis, spinal stenosis, radiculopathy, entrapments of arteries (most commonly the popliteal artery) and nerves, claudication (arterio-sclerosis), and effort induced venous thrombosis (Hutchinson *et al.*, 1998; Mirabelli & Dimeff, 2006; Reinking, 2006).

According to Brukner (2000) and Mirabelli & Dimeff (2006), shin pain can occur in one or more of three anatomical structures, namely: bone, periosteum and muscle compartment. To clarify the previous statement, examples are given: when repetitive strain is applied to the bone, a common reaction to this is the development of a stress fracture. When the periosteum is submitted to stress, inflammation develops at the insertion of muscles. This is especially applicable to the tibialis posterior- and the soleus muscle as well as the fascia attaching to the medial border of the tibia. All the muscle compartments are enveloped by fascia. The deep compartment contains the tibialis posterior-, the flexor hallucis longus- and the flexor digitorum longus muscle which can all refer pain to the posterior-medial part of the leg.

With stress fractures the patient will complain of pain at rest or an ache experienced at night. A stress fracture can be seen on X-rays although radiographic findings are normally negative during the first two to four weeks following the stress fracture. If the



X-rays are still negative after four weeks, a bone scan can be done. The technetium pyrophosphate bone scan is usually positive within the first week after presentation of symptoms if a stress fracture is present. With a stress fracture, it reveals a very focal uptake of technetium in the medial tibia. Clinically, hopping on the affected leg and percussion of the bone can be used as part of the objective assessment and these tests will elicit pain if a stress fracture is present. Ultrasound therapy seems to aggravate the symptoms if a stress fracture is present and can therefore be used as a diagnostic tool (Clanton & Solcher, 1994).

When the pain originates from a tendon or the periosteum in the lower leg, the pain tends to improve after warming up, in contrast to pain caused by a compartment syndrome which gradually worsens with exercise. In contrast to a stress fracture, a technetium pyrophosphate bone scan will show a diffuse linear uptake of the technetium along the posterior-medial tibia with a periostitis. Another interesting difference between pain caused by CPCS and periosteum involvement according to Blackman *et al.* (1998) is that the pain of CPCS is unrelated to ground contact.

Painful isometric muscle contractions of the various muscles of the posterior compartment are normally an indication of a partial muscle tear (Travell & Simons, 1999).

When pain in the lower leg is referred from a lumbar nerve root (radiculopathy), neurological conduction deficits will be identified with a neurological examination. There will be an area of decreased sensation (in a specific dermatome, indicative of a specific nerve root), decreased motor strength, decreased tendon reflexes or a combination of any of these factors (Maitland, 2006). Pain due to mechanical sensitivity of the lumbar-sacral nerve roots should however be differentiated with neural provocation tests (Butler, 1996).

Bilateral leg involvement of CPCS occurs in 95% of subjects with CCS (Reneman, 1975). Lumbar stenosis can however also be the cause of bilateral leg pain. The most characteristic features of spinal stenosis will be described in order to assist clinicians to differentiate between symptoms caused by CPCS and symptoms caused by spinal stenosis. The most common finding in a subject with lumbar stenosis is a decrease in



lumbar spinal extension. Lumbar extension will normally reproduce or exacerbate the symptoms. Subjects will be more comfortable walking uphill than downhill and can walk further if they are bent forward while walking. They sometimes walk with a wide base, but a neurological examination often proves to be negative (in some cases, the various forms of sensation or light touch might be decreased) (Nowakowski *et al.*, 1996).

The following tests may assist the clinician to differentiate between vascular impairment, neurogenic impairment and symptoms of CPCS.

- The “bicycle test”: the subject cycles on a stationary bicycle with his spine first extended then flexed. In the case of vascular claudication, the change in lumbar posture will have no influence on his symptoms. In the case of neurogenic claudication, the subject will be able to cycle for a longer period of time with the spine in a flexed position before he experiences any symptoms (Nowakowski *et al.*, 1996).
- The “stoop test”: the test is positive for neurogenic claudication when flexion of the lumbar spine while walking or stooping relieves the limb symptoms. (Nowakowski *et al.*, 1996).

Other conditions that can mimic the symptoms of CPCS are neural entrapment syndromes. The most common neural entrapment syndrome, causing pain in the lower leg and often paraesthesia over the dorsal aspect of the foot, is that of the superficial peroneal nerve (Clanton & Solcher, 1994). The superficial peroneal nerve runs through the lateral compartment (Travell & Simons, 1999). In order to differentiate it from the posterior compartment syndrome, Tinel’s sign may be elicited by gentle percussions over the path of the superficial peroneal nerve (8 – 15 cm proximal to the ankle). Symptoms may also be elicited by pressing on the nerve where it emerges from the deep fascia (approximately one-quarter of the distance up the lower leg from the lateral malleolus) while doing dorsi flexion and eversion against resistance (Clanton & Solcher, 1994). Pain is normally experienced over the lateral side of the lower quarter of the shin. Swelling and diminished sensation might be present over the before mentioned area without any motor deficits (Kernohan *et al.*, 1985; Travell & Simons, 1999).





Although some of the symptoms (pain, swelling and sensational deficits) are similar to that of CPCS, the area of the symptoms differs.

Other possible nerve entrapments whose symptoms might mimic some of the symptoms experienced with CPCS are:

- entrapment of the posterior tibial nerve (which runs through the deep posterior compartment) with a high tarsal tunnel. Entrapment of the tibial nerve by the tendinous arch of the soleus muscle has been noted a couple of times before. This gives rise to pain in the popliteal fossa which is exacerbated with active or passive dorsi-flexion and by weight bearing. Entrapment of the posterior tibial nerve can cause pain behind the knee and a tingling sensation in the sole of the foot (Mastaglia, 2000; Mastaglia *et al.*, 2000)
- entrapment of the common peroneal nerve at the neck of the fibula (at the origin of the peroneus longus muscle). Entrapment of the common peroneal nerve weakens both the anterior and lateral compartment muscles. Loss of sensation is most marked in a triangular patch on the dorsum of the foot distally between the first and the second toes (Travell & Simons, 1999).
- entrapment of the sural nerve (which runs through the superficial posterior compartment) in the posterior part of the calf. Patients typically have a sub-acute onset of a neuritis type of pain such as burning, tingling or radiation (Clanton & Solcher, 1994). This pain is unlike the pain, stiffness or paraesthesia experienced with the chronic posterior compartment syndrome.

A vascular condition that can mimic symptoms of CPCS is entrapment of the popliteal artery. Popliteal artery entrapment syndrome is a relatively rare entity that causes calf pain in young athletes but it should not be disregarded as a possible diagnosis in a young athlete presenting with unilateral symptoms of intermittent claudication. Early symptoms may be vague and atypical for claudication. The symptoms can include cramping pain in the calf, paraesthesia and discoloration in the foot or toes, as well as temperature changes in the foot (Clanton & Solcher, 1994).

When claudication is present, the most common cause is compression of the artery beneath the gastrocnemius muscle. The pain increases with elevation of the leg as well as with exercise. The pain decreases with the cessation of exercise. The dorsalis pedis





and posterior tibialis pulses should be palpated in order to help with the diagnosis of popliteal artery entrapment. In the case of popliteal artery entrapment, the pulses will be decreased or absent in contrast to the normal pulses in subjects with CCS. The increased intra-compartmental pressure in CCS is able to disturb only the capillary circulation - will not have any effect on the pulses. This capillary compression results in a relative ischemia, while the larger arteries are not involved and have normal pulses. A Doppler ultrasound or angiogram performed directly after the exercise can confirm the diagnosis of popliteal artery entrapment (Touliopolous & Herschman, 1999). Another difference between the symptoms of CPCS and popliteal artery entrapment is that the complaints of intermittent claudication in the popliteal artery entrapment syndrome disappear a few minutes after cessation of the activity. In subjects with CCS, the discomfort will persist until the intra-compartmental pressure is restored to a normal level (Lysens *et al.*, 1983).

Exercise-induced venous thrombosis is another vascular impairment that needs to be excluded as a possible cause of pain in the lower leg. With exercise-induced venous thrombosis, symptoms of swelling of the calf area and point tenderness over the calf will occur unilaterally, in contrast to the symptoms of CPCS which are often present in both legs. Homan's sign will be positive. Elevation of the leg will decrease the pain, in contrast to the symptoms experienced by popliteal artery entrapment. An ultrasonographic or venographic investigation is necessary to confirm the diagnosis of a deep venous thrombosis (Clanton & Solcher, 1994).

In summary, the following conditions that might mimic some of the symptoms of CPCS should be ruled out through a process of differentiation as discussed above:

- Stress fractures
- Tendonitis
- Periostitis
- Radiculopathy
- Spinal stenosis
- Vascular impairments (vascular claudication; exercised-induced venous thrombosis) and popliteal artery entrapment syndrome
- Neurogenic impairments (neurogenic claudication)



### 2.3.2.7. *Intra-compartmental pressure measurement and the diagnosis of CPCS*

Measurement of intra-compartmental pressure may provide an objective tool to confirm the diagnosis of CPCS.

Pedowitz *et al.* (1990) published a paper in which they suggested criteria for the measurement of intra-compartmental pressures in chronic compartment syndrome of the leg by means of the slit-catheter technique. The criteria suggested that a pre-exercise pressure of 15 mm Hg or more and or a one minute post-exercise pressure of 30 mmHg or more and or a five minute post-exercise pressure of 20 mmHg or more would be indicative of chronic compartment syndrome.

Allen & Barnes (1986) propose that pre-exercise resting pressures do not conclusively demonstrate compartment syndrome, and significant differences between normal and abnormal compartment pressures have been found only by means of statistical analysis.

In the measurement of the intra-compartmental pressures, the most emphasis has been placed on the time that it takes for the post-exercise values to return to the pre-exercise level. Reneman (1975), Mubarak *et al.* (1982), Rorabeck *et al.* (1983) and Wallensten (1983) all used this index, and although all concur that pressure in pathological compartments takes longer to return to its pre-exercise level, there is no agreement about precise criteria of pressure and time. Although it is generally agreed that higher pressures are found during exercise in cases of compartment syndrome, only Puranen & Alavaikke (1981) consider this raised pressure to be the most important diagnostic factor.

Several invasive methods are available to obtain intra-compartmental pressures. These include the continuous infusion technique, a wick catheter, a split catheter and the solid state transducer intra-compartmental catheter. Numerous potential sources of error are present with each method (Clanton & Solcher, 1994). According to Detmer *et al.* (1985), proper experience in measurement techniques is needed to assure accurate results and these results still need to be interpreted in the context of the clinical signs and symptoms.



A variety of side-effects and technical problems experienced with the measurement of intra-compartmental pressures have been described in the literature (Schepstis & Lynch, 1998). These side-effects include discomfort to the patient; injury to important deep structures in the proximal deep compartment which can result from a blind needle stab; obstruction of the catheter can occur and it could be argued that even a very slow infusion of saline could be dangerous to the patient. Furthermore, the accuracy of the measurements can be influenced by the exact location of the tip of the needle, the depth of the needle, the position of ankle and foot during pressure measurements and the force of contraction.

In a survey done by Williams *et al.* (1998) on the use of compartment monitoring devices in the diagnosis of compartment syndrome, the results demonstrated that the majority of the trauma- and orthopaedic surgeons surveyed in the United Kingdom, advocate making the diagnosis through a combination of clinical reasoning and compartment pressure measurements. However, less than half of the trauma centres surveyed had pressure monitoring equipment available and there was a marked variation in opinion regarding the threshold level for surgery when pressure monitoring was applied.

Chronic compartment syndrome is currently diagnosed using invasive intra-compartmental pressure measurements (Mubarak, 1981; Puranen & Alavaikke, 1981). Two new techniques that are non-invasive and currently being studied as substitutes for testing intra-compartmental pressures are: the Thallium Stress Test and Near Infrared Spectroscopy. Near Infrared Spectroscopy is an indirect tool developed for the monitoring of tissue oxygenation in exercising skeletal muscle in order to detect the de-oxygenation caused by high intra-compartmental pressures. It is a non-invasive, painless method, but one of its limitations is the fact that light absorption is altered with passage through tissue (Giannotti *et al.*, 2000).

MRI has been used in a limited way in attempts to diagnose CECS. The problem with a MRI test as a diagnostic tool in the condition of CECS, is the fact that 5 min have already elapsed since the running test have been done, and the pressures have already dropped by the time the scanner is ready to operate (Litwiller *et al.*, 2007).



Litwiller *et al.* (2007) feels that the currently accepted method of measuring intra-compartmental pressure is too invasive and potentially quite painful and because of this the test should only be performed when the suspicion for compartment syndrome is very high. They have developed an improved MRI screening protocol for CECS using an in-scanner exercise protocol and novel dual birdcage coil design for improved scanning. Although the concept of the screening protocol is excellent, it still requires refinement for commercial exploitation.

Measurement of the intra-compartmental pressures might be valuable to differentiate between CPCS and medial tibial stress syndrome. However, in the light of the risks associated with possible complications and side effects revealed by the literature research, the question arises as to whether invasive intra-compartmental pressure measurement is worth the risks. It can therefore be argued that no gold standard exists for the measurement of intra-compartmental pressures.

#### ***2.3.2.8. Surgical management of chronic compartment syndrome***

Allen & Barnes (1986) as well as Edwards & Myerson (1996) proposed that in order to make a definitive diagnosis, patients with symptoms of CCS should first be treated conservatively. If there is no improvement and symptoms persist for a period of three to six months or longer, surgery is recommended in the presence of an increased post-exercise intra-compartmental pressure.

Since compartment syndrome of the posterior compartment occurs less frequently than compartment syndrome of the anterior and lateral compartments (Rorabeck *et al.*, 1988; Mouhsine *et al.*, 2006), the outcomes of surgical interventions in compartment syndromes in general will be discussed.

Surgery entails the surgical decompression of the various involved compartments. This is called a fasciotomy (Micheli *et al.*, 1999). Occasionally a part of the fascia is also removed surgically. This procedure is called a fasciectomy (Slimmon *et al.*, 2002). According to Swain & Ross (1999), the deep posterior compartment does not respond as quickly or as well, to fasciotomy as the anterior compartment. Edwards & Myerson (1996) have established that in most studies the results of surgical releases of the fascia of the anterior and lateral compartments are more successful in terms of the alleviation



of symptoms than are those of posterior fascia releases. Schepsis & Lynch (1998) as well as Mouhsine *et al.* (2006) agree that subjects with symptoms of CPCS do not seem to respond as well to surgery as subjects with symptoms of other chronic compartment syndromes. Only 13 out of their 20 patients with symptoms of CPCS responded satisfactory to the surgery. Rorabeck *et al.* (1988) subjected 25 patients with elevated compartment pressures to surgical fasciotomy of the respective compartments (anterior, 13; anterior and posterior, four; deep posterior, eight). There were three failures, all of whom had decompression of the deep posterior compartment. They have then suggested that fasciotomy of the deep posterior compartment should also include a formal release of the tibialis posterior muscle at the same time.

Wallensten (1983) studied the results of fasciotomy of the affected muscle compartment in eight patients with chronic anterior compartment syndrome and in nine patients with medial tibial syndrome (which he also calls a deep posterior compartment syndrome). Clinically, there was a complete relief of pain in all of the patients with chronic anterior compartment syndrome and in five of the nine patients with medial tibial syndrome. It was not mentioned how the pain was measured or how long post-operatively the follow-up assessment was done.

Howard *et al.* (2000) conducted a study in which they evaluated the outcomes in patients following surgical treatment (fasciotomy) of chronic exertional compartment syndrome (CCS) in the leg. Over a period of six years a questionnaire was sent to 62 patients who had received surgery for the symptoms of CCS. A clinically significant improvement was reported by 81% of the anterior/lateral compartment patients compared to 50% of the patients with deep posterior compartment involvement. The mean percentage of pain relief was 68%. Lower activity levels were reported by 22% of the subjects; six percent failed initial surgical procedures and required revision surgery. Five percent had subsequent operations for exercise-induced pain in a different compartment than the compartment that initially underwent surgery. The researchers concluded that despite the possibility that some patients have less favourable outcomes, experience complications, or need subsequent operations, fasciotomy is recommended for patients with CECS as there is no other treatment for this condition. According to the authors, possible reasons for better post-surgical results in the anterior and lateral compartment than in the posterior compartment may have been the presence of sub



compartments, such as the tibialis posterior compartment within the deep posterior compartment; or entrapment of the popliteal artery.

Slimmon *et al.* (2002) combined a partial fasciectomy with fasciotomy for compartment syndrome in order to relieve pain and to prevent recurrences thereof. A self-administered questionnaire was given to 62 patients at a mean follow-up 51 months after the surgery. Of the 50 patients who underwent a single operation, 60% reported an excellent or good outcome. Average pain and pain-on-running were significantly reduced, although some subjects still reported considerable levels of pain. Fifty eight percent of patients were exercising at a lower level than before the operation and, of these, 36% of the patients cited the return of their compartment syndrome or the development of a different lower leg compartment syndrome as the reason for the reduction in exercise levels. Some subjects indicated early improvement followed by subsequent deterioration. Slimmon *et al.* (2002) concluded that this surgical technique reduces pain and allows the majority of patients to return to sports; however, patients should be counselled that they may not be able to return to their pre-injury level of exercise or remain pain free.

Shah *et al.* (2004) are of the opinion that conservative management of CCS has been highly unsuccessful in terms of alleviating or decreasing the symptoms of CCS. They propose that a considerable degree of success (a decrease or alleviation of some of the symptoms), can be expected after a fasciotomy of the involved compartments followed by a rigorous rehabilitation programme, though recurrence of symptoms is a reported complication.

One of the more positive surgical studies was reported by Turnipseed (2002). An open fasciectomy was performed in all patients with CCS and where applicable, the popliteal artery was released. Three hundred and sixteen antero-lateral, 70 deep-posterior and 50 superficial-posterior compartments were treated. The mean follow-up period was 60 months. Of the 276 patients, 92% had full relief of symptoms, eight percent obtained symptomatic relief, but activity was limited because of new compartment symptoms.



According to Howard *et al.* (2000) 13% of patients have experienced post-operative complications. Some of the complications experienced by Detmer *et al.* (1985) during a fasciotomy were:

- artery injury, requiring repair
- haematoma
- superficial wound infection
- peripheral cutaneous nerve damage
- lymphocele and
- deep venous thrombosis.

According to Mouhsine *et al.* (2006) the reported complication rate of fascia release, is between 4,5% and 13% including nerve injury, infections, post-operative haematoma, recurrence of symptoms secondary to incomplete release, cosmetically unacceptable scarring, anaesthetic problems, muscle fascia adhesions, swelling, lymphocele or haemorrhage.

The literature summarized in Tables 2.8 demonstrates that the surgical release of the fascia is successful in a limited number of cases and that there are often surgical complications. A variety of possible complications exists and the results are not always satisfactory in especially the deep posterior compartment. Some of the muscle strength is sacrificed, at least temporarily if not permanently, when the fascial barrier is not present for the muscle to work against (Micheli *et al.*, 1999). It also takes 6 - 12 weeks before the runner is back on his former running programme. This implies that he is back at the stage of base training.

In order to make a definitive diagnosis, the patients with symptoms of CCS should first be treated conservatively for a period of three to six months before they are referred for possible surgical management (Allen & Barnes, 1986; Edwards & Myerson, 1996). It was however found from the literature review that study data for conservative treatments are sparse and largely unsuccessful. Fasciotomy, according to the literature review, is the only effective treatment for chronic compartment syndrome (Fraipont & Adamson, 2003; Bong *et al.*, 2005; Englund, 2005; Godon & Crielaard, 2005; Mouhsine *et al.*, 2006).



**Table 2.8 (a): Outcomes of surgical interventions during 1983 to 1998**

<i>Researchers</i>	<i>Number of subjects</i>	<i>Intervention</i>	<i>Outcome measure</i>	<i>Results</i>	<i>Complications</i>
Wallensten, 1983	Eight subjects with CSCS and nine subjects with chronic medial tibial syndrome.	Fasciotomy.	Clinical assessment and questionnaires.	CACS: no pain. Complete relief of symptoms in five out of the nine subjects with chronic medial tibial syndrome.	Not mentioned.
Allen & Barnes, 1986	110 subjects.	Subcutaneous fasciotomy of the affected compartment.	Reassessment of symptoms. Not mentioned how.	At least one type of CCS was diagnosed in 105 limbs. 12 were treated conservatively, 20 limbs were treated surgically. Of the balance of 73 limbs during follow-up three months later, all but three patients had improved.	Not mentioned.
Rorabeck <i>et al.</i> , 1988	12 subjects with bilateral compartment syndrome. Group 1: seven subjects with anterior or lateral compartment symptoms. Group 2: three subjects with symptoms of deep posterior compartment. Group 3: two subjects with symptoms of both the anterior and the deep posterior compartments.	Bilateral fasciotomy.	Follow-up symptoms were assessed six - 24 months post-operatively. Subjects were questioned about the success of the surgery. Conditions were rated as improved or not improved.	Two subjects did not improve and were both from the deep posterior compartment group.	Not mentioned.
Schepsis & Lynch, 1998	16 subjects – (26 limbs) for anterior decompression and 12 subjects (20 limbs) for deep posterior decompression.	Surgical decompression (fasciotomy).	Follow-up of symptoms after four years.	15 out of the 16 subjects had excellent results for the anterior decompression. In 12 subjects (20 limbs) who underwent fasciotomy of the deep anterior compartment, there were five excellent, eight good, four fair and three poor results.	Not mentioned.



**Table 2.8(b): Outcomes of surgical interventions during 1998 to 2002**

<i>Researchers</i>	<i>Number of subjects</i>	<i>Intervention</i>	<i>Outcome measure</i>	<i>Results</i>	<i>Complications</i>
Howard <i>et al.</i> , 2000	62 subjects treated over a period of seven years.	Fasciotomy.	Questionnaire with regard to pain on VAS; level of improvement; level of maximum activity; satisfaction level and occurrence of re-operations.	A clinically significant improvement was reported by 81% anterior/ lateral compartment subjects and 50% subjects with deep posterior compartment involvement.	13% reported complication which included: haemorrhage, wound infection, nerve entrapment, swelling, artery injury, haematoma, lymphocele, peripheral cutaneous nerve injury and deep vein thrombosis.
Slimmon <i>et al.</i> , 2002	62 subjects in total. 50 subjects underwent a single operation.	Fasciotomy with partial fasciectomy of involved compartment.	Self-administered questionnaire, 51 months after surgery.	60% of the 50 subjects that underwent a single operation reported a reduced average pain and pain-on-running, although some still reported considerable levels of pain. 58% of the 62 subjects were exercising at lower intensities than before the surgery.	13 subjects out of the 62 said their symptoms reoccurred post-surgically or re-appeared in a different compartment.
Turnipseed, 2002	276 subjects.	Surgical procedures: open fasciectomy. Of the 436 treated compartments, 316 were antero-lateral, 70 deep posterior and 50 superficial posterior.	Follow-up 60 months after surgery, inquiring about symptoms, daily or athletic activities.	92% had full relief of symptoms. 8% obtained symptomatic relief, but activity was limited because of new compartment symptoms.	Not mentioned.

### **2.3.2.9. *Conservative management of chronic compartment syndrome***

Since compartment syndrome of the posterior compartment occurs less frequently than compartment syndrome of the anterior and lateral compartments (Godon & Crielaard, 2005), the conservative outcomes of compartment syndromes in general will be reviewed.

The literature available on the conservative management of chronic compartment syndrome is limited. Most of the conservative approaches or modalities used in the management of CCS are only mentioned and not discussed in detail. The following list is a summary of the various conservative treatment modalities/ approaches mentioned in the literature and which will be discussed in this section:

- physical therapy (massage, ultrasound, stretching, heat, cold, myofascial release techniques, whirlpool, electrical stimulations). All of the physical therapy techniques were applied locally to the area of the posterior calf.
- orthotics, modification of shoes, taping
- anti-inflammatory medication, diuretics, steroid injections
- rest, cast immobilizations
- reduced training, different training programmes
- compression, elevation

Schepsis & Lynch (1998) state that once the patient has been diagnosed with chronic exertional compartment syndrome, the only worthy non-operative treatment is the modification of activity. They are of the opinion that if the athlete is unwilling or unable to give up the activity that causes the symptoms, the only other option is surgical decompression by fasciotomy. In their opinion other treatment modalities such as physiotherapy, rest, orthotics and anti-inflammatory medication are of minimal value.

Styf (1998) has investigated recurrent exercise-induced pain in the anterior aspect of the lower leg in 98 patients who were clinically diagnosed with chronic anterior compartment syndrome (CACS). Intra-compartmental pressure measurements



confirmed the diagnosis of CACS in 26 of the patients. According to the subjective assessment done, all of the 98 patients tried various conservative treatment approaches including rest, reduced training, anti-inflammatory drugs, diuretics, modification of shoes, different training programmes and orthotic applications, as well as physiotherapy that included ultrasound therapy, stretching, local heat and cold. Conservative treatment approaches were only mentioned and not described in detail. According to the patients, none of the previously mentioned treatments had any lasting effect on their symptoms.

Davey *et al.* (1984) postulated that the tibialis posterior muscle is contained in its own osseofascial compartment and might be the site of isolated exertional/compartment syndrome. Part of the study was done on two runners complaining of pain to the posterior- medial aspect of the mid and lower third of the tibia. Both of these individuals had failed to respond to conservative modalities including rest, ice, anti-inflammatory drugs, physiotherapy, and foot orthoses. The conservative physiotherapy treatments were not described. The authors concluded that they have found true exertional compartment syndrome to be resistant to anything less than surgical decompression.

According to a study done by Detmer *et al.* (1985) on patients with chronic compartment syndrome, it was mentioned that:

- 41% had tried orthotics which was considered helpful by 15% of the patients;
- stretching programmes were widely used but were not considered helpful;
- 40% of the subjects underwent physiotherapy; and
- 49% of the subjects had used medication.

A third of the patients described the physiotherapy and the use of medication as being only somewhat helpful while the rest said that they had experienced no measurable relief of their symptoms.

In a study done by Martens *et al.* (1984) nine patients diagnosed with chronic compartment syndrome received conservative treatment consisting of prolonged rest, physiotherapy, anti-inflammatory drugs and stretching exercises of the flexor muscles



of the lower leg. According to Martens *et al.* (1984), none of the conservative treatment methods led to an appreciable improvement. All the patients were forced to limit their sports activities to a certain extent and ended up undergoing a fasciotomy. Although the fasciotomy and its results were described in the study reported, conservative approaches were merely mentioned and not dealt with in any depth.

Martens *et al.* (1984) tried a variety of therapeutic modalities to treat the symptoms of shin splints (which they called medial stress syndrome). According to the authors, aspirin, phenylbutazone, heel-cord stretching, heel-pads and cast immobilizations did not have any lasting effect on the patients' symptoms.

Jackson & Bailey (1975) reported that taping or arch supports yielded no success in patients with CPCS and found that aspirin and local injection of steroids were also not beneficial.

According to Garcia-Mata *et al.* (2001), intermittent massage with specific stretching only serves to lengthen the time before onset of pain in patients with chronic exertional compartment syndrome, but does not cure or prevent the condition. The authors' mention the effect of massage together with specific stretches only in passing and then go on to describes surgery as a better option for the symptoms. Their study included 23 legs. Twenty-one patients complained of anterior compartment syndrome, one of posterior compartment syndrome and one of both anterior and posterior compartment syndrome.

Allen & Barnes (1986) have found with medial tibial syndrome (deep posterior compartment) that the outcomes of both surgical (fasciotomy of the medial border of the tibia) and conservative treatment (physiotherapy, steroid injections to the tibial border and shoe inserts) were poor. The physiotherapy techniques used, or their area of application were not mentioned.

Clanton & Solcher (1994) states, that conservative method for treating chronic compartment syndrome, such as ice, medication, shoe modification and orthotics usually provide little benefit. No further description is given with regard to the application of the conservative treatment methods.



According to Melberg & Styf (1989), 28 patients complaining of posterior-medial pain in the lower leg received conservative treatment, with no improvement in the signs and symptoms. The types or number of conservative treatments were not mentioned.

Poor results from non-operative treatment of chronic compartment syndrome were highlighted by Froneck *et al.* (1987) who reported that five out of seven patients were unable to return to sport after undergoing a conservative programme. The authors did not mention the type of non-operative treatment approaches that were used or the duration of the conservative programme.

Biedert & Marti (1997) have found conservative treatment techniques applied to patients with symptoms of CPCS such as anti-inflammatory medication, ultrasound therapy, myofascial release techniques, stretching and orthotics, not useful. This treatment was applied locally to the area of the lower leg but the number of treatments received by the patients was not mentioned. The authors mentioned the effect of these techniques only in passing and went on to describe the surgical management for CPCS.

A study done by Micheli *et al.* (1999) demonstrated that the conservative treatment of chronic compartment syndrome in young female athletes using treatment approaches such as ice, rest, compression, elevation, medication, ultrasound, orthotics, massage, whirlpool, heat, gel packs, electrical stimulation and stretching were not successful. Once again the number of treatments given to the patients was not mentioned.

Blackman *et al.* (1998) determined the effect of massage as a treatment on symptoms of chronic anterior compartment syndrome. The study was performed on seven patients, each receiving six treatments. Each massage session consisted of the following phases: preparatory massage (one minute), longitudinal gliding (one minute), transverse gliding (one minute), mobilizing digital pressure (two minutes) and myofascial release and posterior compartment massage (ten minutes). Between sessions they were given a stretching programme. Stretches were given for the anterior as well as the posterior compartment muscles. There was no significant difference in the three minute post-exercise compartment pressures after the



treatment. There was however a significant increase in dorsi flexion work performed before the onset of pain following the massage (it is normally the plantar flexion action during running that leads to an increase in the intra-compartmental pressure whenever the posterior compartment is involved).

During 2002, Mouhsine *et al.* (2006) recruited 18 subjects for surgical release of the fascia after they have received, to no avail, conservative treatment for a mean period of four months. The conservative treatment entailed modification of exercise programmes, stretching, changing of shoes, shoe inserts, and local injections of xylocaine into the area of maximum swelling. Although the reviewed studies all had poor outcomes, it is difficult to evaluate the effect of manual therapy and other physiotherapy techniques on CCS because in none of the studies neither the type and application of the conservative techniques/approaches used, nor the number of treatments were described in detail.

### **2.3.3. Functional anatomy and biomechanical factors**

In the literature a number of researchers found a correlation between exercise related leg pain (ERLP) and biomechanical factors (Bennett *et al.*, 2001; Dugan & Bhat, 2005; Hreljac, 2005). Dugan & Bhat (2005) found that foot pronation had a distinct influence on the development of ERLP in athletes. They found that athletes with a history of ERLP had a significant greater foot pronation than athletes without such a history. Excessive pronation is however difficult to define due to the fact that the magnitude of normal foot pronation in a large sample of asymptomatic subjects has never been described (Johanson *et al.*, 1994).

Although some research findings contradicts these relationships (Johanson *et al.*, 1994; Lun *et al.*, 2004), these contradictions are probably contributable to the definition of runners and the associated distances run by the different research populations (Hreljac, 2005). Hreljac (2005) however makes a convincing argument for such a correlation based on the increased risks for injury due to increased torque and instability that results from improper biomechanical alignments.



In the following section the function of the muscles of all the compartments of the posterior compartment of the lower leg will be discussed in the light of the effect which they have on movement patterns which will be assessed during the gait analysis.

### ***2.3.3.1. Muscles of the posterior compartments of the lower leg***

In order to determine which muscles are involved and play a role in the cause or the perpetuation of the symptoms of CPCS, it is necessary to analyse the functions of the muscles contained in the posterior compartment of the lower leg.

As previously seen, the deep posterior compartment contains the flexor digitorum longus (FDL) -, the flexor hallucis longus (FHL) - and the tibialis posterior muscle. The main function of the FDL is to flex the distal phalanx of each of the four lesser toes and the main function of the FHL is to flex the distal phalanx of the great toe (Agur, 1991). Both of these muscles also assist in plantar flexion (PF) and inversion (Inv) of the foot when the foot is free to move (Travell & Simons, 1999). The FDL and the FHL muscles are also important in preventing extreme plantar to dorsi flexion movement at the metatarsophalangeal joints when the foot is in contact with the ground. An inflexible shoe sole might prevent normal extension of the metatarsophalangeal joints during running. The stiffness of the sole effectively lengthens the lever arm against which these two long flexor muscles of the toes function, and thus overload them. An inflexible shoe sole might thus be one of the factors perpetuating the symptoms of CPCS if the FHL and the FDL muscles are involved (Travell & Simons, 1999).

The FDL and the FHL stabilize the foot and ankle in the mid- to late-stance phase of the running gait, playing a role in the medio-lateral balance. These muscles also assist other plantar flexors in enabling the individual to transfer weight to the fore foot and they assist in the maintenance of equilibrium when the weight is on the fore foot (Travell & Simons, 1999).

FHL stenosing tenosynovitis is a well-recognised dysfunction amongst ballet dancers and is also occasionally seen in runners. The symptoms typically begin insidiously



and include pain and tenderness in the posterior-medial area of the ankle (might radiate distally along the medial arch). Passive and or active ankle and or hallux movement is painful and a crepitus of the hallux might be present. Weight-bearing exercises exacerbate the symptoms and swelling may be seen in the vicinity of the postero-medial part of the ankle (Oloff & Schulhafer, 1998).

In summary, the following factors need to be assessed in order to determine whether FHL- and the FDL muscle play a role in the symptoms of CPCS:

- muscle strength of the distal phalanxes of the toes;
- the amount of plantar and dorsi flexion range of movement at the metatarsophalangeal joints when the foot is in contact with the ground;
- the medio-lateral balance in the mid- to late-stance phase; and
- the maintenance of equilibrium when the weight is on the fore foot.

After heel contact, the tibialis posterior acts as a shock absorber for the subtalar joint limiting hind foot eversion by eccentric contraction (Mosier *et al.*, 1999). During mid-stance, contraction of tibialis posterior muscle causes inversion of the subtalar joint, locking the calcaneocuboid and talonavicular or transverse tarsal joints. This results in a rigid lever for forward propulsion of the foot over the metatarsal heads (distributes the body weight over the heads of the metatarsals). With dysfunction of the tibialis posterior muscle, there is no rigid lever during the mid-stance, resulting in decreased tarso-metatarsal joint stability and hind foot inversion. The forward propulsion force of the gastrocnemius and soleus muscle complex acts at the mid-foot rather than at the metatarsal heads, creating excessive mid foot stress allowing increased mid foot abduction (Mosier *et al.*, 1999).

When the foot is free, the tibialis posterior muscle acts to invert and to adduct the foot. It also assists in plantar flexion of the ankle joint (Travell & Simons, 1999). Tibialis posterior dysfunction is associated with an increased amount of fore foot abduction, mid-foot collapse and excessive hind foot valgus (Mosier *et al.*, 1999). Excessive hind foot valgus causes an increase in tensile stress to the plantar fascial insertion. During the initial stages of tibialis posterior muscle dysfunction, pain will be experienced along the course of the tibialis posterior tendon (Travell & Simons,



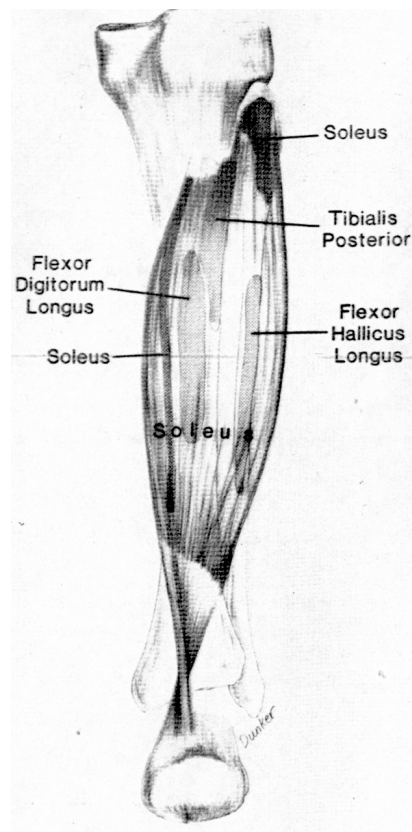


1999; Agur, 1991). In summary, with regard to the possible involvement of the tibialis posterior muscle as a role player in the symptoms of CPCS, the following need to be assessed:

- signs of mid foot collapse
- increased fore foot abduction
- increased hind foot valgus

The superficial posterior compartment contains the gastrocnemius, the plantaris and the soleus muscles. The soleus muscle is a primary plantar flexor of the foot (Travell & Simons, 1999). A study done by Michael & Holder (1985) has shown that the medial one-half of the soleus muscle is also an inverter of the calcaneus. The soleus muscle has a tough aponeurotic covering both anteriorly and posteriorly of the muscle. This fascia, which is firmly attached to the anterior and the posterior surfaces of the soleus muscle, fuse together beyond the edge of the muscle and form an impressively tough attachment to the medial border of the tibia, much stronger than the thin layer of fascia covering the deep layer of muscles. The soleus and its fascia form an unyielding structure under which the deep muscles must function (Michael & Holder, 1985).

An injured soleus muscle can produce pain along the medial border of the tibia. This referred pain was thought to originate from the tibialis posterior muscle (O'Donoghue, 1976), but Michael & Holder (1985) showed that the tibialis posterior muscle takes its origin a considerable distance away from the medial border of the posterior tibia and that the muscle belly of the tibialis posterior muscle is higher in location than the usual site where the pain is experienced. The tendinous portion of the soleus makes up the anterior one- half of the Achilles tendon in association with the gastrocnemius, and as the Achilles tendon approaches the calcaneal insertion, it rotates 90 degrees so that the soleus contribution to the tendon inserts on the medial one-third of the calcaneus, while the gastrocnemius contribution inserts in the lateral two- thirds. From a biomechanical standpoint, the insertion of the soleus muscle on the medial aspect of the calcaneus makes it vulnerable to excessive elongation when the heel is placed in the pronated position (Michael & Holder, 1985) (See Figure 2.4).



**Figure 2.4: Rotation of the Achilles tendon (Michael & Holder, 1985)**

In summary, with regard to the possible involvement of the soleus muscle in the symptoms of CPCS, the following factors need to be assessed:

- tightness of the soleus bridge over the deep posterior compartment
- hind foot pronation (Achilles tendon angle)

The gastrocnemius muscle is most effective as a plantar flexor at the ankle when the knee is extended. As the knee becomes progressively more flexed this muscle loses effectiveness and plantar flexion of the foot is accomplished increasingly by the soleus muscle. The gastrocnemius muscle may also assist flexion at the knee. As proposed by Travell & Simons (1999), the gastrocnemius muscle is not likely to play a role in the cause or perpetuation of symptoms of CPCS.

### **2.3.3.2. Muscles of the anterior compartments of the lower leg**

The anterior compartment contains the tibialis anterior- (TA), the extensor hallucis longus (EHL)-, the peroneus tertius- (PT) and the extensor digitorum longus (EDL)



muscles. The tibialis anterior muscle can give rise to pain superficially over the big toe. During running, the function of the tibialis anterior muscle is to dorsi-flex the foot of the non-weight bearing leg at the talocrural joint and to supinates (inverts and adducts) the same foot at the subtalar and transverse tarsal joints. It is not active as an inverter during plantar flexion (Travell & Simons, 1999). With weakness of the tibialis anterior muscle, a person might have difficulty in clearing the foot during the swing phase (Travell & Simons, 1999). The PT muscle causes dorsi-flexion and assists with eversion (Agur, 1991; Travell & Simons, 1999.).

The EDL acts to dorsi-flex and evert the foot and to extend the four lesser toes. A heel strike position with the calcaneus in a position of too much inversion might therefore be an indication of a weak PT- or EDL muscle. The EHL assists in dorsi-flexion and inversion of the foot and extends the big toe. The EDL- and the EHL muscles functions as assistants in controlling (decelerating) the descent of the fore foot to the floor immediately following heel-strike, thereby preventing foot slap. During the swing-phase, they assist in providing foot-floor clearance. The EDL muscle help to provide a pure dorsi-flexion movement of the foot by balancing the inversion pull of the tibialis anterior muscle. With a weakened EDL muscle, the calcaneus might move into a position of inversion (Travell & Simons, 1999).

#### ***2.3.3.3. Muscles of the lateral compartments of the lower leg***

The lateral compartment contains the peroneus longus- (PL) and the peroneus brevis (PB) muscles. They cause the foot, when free, to abduct and to evert. These two movements together cause pronation. Both the muscles also assist plantar flexion and together with the tibialis posterior – and the FDL muscles, it helps to control medio-lateral balance in walking (Travell & Simons, 1999).

In conclusion, the following measurable factors will be considered as possible role players in the precipitation or perpetuation of symptoms of CPCS and will therefore be taken into account during the individual assessments:

- muscle strength of the muscles in the lower leg;
- tightness of the soleus bridge over the posterior compartment; and



- during running the degree of forefoot abduction, the alignment of the Achilles tendon angle and movement patterns, e.g. the position of the calcaneus during heel strike and the ability to clear the foot easily during the swing phase will be observed.

#### **2.3.3.4. Normal running gait**

In order to identify abnormal movement components and compensatory mechanisms, which is often seen clinically in subject with symptoms of CPCS, it is necessary to analyse and compare the subjects' gait to what is considered as the norm for symptom free runners. From a biomechanical perspective, various types of pathology in the musculoskeletal system can alter mobility and muscular effectiveness in such a way that subjects substitute and develop compensatory mechanisms. This results in a walking/running pattern which is a combination of normal and abnormal movement patterns which increases the energy costs of running and compromises the functional versatility of the walker/runner (Perry, 1992). Running is similar to walking, but there are differences that need to be examined. Running requires greater balance, muscle strength, and range of motion than normal walking. Greater balance is required because running is characterized not only by an absence of double support periods observed in normal walking but also by the presence of float periods in which both feet are out of contact with the supporting surface (Levangie & Norkin, 2001). Walking/running is a complex activity because it is dependent on a series of interactions between two multi-segmental lower limbs, the trunk and the upper limbs. Normal walking/running gait repeats a basic sequence of motions that serve to progress the body along a desired path while maintaining weight-bearing stability, conserving energy and absorbing shock during ground contact. A gait cycle is defined as the time from heel strike to ipsilateral heel strike. Each gait cycle is divided into two periods, stance and swing. To facilitate observational analysis, the walking/running gait cycle is further divided into eight phases, namely: initial contact; loading response; mid-stance; terminal stance; pre-swing; initial swing; mid-swing and terminal swing. For each of these phases, the movement components taking place at the pelvis, the hips, the knees, the ankles and the feet must be analysed (Perry, 1992). The walking gait analysis of Perry (1992) will be applied for the gait analysis of the runners. This is summarized in Table 2.9.

**Table 2.9: Sequence of movement at the pelvis, hips, knees, ankles and feet during running (Perry, 1992)**

	<i>Weight acceptance</i>		<i>Single leg support</i>		<i>Swing leg advancement</i>			
<i>Joint</i>	Initial contact.	Loading response.	Mid stance.	Terminal stance.	Pre-swing.	Initial swing.	Mid-swing.	Terminal swing.
<i>Pelvis</i>	Anterior rotation.	Anterior rotation.	Neutral.	Posterior rotation.	Posterior rotation.	Posterior rotation.	Neutral.	Anterior rotation.
<i>Hip</i>	Flexion.	Flexion.	Neutral.	Extension.	Slight extension to neutral.	Flexion.	Flexion.	Flexion.
<i>Knee</i>	Neutral to slightly flexed.	Flexion.	Extended, but not completely.	Extended, but not completely.	Flexion.	Maximum flexion.	Less flexion.	Neutral.
<i>Ankle</i>	Neutral.	Plantar flexion.	Dorsi flexion.	More dorsi flexion.	Plantar flexion.	Less plantar flexion.	Neutral.	Neutral.
<i>Subtalar</i>		Pronation.		Gradual progression to inversion.	Neutral.			



Observational gait analysis is used to some extent by all health care professionals. It is the easiest and least expensive method of analysis. The gait cycle is observed with gross focus sequentially on stance, swing and float phase. The simplest form of motion analysis is the use of video-recording (Dugan & Bhat, 2005).

The risk factors for developing running injuries depend on a number of extrinsic factors such as velocity, training time, equipment (shoes) and running surface. Intrinsic factors such as the individual's physical characteristics and personality traits can also influence the likelihood of injury. The factors most associated with running related injuries of the foot and ankle include anatomical or biomechanical abnormalities, lack of flexibility (tightness in the gastrocnemius- and soleus muscles which can cause compensatory excessive pronation of the hind foot), poor strength or muscular imbalances, the type of shoe or orthotics used and type of running surface that the runner trains on (Hintermann & Nigg, 1998; Reinking, 2006).

Excessive eversion velocity has been associated with medial tibial stress syndrome (Viitasalo & Kvist, 1983). Excessive pronation of the hind foot will predispose individuals to injuries on the medial aspect of the lower extremities. Increased pronation is associated with injuries such as medial tibial stress syndrome, tibialis posterior tendonitis, bursitis of the tendon Achilles (or tendonitis), patello-femoral disorders, iliotibial friction syndrome and lower extremity stress fractures. However, specific anatomic abnormalities and abnormal biomechanics of the lower extremity are not correlated with specific injuries on a predictable basis (Hintermann & Nigg, 1998). In runners with excessively pronated hind feet, the muscles of the superficial and deep posterior compartments are required to contract harder and longer eccentrically to resist pronation after heel strike. At toe off, they work harder concentrically to accelerate supination. As fatigue sets in, these muscles fail to provide the normal degree of shock absorption which might lead to the development of stress fractures, teno-periostitis or compartment syndrome (Brukner, 2000). According to Brukner (2000), abnormal biomechanics is a major cause of injuries to the bone, periosteum and muscle. Biomechanical events occurring at the lower limb at the time of initial ground contact include shock absorption, joint stabilization of the pelvis, the hip, knee and ankle joints as well as foot flexibility.



Tight calf muscles will also restrict ankle dorsi-flexion and increase the tendency for excessive pronation, leading to increased internal rotation of the tibia (Brukner, 2000). According to Hintermann & Nigg (1998), a combination of excessive pronation and substantial movement transfer of foot eversion into internal tibial rotation is a better predictor of the development of overuse injuries than excessive pronation alone (the transfer will be small for individuals with low arches and high for those with high arches). The tibialis posterior muscle also seems to play a significant role in stabilizing the arch of the foot and as such in controlling the pronation movement at the ankle joint complex (Travell & Simons, 1999).

In summary it is clear that it is important to take biomechanical factors (intrinsic factors), which might play a role in the precipitation and perpetuation of the injury into account, when treating an injury such as CPCS. Based on the literature, it thus seems as though the following biomechanical factors should be taken into account when assessing the patient: dynamic pes planus, restricted ankle dorsi-flexion, increased hind foot inversion and the amount of internal tibial rotation (and the factors that might influence this). It is also important to bear in mind the extrinsic factors that might play a role in the presentation of symptoms of chronic compartment syndrome, namely shoes, training surfaces and intensity of training.

Since it seems as though most authors see excessive pronation as a role player in the precipitation and perpetuation of injuries, it becomes important to find an objective way of measuring the amount of pronation. A simplistic method of measuring the amount of hind foot pronation was proposed by Gould & Davies (1985); Johanson *et al.* (1994) as well as Hunt (1998). Subtalar joint eversion and inversion were determined with the subject positioned prone and the lower half of the calf off the edge of the plinth. Sliding callipers were used to identify midpoints on the calf and calcaneus, and lines were drawn along the midline on the posterior third of the calf and on the calcaneus. The axis of a standard goniometer was placed between the malleoli in the frontal planes. The stationary arm of the goniometer was placed over the line on the posterior calf, and the movable arm was placed over the line on the posterior calcaneus.





Hind foot over pronation accounts for, or is associated with, approximately 10% of all running injuries (Hintermann & Nigg, 1998). The normal degree of hind foot pronation varies between 6° and 12° (Hoppenfield, 2000). Hind foot over pronation, according to measures provided by Clarke *et al.* (1984), is defined as a pronation angle of more than 12° measured during running. None of the aforementioned however provided any information with regard to the standard deviation. Brukner & Khan (2007) indicated that fascia related injuries such as iliotibial band syndrome; shin splints and plantar fasciitis are all associated with an increased degree of hind foot pronation.

Brukner & Khan (2007) proposed that injury of connective tissue leads to an inflammatory response. This is followed by repair with scar tissue which could, if not subjected to the necessary forces during the healing period, result in irregular arrangement of fascia. This could cause restrictions and a compromise in the effective length of the fascia, which results in pain or malfunctioning throughout the body. Manheim & Lavett (1989) are of the opinion that tight fascia leads to abnormal biomechanics.

Soft tissue mobilizing techniques, such as myofascial release techniques (Manheim & Lavett, 1989), trigger point therapy (Travell & Simons, 1999) and specific soft tissue mobilizing techniques (Hunter, 1998) allow therapists to treat soft tissue dysfunctions. The ultimate goal of soft tissue mobilizing techniques is optimal body alignment, which allows for the most efficient use of energy for daily tasks (Barnes, 1990). Optimal body alignment will also facilitate normal movement patterns and normal biomechanics.

In terms of the modified theoretical framework for the pathogenesis of CPCS that was developed and discussed in Chapter 2, one can argue that the stresses that are induced in the fascial web as a result of restricted fascial movement in the clinical significant muscles, would also have an effect on the alignment of the feet with the rest of the body. It was thus decided to investigate the effect of the soft tissue mobilization of the clinical significant muscles in subjects with abnormal pronation. From the literature, abnormal pronation appears to be in excess of 12°. The normal degree of hind foot





pronation which varies between 6° and 12° (Hoppenfield, 2000), thus implies an average of around 9°.

#### **2.3.3.5. *Dorsi flexion at the ankle joint***

As previously stated, tight calf muscles will also restrict ankle dorsi-flexion and increase the tendency for excessive pronation. It therefore becomes important to measure the degree of dorsi flexion at the ankle. Markings need to be made on the lateral side of both ankles. Dots can then be made over the central part of the inferior border of the lateral malleolus and on the lateral side of the foot over the head of the fifth metatarsal bone. Another dot must be made over the tibia-fibular joint and the centre of the lateral malleolus. Thereafter, the subject must run barefoot on a treadmill with a calibrated running speed of 7.6 km/h, whilst being videotaped with a Sony digital video camera. This video material must then be copied to a specific software program (TV 2000 combined with Corel Draw version 11) in order to measure the degree of dorsi flexion at the ankle. The dots over the head of the fifth metatarsophalangeal joint and inferior border of the lateral malleolus must be joined with a straight horizontal line through Coral Draw. The dots over the centre of the lateral malleolus and the tibia-fibular joint must be joined with a straight vertical line using Corel Draw. Every measurement must be taken three times at different intervals of the mid and terminal stance of the running gait to calculate an average value.

The fact that the surgical release of fascia currently provides the only relative successful management of symptoms of CPCS, emphasizes the important role that non-compliant compartment fascia plays in the symptoms thereof. Since no study with positive outcome of conservative treatment aimed locally at the area of the symptoms could be found in the literature (Jackson & Bailey, 1975; Davey *et al.*, 1984; Martens *et al.*, 1984; Detmer *et al.*, 1985; Allen & Barnes, 1986; Froneck *et al.*, 1987; Melberg & Styf, 1989; Clanton & Solcher, 1994; Biedert & Marti, 1997; Blackman *et al.*, 1998; Schepsis & Lynch, 1998; Styf, 1998; Micheli *et al.*, 1999; Garcia-Mata *et al.*, 2001), the next logical step was to explore the literature on the current knowledge on fascia and its characteristics.



## 2.4. KNOWLEDGE OF FASCIA

### 2.4.1. Composition of normal fascia

The word “myofascial” connotes the bundled together, inseparable nature of muscle tissue (myo-) and its accompanying web of connective tissue (fascia) (Comerford, 2000; Myers, 2001). Any soft tissue mobilizations will therefore involve both the muscle and the fascia (myofascia) and various soft tissue mobilizing techniques such as trigger point release techniques (Travell & Simons, 1999), myofascial release techniques (Barnes, 1990; Manheim, 1994) and specific soft tissue mobilizing techniques (Hunter, 1998) will be used to mobilize the soft tissue.

Fascia is composed of connective tissue (CT). Schultz & Feitis (1996) have proposed that the organization of connective tissue in the leg is different from that of the arm. In the leg the connective tissue is structured for stability and in the arm it is more elastic for flexibility. In the forearm the interosseous membrane needs to be elastic so that the bones can rotate with the multidirectional use of the hand. In the lower leg, the interosseous membrane must be denser to support the leg and control rotation between the tibia and the fibula as the foot moves (Schultz & Feitis, 1996).

The properties of CT are determined by the amount, type and arrangement of the extracellular matrix (ECM). The ECM consists of three major types of macromolecules namely fibres, proteoglycans (PGs) and glycoproteins. The two most important fibrous components of the ECM are collagen and elastin, both insoluble macromolecular proteins. The striking feature of the most prominent collagens is their ability to resist tensile loads. Generally they show minimal elongation under tension and a part of this “elongation” is due to the straightening of fibres that are packed in various three-dimensional arrays (Culav *et al.*, 1999). Collagen fibres are coiled and their inter weaving allows for elastic displacement and return. When these fibres are densely matted or not aligned in the direction of movement, their elastic potential is dispersed. This is the case where there is thickening or bunching of the connective tissue (Schultz & Feitis, 1996). An increase in fascial thickness was found during biopsies in subjects diagnosed with CCS (Detmer *et al.*, 1985; Hurschler *et al.*, 1994 and Garcia-Mata *et al.*, 2001).



The second major component of the ECM is the PGs. Their mechanical functions include hydration of the matrix, stabilization of collagen networks and the ability to withstand compressive forces. The mechanical properties of CT such as the ability to resist tension, compression, extensibility and torsion are determined by the proportions of the matrix components. Generally, tissues with high collagen-fibre content and low amounts of PG resist tensile forces and those with high PG content, combined with a network of collagen fibres, withstand compression (Culav *et al.*, 1999).

Dense regular CT is a histological category of CT that includes ligaments, tendons, fascia and aponeuroses. As described above, these dense CT structures all share similar elements but differ in mechanical characteristics, primarily because of the arrangement and various proportions of their basic constituents. Fascia has collagen bundles, but the bundles are organized into multilayered sheets or lamellae. The bundles within individual layers are roughly parallel but often have some undulations or waviness. Adjacent layers may not have the same fibre direction, although fibres will often pass between adjacent layers as well as into adjacent loose CT. Ground substance and elastin content is low in fascia (Threkeld, 1992) and fascia is therefore classified as mainly non-elastic (Brukner, 2000).

According to Schwind (2006), the proportion of collagen and elastin fibres within any area of fascia depends upon the functional demands placed upon the tissue in that area. If there are strong tensile stresses on the tissue, then the collagen portion will predominate and there will be less elastic fibres. In the periphery, such as the lower limb, where potentially dangerous forces are greatest, the fascia tends to be thicker and denser. When the work load is heavy, thickened fascia can completely replace muscle bundle e.g. the iliotibial tract and the lumbosacral aponeurosis (Paoletti, 2006).

The function of fascia is to constrain muscles to a specific area and to protect muscles. Fascia prevents muscles from tearing and also prevents muscle hernias. If muscles were not surrounded by fascia, their action would be uneven and uncoordinated and would rupture and tear more easily. Without the structural protection of the fascia, the ability of the muscle to generate and transfer power and movement would be limited



(Robertson, 2001). In a study done by Huijing & Baan (2001), muscles within the anterior tibial compartment and within the peroneal compartment were excited simultaneously and maximally, first, with the anterior compartment intact, second, after a blunt dissection of the anterior and the lateral interface of extensor digitorum longus- and tibialis anterior muscles, thirdly, after a full longitudinal fasciotomy of the anterior tibial compartment and finally, after full removal of tibialis anterior- and extensor hallucis longus muscles. It was found that the length-force characteristics were significantly changed by these interventions. Blunt dissection caused a force decrease of approximately 10% at all lengths. This indicates that intermuscular connective tissue mediates significant interactions between adjacent muscles. A full lateral compartmental fasciotomy increased optimal length and decreased active slack length, leading to an increase of length range (by approximately 47%), while decreasing optimal force. Based on these results, they concluded that extra muscular connective tissue has a sufficient stiff connection to intramuscular connective tissue to be able to play a role in force transmission.

Every muscle of the body is surrounded by a smooth fascia sheath, every muscular fascicle is surrounded by fascia, every fibril is surrounded by fascia, and every microfibril down to the cellular level is surrounded by fascia (Barnes 1990). Therefore fascia plays a key role in length and function of the muscular component. The word “myofascial” connotes the bundled together, inseparable nature of muscle tissue (myo-) and its accompanying web of connective tissue (fascia) (Comerford, 2000; Myers, 2001).

In summary, patients with CT problems affecting movement are frequently examined and treated by physical therapists. Knowledge of the CT matrix composition and its relationship to the biomechanical properties of these tissues, particularly the predictable responses to changing mechanical forces, offers an opportunity to provide a rational basis for treatments. The complexity of the interplay among the components, however, requires that further research be undertaken to determine more precisely the effects of treatments on the structure and function of CT (Culav *et al.*, 1999).

#### 2.4.2. Manual therapy techniques for connective tissue dysfunction

Connective tissue problems can be caused by CT shortening or by diminished CT mobility (Threkeld, 1992). Since the CT is responsive to changes in the mechanical environment, both naturally occurring and applied (Culav *et al.*, 1999), the following soft tissue mobilizing techniques can be used by physical therapists in order to effect the healing of the CT, following injury:

- specific soft tissue mobilization (Hunter, 1998)
- myofascial release techniques (Manheim, 1994)
- trigger point release techniques (Travell & Simons, 1999)

The actual mechanism responsible for the symptoms of CCS is unclear. It has however been speculated that the process of CCS may result from repeated micro injury to the involved compartment (Ross, 1996). According to the proposed pathogenesis of CPCS as illustrated by Clanton & Solcher (1994) micro tears are caused by the increase in the intra-compartmental pressure. Therefore subjects with symptoms of CPCS are normally seen during the mechanical or remodelling phase of healing. The objective during this stage is to restore the biomechanical properties of the tissue as close as possible to normal by the formation of a functional scar. A functional scar is a scar in which the collagen tissue is formed in the same line as the tissue it is replacing and the scar tissue is as long as the tissue it is replacing. It is not adhered to any adjacent tissue and is of sufficient quality and quantity to accept the compressive, distracting and shearing stresses to which it will be subjected. It must be able to shorten, lengthen and move in relation to its surrounding tissues (McGonigle & Matley, 1994).

If cross link formation is responsible for a degree of stiffness, mobilization techniques should be applied far enough into resistance to disrupt the cross link formation in order to produce change (Hendricks, 1995). Specific soft tissue mobilizations according to him can be used to disrupt the cross link formation. These techniques apply a longitudinal stretch to the tissue by exerting a manual force applied at 90° to the soft tissue. It is applied transversely to the line of collagen fibres but in the same plane as the fibres. The force is increased into resistance and applied for 3 x 60



seconds. The techniques should be followed up with stretches done by the subject, each stretch lasting 30 seconds (Hunter, 1998).

Myofascial release techniques can be used to lengthen shortened CT as well as to disrupt cross links. The aim of myofascial release techniques is to break up any cross restrictions and to restore the ground substance to its normal viscosity. Manheim (1994) is of the opinion that any myofascial restriction at, near or far from a target muscle causes distortions not only in the target muscle but in other muscles as well. She is therefore of the opinion that all myofascial restrictions must be treated and released to restore proper alignment and energy efficient movement to the entire system. When the location of the fascial restriction is determined, gentle pressure is applied in its direction. This has the effect of pulling the elasto-collagenous fibres straight. When pressure is first applied to the elasto-collagenous complex, the elastic component is engaged. This has a springy feel. The elastic component is slowly stretched until the hands stop at a firm barrier. This is the collagenous component. This barrier cannot be forced; it is too strong. Instead, gentle sustained pressure will release it. This fact has to do with viscous flow phenomenon, that is, a low load (gentle pressure) applied slowly will allow a viscous medium to flow to a greater extent than a high load (quickly applied pressure). By increasing the viscosity of the ground substance this allows the collagen fibres to rearrange themselves resulting in increased tissue length (Barnes, 1990). The physical therapist follows the motion of the tissue, barrier to barrier, until freedom from abnormal restriction is felt (Barnes, 1990).

