CHAPTER 1
THE PROBLEM

1.1 Introduction

Traditionally, the most diagnosed characteristic of low back disorders is pain, which has become a very common disorder (Goldby *et al*., 2006). In recent times, pain has been recognized as a disease in itself with its own set of consequences, and no longer just as a symptom of some other type of disease or disorder (Meyer, 2007).

All innervated spinal tissues can be a potential source of pain, and due to the multifactorial etiology of chronic low back problems it has been suggested that there is still a large amount of uncertainty to the cause of low back pain (Kääpä *et al*., 2006). Low back problems can arise from area of the extensive network of intersecting nerve fibres that supply the lumbosacral region, the vertebral periostium, intervertebral discs, neurovascular region, back extensor muscles, tendons, ligaments, vessels, fascias, zygapophyseal joints and sacroiliac joints, or it can even arise from the visceral organs (Schwarzer *et al.,* 1994; Freemont *et al.,* 1997; Simmonds & Dreisinger, 2003; Adams, 2004).

When applied load exceeds the failure tolerance, injury or tissue failure can result, which includes injuries from micro-trauma right up to total failure of the tissue such as ligament avulsion accompanying a fracture (McGill, 2002). The human pain experience is a multidimensional feature with sensory-discriminative, affective-motivational motor and autonomic components, which is critical to the survival of human beings and to maintain the integrity of the body. Pain has been described as an emotional, subjective and complex sensation (Treede *et al.,* 1999).

Pain is not always an indication of underlying problems. There exists a lack of understanding with regard to the multifactorial nature of low back problems, and this is reflected in the large variety of available treatments, ranging from medically oriented treatments such as injection therapy and surgery to behaviour oriented approaches (Nelemans *et al.,* 2001; Van Tulder *et al.,* 2001; Staal *et al*., 2005). A large variety of treatments exists for the treatment of low back pain, from invasive procedures such as surgery and injection therapy to more psychology-based

approaches. Due to this, it has been suggested that there is a lack of understanding with regards to the multifactorial nature of low back pain. It has also been suggested that pain is not always an indicator of underlying problems (Nelemans *et al.,* 2001; Van Tulder *et al.,* 2001; Staal *et al.*, 2005).

Low back pain has a prevalence rate of about 84% (Simmonds & Dreisinger, 2003; Hildebrandt *et al.*, 2004). A prevalence rate of about 63.9% has been reported in South Africa (Van Vuuren *et al.,* 2005). About 44-78% of people suffer relapses in low back pain after an initial episode and 26-37% experience relapses in absence from work (Hildebrandt *et al.*, 2004). In those suffering from low back pain, about 11-12% are disabled by their pain.

In all cases of low back pain, being able to trace the pain back to a specific cause is rare, with only 15% of all cases able to be traced back to a specific cause (Hildebrandt *et al.*, 2004). Chronic low back pain will be the end result for about 5-10% of those who suffer an initial episode of back pain (Linton *et al.*, 2005). These patients undergo considerable suffering because of pain and reduced function as well as accounting for high costs from health care utilization and compensation for lost work (Dionne, 1997).

Direct and indirect costs of low back pain in the United States annually has been reported to be around $25-$50 billion because of pain and disability, which continues to rise and contributes to a substantial economic burden (Frymoyer & Cats-Baril, 1991; Frymoyer, 1992). The losses due to loss of productivity are around $28 billion (Rizzo *et al.,* 1998). Those with low back pain also suffer higher health care costs, which are around 60% greater than healthy individuals (Luo *et al.,* 2004).

This again demonstrates the severe impact of low back pain on society. Primary health care consultation in the United Kingdom for low back pain is the second highest reason why health care is sought (Deyo & Phillips, 1996). Medical expenditure in the United States due to low back pain is responsible for billions of dollars being spent each year (Childs *et al.,* 2004). This impact of low back pain on the economy is of particular concern for poorer continents such as Africa, where epidemics such as HIV/AIDS already take up a majority of the governmental

expenditure towards health care (Walker, 2000). This again demonstrates the severe impact of low back pain on society.

Work absenteeism accounts for a large proportion of the socio-economic burden of low back pain. Restrictions on normal activities and the participation in activities such as the ability to work can lead to absenteeism. This can be caused by low back pain that leads to disability, which will place significant restrictions on these usual activities (Katz, 2006). The largest number of low back pain related economic costs in Western societies is reported to include work absenteeism and disablement (Andersson, 1999). It has also been reported that the majority of low back pain episodes will still result in a full return to work capacity (Phelps *et al.,* 2004), but those that become chronic has been widely reported to be responsible for a large portion of the total number of work absenteeism and accounts for a large portion of the economic and social burden of low back pain (Burton, 2005).

An important part of the treatment for low back pain has been suggested to be a return to work, which should be more than just a treatment goal and an outcome measure that is used in research (Staal *et al.,* 2005). When the disabled worker is involved in the work setting, they can realize that despite the discomfort due to pain that they can still function. Being at work, either in partial or full-capacity, attention is drawn away from the negative issues such as pain and helps to decrease the focus of the disabled worker from pain (Staal *et al.,* 2005).

This demonstrates the need for those with chronic low back pain to remain active regardless of the discomfort suffered. A very important economic implications is that patients who are absent from work for more than six months has a 50% chance of returning to work. This number becomes lower the longer the person is away from work. Being off from work for more than one year has a 25% chance of returning to work and being off for longer than two years has less than a 5% chance of returning to work (Bergquist-Ullman & Larsson, 1977).

The summary of the recommendation comes down to the guidelines that recommend that when a person first represents with acute low back pain, they first have to be examined for the so-called ‘red flags’, which are indications of serious underlying

pathology (Koes *et al.*, 2001). If the patient doesn't present with any red flags, current recommendations state that they should be advised to continue or gradually resume their activities of daily living (Waddell *et al.*, 1997; Koes *et al.*, 2001; Waddell, 2004). Beyond this, further recommendations state that treatment has to be delayed until the patient has been away from work for at least 4-6 weeks. This is only to prevent the slip into chronicity, as many patients will recover spontaneously from an episode of acute low back pain (Frank *et al.*, 1996).

Psychological, social and occupation factors increase in influence of back pain becoming chronic, because the longer that a patient experiences low back pain and subsequent disability, the more the factors will become significant (Waddell *et al.*, 1996). For the treatment of chronic low back pain, conservative treatment has become the most recommended form of treatment (Shirado *et al.,* 2005). Absence from work because of back pain recommended to be prevented by physical exercise, as well as preventing further episodes and the severity of pain episodes (Burton, 2005). Exercise therapy is thus recommended by many guidelines, and it is one of most recommended guidelines for the treatment of chronic low back pain (Spitzer *et al.,* 1987; Albright, 2001; Hayden *et al.,* 2005; Krismer & Van Tulder, 2007). The pathogenesis of low back pain shares a close relationship with impaired function of the trunk muscles (Shirado *et al.,* 1992; Shirado *et al.,* 1995a; Ito *et al.,* 1996).

To strengthen the trunk muscles and improving their flexibility is the primary purpose of therapeutic exercises for chronic low back pain (Shirado *et al.,* 1995b). It is recommended by current evidence to use multidisciplinary/interdisciplinary approaches in the treatment for chronic low back pain, because multiple therapies are incorporated in a coordinated manner which facilitates active interaction and a common philosophy that promotes active involvement from the patient in the rehabilitation programme (Ashburn & Rice, 1998; Ashburn & Staats, 1999; Karjalainen *et al.,* 2001; Karjalainen *et al.,* 2003a; Rome *et al.,* 2004).

This approach includes either one of the psychological, social or occupation dimensions along with the physical exercise therapy (Guzman *et al.,* 2001). Functional capacity and the development of coping strategies was the reason that the multidisciplinary programmes have been developed (Linton & Andersson, 2000).

Providing accurate information about back pain, to lend attitudes that are favourable towards self-care, to reduce fears and worries, to assist patients in developing personalized action plans to manage their back pain and to improve functional outcomes are all established goals of the multidisciplinary approach (Kääpä *et al*., 2006).

The use of exercise therapy as part of the multidisciplinary approach has become very popular for the treatment for chronic low back pain (Staal *et al*., 2005). However, as suggested by McGill (2002), many of the studies focusing on exercise treatments have not made assessments about progressive treatment that change as the patient move through the rehabilitation process, and have only focused on single treatment methods.

Patient needs change throughout the rehabilitation process. Therefore, the applied exercises should also change and adapt to accommodate these changes by increasing in difficulty as the levels of exercise tolerance of the subject increase (McGill, 2002). Also, many of the exercise programmes have been conservative in nature (Richardson *et al.,* 1999; Hides *et al.,* 2001) and it is not clear if this is sufficient to restore full functional capacity.

The important part will be to select exercises that are safe, yet effective. In the rehabilitation process, functional progression is needed to stress tissue sufficiently that will allow it to adapt to other kinds of stresses that a patient will be exposed to in every situations. Otherwise, tissue failure due to weakness can lead to injury (Prentice, 2004). Unfortunately, the effect of more aggressive types of exercises has not been assessed adequately. In order to fully restore functional capacity and provide tissue with enough strength to sustain loads applied to the body, it might be necessary to include more aggressive types of exercises to not only strengthen the muscles of the low back, but also to strengthen the muscles used for functional tasks in a safe and effective way (McGill, 2002). The important part will be to select exercises that are safe, yet effective.

Approaches that put emphasis on functional restoration and the improvement of functional capacity produces the best results in the management of chronic low back

pain. These approaches focus on the development of coping strategies to help those with chronic low back pain in the long term (Linton *et al.,* 2000; Caraggee, 2005). It thus provides them with a type of self-management strategy (Rasmussen-Barr *et al.,* 2009).

Taking this into account, it can be argued that treating musculoskeletal dysfunctions alone may not be beneficial without directly addressing psychosocial factors that contribute to the experience of pain (Geisser *et al.,* 2005), thus combining more than one treatment modality. This is especially true if the type of pain is considered in chronic low back pain, which does not always indicate that a problem exists (Meyer, 2007).

Several recent studies have provided evidence that treatment programmes containing active exercises are equally effective in patients with chronic low back pain, irrespective of the type of exercises compared. This suggests that any type of intensive exercise programme that manages to cause patients to expand the limits of their physical functioning may provide them with a method for increasing the feeling of pain control, thus inhibiting negative pain behaviour relating to a chronic low back problem (Petersen *et al.,* 2002).

In a well-designed randomised controlled trail Petersen *et al*. (2002) compared the effect of McKenzie therapy to intensive strengthening in the treatment of chronic low back pain. They concluded that the McKenzie method and intensive strength training seem to be equally effective in the treatment of chronic low back pain. However, frequently used, popular exercise regiments, like the McKenzie technique, are not adequately researched.

The European Guidelines for the Management of Chronic Non-Specific Low Back Pain suggest that there is a need to investigate the optimal intensity, frequency, duration and specific types of exercises used in the clinical setting and academic literature (Hildebrandt *et al.,* 2004). It also remains unclear whether any specific type of exercise (flexion, extension or strengthening exercises) is more effective than another (Van Tulder *et al.,* 2000).

Problems associated with chronic low back pain such as restricted functioning, disability and absence from work can cause more long-term problems and disability than pain alone, and it has been suggested that pain treatment has to be a secondary goal, as these other problems cause more long-term complications (Staal *et al.,* 2003; Staal *et al.,* 2005). Treatment should thus focus rather on the consequence of pain, such as a loss of function, physical inactivity and work absenteeism. Pain varies from day to day and is thus not necessarily of long-term concern (Staal *et al.,* 2005). A high recurrence rate of low back pain will likely lead to a life that is not totally free of pain, but pain can be managed and restricted (Staal *et al.,* 2005).

Future studies must assess the efficacy of staged programmes in which categorised patients follow progressive treatment involving several sequenced approaches (McGill, 2002). More aggressive types of remedial exercises, as well as specific types of exercises have to be incorporated in order to establish if they are more effective in the treatment of chronic low back pain.

