Functional Anatomy in Low Back Rehabilitation

Balance in the Biopsychosocial Model

Drukwerk: Gildeprint Drukkerijen, Enschede

ISBN 978-94-90122-66-9

Functional Anatomy in Low Back Rehabilitation

Balance in the Biopsychosocial Model

Functionele anatomie
bij revalidatie van lage rugklachten

Balans in het biopsychosociaal model

Proefschrift

ter verkrijging van de graad van doctor
aan de Erasmus Universiteit Rotterdam
op gezag van rector magnificus
Prof.dr. H.G. Schmidt
en volgens besluit van het College voor Promoties.

De openbare verdediging zal plaatsvinden op
woensdag 11 november 2009 om 15.30 uur

door

Jan-Paul van Wingerden

geboren te Rotterdam

ERASMUS UNIVERSITEIT ROTTERDAM

Promotiecommissie

Promotor	Prof.dr. C.I. De Zeeuw
Leden	Prof.dr. B.W. Koes
	Prof.dr. J.F. Lange
	Dr.ir. R.H.M. Goossens
Copromotoren	Dr. G-J. Kleinrensink
	Dr. R. Stoeckart

Personal isn't the same as important
Dat iets persoonlijk is, maakt het nog niet belangrijk

Corporal Carrot Ironfoundersson in
Men at Arms by Terry Pratchett

The following parts of this thesis have been published or submitted for publication

van Wingerden JP, Vleeming A, Snijders CJ, Stoeckart R. A functional-anatomical approach to the spine-pelvis mechanism: interaction between the biceps femoris muscle and the sacrotuberous ligament. Eur Spine J 1993 ; 2:140-144.
Reprinted with permission. Copyright (1993), European Spine journal.

van Wingerden JP, Vleeming A, Buyruk HM, Raissadat K. Stabilization of the sacroiliac joint in vivo: verification of muscular contribution to force closure of the pelvis. Eur Spine J 2004;13:199-205.
Reprinted with permission. Copyright (2004), European Spine journal.

van Wingerden JP, Vleeming A. Ronchetti I. Differences in standing and forward bending in women with chronic low back or pelvic girdle pain; indications for physical compensation strategies. Spine 2008;33:E334-E341.
Reprinted with permission. Copyright (2008), Spine.

van Wingerden JP, Stoeckart R, Ronchetti I, Burdorf A, Kleinrensink GJ. Kinesiophobia in woman with chronic pelvic pain; fear of motion in physical perspective.
Submitted to Spine.

van Wingerden JP, Ronchetti I, Vleeming A, Stoeckart R, Burdorf A, Kleinrensink GJ. Balancing the biopsychosocial model for multidisciplinary treatment of non-specific chronic low back pain: merging motor control education and behavioural principles.
Submitted to Spine.

Contents

Chapter 1

Introduction

Low back pain, especially non-specific chronic low back pain (NCLBP) is an ever-increasing problem for society, despite enormous investments in terms of money and time for scientific research [62]. Finding an adequate solution to this problem remains a challenge. The present thesis accepts this challenge by exploring whether more emphasis on physical aspects within the contemporary biopsychosocial (BPS) disease model may improve the diagnostic process and, consequently, therapeutic results.

For a better understanding of the outline of this thesis it is essential to provide, with respect to treatment of NCLBP, a brief overview of the historical development of disease models and their clinical consequences.

1.1. Historical development of disease models

In the past hundred years, at least until 1977, the traditional disease model was widely accepted as an adequate model to explain and to treat diseases [7,13,60]. According to that model, physical and/or chemical findings should be sufficient to explain physical complaints or diseases (figure 1) [7,60]. Such physical and/or chemical impediments could result in disturbed function and lead to limitations in daily activities, such as work.

When applying this common disease model to back complaints, two specific groups can be distinguished with different results of therapy [35,44]. The first group consists of patients with specific (low) back complaints. In this group anatomical changes, like a herniated disc, fracture or stenosis, can be pointed out. Such specific pathological-anatomical findings allow for adequate (conservative of non-conservative) intervention, often with satisfactory results [3,60,62]. With advancing technologies, such as minimally

Figure 1. Illustration of classical view on relationship between structural damage and pain (Descartes 1664)

invasive techniques, the results of interventions are still improving for these patients.

In the second group of low back pain patients no specific pathologic substrate can be found; they have so called non-specific low back complaints. Lack of a clear relation between lesions in anatomical structures and complaints limits or hampers the therapeutic options. In this group of patients there is no anatomical structure or tissue that can be operated upon, and the results of conservative treatment, like physical therapy, are often disappointing [4,9,46-51]. The limited therapeutic possibilities and results in the NCLBP patient group are also recognized in patients with other certain complaints or diseases [7, 8]. At that time it was argued that the traditional biomedical model failed to provide an adequate explanation for those diseases for which no chemical or physical cause could be found. Apparently, there was a need for another disease model [7,8].

In 1977 an alternative was suggested by a psychiatrist, George Engel: the biopsychosocial model [7,60,62]. A key factor in the BPS model is that it describes disease not only as a purely physical process but also as a complex interaction between biological, psychological and social factors (figure 2). The model was also applied to chronic complaints such as NCLBP [31,60]. Subsequently, the medical world gradually became aware of the fact that NCLBP is not merely the result of tissue damage but the

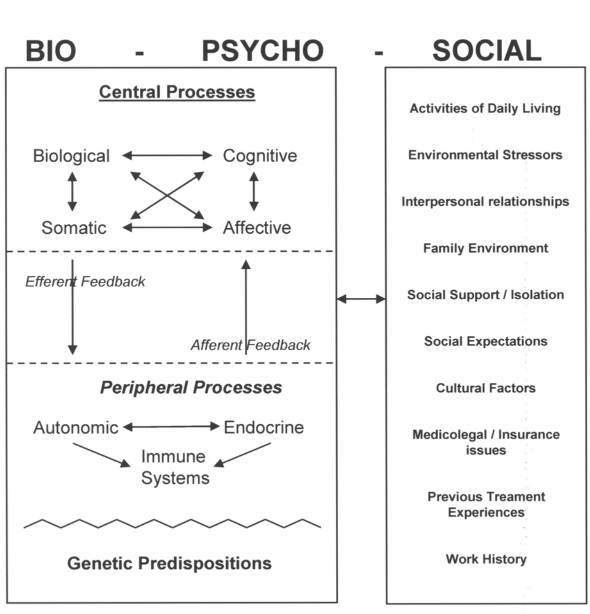


Figure 2. Schematic representation of the biopsychosocial model (Gatchell 2007, permission granted by Gatchell)

result of a complex interaction between physical dysfunction, psychological characteristics (like beliefs and coping), distress, illness behaviour and social interactions.

This new approach to NCLBP, based on Engel's model, allowed for new therapeutic interventions. Especially the psychological domain took advantage of and benefited from this new development [10,37]. The BPS model allowed psychological aspects to be taken into account in explaining NCLBP. It then became possible to demonstrate that a considerable number of NCLBP patients actually avoided activity (figure 3) [30,53]. It was postulated that this type of avoidance could be related to fear of motions and/or activities; fear of motion is primarily triggered by pain [53,55]. Consequently, it was hypothesised that psychological characteristics play an important role in the fear response to pain. Diverse responses to pain, as a consequence of individual psychological characteristics, might explain the differences in behaviour of NCLBP patients with respect to returning to work or other regular daily activities [30,45,53,55,61]. Following this line of thought, fear-reduction therapies as applied in other anxiety (fear) disorders were adopted for NCLBP patients. The first results of these psychology-based therapies (e.g. cognitive-behavioural therapy, graded exposure and graded activity), turned out to be promising [32, 48-54, 56].

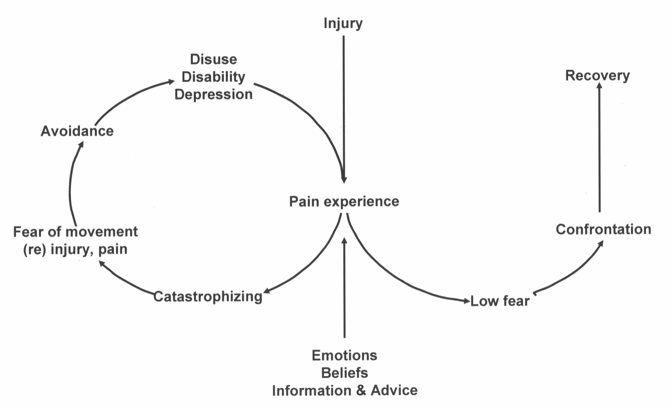


Figure 3. Fear avoidance model by Vlaeyen (Vlaeyen 2000, used with permission from IASP)

Because of these promising results, which were in sharp contrast to the unsatisfactory results of conservative therapy, the emphasis in the treatment of these complaints shifted within the BPS model from physical to psychosocial [20,28,29,48-52].

As a result of this process the focus in contemporary multidisciplinary NCLBP therapies is on the psychological and behavioural aspects [60,62]. The emphasis of the therapy lies on changing behaviour, especially socially-oriented behaviour like returning to work, resuming housekeeping, caring for the children, and re-participation in social life [29]. Obviously, the physical domain is subordinate and consequently the aim of physical therapy within the multidisciplinary programs is rather basic: i.e. general re-conditioning and re-activation of the patient [16,27,40,41]. The focus is primarily on the quantity of activities and not on their quality. This is not surprising. Historically, physical therapy had only limited options for addressing qualitative aspects of function, while more qualitative-based therapy forms (e.g. the Mensendieck or Cesar therapy) still lack an adequate evidence-based foundation [6,14,43]. Consequently, within the BPS based multidisciplinary protocols for NCLBP, physical therapists were assigned only a limited role, as a practical trainer or coach [16,27,40,41].

1.2. New developments in the biological domain

In the last decades our understanding of the functioning of the locomotor system, in particular the pelvis and spine, has significantly increased [1, 2, 5, 12, 15, 17, 38]. In 1990, Vleeming and Snijders introduced the model of 'form and force closure' which provides an explanation of how synovial joints in

Figure 4. Illustration of principle of form and force closure: the combination of surfaces with a specific friction coefficient and compressive force provides stability.

general and more specifically, the sacro-iliac joints can be stabilised by the interaction of a large variety of structures in the proximity of the joint such as ligaments, muscles, etc. (figure 4) [42,57-59].

This model could be well integrated with the new insights on control and stability of the lumbar spine [12,17,18,33,38,39]. It became clear that changes in neuromuscular control can cause sub- or non-optimal motion patterns in the lumbar spine and also in the pelvis, thus compromising the physical capacity (figures 5 and 6) [21,23-25]. Compromised physical capacity will lead to physical overload and pain [5,15]. It is important to note that these mechanisms take place in the absence of visual tissue damage and may last for a prolonged period of time, even years [15,17-19,22,38,39].

A logical consequence of the recent development of functional anatomical knowledge is that the role of the physical aspect within the BPS model needs reconsideration.

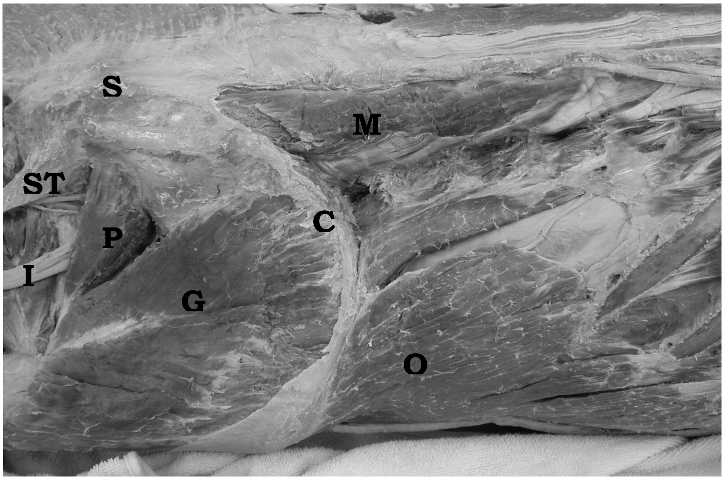


Figure 5. Dorso-lateral picture of deep multifidus muscle:
M: m. multifidus.
C: crista iliaca,
S: sacrum,
ST : ligamentum sacro-tuberale,
P: m. piriformis,
I : n. ischiadicus,
G : m. gluteus medius,
O : m. obliquus externus.

1.3. Reconsideration of the physical aspect in the BPS model

In contemporary multidisciplinary treatments for NCLBP the main focus is on the psychological, behavioural aspects [11,26,34,36,40,41,52,56]. At first glance the results of these behavioural-based therapy forms appear to be better than traditional conservative methods. Closer observation shows that the results of these interventions often reflect their original purpose: the patients return to work and take up their social life. However, when parameters such as experienced pain or improved physical performance are taken into account, the results are far less positive [11,26,34,36,40,41,48,52]. The assumption that the purpose of therapy is to provide a cure and not just to change behaviour leaves the behavioural-based therapies with significant room for improvement.

One option to improve LBP therapy lies in revaluation of the physical domain within the BPS model. New scientific data within the physical domain, especially those based on functional anatomy, may provide possibilities to improve BPS-based interventions, especially by addressing the quality of behaviour. It must be determined whether it is possible to implement functional anatomical principles within existing behavioural therapy leading to better therapy results.

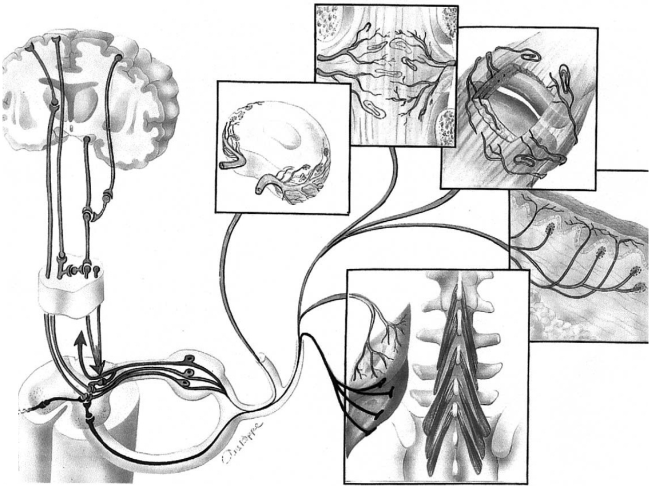


Figure 6. Schematic representation of a reflex system for motion segment stabilization (Holm, 2002).

1.4. Aim of this thesis

In the context of multidisciplinary treatment of NCLBP patients, the aim of this thesis is to answer the following three questions:

1. Taking into account the available recent data on functional anatomy, is there a need to reconsider the role of the physical domain within the BPS model?
2. Will a more pronounced role of functional anatomy in the BPS model contribute to better diagnosis?
3. Will functional anatomy applied in the BPS model contribute to improved therapy?

In answer to the first question, Chapters 2, 3 and 4 present a specific sample of functional anatomy and elaborate on their clinical implications. Chapter 5 deals with the contribution of functional anatomy to the diagnostic process (the second question); this chapter explores the surplus value of combining the results of a physical test (the Active Straight Leg Raise, or ASLR test) with a psychological questionnaire (Tampa Scale of Kinesiophobia Dutch language version (TSK-DV)). Chapter 6 presents an answer to the third question; this chapter describes a multidisciplinary therapy, characterized by a better balance between the physical and psychological domains. The results of this therapy are presented and compared with other behavioural therapies.

Finally, in Chapter 7 the main issues addressed in this thesis are discussed. An answer to the question whether more appreciation for functional anatomy in the BPS model improves diagnosis and therapy of patients with NCLBP is formulated. The results of the studies are discussed in a larger perspective and suggestions for future research are provided.

References

1. Adams M.A. The Biomechanics of Back Pain, Churchill-Livingstone, 2002.

2. Bogduk N. The anatomical basis for spinal pain syndromes. J Manipul Physiol Ther 1995;18:603–5.

3. Carreon LY, Glassman SD, Howard J. Fusion and nonsurgical treatment for symptomatic lumbar degenerative disease: a systematic review of Oswestry Disability Index and MOS Short Form-36 outcomes. Spine J 2007:21.

4. Chavannes A.W. Oefenen bij lage rugpijn. PhD thesis, Utrecht University, The Netherlands, 1992.

5. van Dieen JH, Cholewicki J, Radebold A. Trunk muscle recruitment patterns in patients with low back pain enhance the stability of the lumbar spine. Spine 2003;28:834-41.

6. van Eijsden-Besseling MD, Staal JB, van Attekum A, de Bie RA, van den Heuvel WJ. No difference between postural exercises and strength and fitness exercises for early, non-specific, work-related upper limb disorders in visual display unit workers: a randomised trial. Aust J Physiother 2008;54:95-101.

7. Engel GL. The need for a new medical model: a challenge for bio-medicine. Science 1977;196:129-36.

8. Engel GL. The clinical application of the biopsychosocial model. Am J Psychiatry 1980;137:535-544.

9. Faas A. Oefentherapie bij acute lage rugpijn. PhD thesis, Enschede University, The Netherlands, 1993.

10. Gatchel RJ, Peng YB, Peters ML, Fuchs PN, Turk DC. The biopsychosocial approach to chronic pain: scientific advances and future directions. Psychological Bulletin 2007;133:581-624.

11. van Geen JW, Edelaar MJ, Janssen M, van Eijk JT. The long-term effect of multidisciplinary back training: a systematic review. Spine 2007;32:249-55.

12. Gracovetsky S. The Spinal Engine, Spinger, 1989.

13. Hadler NM. The disabling backache. An international perspective. Spine 1995;20:640-9.

14. Haugstad GK, Haugstad TS, Kirste UM, Leganger S, Klemmetsen I, Malt UF. Mensendieck somatocognitive therapy as treatment approach to

chronic pelvic pain: results of a randomized controlled intervention study. Am J Obstet Gynecol 2006;194:1303-10.

15. Hides JA, Richardson CA, Jull GA. Multifidus muscle recovery is not automatic after resolution of acute, first-episode low back pain. Spine 1996;21:2763-9.

16. Hildebrandt J, Pfingsten M, Saur P, Jansen J. Prediction of success from a multidisciplinary treatment program for chronic low back pain. Spine 1997;22:990-1001.

17. Hodges PW, Richardson CA. Inefficient muscular stabilization of the lumbar spine associated with low back pain. A motor control evaluation of transversus abdominis. Spine 1996;21:2640-50.

18. Hodges PW, Richardson CA. Delayed postural contraction of transversus abdominis associated with movement of the lower limb in people with low back pain. J Spin Disord 1998;11:46-56.

19. Hodges PW, Richardson CA. Altered trunk muscle recruitment in people with low back pain with upper limb movement at different speeds. Arch Phys Med Rehabil 1999;80:1005-12.

20. Hoffman BM, Papas RK, Chatkoff DK, Kerns RD. Meta-analysis of psychological interventions for chronic low back pain. Health Psychol 2007;26:1-9.

21. Holm S, Indahl A, Solomonow M. Sensorimotor control of the spine. J Electromyography Kinesiol 2002;12:219-34.

22. Hungerford B, Gilleard W, Hodges P. Evidence of altered lumbopelvic muscle recruitment in the presence of sacroiliac joint pain. Spine 2003;28:1593-600.

23. Indahl A, Kaigle A, Reikeras O, Holm S. Electromyographic response of the porcine multifidus musculature after nerve stimulation. Spine 1995;20:2652-8.

24. Indahl A, Kaigle A, Reikeras O, Holm S. Interaction between the porcine lumbar intervertebral disc, zygapophysial joints, and paraspinal muscles. Spine 1997;22:2834-40.

25. Indahl A, Kaigle A, Reikeras O, Holm S. Sacroiliac joint involvement in activation of the porcine spinal and gluteal musculature. J Spinal Disord 1999;12:325-30.

26. Johnson RE, Jones GT, Wiles NJ, Chaddock C, Potter RG, Roberts C, Symmons DP, Watson PJ, Torgerson DJ, Macfarlane GJ. Active exercise,

education, and cognitive behavioral therapy for persistent disabling low back pain: a randomized controlled trial. Spine 2007;32:1578-85.

27. Kääpä EH, Frantsi K, Sarna S, Malmivaara A. Multidisciplinary group rehabilitation versus individual physiotherapy for chronic nonspecific low back pain: a randomized trial. Spine 2006;31:371-6.

28. Keller A, Hayden J, Bombardier C, van Tulder M. Effect sizes of non-surgical treatments of non-specific low-back pain. Eur Spine J 2007;16:1776-88.

29. Klaber Moffett J, Mannion AF. What is the value of physical therapies for back pain? Best Pract Res Clin Rheumatol 2005;19:623-38.

30. Kori SH, Miller RP, Todd, DD. Kinisiophobia: a new view of chronic pain behaviour. Pain Manag 1990;3:35-43.

31. Langendoen J. The patient-centredness of evidence-based practice. A case example to discuss the clinical application of the bio-psychosocial model. Man Therap 2004;9:228-33.

32. McCracken LM, Gross RT, Eccleston C. Multimethod assessment of treatment process in chronic low back pain: comparison of reported pain-related anxiety with directly measured physical capacity. Behav Res Ther 2002;40:585-94.

33. McGill SM, Grenier S, Kavcic N, Cholewicki J. Coordination of muscle activity to assure stability of the lumbar spine. J Electromyograph Kinesiol 2003;13:353-9.

34. Nicholas MK, Wilson PH, Goyen J. Comparison of cognitive-behavioral group treatment and an alternative non-psychological treatment for chronic low back pain. Pain 1992;48:339-47.

35. Njoo KH. Non specific low back pain in general practice: a delicate point. PhD thesis, Erasmus University Rotterdam, the Netherlands, 1996.

36. Ostelo RW, van Tulder MW, Vlaeyen JW, Linton SJ, Morley SJ, Assendelft WJ. Behavioural treatment for chronic low-back pain. Cochrane Database Syst Rev 2005;25, CD002014.

37. Pfingsten M. [Functional restoration-it depends on an adequate mixture of treatment] Schmerz 2001;15:492-8.

38. Richardson CA, Jull GA, Muscle control-pain control. What exercises would you prescribe? Man Ther 1995;1:2-10.

39. Richardson CA, Jull GA, Hodges PW, Hides JA. Therapeutic exercise for spinal segmental stabilisation in low back pain: scientific basis and clinical approach. Edinburgh, Churchill Livingstone, 1999.
40. Smeets RJ, Vlaeyen JW, Hidding A, Kester AD, van der Heijden GJ, van Geel AC, Knottnerus JA. Active rehabilitation for chronic low back pain: Cognitive-behavioral, physical, or both? First direct post-treatment results from a randomized controlled trial. BMC Musculoskelet Disord 2006a;7:5.
41. Smeets RJ, Vlaeyen JW, Hidding A, Kester AD, van der Heijden GJ, Knottnerus JA. Chronic low back pain: physical training, graded activity with problem solving training, or both? Pain. 2008;134:263-76.
42. Snijders CJ, Vleeming A, Stoeckart R. Transfer of lumbosacral load to iliac bones and legs. Part 1 and 2. Clin Biomech 1993;8:285-294.
43. Soukup MG, Glomsröd B, Lönn JH, Bö K, Larsen S. The effect of a Mensendieck exercise program as secondary prophylaxis for recurrent low back pain. A randomized, controlled trial with 12-month follow-up. Spine 1999 24:1585-91.
44. Spitzer WO, LeBlanc FE, Dupuis M. Scientific approach to the assessment and management of activity-related spinal disorders. A monograph for clinicians. Report of the Quebec Task Force on spinal disorders. Spine 1987;12:1-59.
45. Thomas JS, France CR. Pain-related fear is associated with avoidance of spinal motion during recovery from low back pain. Spine 2007;32:E460-6.
46. Van Tulder MW. Koes BW. Assendelft WJJ. Bouter LM. The effectiveness of conservative treatment of acute and chronic low back pain. Amsterdam: Fac. Der geneeskunde VU, EMGO instituut, 1999.
47. Van Tulder MW, Ostelo R, Vlaeyen JW, Linton SJ, Morley SJ, Assendelft WJ. Behavioral treatment for chronic low back pain: a systematic review within the framework of the Cochrane Back Review Group. Spine 2001;26:270-81.
48. Van Tulder WM , Waddell G. Evidence-based medicine for non-specific low back pain. Best Pract Res Clin Rheumatol 2005;19:7-9.
49. Van Tulder M, Koes B. Chronic low back pain. Am Fam Phys 2006;74:1577-9.

50. Van Tulder M, Koes B. Low back pain (chronic). Clin Evid 2006;15:1634-53.
51. Van Tulder MW, Koes B, Malmivaara A. Outcome of non-invasive treatment modalities on back pain: an evidence-based review. Eur Spine J 2006;15:S64-81.
52. Vendrig AA, van Akkerveeken PF, Sanders RJ. [Good results from a multidisciplinary and behavioral program for chronic back pain] Ned Tijdschr Geneeskd 2000;144:560-3.
53. Vlaeyen JW, Kole-Snijders AM, Boeren RG, van Eek H. Fear of movement/(re)injury in chronic low back pain and its relation to behavioral performance. Pain 1995;62:363-72.
54. Vlaeyen JW, Haazen IW, Schuerman JA, Kole-Snijders AM, van Eek H. Behavioural rehabilitation of chronic low back pain: comparison of an operant treatment, an operant-cognitive treatment and an operant-respondent treatment. Br J Clin Psychol 1995;34:95-118.
55. Vlaeyen JW, Linton SJ. Fear-avoidance and its consequences in chronic musculoskeletal pain: a state of the art. Pain 2000;85:317-32.
56. Vlaeyen JW, De Jong JR, Onghena P, Kerckhoffs-Hanssen M, Kole-Snijders AM. Can pain-related fear be reduced? The application of cognitive-behavioural exposure in vivo. Pain Res Manag 2002;7:144-53.
57. Vleeming A, Stoeckart R, Volkers ACW, Snijders CJ. Relation between form and function in the sacro-iliac joint, Part 1: Clinical anatomical aspects. Spine 1990a;15:130-2.
58. Vleeming A, Volkers ACW, Snijders CJ, Stoeckart R. Relation between form and function in the sacro-iliac joint, Part 2: Biomechanical aspects. Spine 1990b;15:133-6.
59. Vleeming A. The sacroiliac joint, a clinical-anatomical, biomechanical and radiological study. PhD thesis, Erasmus University Rotterdam, the Netherlands, 1990.
60. Waddell G. 1987 Volvo award in clinical sciences. A new clinical model for the treatment of low-back pain. Spine 1987;12:632–44.
61. Waddell G, Newton M, Henderson I, Somerville D, Main CJ. A Fear-Avoidance Beliefs Questionnaire (FABQ) and the role of fear-avoidance beliefs in chronic low back pain and disability. Pain 1993;52:157-68.
62. Waddell G. The back pain revolution. Second edition. Churchill Livingstone 2004.

Chapter 2

A functional-anatomical approach to the spine-pelvis mechanism: interaction between the biceps femoris muscle and the sacrotuberous ligament

J.P. van Wingerden[*,I], A. Vleeming, PhD[*], C.J. Snijders, PhD[I], R. Stoeckart, PhD[*]

From the Faculty of Medicine, Departments of [*] Anatomy and [I] Biomedical Physics and Technology, Research Group of Clinical Anatomy and Medical Technology, Erasmus University, Rotterdam, The Netherlands

Abstract

Sacroiliac joint dysfunction is often overlooked as a possible cause of "low back" pain. This is due to the use of reductionistic anatomical models. From a kinematic point of view, topographic anatomical models are generally not sufficient since they categorize pelvis, lower vertebral column and legs as distinct entities. This functional-anatomical study focuses on the question whether anatomical connections between the biceps femoris muscle and the sacrotuberous ligament are kinematically useful. Forces applied to the tendon of the biceps femoris muscle, simulating biceps femoris muscle force, were shown to influence sacrotuberous ligament tension. Since sacrotuberous ligament tension influences sacroiliac joint kinematics, hamstring training could influence the sacroiliac joint and as such low back kinematics. The clinical implications with respect to "short hamstrings", pelvic instability and walking are discussed.