Trigger point therapy is another form of myofascial release that is proposed to cause a change in tissue length by allowing the involved muscle to lengthen fully. Travell & Simons (1999) define a myofascial trigger point as a hyperirritable locus within a taut band of skeletal muscle, located in the muscular tissue and/ or its associated fascia. A trigger point prevents full lengthening of the muscle and weakens the muscle. The spot is painful on compression and can evoke characteristic referred pain and autonomic phenomena. The relaxed muscle is stretched to the verge of discomfort, where after a tolerable, painful pressure is exerted on the trigger point. When the pain starts to abate, the pressure is increased until the subject does not experience the pain anymore. According to these authors normal muscles do not contain trigger points.



### 2.4.3. Reflection

As stated the actual mechanism responsible for the symptoms of CCS is unclear, but it has been speculated that the process may result from repeated micro injury to the fascia (Ross, 1996). Eisele & Sammarco (1993) have found that the muscle within a compartment may undergo a 20% volumetric increase. If the compartment margins are unable to accommodate this increase in size, they may be stretched and injured. Each stretching trauma results in inflammation which, as it resolves, contributes to scarring. Because scar tissue tends to contract as it matures, this could decrease the compartment size and further contribute to the compartment's inability to accommodate volumetric enlargement during the next exercise bout. The other possibility is that the scar tissue may hypertrophy, causing it to become thicker and less elastic (Hutchinson & Ireland, 1994). This will further prohibit its ability to compensate for changes in compartmental volume (Ross, 1996). There are research reports that support this explanation in that the fascial tissue from compartments with CCS was demonstrated to be thicker than normal (Detmer *et al.*, 1985).

As mentioned (in section 2.14), the arrangement of the fascial fibres, allows a certain amount of deformation of viscous connective tissue. With injury, as discussed above, together with the formation of adhesions (macro structure), the arrangement of the fibres has been changed and this might affect the amount of deformation/ length that was available. When these fibres are densely matted or not aligned in the direction of movement, their elastic potential is dispersed (Culav *et al.*, 1999). This could be the case where there is thickening or bunching of the fascial connective tissue (Detmer *et al.*, 1985; Hurschler *et al.*, 1994; Schultz & Feitis, 1996; and Garcia-Mata *et al.*, 2001). Fascial thickening develops as a response to forces of tension and mechanical demands on the fascia during running. Hurschler *et al.* (1994) have found that fascia samples removed from the affected compartment of a patient who has been diagnosed with CPCS were thicker and structurally stiffer in the axial direction compared to fascia from a normal compartment.

The problem appears to be self-perpetuating, as the tissues have insufficient extensibility (decrease in the effective functional length of the myofascial system) to allow the desired movement to take place and pain is produced when the tissue is





overstretched (as what happens during running). This triggers the whole inflammatory response again. This result in further thickening and the formation of adhesions at tissue interfaces (Holey, 1995).

The healing of fascia (type 1 connective tissue), following injury, follows the same classic sequence of inflammation, repair and remodelling as found with other soft tissue injuries. Fascia shrinks when it is inflamed and is slow to heal because of a poor blood supply; it is a focus of pain because of its rich nerve supply (Manheim, 1994). However, micro-structurally, the end results of the healing / reorganizing process in connective tissue is that the tissue:

- has a more irregular arrangement (the arrangement and the alignment are a result of the mechanical stresses applied to the tissue);
- has lower water content; and
- contains more random cross-links between fibres, fibre bundles and adjacent tissues.

As the collagen fibres are more randomly aligned with respect to forces applied to the tissue, the fibres must resist forces that are not parallel to their longitudinal axes. This is a task for which collagen is not structurally designed. In addition the loss of water diminishes the ease with which the collagen bundles might slide past one another (Threkeld, 1992).

From the literature it can thus be concluded that fascia, in the normal healthy state, is relaxed and wavy in configuration. It has the ability to move without restriction. When connective tissue experiences physical trauma, scarring or inflammation, the fascia loses its pliability. It becomes tight, restricted and a source of tension to the rest of the body. Trauma, such as repetitive strain injuries, has accumulative effects. The changes which trauma causes in the fascial system influences comfort and the functioning of the body. Fascia is a non-elastic, integrated network, where collagen fibres are coiled and their interweaving allows for elastic displacement and return. Fascia shrinks when it is inflamed and is slow to heal because of a poor blood supply. Fascia of affected compartments in CPCS was demonstrated to be thicker and structurally tighter in the axial direction compared to fascia removed from a normal compartment. Myofascial release techniques, specific soft tissue mobilizations and





trigger point therapy are proposed to cause a change in tissue length, by rearranging the collagen fibres that occurs in the tissue in response to the viscoelastic effects of the techniques. The release of an active trigger point also causes a change in tissue length by allowing the involved muscle to lengthen fully. Myofascial release techniques and specific soft tissue mobilization techniques can be used to disrupt cross link formations. Conservative treatment of the local fascial sheath and contained muscles has to date not been very successful in alleviating the symptoms of CPCS.

Surgical release for compartment syndrome has demonstrated moderately good results, but could have several disadvantages. Several factors affecting normal gait and running patterns are indicated by the literature as possible role players in the symptoms of CPCS. These include tightness of the calf muscles, muscle strength of flexion of the distal phalanges of the toes, the degree of plantar and dorsi flexion range of movement at the metatarsophalangeal joints when the foot is in contact with the ground, the medio-lateral balance in the mid- to late-stance phase, the maintenance of equilibrium when the weight is on the fore foot, an increased amount of fore foot abduction, hind foot valgus and a greater than normal Achilles tendon angle. It also includes external factors such as training time, shoes and running surfaces.

The development of the methodology for the management of the case studies to follow will be based on the aforementioned literature review and will be discussed in the next chapter. The major discrepancies found in the literature research that will be addressed are:

- the lack of success with conservative treatment and
- that the conservative treatments are not aimed specifically at the fascia that plays a crucial role in the condition as determined through the literature research.

## **2.5. THE CONTINUITY OF THE SOFT TISSUE LINKS**

### **2.5.1. Introduction**

This section deals with an extension of the literature research based on the findings of the first three case studies. The interventions had better results with the application of



the intervention techniques to areas external to the calf area. The application of the treatment techniques applied distally, further proximally, as well as anteriorly from the posterior compartment, showed positive results in terms of the outcome measures which were elected, i.e. the intensity of pain and the ability of the subject to run. It was then argued that if the soft tissue mobilization of the posterior compartment had no positive effect, the fact that the treatment of the more proximal and anterior soft tissue led to a reduction in the pressure in the posterior compartment, then the soft tissue of the calf muscles must be linked to these posterior muscles in order to have an effect on the symptoms. Based on this argument it was decided to do an additional literature research in order to establish whether such anatomical links between the calf muscles and the more proximal anterior and posterior myofascial structures existed.

As indicated in Chapter 1, little effective research results emerged on myofascial related issues after the “classic” work done by Travel and Simons (Harden, 2007). The results that emanated from this research make a major contribution in the sense that it provides the first focussed perspective on the anatomical links between the calf muscles and the myofascial structures and associated muscle interdependencies that exist. These interdependencies between the more proximal muscles and myofascial structures allow for the development of successful treatment interventions for the symptoms of CPCS external to the problem. The resulted form the literature research which follows, provides the first comprehensive identification of the interdependencies of muscles linked to the myofascial tissue of the posterior compartment. The progressive development of these links is discussed in the following sections. The final integrated perspectives of these links are covered in Chapter 4 which deals with research results.

### **2.5.2. Description of the myofascial links**

As discussed in the previous section, every muscle of the body is surrounded by a smooth fascia sheath, every muscular fascicle is surrounded by fascia, every fibril is surrounded by fascia, and every micro- fibril down to the cellular level is surrounded by fascia (Steffen, 1996). The word “myofascial” connotes the bundled together, inseparable nature of muscle tissue (myo-) and its accompanying web of connective tissue (fascia) (Comerford, 2000; Myers, 2001). Anatomical links found between the

calf muscles and the more proximal anterior and posterior soft tissue will therefore be described as myofascial links.

In the selection of a starting point for the description of the inter dependence of muscles and ligaments and fascia, it was decided to follow a functional approach. In the analysis of gait, the pelvis was used as the starting point to describe how the different muscles and ligaments are linked through the fascia. The anterior and the posterior myofascial links are described separately.

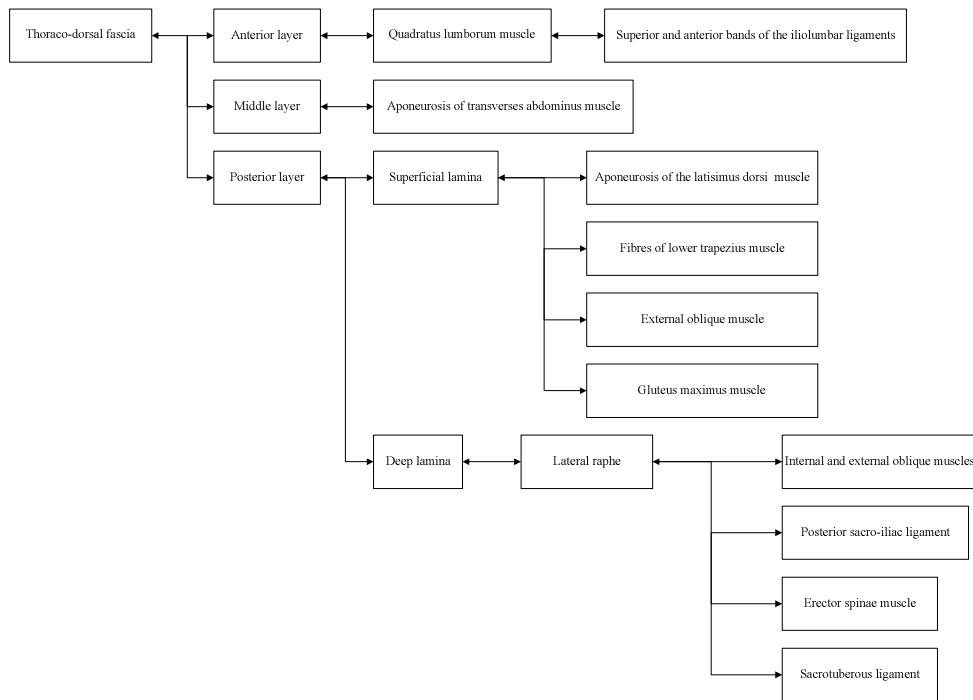
### **2.5.3. Posterior myofascial links of the trunk**

The description of the posterior myofascial links will commence with a description of the thoraco-dorsal fascia. The thoraco-dorsal fascia (TDF) is comprised of three layers, namely the anterior-, the middle- and the posterior layer (Lee, 1996; Bogduk, 1997).

The anterior layer covers the anterior aspect of the quadratus lumborum muscle (Bogduk, 1997), which in turn attaches to the superior and the anterior bands of the iliolumbar ligament (Luk *et al.*, 1986; Bogduk, 1997).

The middle layer lies posterior to the quadratus lumborum and provides origin to the aponeuroses of the transversus abdominus muscle. The posterior layer of the TDF is comprised of two laminae, namely the superficial- and the deep lamina. The superficial lamina is predominantly derived from the aponeuroses of the latissimus dorsi muscle (Lee, 1996) and receives some fibres from the external oblique muscle and the lower fibres of the trapezius muscle (Vleeming, 1995). The superficial lamina blends with the fascia of the gluteus maximus muscle.

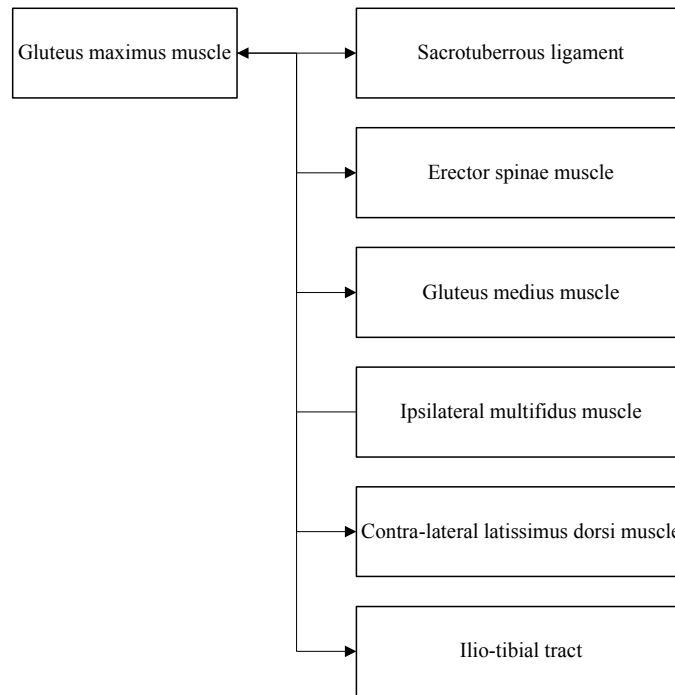
The deep lamina of the TDF attaches to the posterior sacroiliac ligaments, the lateral raphe and blends with the middle layer of the TDF. Some of the fibres of the deep lamina blend with the deep fascia of the erector spinae muscle and the sacrotuberous ligament. These links are illustrated schematically in Figure 2.5.



**Figure 2.5: The thoraco-dorsal fascia and its links**

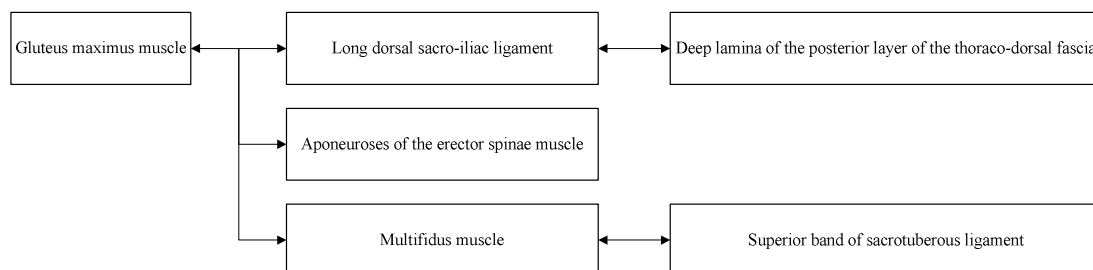
The gluteus maximus muscle links to various muscles through facial attachments as illustrated in Figure 2.6. The fascia of the gluteus maximus muscle has attachments to the sacrotuberous ligament, the erector spinae muscle and the fascia covering the gluteus medius muscle (Lee, 1996). The gluteus maximus muscle further blends with the ipsilateral multifidus muscle through the raphe of the thoraco-dorsal fascia (Willard, 1997) and with the contra-lateral latissimus dorsi muscle through the superficial lamina of the thoraco-dorsal fascia (Vleeming, 1995).

Fibres from the multifidus muscle also blend with the sacrotuberous ligament. The majority of the fibres of the gluteus maximus muscle inserts into the iliotibial tract of the fascia lata (Farfan, 1978). Medially, fibres of this ligament attach to the deep lamina of the posterior layer of the thoraco-dorsal fascia and the aponeuroses of the erector spinae muscle (Vleeming, 1996). Connections have also been noted between the long dorsal ligament and the multifidus muscle (Willard, 1997). Laterally the fibres of the multifidus muscle blend with the superior band of the sacrotuberous ligament.



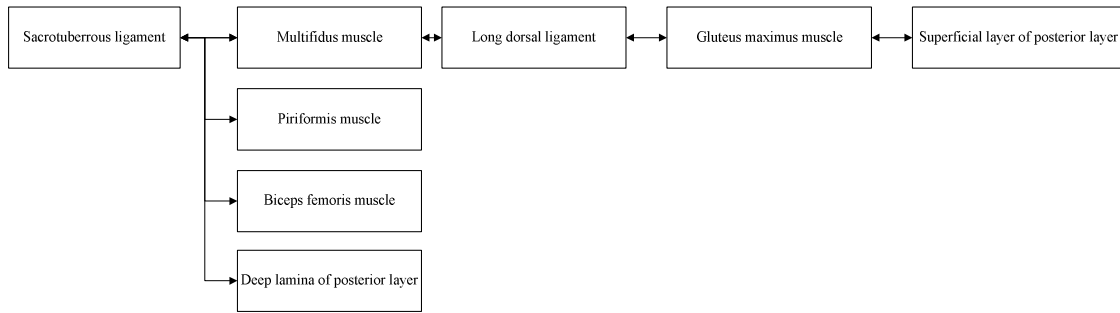
**Figure 2.6: The gluteus maximus muscle and its fascia links**

Myofascial links are also continuous through the sacroiliac ligaments (illustrated in Figure 2.7). The long dorsal sacroiliac ligament is covered by the fascia of the gluteus maximus muscle (Lee, 1996).



**Figure 2.7: The continuity of the myofascial links through the sacroiliac ligament**

The lateral band of the sacrotuberous ligament spans the piriformis muscle (Lee, 1996). In a certain percentage of humans, the sacrotuberous ligament receives some fibres from the biceps femoris muscle (Vleeming, 1996). The fibres of the biceps femoris muscle can bridge the ischial tuberosity completely to attach directly to the sacrotuberous ligament. These links are illustrated in Figure 2.8.



**Figure 2.8: The myofascial links connecting to the sacrotuberous ligament**

#### 2.5.4. Anterior myofascial links of the trunk



**Figure 2.9: An illustration of some of the anterior myofascial links**

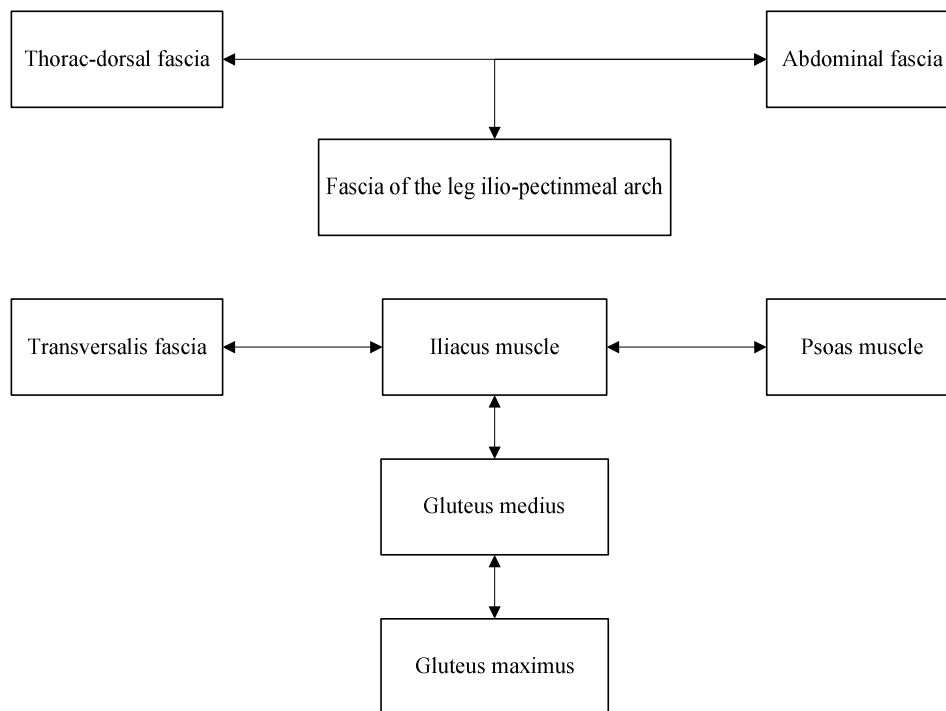


The description of the anterior myofascial links will commence with a description of the myofascial links between the abdominal (core) muscles. Anteriorly, the external oblique muscle (the most superficial of the abdominal muscles) inserts into a complex anterior aponeuroses (Lee, 1996). The internal oblique muscle lies between the external oblique and the transversus abdominus muscles. It arises from a part of the inguinal ligament and the lateral raphe of the thoraco-dorsal fascia. The anterior fibres arising from the inguinal ligament blend with the aponeuroses of transversus abdominus (Lockhart *et al.*, 1974). The transversus abdominus muscle is the deepest abdominal muscle and arises from part of the inguinal ligament, the lateral raphe of the thoraco-dorsal fascia and the internal aspect of the lower six costal cartilages interdigitating with the costal fibres of the diaphragm. The muscle's upper and middle fibres blend with the fascial envelope of the rectus abdominus muscle. Inferiorly the muscle blends with the insertion of the internal oblique muscle (Lockhart *et al.*, 1974).

There is a strong myofascial connection between the trunk and the lower limb. The thoraco-dorsal and the abdominal fascia blend with the fascia of the leg. The transversalis fascia is continuous with the iliac fossa, psoas major and psoas minor muscles. The iliacus muscle fibres, arising from the iliac fossa, converge to merge with the lateral aspect of the tendon of the psoas major muscle. The ilio-pectineal arch is a condensation of the psoas and iliac fasciae. This fascia is laterally continuous with the transversalis fascia (Lockhart *et al.*, 1974). From the iliac crest, the fascia descends over the gluteus medius muscle before splitting to envelop the gluteus maximus muscle (Lee, 1996).

The fascia of psoas muscle blends anteriorly into the anterior longitudinal ligament. The anterior longitudinal ligament is continuous with the pre-vertebral fascia which is the deepest anterior fascia layer and encloses the vertebral column and its muscles (longus colli, longus capitis, rectus capitis anterior and rectus capitis lateralis) (Romanes, 1981).

The pre-vertebral fascia is attached to the anterior scalene muscle, the scalenus medius muscle and the levator scapula and the splenius capitis (Romanes, 1981; Clemente, 1996).



**Figure 2.10: Examples of anterior and posterior trunk fascia links to one another**

The scalenus anterior muscle attaches to the axillary sheath (McMinn, 1995), the brachial fascia on the flexor side (Frick *et al.*, 1991), the latissimus dorsi (Frick *et al.*, 1991) and the clavi-pectoral fascia (Lockhart *et al.*, 1974). The brachial fascia is attached to the deltoid fascia on the extensor side (Frick *et al.*, 1991) and to the ante-brachial fascia. The clavi-pectoral fascia is attached to the subclavius muscle which links the pectoralis major to the serratus anterior muscle and the pectoralis minor muscle (Lockhart *et al.*, 1974). The pre-tracheal fascia which envelops the sterno-hyoid, the sterno-thyroid, the omo-hyoid and the thyro-hyoid muscles has a posterior link to the pre-vertebral fascia via the anterior scalene muscle. The pre-tracheal fascia also has an anterior-lateral link to the investing (most superficial) fascia via the sternocleidomastoid muscle (Romanes, 1981; Clemente, 1996). The investing fascia encloses the sternocleidomastoid and the upper trapezius muscles. The deepest layer of the investing fascia passes behind the clavicle and is attached to the sheath of the subclavius muscle (Romanes, 1981). This layer connects with the pre-vertebral fascia via the pectoral fascia.





### 2.5.5. Fascia of the leg

It has already been shown that there is a strong myofascial connection between the trunk and the lower leg. The tendinous fibres of the anterior-medial half of the tensor fascia lata muscle extend down the thigh and curve anteriorly at the level of the patella to interweave with the lateral patellar retinaculum and the deep fascia of the leg superficially to the patellar ligament (Travell & Simons, 1999). The fascia is continuous in the thigh with two intermuscular septa which attach to the linea aspera, namely the lateral intermuscular septum, which separates the extensors from the flexors, and the medial intermuscular septum, which separates the hamstrings from the adductors (Romanes, 1981). The medial intermuscular septum is strengthened by the fascia from the adductor magnus muscle (Romanes, 1981). Fascia of the adductor magnus muscle is also attached to the medial tibial collateral ligament (Romanes, 1981; Clemente, 1996) and to the medial head of the gastrocnemius muscle (Clemente, 1996).

There is a dense fascia network in the popliteal fossa. The tibial collateral ligament is attached to the fascia of the semimembranosus (Romanes, 1981; Clemente, 1996), and the semimembranosus fascia is attached to the oblique popliteal ligament (Clemente, 1996) and to the popliteal aponeuroses (Clemente, 1996). The oblique popliteal ligament is attached to the arcuate ligament, which is attached to the fibular collateral ligament (Clemente, 1996) and to the popliteal aponeuroses (Crafts, 1981). The popliteal aponeurosis inserts into the soleus line (Romanes, 1981) and the deep fascia of the leg is strengthened on the medial side by the insertion of the tendons of sartorius, gracilis and semitendinosus (pes anserine). The crural fascia forms the anterior, the posterior and the transverse intermuscular septum (Lockhart *et al.*, 1974). The anterior intermuscular septum separates the anterior (tibialis anterior, extensor hallucis longus, extensor digitorum and peroneus tertius) and the lateral (peroneus longus and peroneus brevis) compartments. The transverse intermuscular or interosseous membrane separates the anterior and the lateral compartments from the deep posterior (popliteus, flexor digitorum longus, tibialis posterior and flexor hallucis longus) compartment. The posterior intermuscular septum separates the superficial (gastrocnemius, soleus and plantaris) and the deep posterior compartments. Distal to the flexor retinaculum the crural fascia becomes the plantar aponeuroses.



### 2.5.6. Conclusion

In this chapter the utilisation of case study research as a methodology for exploratory research has been reviewed in a fair amount of detail. This has been done in the light of the fact that research in general in the medical sciences is normally based on quantitative methodologies as a result of the fact that the theoretical models in general are well developed. In these situations it is common to find research designs based on classical research designs with control groups; and double blind procedures aimed at the elimination of any form of bias during the experimentation. The lack of success with conservative treatment and by implication, a lack of a reliable theoretical model that enables the prediction of the results of interventions thus directs the research to a qualitative approach.

From the literature research it is evident that fascia plays a major role in the condition. It is also worthy to note that the only treatment with a degree of success is the surgical release of the fascia. The fact that the nature of this solution is often temporary suggests that the root cause of the problem had not been addressed.

It has also been identified that a number of conditions could mimic symptoms of chronic compartment syndromes. These include stress fractures, tendonitis (for example that of flexor hallucis longus), gastrocnemius strain, periostitis, spinal stenosis, radiculopathy, entrapments of arteries and nerves, claudication (arteriosclerosis), and effort induced venous thrombosis (Hutchinson *et al.*, 1998). These thus provide the basis for the selection criteria for the subjects in this research project.

In the first part of the literature review on CPCS it was established that fascia exposed to physical trauma, scarring or inflammation loses its pliability. Any restriction somewhere along the myofascial chain will cause a decrease in the effective length of the myofascial chain. Such a restriction in length will induce stresses in the web during activities which require extended ranges of movement such as running. These stresses will be transmitted via the inelastic myofascial web to areas such as the posterior compartment where it will induce pressure in the posterior compartment. Whilst running, the additional forces exerted on an already compromised myofascial chain, will cause micro trauma and inflammation. The fascia will then become tight,



restricted and a source of tension to the rest of the body due to the continuous nature of the myofascial web that links muscle and ligaments of various parts of the body with each other. It is therefore conceivable that a restriction or tightness in any of the more proximal muscles linked to that of the posterior compartment might contribute to the stresses in the fascia which will lead to associated pressure in the compartment.

The knowledge gained during this part of the literature review provided a new perspective on the possible causal relationships which exist in the support of the symptoms of CPCS. These new perspectives were also supported by the research results with the latter case studies where interventions outside the calf area solicited positive responses. These findings enabled the development of new treatment interventions based on a firmer theoretical footing.



## CHAPTER 3

### METHODOLOGY

#### 3.1. INTRODUCTION

This chapter deals with the research methodology. The first section deals with the basic research design for the exploratory phase of the research. It provides the basic design which was progressively adapted based on the results achieved with each of the individual case studies. These changes in the approach, research and investigative questions, theoretical framework, associated propositions, rival theories, and techniques will be highlighted with the results of the individual case studies presented in Chapter 4.

#### 3.2. EXPLORATORY RESEARCH DESIGN

The literature research has revealed that the conservative treatment approaches to the condition, is generally not successful (Edwards *et al.*, 2005). The objective with the exploratory research was thus to gain insight into the underlying theoretical framework responsible for the development of the symptoms of Chronic Posterior Compartment Syndrome (CPCS) in order to create an understanding for the lack of success in this regard. As a result of the exploratory nature of this research, it was decided to make use of a case study approach (Holloway, 1997). Due to the exploratory nature of this phase a flexible research design for the first case studies was elected. As the research became progressively more focussed, the research designs were adapted accordingly.

The objective of the first group of case studies was to explore the causal relationships of the condition. As a first step, the adequacy of the current theoretical framework was assessed as basis for the development of a potential treatment approach for the symptoms. If this proved unsuccessful, the next step would be to adapt the current framework or to develop a new one that could be used as basis for replication logic in the research.



In the following sections, the basic design for the case studies used during the exploratory design, will be reflected. This design was progressively adapted based on the insights gained from preceding cases. This typically included changes in investigative questions, the theoretical framework and associated propositions, rival theories and treatment interventions. These changes or modifications to the basic design are not discussed in this chapter, but will be progressively addressed in Chapter 4 as the rival theories are disposed off.

### **3.2.1. The basis for exploratory research design**

The framework (Yin, 2003) which was developed in Chapter 2 for the case study protocol will be used as the basis for research design. This framework included:

#### *General aspects*

- Design classification;
- Unit of analysis;
- Subjects;
- Database; and
- Ethical considerations.

#### *Specific issues*

- Overview;
- The research question;
- Investigative questions;
- Theoretical framework;
- Propositions;
- Rival theories;
- Data collection;
- Schedule and reviews;
- Criteria for interpreting results;
- Variables and associated measures;
- Instrumentation/procedure;

- Intervention;
- Data recording; and
- Quality assurance measures.

The basic design for the exploratory research will be reflected in the following section.

### 3.2.2. Design classification (Scholz & Tietje, 2002)

The group of case studies dealing with the exploratory research is classified as a *Type 4: Multi-case; Embedded design* as reflected in Table 3.1. Each of the individual case studies however is classified as a *Type 2: Single-case; Embedded design* in its own right. Table 3.1 provides a brief classification of the exploratory group of research designs based on a number of dimensions that are self-explanatory in the light of the previous literature review in Chapter 2.

**Table 3.1: Design classification of Case Study 1**

<i>Dimension</i>	<i>Classification</i>
<i>Basic design:</i>	<i>Type 4: Multi-case; Embedded</i>
<i>Motivation:</i>	<i>Instrumental</i>
<i>Epistemological status:</i>	<i>Exploratory</i>
<i>Purpose:</i>	<i>Research</i>
<i>Data:</i>	<i>Qualitative and quantitative</i>
<i>Format:</i>	<i>Unstructured</i>
<i>Synthesis:</i>	<i>Both quantitative as well as qualitative information will be used for a narrative assessment of the current theoretical model</i>
<i>Synthesis strategy:</i>	<i>Pure case or Leibnizian model</i>

### 3.2.3. Unit of Analysis

The main unit of analysis is the *individual subject* who suffers from CPCS. During the experimentation alternative units of analysis will be explored on a progressive basis and could include aspects such as the *treatment process*, or *events* that trigger positive response.

### 3.2.4. Subjects

#### 3.2.4.1. Inclusion criteria

For inclusion in the research project the subjects had to meet the following inclusion criteria. All subjects recruited had to:

- Be between 18 and 50 years of age;
- Actively participate in races with a distance of 10 kilometres or more;
- Be willing to run throughout the intervention period;
- Have symptoms of chronic posterior compartment syndrome in one or both lower legs; and
- Pass a subjective examination (interview) and a process of differentiation as described in the literature, to ascertain whether the symptoms were due to CPCS and not a stress fracture, tendonitis, calf muscle sprain, periostitis, spinal stenosis, radiculopathy, entrapment of arteries and nerves, claudication and effort induced venous thrombosis.

#### 3.2.4.2. Exclusion criteria

Such subjects were excluded based on the following criteria:

##### ***Stress fractures:***

- The subject had to have no pain at night or at rest;
- Percussions all along the length of the tibia with a patella hammer had to be painless;
- Ultrasound therapy applied over the painful posterior-medial aspect of both lower legs had to be painless;

***Tendonitis:***

- The intensity of pain should have increased and not have improved after warming up;

***Lumbar nerve root:***

- The subject should not have had any back pain;
- The subject should have had full range of all the lumbar physiological movements (flexion, extension, rotation and side flexion); and
- The neurological assessment should have been normal with regard to sensation, reflexes and motor strength of the L4, L5 and S1 referral/innervated areas.

***Spinal stenosis:***

- Lumbar spinal extension should have been normal; and
- No correlation should be reported between pain and the gradient of the running surface.

***Nerve entrapments:***

- The subject should not have experienced any tingling or burning sensation or pain behind the knee.

***Vascular conditions (popliteal artery entrapment):***

- There should not have been discoloration of the toes;
- The pain should not have increased with elevation of the leg; and
- The dorsalis pedis and posterior tibialis pulses should have been normal.

***Exercise-induced venous thrombosis***

- The subject should not have/ had any exercise-induced thrombosis.

The rationale for exclusion criteria was to eliminate subjects with conditions which could mimic symptoms of CPCS.

**3.2.4.3. Recruitment of subjects**

Subjects were recruited by means of an e-mail to all the registered running clubs in Pretoria; as well as through referrals by medical practitioners specialising in sport





injuries. The latter also included a podiatrist known in the running community for assisting runners with symptoms of CPCS. The e-mail described the symptoms of CPCS in detail and was distributed to all the different club members via the individual club's weekly newsletter to members. The e-mail is contained in Appendix 3. The medical practitioners consisted of three orthopaedic surgeons and three general practitioners, specialising in sport injuries.

Seven subjects were recruited over a period of six months from January 2002 to June 2002. One of the seven subjects sprained his ankle's lateral ligament (grade 2 ligament injury) one week after being recruited and was therefore excluded from the study. The subjects recruited were assessed by the researcher to ensure that they complied with the inclusion criteria and were then treated in the facilities of a private physiotherapy practice (i.e. the researcher's facilities) in Pretoria.

#### **3.2.4.4. Randomness**

The selection process is considered as random. The researcher, other than the pre-specified inclusion and exclusion criteria, had no way of influencing the selection process and the subjects can therefore be viewed as a representative sample of the running population with symptoms of CPCS.

#### **3.2.5. Database**

All the data are filed in hardcopy in a manual filing system. Transcribed data onto magnetic media are stored in MS Office formats on hard disc as well as removable hard disc (flash discs and DVD). The latter includes video material which is stored in this manner as well as on video tape.

#### **3.2.6. Ethical considerations**

The research was approved by the Student Ethics Committee of the Faculty of Health Sciences and the Postgraduate Committee of the School of Health Care Sciences (at the University of Pretoria) during October 2002 (protocol number: S166/2002; Appendix 1). The informed consent which had to be signed by the individual subjects is contained in Appendix 2.

## *Specific issues*

### **3.2.7. The research question**

The initial research question for Case Study 1 has been formulated as:

*“Does the existing theoretical model for CPCS provide for a logical model of proof for predicting replication in experimental results, and does it allow for inference of the causal relationships under investigation?”*

### **3.2.8. Investigative questions**

The investigative questions are reflected in the table below.

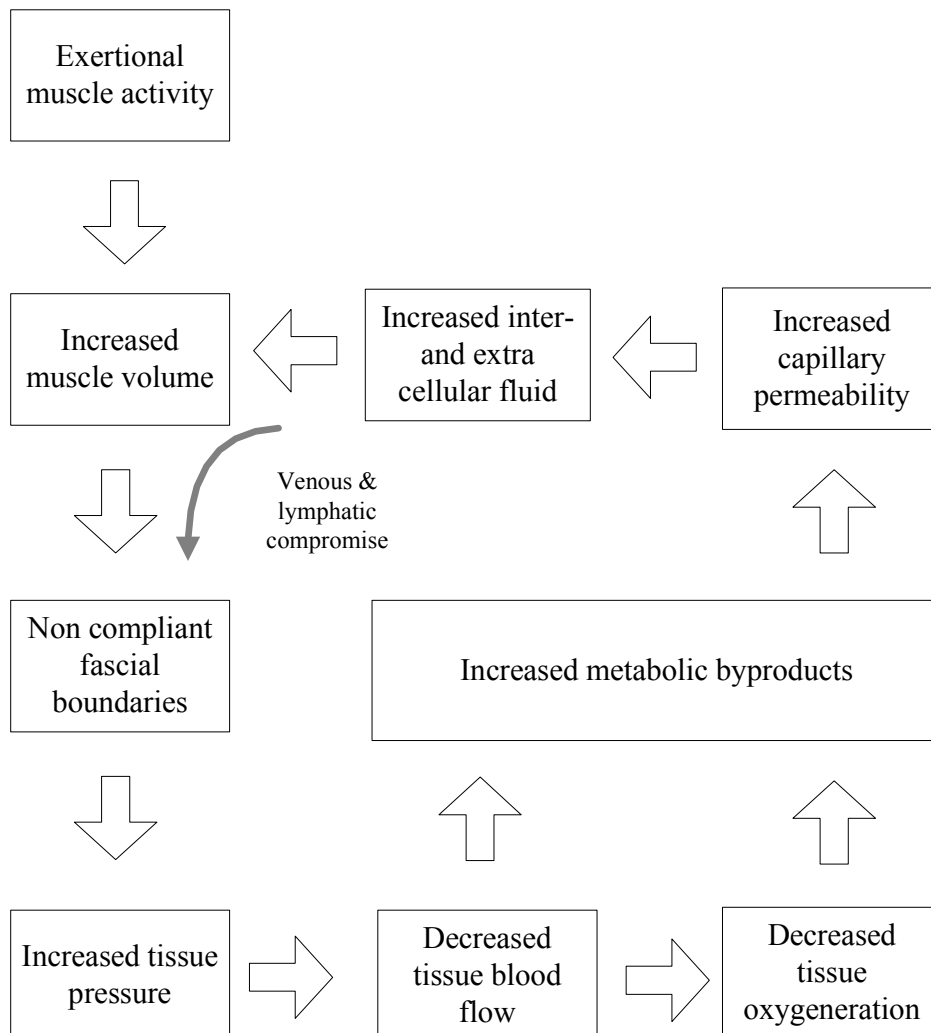
*Note: It is important to keep in mind that these questions are primarily directed towards the researcher as guidance in executing the research.*

**Table 3.2: Case study questions (Yin, 2003)**

<i>Level 1 Questions</i>	<i>Data Source</i>
○ <i>Does the subject qualify for inclusion in the research?</i>	<i>Interview</i>
○ <i>What is the running history of the subject?</i>	<i>Interview</i>
○ <i>Has the subject had any previous injuries?</i>	<i>Interview</i>
○ <i>When were the symptoms experienced for the first time?</i>	<i>Interview</i>
○ <i>Were there any previous treatments for the symptoms?</i>	<i>Interview</i>
<i>Level 2 Questions: Physical examination</i>	<i>Data Source</i>
○ <i>Does the subject have any muscle imbalances?</i>	<i>Isokinetic dynamometer</i>
○ <i>Does the subject have any abnormal movement patterns?</i>	<i>Running gait analysis</i>
○ <i>What is the flexibility/length of the soleus muscle?</i>	<i>Physical measurement</i>
○ <i>Does palpation of the soft tissue in the calf-area reveal any tightness?</i>	<i>Observation</i>
○ <i>What is the impact of the interventions on performance measures?</i>	<i>Observation</i>
○ <i>Why do the measures respond to interventions in the way that they do?</i>	<i>Logical argument</i>

### 3.2.9. Theoretical framework

The current theoretical framework for the pathogenesis of CPCS as reflected in Figure 3.1 was adapted from the model of Clanton & Solcher (1994) which was discussed in detail in Chapter 2. This theoretical perspective of CPCS sees it as a localised condition in the posterior compartment. The non-compliant fascia boundaries play a crucial role in the perpetuation of the condition. As was seen from the literature review, the only marginal successful intervention is the surgical release of the pressure in the posterior compartment. This is achieved through a surgical insertion in the encapsulating fascia. This allows for increased blood-flow to the area, with associated increase in oxygenation.



**Figure 3.1: Pathogenesis of CPCS (Clanton & Solcher, 1994)**

### 3.2.10. Propositions

The mobilization of soft tissue (myofascial tissue) of the lower leg will lead to a disappearance in symptoms of CPCS through:

- *a reduction in the pressure in the calf area which in turn will lead to the alleviation of the symptoms of CPCS; and/or*
- *an increase in tissue blood flow and associated oxygenation that will reverse the process.*

### 3.2.11. Rival theories

The following rival theories can be postulated:

- *The rival proposition is that the conventional treatment of the posterior compartment of the lower leg will not lead to the alleviation of the symptoms of CPCS, as a different theoretical framework is responsible for the pathogenesis of the condition.*
- *The alleviation of the symptoms of the condition is purely due to chance and the intervention has nothing to do with it.*
- *The interventions have not been applied long enough in order to generate the required response.*
- *The researcher is incapable of applying the intervention techniques in an effective manner.*

### 3.2.12. Data collection (Hussey & Hussey, 1997)

The principle data collection methods were:

#### ***Participant Observation:***

The data was collected by the researcher by means of observation of the response of the subjects to the treatments.

#### ***Critical incident technique:***

The researcher focused on interventions with a positive effect on the symptoms of CPCS as reflected in the experimental measures used.



### ***Interviews***

The researcher obtained both qualitative and quantitative information from the participants by means of interviews. Use was made of both a *positivistic* approach by means of structured questions as a *phenomenological approach* with unstructured “open-ended questions” where the subject could give his own opinion with regard to the investigation.

### ***Records***

Where appropriate, use was made of any historical records that the subject may have that had bearing on the condition.

### **3.2.13. Schedule and reviews**

The schedule for the first case study and associated reviews was handled on an ad hoc basis due to the unpredictability of the exploratory nature of the research.

### **3.2.14. Criteria for interpreting results**

The criteria for interpreting results were the degree of conformance to the propositions made from the theoretical framework.

### **3.2.15. Variables and associated measures**

During the exploratory research phase the objective was the search for propositional logic. The identification of the dependant variable, i.e. the symptoms of CPCS, is fairly obvious. With the independent variables, it is however not the case.

The exploratory research process is dominated by the interaction between research question and intervention. The objective is, as stated, to find the causal explanation of the phenomenon under investigation. In this process a proposition is developed from the theoretical framework. The next step is to vary the interventions in order to assess its effect on the dependant variable. If no desired response is obtained, the researcher has to reformulate the theoretical proposition, and redo the iteration. The independent

variables are thus the *research question* and the *treatment intervention*. These variables and associated measures are reflected in Table 3.3.

**Table 3.3: Case study variables and associated measures**

<i>Dependant variable</i>	<i>Measure</i>
<ul style="list-style-type: none"><li>○ <i>The symptoms of CPCS</i></li></ul>	<ul style="list-style-type: none"><li>○ <i>Intensity of pain/discomfort prior to running</i></li><li>○ <i>Intensity of pain/discomfort post running</i></li><li>○ <i>Distance run prior to symptoms</i></li><li>○ <i>Total weekly distance run</i></li><li>○ <i>Palpation findings</i></li></ul>
<i>Independent variable</i>	<i>Measure</i>
<ul style="list-style-type: none"><li>○ <i>Research question</i></li><li>○ <i>Treatment interventions</i></li></ul>	<ul style="list-style-type: none"><li>○ <i>Conformance to the proposition and theoretical framework</i></li><li>○ <i>Credibility of the rival theories</i></li></ul>

### 3.2.16. Research process

#### 3.2.16.1. Subjective assessment - Interview

##### *Running history*

A detailed history of each subject's running career was taken. This included:

- number of years that the subject has been running (running experience);
- his detailed training programme (total kilometres per week, quality training session- and cross training detail);
- race participation (frequency, distance and average speed) as well as
- the type of running shoes used.

The training programme was scanned for:

- sudden increases in either distance run per week or quality work such as speed work or hill training which might have precipitated the injury;



- the stability/motion control ability of the running shoe. This was assessed in order to evaluate the compatibility of shoe alignment with the degree of hind foot pronation; and
- the cumulative distance run with the shoes was also determined in order to assess whether the stability/motion control and shock absorption features were still intact.

### ***Previous running injuries***

All previous running injuries were noted, together with the treatments received and their outcomes. The rationale for recording previous injuries were, where possible, to determine previous fascia-related injuries such as plantar fasciitis, ilio tibial band syndrome and compartment syndrome. It was also noted whether the injury occurred on the left or on the right side in order to determine whether the current injury could be linked to a previous injury.

### ***Symptoms noted***

- Types of symptoms produced by the CPCS;
- Area of symptoms of CPCS;
- Intensity of the symptoms at rest as well as at the end of the training session;
- Duration of the symptoms, once produced; and
- Factors that either eased or aggravated the symptoms.

### ***History of symptoms and any treatment received***

- The history with regard to the symptoms as well as the extent of any medical, surgical and conservative treatments received, and the effect thereof.

### ***3.2.16.2. Objective assessment – Physical examination***

#### ***Muscle strength tests***

Muscle strength tests (plantar- and the dorsi flexors), as determined by means of a calibrated Isokinetic dynamometer, using the same testing protocol for each of the subjects.

### ***Analysis of running gait***

Analysis of running gait (Perry, 1992) as reflected in Table 2.9 was assessed while running at a calibrated speed of 7, 6 kilometres per hour (km/hr) on a treadmill. This was video-taped from posterior as well as from the right side on a digital Sony video-camera. The right side was chosen for convenience since the room space was limited and the treadmill was placed against the left wall of the room. The camera was set up in the centre, three metres posterior to the treadmill as well as in the centre, one and a half metre to the right side of the treadmill. The video-taped information was then transferred via a frame catcher programme onto the computer. Once on the computer, the information was played through IrfanView (version 3, 92), which allowed for the analysis of movement patterns as well as the measurement of specific biomechanical angles such as the degree of hind foot pronation.

### ***Soft tissue***

The soft tissue over the posterior aspect of the lower leg was palpated for muscle spasm, trigger points, swelling and general tissue tension. The palpation was done in a direction from cephalad to caudad on the posterior side of the lower leg. This was done, using the hands and fingers in a general manner over these parts as a circular massage during which a general impression could be gained as to the state of the superficial soft tissues. Thereafter a deeper massage was done, using the tips of the three middle fingers. The purpose was to identify areas of thickness, swelling and tightness in the soft tissue (Maitland, 2006).

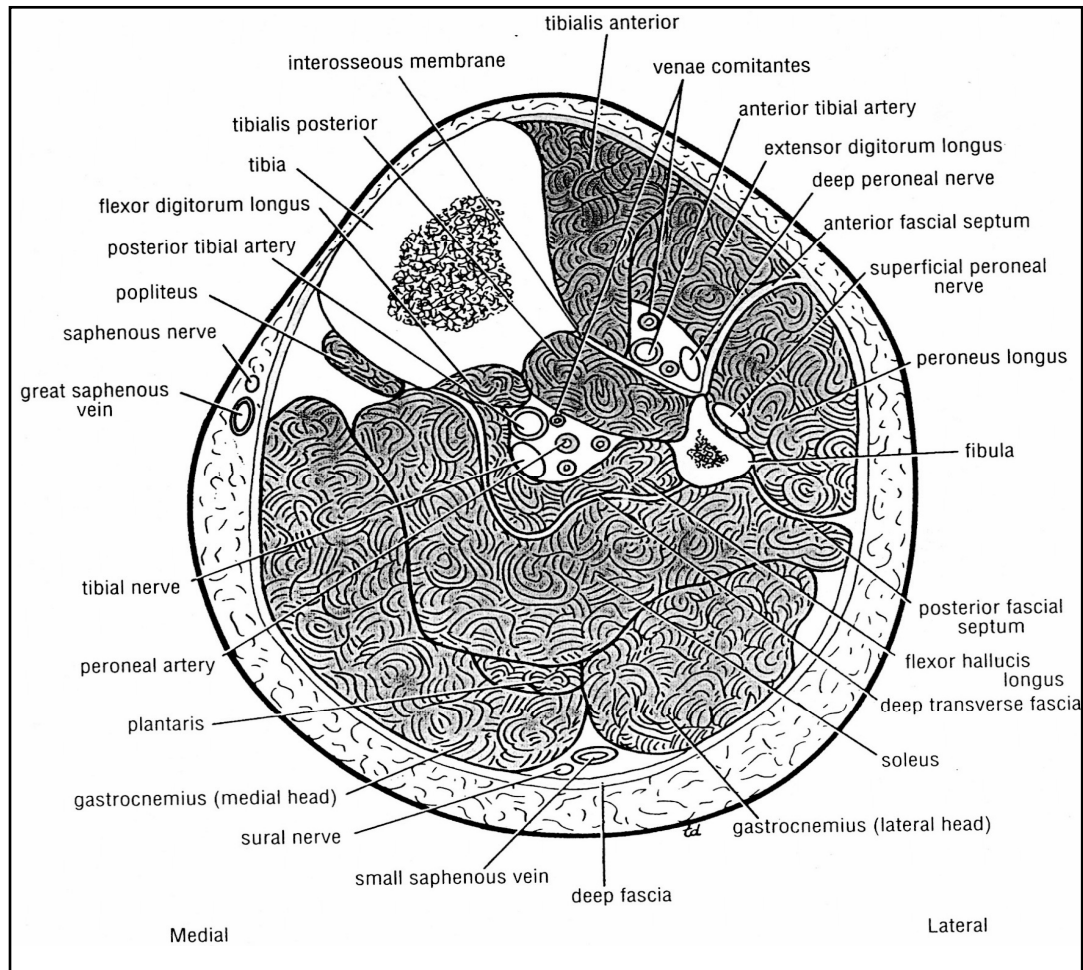
### ***Flexibility of the calf muscle (soleus muscle)***

The soleus muscle was chosen because it is a deeper muscle than the gastrocnemius muscle and is often indicated in the medial tibial syndrome which is often described as a compartment syndrome (Puranen, 1974; Detmer *et al.*, 1986; Touliopolous & Herschman, 1999). The subject was asked to stand flat footed, the maximum possible distance away from the wall, while still able to bend the knee to touch the wall. The distance between the big toe and the wall was measured with a tape measure. An average was then calculated based on three measurements.



### 3.2.17. Intervention

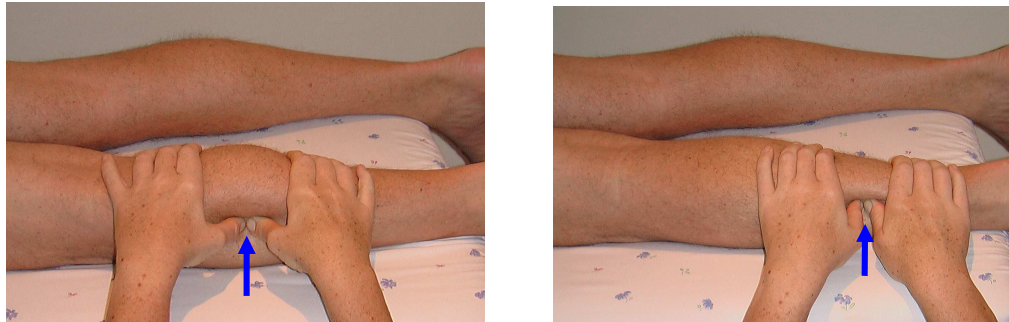
Three specific soft tissue mobilizations were selected which will be discussed in the following paragraphs. In order to provide for a better visualisation of these techniques, Figure 3.2 has been included for reference purposes.



**Figure 3.2: Anatomy of the compartments of the lower leg (Clemente, 1996).**

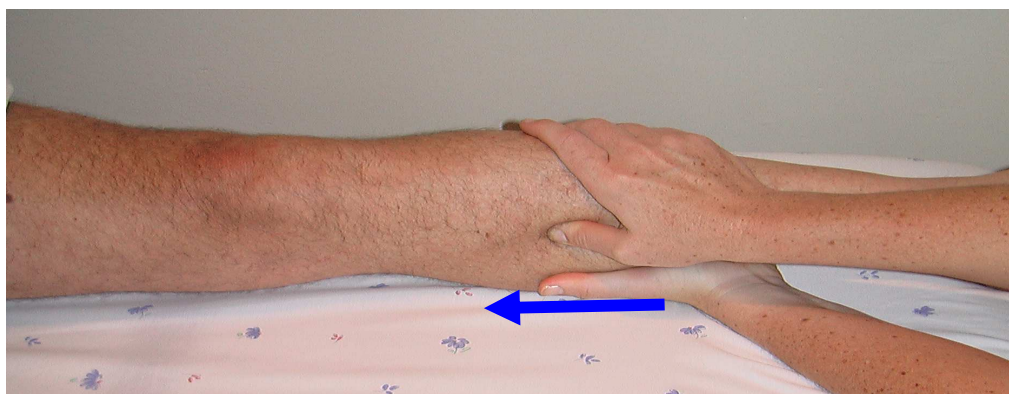
- Soft tissue mobilization techniques (Hunter, 1998), aiming at reducing the pressure in the calf area and improving the blood flow to the area were applied (Figure 3.3) to the posterior intermuscular septum (on the posterior-lateral side of the lower leg- through the gastrocnemius muscle). The posterior intermuscular septum separates the superficial posterior compartment from the lateral compartment. A sustained force, aimed at a 90° angle to the posterior intermuscular septum, with slow oscillations into resistance was used in order to promote hysteresis, creep and plastic deformation of the soft tissue. A force

equivalent to a grade three (Maitland, 1991) was used for 30 seconds at a time. This mobilization technique was started proximally at the posterior-lateral part of the knee and the mobilization was repeated in a distal direction, covering an area of two to three centimetres at a time until the length of the posterior lower leg was covered.



**Figure 3.3: Mobilization of the posterior inter-muscular septum**

- Whilst the subject was lying supine, the deep transverse fascia which lies between the flexor hallucis longus muscle and the soleus muscle was mobilised. The physical therapist faced the subject with her hand resting on the subject's lower leg with her thumb on the side of the subject's tibia where the soleus muscle is palpable next to the tibia. The therapist's lower hand supported and counteracted the movement that wanted to take place by encircling the gastrocnemius muscle from posteriorly (Figure 3.4). The therapist's upper hand glided the soleus muscle in a superior direction at the place where the soleus muscle was palpable. At the end range of the glide, accessory soft tissue mobilization techniques, equal to a grade three, were again applied for 30 seconds.



**Figure 3.4: Soft tissue mobilization of the deep transverse fascia**

- Thereafter the gastrocnemius muscle was glided over a fixated soleus muscle in order to mobilize the fascia of the leg that lies between these two muscles. With the subject lying prone, the researcher's underneath hand fixated the soleus muscle from anteriorly while the upper hand of the researcher rested on the posterior aspect of the gastrocnemius muscle and glided the gastrocnemius muscle superiorly over the fixated soleus muscle (Figure 3.5). At the end range of the glide, accessory soft tissue mobilization techniques, equal to a grade three were again applied for 30 seconds. The posterior lower leg was divided into the upper, middle and lower parts and the glide was applied to each.



**Figure 3.5: Mobilization between the soleus- and the gastrocnemius muscles**

Thereafter the gastrocnemius muscle was glided over a fixated soleus muscle in order to mobilize the fascia of the leg that lies between these two muscles. The intervention was followed with daily stretches of the gastrocnemius- and the soleus muscles. The stretches were taken to the point where a mild tension was felt and the stretch was held for 30 seconds (Hunter, 1998). Stretches were done bilaterally and each stretch was repeated twice.

The following stretches were included:

- *Gastrocnemius muscle (Travell & Simons, 1999):*
  - Wall standing;
  - Point both feet forward with legs apart;



- Keep back knee straight;
- Push hips forward;
- Press heel to ground, keeping it flat;
- Hold the stretch; and
- Repeat stretch on each leg.

➤ *Soleus (Travell & Simons, 1999):*

- Wall standing;
- Legs apart and both feet pointing forward;
- Lower the hips and bend knee of back leg;
- Push back heel flat to ground;
- Hold the stretch; and
- Repeat stretch on each side.

The intervention was scheduled for once a week. End of range treatment grades were used (grades three) and more time was needed in between treatment sessions to allow the soft tissue to recover from treatment tenderness/soreness. Since the condition treated was chronic, a treatment period of three months was decided on (this totalled approximately 12 treatment sessions).

### **3.2.18. Data recording**

The following data were recorded during the treatment interventions:

- The intensity of pain / discomfort at rest (if present) as well as the intensity of pain/discomfort at the end of every training session was plotted on a 100 mm visual analogue scale (VAS);
- The distance run (measured in kilometres) before the commencement of the symptoms was noted at every training session;
- The total weekly distance run (measured in kilometres) was noted for the duration of the study;
- Muscle strength of the dorsi- and plantar flexors of the ankle were tested on the Isokinetic dynamometer before commencement of the intervention;



- Running gait and movement patterns were assessed, as described above, before the intervention as well as three months thereafter;
- Flexibility of the calf muscles (soleus muscle) was assessed, as described above, before and three months after the intervention; and
- The tightness on palpation of the soft tissue of the posterior lower leg was assessed before and after every treatment session.

### 3.2.19. Quality assurance measures

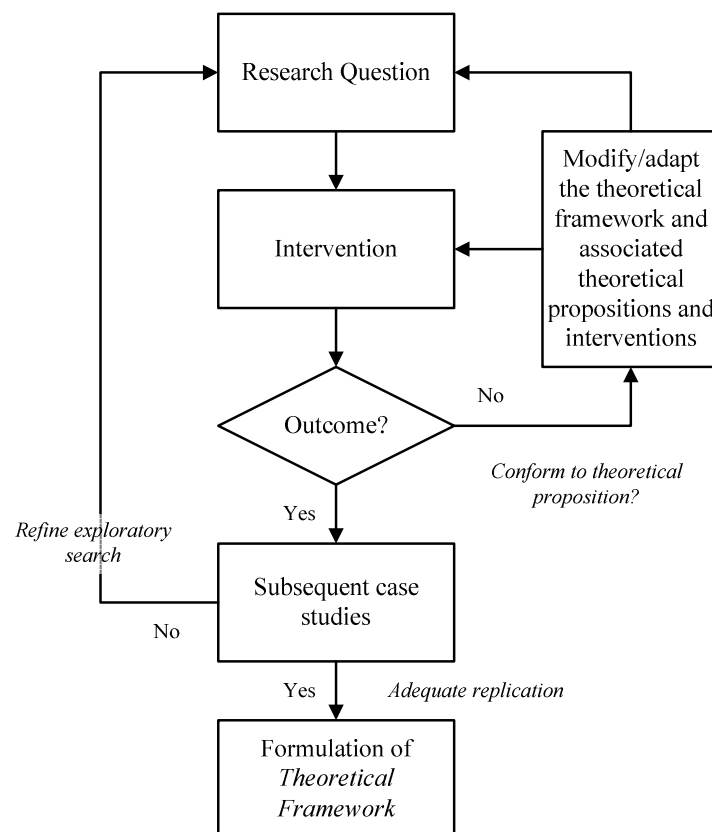
With regard to ensuring the quality of the research, the measures reflected in Table 3.4 was used to ensure the quality of the research design. The reviews of the case study designs were however not limited to peer-reviews. The designs were also reviewed by members of the running club as well as peer physiotherapists who provided a multi-disciplinary perspective. The members included physicists and an engineer. The same approach was followed with draft case study report reviews.

**Table 3.4: Quality assurance measures (Olivier, 2004; Yin, 2003)**

<i>Test</i>	<i>Case Study Measures</i>	<i>Research Phase</i>
<i>Construct Validity</i>	<i>Peer review of the design</i>	<i>Research design</i>
	<i>Use multiple sources of evidence</i>	<i>Data collection</i>
	<i>Establish a chain of evidence</i>	<i>Data collection</i>
	<i>Key informant reviewing draft case study report</i>	<i>Composition</i>
<i>Internal Validity</i>	<i>Pattern matching</i>	<i>Data analysis</i>
	<i>Explanation building</i>	<i>Data analysis</i>
	<i>Addressing rival explanations</i>	<i>Data analysis</i>
	<i>(Arguments based on ) logic models</i>	<i>Data analysis</i>
<i>External Validity</i>	<i>Use of theory</i>	<i>Research design</i>
	<i>Use of replication logic</i>	<i>Research design</i>
<i>Reliability</i>	<i>Use of case study protocol</i>	<i>Research design</i>
	<i>Develop a case study database</i>	<i>Data collection</i>

### 3.2.20. Modification of the exploratory research design

The nature of the exploratory process implies that the research design be adapted progressively in line with the research outcomes. The process that has been followed is depicted in Figure 3.6. In terms of this process the outcomes of the interventions were progressively compared to the expected outcomes as reflected by the propositions that were deduced from the then ruling theoretical framework. In the event of a deviation from this expectation, the first action was to change the intervention that was applied to the subject. If the options with regard to the available treatment interventions were depleted, the researcher was forced to conclude that the proposition did not hold true. In other words one of the rival theories or a derivative thereof applied to the case study under review.