1.2 Research Questions

For this study the following research questions were used:

- ***How effective are progressive-aggressive exercises vs. more traditional exercise in the treatment of chronic non-specific low back pain?***
- ***What exercises will be effective and how effective they will be when progressed?***
- ***How will a more aggressive approach influence the outcomes as compared to the more traditional approaches to remedial exercise therapy?***

1.3 Research Hypothesis

In the light of the aim of this study the following research hypothesis was formulated:

A progressive-aggressive multi-component rehabilitation programme in conjunction with high intensity back school will be more effective in the

improvement of chronic low back pain outcomes than a conservative rehabilitation programme in conjunction with a low intensity back school.

Significance was set at p≤0.01 level of significance.

1.4 Goals of the Study

The following goals were set prior to the study commencing:

Develop a progressive-aggressive exercise programme, a conservative exercise programme and a back school curriculum.

1.5 Objectives of the Study

This study aimed to achieve the goal through the following objectives:

- Building a theoretical frame of reference on existing literature with specific focus on topics such as chronic pain, work absenteeism, remedial exercise therapy and full working capacity adults;
- Identification of the major components involved in chronic low back pain, as well as the mechanisms involved in chronic pain (neuropathic pain, etc.);
- Description of the remedial exercise use and function associated with chronic low back pain;
- Identification and description of a test battery that will be suitable in assessing the critical components of the low back;
- Description and illustration of the different exercises to be used with the progressive-aggressive exercise programmes, as well as the conservative exercise programmes;
- Guidelines for the implementation of the progressive-aggressive exercise programme; and
- Suggestions for future research on the topic of low back pain, which remains a topic with many unanswered questions.

1.6 Research Design

The present study mainly made use of qualitative research methods. Qualitative research methods generally include field observations, case studies, ethnography and narrative reports (Linn, 1986). This type of research seeks to understand the meaning an experience has for the participants in a specific setting and how the components mesh to form a whole (Thomas & Nelson, 2001).

The objectives are primary description, understanding and meaning. Thus the researcher does not manipulate the variables through experimental treatments alone but rather takes more interest in the process than the product (Patton, 1987). The researcher observes and gathers data in the field, which is the natural setting (Thomas & Nelson, 2001). Qualitative research methods, however, are very subjective. Here the researcher is the primary instrument for data collection and analysis, as well as interacting with the subjects to observe their responses and changes (Patton, 1987).

The type of research design used in the present study was that of experimental research. With this type of research the independent variables are manipulated in an attempt to judge their effect on the dependant variable. A well-designed study is one in which the only explanation for the change in the dependant variable will be due to the applied intervention (Thomas & Nelson, 2001). Thus, the major advantage of this type of research is that the researcher is able to manipulate the treatments to establish a cause-and-effect relationship (Thomas & Nelson, 2001). Cause and effect can only be established by the application of logical thinking to well-designed experiments (Kratwohl, 1993).

Three criteria need to be present to establish cause and effect:

1. The cause must precede the effect in time.
2. The cause and effect must be correlated with each other.
3. The correlation between cause and effect cannot be explained by another variable. (Kratwohl, 1993)

The researcher attempts to control all factors except the experimental variable. If extraneous factors can be controlled successfully, then the researcher may presume that the changes in the dependant variable are due to the independent variable (Thomas & Nelson, 2001).

This presumption has to include the following aspects:

- The selection of a good theoretical framework
- The use of appropriate participants
- The application of an appropriate experimental design
- The proper selection and control of the independent variable
- The appropriate selection and measurement of the dependent variable
- The use of the correct statistical model and analysis
- The correct interpretation of the results (Kratwohl, 1993)

The type of study design used in the present study was a True Experimental Design, which basically facilitates the random allocation of groups to ensure that all sources of invalidity and all threats to design have been counteracted (Thomas & Nelson, 2001). More specifically, the present study was a Pre-test-post-test Randomised Groups Design, in which the groups were randomly formed but both groups were also given a pre-test, as well as a post-test. The major purpose of this type of design is to determine whether the experimental group showed more improvement than the control group (Thomas & Nelson, 2001).

1.7 Research Procedure and Strategy

This study included subjects, both male and female, between the ages of 20 and 55 years of age. They were randomly assigned to either a control or experimental group. Each participant was also assigned a unique study number and this number will be randomly linked to the data. Patients' names and file numbers will have no correlation to the study number. The unique study number will prevent patients from

being identified, even by the researcher, and this will ensure the confidentiality of the records in order to protect patients' privacy.

1.7.1 Inclusion Criteria

Inclusion criteria consisted of suffering from back pain for longer than 12 weeks, based on the classification of chronic low back pain recommended by Abenheim *et al.* (2000) and Burton *et al.* (2004). The source had to be chronic in nature, which was confirmed by the physical testing procedures and questionnaires. Subjects could not suffer from low back pain caused by the so-called 'red flag' conditions. Subjects were included who suffered from radiating symptoms in the legs, but this again could not be caused by any 'red flag' conditions.

1.7.2 Exclusion Criteria

Exclusion criteria consisted of any previous back surgery, any known spinal pathology and discogenic diseases, as well as the so-called 'red flag' conditions. These include weakness, particularly if localised in one area such as the leg; pain and/or difficulty controlling the bladder; numbness or tingling in the feet, legs or groin; severe, disabling or night pain; serious pain and a history of cancer or intravenous drug use; pain that does not subside within a couple of days; pain in the abdomen, as well as fever and weight loss along with back pain.

Current pregnancy, as well as ongoing disability and injury compensation cases were also excluded. Subjects were not excluded based on previous treatment modalities such as physiotherapy. However, they could not seek these types of treatments during the course of the study. Subjects were also excluded if they had a Body Mass Index (BMI) of over 40. By default this condition puts too many compressive forces on the low back and may contribute to chronic low back pain (McGill, 2002).

1.7.3 Study Sample

Subjects for this study were recruited by placing advertisements in local newspapers, as well as broadcasting the advertisement on the local radio station. Physicians were also contacted for possible referrals for likely and willing participants seeking long-term help. After initial contact with the possible subjects, they underwent a thorough

screening procedure by the main investigator to ensure that they fitted the criteria of chronic low back pain. Potential subjects then gave written informed consent prior to participation. The subjects selected were then randomly assigned to either the control or the experimental group.

Research subjects were given the questionnaires to complete at the initial testing session. After the 12 week intervention period they were again given the questionnaires to complete. Subjects were given 20 minutes to complete the questionnaires. The researcher administrating the tests was blinded to the allocation of the subjects at each of the test sessions by means of random numbers. Immediately after the questionnaires had been completed, the physical tests were administered on the subjects. The questionnaires were repeated after every four weeks during the intervention period when the exercise programme of the experimental group progressed.

To prevent test results from being confused, the subjects were tested individually and not allowed to interact with one another during the test administration. This prevented subjects from copying one another's answers because of what they thought the answers should be as required by the researchers. Subjects had to provide answers to what they were experiencing; not to what they thought the researchers wanted to hear.

1.7.4. Intervention

- The timeframe for the intervention was 12 weeks.
- Subjects were randomly assigned to two groups. The control group performed the conservative exercise programme while the experimental group performed the progressive-aggressive exercise programme.
- After every four weeks the exercise programme of the experimental group was progressed to the next level of difficulty (at Week 4 and Week 8 up to the maximum of Week 12).
- Both groups performed the exercise sessions twice per week.

- The control group also received low intensity back school intervention while the experimental group received high intensity back school intervention.
- High intensity back school consisted of two sessions per week for 40 minutes, where the principles of the cognitive behavioural approach was applied. This included the exercise session and the back school session.
- In turn, the control group performed the low intensity back school in the form of reading the back school document on their own, but still exercised twice per week. The control group back school was done in the form an informative book given to the subjects to study. This procedure is based on the guidelines suggested by Heymans *et al.* (2006).

1.7.5 Back School

The back school included a high intensity approach for the experimental group and low intensity for the control group. High intensity-based back schools have been shown to be more effective to a certain degree than low intensity back schools (Heymans *et al.*, 2006). High intensity back school consisted of two sessions per week for an hour (including the exercise session) during which the principles of the cognitive behavioural approach were applied.

In turn, the low intensity back school was applied twice a week (with two exercise sessions per week) during which subjects were handed an informative book. This book contained all of the necessary information but subjects had to study the content on their own. The experimental group was given information as well but it was discussed with them in detail. This procedure is based on the guidelines suggested by Heymans *et al.* (2006).

1.8 Definition of Key Concepts

- **Back Pain:** Pain and discomfort localised below the costal margin and above the inferior gluteal folds, with or without leg pain (Van Tulder *et al.,* 2004)

- **Pain:** An unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage (Meyer, 2007).

- **Chronic Low Back Pain:** Low back pain that persists for more than 12 weeks (Burton *et al.,* 2004). This is pain that persists for longer than the time expected for healing or pain associated with progressive, non-malignant disease, usually taken to be three months (Meyer, 2007).

- **Therapeutic Exercise:** Exercise performed for the purpose of cure or restoration of function (Abenhaim *et al.,* 2000).

- **Progressive-Aggressive Exercise:** Exercise that increases in difficulty as the needs of the patient change to create a need for higher levels of intensity and exercise tolerance (McGill, 2002).

For the purpose of this study, conservative exercise will imply exercises that aim to strengthen the local muscle group only. Aggressive exercises will include exercises that strengthen the local muscle group as well as the global muscle group.

- **Full Working Capacity Adults:** Adults who suffer from chronic low back pain but suffer virtually no disability at all due to the pain. They are still working full-time in terms of hours, duties and travelling time, but are required to take part in a back rehabilitation programme.

- **Multidisciplinary/Interdisciplinary Rehabilitation:** Rehabilitation which includes the physical dimension and at least one of the other dimensions: psychological, social or occupational dimensions (Guzman *et al.,* 2001).

- **Neuropathic Pain:** Injury or disease of neurons in the peripheral or central nervous system (Jänig & Baron, 2003).

- **Fear Avoidance Behaviour:** The perception of pain in a threatening, catastrophic manner (as in signs of tissue damage); experiencing of pain-

related fear and anxiety, and consequently engaging in escape or avoidance behaviours to situations that are perceived to be potentially harmful (Thomas & France, 2007).

CHAPTER 2
LITERATURE SURVEY

2.1 Pain and its Physiology

Pain is an important symptom of a wide variety of different diseases and disabilities (Schaible & Richter, 2004). However, as stated by Meyer (2007), chronic pain has been recognised in recent times as not only a symptom, but also as a disease itself.

According to Meyer (2007:20), a current definition of pain proposed by the IASP (International Association for the Study of Pain) states: "*Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage*". The casual mechanism in pain, especially musculoskeletal pain is a very complex phenomenon, as the experience of pain itself may emphasize the negative effects of experienced external stressors, or provoke psychological and biological reactions that maintain or increase pain in a vicious circle (Svebak, 2000).

It has been reported that the experience and regulation of social and physical pain share a common neuro-anatomical basis in the brain (Eisenberger *et al.*, 2003). Pain can be described as a sensation that is evoked by potential or actual noxious (toxic) stimuli or by actual tissue damage (Schaible & Richter, 2004). A potential source of low back pain for example can arise from any part of the extensive network of intersecting nerve fibres that supply the lumbosacral region, the vertebral periostium, intervertebral discs, neurovascular region, back extensor muscles, tendons, ligaments, vessels, fascias, zygapophyseal joints and sacroiliac joints, or it can even arise from the visceral organs (Schwarzer *et al.*, 1994; Freemont *et al.*, 1997; Simmonds & Dreisinger, 2003; Adams, 2004).

Also, advanced atherosclerosis presenting calcific deposits on the posterior wall of the abdominal aorta and in vertebral arteries has been suggested to be associated with advance types of disc degeneration and the subsequence occurrence of low back pain (Kauppila *et al.*, 1997).

Injury or tissue failure can also occur when the applied load exceeds the failure tolerance of the muscle, and includes micro-trauma all the way up to gross tissue failure, such as fractures and ligament avulsion (McGill, 2002). However, it is generally accepted that non-innervated tissue cannot be the origin of pain sensation (Aoki *et al.*, 2006), such as the intact lumbar intervertebral disc, which has been shown to be aneural except in the outermost part of the annulus fibrosus (Bogduk *et al.*, 1981; Coppes *et al.*, 1990; Konttinen *et al.*, 1990; Roberts *et al.*, 1995; Freemont *et al.*, 1997; Palmgren *et al.*, 1999).