Acknowledgement

The authors appreciate mrs. C. Langendoen for her dedicated assistance and mr. J.V. de Bakker for his technical support throughout this study.

Introduction

For a successful treatment of pelvic and spinal disorders, it is essential to have a clear insight into the morphology and function of the connections between spine and pelvis, i.e., the sacrum and its joints. As a rule discussions on "low back" pain are based on classifications used in topographical-anatomical models. In these models spine, pelvis and lower extremities are considered as separate entities. However, from a neurophysiological, biomechanical and functional-anatomical point of view these structures are fully coupled. The topographical-anatomical approach is shown by reductionistic terminology as in the word "back" muscles. After all, these muscles are not only connected to head and ribs but also to "pelvic" structures such as the iliac crests, sacrum and sacroiliac ligaments [2,10,11,12,17]. Obviously, parts of the backmuscles act directly and indirectly at the sacroiliac (SI) joints. Consequently, neglecting SI joint dysfunction as a cause of "low back" pain may well be the result of the use of reductionistic anatomical models leading to an artificial classification.

Preceding studies [18-21,24] were dealing with the intertwined relation between pelvis and spine. Specific symmetrical roughening patterns on the surface of the SI joints, already commencing in the fetal period, were considered as functional adaptations, increasing stability [3]. As shown in a biomechanical study, the specific roughening of the SI joint surfaces goes with a higher friction coefficient. Furthermore, it was shown that the stability of the SI joint was increased by a larger wedge-angle of the joint. As a result, less ligament force is required for bearing the upper part of the body. Vleeming et al. [21] described this as the selfbracing effect of the SI joint. This refers to the dynamic mechanism by which the internal friction in the SI joint can be enlarged.

Since the sacrotuberous ligament influences the selfbracing mechanism, muscles connected to the ligament could play an important role in obtaining SI joint stability [18,19,24]. Connections between the gluteus maximus muscle and the sacrotuberous ligament were found [18]. In the same study the sacrotuberous ligament was shown to be fused with the tendon of the long head of the biceps muscle in six out of twelve cadavers, in four cases even bilaterally.

The anatomical findings were substantiated by a biomechanical study:

when minor loads in the direction of gluteus maximus and biceps femoris muscle were bilaterally applied to the sacrotuberous ligament, ventral rotation (nutation) of the sacrum, as a result of simulated bodyweight, diminished significantly. Since in some cases the long head of the biceps femoris muscle is connected to the sacrotuberous ligament, it is hypothesized that force from this muscle can influence sacrotuberous ligament tension, and in doing so dynamically influence stability of the SI joints [19].

This article deals with the question whether biceps femoris muscle force indeed influences sacrotuberous ligament tension.

Material and methods

Six human bodies (2 male, 4 female) in the age of 70 to 90 were embalmed by vascular perfusion with a medium containing 2.2% formaldehyde. Skin, gluteus maximus muscle and soft tissue covering the sacrotuberous ligament were carefully removed, leaving the sacrotuberous ligament unimpaired. In addition, the distal part of the biceps femoris muscle was removed, leaving intact its proximate tendon and adjacent muscular tissue originating from the ischial tuberosity. Special attention was given to the course of the fibres of the sacrotuberous ligament. Based on the macroscopic findings the sacrotuberous ligaments were classified to be either *totally* or *partially* fixed to the ischial tuberosity.

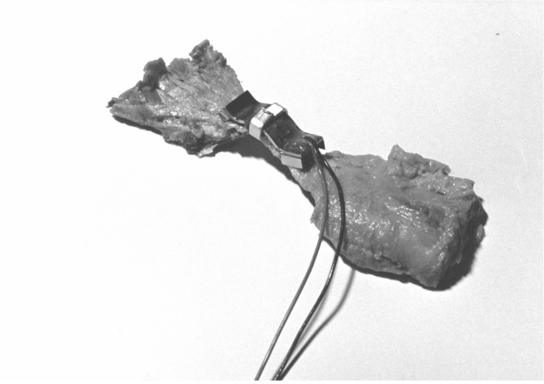


Figure 1. Buckle-transducer attached to sacrotuberous ligament.

Ligament dissected from pelvis after measurement for calibration.

In a previous study the effect of increased sacrotuberous ligament tension on SI joint mobility was demonstrated under loaded circumstances of the lower lumbar spine and pelvis, to simulate trunk weight [19]. This study focuses on the influence of biceps femoris muscle force on sacrotuberous ligament tension. Bodyweight was not simulated. The specimens were lying prone and anchored to the table to prevent sliding.

Ligament tension was recorded by means of a custom-made buckle-transducer (figure 1), as described by Peters [14] and Barry and Achmed [1]. The dimensions of the transducer were adapted to fit a sacrotuberous ligament: 8 x 12 x 34.5 [mm.]. (Strain gauge: Micromeasurements EA-09-062-AP). The buckle-transducer could be applied to the sacrotuberous ligament without affecting its anatomical integrity.

Biceps femoris muscle forces from 0 to 100 N with a 10 N increment were simulated with weights. As site of impact, the biceps femoris muscle tendon was chosen five centimeters caudal from the tuber ischiadicum.

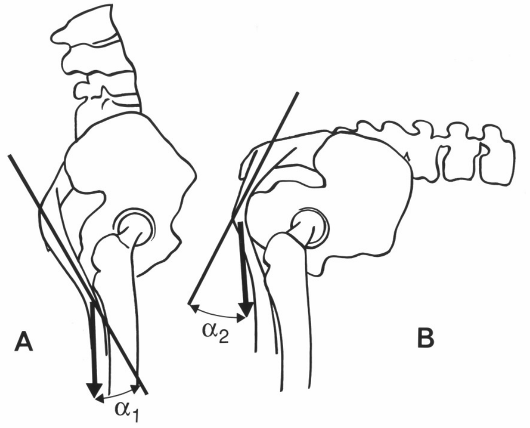


Figure 2. From erect stance (A) to flexed stance (B) the angle between sacrotuberous ligament and biceps femoris muscle changes from α_1 to α_2

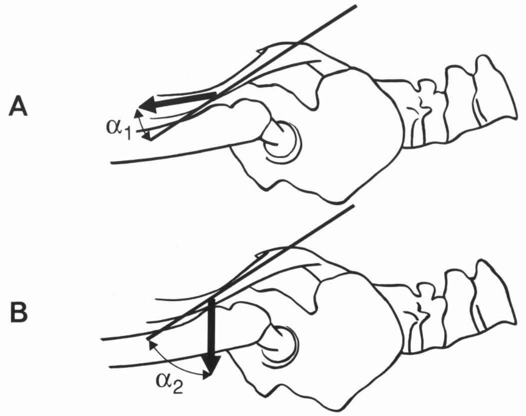


Figure 3. Angle between sacrotuberous ligament and biceps femoris muscle during measurements. Simulated erect stance (A) and simulated flexed stance (B). α_1, approximately longitudinal to the biceps femoris muscle tendon, α_2, vertically downwards

During hipflexion the angle between the sacrotuberous ligament and the biceps femoris muscle tendon changes (figure 2). It can therefore be expected that the amount of force transmitted to the ligament is influenced by the pelvic tilt in the sagittal plane. For this reason measurements were taken in two different directions (figure 3). The primary direction of the applied forces was approximately longitudinal to the course of the biceps femoris muscle, simulating erect stance, to be referred to as erect or upright. Secondary, forces were applied vertically downward to the biceps femoris muscle, simulating hipflexion and to be referred to as flexed stance. To avoid test repetition influence the sequence of force directions was randomized.

To be able to convert the transducer output from millivolts to Newtons the transducer was calibrated for each individual ligament. For this calibration the ligament and transducer were simultaneously removed after the measurements. Calibration was performed twice from 0 to 50 N in steps of 10 N. (Correlation coefficient > 0.995 and mean standard error of estimate = 0.14, range of 0.10).

All tests were repeated three times for each simulated situation. Data of three repetitions were statistically analyzed using two sample ANOVA.

Results

Anatomy

In all preparations the superficial fibres of the sacrotuberous ligament were continuous with the superficial collagenous fibres of the biceps femoris muscle tendon. In six ligaments the deeper part of the ligament was medially connected to the ischial tuberosity. However the lateral deep part of these ligaments was connected to the biceps femoris muscle tendon, and no significant fixation to the ischial tuberosity occurred (to be referred to as *partially fixed* ligaments, figure 4).

The deeper parts of the other four ligaments (No: 1, 2, 9 and 10) did not have any connections with the biceps femoris muscle tendon; they were fully connected to the ischial tuberosity (to be referred to as *totally fixed* ligaments).

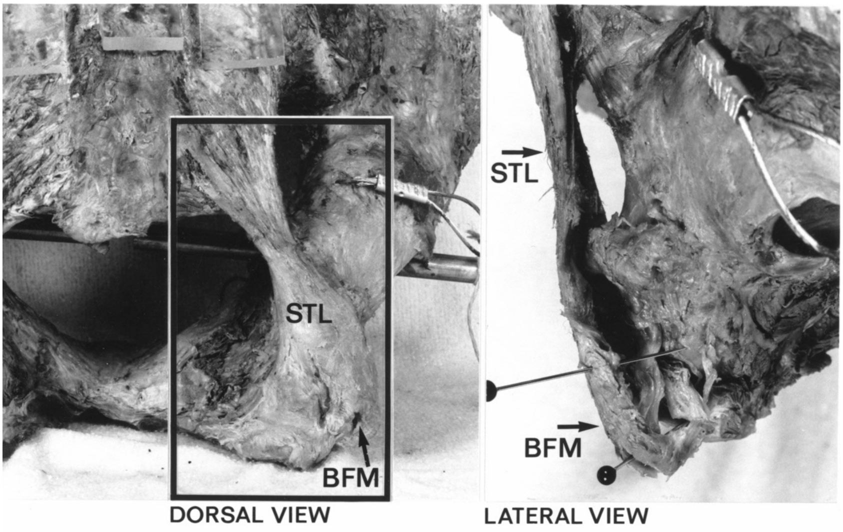


Figure 4. Example of a partially fixed sacrotuberous ligament (STL) and its relation to the biceps femoris muscle (BFM).

Macroscopic observations showed that the fibres of all sacrotuberous ligaments tested were not arranged parallel but spiral in the course of the ligament. As a result, the medial fibres of the ligament cross to the cranial part of the sacrum, while fibres originating more lateral in the ischial tuberosity region, attach to the caudal part of the sacrum. This coiled structure was present in all ligaments.

In the partially, as well as in the totally fixed ligaments the long head of the biceps femoris muscle has the shape of a firm oval tendon on the level of the ischial tuberosity.

Biomechanics

The results are presented as the ratio of the force applied to the biceps femoris muscle tendon and the force measured on the sacrotuberous ligament (table 1). In table 1 every two sequential ligaments belong to one body, except for ligaments 3 and 4 which belong to different bodies.

Table 1. Collected ligament data and applied force/measured force ratio's for simulation of biceps femoris muscle force in erect (upright) stance and flexed stance. Ratio's averaged over three repetitions. Correlation coefficient of all ratio's > 0.98

Ligament	**Side**	**Gender**	**Fixation**	**Upright**	**Flexed**
1	L	F	Total	0.09	0.13
2	R	F	Total	0.08	0.13
3	L	F	Partial	0.20	0.43
4	L	F	Partial	0.54	0.33
5	L	M	Partial	0.08	0.42
6	R	M	Partial	0.16	0.52
7	L	M	Partial	0.69	0.19
8	R	M	Partial	0.19	0.31
9	L	F	Total	0.07	0.15
10	R	F	Total	0.07	0.17

Statistical analysis showed that part of the force applied to the biceps femoris muscle tendon was transferred to the sacrotuberous ligament, in all preparations and in all situations. However, interindividual differences were large (table 1). Transferred forces tended to be higher during the simulated flexed stance than during simulated erect stance (table 1), but differences were not significant. Between genders no significant differences in force transfer could be demonstrated, nor between left and right (table 1).

Table 2. Statistical analysis of all ligaments

	Total Fixation (n=4)		**Partial Fixation** (n=6)
Simulated Erect Stance	0.08 ± 0.01	N.S.	0.31 ± 0.24
	P < 0.01		N.S.
Simulated Flexed Stance	0.15 ± 0.02	P < 0.01	0.36 ± 0.11

More specific results can be summarized as follows:

1. In comparing the sacrotuberous ligaments partially fixed to the ischial tuberosity with the totally fixed sacrotuberous ligaments the following has to be noted:

A. During simulated flexed stance.

Force transfer to the partially fixed ligaments was significantly higher than to the totally fixed ligaments (P < 0.01, table 2).

B. During simulated erect stance.

Although not statistically significant, force transfer to the partially fixed ligaments tends to be four times higher than in the totally fixed ligaments (table 2).

2. In comparing the simulated flexed stance with the simulated erect stance the following has to be noted:

A. For the totally fixed ligaments.

Force transfer in the simulated flexed stance is slightly but significantly higher than during the simulated erect stance (P < 0.01, table 2).

B. For the partially fixed ligaments.

Force transfer in the simulated flexed stances is not significantly different from the simulated erect stance. This is due to the aberrant data for ligaments 4 and 7 (table 1).

Discussion

Insight into the spine-pelvis mechanism can only be obtained on the basis of a functional-anatomical approach [23]. Several anatomical studies [2,7,8,10,11,18,23] show that the influence of soft tissues on lumbar and pelvic kinematics is considerably more complex than presumed by standard anatomical references. The present study emphasizes this view. From a functional-anatomical viewpoint it can be assumed that massive ligaments like the sacrotuberous ligament conduct large forces. From the present study it can be concluded that part of these large forces have a dynamic character. But also the connections of fibres of the gluteus maximus muscle may play an important role in the dynamic aspects of sacrotuberous ligament function. Recently connections of the sacrotuberous ligament with the fascia thoracolumbalis were described [26]. However it is still unclear to what extent the sacrotuberous ligament has the capacity to directly influence lumbar spine function. To understand spine, pelvis and leg kinematics the function of these complex relations must be unraveled.

The leg-back system

The aim of this study is to specify the role of the sacrotuberous ligament and the biceps femoris muscle in the kinematic chain of spine-pelvis-leg. Like the gluteus maximus muscle, the hamstrings are able to tilt the pelvis backwards, thus flattening the lumbar spine. In addition to this "gross" pelvic positioning system we want to distinguish a second, more refined leg-back system. Because of the distinct tendon form of the biceps femoris muscle while approaching and crossing the ischial tuberosity, the muscle is able to conduct its force upwards to the sacrotuberous ligament. As shown in this study, fibres of the biceps femoris muscle tendon are able to alter sacrotuberous ligament tension in all cases. The transfer of force in the fixed ligaments can be explained in two ways: first, superficial fibres that connect ligament and muscle in all preparations, can transduce some force. Secondly, since we noticed a high tension in the sacrotuberous ligament, distortion of the ischial tuberosity (bone elasticity) could easily lead to altered ligament tension.

Increased sacrotuberous ligament tension diminishes sacrum nutation and may consolidate selfbracing of the sacrum [18,19]. Consequently diminished sacrotuberous ligament tension may increase SI joint mobility. This mechanism may even be more subtle: in eight of all ten ligaments tested a relatively higher percentage of force was transferred from the biceps femoris muscle to the sacrotuberous ligament during the flexed situation if compared with the erect situation. From a biomechanical point of view this could be expected, since the flexion torque on the lumbar spine increases when changing from erect stance to flexed stance [9,22]. Therefore, in the flexed position larger contranutating forces are needed to prevent the sacrum from tilting forward. As emphasized by the present findings in most individuals part of this force can be derived from the biceps femoris muscle.

The specific role of the described coiled structure of the sacrotuberous ligament is still unclear however, some speculations can be made. As a result of the coiled structure of the sacrotuberous ligament, the lateral part of the biceps femoris tendon creates a force which is directed to the sacrum horizontally. This force has the same direction as the resultant of ligament forces (F_l), which compress the SI joint and are essential for the selfbracing mechanism as described by Vleeming [21]. It can be noted that the coiled structure of the sacrotuberous ligament resembles the structure of the cruciate ligaments [4,16]. This could imply that different parts of the sacrotuberous ligament, like the cruciate ligaments, are loaded during different stages of motion of the SI joint.

SI joint stabilization during walking

Stabilization of the SI joints during daily activities like walking must be considered a dynamic process. During walking the leg as well as the homolateral SI joint become weight-bearing at heel-strike. On this very moment or better, just before, its selfbracing system must be activated to stabilize the SI joint. Gait analysis shows the hamstrings to become active just before heel-strike [27]. This action increases sacrotuberous ligament tension and presumably selfbracing of the SI joint in addition to limiting knee extension. On heel-strike the homolateral SI joint and the spine will benefit from an optimal stabilization induced by muscular activity of the lower extremity. However, small physical changes, like functional short hamstrings can disturb this leg-spine mechanism.

"Short hamstrings" phenomenon

The phenomenon of "tight-" or "short-" hamstrings is often considered as a secondary effect or residual sign of low back trouble [5,6,12,13,15]. According to the data presented here, shortened hamstrings can affect the selfbracing mechanism of the pelvis. An altered selfbracing mechanism might change the pattern of forces in spine and pelvis. Consequently, short hamstrings may prolong or even initiate low back problems. Whether stretching the hamstrings influences "low back" pain is unclear, since scientific data are lacking [12]. However, it might well be that stretching the hamstrings restores pelvic and lumbar kinematics and breaks the vicious circle of "low back" pain and shortened hamstrings.

Pelvic instability and leg-muscle training

Exercise of muscles, which influence the pelvis directly, or indirectly via the sacrotuberous ligament can be of special importance for women suffering from hypermobility of the pelvis [25]. Pelvic instability is often regarded as exclusively a failure of the pelvic ligaments, the passive structures stabilizing the pelvis. As emphasized here, leg and pelvic muscles can actively influence the mobility of the SI joint and thus influence pelvic stability. By leg-muscle training the selfbracing mechanism can be influenced. Specific muscle training is therefore recommended for women with complaints of pelvic hypermobility [25].

Conclusion

Sacrotuberous ligament tension can be influenced by biceps femoris muscle force. Consequently a leg muscle like the biceps femoris can affect the SI joint and hence pelvic and lumbar stability. In solving complex low back problems, it is essential to see the spine, pelvis and lower extremities as integrated and mutual influencing entities.

References

1. Barry D, and Ahmed AM (1986) Design and performance of a modified buckle transducer for the measurement of ligament tension. J of Biomech Eng 108:149-52.

2. Bogduk N, and Macintosh JE (1984) The applied anatomy of the thoracolumbar fascia. Spine 9:164-70.

3. Bowen V. Cassidy JD (1981) Macroscopic anatomy of the sacroiliac joint from embryonic life until the eight decade. Spine 6:620-8.

4. Butler DL, Guan Y, Kay MD, Cummings JF, Feder SM, Levy MS (1992) Location-dependent variations in the material properties of the anterior cruciate ligament. J. Biomechanics 25:511-18.

5. Gajdosik RL, Giuliani CA, and Bohannon RW (1990) Passive compliance and length of the hamstring muscles of healthy men and woman. Clin Biomech 5:23-9.

6. Göeken LNH (1988) Straight-leg raising in "short hamstrings" [Dissertation]. Groningen (NL):Univ of Groningen.

7. Gracovetsky S, Kary M, Pitchen I, Levy B, and Ben Said R (1989) The importance of pelvic tilt in reducing compressive stress in the spine during flexion-extension exercises. Spine 14:412-6.

8. Gracovetsky S, Kary M, Levy S, Ben Said R, Pitchen I, and Helie J (1990) Analysis of spinal and muscular activity during flexion/extension and free lifts. Spine 15:1333-9.

9. Lindh M (1984) Biomechanica van het skeletsysteem. In: Frankel VH, Nordin M, and Snijders CJ, editors. Biomechanica van het skeletsysteem. Lochem: De Tijdstroom BV, pp 265-303.

10. Macintosh JE, and Bogduk N (1986) The Biomechanics of the Lumbar Multifidus. Clin Biomech 1:205-13.

11. Macintosh JE, and Bogduk N (1991) The Attachments of the Lumbar Erector Spinae. Spine 16:783-92.

12. Mellin G (1988) Correlations of hip mobility with degree of back pain and lumbar spinal mobility in chronic low-back pain patients. Spine 13:668-70.

13. Mierau D, Cassidy JD, and Yong-Hing K (1984) Low back pain and straigth leg raising in children and adolescents. Spine 14:526-8.

14. Peters G (1987) Tools for the measurement of stress and strain fields in

soft tissue [Dissertation]. Eindhoven (NL): Univ of Eindhoven.

15. Stokes IA, and Abery JM (1980) Influence of the hamstring muscles on lumbar spine curvature in sitting. Spine 5:525-8.

16. Strocchi R, De Pasquale V, Gubellini P, Facchini A, Marcacci M, Buda R, Zaffagnini S, Ruggeri A (1992) The human anterior cruciate ligament:histological and ultrastructural observations. J. Anat. 180:515-519.

17. Vink P (1989) Functions of the lumbar back muscles [Dissertation]. Leiden(NL):Univ of Leiden.

18. Vleeming A, Stoeckart R, and Snijders CJ (1989) The sacrotuberous ligament: a conceptual approach to its dynamic role in stabilizing the SI-joint. Clin Biomech 4:201-3.

19. Vleeming A, Van Wingerden JP, Snijders CJ, Stoeckart R, and Stijnen T (1989) Load application to the sacrotuberous ligament; influences on SI-joint mechanics. Clin Biomech 4:204-9.

20. Vleeming A, Stoeckart R, Volkers ACW, and Snijders CJ (1990) Relation between form and function in the sacro-iliac joint, Part 1: Clinical anatomical aspects. Spine 15:130-2.

21. Vleeming A, Volkers ACW, Snijders CJ, and Stoeckart R (1990) Relation between form and function in the sacro-iliac joint, Part 2: Biomechanical aspects. Spine 15:133-6.

22. Vleeming A, Snijders CJ and Stoeckart R, editors (1991) A new and functional approach to the biomechanics of the human pelvis. Proceedings of the First international symposium on the SI-joint: Its role in posture and locomotion; Maastricht(NL):98-109.

23. Vleeming A, Snijders CJ, and Stoeckart R, editors (1991) Progress in vertebral column research; Proceedings of the First international symposium on the SI- joint: Its role in posture and locomotion; Maastricht(NL).

24. Vleeming A, Van Wingerden JP, Dijkstra PF, Stoeckart R, Snijders CJ and Stijnen T (1992) Mobility in the sacroiliac joints in the elderly: A kinematic and radiological study. Clin Biomech 7:170-6.

25. Vleeming A, H.M. Buyruk, Stoeckart R, Karamursel S, Snijders CJ (1991) An integrated therapy for peripartum pelvic instability: A study of the biomechanical effects of pelvic belts. The Am J Obstet Gynacol 166:1243-7.

26. Vleeming A, Pool A, Snijders CJ, Wingerden JP, Stoeckart R. A study of the posterior layer of the lumbo-dorsal fascia. In preparation.
27. Weil S, Weil UH (1966) Mechanik des Gehens. Stuttgart, Georg Thieme Verlag.

Chapter 3

Stabilization of the sacroiliac joint in vivo: verification of muscular contribution to force closure of the pelvis

Wingerden JP van[a], Vleeming A[a], Buyruk HM.[b], Raissadat K[c]

[a] Spine & Joint Centre, The Netherlands, [b] Institute of Rehabilitation, University Hospital Dijkzigt, Rotterdam, The Netherlands, [c] St. Antonius Hospital dept. Surgery, The Netherlands

Abstract

Objectives

To study in vivo whether muscles contribute to force closure of the sacroiliac joint (SIJ).

Summary of background data

A model on SIJ function postulates that SIJ shear is prevented by friction, dynamically influenced by muscle force and ligament tension. Thus, SIJ stability can be accommodated to specific loading situations.

The amount of SIJ friction can be measured as stiffness using a verified method combining Color Doppler Imaging and induced oscillation of the ilium relative to the sacrum.

Study design and methods

SIJ stiffness was measured using Color Doppler Imaging combined with pelvic oscillation in six healthy women. SIJ stiffness was measured both in a relaxed situation and during isometric voluntary contractions (electromyographically recorded). The biceps femoris, gluteus maximus, erector spinae, and contralateral latissimus dorsi were included in this study. Results were statistically analyzed.

Results

SIJ stiffness significantly increased when the individual muscles were activated. This held especially for activation of the erector spinae, the biceps femoris and the gluteus maximus muscles. During some tests significant cocontraction of other muscles occurred.

Conclusions

SIJ stiffness increased even with slight muscle activity, supporting the notion that effectiveness of load transfer from spine to legs is improved when muscle forces actively compress the SIJ preventing shear. When joints are manually tested, the influence of muscle activation patterns must be considered since both inter-and intra tester reliability of the test can be affected by muscle activity. In this respect the relation between

emotional states, muscle activity and joint stiffness, deserves further exploration.

Acknowledgment

The authors wish to acknowledge the department of Biomedical Physics and Technology of the Erasmus University Rotterdam, The Netherlands, for their technical support.

Introduction

This study was initiated to demonstrate in vivo that muscles contribute to force closure of the sacroiliac joint (SIJ). According to the model of form and force closure, shear in the SIJs is prevented by increased friction due to a combination of two factors: 1) specific anatomic features increased the friction coefficient (form closure) and 2) tension of muscles and ligaments crossing the SIJ led to higher friction and hence stiffness (force closure) [16,22,23,26]. Thus, stabilization of the SIJs can be dynamically accommodated to the specific loading situation [16,17,20,21,23,27,28,29,30,31]. Stability of the SIJs is partly realized by tension of ligaments due to SIJ motion [16,20,22,23,24,28]. The model assumed that for effective transfer of load from the spine through the pelvis to the legs, muscles acting on the pelvis must be activated to increase force closure of the SIJ [17,29,30]. Research on joint stability in general and SIJ stability specifically, is mainly focussed on quantitative measurements including recording of the range of motion [10,12,15,18,19,25,26]. No studies were found on qualitative measurements like establishing the stiffness of the SIJ, or to determine the ability of the SIJ to resist shear forces. The need for a reliable and non-invasive method to quantify SIJ stability in vivo resulted in the development of a measuring technique, combining Color Doppler Imaging (CDI) with excitation of the pelvis by means of an oscillation device [1,2,3]. With this method force closure of the SIJ can be measured in vivo as a function of the amount of SIJ friction.

Experimental application of this method on an artificial mechanical model of the pelvis showed reproducible results [1,2,3]. Further validation of this method was performed in three different studies: on embalmed specimen, on healthy subjects, and a comparative clinical study demonstrating this technique to be objective and reproducible in determining SIJ stiffness (Reliability coefficients: left SIJ 0.97 and right SIJ 0.94) [1,2,3].

Former anatomical in vitro studies identified specific muscles that could contribute to SIJ stabilization. Biceps femoris and gluteus maximus muscles could increase force closure of the SIJ, through their specific and massive attachments to the sacrotuberous ligament [20,21,30]. Gluteus maximus and latissimus dorsi were found to be partially coupled by the posterior layer of the thoracolumbar fascia, creating a compressive force acting perpendicular to the SIJ. This was confirmed by a study of Mooney et al [13]. Finally, it was shown that the tendinous aponeurose of the erector muscle was closely linked to the sacrum and posterior superficial SIJ ligaments [24].

The present study attempts to determine whether muscles contribute to force closure in vivo. This study combines CDI and artificially generated oscillation of the SIJ with controlled activation electromyography (EMG) of specific muscles, applied to a group of healthy volunteers. Because of their assumed role in force closure of the SIJ, this study focused on the effect of unilateral activation of the biceps femoris, gluteus maximus and erector spinae, and contralateral activation of the latissimus dorsi muscle [13,20,21,29,30]. It was expected to reject the null hypothesis that muscles cannot stabilize the SIJs.