**Figure 3.6: Modification of exploratory designs**



The next step was thus to adapt or modify the theoretical framework and to postulate a new proposition and rival theories. This iterative approach was followed till convergence of the outputs with the expectations was reached. This implied that some degree of credibility existed in terms of the postulated theoretical framework. In such an event, the researcher proceeded with the subsequent case studies with research objectives aligned with the indications from the preceding case.

### **3.2.21. Conclusion**

In this section the basic design for the exploratory phase of the research project was presented. This design is based on the findings of the literature research on the subject contained in Chapter 2. It also contained the rationale for as well as the process for the adaptation of this design throughout the exploratory design phase.

In the following section, the research design for the explanatory phase of the research will be covered.

## **3.3. THE EXPLANATORY RESEARCH DESIGN**

The research design framework developed in Chapter 2 as applied to the design of the exploratory research will again be used as the foundation for the research design covered in this section. Elements of the design which are common to the exploratory research design are not repeated in this section. This includes elements such as the database, ethical considerations, and data recording.

Although the objective of pressure reduction in the posterior compartment and the normalisation of the blood micro circulation remained the same, the focus of the explanatory research phase shifted to the validation of the concept that this could be achieved through interventions aimed at the so called “*clinically significant muscles*”.

### 3.3.1. The research question

*The research question is whether the revised theoretical model developed as a result of the exploratory research is adequate to explain the phenomenon of the pathogenesis of CPCS; and whether it can be used as a general framework for the development of conservative treatment interventions for the condition.*

The main proposition of this explanatory phase of the research is that the root cause of CPCS lies outside the posterior compartment and this manifests through tightness in the clinically significant muscles. The mobilization of these tight “*clinical significant muscles*” will lead to a disappearance in symptoms of CPCS through:

- a reduction in the pressure in the calf area which will lead to the alleviation of the symptoms of CPCS through:
  - the normalisation of the length of the myofascial chain;
  - which in turn will lead to a reduction in the stresses exerted in the chain;
  - which will lead to a reduction in the radial stresses induced on the calf-area;
  - which will reduce the pressure in the posterior compartment; and
- the normalisation of the blood micro circulation and associated oxygenation which will enable final healing.

### 3.3.2. Theoretical framework

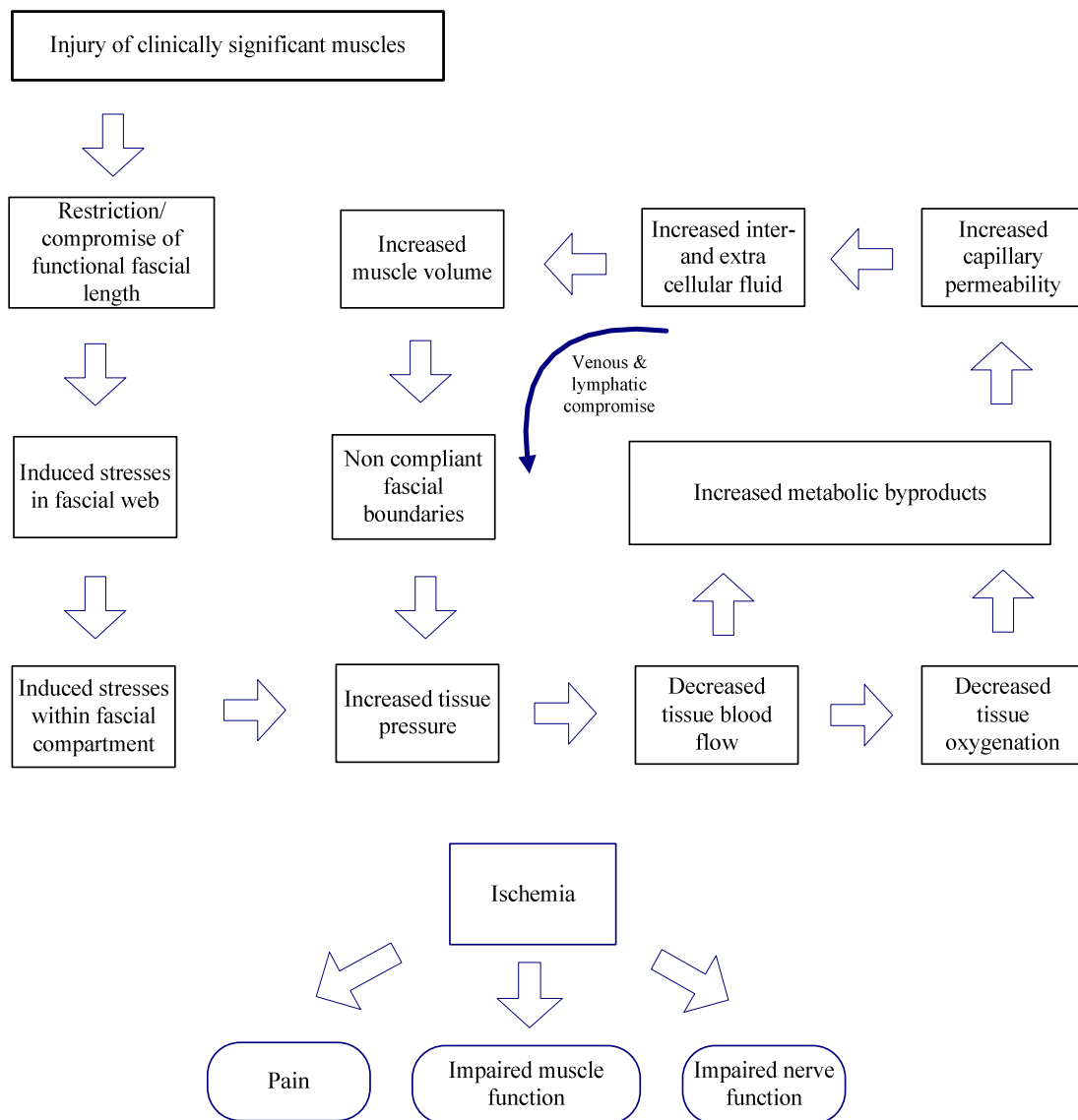
The revised theoretical model which was developed based on the research findings of the exploratory research phase is reflected in Figure 3.7 for the ease of reference.

### 3.3.3. Propositions

The mobilization of soft tissue (myofascial tissue) of the lower leg will lead to a disappearance in symptoms of CPCS through:

- a reduction in the pressure in posterior compartment which in turn will lead to the alleviation of the symptoms of CPCS; and/or
- an increase in tissue blood flow and associated oxygenation which will reverse process.





**Figure 3.7: Modified model for the pathogenesis of CPCS (adapted from the model of Clanton & Solcher (1994))**

### 3.3.4. Rival theories

The following rival theories can be postulated:

- *The rival proposition is that the release of the tightness in the clinically significant muscles will not lead to the alleviation of the symptoms of CPCS, as a different theoretical framework is responsible for the pathogenesis of the condition.*



- *The alleviation of the symptoms of the condition is purely due to chance and the intervention has nothing to do with it.*
- *The interventions have not been applied long enough in order to generate the required response.*
- *The researcher is incapable of applying the intervention techniques in an effective manner.*

### **3.3.5. The Research Process**

In terms of the process the same process as followed during the exploratory phase will be used. The focus is however now on the application of the techniques and approaches developed based on the propositional logic as developed during the exploratory phase. The process in terms of the subjective assessment by means of interviews and the objective assessment through physical examination are the same.

#### ***Interventions***

The following intervention techniques will be used to release the tightness in the clinically significant muscles:

- Trigger point releases;
- Myofascial releases;
- Specific mobilizations; and
- Interventions followed up by stretches.

### **3.3.6. Conclusion**

In this section the basic design for the explanatory phase of the research project was presented. The actual case study research results which flowed from both the exploratory and the explanatory research will be presented in Chapter 4.

In the next section the approach and rationale for the supplementary experimental research will be presented.



## **3.4. EXPERIMENTAL RESEARCH**

### **3.4.1. Introduction**

This section covers the experimental design for the research on the effects of interventions on the extension at the first metatarsophalangeal joint as well as on hind foot pronation. As mentioned in Chapter 2, the FDL and the FHL muscles are important in preventing extreme plantar to dorsi flexion movement at the metatarsophalangeal joints when the foot is in contact with the ground. This implies that measurements of the metatarsophalangeal joints might provide further important information with regard to the running gait and muscles involved.

### **3.4.2. Degree of extension at the metatarsophalangeal joint during terminal stance**

During the assessment of the video-clips of the first three subjects it was noted that these subjects appeared to rise abnormally high onto their forefeet. It was decided to investigate the phenomena. Two injury free runners were selected as a basis for comparison. Their extensions at the first metatarsophalangeal joints during the terminal phase were video-taped and compared with that of the subjects with symptoms of CPCS.

#### ***3.4.2.1. Aim of the study***

The primary aim of this pilot study was to determine whether the subjects with symptoms of CPCS rose higher onto their forefeet during the terminal phase of their running gait in comparison to normal runners without symptoms.

#### ***3.4.2.2. Hypothesis***

Runners with symptoms of CPCS rise higher onto their forefeet during the terminal phase of the running gait than injury-free runners.

### 3.4.2.3. *Research design*

The dimensions and classification of the experiment are reflected in Table 3.5.

**Table 3.5: Dimensions and classifications of the experiment**

<b>Dimension</b>	<b>Classification</b>
<b>Design</b>	Holistic multiple case, single unit (Type 3)
<b>Motivation</b>	Instrumental
<b>Epistemological status</b>	Exploratory followed by explanatory
<b>Purpose</b>	Research
<b>Data</b>	Quantitative
<b>Format</b>	Structured
<b>Synthesis</b>	Formative or method driven

### 3.4.2.4. *Sample selection*

Six subjects with symptoms of CPCS recruited for the research programme were included, as well as two controls from a regular morning training group. This pilot study was conducted during the therapeutic intervention applied to subjects one and two; and before the therapeutic intervention applied to subjects three to six.

### 3.4.2.5. *Inclusion and exclusion criteria for the controls*

#### *Inclusion criteria of the controls*

- Age: 18 – 50 years of age
- Runners participating in races with a distance of 10 km or further
- Male or female
- Had to be running at the time of the pilot study
- No injuries at the time of the study. Had to be running symptom free for at least the previous 12 months.



#### *Exclusion criteria of the controls*

- Any injuries during the last 12 months.
- Not running at the time of the pilot study.
- Not currently participating in road races.

#### **3.4.2.6. Inclusion and exclusion criteria for the subjects**

The inclusion and exclusion criteria for the runners with CPCS were covered in detail in Chapter 4.

#### **3.4.2.7. Outcome measures**

The outcome measure was the range of extension at the first metatarsophalangeal joint, measured in degrees during terminal stance of the running cycle.

#### **3.4.2.8. Procedure**

The degree of extension at the 1<sup>st</sup> metatarsophalangeal joint will be measured as follows:

- Markings will be made on the medial side of both feet. Dots will be made over the head of the first metatarsophalangeal joint and at the central-inferior border of the medial malleolus.
- Thereafter, the subjects will run barefoot on a treadmill with a calibrated running speed of 7.6 km/h.
- The subject's lower legs will be video-taped with a Sony digital video-camera whilst running.
- This video material will be copied to a specific software programme (TV 2000 combined with Corel Draw version 11) which will be used to measure the degree of extension at the first metatarsophalangeal joints.
- The dots over the centre of the inferior border of the medial malleolus and the head of the first metatarsophalangeal joint must be connected with a straight line using Corel Draw.



- A straight connecting line will also be drawn with Corel Draw between the head of the first metatarsophalangeal joint and the tip of the first toe.
- Every measurement must be taken three times at different intervals of the terminal stance of the running gait and the average of the three measurements must be calculated.

### **3.4.3. The effect of soft tissue mobilization on subtalar over pronation in sportsmen**

#### **3.4.3.1. Introduction**

During the treatment of the subjects in case studies 4 to 6, it was noted that a release of tightness in the '*clinical significant muscles*' reduced the degree of hind foot pronation in all three runners. These observations were supportive of the revised or new theoretical model for the pathogenesis of CPCS that was beginning to take shape. In terms this framework one can argue that the stresses that are induced in the fascial web as a result of restricted fascial movement in the clinical significant muscles would also have an effect on the alignment of the feet with the rest of the body. It was thus decided to investigate the effect of soft tissue mobilization of the clinically significant muscles in subjects with abnormal pronation. Abnormal hind foot pronation as was seen in Chapter 2 is defined as in excess of 12° (Hoppenfield, 2000).

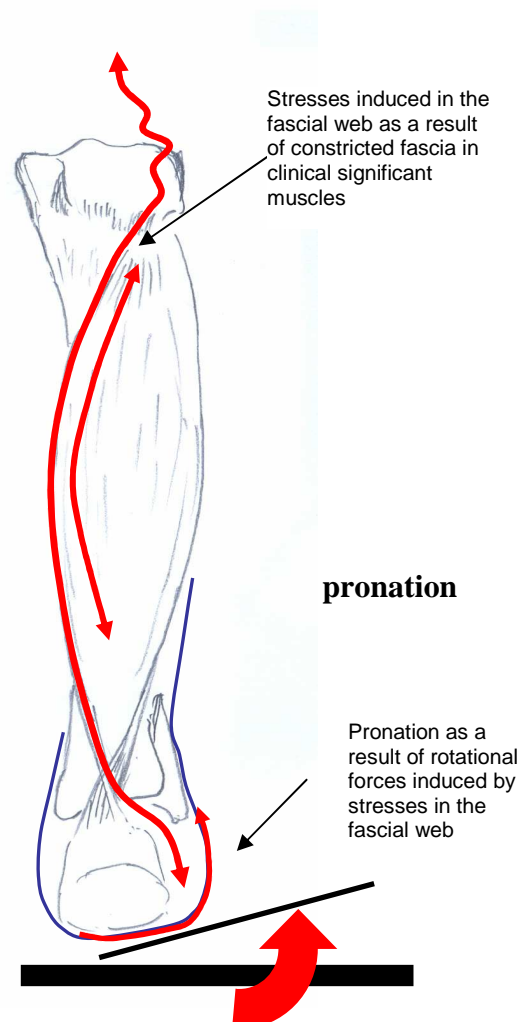
#### **3.4.3.2. Aim of the study**

The aim of the study was three-fold. The objectives were to:

- establish whether subjects with CPCS symptoms had excessive hind foot pronation (if the stresses in the fascial web were sufficient for the pathogenesis of the condition, it should also reflect in biomechanical abnormalities);
- establish whether the release of the tightness in the clinical significant muscles would normalise such biomechanical abnormalities (if these were indeed the effect of tightness, one could argue that the elimination of the root cause would lead to the normalisation of the phenomena); and
- establish whether these intervention techniques could be successfully applied by less skilled physiotherapists.

### 3.4.3.3. Hypothesis

The hypothesis was that abnormal pronation is as a result of tightness in the clinical significant muscles of the subjects which distorts the fascial web which in turn induces stresses in the fascial structures that leads to abnormal pronation. Such abnormal pronation could be normalized by the soft tissue mobilization techniques. It was argued that the tightness in one or more of the ‘clinical significant’ muscles compromise the effective length of the myofascial chain which in turn induces stresses along the fascial web which in turn affects the alignment of the foot and movement patterns which increase the degree of hind foot pronation. This argument is graphically reflected in Figure 3.8.



**Figure 3.8: Fascial stress induced pronation**



#### **3.4.3.4. *Experimental design***

The design for the experiment involving hind foot pronation can be described as follows:

##### ***Single group design***

The subjects were subjected to observation of their behaviour with the *unmodified* system, which in this case represents the degree of pronation prior to intervention, and then subsequently after the application of the intervention. The subject thus becomes its *own control*. In the broader context the results from the literature provide an additional control.

##### ***Blind experiment (application by others)***

The experiment was conducted by five research assistants. These were all final year physiotherapy students at the Pretoria University.

#### **3.4.3.5. *Subjects***

Fifteen subjects consisting of seven males and eight females, between the ages of 18 and 45 years complied with the inclusion criteria. To be included into the study, the subjects had to participate actively in a sport or activity that involved a minimum of one hour's training three times a week, live in the Pretoria area and with a hind foot pronation angle of more than 12°. The subjects were requested to continue their participation in the sport or activity whilst participating in the study.

During the interview, it was noted that ten of the subjects experienced occasional symptoms at the time of inclusion into the study but none of them had requested any form of treatment for their symptoms until then. Five subjects complained of medial shin pain, four subjects complained of lateral thigh pain and one subject complained of plantar foot pain. These symptoms were merely noted and did not influence the application of the intervention in any way.



After being briefed on the procedures, all subjects agreed to participate in the study. Subjects who had undergone surgery of the lower extremities within the previous six months, as well as subjects who had had any connective tissue disease, were excluded from the study. During the intervention period, two male subjects dropped out of the study due to work obligations. A third subject's (female) final measurements of hind foot pronation angles were lost and her results were therefore not included. Ethical clearance was obtained from the relevant ethics committee at the University of Pretoria, and informed consent was signed by all the participants.

#### **3.4.3.6. Procedure**

The degree of hind foot pronation in each subject was measured by the same podiatrist. It was measured by marking the reference landmarks in the frontal plane bisection of the posterior calcaneus, as it relates to the frontal plane bisection of the posterior aspects of the distal one-third of the lower leg (Hunt, 1998).



**Figure 3.9: Measurement by means of Corel Draw software**

Dots representing the axis of motion, as well as distal points of the bisections, were drawn with a pen. Skin lines connecting the dots were drawn (Gould & Davies, 1985). Thereafter, the subjects ran barefoot on a treadmill with a calibrated running

speed of 7.6 km/h. The subjects' lower legs were videotaped with a Sony digital video camera while running. This video material was copied to a specific software program (TV 2000 combined with Corel Draw version 11) which was used in turn to measure the degree of hind foot pronation. Figure 3.9 provides an example of a video frame that has been frozen and the angle measured with the Corel Draw software program.

The 'clinical significant' muscles were assessed in all the subjects prior to and after every intervention session to determine whether any tightness existed in the muscles. All the tight 'clinical significant' muscles were subsequently released by means of myofascial release techniques (Manheim & Lavett, 1989), trigger point release therapy (Travell & Simons, 1999) or specific soft tissue mobilization techniques (Hunter, 1998). For the sake of standardization, the main researcher and author demonstrated the assessment and intervention techniques to the five research assistants (fourth year physiotherapy students). The 15 subjects were divided into five groups and each of the five research assistants was responsible for the assessment and treatment of three subjects. The treatment sessions were scheduled once weekly for each of the subjects and were conducted over eight weeks. A treatment session lasted for 45 to 60 minutes, depending on the number of tight 'clinical significant' muscles. The weekly assessment and treatment sessions of all five research assistants were supervised by the main researcher. Each patient was assessed and treated by the same researcher throughout the intervention period. Each subject received the same home muscle stretching programme (mm. trapezius mid fibres, levator scapula, pectoralis major, rectus abdominus, iliopsoas, piriformis, hamstring, soleus and gastrocnemius) in order to maintain the soft tissue mobilization releases that had been achieved during the treatment session. The subjects' hind foot pronation angles were re-assessed directly after the eight-week intervention period, in the same manner as with the initial assessment.

#### ***3.4.3.7. Data management and analysis***

In terms of the experimentation the independent variable was the application of the soft tissue mobilization techniques with the dependant variable being the degree of hind foot pronation.



A significant improvement in the dependent variable was defined as a change in the degree of hind foot pronation to such an extent that the subjects' measurements would fall within the range considered to be normal for the broad population.

In order to establish a confidence interval for normal pronation, it is necessary to know what the standard deviation for the pronation distribution would be. Based on the work of Hurlburt (1993) one can estimate the standard deviation based on information available on the range of observations. He has developed conversion tables with conversion factors that could be used for this purpose. The range is then divided by the conversion factor which then provides an estimate for the standard deviation.



## **CHAPTER 4**

### **RESEARCH RESULTS**

#### **4.1. INTRODUCTION**

This chapter deals with the results of the all the different phases of the research covered by the thesis. In the first section the results of the exploratory phase is covered. The section is followed by a section that deals with the new theoretical framework which has been developed as a result of the exploratory research. This new theoretical model plays a cardinal role in the research which follows.

In the third section the results of the explanatory research phase is covered. This section is followed by a section that deals with the experimental research which provides new perspectives on the new theoretical model which has been developed.

#### **4.2. EXPLORATORY RESEARCH PHASE**

##### **4.2.1. Introduction to the exploratory research**

The following section deals with the research results of the exploratory phase of the research. It covers the results of the first three individual case studies. The level of understanding is gradually increased during the research process which leads to a progressive refinement of theoretical propositions.

##### **4.2.2. CASE STUDY 1**

###### **4.2.2.1. Introduction**

Case Study 1 forms part of a group of three case studies that deal with the exploratory research phase of this project. The case study consists of four distinct phases.

The first two phases consisted of exploratory research based on the current theoretical understanding of the condition. In phase 1 the interventions were based on

conventional physiotherapy. During phase 2 this was supplemented by calf muscle strengthening and proprioceptive retraining.

In phases three and four the interventions were expanded to include soft tissue mobilization in the lower leg which in turn was expanded to include the more proximal soft tissue.

Although the research started out as exploratory, as the knowledge and insight gained expanded, the case studies eventually provided the basis for the explanation of the new theoretical perspectives developed during the research.

#### ***4.2.2.2. Subject***

The subject in Case Study 1 was a 36 year old female athlete who had been participating in road running for the previous three and a half years. At the time of her first consultation (24/01/02), she participated mainly in ten kilometre races and averaged a running pace of six and a half minutes per kilometre.

#### ***Inclusion criteria***

During December 2001, she was clinically diagnosed with CPCS by a general practitioner who specializes in sport injuries. She complained of pain over the posterior-medial aspects of both lower legs whilst running. This pain usually occurred within the first kilometre of running, gradually grew worse as the run continued and abated with rest.

#### ***Exclusion criteria***

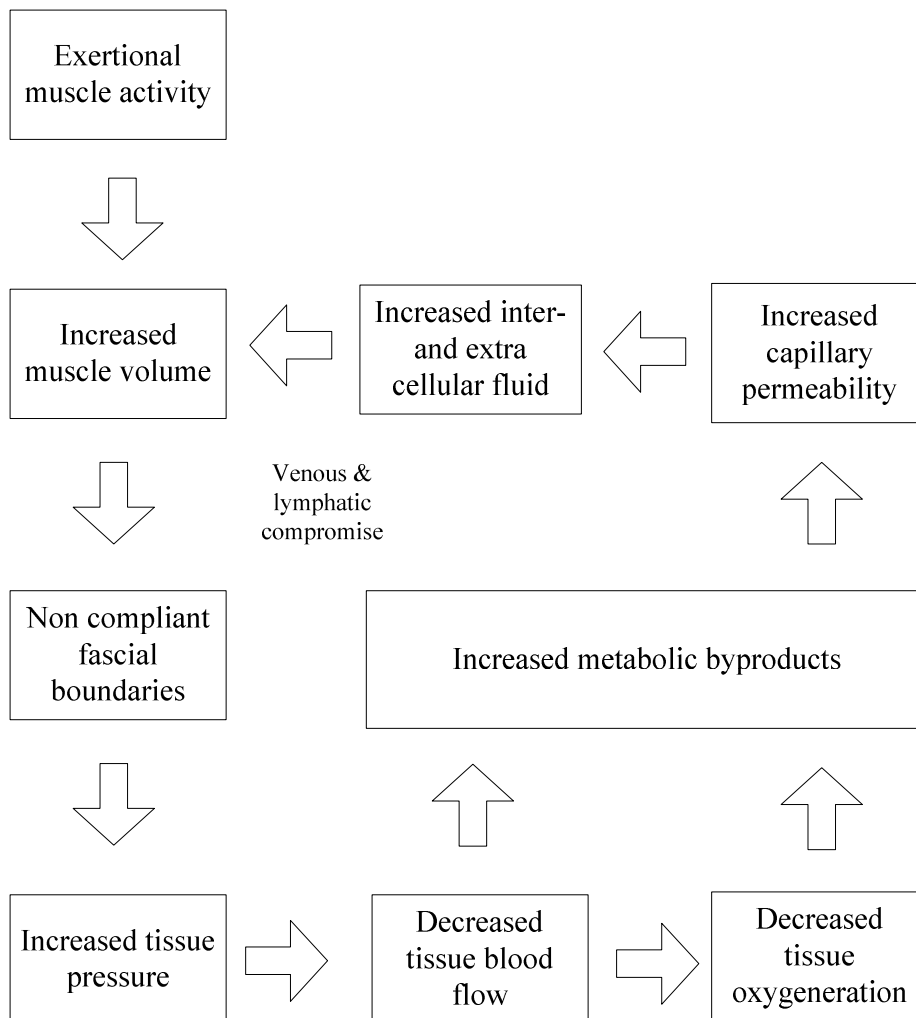
With regard to the exclusion of nerve entrapments, it was noted that the plantar aspect of her feet often turned numb during a run (which could possibly be an indication of nerve entrapment), affecting the placing of her feet, but there was no tingling or burning sensation. She also did not experience any pain behind the knee.

#### ***4.2.2.3. The research question***

*“Does the existing theoretical model for the pathogenesis of CPCS provide for a logical model for describing causal relationships that could be used as the basis for the development of a conservative treatment approach for the condition?”*

#### 4.2.2.4. Theoretical framework

The model of Clanton & Solcher (1994) which has been discussed in Chapter 2 (Figure 2.3, page 55) is reproduced below with minor cosmetic changes. In terms of the model, CPCS is due to the increased muscle volume and the non-compliant fascial boundaries, which in turn leads to a decrease in tissue blood flow. The initial premise was that the blood-flow to the posterior compartment could be restored through conventional physiotherapy. Such conventional physiotherapy interventions would include massages, cross-friction techniques, ultrasound and interference therapy, and stretches that would lead to a reduction in the pressure in the calf area.



**Figure 4.1: Pathogenesis of CPCS (Clanton & Solcher, 1994)**



#### **4.2.2.5. Propositions**

*The conventional treatment of the posterior compartment of the lower leg will lead to a disappearance in symptoms of CPCS through:*

- *reduction in the pressure in calf area which in turn will lead to the alleviation of the symptoms of CPCS; and/or*
- *an increase in tissue blood flow and associated oxygenation that will reverse the process.*

#### **4.2.2.6. Rival theories**

The following rival theories can be postulated:

- *The rival proposition is that the conventional treatment of the posterior compartment of the lower leg will not lead to the alleviation of the symptoms of CPCS, as a different theoretical framework is responsible for the pathogenesis of the condition.*
- *The alleviation of the symptoms of the condition is purely due to chance and the intervention has nothing to do with it.*
- *The interventions have not been applied long enough in order to generate the required response.*
- *The researcher is incapable of applying the intervention techniques in an effective manner.*

#### **4.2.2.7. Schedule and reviews**

The schedule for the first case study and associated reviews was handled on an ad hoc basis due to the unpredictability of the exploratory nature of the research.

#### **4.2.2.8. Criteria for interpreting results**

The criteria for interpreting results were the degree of conformance to the propositions made from the theoretical framework.



#### 4.2.2.9. *The Research Procedure*

##### *Subjective assessment - Interview*

###### ○ *Running history*

The subject in Case Study 1 was a female athlete who had been participating in road running for the previous three and a half years. She participated mainly in ten kilometre races and averaged a running pace of six and a half minutes per kilometre. Her training programme consisted of a very gradual build up. She initially started by running for one minute, followed by walking for one minute. The running time was gradually increased over time to a stage where the total training distance reached a distance of five kilometres, where after the walking time was gradually reduced. This training programme was repeated three times a week.

She started off running with a slight anti-pronation shoe, but changed to a neutral shoe after consulting a podiatrist during February 2001. She had been paying careful attention to the number of kilometres she ran with the shoes and had to date never run more than 800 kilometres with a pair of shoes.

###### ○ *Previous running injuries*

No other injuries apart from the calf muscle pain had been experienced.

###### ○ *Symptoms noted*

Her first consultation with the researcher was on the 24th of January 2002. She complained of pain over the posterior-medial aspects of both lower legs with a pain rating of 20 out of 100 on a 100 mm VAS. This pain usually occurred within the first kilometre of running. The plantar aspect of both feet turned numb, during most of her runs (at least 70% of the runs) within the first four kilometres of the run. This made running very difficult, as she was not always sure of the placing of her feet.





○ *History of symptoms and treatment received*

The subject first experienced the above mentioned symptoms towards the end of December 1998. During February 2001, she consulted a biokineticist who, in turn, recommended her to consult a podiatrist.

The podiatrist measured the degree of hind foot pronation as eight degrees in the right foot and six degrees in the left foot, after which he advised her to change her running shoes from a slight anti-pronation shoe to a neutral shoe. She also underwent six physiotherapy sessions consisting of massage of the calf muscles, stretch exercises for the calf muscles, ultrasound and interferential therapy over the indicated symptomatic calf muscles. She continued running, but despite the physiotherapy, her symptoms gradually became worse.

During September 2001, she consulted a general practitioner who specializes in sport injuries. The practitioner determined, by means of a Isokinetic dynamometer test, that both her plantar flexor muscles and her dorsi flexor muscles were weak and felt that these weaknesses were the cause of her symptoms. She underwent eight supervised strengthening sessions of the plantar- and dorsi flexor muscles on the Isokinetic dynamometer. Through all of this she continued to run, yet her symptoms progressively deteriorated to such an extent that towards the end of January 2002, she was unable to walk a distance of 100 metres. At this stage she presented at the researcher's practice.

In summary, the subjective outcome measures as measured before the intervention:

- The intensity of pain / discomfort was 20 on the 100 mm VAS after a one kilometre run.
- The pain/ discomfort started within the first kilometre of running.
- She averaged a weekly distance of 12 kilometres per week at a pace of six and a half minutes per kilometre (she did not stop running the moment she felt the pain, which was within the first 100 metres. She continued as far as she could tolerate the pain).



### ***Objective assessment – Physical examination***

#### ○ ***Muscle strength tests***

The muscle strength of the plantar- and the dorsi flexor muscles was not retested, since this has been tested on a Isokinetic dynamometer by a biokineticist four months prior to the researcher seeing the subject. These measurements were accepted for baseline purposes. According to the referring practitioner, the strength of these two muscle groups was significantly lower compared to the norm at the time of testing.

#### ○ ***Analysis of running gait, including movement patterns***

The following movement patterns deviated from the normal/ ideal (Table 4.1):

- Throughout the running phases, the pelvis on the right remained slightly more posteriorly rotated in comparison to the left. The pelvis on the right also never seemed to move into a position of anterior rotation as one would normally expect during the initial contact phase.
- During the terminal stance and pre-swing phases, the right hip moved into more lateral rotation and extension compared to the left. Occasionally on every fourth or fifth stride, there was an additional lateral rotation and extension of the hip on the right, followed by an increased amount of medial rotation on the forward movement of the right hip in an attempt to counteract this.
- There was no heel toe action and during the mid stance phase, the calcaneus sunk into more of a valgus position than normal. This also appeared to be more on the right side than on the left. During the pre-swing phase the calcaneus on both sides moved into more inversion as though there was a slight weight shift from the second metatarsal head to the third metatarsal head. During the initial swing phase, there was hardly any push-off to be noticed on both sides.
- It was noticed from the posterior that the tendon Achilles was slightly curved convexly to the medial side in both legs.

From the description of the running movement patterns and the results tabled in Table 4.1, it is clear that abnormalities and asymmetries were present in the running movement patterns, compared to the norm.

**Table 4.1: Outcomes of the running gait analysis for Case Study 1 prior to intervention**

<i>Running gait analysis: Case Study 1 (24/01/2002)</i>								
	<i>Weight Acceptance</i>		<i>Single Leg Support</i>			<i>Swing Leg Advancement</i>		
<i>Joint</i>	<i>Initial Contact</i>	<i>Loading Response</i>	<i>Mid-stance</i>	<i>Terminal Stance</i>	<i>Pre-swing</i>	<i>Initial Swing</i>	<i>Mid-swing</i>	<i>Terminal Swing</i>
<i>Pelvis</i>	On the left side, the pelvis is in anterior rotation. On the right, the pelvis is in a neutral position.		Both sides are in slight posterior rotation. The right side more so than the left side.	Both sides are in posterior rotation; right side more than left side. Vertical movement of the pelvis on the right side is greater in comparison to the left.	Both sides are in posterior rotation; right side more than left side.		Both sides move out of posterior rotation.	The right side moves into a neutral pelvic position and the left side moves into anterior pelvic rotation.
<i>Hip</i>				The right hip is in more lateral rotation and extension compared to the left side.	The right hip is in more lateral rotation and extension compared to the left side.	Both hips start to move into internal rotation from the externally rotated position. Every now and then there is an irregularity on the right side with an increased amount of right hip external rotation and posterior pelvic rotation on the right.	Both hips are in flexion and internal rotation. Increased medial rotation in on the right side, following the occasional irregularity described in the previous column.	Both hips are in flexion.
<i>Knee</i>	Both knees: almost full extension with the tibia in external rotation.	The amount of knee extension decreases slightly in both knees.	The amount of extension in both knees increase slightly again with the tibia being in external rotation.	The tibias move into more external rotation with the knees in extension.	Both knees are extended with some external rotation of the tibias.	Both knees move out of extension and external rotation into flexion.	Both sides: Flexion and internal rotation.	Both sides: Extension and external rotation .
<i>Ankle</i>	Both ankles: no heel – toe action. Lands with both the ankles in a slightly inverted position, fractionally first on the lateral side of the foot (the base of the 5 <sup>th</sup> metatarsal).	On both sides, there is a weight shift to the medial side of the foot (it moves into a more everted position).	The hind foot is in pronation. The left and right calcaneus sinks further into valgus, more so on the right than on the left. Both feet are in a position of abduction.	The hind foot moves out of the pronated position into a neutral position. The ankle starts to move into PF.	The hind foot moves into inversion, more on the right than on the left. Ankles are in plantar flexion.	Hardly any push-off. The hind foot moves into more inversion, especially on the right side. There is a weight shift from the 2 <sup>nd</sup> metatarsal head to the 3 <sup>rd</sup> metatarsal head.	Both ankles are in a position of plantar flexion and slight inversion of the hind foot.	Again: slight plantar flexion and inversion.



○ *Soft tissue palpation*

▪ *Sole of the foot:*

On palpation the area underneath the foot, namely the plantar fascia, felt tight bilaterally compared to the norm, but the left side felt tighter in comparison to the right side.

▪ *Left calf:*

On palpation, the posterior-medial aspect of the calf felt very tight in comparison with the adjacent tissue, especially the area against the medial aspect of the tibia where the soleus muscle is palpable. The medial part of the Achilles tendon felt tight and swollen. A tight area was also palpated over the peroneus longus muscle in the middle one third of the lower leg.

▪ *Right calf:*

On palpation there was tightness in the upper central part of the calf, in the area between the two heads of the gastrocnemius muscle as well as on the medial part of the Achilles tendon. This area again appeared swollen but to a lesser extent than the left.

The motivation and description for the interventions used during this case study will be addressed in each of the phases as described hereafter.

○ *Flexibility/ length of the soleus muscle*

- Right soleus muscle: Big toe six mm from the wall.
- Left soleus muscle: Big toe four mm from the wall.

**4.2.2.10. Intervention and data recording**

The interventions used during the first case study, were changed during the treatment period based on the response of the subject. If an intervention did not lead to any



improvement in the symptoms of CPCS, an alternative approach was adopted. The interventions applied in Case Study 1 consisted of four different phases.

## **Phase 1**

### ***Approach followed***

The first phase consisted of intervention by means of conventional physiotherapy. This entailed the application of massage techniques; stretches of the soleus, and the gastrocnemius muscles; ultrasound; heat; myofascial release techniques of the tight calf muscles; and electrical stimulations to the area of the calf muscles (Biedert & Marti, 1997; Styf, 1998; Micheli *et al.*, 1999; Garcia-Mata *et al.*, 2001).

### ***Treatment period***

24<sup>th</sup> January 2002 to the 15<sup>th</sup> February 2002 (three weeks)

### ***Number of treatments sessions:***

Ten

Initially conventional physiotherapy techniques were applied for the period 24<sup>th</sup> January 2002 to 15<sup>th</sup> February 2002 to treat the symptoms of CPCS. The area of the calf muscles was targeted during the treatment sessions and use was made of massage techniques, cross frictions (where the adhesions could be felt), ultrasound- and interferential therapy, and stretches of the calf muscles.

### ***Outcome measures (as measured on the 15<sup>th</sup> February 2002)***

The following outcome measures demonstrated no improvement on reassessment:

- The intensity of pain / discomfort at rest as well as the amount of pain/discomfort at the end of every training session as plotted on a 100 mm visual analogue scale (VAS). Throughout this period, she rated the pain as a 20 to a 40 on the 100 mm VAS.



- The distances run before the commencement of the symptoms with every training session. The distance varied from one to three kilometres.
- The total weekly distance run. This varied between six and nine kilometres.
- The palpation findings of the soft tissue of the posterior lower leg. This was tight exactly in the same places as with the first assessment.

### ***Discussion***

Since this approach did not lead to an improvement in any of the outcome measures, a different approach was selected. Varelas *et al.* (1993) proposed a strengthening programme of the calf muscles and Chandler & Kibler (1993) proposed proprioceptive retraining as intervention approaches for injuries of the lower limb. Varelas *et al.* (1993) have evaluated muscle function in compartment syndrome and concluded by saying that strengthening may be useful in very mild cases of CPCS or in post fasciotomy patients.

Proprioceptive retraining is recommended after ankle and knee injuries (Lentell *et al.*, 1995; Osborne *et al.*, 2001; and Risberg *et al.*, 2001.) as well as after plantar fasciitis (Chandler & Kibler, 1993). Although these two approaches were not described specifically for the treatment of symptoms of CPCS in the literature, it was decided to apply these two approaches as intervention techniques for CPCS. This decision was based on the fact that all the injuries occur in the lower limb, and both CPCS and plantar fasciitis are fascia-related injuries.

The choice of proprioceptive retraining as an intervention technique was based on the evidence found in the literature that some of the proprioceptive receptors are located in the fascia (Brown, 1980). The researcher thought it was possible that dysfunctional fascia might lead to abnormal feedback via the proprioceptive receptors located in the dysfunctional fascia. If the proprioceptive feedback can be improved by proprioceptive rehabilitation, it might have a positive effect on the symptoms of CPCS by decreasing or alleviating the symptoms.



## **Phase 2**

### ***Approach followed***

The same conventional intervention approach was followed as described during phase 1, together with an eccentric- and concentric strengthening programme of the calf muscles (Varelas *et al.*, 1993) as well as proprioceptive retraining programme (Chandler & Kibler, 1993) to improve the subject's balance reactions.

### ***Treatment period***

15<sup>th</sup> April 2002 to the 3<sup>rd</sup> May 2002 (just more than three weeks)

### ***Number of sessions***

Five

The subject received a training programme for the strengthening exercises. Calf muscles were strengthened concentrically by doing calf raises, one leg at a time. Calf raises were done daily in sets of three with 15 repetitions in each set. Eccentric calf muscle strengthening was combined with proprioceptive retraining on a balance board. The calf muscles were strengthened eccentrically by doing proprioceptive retraining on a balance board, with the knees in a degree of flexion. The subject spent two minutes twice a day on the balance board, throwing a ball against a wall at slightly different angles and catching it. One minute was spent standing with both legs on the balance board, while the remaining time on the board was divided between the left and the right leg (30 seconds per leg).

### ***Outcome measures***

The following outcome measures on the 3<sup>rd</sup> May 2002 showed no improvement:



- The intensity of pain/discomfort at rest as well as the amount of pain / discomfort at the end of every training session as plotted on a 100 mm visual analogue scale (VAS): 20 – 40 mm.
- The distance run before the commencement of the symptoms with every training session: one to three km.
- The total weekly-distance run: six to nine kilometres.
- The palpation findings of the soft tissue of the posterior lower leg. This was tight exactly in the same places as with the first assessment.

### *Synthesis/discussion*

The interventions did not result in the desired outcome, namely the alleviation of the symptoms of CPCS. At this point most of the findings are inconclusive. It is however possible to eliminate some of the issues raised. For convenience the rival theories will be addressed first.

The rival proposition that the treatment of the soft tissue in the calf-area will not lead to the alleviation of the symptoms of CPCS, as a different theoretical framework is responsible for the pathogenesis of the condition, can thus not be discarded.

Similarly, the rival theory that the alleviation of the symptoms of the condition will be due purely to chance, i.e. the intervention has nothing to do with it, can also be discarded. The soft tissue mobilization would in all probability have improved blood flow in the area, but insufficiently to reverse the condition.

The rival theory that the interventions have not been applied long enough also seems unlikely. During the interventions no positive response had been achieved. If interventions are successful, one would expect an incremental response to the application thereof.





The last of the rival theories, which challenges the competence of the researcher, is only of academic importance. It does however need to be addressed. The researcher is nationally acclaimed by the physiotherapy community for her applied skills and this theory is here forth discarded.

The proposition formulated has two components. The first addresses the reduction of pressure in the calf area which clearly has not been achieved. As was shown during the literature review, the surgical release of the pressure in the calf-area is the only intervention that had any degree of success. The second deals with increased blood flow to the area, which has as been stated earlier, insufficient to reverse the situation.

### ***Conclusion***

The current theoretical model does not provide an obvious mechanism for forecasting replication results in the application of treatment interventions for CPCS and calls for further exploration.

### ***Propositions***

Based on the aforementioned the propositions were adapted to the following:

*“An intervention technique that will lead to a reduction in the pressure in posterior compartment will lead to the alleviation of the symptoms of CPCS”.*

### ***Rival theories***

The rival theory was adapted as follows:

*The rival theory is that the treatment of the soft tissue in the calf-area will not lead to the alleviation of the symptoms of CPCS, as a different theoretical framework is responsible for the pathogenesis of the condition.*



### **Phase 3**

#### ***Approach followed***

Interventions were then focused on the fascia and the release of associated muscles. The methodology as described in chapter four was applied (4.3.16 - mobilizations 1 to 3). Specific soft tissue mobilizing techniques were applied to the various fascia septa in the lower leg (such as the posterior inter-muscular septum and the deep transverse fascia). These interventions were supplemented by stretches for the soleus- and the gastrocnemius muscles.

#### ***Treatment period***

10<sup>th</sup> May 2002 to the 2<sup>nd</sup> July 2002 (seven weeks).

#### ***Number of sessions***

Five

During the first week of July 2002, the subject had insoles made by a podiatrist. These however caused severe blistering during running underneath her medial foot bridge. Video assessment of her gait while running on the treadmill with the insoles revealed an increase in abnormal movement patterns. Reassessment the running gait on the treadmill demonstrated an exaggeration of all the previously described gait abnormalities recorded on the video clips, especially the degree of inversion at the calcaneus during the pre-swing phase. During the phase of initial swing, her ankles went into an increased degree of inversion (shifting the weight from the second to the fourth metatarsal head), followed by an increased amount of eversion (seemingly uncontrolled) during the mid- to terminal swing phase in an attempt to correct the exaggerated inversion position. After one week the athlete stopped using the insoles.



*Outcome measures (as measured on the 2<sup>nd</sup> July 2002)*

- The intensity of pain / discomfort at rest as well as the amount of pain/discomfort at the end of every training session as plotted on a 100 mm visual analogue scale (VAS) improved slightly: 20 to 10 mm.
- The distance run before the commencement of the symptoms with every training session improved slightly: two to four kilometres
- The total weekly distance run improved slightly: 9 to 12 kilometres.
- The palpation findings of the soft tissue of the posterior lower leg demonstrated the following changes: The previously described tight area over the peroneus longus muscle in the middle one third of the lower leg on the left side was less tight on palpation.

As can be seen from the above, the interventions generated improvements for the first time. This could be attributed to the fact that the treatment was aimed more specifically at the fascia. This indicated that the solution to the symptoms of CPCS lies in the release of the tightness in the fascial web which is responsible for constricting the function of the muscles enclosed in the posterior compartment. This raised the question as to what the most effective manner would be for releasing the restricting/limiting fascia.

The literature search revealed fascia as a continuum that surrounds, encapsulates, and is intertwined with the muscles. One can thus hypothesise that inflammation, muscle spasm and trigger points in the muscles, etc. would in effect generate stresses on the fascial-web. Such stresses will be transmitted through the web to areas such as the calf area, and could indeed trigger the CPCS condition. This argument is supported by the success with the release of some of the more proximal muscles that are also linked via the fascial chain.

Since the fascia is a continuum, it is possible that there might be an anatomical relation between the muscles encapsulated in the fascia of the posterior compartment and the more proximal soft tissue, i.e. the soft tissue above the knee.



The formation of a non-functional scar, following a period of inflammation, could result in excessive cross-link formation or shortening of the involved connective tissue. Intervention techniques can be used to either break down excessive cross-links or to gain length in the myofascial tissue. These techniques have been described in chapter three.

### ***Conclusion***

Based on these findings and deductions made, the treatment interventions will be adapted as follows:

In addition to the fascial mobilization techniques applied during the third phase, all the tight muscles in the leg and lower back identified during the assessment will be mobilized. The following techniques will be applied:

- The trigger point therapy techniques (Travell & Simons, 1999);
- The myofascial release techniques (Barnes, 1990; Manheim, 1994); and
- Specific soft tissue release techniques (Hunter, 1998).

### **Phase 4**

#### ***Approach followed***

The approach followed was based on intervention by means of the mobilization of the more proximal posterior soft tissue. During this period, the subject followed her exercise programme (stretches of the calf muscles, strengthening of the calf muscles and proprioceptive retraining). The tight posterior soft tissue proximal and distal to the calf muscles was identified through the soft tissue assessment and mobilized. In addition to the calf muscle, the following soft tissue was found to be tight and was mobilized:

- The plantar fascia;
- The hamstring muscles;
- The sacrotuberous ligament and the piriformis muscles.



The plantar fascia was released through a myofascial release technique (Barnes, 1990); the semimembranosus, semitendinosus, biceps femoris and the sacro-tuberous ligament were released using specific soft tissue release techniques (Hunter, 1998) and a trigger point in the piriformis muscle was released (Travell & Simons, 1999).

### ***Treatment period***

5<sup>th</sup> July 2002 to the 25<sup>th</sup> September 2002 (11 weeks).

### ***Number of sessions***

14.

### ***Outcome measures (as measured on the 25<sup>th</sup> September 2002)***

- The intensity of pain/discomfort at rest as well as the amount of pain/discomfort at the end of every training session as plotted on a 100 mm VAS demonstrated good improvement: 0 mm – no pain.
- The distance run before the commencement of the symptoms improved: she could run ten kilometres without pain, discomfort or numbness of the feet.
- The total weekly distance run improved to 27 kilometres. She was still able to run 21 kilometres symptom free, 35 months after the last physiotherapy session.
- The palpation findings of the soft tissue of the posterior lower leg improved slightly: The medial parts of both Achilles tendons were not swollen any more and the alignment more normal (no convex curving any more). Although still tight on palpation bilaterally, the plantar fascia felt less tight.
- Flexibility of the soleus muscles improved: From four to 30 mm on the left side and from six to 32 mm on the right side.

Despite the improvements in the outcome measures, the athlete still felt the need to work hard at her stretches and proprioception in order to remain symptom free. She said that, although she experienced no symptoms, she felt as though the tightness in her calf muscles gradually increased after a two week period of doing no exercises.



### ***Synthesis/discussion***

The intervention by means of the mobilization of the more proximal posterior soft tissue was responsible for the first improvements of the symptoms of CPCS. These improvements created a direct link between the research outcomes and the emerging theoretical reasoning. Although the subject was involved with exercise programme (stretches of the calf muscles, strengthening of the calf muscles and proprioceptive retraining) during this period, it could not have made any significant contribution towards the release of the constraining fascia border within the posterior compartment. Similar to the surgical release of the involved fascia, the intervention had resulted in a loosening of the “non-compliant fascia border”, which alleviated the symptoms.

### ***Conclusion***

These interventions based on the release of fascia through the mobilization of the more proximal posterior soft tissue appear to provide a logical framework for predicting replication of results.

### ***New theoretical framework***

*The entrapment/restriction of the fascia external to the calf area could be responsible for the pathogenesis of CPCS and the release of tightness in more proximal posterior soft tissue will lead to a reduction in the pressure in the calf area, which in turn will lead to the alleviation of the symptoms of CPCS.*

### ***Proposition***

Based on the aforementioned, the propositions were adapted to the following:

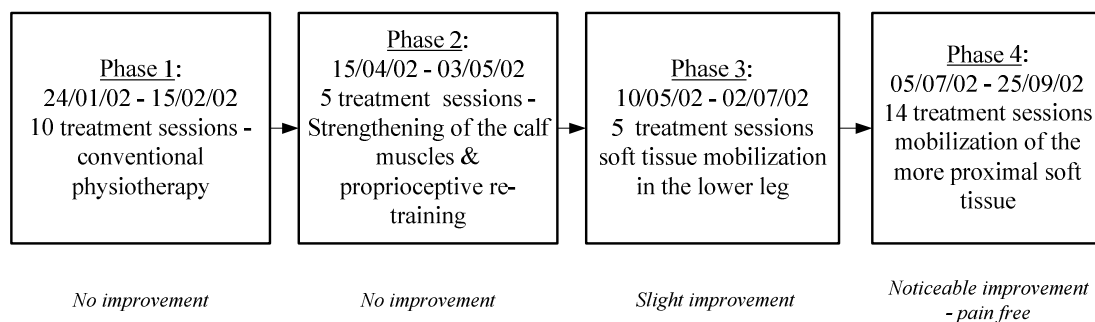
*Interventions based on the release of fascia through the mobilization of the more proximal posterior soft tissue will reduce the pressure in the posterior compartment, which in turn will lead to the alleviation of the symptoms of CPCS.*

### ***Rival theories***

*The rival proposition that the treatment of the soft tissue in the calf-area will not lead to the alleviation of the symptoms of CPCS, as a different theoretical framework is responsible for the pathogenesis of the condition, must be accepted.*

#### **4.2.2.11. Conclusion**

The first case study has revealed that the current theoretical framework does not provide a logical framework for predicting the effects of treatment interventions. The research has indicated a strong possibility that the condition might be resolved by focussing on the fascial web, and particularly at releasing the fascia that are intertwined with more proximal posterior muscles. The chronological development of the first case study is reflected in Figure 5.2.



**Figure 4.2: The chronological development of Case Study 1.**

*Note: Subject 1 was discharged in 2002. In 2006 she completed the Comrades marathon without any problems. The researcher saw her again at the Skukuza half-marathon in August 2007 and she was still running pain free.*

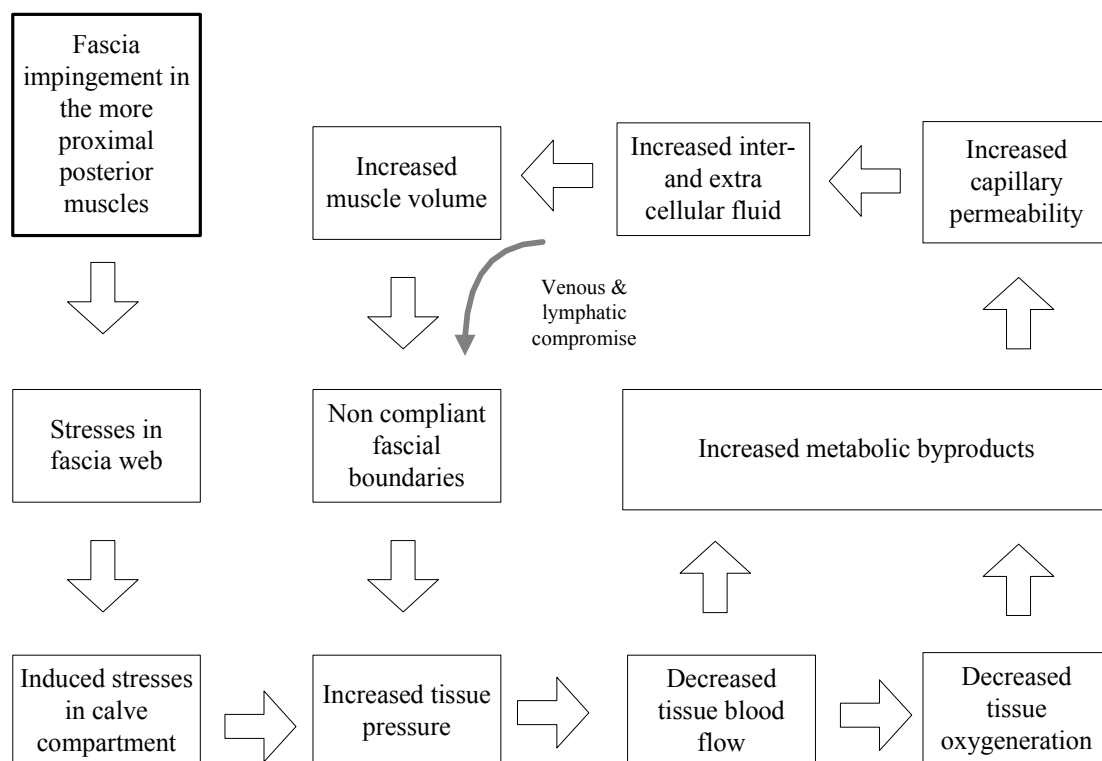
#### **4.2.2.12. Knowledge integration**

The exploratory research has revealed that the fascia, and in particular the impingement thereof in the more proximal posterior muscles play a significant role in the perpetuation of the condition.

Knowledge gained from this study that will be applied in the next case study:

- *The subjects should run bare foot on the treadmill during the video assessment of the running gait to allow surface anatomical markings on the legs to be identified clearly. It might be informative to measure the degree of hind foot pronation in other subjects. The objective is to determine whether the degree of hind foot pronation plays as big a role in the symptoms of CPCS as it does in other running injuries (Hintermann & Nigg, 1998).*
- *It may be necessary to mobilize the other tight proximal soft tissue (myofascial) structures since the subject only started to improve once the hamstring muscles, the piriformis muscles as well as the sacrotuberous ligament have been released. It may therefore be important to identify other more proximal muscles that are tight and that might lead to a further decrease in symptoms when mobilized.*

The modified theoretical framework as a result of these findings is reflected in Figure 4.3.



**Figure 4.3: Modified theoretical framework (Case Study 1)**





### **4.2.3. CASE STUDY 2**

#### ***4.2.3.1. Introduction***

Case Study 2 is the second of the exploratory research cases. This case study is more focused than the first based on the realization from the results of the first that the solution to the problem might lay external to the calf compartment.

#### ***4.2.3.2. Subject***

The subject in Case Study 2 was a 40 year old male athlete who had been participating in road races for the previous eight years.

#### ***Inclusion criteria***

He experienced pain in the centre of the posterior aspect of his lower leg on the left together with a constant feeling of tightness/swelling in the calf, which abated with rest. During October 1997 he was diagnosed with CPCS by a general practitioner who specializes in sport injuries.

#### ***Exclusion criteria***

All the necessary physical tests as described in Chapter 3 were performed to confirm that no exclusion criteria were present. The subject however did not experience lumbar nerve root pain. Although the pain was primarily on the left side, the right calf was also tight/swollen and he had previously experienced the same symptoms on the right side.

#### ***4.2.3.3. The research question***

*Could the entrapment/constriction of the fascia external to the calf area be responsible for the pathogenesis of CPCS and will the release of tightness in more proximal posterior*



*soft tissue lead to a reduction in the pressure in the calf area, which in turn will lead to the alleviation of the symptoms of CPCS?*

#### **4.2.3.4. Theoretical framework**

The revised theoretical framework is reflected in Figure 4.3.

#### **4.2.3.5. Propositions**

*Interventions based on the release of fascia through the mobilization of the more proximal posterior soft tissue will reduce the pressure in the calf area, which in turn will lead to the alleviation of the symptoms of CPCS.*

#### **4.2.3.6. Rival theories**

The following rival theories can be postulated:

- *The exercise programme was responsible for the alleviation of the symptoms.*
- *The interventions responsible for the alleviation of the symptoms of CPCS are not limited to the more proximal posterior soft tissue but extend to other areas as well.*

#### **4.2.3.7. Criteria for interpreting results**

The criteria were the degree of conformance to the propositions made.

#### **4.2.3.8. The Research Procedure**

##### ***Subjective assessment - Interview***

- ***Running history***

The subject had been participating monthly in road races for the previous eight years. The distances varied from 10 to 91 kilometres. His average running pace was five and a half

minutes per kilometre. He never increased his training programme by more than ten percent per week. He had been running in the same brand of neutral running shoe for the past six years and never ran more than 800 kilometres with a pair.

○ ***Previous running injuries***

He was involved in a motorbike accident during 1984 which resulted in reconstructive surgery (1985) for the anterior cruciate ligament of his right knee. Whilst at school, he injured some of the tendons/ ligaments around his right ankle.

○ ***Symptoms noted***

Muscle strength was tested by a biokineticist on a calibrated Isokinetic dynamometer, using a standardized testing protocol. The results are shown in Table 4.2. Statistical significant differences between left and right are reflected in italics.

**Table 4.2: Muscle strength results prior to intervention: Case Study 2**

<i>Movement tested **</i>	<i>Peak torque (Nm)</i>			<i>Work per repetition(Nm/s)</i>		
	<i>Right</i>	<i>Left</i>	<i>Deficit</i>	<i>Right</i>	<i>Left</i>	<i>Deficit</i>
<i>Hip extensors</i>	132	113	<i>-14</i>	158	135	<i>-15</i>
<i>Hip flexors</i>	75	84	<i>-11</i>	87	90	-3
<i>Hip internal rotators</i>	17	24	<i>-29</i>	13	22	<i>-41</i>
<i>Hip external rotators</i>	24	27	<i>-11</i>	17	27	<i>-37</i>
<i>Hip abduction</i>	68	110	<i>-38</i>	38	65	<i>-42</i>
<i>Hip adduction</i>	118	153	<i>-23</i>	80	100	<i>-20</i>
<i>Knee extensors</i>	113	182	<i>-38</i>	111	193	<i>-42</i>
<i>Knee flexors</i>	69	115	<i>-40</i>	79	142	<i>-44</i>
<i>Knee internal rotation</i>	20	28	<i>-29</i>	15	20	<i>-25</i>
<i>Knee external rotation</i>	21	26	<i>-19</i>	17	20	<i>-15</i>
<i>Ankle inversion</i>	37	36	-3	31	46	<i>-33</i>
<i>Ankle eversion</i>	26	38	<i>-32</i>	23	49	<i>-53</i>
<i>Ankle plantar flexors</i>	67	89	<i>-25</i>	41	53	<i>-23</i>
<i>Ankle dorsi flexors</i>	22	37	<i>-41</i>	16	24	<i>-33</i>



○ *History of symptoms and treatment received*

The symptoms had first been experienced on a training run in October 1997, two weeks prior to the Berlin Marathon. His left calf muscles were very sore and felt swollen. He rested for the remaining two weeks prior to running the Berlin Marathon. While participating in the race, his left calf muscles felt tight from the beginning of the race and the associated pain gradually grew worse throughout the race. After the race, his left calf muscles were very sore for a couple of months and the calf muscles of both his left and right legs felt tight and swollen.

Back in South Africa, a couple of weeks after the Berlin Marathon, he consulted a general practitioner, who specializes in the treatment of sport injuries. The practitioner diagnosed him with CPCS and recommended that he rested for eight weeks and during these eight weeks he was instructed to undergo a strengthening programme on the Isokinetic dynamometer for the dorsi flexor - and the plantar flexor muscles of the ankles.