These nerves extend no further than the outer few lamellae of the annulus fibrosis (Coppes *et al.*, 1990; McCarthy *et al.*, 1991; Freemont *et al.*, 1997). Some of these nerves contain neurotransmitters such as substance P and calcitonin gene-relating peptide, indicating a possible nociceptive function and subsequent pain regulation function (Ahmed *et al.*, 1991; Grönblad *et al.*, 1991; McCarthy *et al.*, 1991; Ahmed *et al.*, 1993; Ashton *et al.*, 1994; Roberts *et al.*, 1995).

However, both nerve fibre and blood vessel ingrowth deeper into the annulus fibrosis and even up to the nucleus pulposus have been demonstrated in degenerative discs, acting as a possible source of pain perception (Coppes *et al.*, 1990; Kauppila, 1995; Palmgren *et al.*, 1996; Freemont *et al.*, 1997; Repanti *et al.*, 1998). Even high levels of serum blood cholesterol levels have been associated with low back pain in some people (Kauppila *et al.*, 2004).

This network of tissue is the reason why the source of pain is frequently difficult to determine, as any innervated structure of the low back can trigger a possible nociceptor and thus a painful signal, and most of the structures in the low back are well innervated, relatively small and in close proximity to each other (Simmonds & Dreisinger, 2003).

Pain can be classified as being either acute or chronic in nature (Meyer, 2007). Acute pain is usually of known cause, and there are only minor contributions from the emotional and cognitive dimensions, but in chronic pain patients the pain can last for up to many years, and the emotional and cognitive dimensions can potentially play a much larger role in pain perception over a long period of time (Johnson, 1997;

Simmonds & Dreisinger, 2003). According to Meyer (2007) a large number of brain regions that are also described as the so-called "pain matrix" are activated following a stimulus that causes pain. Rather than registering the pain signal, it is communicated to the body as a perception of pain, and the brain matrix constructs the pain experience by integrating multiple inputs that includes biological (sensory) factors, past and present psychological events and socio-cultural influences.

Recognizing the importance of pain in modern research settings, Schaible & Richter (2004: 237) stated the following: "*Pain is a major symptom of many different diseases. Modern pain research has uncovered important neuronal mechanisms that are underlying clinically relevant pain states, and research goes on to define different types of pains on the basis of their neuronal and molecular mechanisms. This review will briefly outline neuronal mechanisms of pathophysiological nociceptive pain resulting from inflammation and injury, and neuropathic pain resulting from nerve damage."*

The human pain experience is a complex sensation that is of paramount importance to maintain the body integrity and survival of human beings, as it is a multidimensional phenomenon with sensory-discriminative, affective-motivational, motor and autonomic components (Treede *et al.*, 1999). Treede *et al.* (1999) argued that the human pain experience is a complex sensation that is of paramount importance to maintain the body integrity and survival of human beings. It is a multidimensional phenomenon with sensory-discriminative, affective-motivational, motor and autonomic components, making it a very effective phenomenon. Understanding the function of the pain system is of primary concern.

2.1.1 The Pain System

Schaible & Richter (2004: 237) stated that:" *Precisely, the 'pain system' should be called the 'nociceptive system' because pain is a subjective result of nociception. Nociception is the encoding and processing of noxious stimuli in the nervous system that can be measured with electrophysiological techniques."* This results due to the activation of complex and integrated networks of neurons that are prone to string loops consisting of automatic regulation and fast changing neuroplastic responses (Verdu *et al.*, 2008).

Schaible & Richter (2004: 237) further stated that:" *A noxious stimulus activates nociceptors (Aά and C fibres) in the peripheral nerve. Their sensory endings are so-called free nerve endings, i.e. they are not equipped with corpuscular end organs. Most of the nociceptors are polymodal, responding to noxious mechanical stimuli (painful pressure, squeezing or cutting of the tissue), noxious thermal stimuli (heat or cold), and chemical stimuli (Belmonte & Cervero, 1996)."* This opens ion channel transducers to accelerate the nociceptive pain experience (Verdu *et al.,* 2008).

Schaible & Richter (2004: 237) further states that: "*Sensor molecules in the sensory endings of nociceptors transduce mechanical, thermal and chemical stimuli into a sensor potential, and when the amplitude of the sensor potential is sufficiently high, action potentials are triggered and conducted by the axon to the dorsal horn of the spinal cord or the brainstem. Nociceptors can also exert efferent functions in the tissue by releasing neuropeptides [substance P, calcitonin gene-related peptide (CGRP)] from their sensory endings. Thereby, they induce vasodilatation, plasma extravasation and other effects, e.g. attraction of macrophages or degranulation of mast cells. The inflammation produced by nociceptors is called neurogenic inflammation (Foreman, 1987; Lynn, 1996)."*

Schaible & Richter (2004: 237-238) further states that: *"Nociceptors activate synaptically nociceptive dorsal horn neurons. The latter are either ascending tract neurons or interneurons that are part of segmental motor or vegetative reflex pathways. Ascending axons in the spinothalamic tract activate the thalamocortical system that produces the conscious pain sensation. The pain sensation has a sensory discriminative aspect, i.e. the noxious stimulus is analysed for its location, duration and intensity. This is produced in the lateral thalamocortical system, which consists of relay nuclei in the lateral thalamus and the areas SI and SII in the postcentral gyrus. A second component of the pain sensation is the affective aspect, i.e. the noxious stimulus feels unpleasant and causes aversive reactions. This component is produced in the medial thalamocortical system, which consists of relay nuclei in the central and medial thalamus and the anterior cingulate cortex (ACC), the insula, and the prefrontal cortex (Basbaum & Jessell, 1999; Treede et al., 1999).*

The spinal cord is under the influence of descending tracts that reduce or facilitate the nociceptive processing. Descending inhibition is formed by pathways that originate from brainstem nuclei (in particular, the periaqueductal grey, nucleus raphe magnus) and descend in the dorsolateral funiculus of the spinal cord. This system is able to suppress nociceptive information processing via interneurons in the dorsal horn of the spinal cord (Basbaum & Jessell, 1999)".

However, the concept of the nociceptive pain system being the only cause of physiological pain production has been challenged by the so-called 'gate-control theory' of Melzack and Wall in 1965, which integrates the views of neurophysiology and psychology (Melzack & Wall, 1965).

According to Melzack & Wall (1965), the theory states that spinal transmission of pain impulses is continuously modulated by the relative activity in the small (A-delta and C) fibres and the large (A-beta) fibres. The descending impulses from the brain that originate in the cerebral cortex and brainstem also plays a role, and the subsequent irritation that is felt as pain will be reinforced by different parts of the central nervous system.

Under these circumstances, psychological and social factors also have an influence on pain sensation (Nykänen & Koivisto, 2004). Revolutionary was the finding that pain is not just the result of nociceptive information ascending from the periphery, but that it is also profoundly moderated by descending pathways (Vlaeyen & Morley, 2005). It is because of the advent of this theory that the modern paradigm of pain management has moved away from the classic biomedical approach to the broader bio-psycho-social approach, where pain physiology now integrate input from sensory, emotional and cognitive systems (Main & William, 2002; Merskey *et al.*, 2005; Justins, 2005).

The biomedical perspective has thus evolved into a broader conceptual framework, which addresses the influence and effect of psychological and social factors in the pathophysiology and prognosis of pain and subsequent disability (Staal *et al.*, 2005). Moreover, the nociceptive signal is modified during its transmission from the

peripheral nerves to neurons in the spinal cord and then up to the sensory centres of the brain.

The interpretation of the pain signal is now recognized to be influenced by numerous psychological (such as past experience and mood) and social (such as work, leisure activity) factors (Simmonds & Dreisinger, 2003). This change in thinking identifies the complex and multi-dimensional experience of pain where the patient's physical, cognitive, emotional and behavioural characteristics mediate the pain experience and can influence the reaction of the person based on these factors (Katz, 2000). Currently, chronic pain states are attributed to abnormal nociceptive/antinociceptive function on different levels of the neuroaxis with normal brain structure (Wall & Melzack, 1999).

2.1.2 Types of Pain

Different types of pain can be distinguished. On the bases of aetiology and neurobiological mechanisms Treede *et al.* (2008) have identified different types of pain:

1) nociceptive pain, caused by any lesion or potential tissue damage;
2) inflammatory pain, due to inflammatory processes; and
3) neuropathic pain, induced by a lesion or disease affecting the somatosensory system (Treede *et al.*, 2008).

In the absence of a neurological disorder or peripheral tissue abnormality another type of pain has been suggested in the form of functional/dysfunctional pain, which is supported by the existence of an abnormal central operation of inputs leading to pain hypersensitivity (Talley & Spiller, 2002; Desmeules *et al.*, 2003; Banic *et al.*, 2004; Harris *et al.*, 2007).

2.1.2.1 Nociceptive Pain

Schaible & Richter (2004: 238) stated that: "*When a noxious stimulus is applied to normal tissue, acute physiological nociceptive pain is elicited. This pain protects tissue from being further damaged, because usually withdrawal reflexes are elicited. Pathophysiological nociceptive pain occurs when the tissue is inflamed or injured.*

This pain may appear as spontaneous pain (pain in the absence of any intentional stimulation) and/or as hyperalgesia and/or allodynia."

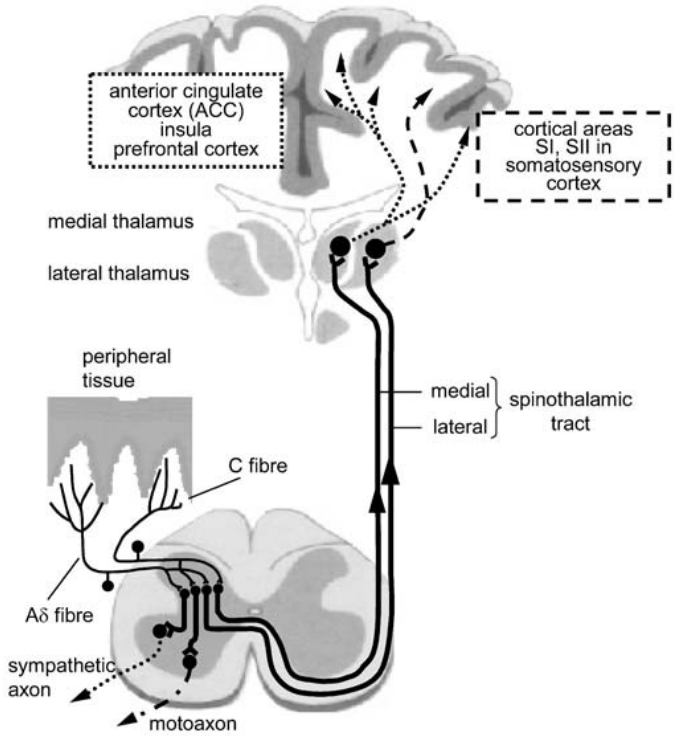

Figure 2.1: The Nociceptive Pain System (Schaible & Richter, 2004)

Seifert and Maihöfner (2008) reported that hyperalgesia can be differentiated into primary and secondary hyperalgesia, where primary hyperalgesia results from the sensitisation of peripheral nociceptive structures, and secondary hyperalgesia results from sensitisation processes within the central nervous system. A hypersensitivity towards heat stimuli know as thermal hyperalgesia is a key component of primary hyperalgesia, whereas secondary hyperalgesia is recognised by hypersensitivity towards mechanical stimulation. Schaible & Richter (2004: 238) stated that:" *Hyperalgesia is a higher pain intensity that is felt upon noxious stimulation, and allodynia is the occurrence of pain that is elicited by stimuli that are normally below the pain threshold*."

2.1.2.1.1 Peripheral Mechanisms of Pathophysiological Nociceptive Pain

The sensitizing of the polymodal nociceptors during inflammation is referred to as peripheral sensitization, and tend to have relatively high levels of mechanical and thermal thresholds in normal tissues. They require a high level of intensity from stimuli to cause an excitation of the involved neurons (Handwerker, 1999; Schaible & Schmidt, 2000).