Material and Methods

Volunteers

Fifteen female volunteers (aged 15 to 30 years) participated in this study. They were all in good physical health with no recent complaints of spine, pelvis or hipjoints. To increase sensitivity of the CDI method only pelves that exhibited considerable motion were included. Joint stiffness was initially measured three times with CDI during application of oscillation to the pelvis. Only in six volunteers (average age 22 sd 2.6 years) threshold values of the CDI were high enough to be included in the study (see results). Average height and weight of the subjects were respectively 170 (sd 4.1) cm and 62 (sd 4.9) kg. Preliminary tests showed the protocol to be fairly straining to the subjects. Because testing both sides may have led to unreliable results due to fatigue [11], during the experiment, tests were performed unilaterally (4 right, 2 left side).

Testing procedure

Volunteers were lying prone with the anterior superior iliac spine in contact with the oscillator plate (figure 1). Before the measurements a maximal voluntary contraction (MVC) of each separate muscle was recorded, using isometric muscle test procedures with manual resistance

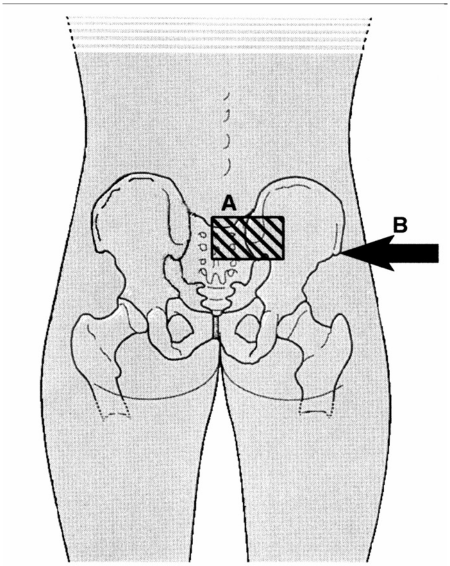


Figure 1. Outline of test position for combined CDI and EMG measurements. A indicates CDI probe location over both sacrum and ilium on one side of the pelvis. B the location where the oscillator plate is positionned against the anterior superior iliac spine.

as described by Kendall et al. [9].

Each measurement started with determination of SIJ stiffness without any muscle activation using CDI. Then the volunteers were asked to activate only one particular muscle for the period of the measurement using the technique as for the MVC test. However, in contrast to the MVC test, no maximal voluntary contraction but only slight effort of the tested muscle was pursued (>10% of MVC), with no or only minimal coactivation of other muscles (<10% of MVC) and minimal disturbance of the initial posture. Since only minimal exertion was required no manual resistance (in contrast with the MVC test) was applied during the tests.

During each test, EMGs of all four muscles were recorded simultaneously to test for cocontractions. Sustained muscle contractions with an average duration of 10 seconds were required to analyze SIJ stiffness by means of the CDI method.

The test sequence was repeated three times with biceps femoris, gluteus maximus, latissimus dorsi and erector muscles tested in randomized order for each subject.

Finally, to verify that EMG signal quality did not change during the measurements, a second maximal voluntary contraction test, similar to the initial MVC test was performed for each muscle.

EMG recording

Electrode location was determined as described by Delagi et al. [4,6,11]. Volunteers were instrumented with surface EMG electrodes (Meditrace pallet electrodes) after the skin was scrubbed and cleaned with alcohol. EMG signals were amplified and 10 - 2 kHz filtered (bipolar EMG amplifier PS-800, Twente Medical System). The signals were rectified, low-pass filtered (10 Hz) and simultaneously fed to a computer with a sample frequency of 50 Hz. Preliminary studies showed no interference of the vibration device with the EMG recordings.

Color Echo Doppler imaging (CDI)

The application of CDI in combination with generated oscillation and the subsequent validation of this method, is described in detail in previous studies on SIJ stiffness [1,2,3]. Vibrations with a frequency of 200 Hz (using a

Derritron VP3 oscillator) were unilaterally applied to the anterior superior iliac spine. The vibrations from ilium and sacrum were measured by a Philips Quantum AD1 CDI transducer covering both sides of one SIJ (see figure 1).

The threshold indicates the necessary signal power to display perceived vibration in color. The height of the threshold is set by the operator by means of the threshold button on the control panel of the CDI apparatus. During a measurement the threshold is precisely set to the level were no vibrations are visible on the CDI screen. A large difference between the thresholds (threshold difference; THD) set at the sacrum and ilium indicates little stiffness of the SIJ. A small or absent THD indicates a stiff joint [1,2,3]. In this study differences between THD in the relaxed position and the THD during a muscle test were used as a measure for change in SIJ stiffness. A decreased THD during the muscle test indicates that the joint has become more stiff.

Analysis

To determine changes in SIJ stiffness during muscle activity, THD's found during muscle tests were subtracted from THD's found during relaxed postures for each individual. The muscle tests were: 1) the biceps femoris test, 2) the gluteus maximus test, 3) the erector spinae test and 4) the latissimus dorsi test. From the three repetitions of each muscle test the mean THD was calculated. The statistical significance of mean differences between THD during relaxed postures and the THD during each muscle test was determined using a paired two sample t-test.

To quantify the activity level of each muscle during the tests, the recorded EMG signals were averaged. From the three repetitions of each muscle test the mean activity level was calculated. To compare between subjects, the muscle activity levels are presented as percentages of the MVC for each muscle.

Muscle activity (in percentage of MVC) during relaxed position and the muscle tests was compared using a paired t-test. A muscle was considered active when the activity level during the tests was more than 10% of MVC. P-values less then 0.05 were considered significant.

Results

Mean results of all subjects are presented in Table 1. Individual results are presented in figures 2 to 6. During the initial SIJ stiffness measurements (no muscle activation) the individual mean THD was 5.8, 3.0, 3.8, 6.0, 4.0, 8.3 respectively (mean 5.2, sd 1.94). The THD in the relaxed position between measurements varied in most cases 0 or 1 level. In one occasion the THD was 2 levels less than the initial measurement. During each muscle test the THDs significantly diminished (Table 1).

Table 1. Mean electromyography levels of muscles as percentage of maximal voluntary contraction and mean decrease of threshold difference (THD) during specific tests when compared to THD measured in the relaxed situation (n=6)

	Biceps	**Gluteus**	**Latissimus**	**Erector**	**THD**
Test for:	Mean (sd)	Mean (sd)	Mean (sd)	Mean (sd)	Mean (sd)
Biceps	54 (22)**	9 (6)	27 (23)	6 (4)	2.5 (0.5)**
Gluteus	19 (5)**	47 (22)**	42 (27)*	18 (14)	2.7 (0.8)**
Erector	10 (6)	8 (3)	46 (19)**	14 (10)	2.7 (1.5)**
Latissimus	13 (8)	9 (9)	27 (21)	34 (13)**	1 (0.6)**

* $p<0.05$, ** $p<0.01$. P-values are calculated with a paired t-test, for muscles: $H_0{:}\mu=10$, for THD $H_0{:}\mu=0$

This effect is particularly strong during the erector, gluteus and biceps muscle test; the mean decrease of THD of 2.7, 2.7 and 2.5 respectively comes to about 50% of the mean relaxed THD of 5.2. The mean results show a significant increase in SIJ stiffness when muscles were activated. Figure 2 shows that there is no change in THD during the latissimus test for subject 3. Also for the other subjects activation of the latissimus dorsi shows the smallest decrease in THD.

With respect to muscle contribution in all tests the highest mean EMG level is especially found for the target muscle (Table 1). In some individual tests however erector EMG level is higher than the target muscle: during the biceps test subject 3 , during the gluteus test subjects 2, 3 and 6, and during the latissimus test subject 4 (Figures 3-6). In most individual tests there is more than 10% of MVC EMG activity of other muscles. However as table 1 shows, this does not result in significant co-activation. Only during the gluteus test the mean EMG activity of another muscle besides the gluteus (erector) is significantly more than 10% of MVC (42%).

For all muscles the MVC before the test sequence highly correlated with the MVC after the tests (ICC; biceps: 0.98, gluteus: 0.98, erector: 0.97 and latissimus: 0.92).

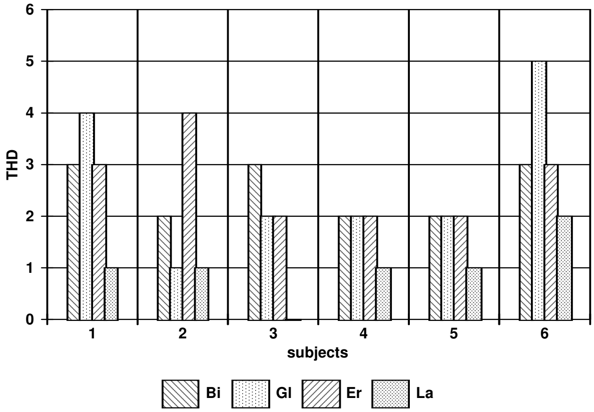


Figure 2. Mean decrease in threshold level for each muscle test (see legend) clustered by volunteer.

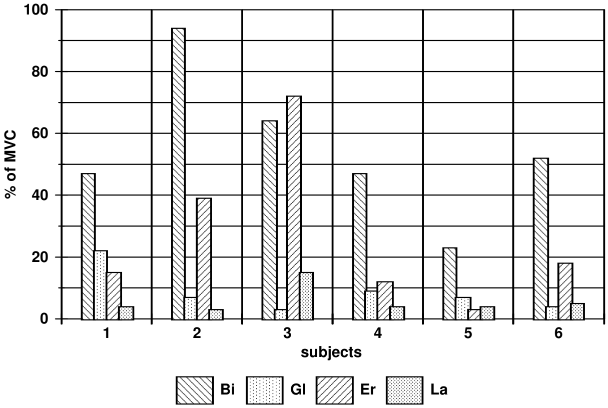


Figure 3. Mean (over 3 repetitions) EMG activity of all muscles as percentage of MVC for each volunteer during biceps test.

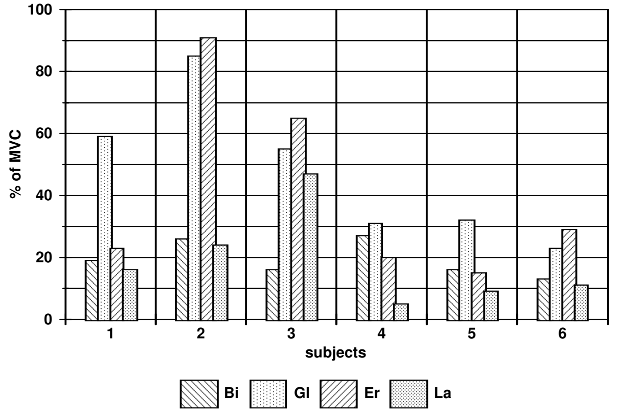


Figure 4. Mean (over 3 repetitions) EMG activity of all muscles as percentage of MVC for each volunteer during gluteus test.

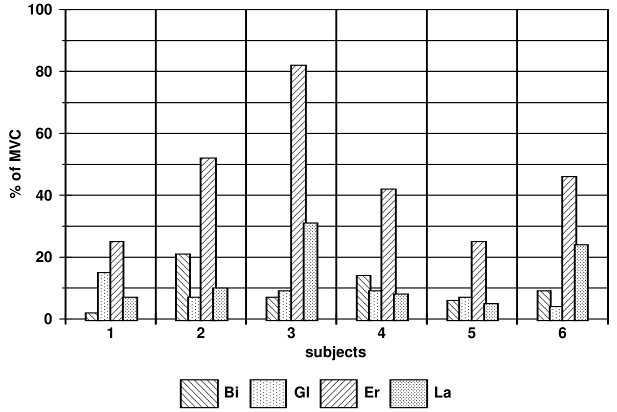


Figure 5. Mean (over 3 repetitions) EMG activity of all muscles as percentage of MVC for each volunteer during erector test.

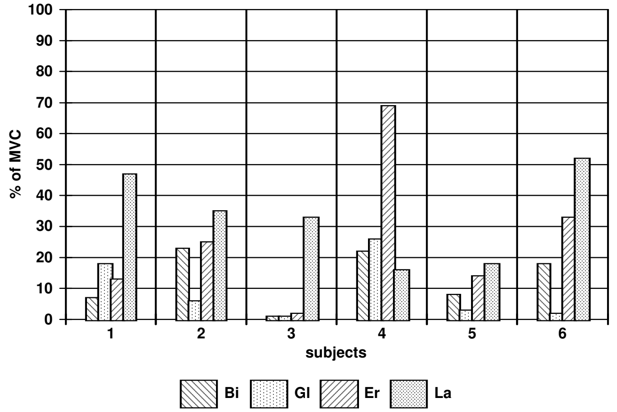


Figure 6. Mean (over 3 repetitions) EMG activity of all muscles as percentage of MVC for each volunteer during latissimus test.

Discussion

SIJ motion is characterized by minute movements [18,19,20]. Color doppler imaging in combination with pelvic oscillation can be applied to study sacroiliac stiffness in vivo [1,2,3]. This method was used to analyze the influence of muscle activity on SIJ stiffness. It showed that contraction of the selected muscles increased SIJ stiffness. The null hypothesis that SIJ stiffness cannot be influenced by muscle activation must therefore be rejected. The erector spinae, the biceps femoris and the gluteus maximus muscles were shown to have the greatest effect on SIJ stiffness. The latissimus dorsi muscle was shown to have a small effect on SIJ stiffness. Subject three was able to activate the latissimus dorsi nearly in isolation (figures 2 and 6), with no change in SIJ stiffness. It can be argued that the increased SIJ stiffness during the latissimus test in other subjects was due to action of other muscles than the latissimus dorsi. Besides statistical significance of the results some intriguing inter-individual differences occurred in both muscle activation and diminishing of THD (figures 2-6). These differences may be partly due to individual initial threshold values, but also to individual muscle activation patterns. Therefore the relative contribution of specific muscles to SIJ stiffness needs further study.

Although the activated muscle was the most electromyo-graphically active muscle during all tests (Table 1), the coactivation of other muscles occurred. The significant cocontraction of biceps femoris and erector spinae muscles during the gluteus maximus test can be expected, since effective movement requires orchestrated contractions of multiple muscles to evoke tailored joint reaction forces [23]. Cocontractions could have been precluded by using electric muscle stimulation instead of intentional voluntary isometric muscle activation. A reason for not opting for this latter solution is that optimal recording of CDI threshold values and thus establishing realistic values for SIJ stiffening, requires maximal relaxation of the volunteers. Electric stimulation can be painful with possible involuntary increase of muscle tone, directly affecting the measurements.
The considerable coactivation of the erector muscle during the biceps, latissimus and gluteus maximus tests, could be expected since it has been shown that the aponeurosis and muscle strains of the erector spinae insert

on the sacrum, the ilium (PSIS) and partially the long dorsal sacroiliac ligament and sacrotuberous ligament [24,28]. These anatomical connections explain how the muscle can contribute to stability of the SIJ. This coactivated function of the erector as described here, is also in agreement with the stabilizing function of the multifidus part of the muscle as described by Hides et al. [5]. Their study shows that the multifidus is coactive with the transverse abdominals and possibly oblique abdominals as primary stabilizers of spine and pelvis [5,6,7]. Since in the present study surface electrodes were used, the abdominal muscles could not be included.

During the gluteus maximus test the activity of erector spinae is particularly high. An additional reason for this activity could be that the subjects were asked to 'take the weight of their upper leg from the table', thus activating the erector in the process of stabilizing pelvis and spine.

The influence of muscles on SIJ stiffness as demonstrated in this study could have clinical consequences. In the clinic, joint stiffness is commonly determined by means of the manual skills of the clinician. However, it was shown that the intra and inter tester reliability of manual tests is low [14]. To our knowledge no studies have been performed to reveal to what extent poor reproducibility of manual tests, could be related to variance of muscle tension and hence joint stiffness between tests (in fact intra-joint or patient reliability). The present study showed that SIJ stiffness is influenced by muscle activity and thus by motor patterns. It can be expected that this also holds for joint stiffness in general. Small variations in the excitation pattern of muscles can lead to differences in joint stiffness. Consequently, during retesting of joints in patients, relatively small postural changes can result in altered muscle contraction patterns and subsequently influence the inter and intra tester reliability of manual joint play tests.

The use of CDI in combination with bone oscillation gives valid results; however, the method is not easy to use in daily practice [1,2,3]. To ascertain valid results in this study only subjects with a relatively high (more than 2.5) THD during the relaxed posture were chosen. The particular aim of the study was only to demonstrate the effect of muscle contraction on SIJ stiffness. Therefore the small number of included subjects [6] as a consequence of the high THD criterion, was considered acceptable for this study. New studies on specific muscles like the transverse and oblique abdominous, using selective electro-stimulation, are necessary [5, 6, 7,17].

This study wanted to show that joint stiffness is not only influenced by structural quality and integrity of the joint but is also influenced by the dynamics of muscle activity. It therefore can be assumed that even when no muscle activity is detected on EMG, basic muscle tone already influences joint stiffness. Emotional states are known to influence basic muscle tone and patterning [8]. The effect of emotional states on specific muscle patterns needs to be taken into account when analyzing SIJ function.

In conclusion, this in vivo study showed that stiffness of the SIJ was increased by certain muscle activity. This supported the model proposed that load transfer from spine to legs is enhanced when muscles actively compress the SIJ thus preventing shear [16, 17,21,22,23]. This agrees with a recent study by Sturesson et al. who demonstrated that in postures with long lever arms, as in stooped positions, SIJ motion became restricted [18, 19].

This in vivo study enhanced our understanding on how muscles dynamically influence SIJ stiffness. The results however, could have implications for joints in general. When joints are manually tested, the influence of muscle activation patterns must be taken into consideration to recognize how both inter and intra tester reliability can be influenced. In this respect the relation between emotional states, muscle activities, SIJ stiffness and joint stiffness in general deserves further exploration.

Literature

1. Buyruk HM, Snijders CJ, Vleeming A, Laméris JS, Holland WPJ, Stam HJ (1995) The measurements of sacroiliac joint stiffness with Color Doppler imaging: a study on healthy subjects. Eur J Radiol 21:117-121.
2. Buyruk HM, Stam HJ, Snijders CJ, Laméris JS, Holland WPJ, Stijnen TH (1999) Measurement of sacroiliac stiffness in peripartum pelvic patients with Doppler imaging of vibrations (DIV). Eur J Obs Gynea Rep Biol 83: 159-163.
3. Buyruk HM (1996) Color Doppler Imaging: new applications in musculoskeletal system pathology. Thesis, Erasmus University Rotterdam, the Netherlands.
4. Delagi EF, Perotto A, Lazetti J, Morrison D (1981) Anatomic guide for the electromyographer. Charles C. Thomas, Illinois, USA.
5. Hides J (1998) The lumbar multifidus: evidence of a link to low back pain. Proceedings of the third interdisciplinary world congress on low back and pelvic pain, Vienna, 117-122.
6. Hodges PW, Richardson CA (1996) Inefficient muscular stabilization of the lumbar spine associated with low back pain. A motor control evaluation of transversus abdominis. Spine 21:2640-2650.
7. Hodges PW (1999) Is there a role for transversus abdominis in lumbo pelvic stability? Man Ther 4:74-86.
8. Holstege G, Bandler R, Saper CB (eds)(1996). The Emotional Motor System. Elsevier Science.
9. Kendall HO, Kendall FP, Wadsworth GE (1971) Muscles, testing and function, Williams and Wilkins Company, Baltimore, Maryland USA.
10. Kissling RO (1995) The mobility of the sacro-iliac joint in healthy subjects. Proceedings of the second interdisciplinary world congress on low back pain. San Diego 411-422.
11. Kumar S, Narayan Y, Zedka M (1996) An electromyographic study of unresisted trunk rotation with normal velocity among healthy subjects. Spine 21:1500-1512.
12. Miller JA, Schulz AB, Andersson GB (1987) Load displacement behavior of sacroiliac joints. J Orthop Res 5:92-101.
13. Mooney V, Pozos R, Vleeming A, Gulick J, Swenski D (2001) Exercise treatment for sacroiliac pain. Orthopedics 24:29-32.

14. Potter NA, Rothstein JM (1989) Intertester reliability for selected clinical tests of the sacroiliac joint. Phys Ther 65:1671-1675.
15. Smidt GL, Wei S-H, McQuade K, Barakatt E, Tiansheng S, Stanford W (1997) Sacroiliac motion for extreme hip positions. Spine 22:2073-2082.
16. Snijders CJ, Vleeming A, Stoeckart R (1993) Transfer of lumbosacral load to iliac bones and legs. Part 1 and 2. Clin Biomech 8: 285-294.
17. Snijders CJ, Slagter AHE, Strik R van, Vleeming A, Stoeckart R, Stam HJ (1995) Why leg-crossing? The influence of common postures on abdominal muscle activity. Spine 20:1989-1993.
18. Sturesson B, Selvik GA, Udén U (1989) Movements of the sacroiliac joints. Spine 14:162-165.
19. Sturesson B, Udén U, Vleeming A (2000) A radiostereometric analysis of movement of the sacroiliac joints during the standing hip flexion test. Spine 25:364-368.
20. Vleeming A, Stoeckart R, Snijders CJ (1989a) The sacrotuberous ligament: a conceptual approach to its dynamic role in stabilizing the sacroiliac joint. Clin Biomech 4:201-203.
21. Vleeming A, Wingerden JP van, Snijders CJ, Stoeckart R, Stijnen T (1989b) Load application to the sacrotuberous ligament; influences on sacroiliac joint mechanics. Clin Biomech 4:204-209.
22. Vleeming A, Stoeckart R, Volkers ACW, Snijders CJ (1990a) Relation between form and function in the sacro-iliac joint, Part 1: Clinical anatomical aspects. Spine 15:130-132.
23. Vleeming A, Volkers ACW, Snijders CJ, Stoeckart R (1990) Relation between form and function in the sacro-iliac joint, Part 2: Biomechanical aspects. Spine 15:133-136.
24. Vleeming A, Pool-Goudzwaard AL, Hammudoghlu D, Stoeckart R, Snijders CJ, Mens JMA (1996) The function of the long dorsal sacroiliac ligament; its implication for understanding low back pain. Spine 21:556-562.
25. Vleeming A, Wingerden JP van, Dijkstra PF, Stoeckart R, Snijders CJ, Stijnen T(1992) Mobility in the sacroiliac joints in the elderly: A kinematic and radiological study. Clin Biomech 7:170-176.
26. Vleeming A (1990) The sacroiliac joint, a clinical-anatomical, biomechanical and radiological study. Thesis, Erasmus University Rotterdam, the Netherlands.

27. Vleeming A, Buyruk HM, Stoeckart R, Karamürsel S, Snijders CJ (1992) An integrated therapy for peripartum pelvic instability: A study of the biomechanical effects of pelvic belts. Am J Obstet Gynaecol 166:1243-1247.
28. Vleeming A, Pool-Goudzwaard AL, Stoeckart R, Wingerden JP van, Snijders CJ (1995) The posterior layer of the thoracolumbar fascia; its function in load transfer from spine to legs. Spine 20:753-758.
29. Vleeming A, Snijders CJ, Stoeckart R, Mens JMA (1997) The role of the sacroiliac joints in coupling between spine, pelvis, legs, and arms. In: Vleeming A, Mooney V, Dorman T, Snijders CJ, Stoeckart R (eds.) Movement, Stability & Low Back Pain. Churchill Livingstone, 53-71.
30. Wingerden JP van, Vleeming A, Snijders CJ, Stoeckart R (1993) A functional-anatomical approach to the spine-pelvis mechanism: interaction between the biceps femoris muscle and the sacrotuberous ligament. Eur Spine J 2:140-144.
31. Wingerden JP van, Vleeming A, Stam HJ , Stoeckart R (1995) Interaction of spine and legs: influence of hamstring tension on lumbo-pelvic rhythm. Proceedings of second interdisciplinary world congress on low back pain.

Chapter 4

Differences in standing and forward bending in women with chronic low back or pelvic girdle pain; indications for physical compensation strategies

J.P. van Wingerden BSc, PT, A. Vleeming PhD, I. Ronchetti MSc.

Spine & Joint Centre, The Netherlands

Abstract

Study design

This cohort study compares motion characteristics during forward bending of a group of chronic female patients either with low back pain (LBP) or pelvic girdle pain (PGP) and healthy subjects using computer-video analysis.

Objective

This study determines whether subcategories of back pain patients could be distinguished by motion characteristics of the pelvis and lumbar spine.

Summary of background data

Compared with healthy subjects, patients with low back pain bend forward in distinct manners. Clustering these motion patterns into specific patient subgroups has been challenging since a basis for subcategorizing was lacking.

Chronic LBP can be distinguished from PGP using specific evidence based diagnostic tests. This allows comparing the motion characteristics of subgroups of chronic patients with either LBP or PGP.

Methods

Forward bending was recorded in both female patients groups and healthy female individuals, using a computer video analysis system.

Trunk motion, pelvic tilt and lumbar lordosis are represented as sagittal plane angles. From these angles the relative contribution of the lumbar spine and hip joint to forward bending can be derived.

Results

Specific and discriminating motion characteristics were found between groups. During erect stance in the PGP group the pelvis is significantly tilted backwards. At maximally forward bending the ROM of the trunk is limited in all patient groups, but only the PGP group has significantly limited hip motion. During the initial part of forward bending lumbar motion is increased in PGP patients and decreased in LBP patients. In the

final part of forward bending contribution of the lumbar spine is increased in both patient groups.

Conclusions

LBP and PGP patients show specific, consistent and distinct motion patterns. These motion patterns are assumed to be functional compensation strategies, following altered neuromuscular coordination.

Introduction

One of the main problems in non-specific low back pain is the lack of adequate sub- categories allowing specific fine-tuning of therapeutic interventions. Since in the past sub-categorizing on a structural basis was found to be fruitless, present research predominantly aims at sub categorisation based on functional analysis [1-11]. For successful functional sub categorisation both an adequate parameter and a preliminary sub classification preferably based on a "gold standard" are essential. This study aims at subcategorizing two back pain groups using forward bending as a discriminating tool.

In healthy subjects forward bending consists of trunk flexion, which is flexion of the (lumbar) spine combined with pelvic tilt (hip flexion). The coordination of the lumbar spine and pelvis during this motion is not arbitrary, but specifically and consistently coupled [12-19].

In the 1960's, Cailliet described the specific motion pattern of spine and pelvis, coined the lumbar-pelvic rhythm, similar to the scapulo-thoracal rhythm [20]. Compared with healthy subjects, patients with low back pain (LBP) usually bend forward in a distinct manner [5,7,15,20-28].

Because of the assumed relation between low back pain and the specific motion patterns during forward bending, this subject has been well studied [4,5,7,10,15,21,22,26,27,29]. Most studies found differences in the motion patterns between healthy individuals and LBP patients. However attempts to cluster motion patterns of specific subgroups of "non-specific" low back pain patients remains difficult especially because of the considerable variation in the motion patterns found and also the lacking of evidence based diagnostic tests to discriminate subgroups [1,29,30,31].

With respect to the latter problem, in a recent European Guideline on Pelvic Girdle Pain (PGP) a definition was constructed for pelvic musculoskeletal pain as follows:

"Pelvic girdle pain (PGP) generally arises in relation to pregnancy, trauma or reactive arthritis. Pain is experienced between the posterior iliac crest and the gluteal fold, particularly in the vicinity of the sacroiliac joints (SIJ). The pain may radiate in the posterior thigh and can also occur in conjunction with/or separately in the symphysis. The endurance capacity for standing, walking, and sitting is diminished. The diagnosis of PGP can be reached

after exclusion of lumbar causes. The pain or functional disturbances in relation to PGP must be reproducible by specific clinical tests” [32].

This European PGP guideline considers, among valid tests like the the Gaenslen and Patricks Faber test, specific tests such as the Active Straight Leg Raise (ASLR), the Long Dorsal Ligament (LDL) and the Posterior Pelvic Pain Provocation (PPPP) test valuable in discriminating PGP patients from healthy subjects and low back pain patients [31-35]. Functionally, PGP patients can be distinguished from regular LBP patients by certain motion characteristics like in walking [3,11,36]. As shown in the study by Wu et al., PGP patients do not only walk at lower speed, their coordination during walking is also distinct from LBP patients and healthy subjects [11]. This leads to the assumption in the present study that also the coupled motion of lumbar spine and pelvis could differ between LBP and PGP patients

Comparison and analysis of the motion patterns of LBP and PGP patients may provide new insight in the aetiology of chronic of low back and pelvic pain.