Eight weeks later, he resumed his training programme. During February 1998, he felt fit enough to participate in a 32 kilometre race. About six kilometres into the race, he felt a pain in his right calf muscles. The further he ran the more painful and the tighter his right calf muscles became. Although he completed the race, he was forced by the pain in his right calf to walk a great part of it. After the race, his right calf muscles were very sore and both calves felt very tight. Following this second incident of calf muscle pain, he tried a variety of treatment approaches namely:

- Physiotherapy, consisting of massage of the muscles of the lower leg, cross-frictions of “adhesions” in the calf muscles, ultrasound- and interferential therapy over the area involved as well as calf muscle stretches. The physiotherapy had no beneficial effects and the stretches made his symptoms worse.
- After a number of physiotherapy sessions, he underwent a programme on the Isokinetic dynamometer to strengthen the plantar- and dorsi flexor muscles of the ankles. After confirmation that his plantar- and dorsi flexor muscles were again of normal strength, he had still not experienced any relief from the symptoms of PCS.



He decided to rest again for eight weeks, after which he resumed his training. He followed a very conservative running programme, increasing the distance by not more than ten percent every second week. On the 10<sup>th</sup> of February 2003, a pain in his left calf forced him to walk after he had been running a distance of three and a half kilometres. He commenced running again on the 24<sup>th</sup> of February 2003, but was forced to walk due to pain in the left calf after running four kilometres. He rested his injury until he could walk comfortably again. He ran again on the 6<sup>th</sup> of March 2003. After two and a half kilometres he was forced by pain in his left calf to walk. On the 10<sup>th</sup> of March 2003 after a screening assessment, he was included into the study. In summary, the subjective outcome measurements as measured before the intervention:

- The intensity of pain/discomfort at rest was 60 on the 100 mm VAS.
- He could not run at all.

#### ***The Objective assessment - Physical examination***

##### ○ ***Muscle strength tests***

Muscle strength was tested by a biokineticist on a calibrated Isokinetic dynamometer, using a standardized testing protocol. The results are shown in Table 4.2.

##### ○ ***Analysis of running gait, including movement patterns***

The subject was videotaped while running, from both sides as well as from posterior. The subject ran bare footed on the treadmill during the videotaping. Details of the observations found during the gait analysis are provided in Table 4.3. The following movement patterns deviated from the normal/ideal running patterns:

**Table 4.3: Outcomes of running gait analysis for Case Study 2 prior to intervention**

<i>Running gait analysis: Case Study 2 - 10/03/2003</i>								
	<i>Weight Acceptance</i>		<i>Single Leg Support</i>			<i>Swing Leg Advancement</i>		
<i>Joint</i>	<i>Initial Contact</i>	<i>Loading Response</i>	<i>Mid-stance</i>	<i>Terminal Stance</i>	<i>Pre-swing</i>	<i>Initial Swing</i>	<i>Mid-swing</i>	<i>Terminal Swing</i>
<i>Pelvis</i>						Occasionally, there is an increased degree of posterior pelvic rotation on the left compared to the right.		
<i>Hip</i>				The left hip is in more lateral rotation and extension in comparison to the right side.	The left hip is in more lateral rotation and extension in comparison to the right side.	Both hips start to move into internal rotation from the externally rotated position. Every 3 <sup>rd</sup> or 4 <sup>th</sup> stride, there is an irregularity on the left side with an increased amount of left external rotation accompanied with more of a posterior pelvis rotation on the left.	Both hips are in flexion and internal rotation. An increased degree of internal rotation on the left follows on the occasional increased lateral rotation on the left.	
<i>Knee</i>	Both knees: almost full extension with the tibias in external rotation. A slightly greater right knee extension in comparison to the left.	The degree of knee extension decreases slightly in both legs.	The amount of knee extension increases slightly again with the tibia being in external rotation.	The tibias move into more external rotation with the knees in extension.	Both knees are extended with some external rotation of the tibias.	Both knees move out of extension and external rotation into flexion.	Both sides: flexion and internal rotation.	Both sides: extension and external rotation.
<i>Ankle</i>	Both ankles: no heel-toe action. Lands with both the feet fractionally first on the lateral side of the foot (5 <sup>th</sup> metatarsal bone).	On both sides, there is a weight shift to the medial side of the foot.	Both hind feet are in pronation. The eccentric control is less on the left side; after weight acceptance, the hind foot on the left side sinks further into valgus. Both feet are in a position of abduction.	The hind foot move out of a pronated position into neutral.	The hind foot on the right moves more into inversion in comparison to the left side.	The hind foot on the right moves more into inversion in comparison to the left.	Both ankles are in slight plantar flexion and slight inversion.	Slight plantar flexion and inversion.
<i>Toes</i>				The subject rises higher onto his fore foot than normal.				The extensor tendons of the lateral 4 toes are very active.

- Throughout the running phases, the pelvis on the left side remained slightly more posteriorly rotated in comparison to the right side.
  - During terminal stance and pre-swing phases, the left hip moved into more lateral rotation and extension in comparison to the right side. Occasionally, every third or fourth stride, there was an increase in the amount of lateral rotation and extension on the left, followed by an increased medial rotation on the forward movement of the left hip as counter action. This phenomenon was observed as a “wobble” in the left ankle.
  - There was no heel toe action and during mid stance, the calcaneus sunk into more of a valgus position on the left than would normally be expected. During the pre-swing phase the calcaneus on the right sides moved into inversion as though there was a slight weight shift from the second metatarsal head to the third metatarsal head.
  - The subject had a greater degree of metatarsophalangeal extension in the forefoot during the push off than what one would normally expect.
- *Measurement of specific biomechanical angles*

The measurements of the degree of dorsi flexion at the ankle joint, and the degree of extension at the first metatarsophalangeal joint was done as described respectively in Chapter 3 sections 3.4.2.8. The biomechanical measurements are provided in Table 4.4.

**Table 4.4: Biomechanical angles: Case Study 2 during intervention – (15/05/2003)**

<i>Movement</i>	<i>Joint Range of Movement (degrees)*</i>	
	<i>Mid stance</i>	<i>Terminal stance</i>
<i>Dorsi flexion left</i>	7, 33°	15, 33°
<i>Dorsi flexion right</i>	10, 33°	18, 00°
<i>Metatarsophalangeal extension left</i>		47, 00°
<i>Metatarsophalangeal extension right</i>		40, 33°
<i>Hind foot pronation left</i>		9, 00°
<i>Hind foot pronation right</i>		9, 00°

\* *Averages of three measurements*



○ ***Soft tissue palpation***

The left and right sides of the body were compared to each other, and all the soft tissue palpated in order to assess the extent of tightness.

*Left side:*

The plantar fascia felt tight as well as the lateral side of the calf muscles (gastrocnemius muscle). The soleus muscle, palpated from anterior, felt tight. The iliotibial band and the central part of the hamstring felt tight. This palpated tightness extended into the gluteus maximus muscle on the left side. Higher up, the fascia- junction between the latissimus dorsi muscle and the trapezius muscle in the vicinity of T8 felt tight. In the trunk area, the quadratus lumborum and the erector spinae muscles felt tight.

*Right side:*

The lateral part of the calf muscles felt tight (tighter compared to the left side, but along a smaller area). The soleus muscle, palpated from the anterior also felt tighter than the left side. The right iliotibial band felt tight as well as the fascia-junction of the latissimus dorsi muscle and the trapezius muscle in the vicinity of T8. This fascia junction seemed tighter on the right than the left side. In the area of the trunk, the quadratus lumborum and the erector spinae felt tighter on the right than on the left.

○ ***Flexibility/length of the soleus muscle***

- *Right soleus muscle: Big toe ten mm from the wall*
- *Left soleus muscle: Big toe 12 mm from the wall*

**4.2.3.9. Intervention and data recording**

With this subject, the mobilizing of the distal and the more proximal tight soft tissue structures were included from the start. All the tight soft tissue identified during the assessment was mobilized.





## Phase 1

*Approach followed:* Intervention by means of the mobilizing of the distal and the more proximal posterior soft tissue.

The following soft tissues were mobilized:

- plantar fascia (myofascial release according to Barnes, 1990),
- the gastrocnemius and soleus muscles. The soleus muscle was mobilized through specific soft tissue techniques according to Hunter (1998), working from the anterior aspect medial to the tibia. The gastrocnemius muscle was released with myofascial release techniques (Mannheim, 1994),
- the pes anserine (myofascial release technique of the adductor muscles) (Mannheim, 1994),
- the ilio tibial band (myofascial release techniques) (Mannheim, 1994),
- the hamstring muscles (specific soft tissue mobilization techniques) (Hunter, 1998),
- the gluteus maximus (trigger point release) (Travell & Simons, 1999),
- the piriformis muscles (trigger point release) (Travell & Simons, 1999),
- the sacrotuberous ligament (specific soft tissue mobilization techniques) (Hunter, 1998),
- the quadratus lumborum muscle (trigger points release) (Travell & Simons, 1999),
- the erector spinae (myofascial release technique) (Mannheim, 1994) and
- the fascial link between the latissimus dorsi- and the lower fibres of the trapezius muscle in the vicinity of T8 (Hunter, 1998).

Stretches for the piriformis muscle, as well as the hamstring muscles was introduced at this stage. This was in addition to stretches of the calf muscles introduced in the initial treatment programme. These stretches did not aggravate his symptoms. The descriptions of the newly introduced stretches are as follows:



### ***Piriformis stretch***

- The supine subject crossed the leg on the side to be stretched over the opposite thigh, and rested the opposite hand on the knee of the uppermost limb to be stretched.
- This hand is used, when needed, to assist gravity in adducting the leg to be stretched, which is flexed 90°.
- The subject stabilizes the hip on the side to be stretched by pressing down on the iliac crest with the ipsilateral hand. (Travell & Simons, 1999)

### ***Hamstring stretch in sitting***

- Sit on the floor with the leg to be stretched extended, and the other leg bent with the foot towards the body.
- Reach out with the hands, lean the upper body forwards, and bring the chest towards the thigh.
- Care must be taken not to round the upper back; the lower back should be slightly curved.
- Get to the point of a mild stretch and hold.
- Repeat for each leg.

### ***Hamstring stretch in supine***

- Lie on the back with the legs out.
- Raise the leg to be stretched with the knee bent.
- Grasp the calf or thigh and gently pull the leg towards the body.
- Get to the point of a mild stretch and hold.
- Repeat for each leg.

### ○ ***Treatment period***

10<sup>th</sup> March 2003 – 28<sup>th</sup> May 2003 (six weeks)



○ ***Number of sessions***

11 treatments were performed between the 10<sup>th</sup> March 2003 and the 28<sup>th</sup> May 2003.

○ ***Outcome measures (as measured on the 28<sup>th</sup> May 2003)***

At the end of May, he was running 12 kilometres per week without any pain. A videotape of his running gait was repeated at this stage. His running gait had improved in terms of symmetry and abnormal movement patterns. The increased lateral hip rotation, followed by an increased medial rotation upon hip flexion that occurred on the left side with every third or fourth stride, disappeared. His calf (soleus) muscle flexibility improved from ten mm to 28 mm on the right and from 12 mm to 29 mm on the left (distance of toe from wall in knee-to-wall test).

○ ***Synthesis/discussion***

Subject two has had symptoms of CPCS since October 1997 and had previously tried physiotherapy, strengthening of the plantar flexor muscles on the Isokinetic dynamometer, and stretches of the calf muscles to no avail. With the new approach of mobilizing the more proximal soft tissue, the subject was pain free within a period of two and a half months (11 treatment sessions).

○ ***Conclusion***

This led to the notion that the anterior soft tissue distally as well as proximally might also be tight and that the mobilizing of these soft tissue links might further aid the outcomes of the treatment approach. Working additionally on the anterior tight soft tissue (myofascial links) might cause the symptoms of CPCS to disappear more quickly. With the next subject the anterior myofascial tissue would be mobilized additionally. The exercise programme as a possible causal factor for success can safely be discarded as a rival theory in the light of that with Case Study 2, no strengthening programmes were utilised.



○ ***New theoretical framework***

The entrapment/constriction of the fascia external to the calf area could be responsible for the pathogenesis of CPCS and the release of tightness in more proximal posterior as well as the anterior soft tissue will lead to a quicker response to the interventions.

○ ***Propositions***

Based on the aforementioned the proposition was adapted to the following:

*Interventions based on the release of fascia through the mobilization of the more proximal posterior as well as anterior soft tissue will lead to a quicker response to treatment interventions.*

○ ***Rival theories***

- *The positive response to the interventions is due to the stretching programmes.*
- *The improvements are purely by chance and there is a different theoretical framework responsible for the pathogenesis of CPCS.*

An argument that could have been raised with Case Study 1 is that the improvements were due to the fact that the interventions had been applied long enough in order to be effective. The quick response of Case Study 2 with its focus on the more proximally muscles, defeats this argument.

#### **4.2.4. CASE STUDY 3**

##### ***4.2.4.1. Introduction***

Case Study 3 is the last of the exploratory case studies and is more focused than the first two due to the realization that the solution to the problem lay external to the calf compartment.



#### **4.2.4.2. Subject**

The subject was a 32 year old female athlete participating in ten kilometre road races.

#### ***Inclusion criteria***

She complained of symptoms of CPCS in both legs that increased during running and decreased with rest. She complained of pain and stiffness over the posterior-lateral and especially over the posterior-medial aspect of the middle one-third of the posterior part of both lower legs. She also experienced stiffness, but to a lesser extent, over the anterior-lateral and the anterior-medial aspect of both the lower legs. She experienced constant discomfort with walking and climbing stairs. She rated the intensity of the pain as a 65 on a 100mm VAS.

#### ***Exclusion criteria***

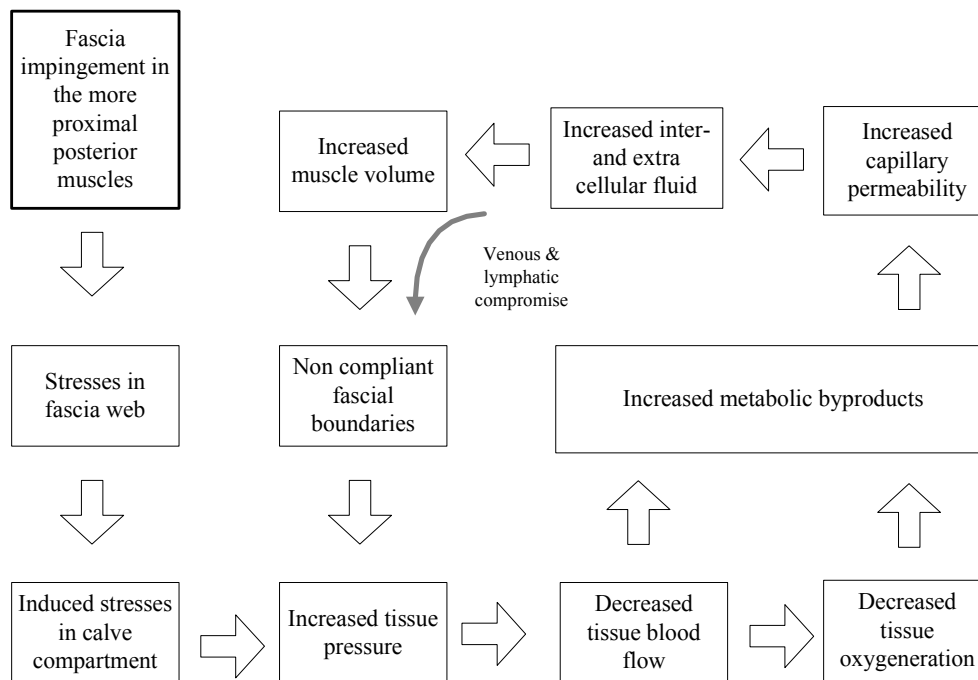
None.

#### **4.2.4.3. The research question**

*Would the release of tightness in more proximal posterior as well as anterior soft tissue lead to a quicker response in the alleviation of the symptoms of CPCS?*

#### **4.2.4.4. Theoretical framework**

The theoretical framework that evolved from the exploratory research is reflected in Figure 4.4.



**Figure 4.4: The evolved theoretical framework**

#### 4.2.4.5. Propositions

*Interventions based on the release of fascia through the mobilization of the more proximal posterior soft tissue will reduce the pressure in the calf area, which in turn will lead to the alleviation of the symptoms of CPCS.*

#### 4.2.4.6. Rival theories

The following rival theories can be postulated:

- *The exercise programme of the subject in Case Study 1 was responsible for the alleviation of the symptoms of CPCS.*
- *The interventions responsible for the alleviation of the symptoms of CPCS are not limited to the more proximal posterior soft tissue but extend to other areas as well.*



#### **4.2.4.7. The Research Procedure**

##### ***Subjective assessment - Interview***

###### ***o Running history***

The subject in case 3 was a 32 year old female athlete who had been participating in road races. She ran in ten kilometre races at an average pace of just over six minutes per kilometre. Her programme consisted of a gradual build-up of running distance. She started on the walk/ run programme and her running time gradually increased to the point where she was running for a full five kilometres. This programme was followed three times a week. She was running in slight anti-pronation shoes.

###### ***o Previous running injuries***

The subject had no history of any previous running injuries. She had had an operation for the removal of bunions on both feet two years previously.

###### ***o Symptoms noted***

She was assessed on the 13<sup>th</sup> May 2003. She complained of pain and stiffness over the posterior-lateral and especially over the posterior-medial aspect of the middle one-third of the posterior aspect of both lower legs. She also experienced stiffness, but to a lesser extent over the anterior-lateral and the anterior-medial aspect of both the lower legs. At the time of her first consultation, she found it too painful to run. She experienced constant discomfort with walking and climbing stairs. The intensity of the pain was 65 on a 100 mm VAS.

###### ***o History of current symptoms and previous treatment received***

She had experienced the symptoms for a period of four months. When she reached the stage where her calves became too painful to run, she exchanged the running for a two-week programme of stretching and strengthening work in the gymnasium. Her calves

improved and she could resume the running again, which was followed by a gradual build-up of pain and tightness. She had no treatment. Subjective outcome measures were:

- The intensity of pain / discomfort at rest was 65 on the 100 mm VAS.
- She could not run at all.

**Objective assessment – Physical examination**

○ **Muscle strength tests**

Muscle strength was tested on a calibrated Isokinetic dynamometer, using a standardized testing protocol.

**Table 4.5: Muscle strength results\* prior to intervention: Case Study 3**

<i>Movement tested**</i>	<i>Peak torque (Nm)</i>			<i>Work per repetition(Nm/s)</i>		
	<i>Right</i>	<i>Left</i>	<i>Deficit</i>	<i>Right</i>	<i>Left</i>	<i>Deficit</i>
<i>Hip extensors</i>	85	99	<i>-14</i>	123	144	<i>-14</i>
<i>Hip flexors</i>	46	39	<i>-15</i>	60	62	<i>-4</i>
<i>Hip internal rotators</i>	5	5	<i>0</i>	4	5	<i>-25</i>
<i>Hip external rotators</i>	8	9	<i>-14</i>	7	8	<i>-17</i>
<i>Hip abduction</i>	30	43	<i>-31</i>	11	20	<i>-47</i>
<i>Hip adduction</i>	4	26	<i>-84</i>	1	11	<i>-88</i>
<i>Knee extensors</i>	71	79	<i>-10</i>	81	84	<i>-3</i>
<i>Knee flexors</i>	46	43	<i>-6</i>	61	56	<i>-9</i>
<i>Knee internal rotation</i>	9	8	<i>-14</i>	8	8	<i>0</i>
<i>Knee external rotation</i>	12	11	<i>-11</i>	12	11	<i>-11</i>
<i>Ankle inversion</i>	15	11	<i>-27</i>	22	14	<i>-38</i>
<i>Ankle eversion</i>	15	12	<i>-18</i>	22	14	<i>-38</i>
<i>Ankle plantar flexors</i>	57	53	<i>-7</i>	39	34	<i>-14</i>
<i>Ankle dorsi flexors</i>	18	19	<i>-7</i>	14	12	<i>-10</i>

\*Statistically significant values between the left and the right sides are reflected in *italics*

\*\* Tested at a speed of 30/30 and with five repetitions

○ **Analysis of running gait, including movement patterns**

A summary of the gait analysis is provided in Table 4.6.



**Table 4.6: Outcomes of the running gait analysis for Case Study 3 prior to intervention**

<i>Running gait analysis: Case Study 3 (13/05//2003)</i>								
	<i>Weight Acceptance</i>		<i>Single Leg Support</i>			<i>Swing Leg Advancement</i>		
<i>Joint</i>	<i>Initial Contact</i>	<i>Loading Response</i>	<i>Mid-stance</i>	<i>Terminal Stance</i>	<i>Pre-swing</i>	<i>Initial Swing</i>	<i>Mid-swing</i>	<i>Terminal Swing</i>
<i>Pelvis</i>	On the left side, the pelvis is in anterior rotation. On the right, the pelvis is also in an anteriorly rotated position, but less compared to the left side.			Both sides are in posterior rotation; right side more than left side.	Both sides are in posterior rotation; right side more than left side.		Both sides move out of posterior rotation.	Both sides move into anterior rotation. Left more than right.
<i>Hip</i>			The hip on the left side is in slightly more adduction than on the right side.	The right hip is in a greater lateral rotation and extension compared to the left side.	The right hip is in a greater lateral rotation and extension in comparison to the left side.	Both hips start to move into internal rotation from the externally rotated position.  A periodic irregularity on the right side with an increased amount of external right hip rotation and posterior pelvic rotation on the right.	Both hips are in flexion and internal rotation. Increased medial rotation in on the right side, following the occasional irregularity described in the previous column.	Both hips are in flexion.
<i>Knee</i>	Both knees: almost full extension with the tibia in external rotation.	The amount of knee extension decreases slightly in both knees.	The amount of extension in both knees decrease further with the tibia being in external rotation.	The tibias move into a greater external rotation. With the knees in extension.	Both knees are extended with some external rotation of the tibias.	Both knees move out of extension and external rotation into flexion.	Both sides: Flexion and internal rotation..	Both sides: Extension and external rotation.
<i>Ankle</i>	Definite heel- toe action.	A weight shift to the medial side of the foot on both sides (it moves into a greater everted position).	Hind foot is in pronation. Left and right calcaneus sinks slightly further into valgus, more on the right than on the left. Both feet are in abduction.	The hind foot moves out of the pronated position into a slightly everted position, more on the right. The ankle starts to move into PF.	The hind foot moves into inversion. Ankles are in plantar flexion.	Hardly any push-off. The hind foot moves into greater inversion.	Both ankles are in a position of plantar flexion and a neutral position of the hind foot.	Again: slight plantar flexion in a neutral position of the hind foot.
<i>Toe</i>							Extension, especially of the big toe.	Extension, especially of the big toe.

The following movement patterns deviated from the normal/ ideal running patterns:

- Throughout the running phases, the pelvis on the right side remained slightly more posteriorly rotated in comparison to the left. Although the pelvis on the right moved into anterior rotation during the initial contact phase, the degree of anterior rotation was still less than that on the left.
  - During the terminal stance and pre-swing phases, the right hip moved into more lateral rotation and extension compared to the left. Occasionally a further increase in lateral rotation and extension on the right side occurred, followed by increased medial rotation on the forward movement of the right hip.
  - During the phase of mid stance, the calcaneus on the right sunk into more of a valgus position than would normally be expected. Both feet were in a position of abduction. During the initial swing phase, there was hardly any push-off on both sides.
  - During the mid swing and the terminal swing phase, the tendon on both the big toes appeared very active.
- *Measurement of biomechanical angles*

The Biomechanical angles as measured with Corel Draw are provided in Table 4.7.

**Table 4.7: Biomechanical angles for Case Study 3 during intervention – (15/05/2003)**

<i>Movement</i>	<i>Joint Range of Movement (degrees)*</i>	
	<i>Mid-stance</i>	<i>Terminal stance</i>
<i>Dorsi flexion left</i>	5,25°	11,67°
<i>Dorsi flexion right</i>	4,67°	12,33°
<i>Metatarsophalangeal extension left</i>		45,67°
<i>Metatarsophalangeal extension right</i>		53,00°
<i>Hind foot pronation left</i>		10,67°
<i>Hind foot pronation right</i>		7,67°

*\*Averages of three measurements*



○ *Flexibility/ length of the soleus muscle*

- Right soleus: Big toe five mm from the wall.
- Left soleus: Big toe seven mm from the wall.

○ *Soft tissue palpation*

The left and right sides of the body were compared to each other and all the soft tissue palpated in order to assess the tightness.

○ *Posterior soft tissue assessment*

- The plantar fascia as well as the soleus muscles felt tight on both sides.
- The gastrocnemius muscles felt very tight on both sides.
- The iliotibial band, the gluteus medius-, gluteus maximus- and quadratus lumborum muscles felt tight on both sides, right more than left.
- The hamstring muscles felt tight on both sides, left more than right.

○ *Anterior soft tissue assessment*

- The pes anserine felt tight on both sides, right more than left.
- Both soleus muscles felt very tight.
- The tensor fascia lata felt tight on the right side.
- Iliopsoas muscles felt tight on both sides, but left more than right.
- The pectoralis major muscle felt tight on the left side.

#### **4.2.4.8. Intervention and data recording**

##### **Phase 1:**

The release of the posterior tight myofascial links, as described in Case Study 2, was done during this phase together with stretches of the calf muscles, the hamstring muscles and the piriformis muscles.



○ ***Treatment period***

14<sup>th</sup> May 2003 to 13<sup>th</sup> June 2003 (four weeks).

○ ***Number of session:***

Six.

○ ***Interventions***

During the first six sessions only the posterior links were treated in order to determine the effect of the release of the anterior links. During the first six treatments, the researcher mobilized:

- the plantar fascia (myofascial release according to Barnes, 1990);
- the gastrocnemius and soleus muscles (the soleus muscle was mobilized through specific soft tissue techniques according to Hunter (1998), working from the anterior aspect; medial to the tibia. The gastrocnemius muscle was released with myofascial release techniques (Mannheim, 1994);
- the ilio tibial band (myofascial release techniques according to Mannheim (1994);
- the hamstring muscles (specific soft tissue mobilization techniques according to Hunter);
- the gluteus maximus (trigger point release according to Travell and Simons (1999);
- the quadratus lumborum muscle (trigger point release according to Travell & Simons, 1999); and
- the sacrotuberous ligament (specific soft tissue mobilization techniques according to Hunter, 1998).

All the muscles that were mobilized were followed up by stretches in order to try and maintain the length that had been gained with the soft tissue mobilization. The following stretches were also added to the stretches of the soleus-, the gastrocnemius, the piriformis and the hamstring muscles:



○ ***The quadratus lumborum muscle***

Side lying: the lower limb on the side involved was uppermost and extended behind the other lower limb. The subject anchored the ribcage in an elevated position by raising the arm and grasping the head of the treatment table. A pillow underneath the lumbar region further increased the stretch (Travell & Simons, 1999).

○ ***Outcome measures (as measured on the 13<sup>th</sup> June 2003)***

The subject improved and she was able to run faster (she managed a personal best time during a ten kilometre race) and the distance that she ran per week, increased from nought to 15 to 20 kilometres per week. She ran pain free every second or third run, but during the other runs, she still experienced pain during the run. The pain as indicated on a 100mm VAS, decreased from a 65 to a 40 during a run.

**Phase 2**

○ ***Treatment period***

26<sup>th</sup> June 2003 to 1<sup>st</sup> September 2003

○ ***Number of sessions***

Five

During the last five treatments, the following tight anterior soft tissue was also mobilized in addition to the posterior muscles:

- both soleus muscles from anterior (specific soft tissue mobilization techniques according to Hunter, 1998);
- the tensor fascia lata on the right (trigger point release according to Travell and Simons (1999) as well as myofascial release technique according to Mannheim (1994);



- both iliopsoas muscles (trigger point release according to Travell and Simons (1999) and myofascial release technique according to Mannheim (1994); and
- the pectoralis muscle on the left (trigger point release according to Travell and Simons (1999); and myofascial release technique according to Mannheim (1990).

Stretches for the iliopsoas muscles and the pectoralis muscles were added:

#### ***Iliopsoas muscle***

In side-lying: The subject extended the hip of the upper leg and grasped the upper leg with the uppermost hand. Very carefully without rolling backwards, the subject then increased the amount of hip extension in the uppermost leg by pulling the leg as far backwards as possible (Travell & Simons, 1999).

#### ***Pectoralis muscle***

The in-doorway stretch exercise was useful to stretch all of the adductors and internal rotators at the shoulder. To achieve this, the subject stood in a narrow doorway with the forearms against the doorjambs. One foot was placed in front of the other, and the forward knee was bent. The subject held the head erect looking straight ahead, neither craning the head forward nor looking down at the floor. As the forward knee bent and the subject leaned through the doorway, a slow, gentle passive stretch was exerted bilaterally on the pectoralis muscles. The hand position against the door jambs was adjusted to vary the stretch on different sections of the muscle. Fibres of the clavicular section were best stretched in the low hand position. By raising the hands to the middle hand-position with the upper arms horizontal, the sternal section was stretched. By moving the hands as high as possible, while keeping the forearms against the doorjambs, the costal and more vertical abdominal fibres that form the lateral margin of the muscle were stretched (Travell & Simons, 1999).



○ ***Outcome measures (as measured on the 1<sup>st</sup> of September 2003)***

The following noticeable changes took place in the parameters following the five treatment sessions in phase two:

- After the second treatment discomfort in the lower legs disappeared after five kilometres.
- After the third treatment, the pain on a 100 mm VAS went down to ten. The area of discomfort reduced to a small area located over the medial aspect of the posterior calf.
- After the fifth treatment she was running pain free for the whole ten kilometres.
- The outcome measures after a total of 11 treatment sessions during the period 13<sup>th</sup> of May 2003 to the 1<sup>st</sup> of September 2003 were:
  - ✚ She was running 17, 5 kilometres per week pain free and also completed her first 21 kilometre race on the 1<sup>st</sup> of September 2003. (In 2005 she was still running pain free and had completed her first 32 kilometre race.)
  - ✚ Her calf mobility improved from five mm to 30 mm on the right and from seven mm to 30 mm *on the left*.

The knowledge gained from this study which would be applied in the next Case Study, is as follows: Working on the anterior tight muscles accelerated the rate at which the symptoms improved. It was also important to establish which physically palpable muscles are linked via the fascia to the involved calf muscles. If these muscles could be identified through a literature study, they would form the basis of the soft tissue assessment and the soft tissue mobilizations. If these were tight, they might have an influence on the symptoms of CPCS.

#### **4.2.4.9. Integration**

##### ***Pain/discomfort***

The symptoms of the subjects progressively improved as the interventions were adapted to include the mobilization of the more proximal muscles. In terms of pain/discomfort all

the subjects were symptom free at the end of the interventions. The intensity of pain/ discomfort as measured prior- and post intervention is summarized in Table 4.8.

**Table 4.8: The intensity of pain/ discomfort before and after intervention**

<i>Case Studies</i>	<i>Intensity of Pain VAS- 100mm (visual analogue scale)</i>		
	<b>1</b>	<b>2</b>	<b>3</b>
<i>Prior to intervention</i>	20	60	65
<i>Post intervention</i>	0	0	0

***Distance run before commencement of symptoms***

The distance run before commencement of symptoms is reflected in Table 4.9 below.

**Table 4.9: The distance run before commencement of symptoms**

<i>Case Studies</i>	<i>Distance run (km)</i>		
	<b>1</b>	<b>2</b>	<b>3</b>
<i>Prior to intervention</i>	1	0	0
<i>Post intervention</i>	21	21	21

***Total weekly distance run***

The same argument applies in terms of the third outcome measure used, namely the total weekly distance run. The cross case results for this measure are reflected in Table 4.10.

**Table 4.10: The total weekly distance run before and after intervention.**

<i>Case Studies</i>	<i>Total weekly distance run (km)</i>		
	<b>1</b>	<b>2</b>	<b>3</b>
<i>Prior to intervention</i>	12	0	0
<i>Post intervention</i>	17	12	18



### **Conclusion**

The improvements in all three of the outcome measures were significant.

### **Muscle imbalances**

A comparison of the abnormalities in muscle imbalances between case studies 2 and 3, based on *peak torque performance* is reflected in Table 4.11.

**Table 4.11: Muscle imbalances in peak torque performance prior to intervention**

<i>Movement tested **</i> <i>Peak torque</i>	<i>Imbalances between Left and Right (Nm)</i>	
	<i>CS 2</i>	<i>CS 3</i>
<i>Hip extensors</i>	<i>-14</i>	<i>-14</i>
<i>Hip flexors</i>	<i>-11</i>	<i>-15</i>
<i>Hip internal rotators</i>	<i>-29</i>	<i>0</i>
<i>Hip external rotators</i>	<i>-11</i>	<i>-14</i>
<i>Hip abduction</i>	<i>-38</i>	<i>-31</i>
<i>Hip adduction</i>	<i>-23</i>	<i>-84</i>
<i>Knee extensors</i>	<i>-38</i>	<i>-10</i>
<i>Knee flexors</i>	<i>-40</i>	<i>-6</i>
<i>Knee internal rotation</i>	<i>-29</i>	<i>-14</i>
<i>Knee external rotation</i>	<i>-19</i>	<i>-11</i>
<i>Ankle inversion</i>	<i>-3</i>	<i>-27</i>
<i>Ankle eversion</i>	<i>-32</i>	<i>-18</i>
<i>Ankle plantar flexors</i>	<i>-25</i>	<i>-7</i>
<i>Ankle dorsi flexors</i>	<i>-41</i>	<i>-7</i>
<i>Total per Case Study</i>	<i>14</i>	<i>12</i>

\*Statistically significant differences are reflected in *italics*

\*\* Tested at a speed of 30/30 and with five repetitions

### **4.2.5. Conclusion**

During the interventions with these three case studies better outcomes were achieved through treatment techniques applied distally, further proximally, as well as anteriorly

from the posterior compartment. This implied that the more proximal and the anterior soft tissue could be connected to the soft tissue of the calf muscles. Based on this argument the anatomical links between the calf muscles and the more proximal anterior and posterior myofascial structures were explored. The progressive improvements in outcomes are reflected in Table 4.12.

**Table 4.12: A summary of the chronological progression of case studies 1 to 3**

<i>Subjects</i>	<i>Main approach used</i>	<i>Main outcome</i>
<b>Subject 1</b>	<i>Phase 1: 24/01/02 - 05/02/02</i> - Conventional physiotherapy	No improvement
	<i>Phase 2: 15/04/02 –03/05/02</i> - Calf muscle strengthening and proprioceptive retraining	No improvement
	<i>Phase 3: 10/05/02 - 02/07/02</i> - Soft tissue mobilization aimed specifically at the fascia	Slight improvement
	<i>Phase 4: 05/07/02 - 25/09/02</i> - Mobilization of the more proximal soft tissue plus stretches	Noticeable improvement
<b>Subject 2</b>	<i>Phase 1: 10/03/03 –28/05/03</i> - Mobilization of the more proximal posterior soft tissue plus stretches	Progressive improvement
<b>Subject 3</b>	<i>Phase 1: 14/05/03 -13/06/03</i> - Mobilization of the more proximal posterior soft tissue plus stretches	Noticeable improvement
	<i>Phase 2: 26/06/03 -01/09/03</i> - Mobilization of the more proximal anterior and posterior soft tissue plus stretches	Noticeable improvement

#### **4.3. THE DEVELOPMENT OF THE CONCEPT OF CLINICALLY SIGNIFICANT MUSCLES**

##### **4.3.1. Introduction**

The relative successes which were achieved with the mobilization of muscles external to the calf area, led to the exploration of the relationships that exist between these muscles and the fascia that link them to the fascia surrounding the muscles of the posterior compartment. The required knowledge with regard to these relationships however did not



exist at the outset of this research. Information on fascia obtained from the literature research covered in Chapter 2, was fragmented. In order to create a holistic perspective of these relationships the contributions of various researchers had to be integrated into a bigger picture.

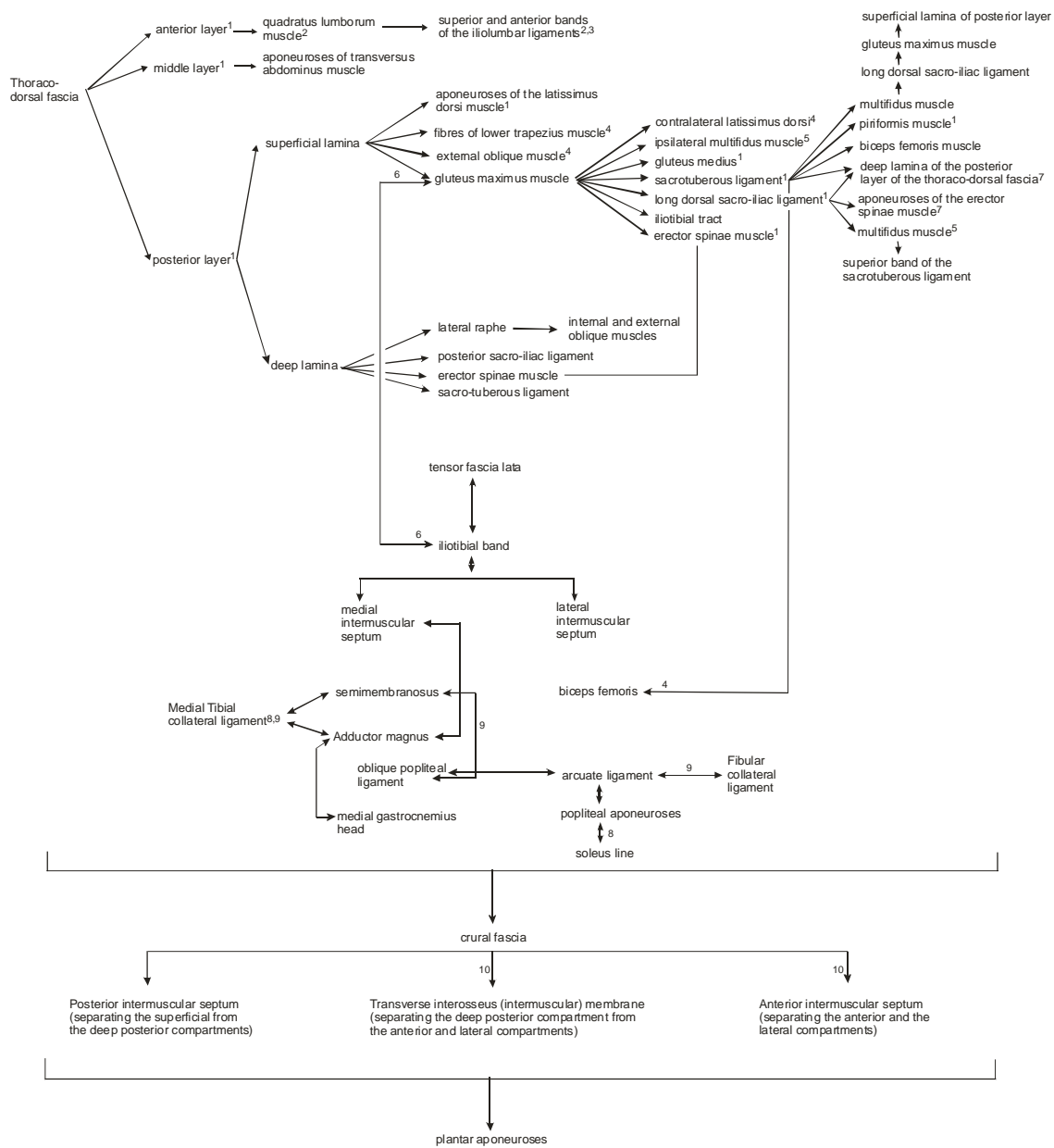
#### **4.3.2. Myofascial links and clinically significant muscles**

Comprehensive maps were created through the integration of information from various researchers that reflects these relationships that exist between the muscles and the myofascial web. This led to a clearer understanding of the relationships between the muscles external to the calf-area and the fascia surrounding the posterior compartment, and how these links could influence stresses in the fascia of the posterior compartment. The results of the knowledge integration of these myofascial links of the trunk and the leg are summarized in Figures 4.5 and 4.6. These figures represent a significant and original contribution of the research project. The muscles that could influence the stress in the fascia of the posterior compartment were classified as the *clinically significant muscles* and are reflected in Table 4.13

#### **4.3.3. Significance of the myofascial links**

Through these links, restrictions of the fascia in any of these muscles could have an effect on the fascia that surrounds the posterior compartment. The mobilizing of these muscles could thus release the stress in the fascia surrounding the posterior compartment. As these *clinically significant* muscles are all linked to each other and ultimately to the muscles of the posterior compartment of the lower leg via the myofascial web, the release of restrictions of the fascia in other muscles external to the calf-area, could normalize the length of the myofascial chain which could influence the root cause of the problem, namely stress in the surrounding fascia of the posterior compartment. The clinically significant muscles thus formed the basis for the soft tissue assessment and treatment of the next three subjects.

Posterior Myofascial Links

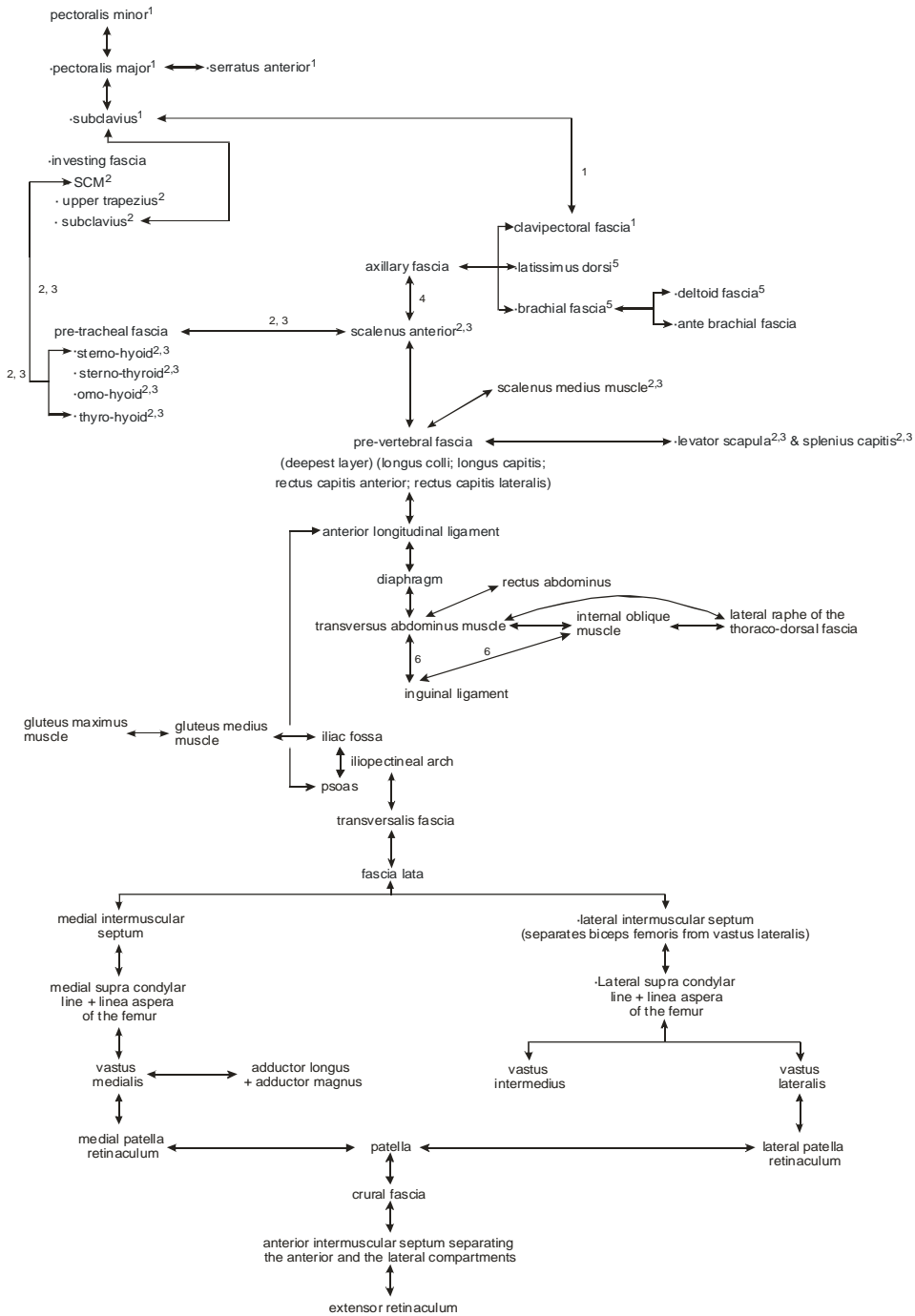


References:

- <sup>1</sup>Lee D. 1996: The pelvic girdle: An approach to the examination and treatment of the lumbo-pelvic-hip region, 2nd Ed., Churchill Livingstone, New York
- <sup>2</sup>Bogduk N.L.T. 1977: Clinical Anatomy of the Lumbar Spine and Sacrum, 3rd Ed., Churchill Livingstone, New York.
- <sup>3</sup>Luk et al. 1986: The ilio-lumbar ligament: a study of its anatomy, development and clinical significance. Journal of Bone and Joint surgery 68B:197
- <sup>4</sup>Vleeming et al. 1995a: The posterior layer of the thoracolumbar fascia. Its function in load transfer from spine to legs. Spine 20 (7): 753-8, 1995 Apr 1
- <sup>5</sup>Willard F.M. 1997: The muscular, ligamentous and neural structure of the low back and its relation to back pain in: Vleeming A, Mooney V, Dorman T, Snijdes C, Stoecart R (eds.) Movement, stability and low back pain. Churchill Livingstone, Edinburgh.
- <sup>6</sup>Farfan 1978. The biomechanical advantage of lordosis and hip extension for upright activity. Spine 3: 336
- <sup>7</sup>Vleeming A 1996: The function of the long dorsal sacroiliac ligament: its implication for understanding low back pain. Spine 21(5): 556 -62.March 1
- <sup>8</sup>Romanes G.J. 1981: Cunningham's Textbook of Anatomy, 12th Ed., Oxford University Press.
- <sup>9</sup>Clemente C.D. 1996: Anatomy: A regional atlas of the human body, 4th Ed., Williams and Wilkins.
- <sup>10</sup>Lockhart R.D., Hamilton G.F. and Fyfe F.W. 1974: Anatomy of the human body, Faber and Faber, London.

Figure 4.5: A summary of the different posterior myofascial links

Anterior Myofascial Links



References

- <sup>1</sup>Lockhart R.D., Hamilton G.F. and Fyfe F.W. 1974: Anatomy of the human body, Faber and Faber, London.
- <sup>2</sup>Romanes G.J. 1981: Cunningham's Textbook of Anatomy, 12th Ed., Oxford University Press.
- <sup>3</sup>Clemente C.D. 1996: Anatomy: A regional atlas of the human body, 4th Ed., Williams and Wilkins.
- <sup>4</sup>McMinn R.M.H. 1995: A color atlas of head and neck anatomy, Mosby-Wolfe, London.
- <sup>5</sup>Frick H., Leonhardt H. and Starck D. 1991: Human anatomy 1: General anatomy, special anatomy: Limbs, trunk wall, head and neck, Thiemo Medical Publishers
- <sup>6</sup>Rizk 1980. A new description of the anterior abdominal wall in man and mammals. Journal of Anatomy 131:373

Figure 4.6: A summary of the different anterior myofascial links

**Table 4.13: Clinically significant muscles**

<u><i>Posterior fascia links</i></u>	<u><i>Anterior fascia links</i></u>
<i>Levator scapula</i>	<i>Sternocleidomastoid</i>
<i>Trapezius</i>	<i>Scalenii</i>
<i>Latissimus dorsi</i>	<i>Pectoralis major</i>
<i>Erector spinae</i>	<i>Pectoralis minor</i>
<i>Quadratus lumborum</i>	<i>Serratus anterior</i>
<i>Gluteus medius</i>	<i>Subscapularis</i>
<i>Gluteus maximus</i>	<i>External oblique</i>
<i>Piriformis</i>	<i>Rectus abdominus</i>
<i>Semimembranosus</i>	<i>Psoas- umbilicus head</i>
<i>Semitendinosus</i>	<i>Psoas- iliac head</i>
<i>Biceps femoris</i>	<i>Psoas- groin</i>
<i>Gastrocnemius</i>	<i>Iliotibial band</i>
<i>Soleus</i>	<i>Vastus lateralis</i>
<i>Plantar fascia</i>	<i>Vastus medialis</i>
	<i>Adductor longus</i>
	<i>Adductor magnus</i>
	<i>Pes anserinus</i>

#### **4.3.4. Conclusion**

In the first literature review it was established that fascia exposed to physical trauma, scarring or inflammation loses its pliability. Any restriction somewhere along the myofascial chain will cause a decrease in the effective length of the myofascial chain. Such a restriction in length will induce stresses in the web during activities which require extended movement patterns such as running. These stresses will be transmitted via the inelastic myofascial web to areas such as the posterior compartment where it will induce pressure in the posterior compartment. During running, the additional forces exerted on an already compromised myofascial chain, will cause micro trauma and inflammation. The fascia will then become tight, restricted and a source of tension to the rest of the body due to the continuous nature of the myofascial web that links muscle and ligaments of various parts of the body with each other. It is therefore conceivable that a



restriction or tightness in any of the more proximal muscles linked to that of the posterior compartment might contribute to the stresses in the fascia which will lead to associated pressure in the compartment.

#### **4.4. THE REVISED THEORETICAL FRAMEWORK**

##### **4.4.1. Introduction**

The role of a theoretical model that reflects causal relationships is vital to qualitative research (Yin, 2003). He proclaims that theory development as part of the design phase is essential. The complete research design embodies a “theory” of what is being studied and the goal is to have some theoretical basis for formulating theoretical propositions that are to be tested by means of experimentation.

With the initial exploratory research the interventions which were primarily based on the current perspectives of CPCS, did not lead to any successes. This resulted in an additional literature study which was directed to the characteristics and properties of fascia. Although the role that fascia plays in creating pressure within the enclosed compartment is acknowledged in the current theory, the nature and characteristics of this variable are not explored.

This chapter reviews the current theoretical framework for CPCS and highlights the gap that exists. A modified model is proposed which provides a more credible basis for causal relationships of the condition.

##### **4.4.2. The current theoretical model**

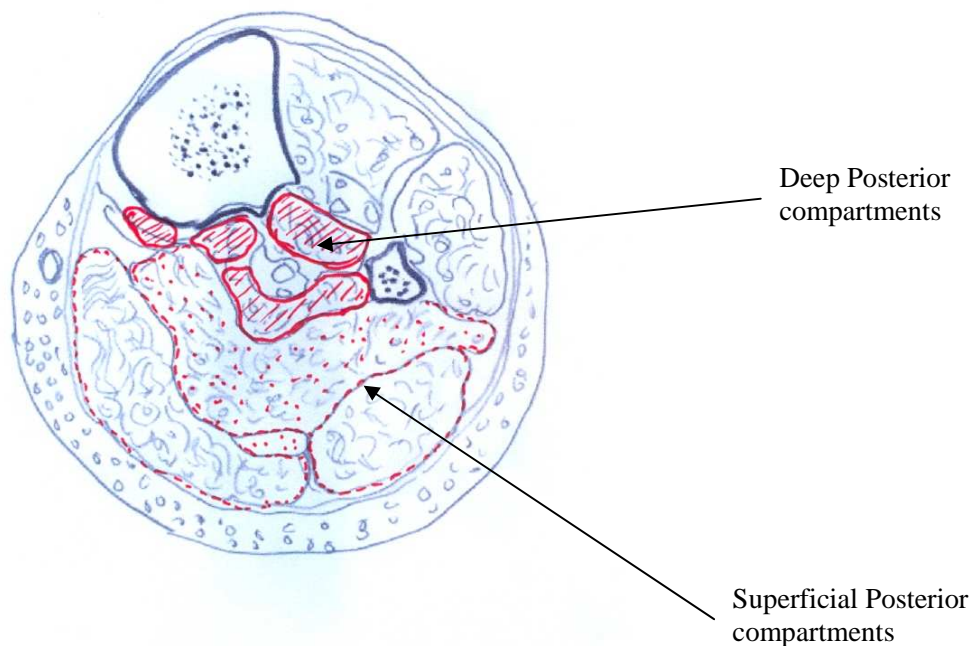
In order to create a broader contextual framework for a discussion of the current model, some of the viewpoints as emerged during the literature research in chapter 2 will be briefly recaptured. The following reflect the wide acceptance of the model, or at least elements which are encapsulated in the model:



- With repeated contraction, an exercising muscle can increase its volume by 20% (Bourne & Rorabeck, 1989; Eisele & Sammarco, 1993);
- If this occurs with compartment surrounded by a non-compliant fascia envelope (Clanton & Solcher, 1994; Nicholas & Herschman, 1995b), compartment pressures are expected to increase;
- This increase in pressure impedes blood flow and produces ischemic pain (Nicholas & Herschman, 1995b). The elevated compartment pressure increases pressure within intra-compartmental veins which reduces blood flow within the compartment;
- The circulation and function of the muscles and the neurovascular components are compromised in the closed anatomical space of the posterior compartment of the lower leg (Reneman, 1975);
- People presenting with the syndrome are predominantly runners (Detmer *et al.*, 1985);
- The propagation that the whole cycle is initiated by exertional muscle activity (Clanton & Solcher, 1994), and
- The role of fascia in perpetuating the condition has been adequately demonstrated with the relative success of a fasciotomy or a fasciectomy (Howard *et al.*, 2000; Slimmon *et al.*, 2002; Turnipseed, 2002; Shah *et al.*, 2004).

This model proposes that symptoms are caused by exertional muscle exercise, creating abnormally high intramuscular pressure as the result of non-compliant fascial borders. It is suggested that this high pressure occurs during exercise or shortly thereafter. The syndrome then allows several deleterious processes to develop. Circulation to the microvasculature is impeded, and the metabolic demands of the intra-compartmental musculature are compromised. Pressure is then thought to develop secondary to increased intra- and extracellular fluid accumulation within a non-compliant fascial space. There is often venous and lymphatic compromise that contributes to the vicious cycle of increasing tissue pressure resulting in further vascular compromise.





**Figure 4.7: Posterior compartments**

#### 4.4.3. The theoretical gap

There is however one question that vividly reflects the lack of a comprehensive understanding of the condition, namely:

*“Why do only a selected few runners develop this syndrome, if it is caused by muscle exertional exercises?”*

In order to answer this question it is necessary to review the shortcomings of the current theoretical base. The glaring shortcoming which emerged from the literature review is the fact that the nature and the characteristics of fascia have largely been ignored in the theoretical approach towards the syndrome. The current model proposes that symptoms are caused by *exertional muscle exercise*, creating abnormally high intramuscular pressure as the result of non-compliant fascial borders. It is suggested that this high pressure occurs during exercise or shortly thereafter. The syndrome then allows several



deleterious processes to develop. Circulation to the microvasculature is impeded, and the metabolic demands of the intra-compartmental musculature are compromised. Pressure is then thought to develop secondary to increased intra- and extracellular fluid accumulation within a *non-compliant fascial space*. There is often venous and lymphatic compromise that contributes to the vicious cycle of increasing tissue pressure resulting in further vascular compromise. The role of the non-compliant fascial space has been adequately demonstrated by the success of a fasciotomy or a fasciectomy (Turnipseed, 2002; Shah *et al.*, 2004).

The one aspect of the model that has however not been verified is the assumption that the condition is triggered by muscle exertion. In the revised or modified model that is presented in the next paragraph, a more credible rationale for the development of CPCS is presented.

#### **4.4.4. A revised theoretical model for CPCS**

The fact that non-compliant fascia, surrounding muscle compartments, play a crucial role in CPCS is widely accepted. This is also illustrated by the fact that the only successful treatment to date, results from the surgical release of the involved fascia (Froneck *et al.*, 1987; Melberg & Styf, 1989; Biedert & Marti, 1997). The surgical release leads to a decrease in the intra-compartmental pressure which in turn leads to the restoration of circulation and the decrease of symptoms.

The long term result of this approach is however not successful, since symptoms often re-occur shortly after the athlete resumes training (Shah *et al.*, 2004). The fact that running re-induces this condition implies that the root cause for the condition has not been addressed. From the literature review it became evident that the continuous nature and non-elasticity of fascia have been ignored in the theoretical deliberation of the condition. The characteristics of fascia as an ‘integrated continuum’ suggest that the cause of the symptoms of CPCS might lie somewhere outside the enclosure of the posterior compartment. With the continuous nature of the fascia and its relative inelasticity it seem logical to assume that a restriction anywhere along the myofascial chain could influence

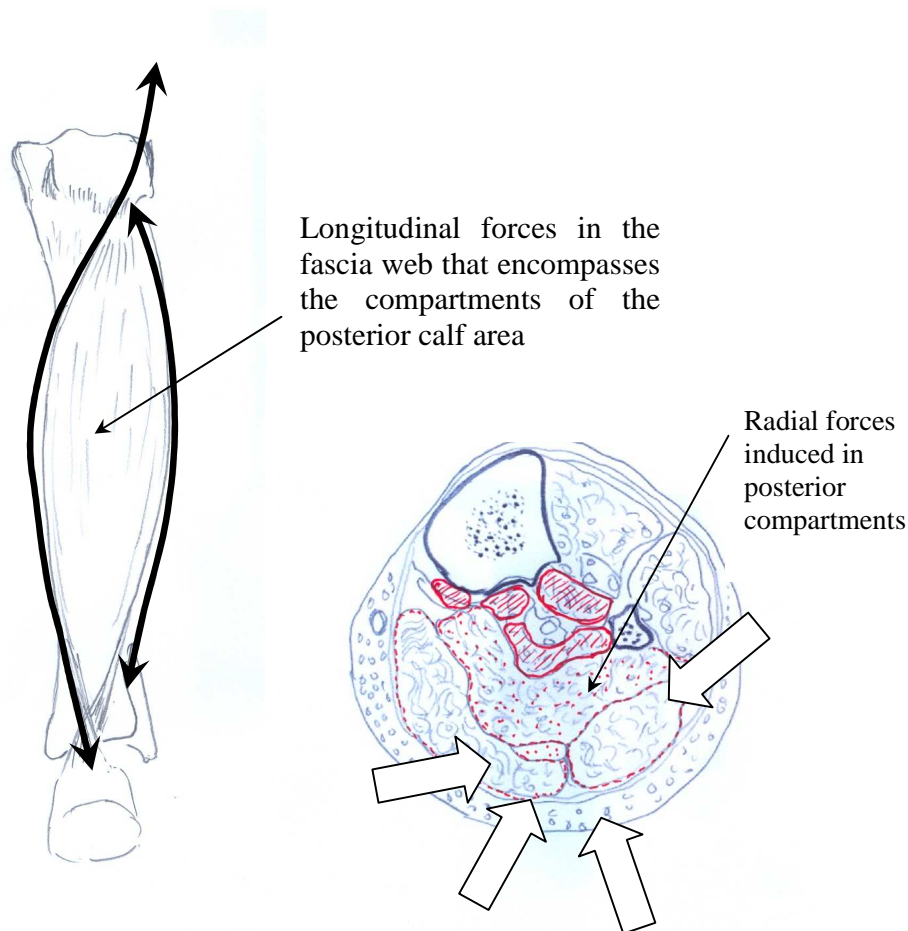


an area remotely distant from the source of the restriction. To illustrate this argument, the analogy of an insect in a spider web can be used. The entangled insect will induce forces along the links, destroying the symmetry of the web. If the spider web was inelastic, the length in the remaining part of the web will also be affected. Likewise any restrictions in the myofascial chain will induce longitudinal stresses in the myofascial web. This will be especially relevant during exercises where extended movement ranges are required during activities such as running. Such restrictions of the fascia will decrease the available fascial length which will prevent the myofascial chain from working optimally. These induced stresses in the fascial web will be transmitted along the chain and can be reflected at any point in the continuous web.