This leads to a situation in which even very slight and non-significant stimuli activates the fibres due the excitation levels of the fibres decreasing during the inflammation process. This causes pain to be experienced due to the sensitized pain fibres which react to non-painful stimuli. Noxious stimuli will still cause a much stronger reaction when compared to the non-sensitized state (Handwerker, 1999; Schaible & Schmidt, 2000).

According to Schaible and Richter (2004) silent nociceptors can be activated by the inflammation process and these C-fibres are, under normal conditions, inexicitable by stimuli that are either thermal or mechanical. However, inflammation changes this condition, in that the primarily mechanosensitive fibres become sensitized and activate when a stimulus is applied. Thus the enhanced activity of the sensitized polymodal nociceptors and the subsequent recruitment of the silent nociceptors cause the pathophysiological nociceptive input to the spinal cord.

Interaction from inflammatory response mediators which include prostaglandins, bradykinin, histamine, ATP and acetylcholine on receptors on sensory endings (Kress & Reeh, 1996; McCleskey & Gold, 1999; Schaible, 2004) activates the neurons directly or causes a sensitization effect for other forms of stimuli (Kress & Reeh, 1996). The activation of secondary messenger by the mediators can then influence specific ion channels in the membrane. This type of reaction then causes an enhanced excitability of the neuron with decreased threshold levels and an increased action potential frequency during higher than normal activation (Bevan, 1996).

Schaible & Richter (2004:239-240) stated that: "*Primary afferent neurons also express receptors for neurotrophins. Neurotrophins are survival factors during the*

development of the nervous system, but during inflammation of the tissue, the level of nerve growth factor (NGF) is substantially enhanced. By acting on the tyrosine kinase A (trk A) receptors, NGF increases the synthesis of substance P and CGRP in the primary afferents. The release of these peptides from the endings produces neurogenic inflammation. NGF may also act on mast cells and, thereby, activate and sensitise sensory endings by mast cell degranulation (Lewin et al., 1994). The sensitisation of nociceptors is rapidly induced, i.e. the changes mentioned can be observed within a few minutes. If noxious stimuli persist, changes in the expression of receptors in the primary afferents are induced. For example, the expression of neurokinin 1 receptors (activated by substance P) and bradykinin receptors is enhanced in rat dorsal root ganglia and in peripheral nerve fibres during persistent inflammation (Segond von Banchet et al., 2000).”

2.1.2.2 Neuropathic Pain

Nociceptive pain has been described as pain that is caused by the stimulation of the sensory endings by noxious stimulation. Another type of pain known has neuropathic pain has also been described. This type of pain occurs due to an injury or a disease of the neurons specifically in the periphery or the central nervous system (Jänig & Baron, 2003). It can also include a lesion in the peripheral or central nervous system; for example, in patients with diabetic or AIDS poly-neuropathy and post herpetic neuralgia (Shipton, 1999; Woolf, 2004; Meyer, 2007).

Hyper-excitability and ectopic activity are unique to neuropathic pain, in that altered membrane excitability and abnormal electrogenesis results lead to the generation of inappropriate action potentials and repetitive firing without a peripheral stimulus (Verdu *et al.*, 2008). This type of pain doesn’t cause normal noxious stimulation of the tissues and tends to feel like an abnormal type of pain, and has been described as a feeling of being burnt and/or a feeling similar to electricity passing through the area, which can become longstanding in nature, or occur in intermitted bursts and can even occur together with hyperalgesia and allodynia (Jänig & Baron, 2003). According to Verdu *et al.* (2008) hyper-excitability and ectopic activity is unique to neuropathic pain. Altered membrane excitability and abnormal electrogenesis result leads to the generation of inappropriate action potentials and repetitive firing without a peripheral stimulus

In the state of allodynia, sensitivity increases dramatically, so that even slight contact with the skin with non-painful touches will cause an intensive amount of pain (Schaible & Richter, 2004). Schaible & Richter (2004: 238) stated that "*Numerous pathological processes can cause neuropathic pain, e.g. axotomy or nerve or plexus damage, metabolic diseases such as diabetes mellitus, or herpes zoster. The complex regional pain syndrome (CRPS) is a neuropathic pain syndrome that involves the sympathetic nervous system (one form was previously called sympathetic reflex dystrophy or Sudeck's disease) (Jänig & Baron, 2003). Damage to central pain processing neurons (e.g. in the thalamus) can cause central pain (Basbaum & Jessell, 1999).*

2.1.2.2.1 Peripheral Mechanisms of Neuropathic Pain

Schaible & Richter (2004: 240) stated that: "*Ectopic discharges do not only occur in Aά-fibres and C-fibres, but also in thick myelinated Aβ-nerve fibres that encode innocuous mechanosensory information. This has led to the idea that, after nerve injury, low threshold Aβ-fibres, as well as Aά-fibres and C-fibres, are involved in the generation of pain. In particular, two mechanisms have been proposed as to how impaired Aβ-nerve fibres might cause pain. First, Aβ-fibres might evoke exaggerated responses in spinal cord neurons that have undergone the process of central sensitisation. Second, Aβ-fibres might sprout into spinal cord layers that are usually only a target of C-fibres, and, thus, these fibres might activate the 'wrong' neurons (Woolf et al., 1992).*"

Schaible & Richter (2004: 240) further stated that: "*Different mechanisms are thought to produce ectopic discharges: changes in the expression of ionic channels, pathological activation of axons by inflammatory mediators, and pathological activation of injured nerve fibres by the sympathetic nervous system. At least six different types of sodium channels were found in primary afferents, two of them being tetrodotoxin (TTX)-insensitive (McCleskey & Gold, 1999). Sodium influx through TTX-sensitive sodium channels into the neuron inactivates very quickly: sodium influx through TTX-insensitive sodium channels is more slowly inactivating (Cummins et al., 2000). After nerve injury the expression of TTX-sensitive sodium channels is increased, and the expression of TTX-insensitive sodium channels is*

decreased, These changes are thought to alter the membrane properties of the neuron, such that rapid firing rates (bursting ectopic discharges) are favoured (Cummins et al., 2000). Changes in the expression of potassium channels of the neurons have also been shown (Everill & Kocsis, 1999).

Injured axons of primary afferent neurons might be excited by inflammatory mediators, e.g. by bradykinin, NO (Michaelis et al., 1998; Ramer et al., 1998; Liu et al., 2000; Levy et al., 2000; Perkins & Tracey, 2000), and by cytokines (Cunha & Ferreira, 2003). The source of these inflammatory mediators might be white blood cells and Schwann cells around the damaged nerve fibres. The sympathetic nervous system does not activate primary afferents in normal tissue. Injured nerve fibres, however, might become sensitive to adrenergic mediators (Lee et al., 1999; Moon et al., 1999; Kingery et al., 2000). This cross-talk might occur at different sites. Adrenergic receptors might be expressed at the sensory nerve ending. A direct connection between afferent and efferent fibres (so-called ephapses) is considered. Sympathetic endings are expressed in increased numbers in the spinal ganglion after nerve injury. The cell bodies of the injured nerve fibres are surrounded by 'baskets', consisting of sympathetic fibres (Jänig et al., 1996). Currently, the best treatment is the application of drugs that reduce the excitability of neurons (e.g. carbamazepine or gabapentin)." The role of exercise in this regard needs to be further researched.

2.1.2.3 Dysfunctional Pain

According to Meyer (2007), large groups of chronic pain patients tend to demonstrate no existing peripheral abnormality or neurological deficit. The mechanism of pain has been described as an abnormal sensory processing of non-painful stimuli once the central nervous system has undergone changes that cause it to become sensitised (Bennett, 1999; Nielson & Nielson, 2003; Woolf, 2004). These disorders include the much described idiopathic pain disorders such as irritable bowel syndrome, chronic headaches, post-whiplash disorders, and fibromyalgia syndrome and others (Diatchenko *et al.*, 2006).

It is important to note that both neuropathic and dysfunctional pain may be present in the absence of an ongoing peripheral stimulus or 'organic cause'. It is wrong to assume that these patients are only psychologically unbalanced or even hysterical

(Nielson & Nielson, 2003; Woolf, 2004; Diatchenko *et al.*, 2006). It is important to note that both neuropathic and dysfunctional pain may be present in the absence of an ongoing peripheral stimulus or organic cause. It is wrong to assume that these patients are only psychological unbalanced or even hysterical OR both neuropathic and dysfunctional pain may be present in the absence of an ongoing peripheral stimulus or "organic cause", and it is wrong to assume that these patients are only "psychological" or "hysterical" (Nielson & Nielson, 2003; Woolf, 2004; Diatchenko *et al.*, 2006).

2.1.2.4 Mixed Pain

These people include those displaying conditions such as cancer pain and low back pain, in particular low back pain following surgery, or failed back surgery syndrome where neuropathic, nociceptive and myofascial components may contribute to the patient's increased pain perception (Shipton, 1999; Woolf, 2004; Meyer, 2007).

2.1.3 Chronic Pain and its Effects

Chronicity can be described in terms of persisting symptoms, disability and work status changes (Pincus *et al.*, 2002). Evidence of distorted local brain chemistry and functional reorganisation in chronic back pain patients support the idea that chronic pain could be understood not only as an altered functional state, but also as a consequence of central plasticity (Flor, 2003). 'Plasticity' is a term used to refer to changes that occur in the established nervous system. It has been observed that plasticity at several levels of the nervous system is related to the transmission of pain signals long after the original cause is gone, depriving pain of its functional role and thus becoming the disease itself (May, 2008).

Recent neurobiological findings suggest cortical reorganisation on a functional level (Grusser *et al.*, 2004). For example, amputation of a limb is often accompanied by phantom pain. In these patients the deafferentation leads to cortical reorganisation where the representational fields of adjacent areas move into the representation zone of the deafferented limb (Flor *et al.*, 2006). This 'functional reorganisation' has not only been detected in patients suffering from phantom limb pain but has also been observed in those suffering from chronic low back pain (May, 2008).

The incidence and prevalence of chronic pain do not appear to be diminishing, but may in fact be on the increase (Harstall & Ospina, 2003). Disability related to back pain has increased dramatically over the past 20 years due to influences in the form of psychological and social factors that influence adaptation to back pain early in the process (Waddell, 1996).

Pain and loss of function associated with musculoskeletal conditions primarily lead to disability (Woolf & Pfleger, 2003). For example, fear of pain and other psychosocial variables during an acute episode of back pain is related to chronic pain status at follow-up (Gatchel *et al.*, 1995; Klenerman *et al.*, 1995; MacFarlane *et al.*, 1999).

Disability is a very important factor regarding low back pain, for it has been reported that an improvement in the patient's subjective perception of functional disability is the most powerful predictor of treatment outcome (Pfingsten *et al.*, 1997).

Chronic pain has always been classified and diagnosed when it has lasted for more than six months (Schaible & Richter, 2004). However, an attempt has been made to define chronic pain by its characteristics rather than its duration (Schaible & Richter, 2004). Meyer (2007: 20) provides an accepted definition of chronic pain as state by the IASP: "...*pain that persists for longer than the time expected for healing, or pain associated with progressive, non-malignant disease, usually taken to be three months"*.

Others have defined chronic pain syndromes as any set of behaviours that involve the complaint of enduring or recurring pain which has persisted for longer than typical for an associated condition, is associated with an intermittent or chronic disease process, has inadequately responded to appropriate medical or invasive care, and is associated with significant and reliable impairment of functional status (Sanders *et al.*, 2005). Others have defined chronic pain by other sets of criteria. Sanders *et al.* (2005) reported chronic pain syndromes as any set of behaviours that involve the complaint of enduring or recurring pain which has persisted for longer than typical for an associated condition, is associated with an intermittent or chronic

disease process, has inadequately responded to appropriate medical or invasive care, and is associated with significant and reliable impairment of functional status.

This has given support to the hypothesis that chronic low back pain should not be regarded as an isolated spinal disorder (Kikuchi, 2008). It has been reported that two-thirds of subjects with chronic low back pain have at least another chronic pain conditions. About one-third has a diagnosable mental disorder (Von Korff *et al.*, 2005).

