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Abstract

A Multidimensional Manual Therapy Model for managing patients with CNSLBP

Low back pain (LBP) is regarded as a major health and economic problem in western industrialised countries even at this time in the twenty-first century. Researchers estimate that it has increased to affect about 45% of the population in 2011. This increase creates a major burden on the health care services, social structures and the economy in terms of absenteeism from work. CNSLBP is still poorly understood. Main reasons for the poor understanding of CNSLBP discussed in this study are the limited understanding of the effect of the spine as kinetic chain which includes the head and pelvic girdle and with its attachments to the scapulae . The process of development of ISMS dysfunction are discussed as a combination of abnormal spinal loading, soft and neural tissue plasticity that result in biomechanical malalignment, adaptive and maladaptive movement patterns, pain processing integrated with psychosocial factors that influence the biomechanical, pain processing and psychological responses are discussed as possible mechanisms in the development of CNSLBP.

The researcher developed a multidimensional manual therapy model to manage patients with CNSLBP based on metacognitive reflection on her clinical reasoning over a period of 40 years as the research methodology. The metacognitive reflection has been performed within the interpretive paradigm
The model that resulted from the metacognitive reflection is dialectic in nature because it entails the understanding of the patient's problem from an interpretive as well as from an empirico-analytical perspective.

The model is conceptualised in three stages: Firstly the conceptualisation of the integrated spinal movement system (ISMS), to indicate that the spine, head,

shoulder and pelvic girdles function as a closed kinematic chain. Secondly the process of the development of ISMS dysfunction as a major concept in the clinical picture of patients with CNSLBP is based on functional anatomy of the ISMS and the researcher's clinical observation in clinical practice. The researcher indicates how the development of ISMS dysfunction and characteristic adaptive behaviour are integrated components of the patient's complex heterogenic clinical picture. The underlying process for the development of ISMS dysfunction as a possible mechanism for CNSLBP is described as plasticity of soft and neural tissues (including the brain) which result in chronicity over time.

Thirdly a multidimensional manual therapy model to manage patients with CNSLBP's heterogenic condition is discussed. The model indicates how the mechanisms underlying the development of ISMS dysfunction is addressed in a multidimensional approach to patient management. Finally the multidimensional manual therapy model is discussed in relation to other relevant intervention approaches. The model finally serves as a point of departure for planning and conducting appropriate research in basic and clinical sciences.

The multidimensional manual therapy model for the management of patients with CNSLBP has been developed in clinical practice and is presented as a practice-theory in the form of a model.

Key words: chronic non-specific low back pain, chronicity, plasticity, pain processing, multidimensional manual therapy, clinical reasoning, holistic approach, spinal dysfunction, hermeneutic research approach, practice-theory model.

CHAPTER 1

Background and rationale

1.1 Introduction

Low back pain (LBP) has affected human beings throughout recorded history (Allan & Waddell, 1989). The biggest changes in the understanding of the pathophysiology, diagnostics, and surgery that formed the basic management of patients with LBP have taken place during the 20th Century.

From the 1900s, important milestones (Bucy, 1988) first showed that the pressure of disc herniation could produce neurological symptoms.

In 1934 Mixter and Barr (White & Anderson, 1991) performed surgery on a patient with low back pain and gave the first complete clinical, pathologic and surgical description of disc prolapse as the cause of sciatica. Their paper in 1934 was regarded by surgeons as a classic contribution to surgery and showed that surgery for disc prolapse was possible.

In 1965 Melzack and Wall proposed the gate-control theory of pain (Melzack & Wall, 1996). The gate control theory proposes that pain is a multidimensional experience produced by characteristic 'neuro-signature' patterns of neuro-impulses generated by a widely distributed neural network – the body-self neuromatrix in the brain (Melzack & Wall, 1996).

Around 1970, the patho-physiology of lumbar degeneration, work done by Yong-Hing and Kirkaldy-Willis (1984) resulted in a major breakthrough towards the understanding of degenerative back disease and its effects on the components of the vertebrae and soft tissues, resulting in changes within the biomechanics of the patient's alignment and movements. This knowledge at that time revealed two 'new' clinical entities, namely nerve root entrapment and spinal stenosis, which gave rise to the conceptualisation of the 'structural–anatomical–biomechanical' (SAB) or

'disease model' (Alan & Waddell, 1989) for assessment and treatment of LBP. The SAB model of management of LBP withstood the test of time, and it is still practiced (Zusman, 2007).

The SAB era was followed by growing evidence that biological pain perception is exacerbated by psychological as well as by social factors and may contribute to the chronic pain and dysfunction in these patients Main & Watson (1999). Researchers also found that pain may originate and be maintained by altered pathways in the brain (Apkarian, Baliki & Geha 2009; Tracey and Bushnell, 2009; Kuner 2010).

Waddell (2004) concludes that LBP is a 20th Century medical disaster, and that physiotherapists and medical practitioners together have failed in their attempts to improve the manifestations of LBP. If properly managed by these professions chronic back pain and disability should be reducing, but instead the opposite is true. He also concludes that clinicians have lost sight of the basic principles to understand pain and disability in their approach to management of CNSLBP.

The influence of the psychological and social factors on biological pain perception contributed to the fact that the 'biopsychosocial' (BPS) model was conceptualised for more accurate multidimensional management of acute as well as chronic LBP (McCarthy, Arnall, Strimpakos, Freemont, & Oldham, 2004).

Waddell's classification of LBP into specific and non-specific low back pain brought much clarity on the understanding of the problem (Figure 1.1).

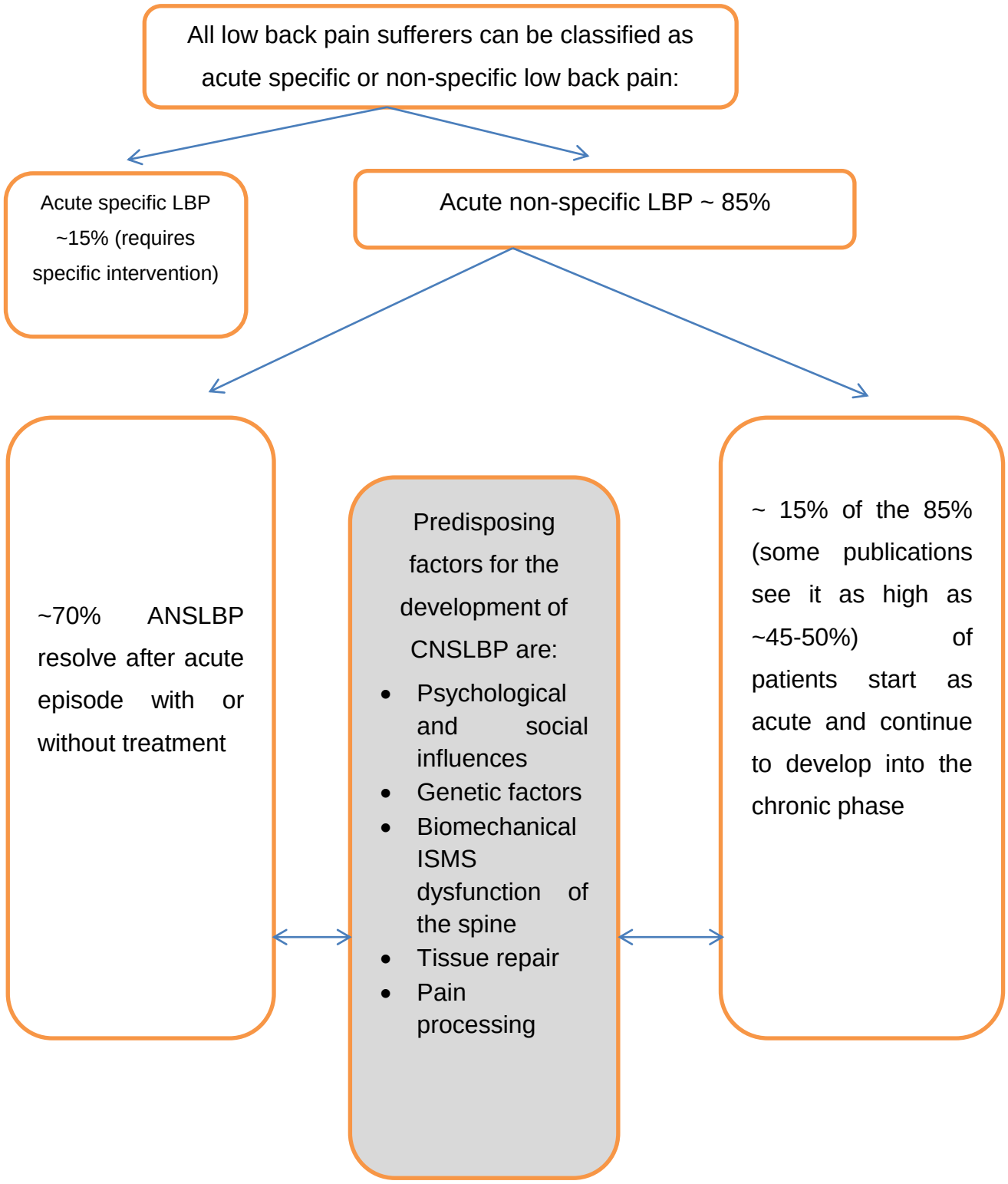


Figure 1.1: Differentiation between specific and non-specific low back pain

(Waddell, 2004; Adams, Bogduk, Burton, and Dolan 2002; Porterfield and DeRosa, 1990)

Henchoz and So (2008); Chanda, Alvin, Schnitzer and Apkarian (2011) estimate that among the adult population between 60% and 85% of the general population suffers from LBP at least once in their life time. Back pain affects at least 20% of people at any time in their lives and about half the global population has had at least one episode of LBP by the age of 30 (Docking, Fleming, Brayne, Zhao, Macfarlane & Jones, 2011). Between 44% and 78% of people suffer relapses of LBP after an initial episode of LBP. According to the European Guidelines for the Management of Chronic Nonspecific Low Back Pain (CNSLBP) (2006) there is little scientific evidence regarding the prevalence of CNSLBP: best estimates suggest that the prevalence of people who are disabled by chronic low back pain is approximately 11% to 12% of the population.

More specifically, the problem of non-specific low back pain (NSLBP) is regarded as the leading cause of disability among the population in developed countries (Bunzli, Gilham & Esterman, 2011) and the risk of disabling back pain rises with ageing (Docking *et al*., 2011). Chronic non-specific (or 'idiopathic') back pain accounts for the majority of patients treated in primary care because they make use of all the health care resources available (Chanda *et al*., 2011). Any primary care intervention has resulted in a disappointing outcome in terms of decreasing the burden of suffering in patients with LBP (Pransky, Borkan, Young & Cherkin, 2011).

Pransky *et al.* (2011) report that despite the enormous increase in the number, quality and variety of the research studies on LBP, since 1990, the progress in terms of the impact it had on primary care resulted in a disappointed outcome. The International Forum on Primary Care Research on Low Back Pain which is the premier global conference on LBP concluded that few of the treatment approaches for LBP could withstand the test of a randomised controlled trial. Pransky *et al.* (2011) are of the opinion that the evidence-based guidelines and systematic reviews that were carried out had little impact on primary care clinical practice for patients with LBP.

In 2004 Bogduk published a clinical update on the management of chronic low back pain in which he stated that 70% of patients with acute low back pain could expect to

become pain free with a recurrence rate of less that 25%. The management of patients with CNSLBP is, however, a different situation because patients with this condition have a complex clinical picture including physical disabilities and psychological distress with a duration of more than three months. The management approaches for these patients fell into three categories: monotherapy (analgesics, non-steroidal anti-inflammatories, muscle relaxants anti-depressants, physiotherapy, manipulative therapy and surgery) multidisciplinary therapy (intensive exercises) and reductionism (pursuit of a patho-anatomical diagnosis in order to target specific treatment). However treatment approaches in all three broad categories have shown limited efficacy in the management of patients with CLBP (Croft, Papageorgiou & McNally, 1997).

The impact of CNSLBP on the economy is described in terms of lost work-days due to workers' absence from work and the increased number of medical visits as a result of LBP which increases the demand on health care. It is estimated that after an initial episode of LBP between 26% and 37% of people have relapses of absence from work (Pransky *et al.*, 2011; European Guidelines for the Management of Chronic Nonspecific Low Back Pain, 2006).

According to Fourney Andersson, Arnold, Dettori, Cahana, Fehlings, Norvell, Samartzis and Chapman (2011) the fact that CNSLBP affects the patients cognitive and emotional status as a result of the involvement of the neuromatrix specifically the frontal and parietal brain lobes makes CNSLBP a deleterious condition in which around 5% of patients account for 75% of the health care cost and absenteeism from work.

Based on a review of the literature by Fourney *et al*. (2011), the authors concluded that (CNS)LBP is a multidimensional problem which affects people of all age groups occupations, races and cultures. The authors confirm that CNSLBP is a **condition** that is a problem to diagnose and manage despite the advances of modern medicine. Fourney *et al.* (2011) also state that CNSLBP is a symptom but that the ramifications of these symptoms manifests as a **disease**. The authors (Fourney *et al*., 2011) confirm Waddell's (2004) opinion that for most people with (acute

nonspecific) LBP it may be a time-limited and harmless episode while for an unpredictable 15% it can become a chronic life-changing phenomenon.

From the literature it is clear that a diagnosis of CNSLBP is based on a number of characteristics namely, the fact that a diagnostic radiological investigation does not show any specific origin of the patients' signs and symptoms. The duration of the patients' symptoms is at least 12 weeks or longer. Patients present with physical signs characteristic of impaired postural control (decreased range of motion, trunk muscle strength, muscle imbalance and endurance, impaired tactile awareness and spatial orientation) psychological behaviour such as fear avoidance, catastrophisation, hypervigilance, depression. The symptoms are strongly influenced by social stressors (Waddell, 2004; Dankaerts, O'Sullivan, Burnet & Straker, 2006; O'Sullivan, Twomey, Allison, Sinclair, Miller & Knox, 1997).

1.2 The mechanisms in the development of CNSLBP

In Figure 1.1 Waddell's (2004) opinion on the transition from ANSLBP to CNSLBP is illustrated. Acute non-specific LBP is defined as an acute episode of LBP with no structural tissue damage that can be detected by radiological investigations which can explain the patient's widespread combination of signs and symptoms (European Clinical Practice Guidelines, for the Management of Chronic Non-Specific Low Back Pain, 2005). Within this definition of CNSLBP there is thus no detectable patho-anatomical cause underlying the condition (Fersum, 2010; O'Sullivan, 2005).

Reasons why around 15% of the 85% of patients with acute non-specific LBP (ANSLBP) develop sub-acute LBP and the fact that CNSLBP is only diagnosed after 12 weeks suggest that CNSLBP develops over time (Spitzer, Leblanc, Dupuis *et al*., 1987) (refer to Figure 1.1). When and how it becomes chronic during this time is still a researcher's and clinician's challenge (Fourney *et al*., 2011).

Social and psychosocial factors impact on patients' pain modulation by hypersensitising the central nervous system due to hypervigilance and catastrophising in vulnerable patients (Zusman, 2002; Waddell, 2004). Negative

thinking patterns, fear of pain and maladaptive coping strategies in addition to stressful relationships in all or some spheres of life (family, friends or work-related relationships), work-structure, support structures, cultural factors, medical advice, compensation and socio-economic factors (Waddell, 2004) can in various combinations become drivers of the patient's pain perception (O'Sullivan, 2005)

CNSLBP is a condition that develops over approximately 12 weeks after an acute incident of non-specific LBP (ANSLBP) (Waddell, 2004). Factors that can contribute towards the development of chronicity of the LBP are numerous and most probably according to the research heterogenetic.

The nature of the mechanism that is behind the development of CNSLBP is still unclear but a number of factors which are thought to contribute towards the condition are biological (Fourney *et al*., 2011) or physical factors, mechanical forces and physiological processes in the neuromusculoskeletal systems. Fersum *et al*. (2012) describe it in more detail by listing pain provocative postures and movement patterns related to altered body schema, muscle guarding, pain behaviours and physical deconditioning as the physical factors.

Reasons why it takes time to develop are unclear at present. Field (2009); is of the opinion that some people may have a genetic predisposition towards the development of CNSLBP while in others there is a 'process of development' towards the chronicity of the pain.

From a biomechanical perspective, Panjabi (2003) and O'Sullivan (2005) describe abnormal spinal loading as an originating factor to CNSLBP. Axial loading of the spine involves impairment to the neutral zone of the disc resulting in adaptive stiffening of the spine by the stabilising effect of the muscular component which over time results in articular segmental spinal stiffness (Panjabi, 2003). Acute spinal dysfunction that results in inflammation or swelling with or without accompanying disease processes such as joint degeneration can be the origin of ongoing nociceptive stimulation and as such over time, result in CNSLBP. Spinal loading as a cause or contributing factor towards the development of CNSLBP can

characteristically be the cause of the initial and/or of the recurrent episodes of (C)NSLBP (O'Sullivan, 2005) and may exacerbate the condition. Patients with CNSLBP have a typical history of recurrent episodes of ANSLBP or acute ANSLBP episodes superimposed on CNSLBP (Croft *et al*., 1997). Van Korff, Deyo, Cherkin and Barlow (1993) caution that the duration of each acute episode and its remission may not give a true clinical picture of its outcome because the distinction between acute and chronic pain may not be clear cut.

Sahrmann (2002) mentions that abnormal spinal loading can be caused by obesity, poor posture which is also associated with muscle imbalance and endurance. Abnormal motor control has also been identified by various other authors as a contributing or characteristic factor of patients' clinical picture of CNSLBP (Fersum *et al.*, 2009; O'Sullivan, 2005). However, O'Sullivan (2005) is of the opinion that the cause and effect of poor motor control are variable and unclear. Hodges and Moseley (2003) and Van Dieen, Selen and Cholewicki (2003) are of the opinion that abnormal motor control occurs secondary as a result of the pain.

Abnormal motor control is intimately associated with abnormal muscle recruitment which is observable as changes in the quality of movement or abnormal movement components during functional activities of daily life such as gait. Whether abnormal motor control (for instance during gait) results in CNSLBP or is a result of the patient's pain response is not clear (Richardson & Jull, 1995; O'Sullivan, Twomey, Allison, Sinclair, Miller & Knox, 1997; O'Sullivan, 2000; Dankaerts *et al*., 2009).

With specific reference to patients diagnosed with CNSLBP who present with abnormal motor (postural) control, O'Sullivan (2005) distinguishes between patients with CNSLBP in whom the pathophysiological process drives the pain which is characterised by pain avoidance behaviour and a second group of patients in whom psychological and/or social factors drive the pain. Patients in whom the pathophysiological process drives the pain adapts to the nociceptive stimuli and their movement is characteristic of adaption to the painful (pain avoidance behaviour). Patients in whom pain is driven by psychological and/or social factors' movement

patterns characteristically display provocative movement strategies which are typical of maladaptive coping strategies which become ongoing sources of pain.

Neurological pain processing is closely associated with these factors. Continuous stimulation of the nociceptors results in peripheral and central sensitisation, and adaptive pathways in the brain. Chronic pain is further associated with cortical thinning or degeneration in the frontal and parietal grey matter in the brain. The changes in the peripheral, autonomic and central nervous system as a result of continuous nociceptor stimulation is the link between pain processing and psychological factors (e.g. fear avoidance and guarded movements) (Kuner 2010).

However, depression is often present in patients diagnosed with CNSLBP (Kuner, 2010), which can be associated with pain processing. Pain processing per sé has been identified as one of the driving factors of CNSLBP, specifically the cortical thinning and altered pathways in the brain. These changes in the brain also have an effect on cognitive and emotional functioning (Fourney *et al*., 2011)

The cognitive factors that are identified as playing a role in the development of CNSLBP are negative beliefs, fear-avoidance behaviours, catastrophising, hypervigilance, anxiety, depression, stress, poor pacing and maladaptive coping. Stress, catastrophisation, anxiety, depression are also regarded as psychological factors that influence CNSLBP (Fourney *et al*., 2011).

Social factors that influence CNSLBP are lifestyle and interpersonal interaction (Fourney *et al*., 2011). Lifestyle factors associated with the condition include sedentary behaviour, inactivity and sleep deficits (Fersum *et al*., 2012).

Socio-demographic factors that are associated with CNSLBP are gender, age (>50-55), marital/family status (single parent/young children, partner retired or disabled), health condition (mental health conditions musculoskeletal conditions, comorbidities), occupational/educational level, time since last worked, and occupational status (no longer employed or unemployed) (Waddell, 2004).

Fourney *et al.* (2011) describe CNSLBP as a heterogeneous condition characterised by multidimensional interaction between various factors which interact with each other. The condition can be driven by one or various combinations of factors and is therefore considered as a complex multifaceted problem of which the underlying mechanism is still unclear. Fourney *et al.* (2011) call it a heterogeneous problem which poses a challenge for an evidence-based approach.

1.3 Management of patients with CNSLBP

In a systematic review Middelkoop, Rubinstein, Kuijpers, Verhagen, Ostelo, Koes *et al*. (2010) assessed the effectiveness of single treatment modalities in the management of CNSLBP. The result of the systematic review was that exercise therapy compared with no exercise; back school/education; behavioural therapy in the short- and long term; manual therapy/manipulation in the short- and long term and different exercise interventions with each other showed low-quality evidence or no statistically significant difference of the effect of the modalities on pain and disability. A 12-week viniyoga programme compared to a 12-week conventional exercise class showed that the viniyoga programme improved participants' back-related function superiorly compared to the conventional exercise programme; motor control proved to have slightly significantly better outcomes when compared to a general exercise group after 12 weeks.

A statistically significant difference in the decrease of disability was found in favour of exercise therapy when the effect of exercise and psychotherapy in favour of exercise therapy. No difference was found between the groups in post-treatment pain intensity and also not after six months.

When 'back school' was compared to education/information, a significant difference in the outcome of disability has been shown but there was no statistical improvement in reduction of the patient's pain perception.

A statistically significant decrease in the number of days 'sick leave' that participants took during the four months after they received multidisciplinary treatment was found in the exercise group compared to a control group who received no treatment.

In a systematic review on the effectiveness of behavioural treatment of patients with CNSLBP, the authors (Ostelo, van Tulder, Vlaeyen, Linton, Morley, & Assendelft, 2000) showed that behavioural treatment has a small positive effect on behavioural outcomes and a moderate positive effect on pain intensity.

From the systematic review by Middelkoop *et al*. (2010) it is clear that exercise is a popular form of treatment for patients with CNSLBP, although there is no evidence that one form of exercise is more effective than another (Liddle, Baxter & Gracey, 2004).

Fourney et al. (2011) confirm the disappointing results of single management approaches for patients with CNSLBP and are of the opinion that the problem is still treated from a homogenous (fragmented) perspective rather than from an integrated heterogeneous perspective. Based on their research Fourney *et al.* (2011) clinically categorise patients with CNSLBP into five treatment spheres: those who need procedural-based specialities, those who need strength-based rehabilitation, cognitive behavioural therapy, pain management and manipulative care. These authors (Fourney *et al.*, 2011) argue that C(NS)LBP is a heterogeneous condition which requires a **multidisciplinary** intervention approach. The multidisciplinary team these authors suggest include a spinal surgeon, anaesthesiologist, psychiatrists, radiologist, physical therapist, rehabilitation psychologist, pain medicine practitioners, chiropractors and osteopaths.
Fourney *et al*. (2011) also strongly suggest that converging the five categories of intervention into a conjoined approach to the management of patients with CNSLBP would be a major step towards research, knowledge and to address the various facets with which patient's presents.

Wand and O'Connell (2008) concluded that the disappointing results from the clinical trials on the intervention for patients with CNSLBP could be ascribed to the fact that

patients might not have been appropriately selected for the research in the sense that unsuitable (single) treatment protocols might have been selected to address the heterogeneity of the patients problem; or clinical trials failed to capture the true effectiveness of current practice. These authors also suggest that the current approaches to the management of CNSLBP should be revisited (Wand & O'Connell, 2008).

Fersum *et al*. (2012) also concluded that the possible reasons for the lack of effective management of patients with CNSLBP could probably be found in the fact that single treatment approaches do not address the complex heterogenetic nature of CNSLBP. The heterogeneity of the conditions lies in the fact that in patients with CNSLBP cognitive, physical and lifestyle factors could all or in varying combinations be the provocative of driving factor(s) of the condition.

Manual therapy is commonly accepted as a treatment approach for patients with CNSLBP. However, in a systematic review on the effectiveness of manual therapy for patients with CNSLBP, the researchers concluded that manual therapy only shows a minimal clinical meaningful effect compared to other treatment options (European Guidelines for the Management of Chronic Nonspecific Low Back Pain, 2006; Assendelft, Morton, Suttorp & Shekelle, 2004; Dagenais *et al*., 2010) Rubinstein, van Middelkoop, Assendelft, de Boer, & van Tulder *et al.,* (2011) found no clinical relevant difference between spinal manipulative therapy (SMT) and other intervention to reduce pain.
From the systematic review by Middelkoop *et al*. (2010) it is clear that exercise is a popular form of treatment for patients with CNSLBP, although there is no evidence that one form of exercise is more effective than another (Liddle, Baxter & Gracey, 2004).

Ferreira, Ferreira, Latimer, Herbert, Hodges, Jennings Maher and Refshauge (2003) and Kääpä, Frantsi, Sarna, and Malmivaara (2006) conducted a randomised clinical trial to evaluate the effect of an intervention which consisted of individual physiotherapy, ultrasound and light active exercise, and advice compared to a multidisciplinary intervention program.

The individual physiotherapy consisted of (1) passive pain treatment which was a combination of massage, spine traction and manual mobilisation of the spine, (2) the exercise therapy part of the intervention consisted of muscle stretching, spine mobilisation and deep trunk exercises and (3) advice to keep active with large movement activities. The intervention was applied in a cognitive behavioural way.

The multidisciplinary rehabilitation programme (presented by a multidisciplinary team), entailed group sessions consisting of cognitive behavioural stress management methods, (rational emotive), applied relaxation session, back school education, including occupational intervention, and a physical exercise programme. The researchers found that the multidisciplinary rehabilitation programme does not offer incremental benefits when compared to individual physiotherapy. The majority of the participants (98%) were female.

The limited evidence for the effectiveness of manual therapy for 'LBP' (CNSLBP, LBP or acute LBP) may be contributed to the heterogeneity of the sample of patients who participated in the RCTs to investigate the effectiveness of manual therapy. Heterogeneous sample groups are known to reduce the likelihood of a significant treatment effect especially if the sample-size of the RCTs is small (Slater *et al.*, 2012; Kent *et al*., 2005).

To overcome this problem of heterogeneity in RCTs, sub-grouping of the heterogeneous population of CNSLBP has been suggested to compile homogenous groups of patients with CNSLBP who are likely to respond similarly to manual therapy (McCarthy *et al*., 2004; O'Sullivan, 2005).

Sample selection based on reliable sub-classification strategies within a biopsychosocial framework will result in more patient-centred targeted management and reliable and valid outcomes (research results) on patients' with similar underlying mechanisms driving the complex condition of CNSLBP (Fersum *et al*., 2012).

The authors (Fersum *et al*., 2012) also state that the reason for clinicians' failure to manage patients with CNSLBP in clinical practice is that patients are not managed within a multidimensional biopsychosocial framework. Fersum *et al*. (2012) therefore conducted a randomised controlled trial in which they compared classification based 'cognitive functional therapy' (CBFT) with 'manual therapy and exercise' (MT-EX). The patients in the classification-based cognitive functional therapy demonstrated superior outcomes on the Oswestery Disability Index (ODI), pain intensity (PINRS), Hopkins Symptom Checklist (for anxiety and depression), fear avoidance in the physical social environment and fear avoidance work environment, and the total range of spinal motion 12 weeks and 12 months post-intervention.

In essence the researchers (Fersum *et al*., 2012) used a multidimensional approach in the CB-CFT as well as the MT-EX to address the complex multidimensional aspects. The authors (Fersum *et al.* (2012), found that CB-CFT resulted in superior outcomes in reducing the patients' pain, disability, fear, beliefs, mood and sick leave at the 12 month follow-up compared to MT-EX. They (Fersum *et al*., 2012) concluded that it is unclear as to the exact basis for the superior outcomes of the multidimensional nature of CB-CFT intervention. Their hypothesis indicates that the mechanisms for change in the patient-centred body-mind behavioural approach most likely addressed the heterogeneous nature of the condition by having an impact of the cognitive factors known to have an effect on pain sensitivity and disability. These cognitive factors include the generation of positive beliefs, control of pain, reducing fear of pain, *adaptive coping enhanced self-efficacy, confidence and improved mood* (Fersum *et al*., 2012 p 10). The authors achieved these effects by enhancing body awareness relaxation of guarded muscles, normalising maladaptive movement patterns, body schema retraining and extinguishing pain behaviours.

The study by Fersum *et al*. (2012) emphasises the importance of patients to receive multidimensional intervention as was the case with the group who received CB-CFT. It appears that the MT-EX group did not receive a multidimensional intervention to the same extent as the group who received the CB-CFT.

In conclusion it is clear that single treatment procedures, although possibly relevant to patients' signs and symptoms, do not address the heterogeneous nature of CNSLBP. Because of the heterogeneous nature of CNSLBP various authors have expressed the importance of sub-classification in research studies in order to plan and implement patient-specific targeted studies on homogenous subgroups.

Identifying the origin of CNSLBP is another aspect that has been studied over the last few decades.

Panjabi (1992; 2003) and O'Sullivan (2000) described a lumbar motion segment instability model (due to enlargement of the neutral zone of the motion segment) as the origin of CNSLBP. O'Sullivan (2000) suggests an exercise intervention program on the levels of body function (local stabilisation exercises) functional activity and participation levels for the treatment of these patients. The model by Panjabi (1992, 2003) and O'Sullivan (2000) is not based on a detailed analysis of the functional anatomy of the spine and only covers the motion segments in the lumbar spine and not the whole spine. In Chapter 3 the researcher shows how CNSLBP can originate in the lumbar spine (due to abnormal spinal loading as suggested by Panjabi (1993) and O'Sullivan (2000) but also indicates that pain in the lumbar spine can be referred from other areas in the spine to the lumbar region due to ISMS dysfunction.

None of the research studies describe or discuss the importance of the thoraco-lumbar fascia and its integrated functioning with the segmental and multisegmental muscle groups to affect its functioning and nociceptive input in case of musculoskeletal dysfunction of the spine, head, shoulder and pelvic girdle positions

In summary it is therefore essential that the combination of the driving factors in patients with CNSLBP should be addressed in a multidimensional or multidisciplinary approach to intervention. The multidimensional or multidisciplinary intervention should therefore address the inseparable interaction between the neuromusculoskeletal systems and the psychosocial factors influencing the patients clinical picture within the biospychosocial framework.

1.3.1 The researcher's multidimensional manual therapy approach to the management of patients with CNSLBP

The researcher has developed a multidimensional manual therapy approach to the management of patients with CNSLBP over a period of 30 years. This period was also characterised by the development of manual therapy on a timeline as shown in Figure 1.2. As a clinician the researcher attended the relevant courses and conferences on national and international level to keep abreast of the development of manual therapy as it occurred. What is characteristic of the development of manual therapy at the time is that it occurred in parallel with the development of scientific knowledge in the basic sciences of especially Physiology, biomechanics (functional Anatomy) and Pathology, and the clinical sciences in Medicine, e.g. Orthopaedics and Radiology. Development in the basic and clinical sciences contributed to the accuracy of diagnoses and understanding of underlying disease processes which paved the way for the manual therapy researchers.

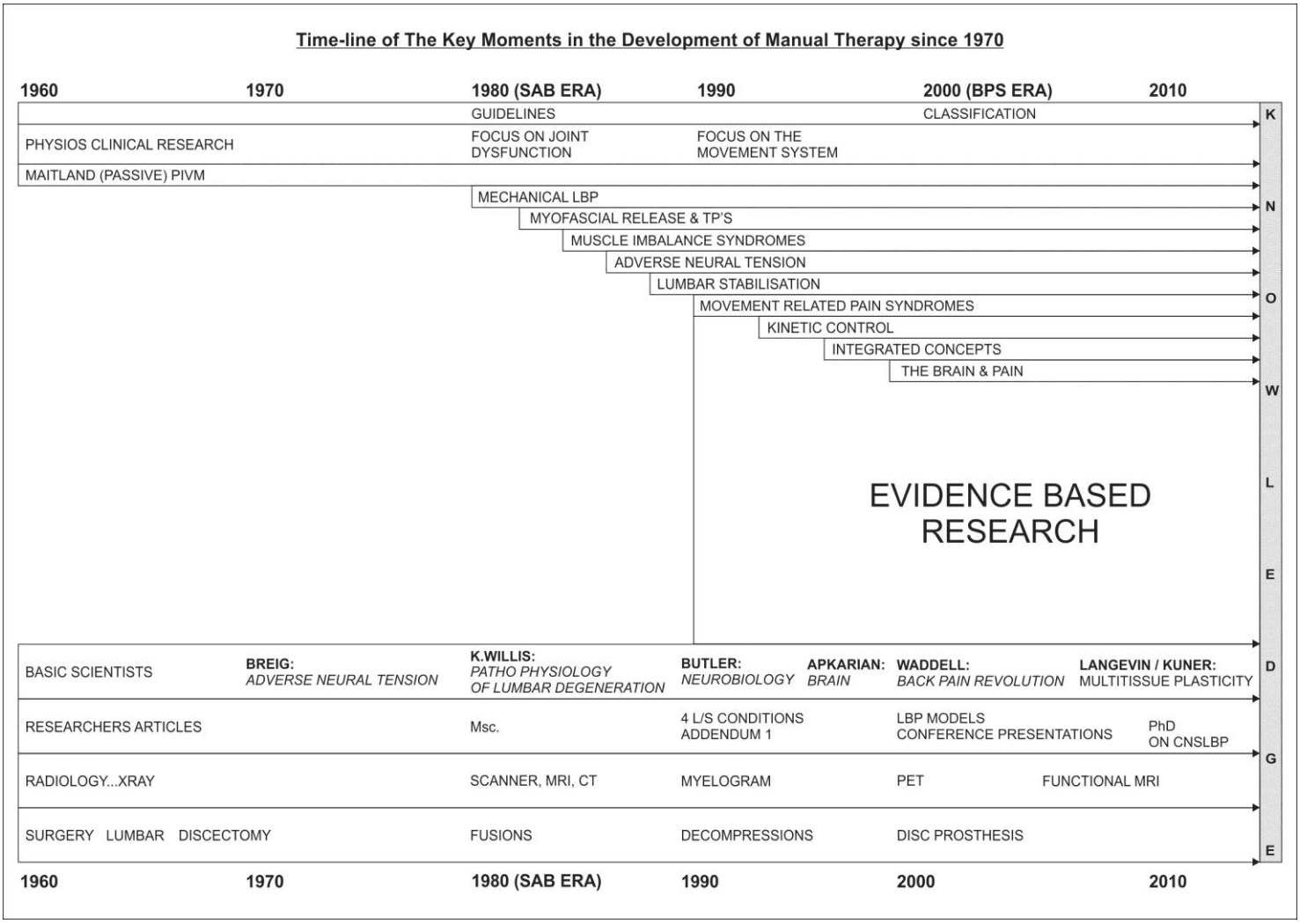


Figure 1.2: Comparison of the key moments in the development of manual therapy, diagnostic medicine, and neuro- and orthopaedic surgery experienced by the researcher since 1970

As a clinician who has had a dialectic approach to the management of patients the researcher has identified the complexity of all the contributing factors to the complex clinical picture of the patients with CNSLBP. As a manual therapist who practiced as an integral member of a multidisciplinary team in a spinal unit the researcher developed a multidimensional manual therapy approach to the management of patient with CNSLBP to address the 'multiple components' of the patient's complex clinical picture (Curriculum Vitae M C Steffen, Addendum 1).

The essence of the researcher's multidimensional manual therapy to the management of patients with CNSLBP is patient-centred (from the patient's lived experience) that is interpreted against the biomedical approach to management of

these patients. The principles of the researcher's multidimensional manual therapy had always been to manage the patients' pain through facilitation of endogenous pain-inhibiting mechanisms which involve an integrated interaction between:

- The therapist as a pain inhibiting agent in a professional therapist-patient relationship. By putting the patient at ease and re-assuring him/her about the nature and seriousness of his/her condition within a professional therapist-patient relationship the researcher explained the patient's condition and findings of her treatment to him/her as the treatment progressed.
- Manual therapy is applied to patients based on the presentation of the patient's signs and symptoms to
    - Inhibit ascending pain modulation by releasing soft tissue and joint restrictions on segmental and multisegmental levels and re-alignment of the (integrated spinal movement system (ISMS which entails the whole spine including the head, shoulder and pelvic girdles). (The researcher discovered that patients with CNSLBP need manual therapy to the ISMS because the ISMS (all spinal structures) were affected: patients with CNSLBP often experience diffuse pain simultaneously with LBP at various sites. By releasing muscle spasm, and restoring soft tissue mobility muscles are prepared for better recruitment.
    - At the time the researcher also 'discovered' that patients who received feedback on where they experienced symptoms and how it responded to her treatment, were more aware of their proprioceptive awareness and responded better to specific exercises. This concept was only described in 2012 by Moseley, Gallagher and Gallace as tactile awareness and spatial orientation but has been used in principle by the researcher over many years during manual therapy.
    - Patients are encouraged to engage in normal activities of daily life within the limits of their pain perception before they are given an exercise program.
    - The researcher found that manual therapy as she practised it addressed the patient's pain and dysfunction including that of the autonomic nervous system effectively.

- Because the researcher practised as a member of the multidisciplinary team pharmacology was introduced as an integral part of the multidimensional manual therapy management.
- Re-education of postural control and characterised adaptive behaviour had always been part of the researcher's multidimensional manual therapy management of patients to maintain mobility and alignment of the ISMS and facilitate their functional restoration.

1.4 Problem statement

From the discussion in Section 1.3 it is clear that many studies have not addressed the heterogenic nature of CNSLBP in patients with a multidisciplinary or a multidimensional approach. Results from these studies have therefore either not shown a statistically significant difference, or showed a small effect size, or only had a short term effect on the patients signs and symptoms (Middelkoop et al, 2010; Wand & O'Connell, 2008).

From the systematic review by Middelkoop *et al*. (2010) it is also evident that exercise is a popular form of treatment for patients with CNSLBP, although there is no evidence that one form of exercise is more effective than another (Liddle, Baxter & Gracey, 2004).

The heterogenic nature with which CNSLBP can present in patients entails the fact that varying combinations of biopsychosocial factors may drive the condition which may be the major contributing factor in the poor effect size observed in the randomised clinical trials. Various authors have therefore investigated and suggested ways in which patients with CNSLBP can be subgrouped (O'Sullivan, 2005; McKenzie, 2003; Herbert, 2007; Cook, Gebski & Keech, 2004; McCarthy *et al*., 2004) into more homogenic groups who have the same or similar driving factors so that interventions can be more patient-specific within the biopsychosocial nframework.

Despite this recommendation that subgrouping is a way to create homogeneous subsets within the CNSLBP population, Fersum *et al*. (2010) found that the application of a classification system to plan and implement RCTs to evaluate the

efficiency of manual therapy and exercise with other matched treatments are very limited to non-existing. The alternative is that clinicians and researchers may not understand the complexity of the underlying mechanism(s) that drive the patient with CNSLBP's clinical picture and may not be able to select appropriate classification systems; that clinicans have a perception that a classification system is not very valuable; that clinicians use other methods to assess and implement targeted patient-specific ingtervention, or that the classification systems do not integrate the multidimensional nature of CNSLBP (Karayannis, Jull & Hodges, 2012)

Fersum *et al*. (2012) used the systematic review by applying a person-centered 'mechanical behaviour' (O'Sullivan, 2005) classification system to assess the effect of 'classification based cognitive functional therapy' (CB-CFT) versus a 'manual therapy and exercise' (MT-EX) approach to treatment of patients with CNSLBP.

The manual therapy section of the MT-EX group was administered to the spine OR the pelvis to address patients' signs and symptoms of the patient's condition. The exercise section of the MT-EX group was administered to isolated muscle contraction such as abdominal muscles in different functional positions OR a home exercise programme consisting of 'general exercise' or 'abdominal muscle contraction'.

The manual therapy in this RCT does not appear to address the soft tissue shortening (muscles, fascia, ligaments and joint capsules) and realignment of the biological (biomechanical stiffness and malalignment and physiological processes involved in the development and clinical presentation of patients with CNSLBP) heterogeneity typical in patients with CNSLBP. Neither does it mention the importance of addressing the functioning of the spine as a closed kinetic chain, nor the effect that the attachments of the head, shoulder and pelvic girdles to the spine may have on pain or dysfunction in the lumbar region.

It is not clear from the publication whether the exercise section of the MT-EX group have addressed 'pain avoidance' or 'pain provocation behaviour' (O'Sullivan 2005). It is therefore not unexpected that CB-CFT was shown to be superior to the MT-EX. The manual therapy (MT-EX) applied as a treatment procedure in this RCT was left

to the discretion of experienced manual therapists and seem to differ from the way manual therapy is practised in more specific detail by the researcher (Section 1.3.1).

Kääpä et al. (2006), who compared 'individual therapy' which included passive mobilisation and spinal traction, education and exercises, with multidisciplinary rehabilitation that included education, exercises, relaxation, stress management and advice, do not discuss the expected difference between the benefits of the two management approaches. The passive mobilisation and spinal traction given to the group who received the individual therapy was not applied based on specific biomechanical or biological criteria or aims of treatment.

The authors that recommend a multidimensional or a multidisciplinary treatment approach do not explain the expected structural and physiological and psychological mechanisms that could have played a role in the explanation of the interventions that they compared (Fersum *et al*., 2010; Fourney et al, 2011; Kääpä et al. 2006).

The limited understanding of the mechanisms underlying CNSLBP which should also be the mechanisms that should be addressed in a multidicisplinary or multidimensional intervention for these patients, create in the researcher's opinion a limitation in the management of this heterogeneous condition.

None of the research studies describes or discusses the importance of the thoraco-lumbar fascia and its integrated functioning with the segmental and multisegmental muscle groups working in on the spine and its nociceptive input in the case of musculoskeletal dysfunction of the spine as a kinetic chain (Middleditch and Oliver, 2005). Although many studies recognise the importance of addressing the single or combination of driving factors in patients with CNSLBP, no study addressed the inseparable biological interaction between the neuromusculoskeletal systems and the psychological response of the patient as a result of the pain perception in the brain.

The process of plasticity that play a major role in the development of soft and neural tissue shortening and motion segment stiffness throughout the spine (integrated spinal movement system (ISMS), and the remodelling of these tissues and

realignment of the motion segments throughout the spine, head, shoulder and pelvic girdles (ISMS) (through the process of plasticity) is not addressed as part of the research or treatment interventions. The process of neural plasticity in the development as well as the 'unlearning' of the altered pathways and changes in the neuromatrix as a result of adaptive or maladaptive motor behaviour and pain processing is not discussed as the **mechanisms** behind the cognitive and psychosocial driving factors of the condition (Flor, Braun, Elbert, & Birbaumer, 1997; Kuner, 2010). By understanding these complex neurophysiological processes, clinicians and researchers can optimise intervention (education to understand the condition, advice, exercise to address adaptive and maladaptive motor behaviour) researchers will be able to explore the exceptional results of the CB-CFT achieved by Fersum et al (2012).

1.5 Research questions

The research questions of this study were:

- Can the concept of an 'integrated spinal movement system' ISMS be conceptualised based on the anatomy of the trunk?

- What are the underlying systems, processes and influences that result in ISMS dysfunction and contribute to the clinical picture of patients with CNSLBP?

- What contribution can the professional craft knowledge and the personal tacit knowledge acquired by the researcher over many years of clinical practice, make towards the declarative professional knowledge of manual therapy?

1.6 Research aims and objectives

The primary aim of this research was to develop a multidimensional manual therapy model for patients with CNSLBP based on clinical observations, clinical reasoning, professional craft knowledge and personal tacit knowledge

The process of model development requires the following sub-aims:

(1) To discuss the theoretical basis for the conceptualisation of the ISMS and ISMS dysfunction as the focus of the multidimensional manual therapy model regardless of whether the origin of the CNSLBP is more biomechanical or as a result of increased muscle tone due to hypervigilance in the brain as a result of social stressors.
(2) To discuss the underlying process involved in the development of ISMS dysfunction and the possible reason for the variations in ISMS dysfunction that can occur.
(3) To discuss the assessment of a patient with CNSLBP as part of the multidimensional manual therapy model.
(4) To discuss the principles of a multidimensional manual therapy model for managing patients with CNSLBP.
(5) To conceptualise a multidimensional manual therapy model for managing patients with CNSLBP.
(6) To discuss the multidimensional manual therapy model in the context of other relevant models for managing patients with CNSLBP.

1.7 Research approach

Model development based on a grounded theory development

Model development as a research design and as the outcome of this study was chosen because in a model the relationship between the different components and concepts related to the development and management of CNSLBP could be illustrated instead of extensively described in terms of management processes, guidelines, services to patients and the identification of new fields for further research. When the relationships between the components and concepts are illustrated they can be tested with empirico-analytical research (Higgs *et al*., 2010).

The basic function of a model is to promote, explain and define relationships, structure, and linkages between concepts to enhance understanding of a phenomenon: in other words it is '*heuristic, i.e. discovering or "exposing" certain relationships between concepts'* (Mouton & Marais, 1990 p 60).

The process for developing a model (Polit & Beck, 2008 p 85) will therefore be the same as the process for developing a theory. This process is described by Walker and Avant (1995) as:

- *Select a topic of interest (may be one concept / variable or a framework of several concepts)*
- *Conduct a review of the literature or use field observations and note related variables*
- *Organize relational statements in terms of patterns of relationships amongst the variables. Diagrams may be used to express relationships amongst concepts and to organize the components of the theory.*

The study is divided into three main sections:

- A discussion of the theoretical basis for the conceptualization the ISMS.
- Secondly, a discussion on the proposed process of the development of ISMS dysfunction which include the associated pain processing and characteristic adaptive behaviour typically observed by the researcher in patients with CNSLBP. The discussion of the proposed process of development of ISMS dysfunction is based on the functional anatomy
- Thirdly, a multidimensional manual therapy model for the management of a typical patient with CNSLBP is which is grounded in the biopsychosocial framework, is presented and discussed. The researcher further indicates how the model fills a gap in the understanding and management of patients with CNSLBP.

Table 1.1: Summary of the components of the model

Component of the model	Description of the component	Application/Contribution of component to the model
Conceptualisation of the ISMS	The conceptualisation of the ISMS is based on (1) the discussion of the functional anatomy and the processes which form the ISMS	The conceptualisation of the ISMS and the processes working within the system serve as the premise for understanding the development of ISMS dysfunction and multidimensional manual therapy of patients with CNSLBP (assessment and treatment).
Development of the ISMS dysfunction, associated pain processing and characteristic adaptive behaviour.	The development of ISMS dysfunction is discussed as the basis for the development of CNSLBP which cannot be diagnosed based on radiological investigations or other objective tests. Pain processing and characteristic adaptive behaviour, form an integrated part of the development of ISMS dysfunction.	The proposed process of development of ISMS dysfunction, pain processing and characteristic adaptive behaviour forms the basis of the understanding and manual therapy management patients with CNSLBP.
Conceptualisation of the multidimensional manual therapy management model for patients with CNSLBP	The multidimensional manual therapy model for managing patients with CNSLBP indicates the integrated multidimensional approach to address ISMS dysfunction which includes the management of pain processing and characteristic adaptive behaviour.	The multidimensional manual therapy model for management of patients with CNSLBP, falls within the biopsychosocial framework and specifically within the movement and control impairment groups described by O'Sullivan (2005)

1.8 The nature of this study

The research problem emanated from the fact that the researcher through her clinical experience observed some mechanisms involved in the development of ISMS dysfunction and became aware that manual therapy for patients with CNSLBP applied to the ISMS and not only to the low back address the patient's diffuse and specific pain patterns, general ISMS mobility which prepared the patient for muscle activation and postural re-education. Furthermore the researcher observed that manual therapy and her professional interpersonal interaction with the patient, contributed to pain modulation and enhanced re-education of postural control. Pharmacology (anti-inflammatories) post treatment was found to maintain patients' mobility and reduced post treatment effects. Based on her clinical experience, clinical reasoning, gaining knowledge in the field over many years, the researcher developed a practice theory on the multidimensional manual therapy for patients diagnosed with CNSLBP.

According to McEwen and Wills (2002), theories to explain a phenomenon from the perspective of clinical practice situations can be inductively developed to describe or explain such a phenomenon.

The insight gained from describing a phenomenon in a particular situation can in turn contribute to the understanding of similar situations in clinical practice. The authors (McEwen & Wills, 2002) call this process the practice-theory approach to theory development. The research strategy to generate or develop a practice-theory is based on the grounded theory approach (McEwen & Wills, 2002). Grounded theory is defined as: '*an approach to collecting and analyzing qualitative data that aims to develop theories grounded in real-world observations*' (Polit & Beck 2008 p 755).

This research was initiated by the researcher's reflection on her treatment of patients with CNSLBP and her clinical reasoning during:

- The treatment of patients and discussions with colleagues at national and international level on the topic of CNSLBP;

- Attending courses on spinal rehabilitation and manipulation/mobilisation nationally and internationally;
- Presenting continuing professional development courses nationally;
- Clinical training of postgraduate students in manual therapy and an international workshop on manual therapy; and
- Observing and analysing patient responses to treatment and adapting treatment to the patients' physical responses during and after treatment, and patient feedback during and after treatment.

The research was further initiated by a critical analysis of the literature on the management of patients diagnosed with CNSLBP, and the fact that present RCT which compared the effect of manual therapy with other physiotherapy modalities in the management of the heterogeneous condition of CNSLBP.

In essence, the researcher presents a theory on the development of CNSLBP and the management of patients in the form of a multidimensional model for managing patients with CNSLBP and, in this way, contributes to the knowledge basis of physiotherapy and in particular manual therapy.

Higgs and Titchen (1995) and Higgs, Jones and Titchen (2010) distinguish between three types or domains of knowledge: (1) discursive research and declarative knowledge (also called propositional knowledge); (2) personal knowledge; and (3) professional craft knowledge ('knowing how' or non-propositional knowledge/practical and procedural knowledge). The three types of knowledge and the interaction between them are illustrated in Figure 1.2.

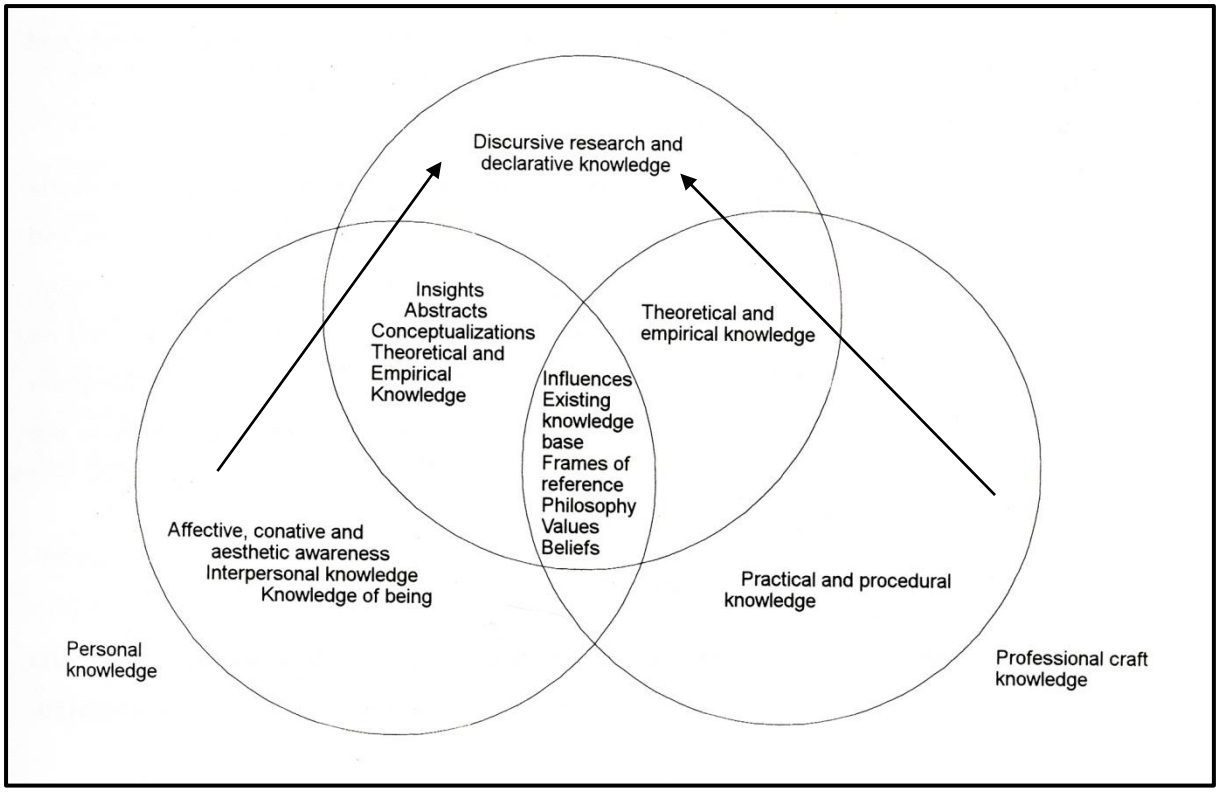


Figure 1.3: Types of knowledge and internal influences on knowledge generation

(Adapted from Higgs & Titchen, 1995 p 137)

The arrows indicate the domains of knowledge from which the practice theory was developed.