The specific patient population of a Dutch rehabilitation centre specialized in the treatment of severe low back pain and pelvic girdle pain allowed to compare the motion patterns of LBP and PGP patients.

The aim of the present study was to demonstrate that consistent and discriminating motion patterns exist for the mentioned subgroups. When this assumption is proven correct, analysis of coordination may provide useful information for therapy of LBP and PGP.

Material & methods

Subjects

In a Dutch rehabilitation centre, as part of the standard diagnostic procedure, motion of the lumbar spine and pelvis during forward bending was recorded using video analysis. From the general patient population, a group with specific PGP (29 women, age 33 years SD 5 years) was selected. The cut off scores for the inclusion criteria for PGP were raised to exclusively select severe PGP patients in this group. In the PGP group, pain was mainly experienced in the pelvic area and commenced during pregnancy or within three weeks after delivery. There was no history of low back pain. The Active Straight Leg Raise test (ASLR test) was positive (score summed for both legs was more than 4 on a scale of 0 to 10). The score of the Long Dorsal Ligament (LDL) test, summed for left and right posterior superior iliac spine was more than 2 and the Posterior Pelvic Pain Provocation test (PPPP) test was positive.

In the group with LBP (22 women, aged 36 years SD 9 years) patients were selected whose pain had no relation with pregnancy; they had explicit pain in the lumbar spine but no pain in the pelvic area. The ASLR test was over all negative (summed score of both legs not more than 2, (0.9 on average for both sides). The summed score of the LDL test (left and right posterior superior iliac spine) was less than 2 (0.4 on average for both sides), and the PPPP test was negative.

In both patient groups complaints were present for more than three months. Impact of the complaints on daily life was measured using the Quebec Disability scale, experienced pain was measured with VAS scales and the Tampa scale for kinesiophobia was used to record fear avoidance beliefs. Furthermore, as measure of physical impairment, abduction and adduction strength of the hips was measured using a handheld dynamometer. Finally patients were asked how long they could stand, walk, sit or lie down before their pain significantly increased. An overview of these results is presented in table 1.

Both patient groups were compared to a control group of 53 healthy women (aged 25 years SD 9 years). In this control group none of the women had any history of spine, pelvic, hip, knee or ankle complaints.

Table 1. Overview of severity and impact of complaints of LBP and PGP group.

	LBP	PGP
Quebec*	45 ± 15	61 ± 10
Pain Actual	55 ± 25 mm	54 ± 24 mm
Pain Minimal	32 ± 20 mm	28 ± 16 mm
Pain Maximal	86 ± 15 mm	89 ± 11 mm
Tampa	33 ± 10	36 ± 7
Abduction Strength*	245 ± 83 N	146 ± 74 N
Adduction Strength*	176 ± 55 N	83 ± 51 N
Standing time	12 ± 13 min	10 ± 9 min
Walking time¶	30 ± 20 min	17 ± 14 min
Sitting time	22 ± 18 min	27 ± 16 min
Lying down time	37 ± 25 min	45 ± 20 min

Presented are limitations in daily life (Quebec Disability Scale), experienced pain (actual, minimal and maximal), Tampa list for kinesiophobia, measured ab- and adduction strength of the hips and duration of standing, walking, sitting or lying down before experienced pain significantly increases.
Values are mean ± SD.
* Difference between LBP and PGP significant at P<0.001
¶ Difference between LBP and PGP significant at P<0.01
(mm = millimetres, N = Newtons, min = minutes)

Video method

Women were instrumented with four markers (infra-red LEDs, Figs. 1-3) attached to the skin: one directly to the lateral side of the anterior superior iliac spine, one in the middle on the sacrum at the level of the posterior superior iliac spine, one at the level of the spinal process of the first lumbar vertebra (L1), and one rigidly connected to the marker on L1 (7 cm above the L1 marker).

Marker positions were recorded in the sagittal plane using a CCD video-camera (Javelin JE7642) equipped with a black filter. Frames were sampled at 50 Hz by a standard Personal Computer (Windows based) equipped with a customized video digitizer board (M3156b) and customized software.

Accuracy, inter- and intra-observer reliability and reproducibility of the method were extensively tested with good results (Accuracy: 1°, interobserver reproducibility 0.80, internal data)

Figure 1. Upright position at the beginning of the measurement. Note the marker positions on spine and pelvis.

Figure 2. Maximally flexed posture during measurement. Note the marker positions on spine and pelvis.

Recording

At the beginning of the recording the subject stood upright for one second, with both hands on the contra lateral shoulder to avoid the arm crossing the anterior pelvic marker (Figure 1). Next, subjects were asked to bend forward with straight knees as far as possible in a moderate pace without forcing or jerking and then return to the initial position (Figure 2). The motion was repeated five times without interruption. Minimally three repetitions are used for analysis.

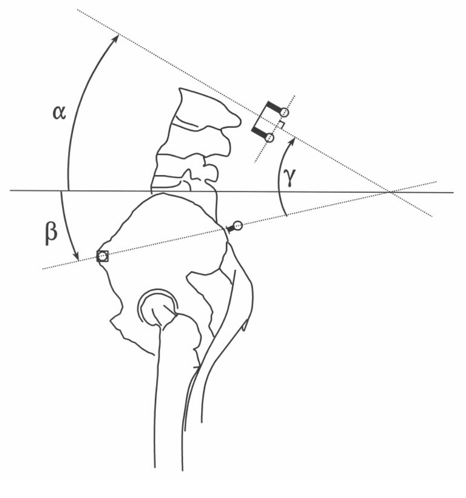


Figure 3. Outline of LEDs and calculated angels of trunk (α), pelvis (β) and lumbar spine (γ).

Analysis

The four pairs of coordinates obtained from each video image were converted into three angles in the sagittal plane (Figure 3):

α, the angle between the horizontal and the line perpendicular to the tangent of the lumbar curve at the level of L1. This angle represents the combined pelvic tilt (hip flexion) and lumbar lordosis (*trunk flexion*). α as shown in Figure 3 has a negative value.

β, the angle between the horizontal and the line through the pelvic markers, representing pelvic tilt (hip flexion).

γ, representing the *lumbar lordosis* was calculated by subtracting angle β from angle α as described by Gracovetsky et al. [14,37].

Regressions were performed on the *lumbar lordosis* (γ) as a function of *trunk flexion* (α) for the first and final one third of *trunk flexion* ROM. In this study ROM was measured from the upright position to maximal flexion as obtained during the video recording.

The slopes, resulting from the regression analysis, represent the relative contribution of the lumbar spine (lordosis) and pelvis to flexion. A slope of 100 reflects exclusively lumbar motion, while a slope of 50 indicates that 50% of the motion consists of lumbar motion and 50% of pelvic tilt.

For between group comparison an unpaired t-test was used. A p-value ≤ 0.05 was considered significant for all tests.

Table 2. Upright position of trunk, pelvis and shape of lumbar spine of the no complaints group, low back pain (LBP) and pelvic girdle pain (PGP) patients.

	Trunk (°)	**Pelvis (°)**	**Lumbar Spine (°)**
No Complaints	-14 ± 5	11 ± 6	-25 ± 7
LBP	-13 ± 5	10 ± 5¶	-23 ± 6
PGP	-13 ± 5	7 ± 4*¶	-20 ± 6*

Values are mean ± SD.
* Compared with no complaints group difference significant at P<0.001
¶ Compared with other patient group difference significant at P<0.01

Results

The quebec and pain scores as presented in table 1 show that both patient groups are mildly to severely impaired. While they experience equal pain, the impact on daily life is significantly higher in the PGP group. Furthermore strength of the hips is lower in the PGP group, and walking is significantly more limited.

The data in Table 2 show that while standing upright, the position of the trunk was similar in all three groups (13°-14°) and pelvic tilt was similar between subjects without complaints (11°) and LBP patients (10°). In PGP patients however, there was a significant backward tilt of the pelvis (7°) compared to both the healthy group and LBP patients. In PGP patients lumbar lordosis was significantly flattened (20°) compared with the healthy subjects (25°) but not with LBP patients (23°) (Table 2 and Figs. 3-6).

Table 3. Range of motion to flexion from upright position of trunk, pelvis and shape of lumbar spine of no complaints group, low back pain (LBP) and pelvic girdle pain (PGP) patients.

	Trunk (°)	**Pelvis (°)**	**Lumbar Spine (°)**
No Complaints	-14 ± 5	11 ± 6	-25 ± 7
LBP	-13 ± 5	10 ± 5¶	-23 ± 6
PGP	-13 ± 5	7 ± 4*¶	-20 ± 6*

Values are mean ± SD.
* Compared with no complaints group difference significant at P<0.001
¶ Compared with other patient group difference significant at P<0.005

Table 3 shows that compared with the healthy group (116°) the ROM of the trunk was significantly decreased in both LBP and PGP patients (81° and 83°, respectively). However, in the LBP group this diminished motion is caused by a specific limitation of the lumbar motion (30°), whereas in the PGP group not only lumbar motion is limited (47°), but also pelvic tilt (37°) is significantly limited. There is a significant difference in both pelvic tilt (51° and 37° respectively) and lumbar motion (30° and 47° respectively) between the LBP and PGP patients (Table 3).

Table 4 provides data on the relative contribution of the lumbar spine and pelvis to forward bending (Slope 1 and Slope 2). Slope1 represents the initial one third and Slope 2 represents the final one third of the forward bending motion.

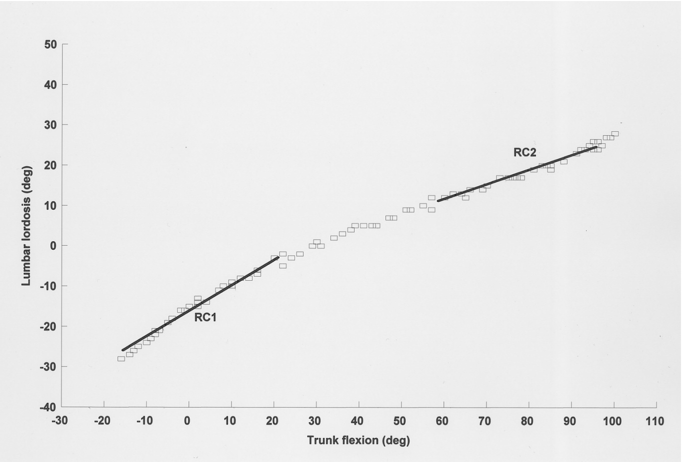


Figure 4. Example of motion pattern typical for healthy subjects, with trunk flexion (α) on the x-axis and lumbar lordosis (γ) on the y-axis. Slope1 and Slope2 represent the relative contribution of the lumbar spine (lordosis) to the first and final one third of flexion respectively.

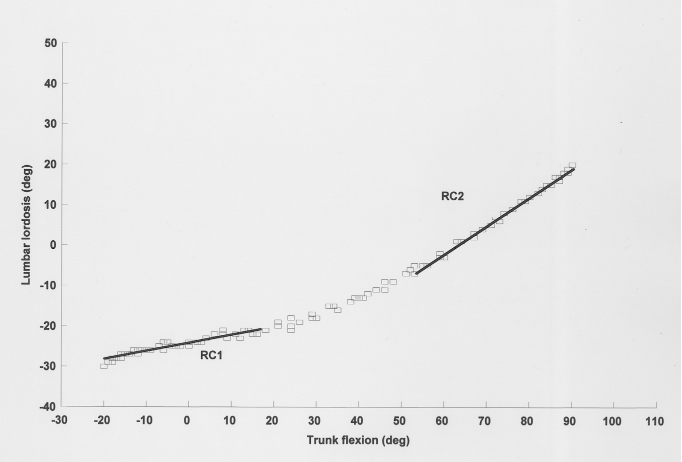


Figure 5. Example of motion pattern typical for LBP patients subjects, with trunk flexion (α) on the x-axis and lumbar lordosis (γ) on the y-axis. Slope1 and Slope2 represent the relative contribution of the lumbar spine (lordosis) to the first and final one third of flexion respectively.

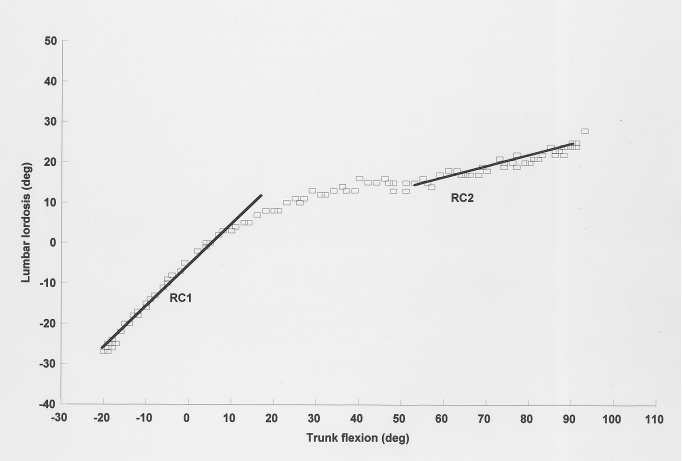


Figure 6. Example of motion pattern typical for PGP patients, with trunk flexion (α) on the x-axis and lumbar lordosis (γ) on the y-axis. Slope1 and Slope2 represent the relative contribution of the lumbar spine (lordosis) to the first and final one third of flexion respectively.

For the LBP patients the Slope 1 is significantly smaller compared to healthy controls (57.7% and 66.9%, respectively), indicating that LBP patients maintain lordosis in the initial flexion. In contrast, the Slope 1 of PGP patients (71.2%) is significantly increased compared to healthy controls (Table 4 and Figs. 4-6). This result shows that in contrast to both healthy subjects and LBP patients, PGP patients emphasise lumbar motion in the initial phase of forward bending.

In the final phase of forward bending the Slope 2 is significantly increased in both LBP and PGP patients compared to healthy controls, demonstrating that both patient groups have more lumbar motion in the final stage of flexion.

Table 4. Range of motion to flexion from upright position of trunk, pelvis and shape of lumbar spine of no complaints group, low back pain (LBP) and pelvic girdle pain (PGP) patients.

	Trunk (°)	**Pelvis (°)**	**Lumbar Spine (°)**
No Complaints	116 ± 14	56 ± 13	60 ± 9
LBP	81 ± 23*	51 ± 18¶	30 ± 16*¶
PGP	83 ± 28*	37 ± 19*¶	47 ± 14*¶

Values are mean ± SD.
* Compared with no complaints group difference significant at P<0.001
¶ Compared with other patient group difference significant at P<0.005

Table 5. Relative contribution (RC) of lumbar spine to forward bending of no complaints group, low back pain (LBP) and pelvic girdle pain (PGP) patients.

	Slope 1 (%)	**Slope 2 (%)**
No Complaints	66.9 ± 7.4	29.6 ± 12.0
LBP	57.7 ± 14.7*¶	49.3 ± 17.5*
PGP	71.2 ± 12.7*¶	47.0 ± 17.3*

Values are mean ± SD.
* Compared with no complaints group difference significant at P<0.05
¶ Compared with other patient group difference significant at P<0.001
Slope1 = the relative contribution of the lumbar spine (lordosis) to the first one third of flexion.
Slope2 = the relative contribution of the lumbar spine (lordosis) to the final one third of flexion.

Discussion

This study investigated motion strategies in female patients with female chronic LBP and chronic PGP compared to healthy female controls. Firstly, before the initiation of movement PGP patients stand with especially more backward pelvic tilt but also with a slight flattened lordosis compared to both healthy subjects and LBP patients. Secondly, during forward bending the coupled motion of lumbar spine and pelvis during the initial phase of the motion differed significantly between both patient groups. Especially during the first one third of forward bending LBP patients tend to maintain lordosis, whereas PGP pain patients emphasise lumbar flexion. Although the coupled motion of lumbar spine and pelvis has been well investigated, this distinct motion pattern between two groups of "non-specific" back pain patients has not been reported previously. In two earlier studies, LBP patients could be divided in two subgroups: one with normal coupled motion of lumbar spine and pelvis and one with altered coupled motion [7,31], however, no satisfactory reason for these differences was provided. In the study by Paquet et al., it is unclear whether male or female subjects (or both) were included [7], so it is likely that LPB and PGP patients were mixed. In the study by Porter et al. [26] a subgroup was found with reduced hip flexion (e.g. limited pelvic tilt during forward bending). Although this motion pattern is similar to that found in the PGP group in this study there are no clear indications in the Porter study that their subgroup had PGP rather than LBP. This also applies to the study by Esola et al. which compared the coupled motion of spine and pelvis during forward bending of 14 males and 6 females [9]. In their study, the spine/hip ratios (as a measure of relative contribution of spine and pelvis to forward bending) have large standard deviations, especially for the first part of the flexion, indicating a substantial variation in the spine/hip ratios. Such variation can occur when LBP and PGP patients, with distinct motion patterns as shown in the present study, are mixed in the same study population.

Possible explanations why LBP patients maintain lordosis

In contrast to the motion strategies of healthy subjects, LBP patients tend to maintain lordosis during forward bending. Many authors consider this specific motion pattern as a natural protection response of the body during a back problem [14,22,28,35,37-42]. Consequently this motion pattern is often advised to patients as the "squat" lifting technique [43]. However, it can be argued that maintaining lordosis is not a solution for a back problem, but a direct consequence of the back problem. In LBP patients the recruitment pattern of the m. multifidus frequently changes, diminishing its anticipatory, stabilizing effect [15,39,44,45]. To guarantee stability, despite this altered activity of multifidus, other muscles (especially the m. erector spinae) become more active [2,39]. Due to its anatomic orientation the m. erector spinae does not stabilize the lumbar spine on a segmental level, but merely increases compression, pulling the lumbar spine into lordosis. Consequently, coordination of segmental motion during forward bending is disturbed when using m. erector spinae predominantly. Therefore, it could be speculated that when m. erector spinae activity is increased to compensate for diminished m. multifidus activity this results in maintained lordosis during forward bending, as shown in the present study. Since many other factors could lead to the described patterning this is still an incomplete analysis which requires further study.

Possible explanations why PGP patients emphasise lumbar flexion

In contrast to LBP patients, PGP patients emphasise lumbar flexion in the initial phase of forward bending. Like in the LBP group the motion pattern found could be a consequence of the specific pelvic problem.

To comprehend stability of the pelvis, a joint model of form and force closure has been introduced [46]. According to this model several structures surrounding the SIJ can stabilize the joint by increasing joint compression [19,34,46-49]. The sacrotuberous ligament is one such structure that stabilizes the SIJ [32,49,50]. Since the sacrotuberous ligament is connected to the long head of the m. biceps femoris and the m. gluteus maximus, by increasing tension of the sacrotuberous ligament these muscles can dynamically stabilize the SIJ [13,19,32,48,49]. Indahl et al. showed that, in analogy to zygapophysial joints, the capsule of the SIJ plays an important role in the neuromuscular control of its stabilizing muscles [51]. When neuromuscular control of the SIJ is disturbed compensatory means of stabilization could be addressed, such as increased activation of the biceps femoris muscle, which was elegantly explained in a study by Hungerford et al, but also indicated by other studies [3,5,6,32,49,50]. Because increased tension of the biceps femoris or m. gluteus maximus also resists the pelvic rotation in the hip joint this limits the contribution of the pelvis to forward bending. Consequently, lumbar motion will be emphasised, as was shown in the present study. However, other explanations for the presented phenomenon can not be excluded.

The present study compared two specifically selected groups of patients with low back problems. However, it can be expected that when groups with less outspoken differences are compared, the motion patterns will be less distinct: the lumbar spine and pelvis are not separate entities but are, from a functional perspective, mutually dependent systems [19,46,34]. Low back problems and their compensatory strategies will have an impact on pelvic function, and vice versa. The explicit distinction between groups in this study was made for methodological reasons but LBP and PGP can occur in mixed variations. Therefore in a clinical setting pelvic function should also be examined in LBP patients, and consequently, lumbar function should be analysed in PGP patients.

In this study no men were included because it was assumed that specific differences in motion patterns could occur between sexes. Motion patterns in men, as the differences in motion patterns between men and women, needs further study.

Conclusion

This study shows that coupled motion of the low back and pelvis, studied in strictly classified subgroups of LBP and PGP patients is specific and discriminating between groups. It is postulated that the specific motion patterns in patients could be functional compensation strategies of the body possibly following adjusted neuromuscular coordination [45, 51].

The distinct coupled motion of lumbar spine and pelvis, combined with more impaired walking and lower hip strength in PGP patients emphasises the notion that LBP and PGP patients belong to distinct patients groups. Analysis of their specific compensatory patterns may elucidate how our body attempts to compensate for functional disturbances. In the clinical setting this may enable more specific exercise programmes to be developed for both LBP and PGP patients.

References

1. Dankaerts W, O'Sullivan P, Burnett A. Straker L. Altered patterns of superficial trunk muscle activation during sitting in non-specific chronic low back pain patients: importance of subclassification. Spine 2006 Aug 1;31(17):2017-2023.

2. Danneels LA, Coorevits PL, Cools AM, Vanderstraeten GG, Cambier DC, Witvrouw EE. Differences in electromyographic activity in the multifidus muscle and the iliocostalis lumborum between healthy subjects and patients with sub-acute and chronic low back pain. Eur Spine J 2002;11:13-9.

3. Hungerford B, Gilleard W, Hodges P. Evidence of altered lumbopelvic muscle recruitment in the presence of sacroiliac joint pain. Spine 2003 28:1593-600.

4. Leinonen V, Kankaanpaa M, Airaksinen O, Hanninen O. Back and hip extensor activities during trunk flexion/extension: effects of low back pain and rehabilitation. Arch Phys Med Rehabil. 2000 Jan;81(1):32-37.

5. Neblett R, Mayer TG, Gatchel RJ, Keeley J, Procter T, Anagnostis C. Quantifying the lumbar flexion-relaxation phenomenon: theory, normative data, and clinical applications. Spine 2003 Jul 1;28(13): 1435-1446.

6. O'Sullivan PB, Beales DJ, Beetham JA, Cripps J, Graf F, Lin IB, Tucker B, Avery A. Altered motor control strategies in subjects with sacroiliac joint pain during the active straight-leg-raise test. Spine 2002;27:E1-8.

7. Paquet N, Malouin F, Richards CL. Hip-spine movement interaction and muscle activation patterns during sagittal movement in low back pain patients. Spine 1994;19:596-603.

8. Pirouzi S, Hides J, Richardson C, Darnell R, Toppenberg R. Low back pain patients demonstrate increased hip extensor muscle activity during standardized submaximal rotation efforts. Spine 2006 Dec 15;31(26):E999-E1005.

9. Pool-Goudswaard AL, Vleeming A, Stoeckart R, Snijders CJ, Mens JMA. Insufficient lumbopelvic stability: a clinical and biomechanical approach to 'specific' low back pain. Man Ther 1998;3:2-20.

10. Silfies SP, Squillante D, Maurer P, Westcott S, Karduna AR. Trunk muscle recruitment patterns in specific chronic low back pain populations. Clin Biomech 2005 Jun; 20(5):456-473.

11. Wu W, Meijer OG, Jutte PC, Uegaki K, Lamoth CJ, de Wolf SG., van Dieen JH, Wuisman PI, Kwakkel G, de Vries JI, Beek PJ. Gait in patients with pregnancy-related pain in the pelvis: an emphasis on the coordination of transverse pelvic and thoracic rotations. Clin Biomech 2002;17:678-86.
12. Adams MA, Dolan P, Marx C, Hutton WC. An electronic inclinometer for measuring lumbar curvature. Clin Biomech 1986;1:130-34.
13. Gajdosik RL, Hatcher CK, Whitsell S. Influence of short hamstring muscles on the pelvis and lumbar spine in standing and during the toe-touch test. Clin Biomech 1992;7:38-42.
14. Gracovetsky S, Kary M, Levy S, Ben Said R, Pitchen I, Helie J. Analysis of spinal and muscular activity during flexion/extension and free lifts. Spine 1990;15:1333-9.
15. Sihvonen T, Partanen J, Häninnen O, Soimakallio S. Electric behaviour of low back muscles during lumbar pelvic rhythm in low back pain patients and healthy controls. Arch Phys Med Rehabil 1991;72:1080-7.
16. Stokes IA, Abery JM. Influence of the hamstring muscles on lumbar spine curvature in sitting. Spine 1980;6:525-8.
17. Toppenberg R, Bullock M. The interrelation of spinal curves, pelvic tilt and muscle length in the adolescent female. Aust J Physiother 1986;32:6-12.
18. Tully EA, Wagh P, Galea MP. Lumbofemoral rhythm during hip flexion in young adults and children. Spine 2002;27:432-40 .
19. van Wingerden JP, Vleeming A, Stam HJ, Stoeckart R. Interaction of spine and legs: influence of hamstring tension on lumbo-pelvic rhythm. Proceedings of second interdisciplinary world congress on low back pain, San Diego, 1995.
20. Cailliet R. Low back pain syndrome. Second edition, Philadelphia: F.A. Davis Company. 1968:23-26.
21. Esola MA, McClure PW, Fitzgerald GK, Siegler S. Analysis of lumbar spine and hip motion during forward bending in subjects with and without a history of low back pain. Spine 1996;21:71-8.
22. Kaigle AM, Wessberg P, Hansson TH. Muscular and kinematic behaviour of the lumbar spine during flexion-extension. J. Spinal disorders 1998;11:163-74.

23. Mayer TG, Tencer AF, Kristoferson S, Mooney V. Use of non-invasive techniques for quantification of spinal range-of-motion in normal subjects and chronic low-back dysfunction patients. Spine 1984;9:588-95.

24. Mellin G. Correlations of hip mobility with degree of back pain and lumbar spinal mobility in chronic low-back pain patients. Spine 1988;13:668-70.

25. Nelson JM, Walmsley RP, Stevenson JM. Relative lumbar and pelvic motion during loaded spinal flexion/exension. Spine 1995;20:199-204.

26. Porter JL, Wilkinson A. Lumbar-hip flexion motion. A comparative study between asymptomatic and chronic low back pain in 18- to 36-year-old men. Spine 1997;22:1508-13.

27. Shirado O, Ito T, Kaneda K, Strax TE. Flexion-relaxation in the back muscles. A comparative study between healthy subjects and patients with chronic low back pain. Am J Phys Med Rehabil 1995;74:139-44.

28. Toussaint HM, de Winter AF, de Haas Y, de Looze MP, van Dieen JH, Kingma I. Flexion-relaxation during lifting: implications for torque production by muscle activity and tissue strain at the lumbo-sacral joint. J Biomech 1995;28:199-210.

29. McClure PW, Esola M, Schreier R, Siegler S. Kinematic analysis of lumbar and hip motion while rising from a forward, flexed position in patients with and without a history of low back pain. Spine 1997;22:552-8.

30. Östgaard HC, Andersson GBJ, Karlsson K. Prevalence of back pain in pregnancy. Spine 1991;16:549-52.

31. Östgaard HC, Zetherstrom GBJ, Roos-Hansson E. The posterior pelvic pain provocation test in pregnant woman. Eur Spine J 1994;3:258-60.

32. Vleeming A, Albert H, Östgaard HC, Stuge B, Sturesson B. COST B14, working group 4 on pelvic girdle.

33. Mens JMA. Pregnancy related low back pain. PhD thesis, Erasmus University, Rotterdam, the Netherlands, 2000.

34. Vleeming A, Pool-Goudzwaard AL, Hammudoghlu D, Stoeckart R, Snijders CJ, Mens JM. The function of the long dorsal sacroiliac ligament: its implication for understanding low back pain. Spine 1996;21:556-62.

35. Vleeming A. de Vries HJ, Mens JMA, van Wingerden JP. Possible role of the long dorsal sacroiliac ligament in women with peripartum pelvic pain. Acta Obstet Gynecol Scand 2002;81:430-6.

36. Lamoth CJ, Meijer OG, Daffertshofer A, Wuisman PI, BeekPJ. Effects of chronic low back pain on trunk coordination and back muscle activity during walking: changes in motor control. Eur Spine J 2006 Jan(1):23-40.