Patients who suffer from chronic pain syndromes may also demonstrate significant mood disturbances and/or anger-hostility, but these are not considered as necessary to make a diagnosis (Sanders *et al.*, 2005). Petersen *et al.* (2002) reported that there is a need for a classification system highlighted by Borkan & Cherkin (1996) to increase researchers' ability to identify differences in treatments for low back pain patients.

Petersen *et al.* (2002) reported that in diverse samples of non-specific low back pain patients, subgroups of patients for whom a specific treatment has been of benefit may be masked by subgroups for whom no measurable benefits was achieved. It can therefore be argued that the aims of such a classification system would be to identify patient characteristics that could predict the effects of different types of treatment procedures, and to distinguish clinically relevant subgroups for testing the relevance of specific treatment effectiveness.

Chronic pain can often persist long after the tissue trauma that triggered its onset, has healed and may be present in the absence of recognized ongoing tissue damage (Holdcroft & Jagger, 2005). Chronic pain is a dysfunctional response which mostly does not warn the individual of underlying disease or injury that will trigger an aversion response, and has thus accordingly been widely acknowledged as a disease in its own right (Niv & Devor, 2007; Kikuchi, 2008).

Chronic pain may be associated with an underlying chronic disease such as arthritis, but the largest group of chronic pain patients in the current epidemic in developing countries consists of the chronic pain syndromes of unknown etiology (Shipton,

1999). These pain syndromes have no confirmed laboratory evidence and are diagnosed on the basis of clinical criteria, such as the headache syndromes, irritable bowel syndrome, fibromyalgia and non-specific low back pain (Meyer, 2007). According to Meyer (2007) chronic pain may be associated with an underlying chronic disease such as arthritis. However, the largest group of chronic pain patients in the current epidemic in developed countries, comprises the chronic pain syndromes of unknown etiology (Shipton, 1999). These pain syndromes have no confirmed laboratory evidence and are diagnosed on the basis of clinical criteria, e.g. the headache syndromes, irritable bowel syndrome, fibromyalgia and non-specific low back pain.

Schaible & Richter (2004: 239) stated that "…*chronic pain might also result from a chronic disease and might then actually result from persistent nociceptive processes. It may be accompanied by neuroendocrine dysregulation, fatigue, dysphoria, and impaired physical and even mental performance (Chapman & Gavrin, 1999).* It has also been suggested that physical and sexual abuse in childhood can highly influence the incidence of chronic pain, especially in adulthood (McMahon *et al.*, 1997).

It is important to note that conservative treatment should be the first choice recommended treatment when treating patients with chronic low back pain (Zachrisson-Forssell, 1981; Hall & Iceton, 1983; Mayer *et al.*, 1985).

2.1.3.1 Psychological/Psychosocial Consequence of Chronic Low Back Pain

There is increasing acceptance that psychosocial factors play a crucial role in the transition from an acute episode of low back pain to a chronic back disorder, as well as containing etiologic factors (Bigos *et al.*, 1994; Kendall, 1999). Schaible & Richter (2004: 238) stated that: "*In many chronic pain states the causal relationship between nociception and pain is not tight and the pain does not reflect tissue damage. Rather, psychological and social factors seem to determine the pain, e.g. in many cases of low back pain (Kendall, 1999).* It has been reported that back pain can be triggered by psychological problems such as distress (depression), poor health or excessive fear of illness (Carragee *et al.*, 2000). However, the tendency to consider chronic

pain as either psychological or physical is a false implication, because both play a role in most chronic pain disorders, although the balance between organic pathology and psychosocial contributions may differ in different pain disorders (Meyer, 2007). According to Meyer (2007) however, the tendency to consider chronic pain as either psychological or physical implies a false dichotomy - both play a role in most chronic pain disorders, although the balance between organic pathology and psychosocial contributions may differ in different pain disorders.

The emotional component of pain is complex and is influenced by past experiences, patient beliefs and fears (Turk, 2002). Catastrophising, described as an exaggerated orientation towards pain stimuli and pain experience (Sullivan *et al.*, 1995), is considered to be a maladaptive coping mechanism. It is interesting that it has been described as an explanatory concept for variations in pain and depression in chronic pain patients (Keefe *et al.*, 1989).

According to Meyer (2007) negative beliefs and an attitude of hopelessness may generate maladaptive illness behaviour with increased pain reporting (Baumann, 1994). Improvement in the understanding of the psychosocial aspect of suffering in chronic pain has led to an improvement in the effectiveness of rehabilitation programmes (Kääpä *et al.*, 2006).

Fear avoidance behaviour also plays an important part in the development of chronic low back pain. Fear avoidance beliefs are suggested to contribute to the development of chronic low back pain earlier than previously believed (Klenerman *et al.*, 1995; Linton *et al.*, 2000; Fritz *et al.*, 2001; Picavet *et al.*, 2002; Sieben *et al.*, 2002). It is reported that patients who perceive pain in a threatening, catastrophic manner (as in signs of tissue damage) are much more likely to encounter pain related fear and anxiety. This will consequently lead to escape or avoidance behaviours to situations that they perceive to be potentially harmful (Thomas & France, 2007). Confrontation and avoidance have been suggested as the two extreme responses to the fear of pain (Lethem *et al.*, 1983). Consequences of avoidance include escape and avoidance behaviour, resulting in poor performance and muscle reactivity, physical disuse and guarded movements (Vlaeyen & Linton, 2000).

Distress has been identified as a predictor of chronic pain and disability. This effect is independent of clinical factors, such as pain and function at baseline level measurements (Dionne *et al.*, 1995; Cherkin *et al.*, 1996; Dionne *et al.*, 1997; Epping *et al.*, 1998; Thomas *et al.*, 1999; Linton *et al.*, 2000; Pincus *et al.*, 2002). Distress deals with aspects such as anxiety, somatisation and depression in general, whereas fear avoidance beliefs focuses more on specific back pain related anxiety (Grotle *et al.*, 2006). Distress contributes about the same amount as fear avoidance beliefs to the changes in variance in disability scores (Grotle *et al.*, 2004).

It has also been reported that distress is a significant prognostic factor of non-recovery at three months post-onset, whereas fear avoidance beliefs are not (Grotle *et al.*, 2005). Thus, it has been reported that leaving out the distress variable may result in an over-optimistic conclusion regarding the prognostic role of fear avoidance beliefs (Grotle *et al.*, 2006).

Thomas and France (2007) has reported that over the long-term, the avoidance of activities of everyday life that are perceived by the person to increase pain or a risk for re-injury is repeatedly reinforced, can cause anxiety symptoms towards these activities. It can then be argued that pain-related fear and anxiety eventually contribute to symptoms of disuse and disability.

Grotle *et al.* (2006) reported that fear avoidance beliefs for physical activity follow the same clinical pattern as pain and pain-related disability. Consistent with this model, studies have shown that individuals suffering from chronic low back pain who also experience high levels of pain-related fear demonstrates sub-maximal performance on a variety of physical tasks (Waddell *et al.*, 1993; Klenerman *et al.*, 1995; Vlaeyen *et al.*, 1995; Crombez *et al.*, 1999; Geisser *et al.*, 2000; Burns *et al.*, 2000; Sieben *et al.*, 2002; Fritz & George, 2002; Swinkels-Meewisse *et al.*, 2003; Al-Obaidi *et al.*, 2000; Al-Obaidi *et al.*, 2003).

Moore *et al.* (2000) have reported that approximately two months after consultation with a primary care physician for back pain, approximately 64% of patients still feel that incorrect movement could lead to a serious problem, and over 46% still feel they

could suffer disability for a long time. This type of thinking can contribute to the avoidance of activities or to additional health care visits, making the levels of anxiety of a patient an important target of clinical care. These patients however feel that they have a reason for concern, as it has been shown that guarded movements and hyperactivity in the lumbar paraspinal muscles are directly correlated with pain-related fear (Maffey-Ward *et al.*, 1996; Main & Watson, 1999), as they feel discomfort with movement.

Thomas and France (2007) reported that prior studies have usually defined physical impairment on the basis of reduced exertion. This classification may show that individuals with pain-related fear alter the manner in which they move in an effort to avoid pain or further damage (Thomas & France, 2007). As an example, research of motor coordination have shown that individuals with low back pain have reduced peak velocity and acceleration of the upper body (Marras *et al.*, 1995b; Ferguson *et al.*, 2000; Tawfik, 2001), and limitations and asymmetries in range of motion (Bishop *et al.*, 1997; Magnusson *et al.*, 1998).

These kinds of changes in motor behaviour may possibly reflect adaptations that reduce spinal load, and to avoid large forces on healing muscles and irritated joints. More likely they are a learned response due to fear avoidance behaviour with no actual reduction in spinal loading (Thomas & France, 2007).

Whichever the case, such adaptations may contribute to increased risk for subsequent injury. Thomas and France (2007) reported that this may explain the association between pain-related fear and disability. The authors also suggested that patients with high pain-related fear tend to adopt alternative movement patterns and strategies to avoid motion of the lumbar spine when performing everyday reaching movement tasks. Also, patients performing movement tasks have no preference to move at slow or fast speeds, but it is a specific position that they aim to avoid (Thomas & France, 2007).

Simmonds and Dreisinger (2003) reported that an approach to exercise will be influenced by the individuals beliefs about their back pain. Beliefs differ from person to person, as some people will ignore their own pain and discomfort and carry on

with normal every day activities while others will stop all forms of daily activity and seek medical attention. If certain activities aggravate pain, certain types of people will avoid activities or even attempt to avoid those activities that they expect will cause pain. These beliefs will influence the probability of these people to partake in exercise treatment modalities, and it can be severely compromised if their believes are negative.

Psychological issues of motivation and fatigue must be taken into consideration in relation to isometric extension endurance testing before a statement can be made regarding their diagnostic value (Moreau *et al.*, 2001). It has been suggested that a disadvantage of endurance testing is its dependence on subject motivation which can be influenced by a subjects perception of being able to maintain a given sub-maximal target force as measured to their own perceived limit of endurance (Biering-Sorensen, 1984; Mannion & Dolan, 1994; Ng & Richardson, 1996; Van Dieen & Heijblom, 1996).

Many factors influence a person's perception of exertion. Moreau *et al.* (2001) reported that some subjects who possess personalities motivated by achievement and competition tend to be better at endurance based tests, traits of which include competitiveness (Hellandsig, 1998), self-motivation (Raglin *et al.*, 1990), leadership qualities (Clingham & Hilliard, 1987), ambition, organisation, deference, dominance, endurance, self-control, tough-mindedness (Ogilvie, 1968), lower rates of perceived exertion (Beaudoin *et al.*, 1998), less negative feelings during endurance tasks (Beaudoin *et al.*, 1998), the ability to activate an emotion appropriate for the task (Forbes, 1986), and control of fatigue and pain (Forbes, 1986). These traits might mean a critical difference when measuring poor endurance performers from good performers, even when all other physiological variables are similar (Moreau *et al.*, 2001).

Moreau *et al.* (2001) further reported that the effect of personality traits on endurance variables may extend beyond athletes to non-athletes. This could imply that certain personality types are susceptible to working beyond their endurance and recuperative capacities (Moreau *et al.*, 2001). These people include business executives, doctors, lawyers, accountants, clergymen and housewives (Rhoads,

1977). Personality traits that are often seen in those who perform well on endurance tasks may suggest that white-collar workers perform better because they are more psychologically suited to the tasks and they respond as a result (Moreau *et al.*, 2001).

To add to the psychological problem, most patients presenting with first-time low back pain will be diagnosed according to a scientific standpoint as having non-specific low back pain (simple back pain) (Staal *et al.*, 2005). Many of these patients, however, may desire a more somatic name for their pain, such as a 'slipped disc' in order to justify their complaints and to show to others that their pain is indeed real and that they can prove it (Staal *et al.*, 2005).

The assumed disadvantages of receiving a somatic diagnostic label or name is that besides the possible lack of validity, they may enhance pain-related fears such as fear avoidance beliefs and encourage a dependant 'sick-role' (Staal *et al.*, 2005). The message that needs to be communicated to the patients is that their pain is real but that it does not necessarily imply harm. This should not prevent patients from being active (Frank *et al.*, 1998).