The fact that the surgical release of the involved fascia reduces the pressure in the posterior compartment to such an extent that circulation is restored, supports the deduction that the induced intra-compartmental pressure caused by stresses in the fascial web could be sufficient to trigger the condition in the first place. The fact that the condition reoccurs after surgical release once the subject starts to run again, implies that running somehow triggers the condition again. In other words the running creates a process that interferes with the microcirculation to the extent that the symptoms reoccur. This implies that sufficient pressure has been induced in the posterior compartment to impede the microcirculation again.

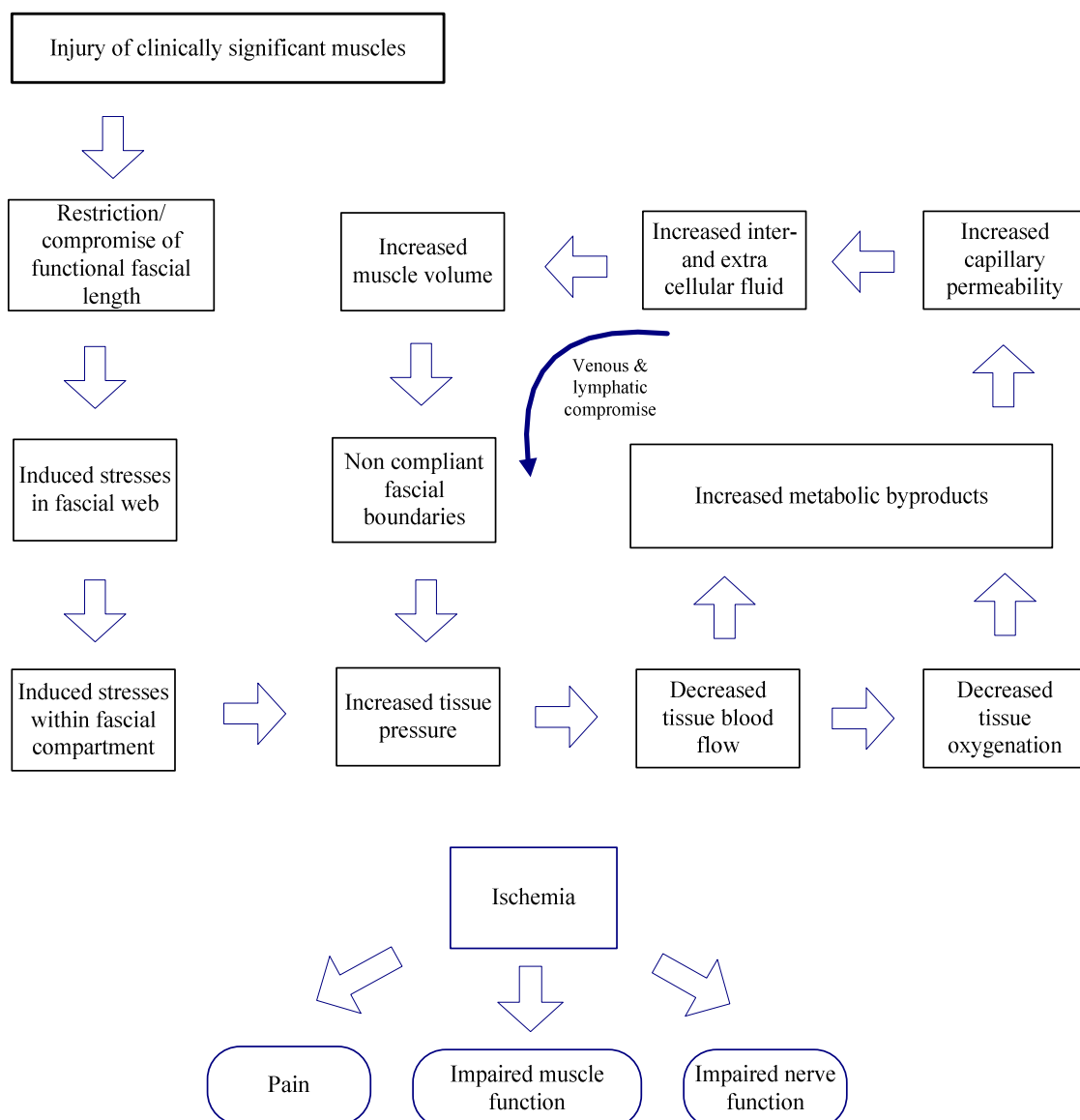
A critical analysis of the running activity reveals that during running the stresses in the fascial web will be increased due to an increased range of movement and dynamic forces induced by the weight bearing nature of the running exercise. It is thus argued that the pressure induced in the posterior compartment as a result of running following surgical release is sufficient to increase the intra-compartmental pressure to such an extent that the symptoms recur. It is also argued that if the induced stresses caused by running in these subjects are sufficient to impede microcirculation, the same argument can be applied to subjects with a compromised fascial length. The pressure that is induced in the posterior compartment could be sufficient to impede the micro circulation. This in turn creates the currently accepted conditions for the development of the symptoms of CPCS as proposed

by Clanton & Solcher (1994). In the previous chapter the continuous nature of fascia has been explored which led to the identification and classification of the “significant muscles”. It is postulated that a restriction or tightness in any of these proximal muscles that are linked to the posterior compartment might have an effect on the fascia of the posterior compartment. In exercises such as running the range of movements are more accentuated and a greater degree of stresses are therefore induced in the fascia. These longitudinal forces in the fascia web that surrounds the muscle compartments induce abnormal high intramuscular pressure within the compartments. The pressure is thus not only as the result of non-compliant fascial borders, but also due to the fact that these fascial borders create the initial conditions for the development of the said deleterious processes. The proposed longitudinal forces in the myofascial web which could be responsible for the initiation of CPCS are depicted in Figure 4.8.



**Figure 4.8: Induced stresses in posterior compartments**

It is proposed that the injury and inflammation of any of the clinically significant muscles could compromise the myofascial web, which in turn could lead to the initiation of CPCS through the involvement in weight bearing exercises such as running. The proposed modified model for the pathogenesis of CPCS is depicted in Figure 4.9. It should be noted that the only modification to the current model is the initiating conditions. The root cause thus in all probability lies external to the posterior compartment.



**Figure 4.9: Modified model for the pathogenesis of CPCS**



#### **4.4.5. Rationale for the treatment approach**

If the proposed model or framework is a fair reflection of the pathogenesis of CPCS, a number of deductions should be valid. Any distortions in the fascial web should be evident from tightness in the ‘clinically significant’ muscles.

Stresses in the fascial web and a compromised myofascial chain should also lead to altered movement patterns and biomechanical measures. These should be evident during exercises such as running, especially where extended or full ranges of movement are required.

Similarly it can be argued that if the root cause for the condition is indeed determined by injuries in the clinically significant muscles. The alleviation of the condition will also lead to the normalisation of such movement patterns and biomechanics.

#### **4.4.6. Conclusion**

The problem with the current model for the pathogenesis of CPCS of Clanton & Solcher (1994), is that based on clinical experience, exertion of muscle activity does not lead to CPCS in the majority of athletes. Exertional muscle activity is thus not solely responsible for the precipitation of symptoms of CPCS.

It was hypothesized that it is probable that the cause of the symptoms of CPCS might lie somewhere outside the involved compartment. This would imply tightness in the clinically significant muscles as far proximally as where the restriction in the myofascial web lies. Due to the fact that such restrictions might lie anywhere along the continuum, it can be expected that the patterns of associated tightness in the ‘clinically significant’ muscles would in all probability differ from subject to subject. This would imply that the patterns of muscle tightness of the subjects will differ from subject to subject, depending on where the restriction in the web lies.



It could also be argued that tightness in the soft tissue would lead to altered running movement patterns and altered biomechanics. It is also possible that if the mobilization of the 'clinically significant' muscles leads to the clearance of the symptoms of CPCS, the restoration of the functional length of the fascial web will also lead to an improvement in running patterns and biomechanics.

## **4.5. EXPLANATORY RESEARCH RESULTS**

### **4.5.1. Introduction**

This section deals with the results of case studies 4 to 6 of the explanatory research phase. The objective of the research is primarily the validation of the model for the pathogenesis of CPCS as developed towards the end of the exploratory research phase. A number of quantitative measurements were done during the explanatory research phase. The assessments of these results will also be covered in group context.

Case Study 4 is the first of the case studies dealing with the validation of the theoretical model as discussed in the previous section. To some extent it also reflects the transition from the exploratory to the explanatory. In the initial interventions these have not been fully developed and only in the latter part of the interventions were the theory fully applied. In terms of this model, the basic proposition is that the cause of the condition lies outside the posterior compartment and that the mobilization of soft tissue (myofascial tissue) of the clinical significant muscles involved will lead to a disappearance in symptoms. This proposition relies to a large extent on the existing knowledge base as reflected by the model for the pathogenesis of CPCS (Clanton & Solcher, 1994) and supplemented by the findings in the literature research. In terms of this knowledge (Reneman, 1975; Detmer *et al.*, 1985; Clanton & Solcher, 1994; Nicholas & Herschman, 1995a; Howard *et al.*, 2000; Turnipseed, 2002; Slimmon *et al.*, 2002; Shah *et al.*, 2004), healing is effected by a reduction in the pressure in the posterior compartment. These claims are adequately demonstrated by the success of the release of pressure by means of fasciectomy which increases tissue blood flow and associated oxygenation which reverse the process.





With regard to the measures employed during this research, none have the sophistication to actually measure the pressure within the posterior compartment, nor the increase in tissue blood flow. The measurement of pressure in the posterior compartment is based on the palpation findings which to some extent is a qualitative measure. Some degree of reliance is thus placed on the current understanding of the healing process as was demonstrated during the literature research with the surgical release of the fascia.

### ***The propositions***

#### ***Main proposition***

*The main proposition of this explanatory phase of the research is that the root cause of CPCS lies outside the posterior compartment-compartment and this manifests through tightness in the clinically significant muscles. The mobilization of the soft tissue (myofascial tissue) of these “clinical significant muscles” will lead to a disappearance in symptoms of CPCS through:*

- *a reduction in the pressure in calf area which in turn will lead to the alleviation of the symptoms of CPCS; and/or*
- *an increase in tissue blood flow and associated oxygenation that will reverse the process.*

#### ***Secondary propositions***

*As a result of the continuous nature of the fascia and its relatively inelasticity the condition will reflect in a number of movement abnormalities which will be restored once the condition of CPCS has been eliminated.*

In terms of these propositions, the main proposition and its associated rivalry theories will be discussed on a case-by-case basis.





#### 4.5.2. CASE STUDY 4

##### 4.5.2.1. *The subject*

The subject in Case Study 4 was a 24 year old male athlete who had been forced by pain to focus on ten kilometre races and duathlon events.

##### *Inclusion criteria*

- He experienced constant discomfort in both Calves, over the posterior-medial aspect in the middle one third of both lower legs. His discomfort rated as a 25 on the 100mm VAS. This discomfort steadily increased to reach a pain level of 75 as indicated on a 100mm VAS, at the end of a seven kilometre run.
- He was diagnosed with CPCS after intra-compartmental pressure measurements were taken by an orthopaedic surgeon at the beginning of 1996.

##### *Exclusion criteria*

With regard to the exclusion of vascular conditions:

- He already had two popliteal artery entrapment release operations (November 1994), one in each leg.

##### 4.5.2.2. *Research Procedure*

##### *Subjective assessment – Interview*

- *Running history*

This 24-year old male subject had been a competitive athlete since high school (1994). He participated in 800 m and 1500 m track events. During recent years, he had been forced by injuries to focus on ten kilometre races and duathlon events. These consist of



standard and mini duathlon. The duathlon consists of a ten kilometre run followed by 40 kilometres cycling, and then a five kilometre run; while the mini duathlon consists of a five kilometre run followed by a 30 kilometre cycling, and then a five kilometre run. At the time of his inclusion into the study, he managed to run a distance of seven kilometres twice a week, providing that he had a period of at least two days rest in between. He ran at a pace of four-and-a-half minutes per kilometre. He increased his fitness by following a cross-training programme. He cycled a distance of 55 to 120 kilometres twice a week and also attended a gymnasium for weight training three times a week. At the time of inclusion into the study, he was running with strong motion control shoes. His hind foot pronation was 20° on the left and 18° on the right as measured by a podiatrist in the early half of 2002. He used different shoes (clip-on) for cycling.

○ ***Previous running injuries***

Previous injuries that he had encountered included:

- A right-sided iliotibial band syndrome;
- Runner's knee on the right side; and
- Mid thoracic pain during cycling.

○ ***Current symptoms***

He experienced a constant discomfort in both calves, over the posterior-medial aspect in the middle one-third of both lower legs. He rated this constant discomfort as a 25 on the 100mm VAS. This discomfort steadily increased to 75 as indicated on a 100mm VAS towards the end of a seven kilometre run. He ran in the late afternoons and the intensity of pain would remain at that level for a couple of hours. If he attempted to run more than twice a week without the break, the pain would remain at a level of 75 on a 100mm VAS for a period of two to three days thereafter. With his entrance into the study, he was averaging a running distance of 14 kilometres per week.



○ *History of symptoms and previous treatment received*

He first experienced symptoms during 1994 whilst he was participating in 800 m and 1500 m track events. He described the symptoms as deep sharp pains behind the posterior-medial aspect of both tibias. At times, he also experienced a numb sensation or a sensation of pins and needles in both feet. His symptoms commenced within ten to 20 minutes after the commencement of the training session. The pain would then continue for a couple of hours after the cessation of the exercise session. He also complained of a sensation of weakness in his calf muscles.

When the symptoms appeared for the first time, he received conventional physiotherapy treatment that consisted of ultrasound, interferential, massage and stretch exercises, all aimed locally at the area of the calf muscles. He also underwent a calf muscle strengthening programme on the Isokinetic dynamometer. Despite this treatment, his symptoms remained unchanged. He was then referred for further investigations which included: a bone scan, a Doppler test and an arteriogram. The results of the bone scan and the Doppler test were negative, but the arteriogram was indicative of a bilateral popliteal entrapment syndrome with a stenosis developing in the popliteal artery.

During November 1994, he underwent a bilateral popliteal entrapment release. Post-operatively, he started to train gradually, but his symptoms re-appeared two to three weeks after the commencement of his training programme. Intra-compartmental pressure measurements were taken. The pressure was found to be raised significantly enough to be indicative of posterior compartment syndrome. A bilateral posterior compartment release was done during April 1996. The symptoms again re-appeared two to three weeks after he had started to train, the only difference being a decrease in the frequency of pins and needles in the feet. He again received three to four sessions of physiotherapy consisting of ultrasound, interferential, massage and stretch exercises over the area of the lower leg, which once more had no effect on his symptoms.

During May 1998, he underwent a revised surgical posterior compartment release (bilaterally). Thereafter he experienced no symptoms with cycling, but with running, his



symptoms became progressively worse, despite the fact that he was receiving conventional physiotherapy for his calf muscles on a daily basis. The physiotherapy consisted of massage, ultrasound- and interferential treatment over the area of the calf muscles, calf muscle stretches and calf muscle strengthening exercises. The latter consisted of both concentric as well as eccentric exercises.

During the latter half of 2001, he reached the stage where he had constant pain and a sensation of weakness in his calf muscles. He was again referred for further tests of the lower legs. These tests included ultrasonic imaging, an arteriogram, an electromyographic test (EMG) as well as magnetic resonance imaging (MRI). The test results were normal.

In summary, the subjective outcome measurements as measured before the intervention:

- The intensity of pain / discomfort at rest was 25 on the 100mm VAS and 75 on the 100mm VAS after a seven kilometre run.
- He averaged a distance of 14 kilometres per week at a pace of four and a half minutes per kilometre.

### ***Objective assessment – Physical examination***

#### ***○ Muscle strength tests***

Muscle strength was assessed by a biokineticist on a calibrated Isokinetic dynamometer, using a standardized testing protocol (Date: 22/11/2002). The subject presented with several significant differences in muscle strength between the left and the right sides as reflected in Table 4.14.

**Table 4.14: Isokinetic dynamometer test results\* prior to intervention: Case Study 4.**

<i>Movement tested**</i>	<i>Peak torque (Nm)</i>			<i>Work per repetition (Nm/s)</i>		
	<i>Right</i>	<i>Left</i>	<i>Deficit</i>	<i>Right</i>	<i>Left</i>	<i>Deficit</i>
<i>Hip extensors</i>	175	152	<i>13</i>	223	183	<i>18</i>
<i>Hip flexors</i>	92	110	<i>-16</i>	108	117	-8
<i>Hip internal rotators</i>	28	21	<i>25</i>	31	19	<i>39</i>
<i>Hip external rotators</i>	26	20	<i>23</i>	28	19	<i>32</i>
<i>Hip abduction</i>	104	121	<i>-14</i>	55	59	-7
<i>Hip adduction</i>	120	176	<i>-32</i>	69	91	<i>-24</i>
<i>Knee extensors</i>	192	173	<i>10</i>	194	176	9
<i>Knee flexors</i>	107	100	7	130	132	-2
<i>Knee internal rotation</i>	34	35	-4	33	33	0
<i>Knee external rotation</i>	26	27	-5	30	31	-4
<i>Ankle inversion</i>	33	32	3	27	26	4
<i>Ankle eversion</i>	21	21	0	23	17	<i>26</i>
<i>Ankle plantar flexors</i>	85	79	7	47	47	0
<i>Ankle dorsi flexors</i>	34	36	-6	21	20	5

\*Statistically significant differences between the right and the left sides are given in italic

\*\* Tested at a speed of 30/30 and with five repetitions

○ ***Analysis of running gait, including movement patterns***

The following movement patterns deviated from the normal/ ideal running patterns (see Table 4.15 for full description):

- The right shoulder moved into more extension and retraction than the left shoulder. It was swinging more than the left. It was also more depressed than the left.
- There was more thoracic rotation towards the left compared to the right side.
- The pelvis on the right side moved into anterior rotation during initial contact whilst the pelvis on the left only moved into a position of neutral. During the single leg support phase, the left pelvis dropped more in comparison to the right.

**Table 4.15 (a): Running gait analysis: Case Study 4 prior to intervention (Upper body)**

<i>Running gait analysis: Case Study 4 (28/01/2003)</i>								
<i>Joint</i>	<i>Weight acceptance</i>		<i>Single leg support</i>			<i>Swing leg advancement</i>		
	<i>Initial contact</i>	<i>Loading response</i>	<i>Mid-stance</i>	<i>Terminal stance</i>	<i>Pre swing</i>	<i>Initial swing</i>	<i>Mid-swing</i>	<i>Terminal swing</i>
Shoulders	Left leg: right shoulder in more extension and retraction than left shoulder with right leg initial contact. During this phase the right shoulder-blade is swinging more than the left and is also in more depression than the left.							
Thoracic region	There is more thoracic rotation to the left (initial contact left leg) than to the right (initial contact right leg).							
Pelvis	Right pelvis is in anterior rotation. The left pelvis is in a neutral position.		Both sides are in slight posterior rotation. The left side more so than the right side. The left side seems to drop more than the right side.	Both sides are in posterior rotation; left side more than right side.	Both sides are in posterior rotation; left side more than right side.		Both sides move out of posterior rotation.	The left side moves into a neutral pelvic position and the right side moves into anterior pelvic rotation.
Hip	Right hip: flexion plus slight adduction. Left hip: flexion plus slight external rotation.		The right hip is in neutral. The left hip is in slight abduction.  The subject has an uneven gait. He seems to have more of a push-off action on the right side and a landing action on the left side.	Both hips are in extension and external rotation. The left hip is in more external rotation than the right.	Both are in extension and external rotation. The left side is in more external rotation than the right side.	Both hips start to move into internal rotation from the externally rotated position.	Both hips: Flexion and internal rotation.	Both hips: Flexion.

**Table 4.15 (b): Running gait analysis: Case Study 4 prior to intervention (Lower body)**

<i>Running gait analysis: Case Study 4 (28/01/2003)</i>								
<i>Joint</i>	<i>Weight acceptance</i>		<i>Single leg support</i>			<i>Swing leg advancement</i>		
	<i>Initial contact</i>	<i>Loading response</i>	<i>Mid-stance</i>	<i>Terminal stance</i>	<i>Pre swing</i>	<i>Initial swing</i>	<i>Mid-swing</i>	<i>Terminal swing</i>
Knee	Both knees: almost full extension with the tibia in external rotation.	The amount of knee extension decreases slightly in both knees.	The amount of extension in both knees increase a bit again with the tibia being in external rotation.	The tibias move into more external rotation with the knees in extension.	Both knees are extended with a degree of external rotation of the tibias.	Both knees move out of flexion and external rotation into flexion. There is a frequent irregularity on the left side with an increased amount of left hip external rotation and posterior pelvic rotation.	Both sides: Flexion and internal rotation.	Both sides: Extension and external rotation.
Ankle	Right ankle: no heel –toe action. Lands with the ankle in a neutral position, fractionally first on the lateral side of the foot.  Left ankle: lands in a slightly inverted position; laterally on the 5 <sup>th</sup> metatarsal. No heel-toe action.	Left and right sides: shift the weight to the medial side of the foot (moves into a greater everted position).	The hind foot is in pronation. The left and the right calcaneus sink further into valgus (loss of eccentric control). This occurs more on the left than the right. Both feet are in abduction.	The hind foot moves out of the pronated position into neutral. The right calcaneus starts to move earlier than the left one. The ankle moves into plantar flexion.	Left and right sides are in a neutral hind foot position with plantar flexion.	There is no real push-off. It is initiated (more so on the left than on the right) by a backward rotation of the pelvis, hip extension and external rotation. With the push-off the weight is shifted from the 2 <sup>nd</sup> to the 3 <sup>rd</sup> metatarsal head so that the foot moves into inversion. It returns to the position of the 2 <sup>nd</sup> metatarsal head as soon as the weight is off. Less subtle on the right side than on the left side.	Left and right sides: slight inversion and plantar flexion.	Left and right sides: slight inversion and plantar flexion.
Toes	Excessive MTP extension, especially of the big toes.	Less extension.	Toes in neutral position.	Toes move into extension.	Extension.	Extension.	Extension.	More extension.

- The left hip was in slight abduction in comparison to the right side. It was almost as though the subject had an uneven push-off (right side) and landing (left side) gait. The hip on the left side was throughout in a position of increased lateral rotation.
  - There was no heel-toe action and during the mid stance phase, the calcaneus on both sides, sank into an increased degree of valgus. This was more noticeable on the left side. Both feet were in a position of abduction. During the terminal stance phase, the right calcaneus started to move earlier out of the valgus position in comparison to the left side.
  - There was no push-off. There was again an irregularity noticeable when the weight was shifted from the 2<sup>nd</sup> to the 3<sup>rd</sup> metatarsal heads during the phase of terminal stance. This was less subtle on the right than on the left.
- ***Flexibility/ length of soleus muscle ( 28/01/2003)***

Right soleus muscle: Big toe 120 mm from the wall.

Left soleus muscle: Big toe 140 mm from the wall.

- ***Biomechanical angles ( 28/01/2003)***

The biomechanical angle measurements are provided in Table 4.16.

**Table 4.16: Biomechanical angles: Case Study 4.**

<i>Movement</i>	<i>Joint Range of Movement (degrees)*</i>	
	<i>Mid stance</i>	<i>Terminal stance</i>
<i>Dorsi flexion left</i>	9, 33°	19, 00°
<i>Dorsi flexion right</i>	5, 67°	20, 75°
<i>Metatarsophalangeal extension left</i>		50, 67°
<i>Metatarsophalangeal extension right</i>		48, 50°
<i>Hind foot pronation left</i>	20, 00	
<i>Hind foot pronation right</i>	18, 00	

\* Averages of three measurements



**Table 4.17: Tightness of clinically significant muscles: Case Study 4 prior to intervention: (Date: 28/01/2003)**

<i>Muscle</i>	<i>Left Side</i>			<i>Right Side</i>			<i>Muscle</i>	<i>Left Side</i>			<i>Right Side</i>		
	<i>0</i>	<i>1</i>	<i>2</i>	<i>0</i>	<i>1</i>	<i>2</i>		<i>0</i>	<i>1</i>	<i>2</i>	<i>0</i>	<i>1</i>	<i>2</i>
<u><i>Posterior fascia links</i></u>							<u><i>Anterior fascia links</i></u>						
<i>Levator scapula</i>		1				2	<i>Sternocleidomastoid</i>		1				2
<i>Trapezius</i>		1				2	<i>Scalenii</i>		1				2
<i>Latissimus dorsi</i>		1				2	<i>Pectoralis major</i>		1				2
<i>Erector spinae</i>		1				2	<i>Pectoralis minor</i>			2		1	
<i>Quadratus lumborum</i>			2			2	<i>Serratus anterior</i>		1				2
<i>Gluteus medius</i>		1				2	<i>Subscapularis</i>		1				2
<i>Gluteus maximus</i>			2			2	<i>External oblique</i>		1				2
<i>Piriformis</i>			2			2	<i>Rectus abdominus</i>			2			2
<i>Semi-membranosus</i>		1				2	<i>Psoas- umbilicus head</i>		1				2
<i>Semitendinosus</i>		1			1		<i>Psoas- iliac head</i>		1				2
<i>Biceps femoris</i>			2			2	<i>Psoas- groin</i>		1				2
<i>Gastrocnemius</i>			2			2	<i>Iliotibial band</i>		1				2
<i>Soleus</i>			2			2	<i>Vastus lateralis</i>	0				1	
<i>Plantar fascia</i>		1			1		<i>Vastus medialis</i>	0				1	
							<i>Adductor longus</i>	0				1	
							<i>Adductor magnus</i>	0				1	
							<i>Pes anserinus</i>			2			2

Key: 0= normal fascia

1= tight fascia

2=very tight fascia



○ *Soft tissue palpation (clinically significant muscles)*

All the clinically significant muscles were palpated for tightness and spasm and rated on a scale from 0 to 2. These outcomes are reflected in Table 4.17.

**4.5.2.3. Interventions**

All the clinically significant muscles with a relative tightness rating of one or two were mobilized. Different soft tissue mobilising techniques were used, depending on the muscle involved. Myofascial release techniques were applied according to the approach of Barnes (1990) and Manheim (1994). Trigger point release techniques were applied according to the approach of Travell & Simons (1999) and specific soft tissue mobilization was applied according to Hunter's approach (1998).

The subject was seen twice a week during the first two weeks. Thereafter the frequency decreased to once every seven to 14 days for a period of one year. The objective was an intervention period of every ten days but the schedule was influenced by external commitments of the subject. He was instructed to stretch on a daily basis. Stretches were provided for the following muscles:

- the trapezius
- the levator scapula
- the pectoralis
- the abdominal muscles
- the iliopsoas
- the piriformis
- the hamstrings
- the gastrocnemius and
- the soleus muscles

He was instructed to hold each stretch for 30 seconds and to repeat it.



*Clinical observations during the intervention period:*

- During the first half of the intervention period (23/08/02 – 25/10/03), on eight different occasions there were so many tight clinically significant muscles that it was impossible to mobilize all effectively during the 45 minute intervention. During the 2<sup>nd</sup>, the 4<sup>th</sup>, the 6<sup>th</sup> and the 8<sup>th</sup> occasions, only the tight clinically significant muscles of the pelvis and the area above the pelvis were mobilised. During the 1<sup>st</sup>, the 3<sup>rd</sup>, the 5<sup>th</sup> and the 7<sup>th</sup> occasions only the tight clinically significant muscles of the legs were mobilized. The differences in the subjective outcome measurements between these two were then compared. In the week following the mobilization of the legs, there was no improvement in the measures (i.e. pain as indicated on a 100mm VAS; the distance run before the commencement of the pain; or the total weekly distance run). In the week following the intervention where the clinically significant muscles of the pelvis area and above were mobilized, a noticeable improvement was recorded in terms of all three the measures. This was an indication that the release of the more proximal tight myofascial links played a very important role in the amelioration of the symptoms of CPCS in the fourth subject.
- As can be seen in the Figures 2.5 (posterior myofascial links), the superficial lamina of the posterior layer of the thoraco-dorsal fascia links both to the aponeurosis of the latissimus dorsi muscle and also to the lower fibres of trapezius muscle. Clinically, the fascia overlap between these two muscles could be felt bilaterally at the T8 spinous level. It could also be observed when the subject was asked to lift the arms up in elevation, adduction and lateral rotation while lying in prone. Specific soft tissue mobilizations as developed by Hunter (1998) in the area where the fascia of the two muscles overlap, led to the disappearance of the thoracic pain that he previously experienced every time that he cycled a distance of more than 70 km. This was not mentioned earlier since it did not occur while running and seemingly had no direct correlation with the symptoms of CPCS.
- The link between the semimembranosus, the medial tibial ligament and the adductor magnus is also reflected in Figure 4.5. Palpation of his right semimembranosus initially reproduced his right knee pain (so called “runners knee”) and the



mobilization of this muscle with Hunter's specific soft tissue mobilizations led to the disappearance of his right knee pain. In addition to this, it also improved the range of his straight leg raise.

- It was noted throughout the intervention period that his symptoms increased slightly, despite the interventions, whenever he had to spend more time sitting and studying; or when he trained on loose sea sand during the holiday periods. It seemed as though posture plays a definite role in the symptoms of CPCS. Whenever he spent more time in a seated position, slumping as during examination times, his symptoms increased. A plausible explanation for this is that the sitting and slumping positions probably places a bigger demand on the already compromised myofascial chain. As for the training on loose sea sand, a greater force is required for forwards propulsion in the loose sand which also requires a greater range of movement with associated increased stresses in the fascial chain.

- *Treatment period*

(23<sup>rd</sup> August 2002 – 25<sup>th</sup> October 2003)

***Final assessment results (Date: 25/10/2003)***

***Interview***

- The intensity of pain / discomfort at rest as well as the intensity of pain / discomfort at the end of every training session was plotted on a 100 mm VAS: he had no discomfort at rest (0 on the 100mm VAS). He experienced occasionally a discomfort of ten in the right calf muscle on the 100 mm visual analogue scale after 15 minutes of running; or at the end of a ten kilometre run; or once cooled down.
- He ran pain free most of the time.
- He ran a weekly distance of 20 kilometres.
- He ran at a pace of less than four minutes per kilometre.
- He received provincial colours for triathlon during 2004, for duathlon during 2005, and represented South Africa in duathlon during the 2005 world championships.

**Physical examination**

○ **Muscle strength tests**

The strength of the hip extensors, the hip flexors, the hip abductors, the hip adductors, the medial- and lateral rotators of the hip, the knee extensors, the knee flexors, the medial- and lateral rotators of the knee, the invertors and evertors of the ankle as well as the dorsi- and plantar flexors of the ankle were assessed by a biokineticist on a calibrated Isokinetic dynamometer, using a standardized testing protocol. The results of the tests as measured on 25/10/2003 are provided in Table 4.18.

**Table 4.18: Isokinetic dynamometer test results\* after intervention: Case Study 4.**

<i>Movement tested**</i>	<i>Peak torque (Nm)</i>			<i>Work per repetition (Nm/s)</i>		
	<i>Right</i>	<i>Left</i>	<i>Deficit</i>	<i>Right</i>	<i>Left</i>	<i>Deficit</i>
Hip extensors	133	155	<i>-14</i>	181	195	<i>-7</i>
Hip flexors	102	104	<i>-2</i>	140	117	<i>16</i>
Hip internal rotators	37	48	<i>-23</i>	34	68	<i>-50</i>
Hip external rotators	30	33	<i>-9</i>	37	41	<i>-10</i>
Hip abduction	129	135	<i>-4</i>	119	114	<i>4</i>
Hip adduction	104	139	<i>-25</i>	117	135	<i>-13</i>
Knee extensors	197	192	<i>3</i>	187	204	<i>-8</i>
Knee flexors	115	117	<i>-2</i>	142	178	<i>-20</i>
Knee internal rotation	***	***	***	***	***	***
Knee external rotation	***	***	***	***	***	***
Ankle inversion	58	50	<i>14</i>	74	49	<i>34</i>
Ankle eversion	34	33	<i>3</i>	53	37	<i>30</i>
Ankle plantar flexors	109	90	<i>17</i>	61	53	<i>13</i>
Ankle dorsi flexors	46	36	<i>22</i>	33	24	<i>27</i>

\*Statistically significant differences between right and left are given in italics

\*\*Tested at a speed of 30/30 and with 5 repetitions \*\*\* Not measured

○ **Reassessment of running gait and movement patterns**

Running gait and movement patterns were reassessed and the results are reflected in Table 4.19.

**Table 4.19: Running gait analysis: Case Study 4 after intervention (Date: 13/07/2003)**

<i>Running gait analysis: Case Study 4 (13/07/2003)</i>								
<i>Joint</i>	<i>Weight acceptance</i>		<i>Single leg support</i>			<i>Swing leg advancement</i>		
	<i>Initial contact</i>	<i>Loading response</i>	<i>Mid-stance</i>	<i>Terminal stance</i>	<i>Pre- swing</i>	<i>Initial swing</i>	<i>Mid-swing</i>	<i>Terminal swing</i>
Shoulders	Left leg: right shoulder in more extension and retraction than left shoulder with right leg initial contact, but less than on the 28/01/03.							
Thoracic region	Left pelvis in more anterior rotation than previously.							
Pelvis	Left pelvis in more anterior rotation than previously.		Pelvis on the left and right in neutral. Still tends to drop a bit on the left side.	The amount of posterior rotation of the pelvis is equal on the left and the right.	The amount of posterior rotation is equal on the left and the right.			The left side now also moves into anterior rotation.
Hip	Left and right sides are in flexion.							
Knee	All movements were normal.							
Ankle	All movements were normal.							
Toes	All movements were normal.							

○ ***Flexibility/ length of soleus muscle (25/10/2003)***

Right soleus muscle: Big toe 160 mm from the wall.

Left soleus muscle: Big toe 180 mm from the wall.

○ ***Biomechanical measurements***

The biomechanical measurements for subject 4 were done twice, the first measurement after six months and the second measurement after a further three months. This was due to the fact that the treatment period for subject 4 was far longer than the other subjects and provided for an interim reflection on results. The measurements made on 15/05/2003 are reflected in Table 4.20.

**Table 4.20: Biomechanical angles: Case Study 4**

<i>Date</i>	<i>Joint Range of Movement (degree)</i>			
	<i>13/07/2003</i>		<i>25/10/03</i>	
	<i>Stance</i>		<i>Stance</i>	
	<i>Mid</i>	<i>Terminal</i>	<i>Mid</i>	<i>Terminal</i>
<i>Dorsi flexion left</i>	9,67	15	10,75	16,5
<i>Dorsi flexion right</i>	8,67	20	5,33	16,67
<i>MTP extension left</i>		47,33		47
<i>MTP extension right</i>		47,33		38
<i>Hind foot pronation left</i>	13		11,3	
<i>Hind foot pronation right</i>	13		12	

○ ***Soft tissue palpation (clinically significant muscles)***

All the clinically significant muscles were palpated for tightness and spasm and rated on a scale from 0 to 2. These results appear in Table 4.21.

**Table 4.21: Tightness of clinically significant muscles: Case Study 4 after intervention: (Date: 25/10/2003)**

<i>Muscle</i>	<i>Left Side</i>			<i>Right Side</i>			<i>Muscle</i>	<i>Left Side</i>			<i>Right Side</i>		
	<i>0</i>	<i>1</i>	<i>2</i>	<i>0</i>	<i>1</i>	<i>2</i>		<i>0</i>	<i>1</i>	<i>2</i>	<i>0</i>	<i>1</i>	<i>2</i>
<u><i>Posterior fascia links</i></u>							<u><i>Anterior fascia links</i></u>						
<i>Levator scapula</i>	0				<1		<i>Sternocleidomastoid</i>	0				<1	
<i>Trapezius</i>	0				1		<i>Scalenii</i>	0				<1	
<i>Latissimus dorsi</i>	0				1		<i>Pectoralis major</i>		<1			1	
<i>Erector spinae</i>	0				1		<i>Pectoralis minor</i>		1			<1	
<i>Quadratus lumborum</i>		<1			<1		<i>Serratus anterior</i>		<1			1	
<i>Gluteus medius</i>		<1			1		<i>Subscapularis</i>		<1			1	
<i>Gluteus maximus</i>		1			<1		<i>External oblique</i>		<1			1	
<i>Piriformis</i>		<1			<1		<i>Rectus abdominus</i>		1			1	
<i>Semimembranosus</i>	0				1		<i>Psoas- umbilicus head</i>	0				1	
<i>Semitendinosus</i>							<i>Psoas- iliac head</i>	0				0	
<i>Biceps femoris</i>	0			0			<i>Psoas- groin</i>	0				0	
<i>Gastrocnemius</i>		<1			<1		<i>Iliotibial band</i>	0				1	
<i>Soleus</i>	0			0			<i>Vastus lateralis</i>	0				0	
<i>Plantar fascia</i>	0				<1		<i>Vastus medialis</i>	0				<1	
							<i>Adductor longus</i>	0				<1	
							<i>Adductor magnus</i>	0				<1	
							<i>Pes anserinus</i>	0				0	

Key: 0= normal fascia

1= tight fascia

2=very tight fascia





#### 4.5.2.4. *Synthesis/discussion*

The discussion will be limited to the conclusions drawn in terms of the main proposition and associated rival theories.

##### ○ *The proposition*

The mobilization of soft tissue (myofascial tissue) of the tight clinical significant muscles did lead to a disappearance in symptoms of CPCS. This was accompanied with a reduction in the pressure in the posterior compartment manifested by means of the palpation of the posterior compartment area. This reduction in pressure in posterior compartment in all probability led to the alleviation of the symptoms of CPCS. This includes the increase in tissue blood flow to the posterior compartment area and associated oxygenation which reversed the process.

##### ○ *Rival theories*

- The subject had significant tightnesses in the clinical significant muscles. This negates the rival proposition that subjects will not have any significant tightness in the clinically significant muscles. At this point it does not exclude the possibility that a different theoretical framework could be responsible for the pathogenesis of the condition.
- The treatment of the tightness in the clinical significant muscles did lead to the alleviation of the symptoms of CPCS. This is however not a conclusive fact that this was solely responsible for the alleviation of the symptoms. It could be as a result of chance, and that a different theoretical framework is responsible for the pathogenesis of the condition.
- The rival theory that the alleviation of the symptoms of the condition is purely due to chance and the intervention has nothing to do with it can not be ruled out at this stage.
- The rival theory that the interventions have not been applied long enough in order to generate the required response is ruled out as the desired response has been achieved.



- Similarly the last rival theory that the researcher is incapable of applying the intervention techniques in an effective manner is also ruled out as the desired response has been achieved.

### **4.5.3. CASE STUDY 5**

#### ***4.5.3.1. The subject***

The subject in Case Study 5 was a 35 year old male athlete who participated competitively in road running races, duathlon- and cycling events.

#### ***Inclusion criteria***

He complained of a constant bilateral calf pain over the posterior-medial aspect of the middle one-third of his lower legs. The pain therefore increased with exercise and decreased with rest.

#### ***Exclusion criteria***

The pins-and-needles sensation that he occasionally experienced after sitting for a prolonged period of time behind the computer was present in both feet and affected the whole foot; it was not in the distribution area of a specific nerve.

#### ***4.5.3.2. Research Procedure***

##### ***Subjective assessment - Interview***

- ***Running history***

The subject was a 35 year old male athlete who participated competitively in road running races, duathlon- and cycling events. He had won the Cullinan Duathlon event (6



km run; 30 km cycle; 6 km run) the previous year and had also won a two year bursary to cycle in Belgium during the Nineties. He usually cycled a distance of between 60 and 120 kilometres on a Sunday and was running a total of 28 kilometres per week when he was seen by the researcher for the first time. At the time of inclusion into the study, he was running with mild anti-pronation shoes. He used different shoes (clip-on) for cycling.

○ ***Previous running injuries***

Previous injuries that he had encountered included:

- bilateral iliotibial band syndromes (18 months before)
- left sided hip pain (18 months before)
- right knee pain following a hit and run accident during 1989 whilst he was cycling
- a left ankle pain

○ ***Current symptoms***

He complained of a constant bilateral calf pain with an intensity of 40 on a 100mm VAS over the posterior-medial aspect of the middle one-third of his lower legs. He also experienced pain over the anterior-lateral aspects of both lower legs which developed during running, but was never as bad as the calf pain. The pain was rated as a 50 on the 100mm VAS directly after a run. The pain in the left lower leg was worse than that in the right leg. After a hard run, when the pain increased, a haematoma always appeared over the superior-lateral aspect of his left leg in the vicinity of his superior tibio-fibula joint. He often experienced pins and needles in both feet after sitting behind the computer for a prolonged period of time. He also complained of experiencing abdominal cramps (stitches) during hard runs.

○ ***History of symptoms and previous treatment received***

He had first experienced these symptoms six months before, when he increased his training from eight kilometres to 28 kilometres per week. Since then the symptoms in his legs, had grown gradually worse. He stopped training for a period of three weeks in order

to recuperate but with his first training run all symptoms returned immediately. In summary, the subjective outcome measures before the intervention were as follows:

- Pain/discomfort at rest was 40 on the 100mm VAS and 50 after a training session.
- He averaged a weekly distance of 28 kilometres per week.

**Objective assessment – Physical examination**

○ **Muscle strength tests**

Muscle strength was measured by a biokineticist on 14/06/2003 on a calibrated Isokinetic dynamometer, using a standardized testing protocol. The results are reflected in Table 4.22.

**Table 4.22: Isokinetic dynamometer test results\* prior to intervention: Case Study 5.**

<i>Movement tested**</i>	<i>Peak torque (Nm)</i>			<i>Work per repetition(Nm/s)</i>		
	<i>Right</i>	<i>Left</i>	<i>Deficit</i>	<i>Right</i>	<i>Left</i>	<i>Deficit</i>
<i>Hip extensors</i>	252	237	-6	325	312	-4
<i>Hip flexors</i>	115	108	-6	144	137	-5
<i>Hip internal rotators</i>	47	47	0	46	50	-8
<i>Hip external rotators</i>	35	43	<i>-19</i>	38	49	<i>-22</i>
<i>Hip abduction</i>	130	137	-5	99	95	-4
<i>Hip adduction</i>	134	145	-7	111	119	-7
<i>Knee extensors</i>	217	188	<i>-13</i>	241	213	<i>-12</i>
<i>Knee flexors</i>	137	132	-4	165	156	-6
<i>Knee internal rotation</i>	42	50	<i>-16</i>	33	34	-4
<i>Knee external rotation</i>	47	42	<i>-11</i>	30	34	<i>-12</i>
<i>Ankle inversion</i>	46	50	27	35	46	***
<i>Ankle eversion</i>	24	36	51	22	28	***
<i>Ankle plantar flexors</i>	89	103	<i>-13</i>	49	57	<i>-14</i>
<i>Ankle dorsi flexors</i>	34	38	<i>-11</i>	18	24	<i>-28</i>

\*Statistically significant differences between right and left are given in italic

\*\*Tested at a speed of 30/30 and with 5 repetitions

\*\*\*Not determined.

**Table 4.23(a): Running gait analysis: Case Study 5 prior to intervention (Upper body)**

<i>Running gait analysis: Case Study 5:24/05/03</i>								
<i>Joint</i>	<i>Weight acceptance</i>			<i>Single leg support</i>		<i>Swing leg advancement</i>		
	<i>Initial contact</i>	<i>Loading response</i>	<i>Mid-stance</i>	<i>Terminal stance</i>	<i>Pre- swing</i>	<i>Initial swing</i>	<i>Mid-swing</i>	<i>Terminal swing</i>
Shoulders	Left leg: right shoulder in more extension and retraction than left shoulder with right leg initial contact. During this phase there is also a swing at the right shoulder blade. The right shoulder is in more depression than the left.							
Thoracic region	There is more upper thoracic rotation to the left (initial contact left leg) than to the right (initial contact right leg).							
Pelvis	The pelvis on the right moves into more anterior rotation than on the left.	Left: anterior rotation. Right: anterior rotation.	Left: mid position. Right: mid position.	The pelvis on the right moves into less posterior rotation than on the left.	Left: posterior rotation. Right: posterior rotation (less than on the left.)	Both move out of posterior rotation.	Both are in a neutral position.	Both in anterior rotation: right more than left.
Hip	Both are in flexion.	Both are in flexion and slight external rotation.	Both are in slight flexion and external rotation.	Both are in extension and external rotation. The left is in more external rotation than the right.	Both are in extension and starting to move out of external rotation.	Both moves into flexion and out of external rotation.	Both are in flexion and internal rotation. The right hip is in more internal rotation than the left.	Both are in flexion.

**Table 4.23(b): Running gait analysis: Case Study 5 prior to intervention (Lower body)**

<i>Running gait analysis: Case Study 5:24/05/03</i>								
<i>Joint</i>	<i>Weight acceptance</i>		<i>Single leg support</i>			<i>Swing leg advancement</i>		
	<i>Initial contact</i>	<i>Loading response</i>	<i>Mid-stance</i>	<i>Terminal stance</i>	<i>Pre- swing</i>	<i>Initial swing</i>	<i>Mid-swing</i>	<i>Terminal swing</i>
Knee	Both are in very slight flexion.	Slightly more flexion on both sides.	Slightly more flexion and external rotation on both sides.	Extension and external rotation on both sides. The right side is more externally rotated than the left.	Still in external rotation. Flexion is initiated.	On both sides the amount of flexion is increased and both start to move into internal rotation.	Both are in flexion and starting to move into external rotation and extension. There is more external rotation on the right than on the left.	Both are in slight flexion and external rotation.
Ankle	On both sides there is a definite heel contact. The ankles are in neutral with the feet a little in abduction.	Both ankles are in neutral: midway between supination and pronation and both feet are in abduction.	The degree of dorsi flexion at the ankle is 0 degrees. Both feet moves into hind foot pronation.	There is an irregularity in the right foot. Both feet are in plantar flexion. On the left, the push-off is in line with the 2 <sup>nd</sup> metatarsal head. On the right., it is in line with the 3 <sup>rd</sup> metatarsal and the foot moves slightly into inversion.	Both are in plantar flexion.	Both are in plantar flexion.	Both are in plantar flexion.	Both are in dorsi flexion.
Toes	Both are in extension. Very active tendons.	Both are in extension.	Both are still slightly in extension.	Both are in extension. He is very high on the toes. Seems to be higher on the toes of the left foot.	Both are in extension, but less.	Both are in extension, but less.	Almost neutral: both sides.	Both sides move into a lot of extension.

○ *Analysis of running gait, including movement patterns*

Movement patterns which deviated from the normal/ ideal running patterns are:

- The right shoulder moved into more extension and retraction than the left shoulder. There was more thoracic rotation towards the left compared on to the right side.
- The pelvis on the right side moved into anterior rotation during initial contact whilst the pelvis on the left side only moved into a neutral position.
- The hip on the left side was in a position of increased lateral rotation throughout compared to the right side.
- There is an irregularity in the right foot. On the left side, the push-off is in line with the second metatarsal head. On the right side, it is in line with the third metatarsal and the foot moves slightly into inversion.

○ *Flexibility/ length of soleus muscle 24/05/2003*

Right soleus muscle: Big toe 80 mm from the wall.

Left soleus muscle: Big toe 90 mm from the wall.

○ *Biomechanical angles 24/05/2003*

The biomechanical angles as measured on 24/05/2003 are reflected in Table 4.24.

**Table 4.24: Biomechanical angles: Case Study 5 during intervention**

<i>Movement</i>	<i>Joint Range of Movement (degrees)*</i>	
	<i>Mid stance</i>	<i>Terminal stance</i>
<i>Dorsi flexion left</i>	11, 67°	18, 00°
<i>Dorsi flexion right</i>	4, 33°	14, 67°
<i>Metatarsophalangeal extension left</i>		56, 67°
<i>Metatarsophalangeal extension right</i>		49, 33°
<i>Hind foot pronation left</i>	11, 67°	
<i>Hind foot pronation right</i>	9, 33°	

*\*Average of three measurements*

**Table 4.25: Tightness of clinically significant muscles: Case Study 5 prior to intervention (Date: 24/05/03)**

<i>Muscle</i>	<i>Left Side</i>			<i>Right Side</i>			<i>Muscle</i>	<i>Left Side</i>			<i>Right Side</i>		
	<i>0</i>	<i>1</i>	<i>2</i>	<i>0</i>	<i>1</i>	<i>2</i>		<i>0</i>	<i>1</i>	<i>2</i>	<i>0</i>	<i>1</i>	<i>2</i>
<b><i>Posterior fascia links</i></b>							<b><i>Anterior fascia links</i></b>						
<i>Levator scapula</i>		1				2	<i>Sternocleidomastoid</i>		1				2
<i>Trapezius</i>		1				2	<i>Scalenii</i>			2			2
<i>Latissimus dorsi</i>		1				2	<i>Pectoralis major</i>		1				2
<i>Erector spinae</i>		1				2	<i>Pectoralis minor</i>			2		1	
<i>Quadratus lumborum</i>			2		1		<i>Serratus anterior</i>			2		1	
<i>Gluteus medius</i>		1				2	<i>Subscapularis</i>	0				1	
<i>Gluteus maximus</i>		1				2	<i>External oblique</i>			2		1	
<i>Piriformis</i>		1				2	<i>Rectus abdominus</i>			2			2
<i>Semimembranosus</i>			2		1		<i>Psoas- umbilicus head</i>			2		1	
<i>Semitendinosus</i>			2		1		<i>Psoas- iliac head</i>			2		1	
<i>Biceps femoris</i>		1				2	<i>Psoas- groin</i>			2		1	
<i>Gastrocnemius</i>			2		1		<i>Iliotibial band</i>			2		1	
<i>Soleus</i>			2		1		<i>Vastus lateralis</i>	0			0		
<i>Plantar fascia</i>	0			0			<i>Vastus medialis</i>	0			0		
							<i>Adductor longus</i>			2		1	
							<i>Adductor magnus</i>			2		1	
							<i>Pes anserinus</i>			2		1	

Key: 0= normal fascia

1= tight fascia

2=very tight fascia





○ *The tightness of the clinically significant muscles*

All clinically significant muscles were palpated for tightness and rated on a scale of 0 – 2. The outcomes are provided in Table 4.25.

**4.5.3.3. Intervention**

All the tight clinically significant muscles were released. Different release techniques were used, depending on the muscle involved. Myofascial release techniques were used according to Mannheim (1994) and Barnes (1990). Trigger point release techniques were done according to Travell & Simons (1999) and specific soft tissue mobilization were done according to Hunter (1998).

The subject was seen twice a week during the first two weeks. Thereafter the frequency decreased to once every ten days for a period of five months. The intervals however varied from seven to fourteen days due to uncontrollable events.

He was instructed to stretch the following muscles on a daily basis:

- the trapezius
- the levator scapula
- the pectoralis
- the abdominal muscles
- the iliopsoas
- the piriformis
- the hamstrings
- the gastrocnemius and
- the soleus muscles

He was instructed to hold each stretch for 30 seconds and to repeat it.

The following clinical observations were made during the intervention period:



- the sensation of pins-and-needles in both feet which occurred after spending prolonged periods of time behind the computer disappeared.
- he could run hard without afterwards noticing any signs of haematomas over the superior tibio-femoral joint on the left.
- the release of the external oblique muscles led to the disappearance of the abdominal stitches he experienced whilst running hard. This was observed with the first run after these muscles were released.

### ***Treatment period***

24 May 2003 to 25 October 2003.

### ***Final assessment results (25/10/2003)***

#### ***Interview***

- He had no discomfort/pain at rest or during or after a run anymore.
- He ran a weekly distance of 55 to 65 kilometres.
- He ran a personal best time of 45 minutes for a ten kilometre run; as well as a best time of 94 minutes for a 21 kilometre run during November and October 2003 respectively.

#### ***Physical examination***

##### ***○ Re-assessment of running gait and movement patterns***

The following movement patterns deviated from the normal/ideal running patterns (see Table 4.26 for more detail):

- The pelvis was still slightly more posteriorly rotated on the left than on the right, but the difference was much less than with the initial assessment.
- The external hip rotation on the left was less. Both sides were more equal.

**Table 4.26: Running gait analysis: Case Study 5 after intervention (Date: 25/10/2003)**

<i>Running gait analysis: Case Study 5: 25/10/2003</i>								
<i>Joint</i>	<i>Weight acceptance</i>		<i>Single leg support</i>			<i>Swing leg advancement</i>		
	<i>Initial contact</i>	<i>Loading response</i>	<i>Mid-stance</i>	<i>Terminal stance</i>	<i>Pre- swing</i>	<i>Initial swing</i>	<i>Mid-swing</i>	<i>Terminal swing</i>
Shoulders	As above but to a lesser extent.							
Thoracic region	As above but to a lesser extent.							
Pelvis	The right side of the pelvis still moves into more anterior rotation than the left but the difference between the 2 sides are much less.				The pelvis is still slightly more posteriorly rotated on the left than on the right., but the difference is much less than with the previous assessment.			
Hips							The left and right sides are more equal. There is less medial rotation of the left hip.	
Knees							The external rotation on the left side is less. Both sides are more equal.	
Ankles		The right foot is less in abduction than with the previous assessment.		With the right foot's push-off, there is now only a slight inclination towards the 3 <sup>rd</sup> metatarsal head.				Appears not to go so high on the toes. The amount of dorsiflexion is less.
Toes								Appears not to go so high on the toes. The amount of MTP extension is less.

- With the right foot's push-off, there was now only a slight inclination towards the third metatarsal head.
- He did not rise as high as previously onto his front foot during the terminal phase. The measurements of the degree of metatarsophalangeal extension at the first metatarsophalangeal joint angle confirmed this observation (see Table 4.27).

○ ***Flexibility/ length of soleus muscle***

Right soleus muscle: Big toe 100 mm from the wall.

Left soleus muscle: Big toe 110 mm from the wall.

○ ***Biomechanical angles***

The biomechanical angles as measured on 25/10/2003 are reflected in Table 4.27.

**Table 4.27: Biomechanical angles: Case Study 5**

<i>Movement</i>	<i>Joint Range of Movement (degrees)*</i>	
	<i>Mid stance</i>	<i>Terminal stance</i>
<i>Dorsi flexion left</i>	13, 33°	18, 33°
<i>Dorsi flexion right</i>	5, 50°	14, 33°
<i>Metatarsophalangeal extension left</i>		52, 66°
<i>Metatarsophalangeal extension right</i>		40, 33°
<i>Hind foot pronation left</i>	9, 67°	
<i>Hind foot pronation right</i>	7, 00°	

\* *Averages of three measurements*

○ ***The tightness of the clinically significant muscles***

The tightness of the clinically significant muscles was again rated on a scale of 0 – 2 as indicated in Table 4.28.

**Table 4.28: Tightness of clinically significant muscles: Case Study 5 after intervention: (Date: 15/10/2003)**

<i>Muscle</i>	<i>Left Side</i>			<i>Right Side</i>			<i>Muscle</i>	<i>Left Side</i>			<i>Right Side</i>		
	<i>0</i>	<i>1</i>	<i>2</i>	<i>0</i>	<i>1</i>	<i>2</i>		<i>0</i>	<i>1</i>	<i>2</i>	<i>0</i>	<i>1</i>	<i>2</i>
<b><u>Posterior fascia links</u></b>							<b><u>Anterior fascia links</u></b>						
<i>Levator scapula</i>	0			0			<i>Sternocleidomastoid</i>		<1			1	
<i>Trapezius</i>	0				1		<i>Scalenii</i>	0			0		
<i>Latissimus dorsi</i>	0				1		<i>Pectoralis major</i>		<1			1	
<i>Erector spinae</i>	0				1		<i>Pectoralis minor</i>		1			<1	
<i>Quadratus lumborum</i>		1		0			<i>Serratus anterior</i>		1		0		
<i>Gluteus medius</i>	0			0			<i>Subscapularis</i>	0			0		
<i>Gluteus maximus</i>	0			0			<i>External oblique</i>		1			<1	
<i>Piriformis</i>	0			0			<i>Rectus abdominus</i>		1			1	
<i>Semi-membranosus</i>		1		0			<i>Psoas- umbilicus head</i>		1		0		
<i>Semitendinosus</i>	0			0			<i>Psoas- iliac head</i>		1		0		
<i>Biceps femoris</i>	0			0			<i>Psoas- groin</i>		1		0		
<i>Gastrocnemius</i>		1		0			<i>Iliotibial band</i>		1		0		
<i>Soleus</i>		1		0			<i>Vastus lateralis</i>	0			0		
<i>Plantar fascia</i>		1		0			<i>Vastus medialis</i>	0			0		
							<i>Adductor longus</i>		1		0		
							<i>Adductor magnus</i>		1		0		
							<i>Pes anserinus</i>		1		0		

Key: 0= normal fascia

1= tight fascia

2=very tight fascia



#### 4.5.3.4. *Synthesis/discussion*

The discussion will again be limited to the conclusions drawn in terms of the main proposition and associated rival theories.

##### ○ *The proposition*

The mobilization of the tightness (myofascial tissue) of the clinical significant muscles did lead to a disappearance in symptoms of CPCS. This was accompanied with a reduction in the pressure in the posterior compartment as manifested by means of the palpation of the posterior compartment area. This reduction in pressure in posterior compartment in all probability led to the alleviation of the symptoms of CPCS. This reduction in pressure can in all probability be contributed to the normalisation of the myofascial chain.

This normalisation leads to a reduction in the stresses in the chain and the pressure which these stresses induce into the posterior compartment. The pressure relief is associated with an increase in tissue blood flow to the posterior compartment area and an associated oxygenation which reversed the process.

##### ○ *Rival theories*

- The subject had significant tightnesses in the clinical significant muscles. This negates the rival proposition that subjects will not have any significant tightness in the clinically significant muscles as a different theoretical framework is responsible for the pathogenesis of the condition.
- The treatment of the tightness in the clinical significant muscles did lead to the alleviation of the symptoms of CPCS. This is however not a conclusive fact that this was solely responsible for the alleviation of the symptoms. It could be as a result of chance, and that a different theoretical framework is responsible for the pathogenesis



of the condition. In the light of the replication of the previous case study results this is considered to be highly unlikely.

- The rival theory that the alleviation of the symptoms of the condition is purely due to chance and the intervention has nothing to do with it can be considered as highly unlikely.

#### **4.5.3.5. *Quality assurance measures***

The review of the quality assurance measures will be done on a collective basis with regard to the explanatory research involving the main propositions at the end of this chapter.

#### **4.5.4. CASE STUDY 6**

**(24 May 2003 – 25 October 2003)**

##### **4.5.4.1. *The Subject***

The subject in Case Study 6 was a 35 year old male athlete, who had participated competitively in duathlon, cycling and running events for the previous four years.

##### ***Inclusion criteria***

He complained of symptoms of CPCS. The pain was situated over the posterior-medial aspect of the middle third of both calves. He described the pain as an intense cramp. Once he experienced this pain, it became impossible to run; he completed the race by walking. The pain increased with exercise and abated with rest.

##### ***Exclusion criteria***

Nothing abnormal was noted whilst testing the exclusion criteria.



#### **4.5.4.2. Research procedure**

##### ***Subjective assessment - Interview***

###### ***○ Running history***

The subject in Case Study 6 was a 35 year old male athlete. He had participated competitively in standard duathlon events, 100 km cycling events and ultra distance running events for the previous four years. His weekly training programme included a 100 kilometre bicycle ride on a Sunday, two spinning sessions in the gymnasium and a total of 40 kilometres of running. Whenever he participated in a long run such as a 42 km race over a weekend, he did very little running during the remainder of the week (at the most, distances of eight kilometres and no later than Wednesday). He did a 'brick session' twice a week, i.e. a spinning session followed by an eight kilometre running session.

At the time of inclusion into the study, he was running with strong anti-pronation shoes.

###### ***○ Previous running injuries***

He had right sided iliotibial band syndrome (2002).

He had left sided plantar fasciitis (2002).

He had received cortisone injections for both of the above injuries, which had left him pain free until the commencement of the intervention.

###### ***○ Current symptoms***

His symptoms usually started 35 to 40 kilometres into a race or towards the end of a brick session. He found that running over hilly terrain or on a treadmill, caused the symptoms to appear more quickly. The pain was situated over the posterior-medial aspect of the middle third of both calves. He described the pain as an intense cramp. Once he experienced this pain, it became impossible to run; he completed the race by walking. On





photographs that had been taken during one of his previous races, dentations caused by a cramp in the vicinity of the soleus muscles were clearly visible. He rated the intensity of the pain as 80 on a 100mm VAS. He also often experienced cramps in his adductors during a longer cycling race of 70 kilometres plus.