Propositional knowledge, which is formally generated through research and scholarship, is regarded in modern society as having a higher status than non-propositional knowledge, which is generated through practical experience. However, already in 1949 Ryle (Higgs *et al.,* 2010 p 154) argued that propositional knowledge **follows** rather than **drives** procedural knowledge (non-propositional knowledge) while Barnett (1990) is of the opinion that the cognitive framework dominates other forms of knowledge and regards non-propositional knowledge as downgraded forms of knowledge. This opinion is slowly being challenged and researchers are increasingly realising that knowledge generated in clinical practice is rather driving the generation of propositional knowledge than following it. Ryle argued that an aspect of theory that is developed in clinical practice is inherently part of clinical

practice but is different from applying propositional knowledge in practice (Higgs *et al.,* 2010 p 154).

Higgs *et al*. (2010 p 154) furthermore state that knowledge derived from clinical experience can be transformed to propositional or declarative knowledge '*through a process of theorization and/or rigorous critique and debate among practice communities*'.

Once the non-propositional tacit knowledge is clarified or identified, described and tested through empirical research, it becomes part of the declarative (propositional) knowledge (Higgs & Titchen, 1995).

The opposite can also happen, that propositional knowledge can be derived from basic or applied research and be elaborated and particularised through clinical practice and as such become part of the personal experience of the individual (clinician).

The characteristics of the three types of knowledge are as follows. Propositional knowledge is derived through research and/or scholarship. It is formal and explicit knowledge that is expressed in propositional statements, which enunciate, for example, relationships between concepts or causes and effects and which identify the generalisability or transferability of research knowledge to populations and settings. Theoretical knowledge may be developed from arguments of principle, from dialogue and logic, and through use or application of existing empirical and theoretical knowledge (Higgs & Titchen, 1995).

Professional craft knowledge and personal knowledge are collectively called non-propositional knowledge. Both these types of knowledge are derived from the processing (e.g. through reflection) of professional and personal experiences, respectively, and may be tacit and embedded in practice or in the personal identity and lives of the patients, and researcher (the 'knowers'). Cervero (1992) describes professional craft knowledge as a 'repertoire' of examples, images, practical principles, scenarios or rules of thumb that have been developed through prior

experience. Professional craft knowledge comprises general knowledge gained from practical experience (e.g. knowledge about how a population of patients responds to disease or disability) and specific knowledge (e.g. about a particular patient), in a particular situation and context at a particular time (Higgs, Fish & Rothwell, 2010).

Personal knowledge is accrued from life experiences, such as relationships and cultural influences that contribute to shaping individual perspectives; as such, it influences personal interactions, personal values and beliefs. This knowledge, in its general form, is gained, as in the case of professional craft knowledge, through socialisation into a society, group or professional community. In its particular form personal knowledge is acquired more consciously by reflecting upon one's knowing, being, doing and feeling in each unique situation.

Practitioners use all three forms of knowledge in practice. Propositional knowledge can provide the basis for understanding the medical, psycho-social and cultural context and the physical and psycho-social nature of the client's needs and problems. In relation to clinical reasoning, practitioners need to accumulate and update carefully a rich and dependable knowledge base, and have to be vigilant in checking for potential errors in the currency and use of this knowledge, in particular when making important decisions of diagnosis, treatment and prognosis.

Professional craft knowledge enables practitioners to tailor management procedures to individual patients' needs based on clinical decision making and recognition of the individual client's needs (Higgs, Fish & Rothwell, 2010). Such knowledge enables clinicians to plan, modify and critique their treatments to consolidate their understanding of the particular clinical problem (Jensen *et al*., 1992) and to implement sound, efficient and timely decision making.

Research has demonstrated that it is the ability of experienced professionals to integrate propositional knowledge with professional craft knowledge that enables them to assess the relevance of clinical data and to distinguish and comprehend the significance of crucial cues (Dreyfus & Dreyfus, 1980; Elstein, Shulman & Sprafka, 1990).

Health professionals draw on their professional craft knowledge and their personal knowledge to interact effectively with patients and their carers. Such knowledge, combined with skills in communication, listening and problem solving, facilitates interpersonal interactions and enables practitioners to relate well to their clients as individuals with their unique needs, fears, hopes and expectations. Carper (1978, argued that personal knowledge promotes wholeness and integrity in the personal encounter, the achievement of engagement rather than detachment. The ability to place the clinical problem within the patient's world and to design personalised care and interventions that take the patient's experience into account is recognised across the health sciences as a key element of expertise that develops from clinical practice experience (Crepeau, 1991; Jensen, Shepard & Hack, 1992; Jones *et al*., 2002).

The interaction between theory, research and practice is a reciprocal, cyclical interaction. McEwen and Wills (2002) illustrate the interaction between research, practice and theory in a cycle presented in Figure 1.3.

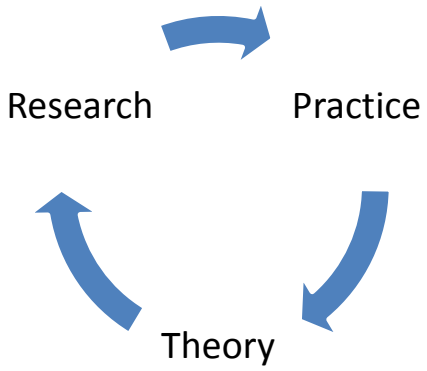


Figure 1.4: The research, theory, practice cycle
(McEwen & Wills, 2002 p 80)

A theory developed from clinical practice through a process of inductive reasoning is called a practice-theory (refer to Section 1.7). A practice-theory assumes that the phenomenon observed and experienced in clinical practice is important enough to

pursue, and that it presents an understanding of the clinical phenomenon that has not been articulated (McEwen & Wills, 2002).

McEwen and Wills (2002 p 80) further explain the relationship between a theory and a model as:

> *A theory has several components, including purpose, concepts and definitions, theoretical statements, structure, and assumptions (Bishop, 1998; Chinn & Kramer, 1999). Creation of conceptual models or maps is also a component of theory development that is promoted to further explain and define relationships, structure, and linkages.*

Mouton and Marais (1990) are of the opinion that the relationship but also the distinguishing factor between a theory and a model is that a theory classifies knowledge (or explains a phenomenon in clinical practice in this case) and explains or enhances understanding of a phenomenon, while a model not only classifies knowledge *'but the basic function* [of the model] *is heuristic, i.e. discovering or "exposing" certain relationships between concepts'* (Mouton & Marais, 1990 p 60). A model helps to illustrate the processes through which outcomes occur by specifying the relationships among the variables in graphic form. In this graphic form, a model can be examined for inconsistency, incompleteness or errors (McEwen & Wills, 2002).

1.9 Clarification of terminology

The terminology that is discussed in this paragraph includes low back pain, acute specific low back pain, acute non-specific low back pain; chronic nonspecific low back pain, manual therapy, plasticity, chronicity, dysfunction and disability, integrated spinal movement system (ISMS), integrated spinal movement system dysfunction (ISMS dysfunction), characteristic adaptive behaviour and pain modulation.

1.9.1 Low back pain

Low back pain is defined in terms of the location of pain. This implies 'pain in the low back'. The low back is identified by Waddell (2004) as pain in the area between the

lowest ribs and the inferior gluteal folds and is often accompanied by referred pain. Referred pain can be somatic or neurological in origin. Pain which is neurological in origin can be categorised as specific LBP. The referred pain which is somatic in origin is more associated with non-specific low back pain (Bogduk & Twomey, 2005). All low back pain starts as acute and can be divided into specific LBP or non-specific low back pain (refer to Figure 1.1).

1.9.2 Acute specific low back pain

Specific low back pain is defined as low back pain from a specific origin such as anatomical tissue damage in the lumbar spine (i.e. an annulus tear, disc prolapse, subluxation of the facet joint) that can be detected by CT and MRI scans. The patients' pain perception is characterised by a direct proportion to the physical findings (acute tissue damage) and the, nociception, pain, suffering, and pain behaviour. Acute specific LBP responds well to physical treatment and/or surgery and patients show a natural tendency to recover as response to treatment (European Clinical Guidelines for the Management of Chronic Non-Specific Low Back Pain, 2005).

1.9.3 Manual therapy

Manual therapy by definition in this thesis is the release of soft tissues (muscle spasm, taut bands, trigger points and joint capsules, myofascia and neural tissue restrictions – all of which may restrict the skeletal system and contribute to malalignment of the skeletal system) through the direct contact between the patient and the manual therapist's hands. It further entails the realignment of the skeletal system by passive oscillatory physiological and accessory movements of joints that do not exceed the normal end-range of joints (Elvey & O'Sullivan, 2004).

Spinal manipulative therapy (SMT) on the contrary entails a sudden available end of range thrust to increase joint range when the window of opportunity presents. A manual therapist would use SMT in combination with manual therapy to release restrictions (scar tissue) in order to integrate other forms of manual therapy to restore alignment, muscle recruitment and function (Haynes, 2003).

Treatment techniques to release soft tissues used by the researcher include myofascial release (Barnes, 1997), trigger point therapy (Dommerholt, 2012), Rolfing structural integration (Caspari & Massa, 2012), myofascial induction approaches (Pilat, 2012), and neural release (Wander & Weinschenk, 2012). Other techniques in the literature include osteopathic manipulative therapies (King, 2012), connective tissue manipulation (Prendergast & Rummer, 2012), fascial manipulation (Stecco & Stecco, 2012), accupuncture as a fascia oriented therapy (Irnich & Fleckenstein, 2012), and stretching of fascia (Myers & Frederick, 2012).

Manual therapy as the passive release of soft and neural tissues as well as joint restrictions through physiological and accessory movement in this thesis is accompanied by communication with the patient, reassuring him/her about the causes / contributing factors to his condition and how it presents, as well as giving the patient feedback on his/her tissue response during the manual therapy, re-education of tactile awareness and spatial orientation during manual therapy.

The researcher prepared a timeline (Figure 1.2) of the development in manual therapy to indicate the sequence in which the concepts became common practice in physiotherapy. The relevance of the timeline for this thesis is that it presents the era during which the researcher developed her own approach to a multidimensional approach to the management of patients with CNSLBP. The timeline (Figure 1.2) also indicates the major developments in diagnostic medicine and neuro and orthopaedic surgery because it influenced or coincided with the development of the manual therapy concepts.

1.9.4 Multidisciplinary approach to management of CNSLBP

A multidisciplinary approach to management of patients with CNSLBP entail the treatment of the patient by all relevant health care professionals from different disciplines such as spine surgeons, psychiatrists, neurologist, radiologist, anaesthesiologists, pain medicine practitioners; chiropractors, osteopaths, physical therapists (Fourney *et al*., 2011).

1.9.5 Multidimensional manual therapy for the management of CNSLBP

A multidimensional manual therapy approach to the management of patients with CNSLBP, entail the treatment of a patient by a single health care professional such as a physiotherapist who addresses the multiple components (heterogeneity) of CNSLBP in a particular management approach.

1.9.6 Chronicity

Chronicity is the state where the clinical signs and symptoms of CNSLBP become self-sustainable and continuously worsens. Chronicity is categorised in different stages dependent on pain intensity, pain duration and disability due to pain processing (Buchner, Neubauer, Zahlten-Hinguranage & Schiltenwolf, 2007).

The non-variable factor in chronicity in patients with CNSLBP is a person's genetic make-up which predetermines the extent to which each factor plays a role in the development of chronicity OR the ability to adapt to the condition (Field, 2009; O'Sullivan, 2005). Clinically it is apparent that some people are set up for the development of chronic pain before the pain starts, while others develop it soon after the acute episode of NSLBP and others drift into it (Field, 2009). Typically the largest group of patients with CNSLBP drifts into chronic pain after trying various available treatment options which did not address their problem adequately (Field, 2009).

Socio-demographic factors associated with patients developing CNSLBP are gender, age, marital/family status (single parent/young children, partner retired or disabled), health condition (mental health conditions musculoskeletal conditions, comorbidities) occupational or educational level, time since last worked, occupational status (no longer employed) and local employment rate (Waddell, 2004).

1.9.7 Dysfunction and disability in patients with CNSLBP

Dysfunction and disability are the two ends of a continuum. Dysfunction is the restricted activity due to CNSLBP.

Dysfunction is the malfunction of anatomical structures and physiological and psychological processes to limit the patient's daily functional activities. Dysfunction may become self-perpetuating as a result of any one or a combination of the factors that initiate the physiological change and/or change in the pattern of motor control (movement or activity). It is also possible that emotional stress can result in biomechanical dysfunction due to the fact that it changes muscle recruitment, posture and motor control. If the biomechanical dysfunction from whatever cause perpetuates then it becomes CNSLBP (Waddell, 2004).

When a person is not able to perform the activities of daily living and/ or meet personal, social or occupational demands characteristic of people of his/her age, gender and culture because of CNSLBP, such a person can be regarded as disabled due to his/her CNSLBP (WHO, 2011; The American Medical Association, 2000).

1.9.8 Plasticity

Plasticity is a dynamic physiological property of all the soft tissue systems to adapt their structural organisation and biochemical, physiological and morphological characteristics temporarily or permanently due to new emerging situations as a result of inherent and environmental situations (which can be internal or external) as well as due to other factors (e.g. injuries) affecting the systems. Plasticity is a constant dynamic adaption in all the systems of the ISMS and enables the body (soft tissues) to adapt to injury and psycho-social and environmental demands (Dorland's Illustrated Medical Dictionary, 32nd edition, 2012).

In CNSLBP plasticity occurs in muscular, connective tissue, peripheral, central and autonomic neural tissue due to the tissues' ability to adapt to the stimuli they are subjected to. It can also influence behaviour based on beliefs and fear avoidance. The process of plasticity is the basis for the adaptation of soft tissues and neural tissue to adapt to internal or external stressors and is proposed by the researcher as an important process in the development of ISMS dysfunction and as such the clinical picture of CNSLBP.

1.9.9 Integrated spinal movement system (ISMS)

The term 'ISMS' was conceptualised in this thesis to portray the complex integrated functioning of the spine, pelvis, shoulder girdle, and head as a kinematic chain due to the fact that the:

- Multiple segmental motion segments functions as a unit on segmental and multisegmental levels (Comerford & Mottram, 2003)
- The ligaments stabilise the spine on segmental and multisegmental (global) level
- Muscles of the spine control movement on segmental and multisegmental (global) levels)
- The neural structures emerge from the spinal cord on segmental level but functions as a whole (Middleditch & Oliver, 2005)

1.9.9.1 Integrated spinal movement system (ISMS) dysfunction

'ISMS dysfunction' is the term used in this thesis to describe the dynamic integration between all the systems within and around the ISMS. The integration between the biomechanical component of the ISMS, pain processing, guarded movement, fear of movement, catastrophisation which eventually results in altered pathways in the brain and altered (dysfunctional) motor control, result in poor posture, and impaired coordination which permeates the patients psychological frame of mind and social behaviour to merge into characteristic adaptive behaviour.

1.10 Outline of the study

This chapter indicated that the management of patients with CNSLBP is still a problem in this day and age and that the problem is still growing. The essence of the reasons for the continued lack of success of medical and physiotherapy management of patients with CNSLBP is discussed.

In Chapter 2 the research methodology is described as the researcher's metacognitive reflection on her clinical reasoning, an integrated literature review and the knowledge and skills she obtained from attending courses nationally and

internationally to develop a multidimensional manual therapy model for the management of patients with CNSLBP.

Chapter 3 gives an account of the literature which serves as a knowledge base for the conceptualisation of the ISMS and development of ISMS dysfunction in patients with CNSLBP.

In Chapter 4 the researcher discusses the assessment of a typical patient diagnosed with CNSLBP. The chapter ends with a model on the assessment of patients with CNSLBP.

The multidimensional manual therapy management of patients with CNSLBP is discussed in Chapter 5. The multidimensional manual therapy model for management of these patients is presented in this chapter.

In Chapter 6 the researcher reflects on the multidimensional manual therapy model for managing patients with CNSLBP and discusses the relation of this model with other models used in the management of patients with CNSLBP.

CHAPTER 2

Research methodology

2.1 Introduction

In Chapter 1 the nature of the study is described as model development based on a practice-theory approach. According to McEwen and Wills (2002), a 'practice-theory' is developed from an interaction between practice, theory and research. The development of a practice theory is based on the assumption that a particular phenomenon observed in clinical practice has the potential to enhance understanding in a particular field through the development of propositional knowledge (McEwen and Wills, 2002)

The core problem identified in Section 1.4 is the limited understanding of the biomechanical dysfunction in patients with CNSLBP which is a result of a combination of the different driving factors in the development of CNSLBP. From the researcher's perspective of managing patients with CNSLBP, researchpublications on the topic focus on the treatment of the 'low back' when they treat patients with CNSLBP while the symptoms 'in the low back' can be referred from elsewhere in the spine to the 'low back' (Kääpä *et al*., 2006; Fersum *et al*., 2012).

Another aspect of the problem is that the researcher observed that it is not only patients with CNSLBP's lumbar area that is affected but the whole spine including the position of the head, shoulder and pelvic girdles. From the researcher's perspective manual therapy applied to the lumbar area only, will not address the heterogenetic nature of the patient's CNSLBP. Instead manual therapy should be applied to the whole spine to release the soft tissue connections between the head and the cervical spine, and the soft tissue attachments to the shoulder and pelvic girdles to realign the motion segments, head and girdles. The role of the thoraco-lumbar fascia and the process of plasticity in the shortening of the soft and neural tissues working in on the spine, position of the head, shoulder and pelvic girdles in

the development and management of patients with CNSLBP has not been described in the literature.

2.2 The frame of reference

The frame of reference of the thesis developed from the researcher's professional (declarative or propositional knowledge), personal and professional craft knowledge acquired in clinical practice and through interaction with professionals, patients and their significant others, and by continuously attending courses and congresses related to this field and teaching in this field of practice. Because the researcher is an integral part of the knowledge generation in this study, it is important to state the frame of reference of this study in terms of paradigms and assumptions.

2.2.1 The paradigm of this study

Manual therapy is a clinical area of specialisation in physiotherapy where an aspect of the field of knowledge falls within the empirico-analytical paradigm (biomedical evidence-based knowledge and management of patient problems). Simultaneously due to the fact that manual therapy also requires an interaction between the manual therapist and patient, the patient's perspective of his/her problem should also be interpreted and understood to formulate the problem holistically. The manual therapist's critical reflection on the patient's perspective (illness experience, story, context and culture) and post treatment feedback as well as the patient's clinical presentation (biomedical problem) and process of clinical reasoning to manage the patient's problem is known as dialectical reasoning (Figure 2.2, Section 2.3.1.2) and is typical of the reasoning process used by clinical experts to generate knowledge (Edwards & Jones, 2007). As a result of the dialectical reasoning process and the fact that the researcher performed metacognitive reflection on the clinical presentation of her patients, her management of the patient's heterogenetic CNSLBP problem and the patients' responses this thesis also falls within the interpretive paradigm where knowledge is socially constructed. Working in a multidisciplinary team, attending congresses and conferences, presenting post graduate discussions and courses also contribute to the fact that knowledge is socially constructed. The knowledge obtained from the two different paradigms needs to be integrated to obtain a holistic view on patient problems. This ability to

integrate knowledge generated from two different paradigms to formulate the patient's problem requires dialectical thinking (Edwards & Jones, 2007).

In this study the researcher obtained a deep understanding of the complexity of the problem of patients with CNSLBP through knowledge generated through clinical reasoning. The knowledge that she generated in the management of patients with (C)NSLBP entails knowledge from the empirico-analytical paradigm (biomedical knowledge) as well as from an interpretive paradigm (knowledge from the patient's lived experiences).To verbalise and write up this deep understanding and knowledge generated in clinical practice through clinical reasoning, the researcher performed metacognitive reflection in a heuristic approach to develop a model based on her individualised patient management. The paradigm within which this study was conducted is therefore the interpretive paradigm.

In any scientific research it is important to state the assumptions on which the research was conducted. In this research, which falls within the qualitative research framework in which the researcher is an integral part of the data generation and analysis, these assumptions need to be explicitly stated to interpret the researcher's frame of reference when she developed the model.

2.2.1.1 Assumptions

Assumptions are statements that are not necessarily empirically tested but which are regarded as true. These assumptions influence the logic behind the study and therefore need to be explicitly stated, particularly in qualitative research (Cresswell, 2007).

The assumptions underlying this study and listed below are ontological, epistemological, axiological and methodological assumptions and assumptions regarding the rhetorical structure of the study.

Ontological assumptions

Ontological assumptions deal with the question: 'What is reality?' From an empirico-analytical paradigm ontological assumptions indicate that the real world is driven by real natural causes (Polit & Beck, 2007).

In terms of the empirico-analytical paradigm the most important ontological assumptions in this study are:

- The anatomical structures of the spine function as an integrated spinal movement system including the head, shoulder and pelvic girdles
- The development of integrated spinal movement system (ISMS) dysfunction entails the inseparable interaction between the dysfunction of the neuromusculoskeletal systems pain processing and characteristic adaptive behaviour due to a process of plasticity.

In terms of the interpretive or naturalistic paradigm the most important assumptions are:

- Principles for the holistic management of patients with CNSLBP can be deduced from multiple realities of patients' lived experiences.
- Patients and their significant others can contribute to the generation of new understanding and, as such, new knowledge in the context of the clinical reasoning process.

Epistemological assumptions

Epistemological assumptions answer the question: 'How is the inquirer related to those being researched?' (Polit & Beck, 2008). Higgs, Fish and Rothwell (2010 p 163) state that:

Knowledge is constructed in the framework of socio-political, cultural and historical contexts. Practice knowledge evolves within a dynamic history of ideas contained in the particular practice domain and within the history of how ideas born in that practice domain have been shaped by that practice. Each of these dimensions and contexts of knowledge has particular relevance to how we use knowledge in reasoning and generate knowledge from within reasoning. During professional socialization, practitioners learn the ways of being, acting, thinking and communicating that characterize their profession

The researcher's thinking processes are an integral part of the knowledge generated in this research. The way in which she has kept abreast with the knowledge generation in the field of manual therapy over the past 40 years is given in Addendum 1.

The epistemological stance of the researcher's practice is dialectic in nature (Figure 2.2). This implies that her practice is conducted within a biomedical practice framework in which '*knowledge is seen as [an] objective, predictive, empirical generalizable, explanatory phenomenon that arises from the use of the natural scientific method and theorization in a world of external objective reality'* (Higgs *et al*., 2010).

However, the researcher also practises within the interpretive paradigm, which implies that she practises from a patient/client-centred approach in which each patient's problem is unique within his/her history of the development of the clinical picture, and the socio-cultural and psycho-social factors influencing the presentation of the clinical picture. The researcher's interaction with patients and their significant others has contributed to the construction of the researcher's professional craft and personal (tacit) knowledge.

The professional craft and personal (tacit) knowledge the researcher has acquired over 40 years of clinical practice has occurred by sharing knowledge, ideas and experience, in collaboration with the multi-professional team she works with, with colleagues during courses and conferences at national and international level. Teaching postgraduate students has also contributed to the development of the researcher's clinical reasoning and research (and metacognitive reflection on clinical practice) as it is presented in this thesis.

The researcher has also indicated in Figure 1.2 of Chapter 1 the approximate timeline of the development of the manual therapy concepts which she has kept pace with over the last 40 years (also refer to Addendum 2). As a manual therapist the researcher has developed as a professional over the same time and, as such, can reflect on the development of manual therapy in the context of the present day evidence that emanates from basic sciences and randomized clinical trials.

In this context the following epistemological assumptions are stated:

- Propositional knowledge on the development of ISMS dysfunction integrated with pain processing and characteristic adaptive behaviour of patients with CNSLBP can be observed and verbalised on the basis of the interpretation of observations, and all the dimensions of clinical reasoning.
- The researcher can generate knowledge based on her own personal knowledge, professional craft knowledge, tacit knowledge, and propositional knowledge.
- The researcher's knowledge was shaped by interaction with colleagues, team members, patients and their significant others and metacognitive reflection on her clinical practice and clinical reasoning.

Axiological assumptions

'Axiological assumptions' refer to the way in which values mediate and shape our understanding of knowledge and clinical practice. Axiological assumptions are critical in the interpretive paradigm, especially in the context of this study because the holistic management approach for patients with CNSLBP is based on a patient-centred approach to clinical management in which the holistic understanding of the patient's illness behaviour, experience of his/her problem, his/her history, beliefs and culture is part of the problem identification. Patient feedback during and after treatment also plays an integral part of the researcher's understanding of the problem and its management. The axiological assumption on which this study is based is that:

- Integrating the patient's biomedical diagnosis of impairment/disability and addressing the patient's 'lived experience' in a diagnosis of the patient's problem are an integral part of manual therapy.

Methodological assumptions

Methodological assumptions ask the question: 'how is evidence best obtained?' (Polit & Beck, 2008 p 14) or 'what are the processes and procedures of the

research?' Methodological assumptions involve the scientific criteria for obtaining knowledge and refer to the methods used to validate a study scientifically (Polit & Hungler, 1997).

The methodological assumptions pertaining to clinical reasoning as a knowledge-generating activity are:

- Clinical reasoning entails a complex combination of thinking processes:
  These processes consist of a cognitive process, which includes hypothetical deductive reasoning, pattern recognition, forward and backward reasoning (inductive and deductive reasoning), knowledge reasoning integration, and intuitive reasoning.
  - An interactive process, which includes multidisciplinary reasoning, conditional reasoning, narrative reasoning, interactive reasoning (collaborative reasoning), ethical reasoning, and teaching as reasoning (Higgs & Jones, 2010 p 7-8);
  - Clinical experts use various combinations of all forms of clinical reasoning during the treatment of patients and as a result are able to generalise the principles of patient management;
  - Clinical reasoning requires capability in four dimensions: reflective thinking, critical thinking, dialectical thinking, and complex thinking;
  - Clinical reasoning during the holistic manual therapy management of patients with CNSLBP is performed within the biopsychosocial paradigm.
  - Propositional knowledge can be generated through metacognitive reflection on clinical reasoning to identify the principles of a holistic manual therapy approach for managing patients with CNSLBP.
  - Experiential tacit knowledge, professional craft knowledge and personal knowledge are validated by narrative (thick) description of clinical reasoning and metacognitive reflection.
  - An expert clinician's tacit knowledge (personal and professional craft knowledge) can be made explicit through a process of metacognitive reflection on his/her clinical reasoning.
  - The research design of the study (metacognitive reflection on the process of clinical reasoning) is flexible and described in a narrative form.

- A qualitative research design facilitates an in-depth exploration of the phenomenon and favours no methodological approach above another.

2.3 The research approach

The research approach of this study was embedded in the interpretive paradigm as the researcher used a hermeneutic approach to obtain a deeper understanding of the development (causative factors) of CNSLBP and patients' responses to manual therapy.

The hermeneutic approach to the research was completely integrated and relied on metacognitive reflection on the clinical reasoning process. The result of the hermeneutic approach was a model for the holistic manual therapy approach for managing patients with CNSLBP.

The research approach to this study was therefore a hermeneutic metacognitive approach to the development of a clinical management model. Because model development is strongly related to theory development, the study also falls within a grounded theory approach (McEwen & Wills, 2002).

A model was chosen as the outcome of the study because in a model the relationships between the concepts that have been identified can be indicated in a way that is inherent to the clinical problem-solving process (McEwen & Wills, 2002). Figure 2.1 presents the research approach in diagram form.

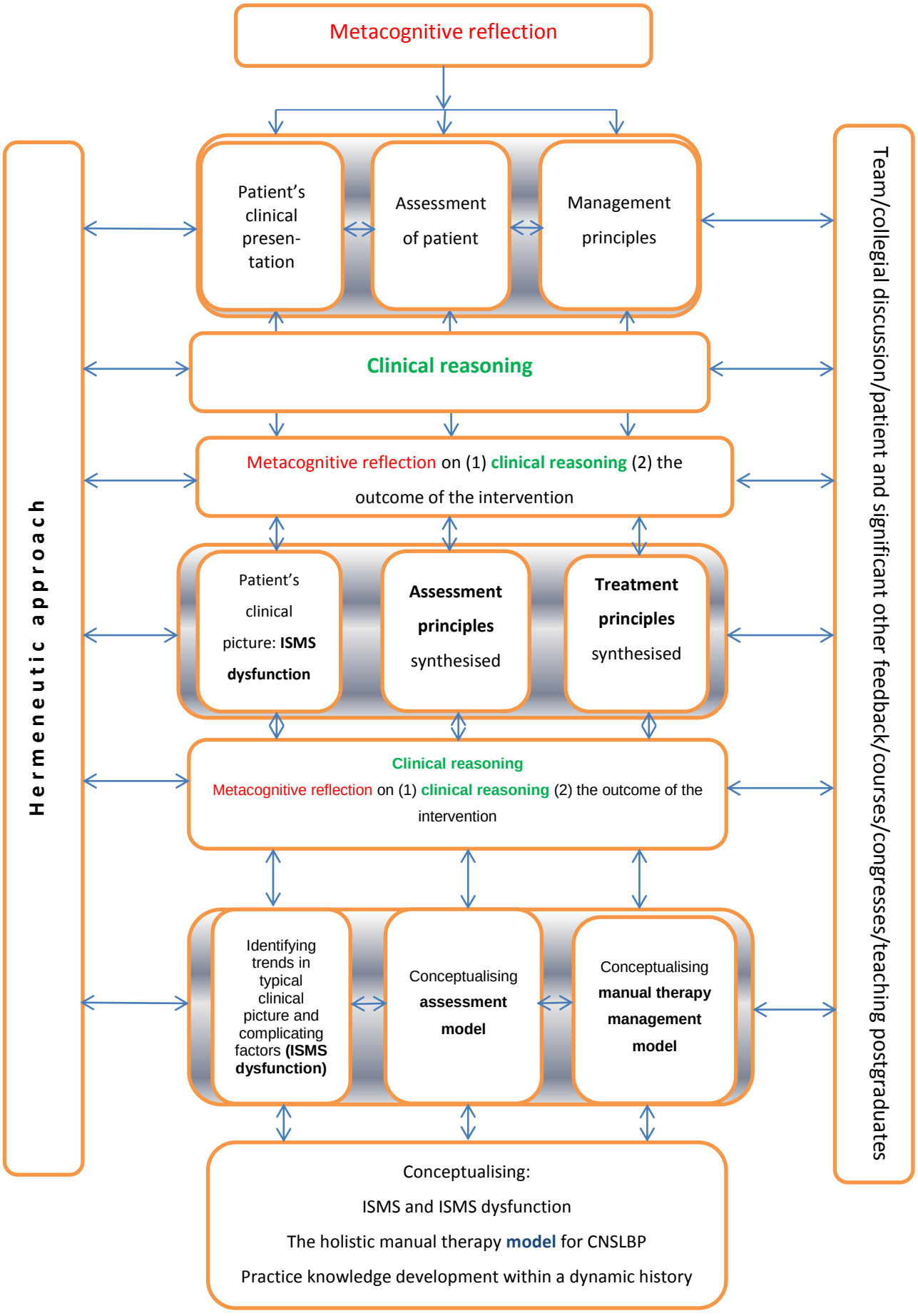


Figure 2.1: Summary of the research approach and research methodology

2.3.1 The hermeneutic process as knowledge-generating process

Hermeneutics is defined by Polit and Beck (2007 p 755) as a Q*ualitative research tradition drawing on interpretive phenomenology that focuses on the lived experiences of humans and on how they interpret those experiences. Fonteyn and Ritter (2010 p 237) states that: 'Hermeneutics is based on the phenomenological tradition that meaning is subjective and contextually constructed*. The term 'hermeneutic circle' is used to describe '*the experience of 'moving' one's thinking dialectically between the parts and the whole'* to understand the phenomenon (Paterson and Higgs, 2010 p 182-183). Loftus and Higgs (2010) call hermeneutics the '*art and study of interpretation'*.

In this study the researcher reflected on human actions and situations that she was involved in herself. During this process she shifted her thinking repeatedly between all dimensions of clinical reasoning and metacognitive reflection to understand the patient's problem and their response to treatment as well as her own therapeutic actions. This process was repeated until a perspective of the development of the clinical picture of a typical patient diagnosed with CNSLBP and his/her response to treatment became clear. This repeated thinking and the reflective processes revealed the fact that she treated the ISMS dysfunction of a patient with CNSLBP and his/her low back pain problem as a person: in other words from an interpretive paradigm she developed in an intuitive way and based on observation and interpretation, developed an understanding of the patient's complex clinical picture and how to address it effectively.

Various schools of thought exist on the process of analysis of hermeneutic data (also called phenomenological data). The characteristic of analysing hermeneutic data (also called interpretive phenomenology) '*is the notion of the **hermeneutic circle.*** [Researcher's emphasis in bold] *The circle signifies a methodological process in which, to reach understanding, there is continuous movement between the parts and the whole of the text being analysed*' (Polit & Beck, 2008 p 521) as mentioned above. This process is also relevant for analysing data from any human action or situation.

The multidimensional manual therapy model for managing patients with CNSLBP discussed in this thesis is the result of the 'analysis of the parts in the context of the whole' and a synthesis of a new holistic approach to the management of these patients.

2.3.1.1 Metacognitive reflection as knowledge-generating process

> *Metacognition is described by the integrative link between knowledge and cognition in the clinical reasoning process (Higgs, 2004), and as the self-monitoring employed by the therapist in order to detect links or inconsistencies between the current situation and expectations based on learning from past clinical experience (Higgs and Jones, 2000). Metacognition may involve reflecting on and critiquing data collection processes and results, considering different strategies of reasoning and reviewing personal biases or limitations in knowledge depth, breadth or organization* (Christensen, Jones, Higgs and Edwards, 2010).

Metacognition is also called '*reflective self-awareness*' (Christensen *et al*., 2010). This ability in a clinician enables him/her to:

- Identify limitations in the quality of the information that she/he gathered;
- Identify inconsistencies or unexpected findings;
- Monitor his/her own practice or clinical performance and clinical reasoning by seeking errors and credibility;
- Recognise when their knowledge or skills are insufficient and when remedial action is needed;
- Recognise, analyse and discuss their thinking processes and, as such, develop their thinking processes
- Self-modify their actions, and by practising metacognition develop into 'a thinking' professional (Cahill & Fonteyn, 2010).

2.3.1.2 Clinical reasoning as knowledge-generating process

The clinical reasoning process that clinicians engage in during patient care is a complex process in which various thinking strategies can be identified. Clinical reasoning is inherent in patient care and therefore played a major role in generating the researcher's perspectives of the development of ISMS dysfunction and the way in which the multidimensional manual therapy management model presented in this study was developed.

Christensen *et al*. (2010) distinguish four dimensions in clinical reasoning: reflective thinking, critical thinking, dialectical thinking and complexity thinking.

(1) Reflective thinking as dimension of clinical reasoning

Reflective thinking by clinicians can take place over a period of time when the clinician reflects on his/her past performance with a particular patient. It can also take place while the clinician is treating a patient, in which case it is called 'reflection in action' and 'reflection on action' (reflection in the midst of action without interrupting the action) as well as reflection on action which entails reflection after treatment/intervention (Christensen *et al*., 2010). 'Reflection on action' refers to thinking back on experiences *'to discover how our knowing in action may have contributed to an unexpected outcome'* (Schon, 1987 p 28). '*In this sense reflection becomes a way of cognitively organizing experience through construction of a sense of coherence, and facilitating planning for future action*' (Forneris, 2004, in Christensen *et al*., 2010 p 105). Reflection on action and reflection in action specifically create an awareness of critiquing one's own thinking and other actions ('metacognition') and is an essential element of sound clinical reasoning (Christensen *et al*., 2010).

Reflective self-awareness associated with concurrent learning is viewed the same as metacognition (Christensen *et al.,* 2010). Reflection is also intimately linked with 'critical thinking', although reflection is by definition not 'critical'. Reflection as part of critical thinking may bring to light the underlying assumptions that people use to justify their beliefs and, as such, contribute to a new perspective based on experience (Christensen *et al*., 2010).

(2) Critical thinking as dimension of clinical reasoning

Critical thinking is defined as:

> *… the intellectually disciplined process of activity and skillfully conceptualizing applying, analysing, synthesizing and/or evaluating information gathered from or generated by observation, experience, reflection, reasoning or communication as a guide to belief and action. It is a skill that can be applied when developing an understanding of a particular situation or context, and can also be applied to the examination of thinking (one's own or that of others) in the context of particular situations* (Christensen *et al*., 2010 p 105).

Critical thinking is also defined as the '*art of analysing and evaluating thinking with the view to improve it and to learn from it*' (Paul & Elder, 2004 in Christensen *et al*., 2010 p 105).

In this context critical thinking links with metacognition. Metacognitive reflection is described in Section 2.3.1.1.

The outcome of thinking critically in practice is the achievement of '*a coherence of understanding'* (Forneris, 2004 in Christensen *et al*., 2010 p 105). Forneris also identified four core attributes to critical thinking: reflection, context, dialogue and time. When the four attributes are applied in clinical reasoning they form a useful framework within which all the different elements of practice and the factors influencing collaborative clinical reasoning are linked to critical thinking.

Through reflection the researcher can attach meaning to information and illuminate '*the why and the reason for what we do and how we critically discriminate what is relevant*' (Forneris, 2004 in Christensen *et al*., 2010 p 105). Mezirow (2000) explains:

> *… 'reflection' allows for interpretation of experience; as part of reflexion the thinker comes to know the 'why' of a situation by subjectively and objectively reframing the context to bring to light the underlying assumptions used to justify beliefs. New knowledge may then be produced if a new perspective on experience is achieved.* (Christensen *et al*., 2010 p 105)

The context in which the researcher developed a new understanding of the problem of how and why ISMS dysfunction develops and forms a major part of patients with CNSLBP's clinical picture was her own clinical practice in which she has treated and still treats out-patients and in-patients (patients admitted to hospital) as an independent practitioner (individually) as well as in the context of a multidisciplinary team. In the same context she performs the clinical training of postgraduate students.

The researcher has a client/patient-centred approach to clinical practice. In a client/patient-centred approach to clinical practice patients are active participants in their problem identification, and multidimensional management plan. Person-centred care is grounded in a particular philosophical tradition in which the practitioner draws on non-propositional knowledge of various kinds such as aesthetic and ethical patterns of knowing, professional craft knowledge and the personal knowledge of the patient/client/family member to manage each individual's (patient's) problem in a unique way that will suit the individual's needs and preferences.

In a client-centred approach practitioners use a unique blend of propositional and non-propositional knowledge to treat the patient in his/her unique context. This unique blending of the different kinds of knowledge is intermingled with the practitioner's qualities, intelligence, practical wisdom, practical skills and therapeutic use of self. The unique blend of all these practical and 'wisdom' 'skills' in the 'hot action' of practice is called '*professional artistry'* (Paterson & Higgs, 2010).

Dialogue as inherent process in critical thinking

'Dialogue' refers to the '*interactive process of evaluating perspectives and assumptions within context, in order to develop an understanding*' (Forneris, 2004 in Christensen *et al*., 2010 p 105). '*Dialogue involves an ongoing evolving exploration of how the context of a situation influences the way in which that situation is understood*'. The interactive ongoing discussion or constructive conversation (dialogue) can take place with oneself, patients, peers and/or team members to reflect on or share ideas on an experience, or to assess reasons and justifications for

assumptions in order to reach new insight. The purpose of the constructive conversation is to get ideas from different perspectives that will facilitate experiential learning.

Time taken to reflect on experience is an absolute necessity to obtain a deeper understanding of a phenomenon. During this reflection, insight gained from past experiences can be integrated into present experiences and may inform future action.

(3) Dialectical thinking as dimension of clinical reasoning

Dialectical reasoning during clinical reasoning is typical of the clinical reasoning of experts. Clinicians who are able to do dialectical reasoning can shift their thinking between two potentially opposing ways of thinking; namely, from empirico-analytical thinking to interpretive thinking. The clinicians are therefore able to collaborate with their patients to get a holistic understanding of the 'lived experience' (in the interpretive paradigm) of the patient as well as a biomedical experience (in the empirico-analytical paradigm) of the patient's problem. Dialectic thinking therefore also includes both deductive and inductive thinking.

The dialectic thinking process enables a clinician to get an understanding of the complexity of the patient's problem. Dialectic thinking is also an integral part of complexity thinking. Edwards and Jones (2007) describe a dialectical model of clinical reasoning which reflects the clinical reasoning that the researcher has done during the diagnosis and management of patients with CNSLBP. The dialectical model is displayed in Figure 2.2.

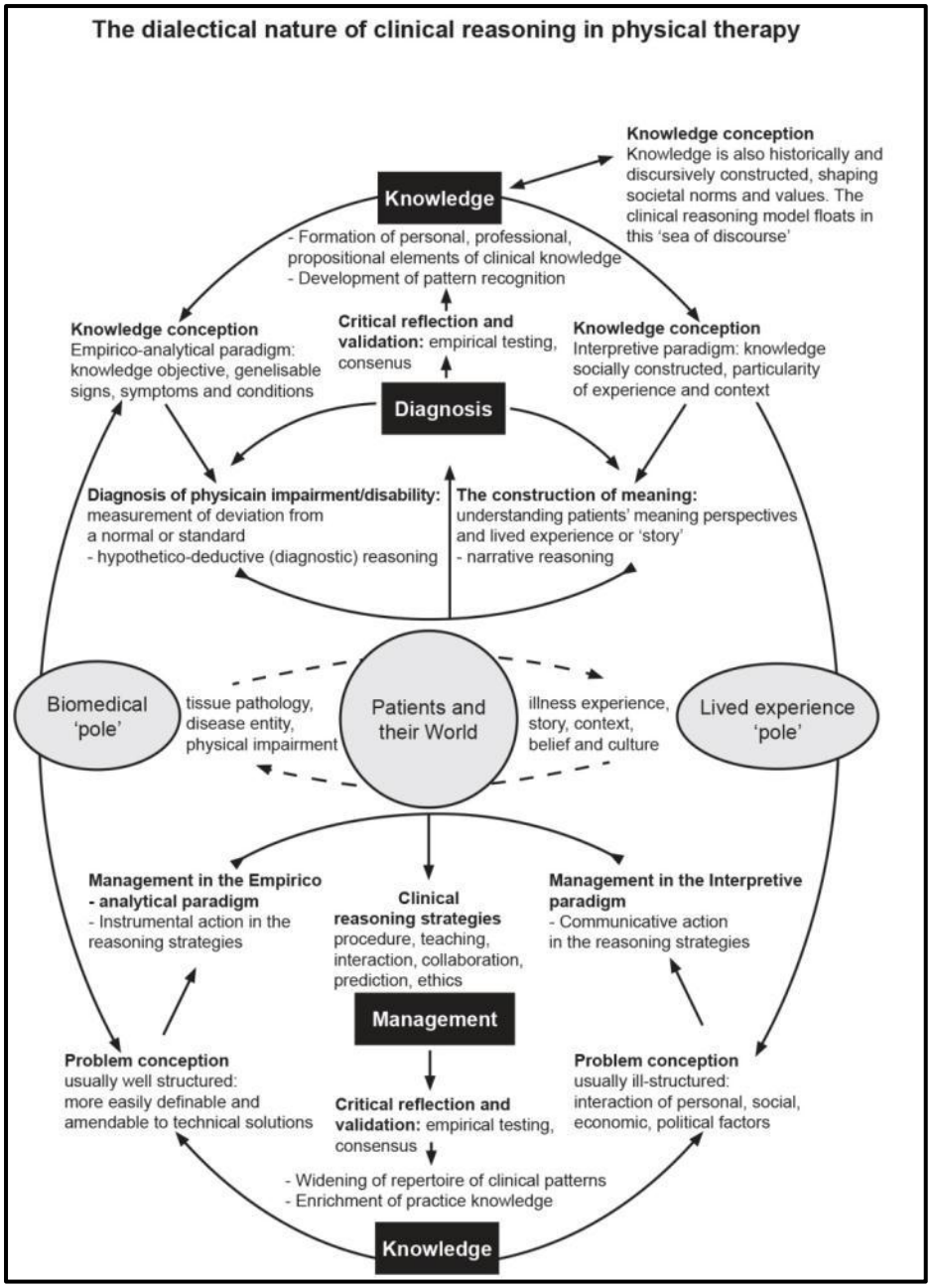


Figure 2.2: Dialectical reasoning in the diagnosis and management of patients

(Edwards & Jones, 2007 in Jensen, Gwyer, Hack & Shepard, 2007 p 210)

(4) Complexity thinking as dimension of clinical reasoning

Complexity thinking is characterised by contemporary systems thinking. It recognises the complex relationship between the many elements and influences in a given situation. It entails forward reasoning (induction) based on specific cues toward a general judgment, deduction which entails reasoning from a general premise toward a specific conclusion, and dialectical thinking.

In the context of this study an example of complexity thinking (systems thinking) would entail understanding the complexity of human behaviour (thoughts, beliefs, emotional arousal, communication) integrated with a dysfunctional ISMS in which neuromusculoskeletal dysfunction is integrated with pain processing and psychological responses which develops into characteristic adaptive behavior in which each of these components can be a driving force behind the patient's clinical picture influenced by external as well as internal conditions (Stephenson, 2002).

By implementing the various forms of thinking and reasoning discussed in the preceding paragraphs, the researcher was able to generate knowledge and insight into the management of patients with CNSLBP from the clinical perspective that was not yet part of the declarative knowledge of the profession.

2.3.1.3 Knowledge-generation process through clinical reasoning

Edwards and Jones (2007) describe a process through which clinical knowledge is generated and conceptualised through clinical reasoning. This process (Figure 2.3) reflects the generic process that the researcher used to conceptualise the components of the multidimensional manual therapy model for managing patients with CNSLBP.

The process displayed in Figure 2.3 was developed on research conducted to identify the processes that clinical experts use to generate new clinical knowledge

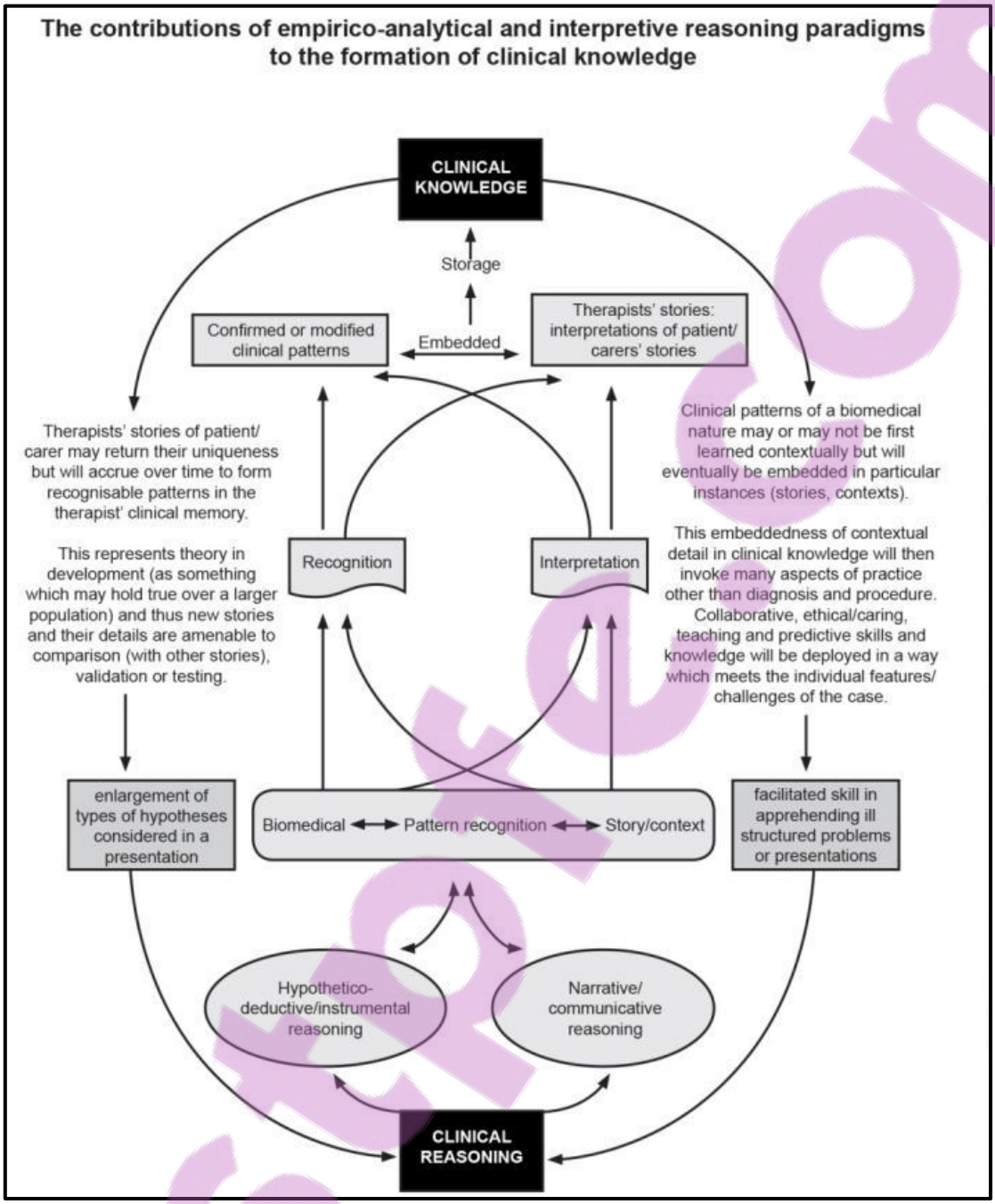


Figure 2.3: The contributions of empirico-analytical and interpretive reasoning paradigms to the formation of clinical knowledge

(Edwards & Jones, 2007 in Jensen, Gwyer, Hack & Shepard, 2007 p 204)

2.3.2 Model development

Model development as a research design and as the outcome of this study was chosen because in a model the relationship between the different components and concepts related to the development and management of CNSLBP could be illustrated instead of extensively described in terms of management processes, guidelines, services to patients and the identification of new fields for further research. When the relationships between the components and concepts are illustrated they can be tested with empirico-analytical research (Higgs *et al*., 2010).

The basic function of a model is to promote, explain and define relationships, structure, and linkages between concepts to enhance understanding of a phenomenon: in other words it is '*heuristic, i.e. discovering or "exposing" certain relationships between concepts'* (Mouton & Marais, 1990 p 60).

The process for developing a model (Polit & Beck, 2007 p 85) will therefore be the same as the process for developing a theory. This process is described by Walker and Avant (1995) as:

- *Select a topic of interest (may be one concept / variable or a framework of several concepts)*
- *Conduct a review of the literature or use field observations and note related variables*
- *Organize relational statements in terms of patterns of relationships amongst the variables. Diagrams may be used to express relationships amongst concepts and to organize the components of the theory.*

The study is divided into three main sections: the conceptualisation of the concept of the ISMS; the discussion on the development of ISMS dysfunction integrated with pain processing and psychological responses which develops into characteristic adaptive behaviour. The management (assessment and multidimensional manual therapy) of the patients is displayed in a model.