2.1.3.2 Physical Changes and Deconditioning

Leboeuf-Yde (2004) reported that a sedentary lifestyle is probably one of the most causative factors for low back pain, as lack of physical activity can lead to reduced muscle strength and flexibility, as well as having an undesirable effect on proprioception. All of these factors can contribute to a maladapted and weakened spine. Such persons are therefore more prone to injuries (Leboeuf-Yde, 2004).

Empirical research has demonstrated that physiological changes such as muscle dysfunction occur in the lumbar spine in conjunction with an initial episode of pain. These changes remain after the pain episode has subsided (Hides *et al.*, 1996; Hodges & Richardson, 1996). People with low back pain often have declining muscle strength as well as endurance, along with greater atrophy of the back muscles. This may compromise the functional capacity of the spine and increase the likelihood of re-injury (Jackson & Brown, 1983; Reid *et al.*, 1991; Parkkola *et al.*, 1993b). This includes stiffness of the lumbar spine-pelvic-femoral unit, decreased muscle strength

and endurance, a loss of adequate cardiorespiratory response to increased physical exertion as well as a inhibition of neuromuscular activation (Mayer *et al.*, 1985).

Thomas and France (2007) proposed that changes that occurs due to low back pain includes the restriction of low back motion, which leads to changes in extensibility of noncontractile connective tissues, muscle recruitment patterns, or alteration in potential feedback signals provided from the muscle spindles and mechanoreceptors in the paraspinal muscles (Thomas & France, 2007). Structural changes in any of these variables could increase the risk of exacerbation of symptoms by interfering with the ability of the individual to control spinal motion (Thomas & France, 2007).

Limitation of the range of motion at a joint may possibly result in reduction in length of peri-articular connective tissues as well as changes in the surrounding muscles (Lieber, 2002). Kendall *et al.* (1993) reported that at least in theory, this may lead to a condition called 'adaptive shortening', which results in tightening that occurs because of the muscle remaining in a shortened position. Unless the opposing antagonistic muscle is able to pull the part back to the neutral position or some type of outside force is exerted to increase the length of the shortened muscle (e.g. stretching), it will remain in a shortened position (Kendall *et al.*, 1993), and can lead to a decrease in muscle length and a corresponding limitation in range of motion (Kendall *et al.*, 1993).

Thomas and France suggested that individuals who demonstrate high levels of pain-related fear who continue to restrict motion may be at greater risk for pain and re-injury when faced with physical challenges that necessitate them to move the spine into and beyond its restricted range of motion. The authors has thus proposed that there are increased demands on shortened connected tissues and muscles that are no longer able to adequately maintain the integrity of the motion segment through normal range of motion, especially in movement tasks that require high velocities (Thomas & France, 2007).

In the past, wasting and denervation of the multifidus muscle in acute and sub-acute low back pain populations has been suggested (Hides *et al.*, 1994; Hides *et al.*, 1996). For example, the multifidus muscle denervation and atrophy have been

reported in patients with lumbar disc herniation injuries (Mattila *et al.*, 1986; Rantanen *et al.*, 1993). This may have an effect on muscle receptors, which in turn will influence trunk proprioception and position sense (Leinonen *et al.*, 2003).

Previous studies have found that a denervation of the multifidus muscle is frequently present in patients suffering from sciatica as well (Mattila *et al.*, 1986; Rantanen *et al.*, 1993). This denervation often occurs in the lower limb muscles innervated by the specific nerve root. The subsequent reinnervation process may take longer than three months (Leinonen *et al.*, 2003).

There is a large body of evidence suggests that poor back extensor muscle endurance and co-ordination cause excessive mechanical loading on the passive structures of the lumbar spine and can exacerbate existing low back pain symptoms (Wilder *et al.*, 1996; McGill, 1997; Taimela *et al.*, 1999). It has been shown previously that lumbar fatigue restricts the ability to sense a change in lumbar rotation location (Kankaanpää *et al.*, 2005). Dynamic testing has also shown pathologically low muscle activities and asymmetries in dynamic movements in back pain patients (Hoyt *et al.*, 1981; Cram & Steger, 1983).

Interestingly, patients with low back pain have shown unsuccessful the ability to increase their paraspinal activity level during the Valsalva manoeuvre or a sit-up test (Soderberg & Barr, 1983). This feature, however, was found in subjects and healthy controls, but subjects with low back pain demonstrates poorer ability to sense a change in lumbar position than healthy controls even when they are not fatigued (Kankaanpää *et al.*, 2005).

Sung *et al.* (2008) reports that subjects with low back pain demonstrate a greater level of fatigue of the erector spinae muscles at the thoracic part than at the lumbar part regardless of gender. The thoracic portion of the erector spinae muscle may play a significant role in spinal endurance for subjects with low back pain compared to subjects without low back pain (Sung *et al.*, 2008). Thus, the thoracic part of the erector spinae muscle shows higher fatigue levels than the lumbar portion. This is in part due to the lumbar region extending over a longer period during tests such as the Sorensen back extensor endurance test (Sung *et al.*, 2008). There seems to be a

phase after a fatiguing task during which the existing information on lumbar position and its changes is faulty (Taimela *et al.*, 1999; Leinonen *et al.*, 2003).

Adequate blood supply is obviously the most essential for the lumbar muscles to withstand fatigue and prevent the loss of sense organ functions that are driving the co-coordinative feedback mechanisms The most essential factor for the lumbar muscles to withstand fatigue and prevent the loss of sense organ functions that are driving the co-coordinative feedback mechanisms is adequate blood supply (Kankaanpää *et al.*, 2005). Suitable rehabilitation strategies based on lumbar erector spinae muscle fatigability may advance the retraining of trunk musculature and prevent re-injury in patients who have a history of low back pain (Sung *et al.*, 2008).

Chronic low back pain has been linked with histomorphological and structural changes in the paraspinal muscles, to an extent that the back muscles are smaller, contain higher levels of fat, demonstrates a degree of selective muscle fibre atrophy (Verbunt *et al.*, 2003) and their blood circulation may be constrained because of calcific deposits in the abdominal aorta and vertebral arteries (Kauppila *et al.*, 1997; Kauppila *et al.*, 2004). Consequently, the lumbar paraspinal muscles are weaker (Hakkinen *et al.*, 2003) and show signs of excessive fatigability, as compared to those without low back pain (Mannion *et al.*, 1997; Greennough *et al.*, 1998; Humphrey *et al.*, 2005).

Also, reduced co-ordination of paraspinal muscles has been associated with chronic low back pain and with excessive lumbar muscle fatigability (Wilder *et al.*, 1996; Taimela *et al.*, 1999; Leinonen *et al.*, 2003). These changes are generally consideration to be a consequence of disuse and deconditioning that are secondary to pain and illness, a process that has been named the ‘deconditioning syndrome’ (Nachemson & Lindh, 1969; Thorstensson & Arvidson, 1982).

Recent studies suggest that pain induced muscle spasms and reflex inhibition of the paraspinal muscles may also lead to the physical deconditioning (Hides *et al.*, 1996; Indahl *et al.*, 1997; Verbunt *et al.*, 2003). Radebold *et al.* (2000) has suggested that patients with low back pain have increased reaction times in muscle response patterns to sudden trunk loading when compared to healthy controls (Radebold *et*

al., 2000). Patients seem to be able to maintain agonistic muscle contraction while their antagonistic muscles become concurrently activated. There seems to be no definite change from agonistic to antagonistic status during movement (Radebold *et al.*, 2000).

Delayed reaction times to unexpected and sudden loading (as in slipping or tripping) for subjects with low back pain could be interpreted either as a predisposing factor to injury or as a consequence of soft tissue damage that necessitates an altered motor control approach to stabilise the lumbar spine (Magnusson *et al.*, 1996; Wilder *et al.*, 1996). Previous soft tissue injuries may have irreversibly damaged proprioceptors and therefore an adequate fast reflex response to a sudden load may not be possible (Radebold *et al.*, 2000). Proprioceptors that have been irreversible damaged by past soft tissue injuries has an inadequate reflex response time to sudden loads, and thus protective response is not possible.

Accordingly, a response time delay must be compensated for by an altered recruitment pattern. Co-activation of the antagonistic and agonistic muscle groups has been reported to stiffen and consequently stabilise the lumbar spine in order to protect the spine form injury (Bergmark, 1989a; Gardner-Morse *et al.*, 1995; Cholewicki & McGill, 1996; Gardner-Morse & Stokes, 1998; Quint *et al.*, 1998). The continued contraction of the agonistic muscles, which are similar in nature to muscle spasms, in subjects with low back pain serve to increase the joint stability and consequently providean adequate method of compensation to protect them from pain and harm progression due to lumbar spine instability (Radebold *et al.*, 2000).

Oldervoll *et al.* (2001) reported that poor muscle strength in the thigh, dorsal and abdominal muscles can cause back pain indirectly, in that weakness-related fatigue in the thigh muscle at the end of a working day can cause a person to lift an object with straight knees and a bent back rather than bending at the knee and hip joint (Oldervoll *et al.*, 2001). This style of lifting can cause amplified load on the passive structures in the low back (Nutter, 1988).

Kendall *et al.* (1993) reported that painful conditions such as the so-called 'periformis syndrome' can indicate how nerve irritation associated with muscle stiffness can cause pain, because the applied pressure can be through pressure on nerve roots, nerve trunks, nerve branches or nerve endings and can be caused by some adjacent firm structure such as bone, cartilage, fascia, scar tissue or stiffened muscles applying pressure to the nerves. All of this can lead to pain.

Apkarian *et al.* (2004) has reported that it seems that a reduction in grey matter has been observed in the dorsal-lateral prefrontal cortices (DLPFC) of the brain. This phenomenon has been observed bilaterally, as well is in the right thalamus. Neural degeneration has been identified as a possible causative factor rather than outright tissue shrinkage.

Schmidt-Wilcke *et al.* (2006) reported an increase in gray matter in the thalamus with an additional level of decrease in the dorsal-lateral pons and the somatosensory cortex. Pain intensity and the unpleasant experience of pain has been suggested to be involved with the reduction in grey matter in the brainstem, rather the timeframe of the pain experience.

Chronic pain has been identified as a multi-factorial condition that can lead to chronic disability the longer the pain persists. Psychological, social and occupation factors all become involved the longer that pain persists, and they can all contribute to a longer term problem (Waddel *et al.,* 1996).

2.1.3.3 The Concept of Central Sensitisation

This concept is very important in the management of chronic pain syndromes, for it is a key mechanism involved in the persistence of pain.

2.1.3.3.1 The Role of Acute Pain in Central Sensitisation

Meyer (2007: 22) stated that: "*Acute pain is a normal biological response to injury or tissue trauma and a signal of ongoing or impending tissue damage, e.g. post-operatively. It protects the organism from further injury and promotes healing after injury*". The cause of acute pain has to be treated appropriately due to the fact that it is a symptom of something else (Shipton, 1999; Woolf, 2004). Meyer (2007)

reported that unnecessary suffering and increased morbidity will result if acute pain is left untreated. It can also cause an increase in recovery time. Meyer (2007: 22) further stated that: "*There is increasing recognition that long-term changes may occur within the peripheral and central nervous system following the noxious input of painful stimuli*". Meyer (2007:22) wrote: "*There is increasing recognition that long-term changes may occur within the peripheral and central nervous system following the noxious input of painful stimuli. Even brief interval of untreated acute pain can induce long-term neuronal remodelling and central sensitisation ("plasticity"), and may lead to chronic pain in some patients*". This sensitization of the nervous system then changes the response of the body to further sensory input which then causes it to become more sensitive to pain impulses and even harmless stimuli can trigger pain perception (Melzack & Wall, 1965; Shipton, 1999; Carr & Goudas, 1999; Woolf, 2004).