37. Gracovetsky S, Kary M, Pitchen I, Levy S, Ben Said R. 1989 The importance of pelvic tilt in reducing compressive stress in the spine during flexion-extension exercises. Spine 1989;14:412-6.

38. Adams MA, Bogduk N., Burton K., Dolan P. The Biomechanics of back pain. Chapter 7, Forces acting on the lumbar spine, 95-105, Churchill Livingston, 2002.

39. van Dieen JH, Cholewicki J, Radebold A. Trunk muscle recruitment patterns in patients with low back pain enhance the stability of the lumbar spine. Spine 2003;28:834-41.

40. Dolan P, Mannion AF, Adams MA. Passive tissues help the back muscles to generate extensor moments during lifting. J Biomech 1994;27:1077-85.

41. Macintosh JE, Bogduk N, Gracovetsky S. The biomechanics of the thoracolumbar fascia. Clin Biomech 1987;2:78-83.

42. McGill SM, Kippers V. Transfer of loads between lumbar tissues during the flexion-relaxation phenomenon. Spine 1994;19:2190-6.

43. van Dieen JH, Hoozemans MJ, Toussaint HM. Stoop or squat: a review of biomechanical studies on lifting technique. Clin Biomech 1999 14: 685-96.

44. Hides JA, Richardson CA, Jull GA. Multifidus muscle recovery is not automatic after resolution of acute, first-episode low back pain. Spine 1996 21:2763-9.

45. Indahl A, Kaigle AM , Reikeras O, Holm SH. Interaction between the porcine lumbar intervertebral disc, zygapophysial joints, and paraspinal muscles. Spine 1997 22:2834-40.

46. Snijders CJ, Vleeming A, Stoeckart R. Transfer of lumbosacral load to iliac bones and legs. Part 1 and 2. Clin Biomech 1993;8:285-94.

47. Vleeming A, Pool-Goudzwaard AL, Stoeckart R, Wingerden JP van, Snijders CJ. The posterior layer of the thoracolumbar fascia; its function in load transfer from spine to legs. Spine 1995;20:753-8.

48. van Wingerden JP, Vleeming A, Snijders CJ, Stoeckart R. A functional-anatomical approach to the spine-pelvis mechanism: interaction between

the biceps femoris muscle and the sacrotuberous ligament. Eur Spine J 1993;2:140-4.
49. van Wingerden JP, Vleeming A, Buyruk HM, Raissadat K. Stabilization of the sacroiliac joint in vivo: verification of muscular contribution to force closure of the pelvis. Eur Spine J 2004;13:199-205.
50. Vleeming A, Stoeckart R, Snijders CJ. The sacrotuberous ligament: a conceptual approach to its dynamic role in stabilizing the sacroiliac joint. Clin Biomech 1989a;4:201-3.
51. Indahl A, Kaigle A, Reikeras O, Holm S. Sacroiliac joint involvement in activation of the porcine spinal and gluteal musculature. J Spinal Disord 1999;12:325-30.

Chapter 5

Kinesiophobia in women with chronic pelvic girdle pain: fear of motion in a physical perspective

J.P. van Wingerden[1,2], R. Stoeckart[2], I. Ronchetti[1], A. Burdorf[3], G-J. Kleinrensink[2]

[1]Spine & Joint Centre, the Netherlands, [2]Erasmus MC, Department of Neurosciences, [3]Erasmus MC, Department of Public Health

Abstract

Study Design
A cohort study investigating female patients with chronic pelvic girdle pain (PGP).

Objective
The study was designed to determine the interrelationship between fear of motion and physical impairment in chronic PGP patients.

Summary of Background Data
Since the introduction of the biopsychosocial model, fear of motion has become an unequivocal factor in the aetiology of chronic back pain. Presuming that injured tissue regenerates in about 12 weeks, avoidance behaviour persisting for more than 12 weeks is considered to be predominantly based on psychological factors and not on pain stimuli from damaged tissue.

The present study challenges this assumption. In chronic PGP patients, a subgroup of non-specific chronic low back pain patients, significant impairment of the mechanical function of the pelvis is demonstrated. This patient group also displays fear avoidance behaviour. In this specific patient group, the interrelationship between fear of motion and physical impairment was determined.

Methods
In a group of 582 chronic PGP patients, Pearson correlation coefficients were determined between fear of motion and physical impairment. Fear of motion was measured with the Tampa Scale for Kinesiophobia Dutch language Version (TSK-DV), and physical impairment with the Active Straight Leg Raise (ASLR) test, the Quebec Back pain Disability Questionnaire (QBDS) and pain VAS, as well as standing, walking, sitting and lying down. To evaluate agreement between outcome measures simple linear regression analyses were conducted.

Results
Correlation coefficients between scores on the TSK-DV (as measure of fear of motion) and the ASLR, QBDS, VAS, standing, walking, sitting and lying down were 0.04, 0.13, 0.12, -0.03, -0.05, -0.07 and -0.05, respectively.

Conclusions
No interrelationship was found between fear of motion and physical impairment in chronic PGP patients. However, based on specific combinations of TSK-DV and ASLR scores subgroups of patients can be recognized that require a different therapeutic approach. Especially patients with low TSK-DV scores and a high level of physical impairment need more attention.

Introduction

Fear of motion

The introduction of the biopsychosocial (BPS) model in the medical world allowed psychosocial factors to be considered as potential aetiological factors for non-specific chronic low back pain (NCLBP) [1-3]. Due to limited evidence for structural damage in NCLBP, there is even a tendency towards accepting psychosocial factors as the primary explanation for NCLBP [3]. In this context, fear of motion (kinesiophobia) has received specific attention with respect to NCLBP [4-8]. Some authors consider kinesiophobia to be one of the most important factors responsible for the chronicity of back pain [5,7,8].

Avoidance behaviour is a normal psycho-physiologic response to pain that prevents the organism from further injury [4]. Injured tissue is expected to recover within 12 weeks. Consequently, avoidance behaviour persisting for more than 12 weeks is assumed to be predominantly based on psychological and no longer on physical factors. Prolonged avoidance behaviour may lead to detrimental physical deconditioning and is considered counterproductive for recovery [7,8]. An important flaw in this reasoning is that it is difficult to determine the actual state of (internal) tissue regeneration. Therefore, the assumed absence of pain stimuli after 12 weeks is generally based on common physiological rules and not on actual physical assessment.

Functional anatomy and pelvic pain

Chronic pelvic girdle pain (PGP) is a subgroup of NCLBP [9,10]. The symptoms of this sub-group of NCLBP patients are described in the European COST guideline as follows:

"Pelvic girdle pain (PGP) generally arises in relation to pregnancy, trauma or reactive arthritis. Pain is experienced between the posterior iliac crest and the gluteal fold, particularly in the vicinity of the sacroiliac joints (SIJ). The pain may radiate in the posterior thigh and can also occur in conjunction with/or separately in the symphysis. The endurance capacity for standing, walking, and sitting is diminished. The diagnosis of PGP can be reached after exclusion of lumbar causes. The pain or functional disturbances in relation to PGP must be reproducible by specific clinical tests" [11].

Chronic PGP patients, like other NCLBP patients, often display fear avoidance behaviour even after 12 weeks [9,12]. In chronic PGP patients, however, the mechanical function of the pelvis is in fact impaired [10,13-15]. To determine the level of physical impairment of the pelvis, Mens et al. introduced the Active Straight Leg Raise (ASLR) test [16,17]. The ASLR test establishes the mechanical load capacity of the pelvis. Impaired capacity of the pelvis can lead to limitations in daily activities, re-injury and pain. These symptoms are present in the absence of visual tissue damage and can last for a prolonged period of time, even years [10,12,13]. Pain resulting from impaired pelvic function can induce fear of motion. In this particular situation, despite the ever present psychological mechanisms, fear of motion may primarily have a biological cause.

The present study was designed to determine the interrelationship between fear of motion and the level of physical impairment in patients with chronic PGP.

Material and Methods

Study population

From 2002 to 2007, 582 females with chronic pelvic pain visited a Dutch outpatient-rehabilitation centre for treatment. As part of the standard diagnostic procedure, all patients completed the Tampa Scale for Kinesiophobia-Dutch Version (TSK-DV), and all were seen by a physician who also performed the tests.

Variables

Kinesiophobia was measured using the TSK-DV [7,8]. This 17-item questionnaire, based on a 4- point Likert scale (range 17-68), determines the level of fear of motion [7,8,18]. This questionnaire has been extensively studied and found adequate to determine the fear of motion in patients with low back pain [18-21].

As a measure of physical impairment of the pelvis the ASLR test is routinely used. The test establishes the level of compromised load capacity of the pelvis and is designed for patients with pelvic pain. In the present study, the ASLR was performed as described by Mens et al., asking the (supine) patient to lift one straight leg 20 cm from the table [16,17]. Patients were asked to estimate the effort needed to perform this task (as measure of impairment and *not* the pain involved), on a 6-point scale: not difficult at all = 0, minimally difficult =1, somewhat difficult = 2, fairly difficult = 3, very difficult = 4, unable to do = 5. The scores of both sides were summed, giving a final score ranging from 0 to 10. In addition to the patient's interpretation (ASLR-P), the patient's effort was also estimated (using the same scale) by the physician performing the test (ASLR-A).

As a second measure of physical impairment the Quebec Back Pain Disability Scale (QBDS) was used. The QBDS is a 20-item self-administered instrument designed to assess the level of functional disability (range 0-100) in individuals with back pain [22, 23].

As a third measure of compromised physical performance, patients reported how long they could stand, walk, sit or lay down before their pain significantly increased. For this measure a scale from 5 to 60 minutes (with a 5-minute interval) was used. Five minutes indicated an immediate increase in pain and 60 minutes or longer indicated that the activity could be performed without an increase of pain (range 5-60).

A Visual Analogue Scale for pain (Pain VAS) is used as a measure for experienced subjective pain. Patients were asked to mark their current level of pain on a 100 mm long horizontal line ranging from 'no pain' to 'unbearable pain' (range 0-100 mm) [24].

Statistical analysis

The interrelationships between fear of motion (TSK-DV), physical impairment (ASLR), perceived pain (pain VAS), QBDS, and standing, walking, sitting and lying down were determined by means of Pearson correlation coefficients. To evaluate agreement between outcome measures as independent continuous variables and the TSK-DV as dependent continuous variable, simple linear regression analyses were conducted. In this analysis the intercept represents the systematic difference between the TSK-DV and another outcome measure and the regression coefficient expresses the agreement between two measures with a value of 1 indicating a perfect agreement and a value of 0 a complete lack of agreement.

The explained variance (R^2) represents how much of the variance in the TSK-DV across all subjects can be explained by another outcome measure [25].

Results

Table 1 shows that the patients in the present study were relatively young (mean 34 years), experiencing their complaints for an average of 4.0 years. Over time their ability to work had decreased from 27.2 to 7.8 hours per week.

Table 2 shows that estimation of the ASLR by the patient (ASLR-P) closely matched the interpretation made by the physician (ASLR-A) (r=0.77). Furthermore the ASLR-P and ASLR-A show a similar trend in correlations regarding the other parameters (Table 3).

There is no correlation between TSK-DV and ASLR-P (r=0.04) and only a slight correlation between TSK-DV and QBDS (r=0.13) and between TSK-DV and pain VAS (r=0.12) (Table 3). In addition, no relationship was found between TSK-DV and standing, walking, sitting or lying down (r = -0.03, -0.05, -0.07 and -0.05, respectively). Strong correlations were found between ASLR-P and QBDS, pain VAS, standing and walking (r= 0.50, 0.30, -0.28 and -0.26, respectively). The correlation between ASLR-P and sitting and lying down was weaker (-0.13 and -0.15, respectively). QBDS was strongly correlated with all other parameters, except for TSK-DV (r=0.13). The strongest correlation was found between QBDS and pain VAS (r=0.54). Pain VAS was also strongly correlated with standing, walking, sitting and lying down (r=-0.31, -0.26, -0.30, -0.28, respectively).

The scores for duration of standing and walking are strongly correlated (0.39), while the correlations between standing and sitting or lying down are somewhat weaker (both 0.20). The correlation between walking and sitting or lying down were only 0.16, whereas the correlation between sitting and lying was 0.28 (Table 3).

Table 4 again indicates the poor agreement between the TSK-DV and the other outcome measures of interest. The regression coefficients and explained variance are close to zero for each comparison. For those measures with a significant correlation with the TSK-DV, the linear regression coefficients were very low and the intercept were very high, the latter indicating large systematic differences and a lack of agreement.

Table 1. Data on the study population: **work pr** indicates the hours/week the patient worked before the complaints, **work ac** indicates the hours/week the patient is currently able to work..

	Mean	SD	N
Age (years)	34.0	7.2	574
Duration of complaints (years)	4.0	4.1	578
Work pr (hours/week)	27.2	10.8	509
Work ac (hours/week)	7.8	11.4	526

Table 2. Data on the Tampa Scale for Kinesiophobia (TSK-DV; range 17-68), Active Straight Leg Raise assessed by the patient (ASLR-P), Active Straight Leg Raise assessed by the physician (ASLR-A; range 0-10), Quebec Back Pain Disability Scale (QBDS; range 0-100) and pain VAS (range 0-100), and standing, walking, sitting and lying down (in minutes; range 5-60).

	Mean	Median	SD	N
TSK-DV	35.7	35	7.0	582
ASLR-P	4.9	5	2.2	582
ASLR-A	5.3	5	1.8	582
Pain VAS	56.1	60	22.4	580
QBDS	55.2	55	15.3	580
Standing	9.2	5	11.5	582
Walking	14.5	10	17.1	582
Sitting	18.7	10	20.8	581
Lying down	35.5	60	26.3	582

The weak agreement between TSK-DV and ASLR is illustrated in Figure 1; the diffuse cloud shape in the graph clearly shows the absence of an agreement between these two parameters. In this graph four areas can be distinguished. Subjects in the lower-left corner of the graph (Low, expected) have a low ASLR score (indicating little physical impairment) and a corresponding low TSK-DV score. Subjects in the upper right corner of the graph (High, expected) have a high ASLR score (indicating a high level of physical impairment) and a high level of fear of motion (indicated by the high TSK-DV score). Subjects in the lower right corner of the graph (Too

Table 3. Pearson correlation coefficients (r) between the Tampa Scale for Kinesiophobia-Dutch version (TSKDV), Active Straight Leg Raise assessed by the patient (ASLRP), Active Straight Leg Raise assessed by the physician (ASLRA), Quebec Back Pain Disability Scale (QBDS), pain VAS (pain), and standing (Stand.), walking (Walk.), sitting (Sit.) and lying down(Lying d.).

	TSKDV	**ASLRP**	**ASLRA**	**QBDS**	**Pain**	**Stand.**	**Walk.**	**Sit.**
ASLRP	0.04	-	-	-	-	-	-	-
ASLRA	0.01	0.77§	-	-	-	-	-	-
QBDS	0.13¶	0.50§	0.49§	-	-	-	-	-
Pain VAS	0.12¶	0.30§	0.24§	0.54§	-	-	-	-
Standing	-0.03	-0.28§	-0.22§	-0.41§	-0.31§	-	-	-
Walking	-0.05	-0.26§	-0.21§	-0.42§	-0.26§	0.39§	-	-
Sitting	-0.07	-0.13¶	-0.10§	-0.28*	-0.30§	0.20§	0.16§	-
Lying d.	-0.05	-0.15§	-0.12¶	-0.29§	-0.28§	0.20§	0.16§	0.28§

* $p<0.01$, ¶ $p<0.001$, § $p<0.0001$

Table 4. Results of linear regression presented by Intercept, slope and R^2 of TSK-DV as dependent variable and Active Straight Leg Raise assessed by patient (ASLR-P), Active Straight Leg Raise assessed by physician (ASLR-A), Quebec Back Pain Disability Scale (QBDS), pain VAS, and standing, walking, sitting and lying down as independent variables.

	Intercept	**Slope**	**R^2**
ASLR-P	35.0	0.13	0.2
ASLR-A	35.5	0.04	0.0
QBDS	32.3	0.06	1.7
Pain VAS	33.6	0.04	0.4
Standing	35.9	-0.02	0.1
Walking	36.0	-0.02	0.3
Sitting	36.1	-0.02	0.4
Lying down	36.2	-0.01	0.27

much) experience a high level of fear of motion, while their level of physical impairment is low. Finally, the subjects in the upper left corner of the graph (Too little) have a high level of physical impairment and experience little fear of motion.

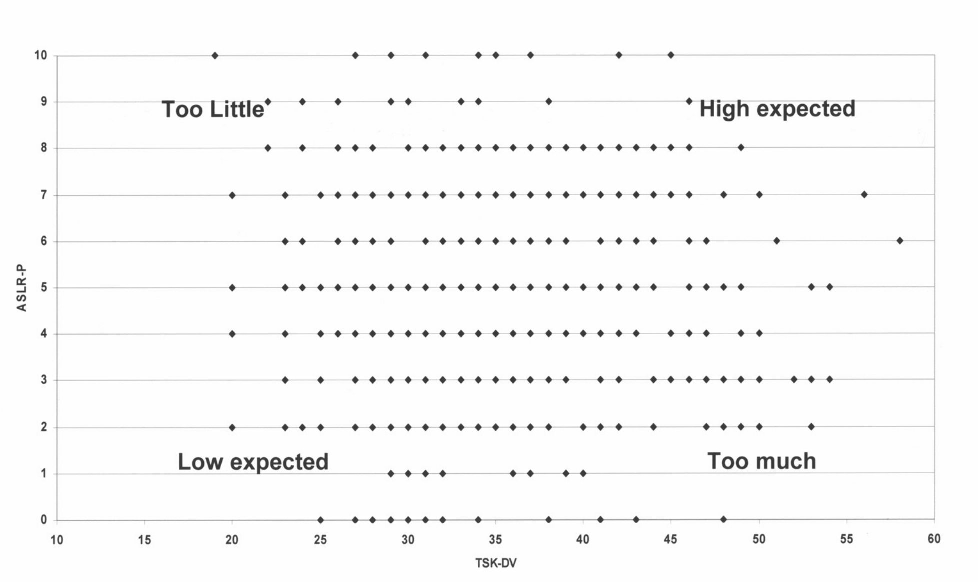


Figure 1. Visual representation of TSK-DV values versus Active Straight Leg Raise patient (ASLR-P) scores. Data are in a diffuse cloud shape; correlation coefficient = 0.04.

Discussion

Relation between fear of motion and physical impairment

This study examined whether there is an interrelationship between fear of motion (kinesiophobia) and the level of physical impairment in chronic PGP patients. The results show only a slight agreement between the TSK-DV and the QBDS and between TSK-DV and pain VAS, whereas QBDS and pain VAS show the highest agreement. Obviously TSK-DV has no or only a very limited relation to physical function or pain.

No agreement was found between the TSK-DV and the parameters used to assess physical impairment, i.e. ASLR, standing, walking, sitting or lying down. These latter parameters show interrelations with each other as to be expected, i.e. the more physically demanding activities (standing and walking) agree relatively strong with each other and with the ASLR. Sitting and lying down, being less physically demanding, are mutually correlated but have a weaker agreement with ASLR.

The very limited interrelationship of TSK-DV with all other parameters provides strong evidence that fear of motion is predominantly determined by factors (or combinations of factors) other than physical impairment. This conclusion is consistent with reports by Vlaeyen et al. and Reneman et al. [18,26,27].

ASLR as measure of physical impairment

Mens et al. described the ASLR as a measure of physical impairment and not as a pain provocation test [16]. As designed, the test is based on the interpretation of the patient [16]. Thus it can be argued that the ASLR is only a subjective interpretation and therefore invalid for objective analysis of impairment. To overcome this problem, in the present study the effort to actively perform a straight leg raise was also interpreted by the physician. The fact that both interpretations show a very strong correlation (0.77; Table 3) allows us to conclude that patients are able to provide an accurate estimation of their ASLR effort.

Physical impairment in patients with fear of motion

Fear of motion discourages patients in being physically active. This is both a problem and a challenge for clinicians who aim to increase the level of physical activity in chronic pelvic or low back pain patients. Questionnaires such as the TSK-DV help to estimate the level of fear of motion. Combined with an understanding of the patient's underlying psychological mechanisms, this can help to customise behavioural-based therapy to suit the individual's specific needs. However, in the present study no correlation was found between the TSK-DV and the ASLR. Consequently, chronic PGP patients with high levels of fear of motion show different levels of physical impairment. Fear of motion in chronic patients is usually considered in the context of little or no physical impairment [3-6,8]. Figure 1 demonstrates that this situation is more complex. Subjects in 'Low expected' and 'High expected' areas experience a fear of motion level that corresponds with their actual physical impairment, i.e. with little impairment there is little fear, and vice versa. Subjects in the 'Too much' demonstrate too much fear in relation to their relatively slight impairment. These latter patients can be considered as classic kinesiophobic patients, i.e. their (high level of) fear does not match their minor physical impairment. Finally, subjects in the 'Too low' area show too little fear in relation to their substantial physical impairment.

This clinically relevant, specific subgroup of chronic PGP patients with relatively little fear of motion has not been described in literature. For these patients, their daily activities may lead to physical overload thus contributing to the persistence and/or exacerbation of complaints. Perceiving too little fear of motion might cause these patients to have problems regarding the appropriate boundaries of their physical capacity. Consequently, it is difficult to motivate this type of patient to diminish their daily activities in order to make a proper recovery.

ASLR and fear of motion

Some comment is required regarding the set-up of the present study. To assess the level of physical impairment a physical performance test (ASLR) was used. Since fear of motion is known to affect physical performance, the question arises to what extent fear of motion might have affected the ASLR test [26-30]. To answer this question the ASLR procedure needs to be clarified. The initial posture for the ASLR is laying face upward on a couch. The required action is to raise one straight leg only 20 cm at a time. The test does not demand direct motion of the spine or of the pelvis. The requested activity is of a low velocity, low energetic level and is evaluated on effort, not on pain. However, it can not be totally excluded that fear of motion may to some extent affect the patient's performance of the ASLR test. On the other hand, because performing the test is not hazardous, does not cause strain, and does not directly involve painful areas of the patient, it was assumed that the impact of fear of motion on the ASLR test is minimal. This assumption is supported by the very low correlations between the TSK-DV and ASLR score (Tables 3, 4).

Conclusions

1. No clear-cut interrelationship was found between fear of motion and physical impairment in patients with chronic PGP.
2. Despite the presumed absence of tissue damage, chronic PGP patients can have slight to severe physical impairment, even after a prolonged period of time (i.e. more than 12 weeks).
3. Consequently, for proper assessment of a patient, it is essential to determine both the fear of motion and the physical impairment. The combination of this information is crucial when considering whether fear of motion in a patient is rational or not.
4. When fear of motion is not within the context of the physical impairment (irrational fear) there can be either too much or too little fear. Too little fear has never been addressed in literature, but is assumed to be clinically relevant. It is recommended that this specific subgroup of patients receives more attention because too little fear of motion may prevent a subject from finding an appropriate balance between load and capacity, thereby prolonging and/or exacerbating the complaints.
5. This study provides an explanation for the finding that not all patients with fear avoidance will benefit from graded exposure-based therapy.

6. Specific interrelationships between physical impairment, psychological factors and fear of motion need further study in different patient groups and for different pathologies and complaints.

References

1. Engel GL. The need for a new medical model: a challenge for biomedicine. Science 1977;196:129-36.
2. Engel GL. The clinical application of the biopsychosocial model. Am J Psychiatry 137:5 535-44.
3. Waddel G. The back pain revolution. Churchill Livingstone 1998.
4. Asmundson GJ, Norton PJ, Norton GR. Beyond pain: the role of fear and avoidance in chronicity. Clin Psychol Rev 1999;19:97-119.
5. Crombez G, Vlaeyen JW, Heuts PH et al. Pain-related fear is more disabling than pain itself: evidence on the role of pain-related fear in chronic back pain disability. Pain 1999; 80:329-39.
6. Grotle M, Vollestad NK, Brox JI. Clinical course and impact of fear-avoidance beliefs in low back pain: prospective cohort study of acute and chronic low back pain: II. Spine 2006;31:1038-46.
7. Kori SH, Miller RP, Todd, DD. Kinesiophobia: a new view of chronic pain behaviour. Pain Manage 1990;3:35-43.
8. Vlaeyen JW, Linton SJ. Fear-avoidance and its consequences in chronic musculoskeletal pain: a state of the art. Pain 2000;85:317-32.
9. O'Sullivan PB, Beales DJ. Diagnosis and classification of pelvic girdle pain disorders--Part 1: a mechanism based approach within a biopsychosocial framework. Man Ther 2007;12:86-97.
10. Ronchetti I, Vleeming A, van Wingerden JP. Physical characteristics of women with severe pelvic girdle pain after pregnancy: a descriptive cohort study. Spine 2008;33: 145-51.
11. Vleeming A, Albert HB, Ostgaard HC et al. European guidelines for the diagnosis and treatment of pelvic girdle pain. Eur Spine J 2008;17:794-819.
12. O'Sullivan PB, Beales DJ. Diagnosis and classification of pelvic girdle pain disorders, Part 2: illustration of the utility of a classification system via case studies. Man Ther 2007;12:e1-12.
13. O'Sullivan PB, Beales DJ, Beetham JA et al. Altered motor control strategies in subjects with sacroiliac joint pain during the active straight-leg-raise test. Spine 2002; 27:E1-8.

14. O'Sullivan P. Diagnosis and classification of chronic low back pain disorders: maladaptive movement and motor control impairments as underlying mechanism. Man Ther 2005;10:242-55.
15. Wu WH, Meijer OG, Bruijn SM et al. Gait in Pregnancy-related Pelvic girdle Pain: amplitudes, timing, and coordination of horizontal trunk rotations. Eur Spine J 2008;17:1160-9.
16. Mens JM, Vleeming A, Snijders CJ et al. Validity of the active straight leg raise test for measuring disease severity in patients with posterior pelvic pain after pregnancy. Spine 2002;27:196-200.
17. Mens JM, Vleeming A, Snijders CJ et al. Reliability and validity of the active straight leg raise test in posterior pelvic pain since pregnancy. Spine 2001;26:1167-71.
18. Vlaeyen JW, Kole-Snijders AM, Boeren RG et al. Fear of movement/(re)injury in chronic low back pain and its relation to behavioral performance. Pain 1995; 62:363-72.
19. Houben RM, Leeuw M, Vlaeyen JW et al. Fear of movement/injury in the general population: factor structure and psychometric properties of an adapted version of the Tampa Scale for Kinesiophobia. J Behav Med 2005;28:415-24.
20. French DJ, France CR, Vigneau F et al. Fear of movement/(re)injury in chronic pain: a psychometric assessment of the original English version of the Tampa scale for kinesiophobia (TSK). Pain 2007;127:42-51.
21. Leeuw M, Houben RM, Severeijns R et al. Pain-related fear in low back pain: a prospective study in the general population. Eur J Pain 2007;11:256-66.
22. Kopec JA, Esdaile JM, Abrahamowicz M et al. The Quebec Back Pain Disability Scale. Measurement properties. Spine 1995;20:341-52.
23. Davidson M, Keating JL. A comparison of five low back disability questionnaires: reliability and responsiveness. Phys Ther 2002;82:8-
24. Dyer, J.S. and Sarin, R.K. Measurable multiattribute utility functions. Operations Res 1979;810-22.
25. Bland JM, Altman DG. Correlation, regression, and repeated data. BMJ 1994;308:896.

26. Reneman MF, Jorritsma W, Dijkstra SJ et al. Relationship between kinesiophobia and performance in a functional capacity evaluation. J. Occup Rehabil 2003;13:277-85.
27. Reneman MF, Schiphorts HR, Geertzen M et al. Are pain intensity and Pain related fear related to capacity evaluation performances of patients with back pain? J Occup Rehabil 2007;17:247-58.
28. Geisser ME, Haig AJ, Wallbom AS et al. Pain-related fear, lumbar flexion, and dynamic EMG among persons with chronic musculoskeletal low back pain. Clin J Pain 2004;20:61-9.
29. Koho P, Aho S, Watson P et al. Assessment of chronic pain behaviour: reliability of the method and its relationship with perceived disability, physical impairment and function. J Rehabil Med 2001; 33:128-32.
30. Thomas JS, France CR. The relationship between pain-related fear and lumbar flexion during natural recovery from low back pain. Eur Spine J 2008;17:97-103.