2.3.3 The role of literature in the development of a holistic manual therapy model for managing patients with CNSLBP

Owing to the fact that the research topic (CNSLBP) is such a major clinical problem, a dearth of literature is available on the topic. Because the researcher presents a different framework (model) on the manual therapy management of patients with CNSLBP, very little literature relevant to the topic could be found. The main focus of the literature review in this study was therefore to search for and where possible to provide evidence for the observations, statements (inductive reasoning statements) and conclusions (deductive reasoning statements) made by the researcher in the discourse throughout the study.

The fact that the literature was used extensively in the text to support the researcher's arguments, statements and conclusions, and served as a contribution to the trustworthiness of the observations, arguments, statements and conclusions conceptualised and presented in this study that were not based on evidence based literature.

The literature consulted in this study included textbooks, research articles, systematic reviews and review articles.

2.4 Trustworthiness of the conceptualisation of ISMS dysfunction and the development of the multidimensional manual therapy model

The phenomenon of ISMS dysfunction, the problem of the patient with CNSLBP from a biomedical as well as a lived experience perspective, and the holistic manual therapy management of the problem are discussed extensively in this thesis so that readers are able to follow the processes and arguments.

The discussion in this thesis as described in Chapters 3, 4 and 5 can be considered to be a 'thick description' of the researcher's manual therapy management of patients with CNSLBP as it developed from metacognitive reflection on clinical reasoning during the treatment of patients from a hermeneutic approach (Polit & Beck, 2007). Denzin and Lincoln (2011) call thick descriptions '*describing a line of*

argumentation' to make the researcher's thoughts visible. In the context of the empirico-analytical and interpretive paradigms of patient management, the researcher is bound to ensure that the statements and conclusions are in line with the latest research evidence published in the literature (Polit & Beck, 2008). By doing this she has shown the trail of evidence-based clinical reasoning throughout the thesis.

The researcher is an experienced manual therapist who has kept abreast with the development in the field of manual therapy over the years. She is therefore experienced in practising the theoretical and clinical principles of manual therapy and the dimensions of clinical reasoning. The clinical reasoning and metacognitive reflection on the clinical reasoning presented in this study are therefore embedded in the practice of manual therapy, which contributes to the validity of the arguments and statements made in this thesis.

The researcher has developed the multidimensional manual therapy over the past 40 years and although the basic principles of the multidimensional manual therapy approach to manage patients with CNSLBP, stayed the same it expanded over the years as new knowledge was generated.

2.5 Significance of the study

The present Clinical Practice Guidelines for the management of patients with CNSLBP (European Guidelines for the Management of Chronic Non-specific Low Back Pain, 2004; National Collaborating Centre for Primary Care, 2009) do not give a clear understanding of the causes of or the reasons for patients developing CNSLBP. Manual therapy based on these Clinical Practice Guidelines also does not lead to satisfactory clinical outcomes for the patients suffering from CNSLBP.

The model developed from this study was grounded in clinical practice supported by research evidence from the literature and therefor brings a different approach to the clinical and research area focusing on CNSLBP which may stimulate further research in this area.

The model developed in this study can also shed new light on other aspects of neuromusculoskeletal physiotherapy, which can contribute to further research to expand the evidence-based (propositional) knowledge in the profession of physiotherapy.

The model addresses the limited understanding of the biomechanical origin of CNSLBP within the complexity of the heterogenetic nature of CNSLBP and the manual therapy management of the problem. From this model new research protocols can be developed not only to test the model empirically but also to develop research protocols in areas in which research evidence is not yet available. The clinical reasoning in this model leans heavily on research from the basic sciences and because this basic scientific knowledge has been applied in clinical practice, it may open up new understanding of and avenues for research in the basic sciences such as physiology, pathology, endocrinology and genetics.

The research process in this study can serve as a basis for the development of new knowledge from clinical practice (practice-theories) in other complex conditions treated by physiotherapy.

2.6 Ethical considerations

This study is based on a hermeneutic enquiry approach, which focuses on the researcher's reflection on the 'parts' within the holistic clinical picture of the patient as well as the holistic clinical picture and the patient's response on treatment by analysing the particular 'parts' of the patient's problem/response The researcher reflected on her own clinical procedures, actions and thinking during clinical reasoning (problem-solving strategies). The only ethical consideration relevant to the research in this study was related to recognising authorship and avoiding plagiarism. The researcher's reflection on her own thinking/clinical reasoning during the treatment of patients did not involve the patients at all and no patients' identity was ever revealed.

2.7 Summary of the chapter

In this chapter the research methodology for developing a multidimensional manual therapy model through metacognitive reflection in a hermeneutic approach on the researcher's clinical reasoning during clinical practice is discussed. The research process is graphically displayed in Figure 2.1 and a summary of the components of the model is given in Table 1.1 (Section 1.7). The nature of qualitative research is such that the research methodology is not usually reproducible - which is the case in the research methodology of this study. However, the researcher has constructed this thesis on the principles discussed in this chapter to optimise the trustworthiness of this work.

CHAPTER 3

Mechanisms generating the development of a dysfunctional integrated spinal movement system

3.1 Introduction

Although much is written about the different aspects of the heterogenic clinical picture with which patients with CNSLBP present, an understanding of the close interactive relationship between the biomechanical, neurological and psychological systems as part of the complex clinical picture of these patients has not been found in the literature. A detailed description of how the complex clinical picture originates or might develop is also not discussed in the literature. Parts of the biomechanical origin of CNSLBP are discussed in the literature by Panjabi (2003) on the role of the neutral zone in motion segment dysfunction. O'Sullivan (2000 p 2) discussed a lumbar segmental instability model based on the *movement dysfunction within the neutral zone resulting in excessive intervertebral motion at the symptomatic* lumbar.
In 2007 Zusman states that validation for a motion segment instability model due to muscular insufficiency is currently been sought. The lack of a comprehensive understanding of the role of the motion segment dysfunction in relation to the whole spine as a kinetic chain may be a contributing factor in varying outcomes in the management of these patients on the basis of the biomedical model (O'Sullivan, 2011). The management of patients with CNSLBP should be based on the biopsychosocial model for the understanding and management of the individual variations in patients with the clinical picture of CNSLBP (O'Sullivan, 2011; Fourney *et al*., 2011; Fersum *et al*., 2012; O'Sullivan 2005) because it is such a complex interaction between various physical, behavioural, lifestyle, neurophysiological, psychological and cognitive factors (O'Sullivan, 2011).

In this chapter the concept 'integrated spinal movement system' (ISMS) is discussed as the premise for the researcher's hypothesis on the development of ISMS dysfunction and how it integrates with the patient's pain processing and

characteristic adaptive behaviour to culminate in the clinical picture of the patient with CNSLBP.

The researcher first discusses how she has conceptualised the 'ISMS', which serves as the premise for the deduction of principles for the assessment and treatment of the lumbar spine as a part of a functional biomechanical system. The discussion is based on a literature review on functional biomechanics and on the generalisation of observation of the most common clinical features in the presentation of the clinical picture of patients with CNSLBP. The researcher's explanation of how ISMS dysfunction develops and culminates in the clinical picture of patients with CNSLBP is based on her clinical observations and reasoning over a period of 40 years. During this time the researcher worked as a member of the multidisciplinary team in a spinal unit and interacted with colleagues on the holistic management of the patients of that unit.

The researcher agrees with Waddell (2004) that CNSLBP develops primarily or secondarily in the low back. The low back pain (regardless of whether it is primary or secondary) sensitises the pain mechanisms to the higher centres with its modulatory influences. From here a dynamic self-perpetuating cycle develops, from which any component (biomechanical, higher centres of the brain under the influence of psycho-social stress) of the cycle can become a dominant driver of the CNSLBP. This cycle implies that low back dysfunction may heal after an initial incident or may become vulnerable to recurrences, which may maintain the spinal dysfunction. From an alternative perspective the researcher assumes that despite clinical healing of the 'back pain' the altered pathways in the higher centres may perpetuate the (phantom) pain perception. The researcher concludes that regardless of the primary component that drives the CNSLBP, patients will always present with a biomechanical component and pain processing cycle and will display characteristic adaptive behaviours (Norkin & Levangie, 2005; Waddell, 2004).

3.2 Conceptualisation of the ISMS

The ISMS is a complex integration between the articular, ligamentous, muscular, neural and connective tissue systems of the motion segment, which is a component

of the entire spine. The entire spine is composed of a series of motion segments and therefore functions as a kinetic chain but functions also inseparably with its adjacent connections: the head, shoulder girdles and pelvis.

The spine is characterised as a series of 24 integrated and interconnected motion segments that function as a closed kinematic chain. The term 'motion segment' refers to the intervertebral disc and its articulations with the adjacent vertebral bodies below and above. The integrated interconnected series of motion segments, which forms the vertical axis of the body, has to reconcile stability and mobility as two opposing mechanical requirements for the vertebral column (core segment) of the body (Middleditch & Oliver, 2005).

Stability of the chain of motion segments is achieved by a series of segmental and longitudinal ligaments and muscles (local and global stability muscles), which stabilises the vertebral column from the pelvis to the occipit (Sahrmann, 2002; Middleditch & Oliver, 2005; Comerford & Mottram, 2012). Muscular stability is achieved by the segmental musculature between the motion segments as well as the global musculature (quadratus lumborum and psoas major between the pelvis and the ribcage), muscles between the shoulder girdle and pelvis (latissimus dorsi) and postural muscles (trapezius and rhomboid minor and major), which stabilise the vertebral column to the shoulder girdle (Kapandji, 2008).

Mobility of the spine is controlled by the global mobilisers in coordination with local and global stabilising muscles. The design for the stability of the spinal column therefore is characterised by mobility and stability. The muscular system under control of the CNS, PNS and ANS controls the muscle tension and contraction and adapts automatically to maintain or restore equilibrium and to achieve voluntary functional movement (Kapandji, 2008).

3.2.1 The articular components of the ISMS

The motion segment is the traditional unit of study in spinal kinematics and is therefore used as the premise for discussing normal and abnormal functional biomechanics of the spine (Middleditch & Oliver, 2005). At a segmental level the vertebral body has six degrees of freedom: forward and backward gliding (anterior

and posterior translation), forward and backward tilt (anterior and posterior sagittal rotation), lateral glide (lateral translation), lateral tilt around a sagittal plane (lateral flexion), distracting and compression in the horizontal spinal axis, and rotation around a vertical axis (Middleditch & Oliver, 2005). During movement of a vertebral body its axis differs from moment to moment. The instantaneous axis of rotation represents a mean axis around which coupled accessory movements occur during movement. The movement of a rigid vertebral body in a three-dimensional space can be analysed at a particular instant as a simple screw motion. The screw motion is a combination of rotation and translation about and along the same axis as illustrated in Figure 3.1.

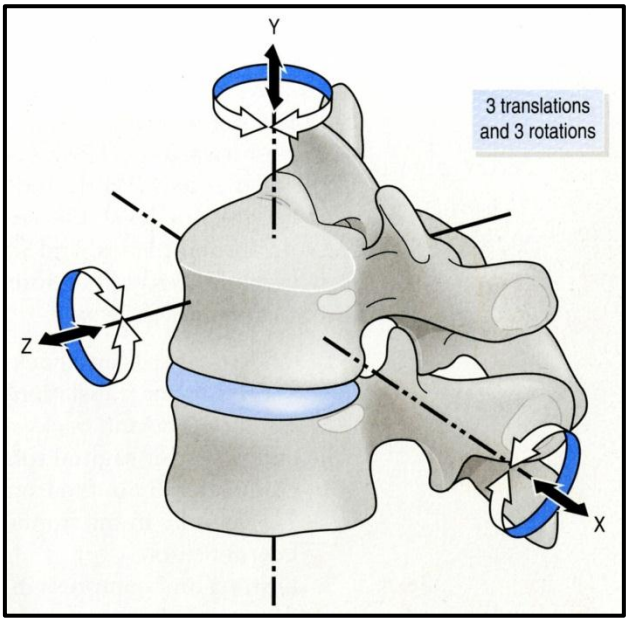


Figure 3.1: Degrees of motion of the motion segment

(Middleditch & Oliver, 2005, p 178)

Movement at a motion segment is determined by the thickness of the intervertebral disc, the compliance of its fibrocartilage and the dimensions of the shape of the adjacent vertebral end plates. The shape and the orientation of the articular apophyseal joints determine the type and amount of movement that is possible at a particular motion segment. The orientation of the apophyseal joints varies in the different spinal regions and allows movement around two or three movement axes at the same time (Middleditch & Oliver, 2005).

In terms of physiological spinal movements, rotation of the spine takes place simultaneously with lateral flexion and vice versa and is known as coupling movements. Tripled movements take place when movement occurs in three dimensions around three axes. The implication of the phenomena of coupled or tripled movements is of importance in the interpretation of abnormal movement patterns in relation to pathology or abnormal spinal biomechanics as a result of abnormal spinal loading (Middleditch & Oliver, 2005).

The global mobility of the spine as a functional biomechanical chain of motion segments is enhanced by the fact that it has two mobile lordotic spinal curves (the cervical and lumbar spines) on either side of the thoracic kyphosis, which is stabilised by the ribcage. The sacrum as a rigid kyphosis links to the pelvis through the two sacro-iliac joints and is stabilised by the pelvis. *Body weight from the vertebral column is transmitted via the 5th lumbar vertebra to the sacrum through the SI joints along the alar of the sacrum and through the ischial tuberosities towards the acetabulum* (Middleditch & Oliver, 2005 p 215).

> Changes in the position of any one motion segment will result in changes in position of the adjacent superior and inferior [motion] segments and as such will affect the cervical, thoracic and lumbar spinal curves (Norkin & Levangie, 1992).

The spinal curves are interdependent and if the face is kept vertical the head has to remain balanced over the sacrum: the relationship between the head and the pelvis behaves as if it is part of a closed kinematic chain.

Owing to the differences in the size of the vertebral bodies, intervertebral discs and the orientation of the zygapophyseal joints in the cervical spine together with the presence of ribs and sternum in the thoracic region, the regions of the spine respond differently to abnormal loading. The L4-S1 motion segments are especially vulnerable for abnormal loading due to the forward tilt of the sacrum in standing. With a decreased anterior stabilising force of transvers abdominus, the L5 vertebra

has a tendency to slip anterior on the sacrum and the L4 has a lesser tendency to slide anteriorly on the L5 motion segment. When the abdominal muscles are weak, particularly the transvers abdominus muscle, the lumbar lordosis increases and also the strain on the L4-S1 segments (Kapandji, 2008; Middleditch & Oliver, 2005).

Mobility at the lumbosacral joint is limited by the iliolumbar ligaments, which unite the L4 and L5 lumbar vertebrae directly to the ilium. The superior band of the iliolumbar ligament between the tip of the L4 transvers process and the iliac crest and the inferior band of the iliolumbar ligament runs between the tip of the lower border of the transvers process of L5 and the iliac crest to insert anterio-medially to the superior band. Thus, the two iliolumbar ligaments stabilise the L4-S1 motion segments during movement (Kapandji, 2005; Bogduk, 2011). The implication of this stabilisation of the L4 and L5 motion segments by the superior and inferior bands of the iliolumbar ligaments is that one can logically assume that during lumbar flexion most strain will occur between L3 and L4. However, the L3 and L4 motion segments are stabilised by the lumbar fibres of the longissimus longus that run from the ilium to the transvers process of L3 and the fibres of the interspinalis thoracis muscles from the thoracic spine and insert on the spinous process of L3.

The clinical implication of this mechanism is that the L3 vertebra is the first true mobile lumbar segment above the stabile L4-L5 and is therefore vulnerable to being pulled posteriorly by the longissimus longus as well as the fibres of the interspinalis thoracis (Kapandji, 2008). The largest amount of intersegmental movement takes place between L4 and L5 while the greatest amount of variability occurs at the L5-S1 level (Middleditch & Oliver, 2005).

It is important to realise also that during a change in the person's basic position; i.e. from standing to unsupported sitting with the hips and knees in 90^{o} flexion, the mobile lumbar spine is flexed into a similar position as during maximal lumbar flexion in standing. In the unsupported sitting, 60% of flexion occurs at the hip joint and 30% of flexion occurs in the lumbar spine. Of this 30% lumbar flexion that occurs, 80% to 90% (of the lumbar flexion) takes place at L4 and L5. This allows one to conclude

that the largest amount of intersegmental flexion occurs at L4-L5 (Middleditch & Oliver, 2005).

During a prolonged standing position, the impacted joints at each level of the spine bear an average axial load of 16%. In people with a lumbar lordosis, the lumbar joints between L3 and S1 bear approximately 19% of the axial load (Middleditch & Oliver, 2005). However, from a sagittal view, the apophyseal joints do not impact on each other but rather slide upon each other because the joint surfaces run more parallel to each other. Because of this sliding action, they may not play a role in load-bearing (Twomey & Taylor, 1987).

In the upright posture of humans, especially, the lower segments of the lumbar spinal column are exposed to considerable pressure. The weight of all body parts above these lumbar segments is borne on a small area of only a few square centimetres. Shifting of the trunk out of midline increases pressure on the lumbar segments considerably. As much as 70% of all movements in flexion and extension of the entire spinal column takes place in these segments (Middleditch & Oliver, 2005; Kapandji, 2005).

In clinical practice, patients with CNSLBP treated by the researcher commonly present with L4-S1 dysfunction, although involvement of L3 is not always excluded. Based on the discussion in the preceding paragraphs, the researcher argues that because the largest amount of intersegmental movement takes place between L4 and L5 because the greatest amount of variability occurs at the L5-S1 level, these segments are most vulnerable to any abnormal loading forces (Kapandji, 2008; Middleditch & Oliver, 2005; Moore, Dally & Agur, 2006).

The clinical implication of the discussion is that the lumbar area should always be assessed and treated by taking into consideration the alignment of the thoracic and cervical curves and the position, control and range of movement of the scapulae and gleno-humeral joints as well as the pelvis and hip complex.

3.2.2 The muscle system of the ISMS

Similar to the spinal ligaments that provide local and global stability to the intervertebral column, the muscular system of the trunk also provides segmental, local and global stability to the trunk (intervertebral column) (Comerford, Mottram and Gibbens, 2008).

The intersegmental and multisegmental muscle attachments shown in Figure 3.2(a) and 3.2(b) illustrate the complex and integrated connectivity of the posterior spinal muscle system as a holistic muscle system.

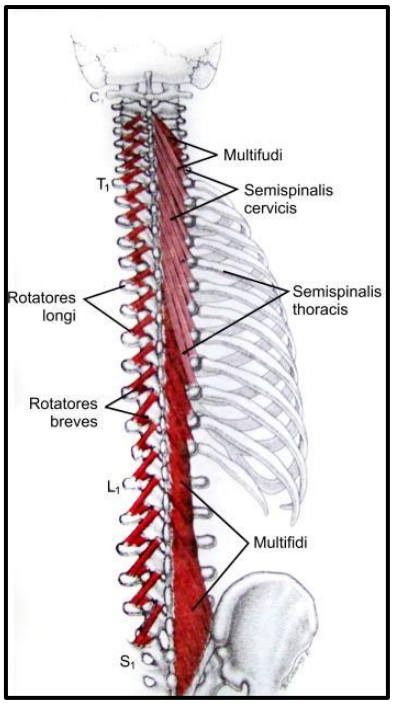


Figure 3.2(a): Intersegmental posterior spinal muscles

(Travell & Simons, 1983)

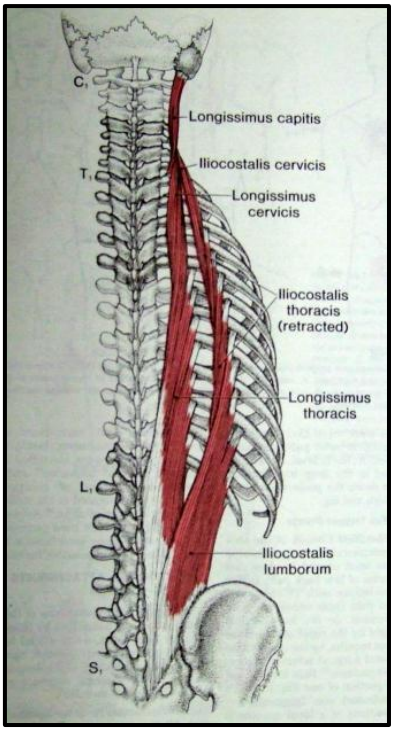


Figure 3.2(b): Multisegmental posterior spinal muscles

(Travell & Simons, 1983)

(i) Core muscles of the trunk

Richardson *et al*. (1999) categorise the trunk muscles into 'inner core muscles' and 'outer core muscles' of the spine. The 'core' also includes the neck and head as well as the shoulder girdle and pelvic girdle. This statement emphasises the interconnectedness of the systems of the ISMS.

The inner core muscles of the trunk are described by Comerford *et al*. (2008) as the diaphragm, posterior psoas, transvers abdominus, segmental multifidus and the pelvic floor. These muscles originate and insert segmentally on the lumbar vertebrae, control the spinal curves, maintain the mechanical stiffness of the spine by controlling intersegmental motion, and respond to changes in posture and to changes in low extrinsic load (Comerford *et al*., 2008). These are the local stabilising muscles.

The outer core muscles are divided into global trunk stabilisers and global mobilisers. The global trunk stabilisers are described as the obliquae abdominals, superficial multifidus and spinalis, anterior psoas, and oblique fibres of quadratus lumborum. The global trunk mobilisers are described as the rectus abdominus, longissimus, iliocostalis, lateral fibres of quadratus lumborum, semispinalis and latissimus dorsi (Comerford *et al*., 2008).

Table 3.1: The global stabilisers and mobilisers of the neck and limb girdles (Comerford & Mottram, 2012)

Description	Global stabilisers	Global mobilisers
Neck	Upper cervical cuff, Longus Colli Semispinalis	Sterno-cleido-mastoid Scalenes Splenius
Pelvic girdle (Pelvis)	Gluteus Maximus, Medius and Minimum Iliacus Pectinius Adductor Brevis	Piriformis Hamstrings Ilio-tibial Band and Tensor Fascia Latae Ilio-tibial Band and superior Gluteus Maximus Rectus Femoris
Shoulder girdle (Scapula)	Trapezius Serratus Anterior Gleno-humeral rotator cuff	Levator Scapula Rhomboids Pectorialis Minor Latissimus Dorsi

(ii) Local and global muscle roles

The local (stabilising) muscles are responsible for increasing the segmental stability of the spine, decreasing excessive intersegmental motion and maintaining muscle control (stabilisation) of the spine during low load tasks and activities They contract regardless of the direction of loading or movement that is taking place and are prone to be activated during low load functioning. During their functioning, the local muscles do not significantly change length but work mostly isometrically and therefore do not primarily contribute to range of motion. During functional activities, they maintain their stabilising function during all ranges of motion.

Global stabilising and mobilising muscles (Table 3.1) are responsible for the production of movement. They control movements that require high physiological load and therefore work against the direction of the loading. These muscles contract, depending on the demands of the environment/task at hand and the load. The global muscles change length significantly (concentric and eccentric muscle contraction) and therefore are the muscles that control the range of motion of a joint or series of motion segments in the case of the spinal column. These global muscles may have a primary global trunk stability or trunk mobility role during activities of daily living.

For normal efficient functional activity both the local and global muscles should work in an integrated way. Neither the local nor the global muscle ‘systems’ can control functional stability during body movement that demands trunk mobility. Trunk stability is controlled by the local stabilisers. The global muscles influence a patient’s postural alignment and contribute to the production and control of range of motion of the spine (trunk).

(iii) Diaphragms attached to the spine

At each junction of the spinal curves C7/T1, T12/L1 and L5/S1 there are three very important circular muscle planes: the cervical or thoracic inlet, the respiratory diaphragm and the pelvic diaphragm. These circular muscle planes function as transverse diaphragms to separate the three chambers of the trunk and permit transmission of vital structures such as vascular and neural structures. These

diaphragms can be areas of major dysfunction and mechanical stress due to their insertion on the spinal vertebrae at the appropriate levels.

The biomechanics of breathing is of vital importance to spinal functioning due its influence primarily on the thoracic area and the cervical and lumbar spinal regions (Kapandji, 2008).

The respiratory diaphragm as the main inspiratory muscle is a musculotendonous dome that attaches to the lower thoracic outlet and separates the thorax from the abdomen. Posteriorly the dome specifically attaches to the deep surfaces of the costal cartilages, the costal arches, the tips of the eleventh and twelfth ribs and the T12-L1 vertebral bodies. The elevation of the ribs by the inspiratory muscles and the depression of the ribs and sternum by the expiratory muscles require mobility of the costovertebral, costochondral and costosternal joints. Any dysfunction that can affect the mobility of these joints will have an effect on the person's breathing. These joints can also be a major source of nociception, which can refer pain to the lumbar spine and/or restrict the patient's breathing pattern.

Other inspiratory muscles are the accessory inspiratory muscles; for instance, the sternocleidomastoid, scalenes, pectoralis major and minor, and the lower fibres of serratus anterior and latissimus dorsi, serratus posterior superior and the iliocostalis thoracis. All these muscles are prone to developing trigger points and can refer pain. Overuse of the accessory muscles results in elevation of the shoulder girdles, which can affect the alignment of the cervical and thoracic spinal curves, which in turn will influence the lumbar curve.

The primary expiratory muscles such as the internal intercostals and the accessory expiratory muscles are involved in forced expiration (valsalva manoeuvre). These muscles are the rectus abdominus, external oblique and internal oblique muscles and they strongly depress the abdominal outlet. Other accessory expiratory muscles are the iliocostalis thoracic, longissimus, serratus posterior inferior and the quadratus lumborum.

Of clinical importance is the fact that muscle imbalance, trigger points in these muscles and dysfunction of the costochondral, costovertebral and costo sternal joints can affect not only breathing but also spinal dysfunction.

Further, the interaction between the stabilisation of the spine by the local and global trunk muscles (in which abdominal muscle control plays an important role), the pelvic floor and the respiratory diaphragm is of vital importance for optimal alignment of the spine as a closed kinematic chain.

During functional activities the forces generated in the local and global trunk muscles are transmitted across the cervical spine to the head and vice versa and the shoulder and pelvic girdles.
So in the context of the biomechanical part of the ISMS, postural adjustments of the trunk include the head, spine, shoulder girdle and pelvic girdle control.

(iv) The head as part of the ISMS

The weight and position of the head have a profound influence on the alignment of the cervical spine. Stability and mobility of the cervical spine are of utmost importance because the spine has to move the head in space on a relative stabile base. Owing to the interaction of the vestibular ocular reflex, the position of the head is usually vertical (eyes are horizontal) and the cervical spine adapts continuously to maintain the head's vertical position. Clinically, it is observed that the junction between the mobile cervical spine and the stabile thoracic spine places the transition vertebrae at the cervico-thoracic junction under great strain (Middleditch & Oliver, 2005).

The design of the musculature of the cervical spine is specialised to move the 'head on the cervical spine' ('head on neck'), to move the 'cervical spine' as such and to move the 'head with the cervical spine' (Middleditch & Oliver, 2005). As the head's stability and movement depend on the cervical spine, it is seen as part of the functioning of the ISMS.

Muscles anterior to the cervical area include muscles that 'flex the neck' (longus colli, sternocleidomastoid and scalenus anterior), muscles that 'flex the neck and head' (sternocleidomastoid and longus capitis) and the muscle that flexes the 'head on the neck' (rectus capitis anterior).

Muscles that laterally flex the neck include the scalenus anterior, scalenus medius, scalenus posterior, splenius cervices, levator scapulae and sternocleidomastoid. Muscles that laterally flex the head and the neck include the sternocleidomastoid, splenius capitis, trapezius, and erector spinae. The muscle that laterally flexes the head on the neck is the rectus capitis lateralis.

In similar fashion the muscles that 'extend the neck' are the levator scapulae and splenius cervicis; the muscles that 'extend the head and neck' are the trapezius, splenius, and erector spinae; the muscles that 'extend the head on the neck' are the rectus capitis posterior major, rectus capitis posterior minor, and obliquus capitis superior (the last-mentioned three muscles are also called the suboccipital muscles).

The muscles that rotate the neck are the semispinalis cervices, multifidus, scalenes anterior and splenius cervicis; the muscles that rotate the neck and the head are the sternocleidomastoid and splenius capitis; the muscles that rotate the head on the neck are the obliquus capitis inferior and rectus capitis posterior major.

An important clinical implication is that the trapezius upper fibres and levator scapulae elevate the scapulae. Longstanding elevation of the scapulae due to prolonged periods of poor sitting posture is associated with an increase in the thoracic kyphosis and cervical extension. The change in both these curves will flatten the lumbar lordosis and put strain on the thoracolumbar fascia (TLF) and result in abnormal muscle activation.

(v) The shoulder girdle as part of the ISMS

The position of the shoulder girdle (scapulae, clavicle and gleno-humeral joints) strongly influences the alignment of the cervical and the thoracic regions of the spine owing to its muscle attachments to the spine.

The medial borders of the scapulae can be in adduction (associated with a flat thoracic curve) or in abduction (associated with an increased thoracic kyphosis). The stabilisation of the scapulae is controlled by the trapezius upper, middle and lower fibres, the rhomboid major and minor, latissimus dorsi, levator scapulae, and serratus anterior. The global mobilisers of the shoulder girdle are the levator scapula, rhomboids, pectoralis minor, and latissimus dorsi muscles.

The rotator cuff, together with the deltoid muscle and supraspinatus, controls all six degrees of freedom of the gleno-humeral joint but needs a dynamic but stabile scapula to control the full range of motion of the gleno-humeral joint.

With an increased thoracic kyphosis and winging of the scapulae (weak rhomboid major and minor as well as serratus anterior and middle and lower trapezius muscles) the rotator cuff muscles tend to shorten and the patient's posture becomes one of an elevated abducted scapulae and anterior (medially) rotated gleno-humeral joints, associated with an increased cervical and lumbar lordosis or a lumbar kyphosis. The upper trapezius and levator scapulae contribute to the increased cervical lordosis with shortening. Simultaneously the anterior pectoralis major and minor shorten and also contribute to scapulae abduction. This muscle imbalance results in a generally 'poor posture' typical of a sedentary lifestyle and/or of a person working in a prolonged poor sitting posture (Middleditch & Oliver, 2005).

When latissimus dorsi is shortened by unilateral muscle spasm and/or as part of stiffening (due to guarded movement) it can create anterior shoulder pain at its insertion on the intertubercular groove of the humerus. When the latissimus dorsi performs its action of adduction, medial rotation and extension, it contributes towards the abduction of the scapulae. This is because it pulls the inferior angle of the scapula into abduction (one of the origins of the muscle) when it contracts and as such contributes to an increased thoracic kyphosis.

Of clinical significance is the fact that the latissimus dorsi inserts on the intertubercular sulcus of the humeral head shoulder joint of the spinous process of

the T7-T12 vertebrae, the ribs, and through the TLF, which attach to the spinous processes of all the lumbar and sacral vertebrae, supraspinous ligaments and iliac crests. It plays an enormous role in controlling a person's posture and through the TLF plays an enormous role in lumbar pain and dysfunction. It is innervated by the thoraco-dorsal nerve (C6-C8). As such it can become involved with lower cervical pathology (Middleditch & Oliver, 2005).

Another muscle of clinical significance is the trapezius muscle, which attaches the occipit, the scapulae (shoulder girdle) and thoracic vertebrae down to the T12 spinous process. It receives the motor innervated from the eleventh cranial nerve and sensory branches from the ventral rami of C3-C4 (Middleditch & Oliver, 2005). The position of the shoulder girdle in relation to the spine is therefore greatly influenced by the directional forces or lack of these forces exercised by the latissimus dorsi and trapezius muscles.

The anatomy and function of the latissimus dorsi and trapezius muscles reveal that the head, cervical thoracic and lumbar spinal regions are intimately connected to the shoulder girdle, trunk and pelvis. This is of significance in the clinical application of manual therapy for CNSLBP.

(vi) The pelvis as part of the ISMS

Because the lumbar spine articulates directly with the sacrum, any movement (anterior, posterior or lateral tilt) of the pelvis will affect the (depth/angulation) lumbar lordosis. A posterior tilt flattens the lumbar curve, an anterior tilt increases the lumbar lordosis and a lateral tilt results in lumbar sideflexion. The muscles that control the anterior-posterior pelvic tilt are the erector spinae and psoas major, rectus abdominus, oblique internus and externus, gluteus maximus, and the hamstrings (semitendonosus, semimembranosus and biceps femoris) (Middleditch & Oliver, 2005).

The muscles acting as mobilisers of the pelvis on the hip and secondarily on the lumbar spine are piriformis, hamstrings, ilio-tibial band and tensor fascia latae, superior gluteus maximus and rectus femoris because they control the tilting of the

pelvis on the hip joint and therefore in nutation and counter-nutation (Middleditch & Oliver, 2005; Kapandji, 2008; Comerford & Mottram, 2008). The control of nutation and counter-nutation takes place through the interaction among the abdominal, lumbar-thoracic and the pelvis-hip stabilisers and mobilisers. One can therefore talk about the 'spine-pelvis-hip complex' (Middleditch & Oliver, 2005).

Muscle imbalance and motor recruitment result in muscle stiffness and as such the 'spine-pelvis-hip complex' during physiological movements of the lower limb and functional activities such as gait. Boyling *et al*. (2004) state that control of pelvis stability and balance is essential for pain-free functioning of the spine.

The clinical implication of this interaction among the spine and head; spine and shoulder girdle and upper limbs; and spine and pelvis and lower limbs is that manual therapy for the lumbar spine should include the release of soft tissues and/or muscle recruitment around the shoulder girdle and gleno-humeral joints, pelvis and hip joints as distal as the hamstring muscles and tensor fascia latae (Middleditch & Oliver, 2005).

3.2.3 The neural components of the spine

Under this heading a brief explanation of the link between the gross anatomy of the nervous system – peripheral (PNS), central (CNS) and autonomic nervous systems (ANS) – is given. The aim of providing this explanation is to indicate how interlinked the neural system related to the motion segment is in terms of the mechanical and physiological responses that can occur at the level of the motion segment. The mechanical and physiological neural components at the level of each motion segment are also related to each other as well as to the brain and the filum terminale in the sacrum.

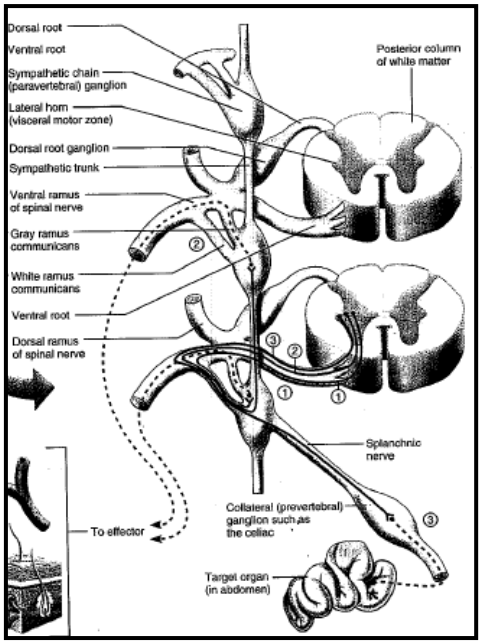
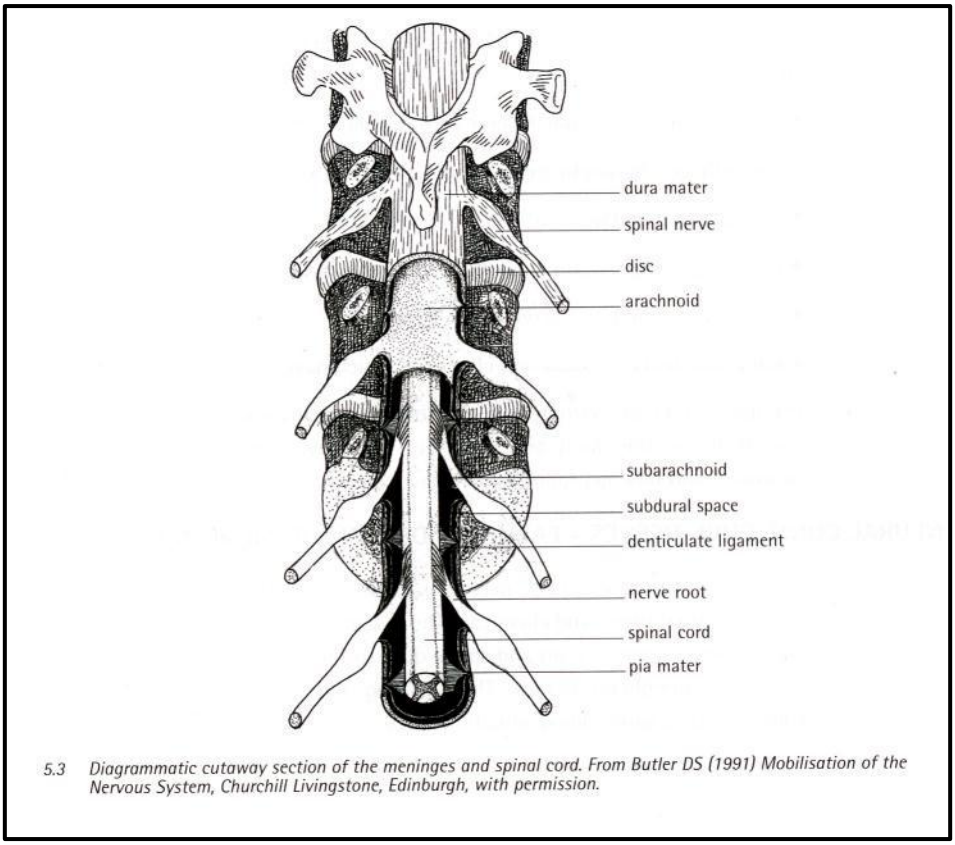


Figure 3.3: The interconnectedness between the spinal cord, the dorsal and ventral rami of the spinal nerve and the sympathetic chain adjacent to the vertebral column

(Marieb, 2004 p 539)

Understanding the mechanical unity among the different parts of the total nervous system is central to understanding the effect of spinal torsion that may occur during the development of ISMS dysfunction pain processing. In Figure 3.3 one can see the interconnectedness between the three nervous systems: the PNS, ANS and CNS, at the level of a motion segment. Each of these systems is a continuum in its own right.

Each of the peripheral nerves/nerve root trunks and autonomic sympathetic chain is ensheathed by fascia. (The dura, arachnoid and pia mater (the meninges) are all part of the connective tissue (fascia) system of the body.) The nerve root complex consists of the root sleeve; the motor and ventral roots; the dorsal root ganglion, which is highly vascularised; and the spinal sleeve. The epineurium in general is continuous with the dura mater while a few layers of the peripheral nerve's epineurium and the endoneurium are continuous with the pia mater.



5.3 *Diagrammatic cutaway section of the meninges and spinal cord. From Butler DS (1991) Mobilisation of the Nervous System, Churchill Livingstone, Edinburgh, with permission.*

Figure 3.4: Diagrammatic sketch of the (specialised) connective tissues around the spinal cord and nerve roots

(Butler, 2000 p 102)

Because of this continuous tract (interconnected systems), any change in part of the system will have repercussions on the whole system. A major characteristic of the neural system is that it should be mobile enough to move as part of the trunk and limbs. It should therefore be able to glide and stretch in relation to the surrounding tissues. Stiffening of fascia or mechanical ‘tightness’ in the neural fascia can be detected during neurodynamic tests by putting the mechanical interface between the neural structures and the surrounding tissues in a stretched position.

The neuraxis is a closed semihydraulic system that houses the cerebrospinal fluid (CSF) and is vital to the nourishment of the neuraxis. The nerve roots, spinal cord and meninges (neuraxis) in the normal spine adapt freely to changes of spinal movement, posture and loading. As a result of the cephalad and caudad attachments of the dura to the spine, the neuraxis has to change in shape or position

to accommodate and adapt to changes in posture and has to adapt to loading (Middleditch & Oliver, 2005; Brieg, 1978). These changes in the neuraxis entail changes in length, diameter, shape and direction during movement and occur because the collagen network of the pia mater has a rhomboid nature, which offers the neuraxis its mobility when deformed by movement. The extensibility of the filum terminale allows it to elongate in a linear fashion when it is stretched in a caudad direction. The changes in the neuraxis and meninges that take place during movement can entail ventral displacement, lengthening, axial sliding and angulation of the nerve roots (Middleditch & Oliver, 2005). During flexion of the spine, the cervical and lumbar regions can increase up to 28 mm each while the thoracic region only increases about 3 mm in length.

The increased tension in the neuraxis and meninges during flexion of the spine is caused by the fact that it attaches cranially to the foramen magnum, which causes rostral pulling on the pons and medulla, and caudally it attaches to the sacrum. In the cervical region the greatest strain occurs at the level of C5-C6 while in the lumbar spine the area of greatest strain occurs on the level of L5-S1. During flexion, caudal sliding of 3 mm in the dura at the levels of C6 and L4 occurs. At the level of L5 rostral displacement of about 3 mm takes place while at the level of T5 rostral movement occurs above T5 and caudal movement occurs below the T5 level. The ability to undergo longitudinal extensibility protects the neuraxis from positions of high tension and from positions of low tension. The neuraxis is relaxed in the neutral position, which is midway between flexion and extension (Middleditch & Oliver, 2005).

The change in dural length is smaller than the change in the bony canal during flexion (Breig, 1978). The clinical implication of this fact is that any change in the curves of the spinal canal will necessitate adaptability of the cord and its dural covering. Depending on where the adaptation has to occur, interfacing between the cord/dural sac and the bony canal occurs. With torsioning of the canal at any level, the appropriate nerve roots will be torsioned accordingly. The torsioning can create changes in alignment of the intervertebral canal and these changes are therefore

possible areas of interfacing of the nerve root sleeve. Central interfacing and lateral interfacing are both potent sources of nociception.

Another factor that contributes to the stability of transitional areas ~ C6-T3 and ~ T10-L2 is the fact that the plexuses originate in these areas from the spinal cord. The spinal canal also narrows slightly in these areas, which influences the mobility of the spine (Middleditch & Oliver, 2005).

The continuum between the three aspects of the nervous system (PNS, ANS and CNS) is the key concept in neurodynamics. The study of neurodynamics of the nervous system is based on the interconnectedness between these three systems because it can reveal the site of restrictions between the nervous system and any other surrounding somatic tissues, in particular the different types of fascia. Fascial restrictions are the most likely source of soft-tissue restriction, pain and dysfunction of trunk and limb movement revealed through adverse neural tension, which can restrict movement.

Neurodynamics indicates adverse neural tension, in particular of the PNS, which is revealed by interfaces frictioning because of a lack of glide or restriction of movement at interfaces due to fibrosis or points of tension within the system.

Restriction of soft tissue surrounding pain-sensitive structures, such as the nerve root sleeve (the classic example) at the local lumbar motion segment, can occur as a result of pathology that develops from strain typically at the L4-S1 motion segments. Structures that are typically strained are the synovium of the facet joint capsules or ligamentous structure of the annular disc or any of the insertions of the muscle (multifidi) local ligaments (for example, anterior or posterior sacro-tuberous ligaments or ligamentum flavum). Healing by fibrous tissue and shortening due to local spasm enhance this restriction and pain (Butler, 2000).

The clinical implication of the neural system as integral part of the ISMS is given at the end of Section 3.2.4.

3.2.4 The connective tissues in the trunk

Apart from the interconnectedness of the systems mentioned in the preceding paragraphs, the muscular, skeletal and ligamentous systems as well as the neural systems are infiltrated and surrounded by organised specialised and loose unspecialised connective tissue (Langevin & Sherman, 2006). Langevin and Sherman (2006) list 14 specific terms that are used to describe fascia but which also indicate specific aspects of connective tissue. These are 'dense connective tissue', 'areolar connective tissue', 'superficial fascia', 'deep fascia', 'intermuscular septa', 'interosseal membrane', 'periost', 'neurovascular tract', 'intra- and extramuscular aponeurosis', 'epimysium', 'perimysium', 'endomysium' and the 'dura'.

The function of fascia is to link all body systems and give structure and support to all anatomical structures of the body, protect and separate specialised organs and contribute to cellular respiration, elimination of toxins, metabolism and fluid and lymphatic flow. Fascia contributes to the energy storage in the body, acts as a passive force transmission and is a major source of proprioceptive feedback to the CNS in the control of movement through the mechanoreceptors found in the fascia (Cantu & Grodin, 2001). Trauma or malfunction of the fascia can result in poor cellular efficiency, necrosis, disease, pain and dysfunction throughout the body because it restricts the environment in which other structures function.

Langevin and Sherman (2006) confirm the fascial network as a continuous web throughout the whole body. The implication of this continuous web of connective tissue is that stiffness of the fascia at one end of the system will cause pain and dysfunction at a distant point in the system.

Connective tissue is an integral part of each muscle, which is classically organised into (1) the epimysium, which encloses the muscle; (2) the perimysium, which binds groups of muscle fibres into fascicles (bundles); and (3) the endomysium, which ensheathes individual muscle fibres (Middleditch & Oliver, 2005). The organised fascial layers in intra-muscular tissue (epimysium, perimysium and endomysium) are classified as part of the deep fascial layers (Findley, Chaudhry, Stecco & Roman, 2012).

Specialised as well as unspecialised loose connective tissue also surrounds and is an integral part of the PNS, ANS and CNS, making up on average 50% of these systems (Coppieters & Nee, 2012; Langevin & Sherman, 2006). The fascial network acts as a passive force transmission and is a major source of proprioceptive feedback through the mechanoreceptors found in fascia. to the CNS in the control of movement (Cantu & Grodin, 2001).

A specialised fascial structure, the TLF, plays an important role in the functional stability of the lumbar-thoracic area, the development of ISMS dysfunction and, as such, CNSLBP. The TLF is a complex fascial structure and consists of posterior, middle and anterior layers of dense connective tissue that are also separated from each other by loose areolar connective tissue that allow the adjacent layers to glide past each other (Langevin, 2011; Middleditch & Oliver, 2005). The posterior layer is also called the superficial layer of the TLF.

Most of the fibres of the posterior TLF derive from the aponeurosis of the latissimus dorsi and attach superior (cranial) to L4 to the supraspinous ligaments. Inferior to L4, the superficial laminae of the posterior TLF attach to the sacrum, posterior superior liac spine of the iliac crest on the contralateral side. The superficial part of the posterior TLF blends with the fascia of the gluteus maximus muscle and superolaterally with the fascia of the contralateral latissimus dorsi. Because these two muscles are mechanically linked through the TLF they increase the tension in the posterior layer of the TLF when they contract or are in spasm (Middleditch & Oliver, 2005).

The deep layer of the posterior TLF medially to the interspinous ligaments, posterior superior iliac spine, iliac crest and long dorsal sacroiliac ligament and some fibres attach to the deep fascia of the erector spinae. The deep laminae also attach to the lateral raphae and as such are indirectly linked to the internal oblique and transversus abdominus muscles. At the sacral level the superficial and deep layers of the TLF blend together.

The middle layer of the TLF attaches medially to the lumbar transvers processes and is continuous with the intertransvers ligaments. Laterally it gives rise to the aponeurosis of the transversus abdominus muscle. It lies posterior to the quadratus lumborum muscle and compartmentalises the erector spinae muscles (Kapandji, 2008).

The anterior TLF layer attaches medially to the lumbar transvers processes and the intertransvers ligaments. Inferiorly it attaches to the iliolumbar ligament and adjacent iliac crest. Superiorly it forms the lateral arcuate ligament. It covers the anterior surface of the quadratus lumborum and laterally it blends into the other layers of the TLF.

The visible areas of superficial and deeper fascia on the posterior spine are shown in Figure 3.6(a) and 3.6(b)

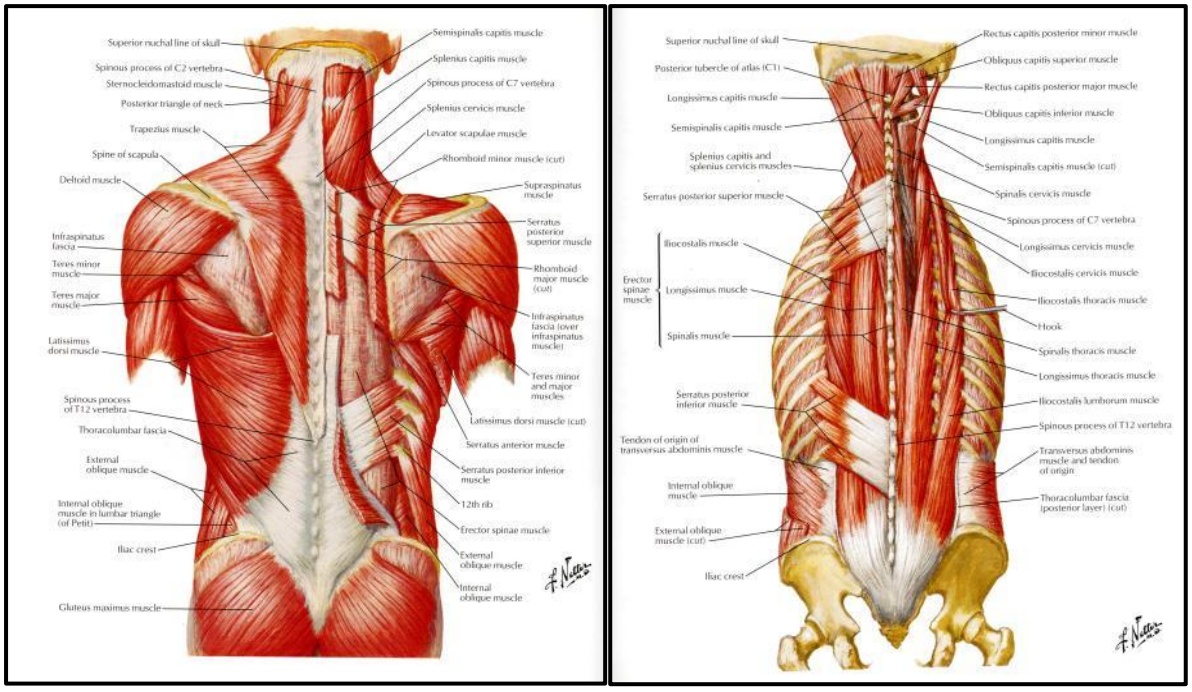


Figure 3.5(a) and 3.5(b): The visible areas of superficial and deeper fascia on the posterior spine

(Netter, 2011 plates 168 and 169)

Fascia is densely innervated by myelinated sensory nerve endings, which are assumed to serve a proprioceptive function. These include Pacini (and paciniform) corpuscles, Golgi tendon organs and Ruffini endings (Stecco *et al*., 2010). In addition

they are innervated by free endings (nociceptors). When including periosteal, endomysial and perimysial tissues as part of a body-wide interconnected network, the fascial network can be seen as the human body's largest sensory organ, particularly proprioceptive sensation (Scleip, 2003).

Of clinical implication is the fact that the CNS, PNS and ANS are interconnected at segmental and global levels throughout the spine and continuous with the brain. That connective tissue is an integral part of the nervous system might relate to the fact that shortening/stiffening of the fascia within and around the total nervous system can limit neural mobility and be a powerful source of nociception. As the fascial system in the human body is a global network, stiffening of the fascia in the limbs may not only affect the mobility of the ISMS but also refer pain to the lumbar spine.

Any imbalance or shortening of the muscles that insert onto the fascial sheet of the TLF will probably result in an uneven pull on the TLF, causing pain in the lumbar area because it is so rich in proprioception and nociception (Stecco, 2010). Imbalance/dysfunction in the TLF will not only affect the lumbar area but, if shortened, will affect the fascial running up in the spine and cause headache.

3.3 Postural control of the ISMS

Posture and coordinated movement are the result of integration between the multiple inputs from spinal, medullary, midbrain and cortical levels (Horak & Macpherson, 2006). The result of the input from multiple levels is: (1) voluntary activity; (2) adjustment of the body (posture) to provide a stable background for movement; and (3) coordination of various muscle activities so that smooth and accurate movement can take place. Horak and Macpherson (2006), Shumway-Cook and Woollacott (2007) and Ganong (2003) describe postural control as the ability to control a stabile position of the body in space while maintaining an appropriate relationship between the body segments and between the body and the environment relevant to the task that the person is performing.

The ability to maintain stability in a standing posture is, according to Levangie and Norkin (2005), a learned skill based on the sensory impulses that the brain receives

from the skeletal, articular system; the sensory systems; and the muscular (myofascial), fascial system; and also cognitive input or higher centre input.