Verdu *et al.* (2008: 2613) stated that: "*In inflammatory pain, the peripheral terminals of nociceptors are subjected to major changes in their chemical environment leading to peripheral sensitization. The numerous inflammatory mediators (prostaglandins, cytokines, bradykinin, amines, and neurotrophic factors, etc.) can directly sensitize the terminal in a way that it becomes more receptive."*

2.1.3.3.2 The Physiology of Central Sensitisation

Meyer (2007:22) reported that "*Central sensitisation is a complex process involving many neurochemical and molecular processes*". It seems to be triggered by intense activation of nociceptors as well as by humoral factors released by inflamed peripheral tissue (Woolf, 1983). Schaible & Richter (2004: 240) stated that: "*Pathological nociceptive input often causes central sensitisation. This is an increase of excitability of spinal cord neurons (Woolf, 1983)."*

Verdu *et al.* (2008:2612) stated that: "*In chronic pain syndromes, the activation of multiple pathophysiological mechanisms leads to a shift towards hyperexcitability of the somatosensory system*." Schaible & Richter (2004: 240) further stated that: "*Hyperexcitable spinal cord neurons are more susceptible to peripheral inputs and respond, therefore, more strongly to stimulation. Central sensitisation amplifies the processing of nociceptive input and is thus an important mechanism that is involved*

in clinically relevant pain states. It consists of the following phenomena: a) increase of the response to input from the injured or inflamed region; b) increase of the responses to input from regions adjacent to and even remote from the injured/inflamed region, although these areas are not injured/inflamed; c) expansion of the receptive fields of the spinal cord, i.e. the total area from which the neuron is activated, is enlarged. Presumably the latter accounts for secondary hyperalgesia, i.e. hyperalgesia in normal tissue surrounding the injured/inflamed area (Schaible et al., 1987).”

According to Meyer (2007) central sensitization is a complex process involving many neurochemicals and molecular processes, and is induced by the release of neuropeptides such as substance P and glutamate, which then activates the NMDD-receptor complex (Bennett, 1999; Bonica, 1953; Carr & Goudas, 1999). The subsequent intra-cellular events may lead to long-term neuronal changes, characterized by a more sensitive nervous system and hyperalgesia.

Schaible and Richter (2004:240-241) reported that: “*Pressure on this area causes a response of the neuron. Stimulation of the surrounding adjacent area does not cause a response, although some afferent fibres from this fringe area project to the same neuron. Under normal conditions, synaptic activation by these afferents is too weak to evoke a suprathreshold response. During injury, nociceptors in the receptive field are sensitised, and their increased activity causes activation and sensitisation of the spinal cord neuron. When the spinal cord neuron is rendered hyperexcitable, the weak inputs from the adjacent regions outside the original receptive field are sufficient to excite the spinal cord neuron, and, hence, the receptive field shows an expansion. Another consequence of peripheral inflammation and spinal sensitisation is that, in the spinal segments with input from the lesioned/injured regions, a higher proportion of neurons respond to stimulation of peripheral tissue(Woolf, 1983; Schaible et al., 1987; Neugebauer & Schaible, 1990; Grubb et al., 1993*).”

Normally, when the nociceptive input is removed, central sensitization decreases as well, but it has been reported that central sensitization may last longer than the peripheral nociceptive stimulation (Sandkühler & Liu, 1998). Hours and days after injury the pain signal can remain sustained by transcriptional changes in the dorsal

horn. These changes, restricted to the activated synapse or spread to adjacent synapses, are responsible for pain produced by low-threshold afferent inputs and pain in regions beyond the tissue injury (Verdu *et al.*, 2008). It has been suggested that as long as a painful nociceptive signal remains that central sensitization will remain until the signal is removed (Sandkühler & Liu, 1998). According to Verdu *et al.* (2008) however, the pain signal can still remain by transcriptional changes in the dorsal horn, and these changes, restricted to the activated synapse or spread to adjacent synapses, are responsible for pain produced by low-threshold afferent inputs (allodynia) and pain in regions beyond the tissue injury (secondary hyperalgesia), and even when the nociceptive signal has been removed.

It is theorised that nociceptive inputs may trigger a so-called long-term potentiation and a persistent increase of synaptic efficacy will occur. Such a process could account for pain states that persist even when the peripheral nociceptive process has been removed (Sandkühler & Liu, 1998). Repetitive end-range loading of pain-sensitised spinal tissue may act towards the maintenance of a chronic pain disorder in the absence of pathoanatomic abnormalities (O'Sullivan *et al.*, 2003). After nerve injury, the amount of input produced by peripheral ectopic activity, in addition to changes in gene expression, will contribute to central sensitisation (Verdu *et al.*, 2008). It has been proposed that a process of persistent increased synaptic efficacy can occur which can trigger a long-term potentiation. This type of response could account for painful situations that occur when the peripheral nociceptive signal has been removed (Sandkühler & Liu, 1998). O'Sullivan *et al.* (2003:1077) stated that "...*repetitive end-range loading of pain-sensitised spinal tissue may act towards the maintenance of a chronic pain disorder in the absence of pathoanatomic abnormalities*". According to Verdu *et al.* (2008) after nerve injury, the amount of input produced by peripheral ectopic activity, in addition to changes in gene expression, will contribute to central sensitization.

2.2 The Problem of Low Back Pain

Low back pain is a very common disorder and is one of the most common types of musculoskeletal pain that affects a larger number of people from many walks of life (Frymoyer & Cats-Baril, 1991; Van Tulder *et al.*, 2000).

Punnett *et al.* (2005:2) stated that "...*low back pain has been defined as any non-traumatic musculoskeletal disorder affecting the low back. It includes all back pain, regardless of the diagnosis, that was not secondary to another disease or injury cause (e.g. cancer or motor vehicle accident). It included lumbar disc problems (displacement, rupture) and sciatica".*

However, it has been reported that the definition of low back pain may vary substantially across studies and prevalence estimates may therefore vary substantially (Loney & Stratford, 1999). Such differences in definitions are not likely to affect the estimation of relative risk as long as they are applied in a regular manner within each study (Punnett *et al*., 2005).

Low back pain can result from various sources, which can include either one serious event such as major trauma or even multiple episodes of microtrauma. It can include a muscular and joint component and can involve single or multiple sites and could continue from anything up to a couple of weeks, months or even span a lifetime (Simmonds & Dreisinger, 2003). A specific diagnosis is thus very difficult, resulting in signs and symptoms being the main considerations that will determine a course of treatment, because even the most objective data is vulnerable to subjective interpretation of its' significance (Kendall *et al.,* 1993).

There exists a lack of understanding with regard to the multifactorial nature and specific cause of low back problems, and this is shown in the large variety of obtainable treatments, ranging from medically oriented invasive treatments such as injection therapy and surgery to more psychological approaches such as behaviour based approaches (Nelemans *et al*., 2001; Van Tulder *et al*., 2001).

Radebold *et al.* (2000:947) reported that: "*Many of the factors associated with low back pain are mechanical. These factors either cause low back problems initially or aggravate them by increasing the risk of recurrence and are thus important for disability considerations".* Burton (2005) has suggested that low back pain tends to present itself as a disorderly collection of periods with increased discomfort due to symptom elevation which is interchanged with periods of less symptom activity

(Croft*et al.*, 1998; De Vet *et al.*, 2002; Hestbaek *et al.*, 2003a). For a group of people, these symptoms can become chronic, as will the disability associated with it.

2.2.1 Lifetime Occurrence of Low Back Pain

Kankaanpää *et al.* (2005) reported that because of the lack of understanding with regards to the underlying mechanisms of low back pain, even modern scanning techniques such as X-rays or MRI scanning procedures along with clinical examinations cannot make a specific diagnosis due to a lack of sufficient information. Burton (2005) reported that in 85% of people that pathology and/or neurological encroachment cannot be attributed directly to pain. The tendency then is to make either a diagnosis that is descriptive of symptoms rather than pathology or a diagnosis of nonspecific low back pain (Nachemson, 1992; Deyo, 1994).

The prevalence for low back pain in industrial developed countries over a lifetime, which will account for at least one episode over a lifetime is reported to be at around 84-85% (Walker, 2000; Simmonds & Dreisinger, 2003). Hildebrandt *et al.* (2004) reported that after an initial episode of low back pain 44-78% of patients will suffer from future relapses in pain and 26-37% will suffer relapses in occupational absence. Sweden reports a prevalence of 69% (Ihlebaek *et al.*, 2006). In South Africa prevalence has been reported to be at 63.9% (Van Vuuren *et al.*, 2005).

Hildebrandt *et al.* (2004) reported that around 11-12% of patients that suffer from low back pain are actually disabled by their pain, and similarly to other, also report that specific causes are unusual, with less that 15% of all cases of low back pain that can be directly related to a specific diagnosis. But, more important from the aspect of the current study, about 5-10% of those who do suffer an initial episode of low back pain will go on to become chronic and thus long-term (Linton *et al.*, 2005).

Goldby *et al.* (2006) reported that over a one year time period, patients who suffer a first-time episode of low back pain that 20% will be without symptoms and 70-80% of patients have at least one reoccurring episode of low back pain (Klenerman *et al.*, 1995; Croft *et al.*, 1997). Many of these patients will continue to have recurring episodes of low back pain that has been reported up to 20 years (Carey *et al.*, 2000). About 3-4% of these patients will develop a specific chronic pain syndrome, but the

largest percentage is those with chronic low back pain, and they compose an estimated 73-77% of all the reported low back pain problems.

Punnett *et al.* (2005) reported that around 37% of all low back pain episodes worldwide is attributable to occupational risk factors. Epidemiologic evidence suggests that patients with chronic low back pain disorders have recurrences from which they rapidly recover over a period of time (Goldby *et al.*, 2006).

2.2.2 Impact of Low Back Pain

Reported estimates have shown that disability resulting from low back pain can contribute to a significant economic burden due to direct and indirect costs that can exceed $25-50 billion per annum in the United States (Frymoyer & Cats-Baril, 1991; Frymoyer, 1992) with associated productivity losses accounting for about $28 billion per annum (Rizzo *et al.*, 1998). Even personal costs are high, as it is estimated that people who suffer from low back pain incur health care costs that are around 60% higher than those without low back pain (Luo *et al.*, 2004).

In the United Kingdom, primary health care consultation for low back pain is the second highest leading cause of health care consultation (Deyo & Phillips, 1996). It also appears that the economic, societal and public health effects of low back pain appear to be increasing (Louw *et al.*, 2007). Punnett *et al.* (2005) reported that low back pain is not responsible for premature mortality but it can lead to severe consequences, such as substantial levels of disability. This is of particular concern if these consequences are suffered at a young age.

Low back pain incurs billions of dollars in medical expenditure each year (Childs *et al.*, 2004). This economic burden is of particular concern for poorer nations such as those in Africa where HIV/AIDS takes preference in terms of funding towards health care management and funds are thus restricted to deal with musculoskeletal problems (Walker, 2000). It has been suggested that most of the research on low back pain has been conducted in the developed world that doesn't have the same social and economic conditions such as those in Africa and other developing nations (Worku, 2000). Louw *et al.* (2007) suggested that Africa is considered to be a developing continent, and it is characterized by factors such as racial, economic and

social heterogeneity. It can thus be argued that reported differences in the prevalence of low back pain between developed and developing nations can be obscured by differences in social and economic structure as well as genetic diversity.

Lopez *et al.* (2006) reported that that prevalence among Africans are also influenced by other factors that are unique to Africa, such as the high HIV/AIDS incidence, types of working tasks unique to Africans as well as poor nutrition. Even in South Africa the cost and impact are high. Sick days taken because of back pain costs companies around R1.2 billion a year, and sick days taken is second only to flu with around 6.4% of all sick days taken (SAPA, 2009).

Significant levels of low back pain results in considerable levels of associated disability, which places restrictions on usual activities of daily living. This includes the ability of the person to continue to work (Katz, 2006). Western societies are influenced greatly by worker absenteeism and disablement, which results in the largest amount of related economic costs (Andersson, 1999). Burton (2005) reported that the greatest number of back pain episodes usually return to work in due time (Phelps *et al.* 2004), but recurrent and chronic low back pain are considered to be responsible for a large portion of the total number of work absenteeism. Work absenteeism accounts for a large proportion of the socio-economic burden of low back pain.

Staal *et al.* (2005:492) stated that ”*Return to work (in the case of work absenteeism due to low back pain) should not only be considered as an important treatment goal and outcome measure in research, but also an important part of the treatment. While working, the disabled worker realises that he/she is still able to be active, despite discomfort. Being at work, in a partial or full capacity, also draws the attention of the disabled worker away from negative issues such as pain, and helps to decrease the focus on disablement*”.