Chapter 6

Balancing the biopsychosocial model for multidisciplinary treatment of non-specific chronic low back pain: merging motor control education and behavioural principles

J.P. van Wingerden[1,2], I. Ronchetti[1], A. Vleeming[1], R. Stoeckart[2], L. Burdorf[3], G-J. Kleinrensink[2]

[1]Spine & Joint Centre, the Netherlands, [2]Erasmus MC, Department of Neurosciences, [3]Erasmus MC, Department of Public Health

Abstract

Study Design

An uncontrolled open label trial investigating 245 patients with non-specific chronic low back pain (NCLBP).

Objective

The aim was to describe from a biopsychosocial (BPS) perspective the integration of physical exercise and behavioural principles within a therapeutic protocol for NCLBP and to demonstrate its therapeutic potential.

Summary of Background Data

Since the introduction of the BPS model, psychosocial aspects have dominated the multidisciplinary treatment of NCLBP. However, there are new and promising developments with respect to physical exercise for recovery from low back pain. It is assumed that integration of these new exercises with cognitive behavioural treatment protocols may be beneficial for therapy outcome.

Methods

A treatment protocol was developed with specific physical exercises integrated with cognitive behavioural principles. A group of 245 severe, seriously impaired and therapy-resistant NCLBP patients with average duration of complaints more that 9 years was treated according to this 8-week protocol. The protocol is described and the impact of the therapeutic approach is evaluated using the Quebec Back Pain Disability Scale (QBDS), a visual analogue scale for pain (VAS) basic function scores, medical consumption, and the patient's global impression of change.

Results

Medical consumption and use of medication decreased significantly after 8 weeks of therapy. Based on the patient's global impression of change, 73% of all patients experienced improvement. Based on the criteria of the minimal clinically important change, 62% of the patients significantly

improved on the QBDS and 53% on the pain VAS; in specific subgroups these beneficial results were even better (70% and 81%, respectively).

Conclusions

This study demonstrates that physical exercise can be successfully merged with cognitive behavioural principles in a multidisciplinary treatment protocol for NCLBP. Our findings support the assumption that therapy outcome improves when more attention is paid to the physical aspects of a multidisciplinary program. Creating and maintaining the balance between the BPS domains within a therapeutic protocol is a challenge for multidisciplinary teams.

Introduction

Multidisciplinary therapy based on the biopsychosocial (BPS) model has gained ground in the search for the most effective treatment of non-specific chronic low back pain (NCLBP) [1-5]. A major advantage of the BPS model is that besides biological factors it incorporates psychological and social aspects in the aetiology of chronic back pain thus broadening the spectrum of factors that may contribute to chronic back pain [6,7]. It was subsequently demonstrated that specific behaviours, such as fear avoidance, play a role in chronicity of low back pain [8-10]. Following this line of thought, behaviour modification therapies, developed for other behaviour-oriented disorders, were adopted for NCLBP patients [1,4,11-13]. The first results of these predominantly psychology-oriented therapies (e.g. cognitive-behavioural therapy, graded exposure and graded activity) were promising and in sharp contrast to the unsatisfactory results of traditional, monodisciplinary physical therapy [14-16]. Consequently, within the BPS model, in the treatment of these complaints the emphasis shifted from the physical to the psychosocial elements [3,16-21]. Presently, in many multidisciplinary NCLBP therapies, psychological aspects are predominant [1,4,13]. In these therapies changing the patient´s behaviour (especially socially-oriented behaviour such as returning to work, resuming housekeeping, child care, and re-participation in social life) is emphasised [14,22]. The physical domain is subordinate and the goals of physical therapy within the multidisciplinary programs are relatively shallow, i.e. general re-conditioning and physical re-activation of the patient [1,15,23-25.] The focus is on the quantity of activity, not the quality. This is not surprising since historically physical therapy has only limited options for addressing the qualitative aspects of function (flexibility, strength), while more quality-based therapies (e.g. the Mensendieck or Caesar therapy) still lack an adequate evidence-based foundation [26-28]. As a result, the role of a physical therapist within multidisciplinary protocols for NCLBP has diminished to that of a practical trainer or coach [15, 23-25].

Behavioural-based therapies appear to produce better results than regular exercise programs. Closer inspection of earlier studies reveals that the parameters used to evaluate the results of these therapies often specifically reflect their main objective, i.e. to have patients return to work and regain their social life. However, when parameters such as experienced pain or physical performance are taken into account, the results are far less positive [14,19,22,24,25,29-31]. Behavioural-based therapies may excel in changing behaviour, but the question remains whether they provide a cure for the patient's actual complaints, i.e. pain and functional limitations. In the process of learning how to change behaviour from the psycho-social perspective, the biological aspects may have been neglected.

In the last decades our understanding of the functioning of the locomotor system, in particular the pelvis and spine, has increased substantially[32-38]. In 1990, Vleeming et al. and Snijders et al. introduced the model of *'form and force closure'* which provides an explanation of how the joints of the pelvis (sacro-iliac joints) specifically, but also synovial joints in general, can be controlled by the interaction of a large number of different structures (like muscles, ligaments and capsules) in the proximity of the joint [39-42]. This model could be well integrated with new insights on control and stability of the lumbar spine [35,38,43-46]. It became clear that changes in neuromuscular control can cause non-optimal motion patterns in both the lumbar spine and in the pelvis, thus compromising physical capacity [47,48-51]. In turn, compromised physical capacity can lead to physical overload and pain [34,36,52]. It is important to note that these mechanisms operate in the absence of perceptible tissue damage and may last for a long period of time, even years [36,38-43,45,52,53] Based on this knowledge new forms of physical training have been developed and were found to be effective in patients with low back pain [45,53,54].

It is proposed that more focus on physical aspects within multidisciplinary programs will improve therapy outcome in NCLBP. To test this assumption the multidisciplinary team of a Dutch outpatient rehabilitation centre merged cognitive behavioural principles with specific physical training. The goal of the present study was to outline this integration of physical exercise and behavioural principles within the therapeutic protocol, and to provide preliminary results to endorse the therapeutic potential of this balanced multidisciplinary approach to NCLBP.

Patients and methods

Population

Between 2000 and 2007 regional pain clinics referred 402 patients diagnosed with chronic back and neck pain to a Dutch outpatient rehabilitation clinic (Rotterdam). From this latter group, 245 met the following inclusion criteria: pain predominantly in the region of the lower back; no significant structural deformations; complaints persisting for longer than 6 months; no acute radicular symptoms; received extensive treatment from a physical therapist; evaluated by at least one medical specialist (e.g. a rheumatologist, neurologist or orthopaedic surgeon); and adequate command of the Dutch language. Patients with multiple back surgery were excluded from the study.

Although 33% of the study population reported having experienced radicular symptoms in the past no acute radicular symptoms were present at the start of therapy. Since none of the patients had acute radicular symptoms, reported disc herniations (30%) were considered to be unrelated to the present complaints.

Of the original 402 selected patients, 148 were excluded because they did not meet the inclusion criteria and another 9 patients were excluded because of insufficient/unreliable data or because they stopped treatment. Therefore, the final evaluation is based on data of 245 (150 females, 95 males) NCLBP patients.

Treatment protocol

In the present therapy protocol, cognitive behavioural principles are applied to stimulate patients to adopt adequate behaviour aimed at physical recovery. The program, which was no isolated study design, but part of regular care, consists of 16 sessions of 3 hours each, over an 8-week period (total of 48 hours). Patients are divided into groups of 6 patients accompanied by three therapists. Each session includes training time (1 hour), a group lesson (1 hour) and individual coaching of the patient (1 hour). The objective of the lessons is to modify the patient's cognitions with respect to their complaints thus reinforcing proper behaviour. The lessons include information on functional anatomy of the spine, principles of chronic pain, the role and impact of emotions, communication, and finding the balance between the load of daily life and physical capacity.

The training is performed in a progressive sequence adjusted to the patient's specific situation and progress. Initially the patient's awareness of excessive or aberrant muscle tension is enhanced by means of relaxation and breathing exercises. Subsequently, patients learn to normalise muscle tension in unloaded situations using breathing and relaxation exercises, e.g. lying down, sitting and standing upright. Basic lumbar and pelvic stability is restored by motor control education using various stabilisation exercises for m. multifidus, m. transversus abdominis, the diaphragm and pelvic floor as suggested in literature [37,38,44-46+2]. When the patient is able to maintain basic lumbar stability in unloaded postures and without compensatory muscle activity, intensity of the exercises is increased in time, load and impact[44]. Control of proper muscle activation patterns is emphasised throughout the entire therapeutic process. Behavioural aspects that are specifically addressed include balancing physical capacity with the load of daily activities, and performing common activities like walking, running, bending forward and lifting.

In addition to the training and learning program, the patient is coached on an individual basis to cope with physical or mental impediments in performing the desired behaviour. Additional assistance can be provided by a manual therapist, psychologist or therapist specialised in body awareness (in Dutch called hapto-therapist).

Evaluation

Results of the therapy were evaluated on six parameters:

1. *Pain*, determined using the Visual Analogue Scale (Pain VAS).
2. *Limitations in daily life*, determined using the Quebec Back Pain Disability Scale (QBDS). [55,56]
3. *Basic function*, based on duration of walking, standing, sitting, lying down.
4. *Isometric trunk strength*, extension and rotation, using isometric strength testing equipment.
5. *Medical consumption*, obtained from questionnaires.
6. *Patient's global impression of change* (PGIC). [57]

Data were obtained during the intake procedure, after 8 weeks of therapy, and at the follow-up 3 months after the end of therapy.

All continuous variables were tested with the Student's t-test and in case of non-normally distributions with the Mann-Whitney U test. All categorical variables were tested with the Chi-Square test. Linear mixed models for analysis of variance with repeated measures was used to evaluate the change in the outcome measures limitations in daily life, pain, basic functions, and isometric strength directly after treatment and 3 months after the end of treatment. These models take account of the correlation between repeated measures on the same subject and allow for incomplete outcome data. In each model, the potential determinants of outcome measures were included as fixed (categorical) effects and the variances between and within subjects were regarded as random effects. The random variance component was pooled across all determinants of exposure and assumed to be equal across all fixed determinants (compound symmetry covariance structure). In all linear mixed models sex and education were included as determinant, since both variables had a significant effect on some outcomes measures and, thus, for reason of comparability were included in all models (table 3). SPSS version 10.01 and SAS version 8.02 were used for the statistical analyses of the data.

To demonstrate clinical relevance, for the QBDS and the pain VAS the minimal clinically important change (MCIC) was calculated, as proposed by Ostelo et al [58,59]. The MCIC was calculated as the percentage of patients with a more than 30% change compared with baseline.

Since baseline values may affect the MCIC outcome, the results were also analysed in subgroups based on three ranges of initial values. For the QBDS, patients were divided into groups with baseline values less than 45 (<45), from 45 to 65 (≥45 to <65), and 65 or higher (≥65). For the Pain VAS, patients were divided into groups with baseline values less than 50 (<50), from 50 to 70 (≥50 to <70), and 70 or higher (≥70).

Table 1. Demographic data of patients included in this study.

	Females **n = 150**	**Males** **n = 95**
Age (years)	43.9 ± 10.8	44.8 ± 8.6
Height (cm)	169.5 ±7.2	180.6 ± 12.8
Weight (kg)	71.6 ± 12.3	86.8 ± 15.9

Table 2. Anamnestic data as indication of the severity of complaints and average medical consumption.

Variable	**Value**
Duration of complaints (years)	9.2 ± 8.9
Legal procedure (claim or work related) (%)	20
Radicular symptoms (in history) (%)	33
Diagnosis of HNP* in history (%)	30
Denervations (%)	14
Previous rehabilitation (%)	14
Use of medication (%)	95
Previous pain injections (%)	82
Mean number of specialists consulted	3.8 ± 1.5
Mean number of paramedic sessions	73.2 ± 71.6
Use of aids for daily activities (%)	85

*HNP; herniated nucleus pulposus

Results

The subjects included in this study (Table 1) are NCLBP patients with an average duration of complaints of 9.2 years (SD 8.9) (Table 2). There is a significant, but small difference in intake values between men and woman for QBDS, standing and walking, and isometric strength (Table 3). At the start of therapy medical consumption was high: 95% of the patients used medication on a frequent basis which diminished to 40% after therapy.

After 8 weeks of therapy all outcome parameters significantly changed towards improvement compared to the level at intake (Table 3). This result was found for the whole study population, but also when men or woman were analysed separately. The Cohen's D for QBDS and Pain VAS (mean difference / baseline sd) at 8 weeks is 0.9 and 1.38, respectively, indicating a large improvement on physical limitations and pain. This change was maintained at 3-months follow-up. There was no significant change in data between the end of therapy and follow-up. The use of aids decreased from 85% to 9%.

Scores on the PGIC indicate that 73% of the patients experienced improvement (30% much better and 34% better); 20% of patients reported no change and in 7% the outcome was negative.

Based on the MCIC criteria, 62% of all patients significantly changed towards improvement on the QBDS and 53% on the pain VAS. Table 4 gives the MCIC results for subgroups of patients grouped by baseline scores on the QBDS and pain VAS. The best results were obtained by the subgroup with initial scores of 45 to 65 on the QBDS (70%) and scores from 50 to 70 on the pain VAS (81%).

Table 3. Therapy outcome measures. Represented are limitations in daily lifo (QBDS), Perceived pain (Pain VAS), basic functions and isometric strength. Sex ('Woman') was included as determinant, since it had a significant effect on most intake value. Values of 'Woman' and 'After 8 weeks treatment' are represented as differences from 'Intake'. '3 months after treatment' values are represented as differences from 'After 8 weeks treatment'.

	Intake	**Determinants**		
		Women	**After 8 weeks treatment**	**3 months after treatment**
QBDS	56 ± 16	7 ± 2*	-22 ± 1*	1 ± 1
Pain (VAS)	60 ± 21	4 ± 2	-19 ± 2*	0 ± 2
Basic functions				
Standing (min)	12 ± 15	-6 ± 2*	12 ± 1**	0 ± 1
Walking (min)	25 ± 22	-7 ± 2*	15 ± 1**	-1 ± 1
Sitting (min)	26 ± 21	-3 ± 2	14 ± 1**	1 ± 1
Lying down (min)	37 ± 24	-4 ± 2	9 ± 2**	1 ± 1
Isometric strength				
Extension (N.m)	119 ± 74	-87 ± 9*	66 ± 3**	0 ± 1
Rotation Left (N.m)	74 ± 46	-68 ± 5*	39 ± 2**	-6 ± 2*
RotationRight (N.m)	64 ± 47	-68 ± 5*	38 ± 2**	+4 ± 2*

* P < 0.05

Table 4. Clinical relevance of changes in limitations in daily life (QBDS) and pain (VAS) represented as the percentage of patients who met the MCIC criterion of a 30% change from the baseline value.

Parameter	**Group**	**Patients (%)**	**Group size (n)**
QBDS	**mean**	62	241
	< 45	57	60
	≥45, <65	70	105
	≥65	55	76
Pain VAS	**mean**	53	238
	< 50	35	63
	≥50, <70	81	87
	≥70	66	88

Discussion

Impact of combining physical exercise and behavioural principles
The goal of this study was to demonstrate the possibility and therapeutic potential of emphasising specific physical exercises within a multidisciplinary rehabilitation programme for NCLBP. The results of this therapy programme must be considered in perspective of the specific patient group. The patients in this group had complaints that persisted for more than 9 years on average. There were severe limitations in daily life (QBDS) and extensive consumption of medication. All patients had received extensive paramedical and medical treatment without adequate relief. Therefore, it can be concluded that the subjects in the present study were severe, seriously impaired and therapy-resistant NCLBP patients. Thus it is encouraging that, in contrast to their history of unsuccessful treatments, most of the patients in the present study experienced improvement (PGIC: 73% much better or better). This subjective experience is in line with the significant decrease in experienced pain (Pain VAS) and in diminished limitations in daily activity (QBDS). Isometric strength and function scores (duration of walking, standing, sitting and lying down) improved significantly. In the light of the long history of treatment and recurrence of complaints, the finding that the therapeutic effects remained after three months is also encouraging. Furthermore, medication use dropped from 95% to 40%, and in those patients still using medication the daily dosage was also reduced. The number of patients using aids for support reduced from 85% to only 9% indicating functional improvement in the majority of patients.

Results using the MCIC criterion
Evaluation of the QBDS and pain VAS using the MCIC criteria shows promising results. Limitations in daily life showed a significant decrease in 62% of all patients (QBDS) and 55% had significantly less pain (Pain VAS). For some subgroups this outcome was even more impressive: in patients with a baseline QBDS score of 45-65 and in patients with a baseline pain VAS of 50-70 almost 70% and 80%, respectively, showed a clinically significant improvement. Results are slightly less positive in patients with lower or higher baseline scores on the QBDS and pain VAS. Although it is

tempting to conclude that therapy is less effective in these latter groups this result may be more complex than it seems. For the more 'severe' patient group it is logical to assume that they need more time or care to obtain the results achieved in the 'average' group. However, this does not hold for the patients with lower baseline scores. It is more likely that the low baseline scores thwart a significant decrease in the outcome value, despite the use of a relative change (30% of the baseline value) instead of an absolute value (20 or 15 points on the QBDS or pain VAS, respectively) [58,59].

Additional value of specific physical training in a behavioural program

This study shows that specific physical training can be successfully emphasised within a behavioural program. Since the emphasise on physical training is the only adaptation in the behavioural programme this study also provides strong indications for the additional value of this specific combination. This latter result supports the assumption that physical functioning may be impaired in CLBP patients, despite the absence of recognisable tissue damage [38,52]. With impaired functioning, graded exposure or graded activity programs may not succeed without specific physical training. When physically impaired patients increase their level of activity without concern for the quality of the motion, overburdening may occur and consequently inflammation, leading to an increase of pain [52]. This undesired response to the increased level of activity leads to a negative experience and discourages the patient from continuing therapy.

Balance not dominance

This study advocates more appreciation for the role of physical aspects in multidisciplinary programs for non-specific CLBP; it does not claim that the physical aspect is the most important factor. Basically, the BPS model does not consider any single factor to be the most prominent. Important questions are how, and to what extent, should these factors be implemented in therapy. It seems logical to assume that these considerations largely depend on the individual's needs. In daily practice, however, it is not uncommon that the most dominant discipline in the multidisciplinary team will determine the focus of attention [15,24]. This often results in cognitive behavioral therapy, with physical training being subordinate to the behavioural aspects of the treatment; this is not in line with the essence of the BPS model [6,7].

Conclusions

The present study allows to draw the following conclusions:

1. It is possible to have a cognitive behavioural therapy for NCLBP with more focus on specific exercises, contributing to quality of behaviour.
2. There are strong indications that merging physical exercises with cognitive behavioural principles can improve the results of multidisciplinary treatment for NCLBP.
3. Finding and maintaining the balance between the BPS domains in a therapeutic protocol is a challenge for multidisciplinary teams.

References

1. Gatchel RJ, Peng YB, Peters ML, Fuchs PN, Turk DC. The biopsychosocial approach to chronic pain: scientific advances and future directions. Psychol Bull 2007;133:581-624.
2. Guzmán J, Esmail R, Karjalainen K, Malmivaara A, Irvin E, Bombardier C. Multidisciplinary bio-psycho-social rehabilitation for chronic low back pain. Cochrane Database Syst Rev. 2002;(1):CD000963.
3. Van Tulder MW, Koes B, Malmivaara A. Outcome of non-invasive treatment modalities on back pain: an evidence-based review. Eur Spine J 2006;15:S64-81.
4. Waddell G. 1987 Volvo award in clinical sciences. A new clinical model for the treatment of low-back pain. Spine 1987;12:632–44.
5. Waddell G, Burton AK. Concepts of rehabilitation for the management of low back pain. Best Pract Res Clin Rheumatol 2005;9:655–70.
6. Engel GL. The need for a new medical model: a challenge for biomedicine. Science 1977;196:129-36.
7. Engel GL. The clinical application of the biopsychosocial model. Am J Psychiatry 137:5 535-44.
8. Kori SH, Miller RP, Todd, DD. Kinisiophobia: a new view of chronic pain behaviour. Pain Manag 1990;3:35-43
9. Vlaeyen JW, Kole-Snijders AM, Boeren RG, van Eek H. Fear of movement/(re)injury in chronic low back pain and its relation to behavioral performance. Pain 1995;62:363-72.
10. Asmundson GJ, Norton PJ, Norton GR. Beyond pain: the role of fear and avoidance in chronicity. Clin Psychol Rev 1999;19:97-119.
11. Vlaeyen JW, Haazen IW, Schuerman JA, Kole-Snijders AM, van Eek H. Behavioural rehabilitation of chronic low back pain: comparison of an operant treatment, an operant-cognitive treatment and an operant-respondent treatment. Br J Clin Psychol 1995;34:95-118.
12. Vlaeyen JW, De Jong JR, Onghena P, Kerckhoffs-Hanssen M, Kole-Snijders AM. Can pain-related fear be reduced? The application of cognitive-behavioural exposure in vivo. Pain Res Manag 2002;7:144-53.
13. Waddell G. The back pain revolution. Second edition. Churchill Livingstone 2004.

14. Vendrig AA, van Akkerveeken PF, Sanders RJ. [Good results from a multidisciplinary and behavioral program for chronic back pain] Ned Tijdschr Geneeskd 2000;144:560-3.
15. Hildebrandt J, Pfingsten M, Saur P, Jansen J. Prediction of success from a multidisciplinary treatment program for chronic low back pain. Spine 1997;22:990-1001.
16. Hoffman BM, Papas RK, Chatkoff DK, Kerns RD. Meta-analysis of psychological interventions for chronic low back pain. Health Psychol 2007;26:1-9.
17. Keller A, Hayden J, Bombardier C, van Tulder M. Effect sizes of non-surgical treatments of non-specific low-back pain. Eur Spine J 2007;16:1776-88.
18. Klaber Moffett J, Mannion AF. What is the value of physical therapies for back pain? Best Pract Res Clin Rheumatol 2005;19:623-38.
19. Van Tulder WM , Waddell G. Evidence-based medicine for non-specific low back pain. Best Pract Res Clin Rheumatol 2005;19:7-9.
20. Van Tulder M, Koes B. Chronic low back pain. Am Fam Phys 2006;74:1577-9.
21. Van Tulder M, Koes B. Low back pain (chronic). Clin Evid 2006;15:1634-53.
22. van Geen JW, Edelaar MJ, Janssen M, van Eijk JT. The long-term effect of multidisciplinary back training: a systematic review. Spine 2007;32:249-55.
23. Kääpä EH, Frantsi K, Sarna S, Malmivaara A. Multidisciplinary group rehabilitation versus individual physiotherapy for chronic nonspecific low back pain: a randomized trial. Spine 2006;31:371-6.
24. Smeets RJ, Vlaeyen JW, Hidding A, Kester AD, van der Heijden GJ, van Geel AC, Knottnerus JA. Active rehabilitation for chronic low back pain: Cognitive-behavioral, physical, or both? First direct post-treatment results from a randomized controlled trial. BMC Musculoskelet Disord 2006;20; 7:5 doi:10.1186/1471-2474-7-5.
25. Smeets RJ, Vlaeyen JW, Hidding A, Kester AD, van der Heijden GJ, Knottnerus JA. Chronic low back pain: physical training, graded activity with problem solving training, or both? Pain 2008;134:263-76.
26. van Eijsden-Besseling MD, Staal JB, van Attekum A, de Bie RA, van den Heuvel WJ. No difference between postural exercises and strength and

fitness exercises for early, non-specific, work-related upper limb disorders in visual display unit workers: a randomised trial. Aust J Physiother 2008;54:95-101.

27. Haugstad GK, Haugstad TS, Kirste UM, Leganger S, Klemmetsen I, Malt UF. Mensendieck somatocognitive therapy as treatment approach to chronic pelvic pain: results of a randomized controlled intervention study. Am J Obstet Gynecol 2006;194:1303-10.

28. Soukup MG, Glomsröd B, Lönn JH, Bö K, Larsen S. The effect of a Mensendieck exercise program as secondary prophylaxis for recurrent low back pain. A randomized, controlled trial with 12-month follow-up. Spine 1999;24:1585-91.

29. Johnson RE, Jones GT, Wiles NJ, Chaddock C, Potter RG, Roberts C, Symmons DP, Watson PJ, Torgerson DJ, Macfarlane GJ. Active exercise, education, and cognitive behavioral therapy for persistent disabling low back pain: a randomized controlled trial. Spine 2007;32:1578-85.

30. Nicholas MK, Wilson PH, Goyen J. Comparison of cognitive-behavioral group treatment and an alternative non-psychological treatment for chronic low back pain. Pain 1992;48:339-47.

31. Ostelo RW, van Tulder MW, Vlaeyen JW, Linton SJ, Morley SJ, Assendelft WJ. Behavioural treatment for chronic low-back pain. 2005;25:Cochrane Database Syst Rev CD002014.

32. Adams MA. The Biomechanics of Back Pain, Churchill-Livingstone, 2002.

33. Bogduk N. The anatomical basis for spinal pain syndromes. J Manipul Physiol Ther 1995;18:603–5.

34. van Dieen JH, Cholewicki J, Radebold A. Trunk muscle recruitment patterns in patients with low back pain enhance the stability of the lumbar spine. Spine 2003;28:834-41.

35. Gracovetsky S. The Spinal Engine, Spinger, 1989.

36. Hides JA, Richardson CA, Jull GA. Multifidus muscle recovery is not automatic after resolution of acute, first-episode low back pain. Spine 1996;21:2763-9.

37. Hungerford B, Gilleard W, Hodges P. Evidence of altered lumbopelvic muscle recruitment in the presence of sacroiliac joint pain. Spine 2003;28:1593-600.

38. Hodges PW, Richardson CA. Inefficient muscular stabilization of the lumbar spine associated with low back pain. A motor control evaluation of transversus abdominis. Spine 1996;21:2640-50.
39. Snijders CJ, Vleeming A, Stoeckart R. Transfer of lumbosacral load to iliac bones and legs. Part 1 and 2. Clin Biomech 1993;8:285-94.
40. Vleeming A, Stoeckart R, Volkers ACW, Snijders CJ. Relation between form and function in the sacro-iliac joint, Part 1: Clinical anatomical aspects. Spine 1990a;15:130-2.
41. Vleeming A, Volkers ACW, Snijders CJ, Stoeckart R. Relation between form and function in the sacro-iliac joint, Part 2: Biomechanical aspects. Spine 1990b;15:133-6.
42. Vleeming A. The sacroiliac joint, a clinical-anatomical, biomechanical and radiological study. PhD Thesis, Erasmus University Rotterdam, the Netherlands, 1990.
43. Hodges PW, Richardson CA. Delayed postural contraction of transversus abdominis associated with movement of the lower limb in people with low back pain. J Spin Disord 1998;11:46-56.
44. McGill SM, Grenier S, Kavcic N, Cholewicki J. Coordination of muscle activity to assure stability of the lumbar spine. J Electromyograph Kinesiol 2003;13:353-9.
45. Richardson CA, Jull GA, Muscle control-pain control. What exercises would you prescribe? Man Ther 1995;1:2-10.
46. Richardson CA, Jull GA, Hodges PW, Hides JA. Therapeutic exercise for spinal segmental stabilisation in low back pain: scientific basis and clinical approach. Edinburgh, Churchill Livingstone, 1999.
47. Holm S, Indahl A, Solomonow M. Sensorimotor control of the spine. J Electromyography Kinesiol 2002;12:219-34.
48. Indahl A, Kaigle A, Reikeras O, Holm S. Electromyographic response of the porcine multifidus musculature after nerve stimulation. Spine 1995;20:2652-8.
49. Indahl A, Kaigle A, Reikeras O, Holm S. Interaction between the porcine lumbar intervertebral disc, zygapophysial joints, and paraspinal muscles. Spine 1997;22:2834-40.
50. Indahl A, Kaigle A, Reikeras O, Holm S. Sacroiliac joint involvement in activation of the porcine spinal and gluteal musculature. J Spinal Disord 1999;12:325-30.