The continuous integrated activity of the posture-regulating systems results in continuous adjustment of posture before and during movement. These posture-regulating systems (Shumway-Cook & Woollacott, 2007) include the integration of the musculoskeletal components, the neuromuscular synergies, individual sensory systems, sensory strategies, anticipatory mechanisms, adaptive mechanisms, the respiratory system and internal representation.

Impaired postural control can result in biomechanical malalignment and dysfunction and, as such, cause mechanical pathology (wear and tear on joints). This implies that musculoskeletal pain syndromes are seldom caused by isolated events and that movement systems should be assessed holistically in the context of the person's psycho-social influences as well. Habitual movements and sustained postures also play a role in the development of dysfunction (Sahrmann, 2002).

A painful stimulus originating from the musculoskeletal system stimulates the appropriate nociceptors of the sensory system and results in a particular sensory strategy conducted by the nervous system and integrated with the higher centre control. Through the neuromuscular system the person's muscle recruitment changes and movement patterns adapt to avoid the painful stimuli or to 'hold the body still' (fixation) to avoid the painful stimulus. This response in a person to adapt their movement will affect their adaptive control (balance) and anticipatory control. The person's breathing control or breathing pattern will change if the fixation affects the trunk muscle control (abdominal and erector spinae cocontraction). If the breathing pattern is limited due to the fixation it might lead to a decrease in oxygenation and to tiredness. The fixation of the trunk muscles will also lead to an increase in the muscle metabolism and a feeling of tiredness. Other internal organs such as the heart and blood pressure might also be affected by the adapted postural control. The adapted postural control will probably cause the person to decrease their general level of activities of daily living (ADL) and adapt their lifestyle (if their lifestyle has not been adapted already before the painful incident).

The ISMS is graphically displayed in Figure 3.6.

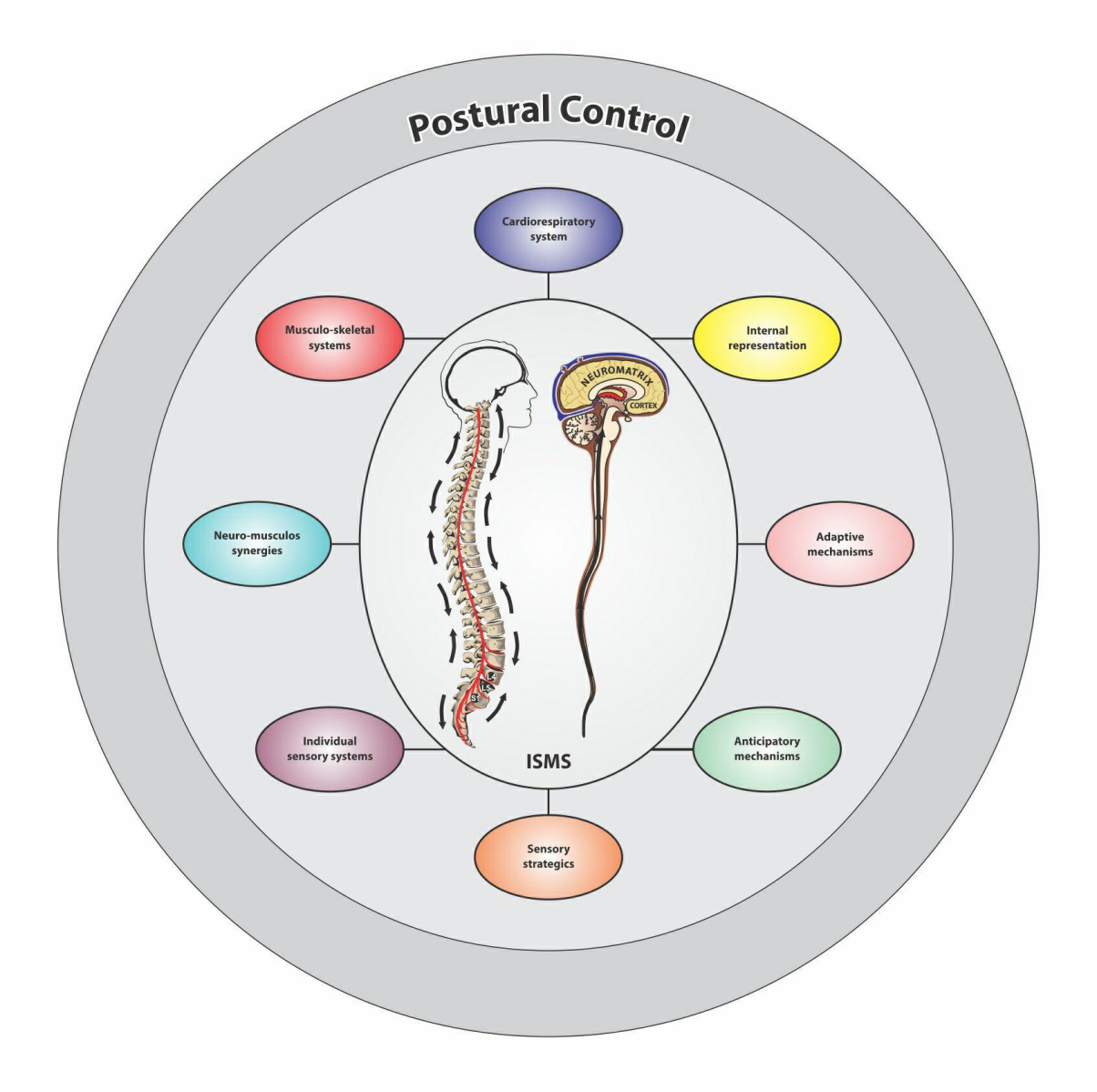


Figure 3.6: The integrated spinal movement system

The functioning of the ISMS at the local and global levels is influenced by the patient's genetic inheritance of their morphology (body build) (Porterfield & DeRosa, 1992), which determines the integrity of the neuromusculoskeletal tissues and of the process of degeneration that may be present in the zygapophyseal joints and intervertebral discs. Because movement is strongly influenced by sensory input, nociceptive stimuli will affect the functioning of the ISMS. The influence of nociceptive stimuli on the ISMS is discussed in Section 3.4.

Characteristics of normal ISMS functioning

- The spine consists of 24 motion segments (local segments) but functions as a (global) multisegmental kinetic chain.

- The ligamentous system that stabilises the multisegmental system is arranged to stabilise the local motion segments as well as the global multisegmental kinetic chain.

- The muscular system is arranged to stabilise and mobilise the multisegmental system at the level of the local motion segment as well as the global multisegmental kinetic chain.

- The connective tissue, which is an integral part of every system and in particular the TLF in the lumbar spine, is continuous throughout the spine.

- The neural system controls the musculoskeletal system by its peripheral, autonomic and central innervation of all structures. The sympathetic ANS prepares the body for 'fright, flight and fight' and the parasympathetic ANS assists the body in conserving the energy resources of the body and is the effector for visceral motor systems.

- Anatomically the PNS, ANS and CNS form an interconnected continuous network from the periphery to the brain.

- The shoulder girdles, pelvic girdle and head structurally and functionally influence the spinal alignment and, as such, the functioning of the multisegmental kinetic chain. The shoulder girdles, pelvic girdle and head therefore are an integral part of the ISMS.

- The position of the thoracic spine influences the ribcage and, as such, the patient's breathing pattern and volume.

3.4 Patho-physiological responses underlying the development of ISMS dysfunction

The pathophysiological responses of muscle spasm, overuse and disuse and trigger points that occur in muscular tissue, connective tissue stiffening, and the effect of nervous tension on muscle tissue are processes integrated in the development of ISMS dysfunction. These processes accumulatively over time contribute to the clinical picture of CNSLBP.

For the sake of systematic discussion on the development of ISMS dysfunction, an overview of these processes is given based on the synthesis of the researcher's clinical experience and an overview of the relevant literature.

3.4.1 The effect of muscle spasm, overuse and disuse on muscular tissue

Muscle spasm is a response to injury to the muscle itself or a reflex response to nociceptor irritation of joints and associated tissues. It is a protective mechanism to splint a painful lesion and may be present segmentally or span a whole spinal region. If short-lived, muscle spasm may be asymptomatic. Once it is present, spasm increases the compressive forces on the structures of the motion segment, in particular the intervertebral disc, which results in greater pain and dysfunction. Muscle spasm can also cause pain through the tension on its attachment to the periosteum.

Muscle spasm might be the primary mechanism in referred pain arising from the spinal segment. Persisting muscle spasm results in decreased blood flow to the muscle, leading to anoxia and accumulation of metabolic waste products in the muscle, which would normally be dispersed during relaxation. With muscle fatigue, pain may occur within the muscle and internal changes in the muscle occur, leading to a contracture (Middleditch & Oliver, 2005).

Overuse or hypertrophy is the result of excessive use of a muscle where lack of use (disuse) leads to atrophy. Excessive use of a particular muscle or altered motor recruitment, which results in the habitual activation of certain muscle groups within

small and abnormally restricted amplitude of their available extensibility ranges, can lead to a state of overactivation in these muscles and eventually they shorten. The antagonists of the shortened muscles respond by inhibition, weakness and lengthening (Janda, 1996). Postural muscles shorten or tighten while their antagonistic phasic muscles become weakened and tend to lengthen.

Muscle atrophy as a result of disuse clinically presents as a decrease in the cross-section area (CSA) of the muscle. Other clinical signs of muscle atrophy are decreased muscle strength and endurance. Reduced muscular support will increase the load on the joints and lead to abnormal movement patterns. A decrease in the CSA of lumbar multifidus has been demonstrated in both acute and chronic LBP pathology (Hides, Richardson & Jull, 1996;). Changes have also been reported in the CSA of the psoas muscle in a patient with chronic LBP (Cooper, St Clair Forbes & Jayson, 1992).

3.4.2 The development of trigger points

Trigger points are sustained contractions (previously known as fibrositis) of isolated groups of muscle fibres. The most likely aetiology of trigger points, according to Middleditch and Oliver (2005), is irritation of the nerve supply to the muscle, which causes localised muscle spasm or a reflex response to irritation of deeper structures supplied by the same segmental innervation. Trigger points are clinically tender on palpation and can refer pain to other areas of the body (Middleditch & Oliver, 2005).

Kostopoulos and Rizopoulos (2001) do not relate the development of trigger points only to the irritation of neural tissue. The authors illustrate the activation of myofascial trigger points in Figure 3.7.

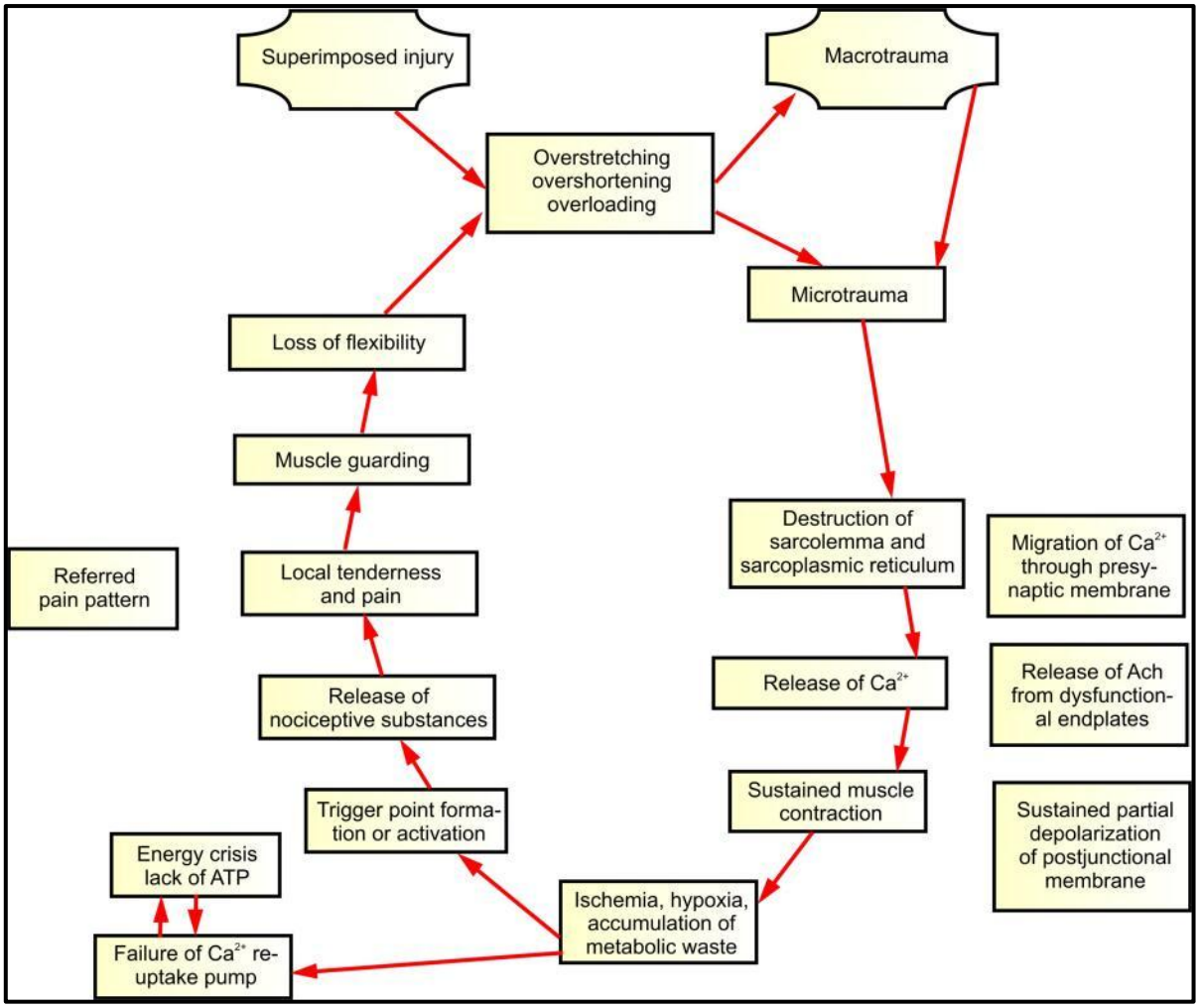


Figure 3.7: The activation of myofascial trigger points

(Kostopoulos & Rizopoulos, 2001)

Somatic referred pain by trigger points into other areas of the body as well as the lower limb can mimic nerve root involvement. It is therefore vital for the therapist to discriminate between somatic and nerve root referred pain during the assessment of the patient. The somatic referral patterns of trigger points that commonly contribute to pain in patients with CNSLBP are shown in the figures below.

The manual therapist should also identify all other trigger points that can develop in other parts of the ISMS due to ISMS dysfunction. Typical trigger points in muscles that refer pain in a similar distribution to neural referred pain in the lower limb are: quadratus lumborum, psoas and iliacus, gluteus maximus, gluteus medius and gluteus minimus, and piriformis muscles (Middleditch & Oliver, 2005). Muscles that refer pain to the lumbar and gluteal areas typically associated with low back pain are

iliocostalis lumborum, psoas and iliacus muscles and quadratus lumborum muscles. Referred pain from trigger points is characteristic of somatic referred pain: when the trigger points are released the referred pain disappears.

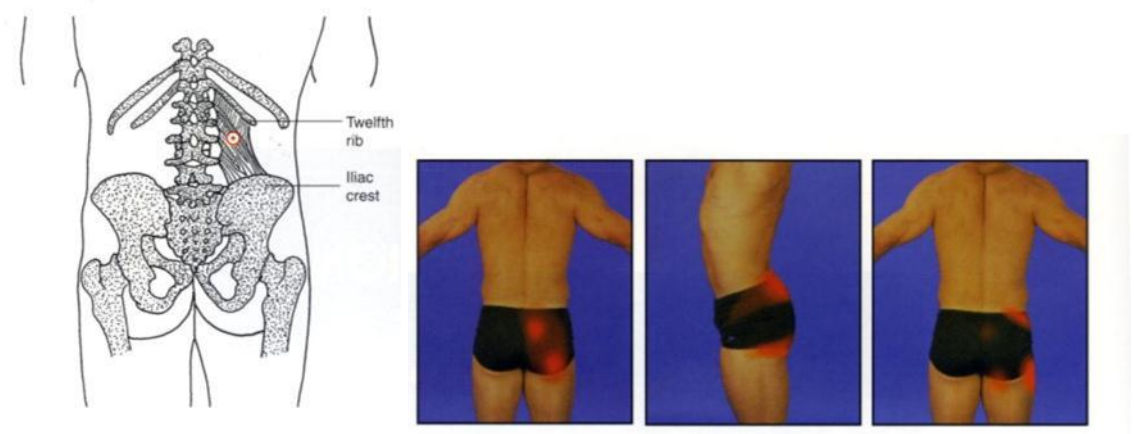


Figure 3.8(a): Anatomy of the quadratus lumborum muscle trigger point; Figure 3.8(b): Area of referral of the quadratus lumborum muscle (Kostopoulos and Rizopoulos, 2001).

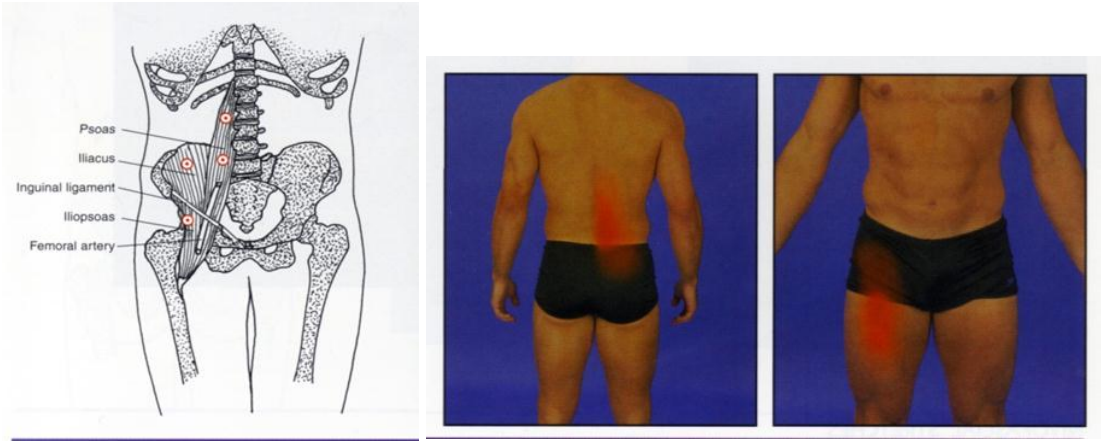


Figure 3.8(c): Anatomy of trigger points of the psoas and iliacus muscles; Figure 3.8(d): Area of referred pain of psoas and iliacus muscles (Kostopoulos and Rizopoulos, 2001).

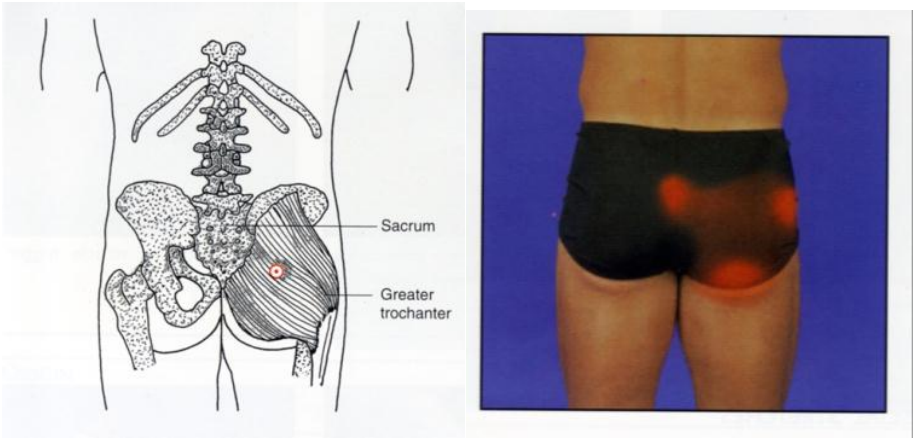


Figure 3.8(e): Anatomy of the trigger point of the gluteus maximus muscle; Figure 3.8f): Area of referred pain of the gluteus maximus muscle (Kostopoulos and Rizopoulos, 2001).

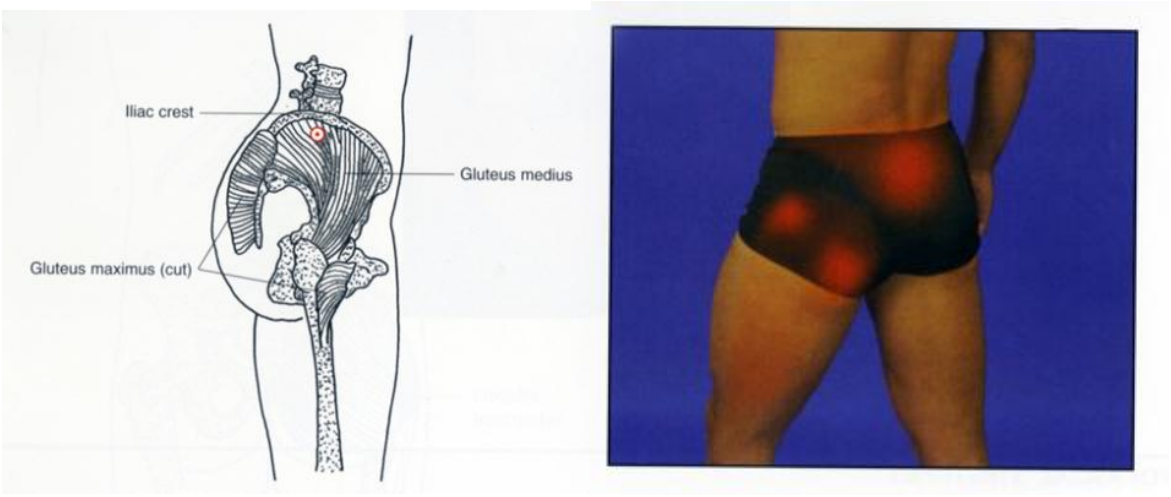


Figure 3.8(g): Anatomy of the gluteus medius trigger muscle; Figure 3.9(h): Gluteus medius muscle point referral pain pattern (Kostopoulos and Rizopoulos, 2001).

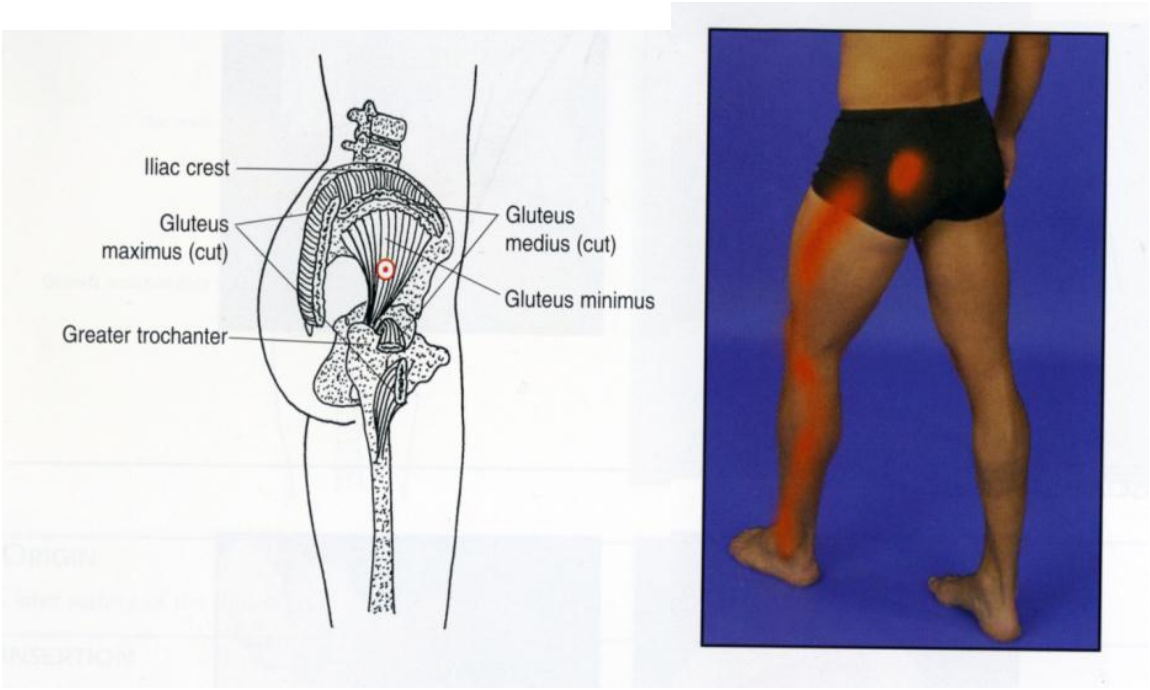


Figure 3.8(i): Anatomy of the gluteus minimus trigger muscle; Figure 3.8(j): Gluteus minimus point pain referral pattern (Kostopoulos and Rizopoulos, 2001).

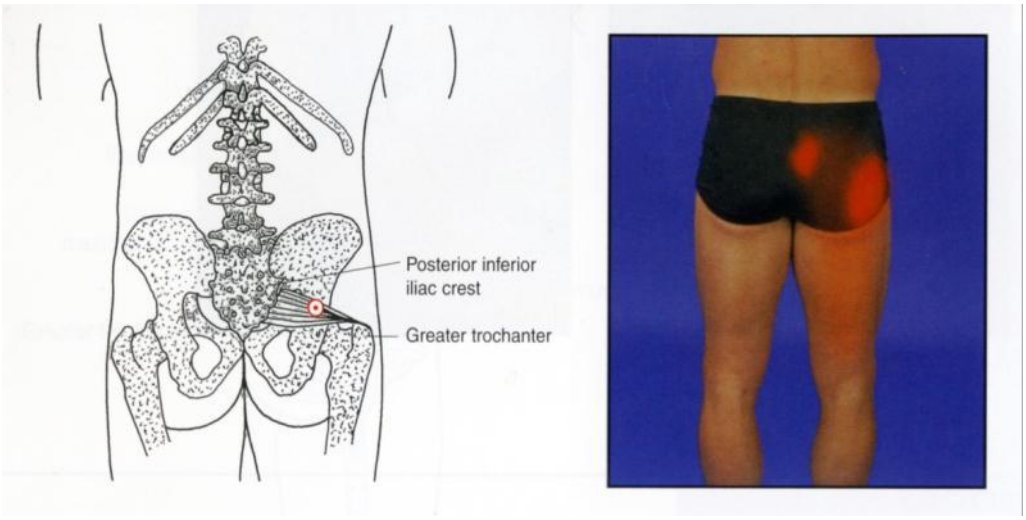


Figure 3.8(k): Anatomy of the piriformis muscle trigger point; Figure 3.8(l): The piriformis muscle pain referral pattern (Kostopoulos and Rizopoulos, 2001).

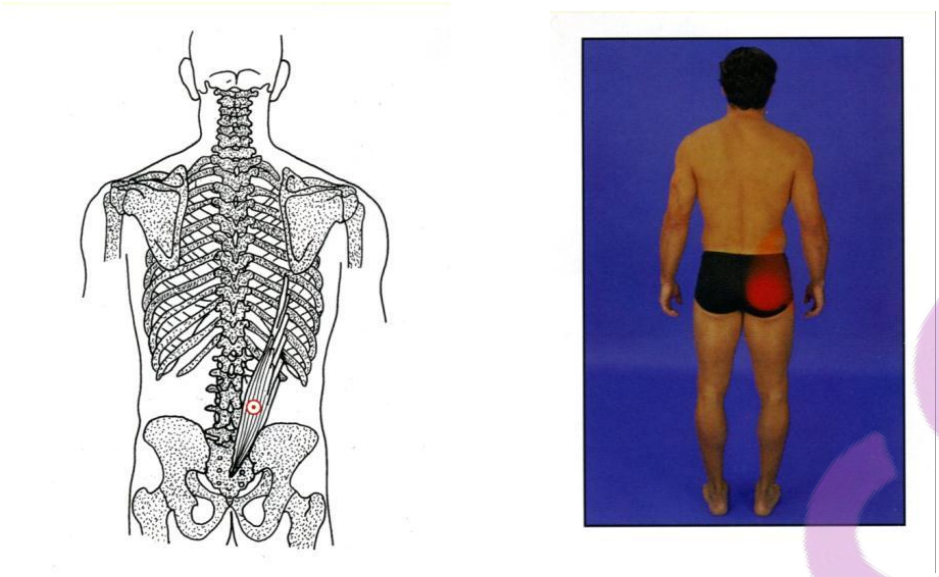


Figure 3.8(m): Anatomy of the trigger point in the iliocostalis lumborum muscle; Figure 3.8(n): Pattern of pain referral of the iliocostalis lumborum muscle (Kostopoulos and Rizopoulos, 2001).

It is of utmost importance that the manual therapist distinguish between various possible origins of the referred pain to accurately identify the variety of structures that can contribute to the complex pain syndrome of the patient with CNSLBP.

3.4.3 The process of connective tissue stiffening in patients with CNSLBP

As connective tissue surrounds and is inherently a part of the structure of muscles, it is subject to mechanical stress as a result of overuse, disuse, repetitive movement and/or hypermobility. Owing to the fact that it is not always possible to determine the tissue pathology in a clinic, the researcher has treated ‘soft tissue restrictions’ as shortening or stiffness in the connective tissue and muscular system, which have possibly developed as a result of fibrosis. Fibrosis in connective tissues can develop in two ways: (1) chronic local increase in stress (contraction of myofascial tissues), which may be due to overuse, might cause microinjury and inflammation; and (2) there may be a concurrent presence of inflammation, tissue hypo-oxygenation and cytokines, such as TGFβ-1, during immobility or lack of stress on connective tissues (Langevin & Sherman, 2006).

A consistent presence of hypomobility or immobilisation leads to atrophy of connective tissue, architectural disorganisation, fibrosis, adhesions and contractures (Langevin & Sherman, 2006). During the early phase of immobilisation the muscle-associated connective tissue shortens before actual shortening of the muscle fibres occurs. The presence of (myofascial) trigger points, taut bands in muscles and muscle spasm may also contribute to connective tissue remodelling and fibrosis. Shah, Phillips, Danoff and Gerber (2005) found a decrease in tissue pH and increased levels of inflammatory cytokines in trigger points in the presence of pain. The authors therefore argue that the presence of painful muscle contraction or tender foci within the perimuscular fascia may be a contributing factor that promotes hypomobility and tissue fibrosis.

Langevin and Sherman (2006) propose that connective tissue fibrosis in patients with CNSLBP occurs as a result of one or a combination of the following factors: decreased activity (sedentary lifestyle), which is characteristic of patients with CNSLBP; changes in the patterns of muscle activation, which can result in muscle cocontraction; muscle spasm or tissue microtrauma; and neuro-mediated inflammation.

The fact that the TLF is so closely integrated with the muscles inserted onto it means that muscle imbalance or spasm in one of these muscles such as latissimus dorsi and the opposite gluteus maximus can result in a change in the biomechanics of the TLF and can be the origin of severe pain due to the stimulation of the rich nociceptor innervation of the TLF.

3.4.4 Effects of nervous tension (stress) on the musculature of the body

Nervous tension results in major chemical changes and alterations in the musculature of the body. The causes of nervous tension can be anxiety, depression, frustration or general stressors of life and can manifest as muscular tension, more commonly seen in the cervical than the lumbar muscles. Elevation of the scapulae results in an increased thoracic lordosis and can either lead to an increased or a flattened lumbar lordosis (sway back) (Norkin & Levangie, 2005). So over time the

tension in the cervical muscles can lead to spinal imbalance and thoracic and lumbar strain, as indicated in Section 3.4.

The researcher observed that the effect of emotional stress on muscle activity results in shortening of soft tissues. Patients who exhibit emotional stress show an overall pattern of shortening which differs from the pattern of shortening which originated from a localised lumbar motion segment(s).

Prolonged muscle tension results in pain produced by the accumulation of metabolites in the muscles. The metabolites become a source of irritation, setting up a vicious cycle that eventually sustains the contraction and leads to joint restriction (Middleditch & Oliver, 2005). Sustained contraction in the whole spinal system becomes self-perpetuating and leads to widespread symptoms that pertain to CNSLBP.

3.5 Development of integrated spinal movement system (ISMS) dysfunction

The exact nature of the mechanisms at work during movement of the human ISMS as well as the development of abnormal function of the ISMS is neither well understood nor well defined. Zusman (2007, p 2) states that researchers are presently looking for validation of a *motion segment 'instability' model that revolves around pain-induced muscular insufficiency with continuing vulnerability to tissue 'injury'.* O'Sullivan (2005) describes eight models or different approaches for the diagnosis and classification of patients with CNSLBP to assist clinicians to understand the complex heterogenetic condition of CNSLBP. One of these classification models described by O'Sullivan (2005) is the mechanical loading model. In the following paragraphs the researcher starts the discussion on her multidimensional explanation of the multiple components that can drive the clinical picture of CNSLBP from the perspective of the abnormal biomechanical loading on the lower lumbar segments. This is followed by a discussion on the effect of the abnormal biomechanical loading on the soft and neural tissues, nociceptors

(peripheral and central sensitisation and altered pathways in the brain) and the influence of the latter on the patients' psychosocial behaviour and vice versa.

The discussion in the following paragraphs is therefore based on the researcher's clinical observation, clinical reasoning integrated with a review of the literature.

influenced by the alignment of the spinal curves. The interconnectedness between the systems around and within the motion segments, as well as the anatomical muscle configuration of the deep z-shaped intersegmental spinal muscles found bilaterally throughout the spine, attach all the motion segments of the lumbar, thoracic and cervical regions of the spine and the O1. This deep z-shaped configuration of the intersegmental spinal muscles lies at the core of the development of the rotational strain typically found in the spine of a patient diagnosed with CNSLBP. The rotational strain initiated by these z-shaped intersegmental muscles when they contract unilaterally is enforced throughout the spine by the global mobilisers and stabilisers. The opposite is also true: that change in the position of the shoulder and pelvic girdles and the head can initiate change in the spinal alignment and biomechanics of the spine to result in an asymmetrical (rotational) strain on the z-shaped intersegmental muscles.

The characteristic configuration of each vertebra (motion segment) in the spinal kinetic chain determines its specific action in posture and movement. The areas of transition in the spine are the occipito-cervical, cervico thoracic, thoraco-lumbar and lumbar-sacral areas. These transition areas undergo strain when a person's posture changes or due to external forces working within and onto the spinal system and create conflict between the demands for mobility and stability of the entire spinal structure (Middleditch & Oliver, 2005).

Adams, Bogduk, Burton and Dolan (2002) postulate that relatively small forces if concentrated onto a motion segment can produce pain. These small forces can result in sudden strain or sprain of the soft tissues of the motion segment due to bending and twisting of the vertebral column during lifting or other occupational,

recreational or home environment activities (Frymore *et al*., 1983; Troup, Martin & Lloyd, 1996; Radebould, Cholewicki, Panjabi & Patel, 2000). Zusman (2007) confirms that the validation is being sought for the argument that muscular insufficiency can be the origin of pain-induced 'motion segment instability' which increases the soft tissues vulnerability to injury.

It is therefore logical to assume that small changes in posture can also result in major increases in spinal loading, depending on the spinal region (Omino & Hayashi, 1992). Over time, the spinal loading brought about by any of the potential causes can generate a concentration of strain in innervated tissues, the annulus of the disc and the periosteum (which is part of the deep fascial connective tissue). The innervated soft tissues in the motion segment are rich in nociceptors and when provoked, through a process of adaptation, initiate the process of peripheral sensitisation (Brumagne, Cordo, Lysens, Verschueren & Swinnen, 2000). The abnormal spinal loading that causes the periosteal or soft tissue strain is not detectable on CT and MRI scans and may therefore easily not be identified.

The soft tissue adaptation is driven by pain processing, which is discussed in Section 3.5, and characteristic adaptive behaviour, which is explained in Section 3.6.

Radebould *et al*. (2000) found that during an experimental spinal loading incident patients' with chronic LBP's muscular response pattern of their trunk muscles differed from the muscular response pattern of healthy control subjects. Patients with low back pain maintain their agonistic trunk muscle contraction while the antagonistic muscles become concurrently activated. The healthy control subjects did not show the same muscle cocontraction as the subjects with CLBP. The authors (Radebould *et al.*, 2000) concluded that patients with CLBP stabilise their lumbar spine in response to sudden loading and that their response lasted longer than healthy control subjects. The muscles that were monitored with surface EMG electrodes were rectus abdominus, external oblique, internal oblique, latissimus dorsi, thoracic and lumbar erector spinae. The deep trunk muscles were not monitored.

3.5.1 Musculoskeletal adaptation to unilateral abnormal spinal loading

In the case of strain on one or more of the L4-S1 motion segments, nociceptors are stimulated in the area of strain by the abnormal loading. This results in unilateral muscle spasm to protect the segment(s). The deep short powerful z-shaped intersegmental muscles multifidus, rotatores and intertransversarius, which are innervated by the posterior rami of the spinal nerves, are probably the first muscles to respond to the strain by a muscle spasm (this is assumed by the researcher based on the fact that it will be a protective reflex contraction in the area of abnormal spinal loading to stiffen or immobilise the affected motion segments) (Middleditch & Oliver, 2005). The severity of the spasm will depend on the severity of the irritation of the nociceptors (intensity of the pain response) and primarily on the extent of the soft tissue strain due to the abnormal spinal loading. The muscle spasm occurs as a protective splinting response of the painful motion segment. If the pain intensity is not severe, the muscle spasm is short lived. If it is more severe, it may involve the deep intersegmental muscles over several segments and may include the entire spine from sacrum to occiput (Middleditch & Oliver, 2005).

Because the protective muscle spasm is unilateral, clinically it appears that the synergistic multifidus, rotatores and intertransversarius on the contralateral side are less active. The principle of reciprocal innervation will probably further result in the inhibition of the deep anterior postural muscles, in this case especially the transversus abdominus (Middleditch & Oliver, 2005; Hodges, 2013).

The effect of psycho-social stressors on a locally provoked pain response is discussed in Section 3.8.3 but it is important to realise that when the nociceptors are activated the pain impulses feed into the central pain mechanisms. The central pain mechanism activates the higher centres, which can be reinforced by emotional stress, fear avoidance and guarded movements. The researcher hypothesises that pain provoked by the abnormal loading response is increased by the presence of psycho-social factors, which may influence the patient's pain processing (perception) from the beginning (Adams *et al*., 2002; Waddell, 2004).

Biomechanically the unilateral muscle spasm will cause an increased compression force on the zygapophyseal joint on the ipsilateral side while at the contralateral side a distraction force will be created at the zygapophyseal joint and, as such, increase the abnormal spinal loading on the primary affected side. These compression and distraction forces will reinforce the peripheral sensitisation (reinforcing pain processing as discussed in Section 3.8) as a result of nociceptive provocation of structures such as the synovial membrane and joint capsules of the zygapophyseal joints and the annuli of the intervertebral discs, which might also be affected by the strain (Middleditch & Oliver, 2005).

The quadratus lumborum and the psoas major muscles, which are two posterior global muscles (innervated by the anterior rami of the spinal nerves T12-L3/4 and L1-L3 respectively), will most likely contract in a protective compensatory 'stiffening' of the trunk (called cocontraction between agonists and antagonists by Radebould *et al*., 2000). The global abdominal oblique external muscles (innervation anterior rami of T7-T12) on the contralateral side, together with the intermediate internal oblique muscles (innervation anterior rami of T7-T12 and L1) on the ipsilateral side, which contract in synergy with the quadratus lumborum and the psoas major muscles, will most probably also contract. The rectus abdominus muscle (innervation ventral rami of T6/7-T12) will probably contract asymmetrically more on the ipsilateral side of the posterior muscle spasm due to the sideflexion caused by the spasm of the unilateral muscles. These global muscles, which should be participating in the physiological movements of the spine, now become 'fixators' of the trunk (Middleditch & Oliver, 2005). The muscle activation pattern of the trunk muscles therefore changes.

The contraction of the quadratus lumborum on the painful side elevates and rotates the pelvis posteriorly on the ipsilateral side. The posterior rotation of the pelvis on the painful side is reinforced by the rotational action of the internal oblique on the ipsilateral side and the external oblique on the contralateral side. The sideflexion on the painful side, which occurs as a result of the spasm in the quadratus lumborum, is reinforced by the psoas major muscle, which also exerts a rotational force on the vertebral bodies of the spine onto which it inserts towards the contralateral side (Middleditch & Oliver, 2005).

The effect of the unilateral muscle spasm by the deep unilateral intersegmental muscles as well as the global mobilisers of the trunk (latissimus dorsi and psoas major), resulting in sideflexion of the lumbar vertebral column, tilts the vertebrae inferiorly on the ipsilateral side and causes the intertransverse ligament to slacken. The articular processes in the lumbar region glide relative to each other, with the superior articular process of the lower vertebra on the ipsilateral side gliding upwards while the inferior articular process of the upper vertebra glides inferiorly. On the contralateral side the inferior articulating processes of the upper vertebra glide downwards and the corresponding articular process of the inferior vertebra glides upwards. The implication of this gliding in opposite directions is that the vertebra and its zygapophyseal joints that were originally affected by the abnormal loading do not stay local but spread to the adjacent vertebrae via the inferior and superior zygapophyseal joints and the discs, which become wedge shaped to the side of lateral flexion and thicker on the contralateral side.

The muscular component exerts a rotational force on the lumbar zygapophyseal joints in addition to the superior and inferior gliding of the articular surfaces because the superior zygapophyseal joints of the lumbar spine face posteriorly and medially. The inferior zygapophyseal joints of the lumbar motion segment face laterally and anteriorly. The articular surfaces are concave in the transvers plane and are vertically oriented, which limits rotation. The total range of axial rotation in the lumbar region is therefore approximately 10^{o} with 2^{o} of bilateral segmental (1^{o} unilateral) axial rotation (Middleditch & Oliver, 2005). When rotation occurs around the inferior lumbar vertebra, the rotational movement is associated with a gliding movement of the upper (superior) vertebra in relation to the lower (inferior) vertebra. This gliding movement results in the disc also being subjected to gliding and shear force. The disc limits rotation of the lumbar spine at segmental and global levels. The rotational strain that is imposed on the vertebrae by the muscle spasm in the deep z-shaped intersegmental muscles, and the global muscles described in the preceding paragraphs, will create additional loading force on the zygapophyseal joints and the annuli of the intervertebral discs, aggravating pain provocation and, as such, muscle spasm.

In the upper four segments of the lumbar spine, lateral flexion is normally accompanied by axial rotation of the vertebrae to the opposite side and rotation is accompanied by lateral flexion to the opposite side. Conversely at the joints of the fifth lumbar vertebra and the sacrum, axial rotation of the vertebra is accompanied by lateral flexion to the same side and lateral flexion of the joint is accompanied by rotation to the same side (Bogduk & Twomey, 1987). Clinically, because of the strain caused by abnormal spinal loading in the upper lumbar vertebrae, the manual therapist could find more muscle spasm on the contralateral side than on the ipsilateral side. In an acute episode of NSLBP superimposed on CNSLBP, clinically the manual therapist may find that longissimus thoracis spasm can flatten the upper lumbar lordosis, which puts strain on the thoracolumbar junction.

In strain on the L5-S1 segments the manual therapist may find muscle spasm more on the ipsilateral side. With these anatomical features it is also possible to get fluctuating muscle spasms and flattening of the lumbar lordosis as part of the patient's pain response. The malalignment of the joints described above results in strain on the annulus fibrosis, which plays a stabilising and restricting role during all the movements of the interbody joint. All collagen fibres of the annulus fibrosis are involved in weight bearing and resist distraction. When the collagen fibres of the annulus fibrosis are separated, they are stretched and resist movement. In movements other than distraction, the collagen fibres' oblique orientation will determine their participation or restriction in the movement (Kapandji, 2008).

The straining force on the zygapophyseal joints involves the ligaments as well. The ligaments under direct strain because of the rotational force created by the unilateral muscle spasm will be the ligamentum flavum and the anterior and posterior longitudinal ligaments (Middleditch & Oliver, 2005). Owing to the unilateral (side)flexion and rotation of the motion segments, these longitudinal ligaments will experience unilateral strain. The superior/inferior displacement of the zygapophyseal joints, which occurs as a result of the sideflexion and rotation, might cause a rotational/unilateral strain on these longitudinal ligaments, which might not be localised to the lumbar region only but extend to the sacrum and cervical regions

where it might also put strain on the nuchal line (Bogduk, 2011). The TLF plays an important role in extending the strain from the lumbar region to the thoracic and cervical regions to the nuchal line.

Another ligament that will also be strained by the lateral flexion and rotational force created by the muscle spasm is the interspinous ligament, which runs between adjacent spinous processes. The extent to which the supraspinous ligament will be strained depends on the level of the abnormal spinal loading and the muscle spasm because the supraspinous ligament is well developed in the upper lumbar area but terminates at L3 or L4 in most individuals and is present in only 5% of individuals and mostly lacking in L5-S1 (Middleditch & Oliver, 2005). Strain on the interspinous and supraspinous ligaments will therefore depend on the level of the original abnormal spinal loading that resulted in the muscle spasm that caused the sideflexion and rotational strain.

The iliolumbar ligament, which connects the transverse process of L5 to the ilium bilaterally, will be affected by the unilateral sideflexion and rotational force created by the muscle spasm because, apart from preventing forward sliding of L5 on the sacrum, it further resists twisting, flexion, extension and lateral bending. The iliolumbar ligament may therefore be strained unilaterally.

The rotational strain that can develop due to the unilateral muscle spasm and rotation of the pelvis will not only have an upward effect via the superior zygapophyseal joints but also a downward effect via the inferior zygapophyseal joints of the same vertebra. As such it can also result in strain on the sacro-iliac joints and the iliolumbar ligaments.

The T12 vertebra is regarded as a true swivel joint between the thoracic kyphosis and the lumbar lordosis. The superior articular facets have the characteristics of the thoracic vertebrae while the inferior articular surfaces have the characteristics of the lumbar vertebrae (Middleditch & Oliver, 2005). As a transition area where one spinal curve changes into the next opposite curve (like the cervico-thoracic junction), the joints and disc at these junctions take more strain.

Movement in the thoracic spine is limited and complicated by the costotransvers, costovertebral and sternocostal joints; the ribcage and sternum as well as the shape and size of the thoracic discs are all articular components that contribute to the stability of the thoracic spine.

The shape of the superior zygapophyseal joints is almost flat and oval and faces posteriorly and slightly laterally and superiorly. The inferior articular surfaces face superiorly, slightly inferiorly and slightly medially (Middleditch & Oliver, 2005). Rotation of the thoracic spine to one side accentuates the concavity of the rib to the side of the rotation. On the opposite side the rib-concavity is flattened; the costochondral rib angle is flattened on the same side of rotation while the costochondral rib angle on the opposite side to the rotation is accentuated.

Owing to the cylindrical shape of the zygapophyseal joints, the rotational force that was limited in the lumbar region will extend superiorly and be exaggerated in the thoracic region and result in axial rotation. Axial rotation results in rotation-torsion of the annulus fibrosis of the disc, which can be at least three times greater than the axial rotation that occurs in the lumbar spine. Although rotation is the largest range of movement that can take place in the thoracic spine, it is limited by the resisting forces of the corresponding pair of ribs, which resultantly distort the corresponding ribs and their cartilages (Middleditch & Oliver, 2005; Kapandji, 2008).

The capsular ligaments of the zygapophyseal facet joints are very short but strong and provide strong resistance to flexion movements. They also resist extension and may resist lateral flexion. This means that they appear to be stabilisers of the spine and protect the disc from excessive bending in any direction (Middleditch & Oliver, 2005). During the torsional strain of the spine these joints will also be strained and become a source of nociception.

The over-activation of the quadratus lumborum muscle spasm with its origin and insertion on the pelvis and the ribcage could cause thoracic sideflexion strain. During sideflexion the articular surfaces on the contralateral side glide upwards (superiorly)

while the articular surfaces on the ipsilateral side glide downwards (inferiorly). Sideflexion is limited by the impact of these articular processes and by the tension on the ligamenta flava and the intertransvers ligaments.

During sideflexion of the thoracic spine the contralateral side of the thorax elevates, the intercostal spaces widen, the costochondral angle of the rib tends to gape and the thoracic cage is enlarged. On the ipsilateral side as the thorax moves inferiorly and inwards, the intercostal spaces are narrowed and the costochondral angles decrease. Rotation and some sideflexion strain in the thoracic spine are associated with the flattening of the ribcage on the one side and accentuation of the ribcage on the other side, which will affect the patient's breathing pattern as well as forced expiratory volume (FEV) and tidal volume (Middleditch & Oliver, 2005). Depending on the increase or the decrease in the curve of the thoracic spine, the ribs will accommodate by changing the articulation and therefore the angulation of the ribs at these joints. Changes in the articulation of the costovertebral, costotransvers, and sternocostal angulation will result in additional strain on these synovial joints, which are rich in peripheral nociceptors. Because the thoracic spine is stabilised by the ribcage, thoracic pain and breathing dysfunction are often a result of the changed angulation. The nociception in the soft tissues around and within the costovertebral, costotransvers, and sternocostal joints is irritated or stimulated and not necessarily at the zygapophyseal joints. Overactivation (spasm) of the quadratus lumborum will also limit lateral costal and diaphragmatic breathing (Kapandji, 2008).

The thoracic kyphosis is influenced by the position of the scapulae and the shoulder girdle. Abduction and elevation of the scapulae are associated with an increased thoracic kyphosis, resulting in an increase in the cervical and lumbar lordoses and strain at the transitional junctions. If elevation of the scapulae occurs in conjunction with a flattened thoracic region, it will most probably be associated with a flattened cervical and lumbar lordoses (flat back).

3.5.1.1 Somatic pain referral patterns of the musculoskeletal system

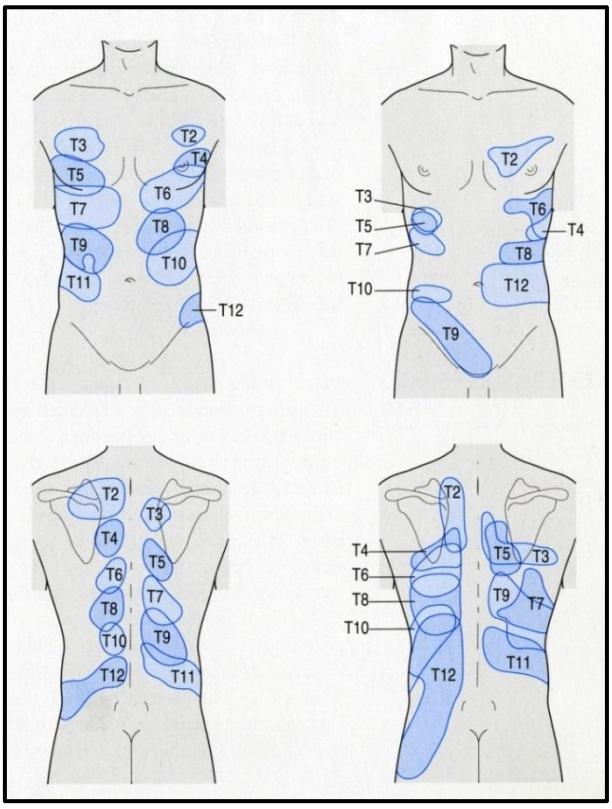


Figure 3.9(a): A presentation of the somatic referred pain from thoracic zygapophyseal joints;

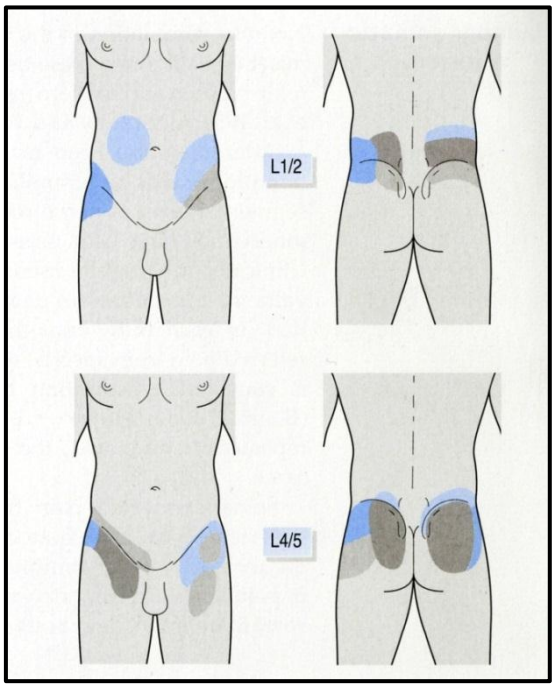


Figure 3.9(b): A presentation of the somatic referred pain from lumbar zygapophyseal joints

(Middleditch & Oliver, 2005 p 259)

3.5.1.2 The importance of the dysfunctional diaphragms in ISMS dysfunction

ISMS dysfunction is intimately associated with dysfunction in the diaphragms. The functioning of the respiratory diaphragm is compromised by the torsioning of the spine and especially the thoracolumbar junction, where the posterior part of the respiratory-dome specifically attaches to the deep surfaces of the costal cartilages, the costal arches, the tips of the eleventh and twelfth ribs and the T12-L1 vertebral bodies. Torsioning at the T12/L1 segmental level might result in a pulling force of the diaphragm on the T12/L1 segment and on the costal cartilages, the costal arches, and the tips of the eleventh and twelfth ribs, which may become a source of nociception.