○ ***History of symptoms and previous treatment received***

The symptoms had started three years previously. He had previously received massage of the legs as a treatment and had been stretching the calf muscles (gastrocnemius- as well as soleus muscles); the hamstring muscles, the adductor muscles, the quadriceps-, the iliopsoas- and the piriformis muscles on a daily basis (each stretch was maintained for 30 seconds).

*In summary, the subjective outcome measures before the intervention were:*

- He had no pain at rest.
- The intensity of pain/ discomfort was rated as an 80 on the 100mm VAS and appeared after a brick session or 35 to 40 kilometres into the race.
- He averaged a running distance of 40 kilometres per week.

***Objective assessment – Physical examination***

○ ***Muscle strength tests***

*Muscle strength* was assessed by a biokineticist on a calibrated Isokinetic dynamometer, using a standardized testing protocol. It is clear from the measurements made on 06/06/2003 reflected in Table 4.29 that there were some muscle imbalances between the left and right sides of the athlete.

**Table 4.29: Isokinetic dynamometer test results\* prior to intervention: Case Study 6.**

<i>Movement tested**</i>	<i>Peak torque (Nm)</i>			<i>Work per repetition (Nm/s)</i>		
	<i>Right</i>	<i>Left</i>	<i>Deficit</i>	<i>Right</i>	<i>Left</i>	<i>Deficit</i>
<i>Hip extensors</i>	106	115	-8	141	154	-8
<i>Hip flexors</i>	78	71	9	90	90	0
<i>Hip internal rotators</i>	14	12	<i>14</i>	14	13	7
<i>Hip external rotators</i>	14	14	0	16	16	0
<i>Hip abduction</i>	66	76	<i>-13</i>	33	42	<i>-21</i>
<i>Hip adduction</i>	66	61	8	35	38	-8
<i>Knee extensors</i>	137	122	<i>11</i>	150	146	3
<i>Knee flexors</i>	81	72	<i>11</i>	97	89	8
<i>Knee internal rotation</i>	23	23	0	21	22	-5
<i>Knee external rotation</i>	17	15	<i>12</i>	18	17	6
<i>Ankle inversion</i>	21	12	***	18	9	***
<i>Ankle eversion</i>	16	12	***	14	9	***
<i>Ankle plantar flexors</i>	45	45	0	32	29	9
<i>Ankle dorsi flexors</i>	17	17	0	13	13	0

\*Statistically significant differences between the left and right legs are given in italic

\*\*Tested at a speed of 30/30 and with 5 repetitions

\*\*\*Not calculated

○ ***Analysis of running gait, including movement patterns (24/05/03)***

The following movement patterns as summarised in Table 4.30 deviated from the normal/ideal running patterns:

- The left shoulder moved into more extension and retraction than the right.
- There was more thoracic rotation towards the right in comparison to the left side.
- The pelvis on the left side moved into anterior rotation during initial contact whilst the pelvis on the right only moved into a neutral position.
- The right hip was throughout in an increased lateral rotation in comparison to the left.
- There is an irregularity in the right foot. On the left, the push-off is in line with the 2<sup>nd</sup> metatarsal head. On the right, it is in line with the 3<sup>rd</sup> metatarsal and the foot moves slightly into inversion.

**Table 4.30 (a): Running gait analysis: Case Study 6 prior to intervention (Upper body)**

<i>Running gait analysis: Case Study 6 (24/05/2003)</i>								
<i>Joint</i>	<i>Weight acceptance</i>		<i>Single leg support</i>			<i>Swing leg advancement</i>		
	<i>Initial contact</i>	<i>Loading response</i>	<i>Mid-stance</i>	<i>Terminal stance</i>	<i>Pre-swing</i>	<i>Initial swing</i>	<i>Mid-swing</i>	<i>Terminal swing</i>
Shoulders	Right leg: left shoulder in more extension and retraction than left leg with right shoulder (phase of initial contact). There is a bit of swinging of the right shoulder with extension of the right shoulder.							
Thoracic region	There is more thoracic rotation to the right than to the left.							
Pelvis	The pelvis on the left rotates more anteriorly than the pelvis on the right side.	The left pelvis is in anterior rotation, while the pelvis on the right side moves into a neutral position.	The pelvis on the left is in anterior rotation while the pelvis on the right is in a neutral position.	The pelvis on the right side is in more posterior rotation than the pelvis on the left side.	Both sides are in posterior rotation.	Both sides are in posterior rotation.	Both sides move into a position of neutral.	Left side of the pelvis moves into anterior rotation. The right side stays in neutral.
Hip	Both hips are in flexion. The left hip is slightly in adduction.	The left hip is lower than the right. Both hips are in flexion.	Both hips are in flexion.	Right hip in more extension and external rotation than the left. The left hip only moves into a neutral position.	Right hip: extension and external rotation. Left hip: neutral and external rotation.	The right hip starts to move into internal rotation. The left hip is in neutral.	The left hip is lower than the right hip. Both hips move into flexion.	The left hip is lower than the right. Both hips are in flexion.

**Table 4.30 (b): Running gait analysis: Case Study 6 prior to intervention (Lower body)**

<i>Running gait analysis: Case Study 6 (24/05/2003)</i>								
<i>Joint</i>	<i>Weight acceptance</i>		<i>Single leg support</i>			<i>Swing leg advancement</i>		
	<i>Initial contact</i>	<i>Loading response</i>	<i>Mid-stance</i>	<i>Terminal stance</i>	<i>Pre-swing</i>	<i>Initial swing</i>	<i>Mid-swing</i>	<i>Terminal swing</i>
Knee	Both are almost fully extended.	Slightly more flexion in both knees.	A further slight increase in the amount of knee flexion in both knees.	Both knees: almost full extension.	Left knee: slightly more flexion. Right knee: slightly more flexion and external rotation.	Both knees: external rotation and flexion.	Both knees: external rotation and flexion.	Both knees: almost full extension.
Ankle	Definite heel contact first (both feet) with very active extensor. Tendons.	Both feet in abduction.	Dorsi-flexion – both feet.	Plantar flexion – both feet.	Slight plantar flexion –both feet. Right foot: tends to shift weight to the 3 <sup>rd</sup> metatarsal head, the foot then moves into a position of inversion. This happens much less in the left foot. The amount of inversion is also less in the left foot.	Plantar flexion: left and right sides.	Right side: plantar flexion. Left side: neutral.	Right side: dorsi-flexion. Left side: slight dorsi-flexion.
Toes	Strong extension action on both sides.	Somewhat less extension on both sides.	Left and right sides are in neutral.	High on toes with push-off.	Extension.	Very slight extension on both sides.	Extension: left and right sides.	Extension: left and right sides.

○ *Flexibility/ length of soleus muscle*

Right soleus muscle: big toe 92 mm from the wall.

Left soleus muscle: big toe 86 mm from the wall.

○ *Biomechanical angles (Date: 24/05/2003)*

The biomechanical angles that were measured are provided in Table 4.31.

**Table 4.31: Biomechanical angles: Case Study 6 prior to intervention**

<i>Movement</i>	<i>Joint Range of Movement (degrees)*</i>	
	<i>Mid stance</i>	<i>Terminal stance</i>
<i>Dorsi flexion left</i>	5, 40°	15, 00°
<i>Dorsi flexion right</i>	9, 50°	20, 00°
<i>Metatarsophalangeal extension left</i>		54, 00°
<i>Metatarsophalangeal extension right</i>		51, 00°
<i>Hind foot pronation left</i>	11, 67°	
<i>Hind foot pronation right</i>	9, 00°	

\* *Averages of three measurements*

○ *The tightness of the clinically significant muscles*

Tightness of clinically significant muscles before intervention is reflected in Table 4.32.

**Table 4.32: Tightness of clinically significant muscles: Case Study 6 prior to intervention (Date: 24/05/2003)**

<i>Muscle</i>	<i>Left Side</i>			<i>Right Side</i>			<i>Muscle</i>	<i>Left Side</i>			<i>Right Side</i>		
	<i>0</i>	<i>1</i>	<i>2</i>	<i>0</i>	<i>1</i>	<i>2</i>		<i>0</i>	<i>1</i>	<i>2</i>	<i>0</i>	<i>1</i>	<i>2</i>
<u><i>Posterior fascia links</i></u>							<u><i>Anterior fascia links</i></u>						
<i>Levator scapula</i>		1			1		<i>Sternocleidomastoid</i>		1			1	
<i>Trapezius</i>		1				1,5	<i>Scalenii</i>		1				2
<i>Latissimus dorsi</i>		1				2	<i>Pectoralis major</i>		1			1	
<i>Erector spinae</i>			2			2	<i>Pectoralis minor</i>			2		1	
<i>Quadratus lumborum</i>			2		1		<i>Serratus anterior</i>		1			1	
<i>Gluteus medius</i>			2			2	<i>Subscapularis</i>		1				2
<i>Gluteus maximus</i>			2		1		<i>External oblique</i>			2			1,5
<i>Piriformis</i>			2			2	<i>Rectus abdominus</i>		1			1	
<i>Semimembranosus</i>			2			2	<i>Psoas- umbilicus head</i>			1,5			2
<i>Semitendinosus</i>		1			1		<i>Psoas- iliac head</i>	0			0		
<i>Biceps femoris</i>	0			0			<i>Psoas- groin</i>	0			0		
<i>Gastrocnemius</i>			2		1		<i>Iliotibial band</i>		1			1	
<i>Soleus</i>			2			1,5	<i>Vastus lateralis</i>	0			0		
<i>Plantar fascia</i>			2		1		<i>Vastus medialis</i>		1			1	
							<i>Adductor longus</i>		1				2
							<i>Adductor magnus</i>		1				1,5
							<i>Pes anserinus</i>			2	0		

Key: 0= normal fascia

1= tight fascia

2=very tight fascia



#### 4.5.4.3. Intervention

All the tight clinically significant muscles were mobilized. Different release techniques were used, depending on the muscle involved. Myofascial release techniques were used according to Barnes (1990) and Mannheim (1994), Trigger point release techniques were done according to Travell & Simons (1999) and Cross-frictions were done according to Cyriax (1988). The subject was for the first two weeks treated twice a week, thereafter once every ten days for a period of five months. These intervals again varied between seven and 14 days due to factors beyond the control of the subject or the researcher.

He was instructed to stretch the following muscles on a daily basis:

- the trapezius
- the levator scapula
- the pectoralis
- the abdominal muscles
- the iliopsoas
- the piriformis
- the hamstrings
- the gastrocnemius and
- the soleus muscles

He was instructed to hold each stretch for 30 seconds and to repeat it.

- ***Clinical observations during the intervention period:***

No new knowledge/ observations came to the fore.

- ***Treatment period***

32 weeks.



***Interim results (25/10/2003)***

***Interview***

- The intensity of pain was still rated as 80 on a 100mm VAS scale when he experienced the symptoms 35 to 40 km into a run.
- He no longer experienced cramps after a brick session and his adductors also did not cramp during cycling races.
- He averaged a weekly distance of 60 kilometres.
- The subject ran a 42 kilometre race during the second week in October 2003. It was a hilly course, partly on loose sand and from the 16 kilometres mark onwards the runners were running against a very strong head wind. The subject started cramping very badly at 16 kilometres.

*Note: In Case Study 4 a similar situation was experienced where loose sand triggered cramping faster than normal.*

In general the situation improved and he no longer experienced cramps during or after a brick session. He averaged a distance of 60 kilometres per week versus 40 kilometres. The subject also started out running harder than normal in order to improve his times.

***Physical examination***

- ***Re-assessment of running gait and movement patterns***

With regard to the movement patterns which are summarised in Table 4.33:

- In general his running movement patterns in the pelvic and hip area had improved.
- The anterior rotation on the right improved during the initial contact phase. During terminal stance, the lateral rotation of the hip was less.



**Table 4.33: Running gait analysis: Case Study 6 after intervention**

<i>Running gait analysis: Case Study 6 (25/10/2003)</i>								
<i>Joint</i>	<i>Weight acceptance</i>		<i>Single leg support</i>			<i>Swing leg advancement</i>		
	<i>Initial contact</i>	<i>Loading response</i>	<i>Mid-stance</i>	<i>Terminal stance</i>	<i>Pre--swing</i>	<i>Initial swing</i>	<i>Mid-swing</i>	<i>Terminal swing</i>
Shoulders	As above.							
Thoracic region	As above.							
Pelvis	The amount of anterior rotation on the left side is more, although it is still less than on the right.			The pelvis on the left is in less posterior rotation than before, but it is still more than that on the right hand side.				
Hip	The left hip is in less adduction than before.			The left hip now moves into extension but it is still less than that on the right side.				
Ankle				Left push-off is in line with the 2 <sup>nd</sup> metatarsal head. With the push-off on the right, there is still a weight shift towards the 3 <sup>rd</sup> metatarsal head.				

○ ***Flexibility/length of soleus muscles***

Right soleus muscle: big toe 95mm from the wall (was 92).

Left soleus muscle: big toe 92mm from the wall (was 86).

○ ***Biomechanical measurements***

The biomechanical measurements as measured on 25/10/2003 are given in Table 4.34.

**Table 4.34: Biomechanical angles: Case Study 6 after intervention**

<i>Movement</i>	<i>Joint Range of Movement (degrees)*</i>	
	<i>Mid stance</i>	<i>Terminal stance</i>
<i>Dorsi flexion left</i>	12, 00°	17, 33°
<i>Dorsi flexion right</i>	7, 67°	17, 67°
<i>Metatarsophalangeal extension left</i>		50, 00°
<i>Metatarsophalangeal extension right</i>		42, 67°
<i>Hind foot pronation left</i>	10, 33°	
<i>Hind foot pronation right</i>	8, 67°	

\* Averages of three measurements

○ ***The tightness of the clinically significant muscles***

From Table 4.35 it can be seen that the tightness of the clinically significant muscles at the end of October 2003, have improved except for the tightness of the pectoralis minor and the external oblique muscles. The pectoralis minor muscle still rated as a two on the left side and a one on the right. The external oblique muscle rated as a two on the left, and as 1,5 on the right. It was thus decided to mobilize only these two muscles whilst the subject continued with all the stretches. The objective was to determine whether the tightness in these two muscles caused the perpetuation of the symptoms.

**Table 4.35: Tightness of clinically significant muscles: Case Study 6 after intervention (Date: 25/10/2003) (Interim)**

<i>Muscle</i>	<i>Left Side</i>			<i>Right Side</i>			<i>Muscle</i>	<i>Left Side</i>			<i>Right Side</i>		
	<i>0</i>	<i>1</i>	<i>2</i>	<i>0</i>	<i>1</i>	<i>2</i>		<i>0</i>	<i>1</i>	<i>2</i>	<i>0</i>	<i>1</i>	<i>2</i>
<u><i>Posterior fascia links</i></u>							<u><i>Anterior fascia links</i></u>						
<i>Levator scapula</i>	0			0			<i>Sternocleidomastoid</i>		<1			1	
<i>Trapezius</i>	0				1		<i>Scalenii</i>	0			0		
<i>Latissimus dorsi</i>	0				1		<i>Pectoralis major</i>		<1			1	
<i>Erector spinae</i>	0				1		<i>Pectoralis minor</i>			2		1	
<i>Quadratus lumborum</i>		1		0			<i>Serratus anterior</i>		1		0		
<i>Gluteus medius</i>	0			0			<i>Subscapularis</i>	0			0		
<i>Gluteus maximus</i>			2		1.5		<i>External oblique</i>			2			1.5
<i>Piriformis</i>		1		0			<i>Rectus abdominus</i>		1			1	
<i>Semimembranosus</i>		1		0			<i>Psoas- umbilicus head</i>		1		0		
<i>Semitendinosus</i>	0			0			<i>Psoas- iliac head</i>		1		0		
<i>Biceps femoris</i>	0			0			<i>Psoas- groin</i>		1		0		
<i>Gastrocnemius</i>		1		0			<i>Iliotibial band</i>		1		0		
<i>Soleus</i>		1		0			<i>Vastus lateralis</i>	0			0		
<i>Plantar fascia</i>		1		0			<i>Vastus medialis</i>	0			0		
							<i>Adductor longus</i>		1		0		
							<i>Adductor magnus</i>		1		0		
							<i>Pes anserinus</i>		1		0		

Key: 0= normal fascia

1= tight fascia

2=very tight fascia



After two more treatment sessions during November and two during December 2003, the subject ran a difficult, hilly marathon at the end of January 2004. He completed this race without any incidence in a time of 4h08, compared to 4h53 for the race in October 2003. The release of the remaining two tight muscles led to the alleviation of all symptoms.

#### ***Final results (January 2004)***

##### ○ ***Interview***

He participated in a difficult and hilly 42 kilometre race the last weekend in January on a day during which the maximum temperature increased to 28 degrees Celsius and completed the distance symptom free. He averaged a running distance of 76 kilometres per week.

##### ○ ***Physical examination***

The tightness of the clinically significant muscles at the end of January 2004 is presented in Table 4.36.

#### ***4.5.4.4. Synthesis/discussion***

The discussion will again be limited to the conclusions drawn in terms of the main proposition and associated rival theories.

##### ○ ***The proposition***

The mobilization of the clinical significant muscles did lead to a disappearance in symptoms of CPCS. This was accompanied with a reduction in the pressure in the posterior compartment as manifested by means of the palpation of the calf area. This reduction in pressure in the posterior compartment in all probability led to the alleviation of the symptoms of CPCS by an increase in tissue blood flow to the posterior compartment area and associated oxygenation which reversed the process.

**Table 4.36: Tightness of clinically significant muscles: Case Study 6 (Final) (Date: 24/01/2004)**

<i>Muscle</i>	<i>Left Side</i>			<i>Right Side</i>			<i>Muscle</i>	<i>Left Side</i>			<i>Right Side</i>		
	<i>0</i>	<i>1</i>	<i>2</i>	<i>0</i>	<i>1</i>	<i>2</i>		<i>0</i>	<i>1</i>	<i>2</i>	<i>0</i>	<i>1</i>	<i>2</i>
<u><i>Posterior fascia links</i></u>							<u><i>Anterior fascia links</i></u>						
<i>Levator scapula</i>	0			0			<i>Sternocleidomastoid</i>	0			0		
<i>Trapezius</i>	0			0			<i>Scalenii</i>	0			0		
<i>Latissimus dorsi</i>	0			0			<i>Pectoralis major</i>	0			0		
<i>Erector spinae</i>	0			0			<i>Pectoralis minor</i>		1		0		
<i>Quadratus lumborum</i>	0			0			<i>Serratus anterior</i>	0			0		
<i>Gluteus medius</i>	0			0			<i>Subscapularis</i>	0			0		
<i>Gluteus maximus</i>	0			0			<i>External oblique</i>		1		0		
<i>Piriformis</i>		1		0			<i>Rectus abdominus</i>	0			0		
<i>Semimembranosus</i>	0			0			<i>Psoas- umbilicus head</i>	0			0		
<i>Semitendinosus</i>	0			0			<i>Psoas- iliac head</i>	0			0		
<i>Biceps femoris</i>	0			0			<i>Psoas- groin</i>	0			0		
<i>Gastrocnemius</i>		1		0			<i>Iliotibial band</i>	0			0		
<i>Soleus</i>	0			0			<i>Vastus lateralis</i>	0			0		
<i>Plantar fascia</i>	0			0			<i>Vastus medialis</i>	0			0		
							<i>Adductor longus</i>	0				1	
							<i>Adductor magnus</i>	0				1	
							<i>Pes anserinus</i>	0			0		

Key: 0= normal fascia

1= tight fascia

2=very tight fascia



- ***Rival theories***
  - The subject had significant tightness in the clinical significant muscles. This again negates the rival proposition that subjects will not have any significant tightness in the clinically significant muscles and that a different theoretical framework is responsible for the pathogenesis of the condition.
  - The treatment of the tightness in the clinical significant muscles did lead to the alleviation of the symptoms of CPCS. With three replications it appears as a reasonable conclusion that the interventions of releasing the tightness in the clinically significant muscles are responsible for the alleviation of the symptoms. This perspective is further enforced by the fact that the only other intervention with some degree of success is the surgical release of the involved fascia in the posterior compartment. It is highly unlikely that a totally different healing mechanism applies. It is also highly unlikely that the results are due to chance.

## 4.6. CROSS CASE STUDY COMPARISON

### 4.6.1. Introduction

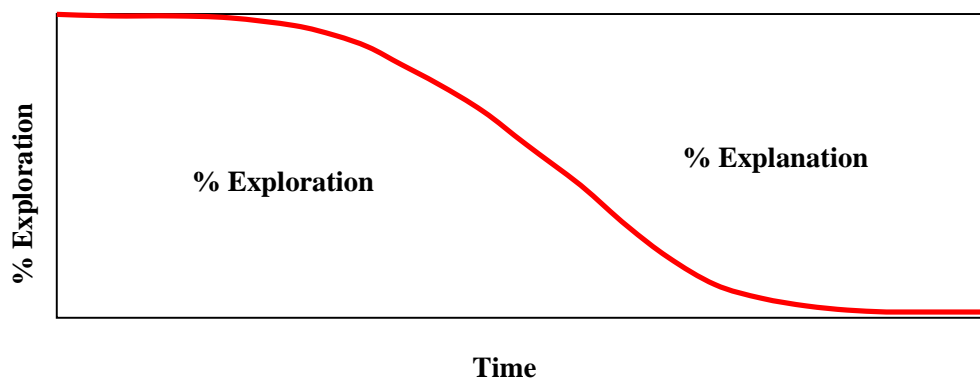
In previous sections the focus was on the individual case studies. In this chapter the focus will be on the consolidation of these observations in the form of cross case comparisons. With regard to the symptoms of CPCS, five measures were elected. These measures consisted of the *Intensity of pain/discomfort prior to running*; *Intensity of pain/discomfort post running*; *Distance run prior to symptoms*; *Total weekly distance run*; and *Palpation findings*. These measures were supplemented by observations made during the pre-assessment of the subjects, as well as post intervention assessments.

Although the explanatory research phase theoretically only dealt with case studies 4 to 6, the final results of case studies 1 to 3, are also included as supplementary evidence to the findings of the explanatory phase. Due to the additional interventions during the

exploratory phase, these results do not bear the same significance as the findings of case studies 4 to 6. From an ethical perspective however, the success that were achieved with the new interventions based on the new theoretical model for the pathogenesis of CPCS had to be shared with the first three subjects.

This section reviews the collective outcomes of the individual case study experiments discussed in previous sections. These outcomes consist of both quantitative and qualitative results. It includes the qualitative observations made during the initial assessment as well as those at the end of the interventions.

Some of these abnormal movement observations triggered the supplementary biomechanical experimentation covered in this section. A summary of the chronological progression of the case studies are presented in Table 4.37 for the ease of relating outcomes to the chronological sequencing of the interventions that were applied. Subject 4, as can be seen from Table 4.37, provided some overlap with the previous three case studies. In this regard it is important to note the gradual progression from pure exploration to explanation of new theoretical concepts formalised. This progression is graphically illustrated in Figure 4.10.



**Figure 4.10: Progressive change from *Exploration* to *Explanation***

**Table 4.37: A summary of the chronological progression of case studies 1 to 6**

<i>Subjects</i>	<i>Main approach used</i>	<i>Main outcome</i>
<b>Subject 1</b>	<b>Phase 1: 24/01/02 - 05/02/02</b> - Conventional physiotherapy	No improvement
	<b>Phase 2: 15/04/02 - 03/05/02</b> - Calf muscle strengthening and proprioceptive retraining	No improvement
	<b>Phase 3: 10/05/02 - 02/07/02</b> - Mobilization aimed specifically at the fascia of the lower leg	Slight improvement
	<b>Phase 4: 05/07/02 - 25/09/02</b> - Mobilization of the more proximal soft tissue*	Pain free
<b>Subject 2</b>	<b>Phase 1: 10/03/03 - 28/05/03</b> - Mobilization of the more proximal posterior soft tissue*	Progressive improvement
<b>Subject 3</b>	<b>Phase 1: 14/05/03 - 13/06/03</b> - Mobilization of the more proximal posterior soft tissue *	Noticeable improvement
	<b>Phase 2: 26/06/03 - 01/09/03</b> - Mobilization of more proximal anterior and posterior soft tissue*	Noticeable improvement
<b>Subject 4</b>	<b>Phase 1: 23/08/02 - 10/03/03</b> - Mobilization aimed specifically at the fascia of the lower leg - Mobilization of the soft tissue of the trunk and pelvis*	No improvement Noticeable improvement
	<b>Phase 2: 10/03/03 - 25/10/03</b> - Mobilization of the clinically significant muscles*	Pain free
<b>Subject 5</b>	<b>Phase 1: 24/05/03 - 25/10/03</b> - Mobilization of the clinically significant muscles*	Pain free
<b>Subject 6</b>	<b>Phase 1: 24/05/03 - 25/10/03</b> - Mobilization of the clinically significant muscles* (pectoralis minor- and external oblique not mobilized effectively)	Initial improvement
	<b>Phase 2: 25/10/03 - 04/12/03</b> - Mobilization of all the clinically significant muscles*	Pain free

\* Plus stretches



#### 4.6.2. The symptoms prior and post interventions

##### *Pain/discomfort*

All the subjects had symptoms of *pain and discomfort* prior to the interventions. These symptoms progressively responded positively to the interventions as more of the ‘clinically significant’ muscles were mobilized. In terms of pain/discomfort all the subjects were symptom free at the end of the interventions. The intensity of pain/discomfort as measured on a 100mm VAS prior- and post intervention is summarized in Table 4.38

**Table 4.38: Intensity of pain and discomfort before and after intervention**

	<i>Intensity of pain VAS- 100mm (Visual analogue scale)</i>					
<i>Case Studies</i>	<b>1</b>	<b>2</b>	<b>3</b>	<b>4</b>	<b>5</b>	<b>6</b>
<i>Prior to intervention</i>	20	60	65	75	50	80
<i>Post intervention</i>	0	0	0	0	0	0

The fact that all the subjects were pain free after the interventions is considered to be a significant improvement.

##### *Distance run before commencement of symptoms*

The second outcome measure used was the *distance run before commencement of symptoms*. The results of this second outcome measure are given in Table 4.39.

**Table 4.39: Commencement of symptoms before and after intervention**

	<i>Distance run before commencement of symptoms (km)</i>					
<i>Case Studies</i>	<b>1</b>	<b>2</b>	<b>3</b>	<b>4</b>	<b>5</b>	<b>6</b>
<i>Prior to intervention</i>	1	0	0	0	0.5	35*
<i>Post intervention</i>	21	21	21	10**	32	42

\* Or after a brick session

\*\* Sometimes a slight discomfort after 10 km.

These results represent a significant improvement with the exception of subject 6 who's distances did not increase, but whose symptoms decreased.

### ***Total weekly distance run***

The results for the third outcome measure used, namely the *total weekly distance run*, are reflected in Table 4.40.

**Table 4.40: The total weekly distance run before and after intervention.**

<i>Case Studies</i>	<i>Total weekly distance run (km)</i>					
	<b>1</b>	<b>2</b>	<b>3</b>	<b>4</b>	<b>5</b>	<b>6</b>
<i>Prior to intervention</i>	12	0	0	14	28	40
<i>Post intervention</i>	17	12	18	20	60	60

The minimum improvement listed in Table 10.4 was 42% (from 12 km to 17 km).

### ***Conclusion***

The improvements in all three of the outcome measures were significant.

#### **4.6.3. Tightness in the clinically significant muscles**

The last of the quantitative measure used, is the palpation findings. In the previous sections it was seen that the successes of the interventions correlated well with the release of tightness in the 'clinically significant' muscles. In this section the pre- and post soft tissue intervention measurements will be reviewed on a comparative cross-case basis. The "clinically significant" muscles as shown earlier can be grouped according to their posterior and anterior fascia links. The posterior and anterior fascia links consists of 14 and 17 muscles respectively. With regard to these groups, two aspects will be reviewed in this section. The first is the number of muscles that were tight prior to the interventions. The second is the extent, or degree of tightness in the muscles. The tightness as indicated earlier, were measured on a scale of 0 to 2. The overall extent of tightness in a subject can

therefore be expressed as the product between the number of tight links, and the degree of tightness in the muscles. The review of the cross case results will firstly be done between the left and right sides, and thereafter between the posterior and anterior links.

#### 4.6.3.1. *Relative tightness on the left*

##### Posterior fascia links – prior to intervention

Each of the subjects had 14 posterior links that could have been tight. Collectively this provides a possible 42 posterior *fascia links* that could be tight. As seen from Table 4.41 (a), 39 out of these possible 42 links were tight. This represents ninety three percent (93%) of the posterior muscles. The average tightness per subject varied between 1.29 and 1.57, with an average tightness for the three subjects of 1.40.

**Table 4.41 (a): Relative tightness of posterior links prior to intervention (left)**

<i>Muscle</i>	<i>Relative Tightness</i>				
	<i>Case 4</i>	<i>Case 5</i>	<i>Case 6</i>	<i>Qty</i>	<i>Average</i>
<u><i>Posterior fascia links</i></u>					
Levator scapula	1	1	1	3	1.00
Trapezius	1	1	1	3	1.00
Latissimus dorsi	1	1	1	3	1.00
Erector spinae	1	1	2	3	1.33
Quadratus lumborum	2	2	2	3	2.00
Gluteus medius	0	1	2	2	1.00
Gluteus maximus	2	1	2	3	1.67
Piriformis	2	1	2	3	1.67
Semimembranosus	1	2	2	3	1.67
Semitendinosus	1	2	1	3	1.33
Biceps femoris	2	1	0	2	1.00
Gastrocnemius	2	2	2	3	2.00
Soleus	2	2	2	3	2.00
Plantar fascia	1	0	2	2	1.00
<b>Number of tight posterior links</b>	<b>13</b>	<b>13</b>	<b>13</b>	<b>39</b>	<b>13</b>
<b>Average posterior tightness rating</b>	<b>1.36</b>	<b>1.29</b>	<b>1.57</b>		<b>1.40</b>

The *product* of the *average tightness rating* (1.40) and the *average number of tight muscles* (13) thus provides an *overall tightness measure* of 18.2. The Overall tightness rating can thus be expressed as follows:

$$\text{Overall tightness rating} = \text{average tightness rating} \times \text{average number of tight muscles}$$

In subjects 4 to 6, the Quadratus lumborum, Gastrocnemius and Soleus muscles as highlighted in Table 4.41(a), were very tight with ratings of 2. With weight bearing exercises such as running, the most stress is expected in these muscles. The Quadratus lumborum is a stabilising muscle. With weakness of the Gluteus medius muscles, which acts as a stabiliser during weight bearing, the Quadratus lumborum muscle takes more strain. This leads to tightness in the Quadratus lumborum muscle.

**Anterior fascia links – prior to intervention**

**Table 4.41 (b): Relative tightness of anterior links prior to intervention (left)**

<i>Muscle</i>	<i>Relative Tightness</i>				
	<i>Case 4</i>	<i>Case 5</i>	<i>Case 6</i>	<i>Qty</i>	<i>Average</i>
<b><u>Anterior fascia links</u></b>					
Sternocleidomastoid	1	1	1	3	1.00
Scaleni	1	2	1	3	1.33
Pectoralis major	1	1	1	3	1.00
Pectoralis minor	2	2	2	3	2.00
Serratus anterior	1	2	1	2	1.33
Subscapularis	1	0	1	2	0.67
External oblique	1	2	2	3	1.67
Rectus abdominus	2	2	1	3	1.67
Psoas- umbilicus head	1	2	1,5	3	1.50
Psoas- iliac head	1	2	0	2	1.00
Psoas- groin	1	2	0	2	1.00
Iliotibial band	1	2	1	3	1.33
Vastus lateralis	0	0	0	0	0.00
Vastus medialis	0	0	1	1	0.33
Adductor longus	0	2	1	2	1.00
Adductor magnus	0	2	1	2	1.00
Pes anserinus	2	2	2	2	2.00
<b>Number of tight anterior links</b>	<b>13</b>	<b>14</b>	<b>15</b>	<b>42</b>	<b>14</b>
<b>Average anterior tightness rating</b>	<b>0.94</b>	<b>1.53</b>	<b>1.00</b>		<b>1.33</b>

The *anterior links* totalled 17 links per side as reflected in Table 4.41(b). This provides a grand total of 51 anterior links for the three subjects. Of these, 42 of the links were tight. This gives an average of 76% (82%) of the links that were tight. The *average tightness* of these links was 1.33. The *overall tightness rating* for the *anterior* left side is the product between the average number of tight links, namely 14, and the average tightness of 1.33. This provides an *overall tightness rating* of 18.62. In the case of the anterior links, only two muscles were very tight in all three subjects with ratings of 2. These muscles were Pectoralis minor and Pes anserinus.

**Posterior fascia links – post intervention**

The relative tightness for the Posterior fascia links, post intervention for the left side, is illustrated in Table 4.42(a).

**Table 4.42 (a): Relative tightness of posterior links post intervention (left)**

<i>Muscle</i>	<i>Relative Tightness</i>				
	<i>4</i>	<i>5</i>	<i>6</i>	<i>Qty</i>	<i>Average</i>
<b><u>Posterior fascia links</u></b>					
Levator scapula	0	0	0	0	0
Trapezius	0	0	0	0	0
Latissimus dorsi	0	0	0	0	0
Erector spinae	0	0	0	0	0
Quadratus lumborum	0.5	1	0	2	0.50
Gluteus medius	0.5	0	0	1	0.17
Gluteus maximus	0.5	0	0	1	0.17
Piriformis	0.5	0	0	1	0.17
Semi-membranosus	0	1	0	1	0.33
Semitendinosus	0	0	1	1	0.33
Biceps femoris	0	0	0	0	0
Gastrocnemius	0.5	1	0	2	0.50
Soleus	0	1	0	1	0.33
Plantar fascia	0	1	0	1	0.33
<b>Number of tight posterior links</b>	<b>5</b>	<b>5</b>	<b>1</b>	<b>11</b>	<b>5.5</b>
<b>Average posterior tightness rating</b>	<b>0.18</b>	<b>0.36</b>	<b>0.07</b>		<b>0.2</b>

As can be seen from the table the number of tight links after intervention, was reduced to 5, 5 for the posterior links. This is thirty-nine percent (39%) of the posterior ‘clinically significant’ muscles. The average tightness rating for the three subjects was reduced to 0,2. This provides an overall tightness measure of 1,1. Prior to intervention this figure was 18,2 which provides an improvement in the overall tightness measure of 17,1 or 94%.

**Anterior fascia links – prior to intervention**

The relative tightness for the anterior fascia links, post intervention for the left side, is illustrated in Table 4.42(b). As can be seen from the table the number of tight links after intervention, was reduced to 9,67. This is sixty-eight percent (68%) of the anterior ‘clinically significant’ muscles.

**Table 4.42 (b): Relative tightness of the anterior links prior to intervention (left)**

<i>Muscle</i>	<i>Relative Tightness</i>				
	<i>4</i>	<i>5</i>	<i>6</i>	<i>Qty</i>	<i>Average</i>
<b><u>Anterior fascia links</u></b>					
Sternocleidomastoid	0	0.5	1	2	0.88
Scalenii	0	0	1	1	0.50
Pectoralis major	0.5	0.5	1	3	0.67
Pectoralis minor	1	1	0	2	0.67
Serratus anterior	0.5	1	1	3	0.83
Subscapularis	0.5	0	1	2	0.88
External oblique	0.5	1	0	2	0.50
Rectus abdominus	1	1	1	3	1.50
Psoas- umbilicus head	0	1	0	1	0.50
Psoas- iliac head	0	1	0	1	0.33
Psoas- groin	0	1	0	1	0.33
Iliotibial band	0	1	1	2	0.67
Vastus lateralis	0	0	0	0	0.00
Vastus medialis	0	0	1	1	0.33
Adductor longus	0	1	1	2	1.00
Adductor magnus	0	1	1	2	0.67
Pes anserinus	0	1	0	1	0.33
<b>Number of tight anterior links</b>	<b>6</b>	<b>13</b>	<b>10</b>	<b>29</b>	<b>9.67</b>
<b>Average anterior tightness rating</b>	<b>0.24</b>	<b>0.71</b>	<b>0.59</b>		<b>0.62</b>

The relative tightness of the muscles changed from 1,33 to 0,62 after the intervention. The tightness of the ‘clinically significant’ muscles has thus improved by 86% for the posterior and 68% for the anterior muscles respectively.

#### 4.6.3.2. *Relative tightness on the right*

##### *Posterior fascia links – prior to intervention*

As can be seen from Table 4.43(a) the average number of tight ‘clinically significant’ muscles on the right was 13,33 for the posterior links. Prior to intervention the average posterior muscle tightness rating was 1,54. This provides an *overall tightness rating* for the three subjects of 20,5 prior to intervention.

**Table 4.43(a): Relative tightness of posterior links prior to intervention (right)**

<i>Muscle</i>	<i>Relative Tightness</i>				
	<i>4</i>	<i>5</i>	<i>6</i>	<i>Qty</i>	<i>Average</i>
<b><i>Posterior fascia links</i></b>					
Levator scapula	2	2	1	3	1.67
Trapezius	2	2	1,5	3	2.00
Latissimus dorsi	2	2	2	3	2.00
Erector spinae	2	2	2	3	2.00
Quadratus lumborum	2	1	1	3	1.33
Gluteus medius	2	2	2	3	2.00
Gluteus maximus	1	2	1	3	1.33
Piriformis	2	2	2	3	2.00
Semi-membranosus	2	1	2	3	1.67
Semitendinosus	1	1	1	3	1.00
Biceps femoris	1	2	0	2	1.00
Gastrocnemius	2	1	1	3	1.33
Soleus	2	1	1,5	3	1.50
Plantar fascia	1	0	1	2	0.67
<b>Number of tight posterior links</b>	<b>14</b>	<b>13</b>	<b>13</b>	<b>40</b>	<b>13.33</b>
<b>Average posterior tightness rating</b>	<b>1.71</b>	<b>1.50</b>	<b>1.33</b>		<b>1.54</b>

**Anterior fascia links – prior to intervention**

For the anterior links reflected in Table 4.43(b) the average number of tight clinically significant muscles was also 13,33 with an average tightness rating of 1,45. This provides an *overall tightness rating* of 19,3 prior to intervention.

**Table 4.43(b): Relative tightness of anterior links prior to intervention (right)**

<i>Muscle</i>	<i>Relative Tightness</i>				
	<b>4</b>	<b>5</b>	<b>6</b>	<i>Qty</i>	<i>Average</i>
<b><u>Anterior fascia links</u></b>					
Sternocleidomastoid	2	2	1	3	2.00
Scalenii	2	2	2	3	2.25
Pectoralis major	2	2	1	3	1.67
Pectoralis minor	1	1	1	3	1.00
Serratus anterior	2	1	0	2	1.00
Subscapularis	2	1	2	3	2.00
External oblique	2	1	1,5	3	1.50
Rectus abdominus	2	2	1	3	2.00
Psoas- umbilicus head	2	1	2	3	2.00
Psoas- iliac head	2	1	0	2	1.00
Psoas- groin	2	1	0	2	1.00
Iliotibial band	2	1	1	3	1.33
Vastus lateralis	1	0	0	1	0.33
Vastus medialis	1	0	1	2	0.67
Adductor longus	1	1	2	3	1.75
Adductor magnus	1	1	1,5	3	1.00
Pes anserinus	2	1	0	2	1.00
<b>Number of tight anterior links</b>	<b>14</b>	<b>13</b>	<b>13</b>	<b>40</b>	<b>13.33</b>
<b>Average anterior tightness rating</b>	<b>1.71</b>	<b>1.50</b>	<b>1.33</b>		<b>1.45</b>

**Posterior fascia links – post intervention**

As can be seen from Table 4.44(a) the average number of tight ‘clinically significant’ muscles on the right was reduced from 13,33 for the posterior links to 5,44. Prior to intervention the average posterior muscle tightness rating was 1,54. This was reduced to an average rating of 0,40 for the posterior links. This provides an overall tightness rating



for the three subjects of 2,2 post interventions. The overall tightness rating was thus reduced by 18,3. This is a reduction in the overall tightness rating of 89%.

**Table 4.44 (a): Relative tightness of the posterior links post intervention (right)**

<i>Muscle</i>	<i>Relative Tightness</i>				
	<i>4</i>	<i>5</i>	<i>6</i>	<i>Qty</i>	<i>Average</i>
<b><i>Posterior fascia links</i></b>					
Levator scapula	0.5	0	1	2	0.50
Trapezius	1	1	0	2	0.67
Latissimus dorsi	1	1	0	2	0.67
Erector spinae	1	1	0	2	0.67
Quadratus lumborum	0.5	0	1	2	0.50
Gluteus medius	1	0	0	1	0.33
Gluteus maximus	0.5	0	1	2	0.50
Piriformis	0.5	0	0	1	0.17
Semi-membranosus	1	0	0	1	0.33
Semitendinosus	0	0	1	1	0.33
Biceps femoris	0	0	0	0	0.00
Gastrocnemius	0.5	0	1	2	0.50
Soleus	0	0	0	0	0.00
Plantar fascia	0.5	0	1	2	0.50
<b>Number of tight posterior links</b>	<b>11</b>	<b>3</b>	<b>6</b>	<b>20</b>	<b>5.44</b>
<b>Average posterior tightness rating</b>	<b>0.57</b>	<b>0.21</b>	<b>0.43</b>		<b>0.40</b>

**Anterior fascia links post intervention**

For the anterior links, post interventions reflected in Table 4.44(b), the average number of tight clinically significant muscles was reduced to 8 with an average tightness rating of 0,5. This provides an overall tightness rating of 4 post interventions. This represents a reduction in this measure of 15,3. This is a 79% reduction in the overall tightness rating.

**Table 4.44 (b): Relative tightness of the anterior links post intervention (right)**

<i>Muscle</i>	<i>Relative Tightness</i>				
	<i>4</i>	<i>5</i>	<i>6</i>	<i>Qty</i>	<i>Average</i>
<b><i>Anterior fascia links</i></b>					
Sternocleidomastoid	0.5	1	1	3	1.38
Scaleni	0.5	0	0	1	0.38
Pectoralis major	1	1	1	3	1.00
Pectoralis minor	0.5	0.5	1	3	0.67
Serratus anterior	1	0	0	1	0.33
Subscapularis	1	0	0	1	0.50
External oblique	1	0.5	0	2	0.50
Rectus abdominus	1	1	1	3	1.50
Psoas- umbilicus head	1	0	0	1	0.50
Psoas- iliac head	0	0	0	0	0.00
Psoas- groin	0	0	0	0	0.00
Iliotibial band	1	0	1	2	0.67
Vastus lateralis	0	0	0	0	0.00
Vastus medialis	0.5	0	1	2	0.50
Adductor longus	0.5	0	0	1	0.38
Adductor magnus	0.5	0	0	1	0.17
Pes anserinus	0	0	0	0	0.00
<b>Number of tight anterior links</b>	<b>13</b>	<b>5</b>	<b>6</b>	<b>24</b>	<b>8.00</b>
<b>Average anterior tightness rating</b>	<b>0.59</b>	<b>0.24</b>	<b>0.35</b>		<b>0.50</b>

#### **4.6.3.3. Summary**

From the previous section on the changes in the tightness of the clinically significant muscles, it can be seen that there is a strong correlation between this reduction in the tightness of the clinically significant muscles and the other outcome measures used.

#### **4.6.4. Assessment of other abnormalities**

##### **4.6.4.1. Muscle imbalances**

The abnormalities in terms of muscle imbalances were assessed based on the differences in muscle strength and peak torque performance of muscles between the left and right sides of the subjects. These measurements were made on an Isokinetic dynamometer.

### *Peak torque performance*

The muscle imbalances based on *peak torque performance* is reflected in Table 4.45. The values in *italics* reflect statistically significant differences as determined by the imbedded software program resident in the Isokinetic dynamometer (Cybex, 1995). From Table 4.45 it can be seen that in 43 out of the 75 cases significant muscle imbalances existed. These imbalances represent 60% of the measurements. Significant differences existed for all subjects in terms of strength of the knee extensor muscles. In four out of the five there were significant differences in the strength of hip internal- and external rotator muscles as well as the knee external rotator muscles. The imbalances showed little correlation with each other. The correlation coefficients between case studies 2 and 3; 2 and 4; 5 and 6 are 0,127; 0,124; and 0,166 respectively. This indicates that muscle tightness of the subjects differed in terms of both location and the extent of tightness (Table 4.45).

**Table 4.45: Muscle imbalances in peak torque performance (Prior to treatment)**

Movement tested **	Imbalances between Left and Right (Nm)					Number of imbalances
	CS 2	CS 3	CS 4	CS 5	CS 6	
Peak torque						
Hip extensors	<i>-14</i>	<i>-14</i>	<i>13</i>	-6	-8	<i>3</i>
Hip flexors	<i>-11</i>	<i>-15</i>	<i>-16</i>	-6	9	<i>3</i>
Hip internal rotators	<i>-29</i>	<i>0</i>	<i>25</i>	0	<i>14</i>	<i>4</i>
Hip external rotators	<i>-11</i>	<i>-14</i>	<i>23</i>	<i>-19</i>	0	<i>4</i>
Hip abduction	<i>-38</i>	<i>-31</i>	<i>-14</i>	-5	<i>-13</i>	<i>4</i>
Hip adduction	<i>-23</i>	<i>-84</i>	<i>-32</i>	-7	8	<i>3</i>
Knee extensors	<i>-38</i>	<i>-10</i>	<i>10</i>	<i>-13</i>	<i>11</i>	<i>5</i>
Knee flexors	<i>-40</i>	<i>-6</i>	7	-4	<i>11</i>	<i>3</i>
Knee internal rotation	<i>-29</i>	<i>-14</i>	-4	<i>-16</i>	0	<i>3</i>
Knee external rotation	<i>-19</i>	<i>-11</i>	-5	<i>-11</i>	<i>12</i>	<i>4</i>
Ankle inversion	-3	<i>-27</i>	3	***	***	<i>1</i>
Ankle eversion	<i>-32</i>	<i>-18</i>	0	***	***	<i>2</i>
Ankle plantar flexors	<i>-25</i>	-7	7	<i>-13</i>	0	<i>2</i>
Ankle dorsi flexors	<i>-41</i>	-7	-6	<i>-11</i>	0	<i>2</i>
Total per case study	<i>14</i>	<i>12</i>	<i>7</i>	<i>6</i>	<i>5</i>	<i>43</i>

\*Statistically significant differences are reflected in *italics*;

\*\* Tested at a speed of 30/30 and with five repetitions; \*\*\* Not measured

***Work performance per repetition***

Muscle imbalances in terms of *work performance per repetition* are provided in Table 4.46.

**Table 4.46: Muscle imbalances: work performance per repetition (Prior to treatment)**

Movement tested ** Work per repetition	Imbalances between Left and Right (Nm/sec)					Number of imbalances
	CS 2	CS 3	CS 4	CS 5	CS 6	
Hip extensors	<i>-15</i>	<i>-14</i>	<i>18</i>	-4	-8	<i>3</i>
Hip flexors	-3	-4	-8	-5	0	0
Hip internal rotators	<i>-41</i>	<i>-25</i>	<i>39</i>	-8	7	<i>3</i>
Hip external rotators	<i>-37</i>	<i>-17</i>	<i>32</i>	<i>-22</i>	0	<i>4</i>
Hip abduction	<i>-42</i>	<i>-47</i>	-7	-4	<i>-21</i>	<i>3</i>
Hip adduction	<i>-20</i>	<i>-88</i>	<i>-24</i>	-7	-8	<i>3</i>
Knee extensors	<i>-42</i>	<i>-3</i>	9	<i>-12</i>	3	<i>3</i>
Knee flexors	<i>-44</i>	<i>-9</i>	-2	-6	8	<i>2</i>
Knee internal rotation	<i>-25</i>	<i>0</i>	0	-4	-5	<i>2</i>
Knee external rotation	<i>-15</i>	<i>-11</i>	-4	<i>-12</i>	6	<i>3</i>
Ankle inversion	<i>-33</i>	<i>-38</i>	4	***	***	<i>2</i>
Ankle eversion	<i>-53</i>	<i>-38</i>	<i>26</i>	***	***	<i>3</i>
Ankle plantar flexors	<i>-23</i>	<i>-14</i>	0	<i>-14</i>	9	<i>3</i>
Ankle dorsi flexors	<i>-33</i>	<i>-10</i>	5	<i>-28</i>	0	<i>3</i>
Total per case study	<i>13</i>	<i>13</i>	<i>5</i>	<i>5</i>	<i>1</i>	<i>37</i>

\*Statistically significant differences as determined by the Cybex in *italic*;

\*\* Tested at a speed of 30/30 and with five repetitions; \*\*\* Not measured

The statistically significant differences represented 37 out of the 75 measurements, i.e. 60%. In four out of the five subjects there were a statistically significant difference in muscle strength of the right and the left hip external rotators. In three of the five subjects there were a statistically significant difference in muscle strength between the left and the right hip extensor, -hip internal rotator, -hip abduction, -hip adduction, knee external rotation, ankle eversion, -ankle plantar flexion and- ankle dorsi flexion muscles. Similar to the muscle imbalances as reflected by the measurements in terms of maximum torque performance, similar patterns existed with the imbalances reflected by the work performed per cycle. This was again reflected by the correlation between the



measurements of the different subjects. The correlation coefficients between case studies 2 and 3; 2 and 4; 5 and 6 are 0,3; -0,38; and -0,25 respectively.

These observations suggest that distortions in the fascia affect muscle performance. The root cause of the distortions in the fascia is however tightness in the clinically significant muscles and it seems logical that such tightness would influence muscle performance.

### *Effect of intervention on muscle performance*

The initial objective did not include the retesting of the subjects. It was however decided to retest subject 4 after 11-months. The objective was to determine whether the soft tissue mobilization techniques had any effect on the subject's muscle strength and imbalances. This retesting was considered to be specifically relevant due to the fact that subject four did not do any form of strengthening exercises during the preceding 11-month time period. Any differences in muscle performance could therefore only reflect the effect of the soft tissue mobilization techniques.

### *Peak torque performance*

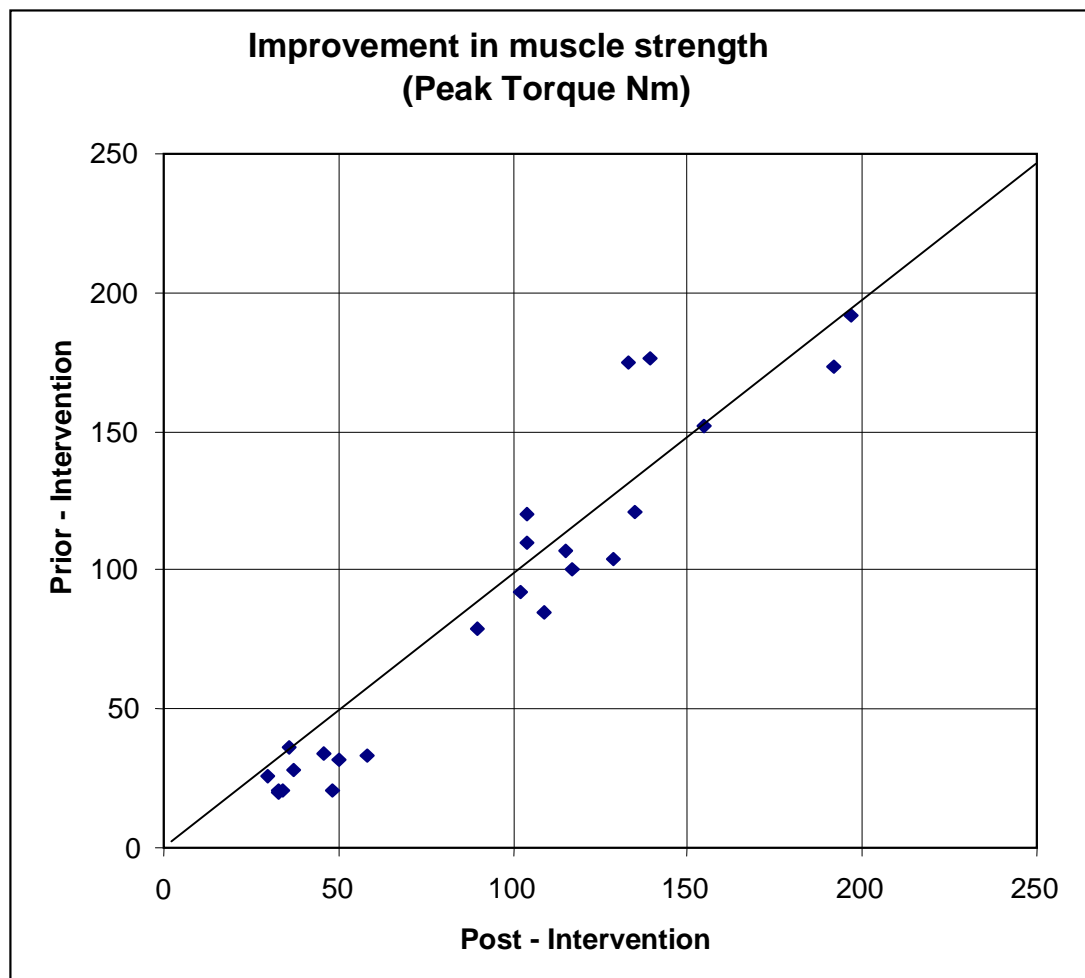
The comparison of pre- and post treatment results in terms of peak torque performance are listed in Table 4.47. The impact of the intervention on the muscle strength as reflected by peak torque performance is reflected in Figure 4.11. The fact that most of the data, as illustrated in Figure 4.11, fall to the right of the diagonal line imply a general improvement in muscle performance over a broad spectrum of muscles.

**Table 4.47: The effect of the treatments on peak torque performance (Nm)**



<b>Peak Torque Measurements – Right</b>			
<b>Movement Tested</b>	<b>Actual Measurements (Nm)</b>		
	<i>Prior</i>	<i>Post</i>	<i>%Change</i>
Hip extensors	175	133	-24%
Hip flexors	92	102	11%
Hip internal rotators	28	37	32%
Hip external rotators	26	30	15%
Hip abduction	104	129	24%
Hip adduction	120	104	-13%
Knee extensors	192	197	3%
Knee flexors	107	115	7%
Ankle inversion	33	58	76%
Ankle eversion	21	34	62%
Ankle plantar flexors	85	109	28%
Ankle dorsi flexors	34	46	35%

<b>Peak Torque Measurements – Left (Nm)</b>			
<b>Movement Tested</b>	<b>Actual Measurements</b>		
	<i>Prior</i>	<i>Post</i>	<i>%Change</i>
Hip extensors	152	155	2%
Hip flexors	110	104	-5%
Hip internal rotators	21	48	129%
Hip external rotators	20	33	65%
Hip abduction	121	135	12%
Hip adduction	176	139	-21%
Knee extensors	173	192	11%
Knee flexors	100	117	17%
Ankle inversion	32	50	56%
Ankle eversion	21	33	57%
Ankle plantar flexors	79	90	14%
Ankle dorsi flexors	36	36	0%



**Figure 4.11: Correlations between peak torque performance pre- and post intervention**

### *Work performance per cycle*

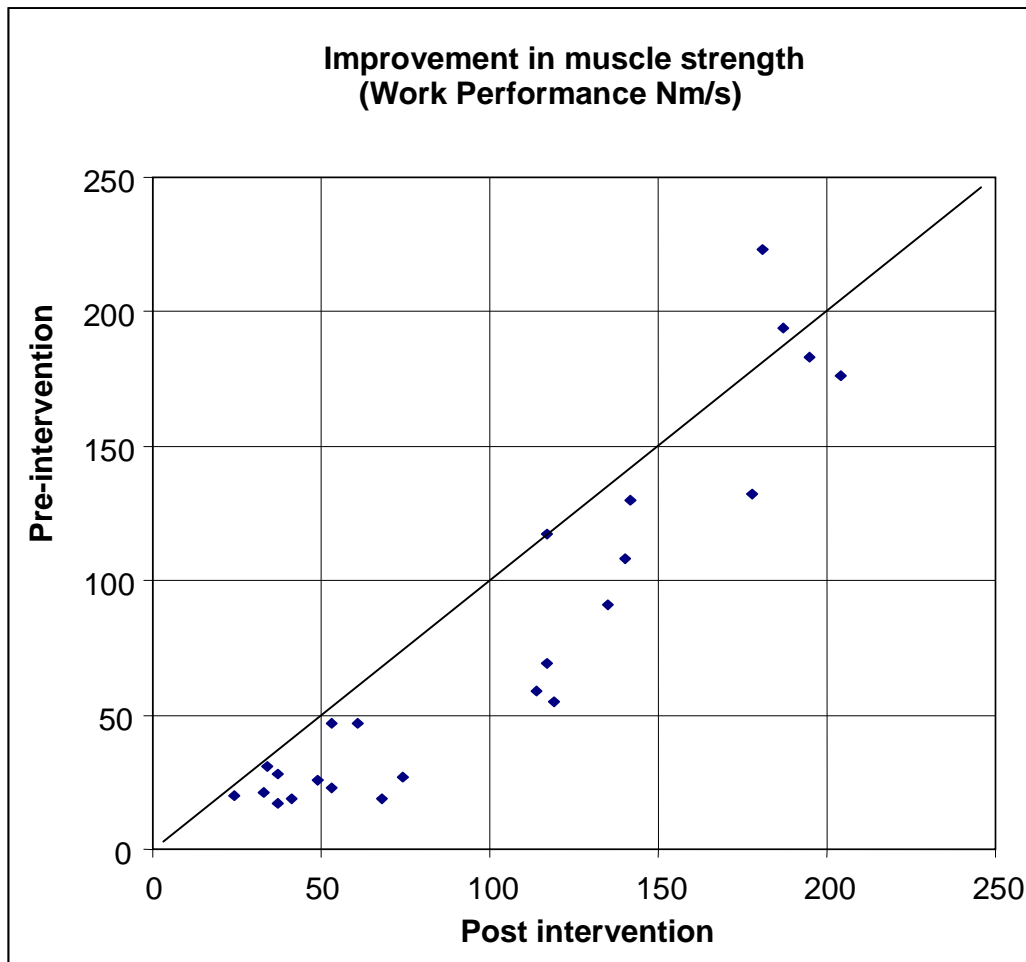
The comparison of the pre- and post treatment results is listed in Table 4.48. As can be seen from the Table 4.48 muscle strengths have improved over a broad spectrum.

**Table 4.48: Analysis of the effect of treatment on work performance (Nm/sec)**

<b>Work per Repetition – Right</b>			
<b>Movement Tested</b>	<b>Actual Measurements (Nm)</b>		
	<i>Prior</i>	<i>Post</i>	<i>%Change</i>
Hip extensors	223	181	-19%
Hip flexors	108	140	30%
Hip internal rotators	31	34	10%
Hip external rotators	28	37	32%
Hip abduction	55	119	116%
Hip adduction	69	117	70%
Knee extensors	194	187	-4%
Knee flexors	130	142	9%
Ankle inversion	27	74	174%
Ankle eversion	23	53	130%
Ankle plantar flexors	47	61	30%
Ankle dorsi flexors	21	33	57%
<b>Work per Repetition - Left</b>			
<b>Movement Tested</b>	<b>Actual Measurements (Nm)</b>		
	<i>Prior</i>	<i>Post</i>	<i>%Change</i>
Hip extensors	152	155	7%
Hip flexors	110	104	0%
Hip internal rotators	21	48	258%
Hip external rotators	20	33	116%
Hip abduction	121	135	93%
Hip adduction	176	139	48%
Knee extensors	173	192	16%
Knee flexors	100	117	35%
Ankle inversion	32	50	88%
Ankle eversion	21	33	118%
Ankle plantar flexors	79	90	13%
Ankle dorsi flexors	36	36	20%

These improvements in muscle strength as reflected by the improvement in work performance per cycle are reflected in Figure 4.12





**Figure 4.12: Correlation between work-performed per repetition pre- and post intervention**

The fact that most of the data points fall to the right of the diagonal line again illustrates the spectrum of improvement in muscle performance.