51. van Wingerden JP, Vleeming A, Ronchetti I. Differences in standing and forward bending in women with chronic low back or pelvic girdle pain: indications for physical compensation strategies. Spine 2008;15;33:E334-41.
52. MacDonald D, Moseley GL, Hodges PW. Why some patients keep hurting their back? Evidence of ongoing back muscle dysfunction during remission from recurrent back pain. Pain (2009), doi:10.1016/j.pain.2008.12.002.
53. Taimela S., Negrini, S, Paroli C. Functional rehabilitation of low back disorders. Eur Med Phys 2004; 40:29-36.
54. Rasmussen-Barr E, Ang B, Arvidsson I, Nilsson-Wikmar L. Graded exercise for recurrent low-back pain: a randomized, controlled trial with 6-, 12-, and 36-month follow-ups. Spine 2009;34:221-8.
55. Hodges PW, Eriksson M.A.E., Shirley D et al. Intra-abdominal pressure increases stiffness of the lumbar spine. Journal of Biomechanics 2005;38:1873–1880.
56. Pool-Goudzwaard AL, Slieker ten Hove MC, Vierhout ME et al. Relations between pregnancy-related low back pain, pelvic floor activity and pelvic floor dysfunction. Int Urogynecol J Pelvic Floor Dysfunct. 2005;16:468-74.
57. Kopec JA, Esdaile JM, Abrahamowicz M et al. The Quebec Back Pain Disability Scale. Measurement properties. Spine 1995;20:341-52.
58. Davidson M, Keating JL. A comparison of five low back disability questionnaires: reliability and responsiveness. Phys Ther 2002;82:8-24.
59. Jaeschke R, Singer J, Guyatt GH. Measurement of health status. Ascertaining the minimal clinically important difference. Controlled Clin Trials 1989;10:407-15.
60. Ostelo RW, Deyo RA, Stratford P, Waddell G, Croft P, Von Korff M, Bouter LM, de Vet HC. Interpreting change scores for pain and functional status in low back pain: towards international consensus regarding minimal important change. Spine 2008;33:90-4.
61. van der Roer N, Ostelo RW, Bekkering GE et al. Minimal clinically important change for pain intensity, functional status, and general health status in patients with nonspecific low back pain. Spine 2006;31:578-82.

Chapter 7

Discussion

The main goal of this thesis is to explore whether a more prominent role of functional anatomy, as part of the physical domain within the biopsychosocial (BPS) model, improves multidisciplinary NCLBP therapy. This goal is pursued by defining the following research questions in perspective of multidisciplinary treatment of NCLBP:

1. Taking into account recent data on functional anatomy, is there a need to reconsider the role of the physical domain within the BPS model?
2. Will a more pronounced role of functional anatomy in the BPS model contribute to better diagnosis?
3. Will functional anatomy applied in the BPS model contribute to improved therapy?

7.1. Should the role of functional anatomy within the BPS model be reconsidered?

The question whether the role of functional anatomy within the BPS model should be reconsidered is subdivided in two other questions: First, is there actually new knowledge on functional anatomy and second, if so, does this knowledge have clinical significance?

7.1.1. Development of functional anatomical knowledge, an example

The description of the connection and interaction of the long head of the biceps femoris muscle with the sacrotuberous ligament (**chapter 2**) is an example of new knowledge in functional anatomy. The described dynamic connection has a clinical consequence since it implies that intrinsic stability of the sacroiliac (SI) joints is determined not only by local muscles. More distant muscles, in this case the m. biceps femoris, may contribute to SI stiffness as well. The study in **chapter 3** confirms the clinical implication of the anatomical finding *in vivo*. It is demonstrated in healthy subjects that activity of the m. biceps femoris indeed increases SI joint stiffness. The study also shows the influence of other muscles (m. gluteus maximus, m. erector spinae and m. latissimus dorsi) on SI joint stiffness. The study was designed to demonstrate the stabilizing effect of m. biceps femoris on the SI joint; it was not intended to determine the relative contribution of m. biceps femoris or other muscles to SI joint stability. However, this would be a logical next step in understanding the complex mechanism of SI joint stability.

The first two studies focus on the SI joint and the influence of a limited number of muscles was observed. Notwithstanding the limited scope, this information clearly demonstrates the importance and potential of the study of functional anatomy.

The next question concerns the clinical relevance of this functional anatomical information.

7.1.2. Functional anatomy in healthy subjects and patients; clinical relevance

The third study as described in **chapter 4** focuses on the clinical relevance of functional anatomical knowledge as presented in the previous studies. This third study is performed on healthy female subjects and female patients with NCLBP. In none of the patients an anatomical substrate explaining their complaints could be pointed out. And on gross observation they appear to be able to perform normal daily activities like bending forward. However, objective analysis of the relative contribution of the lumbar spine and pelvis (hip flexion) to forward bending shows specific and different motion patterns. Furthermore, in the patient group, two subgroups were identified with distinct altered motion patterns: the low back pain (LBP) and pelvic girdle pain (PGP) patients. Based on the first two studies and literature a functional anatomical explanation for the distinct patterns is proposed [2,11,18,25]. When joint stability is compromised, daily activities can only be performed with altered muscle function.

Consequently, altered muscle function changes motion patterns. It is to be expected that depending on the specific joint, part of a joint or combination of joints, other motion patterns will occur. Moreover it must be taken into account that such compensation patterns also can depend on the subjects preferred motion strategies.

As expected the human body is capable of using various motion strategies to perform daily activities. But not all possible strategies will be equally economic [6,7,32]. This leads to the assumption that motion strategies which compensate compromised joint stability are not economically optimal. Thus, abnormal motion patterns may lead to physical overload and consequently induce or prolong physical complaints such as pain. From this perspective functional anatomical analysis helps us to understand that physical factors can play a role in chronic back pain, even without detectable structural disorders [17]. This is in contrast with the present common clinical attitude towards chronic pain. Nowadays, many clinicians consider physical aspects, besides maybe deconditioning, irrelevant in chronic pain patients [30,31]. The present studies and recent literature suggest that this situation is more complex [17]. Based on physiological rules damaged tissue recovers within 12 weeks. However, this does not imply that proper function has restored also. Actually it is demonstrated by Richardson et al that functional disturbances can persist, sometimes even for years [9,17,23]. Consequently, physical function of a NCLBP patient should not be considered irrelevant. Functional anatomical analysis is crucial to provide information on physical (dys-) function of a patient.

7.1.3. Application of functional anatomy in other areas

At present many physical complaints have obscure aetiology, or worse, are primarily attributed to psychosocial factors [5,13,30]. More knowledge on functional anatomy will enhance our understanding of the biological aspects of these complaints. In this thesis the connection between the m. biceps femoris and the sacrotuberous ligament (**chapter 2**) is described in detail as an example of the complexity of functional anatomical mechanisms. Although this is only one relatively straightforward example, other functional anatomical systems have been studied, or are waiting to be unravelled. Examples of such systems are the function of m. transverse abdominis and m. multifidus as stabilizing muscles for the lumbar spine

[16,23]. In this perspective the discussion on the function of the thoracolumbar fascia is intriguing [1,7,8,29]. Several studies point towards a function of the pelvic floor and diaphragm in lumbar and pelvic support [19,21,22]. These new insights do not only provide guidelines for therapy, they may also provide explanations for numerous symptoms that could not be explained from the traditional descriptive anatomical perspective. Examples of these symptoms are the piriformis syndrome, pelvic floor incontinence, hyperventilation syndrome, short hamstrings and hypertone erector spinae.

The number of studies on functional anatomy still increases and it is clear that we are only at the beginning of the functional anatomical exploration of the human body. More research will be necessary to unravel and fully understand the functional anatomical mechanisms of the human body and its clinical consequences. Based on the first three studies in this thesis, and supported by literature, it can be concluded that the role of the physical domain within the BPS model with respect to NCLBP definitely needs reconsideration.

7.2. Better diagnosis with functional anatomy

The results of the study on coordination in **chapter 4** imply that functional anatomy contributes to quality of diagnosis. Obviously, more improved functional anatomical knowledge will make the diagnostic process more thorough and will provide valuable information for therapy; evidence based medicine includes evidence based diagnostics. The strength of the BPS model, however, lies especially in the interaction between the domains.

7.2.1. Difficulties in integration of information from domains within the BPS model

Combining information from different domains is difficult. One important factor is that traditionally disciplines work in one specific domain. Physical therapists work in the physical domain, whereas the psychologist works in the psychological domain and the social worker in the social domain. This creates a problem since therapists working within a specific domain are inclined to interpret clinical findings from their own perspective. Raised shoulders in a patient can be interpreted by the physical therapist as compensating behaviour due to shoulder or neck problems, while the psychologist may interpret this behaviour as a sign of defence or psychological discomfort.

Another problem is the different terminology between disciplines. For example a ‘functional complaint’ has a completely different meaning in the biological or psychological domain (disturbance of physical function or change in physical function related to psychological distress, respectively). Consequently, combining information from the domains is difficult and therefore, often omitted.

The study in **chapter 5** emphasises the importance of combining information from the different domains in achieving a balanced diagnosis.

7.2.2. Integrating information from the biological and psychological domains

The study in **chapter 5** focuses on combining a physical parameter (Active Straight Leg Raise, ASLR) with a psychological parameter (Tampa Scale for Kinesiophobia Dutch language Version, TSK-DV). The study shows that the information of the physical test alters the interpretation of the kinesiophobia scale. The TSK-DV provides information on fear of motion. According to physiological principles, damaged tissue recovers within 12 weeks. This principle easily leads to the assumption that in chronic pain (lasting longer than 12 weeks) a high score on the TSK scale is irrational since it can not be related to tissue damage. However, the ASLR test, as measure of physical impairment, shows that several patients have physical limitations, despite the presumed absence of tissue damage. Since impaired function is a potential threat for injury it can be expected that patients with such limitations are reluctant to move [17]. In these patients a high TSK-DV score is rational. In patients with only limited impairment, a low TSK-DV score is also consistent. Only in patients with a high TSK-DV score and limited impairment and in patients with a low TSK-DV score and vast impairment, the fear of motion could be considered disproportional or irrational. The first combination (High TSK-DV, Low ASLR) is the classical irrational fear of motion patient and frequently referred to in literature [13,28], the second group (Low TSK-DV, High ASLR = ‘too little fear’) is underexposed [15]. This latter group, however, is clinically relevant and difficult to treat. It consists of patients who according to their TSK-DV scores do not experience enough fear to move, causing them to frequently

cross their physical boundaries. This can lead to chronic physical overloading and, consequently, persisting pain. It is postulated that in this specific group of PGP patients, but possibly in other chronic pain patients also, other motivators than pain, e.g financial security or social status (family or work), are predominant in steering the patients behaviour [15].
The study in **chapter 5** shows that combining information from the biological domain (ASLR test) and the psychological domain (TSK-DV) contributes to a more subtle diagnosis. Combining TSK-DV and ASLR is only a small, but promising example of integration of domains within the BPS model. Integrating information from different domains is not easy but, as shown in this study, has potential to improve the diagnostic process. Therefore further exploration in this area is highly recommended.

7.3. Can functional anatomy improve therapy?

The first studies in this thesis show the potential of functional anatomy in analyzing physical function in healthy subject and chronic pain patients. It is also shown that combining functional anatomical information with psychological information enhances the diagnostic process. This leads to the final question: will functional anatomy applied within the BPS model contribute to a more successful therapy?

7.3.1. Acquisition and evaluation of therapy data

The study in **chapter 6** describes a multidisciplinary therapy where the physical domain has a more predominant role compared to regular cognitive behavioural therapy, graded exposure or graded activity. In this study the treatment protocol was applied within regular patient care and data were obtained from the regular therapy process. For evaluation purposes the Minimal Clinical Important Change (MCIC) criterion was used as described by Ostelo [20,24]. This method allows to determine effectiveness of the treatment based on cut-off values of the data. This method also allowed comparing the outcome of this study with other studies. Based on this method we could demonstrate the effectiveness of the present treatment protocol.

7.3.2. Comparison of therapy protocols

A problem related to the study in **chapter 6** was how to distinguish the content of the therapy protocol from other protocols. Most multidisciplinary approaches to back pain use combinations of physical treatment, lessons, group conversations and individual coaching [5,10,26,27]. On this level there are no outspoken differences between the protocols. Only when looking at the detailed content of the programme, differences can be pointed out. For that reason it was attempted to describe the treatment protocol as detailed as possible. Regrettably this is not done in most other study descriptions. Despite extensive protocol descriptions, minute but important details are often left out. It is these details that distinguish the protocols and contribute to the effectiveness of the method. Also in the present study it was difficult to describe the protocol in sufficient detail. A major reason for this problem is the limited space available in scientific articles. To overcome this problem we strongly recommend to supply such information by referring to an internet address or website where elaborate information is provided.

7.3.3. The surplus value of functional anatomy

The results of the multidisciplinary protocol as described in **chapter 6** are very promising. As far as could be compared, they are even better than the results of other multidisciplinary treatments [10,26,27]. However, the question remains whether it is especially the functional anatomy that makes the difference. First of all there is the limited description of therapy protocols which obstructs proper comparison of therapy content. Secondly the BPS model dictates an interaction of all contributing factors, not only in aetiology, but also in the healing process. This interaction makes it difficult to identify and rate the specific contributing factors. Communication between the patient and therapist will not be similar between the different multidisciplinary therapies. Social, environmental and even cultural circumstances will not be alike between populations. And finally behavioural principles will not be implemented in all protocols in the same manner. Therefore it can not be concluded that the main difference in the study in **chapter 6** is the attention for functional anatomy. However what we do know is that a vast majority of patients in this study experienced significant improvement both in pain and function. Therefore it is absolutely worthwhile to further investigate this treatment protocol in controlled studies. However, it must be taken into account that because of the fundamental interaction of domains, therapies based on the BPS model are very hard to study with current statistical methods.

7.4. Considerations

7.4.1. The contribution of functional anatomy in behavioural programs

Most contemporary therapies for NCLBP are based on changing behaviour quantitatively. There is no or only little attention for the quality of the behaviour.

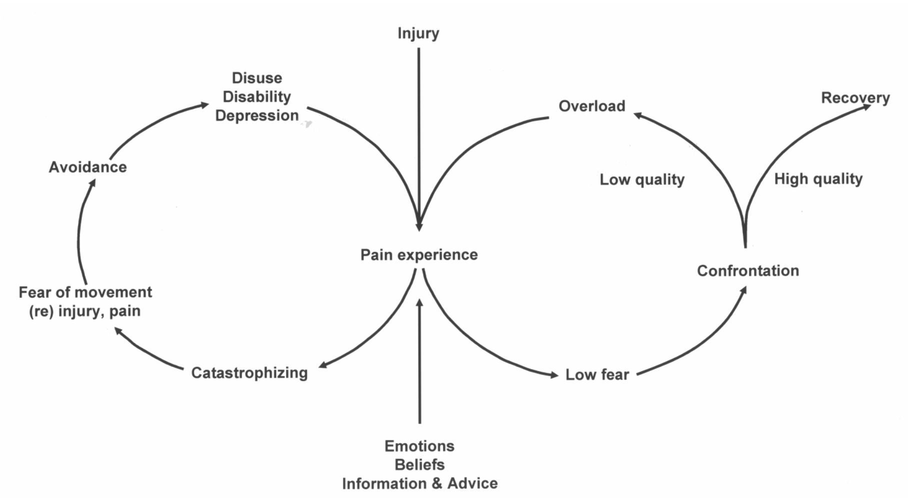


Figure 1. Fear avoidance model as shown in the introduction, figure 3, but adopted for quality of motion. If exercise is pursued in a low quality manner there is a potential risk of overload and re-injury leading back into the fear avoidance model. Only when confrontation is performed with high quality exercises, recovery will be achieved (adopted from Vlaeyen).

One example

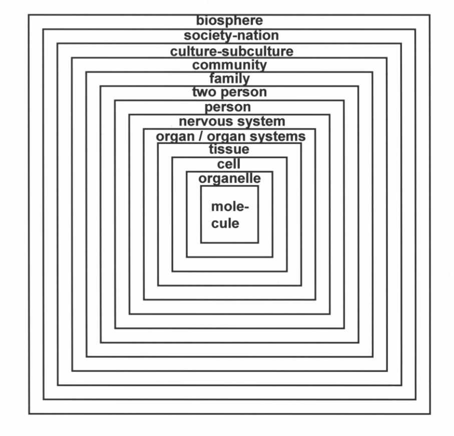
A patient suffering NCLBP states that he can walk only a hundred metres. For his daily work he must be able to walk a thousand metres. Most behavioural therapies aim at a gradual increase of the walking distance until the desired thousand metres are achieved. In this process there is no or little attention for the quality of the walking pattern. However, from a functional point of view low back pain patients often are not able to stand in a neutral lordosis, but adopt some lumbar flexion. In doing so, the patient can not efficiently transfer torsional energy from his legs to his trunk and vice versa. This energy transfer is crucial for effective, energy-economical walking [7,8,14].

Increasing the walking distance of the patient without improving the quality of walking will disproportionably load the spine. As a result this can induce overload, pain and in the end structural change of or even damage to the spine. The possibility that the patient straightens his spine and corrects his posture by coincidence is small. Therefore it should be considered a professional mistake not to focus on improving the quality of posture and the patient's walking pattern. It is to be expected that incorporation of qualitative behavioural modification based on functional anatomy significantly improves therapy results as illustrated in the modified model of Vlaeyen in figure 1.

7.4.2. The BPS model and medical disciplines

This thesis showed how functional anatomy can play a role in improving quality of a multidisciplinary treatment for NCLBP. It can be taken for granted that there will be more functional anatomical relations that play a role in low back pain aetiology. A vast amount of fundamental research on this specific topic is necessary to unravel these functional relations and validate their clinical impact. However, this research does not guarantee optimal balance of the BPS model. Finding a balance in the BPS model also includes more research with respect to the psychological and social domains. And finally balance can only be achieved when communication between medical disciplines is optimized [12]. This aspect of the BPS model is especially challenging because of the traditional separation of disciplines in the (para-)medical world However, if this succeeds their potentials can be merged to truly optimize the BPS model, as proposed by Engel [3,4].

Figure 2. Hierarchy of Natural systems (from: Engel 1980, Reprinted with permission of the American Journal of Psychiatry, (copyright 1980) American Psychiatric Association).



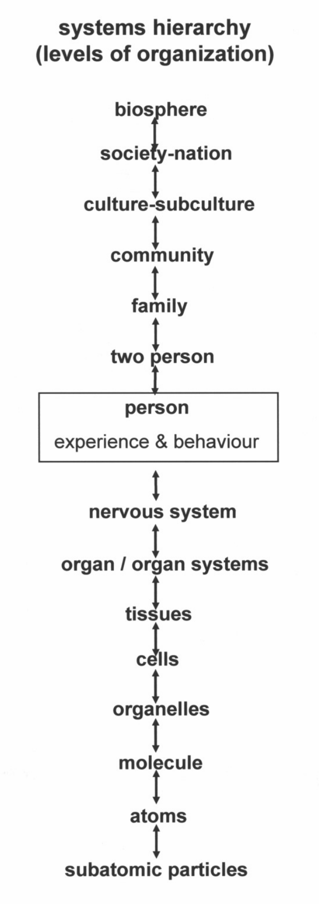


Figure 3. Continuum of Natural Systems (from: Engel 1980, Reprinted with permission of the American Journal of Psychiatry, (copyright 1980)American Psychiatric Association).

7.4.3. The social domain in the BPS model

The present thesis focuses on the balance between the biological and psychological domains in the BPS model. The fact that the social domain is left out is not coincidental. Engel's BPS model is based on the systems theory [3]. System theory states that nature is ordered as a hierarchically arranged continuum, with its more complex, larger units superordinate to the less complex, smaller units. This principle can be represented schematically by a vertical stacking to emphasize the hierarchy (see figure 2) and by a nest of squares to emphasize the continuum (see figure 3).

Engel places the individual in two hierarchies: the single individual (person) is the highest level of the organismic hierarchy and at the same time the lowest unit of the social hierarchy [4]. So Engel distinguishes the individual as an organism with a biological and psychological aspect. Furthermore the individual interacts with his social environment. From this viewpoint the biological and psychological domains belong to the first hierarchy, the social domain to the second hierarchy. In other words, the biological and psychological domains act from within the individual whereas the social domain remains outside the individual.

Although theoretical, this distinction has clinical relevance: in interaction with a patient medical workers can only affect biological and psychological aspects. To change environmental aspects of a patient is beyond the scope of the therapists.

An elementary example

A young man suffers from NCLBP. Because of the duration of absence of work as a result of the back complaints his employer will end his employment contract. Due to this fact and the resulting stress the young man is not able to follow the advice of the therapist to relax his low back and build up gradually.

In this example social factors determine the negative result of the therapeutic process. The therapist may try to alter the patient's attitude with respect to his social situation (psychologic domain). However, changing the actual social situation is not possible for the therapist. A social worker could in this case assist the young man in obtaining another job. But when mental stress is elicited by, for example, health problems of relatives, the options of a social worker are limited also.

In conclusion: the BPS model is designed as a multifactorial etiological model. To apply the model as a basis for treatment does not imply that every contributing factor can or should be addressed. Especially social factors are difficult to incorporate in the treatment.

7.5. Conclusions

This thesis provides strong arguments for the presumption that more emphasise on physical aspects within the contemporary BPS disease model improves the diagnostic process and therapeutic results. Of course this thesis does not reject that other (psycho-social) factors contribute to low back pain aetiology. It only propagates more appreciation for the biological domain, especially functional anatomy, within the BPS model.

It is to be expected that further study of human functional anatomy contributes to the revaluation of the bio-domain within the BPS model. This not only holds for the low back and pelvis, but for every other part of the human body, including the neck and extremities. New functional anatomical knowledge may contribute to comprehension of the aetiology of until now obscure syndromes like fibromyalgia, the post whiplash syndrome, and chronic fatigue syndrome.

The BPS model provides a profound and fertile basis for contemporary therapy. Extensive application of the model is not limited by the amount of research in this area. The prime restraint is found in the troublesome and often disturbed communication between disciplines isolated in their domain. To fully benefit from the principles of the BPS model it is strongly suggested to improve interdisciplinary communication and reconsider the traditional boundaries between medical, psychological and social disciplines.

Non-specific low back pain does not exist: it is only our lack of insight in aetiological mechanisms that leaves back pain unexplained. Balancing the factors within the BPS model will help to improve this insight.

References

1. Barker PJ, Briggs CA, Bogeski G. Tensile transmission across the lumbar fasciae in unembalmed cadavers: effects of tension to various muscular attachments. Spine 2004;29:129-38.
2. Dankaerts W, O'Sullivan PB, Straker LM, A.F. Burnett AF, Skouen JS. The inter-examiner reliability of a classification method for non-specific chronic low back pain patients with motor control impairment. Man Ther 2006;11:28–39.
3. Engel GL. The need for a new medical model: a challenge for bio-medicine. Science 1977;196:129-36.
4. Engel GL. The clinical application of the biopsychosocial model. Am J Psychiatry 1980;137:5 535-44.
5. Gatchel RJ, Peng YB, Peters ML, Fuchs PN, Turk DC. The biopsychosocial approach to chronic pain: scientific advances and future directions. Psychol Bull 2007;133:581-624.
6. Gracovetsky S, Kary M, Pitchen I, Levy S, Ben Said R. 1989 The importance of pelvic tilt in reducing compressive stress in the spine during flexion-extension exercises. Spine 1989;14:412-6.
7. Gracovetsky S. The Spinal Engine, Spinger, 1989.
8. Gracovetsky S. Is the lumbodorsal fascia necessary? J Bodyw Mov Ther. 2008;12:194-7.
9. Hides JA, Richardson CA, Jull GA. Multifidus muscle recovery is not automatic after resolution of acute, first-episode low back pain. Spine 1996; 21:2763-9.
10. Hildebrandt J, Pfingsten M, Saur P, Jansen J. Prediction of success from a multidisciplinary treatment program for chronic low back pain. Spine 1997;22:990-1001.
11. Hungerford B, Gilleard W, Hodges P. Evidence of altered lumbopelvic muscle recruitment in the presence of sacroiliac joint pain. Spine 2003;28:1593-600.
12. Jones M,Edwards I, Gifford L. Conceptual models for implementing biopsychosocial theory in clinical practice. Man Ther 2002;7:2–9.
13. Kori SH, Miller RP, Todd, DD. Kinisiophobia: a new view of chronic pain behaviour. Pain Manag 1990;3:35-43.

14. Lamoth CJ, Meijer OG, Wuisman PI, van Dieën JH, Levin MF, Beek PJ. Pelvis-thorax coordination in the transverse plane during walking in persons with nonspecific low back pain. Spine 2002;27:E92-9.
15. Lundberg M, Styf J. Kinesiophobia among physiological overusers with musculoskeletal pain. Eur.J. Pain:10.1016/j.ejpain.2008.08.004.
16. MacDonald DA, Moseley GL,Hodges PW. The lumbar multifidus: Does the evidence support clinical beliefs? Man Ther 2006;11:254–263.
17. MacDonald D, Moseley GL, Hodges PW. Why some patients keep hurting their back? Evidence of ongoing back muscle dysfunction during remission from recurrent back pain. Pain doi:10.1016/j.pain.2008.12.002.
18. McGill SM, Grenier S, Kavcic N, Cholewicki J. Coordination of muscle activity to assure stability of the lumbar spine. J Electromyograph Kinesiol 2003;13:353-9.
19. Mens J, Hoek van Dijke G, Pool-Goudzwaard A, van der Hulst V, Stam H.J. Possible harmful effects of high intra-abdominal pressure on the pelvic girdle. Biomech 2006;39:627-35.
20. Ostelo RW, Deyo RA, Stratford P, Waddell G, Croft P, Von Korff M, Bouter LM, de Vet HC. Interpreting change scores for pain and functional status in low back pain: towards international consensus regarding minimal important change. Spine 2008;33:90-4.
21. Pool-Goudzwaard A, van Dijke GH, van Gurp M, Mulder P, Snijders C, Stoeckart R. Contribution of pelvic floor muscles to stiffness of the pelvic ring. Clin Biomech 2004;19:564-71.
22. Pool-Goudzwaard AL, Slieker ten Hove MC, Vierhout ME, Mulder PH, Pool JJ, Snijders CJ, Stoeckart R. Relations between pregnancy-related low back pain, pelvic floor activity and pelvic floor dysfunction. Int Urogynecol J Pelvic Floor Dysfunct 2005;16:468-74.
23. Richardson CA, Jull GA, Muscle control-pain control. What exercises would you prescribe? Man Ther 1995;1:2-10.
24. van der Roer N, Ostelo RW, Bekkering GE, van Tulder MW, de Vet HC. Minimal clinically important change for pain intensity, functional status, and general health status in patients with nonspecific low back pain. Spine 2006;31:578-82.
25. Slaboda JC, Boston JR, Rudy TE, Lieber SJ. Classifying subgroups of chronic low back pain patients based on lifting patterns. Arch Phys Med Rehabil 2008;89:1542-1549.

26. Smeets RJ, Vlaeyen JW, Hidding A, Kester AD, van der Heijden GJ, van Geel AC, Knottnerus JA. Active rehabilitation for chronic low back pain: Cognitive-behavioral, physical, or both? First direct post-treatment results from a randomized controlled trial. BMC Musculoskelet Disord 2006;20;7:5.
27. Smeets RJ, Vlaeyen JW, Hidding A, Kester AD, van der Heijden GJ, Knottnerus JA. Chronic low back pain: physical training, graded activity with problem solving training, or both? Pain 2008;134:263-76.
28. Vlaeyen JW, Kole-Snijders AM, Boeren RG, van Eek H. Fear of movement/(re)injury in chronic low back pain and its relation to behavioral performance. Pain 1995;62:363-72.
29. Vleeming A, Pool-Goudzwaard AL, Stoeckart R, van Wingerden JP, Snijders CJ. The posterior layer of the thoracolumbar fascia. Its function in load transfer from spine to legs. Spine 1995;20:753-8.
30. Waddell G. The back pain revolution. Second edition. Churchill Livingstone, 2004.
31. Waddell G, Burton AK. Concepts of rehabilitation for the management of low back pain. Best Pract Res Clin Rheumatol 2005;9:655–70.
32. Wu WH, Meijer OG, Bruijn SM, Hu H, van Dieën JH, Lamoth CJ, van Royen BJ, Beek PJ. Gait in Pregnancy-related Pelvic girdle Pain: amplitudes, timing, and coordination of horizontal trunk rotations. Eur Spine J 2008;17:1160-9.