At a more multisegmental level, unilateral spasm in the longissimus longus and latissimus dorsi might reinforce the torsional force on the thoracolumbar junction. The torsioning action of the local and global muscles will probably lead to an increase in the thoracic kyphosis as well.

With the torsioning and increased tension in the musculature of the spine, the circular muscles of the cervico-thoracic junction might also incriminate the lower cervical structures and the shoulder girdle. Owing to the biomechanical dysfunction of the trunk and pelvis musculature, patients might be involved in pelvic floor dysfunction. Patients' inspiratory capacity might be affected by stiffening and torsion of the thoracic spine and by the involvement of the costovertebral, costochondral and costosternal joints. The fact that torsioning might be more explicit in the thoracic area due to the alignment of the apophyseal joints might mean that the traction effect of the ANS results in symptomsa general feeling of unwellness.

3.5.1.3 The role of the thoracolumbar fascia in the development of ISMS dysfunction

If the transvers abdominus, internal oblique abdominus latissimus dorsi and gluteus maximus contract unilaterally as described in the typical scenario in the preceding paragraphs, these muscles will create a unilateral 'pulling force' distribution on the TLF. This force will add to the abnormal unilateral spinal loading not only on the

affected lumbar motion segments (locally) but also on the lumbar-sacral, thoraco-lumbar and thoracic regions (globally). The stabilising role of the TLF will be altered in the sense that it will increase unilaterally. The latissimus dorsi and the internal oblique abdominus exert a rotational force on the lumbar spine as discussed earlier. The TLF with its connections up to the nuchal line on the occiput will therefore exert a rotational force on the thoracic region and on the cervical region (Middleditch & Oliver, 2005).

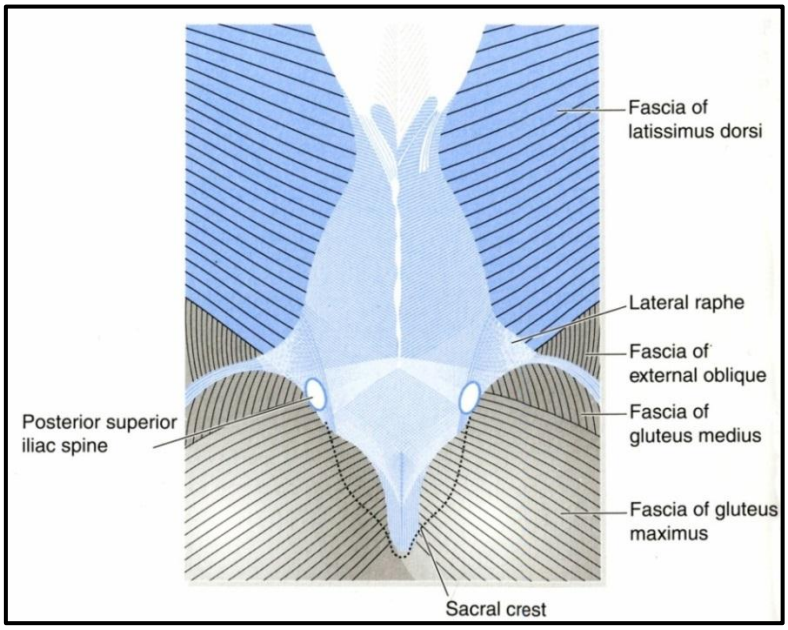


Figure 3.10(a) The superficial thoracolumbar fascia

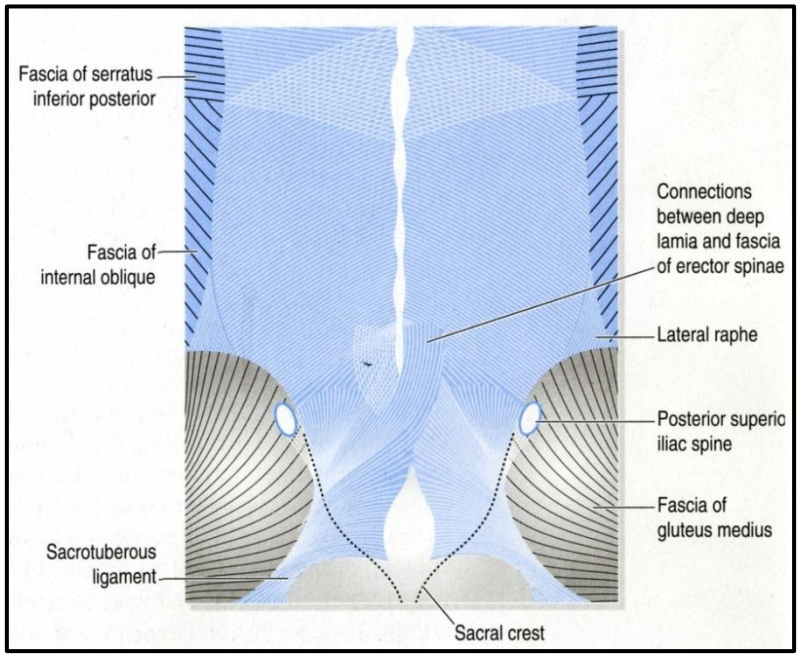


Figure 3.10(b) The deep thoraco-lumbar fascia

(Middleditch & Oliver, 2005 p 128)

The envisaged effect of the rotational force initiated in the lumbar spine by multifidus rotatores and intertransversarius has been enforced and magnified in the thoracic spine and is continued by these deep z-shaped muscles of the core axial skeleton throughout the cervical spine. This is because they have the same anatomical configuration and innervation by the posterior primary rami throughout the spine.

At the cervico-thoracic junction the stiff thoracic spine articulates with the relatively mobile lower cervical spine. At the transitional junction the intervertebral discs and apophyseal joints are particularly subject to stress and strain. Owing to the transitional strain, the C6-T1 segments are commonly subject to degeneration (Middleditch & Oliver, 2005). The spinous processes of C7 and T1 may become prominent and are often tender to palpation.

The uncovertebral joints of Luschka between C3 and C6 provide the cervical spine with additional mobility because they are synovial joints. Because of the mobility of the cervical spine, the effect of the rotational force by the TLF, which extends to the nuchal line, is that the cervical spine is vulnerable to absorb the rotational force that extends from the lumbar spine upwards. The apophyseal joints in the cervical spine C3-C5 have a superior orientation (oval and facing posterior and superior (upwards)) while the inferior facets face forward (anterior) and inferior (downwards) (Middleditch & Oliver, 2005). The orientation of these apophyseal joints allows flexion, extension, rotation and sideflexion to occur. A clinical observation by the researcher is that when the posterior cervical muscles stiffen, the cervical lordosis flattens. The whole cervical spine is incriminated when the flattening of the cervical lordosis occurs. The researcher hypothesises that this stiffening is not necessarily due to the spasm but to shortening of the myofascial system to limit the head movements on the C1 as a compensatory mechanism and also to limit consequential spinal movement. The cervical column does not necessarily react with sideflexion or rotation like the lumbar and thoracic regions but the asymmetrical muscle tension can be palpated and directly related to the patient's headache or cervical symptoms in the presence of a primary lumbar response to abnormal loading (Middleditch & Oliver, 2005).

The condyles of the occiput articulate with the kidney-shaped articular surface of the C1 vertebra. The orientation of these facets and their high lateral margins ensure that movement at the atlanto-occipital joint is mainly flexion and extension. The atlas articulates with the axis below and provides a pivot around which the atlas and the head rotate (Middleditch & Oliver, 2005).

The spinous process of the axis is large, usually bifid and normally provides a prominent bony landmark for palpation. The suboccipital muscles attach onto the spinous process of the axis, which provides powerful leverage for the muscles' actions. The asymmetrical pull of the extension of the TLF on the nuchal line can explain why patients with CNSLBP also complain of headache.

The abnormal biomechanical loading in the lumbar spine also spreads inferiorly towards the lower limb. The stability of the lumbo-pelvic region is dependent on the interplay between the TLF, tensor fascia latae, and the abdominal fascial systems. The TLF covers the muscles in the sacral region and then extends through the thoracic region to the nuchal line. In its course on the posterior trunk, several muscles attach to it, which results in adjusting the tension in the TLF and, as such, contributes to transferring the load from the trunk to the pelvis and lower limbs (Middleditch & Oliver, 2005). The sacral end of the torsioned pelvis initiates the pull on the filum terminale as it winds up within the spinal canal towards the cranium.

3.5.1.4 Effect of ISMS dysfunction on gait

When the pelvis is in elevation and backwards rotation due to the unilateral muscle spasm in the lumbar spine and the spasm in the quadratus lumborum and psoas major, the muscle imbalance that results might cause somatic referred pain in the gluteus medius and maximus. As a result of the distortion of the pelvis on the hip joint, the patient bears weight on a slightly flexed, abducted and slightly laterally rotated hip joint instead of on a hip joint that moves in the habitual movement, which is in and out of the closed packed position. The patient will therefore present with an asymmetrical gait pattern (a shorter step on the ipsilateral side of the muscle spasm).

Weight bearing on a hip joint in slight flexion, abduction and lateral rotation places abnormal loading on the hip joint during the weight-bearing phase of gait. The weight bearing on the hip in slight flexion, abduction and lateral rotation reinforces the asymmetry in the spine that originated from the unilateral muscle spasm in the z-shaped segmental muscles (core axial muscles) around the spine. The asymmetry was also caused by the rotational force created by the additional latissimus dorsi, internal and external oblique, thoracis and longissimus lumborum and the TLF. The asymmetrical gait pattern (decreased weight bearing on the affected side and limited swing through) will therefore reinforce the rotational strain in the spine.

3.6 Soft tissue plasticity as an inherent process in the development of ISMS dysfunction

Plasticity as part of the adaptation of soft and neural tissues (including brain) to continuous stressors is argued to be one of the main reasons for the development of CNSLBP. The fact that plasticity can be reversed (remodulated) (because it is an inherent characteristic of soft and neural tissues (LAngevin & Sherman, 2006) is one of the pillars on which the researcher's multifaceted manual therapy model for the management of patients with CNSLBP is based.

Connective tissue changes its consistency depending on the condition that it is under. When it is put under stress, it maintains its mobility. However, in the presence of inflammation (e.g. due to microtrauma) and immobility it will shorten or stiffen due to atrophy of connective tissue, architectural disorganisation, fibrosis, adhesions and contractures (Langevin & Sherman, 2006) through a process called 'plasticity'.

It has been reported that shortened or stiffened connective tissues can be reversed through a process of stretching these tissues; the gel-like ground substance can be reversed by manual stretching or by stretching these tissues during exercise to become viscous again (Shah, Phillips, Danoff & Gerber, 2005; Langevin & Sherman, 2006; Stecco *et al*., 2004). According to Schleip *et al*. (2006), proteoglycans (which form the gel of the ground substance) play a major role in the absorption and distribution of the compressive forces. When these tissues are stretched during the

myofascial release, the ground substance becomes viscous again. The phenomenon of mobilising the connective tissues (fascia) through stretching is also known as tissue creep.

Langevin and Sherman (2006 p 3) write:

> *A hallmark of connective tissue is its plasticity or 'remodelling' in response to varying levels of mechanical stress*.

On the basis of this assertion by Langevin and Sherman (2006), plasticity can be considered the process of shortening or stiffening of the connective tissue and 'remodelling' indicates the reversing of the process of plasticity with the aim of restoring tissue mobility.

Distinguishing between fibrosis and scar tissue is very difficult to determine clinically. For this reason the researcher suggests that myofascial stretching on shortened/stiffened connective tissue be undertaken on a symptomatic basis for the patient with CNSLBP. Based only on the patient's tissue responses, a therapist can clinically judge whether the 'soft tissue restriction' offers a great deal of resistance. This gives the therapist an idea of the type of tissue restriction, for example a ligamentous, tendonous, facet joint capsule and potentially nerve root sleeve or a combination of nociceptive tissues, involved in the patient's symptoms.

Post-treatment response will vary from treatment to treatment in one patient and across patients, depending on the stage of healing and the skill of the applied techniques. The researcher hypothesises that only in the case of scar tissue will the patient respond with limited improvement in soft tissue mobility. This implies that soft tissue restrictions, whether they are early adhesions or later fibrosis or even scar tissue, may respond with a predictable outcome to manual therapy, which can be identified based on the patient's post-treatment response. The process of mobilising soft tissue restrictions (early adhesions, fibrosis or scar tissue) to restore ISMS alignment and function occurs through a process of remodelling.

Contradictory reports exist in the literature on whether fascia can be over stretched by manual therapy or not. Schleip *et al*. (2006) are of the opinion that it is not possible to over-stretch fascia by means of therapeutic manual stretching (Schleip *et al*., 2006). Langevin and Sherman (2006), however, caution that connective tissue remodelling through stretching can potentially have a harmful as well as a beneficial effect. Direct stretch to ligaments and joint capsules needs a careful approach to avoid tissue inflammation. The challenge to the therapist is to reason clinically how much force is enough to be beneficial and how much is harmful.

Release of muscle shortening is achieved by releasing muscle spasm, taut bands and trigger points. Although the principles for the release of muscle spasm, taut bands and trigger points differ from those for the release of connective tissues, stretching plays an important role as well. The same principles of caution are relevant to the release of muscle spasm, taut bands and trigger points as those applicable to release of shortened/stiff connective tissues.

3.6.1 Plasticity and postural control

It is well known that motor skill can be relearned. In the presence of abnormal muscle recruitment, guarded movements, and adaptation or compensation as a result of malrecruitment and guarded movement, it is vitally important to plan the relearning of normal muscle recruitment and movement patterns after soft tissue release. This is so that mobility and alignment that were achieved through the passive soft tissue release can be maintained.

As a result of the cortical changes that took place and resulted in or contributed to the development of abnormal movement patterns (guarded movement and abnormal muscle recruitment), normal movement has to be relearned through a goal-directed process of motor learning (Shumway-Cook & Woollacott, 2007). The process of motor relearning of movement patterns is closely associated with cognitive control over habitual movement patterns to change/adapt them purposefully to prevent and manage recurrences, avoid harmful behaviour or movement and introduce beneficial movement patterns, health behaviour and lifestyle changes. The last mentioned can

only occur through appropriate health education and empowerment of the patient to take control of their condition.

3.7 Factors that can influence/adapt the typical pattern of ISMS dysfunction

A number of factors can influence the development of the typical pattern of ISMS dysfunction, as described in the preceding paragraphs, resulting in differences in the clinical presentation of the ISMS dysfunction. Dankaerts, O'Sullivan, Burnet and Straker (2006) found that patients with CNSLBP although there is no observable difference in seating posture compared to normal healthy subjects, on sEMG is became clear that there is not a homogenous trunk muscle activation pattern in the patient group. These results may therefore indicate that there are different underlying mechanisms for the abnormal trunk muscle activation. The researcher describes various reasons/factors that may cause the differences, based on clinical experience and the literature reviewed.

3.7.1 Differences in response of the lower and upper lumbar motion segments

The motion segments in the upper lumbar spine (L1-L4) respond differently on rotation than the lower lumbar spine (L5-S1). This means that abnormal loading on the L1-L4 motion segments, resulting in sideflexion due to unilateral muscle spasm, will result in axial rotation of the motion segments to the contralateral side of sideflexion while, in the L5-S1 motion segment, axial rotation towards the ipsilateral side of lateral flexion will take place (Twomey & Bogduk, 1978). This difference in response of the lower and upper lumbar motion segments to sideflexion caused by the unilateral muscle spasm can result in differences in axial rotation that will torsion (corkscrew) upwards in the spine or downwards to the L5-S1 motion segment and as such have an effect on the rest of the spinal regions' response to lumbar dysfunction.

3.7.2 Poor posture and postural control

Poor posture can be the cause or a result of CNSLBP (Sahrmann, 2002). If extension of the lumbar spine increases (as a result of muscle imbalance between

erector spinae and abdominal muscles) it can lead to an increase in the thoracic spinal extension (flat back), or a flat back with a high thoracic kyphosis. In people who work in prolonged sitting positions where so many people sit for lengthy periods of time an increased thoracic kyphosis can develop or short hamstrings, which pull the pelvis into a posterior tilt in sitting, can contribute to the development of a general spinal kyphosis (lumbar as well as thoracic kyphosis). Dankaerts, *et al*., (2006) showed different patterns of trunk muscle activation which may indicate that there are different mechanisms underlying the different patterns of muscle activation in sitting.

3.7.3 Disuse and sedentary lifestyle

Disuse as a result of a sedentary lifestyle has a profound effect on the physical condition of the back, which aggravates and maintains physical dysfunction and leads directly to more severe disability (permanent change in lifestyle and no improvement in CNSLBP).

In any of the different postures mentioned, the spinal deconditioning (as a result of poor postural control and a sedentary lifestyle); the history of the patient's NSLBP, can indicate repetitive incidents of abnormal spinal loading which may change the ISMS biomechanics of the person and manifest as a different pattern of shortening.. A change in ISMS biomechanics will influence the way ISMS dysfunction will develop as a result of the soft tissue response to a 'new incident' of abnormal spinal loading.

3.7.4 The process of spinal loading

The process through which the abnormal biomechanics develop and the end-result may vary due to the different forms and degrees of spinal loading on the already changed biomechanics of the motion segments in the different regions (i.e. due to poor posture) of the biomechanical component of the ISMS.

If a person continually adopts a poor posture, fascia shortens in the pattern of the poor posture. A common example is performing a sitting job in a poor ergonomically designed setup/workstation where gravity loads the intervertebral system and tissue

creep occurs so that the upright posture is difficult to maintain. Owing to the tissue creep, shortening of the fascia will occur at the anterior thoracic spine (anterior shoulder girdle), with resultant aggravated thoracic kyphosis and decreased lumbar lordosis. Continuous strain on fascia in the ISMS due to poor posture will result in fascia shortening because fascia shortening occurs earlier than muscle fibres through the process of 'negative' plasticity (Langevin & Sherman, 2006), which contributes to ISMS dysfunction.

Panjabi (2003) states that people with suboptimal neuromuscular control for some or other reason and who perform dynamic activities are more likely to develop CNSLBP. The implication of this hypothesis is that the pattern of change in the trunk muscle activation might depend on the state of the person's neuromuscular control before abnormal spinal loading took place.

Strain will occur at the transitional intervertebral junctions C7/T1, T12/L1 and L5/S1, which may also result in malalignment of the biomechanical aspect of the ISMS. In this case the shoulder and pelvic girdles are also involved due to the effect of the abnormal biomechanics of the biomechanical ISMS on these girdles and vice versa.

3.7.5 Cervical and thoracic dysfunction

Anecdotally, clinicians observed that patients with neck pain commonly develop LBP. Researchers (Hodges & Richardson, 1996) found that there is an association between neck pain and LBP. Patients who suffer from LBP also suffer from neck pain. Both conditions are associated with dysfunction of the trunk muscles, especially loss of control of the transvers abdominus muscle, which has led to the conclusion that in both cases abdominal muscle dysfunction (which is inherent part of poor posture) may compromise ISMS control.

In a specific study conducted by Hodges *et al*. (1996) their first main finding was that people with sub-acute neck pain have a reduced capacity to perform the abdominal drawing-in task (ADIT). The second main finding was that reduced performance in the ADIT was associated with increased risk of LBP over the following two years, for both patients and control subjects.

The conclusion was made that suggested that maintenance of normal trunk muscle control should be a goal of therapy in patients with sub-acute neck pain as well as in patients with CNSLBP.

3.7.6 Association of chronic unilateral low back pain with disruption of tactile input

Moseley, Gallagher and Gallace (2012) found that chronic unilateral LBP is associated with spatially defined disruption of tactile input. The shortening of fascia, which is known to be a source of nociception, might limit proprioceptive feedback to conscious awareness of the strain on the TLF and other soft tissues and articular structures (ligaments and joint capsules), which can lead to a decrease in tactile discrimination and spatial awareness. It may be possible that a patient with CNSLBP experiences spatial disruption of tactile input that their muscle recruitment may adapt due to the changed (limited or abnormal) sensory input or interpretation of sensory input in the brain. Abnormal muscle recruitment will probably lead to adapted movement.

3.7.7 Neural referred pain through torsioning of the biomechanical ISMS

The researcher hypothesises that in the torsioning of the biomechanical ISMS the disc can be forced against the soft tissue structures to give rise to tension on the nerve root sleeve and can result in neural-referred pain (nerve root sleeve pain) (discussed in Section 3.6) and spasm in the muscle innervated by the affected nerve roots. The relevant muscles in patients with CNSLBP are, for example, the gluteus maximus, medius and minimus; piriformis; quadratus lumborum; iliacus; and psoas major muscles. These muscles may in themselves refer pain to the lumbar area and pelvis and lower limb as well as to the shoulder girdles and upper limb(s). The pain referred from these muscles is somatic-referred pain (discussed in Section 3.5.1.1).

3.7.8 The effect of stress on spinal dysfunction

The researcher observation of patients in clinical practice has revealed that patients may present with soft tissue shortening due to biomechanical dysfunction as well as the generalised soft tissue shortening seen typically in patients with emotional stress. How the two combinations of the pattern of soft tissue shortening relates to the fact that Brooks & Tracey (2005) found that anxiety induced pain results in a different increase in brain activity is still not known (Section 3.8.4.1).

3.7.9 The influence of underlying degeneration in the synovial joints of the spine

Adams *et al.* (2002) argue that a link exists between back pain, mechanical loading, ageing, dysfunction and degeneration. The links between all these factors are complex and justify further research based on clinical data. The researcher hypothesises that the presence of degenerative changes in the spine before an incident of ANSLBP can render the spine vulnerable for episodes of abnormal spinal loading. Abnormal spinal loading on degenerative zygopophyseal joints can result in an aggravated pain response, which can together with psychological responses such as catastrophisation, fear of pain and guarded movement contribute to the development of ISMS dysfunction.

From the moment the abnormal loading occurs and affects a degenerative joint(s) the healing process sets in. Any of the tissue systems (articular, myofascial, connective tissue and neural tissues) in the low back and therefore in the spine can be affected by the wear and tear, strain or microtrauma as a result of the abnormal spinal loading. Each of these tissue types has the *'potential to be repaired by mechanisms unique to its cellular composition and individual biochemistry'* (Porterfield & DeRosa, 1991 p 4).

The healing process can be one of two types: regeneration of the injured tissue *or* replacement of the injured tissue by dissimilar tissue, i.e. connective scar tissue. The latter occurs when the injury exceeds the tissue's capacity to regenerate.

Factors that influence the type of tissue repair that will take place are the availability of vascular supply and the extent of the injury.

3.7.10 Previous history of back pain and response to health care management

One of the best predictors for the development of CNSLBP is the patient's previous history of back pain and his response to the management by health care professionals. Waddell (2004) and Field (2009) are of the opinion that health care professionals' management of patients with acute NSLBP in general can also influence the development of characteristic behaviour in patients with acute/sub-acute NSLBP by the way they manage patients. Doctors and therapists may be treating patients with sub-acute or CNSLBP as if they still had acute pain and, as such, contribute to the establishment of chronic pain and illness behaviour in patients (Waddell, 2004).

3.8 Pain processing as integral component driving the development of ISMS dysfunction

The International Association for the Study of Pain (Merksey & Bogduk, 1994) defines pain as '*an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage*'. Pain is a multidimensional 'sensation' with sensory-discriminative, motivational-affective and cognitive-evaluative dimensions (Melzack- & Casey, 1968).

- The 'sensory-discriminative dimension' refers to the location, intensity, duration and quality of pain.
- The 'motivational-affective dimension' refers to the unpleasant experience of pain such as a feeling of nausea or a sickening feeling.
- The 'cognitive-evaluative dimension' refers to a patient's beliefs, which may arise from their previous pain perceptions and, as such, have a negative or positive effect on the present pain experience.

3.8.1 The biomechanical origin of pain processing in the development of ISMS dysfunction

There is a wealth of experimental data that confirms that ligaments, muscles, neural tissue, joints, annulus of the discs, as well as connective tissue in general, including the specialised TLF of the lumbar spine, are all potential sources of nociceptive stimuli (Langevin & Sherman, 2006). The researcher distinguishes between primary and secondary ANSLBP. Primary ANSLBP can be caused by the tissues directly within or surrounding the lumbar (L5-S1 or other) motion segments. In contrast, secondary ANSLBP might be the result of (often longstanding) forces working within and surrounding the lumbar motion segments but which are created by biomechanical malalignment of the ISMS elsewhere such as the thoracic or cervical spinal regions often associated with poor posture (Porterfield & DeRosa, 1991).

The researcher hypothesises that the unrelenting pain experienced by patients with CNSLBP can be due to, amongst other causes, low grade persistent mechanical strain on nerve, nerve roots and ANS chain. As the biomechanical rotational force on the ISMS dysfunction involves more and more tissue structures or increases, the ongoing irritation of nociceptors causes enduring sensitisation of the peripheral nerve as well as in the dorsal horn (central sensitisation) and altered pathways in the brain. The researcher further reasons that although there is no structural damage or tissue pathology identified in patients with CNSLBP, the unrelenting pain that they experience could possibly be due to constant irritation in the tissues and the neural system as a result of the rotational force.

When stimuli from the various origins (joints, synovia, muscles, ligaments, loose and specialised connective tissue such as the TLF) become more longstanding, peripheral and central sensitisation at the level of the dorsal horn occurs due to the biomechanical rotational strain of the ISMS. These stimuli are received and also interpreted in the sensory-discriminative area (thalamus) as well as by the limbic or emotional aversive area in the brain (Figure 3.11) (Kuner, 2010). Pain perception in this instance is the result of physiological conduction of impulses along a structural network (Figure 3.11).

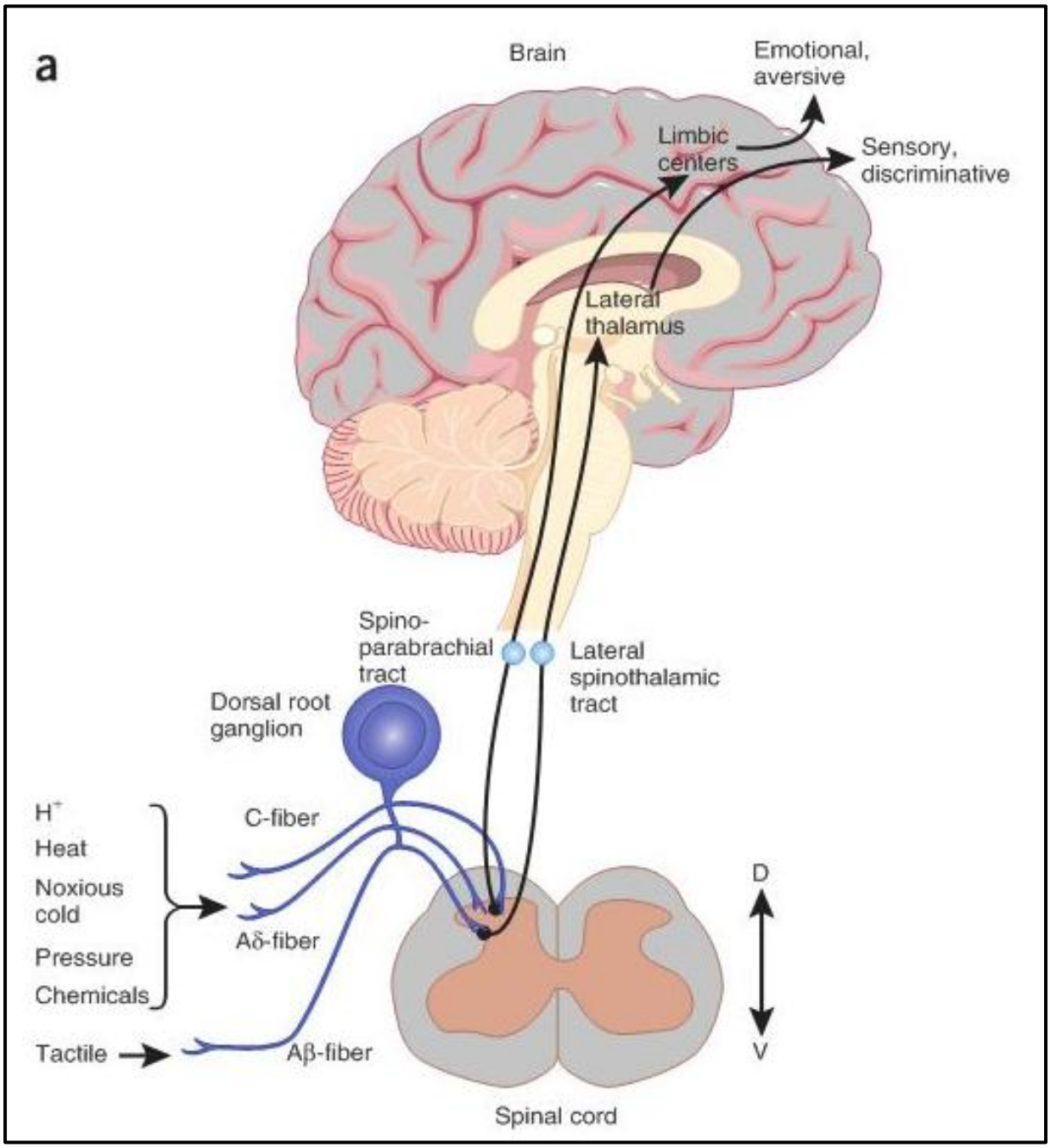


Figure 3.11: A schematic presentation of the circuits that mediate physiological pain

(Kuner, 2010 p 1259)

Depending on the type of nociceptive receptor activated, the stimuli ascend in the contralateral spinothalamic tract (STT) *or* the direct connection to the medulla and brain stem via the spinoreticular tract (SRT), spinomesencephalic tract (SMT) and the hypothalamus via the spinohypothalamic tract (SHT). The ventral posterior

nucleus of the thalamus receives input from the low threshold and wide dynamic range (WDR) nociceptors via the laminae IV-V, which in turn projects input to the somatosensory cortex (S1) (Figure 3.12).

Spinal projections to the ventrolateral medulla, parabrachial nucleus, periaqueductal grey (PAG) and brain-stem reticular formation are also found. Cortical and subcortical areas found to be commonly activated by the nociceptive stimulation are the anterior cingulate cortex (ACC), insula, frontal cortices, the primary somatosensory cortex (S1), secondary somatosensory cortex (S2) and the amygdala. These areas are also referred to as the 'pain matrix' (Brooks & Tracey, 2005). The pain matrix is subdivided into two parts: a medial and a lateral pain system. This distinction between the two parts of the thalamus is a gross distinction based on the projection sites from the thalamus to the cortex and on grouping the brain regions with similar roles in pain perception.

It is thought that the S1 and S2 (lateral pain system) play a role in discriminating the location and intensity of painful stimuli while the ACC is involved in the affective (cognitive-evaluative) component of pain (Brooks & Tracey, 2005).

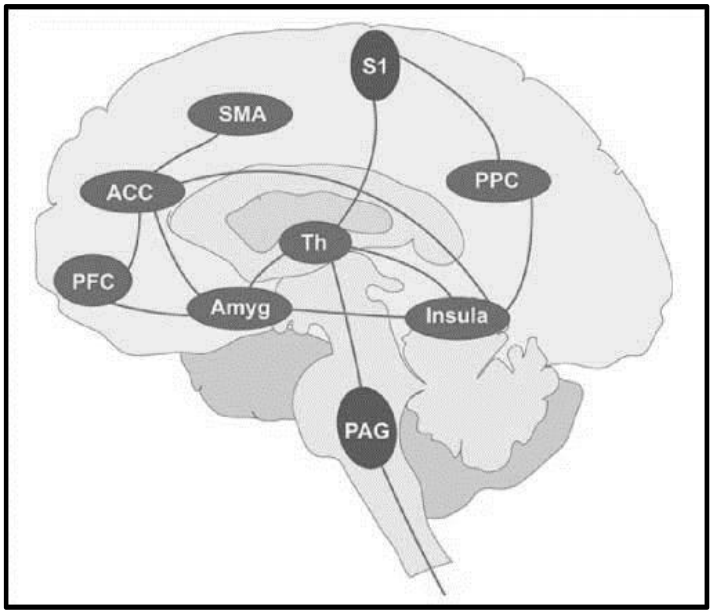


Figure 3.12:Thalamus (Th), the amygdala (Amyg), the insula cortex (Insula), the supplementary motor area (SMA), the posterior parietal cortex (PPC), the prefrontal cortex (PFC), the cingulate cortex (ACC), the periaqueductal grey (PAG), the basal ganglia and cerebellar cortex (not shown) and the primary (S1) and secondary (S2, not shown) sensory cortex

The insula also plays a role in affective pain processing and encodes the intensity and laterality of painful stimuli and thermal non-painful stimuli. The insula therefore integrates stimuli from both the medial and lateral pain systems in the thalamus (Brooks & Tracey, 2005).

Activation of the operculum, which includes the S1 and the insula, is strongly implicated in studies on pain and these are the only cortical areas that, when they are stimulated by direct electrical stimulation, result in pain perception. If the stimulus is short lived, the neuronal response of pain perception will be short lived (Brooks & Tracey, 2005).

The neural adaptive changes in the amygdala (area involved in the emotional state of pain perception) can also exert a powerful inhibitory influence on the prefrontal cortex, which results in interference in a person's cognitive and decision-making ability. If the inhibitory effect of the amygdala on the prefrontal cortex has been established, it will negatively influence the understanding and management of pain and quality of life of the patient with chronic pain. The amygdala is seen as part of

the limbic system, in which the emotions of a person are controlled. Bombardment of the limbic system results in hypervigilance of the limbic system, which is characteristic of patients with chronic pain.

In the presence of hypervigilance, CNS sensitisation because of anxiety and/or emotional stress, beliefs and altered pathways in the brain enhances peripheral and central sensitisation (Field, 2009).

Pain stimuli are caused by chemical irritation due to inflammatory reactions following tissue damage. While it is very difficult to validate experimentally, the mechanism seems to involve the direct stimulation of nerve endings by chemicals, such as hydrogen and potassium ions, or proteolytic enzymes that are liberated from inflammatory cells or damaged tissue cells.

Immune cells and microglia interact with neurons to alter pain sensitivity and to mediate the transition from acute to chronic pain (Ren & Dubner, 2010). When an injury (strain or sprain/microtrauma) occurs, local immune cells are activated and blood borne immune cells, which sensitise the peripheral nociceptors, are recruited to the area.

> *Through the synthesis and release of inflammatory mediators and interactions with neurotransmitters and their receptors, the immune cells, glia and neurons form an integrated network that coordinates immune responses and modulates the excitability of pain pathways. The immune system also reduces sensitization by producing immune-derived analgesic and anti-inflammatory or pro-resolution agents.* (Ren & Dubner, 2010 p 1267)

Langevin and Sherman (2006) found that activation of nociceptors by itself can contribute to the development or worsening of an inflammatory response in connective tissues. In the presence of inflammation hyper-excitability of the peripheral nociceptors occurs, which may result in the patient presenting with primary hyperalgesia (Brooks & Tracey, 2005).

When the central sensitisation occurs, painful stimuli are amplified (due to factors that are not clear yet) and cause peripheral nerves (not normally associated with evoking painful stimuli) to evoke pain. This centrally evoked pain response is called secondary hyperalgesia and is a phenomenon where mechanical non-painful stimuli on the normal skin around the site of primary injury cause a painful stimulus (Brooks & Tracey, 2005). According to Kuner (2010), hyperalgesia is associated with expansion of the peripheral receptive field of neurons, which may cause hyperalgesia to spread to originally unaffected regions.

In a process similar to secondary hyperalgesia, damage to the peripheral nerve induces plastic changes in the CNS, which are maintained by continuous discharge from the damaged afferent, and recruitment of low-threshold mechanoreceptors such as the Aβ fibres, resulting in allodynia (Figure 3.13). Allodynia is associated with withdrawal behaviour in the presence of innocuous stimuli (Brooks & Tracey, 2005).

As the ISMS dysfunction progresses over time so that chronicity sets in, all soft tissues (under strain including the myofascia, TLF and the peripheral dorsal segmental nerve roots and the ANS ganglia; the joint capsules and ligaments of the synovial joints and the annulus of the discs) are compromised in the rotational strain occurring in the ISMS. The repetitive nociceptor drive in the pain cycle causes the loose unspecified and specialised connective tissue structures to stiffen and muscles to shorten as a result of muscle spasticity, taut bands and trigger points. As fascia within and around the nerve roots and sympathetic ganglia of the ANS chain might also stiffen and result in autonomic symptoms, the patient might complain of local lumbar as well as widespread pain and a sickening feeling.

3.8.2 The neuromatrix as part of the ISMS dysfunction

In an ongoing stimulation of the nociceptors (such as in patients with ISMS dysfunction) of the different types of tissue within and surrounding the motion segment(s), neural pathways in the brain change as a result of the process of neural plasticity. When somato-sensory representations in the somato-sensory cortex are stimulated continuously, over time this generates altered processing, which could

lead to the spread of pain and cause various abnormal motor perturbations (Butler, 2000). Abnormal motor perturbations occur probably due to the excessive somatosensory representation of muscles such as erector spinae and multifidus to involve the motor representation of these muscles in the motor cortex through a process termed ‘smudging’. Smudging is associated with compromised activity of these muscles.

Compromised movement (deep segmental as well as multisegmental muscular system dysfunction) will lead to compromised muscle recruitment and fatigue and generalised adaptation of spinal malalignment. On the basis of clinical reasoning the researcher hypothesises that paraspinal muscles have discrete representation at the motor cortex, which may be compromised in patients with LBP (Hodges, 2013). Smudging may be even more likely in the case of patients with CNSLBP.

When the neural pathways in the brain (neuromatrix) change, patients complain of spontaneous ongoing pain, which is initiated by these altered pathways in the brain. These altered pathways can play a key role in the development of chronic pain and in the clinical manifestation of chronic pain (Kuner, 2010; Flor *et al*., 1997). So, the process of plasticity through which the altered pathways in the brain and nervous system has developed is a powerful mechanism for manual therapists to use in dealing with CNSLBP (Butler, 2000).

The macroscopic anatomical changes in the brain (ACC, orbitofrontal cortex, insular cortex, dorsal pons) associated with chronic pain entail a decrease in the grey matter of the brain. What is not clear in the literature yet is whether the changes in the macroscopic grey matter are due to the chronic pain state of the patients or whether the patients’ chronic pain state causes the macroscopic anatomical changes in the brain (Kuner, 2010). Of clinical importance in patients with CNSLBP is the fact that decreased brain volume (grey matter) is also associated with depression, which may be an explanation for the frequent reports that patients with CNSLBP suffer from depression.

Field (2009) states that:

> *Hyper-vigilance in the limbic system has been described as a partial explanation of chronic pain … patients with chronic pain have a heightened sensitivity to pain (lower threshold and tolerance) because of increased attention to external stimulation and a preoccupation with pain sensations; states that are mediated through limbic activity. Limbic dysfunction also manifests as an abnormal efferent innervation of musculature, both visceral and somatic. The musculature undergoes tonic contraction as a result of limbic efferent stimulation which may generate a further sensation of pain.* (Field, 2009 p 48)

3.8.2.1 Plasticity in the neural system

The development of chronic pain occurs through a process of plasticity that takes place in the neural and the soft tissues. Plasticity in the neural system occurs in different forms at different levels: the molecular, synaptic, cellular and network levels. These changes are graphically illustrated in Figure 3.13.

When the physiological pain perception persists or endures, changes take place at various levels of the nervous system; i.e. peripheral sensitisation, central sensitisation and altered pathways in the brain.

A change in the strength of synaptic input is mediated by probable changes in neurotransmitter release. Long-term potentiation of nociceptive transmission at all levels of the nervous system (peripheral and central sensitisation as well as the altered pathways in the ACC) is found in patients with CNSLBP, resulting in the affective (cognitive evaluative) component of the pain experience associated with the patient's beliefs, which may arise from their previous pain perceptions. The long-term potentiation at a molecular level is found to be similar to the processes that are probably involved in the formation of memories. Formation of memory (for instance the memory of previous pain, especially if it is associated with unpleasant emotional experiences such as catastrophisation, as well as the fear of pain) is associated with sensitised neurons in the amygdala and the ACC (area in the brain involved in the affective pain control) in patients with CNSLBP (Kuner, 2010).

At the molecular level the molecules may change in an ‘activity dependent’ manner (i.e. phosphorylation) and in this way alter molecular function (i.e. by a decrease in the activation threshold of an ion channel) or localisation (endocytosis or trafficking) (Kuner, 2010). These changes are illustrated in Figure 3.13.

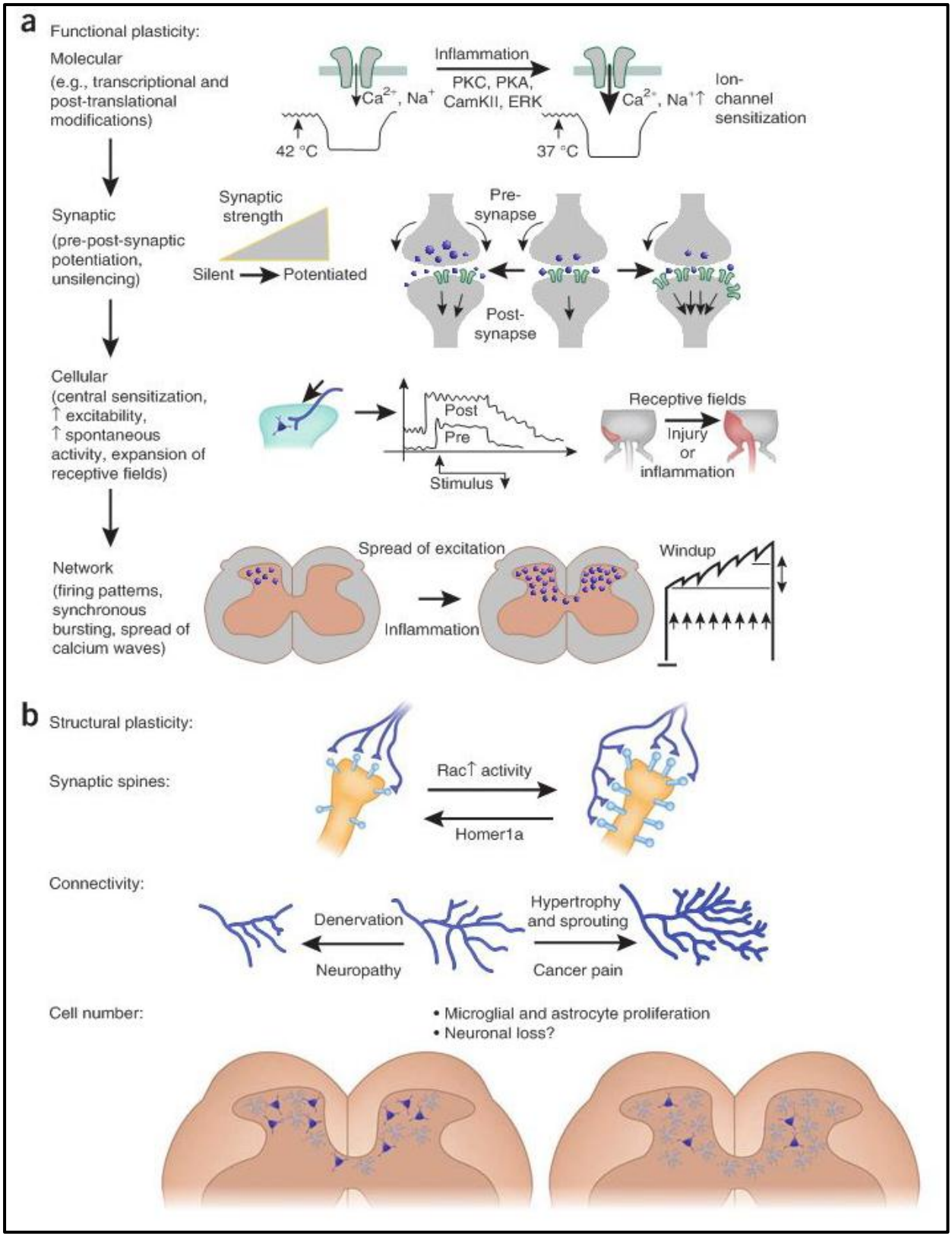


Figure 3.13: Disease-induced functional and structural plasticity in neural substrates of pain on molecular, synaptic, cellular and network levels

(Kuner, 2010 p 1260)

Through a complex process, functional plasticity in the nociceptive pathways gives rise to a process of structural changes, which is detected by a '*change in the increase or decrease in the density of the synaptic spines, degeneration or regeneration of axons leading to aberrant connectivity, degeneration of neurons, and proliferation of asterocytes and microglia which influence nociceptive processing by releasing modulatory substances*' (Kuner, 2010 p1259).

In the end the physiological changes in pain processing lead to structural changes that can become the *origin* of persistent pain as a result of neural plasticity.

> *Plasticity at the level of the neurons in the* ***nociceptive pathways*** *is seen as an increase in the magnitude of responses to a defined sensory stimulus, an increase in the level of spontaneous activity, or after discharges, which represent continued activity after the termination of a nociceptive stimulus, leading to central amplification of pain (central sensitization). Furthermore, the peripheral receptive field of neurons can expand, allowing hyperalgesia to spread to uninjured regions* (Kuner, 2010 p 1259) [bold added by researcher]

The researcher hypothesises that synaptic plasticity and functional plasticity are very important processes in the conceptualisation of the multidimensional management of patients with CNSLBP because they imply that the strengthening of the synaptic connections can be remodulated by decreasing nociceptive stimulation and bombardment of the dorsal horn in a process of desensitisation.

3.8.3 Characteristic adaptive behaviour in patients with ISMS dysfunction

Waddell (2004) links the development of CNSLBP to a timeline in which people who suffer from NSLBP after 12 weeks become chronic. Field (2009), however, states that it is not only a time factor (Waddell, 2004) that determines the development of CNSLBP, but that it depends on the variation of an **individual's response** to an episode of acute NSLBP: … '*some people are set up for chronicity before the pain starts; others develop it soon after the ANSLBP; a third and possibly the largest group drift into it after suffering for a month or so*' (Field, 2009 p 47).

From the earlier discussion in this section it is clear that pain is a multidimensional sensation, which consists of sensory-discriminative, motivational-affective and cognitive-evaluative dimensions (Melzack- and Casey, 1968). The intensity duration and quality of nociceptor stimuli are therefore not only localised in the brain but also associated with a sickening feeling or a feeling of nausea due to the motivational-affective dimension and are cognitively interpreted based on previous pain experiences.

In Section 3.5 discusses the development of ISMS dysfunction from a biomechanical perspective and indicates a possible mechanism through which the torsional strain, fixation of the trunk (guarded movement) and adaptive and/or compensatory movement patterns may develop. These patterns are caused by the neuromuscular responses to pain and the stiffening of the fascia as well as by the fear of pain and injury (fear avoidance behaviour) associated with the motivational affective dimension of pain or are driven by cognitive control of the patient (O'Sullivan 2005). Guarded movements are identified by the fact that the patient holds their body/posture rigid to prevent movement as far as possible to the extent that even their breathing pattern is restricted and shallow (Langevin & Sherman, 2006). The presence of guarded movements is closely associated with psychological processes. Guarded movements may start as a reflex physiologic response to injury or as a primary dysfunction, but may persist due to psycho-physiologic rather than physiologic processes alone. Guarded movements become a learned, protective habit and then persist as physiologic dysfunction associated with abnormal patterns of muscle activity, movement and neurophysiologic activity (Figure 3.14).

Catastrophising is defined by Field (2009 p 47) as the: *'development of inappropriately negative beliefs and understanding relating to pain and its possible consequences'.* Catastrophisation '…. *is felt to be a precursor to pain related fear; with fear going on to cause avoidance behaviour, reducing activity and resultant disability*'.

The development of guarded movement and fear avoidance behaviour can set off the development of unwell/illness behaviour (Field, 2009; Waddell, 2004).

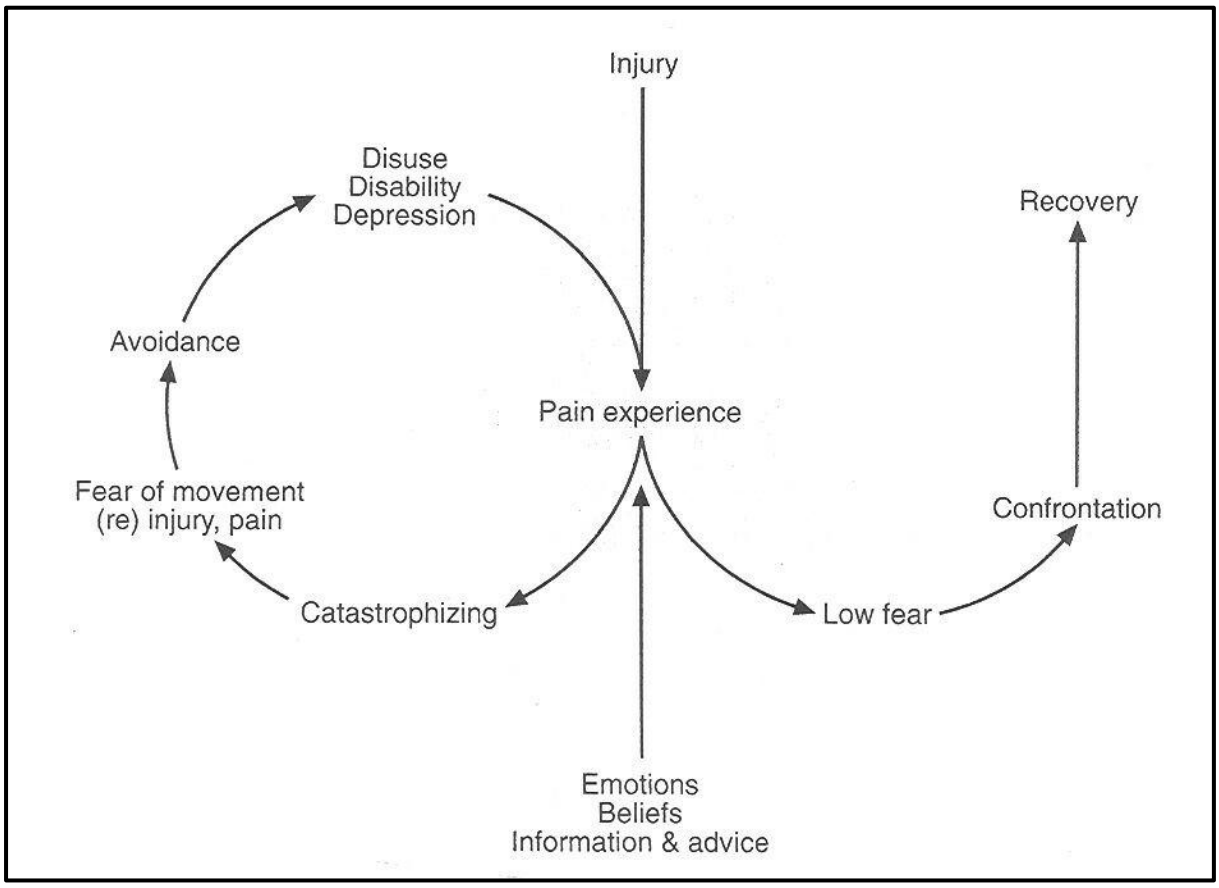


Figure 3.14: The fear avoidance model

(Waddell, 2004)

Hadjistavropoulos and Craig (1994) state that 10% to 15% of patients develop emotional and behavioural problems out of proportion to their physical problem at an early stage after the acute phase of NSLBP followed by CNSLBP.