#### **4.6.4.2. Running movement pattern abnormalities**

The movement abnormalities observed during running and the effect of the mobilization of the tight 'clinically significant' muscles on these are summarized in Tables 4.49 to 4.56.

**Table 4.49 (a): A comparative summary of running gait analysis prior and post intervention: Weight acceptance phase – initial contact (Upper Body - Shoulder)**

<i>Prior Intervention</i>	<i>Post Intervention</i>
<p><b>Case Study 4</b></p> <p>Left leg: Right shoulder in more extension and retraction than left shoulder with right leg initial contact. During this phase the right shoulder-blade is winging more than the left and is also in more depression than the left.</p>	<p>Left leg: right shoulder in more extension and retraction than left shoulder with right leg initial contact, but less than before.</p>
<p><b>Case Study 5</b></p> <p>Left leg: Right shoulder in more extension and retraction than left shoulder with right leg initial contact. During this phase there is also some winging of the right shoulder-blade. The right shoulder is in more depression than the left.</p>	<p>The same movement patterns are observed but the differences between the left and the right sides are less.</p>
<p><b>Case Study 6</b></p> <p>Right leg: Left shoulder in more extension and retraction than left leg with right shoulder (phase of initial contact). There is a bit of winging of the left shoulder blade with extension of the left shoulder.</p>	<p>As before.</p>

***Comment***

Although no significant improvements were observed with Case Study 6, the improvements with the other two were noticeable. The symmetry of their movement patterns improved appreciably. The lack of improvement with the shoulder can be linked to the relative tightness in the pectoralis minor and external oblique muscles which were only effectively released during the last four treatment sessions.

**Table 4.49(b): A comparative summary of running gait analysis prior and post intervention: Weight acceptance phase – initial contact (Upper Body - Thoracic region)**

<i>Prior Intervention</i>	<i>Post Intervention</i>
<p><b>Case Study 4</b> There is more thoracic rotation to the left than to the right.</p>	The difference in the degree of rotation to the left- and the right side is less.
<p><b>Case Study 5</b> More upper thoracic rotation to the left than to the right.</p>	The difference in the degree of rotation to the left- and the right side is less.
<p><b>Case Study 6</b> More thoracic rotation to the right than the left.</p>	As before.

**Comment**

Similar to the shoulder, the movement patterns of the thoracic region were the same. Subject 6 showed no noticeable improvements while those of subjects 4 and 5 were much more symmetrical.

**Table 4.49 (c): A comparative summary of running gait analysis prior and post intervention: Weight acceptance phase – initial contact (Upper Body - Pelvis)**

<i>Prior Intervention</i>	<i>Post Intervention</i>
<p><b>Case Study 4</b> Right pelvis in anterior rotation, the left in a neutral position.</p>	Left pelvis in more anterior rotation than previously.
<p><b>Case Study 5</b> The pelvis on the right moves into more anterior rotation than on the left.</p>	The right still moves into more anterior rotation than the left but the difference between the two sides are much less.
<p><b>Case Study 6</b> The amount of anterior rotation on the left side is more, although less than on the right.</p>	The anterior rotation on the left side is more, although still less than on the right.

**Comment**

The movement patterns of all three the subjects normalised after the interventions.

**Table 4.50(a): A comparative summary of running gait analysis prior and post intervention: Weight acceptance phase – initial contact (Lower Body - Hip)**

<i>Prior Intervention</i>	<i>Post Intervention</i>
<p><b>Case Study 4</b> Right hip: flexion plus slight adduction. Left hip: flexion plus slight external rotation.</p>	Left and right sides are in flexion.
<p><b>Case Study 5</b> Both are in flexion.</p>	As before.
<p><b>Case Study 6</b> Hips in flexion - left slightly in adduction.</p>	The left hip is in less adduction than before.

**Comment**

The improvement in the adduction asymmetry of subjects 4 and 6 was noticeable.

**Table 4.50(b): A comparative summary of running gait analysis prior and post intervention: Weight acceptance phase – initial contact (Lower Body - Knee)**

<i>Prior Intervention</i>	<i>Post Intervention</i>
<p><b>Case Study 4</b> Both knees: almost full extension with the tibia in external rotation.</p>	All movements were normal.
<p><b>Case Study 5</b> Both are in very slight flexion.</p>	As before.
<p><b>Case Study 6</b> Both are almost fully extended.</p>	As before.

**Comment**

With the exception of subject 4, the interventions had little effect of the running gait.

**Table 4.50(c): A comparative summary of running gait analysis prior and post intervention: Weight acceptance phase – initial contact (Lower Body -Ankle)**

<i>Prior Intervention</i>	<i>Post Intervention</i>
<p><b>Case Study 4</b> No heel –toe action –both ankles. Lands in a slightly inverted position on the left ankle; laterally on the 5<sup>th</sup> metatarsal.</p>	All movements were normal.
<p><b>Case Study 5</b> Both are in very slight flexion.</p>	As before.
<p><b>Case Study 6</b> Both are almost fully extended.</p>	As before.

**Comment**

The heel-toe action of subject 4 normalized completely as a result of the interventions. In Subject 5, there was a decrease in the degree of fore foot abduction. Subject 6 was normal at the outset and showed no change.

**Table 4.50(d): A comparative summary of running gait analysis prior and post intervention: Weight acceptance phase – initial contact (Lower Body -Toes)**

<i>Prior Intervention</i>	<i>Post Intervention</i>
<p><b>Case Study 4</b> Excessive MTP extension, especially of the big toes</p>	All movements were normal.
<p><b>Case Study 5</b> Both are in extension. Very active tendons</p>	The extensor tendons are less active than before
<p><b>Case Study 6</b> Very active extensor tendons.</p>	Extensor tendons less active

**Comment**

The extensor tendons became less active in all three subjects.

**Table 4.51(a): A comparative summary of running gait analysis prior and post intervention: Weight acceptance phase – loading response (Pelvis)**

<i>Prior Intervention</i>	<i>Post Intervention</i>
<p><b>Case Study 4</b> Greater anterior rotation on the right.</p>	All movements were normal.
<p><b>Case Study 5</b> Greater anterior rotation on the right.</p>	All movements were normal.
<p><b>Case Study 6</b> The left pelvis is in anterior rotation, while the pelvis on the right side moves into a neutral position.</p>	All movements were normal.

**Comment**

There was more symmetry in the position of the pelvis in all the subjects.

**Table 4.51(b): A comparative summary of running gait analysis prior and post intervention: Weight acceptance phase – loading response (Hip)**

<i>Prior Intervention</i>	<i>Post Intervention</i>
<p><b>Case Study 4</b> Normal - Both hips are on the same level.</p>	Normal - Both hips are on the same level.
<p><b>Case Study 5</b> Both are in flexion and slight external rotation.</p>	Normal - Both hips are on the same level.
<p><b>Case Study 6</b> Left hip lower than the right. Both in flexion.</p>	Normal - Both hips are on the same level.

**Comment**

There was an improvement in the symmetry of the levels of the hips in subject 6.

**Table 4.51(c): A comparative summary of running gait analysis prior and post intervention: Weight acceptance phase – loading response (Ankle)**

<i>Prior Intervention</i>	<i>Post Intervention</i>
<p><b>Case Study 4</b> Shift the weight to the medial side of the foot on both sides.</p>	Moves into a lesser degree of eversion than before.
<p><b>Case Study 5</b> Ankles neutral: midway between supination and pronation; both feet in abduction.</p>	The right foot is less in abduction than with the previous assessment.
<p><b>Case Study 6</b> Both feet in abduction.</p>	A decrease in fore foot abduction.

**Comment**

The degree of eversion was less in subject 4 and the degree of fore foot abduction was less in subjects 5 and 6.

**Table 4.52(a): A comparative summary of running gait analysis prior and post intervention: Single leg support – mid-stance phase (Pelvis)**

<i>Prior Intervention</i>	<i>Post Intervention</i>
<p><b>Case Study 4</b> Both sides are in slight posterior rotation. The left side more so than the right side.</p>	Pelvis on the left and right in neutral. Still tends to drop a bit on the left side.
<p><b>Case Study 5</b> Left: mid position Right: mid position.</p>	Still the same.
<p><b>Case Study 6</b> The pelvis on the left is in anterior rotation - pelvis on the right is in a neutral position.</p>	Still the same.

**Comment**

The symmetry improved marginally.

**Table 4.52(b): A comparative summary of running gait analysis prior and post intervention: Single leg support – mid stance phase (Hip)**

<i>Prior Intervention</i>	<i>Post Intervention</i>
<p><b>Case Study 4</b> The right hip is in neutral - left hip is in slight abduction. Gait uneven - more of a push-off action on the right side.</p>	Gait more even. The left hip is now in a position of neutral.
<p><b>Case Study 5</b> Hips in slight flexion and external rotation.</p>	Still the same.
<p><b>Case Study 6</b> Both hips are in flexion.</p>	Still the same.

**Comment**

The gait became more even in subject 4.

**Table 4.52(c): A comparative summary of running gait analysis prior and post intervention: Single leg support – mid stance phase (Ankle)**

<i>Prior Intervention</i>	<i>Post Intervention</i>
<p><b>Case Study 4</b> Hind foot in pronation. The left and the right calcaneus sink further into valgus (loss of eccentric control). Both feet in abduction.</p>	The calcaneus sinks less into valgus than before. Both feet are in a lesser degree of abduction than before.
<p><b>Case Study 5</b> The degree of dorsi flexion at the ankle is 0 degrees. Both feet moves into hind foot pronation.</p>	The degree of hind foot pronation is less.
<p><b>Case Study 6</b> Dorsi-flexion – both feet.</p>	Still the same.

**Comment**

The symmetry improved marginally – an improvement in hind foot pronation.



**Table 4.53(a): A comparative summary of running gait analysis prior and post intervention: Single leg support – terminal stance phase (Pelvis)**

<i>Prior Intervention</i>	<i>Post Intervention</i>
<p><b>Case Study 4</b> Both sides are in posterior rotation; left side more than right.</p>	<p>The amount of posterior rotation of the pelvis is equal on the left and the right.</p>
<p><b>Case Study 5</b> The pelvis on the right moves into less posterior rotation than on the left.</p>	<p>The degree of hind foot pronation is less.</p>
<p><b>Case Study 6</b> The pelvis on the right side is in more posterior rotation than the pelvis on the left.</p>	<p>The pelvis on the left is in less posterior rotation than before, but still more than right.</p>

**Comment**

There was more symmetry with regard to the position of the pelvis.

**Table 4.53(b): A comparative summary of running gait analysis prior and post intervention: Single leg support – terminal stance phase (Hip)**

<i>Prior Intervention</i>	<i>Post Intervention</i>
<p><b>Case Study 4</b> Both hips are in extension and external rotation, the left more than the right.</p>	<p>The amount of posterior rotation of the pelvis is equal on the left and the right.</p>
<p><b>Case Study 5</b> Both hips are in extension and external rotation, left hip more external than the right.</p>	<p>The degree of hind foot pronation is less.</p>
<p><b>Case Study 6</b> Right hip in more extension and external rotation than the left. Left hip only moves into a neutral position.</p>	<p>The left hip now moves into extension but it is still less than that on the right side.</p>

**Comment**

The symmetry improved marginally.

**Table 4.53(c): A comparative summary of running gait analysis prior and post intervention: Single leg support – terminal stance phase (Ankle)**

<i>Prior Intervention</i>	<i>Post Intervention</i>
<p><b>Case Study 4</b></p> <p>The hind foot moves out of the pronated position into neutral. The right calcaneus starts to move earlier than the left one. The ankle moves into plantar flexion.</p>	
<p><b>Case Study 5</b></p> <p>An irregularity in the right foot. Both feet are in plantar flexion. On the left, the push-off is in line with the 2<sup>nd</sup> metatarsal head. On the right, it is in line with the 3<sup>rd</sup> metatarsal and the foot moves slightly into inversion.</p>	<p>With the right foot's push-off, there is now only a slight inclination towards the 3<sup>rd</sup> metatarsal head.</p>
<p><b>Case Study 6</b></p> <p>Plantar flexion –both feet.</p>	<p>Left push-off is in line with the 2<sup>nd</sup> metatarsal head.</p>

**Comment**

The movement patterns in the ankles of subjects 5 and 6 have improved.

**Table 4.53(d): A comparative summary of running gait analysis prior and post intervention: Single leg support – terminal stance phase (Toes)**

<i>Prior Intervention</i>	<i>Post Intervention</i>
<p><b>Case Study 4</b></p> <p>Toes move into extension.</p>	<p>Less high on the toes.</p>
<p><b>Case Study 5</b></p> <p>Both are in extension. He is very high on the toes. Seems to be higher on the toes of the left foot.</p>	<p>Less high on the toes.</p>
<p><b>Case Study 6</b></p> <p>High on toes with push-off.</p>	<p>Less high on the toes.</p>

***Comment***

After the intervention the decreases in the degree of extension at the metatarsophalangeal joints in subjects 5 and 6 were especially notable. It looked as if restrictions were removed that allowed for a greater range of movement. It was decided to investigate the phenomena further.

**Table 4.54(a): A comparative summary of running gait analysis prior and post intervention: Single leg support – pre-swing phase (Pelvis)**

<i>Prior Intervention</i>	<i>Post Intervention</i>
<p><b><i>Case Study 4</i></b> Both sides are in posterior rotation; left side more than right side.</p>	Posterior rotation equal on the left and right.
<p><b><i>Case Study 5</i></b> The pelvis is more posteriorly rotated on the left than on the right.</p>	Pelvis is still slightly more posteriorly rotated on left than on the right, difference is less.
<p><b><i>Case Study 6</i></b> Both sides are in posterior rotation.</p>	More symmetry with regard to the pelvis.

***Comment***

There is more symmetry with regard to the pelvis.

**Table 4.54(b): A comparative summary of running gait analysis prior and post intervention: Single leg support – pre-swing phase (Hip)**

<i>Prior Intervention</i>	<i>Post Intervention</i>
<p><b><i>Case Study 4</i></b> Both are in extension and external rotation. Left in more external rotation than the right.</p>	Normal.
<p><b><i>Case Study 5</i></b> Normal.</p>	Normal.
<p><b><i>Case Study 6</i></b> Both are in extension and external rotation. The left in more external rotation than right.</p>	Normal.

**Comment**

The symmetry improved marginally.

**Table 4.54(c): A comparative summary of running gait analysis prior and post intervention: Single leg support – pre-swing phase (Ankle)**

<i>Prior Intervention</i>	<i>Post Intervention</i>
<p><b>Case Study 4</b> Left and right sides are in a neutral hind foot position with plantar flexion.</p>	Normal.
<p><b>Case Study 5</b> Normal.</p>	Normal.
<p><b>Case Study 6</b> Slight plantar flexion –both feet. Right foot: tends to shift weight to the 3<sup>rd</sup> metatarsal head, the foot then moves into a position of inversion. Much less in the left foot.</p>	There is less of a weight shift in the right foot.

**Comment**

There is an improvement in the gait symmetry at the ankle in subject 6.

**Table 4.55(a): A comparative summary of running gait analysis prior and post intervention: Swing leg advancement – initial swing phase (Knee)**

<i>Prior Intervention</i>	<i>Post Intervention</i>
<p><b>Case Study 4</b> Knees move out of flexion and external rotation into flexion. An irregularity on the left with increased left hip external and posterior pelvic rotation.</p>	The observed irregularity is less.
<p><b>Case Study 5</b> Flexion is increased on both sides and both move into internal rotation.</p>	
<p><b>Case Study 6</b> Both knees: External rotation and flexion.</p>	

**Comment**

The symmetry has improved.

**Table 4.55(b): A comparative summary of running gait analysis prior and post intervention: Swing leg advancement – initial swing phase (Ankle)**

<i>Prior Intervention</i>	<i>Post Intervention</i>
<p><b>Case Study 4</b> No real push-off. Initiated by a backward rotation of the pelvis, hip extension and external rotation. With push-off the weight is shifted from the 2<sup>nd</sup> to the 3<sup>rd</sup> metatarsal head so that foot moves into inversion. It returns to the position of the 2<sup>nd</sup> metatarsal head as soon as the weight is off. Less subtle on the right.</p>	<p>There is less of a shift from the 2<sup>nd</sup> to the 3<sup>rd</sup> metatarsal head in both feet. In other words, the feet move into less inversion.</p>
<p><b>Case Study 5</b> Both are in plantar flexion.</p>	<p>With right foot's push-off, only a slight inclination towards the 3<sup>rd</sup> metatarsal head.</p>
<p><b>Case Study 6</b> Plantar flexion: Left and right sides</p>	

**Comment**

More symmetry and a lesser degree of inversion were noticeable in subjects 4 and 5.

**Table 4.56(a): A comparative summary of running gait analysis prior and post intervention: Swing leg advancement – mid swing phase (Hip)**

<i>Prior Intervention</i>	<i>Post Intervention</i>
<p><b>Case Study 4</b> Both hips: Flexion and internal rotation.</p>	
<p><b>Case Study 5</b> Both are in flexion and internal rotation. The right hip is in greater internal rotation.</p>	<p>The left and right sides are more equal. There is less medial rotation of the left hip.</p>
<p><b>Case Study 6</b> The left hip is lower than the right hip. Both hips move into flexion.</p>	

**Comment**

The symmetry improved marginally.

**Table 4.56(b): A comparative summary of running gait analysis prior and post intervention: Swing leg advancement – mid-swing phase (Knee)**

<i>Prior Intervention</i>	<i>Post Intervention</i>
<p><b>Case Study 4</b> Both sides: Flexion and internal rotation.</p>	Same.
<p><b>Case Study 5</b> Both are in flexion and starting to move into external rotation and extension. There is more external rotation on the right than on the left.</p>	The external rotation on the left side is less. Both sides are more equal.
<p><b>Case Study 6</b> Both sides: Flexion and internal rotation.</p>	Same.

**Comment**

The symmetry improved marginally.

**Table 4.57(a): A comparative summary of running gait analysis prior and post intervention: Swing leg advancement – terminal swing (Pelvis)**

<i>Prior Intervention</i>	<i>Post Intervention</i>
<p><b>Case Study 4</b> Left side moves into a neutral pelvic position and the right into anterior pelvic rotation.</p>	The left side now also moves into anterior rotation.
<p><b>Case Study 5</b> Both in anterior rotation: Right more than left.</p>	The pelvis is more symmetrical.
<p><b>Case Study 6</b> Left side of the pelvis moves into anterior rotation. The right side stays in neutral.</p>	The pelvis is more symmetrical.

**Comment**

The pelvis was more symmetrical in subjects 4, 5 and 6.

**Table 4.57(b): A comparative summary of running gait analysis prior and post intervention: Swing leg advancement – terminal swing (Hip)**

<i>Prior Intervention</i>	<i>Post Intervention</i>
<b>Case Study 4</b> Both are in flexion.	Both are in flexion.
<b>Case Study 5</b> Both are in flexion.	Both are in flexion.
<b>Case Study 6</b> The left hip is lower than the right. Both hips are in flexion.	Both hips are now almost on the same level.

**Comment**

There is greater symmetry in subject 6.

**Table 4.57(c): A comparative summary of running gait analysis prior and post intervention: Swing leg advancement – terminal swing (Ankle)**

<i>Prior Intervention</i>	<i>Post Intervention</i>
<b>Case Study 4</b> Left and right sides: slight inversion and plantar flexion.	
<b>Case Study 5</b> Both are in dorsi flexion.	The amount of dorsi flexion is less.
<b>Case Study 6</b> Right side: dorsi-flexion. Left side: Slight dorsi-flexion.	

**Comment**

The symmetry improved marginally.

**Table 4.57(d): A comparative summary of running gait analysis prior and post intervention: Swing leg advancement – terminal swing (Toe)**

<i>Prior Intervention</i>	<i>Post Intervention</i>
<p><b>Case Study 4</b> More extension.</p>	
<p><b>Case Study 5</b> A great degree of extension on both sides.</p>	<p>Does not go so high on the toes. The amount of MTP extension is less.</p>
<p><b>Case Study 6</b> Extension: Left and right sides.</p>	

**Comment**

As can be noted throughout from the comments in the right column of the preceding tables, the abnormalities in the common running patterns were significantly reduced by the treatment interventions. The soft tissue mobilization of the ‘clinically significant’ muscles led to an improvement in the running gait of case studies 4 to 6. The running gait became more symmetrical and the abnormal movement patterns normalized to a great extent. Shortening of the myofascial links will lead to asymmetry since the point of gravity will be shifted towards the shortened side. The muscle will follow the path of least resistance and will also add to instability.

**4.6.5. Conclusion**

The effect of the interventions on the symptoms of CPCS has been very successful. The *intensity of pain and discomfort; distances run before commencement of the symptoms; as well as total distances run*, improved to such an extent that all of the subjects were classified as symptom free.

The improvement in these outcome measures was accompanied by a strong correlation between the *tightness of the clinically significant muscles* as determined by the *palpation*





*findings* and the symptoms of CPCS. As the restrictions in the myofascial web were removed, the symptoms of CPCS cleared in the subjects.

With regard to the measurement of the tightness in the clinically significant muscles by means of palpation, the validity of the technique could constrain the external validity of the research. The treatment interventions are dependant on the ability of the health professional to assess and identify tightness in the clinically significant muscles. The calibration of this measurement technique will be covered in section 4.8.4.

In spite of a strong correlation between the symptoms of CPCS as reflected by the tightnesses in the clinically significant muscles, and muscle imbalances as reflected by muscle strength and muscle work performance, these results do not provide any conclusions of significance in terms of the research project. Any restriction in a muscle could affect muscle balances, and the results do not reflect on any aspect that is unique to the symptoms of CPCS. The interventions do however have the distinct advantage of rectifying any muscle imbalances that may be present.

During the running gait analysis, the initial observations made during the exploratory research phase, in terms of hind foot pronation as well as the extension at the metatarsophalangeal joint was again observed. During the exploratory phase, one of the objectives set was to assess whether hind foot pronation plays as big a role in the symptoms of CPCS as it does in other running injuries (Hintermann & Nigg, 1998). The experimental research which was conducted with regard to these two aspects will be covered in the next section.

## 4.7. EXPERIMENTAL RESEARCH

### 4.7.1. Introduction

This section covers the results of the experimentation with the effects of the interventions on the extension at the first metatarsophalangeal joint as well as on hind foot pronation.

### 4.7.2. Degree of extension at the metatarsophalangeal joint during terminal stance

#### 4.7.2.1. Results

From Table 4.58 it can be seen that the range of extension at the first metatarsophalangeal joint during terminal stance of all the subjects complaining of CPCS was greater than that of the two controls. They thus rose higher onto their forefeet during the terminal stance than the runners who served as controls.

**Table 4.58: Metatarsophalangeal extension at the first toe**

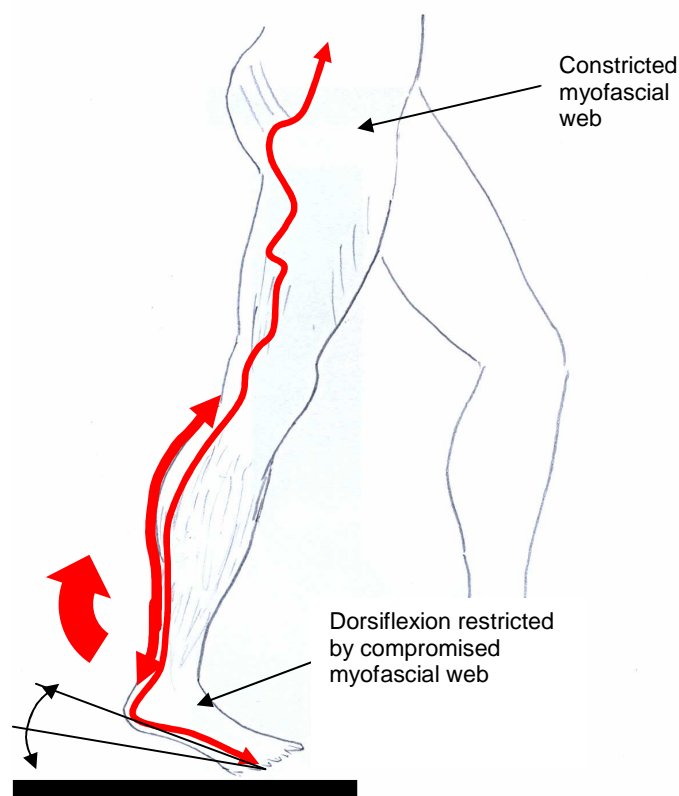
<i>Degree of extension at the metatarsophalangeal joint during terminal stance (degrees)</i>								
	<i>Subject 1</i>	<i>Subject 2</i>	<i>Subject 3</i>	<i>Subject 4</i>	<i>Subject 5</i>	<i>Subject 6</i>	<i>Control 1</i>	<i>Control 2</i>
<i>Left</i>	48, 33	49, 67	50, 67	58, 67	58, 33	57, 67	41, 00	38, 33
<i>Right</i>	47, 00	49, 00	45, 00	49, 33	45, 33	43, 67	36, 67	20, 86

#### 4.7.2.2. Discussion

As was seen during the literature research the FDL and the FHL muscles are important in preventing extreme plantar to dorsi flexion movement at the metatarsophalangeal joints when the foot is in contact with the ground (Travell & Simons, 1999). Tightness in the calf muscles might effectively lengthen the lever arm against which the FHL and the FDL muscles function and overload them. Therefore an increase in the degree of extension at the first metatarsophalangeal joint may be indicative of a dysfunction of the FHL muscle.

An increase in the flexibility of the calf muscles might theoretically effectively shorten the lever arm against which the FHL and the FDL muscles function and decrease the degree of extension at the first metatarsophalangeal joints.

It could also be argued that the degree of extension at the metatarsophalangeal joint during terminal stance could be as the result of the decrease in the functional length of the myofascial chain as a result of the effect of stresses that are induced in the fascial web.



**Figure 4.13: Metatarsophalangeal extension**

In Figure 4.13 this concept is illustrated graphically. As was argued with the development of the revised model for the pathogenesis of CPCS, the constricted myofascial web proximal to the calf area is reflected by the distorted red line above the knee. It is argued that the length of the myofascial web is constricted through tightness in the clinical significant muscles which compromise the length of fascia available to accommodate the increased demand for range of movements such as the dorsiflexion during running.



During running the normal range of movement is increased, and if these movements are restricted by the constriction of the myofascial web in the clinical significant muscles, it will lead to abnormal stresses in the myofascial web. This in turn will exert abnormal pressure in the posterior compartment. A decrease in the dorsiflexion would also lead to an increase in the degree of extension at the first metatarsophalangeal joint due to the unavailability of sufficient length in the myofascial chain to accommodate the requirements for normal movement, thus forcing the subject to rise higher on his toes.

The revised model for the pathogenesis acknowledges the role that the fascia plays in the stiffness in the calf muscle. Should the interventions thus relieve the stiffness in the clinical significant muscles, and this leads to a greater range of movement in dorsiflexion, it would make a strong case for the restricted fascia being the root cause to the problem and not just a general stiffness of the calf area as the current theoretical perspectives would suggest.

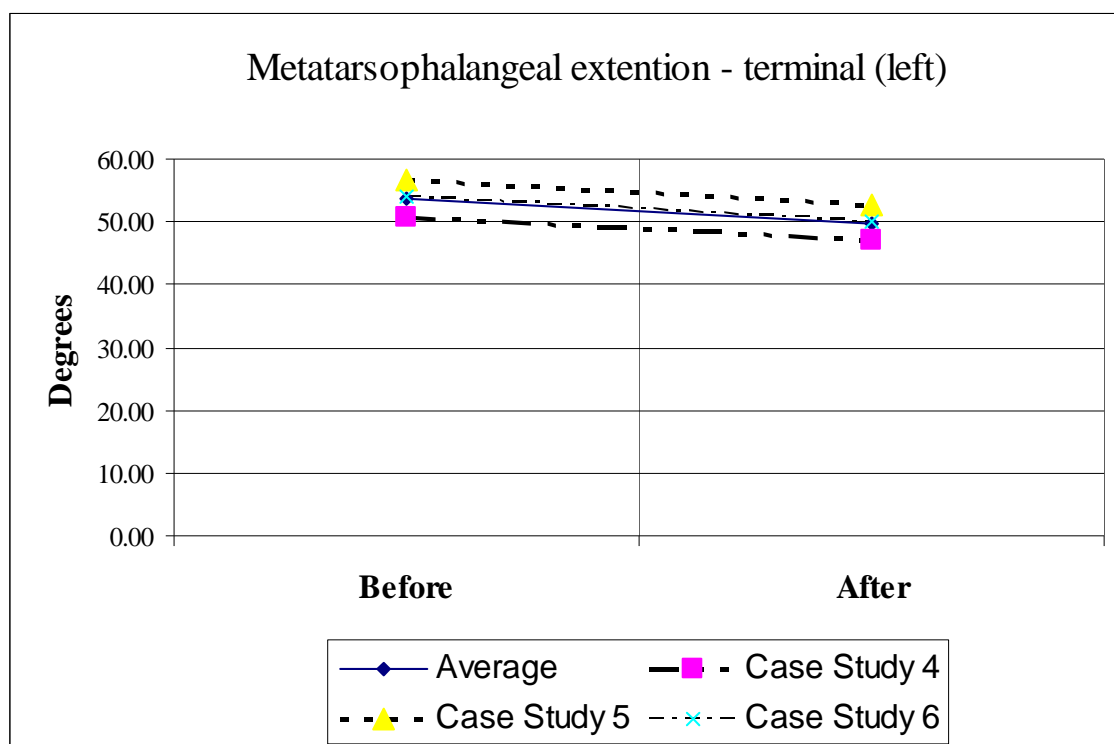
During the three case studies described in Chapter four, a gradual progression in the intervention techniques from soft tissue mobilizing of the posterior part of the lower leg towards the more proximal as well as the anterior soft tissue mobilization have been seen. The flexibility of the soleus calf muscles improved noticeably in case studies one to three after soft tissue mobilization and stretches were included in the interventions. It was therefore interesting to establish whether the intervention led to a decrease in the degree of extension at the first metatarsophalangeal joint.

As a second phase of the experiment it was thus decided to assess the impact of the release of the tight clinical significant muscles on the range of movement of dorsiflexion in the case studies 4 to 6. The results of both the before and after treatment interventions are reflected in Table 4.59.

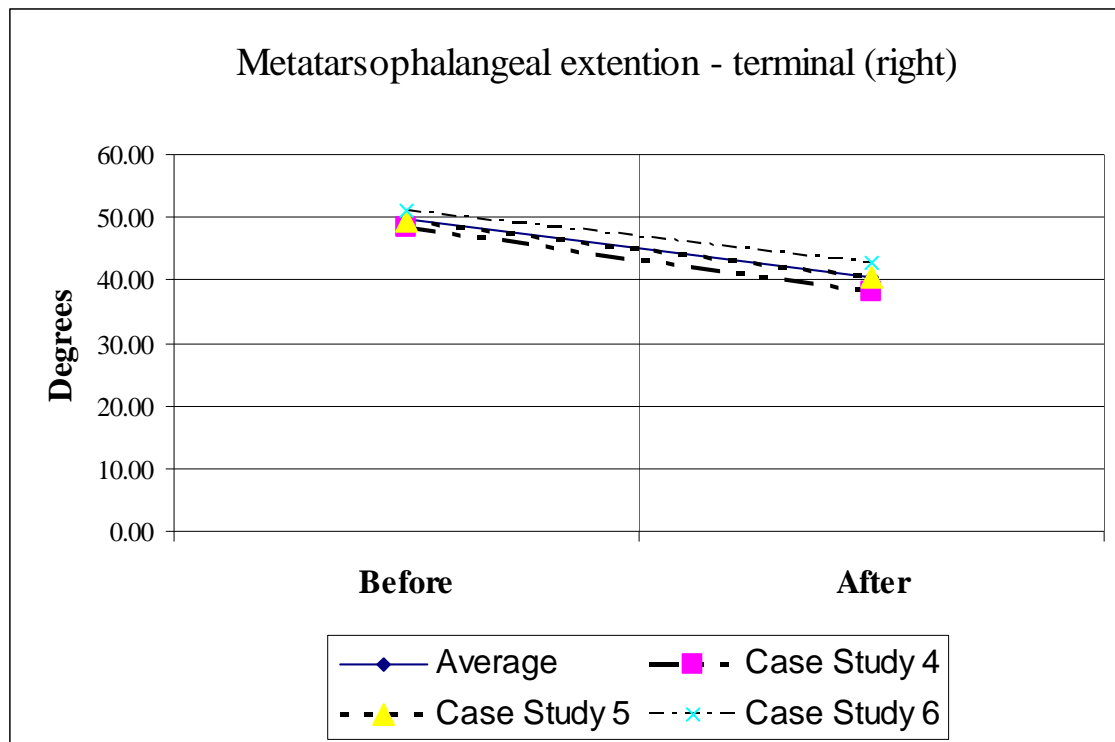
**Table 4.59: Biomechanical measures before and after intervention**

<i>Metatarsophalangeal extension</i>						
<i>(Degrees)</i>						
<i>Side</i>	<i>Case Study 4</i>		<i>Case Study 5</i>		<i>Case Study 6</i>	
	<i>Before</i> 28/01/03	<i>After</i> 25/10/03	<i>Before</i> 24/05/03	<i>After</i> 24/05/03	<i>Before</i> 24/05/03	<i>After</i> 24/05/03
<i>Left</i>	50.67	47.00	56.67	52.66	54.00	50.00
<i>Right</i>	48.50	38.00	49.33	40.33	51.00	42.67

There was a noticeable decrease in the degree of extension at the first metatarsophalangeal joint in all the cases. These results are graphically reflected in Figures 4.14 and 4.15



**Figure 4.14: Improvement of the metatarsophalangeal extension: Left leg**



**Figure 4.15: Improvement of the metatarsophalangeal extension: Right leg**

In both Figures 4.14 and 4.15 the noticeable improvement in the range of movement of both metatarsophalangeal extensions can clearly be seen. This evidence provides a strong support for the new theoretical model which suggests that the restriction of movement is due to fascia that is constricted in the clinical significant muscles. In this process the tightnesses in the posterior compartments were also relieved and one can thus not conclusively argue that the improvement in movement range is solely due to the increased fascia length which resulted from the normalisation of the restrictions in the myofascial web.

In all the cases a distinct improvement in the flexibility at the metatarsophalangeal joint is evident. This effectively rules out the Null Hypothesis which states that the results are purely due to chance. The rival proposition of investigator bias is also ruled out on the basis that the objective evidence is available for scrutiny in the form of the video recordings.

### 4.7.3. The effect of soft tissue mobilization on subtalar over pronation

#### 4.7.3.1. Results

The results of the interventions are tabled in Tables 4.60 and 4.61.

**Table 4.60: Change in left hind foot pronation before and after intervention**

<i>Subject</i>	<i>Left Hind Foot Pronation (Degrees)</i>											
	<i>1</i>	<i>2</i>	<i>3</i>	<i>4</i>	<i>5</i>	<i>6</i>	<i>7</i>	<i>8</i>	<i>9</i>	<i>10</i>	<i>11</i>	<i>12</i>
<i>Before</i>	15	13	18	20	18	21	16	14	14	11	16	22
<i>After</i>	11	4	13	10	14	13	5	7	12	9	12	<b>17</b>
<i>Difference</i>	4	9	5	10	4	8	11	7	2	2	4	5
<i>% Reduction</i>	27	69	28	50	22	38	69	50	14	18	25	23
<i>(N)/(A)*</i>	N	N	N	N	N	N	N	N	N	N	N	<b>A</b>

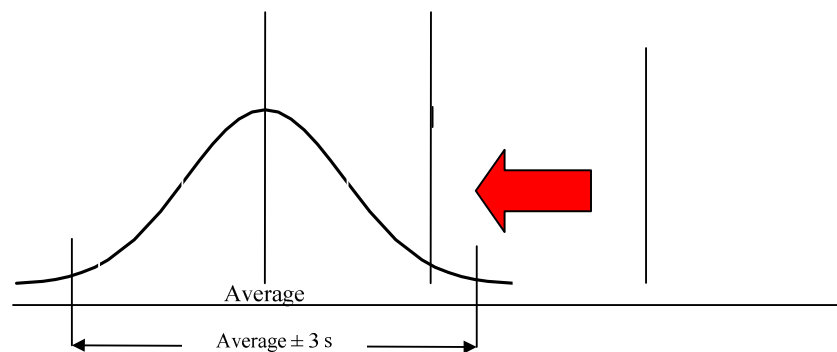
**Table 4.61: Change in right hind foot pronation before and after intervention**

<i>Subject</i>	<i>Right Hind Foot Pronation (Degrees)</i>											
	<i>1</i>	<i>2</i>	<i>3</i>	<i>4</i>	<i>5</i>	<i>6</i>	<i>7</i>	<i>8</i>	<i>9</i>	<i>10</i>	<i>11</i>	<i>12</i>
<i>Before</i>	15	16	17	18	17	22	12	18	17	18	12	22
<i>After</i>	9	10	11	11	14	<b>15</b>	8	13	12	12	11	<b>19</b>
<i>Difference</i>	6	6	6	7	3	7	4	5	5	6	1	3
<i>% Reduction</i>	40	38	35	39	18	32	33	28	29	33	8	14
<i>(N)/(A)*</i>	N	N	N	N	N	<b>A</b>	N	N	N	N	N	<b>A</b>

\*N- Normal; A-Abnormal

In 10 of the 12 cases the extent of improvement was such that the pronation could now be classified as normal. In Table 4.60 subject 12 had a left foot pronation of 17° post intervention. This exceeds the norm of 14° and is accordingly classified as abnormal

pronation. In terms of right foot pronation, both subjects six and 12 have pronation which exceeds  $14^\circ$  and both are accordingly classified as abnormal pronation. The extent of improvement in the degree of pronation for these two subjects was between 23% and 32%. These improvements in both the subjects are considered to be significant, although the final measurements are not quite in the normal range. The correlation coefficient between the pre-intervention and post intervention values is a significant 0.86.



**Figure 4.16: Normalisation of hind foot pronation**

Figure 4.16 provides a graphical picture of how the abnormal pronation has been reduced in order to fall within  $\pm 3 \sigma$  pronation interval that could be considered as normal.

#### **4.7.3.2. Discussion**

According to Hintermann & Nigg (1998) over pronation during the later stages of the stance phase of running is a specific biomechanical abnormality that may cause running injuries, not only in the foot and ankle, but also higher up in the lower limbs. The results of this study however indicate the root cause of such injuries may be linked to underlying problems that exist in the myofascial web. Tightness in the clinical significant muscles leads to over pronation which in turn causes secondary effects as discussed above. Over pronation may therefore be a consequence of an already existing compromise in the effective length of the fascial chain.

This study showed that the application of specific soft tissue mobilizing techniques combined with a home stretching programme produced a significant reduction in the





degree of hind foot pronation. In addition, all the subjects with fascia related injuries experienced a total relief of symptoms. These included the five subjects who complained of medial shin pain, four subjects with lateral thigh pain and one subject with plantar foot pain, who were respectively diagnosed with medial tibial periostitis, iliotibial band syndrome and plantar fasciitis. The treatment of the symptoms was however not the objective of the study and the participants were thus not followed up in order to assess the longer term effects of these interventions. Releasing the tight ‘clinical significant’ muscles led to the release of the constrictions of the fascial web which in turn increased the available effective length of the myofascial chain. This reduced the stresses in the chain which led to the normalisation of the biomechanical angles.

#### **4.7.4. Conclusion**

The experimental research is in conclusion very supportive of the new theoretical perspectives developed during the case study research. In the following section an additional two case studies will be discussed. These case studies were embarked on as a final step towards the validation of the interventions developed and the supportive theoretical framework.

### **4.8. RESEARCH VALIDATION**

#### **4.8.1. CASE STUDY 7**

##### ***4.8.1.1. The subject***

The subject in Case Study 7 was a 37 year old male athlete who has been complaining of symptoms of CPCS in his left calf for more than a year. He participated in road races of 10 kilometre distances as well as duathlon events (running and cycling). He runs at a pace of slightly slower than six minutes a kilometre.



### ***Inclusion criteria***

He experienced pain in his left calf with every run during the previous year. The pain commenced after approximately a kilometre of running. This pain forced him to complete the race by walking. The pain was localized to a small area two thirds down the posterior aspect of the left calf, but whilst running, the pain would spread anteriorly around the lower leg. He rated the intensity of the pain as a 70 on a 100mm VAS.

### ***Exclusion criteria***

He had a sonar scan of the lower left leg done on the 11<sup>th</sup> of May 2007. The results of the showed no abnormalities (tears) of the gastrocnemius- or soleus muscle or the achilles tendon. There was an area of local thickening of the fascia between the medial gastrocnemius- and the soleus muscle, but it was not pressure sensitive in this area.

#### **4.8.1.2. Research procedure**

##### ***Subjective assessment – Interview***

##### ***Running history***

This subject had been competing in running events since he can remember. He participated in road races varying in distance between 5 kilometres and 21 kilometres and started taking part in duathlon events during the last couple of years. He recently joined a cross training group of athletes, alternating running and mountain bike training as part of their weekly training programme. At the time of his inclusion into the study, he couldn't run at all and he was wearing a neutral running shoe (Assics TN 635 Nimbus). He used different shoes (clip-on) for cycling.

##### ***Previous running injuries***

He had no previous running injuries.



### ***Current symptoms***

He felt the pain in his left calf for the first time a year ago during the Kentron 10 km race. He had to walk the last kilometre. Since 2007, he experienced pain in his calf with every run. The pain started after a one kilometre and he had an intensity rating of 70 on a 100mm VAS. The last race he competed in was during the March 2007 where he participated in a duathlon event at the Cradle of Humankind. This duathlon entailed a 5 km run, a 20 km cycle and another 5 km run. He experienced calf pain the whole time.

### ***History of symptoms and previous treatment received***

He received two physiotherapy sessions for the pain in his left calf, but these two treatments had no effect on his symptoms. He received cross-frictions, massage, needling, ultrasound therapy, interferential therapy, stretches and heat; all applied to the area of the painful left calf. Thereafter, he was referred for a sonar scan of the left lower leg. Apart from a small, localized area of adhesions between the medial gastrocnemius and soleus muscles, the results of the scan were normal. It was thought that the local adhesions in the fascia around the calf muscles might be a cause for the symptoms of CPCS.

*In summary, the subjective outcome measurements as measured before the intervention:*

- The intensity of pain / discomfort at rest was 70 on the 100mm VAS.
- He did not run at all, i.e. 0 km per week.

### ***Objective assessment – Physical examination***

#### ***Muscle strength tests***

Muscle strength was assessed on 23/05/2007 by a biokineticist on a calibrated Isokinetic dynamometer, using a standardized testing protocol. Significant differences in muscle strength between the left and right sides were established as reflected in Table 4.62

**Table 4.62: Isokinetic dynamometer test results\* prior to intervention: Case Study 7**

<i>Movement tested**</i>	<i>Peak torque (Nm)</i>			<i>Work per repetition (Nm/s)</i>		
	<i>Right</i>	<i>Left</i>	<i>Deficit</i>	<i>Right</i>	<i>Left</i>	<i>Deficit</i>
<i>Hip extensors</i>	164	126	<i>-23</i>	187	149	<i>-20</i>
<i>Hip flexors</i>	107	125	<i>14</i>	118	138	<i>15</i>
<i>Hip internal rotators</i>	49	24	<i>-50</i>	31	27	<i>-13</i>
<i>Hip external rotators</i>	34	22	<i>-36</i>	39	26	<i>-34</i>
<i>Hip abduction</i>	121	141	<i>14</i>	75	110	<i>-32</i>
<i>Hip adduction</i>	42	35	<i>-16</i>	12	3	<i>-78</i>
<i>Knee extensors</i>	134	130	<i>-3</i>	153	160	<i>4</i>
<i>Knee flexors</i>	94	77	<i>-17</i>	115	103	<i>-11</i>
<i>Knee internal rotation</i>	28	24	<i>-14</i>	22	15	<i>-31</i>
<i>Knee external rotation</i>	30	24	<i>-18</i>	24	16	<i>-33</i>
<i>Ankle inversion</i>	38	57	<i>***</i>	43	35	<i>***</i>
<i>Ankle eversion</i>	43	46	<i>***</i>	43	39	<i>***</i>
<i>Ankle plantar flexors</i>	62	83	<i>25</i>	35	31	<i>-12</i>
<i>Ankle dorsi flexors</i>	30	37	<i>19</i>	18	16	<i>-8</i>

\*Statistically significant differences in italic;

\*\* Tested at a speed of 30/30 and with five repetitions

\*\*\* Not determined

***Analysis of running gait, including movement patterns (23/05/2007).***

The abnormal movement patterns are reflected in Table 4.63.

*Note: The gait analysis assessment was verified by 17 post graduate physiotherapists and 5 post graduate biokineticist during a two day course on fascia presented by the researcher.*

**Table 4.63 (a): Running gait analysis: Case Study 7 prior to intervention (Upper body)**

<i>Running gait analysis: Case Study 7 (23/05/07)</i>							
<i>Joint</i>	<i>Weight acceptance</i>		<i>Single leg support</i>			<i>Swing leg advancement</i>	
	<i>Initial contact</i>	<i>Loading response</i>	<i>Mid-stance</i>	<i>Terminal stance</i>	<i>Pre- swing</i>	<i>Initial swing</i>	<i>Terminal swing</i>
Shoulders	Left leg: right shoulder in more extension and retraction than the left shoulder with right leg in this phase.						
Thoracic Region	No thorax rotation was observed towards the right side.		A thoraco-lumbar curve, convex towards the right side, was evident with weight-bearing on the left as well as on the right leg.				
Pelvis	The pelvis on the right side, stayed in a position of posterior rotation throughout the gait.	Due to the posteriorly rotated position of the right pelvis during the loading phase of the right leg, there is a lot of jarring on the right SI-joint area.			Pelvis is still posteriorly rotated on the right side.		Pelvis on the right side does not move into anterior rotation as is seen on the left side.
Hip	The left hip remained in adduction throughout the different phase of the gait, as a result the left leg, took more weight than the right leg (which explains the thoraco-lumbar convex curve towards the right). This also cause more strain on the left calf muscles, especially on the medial side of the lower leg.	The right hip was constantly in more lateral rotation than the left hip.		The push-off of the right hip also took place from out of a position of lateral hip rotation on the right.	More lateral rotation on the right side.	From this position to mid swing, there is an increased amount of medial rotation on the right side in order to counter act for the initial increased lateral rotation on the right side.	

**Table 4.63 (b): Running gait analysis: Case Study 7 prior to intervention (Lower body)**

<i>Running gait analysis: Case Study 7 (23/05/07)</i>								
<i>Joint</i>	<i>Weight acceptance</i>		<i>Single leg support</i>			<i>Swing leg advancement</i>		
	<i>Initial contact</i>	<i>Loading response</i>	<i>Mid-stance</i>	<i>Terminal stance</i>	<i>Pre- swing</i>	<i>Initial swing</i>	<i>Mid-swing</i>	<i>Terminal swing</i>
Knee	Almost full knee extension on both sides.	The degree of knee extension decreases a little on both sides.	The degree of extension in both knees increase again.	The tibias move into more external rotation with the knees in extension.	Both knees are extended with a degree of external rotation of the tibias.	The right knee in a position of more flexion in comparison to the left knee shortly after the push-off phase. This is most probably necessary because of the increased posterior rotation of the pelvis on the right side and the increased degree of lateral flexion of the right leg in order to swing the leg forwards.	Both knees: flexion and internal rotation.	Both knees: extension and external rotation.
Ankle	The subject runs very flat footed. There is hardly any heel-toe action.	Both sides: shift the weight to the medial side of the foot (move into hind foot pronation).	The right foot is also in a position of more abduction because of the increased degree of lateral rotation at the right hip.	Both feet move into a position of hind foot pronation.	Both feet are in a neutral position with plantar flexion.	The right foot has a weaker push-off in comparison to the left, most probably also because of the position of the right foot (more abduction). The left calf muscles are therefore definitely working harder. The problem is: looking at the results of the Cybex tests: the left calf muscles that seem to work harder have tested the weakest! (Although its work per repetition is more in the left leg than in the right leg).	Both sides: slight inversion and plantar flexion.	Both sides: slight inversion and plantar flexion.



### ***Thorax Area***

No thorax rotation was observed towards the right side. A thoraco-lumbar curve, convex towards the right side, was evident with weight-bearing on the left as well as on the right leg.

### ***Pelvis***

The pelvis on the right side, stayed throughout the gait in a position of posterior rotation. Because of the posteriorly rotated position of the right pelvis during the weight acceptance phase on the right hip, it seems as though there is a lot of jarring on the right SI-joint area during this phase.

### ***Hips***

The left hip remained in a position of adduction throughout the different phase of the gait, which means that the left leg, took more weight than the right leg. This partly explains the thoraco-lumbar convex curve towards the right. This can also cause more strain on the left calf muscles, especially those on the medial side of the lower leg. The right hip was constantly in more lateral rotation than the left hip. The push-off of the right hip also took place from out of a position of lateral hip rotation on the right.

### ***Knees***

The right knee is in a position of more flexion in comparison to the left knee shortly after the push-off phase. This is most probably necessary because of the increased posterior rotation of the pelvis on the right side and the increased degree of lateral flexion of the right leg in order to swing the leg forwards.

### ***Ankles***

The subject runs very flat footed. There is hardly any heel-toe action. The right foot is also in a position of more abduction because of the increased degree of lateral rotation at the right hip. The right foot has a weaker push-off in comparison to the left, probably due



to the position of the right foot, i.e. more abduction. The left calf muscles are therefore definitely working harder. The problem is: looking at the results of the Isokinetic dynamometer tests: the left calf muscles that seem to work harder have tested the weakest! (Although its work per repetition is more in the left leg than in the right leg)

A wobble is also occasionally observed in the right foot shortly after push off. This wobble is caused by a relative increased amount of medial rotation in the right hip to counteract the increased lateral rotation in the right hip. At this point in time, the right foot is in a position of midair, and therefore illustrates the wobble.

If one compares the gait analysis to the tightness found in the clinically significant muscles and the muscle strengths as tested with the Isokinetic dynamometer, the whole picture tends to blend in beautifully. The Quadratus lumborum and Gluteus medius muscles were most probably tight on the right hand side because of the position of relative adduction of the left leg. It is clear that the left calf muscles are working harder, since it tested respectively as a two and a one on the tightness scale. The adductors on the left side have also tested tighter than on the right side.

### ***Gait analysis post intervention***

#### ***Thorax Area***

Good symmetry between the left and the right sides with regard to rotation.

#### ***Pelvis***

The right side of the pelvis is in much less posterior rotation. A better symmetry is seen between the two sides. There is a lot less evident jarring in the vicinity of the right SI-joint during the weight bearing acceptance phase on the right side.

#### ***Hips***

The right hip is in less lateral rotation. The line of extension during the push-off phase in the right leg also looks better. There is less sideway movement and a more energy is





spent directing the subject in a more forward line (forward drive). The movement patterns in the hips are a lot smoother and more symmetrical.

### ***Knees***

There is now an equal degree of knee flexion.

### ***Ankles***

The subject still runs very flat footed, but the push off with the right leg is a lot better and the placing of both feet is now very similar.

### ***Toes***

The muscle imbalances according to the Isokinetic dynamometer tests have definitely improved in terms of the hip rotations, hip flexors and extensors as well as the plantar flexors.

### ***Biomechanical angles (23/05/2007)***

The biomechanical angle measurements of 23/05/2007 are provided in Table 4.64.

**Table 4.64: Biomechanical angles: Case Study 7 before intervention.**

<b><i>Movement</i></b>	<b><i>Joint Range of Movement (degrees)*</i></b>
<i>Hind foot pronation left</i>	15,67
<i>Hind foot pronation right</i>	14,17

*\*Averages of six measurements*

### ***Soft tissue palpation (clinically significant muscles)***

All the clinically significant muscles were palpated for tightness and spasm and rated on a scale from 0 to 2. These outcomes are reflected in Table 4.65.

**Table 4.65: Tightness of clinically significant muscles: Case Study 7 prior to intervention (Date: 23/05/2007)**

<i>Muscle</i>	<i>Left Side</i>			<i>Right Side</i>			<i>Muscle</i>	<i>Left Side</i>			<i>Right Side</i>		
	<i>0</i>	<i>1</i>	<i>2</i>	<i>0</i>	<i>1</i>	<i>2</i>		<i>0</i>	<i>1</i>	<i>2</i>	<i>0</i>	<i>1</i>	<i>2</i>
<u><i>Posterior fascia links</i></u>							<u><i>Anterior fascia links</i></u>						
<i>Levator scapula</i>			2		1		<i>Sternocleidomastoid</i>			2		1	
<i>Trapezius</i>			2		1		<i>Scalenii</i>			2		1	
<i>Latissimus dorsi</i>		1		0			<i>Pectoralis major</i>			2	0		
<i>Erector spinae</i>			2		1		<i>Pectoralis minor</i>			2	0		
<i>Quadratus lumborum</i>		1			1		<i>Serratus anterior</i>	0			0		
<i>Gluteus medius</i>		1		0			<i>Subscapularis</i>			2		1	
<i>Gluteus maximus</i>		1		0			<i>External oblique</i>		1		0		
<i>Piriformis</i>			2		1		<i>Rectus abdominus</i>	0			0		
<i>Semimembranosus</i>	0					2	<i>Psoas- umbilicus head</i>	0			0		
<i>Semitendinosus</i>			2		1		<i>Psoas- iliac head</i>		1		0		
<i>Biceps femoris</i>	0				1		<i>Psoas- groin</i>		1		0		
<i>Gastrocnemius</i>			2	0			<i>Iliotibial band</i>	0			0		
<i>Soleus</i>		1		0			<i>Vastus lateralis</i>		1		0		
<i>Plantar fascia</i>	0			0			<i>Vastus medialis</i>	0			0		
							<i>Adductor longus</i>	0			0		
							<i>Adductor magnus</i>		1		0		
							<i>Pes anserinus</i>	0			0		

Key: 0= normal fascia

1= tight fascia

2=very tight fascia



#### ***4.8.1.3. Interventions***

All the clinically significant muscles with a relative tightness rating of one or two were mobilized. Myofascial release techniques were applied according to the approach of Barnes (1990) and Mannheim (1994) and trigger point release techniques were applied according to the approach of Travell & Simons (1999) and specific soft tissue mobilization was applied according to Hunter's approach (1998). The subject was seen once a week for eight weeks and was instructed to stretch on a daily basis. Stretches were provided for the following muscles (30 seconds and repeated twice):

- the trapezius
- the levator scapula
- the pectoralis
- the abdominal muscles
- the iliopsoas
- the piriformis
- the hamstrings
- the gastrocnemius and
- the soleus muscles

#### ***Clinical observations during the intervention period***

##### ***Treatment period***

23<sup>rd</sup> May 2007 – 17<sup>th</sup> July 2007

##### ***Interview***

- The intensity of pain/discomfort at rest, and at the end of every training session was plotted on a 100 mm VAS: He had no discomfort for both.
- He ran a weekly distance of 25 kilometres.
- He ran at a pace of less than six minutes per kilometre.

**Physical examination**

**Muscle strength tests**

The strength of the hip extensors, the hip flexors, the hip abductors, the hip adductors, the medial- and lateral rotators of the hip, the knee extensors, the knee flexors, the medial- and lateral rotators of the knee, the invertors and evertors of the ankle as well as the dorsi- and plantar flexors of the ankle were assessed on a calibrated Isokinetic dynamometer, using a standardized testing protocol. The results are provided in Table 4.66.

**Table 4.66: Isokinetic dynamometer test results\* after intervention: Case Study 7**

<i>Movement tested**</i>	Peak Torque (Nm)			Work per repetition (Nm/s)		
	Right	Left	Deficit	Right	Left	Deficit
<i>Hip extensors</i>	180	145	-20	191	126	-34
<i>Hip flexors</i>	126	117	-8	152	122	-20
<i>Hip internal rotators</i>	24	19	-22	22	20	-6
<i>Hip external rotators</i>	30	20	-32	28	20	-29
<i>Hip abduction</i>	106	141	25	79	103	24
<i>Hip adduction</i>	56	66	16	27	23	-15
<i>Knee extensors</i>	163	149	-8	205	276	-14
<i>Knee flexors</i>	94	92	-1	123	121	-2
<i>Knee internal rotation</i>	23	28	19	20	24	17
<i>Knee external rotation</i>	37	26	-30	34	23	-32
<i>Ankle inversion</i>	37	30	-19	19	14	-29
<i>Ankle eversion</i>	26	28	10	16	18	8
<i>Ankle plantar flexors</i>	69	72	4	38	31	-18
<i>Ankle dorsi flexors</i>	28	39	28	15	16	8

\*Statistically significant differences between right and left are given in italics

\*\*Tested at a speed of 30/30 and with 5 repetitions; \*\*\* Not measured

**Table 4.67: Running gait analysis: Case Study 7 after intervention (Date: 17/07/2007)**

<i>Running gait analysis: Case Study 7 (17/07/2007)</i>								
<i>Joint</i>	<i>Weight acceptance</i>		<i>Single leg support</i>			<i>Swing leg advancement</i>		
	<i>Initial contact</i>	<i>Loading response</i>	<i>Mid-stance</i>	<i>Terminal stance</i>	<i>Pre--swing</i>	<i>Initial swing</i>	<i>Mid-swing</i>	<i>Terminal swing</i>
Shoulders	A much better rhythm. Shoulder movements more symmetrical.							
Thoracic region	Equal thoracic rotation towards both sides.							
Pelvis	The pelvis on the right side is much less in posterior rotation.	Lesser degree of posterior rotation of right pelvis during loading phase of the right leg - less jarring of the right SI-joint during this phase.					Pelvis on the right side, move into neutral.	
Hip	Right hip is in less lateral rotation.			Extension is done from a better position; definitely less lateral rotation.				
Knee						The degree of knee flexion is now equal in both knees.		
Ankle	Still very flat footed.	Both sides: shift the weight to the medial side of the foot (hind foot pronation).	Foot placing on the right is better: less in abduction.	Both feet move into a position of hind foot pronation.	Both feet are in a neutral position with plantar flexion.	Better push-off with the right leg.	Both sides: slight inversion and plantar flexion.	Both sides: slight inversion and plantar flexion.



### ***Reassessment of running gait and movement patterns***

Running gait and movement patterns were also reassessed and the differences/improvements in comparison to the previously assessed gait are given in Table 4.67.

### ***Biomechanical measurements***

The biomechanical measurements made on 17/07/2007 are reflected in Table 4.68.

**Table 4.68: Biomechanical angles: Case Study 7 after intervention**

<b><i>Movement</i></b>	<b><i>Joint Range of Movement (degrees)*</i></b>
<i>Hind foot pronation left</i>	10,67
<i>Hind foot pronation right</i>	11,33

\* Averages of six measurements

### ***Soft tissue palpation (clinically significant muscles)***

All the clinically significant muscles were palpated for tightness and spasm and rated on a scale from 0 to 2. These results appear in Table 4.69.

### ***Management***

The week after the first intervention the subject ran a distance of 15.5 km. The first session was a 6 km run and he felt good on completion of the run. The second run was a 4 km run. He felt his left calf after 3 km but could complete the run. The intensity of the pain was rated as a 65 on a 100mm VAS. During the third run, two days later he felt uncomfortable from the start and managed a 3 km run (intensity was 75 on a 100mmVAS). After his third run, the treatments focussed on the tight left sided cervical muscles, subscapularis and external oblique muscles. His rotation towards the right side improved and he had no more discomfort during further runs.