Summary

The work presented in this thesis examines whether a more prominent role of functional anatomy within multidisciplinary treatment of non-specific chronic low back pain (NCLBP) will increase its therapeutic effect. The multidisciplinary treatment of NCLBP is based on the biopsychosocial (BPS) model. This model is derived from systems theory and was introduced by Engel in 1977 to replace the traditional biomedical model.

Fundamental to the BPS model is that not only biological but also psychological and social aspects are included in the aetiology of diseases, such as chronic back pain. However, in the ongoing development of new diagnostic and therapeutic techniques based on this BPS model, the behavioural aspects prevailed whilst the physical aspects (especially physical exercises) lagged behind. Consequently, in contemporary multidisciplinary treatment protocols, physical training is subordinate and mainly in service of the desired modification of behaviour.

Recent studies of multidisciplinary programmes for NCLBP show that the results of these predominantly psychological, behaviour-orientated treatments are far from optimal. Therefore, the question arose as to how multidisciplinary treatment can be improved. One option for improvement is to intensify the application of functional anatomical knowledge and incorporate corresponding specific training within existing multidisciplinary programmes.

Functional anatomical research has made significant progress in the last decade; this has led to new knowledge on spine function and,

consequently, to the development of new physical exercises. In the context of multidisciplinary treatment of NCLBP patients, and based on the new functional anatomical knowledge, the aim of this thesis was to address the following questions:

1. Taking into account the available recent data on functional anatomy, is there a need to reconsider the role of the physical domain within the BPS model?
2. Will a more pronounced role of functional anatomy in the BPS model contribute to better diagnosis?
3. Will functional anatomy applied in the BPS model contribute to improved therapy?

Study 1

In the first study, which used human dissection material, an anatomical connection was demonstrated between a pelvic ligament (the sacrotuberous ligament) and a leg muscle (the m. biceps femoris). The study shows that - in every case - a part of the muscle's pulling force tenses the ligament (7%-69% of the pulling force). From this study we concluded that it is theoretically possible that a leg muscle, due to its connection with the pelvic ligament, plays a dynamic role in controlling sacro-iliac (SI) joint stiffness. This implies that SI joint control is more complex than formerly presumed and that anatomical structures, even when not in direct proximity of the joint (e.g. the arm or leg muscles), can be involved in this control. This conclusion holds not only for the SI joints, but for other synovial joints in the body as well.

Study 2

Since the first study was performed on embalmed human material, it was considered essential to confirm the assumed influence of muscles on SJ joint stability on living subjects. Therefore the second study was performed on living, healthy subjects. In 6 young women the effect of muscle tension on SI joint stiffness was recorded using echo-Doppler in combination with vibration and EMG measurements. First, SI joint stiffness without muscle activation was determined using echo-Doppler. Next, the subjects were asked to activate specific muscles in isolation (homolateral: m.biceps femoris, m. gluteus maximus, m. erector spinae; and heterolateral: m. latissimus dorsi). During this muscle activation (controlled with EMG measurement), SI joint stiffness was determined again with echo-Doppler. For many subjects it was difficult to activate the muscles in isolation. Nevertheless, it was demonstrated that the muscles included in this study, even those not in the proximity of the SI joint, were able to increase SI joint stiffness. Consequently, a relationship was assumed between disturbed muscle activation patterns and diminished control of the SI joint. In turn, diminished muscle control may induce overload, even under conditions of normal load. Such relative overload can cause physical complaints. In the context of this thesis, this is the first evidence that functional anatomical information can be of importance in explaining the onset or continuation of complaints of the low back or pelvis.

Study 3

The results of the first and second study led to the conclusion that different muscle activations lead to distinct motion patterns. In patients with low back and pelvic problems, it is presumed that those changes in motion will appear during activities that involve the lower back and pelvis, such as bending forward. This third study explored the relative contribution of the lower back and the pelvis (hip motion) during forward bending. The action of forward bending was investigated and determined in healthy women, in women with pelvic girdle pain (PGP), and in women with low back pain (LBP). The coordination pattern of the women with complaints is distinctly different from that of the healthy subjects. Moreover, the coordination pattern of the women with PGP is opposite to that of the women suffering from LBP. Whereas women with LBP fix their spine during the bending motion, women with PGP emphasise lumbar motion and vice versa. This is a clear indication of a specific complaint and its relation to the motion pattern. Therefore, it seems feasible to assume that more understanding and appropriate therapeutic handling of these specific motion patterns will be of value for the recovery process. This is the second argument in favour of increasing physical information and applications in multidisciplinary therapy for NCLBP.

Study 4

The fourth study emphasises the importance of integration of biological (physical) and psychological factors in the diagnostic process. This study was performed among women with chronic PGP. The physical impairment, as measured with the Active Straight Leg Raise (ASLR) test, is compared with the fear of motion, as measured with the Tampa Scale for Kinesiophobia Dutch language Version (TSK-DV). It was shown that ASLR and TSK-DV measure distinct aspects of the patient and that there is no interrelationship between the physical limitations and the fear of motion. In line with this result, it is suggested that two specific subgroups can be distinguished: patients with a logical relation between the recorded impairment and fear of motion, and patients without a logical relation between physical impairment and fear of motion. It is important to note that this latter group can be further subdivided in two groups. Namely, one group which has little physical impairment, but has considerable fear of motion; this group can be considered as the 'classical' group with too much fear of motion. The other group has, curiously enough, too little fear of motion with respect to the considerable amount of physical impairment. This combination is clinically recognized in patients that ignore their functional limitations and attempt to persist functioning on a relatively high level. Although this behaviour contributes to persistence of the complaints and is, as such, clinically relevant, this specific group has not yet been described in the literature in this particular context.

Study 5

The fifth study describes a multidisciplinary treatment protocol for patients with NCLBP. In this protocol a specific exercise programme is merged with cognitive behavioural principles. The study is performed as part of regular patient care and not as an isolated, randomized study. The population included 245 severe therapy-resistant NCLBP patients. It is concluded that, in contrast to other programmes where physical training is subordinate, physical exercise can be leading within a behaviour-oriented approach. Moreover, this specific combination yielded remarkably positive results in the majority of patients that received this treatment (55%-80% significant improvement, depending on the parameters and group selected).

Based on the studies presented in this thesis the research questions originally posed can be positively answered as follows:

1. Functional anatomy indeed deserves more emphasis in the multidisciplinary treatment of NCLBP.
2. Functional anatomy within the BPS model can improve the diagnostic process in NCLBP.
3. There is sufficient evidence to suggest that functional anatomy can help improve the multidisciplinary treatment of NCLBP.

Samenvatting

Dit proefschrift richt zich op de vraag of een meer prominente rol van functionele anatomie in een multidisciplinaire behandeling voor a-specifieke chronische lage rugklachten (ACLR) het effect van deze behandeling verhoogt. De multidisciplinaire behandeling is gebaseerd op het biopsychosociale (BPS) model. Dit van de systeemtheorie afgeleide model is in 1977 geïntroduceerd door Engel ter vervanging van het traditionele biomedische model. Fundamenteel in het BPS model is dat niet alleen biologische maar ook psychologische en sociale aspecten worden betrokken in de etiologie van aandoeningen zoals chronische rug pijn. Bij de ontwikkeling van nieuwe diagnostische en therapeutische technieken, gebaseerd op het BPS model, traden de gedragsmatige aspecten op de voorgrond terwijl fysieke aspecten, en met name oefenvormen achterbleven. Dit had tot gevolg dat in hedendaagse multidisciplinaire behandelprotocollen de lichamelijke training ondergeschikt is en ten dienste staat van de gewenste gedragssturing.

Uit recente studies van multidisciplinaire behandeling van ACLR blijkt dat de resultaten van deze overwegend psychologische, gedragsgeoriënteerde behandelvormen nog verre van optimaal zijn. Dit is aanleiding om na te gaan of de multidisciplinaire behandeling nog verbeterd kan worden. Een mogelijkheid tot verbetering ligt in het intensiever toepassen van functioneel anatomische kennis en daaraan gekoppelde specifieke training binnen de bestaande multidisciplinaire programma’s. Het onderzoek naar functioneel anatomische kennis van het bewegingsapparaat heeft de laatste jaren een sterke ontwikkeling doorgemaakt die heeft geleid tot nieuwe inzichten in de werking van de wervelkolom en het bekken . Daarop aansluitend zijn nieuwe vormen van fysieke training ontwikkeld. Met deze nieuwe functioneel anatomische kennis als uitgangspunt is het doel van dit proefschrift om, in de context van multidisciplinaire behandeling van patiënten met ACLR, een antwoord te formuleren op de volgende drie vragen:

1. Dient de rol van het fysieke domein binnen het BPS model heroverwogen te worden, de recente ontwikkelingen van functioneel anatomische kennis in aanmerking nemende?
2. Kan een meer uitgesproken rol voor functionele anatomie in het BPS model bijdragen aan een betere diagnose van lage rugklachten?
3. Kan meer aandacht voor functionele anatomie toegepast binnen een multidisciplinaire behandeling bijdragen aan een betere therapie voor ACLR?

Eerste studie

In de eerste studie verricht op menselijke preparaten wordt een anatomische verbinding aangetoond tussen een van de banden van het bekken, de sacrotuberale band, en een van de beenspieren, de m. biceps femoris. De studie laat zien dat in alle gevallen een deel van de kracht waarmee de spier aan de band trekt deze op spanning brengt (7 tot 69% van de trekkracht). Uit deze studie wordt geconcludeerd dat het theoretisch mogelijk is dat de bewuste beenspier, door de beschreven verbinding met de bekkenband, een dynamische rol kan spelen bij het controleren van de stijfheid van de sacro-iliacale (SI) gewrichten. Dit impliceert dat controle over het SI gewicht complexer is dan voorheen werd verondersteld en dat anatomische structuren die verder van het gewricht verwijderd zijn, zoals been- of zelfs armspieren, hierbij betrokken kunnen zijn. De bevinding dat verder weggelegen spieren een rol kunnen spelen bij gewrichtssturing geldt niet alleen voor de SI gewrichten, maar ook voor andere synoviale gewrichten in het lichaam.

Tweede studie

De eerste studie heeft plaats gevonden op gefixeerde en geprepareerde lichamen. Het was daarom noodzakelijk de in de eerste studie veronderstelde werking van spieren op de SI gewrichten ook bij levende proefpersonen (*in vivo*) aan te tonen. Bij 6 proefpersonen is met Echo-Doppler in combinatie met vibratie, en EMG-metingen de invloed van spierspanning op de stijfheid van het SI gewricht gemeten. Eerst is met de Echo-Doppler de stijfheid van de SI gewrichten in rust bepaald. Vervolgens is aan de proefpersonen gevraagd bepaalde spieren (ipsi lateraal: m.biceps femoris, m. gluteus maximus, m. erector spinae en contralateraal: m. latissimus dorsi) aan te spannen. Tijdens deze spieractivatie die werd gecontroleerd met de EMG metingen, is met de Echo-doppler weer de stijfheid van de SI gewrichten bepaald. Voor veel proefpersonen bleek het lastig de verschillende spier(groepen) werkelijk geïsoleerd aan te spannen. Desondanks kon aangetoond worden dat de in deze studie betrokken spieren de stijfheid van het SJ gewricht konden vergroten. Ook wanneer deze spieren niet direct rond het SI gewricht liggen. Op basis van deze bevindingen wordt een samenhang verondersteld tussen een verstoring in activatiepatronen van deze spieren en verminderde controle over het SI gewricht. Een verminderde controle over de SI gewrichten kan, ook bij gelijk blijvende belasting, een relatieve overbelasting veroorzaken. Dergelijke overbelasting kan aanleiding zijn tot klachten. In het kader van dit proefschrift is dit de eerste aanwijzing dat functioneel-anatomische informatie van belang is bij het verklaren van het ontstaan of voortduren van rug- en bekkenklachten.

Derde studie

Op basis van literatuur en de eerste en tweede studie wordt aangenomen dat veranderingen in spieractivatiepatronen kunnen leiden tot veranderingen in bewegingspatronen. Bij mensen met rug- of bekkenklachten ligt het voor de hand te veronderstellen dat dit onder meer tot uiting zal komen bij bewegingen waarbij rug en bekken betrokken zijn, zoals bij voorover buigen. In deze studie wordt de relatieve bijdrage van de lage rug en het bekken (heupbeweging) tijdens het voorover buigen beoordeeld. Deze beweging werd vastgelegd bij gezonde vrouwen, vrouwen met bekkenklachten en vrouwen met rugklachten. De coördinatie van de vrouwen met klachten wijkt af van de coördinatie van de gezonde vrouwen. Tussen de vrouwen met bekkenklachten en de vrouwen met rugklachten is de coördinatie echter precies omgekeerd. Waar de vrouwen met rugklachten de lage rug tijdens de beweging fixeren, leggen de vrouwen met bekkenklachten juist de nadruk op de beweging van de lage rug, en omgekeerd. Dit is duidelijke aanwijzing voor een verband tussen de specifieke klacht (bekken of rug) en de wijze van bewegen. Het ligt daarbij voor de hand dat inzicht in en therapeutisch aangrijpen op dit specifieke bewegingspatroon van meerwaarde kan zijn voor herstel. Dit is een tweede argument voor het intensiever betrekken van informatie over het fysiek functioneren in multidisciplinaire programma's.

Vierde studie

De vierde studie benadrukt het belang van het integreren van biologische (fysieke) en psychische factoren in het diagnostische proces. Deze studie is uitgevoerd bij vrouwen met chronische bekkenklachten. De fysieke beperking, gemeten met de Active Straight Leg Raise (ASLR) test, wordt vergeleken met de mate van bewegingsangst, gemeten met de Tampa Scale voor Kinesiophobia Dutch language Version (TSK-DV). Uit dit onderzoek blijkt dat de ASLR en de TSK-DV geheel verschillende aspecten van een patiënt meten. Er blijkt dan ook geen enkel verband tussen de gemeten fysieke beperking en de angst voor bewegen. Juist daarom is het van belang dat een aantal subgroepen wordt onderscheiden, te weten: a. patiënten waarbij er een logisch verband is tussen de gemeten beperking en de bewegingsangst en b. patiënten zonder een logisch verband tussen de fysieke beperking en de bewegingsangst. Het is van belang op te merken dat deze laatste groep weer in twee geheel verschillende groepen is op te splitsen. Eén deel van de patiënten in deze groep heeft weinig fysieke beperking maar wel veel bewegingsangst. Deze subgroep kan gezien worden als de klassieke groep met teveel angst voor bewegen. Daarnaast is ook een groep die in verhouding tot de gemeten beperking juist merkwaardig weinig bewegingsangst heeft. Dit beeld wordt klinisch herkend als een groep patiënten die de klachten negeert en tracht te blijven functioneren op een verhoudingsgewijs te hoog niveau. Hoewel dit gedrag bijdraagt bij aan het in stand houden van de klachten als zodanig dus klinisch relevant is, is deze laatste groep in literatuur echter nog niet eerder op deze wijze beschreven.

Vijfde studie

In de vijfde studie wordt een multidisciplinaire behandeling voor patiënten met ACLR beschreven waarin een specifiek fysiek oefenprogramma wordt geïntegreerd met cognitief gedragsmatige principes. Deze studie is uitgevoerd als onderdeel van reguliere patiëntenzorg en niet als geïsoleerde, gerandomiseerde studie. De populatie die is gebruikt in deze studie bestond uit 245 ernstige, therapie resistente ACLR patiënten. Het wordt vastgesteld dat het mogelijk is om, in tegenstelling tot andere programma's waar fysieke training ondergeschikt is, meer nadruk te leggen op een fysiek oefenprogramma binnen een gedragsgeoriënteerde benadering. Bovendien blijkt dat deze specifieke combinatie bijzonder positieve resultaten geeft bij een groot deel van de behandelde patiënten (55 tot 80% significante verbetering afhankelijk van de gekozen parameter en groep).

Op basis van de studies in dit proefschrift kunnen de eerder gestelde vragen als volgt positief worden beantwoord:

1. Functionele anatomie dient meer betrokken te worden in multidisciplinaire behandelingen van ACLR.
2. Functionele anatomie kan binnen het BPS model bijdragen aan een betere diagnose bij ACLR.
3. Er zijn duidelijke aanwijzingen dat functionele anatomie kan bijdragen aan een betere therapie voor ACLR.

Dankwoord

Van dit proefschrift heb ik het schrijven van het dankwoord als een van de moeilijkste stukken ervaren. Voor een rechtgeaarde doe-het-zelver is het al niet gemakkelijk hulp te vragen. Maar als je meer dan 10 jaar over het schrijven van je proefschrift doet dan ontkom je er niet aan onderweg hulp te moeten vragen. En hulp heb ik gekregen van veel verschillende mensen op veel verschillende manieren. Graag wil ik al die mensen bedanken voor hun bijdrage aan dit boekje en mijn pad. Iedereen persoonlijk bedanken gaat niet lukken, maar een aantal mensen wil ik zeker noemen.

Beste Chris, onze samenwerking was kort en vooral krachtig. Ik heb je doortastende, no-nonsense begeleiding in het laatste en meest hectische stuk van dit proefschrift zeer gewaardeerd. Jouw rotsvaste vertrouwen in mijn stukken (of was dat meer in Gert-Jan en Rob?), heeft mij zeer geholpen dit proefschrift ook echt af te ronden.

Beste Rob, dank je voor je geduldige, nooit aflatende, liefdevolle, maar meedogenloze begeleiding. Op alle vlakken weet je steeds weer scherpzinnig de juiste snaar te raken, of je rust niet voor hij geraakt wordt. Bovendien heb je me, samen met Corrie, figuurlijk, maar ook letterlijk de ruimte gegeven om dit proefschrift te laten groeien. Wil je me nu leren hoe je mooie teksten schrijft?

Beste Gert-Jan, jouw benadering van wetenschap blijft onnavolgbaar: bodemloze kennis verpakt in razendsnelle humor op zen-boeddistische basis. En dat gecombineerd met ongeëvenaarde sociale vaardigheden. Ik hoop in de toekomst nog veel met je samen te mogen beleven, te leren.... en te jammen?

Lieve Ria, hoe waardeer je het opkomen van de zon in de ochtend? Zo vanzelfsprekend maar tegelijk zo onmisbaar en onbeschrijflijk. Op jou is stelling 11 absoluut van toepassing. Dank je voor alle dingen die je voor me gedaan en geregeld hebt maar bovenal omdat je een van mijn vrienden bent.

Beste Astrid, je hebt me geholpen tijd vrij te maken voor dit proefschrift. Zonder die ruimte was dit werk nooit afgerond.

Andry, het helpen afronden van jouw proefschrift was het begin van dit proefschrift. De jaren die ik voor jou gewerkt heb zijn voor mij een kostbaar leerproces geweest.

Beste Chris, op jouw afdeling aan het ErasmusMC (afdeling Biomedische Natuurkunde en Technologie) heb ik mogen leren hoe onderzoek doen werkt. Ik ben je dankbaar voor de plaats die je me daar gegund hebt.

Professor Voogd, op uw afdeling (Anatomie) mocht ik mij verder ontwikkelen. Dank u dat u mij deze mogelijkheid geboden heeft.

Marcel, bij jou heb ik mijn eerste stappen in de wetenschappelijke praktijk mogen maken. Dank je voor je geduld bij het steeds weer uitleggen hoe bepaalde zaken eigenlijk in elkaar staken. “wacht even, is dat echt wel zo” zit er, tot frustratie van mijn collega’s na al die jaren bij mij nog steeds ingebakken.

Muzaffer, dank je voor je onmisbare hulp bij de Doppler metingen.

Alle andere medewerkers van de afdeling Biomedische Natuurkunde en Technologie, en de afdeling Anatomie , waar ik in het verleden en heden mee heb samengewerkt.

Jan, John, en Annelies dank jullie voor het sparren over de SJC protocollen. Alle discussies over testen, oefeningen, (chronische) pijn. Jullie kennis en visie heeft bijgedragen aan de werkwijze van het Spine & Joint Centre en uiteindelijk aan mijn proefschrift.

Lex, dank je voor je efficiënte en nuchtere statistische ondersteuning gebaseerd op je ongeëvenaarde statistische kennis die je glashelder weet over te brengen.

Laraine, dank je voor je superefficiënte taalcorrecties en bemoedigende woorden.

Inge en Vincent, het wetenschappelijke SJC team. Jullie kunnen het ook, echt!

Alle SJC collega's uit heden en verleden die met elkaar steeds weer het onmogelijk geachte mogelijk maken voor mensen met chronische pijn:
An, Andrea, Annelies, Annemarie, Annemarieke, Annemiek, Anton, Barbara, Britta, Carolien, Cees, Cindy, Colinda, Crispijn, Danielle, Derek, Emmy, Esther, Evelien, Ferry, Gaby, Haitze, Helen, Hubert, Inge, Ingrid, Irene, Irma, Jacqueline, Jan, Jeanet, Jeannette, Jeroen, JohnM, JohnV, Jolanda, Jolan, Joosje, Jorien, Karin, Katarina, Leanne, Liesbeth, Maarten, Maarten, Madelon, Marika, Marja, Merit, Natasja, Nora, Noor, Oliver, Rieneke, Rob, Rosaline, Samantha, Servaas, Suzan, Suzanne, Timothy, Tineke, Twan, Victor, Vincent, Wies, Wil
Alle stagiaires die over de jaren stukken informatie hebben vergaard die mede geleid hebben tot dit proefschrift: Carola, Roland, Renee, Anton, Nesrin, Karim, Bart, Yvonne, Arnold, Bertil, Marijke, Renate, Igor, Aaike, Andries, Karline, Marieke, Danny, Ludo.
De praktijken die het eerste video onderzoek op patienten hebben mogelijk gemaakt Dick Zaanen, Liem Lim, Spanjersberg, Bergweg, Walenburgerweg, Bergpolder.
Irenea, voor het essentiële duwtje in de goede richting.
En omdat ik het beste stukje altijd voor het laatst bewaar: lieve Wilma, dank je dat je me laat zien en ervaren dat het wel kan.

List of publications

van Wingerden JP, Vleeming A, Snijders CJ, Stoeckart R. A functional-anatomical approach to the spine-pelvis mechanism: interaction between the biceps femoris muscle and the sacrotuberous ligament. Eur Spine J 1993 ; 2:140-144.

van Wingerden JP, Vleeming A, Buyruk HM, Raissadat K. Stabilization of the sacroiliac joint in vivo: verification of muscular contribution to force closure of the pelvis. Eur Spine J 2004;13:199-205.

van Wingerden JP, Vleeming A. Ronchetti I. Differences in standing and forward bending in women with chronic low back or pelvic girdle pain; indications for physical compensation strategies. Spine 2008;33:E334-E341.

van Wingerden JP, Stoeckart R, Ronchetti I, Burdorf A, Kleinrensink G-J. Kinesiophobia in woman with chronic pelvic pain; fear of motion in physical perspective.
Submitted to Spine

van Wingerden JP, Ronchetti I, Vleeming A, Stoeckart R, Burdorf A, Kleinrensink G-J. Balancing the biopsychosocial model for multidisciplinary treatment of non-specific chronic low back pain: merging motor control education and behavioural principles. Submitted to Spine

Vleeming A, van Wingerden JP, Snijders CJ, Stoeckart R, and Stijnen T. Load application to the sacrotuberous ligament; influences on SI-joint mechanics. Clin Biomech 1989;4:204-9.

de Wall M, van Riel MP, Snijders CJ, van Wingerden JP. The effect on sitting posture of a desk with a 10 degree inclination for reading and writing. Ergonomics 1991;34:575-84.

Vleeming A, van Wingerden JP, Dijkstra PF, Stoeckart R, Snijders CJ and Stijnen T. Mobility in the sacroiliac joints in the elderly: A kinematic and radiological study. Clin Biomech 1992;7:170-6.

Derksen JC, Van Riel MP, van Wingerden JP, Snijders CJ. A comparison of working postures of parcel sorters using three different working methods. Ergonomics 1994;37:299-309.

Vleeming A, Pool-Goudzwaard AL, Stoeckart R, van Wingerden JP, Snijders CJ. The posterior layer of the thoracolumbar fascia. Its function in load transfer from spine to legs. Spine. 1995 1;20:753-8.

Burdorf A, van Riel M, van Wingerden JP, van Wingerden S, Snijders C. Isodynamic evaluation of trunk muscles and low-back pain among workers in a steel factory. Ergonomics 1995;38:2107-17.

Kleinrensink G-J, Stoeckart R, Vleeming A, Snijders CJ, Mulder PG, van Wingerden JP. Peripheral nerve tension due to joint motion. A comparison between embalmed and unembalmed human bodies. Clin Biomech 1995 ;10:235-239.

Vleeming A, de Vries HJ, Mens JM, van Wingerden JP. Possible role of the long dorsal sacroiliac ligament in women with peripartum pelvic pain. Acta Obstet Gynecol Scand. 2002;81:430-6.

Ronchetti I, Vleeming A, van Wingerden JP. Physical Characteristics of Women With Severe Pelvic Girdle Pain After Pregnancy, A Descriptive Cohort Study. Spine 2008;33:145–51.

Curriculum Vitae

The author of this thesis was born in 1962 in Rotterdam as the son of Jan van Wingerden and Thea van Wingerden-Willebrand. After receiving his VWO diploma (Atheneum) in 1982, he joined the Royal Netherlands Naval College (*Koninklijk Instituut voor de Marine*, KIM) in Den Helder as marine cadet (*'adelborst'*) until October 1983.

In 1988 he graduated at the Academy of Physical Therapy Rotterdam as physical therapist with a *cum laude* paper on the application of artificial intelligence for clinical assessment protocols.

From 1988 untill 1996 he worked at the departments of Biomedical Technology (Prof.dr.ir. C.J. Snijders) and Anatomy (Prof.dr. J. Voogd) of the Erasmus University Rotterdam. During this period as a research assistant he became acquainted with anatomical dissection, biomechanics, measurement techniques, study design, data analysis, statistics and programming.

Teaching is one of the author's passions. At both graduate and postgraduate level he has taught courses on computer science, biomechanics, ergonomics, dissection-room anatomy, anatomy *in vivo* and functional anatomy.

In 1996 he joined the newly-founded Dutch rehabilitation centre - the Spine & Joint Centre – first as head of therapy and since 2001 as managing director.

The author is the delighted father of Thomas (14) and Roos (10).

Glossary

ACLR	A-specifieke lage rugklachten
ASLR	Active Straight Leg Raise
ASLR-A	Active Straight Leg Raise Scored by physician
ASLR-P	Active Straight Leg Raise Scored by patient
BPS	BioPsychoSocial
CDI	Color Doppler Imaging
CLBP	Chronic Low Back Pain
EMG	ElectroMyoGraphy
HNP	Hernia Nuclei Pulposi
LBP	Low Back Pain
LDL	Long Dorsal Ligament
NCLBP	Non specific Chronic Low Back Pain
MCIC	Minimal Clinical Important Change
MVC	Maximal Voluntary Contraction
PPPP	Posterior Pelvic Pain Provocation

PGIC	Patients Global Impression of Change
PGP	Pelvic Girdle Pain
QBDS	Quebec Back Pain Disability Scale
ROM	Range Of Motion
SI	SacroIliac
SIJ	SacroIliac Joint
THD	ThresHold Difference
TSK-DV	Tampa Scale of Kinesiophobia – Dutch language Version
VAS	Visual Analogue Scale