Emotional and behavioural problems culminate in clinical psycho-social characteristics, such as older patients (50% to 55%) who have a previous history of back pain and show signs of catastrophisation and fear avoidance behaviour. They have a poor relation between pain intensity and functional ability (i.e. low pain level with severe disability); poor health perception; psychological distress and

depression; signs of decreased mental health, musculoskeletal conditions; and other comorbidities.

Emotional distress further heightens (lowers the threshold for pain impulses) the patient's pain perception because it increases the patient's awareness of bodily sensations and lowers pain tolerance.

Socio-demographic characteristics include: time since last worked; occupational status (no longer employed); employment rate; educational level; expectations about return to work; job (dis)satisfaction; and duration of sickness absence. Underlying these psycho-social and socio-demographic characteristics are the patient's beliefs about hurting and disease and the fear of experiencing them, personal responsibility and taking control of circumstances in their life and self-sufficiency. The patient's beliefs and expectations regarding the treatment of their condition have a major influence on the successful outcome of the treatment. The patient's experience of their own condition is influenced by stories told about the condition by their peer group or people who have a similar condition.

Psychological factors are seven times more predictive that symptoms will last for a long time than physical tests or other factors identified from the patient's case history. Catastrophisation also influences the patient's coping mechanisms and beliefs on issues that affect health care.

Illness behaviour is an indication of the severity of the patient's problem but it may also reflect the psychological factors mentioned above to such an extent that it is more important than the physical signs and symptoms. Physical function, performance and illness behaviour are inseparable.

Social behaviour and social factors that influence the development of CNSLBP are closely interrelated. The patient's sick role and illness behaviour are typical social phenomena. The patient's social networks influence their beliefs as well as their coping strategies and illness behaviour. The nature, strength and availability of a

patient's social networks either reinforce or discourage illness behaviour (Field, 2009). Pain and pain behaviour are a strong way of communicating with other people and with health care professionals (Waddell, 2004).

Social factors that play a role in the chronicity of a patient's clinical picture are listed by O'Sullivan (2005) as the patient's relationships in all spheres of life (family, friends or at work), work structure, support structures, cultural factors, medical advice, compensation and socio-economic factors.

One of the strongest influences on return to work and work status at six to 12 months post the episode of acute LBP is the patient's own perceptions of their pain. Return to work is determined by the patient's beliefs about what has happened to their backs, by whether they think that their back pain was originally a work-related injury, and about their fear of re-injury if they return to work (Waddell, 2004).

The development of a 'dysfunctional ISMS' based on the process of plasticity in the soft and neural tissues as discussed in the preceding sections implies that ISMS dysfunction is potentially reversible because of the inherent plasticity in the systems of the ISMS. Apart from this physical or physiologic loop, feedback and reinforcement of behaviour play a role in the condition. What we do, our activity level and illness behaviour all reinforce our beliefs about the pain and the coping strategies we use to deal with it. Illness behaviour, disability and sickness absence reinforce distress and depression, which increase illness behaviour and reduced activity, and aggravate and perpetuate physiologic dysfunction and deconditioning.

Field (2009) describes a conceptual framework of the interrelated factors involved in maintaining LBP. The conceptual framework displayed below is adapted from Field's original framework to include the role of social interaction as an important factor in CNSLBP.

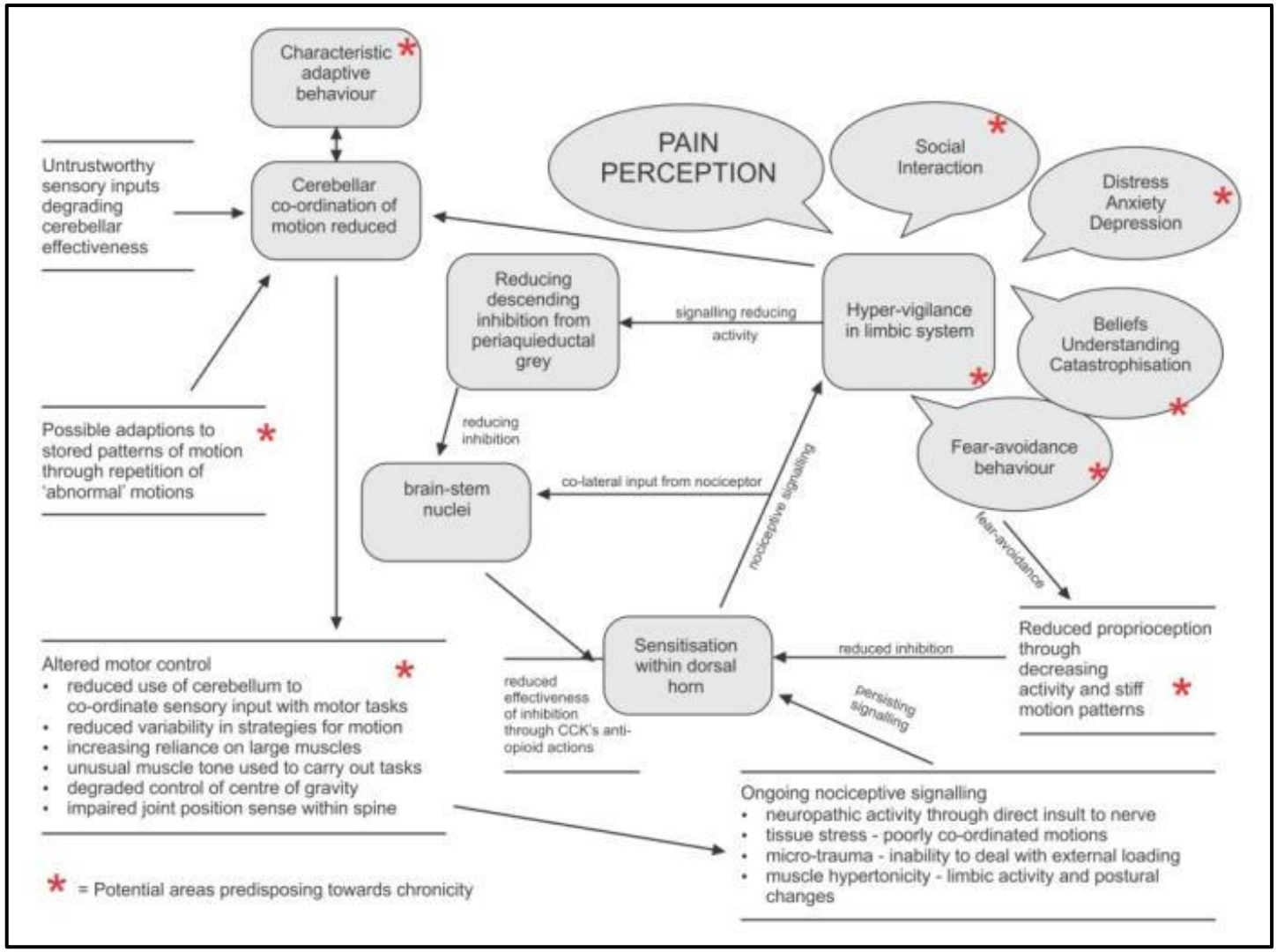


Figure 3.15: Interrelated processes involved in generating and maintaining ISMS dysfunction

(Adapted from Field, 2009 p 49)

3.8.4 Pain modulation

Pain modulation can entail either pain-enhancing mechanisms *or* descending pain inhibition (modulation).

3.8.4.1 Pain-enhancing mechanisms

The perception of pain intensity in relation to the peripheral stimulus that causes the pain depends on many factors such as the level of arousal, anxiety (in which fear avoidance behaviour plays a major role), depression, attention and expectation or anticipation and guarded movement. '*These "pyschological" factors are in turn regulated by overt and covert information, as well as more general contextual cues that establish the significance of the stimulus and help determine an appropriate response to it'* (Brooks & Tracey, 2005 p 24). Studies found that anxiety-induced (anticipation of) pain resulted in a different increase in brain activity than the brain activity generated by a large nociceptive drive. Similar results were shown in studies during which it was found that attention to and anticipation of an upcoming painful stimulus activated the anterior insula (AI) (Brooks & Tracey, 2005).

An inflammatory process plays a major role in pain enhancing as is described in Section 3.8.1.

A person's genetic make-up, previous experience and learning determine how pain stimuli are filtered and modulated through an individual's whole being, current physiological status, emotional state and socio-cultural environment (Turk, 2001*). 'Sensitization may be both neurophysiologic and psychological'* (Eriksen & Ursin, 2002. There is growing evidence that inherited (genetic) factors play a role in the increased incidence of individuals that develop severe or enduring CNSLBP (Mishra *et al*., 2007). Current research implies that there is a genetic link that predisposes vulnerable individuals towards the production or not of an endogenous muscle relaxant, and a predisposition towards the development of major depression when back pain occurs. There is also convincing work on the role of genetic influences affecting disc degeneration (Battie, Videman, Levalahti, Gill & Kaprio, 2007).

Another contributing factor to the persistence of pain and the widespread nature of CNSLBP is a change in the descending pain modulation mechanism. The parabrachial nucleus, PAG, the brain stem and reticular formation, which are known as the descending pain control pathways, can either inhibit or facilitate (modulate) nociceptive transmission and subsequent pain perception (Brooks & Tracey, 2005). In an enduring pain state, as indicated above, the gene expression of the CCK and its receptor protein within the dorsal horn increases. The increased CCK inhibits the effectiveness of opiates released by the descending pathways and, as such, decreases the effectiveness of second order pain inhibition, resulting in the persistence of pain perception (Field, 2009).

The functional changes at the molecular, synaptic, cellular and network levels due to peripheral and central sensitisation as well as hyperactivity in the neural pathways in the cortex midbrain and brain stem result in altered pathways in the higher centres. Over time, as chronicity sets in, these altered pathways become structural changes at these levels.

In a chronic pain state brain areas involved in emotion such as the ACC, insula, and the amygdala also become hyperactive. When this hyperactivity in these areas occurs, peripheral noxious stimuli will not only activate the sensory areas in the brain but also activate in the brain all the areas involved in emotion (ACC, insula, and the amygdala). The pain responses in the ACC are powerfully modulated by a person's mood, placebo and hypnotic suggestion, which indicates that the ACC plays a role in the integration of sensory input with a person's emotional state (Brooks & Tracey, 2005; Field, 2009). The memories of previous pain episodes if reinforced through beliefs and thinking can easily evoke pain (Bouton, 2002). Memories of previous episodes of back pain can lead to chronicity when previous pain memories are reinforced.

Catastrophising, which is the person's expectation, or fear, that pain will be severe and unmanageable, is the likely reason for the development of the affective disturbances associated with chronic pain (Waddell, 2004). Brooks and Tracey (2005) report that the anticipation of pain activates the rostral anterior insula and

medial prefrontal cortices, in contrast to the fact that during nociceptive pain stimulation activation of the insula is more caudal and the expected activity in the prefrontal focus is replaced by activity within the ACC.

Cognitive factors such as fear of pain seem to play a greater role in the development of chronic pain than factors such as pain intensity. It has also been established that psycho-social factors play an important role in chronic pain disorders such as CNSLBP. On the other hand, personality disorders may play a role in a person's ability to develop coping skills to deal with pain and, as such, a lack of developing coping skills can be an antecedent to chronic pain.

Kuner (2010) also explains that cortical thinning due to loss of cortical grey matter occurs in patients with chronic pain, which results in a person experiencing problems with attention and concentration. Cortical thinning is associated with depression in patients with CLBP. Whether it can be viewed as a pain-enhancing mechanism is not clear but one can argue that because depression is strongly associated with the hyper-vigilant limbic system it will enhance pain perception. Wand, Parkitny, O'Connell, Luomajoki, McAuley, Thacker and Moseley (2011) state that (chronic) pain is maintained by changes within the brain of a person with chronic musculoskeletal pain. This finding is in line with the fact that Ploghouse, Narain, Beckmann *et al*. (2001) found that pain induced by mood, emotional stress and/or depression activates different areas in the brain than pain perception from a nociceptive drive.

3.8.4.2 Pain-inhibitory modulation

The ascending STTs send impulses to the PAG via the SMT. When the PAG matter in the midbrain is activated, enkephalin-releasing neurons that project to the raphe nuclei in the brain stem are activated. The nuclei raphae release serotonin to the dorsal horn, where it has an excitatory connection with the inhibitory interneurons in the substantia gelatinosa. When the inhibitory interneurons are activated they release enkephalin or dynorphin (endogenous opioid neurotransmitters), which bind to the mu-opioid receptors on the axons on the A-delta and C fibres that carry incoming nociceptive impulses. The activated mu-opioid receptors inhibit the release

of substance P, which in turn inhibits the activation of the neuron from where the pain impulses are transmitted via the STT to the ventroposteriolateral nucleus of the thalamus. The nociceptive impulse is therefore inhibited (because the pain threshold for mechanical pain is increased) before it can reach the cortical areas responsible for the interpretation of pain.

The researcher associates this change in the role of the ACC, insula and amygdala with the development of characteristic adaptive behaviour displayed in patients with CNSLBP.

3.9 Conclusion

In this chapter the characteristics of the normal ISMS were deduced based on an overview of the functional biomechanics of the spine and head, shoulder and pelvic girdles as discussed in Section 3.6.

The rationale behind the conceptualisation of the characteristics of the *normal* ISMS is to give an overview of the functional biomechanics of the spine, head and pelvis as a close kinematic chain with the shoulder girdle that can function as a closed or an open kinematic chain (ISMS) and to deduce the principles that should guide the management of patients with CNSLBP. The discussion of the normal ISMS is followed by a presentation of the pathophysiological processes closely associated with CNSLBP. From the discussion of the possible mechanism of the development of ISMS dysfunction it is clear that three components of ISMS dysfunction in patients with CNSLBP can be identified: biomechanical, pain processing and characteristic adaptive behaviour. Characteristic adaptive behaviour is strongly associated with a biomechanical and neurological component inherently part of the pain processing which culminate in, or can be driven by psychosocial factors. Characteristic of ISMS dysfunction is that the biomechanical component can be driven by pain processing and/or the characteristic adaptive behaviour component of ISMS dysfunction.

In concluding the discussion on pain processing as a driving factor in the development of ISMS dysfunction, the preceding paragraphs indicate that pain

processing is a complex combination of neurophysiological processes that take place at peripheral and central levels as well as in the higher centres in the brain. These neurophysiological processes result in pathological functioning of the CNS, ANS and PNS in driving the pain processing and abnormal movement, the biological component of the ISMS dysfunction and the characteristic adaptive behaviour.

Clinically it is apparent that some people are set up for the development of chronic pain before the pain starts, while others develop it soon after the acute episode of NSLBP and others drift into it (Field, 2009). Typically the largest group of patients with CNSLBP drift into chronic pain after trying various available treatment options which do not address their problem adequately.

The researcher hypothesises that the process of ISMS dysfunction, which is characteristic of CNSLBP, develops over a period of time. This hypothesis correlates with Waddell's (2004) view that ANSLBP develops progressively over a time period of 12 to 15 weeks into CNSLBP (Figure 3.16) in a process marked by recurrences.

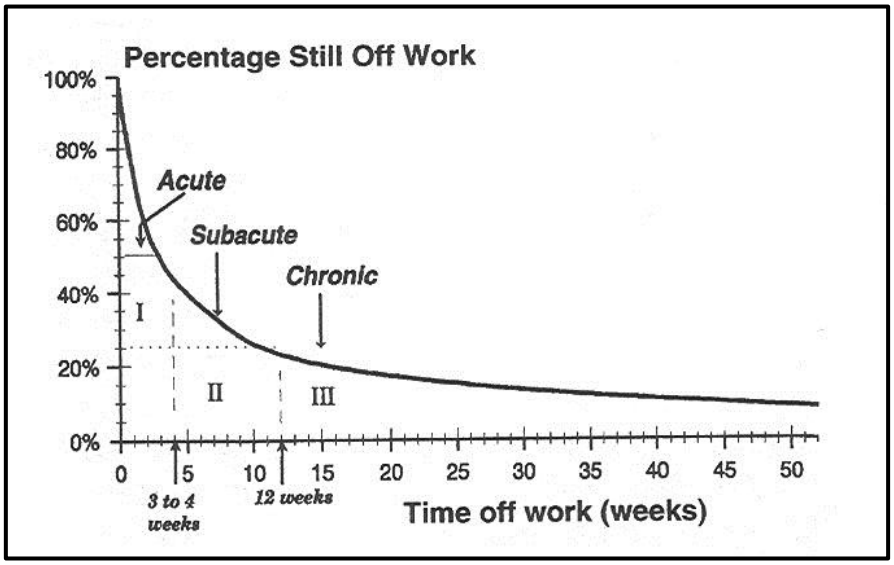


Figure 3.16: Three stages in the development of chronic non-specific low back pain

(Waddell, 2004 p 123)

The development of ISMS dysfunction occurs through a process of plasticity, which over time results in a chronic biomechanical, neural (as a result of pain processing

and/or cortical thinning) and psychological state that is characterised by behaviour typical of a chronic condition. This complex neural network in patients with chronic pain results in altered pathways in the brain that become sensitised to the extent that they can initiate a chronic pain state. The processes are influenced by anxiety, depression, anticipation (fear of pain/fear avoidance and guarded movement), and cognition.

By the time that a patient is diagnosed with CNSLBP a varying degree of chronicity has set in, which depending on the patient can be reversed to a great extent with cooperation from the patient through intensive multidimensional manual therapy management.

Patients who present with a severe degree of chronicity require a specialised aggressive multidisciplinary approach to management by all relevant disciplines to assist them in coping with their condition (Buchner et al., 2007).

The rationale for the multidimensional manual therapy management of patients with CNSLBP therefore would be to desensitise the peripheral and, as such, also central nociceptive activation through a process of release of soft tissues and joint restrictions. Deactivation of the central nervous system in patients in whom the learned memory for pain and its behavioural responses has become sustained, manual therapy together with verbal instructions/ explanation/ education can extinguish the associated learned pain memory (Zusman, 2007). Deactivation of nociceptive stimulation through soft tissue and joint restrictions will also result in decreased sensitisation of the neuromatrix in the brain. A decrease in the hypervigilance in the neuromatrix is achieved by influencing the patient's negative cognitive beliefs to becoming more positive beliefs and by decreasing the influence of pain-enhancing emotional factors by decreasing the effect of the patient's fear avoidance and hypervigilance through education and retraining of motor control. The manual therapist can use relevant education, reassurance and retraining of motor control to empower the patient to manage their condition through cognitive and behavioural control. In this way the effect of the negative pain memories and fears can be addressed by the therapist. This finding of the researcher coincides with the

findings from the randomised clinical trial by Fersum *et al.* (2012). Although the altered pathways in the brain cannot be extinguished through treatment, they can be made less active. At the same time conduction through alternative pathways becomes more dominant through the process of peripheral desensitisation of the nociceptor drive as well as higher centre (cognitive) control over behavioural aspects based on knowledge and understanding of the condition.

The characteristics of ISMS dysfunction typical of patients with CNSLBP is summarised in the following text box to serve as principles for the discussion of the assessment and management of patients with CNSLBP in Chapters 4 and 5 respectively.

Characteristics of ISMS dysfunction typical of patients with CNSLBP

- The initiating factor in the development of ISMS dysfunction is abnormal loading of the lumbar spine resulting in unilateral muscle spasm in the local and global muscular system. . As the muscle spasm shortens upwards in the multisegmental spinal system it generates an imbalance within the entire multisegmental kinetic chain, which over time can incriminate the entire system.

- This shortening of the connective tissues generates strain on the motion segments and can involve the functioning of the multisegmental kinetic chain. Nociception drives the ISMS dysfunction because it causes and aggravates the muscle spasm to spread.

- Because loose and specialised connective tissue forms a network throughout the body it shortens as a result of inactivity, microtrauma and inflammation. In the ISMS it affects local and global muscle recruitment, which promotes soft tissue shortening and encourages ISMS dysfunction. At local and global levels the connective tissue system (especially in the ISMS) is strained and becomes a major source of nociception.

- In the presence of inflammatory mediators (prostaglandins and bradikinin) growth factors and hormones such as adrenaline influence the sensory input to the nervous system.

- A history of recurrent episodes of ANSLBP may indicate the potential presence of soft tissue fibrosis, which is a result of joint degeneration.

- The passive ligamentous system is strained due to the torsioning of the ISMS dysfunction and is a potential cause of local/global instability and a major source of nociception.

- The neural system's (PNS, ANS and CNS) control of the musculoskeletal system is adapted because of the nociceptive bombardment of the sensory receptors, resulting in abnormal sensory strategies which drive the malrecruitment of local and global muscles.

- The connective tissue system is an integral part of the neural system (PNS, ANS and CNS) and when shortening develops the PNS and ANS will be biomechanically affected by the interconnected connective network of connective tissue throughout the body. Shortening of the connective tissue mechanically limits the neural system's biomechanics to become a major source of noxious stimuli.

- ANS responses in patients can be a result of the abnormal nociceptive bombardment, emotional stress/depression and inflammatory tissue responses, which will probably increase the sympathetic activity and which should be considered in the management of patients.

- The shoulder and pelvic girdles and the head structurally and functionally influence the development of ISMS dysfunction. The shoulder and pelvic girdles and head therefore are integral parts of ISMS dysfunction.

Characteristics of ISMS dysfunction typical of patients with CNSLBP (continued)

- ISMS dysfunction affects the strain on the structures of the thoracic spine and ribcage and as such affects the breathing pattern and tidal volume. The costovertebral, costosternal and costochondral joints are major nociceptive sources in patients with ISMS dysfunction.
- Torsioning of the ISMS can be driven by the psychological components (fear avoidance, emotional stress, anxiety, and depression) as well as by configuration of the individual motion segments and the ISMS as a whole and may result result in an incident of unilateral spinal loading causing pain and muscle spasm.
- Chronic pain is generated and driven by a dynamic integrated interaction between the biological, pain processing and the CNS. This interaction is influenced by social stressors.
- A person's sedentary lifestyle reinforces his/her/their lack of movement due to general stiffness and muscle spasm as well as fear avoidance.
- Genetic predisposition influences a person's physical and psychological response to mechanical and environmental influences, which are strong predictors for the development of CNSLBP.
- Patients with CNSLBP present with a combination of ISMS dysfunction, pain processing and characteristic adaptive behaviour and any one of these components can become the primary driver of CNSLBP.

In conclusion ISMS dysfunction is dynamic continuous and over time is progressive without treatment because it is a self-perpetuating cycle within the systems due to the continuous process of plasticity as long as all the components of ISMS dysfunction don't change. Depending on the stage of chronicity, different degrees ISMS dysfunction can be identified: mild ISMS dysfunction can be reversed easier than ingrained longstanding ISMS dysfunction.

ISMS dysfunction is the term used to indicate the dysfunction of the integrated neuromusculoskeletal and psychological systems that forms the essence of the clinical picture of patients with CNSLBP regardless of the origin of the pain.

The clinical picture of a patient with CNSLBP will therefore include ISMS dysfunction as well as other associated signs and symptoms.

CHAPTER 4

The principles of a multidimensional assessment model for patients with chronic nonspecific low back pain

4.1 Introduction

In Chapter 4 the researcher discusses the assessment of the patient with CNSLBP, based on the principles of the multidimensional clinical presentation of a patient with CNSLBP as discussed in Chapter 3.

During the assessment the manual therapist should continuously keep in mind that the interaction between the ISMS dysfunction, pain processing and characteristic adaptive behaviour is a dynamic self-perpetuating cycle in which any of the three components can drive the clinical picture of CNSLBP. The main focus but also the challenge of the assessment, therefore, is that the primary and secondary drivers of the patient's CNSLBP as well as the complex interaction between all three components should be identified in order to plan an appropriate multidimensional management plan for the patient suggested by the researcher.

The premise for the assessment of a patient with NSLBP (it can be acute or chronic) is the bio-psycho-social model (BPS model) (Waddell, 2004; Hodges, 2013). However, the BPS model only gives an indication of the categories in which the patient may experience symptoms. The BPS model serves as a generic model for the approach to management of a patient that presents with (acute or chronic) NSLBP but does not indicate or explain how some people can cope with back pain while others become severely disabled. It is further a model of human illness rather than wellness (Waddell, 2004 p 271).

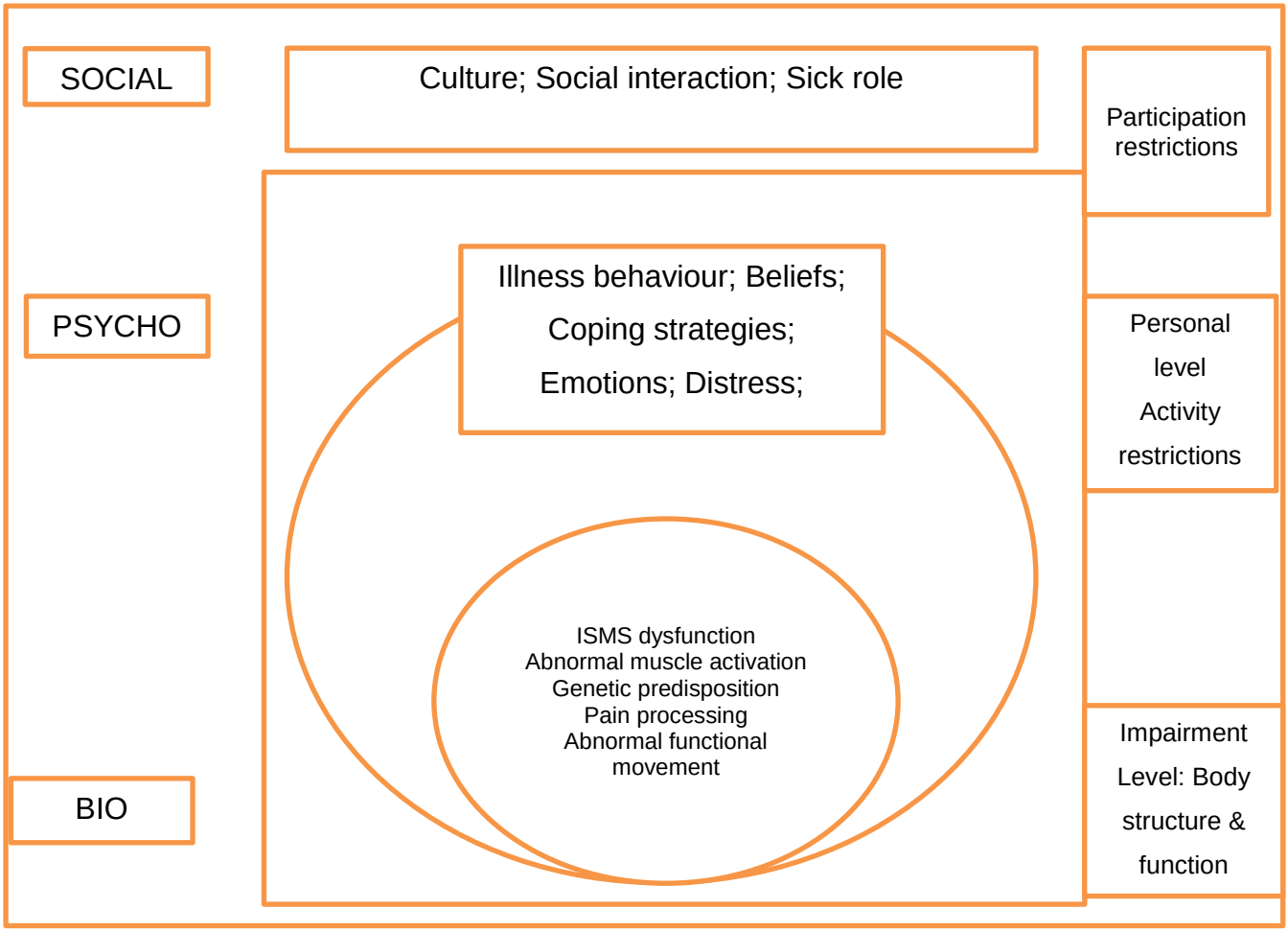


Figure 4.1: A Bio-psycho-social model of CNSLBP

(Adapted from Waddell, 2004 p 272)

Through clinical experience and clinical reasoning the researcher has developed the assessment of the patient, based on the characteristics of ISMS dysfunction.

Typically the history of the patients with (chronic and/or acute NSLBP, posture, functional movement and anatomical structures palpated and emotional, pain or behavioural responses during discussion of the history of the condition, posture and functional assessment as well as the palpation of the structures should assist the manual therapist to identify the primary and secondary drivers of the condition. The therapist is also able to establish the complex interaction between all the contributing factors and 'hidden agendas'.

The differentiation between the signs and symptoms of the primary and secondary drivers of the condition and the interaction between all the factors are not always clear in the case of a patient with CNSLBP and can sometimes only be clarified after observation of the patient's progress, based on management over time rather than a final conclusion after the initial assessment. The conclusion following the assessment of the patient with CNSLBP is a clinical diagnosis and not an evidence-based diagnosis (European Guidelines for the Management of Chronic Non-specific Low Back Pain, 2004).

4.2 Typical clinical appearance of a patient with CNSLBP

Clinically there is no specific distinction between acute and chronic NSLBP (Waddell, 2004). A patient that presents with NSLBP can present with varying degrees and combinations of the following signs and symptoms that may indicate that the patient is developing chronicity. The clinical appearance of the patient described below is based on the researcher's observations during the management of patients in clinical practice.

Typical telltale non-verbal signs that give an indication that the patient has a tendency to develop CNSLBP or has already developed CNSLBP include:

- The patient's posture is 'droopy' (generalised flexion posture), the patient takes small almost guarded steps or lacks the associated arm swing or trunk movements during gait;
- The patient complains of pain in the back and generally elsewhere in the musculoskeletal system; and

- The patient's movements during gait in general are not 'free' and s/he has lost the normal associated flowing movement during gait.

As chronicity becomes embedded in the patient's ISMS towards the more advanced stages of chronicity their clinical appearance becomes one of withdrawal (social withdrawal should be established during the history taking). Advanced stages of chronicity are also revealed in the following symptoms:

- A stooping posture and a facial expression that may indicate a gloomy or negative mindset and/or pain;
- Movement can become so guarded that it is 'locked in' because the patient is terrified to move as it provokes pain. Waddell (2004) calls this disuse syndrome;
- The patient's posture and movement described above may be associated with depression;
- Patients present with a posture that displays a clear torsioning of the trunk that may result in an appearance that the one leg is shorter than the other
- The patient displays feelings of helplessness and hopelessness;
- Owing to the chronicity of the pain and possible involvement of the ANS, the patient can complain of excessive intermittent sweating, nausea, poor sleeping pattern and quality of sleep, light headedness and dry mouth typical of feeling ill. The patient may lose their appetite and show altered breathing due to fear and resulting in decreased lung excursion. The patient often appears generally ill, with limited functional movement such as walking and change in position;
- The patient's complaints of pain do not correlate with the excessive disturbance in their guarded movement. Complaints of pain may decrease although their level of disability may increase;
- The patient's voice may be laboured and monotonous;
- The patient often does not want to be touched because of fear of pain; and
- This patient is full of fear and distrust; they never come to physiotherapy alone but ask someone to bring them to treatment.

4.2.1 History taking

The purpose of the history taking is to distinguish between the dominance of the ISMS dysfunction, characteristic adaptive behaviour, and pain processing as the primary and secondary drivers of the patient's CNSLBP. This approach is in line with the recommendations by O'Sullivan (2011) on the management of patients with CNSLBP.

Specific and non-specific LBP are differentiated through a system of triage. The triage serves as a screening process to distinguish between specific serious spinal pathology or nerve root compression or other 'red flags') and non-specific LBP. If the patient's condition is diagnosed as non-specific LBP, the second challenge is to identify the stage of NSLBP (acute/sub-acute or chronic NSLBP).

4.2.1.1 Triage: Screening of the patient for NSLBP

The Clinical Practice Guidelines on the management of LBP published by the Royal College of Physicians in 2009 recommend an initial triage to facilitate effective assessment and management of the patient's problem. The researcher finds the diagnostic triage described by Waddell (2004) a useful tool to apply in screening the patient to 'identify the red flags' and differentiate between the patient with acute or sub-acute LBP at risk for developing CNSLBP and the patient that presents with CNSLBP (Figure 4.2).

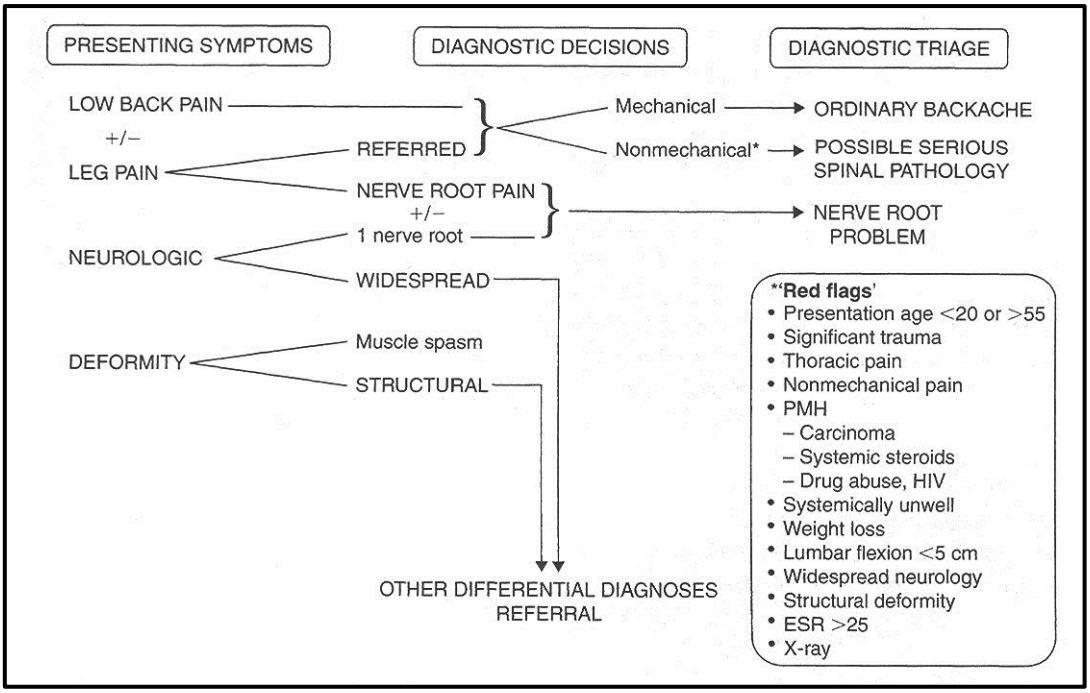


Figure 4.2: Diagnostic triage

(Waddell, 2004 p 13)

In screening for a patient's psycho-social factors (yellow flags) the manual therapist should use appropriate questionnaires such as the Vermont Disability Prediction Questionnaire and the questionnaire for the identification of the patient's risk for developing chronic disability by Linton and Halldèn (1998). This questionnaire assesses not only clinical and psychological factors but also occupational and compensation factors.

A number of screening tools are available for assessing the influence of 'yellow flags', which should be used in combination before a manual therapist is able to predict a patient's risk for developing CNSLBP as a result of a strong influence of the yellow flags (Waddell, 2004). Kendall, Linton and Main (1997) developed a questionnaire to identify the beliefs, behaviours, fear avoidance and activity level, tendency to low mood and withdrawal from social interaction, and expectations regarding treatment in patients that put them at risk for developing CNSLBP.

Waddell (2004) warns that even applying a combination of outcome measures to assess the extent or the role of psycho-social factors as a potential driving factor in the patient's pain perception/pain processing is not an accurate predictor and that each patient should be managed individually, based on their clinical presentation. If positive predictors are identified during the screening process, the therapist should investigate those particular aspects (e.g. fear avoidance) further.

Waddell (2004) describes a combination of socio-demographic, clinical and psycho-social predicting factors that predicts the development of CNSLBP and disability. These factors are listed in Table 4.1.

Table 4.1: Predictors of chronic pain and disability (Waddell, 2004 p 128)

Socio-demographic predictors	Clinical and psycho-social predictors
Gender Age Marital/family status (single parent/young children, partner retired or disabled) Health condition (mental health conditions musculoskeletal conditions, comorbidities) Occupational/educational level Time since last worked Occupational status (no longer employed) Local employment rate	Older age (>50-55) Previous history of back pain Nerve root pain Pain intensity/functional disability Poor perception of general health Psychological distress/depression Fear avoidance Catastrophising Pain behaviour Job (dis)satisfaction Duration of sickness absence Occupational status (no longer employed) Expectations about return to work

During the history taking, the manual therapist should determine the presence of the factors presented in Table 4.1 in the patient's history and observe and interpret the patient's verbal and non-verbal communication regarding their 'LBP' (potential CNSLBP) beliefs, social interaction, and sick role, while enquiring about their physical condition, signs and symptoms, lifestyle, work-related behaviour, and functional performance. The therapist's observations during the history taking should be correlated with the patient's score on the screening questionnaire(s).

Factors that may be identified during the history taking and may indicate the patient's risk of CNSLBP or that they can be clinically diagnosed with CNSLBP are:

- A patient in the sub-acute phase of NSLBP that is not resolving;
- A recurrent attack of NSLBP;
- Poor response to previous therapy/management;
- An acute episode superimposed on an already established CNSLBP;
- Genetic factors (morphology, predisposition for fear of pain);
- ISMS dysfunction during functional activities associated with a higher intensity of pain than expected and environmental factors such as manual labour; i.e. heavy lifting, habitual movements during daily manual tasks, work postures

such as prolonged sitting, and specific job-related demands, which may be mechanical;

- Lack of or limited participation in leisure activities and sports;
- Lack of or limited emotional and physical support, which contributes to inappropriate psychological and social behaviour;
- Cognitive behaviour such as stress responses, provocative coping strategies, and emotional factors such as hypervigilance;
- The underlying dynamic tissue repair process (the patient's response to the tissue repair process is influenced by their psychological frame of mind) (Field, 2009; Waddell, 2004; Adams *et al*., 2002); and
- Symptoms lasting longer than three months.

4.2.1.2 Principles for the (objective) assessment of ISMS dysfunction

The aim of the assessment of a patient at risk of developing CNSLBP or diagnosed with CNSLBP is to determine any malalignment of the spine due to imbalance in the neuromusculoskeletal systems. In essence the objective assessment entails the identification of malalignment to eliminate structural causes such as scoliosis, compensatory postures and movement displayed as guarded movements and pain-provoking coping strategies, and the clinical determination of the possible reasons for and causes of it. The assessment of ISMS dysfunction is totally integrated with assessment of the patient's pain processing and characteristic adaptive behaviour. This behaviour should be correlated with the psycho-social factors such as fear of movement, beliefs and illness behaviour that were identified during the screening of and history taking with the patient.

Finally the aim is to determine the potential reversibility of the local and global shortening and stiffness of the soft tissues that have resulted through a process of plasticity. The patient's neural response on initial palpation to determine the degree of soft tissue stiffness and, potentially, fibrosis and the patient's ability to adapt to correction of postural control may indicate the reversibility of the condition through the process of plasticity.

(1) Observation of the patient's ISMS dysfunction

Observation of ISMS dysfunction entails the integration of the observation of the patient's posture and postural control during functional movement, the patient's pain perception during static posture as well as during functional movement, and the patient's characteristic behaviour during static posture and functional activities.

Assessment of the patient's ISMS dysfunction should also include the identification or confirmation of the risk factors or factors that might have been already identified during the history taking that influence the patient's condition.

Assessment of the patient's clinical picture from the perspective of ISMS dysfunction starts by observation and analysis of the patient's posture to determine baseline markers, from where the patient's movement during functional activities (i.e. gait) can be interpreted. The patient's posture in standing will give the therapist an indication of their postural asymmetry, postural control at the level of the motion segment (local alignment and muscle activation) and global alignment (and global muscle activation that contributes to guarded movements/provocative and avoidance behaviour or strategies). Assessment of posture also includes the limb girdles, shoulder and pelvic girdles, and the position of the head on the cervical spine. All the limb girdles and the head on the cervical spine are inseparably connected to in every aspect of posture and movement.

A patient's posture is seen as an image and expression of the whole patient as described during their history, so the therapist can relate his/her observations to the patient's story of anxiety, frustration, or sheer desperation.

Knowledge block

Grieves (1981) points out that different muscles contain varying proportions of slow and fast muscle fibres. Slow fibres maintain posture; fast fibres give dynamic, voluntary movement (see Section 3.2.1 of the current study). Muscle imbalance gives typical patterns of postural disturbance, which cause abnormal loads on joints and soft tissue structures, abnormal patterns of movement, muscle fatigue, and loss of coordination. In the patient with CNSLBP, the paraspinal muscles are atrophied and contain an increased percentage of fat (Cooper *et al.*, 1992; Mooney *et al.*, 1997). These paraspinal muscles are weaker and fatigue more easily

Hides *et al.* (1994) found local wasting in the multifidus muscle, which is the largest and most medially situated muscle of the erector spinae. The human lordosis is unique because of our upright posture and this involves specific development of the multifidus muscle. The changes were segmental and unilateral, and corresponded to the level and side of symptoms. Hides *et al.*, (1994) found about 30% reduction in the cross-sectional area of multifidus. Because this wasting was so localised and developed so rapidly, they suggested that it was due to segmental inhibition rather than to a general effect of disuse. Even when symptoms settle, multifidus wasting may not recover spontaneously, and this may predispose to recurrent attacks. Hides, Richardson and Jull (1996) showed that specific, localised exercises for the multifidus may not make much difference to symptomatic recovery from the acute attack but do produce better muscle recovery.

(i) Observation of posture

Posture is the alignment of the body based on muscle activity to counteract the force of gravity. In a supported position, such as sitting, posture is assessed during muscle inactivity.

During observation of posture the manual therapist analyses the alignment of the biomechanical component of the ISMS during static posture and postural adaptations during dynamic movement. In essence the assessment of dynamic posture comprises the assessment of the integrated muscle activity that counteracts the force of gravity. In stable posture, regardless of the position, the combined centre of gravity of the various body parts should fall within the base of support. Alignment of the body parts should be maintained to ensure continuous stability in static and dynamic posture. Maintenance of posture can create stress in malaligned joints and poor recruitment of muscle activity. It can also cause muscle atrophy. The influence of the alignment of the limbs on posture, as briefly discussed in Chapter 3, should be taken into account and determined during the assessment of the patient's biological component of ISMS dysfunction. The patient's frame of mind can also be reflected in their posture.

The position of the head on the cervical spine, shoulder girdles in relation to the thoracic spinal region, and the pelvis in relation to the lumbar spine, as well as the position of the hips, knees and feet (in particular the foot arches), should be evaluated (Middleditch & Oliver, 2005; Norkin & Levangie, 2008).

In Figure 4.3 the key points in the observation of a patient's posture are highlighted to illustrate the assessment of posture in the patient with CNSLBP.

The images of the people in Figures 4.3, 4.4 and 4.5 show typical fascial malalignment with dominance in the thoraco-lumbar area. Myers (2011) used the photographs of the people displayed in Figures 4.4 and 4.5 to demonstrate particular patterns characteristic of postural compensations. No history of pathology or signs and symptoms that the people in the photographs may have experienced is given by the author. However, these pictures display the typical postures found in patients

with CNSLBP and are therefore used here to explain the postural observations during the assessment of a patient with CNSLBP.

Observation of posture (1)

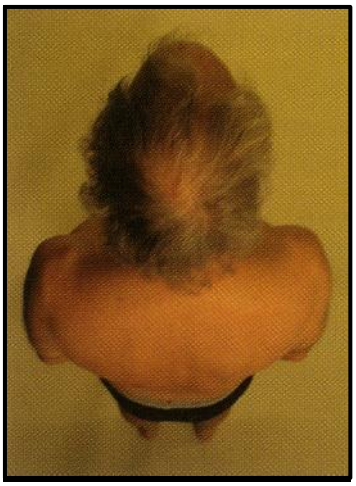


Figure 4.3: An axial view of the position of the shoulder girdle relative to the pelvis due to ISMS dysfunction (observation 1)

(Myers, 2011 p 241)

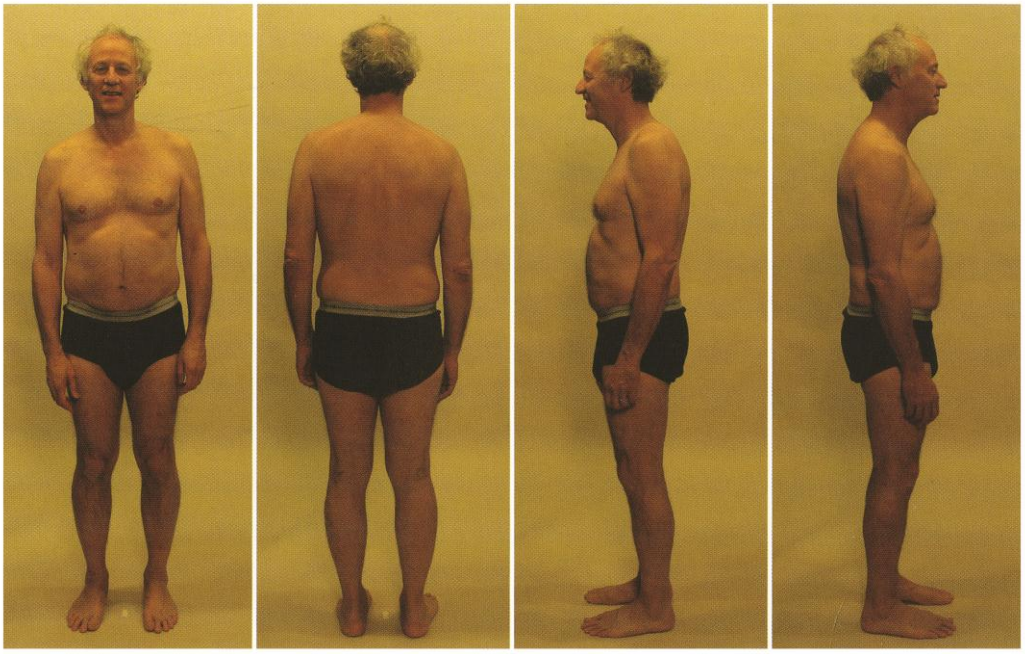


Figure 4.3(b): An anterior, posterior and lateral view of the patients posture typical of ISMS dysfunction

(Myers, 2011, p 241)

The researcher's approach to assessment of a patient's posture is to start observing at the thoraco-lumbar area (including the shoulder girdle), which in the case of the patient shown in Figure 4.3b shows forward (anterior) rotation of the shoulder girdle to the left. The observation from posterior is confirmed by the fact that from the anterior view his right arm is more anteriorly positioned against his thigh. His left shoulder girdle is elevated and rotated posteriorly with the left arm following. This means that he presents with an unlevelled thoracolumbar area with an increased kyphosis with depression and anterior rotation of the right shoulder girdle. The left side has compensated by the reversed pattern; i.e. the left shoulder girdle elevated and posteriorly rotated with the arm following. It appears that he also has weakness of the deep posterior segmental muscles of the spine.

The next observation is the feet and pelvic girdle (adaptive response to the thoracolumbar postural changes). In this case the patient is loading to the right with pelvis rotated towards the right side, with apparent shortening in the angle of the waist. Observation of the compensatory adjustments of the head and the cervical spine shows that his head is tilted to the right and he has a head forward posture (hyperextension of the upper cervical area), which indicates the potential shortening of the anterior neck muscles on the left side.

Observation of posture (2)

Figure 4.4: An axial view of the position of the shoulder girdle relative to the pelvis due to ISMS dysfunction (observation 2)

This person shows a dominant posterior rotation of the right shoulder girdle and a reduced thoracic curve. The scapulae are not symmetrical, which may be indicative of a muscle imbalance between internal oblique abdominus on the left and external oblique abdominus on the right; the latissimus dorsi on the right side seems to be shortened. The lower trapezius, serratus anterior and rhomboids appear to be atrophied and the levator scapulae and middle trapezius appear to be bilaterally overactive.

In this case the pelvic rotation appears to be more anterior on the right side, which results in the patient being loaded more on the right foot.
Her centre of gravity seems to be more posterior, which is probably why she presents with an increased lumbar lordosis.

The compensatory response (position) of the cervical spine and neck is not observable from this view and is not available in the text (Myers, 2011).

Knowledge block

Myers (2011) describes global fascial lines, which indicate not only that fascia may be shortened (stiff) at a local level but that stiffness at a local level can incriminate the whole fascial system. This fact explains why lumbar fascia micro-trauma leads to shortening and fibrosis, which may cause distal signs and symptoms; for example, cervical involvement (immobility and/or headache), restriction of shoulder girdle and gleno-humeral joint involvement (pain and restriction through latissimus dorsi), restriction of breathing movements, and transferring the load from the trunk to the pelvis and lower limbs.

Figure 4.6: Rotational and posterior longitudinal fascia lines

(Myers, 2011 p 72)

(ii) Observation of functional activities

Assessment of the patient's motor control is achieved by assessing their functional activities, such as gait, sitting to standing and/or other functional tasks that will reproduce the patient's symptoms. During functional activities the manual therapist should identify abnormal movement patterns (due to abnormal muscle activation, guarded movement patterns and compensatory mechanisms of movement).

Compensatory movement patterns are characterised by motion segments or spinal regions carrying out a compensatory movement to accommodate the lack of movement (due to stiffness) in another segment or region. Abnormal postural control is identified by abnormal sequence of movement, limited or distorted equilibrium reactions, and a small range of global movement patterns (due to guarded movements). It is also characterised by asymmetry in bilateral cyclical movement patterns (reciprocal trunk rotation and arm swing in, for example, gait), and lack of or limited segmental stability and mobility, which results in abnormal or compensatory movement patterns.

During all functional activities or physiological movements performed by the patient the manual therapist should identify the patient's pain responses. Observation of gait and other functional activities is based on the parameters of gait, and movement analysis (Perry & Burnfield, 2010).

The manual therapist needs to observe the complex, dynamic patterns of spinal movement during functional activities (Esola *et al*., 1996; Steffen *et al*., 1997) to identify a change in the sequence of movement in order to determine the coordination between lumbar and pelvic movements, or between spinal flexion and extension. The relationship of the shoulder girdle and arm in relation to the pelvis and leg during functional activities should be observed. A difference may exist between the mobility of the upper and the lower lumbar spine during simple functional activities and when the upper and/or lower limb movements are part of more complex functional activities.

Pure physiological movements in the spine do not occur because spinal movements are coupled movements and occur in three dimensions (coronal, sagittal and transvers planes). Typical coupled movements are disturbed in a patient with ISMS dysfunction. During observation (assessment) it is essential for the manual therapist to determine how the components of the coupled movements interact during spinal movements without or associated with upper and lower girdle and limb function. Lack of components of coupled movements is an indication of dysfunction in the motion segment and in global mobility and stability of the trunk. This is further investigated by the manual therapist by using passive accessory movements during palpation.

Observation of abnormal movement patterns includes the 'pain-spasm-pain' cycle (reflex sustained co-contraction between agonistic and antagonistic muscles (Langevin & Sherman, 2006)) and 'pain adaptation' (slowing and decreased range of motion due to selective increased activation of antagonists). Altered muscle activation patterns in CNSLBP can stabilise the spine during movement (owing to fixation), to prevent further injuries. This adaptation comes at the cost of restricted range of motion. Patients with CNSLBP appear to have many motion-limiting muscle activation patterns that may be initiated or aggravated by emotional factors (fear and anxiety).

Abnormal movement patterns can have important influences on the connective tissues that surround and infiltrate muscles. Both increased stress due to overuse (repetitive movement) and decreased stress due to immobilisation or hypomobility can cause changes in connective tissue (Langevin & Sherman, 2006).

Within this complex cycle of events, the manual therapist should be able to identify how to interpret components of movement caused by disinhibition, disuse, atrophy and hypertrophy, with the view to isolating the probable source of dysfunction and putting it functionally to the test during activities such as walking and sitting to standing. For example, if the latissimus dorsi is in spasm or hyper active it will fixate the scapula and the trunk and limit arm swing during gait.

Isometric muscle testing is appropriate in the case of severe pain.

(2) Palpation guided by observations

The aim of palpation is to determine specifically (1) the alignment of the palpable components of the vertebral column (spine), pelvic girdle and lower limb and shoulder girdle and upper limb; (2) the mobility of all soft tissue; and (3) the analysis of the texture of the soft tissues.

During palpation of the soft tissues and skeletal landmarks and zygapophyseal joints, rib angles and costal junctions, the manual therapist should correlate with the patient's pain responses. The pattern of soft tissue tension in the ISMS upper and lower limb should be identified. The musculature of the rotator cuff may develop major anterior shortening in response to a thoracic kyphosis and poking chin.