**Table 4.69: Tightness of clinically significant muscles: Case Study 7 after intervention (Date: 17/07/2007)**

<i>Muscle</i>	<i>Left Side</i>			<i>Right Side</i>			<i>Muscle</i>	<i>Left Side</i>			<i>Right Side</i>		
	<i>0</i>	<i>1</i>	<i>2</i>	<i>0</i>	<i>1</i>	<i>2</i>		<i>0</i>	<i>1</i>	<i>2</i>	<i>0</i>	<i>1</i>	<i>2</i>
<u><i>Posterior fascia links</i></u>							<u><i>Anterior fascia links</i></u>						
<i>Levator scapula</i>		1		0			<i>Sternocleidomastoid</i>	0			0		
<i>Trapezius</i>		1		0			<i>Scalenii</i>	0			0		
<i>Latissimus dorsi</i>		1		0			<i>Pectoralis major</i>		1		0		
<i>Erector spinae</i>	0			0			<i>Pectoralis minor</i>	0			0		
<i>Quadratus lumborum</i>	0			0			<i>Serratus anterior</i>	0			0		
<i>Gluteus medius</i>	0			0			<i>Subscapularis</i>	0			0		
<i>Gluteus maximus</i>	0			0			<i>External oblique</i>	0			0		
<i>Piriformis</i>	0			0			<i>Rectus abdominus</i>			2		1	
<i>Semimembranosus</i>	0			0			<i>Psoas- umbilicus head</i>	0			0		
<i>Semitendinosus</i>	0			0			<i>Psoas- iliac head</i>		1		0		
<i>Biceps femoris</i>	0			0			<i>Psoas- groin</i>		1		0		
<i>Gastrocnemius</i>		1		0			<i>Iliotibial band</i>	0			0		
<i>Soleus</i>	0			0			<i>Vastus lateralis</i>	0			0		
<i>Plantar fascia</i>	0			0			<i>Vastus medialis</i>	0			0		
							<i>Adductor longus</i>	0			0		
							<i>Adductor magnus</i>	0			0		
							<i>Pes anserinus</i>	0			0		

Key: 0= normal fascia

1= tight fascia

2=very tight fascia



#### ***4.8.1.4. Synthesis/discussion***

The symptoms of CPCS were accompanied by tightness in the clinically significant muscles as postulated by the revised theoretical model for the pathogenesis of CPCS. The release of the tightness in these muscles by means of soft tissue mobilization techniques did lead to the disappearance of the symptoms. After the interventions the subject was symptom free, although a thickening of the fascia between the medial gastrocnemius- and the soleus muscle was revealed by the sonar scan of the lower left leg.

In terms of the biomechanical measures the subject had abnormal hind foot pronation prior to the interventions. After the interventions the degree of hind foot pronation decreased to below 14° and could thus be classified as normal.

### **4.8.2. CASE STUDY 8**

#### ***4.8.2.1. The subject***

The subject in Case Study 8 was a 38 year old female athlete who participated competitively in road running races. She has been complaining of symptoms of CPCS in her right lower leg since June 2006.

#### ***Inclusion criteria***

She complained of a pain over the anterior-medial (shin) part of her right calf that increased with exercise and decreased with rest.

#### ***Exclusion criteria***

All the other causes of lower leg pain were differentially excluded based on the exclusion criteria described in Chapter 2.





#### **4.8.2.2. Research procedure**

##### ***Subjective assessment – Interview***

###### ***○ Running history***

The subject had been running for a period of 10 years. Her first 42 kilometre race was done two years prior to the interview and her first ultra marathon during April 2006. This was the Loskop Marathon which covered a distance of 50 kilometres. At the time of inclusion into the study, she was running with Assics (TN 661) shoes which were a slight anti-pronation shoe. She ran at a pace of six and a half minutes per kilometre.

###### ***○ Previous running injuries***

Previous injuries that he had encountered included:

- Right hamstring during October/ November 2005.
- Right sided ITB syndrome during March 2006.

###### ***○ Current symptoms***

She complained of a pain over the “shin” of her right leg. It burned like fire and a definite swelling could be seen over the shin of the right lower leg. She described the intensity of the pain as a 75 on a 100mm VAS. She has only managed a 4 km run during the whole of 2007 and this was during April 2007. It was just too painful to run.

###### ***○ History of symptoms and previous treatment received***

She ran the Loskop Marathon during April 2006, after which she rested for a period of 2 months. When she resumed her running, she became aware of the pain in her right shin. During September 2006, she received two physiotherapy treatments for this condition, consisting of massage, ultrasound therapy, interferential therapy and stretches. The degree of pain was no better after the two sessions. During October 2006, she received a



further 2 sessions of needling. The pain was again not reduced by these two treatment sessions. She then decided to rest the injury for a longer period of time. With regular intervals she tried to run around the block to test the calf. The first time it always felt good, but the second time round she was aware of her calf and when she tried the third time, the calf would burn, swell up. The level of pain she rated as a 75 on a 100mm VAS. She finally decided to rest the calf until 2007. She tried to run around the block once, but it was sore immediately. The only other run she did during 2007 was a four kilometre run during April and again, the calf was very sore. She then went to a podiatrist who said that her right leg was shorter than her left and she needed an insert to raise her right foot. She ran once with the shoes with the insert in and the pain in her right calf was even worse.

In summary, the subjective outcome measures before the intervention were as follows:

- She had no pain/discomfort at rest
- The amount of pain/discomfort whilst running was 75 on a 100mm VAS.
- She averaged a weekly distance of 0 kilometres per week.

#### ***Objective assessment – Physical examination***

##### ○ ***Muscle strength tests***

*Muscle strength* was measured by a biokineticist on 04/07/2007 on a calibrated Isokinetic dynamometer, using a standardized testing protocol. The results are reflected in Table 4.70.

**Table 4.70: Isokinetic dynamometer test results\* prior to intervention: Case Study 8**

<i>Movement tested**</i>	<i>Peak torque (Newton-meter)</i>			<i>Work per repetition(Nm/s)</i>		
	<i>Right</i>	<i>Left</i>	<i>Deficit</i>	<i>Right</i>	<i>Left</i>	<i>Deficit</i>
<i>Hip extensors</i>	38	165	<i>77</i>	-5	174	<i>103</i>
<i>Hip flexors</i>	165	95	<i>-43</i>	264	155	<i>-42</i>
<i>Hip internal rotators</i>	66	22	<i>-67</i>	14	27	<i>50</i>
<i>Hip external rotators</i>	71	19	<i>-73</i>	27	22	<i>-20</i>
<i>Hip abduction</i>	100	83	<i>-18</i>	75	52	<i>-31</i>
<i>Hip adduction</i>	20	35	<i>42</i>	0	9	0
<i>Knee extensors</i>	188	178	-6	199	216	8
<i>Knee flexors</i>	71	71	0	76	102	<i>25</i>
<i>Knee internal rotation</i>	16	18	8	15	16	8
<i>Knee external rotation</i>	20	20	0	18	22	<i>19</i>
<i>Ankle inversion</i>	61	35	<i>-42</i>	65	37	<i>-44</i>
<i>Ankle eversion</i>	58	38	<i>-35</i>	68	47	<i>-30</i>
<i>Ankle plantar flexors</i>	28	49	<i>42</i>	11	26	<i>58</i>
<i>Ankle dorsi flexors</i>	18	28	<i>38</i>	8	15	<i>45</i>

\*Significant differences are given in italic; \*\*Tested at a speed of 30/30 with 5 repetitions

Significant muscle imbalances existed between the left and the right sides of his body.

○ *Analysis of running gait, including movement patterns*

The following movement patterns listed in Table 4.71 deviated from the normal/ideal running patterns:

- She in general, had a very irregular and uneven running rhythm.
- She had abnormal stress on the link in her right side. The right hip which was in a position of medial rotation and extension with the push-off (twisting the right gluteus maximus muscle), but at the same time, the pelvis on the left side dipped inferiorly with the weight-bearing on the right leg.

**Table 4.71(a): Running gait analysis: Case Study 8 prior to intervention (Upper body)**

<i>Running gait analysis: Case Study 8:06/07/07</i>								
<i>Joint</i>	<i>Weight acceptance</i>		<i>Single leg support</i>			<i>Swing leg advancement</i>		
<i>Phase</i>	<i>Initial contact</i>	<i>Loading response</i>	<i>Mid-stance</i>	<i>Terminal stance</i>	<i>Pre-swing</i>	<i>Initial swing</i>	<i>Mid-swing</i>	<i>Terminal swing</i>
Shoulders	Right leg: left shoulder, moves into more abduction with extension in comparison to the right. Left leg: the left shoulder is in more protraction and medial rotation than the right shoulder (caused by tightness in pectoralis major and minor).							
Thoracic region	Very little thoracic rotation with rotation to the right more than to the left.							
Pelvis			With weight-bearing on the right side, the left pelvis sinks through.					
Hip			The left hip is in more adduction than the right hip.	Right hip push off: in extension and medial rotation.				

**Table 4.71(b): Running gait analysis: Case Study 8 prior to intervention (Lower body)**

<i>Running gait analysis: Case Study 8:06/07/07</i>								
<i>Joint</i>	<i>Weight acceptance</i>		<i>Single leg support</i>			<i>Swing leg advancement</i>		
<i>Phase</i>	<i>Initial contact</i>	<i>Loading response</i>	<i>Mid-stance</i>	<i>Terminal stance</i>	<i>Pre- swing</i>	<i>Initial swing</i>	<i>Mid-swing</i>	<i>Terminal swing</i>
Knee	Almost full knee extension on both sides.	The degree of knee extension decreases a little on both sides.	The degree of extension in both knees increase again.	The tibias move into more external rotation with the knees in extension.	Both knees are extended with a degree of external rotation of the tibias.		Both knees: flexion and internal rotation.	Both knees: extension and external rotation.
Ankle	Hardly any heel toe action.		Right foot is in a position of more abduction than the left.		Weak push-off on both sides.			

***Biomechanical angles 06/07/2007***

The outcomes of the measured biomechanical angles are reflected in Table 4.72.

**Table 4.72: Biomechanical angles: Case Study 8 before intervention**

<b><i>Movement</i></b>	<b><i>Joint Range of Movement (degrees)*</i></b>
<i>Hind foot pronation left</i>	18,5
<i>Hind foot pronation right</i>	19

*\*Averages of six measurements*

○ ***The tightness of the clinically significant muscles***

All clinically significant muscles were palpated for tightness and rated on a scale of 0 – 2. The outcomes are provided in Table 4.73.

**4.8.2.3. Intervention**

All the tight clinically significant muscles were released. Different release techniques were used, depending on the muscle involved. Myofascial release techniques were used according to Barnes (1990) and Manheim (1994). Trigger point release techniques were done according to Travell & Simons (1999) and specific soft tissue mobilization were done according to Hunter (1998). The subject was seen once a week during eight weeks and instructed to stretch the following muscles on a daily basis. Each stretch had to be held for 30 seconds and repeated twice:

- the trapezius
- the levator scapula
- the pectoralis
- the abdominal muscles

**Table 4.73: Tightness of clinically significant muscles: Case Study 8 prior to intervention (Date: 06/07/2007)**

<i>Muscle</i>	<i>Left Side</i>			<i>Right Side</i>			<i>Muscle</i>	<i>Left Side</i>			<i>Right Side</i>		
	<i>0</i>	<i>1</i>	<i>2</i>	<i>0</i>	<i>1</i>	<i>2</i>		<i>0</i>	<i>1</i>	<i>2</i>	<i>0</i>	<i>1</i>	<i>2</i>
<b><u>Posterior fascia links</u></b>							<b><u>Anterior fascia links</u></b>						
<i>Levator scapula</i>			2		1		<i>Sternocleidomastoid</i>	0			0		
<i>Trapezius</i>		1			1		<i>Scalenii</i>	0			0		
<i>Latissimus dorsi</i>	0			0			<i>Pectoralis major</i>		1		0		
<i>Erector spinae</i>	0			0			<i>Pectoralis minor</i>	0			0		
<i>Quadratus lumborum</i>		1		0			<i>Serratus anterior</i>	0			0		
<i>Gluteus medius</i>			2		1		<i>Subscapularis</i>		1		0		
<i>Gluteus maximus</i>	0				1		<i>External oblique</i>	0			0		
<i>Piriformis</i>		1				2	<i>Rectus abdominus</i>	0			0		
<i>Semimembranosus</i>	0			0			<i>Psoas- umbilicus head</i>					1	
<i>Semitendinosus</i>	0			0			<i>Psoas- iliac head</i>	0			0		
<i>Biceps femoris</i>	0			0			<i>Psoas- groin</i>	0			0		
<i>Gastrocnemius</i>	0				1		<i>Iliotibial band</i>		1			1	
<i>Soleus</i>	0			0			<i>Vastus lateralis</i>		1		0		
<i>Plantar fascia</i>	0			0			<i>Vastus medialis</i>	0			0		
							<i>Adductor longus</i>	0			0		
							<i>Adductor magnus</i>	0			0		
							<i>Pes anserinus</i>	0			0		

Key: 0= normal fascia

1= tight fascia

2=very tight fascia



- the iliopsoas
- the piriformis
- the hamstrings
- the gastrocnemius and
- the soleus muscles

The following clinical observations were made during the intervention period:

○ *Treatment Period (6 July 2007 to 23 August 2007)*

Her running gait was assessed running barefoot, with running shoes on and with the running shoes and the added insert in the right running shoe. The running gait patterns of the right leg deviated more from the norm with the insert than without on the video-clip. It was decided not to tamper with the insert immediately but to continue with the interventions and to reassess the symptoms the week thereafter.

During the first week after her first intervention, she managed to run three times. During the first run, she became aware of a sensation (3 on a 100mm VAS) after a distance of two kilometres but it disappeared again whilst running and she completed her four kilometre run with no symptoms. She ran for the second time that week (three days later). She ran a distance of three kilometres on a treadmill and became aware of a lumbar left sided discomfort. Take note: no calf discomfort. Still in the same week (two days later), she ran three kilometres on the treadmill, but then complained again of a left lumbar discomfort and the right shin pain (an intensity of 28 on a 100mm VAS). With palpation it was found that the area over her right posterior-lateral calf as well as the quadratus lumborum muscle on the left was very tight. She was instructed to remove the insert from her right running shoe.

After she removed the insert from her right shoe and replaced it with the normal insert with which the shoe was bought, she had no more discomfort of her right calf during any





of her other runs during the intervention period of eight weeks. The discomfort in her left lower back also disappeared during the following week.

Two weeks later she ran two days in succession without any pain. During the middle of the intervention period, the subject developed a bad bronchitis and could not run for that week; but she ran again the weeks thereafter and remained symptom free.

### ***Final Assessment Results (23/08/2007)***

#### ***○ Interview***

- She had no discomfort/ pain at rest or during or after a run anymore.
- She ran a weekly distance of 17 kilometres.
- She is enjoying her running again for the first time since the Loskop Marathon.

### ***Physical examination***

#### ***○ Re-assessment of running gait and movement patterns***

The following movement patterns deviated from the normal/ ideal running patterns (see Table 12.2.5 for more detail):

#### ***○ The tightness of the clinically significant muscles***

The tightness of the clinically significant muscles was again rated on a scale of 0 – 2.

**Table 4.74: Running gait analysis: Case Study 8 after intervention (Date: 23/08/2007)**

<i>Running gait analysis: Case Study 8:06/07/07</i>								
<i>Joint</i>	<i>Weight acceptance</i>		<i>Single leg support</i>			<i>Swing leg advancement</i>		
<i>Phase</i>	<i>Initial contact</i>	<i>Loading response</i>	<i>Mid-stance</i>	<i>Terminal stance</i>	<i>Pre--swing</i>	<i>Initial swing</i>	<i>Mid-swing</i>	<i>Terminal swing</i>
Shoulders	Better rhythm; equal shoulder movements.							
Thoracic region	Equal rotation to both sides. Rotation has increased to both sides.							
Pelvis			The left pelvis dips much less with the weight on the right leg.					
Hips			Right leg in less adduction.	Right hip in less medial rotation.				
Knees								
Ankles	Still hardly any heel-toe action.		Right foot in much less abduction.					

**Table 4.75: Tightness of clinically significant muscles: Case Study 8 after intervention (Date: 23/08/2007)**

<i>Muscle</i>	<i>Left Side</i>			<i>Right Side</i>			<i>Muscle</i>	<i>Left Side</i>			<i>Right Side</i>		
	<i>0</i>	<i>1</i>	<i>2</i>	<i>0</i>	<i>1</i>	<i>2</i>		<i>0</i>	<i>1</i>	<i>2</i>	<i>0</i>	<i>1</i>	<i>2</i>
<b><u>Posterior fascia links</u></b>							<b><u>Anterior fascia links</u></b>						
<i>Levator scapula</i>		1		0			<i>Sternocleidomastoid</i>	0			0		
<i>Trapezius</i>	0			0			<i>Scalenii</i>	0			0		
<i>Latissimus dorsi</i>	0			0			<i>Pectoralis major</i>	0			0		
<i>Erector spinae</i>	0			0			<i>Pectoralis minor</i>	0			0		
<i>Quadratus lumborum</i>		1		0			<i>Serratus anterior</i>	0			0		
<i>Gluteus medius</i>		1		0			<i>Subscapularis</i>	0			0		
<i>Gluteus maximus</i>	0			0			<i>External oblique</i>	0			0		
<i>Piriformis</i>	0				1		<i>Rectus abdominus</i>	0			0		
<i>Semimembranosus</i>	0			0			<i>Psoas- umbilicus head</i>	0			0		
<i>Semitendinosus</i>	0			0			<i>Psoas- iliac head</i>	0			0		
<i>Biceps femoris</i>	0			0			<i>Psoas- groin</i>	0			0		
<i>Gastrocnemius</i>	0			0			<i>Iliotibial band</i>	0			0		
<i>Soleus</i>	0			0			<i>Vastus lateralis</i>	0			0		
<i>Plantar fascia</i>	0			0			<i>Vastus medialis</i>	0			0		
							<i>Adductor longus</i>	0			0		
							<i>Adductor magnus</i>	0			0		
							<i>Pes anserinus</i>	0			0		

Key: 0= normal fascia

1= tight fascia

2=very tight fascia

○ **Biomechanical angles (Date: 23/08/2007)**

The measurements of the biomechanical angles made on 23/08/2007 are reflected in Table 4.76.

**Table 4.76: Summary of biomechanical angles: Case Study 8**

<i>Movement</i>	<i>Joint Range of Movement (degrees)*</i>
<i>Hind foot pronation left</i>	9,2
<i>Hind foot pronation right</i>	9,8

\* *Averages of six measurements*

#### **4.8.2.4. Synthesis/discussion**

The symptoms of CPCS were accompanied by tightness in the clinical significant muscles as postulated by the revised theoretical model for the pathogenesis of CPCS. The release of the tightness in these muscles by means of soft tissue mobilization techniques did lead to the disappearance of the symptoms. After the interventions the subject was symptom free.

In terms of the biomechanical measures the subject had abnormal hind foot pronation prior to the interventions. After the interventions the degree of hind foot pronation decreased to below 14° and could thus be classified as normal.

#### **4.8.3. Reflection**

The penultimate section of this chapter deals with reflection on the knowledge gained from both the literature and practical experimentation and the implications that this have on the new or revised theoretical model that has been developed for the pathogenesis of CPCS. These reflections will be cryptic as these issues had been covered in detail in the preceding parts of the thesis.

#### 4.8.3.1. *Knowledge from the literature study*

The most revealing part of the literature research in terms of successful interventions, was the fact that the only interventions that had some degree of success, was the surgical release of the fascia. This led to a reduction in the posterior compartment and the normalisation of the blood flow to the area resulted in the disappearance of the symptoms. These interventions were however not always successful in the longer term as the symptoms often reoccurred once the subject started running again. This fact provides an indication of:

- The fact that the root cause of the problem had not been resolved;
- That the solution to the problem is fascia related in the sense that the surgical release of the fascia leads to the clearance of the symptoms;
- That by implication the solution to the problem lies in the release of the pressure in the posterior compartment; and
- The fact that the symptoms reappear once the subject starts running again provides an indication that the fascia plays a role in the pathogenesis of the condition.

The subject in Case Study 4 had a similar experience as the latter. After surgical intervention his symptoms cleared only to return when he started running again.

The literature also revealed that conventional physiotherapy interventions were limited to the calf area. None of the interventions or the supporting theoretical models took into account the continuous and non-elastic nature of the involved fascia. These findings from the literature were supported by the findings of the exploratory research phase where the traditional conventional physiotherapy interventions did not lead to any noticeable improvements.

The investigation into the nature of the fascia revealed aspects which had a profound implication on the theoretical perspectives of the condition. Although the role of non-compliant fascial boundaries in the perpetuation of the condition is widely recognised, the nature of fascia as such had not been previously explored in this context. The continuous nature of fascia and its relatively inelastic has major implications in terms of the pathogenesis as well as the perpetuation of the condition. The most important



being that the root causes of the initiation of CPCS is the stress in the myofascial web and that the source of the stress could indeed lie external to the calf area. If these stresses are not relieved it would appear logical that the symptoms could re-occur during running when stresses in the fascia are accentuated as a result of the greater ranges of motion required.

The clinically significant muscles were identified by the researcher based on the myofascia which links these muscles to that of the calf area. These linkages were established through the second phase of the literature research dealing with the myofascial web. This also enabled the researcher to hypothesise that constrictions in the myofascial web should manifest in tightness in these muscles. Similarly it also enabled the hypothesis that constrictions in the myofascial web would compromise the length of the fascia which in turn should have an effect on biomechanical measures, especially during activities such as running where the accentuated movement patterns will demand a greater availability of the fascia to execute the running motions. In addition to the restrictions on biomechanical movements it will also place abnormal stresses on the fascia web during such exercises which in turn would increase the pressure in the posterior compartment, restrict blood flow, leading to the pathogenesis of CPCS.

#### ***4.8.3.2. Knowledge gained from experimentation***

In all the case studies the symptoms of CPCS were accompanied by tightness in the clinically significant muscles. The release of the tightness in these muscles in all the cases led to the alleviation of the condition and all the subjects could resume their running careers, even those who have had surgical interventions in the past. In contrast to the limited success with the surgical interventions none of the subjects had any problems with the reoccurrence of the symptoms. This implies that the root cause of the problems with all of the subjects had been resolved.

In one instance one of the subjects had a problem with the reoccurrence of symptoms. This was during the treatment process when all the tightnesses were not released effectively yet and the subject was running in loose sand and in a strong head wind which required additional forces from the muscles. Such accentuated force is



accompanied by increased stresses in the myofascial chain which would tend to increase the pressure in the posterior compartment which in turn could trigger the initiation of the condition.

All the subjects had biomechanical abnormalities which are consistent with the causal relationships propagated by the new theoretical model. The fact that these abnormalities showed significant improvement once the tightness in the clinical significant muscles had been released further enhances the credibility of the new theoretical framework. The hind foot pronation of all the subjects returned to normal after the release of the tightness in the clinical significant muscles. With the experiment on hind foot pronation all the subjects showed significant improvement in the extent of pronation with the release of the tightness in these muscle.

The one aspect which in conclusion provides the greatest credibility for the new theoretical model for the pathogenesis of CPCS is the astonishing degree of replication that has been achieved with the interventions, and the systematic elimination of rival theories. According to Yin (1994) even as little as three replications of results is adequate as validation of a theoretical concept supported by a strong theoretical model.

#### **4.8.4. Validation of tightness assessment technique**

In an attempt to validate the assessment technique used for determining the tightness in the clinically significant muscles, a subject with symptoms of CPCS was recruited for the exercise. Two physiotherapists, who regularly utilize soft tissue mobilization techniques, were recruited to assess the tightness of the clinically significant muscles in the subject. Forty three of these muscles were selected on a random basis and each of the physiotherapists assessed the tightness of these muscles independently from each other. The assessments by the researcher was taken as the “correct” assessment and used as a benchmark for the other results. In tables 4.77(a) and 4.77(b), these values are reflected by the blocks with the shading. In spite of the inherent subjectivity of palpation assessments, the extent of correlation between the individual assessments was encouraging.


**Table 4.77 (a): Validation of soft tissue rating process (Posterior fascia links)**

<b>Date</b>	10 February 2007	<b>Legend</b>
<b>Physiotherapist</b>	1;2;3	<ul style="list-style-type: none"> <li>• - single opinion</li> <li>•• - agreement by two</li> <li>••• - consensus opinion</li> <li><span style="display: inline-block; width: 15px; height: 10px; background-color: #d4edda; border: 1px solid #c3e6cb; margin-right: 5px;"></span> Correct assessment</li> </ul>
<b>Subject:</b>	1	

<i>Muscle</i>	<i>Left Side</i>			<i>Right Side</i>		
	<i>0</i>	<i>1</i>	<i>2</i>	<i>0</i>	<i>1</i>	<i>2</i>
<b><u>Posterior fascia links</u></b>						
<i>Levator scapula (1)</i>		••	•		•	••
<i>Levator scapula (2)</i>		•••			••	•
<i>Trapezius (1)</i>		••	•		••	•
<i>Trapezius (2)</i>		••	•		•••	
<i>Latissimus dorsi</i>		••	•	•	•	•
<i>Erector spinae</i>						
<i>Quadratus lumborum (1)</i>			•••		•••	
<i>Quadratus lumborum (2)</i>	•	••			••	•
<i>Quadratus lumborum(3)</i>		•••			•	••
<i>Quadratus lumborum (4)</i>		••	•		••	•
<i>Gluteus medius</i>						
<i>Gluteus maximus</i>						
<i>Piriformis (1)</i>	••	•		••	•	
<i>Piriformis (2)</i>			•••		•••	
<i>Semimembranosus</i>			•••		•••	
<i>Semitendinosus</i>						
<i>Biceps femoris</i>						
<i>Gastrocnemius</i>						
<i>Soleus(1)</i>		••	•			•••
<i>Soleus (2)</i>		•	••		•	••
<i>Quadratus plantae</i>		••	•		••	•



**Table 4.77 (b): Validation of soft tissue rating process (Anterior fascia links)**

<b>Date</b>	10 February 2007	<b>Legend</b>
<b>Physiotherapist</b>	1;2;3	• - single opinion
<b>Subject:</b>	1	•• - agreement by two
		••• - consensus opinion
		 Correct assessment

<i>Muscle</i>	<i>Left Side</i>			<i>Right Side</i>		
	<i>0</i>	<i>1</i>	<i>2</i>	<i>0</i>	<i>1</i>	<i>2</i>
<i>Sternocleidomastoid (1)</i>		•••			••	•
<i>Sternocleidomastoid (2)</i>		•	••	•	••	
<i>Scalenii</i>						
<i>Pectoralis major (1)</i>		••	•			•••
<i>Pectoralis major (2)</i>		••	•			•••
<i>Pectoralis major (3)</i>		••	•			•••
<i>Pectoralis minor</i>		••	•			•••
<i>Serratus anterior</i>						
<i>Subscapularis</i>			•••		••	•
<i>External oblique (1)</i>			•••		••	•
<i>External oblique (2)</i>		•	••		••	•
<i>Rectus abdominus</i>						
<i>Psoas (1)</i>			•••		•••	
<i>Psoas (2)</i>			•••		•••	
<i>Psoas (3)</i>			•••		•••	
<i>Iliotibial band</i>						
<i>Vastus lateralis</i>						
<i>Vastus medialis (1)</i>			•••		•••	
<i>Vastus medialis (2)</i>		•	••		••	•
<i>Adductor longus</i>		•	••		••	•
<i>Adductor magnus</i>						
<i>Pes anserinus</i>			•••		•••	

The number of links that were assessed in the subject totalled 43 links. This provides a total of a 129 links that were assessed between the three physiotherapists. Out of

these 2 links were classified as being tight, while the researcher was of the opinion that they were not. Similarly 2 links were classified as not tight, while the researcher was of the opinion that they were.

The analysis of the data that appears in table 4.77(a) and 4.77(b) is reflected in Table 4.78. As can be seen from the table, in 96.8% of the possible outcomes the three physiotherapists were in agreement on whether a muscle was tight or not. The application of the intervention techniques developed is not so much dependant on the degree of tightness, as on the fact whether the muscle involved is correctly identified as tight or not.

The three physiotherapists were in general in consensus on the majority of the assessments, i.e. 60.5% of the assessments. The conclusion is thus that a general physiotherapist who is familiar with soft tissue techniques should be able to apply the interventions with success. Specific training, which the two physiotherapists were not exposed to, should greatly enhance the ability of the therapist to accurately and with confidence, identify the tight muscles and the extent of tightness in them.

**Table 4.78: Analysis of validation data on tightness**

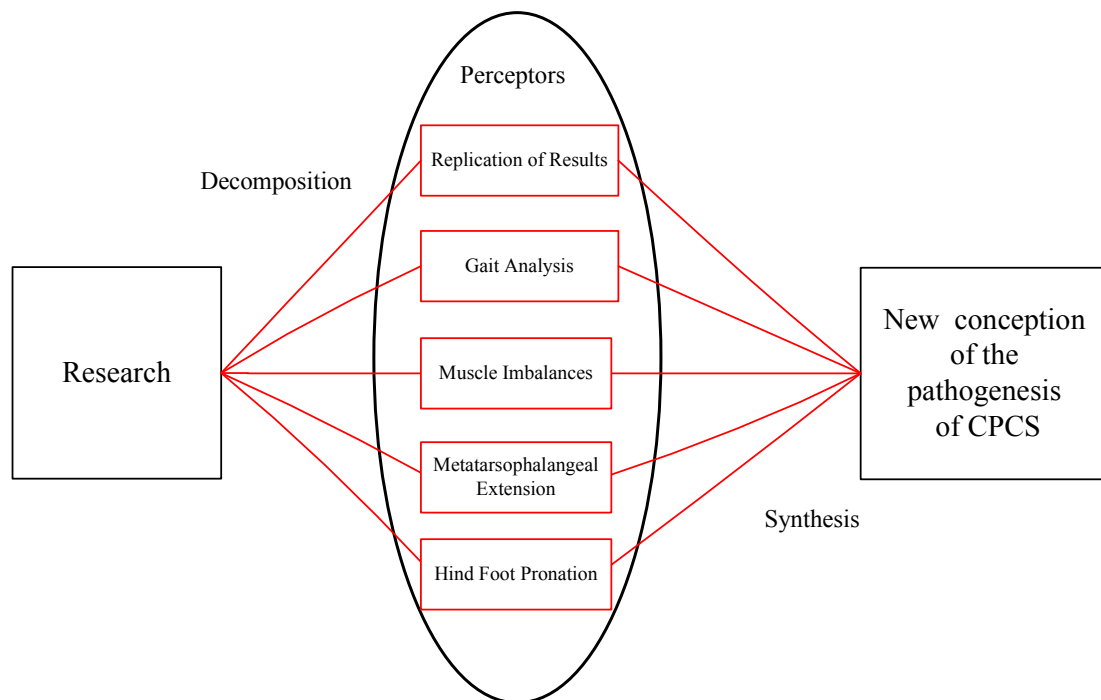
<b>Description</b>	<b>number</b>	<b>%</b>
Total number of links per subject	43	
Grand total of links (43x3)	129	100%
Total number of tight links correctly identified	125	96.8%
Tight links classified as not tight	2	1.6%
Links not tight but classified as tight	2	1.6%
Number of “wrong” classifications	4	3.2%
Consensus on the degree of tightness	78	60.5%

## CHAPTER 5

### CONCLUSIONS AND RECOMMENDATIONS

#### 5.1. INTRODUCTION

The objective of the final chapter is to provide an overview of the research project. It reviews the extent to which the key research objectives have been achieved. It provides the final discussions on the integration of the results that were achieved during the research. The Brunswikian model (Schulz & Tietje, 2002) is used as a conceptual framework for the final integration of results. Through this integration of the various results of the individual research components reflected in Figure 5.1, a new and enhanced understanding of the pathogenesis of CPCS emerge.



**Figure 5.1: Towards a new conception of the pathogenesis of CPCS**

The various research phases of the research project were covered in detail in the individual chapters dealing with each of the research aspects. In this chapter the final perspectives on the research project will be covered. The perspective emanating from the integration of the individual results of the different research phases provides a new understanding of the pathogenesis of CPCS, as well as of the implications which this new perspective has on treatment interventions developed during the course of the



research. Some of the major highlights, short comings and future research questions that need to be addressed will be highlighted.

## **5.2. KNOWLEDGE INTEGRATION**

In this section the knowledge gained during the various sub research projects will be integrated based on the conceptual framework presented by the Brunswikian lens model (Schulz & Tietje, 2002). The aim is to identify those aspects of the research findings of the individual components which enable a clearer and an enhanced picture of the research findings. Central to the integration of the knowledge gained is the new or revised theoretical model that has been developed for the pathogenesis of CPCS.

In Chapter 2 the model by Scholz & Tietje (2002) for the generation of knowledge through case study research has been introduced. In terms of this model three levels of knowledge exist. The first level represents knowledge gained from individual case studies. The knowledge gained on this level is generalised through the creation of a conceptual model on the second level. This model according to them is based on inference drawn from the observations from the individual case studies. This model can be validated by means of its ability to explain outputs from the case study research, such as data, observations, measurements, surveys, documents and expertise, as well as data from the existing body of scientific knowledge in the form of disciplinary propositions.

Contrary to this model, the conceptual model for the pathogenesis of CPCS was based on deductive thinking and the deficiencies in the current theoretical base. In the opinion of the researcher it would not have been possible to formulate the revised theoretical model for the pathogenesis of CPCS through inference made from observations.

### **5.2.1. Replication of results**

The central proposition with each of the case studies was that the symptoms of CPCS will manifest as a result of tightness in the *clinically significant muscles*. This tightness is the result of a decrease in the functional length of the myofascial chain

which reflects in a stiffness/tightness of the *clinically significant muscles*. When the tightness in these muscles is released by means of soft tissue mobilization interventions, the condition will clear and that the subject would be symptom free. In other words, the subject will be pain free and would be able to resume his running career in a normal manner. The results with all eight subjects were successful and through this replication of results the validity of the model was verified.

### **5.2.2. Muscle tightness**

Muscle tightness in the *clinically significant muscles* plays a key role in the revised model for the pathogenesis of CPCS. The identification and classification of these muscles as being significant in the condition of CPCS are one of the key contributions to new knowledge generated by this research. These muscles were identified based on their relationship with the continuous myofascial web which interlinks them with the muscles of the posterior compartment. These muscles were identified based on deductive reasoning from existing anatomical knowledge of fascia, supplemented by clinical verification during the analysis of the subjects during individual case studies.

One of the implications of the *clinically significant muscles* is that tightness in any of these, due to the continuous nature of the myofascial web, could be responsible for the symptoms of CPCS. This implies that the tightness in these muscles could vary from subject to subject, which was indeed the case as observed with the individual case studies.

### **5.2.3. Muscle imbalances**

Tightness of the *clinically significant muscles* lead to a constriction in the length of the myofascial chain which in variably affect the normal functioning of the muscles involved. These results although predictable, do not make any significant contribution to the research per se, other than being consistent with expectations in general. Muscle imbalances per se do not imply that the subject suffers from the symptoms of CPCS, although it could be stated that subjects with symptoms of CPCS would have significant muscle imbalances.



#### **5.2.4. Gait analysis**

Gait analysis as a tool provided the ability to evaluate the effects of CPCS in the subjects in a dynamic manner. Although it is difficult to predict all the effects of CPCS on the dynamic movement patterns, it is relatively easy to motivate the observed abnormal movement patterns based on the theoretical model that has been developed.

The gait analysis was also instrumental to the experimentation with the metatarsophalangeal extension. In retrospect the effect of CPCS on the flexibility at the metatarsophalangeal joint seems obvious. Although the measurement of biomechanical angles was one of the objectives set at the outset, this particular measurement was a direct outflow from the gait analysis conducted. The observations made during the gait analysis were all supportive to the new theoretical constructs that were developed.

#### **5.2.5. Biomechanical measures**

The implication of the continuous and relatively inelastic myofascial web that links the muscles is that stresses in the fascia controls or limits the extent of movement like a puppet on a string. Distortions in the fascia web will manifest in other areas such as the posterior compartment, which could indeed trigger the initiation of CPCS as a result of accentuated forces induced in the posterior compartment during running. The revised model for the pathogenesis of CPCS proposes that this is the most likely cause of the initiation of the condition which is a more credible explanation than the previous model which ascribed the cause to a general *muscle exertion*. Although no evidence could be found to the contrary, the initiation of the condition warrants additional research.

#### ***Metatarsophalangeal extension***

The constraint that compromised myofascia places on movement patterns is adequately demonstrated by this experiment. The restricted available length of the myofascia restricts the movements of the soft tissue. Therefore the subject goes higher



onto his toes in order to compensate for the reduction in the functional length of the myofascia web which restricts the flexibility in the ankle. The release of the myofascia through soft tissue mobilization techniques increases the available length of the myofascia which in turn leads to the normalisation of movement patterns.

### *Hind foot pronation*

Although all the subjects in the hind foot pronation experiment did not suffer from symptoms of CPCS, they all had abnormal hind foot pronation. The experiment however adequately illustrates the causal relationships between the intertwined myofascial web and abnormal movement patterns. The compromised fascia is reflected by the tightness in the clinically significant muscles, and once these tightnesses are released, movement patterns are normalised.

The experiment however also illustrated how the muscle tightness could be identified by relatively inexperienced health practitioners. It also provides evidence that the interventions could be applied by therapists, other than the researcher.

### **5.3. THE RESEARCH PROBLEM**

The research problem that had been investigated was the fact that no known conservative interventions existed which provided reputable successes in the treatment of the symptoms of CPCS (Fraipont & Adams, 2003). The only interventions with some positive responses in the literature were based on the surgical release of the fascia.

This fact, coupled to the lack of emphasis on the nature of myofascia, led to the search for a solution external to the calf area. The revised or modified model developed for the pathogenesis of CPCS were subjected to a rigorous process in search of the replication of results as well as the testing of rival theories that could have caused the initiation and perpetuation of the condition.



The emerging model for the development of CPCS formed the basis the development of the conservative treatment interventions which consisted of the release of the tightness in the so called or defined clinically significant muscles. The application of these techniques resulted in a spectacular success of all the subjects involved with the research project. All subjects were pain free at the end of the programme and could pursue their passion for running.

In conclusion one can state that there is overwhelming evidence that the research problem has been resolved.

#### **5.4. THE RESEARCH AND INVESTIGATIVE QUESTIONS**

In the literature research it was seen that although the condition of CPCS is well defined, it is not easy to identify or diagnose the condition. Some researchers such as Fraipont & Adamson (2003) even consider the measurement of the compartmental pressures as essential for establishing and accurate diagnoses. In the introductory chapter it has been stated that the healthcare practitioner thus often has to deal with a situation where the symptoms are clearly manifested, but the diagnosis of the underlying cause is not that obvious. One of the objectives of the research was to establish a practical methodology for the health professional to deal with this possible ambiguity. The focus of the research has thus been in dealing with these symptoms and the research question as was seen, was thus formulated as:

*Can a successful conservative treatment for runners with symptoms of CPCS be developed?*

From a theoretical perspective the research however focussed on the causal relationships of the condition. The lack of success with conservative interventions was considered to be due to a lack of understanding of the fundamental factors that contributed to the development and perpetuation of the condition. The relative successes that have been achieved with the surgical releases were a major element in the development of the revised theoretical model for the pathogenesis of CPCS, which enabled the development of the interventions. In the humble opinion of the researcher





it was thus possible to develop a successful conservative treatment intervention for the symptoms of Chronic Posterior Compartment Syndrome.

The lack of successful conservative interventions could thus be contributed to gaps that existed in theoretical base which reflects the understanding of the condition. Although the role of fascia in the context of the non-compliant border which leads to the pressure build up in the compartment is widely recognised, the continuous nature and non-elasticity of fascia had not been explored. Once this was identified it led to the exploration of the concept that the condition could be triggered externally to the calf compartments. This in turn led to the identification of the clinically significant muscles which are encapsulated by the continuous myofascial web is considered as the second most important contribution of this research.

It was argued that distortions of the myofascia in the clinically significant muscles would exert stresses and associated pressures along the myofascial web. This insight that the pressure in the calf compartments could be triggered externally, led to the development of the improved theoretical framework for the pathogenesis of the condition. The condition was thus triggered by an injury and not by some vague muscle exertion as proposed by the old theoretical model developed by Clanton & Solcher (1994).

The distortions in the myofascial web could be determined by means of the palpation of the tightness in the clinically significant muscles. Once these were identified it was possible to normalise the condition through the application of soft tissue mobilization techniques.

The application of the newly developed conservative treatment intervention was highly successful as demonstrated by a hundred percent replication of successful results with eight subjects. Through the replication logic of Yin (2003) the rival theories for the condition were systematically eliminated and the replication of the results provided adequate evidence to support the validity of the new model.

One of the investigative questions was whether other biomechanical deviations/abnormalities play a role in the perpetuation of the condition. The results of



the research suggest that such biomechanical deviations are indeed attributable to the condition, and not the other way around. It was also established that the successful treatment of the condition indeed normalised such biomechanical deviations.

The Brunswikian model in Figure 5.1 reflects the various components that were investigated in the research. These individual perspectives ultimately led to a richer understanding of the condition of CPCS. What is considered as significant is the fact that none of these perspectives or insights contradicts the new theoretical model that has been developed for the pathogenesis of CPCS. They all indeed support this new perspective.

## **5.5. KEY RESEARCH OBJECTIVES**

The key research objectives set for the research were all to a greater or lesser degree achieved. The first objective was to develop an enhanced theoretical framework as basis for the treatment of CPCS in runners. The new theoretical model that was developed formed the basis for the second objective which was to develop a successful conservative methodology for the treatment of CPCS in runners.

The new theoretical model is considered as a significant contribution to the current body of knowledge. More significantly however is the underlying implications which the inclusion of the myofascial web in the contextual framework of chronic injuries in general, holds for the development of conservative treatment interventions.

This approach makes a significant contribution to the existing body of knowledge with specific reference to a more holistic approach towards the clinical treatment of myofascial system related dysfunctions in the field of physiotherapy.

As a practising therapist, the most rewarding aspect of the research is the ability to solve the frustrating effects of CPCS in both the runner and the therapist who has to deal with the problem. As the experiment with the hind foot pronation has demonstrated, it enables even the relatively inexperienced practitioner to successfully deal with the problem.



## **5.6. SIGNIFICANCE OF THE RESEARCH**

The successful conservative treatment for the symptoms of CPCS is considered as a major breakthrough. The current physiotherapy approaches reported on in the literature provide no lasting success, whilst the documented surgical option has limited success rates, especially with regard to the posterior compartment.

A successful conservative approach, alleviating the symptoms of CPCS, could form the basis for the successful treatment of other chronic fascia-related injuries such as iliotibial band syndrome, plantar fasciitis and CCS of the other compartments of the lower leg, as well as others such as hamstring injuries where the individual muscle has a great many myofascial links to other areas.

The approach that had been developed for the treatment of the symptoms of CPCS has positive economical and psychological implications. Consequent upon successful treatment through manual, conservative means, costs are saved on surgery, hospitalisation, post-operative rehabilitation and many promising athletes will be able to continue their sport careers.

According to Harden (2007) the research in myofascial pain syndrome (MPS) is essentially at a standstill due to the lack of common, standard diagnostic criteria. In the opinion of the researcher this fact could to a large extent be contributed to the fact that the links which exist between muscles, ligaments and the fascia has been ignored in the theoretical base for the treatment of such related musculoskeletal problems. The application of these interdependencies could make a significant contribution towards the diagnostics of such problems and the treatment thereof.

## **5.7. THE RESEARCH PROCESS**

The use of the case study approach advocated by Yin (2003) was especially rewarding. His emphasis on an underlying theoretical model on which propositions can be based, forced the research to explore the validity of the current theoretical base



on which treatment interventions were based. The development and testing of rival theories that can be postulated to explain phenomena under investigation is a powerful tool that could be used with great success in a variety of situations. The critical evaluation process aimed at looking for evidence to disprove these rival theories is a valuable tool for health practitioners in terms of differentiated diagnostics.

The Brunswikian model in the manner in which it has been used in this research, is in essence nothing very different from *Methodological triangulation*. It however provides a useful graphical representation of how the different perspectives ultimately illuminate the picture under investigation. This approach of decomposing the research into different components also expands the context of analysis. It forces the researcher to look for causal relationships that cross the boundaries of the sub-problem under investigation that link the sub-problems with each other.

If it were not for ethical considerations, the validity of the research could have been enhanced. The fact that the three subjects from the exploratory research phase were included in the later explanatory phase could in certain circles be frowned upon. It would have been unethical not to subject them to the interventions that had been developed during the research project. To some extent there is merit in excluding the successful results achieved with the first three subjects from the results of the explanatory research phase. The complete lack of response with the interventions which focused on the posterior compartment in the mind of the researcher does not disqualify them as ultimate successes of the ultimate interventions that led to the successes.

The experimental research conducted during the research project should probably be viewed as pilot studies. No attempt has been made to subject the design or the analysis of the results to thorough statistical scrutiny. It did however achieve a number of objectives. In the first instance the results of both experiments support the theoretical model that was developed. The second experiment on the hind foot pronation also illustrated that the techniques could be applied by relatively inexperienced therapists. It also eliminated researcher bias through the application by others which increase the external validity of the research. It also provides opportunity for interesting future research which is not limited to runners with symptoms of



CPCS, but all subjects with biomechanical abnormalities where fascia related injuries could be the root cause of the problems.

## **5.8. THE RESEARCH DESIGN AND METHODOLOGY**

Research in the physical and natural world is not normally based on a qualitative approach. In some circles the term qualitative if used in the traditional sciences is frowned upon as research in these domains is normally based on the classical scientific method with well-established experimental approaches (Remenyi *et al.*, 2002). The main thrust of this research project was based on a qualitative, theory-building component, although supplemented by a quantitative component in the form of the experimental research components. If it was not for this very qualitative approach that was adopted, the researcher is of the opinion that it would not have been possible to develop the modified theoretical model for the pathogenesis of CPCS, nor the treatment interventions.

The use of multi-functional approaches in terms of both disciplines and methodologies offer a significant opportunity to create better understanding of unresolved problems. Little to no progress with the treatment of the symptoms of CPCS has been made during the recent past and the researcher is of the opinion that the adoption of the qualitative research approach is the major contributor to the successes that were achieved. In conclusion one must concur with Babbie (2005) that the use of both qualitative and quantitative methodologies forms the basis of a more complete understanding of the subject at hand.

## **5.9. CONTEXTUAL BOUNDARIES AND SHORTCOMINGS**

The research project was constrained to the subjective assessment of the condition in the subjects. This constrained was introduced by choice. By the very nature of the equipment and facilities available to the general physiotherapist, it is not possible to utilise sophisticated diagnostics techniques. In this context the therapist is frequently



forced to deal with the symptoms of the condition. In this regard the research is of immeasurable value as it provides an unsophisticated methodology that could be used to assess the condition of the subject. The muscle tightness that could easily be assessed by means of the soft tissue palpation techniques provides a useful way approach a subject with the symptoms of CPCS.

The fact that the diagnosis of the condition was based on this subjective assessment is in retrospect a possible shortcoming in the research. The actual measurement of the pressures in the involved compartment would have made a significant contribution to the external validity of the research. The results obtained are however so overwhelming that this shortcoming is not considered as too serious.

Greater sample sizes would have enabled the verification of the results on statistical basis. The strong link between the theoretical model and the propositions made based on the model however counters this argument. The degree of replication achieved, coupled to the triangulation of research methodologies provides sufficient evidence for the general validity of the new theoretical constructs that were developed.

From an ethical perspective it was imperative that the research was continuously focussed on the improvement of the subject's performance. The fact that intermittent strategies with controlled periods of no intervention, was not used is however not seen as a major limitation to the validity of the research.

Similarly the delimitations introduced by the researcher did not impose on either the quality or the validity of the research. The inclusion of the anatomically analysis of the myofascial structures would have made no significant contribution to the research and would have taken the focus from the real issue, which was the pathogenesis of the CPCS and the implications thereof on conservative treatment interventions. As stated earlier, the measurements of the actual pressures within the posterior compartment would have made a positive contribution, but is not seen as a serious distraction to the quality of the research project. The measurements of biomechanical effects were limited to aspects that made a direct contribution to the understanding of the theoretical model that was developed. Similarly this aspect is also not seen as a factor or an issue that constrained the quality of the research. It does however provide a vast



opportunity for future research into the effect of myofascial constraints on biomechanical measures.

## 5.10. RECOMMENDATIONS

The soft tissue myofascial web is a new frontier in sports medicine. This research project has demonstrated how the characteristics of the soft tissue web and its interaction with various muscles in the body could lead to the pathogenesis of chronic compartment syndromes in the body. Based on these findings it is recommended that much more attention is paid to fascia and the associated soft tissue in the assessment and treatment of sport injuries.

It is recommended that further research projects be launched in order to apply the same approach to runners with other chronic fascia related injuries such as iliotibial band syndrome, plantar fasciitis as well as chronic compartment syndrome of the other compartments of the lower leg.

The results that have been achieved with the normalization of movement patterns and associated biomechanics suggest that restrictions in the myofascial web should first be released before the introduction of “*permanent*” adaptations such as insoles and inserts. The interaction between the myofascial system and the joints is dynamic and needs to be balanced and stabilized before the introduction of strengthening and final rehabilitation.

The research project also illustrated the power of qualitative research approaches in areas where gaps in the current theoretical bases exists. The utilisation of such qualitative approaches coupled with the concept of the triangulation of results as encountered in mixed-methodological research paradigms are strongly recommended.



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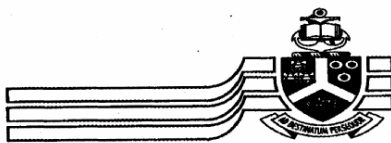
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## **APPENDICES**



**APPENDIX 1: STUDENT PROTOCOL AND THE ETHICAL COMMITTEE**



University of Pretoria

Faculty of Health Sciences Research Ethics Committee

University of Pretoria

Tel (012) 339 8619

Fax (012) 339 8587

E Mail [dbehari@med.up.ac.za](mailto:dbehari@med.up.ac.za)

Soutpansberg Road Private Bag x 385

MRC-Building Pretoria

Level 2 0001

Room 19

Date: 31/07/2002

**Number** : S166/ 2002

**Title** : The effects of manual fascia release on the symptoms of chronic posterior compartment syndrome

**Investigator:** Estelle Erasmus, Department of Physiotherapy, University of Pretoria

**Sponsor** : None

**This Student Protocol has been considered by the Faculty of Health Sciences Research Ethics Committee, University of Pretoria on 30/07/2002 and found to be acceptable.**

Prof P Carstens	BLC LLB LLD (Pret) Faculty of Law
Dr J.E.Dafel	(female) MBChB: Hospital Superintendent
Prof A.P.du Toit	BA; DiplTheo; BA (Hons); MA; DPhil: Philosopher
Prof S.V. Grey	(female) BSc (Hons); MSc; DSc: Deputy Dean
Dr V.O.L. Karusseit	MBChB; MFGP (SA); M.Med (Chir); FCS (SA): Surgeon
Dr S.Khan	(female) MB.BCh.; Med. Adviser (Gauteng Dept.of Health)
Prof M Kruger	(female) MB.ChB.(Pret); Mmed.Paed.(Pret); PhDd. (Leuven)
Miss B Mullins	(female) BscHons; Teachers Diploma;
Snr Sr J. Phatoli	(female) BCur (Et.Al) Senior Nursing-Sister
Prof H.W. Pretorius	MBChB; M.Med (Psych) MD: Psychiatrist
Prof P. Rheeder	MBChB; M.Med (Int); LKI (SA); MSc (CLIN.EPI): Specialist Physician
Dr C F Slabber	BSc (Med)MB BCh, FCP (SA) Acting Head; Dept Medical Oncology
Prof J.R. Snyman	MBChB, M.Pharm.Med: MD: Pharmacologist
Prof De K.Sommers	BChB; HDD; MBChB; MD: Pharmacologist
Dr R Sommers	(female) MBChB; M.Med (Int); MPhar.Med;
Dr TJP Swart	BChD, MSc (Odont), MChD (Oral Path) Senior Specialist; Oral Pathology

**Student Ethics Sub-Committee**

Mrs E Ahrens	(female)B.Cur;
Prof S Meij	(female)BSc(Hons);MSc
Prof P. Rheeder	MBChB;M.Med(Int);LKI(SA);MSc (CLIN.EPI): Specialist Physician
Dr R Sommers	SECRETARIAT (female)MBChB; M.Med (Int); MPharMed;
Dr C van der Westhuizen	(female) D.Cur; M.Ed.
Mrs N Lizamore	(female) BSc(stell), BSc (Hons) (Pret),MSc (Pret) Dept. Anatomy

**PROF J R SNYMAN**

MBChB, M.Pharm.Med MD: Pharmacologist  
CHAIRPERSON of the Faculty of Health Sciences Research  
Main Ethics Committee - University of Pretoria

**PROF P. RHEEDER**

MBChB, M.Med (Int), LKI (SA); MSc (CLIN.EPI): Specialist Physician  
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## **APPENDIX 2: PATIENT INFORMED CONSENT**

### **Patient information leaflet and informed consent**

(Each patient must receive, read and understand this document before the start of the study)

### **Trial Title**

Clinical trial no: S166/ 2002

Multiple single case studies in the form of a clinical trial in patients with chronic posterior compartment syndrome to investigate the effect of manual fascia release techniques on the symptoms.

### **Introduction**

You are invited to volunteer for a research study. The information leaflet is to help you to decide if you would like to participate. Before you agree to take part in this study you should fully understand what is involved. If you have any questions, which are not fully explained in this leaflet, do not hesitate to ask the investigator (Estelle) for clarification. You should not agree to take part unless you are completely satisfied with the procedures involved.





### **What is the purpose of this trial?**

You have a running injury called posterior compartment syndrome, which causes an increase in pressure inside a compartment of the calf muscles. This increase in pressure inside the compartment commences after a certain period of time while running and causes pain and stiffness. A new manual technique is currently being developed in order to release the fascia (a type of soft tissue) involved in the dysfunctional compartment. It is hypothesized that the dysfunctional fascia is the cause of the symptoms of chronic posterior compartment syndrome.

Patients participating will all be runners between the ages of 18 and 50 years, male or female who takes part in road races of distances of ten kilometres or more.

### **What is the duration of this trial?**

Since the study will have an exploratory component it is very difficult to predetermine the duration of the study. Whilst new information is gained, it is likely that the study will continue. It is expected of the participants to commit for a time period of at least six months.

During the intervention, manual fascial release techniques (which can be compared to a soft tissue stretch) will be used. It will be expected of you to continue running while taking part in the study and you will receive home stretches to do on a daily basis.

### **Has the trial received ethical approval?**

This clinical trial protocol was submitted to the Research Ethics Committee and written approval has been granted by that committee. The study has been structured in accordance with the Declaration of Helsinki (last update: October 2000), which deals with the recommendations guiding doctors in biomedical research involving human subjects. A copy of which might be obtained from the investigator (Estelle) should you wish to review it.

### **What are my rights as a participant in this trial?**

Your participation in this trial is entirely voluntary and you can refuse to participate or stop at any time without stating the reason. Your withdrawal will not affect your access to other medical care. The investigator (Estelle) retains the right to withdraw





you from the study if it is considered to be in your interest. If it is detected that you did not give an accurate history or did not follow the guidelines of the trial and the regulations of the trial facility, you may be withdrawn from the trial at any time.

**Is alternative treatment available?**

Alternative treatment in the form of surgical release of the fascia is often used to treat posterior compartment syndromes. If you decide not to take part in this study you might select this option and ask to be referred to an orthopaedic surgeon.

**May any of this trial procedures result in discomfort or inconvenience?**

The only inconvenience experienced might be the time you need to sacrifice to come for the interventions. It will be expected of you to carry on running; so no sacrifices are made in this regard. Manual fascia release is not a painful technique. It is a slow, hold type of release that might feel similar to a stretch sensation.

**What are the risks involved in this trial?**

There are no risks involved. With the involvement of any serious pathology, you will be excluded from this trial.

**Are there any warnings or restrictions concerning my participation in this trial?**

No

**Discontinuation of trial treatment**

You may at any point in time decide to discontinue.

**Insurance and financial arrangements**

Neither you nor your medical scheme will be expected to pay for any of the visits to the investigator (Estelle) if you are included in the trial with the diagnosis of chronic posterior compartment syndrome for the duration of the trial. If you require to be treated for another condition which is not related, it can be expected of you or your medical aid to carry the costs thereof.

You will not be paid to participate in the trial.



### **Source of additional information**

For the duration of the trial you will be under the care of Estelle Erasmus. If at any time between your visits you feel that any of your symptoms are causing you problems, or you have any questions during the trial, please do not hesitate to contact Estelle Erasmus at 082-779-2582 or 991-4499.

### **Confidentiality**

All information obtained during the course of this trial is strictly confidential. Data which may be reported in scientific journals will not include any information which might identify you as a person in this trial.

In connection with this trial, it might be important for domestic and foreign regulatory health authorities and the Research Ethics Committee of the South African Medical Association, as well as your personal doctor, to be able to review your medical records pertaining to this trial. Therefore you hereby authorize your investigator (Estelle) to release your medical records to domestic or foreign regulatory health authorities, the Medicines Control Council and the Research Ethics Committee of the South African Medical Association. You understand that these records will be used by them only in connection with carrying out their obligations relating to this clinical trial.

Any information uncovered regarding your test results or health as a result of your participation in this trial will be held in strict confidence. You will be informed of any findings of importance to your health or continued participation in this trial but this information will not be disclosed to any third party in addition to the ones mentioned above without written permission. The only exception to this rule will be in cases in which a law exists compelling us to report individuals infected with communicable diseases. In this case, you will be informed of our intent to disclose such information to the authorized state agency.

### **Informed consent**

I hereby confirm that I have been informed by the investigator, Estelle Erasmus, about the nature, conduct, benefits and risks of the trial. I have also received, read and



understood the above written information (patient information leaflet and informed consent) regarding this trial.

I am aware that the results of this trial, including personal details regarding my sex, age, date of birth, initial and diagnosis will be anonymously processed into a trial report.

I may, at any time, without prejudice, withdraw my consent and participation in the trial. I have had sufficient opportunities to ask questions an (out of my own free will) declare myself prepared to participate in the trial.

Patient's name: \_\_\_\_\_ (please print)

Patient's \_\_\_\_\_ signature:  
\_\_\_\_\_ Date \_\_\_\_\_

Investigator's name: \_\_\_\_\_ (please print)

Investigator's signature: \_\_\_\_\_  
Date \_\_\_\_\_

I,.....herewith confirm that the above patient has been fully informed about the nature, conduct and risks of the above trial.

Witness's name : \_\_\_\_\_ ( please print)

Witness's signature: \_\_\_\_\_ Date: \_\_\_\_\_



## **APPENDIX 3: E-MAIL FOR THE RECRUITMENT OF SUBJECTS**

**Dear Fellow Runner**

### **PhD study on Sport Injuries:**

**This is a call for all those runners who have been or who are currently suffering with symptoms of chronic posterior compartment to participate in a research study!**

### **What is chronic compartment syndrome?**

Chronic compartment syndrome is a pathological condition of muscle, characterized by increased pressure within an anatomically confined muscle compartment. This increased pressure interferes with the circulation and function of the muscle and neurovascular components of the compartment. When this occurs in the posterior compartment of the lower leg, it is called a posterior compartment syndrome. A condition is usually defined as chronic when it has been present for a period of at least three months.

### **Symptoms of chronic posterior compartment**

The main symptom is activity related pain in the Calves. Both Calves are often affected. The pain increases with exercise and decreases with rest. The area over the calf muscles often feel and appear swollen after exercise. Stretching of the calf muscles often elicit pain. Occasionally there might be a sensation of pins and needles or numbness in the foot. The calf muscles feel tense and tender. Sometimes the pain sensation is also describes as a cramp like sensation.

### **What does participation in the study entail?**

#### **What are the advantages of participation in the study?**

-You might become pain free and will be able to run PB's again (providing you train of course!).

-Free physiotherapy treatment once a week for a couple of weeks specifically for this injury.



-Professional advice with regard to training programmes, correct running shoes, stretches and other information on the injury.

**What are the disadvantages of participation in the study?**

None! (You might not have an excuse for running poorly anymore)

**You qualify for the study if you:**

- are 18 – 50 years of age
- actively participating in road races with a distance of 10 kilometres or more
- are willing to train throughout the intervention period
- have been diagnosed with chronic posterior compartment syndrome or have had symptoms of chronic posterior compartment in one or both legs for a period of more than three months.
- pass an interview and a process of differentiation by the researcher to ensure that the injury is definitely a chronic compartment syndrome.

For further information, please contact Estelle Erasmus at 082-779-2582.

Cheers, see you on the road!