The patient should be asked to report any awareness of abnormal sensation during the palpation. The patient's responses could be malinterpreted because sensory awareness and spatial orientation are inhibited due to the bombardment of nociceptive stimuli from various structures within and around one or several motion segments. Moseley, Gallagher and Gallace (2012) and Langevin and Sherman (2006) have found that tactile discrimination and spatial orientation on the spine are poorly represented in the brain of normal subjects and even more so in patients with CNSLBP.

The manual therapist should be aware that the muscular-tendonous junction, intermuscular septum, ligaments or capsules, junction with the periosteum and bone are more sensitive than other areas and should be taken into account during the interpretation of the patient's response to palpation. Identification of soft tissue abnormality should include the factors outlined below.

Muscle tone (spasm or atrophy) taut bands and trigger points

The manual therapist should be sensitive to detecting any changes in muscle tone and areas of muscle activity or loss of activity. Presence of areas of muscle spasm and tender spots (i.e. trigger points) indicates an active nociceptive source. These observations through palpation should be correlated with the observations of

inactivity, muscle imbalance or malrecruitment, which all contribute to the decreased postural control observed during functional activities.

Taut bands and trigger points in the muscles

On palpation active trigger points give rise to local and referred pain (Travell & Simons, 1983). The presence of painful muscle contraction or tender foci within perimuscular fascia may add to the factors that promote hypomobility and tissue fibrosis because increased muscle tension and contractures may place strain on nociceptive sensitive soft tissue areas.

Referred pain to the lower quadrant due to nerve root involvement should be identified during the triage because it is a red flag and falls under the category of specific LBP (see Section 4.2.1.1). Any other referred pain down the leg is typically due to the activation of trigger points (refer to Sections 3.4.2 and 3.5.1.1). Taut bands and trigger point activity due to L4-S1 dysfunction commonly affect the gluteus maximus, medius and minimus, quadratus lumborum, piriformis, iliacus and psoas muscles. The distribution of the pattern of pain can mimic nerve root pain down the lateral aspect of the thigh and into the lateral lower limb. With careful localisation and successful de-activation of active trigger points, this referred pain should subside if it is somatic (i.e. no nerve root involvement) in origin.

Fascial stiffness (shortening) and fibrosis

'Fascial stiffness' refers to the general fascia as well as the thoraco-lumbar fascia, which includes the gluteus maximus, medius and minimus, quadratus lumborum, piriformis, and iliacus and psoas muscles as well as the internal and external oblique muscles and transversus abdominus.

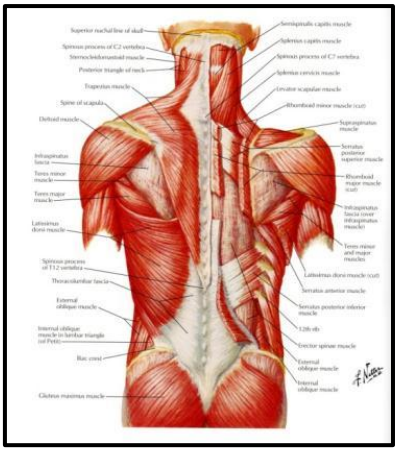


Figure 4.7: Fascia of the posterior aspect of the trunk

(Netter, 2011 plate 168)

Knowledge block: Inflammation and tissue stiffness

Chronic, local increase in mechanical stress on the connective tissues (fascia) can lead to micro-injury and inflammation (overuse injury, cumulative trauma disorder). A consistent absence of mechanical stress on connective tissue leads to connective tissue atrophy, architectural disorganisation, fibrosis, adhesions and contractures. Factors that influence whether atrophy or fibrosis predominates during stress deprivation include the concurrent presence of inflammation, tissue hypo-oxygenation and cytokines such as TGFb-1 that promote fibrosis. Fibrosis therefore can be the direct result of hypomobility or the indirect result of hypermobility via injury and inflammation (Langevin & Sherman, 2006).

Tissue micro-injury, inflammation and fibrosis not only can change the biomechanics of soft tissue (e.g. increased stiffness) but also can profoundly alter the *sensory and nociceptive input* arising from the affected tissues. Connective tissue is richly innervated with chemical, mechanical, and thermal nociceptors and nociceptive neurons (Langevin & Sherman, 2006). Modulation of nociceptor activity has been shown to occur in response to changes in the innervated tissue (Section 3.8.2). The level of protons, inflammatory mediators (prostaglandins, bradykinin), growth factors (NGFs) and hormones (adrenaline) all have been shown to influence sensory input to the nervous system (physiological pain resulting in hypersensitivity). Conversely, nociceptor activation has been shown to modify the innervated tissue (Langevin & Sherman, 2006).

In addition, the release of substance P from sensory C-fibres in the skin enhances the production of histamine and cytokines from mast cells, monocytes and endothelial cells. Increased TGFb-1 production, stimulated by tissue injury and histamine release, is a powerful driver of fibroblast collagen synthesis and tissue fibrosis (Langevin & Sherman, 2006). Thus, activation of nociceptors by itself can contribute to the development or worsening of fibrosis and inflammation, causing more tissue stiffness and movement impairment.

Regardless of its original cause, connective tissue fibrosis is detrimental, as it leads to *increased* tissue stiffness and (*further*) movement impairment. The manual therapist should correlate the tissue fibrosis with the (lack of) mobility of the particular spinal regions during all the functional activities.

Interpretation of referred pain and asymmetry

If the patient complains of referred pain, the therapist should identify the origin of the referred pain as somatic referred pain or neural referred pain. In relation to other findings during palpation the therapist can perform neural tension tests, which in themselves are not diagnostic tests for nerve root pain but involve the nerve due to an adhesion in the neural sheath or nerve root compression (Butler, 2000).

Somatic referred pain patterns have been discussed under 'Taut bands and trigger points in the muscles' and Section 3.4.2. Reflexes such as the ankle (S1) and knee jerk (L3/4), and elbow jerk (C6) should be tested to differentiate between somatic and neural referred pain in order to confirm that referred pain is not neural in origin.

When assessed as part of a clinical picture, posture and functional activities demonstrate disrupted biomechanics. These disruptions are most probably the result of the influence of the shortened soft tissue due to an injury or mechanical failure of one or more of the musculoskeletal structures, commonly due to involvement at the level of L3-S1.

The manual therapist should determine the alignment of the trunk by specifically looking out for the position of the shoulder girdles in relation to the pelvis and the position of the pelvis, in particular, to identify a potential unilateral pull of the quadratus lumborum and sometimes latissimus dorsi muscles that will cause trunk torsion together with a lateral tilt of the pelvis that results in the corkscrew effect on the biomechanical motion segments of the ISMS.

Knowing the patient's pain responses, the therapist should purposefully and carefully palpate deeper to verify the location, level, and pain responses of areas with different tissue textures. During the process of deeper palpation the manual therapist should

also palpate the deep intersegmental muscles of the whole spine to identify on a deeper level the tissue tension, trigger points, taut bands, muscle spasm, differences in tissue textures and, very specifically, the bony alignment of the motion segments. In the thoracic areas the rib angles and costovertebral junctions are also determined. Motion segment distortion may be detected in any motion segment in the spine but especially at the lower lumbar, thoracolumbar, and cervico-thoracic regions because these are areas of transition where great mechanical strain is placed on the transitional vertebrae.

Palpation of the bony prominences of the ISMS

Palpation of the ISMS is performed by using accessory movements to determine the alignment and quality of the motion segments of the whole spine. Alignment of the motion segments of the spine and of the whole spine, including the coccyx, is important to note because the coccyx can be tilted posteriorly or anteriorly due to the potential disposition of the pelvis. If the spine is in a rotoscoliosis, the pelvis and the sacrum will be torsioned. The result of the palpation of the motion segments may reveal hyper- or hypomobility, lack of joint play or altered 'end feel'. These findings should be interpreted in the context of the presenting signs and symptoms; findings observed during the observation of the patient's habitual posture, gait and functional activities; and the soft tissues.

Assessment of the integrity of the neural system

The neural system integrity is assessed as follows:

- Integrity of the neural system is assessed by testing muscle strength, sensation (discrimination between light touch and a pain response) and deep tendon reflexes.
- Assessment of functional muscle strength is based on the observations of the patient's impaired alignment, mobility and stability during static and dynamic postures; characteristic adaptive behaviour, which manifests during gait and relevant functional activities of daily life; and, lastly, abnormal muscle activation patterns observed as asymmetrical muscle bulk that indicates hyper- or atrophy. The challenge to the manual therapist is to identify the interlocking vicious circles between guarded movements, muscle wasting,

loss of strength and endurance, loss of neuro-muscular coordination, muscle imbalance and physical dysfunction, all resulting in deconditioning and illness behaviour. Disuse syndrome develops out of reduced activity and illness behaviour (Bortz, 1984; Mayer & Gatchel, 1988). The effect on the physical condition of the ISMS due to disuse is profound as it aggravates and maintains physical dysfunction and leads directly to more severe dysfunction.

Autonomic nervous system involvement

ANS involvement can be observed and palpated by identification of the pilomotor effect, sweating and decreased skin temperature, vaso-motor effects (such as 'blanching' or excessive redness), pseudomotor effects, and sub-cutaneous skin oedema – also called *peau d'orange* (Waddell, 2004). As explained in Section 3.5 torsioning of the spine and increased thoracic kyphosis can mechanically involve the ANS. With release of all the soft tissue structures involved and re-alignment of the motion segments of the spine, this mechanical strain should be reversible.

The ANS may also be involved through emotional stress via the amygdala and the rest of the limbic system via neurotransmitters. During assessment the patient's awareness and understanding of the influence of emotional stress on their (acute/sub-acute/chronic) NSLBP should be determined. The manual therapist's management of the patient in a reassuring manner is the starting point in addressing the patient's problem.

Altered breathing patterns

The adaptation in the pattern and depth of breathing due to the mechanical distortion of the trunk and associated abnormal muscle activation patterns manifests in poor breathing patterns and limited excursion of the ribcage and lungs. Mechanically and anatomically the ANS is therefore vulnerable to becoming involved in potentially contributing to the patient's poor breathing pattern (Hodges, 2013).

Throughout the observation (assessment) the manual therapist must take into account the dynamics of the strain and pain of the ISMS dysfunction in terms of

inflammation and the repair processes, taking into account the time since the origin and the subsequent course of the NSLBP.

In summary, observing skeletal landmarks, palpating soft tissues, determining ISMS dysfunction and interpreting the patient's pain response (pain processing) and characteristic adaptive behaviour to form a holistic clinical picture is a skill that a manual therapist should acquire. All observations (visual as well as through palpation and muscle testing) should be based on clinical reasoning to explain the reason(s) for malalignment. The observation is therefore an interpretation of the underlying mechanisms that drive the ISMS dysfunction, analysed in terms of the underlying neuro-musculo-skeletal structures and functional activities related to the patient's genetics, pain processing, lifestyle, other psycho-social-related issues, and characteristic adaptive behaviour.

During the assessment the therapist should endeavour to evaluate the patient's potential to respond to direct soft tissue handling to determine the texture and condition of the soft tissues and the patient's pain responses during handling, as well as their response to cognitive demands and instructions in terms of ability to respond to changes in postural control. These responses will enable the therapist to make an estimated judgement on the patient's likelihood of responding to treatment through a process of remodelling of soft tissues and relearning postural control. The degree of improvement is determined by the patient's level of chronicity. The success of the treatment depends on the therapist's clinical judgement during assessment and the selection and performance of the treatment techniques.

> *For most people LBP is a short-lived inconvenience. Even when severe, if the patient can be given appropriate information to disarm or prevent fear and anxiety, and if they can keep their motor systems functioning then the chances for chronicity can be dramatically reduced.* (Field, 2009 p 50)

4.3 A multidimensional model for the assessment of patients with CNSLBP

On the basis of the discussion above the researcher has developed a model for the multidimensional assessment of a patient who presents with CNSLBP of varying degrees of chronicity (Figure 4.8). The model is based on the premise that plasticity in both soft tissues and nervous systems affects pain processing and changes in motor behaviour play a key role in the natural cause and course of CNSLBP.

The researcher hypothesises that with the first incident of ANSLBP, the sensory afferent input system generates pain responses from the peripheral to the CNS, which together with hypervigilance develop altered pathways in the brain. Pain causes active inhibition of the slow motor unit recruitment, which results in dysfacilitation. Even after the pain has been resolved, the dysfacilitation may persist. The initiating response to pain is fear avoidance, which can also cause active inhibition of the slow motor unit, and the central fatigue associated with the fear, stress, or anxiety, all of which contribute to dysfacilitation. The dysfacilitation leads to altered postural control (Comerford *et al*., 2008). In the presence of reduced physical activity connective tissue is not remodelled after the process of inflammation and/or inactivity and because it is so interconnected with all types of tissues it provides a powerful nociceptive input to the CNS.

Through a process of plasticity, the fascial (myofascial and neurofascial) tissues shorten and stiffen (fibrosis) (peripheral sensitisation). The continuous feed-in to the dorsal horn (central sensitisation) and the higher centres of these tissues results in a continuous pain processing within the whole nervous system within the neuromatrix.

The motor output effect of pain processing contributes to the development of the biomechanical malalignment of the ISMS but also to ISMS stiffening (fixation), and guarded movements. When influenced by the modulation of pain processing in the neuromatrix the motor output effect results in characteristic adaptive behaviour which will include the psychosocial responses and illness behaviour of the patients. .

This whole process described thus far is influenced by a person's genetic make-up as well as their genetic perceptibility for pain and psychological ability (make-up) to manage pain. The process of degeneration and tissue healing (which is also genetic in origin) affects the process of ISMS dysfunction.

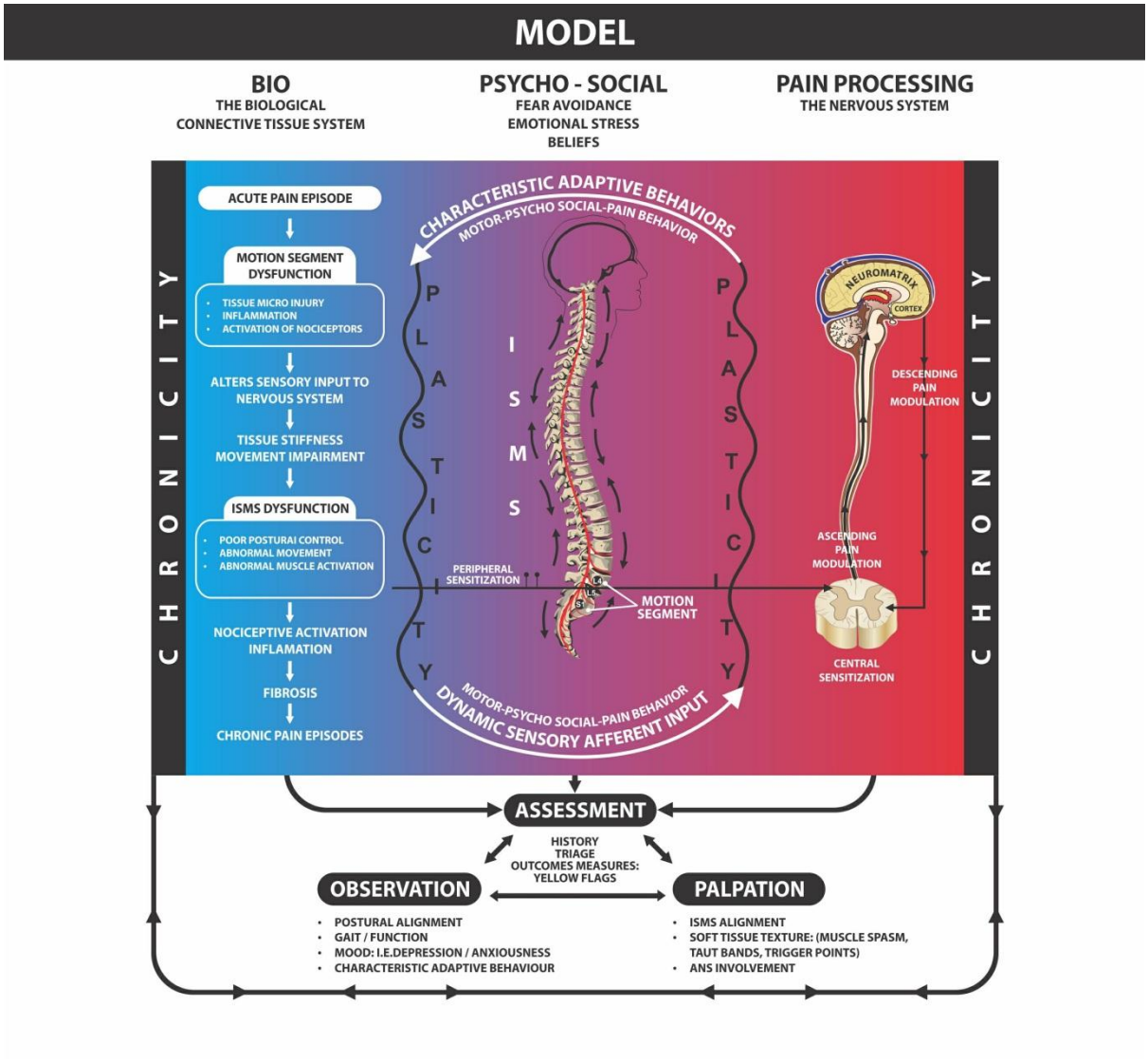


Figure 4.8: Model for the multidimensional assessment of a patient with CNSLBP

4.4 Summary of the chapter and discussion of the holistic integrated model for the assessment of patients with CNSLBP

The model displays the assessment of a patient in the three aspects of ISMS dysfunction: the dysfunction of the biomechanical component of the ISMS, pain processing, and characteristic adaptive behaviour, which are characteristic of patients with CNSLBP.

As discussed in Chapter 3, the biomechanical dysfunction of the ISMS is intimately integrated with the pain processing and characteristic adaptive behaviour. Although the three components of ISMS dysfunction are discussed separately a correlation can be made among them. For this reason, the researcher discusses the assessment from the biological perspective, pain processing perspective, and from a characteristic behavioural perspective.

In this chapter assessment of a patient is discussed from the history taking stage, which includes the patient's psycho-social factors that may contribute to their condition, and the cause and course of the condition. From the history the therapist should be able to evaluate and deduct the patient's ability to manage the condition.

The biological (clinical) manifestation of the signs and symptoms (local as well as widespread) is the result of the processes that have worked within the ISMS over a period of time. These biological processes (clinical manifestation) are reflected in the patient's posture, gait and functional movement, which are strongly affected by the current pain processing as well as the characteristic adaptive behaviour, which can mask the biological origin (signs and symptoms) of the clinical picture. The challenge to the manual therapist is therefore to identify the extent of the ISMS dysfunction in relation to the pain processing and the characteristic adaptive behaviour.

It is possible that the primary driver of the patient's clinical picture cannot be distinguished at the first assessment but may become apparent as the management of the patient progresses and the patient relates to and trusts the therapist. Empathic cognitive feedback from the therapist to the patient during the assessment is critical

for helping the patient understand their condition and address the fear avoidance and characteristic adaptive behaviour from the initial assessment and management.

In Chapter 5 the manual therapy for a patient with the typical presentation of CNSLBP is discussed.

CHAPTER 5

Principles of a multidimensional manual therapy approach to patients with chronic non-specific low back pain

5.1 Introduction

In this chapter the manual therapy model for the management of patients with CNSLBP is presented as a *multidimensional* approach. The multidimensional manual approach, developed by the researcher over years of clinical experience, takes into account the factors discussed in Chapter 3. Chapter 3 outlines the intimate interaction between the biomechanical dysfunction, the neural changes that take place at the peripheral, autonomic and central nervous systems as a result of pain processing and the characteristic adaptive behavior. The biomechanical dysfunction of the ISMS can be the primary reason for the abnormal sensory strategies, which lead to guarded or compensatory movement patterns. The guarded or compensatory movement patterns in turn can become part of the feedback mechanisms that aggravate the pain processing, ISMS dysfunction and the development of characteristic adaptive behaviour.

The pain and ISMS dysfunction can also be driven by the process of plasticity at the peripheral, autonomic and central nervous system due to the effect of the pain processing at these levels, which results in altered pathways in the brain or cortical degeneration (Kuner, 2010). If the hypervigilance of the limbic system is increased by a person's fear avoidance, health belief and illness behaviour (job-related stressors and absence from work),which are all part of the patient's characteristic adaptive behaviour, this facilitates the pain processing cycle, which results in biomechanical dysfunction (Field, 2009).

The researcher hypothesises that regardless of which component is the primary driver in the patient's clinical picture, all three components are present to a greater or lesser extent in one or other combination of dominance. This hypothesis is in line with the fact that Fourney *et al*. (2011) have described CNSLBP as a multidimensional problem. Fourney *et al.* (2011) suggest that patients with CNSLBP should be treated with a multidisciplinary approach. Based on her clinical reasoning the researcher suggests a multidimensional manual therapy approach for patients with CNSLBP so that all three components are addressed in an integrated way.

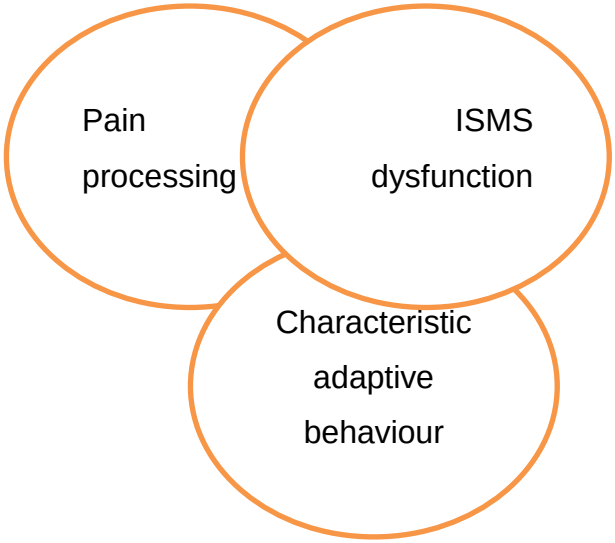


Figure 5.1 Integration of the three driving factors integral to the complexity of CNSLBP

The clinical implication of multidimensional manual therapy is that all three components of ISMS dysfunction (biomechanical dysfunction, pain processing and characteristic adaptive behavior) is always the central focus of patient management CNSLBP management.

The rationale of the multidimensional manual therapy management of patients is that the process of plasticity, which underlies the development of all three components of the patient's clinical picture, can be re-modulated through specific soft tissue stimulation to release restrictions in muscular and connective tissues and peripheral,

and autonomic neural systems. The hypervigilance of the central nervous system is addressed by direct handling techniques to remodel soft tissues as well as reassurance of the patient, implementing cognitive behavioural principles during the handling techniques to educate the patient regarding his /her condition.

The guarded and maladaptive movement responses are influenced from a biomechanical approach by the release of the soft tissue and joint restrictions at segmental and multisegmental levels. The researcher's observation in clinical practice is that by releasing the soft tissue and joint restrictions the muscle tone and elasticity (length) is prepared for recruitment. The researcher hypothesises that by releasing the soft tissues and joint restrictions, it is possible that the restoration of disrupted spatial awareness and tactile processing can be facilitated (Moseley *et al*., 2012). The guarded movements can also be prepared for realignment of the segmental and multisegmental skeletal structures as well as from the perspective of re-learning of postural control (Fersum et al, 2012)..

The maintenance of the released restrictions in the soft tissues and ISMS alignment (through a specifically well-designed exercise programme and lifestyle) can only be achieved through re-education of postural control and movement through appropriate cognitive behavioural input. Based on her clinical experience the researcher found that input regarding a specific exercise program needs to be provided at a frequency that the patient can manage so that she/he is able to take control of the behavioural and lifestyle changes that are needed to manage their condition.

The multidimensional approach does not exclude simultaneous intervention by other health professionals in a multidisciplinary approach but whether other interventions are included will depend on the patient's responses to the manual therapy approach and needs.

The multidimensional manual therapy approach as discussed in this chapter is in keeping with a dialectic approach to the management of patients with CNSLBP. In a

dialectic approach the identification of the patient's problem requires an integrated understanding of their characteristics (identified by the patient's stories or the patient's lived experience) as well as the definable physical or other signs and symptoms. In Section 5.2 the researcher discusses the principles of a multidimensional manual therapy approach to patients with CNSLBP from the perspective that the potentially strong emotional and behavioural factor in these patients makes it necessary for them to have confidence and trust in the therapist to address their problem. The manual therapist should establish an interactive professional relationship with the patient during the assessment of the patient to engender mutual trust and confidence. This professional relationship with the patient forms the background as well as the channel through which the multidimensional management takes place. The manual aspect of the treatment is based on continuous interactive feedback between therapist and patient and for this reason is an inseparable part of patient management from a dialectic perspective.

Pain management through facilitation of endogenous pain-inhibiting mechanisms involves an integrated interaction between:

- The therapist as a pain-inhibiting agent in a professional therapist-patient relationship in which the therapist re-assures the patient regarding their condition;
- Manual therapy to:
    - Inhibit ascending pain modulation by releasing soft tissue and joint restrictions at segmental and multisegmental levels and re-aligning the ISMS;
    - Facilitate the restoration of disrupted tactile processing (Moseley *et al*., 2012); and
    - Facilitate descending pain modulation through the ventral periaqueductal grey (vPAG) and dorsal periaqueductal grey (dPAG);
- Pharmacology; and
- Re-education of postural control.

- Appropriate health education is given on adaptation of lifestyle to maintain and re-enforce the effect of the pain modulation and ISMS dysfunction intervention.

5.2 Principles of pain modulation

The neurophysiology of pain modulation is complex and is the subject of much research at present. The principles of pain modulation discussed below have been identified in the literature as significant in pain modulation in patients with chronic pain (Bioloski *et al*., 2009).

5.2.1 The therapist as pain-inhibiting agent through a professional therapist-patient relationship

Reflection on and discussion of the researcher's clinical experience and clinical reasoning in this thesis have been undertaken from a dialectic perspective, which implies that the patient's stories and participation in their assessment and treatment are crucial.

Evidence is growing that the therapist-patient therapeutic relationship plays a role in the management of pain disorders (Slade, Molloy & Keating, 2009). This suggests that putting the patient at ease about how serious their condition is a very important starting point for managing the patient with CNSLBP. This conversation can take the form of an explanation of what the condition is and what the intervention will entail following the outcome of the assessment.

By putting the patient at ease, explaining why they are experiencing consuming pain and how it can be treated, the manual therapist aims to decrease the activation of the amygdala and insula (hypervigilant limbic system) through decreasing the fear of pain and catastrophising. At the same time the explanation gives the patient information necessary for dealing with the pain and for making deliberate cognitive (prefrontal cortex) decisions on how to understand the pain and manage it.

Empowering the patient to manage all three components of their condition, their lifestyle and the environmental factors influencing the condition is crucial for the success of the total management of the patient. By giving the patient essential, coherent, accessible and valid information about their condition, the manual therapist aims to correct any false beliefs about the condition and understand their role in the holistic management.

5.2.2 The role of cognitive behavioural therapy in the multidimensional manual therapy of patients with CNSLBP

In this thesis the researcher has indicated that cognitive behavioural principles are essential in the multidimensional manual therapy management of patients with CNSLBP. The fact that reassurance, presentation of information and appropriate health education are integrated into the manual therapy is probably the reason why patients are empowered to take control of their condition and manage it in collaboration with the therapist. The appropriate behavioural responses (Buchner *et al*., 2007; Gatchel &Turk, 2002) that a patient needs to be able to achieve include:

- Developing confidence in their skills and ability to re-think and change their beliefs about their pain to decrease the catastrophic thinking about their predicament that if they move they will experience pain;
- Acquiring mental techniques to reduce pain by getting rid of unhelpful thoughts, changing the focus of their attention away from their pain and redefining pain as a different sensation; and
- Managing work-related and social stress more effectively and acquiring skills to cope with the pain and/or recurrences of symptoms.

Relaxation techniques can form part of the cognitive behavioural approach to the management of CNSLBP. The ultimate goal will be for the patient to take responsibility to adapt their lifestyle and to manage their social stressors and achieve job satisfaction.

If the manual therapist observes that the patient needs more intensive cognitive behavioural therapy to achieve more appropriate behavioural responses and self-management of their condition, the therapist can refer the patient to a psychologist (Fourney *et al*., 2011; Buchner *et al.*, 2007).

Health education during the handling techniques and directly afterwards should be directed towards correcting the patient's health beliefs and perceptions and addressing mal-perceptions the manual therapist identifies during the assessment. Empowering the patient to change their lifestyle and manage environmental factors cannot be separated from the segmental and multisegmental soft tissue release and ISMS re-alignment, from re-education of postural control, and from kinetic handling. Empowerment takes place as the patient is able to take control of their postural control and maintain their lifestyle (Zusman, 2002).

5.2.3 Pain modulation through manual therapy

Manual therapy addresses pain modulation through soft tissue release and through stimulation of the mechanoreceptors in the joints. Zusman (2010 p 109) states: '*Constant mechanical stimulation is fundamental to the homeostasis of the musculoskeletal system.*' The mechanical stimulation Zusman (2010) refers to is applicable to soft tissue and to mechanical stimulation of the mechanoreceptors in the joints.

5.2.3.1 Inhibition of ascending pain modulation

Ascending pain modulation through manual therapy is achieved through release of soft tissue and joint restrictions on segmental and multisegmental levels and through re-alignment of the ISMS.

Muscle tone decreases (because ISMS dysfunction starts as a result of local reflex muscle spasm at the L4-S1 motion segments) and muscle stiffness filters throughout the entire muscular component of the ISMS as a result of nociceptive stimulation. Muscle spasm, taut bands and trigger points are released sequentially through

stretching, myofascial release and trigger point release (deep myofascial release techniques applied according to the patient's pain response).

The aim of the release techniques is to restore the viscosity of the ground substance in fascia (actin and myosin filaments) in the muscle, which results in the decrease of the sensory afferent input to the dorsal horn and higher centres. Added to this is the fact that myofascial release stimulates the mechanoreceptors, which results in deactivation of the gamma motor tone regulation system. The slow deep pressure stimulation leads to the stimulation of mechanoreceptors and probably also the slowly adapting Ruffini endings and some of the interstitial receptors, such as the muscle spindles. This leads to an altered proprioceptive input to the CNS, which then results in a changed tonus regulation of motor units (Schleip, 2003). If the functioning of the motor unit is restored, muscle fibres can fire and recruit (Comerford, 2008).

Specialised and unspecialised loose connective tissue (fascia) stiffening, including myofascial shortening within the entire ISMS (with emphasis on the TLF), occurs as a result of a lack of movement (due to compensatory fixation and guarded movements most probably as a result of fear of pain) and/or as a result of adaptive characteristic behaviour and a passive lifestyle.

Poor posture can result in shortened fascia *or* can occur as a result of shortened or dysfunctional fascia (especially TLF) that contributes to poor posture. Either way the mechanism operates initially locally (L4-S1 motion segments) and then filters throughout the entire ISMS.

When fascia is released in the dysfunctional ISMS through manual therapy, a major source of afferent nociceptive input (Langevin & Sherman, 2006) to the dorsal horn and higher centres is also decreased, which results in a decrease of sensitisation in these areas. The decreased afferent input to the dorsal horn is further improved by the fact that muscle ischaemia, which contributes to pain, is also reduced. As Sluka and Milosavljevic (2009) point out, '*Decreased muscle spasm would be expected to decrease muscle ischemia and this reduces nociceptor sensitization and lessens central input to the spinal dorsal horn.'*

The soft tissue release creates the space to restore alignment of the biomechanical aspect of the dysfunctional ISMS through the joint system, by applying passive intervertebral movements in required therapeutic directions with the aim of restoring the dysfunctional ISMS joint mobility and the soft tissues (muscles, joint capsules, ligaments and tendons) to their normal length. Mobilisation of the intervertebral (and costovertebral, costosternal and costochondral joints in the thoracic region) throughout the entire dysfunctional ISMS will also restore the patient's rib excursion and improve their breathing pattern. When this happens, the afferent input via the nociceptors to the dorsal horn and the higher centres is further decreased from this nociceptive source.

Zusman (2010) states that the effect of passive movement procedures on pain modulation is relatively small and changes in tissue position or movement are not lasting. He reports conflicting evidence on the long-term changes in structure and symptoms and disability following passive movement. In the multidimensional manual therapy approach that is the subject of this thesis these limited responses do not affect the short-term effect of the passive movement because they serve as a preparatory technique for the implementation of treatment techniques such as active re-education of postural control.

Pain modulation through manual therapy takes place through the direct muscular reflexogenic response as a result of the effect of manual therapy at the spinal level (Potter, McCarthy & Oldham, 2005). Because the motion segments, head and pelvic girdles of the dysfunctional ISMS is re-aligned the spinal curves is restored and the local reflexogenic muscular (myofascial) response on segmental and multisegmental levels occur.

Stroking over the areas of the skin innervated by primary posterior rami of spinal nerves leads to decrease in muscle tone (relaxation), which contributes to decrease in pain (Schleip, 2003; Kostopoulos & Rizopolous, 2001).

On the basis of clinical reasoning and clinical experience, the researcher recommends that in patients with major tissue stiffness and pain the treatment technique of choice would be to release the motion segment (segmental level) to address pain relief first. Thereafter, the soft tissue release should be performed. The reasoning behind this recommendation is that the primary release (mobilisation) of the motion segment (joints and soft tissues within and surrounding the motion segment) releases the restricted tissues (containing the nociceptors) in response to the movement.

For the lower lumbar motion segments to be released the patient should be in the most neutral position as possible. This neutral position of the lower lumbar vertebrae is reached the easiest in sidelying (Figure 5.1).

Figure 5.1: Lumbar rotation in sidelying (Maitland, 2004)

Segmental joint mobilisation, which should routinely be carried out in prone, is passive accessory movements in all directions of each motion segment, especially in the direction of segmental restriction. Segmental joint mobilisation does not mobilise only the osseus structures but also the deep fibrous structures, such as the ligaments and facet joint capsules (Maitland, 2004; Richardson, Jull, Hodges & Hides, 1999). Graded tensile loading by passive movement onto healing or unhealed soft tissues could facilitate optimal repair and tissue integrity (Zusman, 2010).

Tendons, joint capsules and ligaments are also classified as specialised connective tissue and are also mobilised (Middleditch & Oliver, 2005; Porterfield & DeRosa, 1991; Kapandji, 2008).

Release of segmental joint restrictions in the lower lumbar area will, by definition, be the area of focus for initiating the treatment, followed by releasing the soft tissues and fascia that attach to the pelvis and in the lower limb. The thoracolumbar junction, thoracic spine, and soft tissues that attach to the scapulae and relevant soft structures in the upper limb and the cervico-thoracic junction, cervical region and head on the 01 are also released.

The researcher found clinically that sidelying is the key position for initiating the release of the segmental restrictions in patients who present with a torsioned lower quadrant. The release of the motion segments and the soft tissues is achieved when the range of motion increases and the patient experiences less pain with less somatic referred pain.

Mobilising the motion segments and soft tissue at and around the transitional areas results in elongation of the spine and in some release of the transverse pelvic and respiratory diaphragm and the thoracic inlet. The relevance of mobilising these transition areas is that it is an essential part of the restoration of the patient's breathing pattern.

The soft tissue release includes the muscles (myofascia) (through release of muscle spasm, trigger points and taut bands), TLF and other connective tissue structures such as loose unspecialised connective tissue, joint capsules and, finally, the highly sensitive neural and dural tissues.

Because specialised and unspecialised connective tissue depends on movement and stretch to maintain its mobility, it should be put under lengthening strain such as during normal full range movement (Langevin & Sherman, 2006). Myofascial techniques to remodel connective tissues are therefore skilled applications of manual stretching, trigger point release and positioning to release trigger points, taut bands,

muscle spasm and shortened soft tissue in general (Mannheim & Lavett, 1989; Schleip *et al.,* 2012; Langevin & Sherman, 2006).

Myofascial release creates mobility of soft tissues because the mechanical effect of the stretching of the deeper muscle fibres (elasto-collagenous component of the muscle) changes the ground substance from a solid block to its state of viscosity. *Fascia is the only tissue that modifies its consistency when under stress (plasticity) and which is capable of regaining its elasticity when subjected to manipulation (malleability)* (Stecco *et al.*, 2004).

Treatment techniques for releasing soft tissues used by the researcher include myofascial release (Barnes, 1997), trigger point therapy (Dommerholt, 2012), Rolfing structural integration (Caspari & Massa, 2012) myofascial induction approaches (Pilat, 2012), and neural release (Wander & Weinschenk, 2012). In this thesis these techniques are defined as direct manual mechanical tissue release techniques. The manual therapist should implement the most appropriate techniques for achieving the required outcome.

Other techniques in the literature include osteopathic manipulative therapies (King, 2012), connective tissue manipulation (Prendergast & Rummer, 2012) fascial manipulation (Stecco & Stecco, 2012), accupuncture as a fascia-oriented therapy (Irnich & Fleckenstein, 2012), and stretching of fascia (Myers & Frederick, 2012). Stretching exercises, which are inherently part of yoga and pilates, are also described as techniques to mobilise fascia and are an important part of the discussion on exercise for maintaining soft tissue and joint mobility. In sidelying, release of the soft tissues at segmental level is also achieved more easily (Figure 5.2).

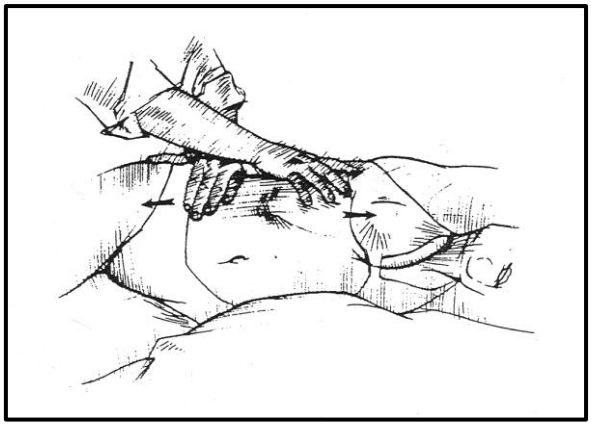


Figure 5.2: Myofascial release of the lateral oblique area

Once joint mobilisation and myofascial release of the whole spine have been addressed, the manual therapist should ensure that mobilisation of the O1 and sacropelvic junction are performed. The O1 and sacro-pelvic junctions can be seen as the anchors of the vertebral column. The researcher has also learned from experience that to get optimal release of the soft components of the scapulae and upper limb the 'hand behind back' is a valuable technique.

Little is known about the remodelling and lengthening response in vivo in anatomically intact tissues as opposed to single structures isolated for research in vitro (Standley, 2009; Solomonow, 2009). Importantly specialised and unspecialised connective tissues should be released at local segmental and global multisegmental levels and in local and global patterns (Myers & Frederick, 2012; Stecco & Stecco, 2012). The researcher therefore recommends that myofascial release in patients with CNSLBP should include the whole ISMS.

The researcher recommends that release of neural restriction is carried out after some mobilising and releasing of the musculoskeletal system, which is preparatory to introducing the pain-sensitive nervous system to stretching.

Torsioning at the pelvis is likely to incriminate the lower lumbar/sacral roots, or the sleeves, so both the slump test (Butler, 2000) and the straight leg raise (SLR) (Butler 1999) are valuable techniques for treating the deepest fascia layer. Tests to assess restriction of neural tissues in the limbs address the peripheral nerves predominantly,

while passive neck flexion (PNF) and slump movements are predominantly tests and treatment (stretching techniques) for restrictions in the neural canal (CNS).

These peripheral and central neural tension techniques can be carried out at different stages of the treatment, based on the stage at which the spinal and limb movements can be stretched into the restriction. These technique(s) can also be carried out together with breathing exercises to increase the range of movement. Breathing exercises serve as a strategy to empower the patient to take control of the movement and the discomfort that accompanies the stretching technique.

Although myofascial release should be achieved by stretching to change the ground substance from a solid block to its proper viscosity, the force should be adapted on the basis of the clinician's interpretation of the patient's response to the clinician's handling skills.

> *It is well known in physical therapy for example that application of direct tissue stretch to ligaments, joint capsules needs to be gauged carefully to avoid causing increased tissue inflammation. To understand how much force (or movement) is beneficial, and how much can be harmful is one of the challenges of these clinical modalities* (Langevin &Sherman, 2006 p 5).

The therapist should also apply the mobilisation of the peripheral and central neural restrictions with caution, taking cognisance of the reasons for underlying strain on the soft tissues and joints and potential underlying inflammatory responses, which will sensitise the nervous system rather than mobilise it.
In addition, the continuum of the nervous system is proof of its potential to reach beyond the physiological limits of the nervous system. This means that with treatment the handling skill of the manual therapist is critical for gauging the extent of the forces and angles applied to the nervous system with this technique. The nervous system is naturally full of nociception but the other hidden problems such as neural and dural adhesions and restrictive interfaces in general can also flare the system to a painful state.

5.2.3.2 The facilitation of the restoration of disrupted tactile processing

The restoration of disrupted tactile processing and spatial discrimination (Moseley *et al*., 2012) is probably facilitated throughout manual therapy when the manual therapist receives feedback from the patient on the localisation of touch and the response of pain during the handling techniques. Tactile processing is disrupted probably due to the descending pain-enhancing mechanism by reducing the effectiveness of the second order inhibitory pathways in the spinal cord (Field, 2009).

Schleip (2003 p 17) states that:

> *In the case of a slow deep pressure, the related mechanoreceptors are most likely the slowly adapting Ruffini endings and some of the interstitial receptors; yet other receptors might be involved too (e.g. spindle receptors in affected muscle fibers nearby and possibly some intrafascial Golgi receptors).*

This statement adds to the support of the argument that manual therapy (myofascial release) will contribute to the restoration of tactile processing and spatial discrimination in addition to the cognitive awareness created by the localisation of touch during manual therapy.

The researcher hypothesises that through the feedback during the manual therapy the prefrontal and limbic centres are stimulated through the spinobrachial tract (Kuner, 2010; Figure 5.3). The localisation of the touch and proprioceptive input through direct handling techniques (deep pressure and stretch) is carried through the lateral spinothalamic tract and posterior columns (together with the cognitive feed-in to the prefrontal cortex and the limbic centre, which is the area for interpreting the cognitive-emotional experience of pain).This enables the patient to regain any tactile spatial orientation that may have been lost / diminished due to the pain and dysfunction the patient has experienced and that overrides the proprioceptive and tactile input. As the manual therapist guides the patient through the process of the handling techniques she attempts to alert the patient to the site of the pain and depth of the pain, and its relationship to other structures. The manual therapist should explain to the patient throughout the treatment what is happening with the soft tissue

release and re-alignment of the motion segments of the spine as the treatment progresses. Through this interaction of therapeutic feedback, the manual therapist aims to facilitate the restoration of disrupted tactile processing by increasing the patient's understanding of the origin of their signs and symptoms and how these signs and symptoms are responding to manual therapy.

During the multidimensional manual therapy the patient is assisted towards taking cognitive control of their emotions evoked by catastrophisation and towards adapting their beliefs regarding their functional limitation caused by CNSLBP.

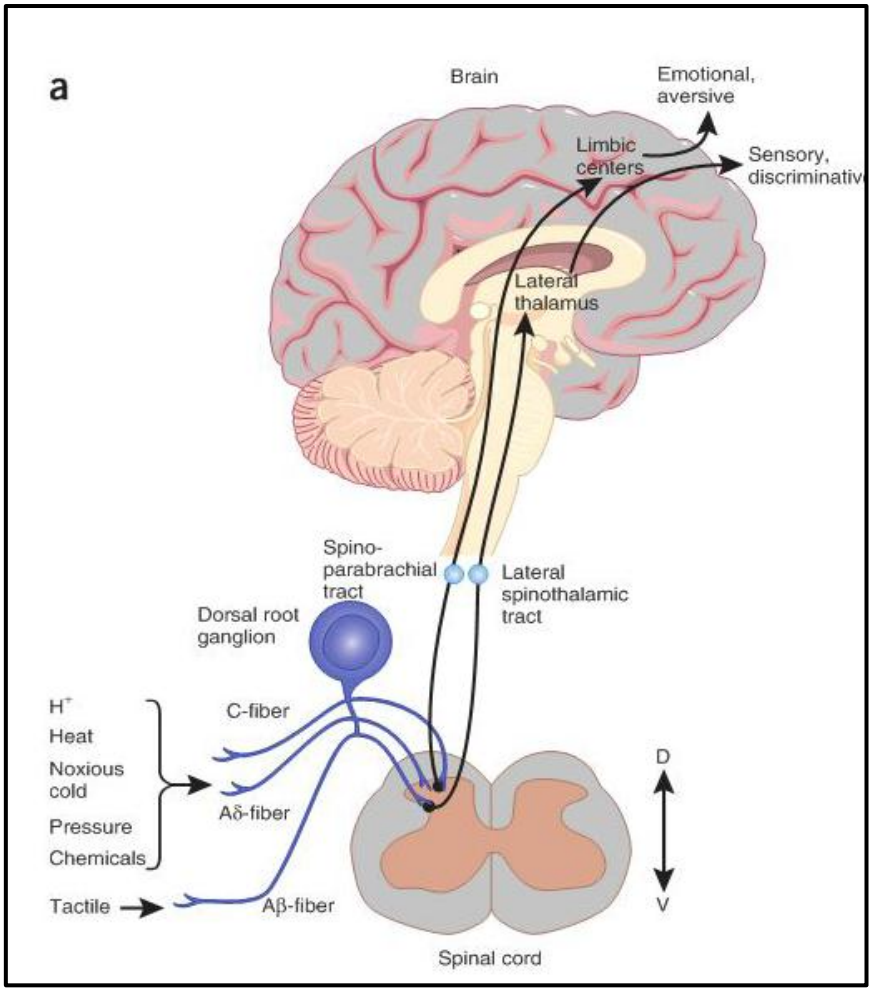


Figure 5.3: A schematic overview of the main circuits that mediate physiological pain

(Kuner, 2010)

The process described above is preparation for active and loaded restoration of muscle balance, which involves posture and movement retraining during activities.

5.2.3.3 Facilitation of descending pain modulation through the vPAG and the dPAG

Another aspect of endogenous pain-inhibiting mechanisms is descending pain modulation through the vPAG and dPAG.

Through the direct stimulation of the intervertebral joints and the myofascial and trigger point release, the 'stimulus' reaches not only the sensory discrimination centre and emotional aversive areas in the limbic centre (see Figure 5.4) (Kuner, 2010; Brooks &Tracey, 2005) but also the dPAG. From the dPAG the descending pathways carry noradrenaline when it synapses with the dorsal horn and activates the inhibitory interneurons.

When the inhibitory interneurons are activated, they release the endogenous opioid neurotransmitters enkephalin or dynorphin, which bind to the μ-opioid receptors on the axons of the A-delta and C fibres that carry afferent nociceptive impulses from the periphery. The activated μ-opioid receptors inhibit the release of substance P, which in turn inhibits the activation of the neuron from where the pain impulses are transmitted to the brain. The nociceptive impulse is therefore inhibited before it can reach the cortical areas responsible for the interpretation of pain and before the limbic centre is activated. The dPAG inhibitory control on the dorsal horn is an immediate hypoalgesic response and is part of the flight or fright reaction (Kuner, 2010).

The vPAG consists of the ventrolateral columns of the PAG and the dorsal raphae nucleus, which is sympatho-inhibitory in action and involves serotonin as the neurotransmitter.

The vPAG is stimulated via the stimulation of the sympathetic trunk when the intervertebral and, especially, the costovertebral joints are being mobilised during manual therapy.

Mobilisation of these joints affects the ANS chain that lies adjacent to the vertebral column. This direct mechanical mobilisation of the sympathetic trunk results in sympathoexitatory activation (Sterling *et al*., 2001; McGuiness *et al*., 1997).

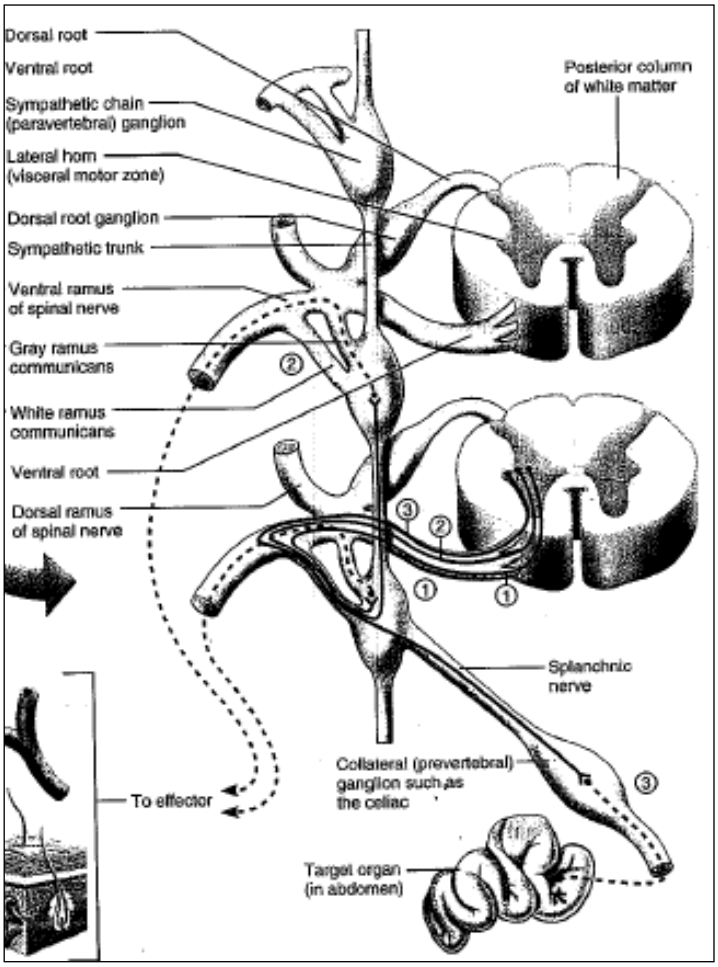


Figure 5.4: Sympathetic pathways

(Marieb, 2004 p 539)

Stimulation of the sympathetic trunk affects the PNS and the target organs (Chui & Wright, 1996; Sterling *et al*., 2001; Cleland *et al.*, 2002) and stimulation of the sympathetic trunk results in the distribution of impulses in three ways:

(a) In the thoracic region, indicated as (1) in Figure 5.4, the sympathetic preganglionic fibres synapse with a peripheral nerve, leading to an effector organ, such as the heart, lungs, and eyes.

(b) The passive therapeutic mobilisation of the motion segments also leads to stimulation of the sympathetic trunk in the adjacent vertebral levels higher and lower than the stimulated level – [number (2) in Figure 5.4] which synapses with neurons in other parts of the sympathetic trunk to join a peripheral nerve. The effect of the stimulation of the sympathetic trunk at one level therefore has a diffuse effect throughout the nervous system because during the passive mobilisation of the motion segments is performed on the entire ISMS system and will therefore mobilise the sympathetic trunk at various levels and have a widespread effect on the ANS.

(c) Mobilisation to the intervertebral and costovertebral joints (ISMS joints) can also activate the preganglionic fibres directly (immediate hypoalgesic effects following SMT, which is specific to mechanical nociception as opposed to thermal nociception).It may be affected via the sympathetic trunk, which can form part of the splanchnic nerve [(3) in Figure 5.4] that synapses with the adrenal medulla in the kidneys. The result of the activation of the adrenal medulla is the release of catecholamines into the blood stream (Marieb, 2004 p 540), which reach the vPAG over time. The release of serotonin from the vPAG to the dorsal horn results in a hypoexitatory effect, known as a descending pain-modulating mechanism, which results in decreased pain perception (Sterling *et al*., 2001; Vicenzino *et al*., 1996).

Descending pain modulation through the dPAG has an immediate effect on pain perception, whereas pain modulation through the vPAG has a delayed pain-modulatory effect because the release of catecholamines from the adrenal medulla stimulates the vPAG via the blood circulation to release serotonin.

Not all researchers who discuss descending pain modulation distinguish very clearly between the effect of pain modulation via the dPAG and the vPAG (Kuner, 2010;

Brooks &Tracey, 2005). Kuner (2010) states that much debate still takes place on the noradrenergic and seronergic pain-modulatory mechanisms.

To conclude the discussion on the pain modulation through manual therapy, the researcher presents an adapted version of the interrelations of processes involved in generating and maintaining CNSLBP in Figure 5.5. This diagram is included to indicate how manual therapy can contribute to the reversing of the interrelated processes that generate and maintain pain processing as described by Field (2009).

The focus of the intervention starts with understanding the patient's 'problem' from their individual (usually ill-structured) perspective to determine in which way it impacts their life during the assessment of the patient. This enables the manual therapist to relate the patient's responses to the activities she/he experiences and relates to his/her CNSLBP problem

Empowering the patient to participate in the management of their CNSLBP problem is something that runs through every aspect of the management from assessment to intervention through health education.

Releasing the soft and neural tissues, mobilising and re-alignment of dysfunctional ISMS on segmental and multisegmental levels to achieve pain modulation on peripheral, central and cognitive level, and

Re-education of postural control, adaptive and maladaptive movement during functional activities

5.2.4 The role of pharmacology as part of the holistic approach to manual therapy for patients with CNSLBP

Release of soft tissue restrictions and mobilising synovial joints in the ISMS may result in inflammatory responses. The most effective way to treat this post-manual therapy inflammation is through appropriate pharmacological agents. The reasoning behind this is the promotion of quicker healing in the soft tissues and limitation of the formation of adhesions. Pharmacological agents can include anti-inflammatories,

muscle relaxants and analgesics and/or anxiolytics to treat pain-induced fear of movement (Langevin &Sherman, 2006).

Improving our understanding of therapeutic mechanisms of drug interactions is key to developing more effective treatment strategies for CNSLBP with minimal adverse effects.

5.2.5 Re-education of postural control

Re-education of postural control is introduced the moment that pain modulation becomes effective. The re-education of movement as such then becomes another pain modulation factor because it actively maintains the passive (manual) release of segmental and multisegmental soft tissue and joint restrictions. General exercise and activity are also known to have a pain modulatory effect due to mechanoreceptor stimulation and result in endogenous opioid release that inhibits pain (Van Tulder & Koes, 2002).

Direct comparisons of different exercises have failed to show that one is any more effective than another or that changes in pain are non-specific and similar in all three groups (Oldervoll *et al*., 2001; Petersen, 2002).

An explanation for the conflicting evidence on the effectiveness of exercise in the management of patients with CNSLBP is that the specific exercises are not as important as physical activity per sé (Van Tulder & Koes, 2002; Macedo, Latimer, Maher, Hodges, Nichola, Tonkin, McAuley & Stafford, 2008; Middelkoop *et al*., 2010). Exercises are aimed at specific (neuro)-musculoskeletal outcomes where (functional) rehabilitation is aimed at restoring a patient's functional ability in their environment on a daily basis and therefore need to be more patient specific than a general exercise programme. Activities of daily life (which may include specific exercises or functional activities to achieve a specific physical effect, i.e. muscle activation) should therefore be adapted to each patient's needs and challenges in their environment.

In keeping with the underlying reasoning in this thesis the researcher suggests that re-education of postural control should aim to teach the patient to recruit appropriate muscles and muscle groups to restore or optimise the patient's posture in the functional positions that they adopt during activities of daily life (ADL). Muscle recruitment should be carried out on appropriate segmental and multisegmental levels as well as during functional movement to optimise segmental and multisegmental stability and mobility. It is, however, also critical to optimise or restore the patient's sequence of movement during functional activities with the aim of addressing guarded, adaptive and provocative movements.

Van Tulder and Koes (2002) and Macedo, Latimer, Maher1, Hodges, Nichola, Tonkin, McAuley and Stafford (2008) found no clear relationship between the type or intensity of exercise and physical performance or improvement in pain and ability. That is also true of rehabilitation and return to work.

O'Sullivan (2011) concludes that contracting stabilising muscles (pelvic floor, transverse abdominus and lumbar multifidus) prior to spinal loading exercises has not addressed the disability associated with CNSLBP. Although randomised controlled trials have shown the small effect of sizes in the treatment of patients with CNSLBP, this is not a superior approach to other conservative approaches. O'Sullivan (2011) acknowledges that stabilising exercises for patients with CNSLBP have some benefit but indicates that the underlying basis for this approach is questioned.

In the RCT by Fersum *et al*. (2012) the results have shown that multidimensional CB-CFT programme (cognitive behavioural education regarding their CNSLBP, health education regarding lifestyle, combined with an exercise programme as well as retraining adaptive and maladaptive movement including in the work place) resulted in superior reduction of patients' pain, disability, fear, beliefs, mood and sick leave at the 12 month follow-up. It therefore appears that exercise should not be given without extensive health education and cognitive behavioural input by health professionals and specifically manual therapists

Based on the multidimensional complexity of the clinical picture of patients with CNSLBP in this study, the researcher suggests that the role of exercise in the management of patients should include not only segmental stabilisation exercises but specifically segmental mobility and multisegmental stability and mobility exercises to address the ISMS dysfunction. However, restoring the patient's segmental and multisegmental stability and mobility with specific exercises addresses only the ISMS components of postural control and not the re-learning of functional movement during ADL, which has also been impaired due to the abnormal changes in the brain.

It is also important that segmental and multisegmental stabilising and mobilising exercises should contribute to the patient's awareness of good posture in all functional positions that they adopt during the day. The rationale behind this statement is that the patient has to be aware of the strain that poor posture has on the segmental and multisegmental levels of the ISMS and how it contributes to their CNSLBP condition.

The researcher suggests that balance exercises and kinetic handling should be included to address the patient's tendency to use guarded and adaptive movement patterns to avoid pain. The patient has to re-learn to move with a normal sequence of movement without fear avoidance. The therapist should therefore ensure appropriate muscle recruitment during functional activities of daily living (FADL), such as climbing stairs, walking, getting in and out of a car, and sitting to standing. The researcher is of the opinion that muscle activation during normal physical activity (ADL) is the key to successful intervention. However, this statement still needs to be verified by research.

The manual therapist should therefore ensure that the patient's postural control is restored by teaching them to normalise their anticipatory control (inhibit excessive anticipatory movement during ADL) and movement strategies (i.e. through kinetic handling) during performance of instrumental activities of daily living (IADL) and general ADL.

These assumptions suggest that for retraining postural control it is essential to work on identifiable functional tasks rather than on movement patterns for movement's sake alone. A task-orientated approach to re-learning postural control during functional activities assumes that patients learn by actively attempting to solve the problems inherent in a functional task rather than repetitively practising normal patterns of movement. Adaptation to changes in the patient's environment is a critical part of recovery of function. In this context, patients are helped to learn a variety of ways to solve a problem to perform a task rather than a single muscle activation pattern through a process of external feedback.

Moseley (2005) describes graded motor imagery as a useful method to retrain movement-related networks without eliciting pain in the CNSLBP population. The researcher supports the concept of using graded motor imagery to retrain movement-related networks (through motor re-learning).This is because, by changing the patient's visual (visuo-motor) input to imitate more normally sequenced movement (without causing pain) during activity, characteristic adaptive behaviour can be changed/adapted. In addition more normally sequenced movement can be systematically re-learned (unlearning maladaptive compensatory movement) by utilising the role of higher centres in re-education of motor (postural) control (Shumway-Cook & Woolacott, 2007).

5.3 Principles of multidimensional pain modulation in patients with CNSLBP

To summarise the discussion on the multidimensional pain modulation discussed in this chapter the researcher presents an adapted version of the interrelations of processes involved in generating and maintaining CNSLBP in Figure 5.5. Figure 5.5 also indicates how the pain-contributing factors can be modulated by addressing chronic pain on various dimensions (Field, 2009).

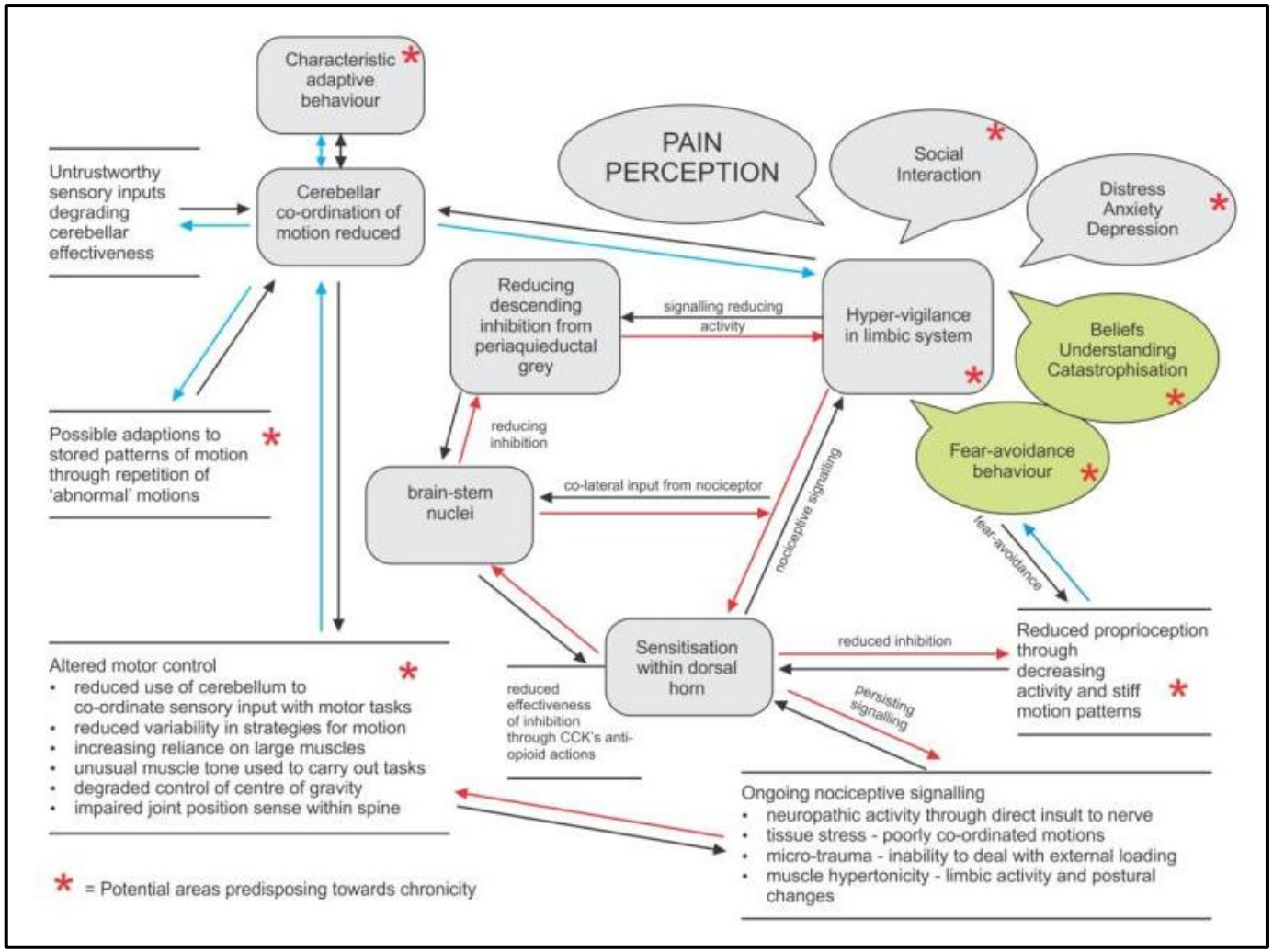


Figure 5.5: Principles of multidimensional pain modulation in patients with CNSLBP

(Adapted from Field, 2009)

In Sections 5.2 and 5.3 ascending pain modulation through a multidimensional manual therapy approach in patients with CNSLBP is discussed. Ascending pain modulation is achieved by decreasing the ongoing nociceptive signalling due to ISMS dysfunction (peripheral desensitisation) and desensitising the dorsal horn (central sensitisation). The hypervigilance in the limbic system is also changed through peripheral and central desensitisation as well as cognitive behavioural input to decrease the patient's fear avoidance, change their health beliefs and improve understanding of their condition, and as such decrease catastrophisation. Through decreasing the ascending pain modulation mechanisms the descending pain modulation is also reduced because the hypervigilance of the limbic system decreases the descending pain inhibition from the PAG. This process is now reversed by the decreased signalling to the limbic system so that the effectiveness of the neuropeptide cholecystokinin in inhibiting the descending pain control via the PAG is decreased, resulting in pain modulation onto the dorsal horn.

Descending pain inhibition is also especially targeted by the segmental joint mobilisation of the ISMS, which also affects the ANS chain and activates the pain modulation process via the vPAG. By facilitating the restoration of disrupted tactile processing, it is possible that the neuropeptide cholecystokinin, which plays a role in inhibiting the descending pain control via the PAG, is reversed. This results in enhancing the recovery of the descending pain inhibition onto the dorsal horn.

A decrease in the hyper-vigilant limbic system, which also plays a role in decreasing cerebellar coordination and in altered motor control (impaired local and global stabilisation and mobilisation), might contribute to the cognitive behavioural input to re-educate postural control (restoring impaired local and global stabilisation and mobilisation, kinetic handling and inhibiting guarded movement) during functional activities through a process of motor re-learning.

5.4 Clinical principles for the treatment of patients with CNSLBP

The clinical principles discussed in this section are based on the researcher's clinical experience. The management of a patient with CNSLBP does not only imply the

application of techniques but also requires that the therapist skillfully apply the appropriate handling techniques based on the reasoning that should be based on the driving factor(s) of the patient's condition and other complicating factors such as:

- The stage (chronicity) of the CNSLBP condition and the presence of joint degeneration (wear and tear on joints);
- The stage of healing of tissues such as adhesion formation and fibrosis, which will probably identify the stage of chronicity;
- A recurrent (ANSLBP) strain on an existing chronic state; and
- The patient's immune-neuronal interaction.

Buchner *et al*. (2007) report that patients with CNSLBP at all stages of chronicity (including the more advanced stages) benefit significantly from a multidisciplinary treatment approach to treatment. The more advanced stage of chronicity is usually associated with adhesions in soft tissues, which became more fibrosed. The researcher agrees with Buchner *et al*. (2007) but also found that patients in the advanced stage of chronicity tissue response show more resistance to treatment and may need more aggressive handling techniques. The frequency of the treatment in the advanced chronic stage of the condition still depends on the patient's response to treatment.

Recurrences are likely to occur in patients who are developing CNSLBP due to the vulnerability of the tissues together with the changes that might have occurred in the PNS, ANS and CNS due to chronic (recurrent) pain together with the associated fear avoidance and other psychosocial factors.

Neuronal function can be dramatically altered by activated immune and immune-like glial cells (Watkins, Hutchinson, Milligan & Maier, 2007). These authors found that activated immune and immune-like glial cells affect the neural function at the levels of the peripheral nerve, dorsal root ganglia and spinal cord and therefore have an effect on patients' neuropathic pain and play a role in the development of chronic pain. This mechanism has been found to disrupt the opioid tolerance and opioid dependence/withdrawal. In a patient with CNSLBP who has altered immune responses pain modulation may be decreased due to the effect on the neural

function. The therapist should be aware of this potential effect of the immune-neural interaction on the patient's pain response during and after treatment.

5.5 Risk factors to take cognisance of during manual therapy

Throughout treatment, feedback to and from the patient is essential to identify non-desirable effects of treatment. Undesirable effects of treatment can include biological and psycho-social factors.

Biologically undesirable effects include the following:

- Any centrally initiated symptoms such as nausea, sweating, light headedness and any changes in sensory disturbance that the patient may have experienced to the spine, head and limb girdles are biologically undesirable effects. The manual therapist should be skilled in identifying these signs and symptoms and make a decision on the progress of the treatment. These signs and symptoms are usually caused by ANS responses. If these signs and symptoms are present, the therapist should evaluate the situation and determine the stage of response before either continuing or stopping the treatment.
- With the direct handling skills the challenge to the manual therapist is to know how much treatment is enough as too much is detrimental to patient compliance. Monitoring pain responses and adapting handling skills accordingly are critical to patient compliance and trust. Excessive tissue stretching or pressure actually causes inflammation and can conceivably worsen the patient's condition.
- The manual therapist's skills for addressing the patient's fear, encouraging activity and for appropriately educating the patient to understand and cooperate in the management of their condition also play a major role in the successful outcome of the treatment.
- The patient is encouraged to tell the therapist everything they experience during the manual therapy and during the first 24 hours after treatment (without putting words into their mouth) to enable the therapist to judge the patient's response to treatment and estimate the patient's outcomes.

- The patient's emotional response and mood may be reflected in their willingness to undergo the treatment, positive involvement, and commitment to participate/collaborate in the treatment.

At the conclusion of the first treatment the manual therapist should:

- Encourage and guide the patient to continue or to resume usual activities, remain as active as possible, including resuming work activities (remaining active is the most widely respected clinical and scientific recommendation in the world today) and avoid bed rest as much as possible (European Guidelines for the Management of Chronic Non-specific Low Back Pain, 2004; National Collaborating Centre for Primary Care, 2009);
- Explain to the patient that they may experience discomfort and why it may happen; and
- Should there be a flare-up of soft tissue reaction inform the patient that they are at liberty to contact the therapist at any time.

The researcher does not give the patient specific exercises at this point in time because she intends to identify the effect of the first treatment on the patient's inherent capability to respond to the first treatment and not to teach the patient any new activities to adapt to. After the first treatment the patient must be given time and be encouraged to use the newly created movement gained during functional activity.

The therapist should know that the repair of tissue should occur within four to five days. A follow-up treatment should only be carried out after a period of tissue repair because of the inflammation of the myofascia that might have occurred as a result of the myofascial release and the passive accessory movements to mobilise the spine. This treatment should have given the patient new movement to work with during their ADL. With the confidence of and encouragement by the manual therapist it is expected that they will have used this movement during their ADL.

Ata the follow-up treatment sessions the manual therapist should re-assess the patient to identify the effect of the treatment on the ISMS dysfunction and on the

patient's posture, muscle activation, adaptive and maladaptive movement during gait and other functional activities that the patient might have engaged in.

If the probability of returning to usual activities is deemed to be low after the first treatment, the manual therapist should seek to identify the reasons preventing the patient from returning to their usual activities. These reasons should have been identified during the assessment of the 'yellow flags' (Waddell, 2004) on the:

- Intensity of pain (Visual Analogue Scale);
- Perceived disability (Quebec Back Pain Disability Scale or Roland-Morris Disability Questionnaire or Oswestry Disability Questionnaire);
- Symptoms (with no signs) of radiating pain below the knee (clinical consultation);
- Fears and beliefs (Tampa Scale for Kinesiophobia);
- Patient's projection regarding return to work (three-month projection question in the Fear-Avoidance Beliefs Questionnaire);
- Catastrophising (Pain Catastrophising Scale); and
- Absence from any type of work (employment status).

Such a patient may be at high risk for an emotional breakdown and should be referred for multidisciplinary management.

Whatever documentation system the manual therapist uses, these findings must be documented to assess future progress of the patient and for legal purposes.

Follow-up treatment should be scheduled for approximately four to five days after the previous treatment. The second treatment will follow the principles discussed in the preceding paragraphs. During the second treatment the therapist should be able to apply the techniques at a deeper level.

At each follow-up treatment the therapist should assess/re-assess:

- The salient warning signs and symptoms that the therapist recorded about the patient's original complaints; and
- The patient's response to the previous treatment.

The therapist would anticipate the leg and back pain to be easier. Should this be the case, the soft tissues in the pelvis and lower limb should be more palpable, revealing the presence of latent active trigger points. Owing to the release of the soft tissue restrictions during the first and second treatments the therapist should be able to palpate and release deeper structures. As the therapist is able to go deeper into the soft tissue she/he will identify and stir up more restrictions in the soft tissues and joints. These structures include releasing trigger points and taut bands in the shoulder and pelvic girdles as well as the attachments between the spine and the head. Other appropriate techniques for soft tissue release, joint mobilisation and neural and dural release can be applied. As indicated earlier over treatment is as bad as under treatment.

The skill of the manual therapist is to identify the dominant components during the re-assessment and through therapist reflection during and after treatment hypothesise what the persistent and recurring symptoms are so that they can plan and perform treatment on the ISMS alignment in terms of posture, gait, abnormal movement and muscle activation. Any abnormal change in posture, gait and muscle activation is an indication that pain and restrictive processes are still active in the ISMS.

As the therapist is able to apply soft tissue release on a deeper level, neural and dural stretches are introduced manually and by using theraband. As the treatment progresses it becomes increasingly complex because the repeated release reveals the structures that may have been the root cause (hidden agenda) of the patient's problem.

As the therapist releases and mobilises the deep lying structures (spine and capsules of the apophyseal joints in the spine, shoulder (glenohumeral) and hip joints, this can awaken emotional responses in the patient as a result of fear of the original pain/catastrophising about the past pain and their unknown response to treatment. In this case the manual therapist needs to deal with the patient tactfully at all levels of fear and trust.

With the introduction of activity (exercises) and cognitive awareness and (re)education of motor control, the mobility of the spine, including the lower and upper quadrants, which have been passively mobilised at segmental and global levels, is maintained.

Treatment can be a mix of several principles, depending on the patient's presentation and response to treatment. Because of the variety of combinations of treatment and the multidimensional nature of the condition, treatment competence of the physiotherapist needs to be monitored over time.

Treatment is not a cure but the improvement of the patient's quality of life in terms of pain responses, which vary from being pain free to experiencing manageable pain and optimised function during daily life.

The multidimensional model for the manual therapy management of patients with CNSLBP is presented in Figure 5.6.

5.6 A multidimensional manual therapy model for management of patients with CNSLBP

The model for the multidimensional manual therapy for management of patients with CNSLBP is an extension of the model for the assessment of these patients presented in Chapter 4.

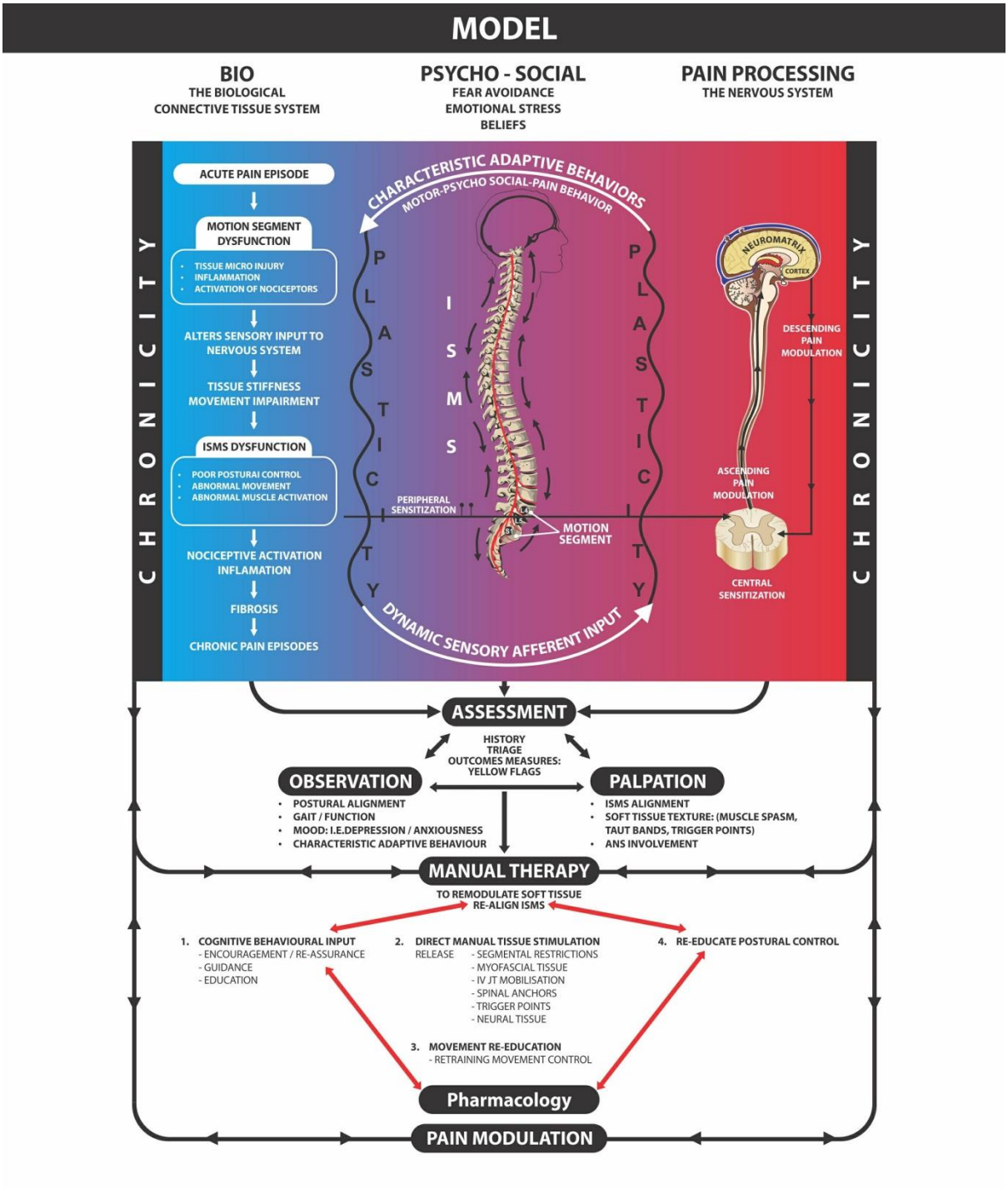


Figure 5.6: A Multidimensional manual therapy model for management of patients with CNSLBP

The focus of the multidimensional manual therapy for patients with CNSLBP is to optimise the re-alignment of the dysfunctional ISMS. Patients should re-learn postural control in order to regain and maintain the optimal ISMS function. The aim of the multidimensional manual therapy is achieved by first releasing soft tissue

restrictions in the muscles, connective tissues (including ligaments, joint capsules and neural and dura tissue) at segmental and multisegmental levels. Soft tissue release is achieved through the process of modulation of these tissues as a result of the initiation of the process of plasticity.

Secondly, the realignment of the ISMS is optimised by segmental and multisegmental mobilisation of the apophyseal and costovertebral, costosternal and adjacent joints in the ISMS. Optimising the realignment of the ISMS is mainly possible due to the release of the soft tissue restrictions.

Thirdly, through the release of the soft tissue restrictions and optimisation of the realignment of the ISMS, pain modulation occurs at peripheral and central levels of sensitisation and by decreasing nociceptive input to the ‘over-used’ neuromatrix in the brain through which the hypervigilance of the limbic system is activated. De-activating of the ‘over-used’ pain-generating neuromatrix is suggested in this thesis as achieved by reassuring the patient regarding their condition, enabling the patient to change their beliefs and providing appropriate education regarding self-management and re-education of postural control.

In Chapter 6 an evaluation of the model is presented, limitations of the study are outlined and suggestions for further research offered.

CHAPTER 6

Conclusion, discussion, limitations and recommendations

6.1 Introduction

The core problem in the limited success that has been reported in the treatment and, specifically, the manual therapy for patients with CNSLBP has been identified as a lack in the understanding of the complex clinical picture and therefore a lack of multidimensional management of the patient's problem.

The research questions of this study were:

- Can the concept of an 'integrated spinal movement system' ISMS be conceptualised based on the anatomy of the trunk?

- What are the underlying systems, processes and influences that result in ISMS dysfunction and contribute to the clinical picture of patients with CNSLBP?

- What contribution can the professional craft knowledge and the personal tacit knowledge acquired by the researcher over many years of clinical practice, make towards the declarative professional knowledge of manual therapy?

The primary aim of this study was to make a contribution to the theoretical foundation which may help clinicians to understand the biomechanical development of the complexity of the clinical picture in patients with CNSLBP and the role of multidimensional manual therapy model in the management of these patients. Through years of experience, metacognitive reflection on her clinical reasoning and social construction of knowledge the researcher has developed a multidimensional manual therapy model for managing the complex clinical picture of patients with CNSLBP.

The development of the components of the model is discussed in Chapters 3, based on the principles that have evolved from the researcher's metacognitive reflection on her clinical reasoning during patient management and the validation of the reasoning, arguments, statements and conclusions with published research evidence. In Chapter 5 the multidimensional manual therapy approach to managing patients with CNSLBP is discussed and the chapter concludes with the presentation of the multidimensional conceptualised practice model (theory).

A multidimensional manual therapy model for managing patients with CNSLBP has been developed and described in three different phases. The phases have not been developed in sequence, but emerged as the researcher performed metacognitive reflection on her (1) clinical reasoning and (2) outcomes of her practice and (3) identified commonalities between patients' with CNSLBP's clinical presentation and their (4) responses to the way she practice manual therapy. This process of interpreting the lived experiences of humans and to generate new knowledge from it is a subjective and contextually research process which falls within the q*ualitative research tradition drawing on interpretive phenomenology* and grounded theory (Polit and Beck, 2007)

The researcher's thinking process to and fro (dialectically) between these aspects to understand and verbalise the whole process/concept of the development of CNSLBP is called the hermeneutic circle. This process was inherently the researcher's process of creation of meaning (learning and gaining experience in clinical practice as clinician) and was expanded by the courses and congresses she attended to broaden her understanding of the research at the time, as well as through the social learning environment in which she worked by discussing and interpreting her views against the meanings of colleagues. This process isinherently based on the phenomenological tradition that meaning is subjective and contextually constructed (Higgs *et al*., 2010).

The process of moving between the 'parts' of the multidimensional manual therapy approach to understand and conceptualise the 'whole' model resulted in

metacognitive reflection and culminated in the formulation of the general manual therapy principles characteristic of the multidimensional manual therapy approach.

6.2 Evaluation of the multidimensional manual therapy model for the treatment of patients with CNSLBP

6.2.1 Summary of the multidimensional manual therapy model

In Chapter 3 (Section 3.5) the researcher has explained that motion segment instability does not only affect the lumbar spine only but the ISMS as a whole and therefore over time ISMS dysfunction becomes the core element of the clinical picture in patients with CNSLBP. The conceptualisation of the ISMS and consequently the discussion on the development of ISMS dysfunction, based on the functional anatomy, is based on the researcher's observation and in clinical practice that patients diagnosed with CNSLBP's whole spine and head position, shoulder and pelvic girdles are affected.

Based on functional anatomy the researchers discussed the possible mechanism through which ISMS dysfunction can develop as the core element of the clinical picture of patients with CNSLBP. Characteristic of this ISMS dysfunction is that it shows the integrated interaction between the neuromusculoskeletal systems and the patient's psychological response to pain and the mechanism through which cognitive beliefs, social and work-related stressors influence patient's pain perception and ISMS dysfunction. The discussion also indicates that due to hypervigilance in the brain, social stressors can result in increased muscle tone and shortening of soft tissues in particular fascia, which triggers ISMS dysfunction (although the mechanism is different from the motion segment instability approach) and can result in CNSLBP.

For this reason the researcher advocates that ISMS dysfunction in patients with CNSLBP should be treated by multidimensional manual therapy regardless of the mechanism through which it developed. Treatment of ISMS dysfunction entails the release of soft and neural tissue shortening and realignment of the spine, position of

the head and shoulder and pelvic girdles by remodelling the soft and neural tissues through a process of plasticity.

Remodelling of the soft and neural tissues within and around the spine, head, shoulder and pelvic girdles and realignment of these structures, pain modulation takes place through a process of activation of the local and central endogenous pain modulatory mechanisms.

The multidimensional manual therapy, pain modulation and re-education of tactile discrimination take place within a dialectic approach to patient care.

Multidimensional manual therapy prepares the patient as a person and his/her neuromusculoskeletal system for appropriate re-education of postural control and functional rehabilitation.

Pharmacology is administered to treat soft tissue swelling, pain and to maintain muscle relaxation.

6.2.2 Evaluation of the multidimensional manual therapy model against other models used in the management of CNSLBP

6.2.2.1 The lumbar segmental instability concept versus the concept of ISMS dysfunction

O'Sullivan (2000) describes lumbar segmental instability as the origin of CNSLBP and also the cause of recurrences of ANSLBP superimposed on CNSLBP. Although the researcher agrees in principle that lumbar segmental instability due to the neutral zone dysfunction can be the primary origin of CNSLBP and that the flexion pattern, extension pattern, lateral shift pattern and multidirectional pattern can be associated with some of the variations in ISMS dysfunction (Section 4.2.1.2).

However in ISMS dysfunction as the core contributing factor to the development of CNSLBP discussed in this thesis the concept of ISMS dysfunction differs from the lumbar segmental instability concept in the sense that:

(1) ISMS dysfunction was observed clinically and then substantiated theoretically based on functional anatomy. There are therefore similarities between ISMS dysfunction and the lumbar segmental instability concept. The difference between the two concepts behind the development of CNSLBP is that the lumbar instability concept is limited to the lumbar spine while the ISMS dysfunction concept includes the whole spine, the position of the head, shoulder and pelvic girdles. The researcher believes that the rotation of the pelvis due to the shortening of unilateral segmental multifidus on the affected lumbar level together with the shortening of quadratus lumborum which will result in pelvic torsioning (Section 3.5) which will spiral up the spinal column to the head and can result in ISMS dysfunction. The researcher hypothesises that the torsioning of the spine involving the neuromusculoskeletal and fascia systems especially the thoracolumbar fascia may be the cause of the widespread signs and symptoms and the heterogenetic nature of CNSLBP.

Lumbar segmental instability due to neutral zone dysfunction is discussed only in terms of the effect on the lumbar spine and does not include the effect of the .

(2) ISMS dysfunction involves the position of the head, shoulder and pelvic girdles in relation to the spine based on the fact that in the presence of ISMS dysfunction, other areas of the spine can refer pain to the low back and as such it can be interpreted as symptoms in the low back while the origin is not in the low back. Hence the motivation behind the multidimensional manual therapy model is to treat the patient in all dimensions of CNSLBP and not only the low back in patients with CNSLBP.

(3) The authors Panjabi (2003) and O'Sullivan (2000) have not reported on the change in the flexion, extension and lateral shift and multidirectional patterns but only mention that the patients' were treated successfully.

Based on the researcher's experience when these flexion, extension and lateral shift and multidirectional patterns observed in patients in the motion segment instability

concept are treated through multidimensional manual therapy the soft and neural tissues can be released, the tactile awareness can be 'alerted' and the motion segments can be realigned in preparation to activate the motor units for better recruitment. The muscle recruitment (re-education of postural control) can then be done in functional movement patterns during activities as well as work and recreational environments.

6.2.2.2 Subgrouping models versus ISMS dysfunction

O'Sullivan (2005) describes eight subgrouping models for sub-classification of patients with CNSLBP in order to administer patient-centered targeted intervention.
The mechanical loading model described by O'Sullivan (2000) is also the basis of the abnormal loading on the lumbar spine as the origin of ANSLBP which according to the researcher's observation, clinical reasoning and discussion based on the functional anatomy slowly develops over time (more or less 12 weeks) into ISMS dysfunction.

ISMS dysfunction as it is described in Section 3.5 encompasses the following: peripheral pain generator model, neurophysiological model, psychosocial model, signs and symptoms model, motor control model and biopsychosocial model. Over time as ISMS dysfunction becomes chronic the characteristics of the different models start to manifest in different combinations. As ISMS dysfunction merges from acute to chronic (CNSLBP progresses from the early to the late stage) so the characteristics of the various models manifest in the patients clinical picture. The longer chronicity persists, the more complex the patient's clinical picture becomes because more of the characteristics of the different models become integrated into the patients clinical picture. The clinical picture can be further complicated by superimposed recurrences of acute on chronic to the point where the strain on all the systems reach a point of break down to the point of failure of adaptive capacity to cope
The models described by O'Sullivan (2005) were designed to guide clinicians on which dimension (model) they should focus their treatment. The multidimensional manual therapy model presented in this thesis was developed on the inseparable interaction between the various systems. Therefore the researcher is of the opinion

that because of the heterogeneity of CNSLBP as a condition, the clinician should identify the dominant drivers of the condition but also address the less dominant drivers of the condition otherwise residual mechanisms will remain to perpetuate the problem (hence the development of the multidimensional manual therapy model).

6.2.2.3 Manual therapy in clinical trials versus the multidimensional manual therapy model

Randomised clinical trials in which manual therapy was compared to various forms of exercise, were conducted by Fersum *et al*. (2012) and Kääpä *et al*. (2006). Kääpä (2006) compared a multidisciplinary rehabilitation which consisted of a combination of (1) General fitness exercise, (2) Muscle strengthening exercise for all main muscle groups in the trunk and the lower limbs, (3) Special exercises to correct mobility of the spine and hip joints, activate the stabilising muscles of the spine, and increase flexibility of the lower limb muscles. (4) Functional exercises to improve postural control dynamic body balance, and coordination; (5) Progressive relaxation exercises to normalise muscle tension, with an individual physiotherapy intervention group. These different types of intervention was presented by a multidisciplinary team consisting of a physiotherapist, occupational therapist, rehabilitation physician, The individual physiotherapy group received (1) Massage, (2) Spine traction, (3) Manual mobilisation of the spine and (4) Therapeutic ultrasound as well as (5) 15-20 minute light active exercise (muscle stretching, spine mobilisation and deep trunk muscle exercises), only from a physiotherapist.

The outcome of the study showed that there were no statistically significant difference between the two groups after the rehabilitation (6-8 week programme), at 6, 12 and 24 months follow-ups.

The difference between the manual therapy that the patients in this individual physiotherapy group in the trial by Kääpä (2006) received and the multidimensional manual therapy model in this thesis, is that the aims of the massage, spine traction and manual mobilisation was not clear from the publication. The aims of the manual therapy which is part of the multidimensional manual therapy model are clearly stated as the soft and neural tissues (which include fascia) modulation, the re-

education of tactile discrimination, and the re-alignment of the ISMS. The soft and neural tissue modulation as well as the realignment of the ISMS results in pain modulation on spinal as well as central level by activating the endogenous pain modulation process in the body.

The individual physiotherapy explained by Kääpä (2006) as well as the multidimensional manual therapy model includes exercise as integral part of the intervention. In the multidimensional manual therapy model the manual therapist also acts as a pain modulating agent in the sense that she/he reassure the patient and give him/her relevant patient-education regarding his/her condition. These aspects were not mentioned as part of the individual physiotherapy intervention by Kääpä *et al*. (2006).

The overall encompassing aim of the multidimensional manual therapy model is to reverse the ISMS dysfunction that occurred in the patients' neuromusculoskeletal systems including the altered pathways in the brain and the patient's thinking patterns through a process of plasticity.

It seems that the individual physiotherapy intervention in the trial by Kääpä *et al*., (2006) was not specifically designed to address the heterogenetic nature of patients with CNSLBP.

Fersum *et al*. (2012) also conducted a randomised clinical trial to compare the outcome of a CB-CFT programme with MT-EX intervention. The CB-CFT programme seemed to address most of the characteristics of patients with CNSLBP. However the MT-EX with which it was compared, was also not reported to be designed to address the heterogenetic characteristics of the clinical picture of patients with CNSLBP.

The MT-EX intervention was according to the publication not designed to modulate the dysfunctional systems in patients with CNSLBP through a process of plasticity on peripheral, central (spinal cord level) and the brain levels. It is therefore not surprising that CB-CFT results in superior outcomes above the MT-EX intervention.

The multidimensional manual therapy model presented in this thesis was specifically designed to address the heterogenetic characteristics of patients with CNSLBP from a manual therapy perspective and can therefore not be compared to the outcome of manual therapy combined with exercise in the trials that was discussed.

6.2.2.4 The mechanisms of a comprehensive manual therapy model for musculoskeletal pain versus the mechanisms of the multidimensional manual therapy model for patients with CNSLBP

Bialosky, Bishop, Price, Robinson and George (2009 p 532) propose a comprehensive model which suggest that a mechanical *stimulus initiates a number of potential neurophysiological effects which produce the clinical outcomes associated with manual therapy in the treatment of musculoskeletal pain.*

The model indicates that the effect of a mechanical stimulus results in a biomechanical mechanism, neurophysiological mechanism, peripheral mechanism, spinal mechanisms and supraspinal mechanisms.

Although lasting changes of the biomechanical mechanism have not been identified, the authors (Bialosky *et al*., 2009) suggest that the outcomes associated with manual therapy are a result of the additional mechanisms initiated by the mechanical stimulus.

Although the authors state that the clinical effectiveness of manual therapy are not established their model serves as a guide for clinicians as well as further research. An important aspect of their conclusion for this thesis is that the effect of manual therapy should not be studied in isolation but that clinicians and researchers should be aware of the other potential non-specific effects of manual therapy through other mechanisms.

The comprehensive model by Bialosky *et al.* (2009) coincides with the multidimensional manual therapy model developed by the researcher based on clinical observation and clinical reasoning. Where Bialosky *et al*. (2009) only

presents the model, the researcher has applied the same principles and explained the outcome thereof in the treatment of the heterogenetic clinical picture of patients with CNSLBP.

6.2.2.5 Evaluation of the multidimensional manual therapy model compared to the challenges for the future management of CSNLBP

O'Sullivan (2011) formulated 12 criteria for the future management of patients with CNSLBP. The researcher evaluates her multidimensional model for the management of patients with CNSLBP against these criteria to establish the extent that the model presented in this thesis met these criteria.

To date there is no clear understanding of the complex heterogenetic nature of CNSLBP. The multidimensional manual therapy model presented in this thesis provides the link between the biomechanical, physiological and behavioural processes inherent in ISMS dysfunction which is suggested by the researcher as the preceding processes to the development of CNSLBP influenced by social factors.
The multidimensional manual therapy model for the management of patients with CNSLBP therefore serves as a point of departure from where patients with CSNLBP can be managed/or managed regardless of the main driver of the condition (ISMS dysfunction, pain processing and/or characteristic adaptive behaviour) because the patients will probably present with a varying combination of these three components.

The multidimensional manual therapy model in this thesis further provides clinicians with a combination of mechanisms within the musculoskeletal, neuromuscular, neural (brain and ANS), sensory receptors (systems), and behavioural systems culminating in adaptive and maladaptive postural control within which the main or multiple driver(s) of chronic pain and the interaction between the systems (and pain driving factors) can be identified.

The multidimensional manual therapy model has been developed from a dialectic perspective and as such the interpretation of information from two different paradigms (psychosocial and biomedical paradigms) is inherently part of the model. In the discussion on the model the researcher has indicated the role of the therapist

as a pain-modulating agent which implies that she/he has to have a thorough understanding of and ability to interpret the neurophysiological responses (sensory-discriminative, peripheral and central pain processing as well as altered pathways in the brain) associated with the biomechanical and social stressors to generate the patients clinical picture and responses and stressors on his/her condition. With a dialectic thinking process the clinician has to interpret information from various domains to synthesise the patient's specific problem which may lie mainly in the biomedical and/or the psychosocial domain. From this synthesis the therapist must be able to plan and conduct a management plan that should address the patient's heterogenetic problem.

Due to the dynamic interaction between the components that can drive the patient's clinical picture the driving component may change as the patient responds to multidimensional manual therapy. Recurrent episodes of ANSLBP superimposed on CNSLBP can therefore be initiated by either social stressors or another biomechanical strain. The researcher has indicated that regardless of what the main driving factor of the patient's recurrent pain episode is, the patient should always be expected to present with a combination of ISMS dysfunction, pain processing and characteristic adaptive behaviour which should be managed to a greater or lesser extent at each treatment.

The researcher has indicated that referral to other health care practitioners is not excluded from the multidimensional manual therapy model. She is in favour of a multidisciplinary approach to management of these patients if and when the need arises.

The distinction between the three components (ISMS dysfunction, pain processing and characteristic adaptive behaviour) which can be single or multiple drivers of the patient's CNSLBP and the various combinations between them make it possible for future research to use the details of this model to refine screening tools or broad categories (subgroups) which will make it possible to identify and manage these patients more effectively.

6.3 Limitations of this study

Limitations in this study are the factors which are inherent in any qualitative research study.

The main limitation in studies from an interpretive paradigm is that the researcher is involved in the data-gathering and data-interpretation processes. In the case of this study the data-generation and interpretation were conducted on the basis of the metacognitive reflection on the researcher's own clinical practice and clinical reasoning processes. A specific limitation with such an approach is that the researcher can 'make up' the clinical reasoning as the study progresses because it is difficult to recall clinical reasoning on aspects of patient management that have taken place quite some time ago. It is also possible that the researcher's clinical reasoning could have been based on her personal opinion at the time of the conceptualisation of the components of the model. These limitations have been limited by the fact that the researcher's conceptualisation of the basic principles of the model was published in 1995 and still remains the same. She continues to treat patients with (C)NSLBP and reflect on her treatment approach all the time while writing up this thesis to confirm the clinical reasoning, statements, arguments, and development of the concepts. These mental activities have also been supported by evidence from the literature to provide a sound foundation and research evidence for her clinical reasoning, statements, arguments and concept development.

The researcher's curriculum vitae is attached to provide evidence of her continuing professional development and participation in courses and congresses at national and international level. She works as a member of a multidisciplinary team where she shapes her ideas on management of patients regularly during discussions. Patient feedback occurs continuously in a patient-centred approach to the management of their CNSLBP. The researcher has also taught postgraduate students and colleagues on continuing professional development courses. Social construction of knowledge therefore still plays a major role in her knowledge base and clinical reasoning during clinical practice.

The diagram on the formation of clinical knowledge from an interpretive and from an empirico-analytical paradigm (Section 2.3.1.2) has been published almost a decade after the researcher conceptualised the ISMS and multidimensional manual therapy management model for patients with CNSLBP. The diagram (Edwards & Jones, 2007), displayed as Figure 2.3, explains the generation of clinical knowledge of clinical experts based on clinical reasoning. This diagram also reflects the knowledge generation of the researcher based on her clinical reasoning and which culminated in the multidimensional manual therapy model for managing patients with CNSLBP conceptualised in this study.

A limitation that could have occurred during writing up the thesis is that the researcher could have implied conclusions without explaining them explicitly because they are part of her intuitive tacit knowledge and could have been overseen in the preparation of this thesis.

This research thesis has been based on a grounded practice theory development in which the results of the research is qualitative conceptualisation based on observations and interpretation of clinical phenomena

6.4 Recommendations

6.4.1 Recommendations for further research

Because the model is dialectic in nature any research that is done to validate the model should take the dialectic nature of the model into consideration. The recommendations for further research therefore include suggestions for research on the ISMS dysfunction as one of the main constructs within the clinical picture of patients with CNSLBP. The main recommendation for further research is that the multidimensional manual therapy model must be empirically validated to be incorporated into the declarative knowledge of Physiotherapy.

The researcher recommends that the different biomechanical manual applications (stimuli) recommended in this multidimensional manual therapy model, be studied by

a multidisciplinary team such as manual therapists to apply manual therapy within the principles of manual therapy, endocrinologists to monitor the effect of manual therapy on inflammatory mediators, neurophysiologists to monitor potential CNSL, PNS and ANS mechanisms, psychologists to monitor patient's non-specific effects such as fear, expectations and catastrophising.

Current research in the process of plasticity in remodelling soft and neural tissues by Langevin (2011) should be followed up and its effect of manual therapy on plasticity should be clinically investigated. The current research looks so promising and should be followed-up clinically and theoretically to enhance the understanding of soft tissue release.

The clinical application of manual therapy should be critically evaluated by manual therapists to develop baseline expertise handling skills and the effects thereof. Research from an empirico-analytical paradigm should be conducted to analyse and validate the development of ISMS dysfunction and its interrelated processes of pain processing and characteristic adaptive behaviour.

Research on the role of manual therapy on the immediate-, medium- and long-term pain modulation should be further investigated. The biochemistry of peripheral and central sensitisation and altered pathways in the brain in patients with CNSLBP and its response to manual therapy is not well understood.

Research on the role of the immune system and tissue irritation (Watkins, Hutchinson, Milligan & Maier, 2007) in pain processing specifically in patients with CNSLBP is suggested.

The skilful application of the manual therapy techniques and the clinicians clinical judgement should also be investigated to determine how 'much is enough' and 'how much is too much' in terms of the restoration of the process of plasticity to restore intervertebral alignment and mobility and tactile discrimination.

Finally the cost-effectiveness of the multidimensional manual therapy management model for patients with CNSLBP should be assessed against the cost-effectiveness of other approaches to treatment such as CB-CFT (Fersum *et al*., 2012).

The risk factors that contribute to the development of CNSLBP, should be investigated so that the high prevalence of CNSLBP can be addressed and the burden on health care services and absenteeism from work be decreased. Research on the risk factors for the development of CNSLBP should have a 'bench to clinic' (laboratory to clinic) approach to include the potential influence of factors from genetic predisposition to psycho-social factors and cognitive behaviour on the development of CNSLBP.

6.5 Summary

This thesis was initiated by the researcher's realisation and the confirmation in the literature that CNSLBP is still a major problem that has not been addressed sufficiently by the health care professions. A review of the literature has revealed opposing and complementary viewpoints on the management of patients with CLBP – all of them between 2007 and 2012.

How it should all be integrated in clinical practice is still not well conceptualised and discussed in the literature. The core of the problem has been identified as the lack of a multidimensional conceptualised framework to explain the possible causes of and contributing factors to the heterogeneous nature of CNSLBP and the principles of management of these patients with CNSLBP.

Based on her experience in clinical practice and ongoing continuing professional education and her approach of working in a team as well as educating postgraduate students, the researcher has developed a multidimensional approach to the management of patients with CNSLBP which addresses their problem(s) with substantial success. Through a process of metacognitive reflection on her clinical reasoning during the management of patients with CNSLBP within a hermeneutic research approach and interpretive paradigm, the researcher aimed to verbally

express her tacit and clinical knowledge, supporting and adapting her observations and reasoning based on evidence from the literature (propositional knowledge). The principles of the research process are described in Chapter 2 and were found to be similar to the principles of the processes used by expert clinicians to generate clinical knowledge (Edwards & Jones, 2007).

As premise for the multidimensional manual therapy model for the management of patients with CNSLBP the researcher substantiated the concept of the integrated spinal movement system (ISMS) from a literature review based on her observations in clinical practice. From her clinical experience the researcher observed that the whole spine, position of the head, shoulder and pelvic girdles of patients with CNSLBP were affected by the 'low back pain' and discovered that pain is referred to the 'low back' from other areas in the spine, and that it was associated with neck pain. From about 1993 the researcher started treating patients with CNSBP's 'whole back by releasing soft and neural tissues, including the position of the head, shoulder and pelvic girdles and re-aligned of the spinal motion segments through intervertebral mobilisation. She then realised the interaction between the 'low back pain' and the neck was probably through the deep structures of the thoraco-lumbar fascia pulling on the base of the skull. The researcher conceptualised the ISMS based on the clinical conclusion that patients who present with CNSLBP's whole ISMS should be treated through manual therapy.

In addition to the conceptualisation of the ISMS in Chapter 3, the concept of ISMS dysfunction was developed in this thesis through a literature review mainly on the biomechanics of the spine as a result of pain processing due to abnormal spinal loading. Further literature review revealed the inseparable link between the psychological response typical in patients with CNSLBP and ISMS dysfunction.

ISMS dysfunction, pain processing and the development of characteristic adaptive behaviour are discussed as the integrated components of the complex heterogenetic clinical picture of patients with CNSLBP.

In Chapter 4 the assessment of patients, based on this complex heterogenetic clinical picture, is discussed and displayed in the second stage of model development as the 'multidimensional integrated model' for the assessment of patients with CNSLBP'.

- The principles of the multidimensional manual therapy management of the typical patient with CNSLBP are discussed in Chapter 5. These principles include the: Release of soft and neural tissues and realignment of all the components of the ISMS by re-modulation of the soft and neural tissues. Retraining of tactile discrimination;
- Modulation of pain on peripheral, central (spinal cord) and brain through cognitive behavioural education;
- Relevant muscle recruitment within the patient's physical, psychological and cognitive limitations/requirements;
- Giving the patient appropriate patient-specific information / education on what the condition entails, to address the patient's anxiety about his/her condition, fear avoidance, catastrophisation healthy lifestyle. and
- Retraining of postural control including during functional ADL to address characteristic adaptive behaviour.

All these aspects are interrelated and are addressed in an interrelated way, which is characteristic of a multidimensional manual therapy model for the management of patients with the complex heterogenetic clinical picture characteristic of CNSLBP.

Finally the researcher evaluates the multidimensional manual therapy model in the context of related models, clinical trials in which the effect of manual therapy was compared to various forms and combinations of exercise therapy and education. Suggestions for further research are briefly presented.

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