Burton (2005) reported that from half the days lost from work due to back pain that it is for a short period of time and they return to work in less than 7 days. This accounts for 85% of people who are absent from work due to low back pain. The other 15% accounts for the other half of days missed and the workers are off from

work for longer than a month. This has important economic implications, in that patients who are absent from work for more than six months has a 50% chance of returning to work. This number becomes lower the longer the person is away from work. Being off from work for more than one year has a 25% chance of returning to work and being off for longer than two years has less than a 5% chance of returning to work (Bergquist-Ullman & Larsson, 1977).

The amount of available academic literature on the epidemiology of low back pain is increasing, but it reported that most of the studies are done in developed, high-income countries, and much less is thus known about the rest of the world (Volinn, 1997). Prevalence in countries such as Australia and the United States for low back pain ranges anywhere from 26.4%-79.2% (Deyo *et al.*, 2006). It is assumed that the prevalence in a developing continent such as Africa is much lower, but this could be due to a lack of reporting (Omokhodion & Sanya, 2003; Omokhodion, 2004; Gilgil *et al.*, 2005).

Walker (2000) reported that a systematic review into the low back pain prevalence globally identified 56 studies, of which only 8% were conducted in developing countries, and only one study was done specifically in Africa. It is clear that there is a lack of research done in developing nations, and this presents a significant shortcoming (Walker, 2000; Gilgil *et al.*, 2005). This lack of hard data presents a problem, as it is predicted that the greatest increase in low back pain incidence in the next decade will occur in developing nations (Louw *et al.*, 2007). Regardless, Louw *et al.* (2007) reported that even due to the lack of evidence, there appears to be not much difference between Africa and developed nations with regards to incidence of prevalence.

2.3 Low Back Anatomy

Understanding the anatomy and function of the lower back is crucial to understanding dysfunctional conditions.

2.3.1 The Bone Structure

The lumbar spine contains a total of five vertebrae (McGill, 2002). The construction of a vertebra consists of relatively stiff cortical bone on the outside walls and more

deformable cartilage plates that are approximately 0.66 mm thick on the sides of the vertebrae (Roberts *et al.*, 1989). These sides of the vertebrae are known as endplates, and they are porous in terms of their construction in order for nutrients such as oxygen and glucose to be transported. The inside of the barrel of the vertebral body is filled with cancellous bone (Roberts *et al.*, 1989).

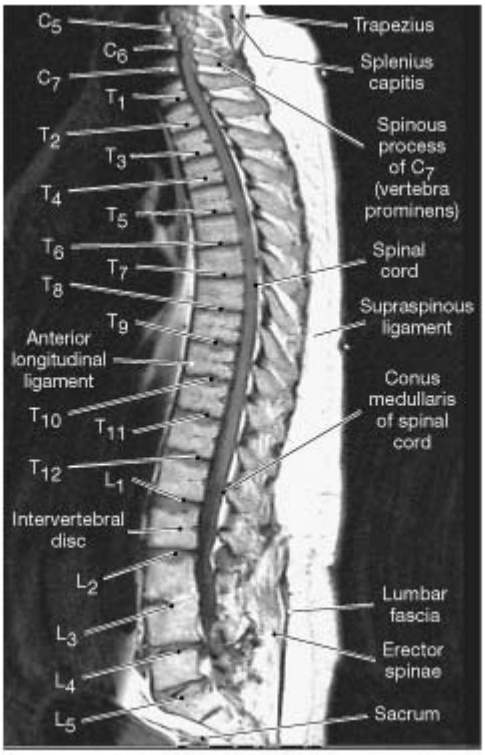

Figure 2.2: A Radiological View of the Spine (Martini, 1998)

Within the cancellous bone of the vertebral body, the trabecular arrangement is aligned with stress trajectories that develop during activity, and three orientations domination, with one being vertical and two are oblique (Gallois & Japoit, 1925).

McGill (2002) reported that the ability of the vertebral body to cope with compressive loads or either to fail under extreme loads is determined by its construction. The vertebral loads will remain fairly ridged under compressive loads, but the pressure will affect the nucleus of the disc (Nachemson, 1960). This will cause the cartilaginous endplates of the vertebra to bulge inward, and to consequently compress the cancellous bone (Brinckmann *et al.*, 1989). This cancellous bone tends to fail first (Gunning *et al.*, 2001). This is then considered to be a failure

determination point of the spine, at least when the spine is not located at the end range of motion. It is unlikely that the annulus itself will be injured by this mechanism.

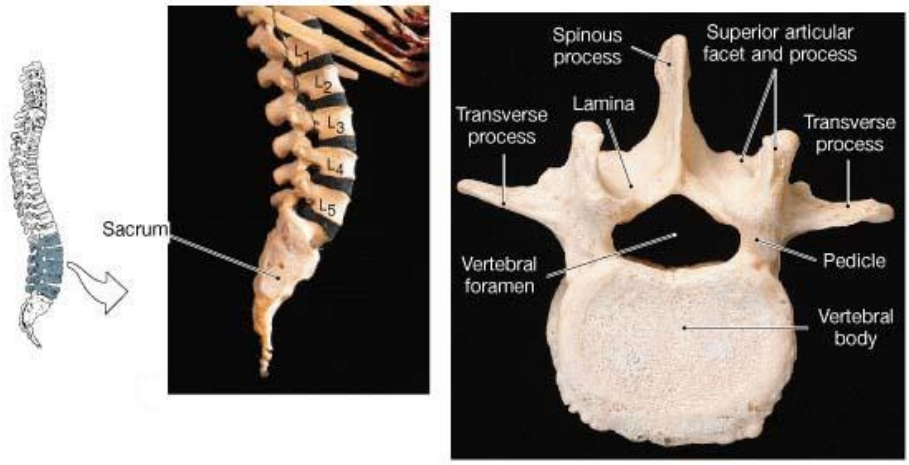

Figure 2.3: The Lumbar Vertebrae (Martini, 1998)

Upon axial compression, as the endplates bulge into the vertebral bodies, these columns experience compression and appear to bend. Under excessive compressive load the bending columns will buckle as the smaller bony transverse trabeculae fracture (Fyhrie & Schaffler, 1994). In this way the cancellous bone may rebound back to its original shape or to at least 95% of the original unloaded shape when the load is removed, even after suffering fracture and delamination of the transverse trabeculae (Fyhrie & Schaffler, 1994). This architecture appears to afford excellent elastic deformation, even after marked damage, and then to regain its original structure and function as it heals (McGill, 2002). McGill (2002: 47) stated that: "...*upon axial compression, as the endplates bulge into the vertebral bodies, these columns experience compression and appear to bend".* Under greater than normal loads, the smaller bony transverse trabeculae will fracture under greater than normal spinal loads, allowing the cancellous bone the opportunity to return to at least 95% of its' original pre-loaded shape, even if fractures and delamination occurs (Fyhrie & Schaffler, 1994). This organization tends to offer good elastic deformation

properties, in that the original structure and function can return as healing starts to occur after injury (McGill, 2002).

McGill (2002: 51) reported that: "*The posterior elements of the vertebrae (pedicles, laminae, spinous processes and facet joints) have a shell of cortical bone but contain a cancellous bony core in the thick sections. The transverse processes project laterally together with a superior and an inferior pair of facet joints. On the lateral surface of the bone that forms the superior facets are the accessory and mamillary processes that, together with the transverse processes, are major attachment sites of the longissimus and iliocostalis extensor muscle groups. The facet joints are typical synovial joints, in that the articulating surfaces are covered with hyaline cartilage, and are contained within a capsule".* Around the rim of the facet, fibro-adipose enlargements are found at the proximal and distal edges, and these structures have been implicated as structures that could lock a facet joint (Bogduk & Engel, 1984).

McGill (2002:48) also reported that: "*Micro-fracturing of the trabeculae can occur with repetitive loading at levels well below the failure level from a single cycle of load."* Lu *et al*. (2001) demonstrated that cyclic loading at 10% of ultimate failure load caused no damage or change in stiffness, but with 20 000 cycles of load at 20-30% of the ultimate failure load, both stiffness and energy absorbed at failure were decreased. Highly repetitive loads, even at quite low magnitudes, appear to cause micro-damage. According to Lu *et al.* (2001) cyclic loading at 10% of failure load seems to cause no damage or change in stiffness. It was found however, that at 20 000 cycles of load at 20-30% of ultimate failure load, there was a marked decrease in both stiffness and energy absorbed at failure. For this it would appear that even at low levels, highly repetitive loads appear to cause micro damage.

2.3.2 The Intervertebral Disc

The intervertebral discs are cartilaginous, articulating structures between the vertebral bodies and link them together, allowing movement (flexion, extension and rotation) in the otherwise rigid anterior portion of the vertebral column (Roberts *et al.*, 2006; Prithvi Raj, 2008). The intervertebral discs are the links between vertebrae that allow for movement such as flexion, extension and rotation. The vertebral column

would have been a rigid structure if not for the intervertebral discs. They are cartilaginous in terms of their structure (Roberts *et al.*, 2006; Prithvi Raj, 2008).

The discs are approximately 7-10 mm thick and 4 cm in diameter in the anterior-posterior plane in the lumbar region of the spine (Twomey & Taylor, 1987; Roberts *et al.*, 1989). McGill (2002:53) reported that: "*The intervertebral disc has three major components: the nucleus pulposus, annulus fibrosus and the endplates".* The discs consist of a thick an outer ring called the annulus fibrosus which consists of a thick outer ring of fibrous cartilage. This surrounds an inner core known as the nucleus pulposus, which is more gelatinous (Roberts *et al.*, 2006; Prithvi Raj, 2008). The discs consist of a network of collagen fibres that are composed of mostly Type 1 and Type 2 fibres, and is responsible for about 70% of the dry weight of the annulus and about 20% of the nucleus weights. This arrangement provides tensile strength to the discs and connects the tissue to the bone (Eyre & Muir, 1977).

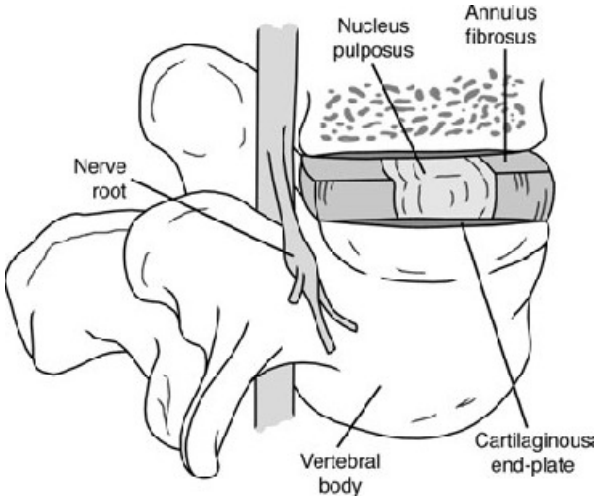

Figure 2.4 : The Intervertebral Disc Between Two Adjacent Vertebrae (Prithvi Raj, 2008)

2.3.2.1 The Nucleus Pulposus

McGill (2002:53) reported that "...*the nucleus has a gel-like character with collagen fibrils suspended in a base of water and various mucopolysaccharides giving it both viscosity and some elastic responses."* The nucleus pulposus is located in the central part of the disc and contains randomly organized collagen fibres (Inoue, 1981) as well as radially organized elastin fibres, all of which is organized in highly hydrated aggrecan-containing gel (Yu *et al.*, 2002).

McGill (2002:53) reported that: "*While there is no distinct border between the nucleus and the annulus, the lamellae of the annulus become more distinct, moving radially outward. The collagen fibres of each lamina are obliquely orientated (the obliquity runs in the opposite direction in each concentric lamella). The ends of the collagen fibres anchor into the vertebral body with Sharpey's fibres in the outermost lamellae while the inner fibres attach to the end plate".* To add to this, Roberts *et al.* (2006:10) reported that: "*Collagen fibres continue from the annulus into the adjacent tissues, tying this fibrocartilaginous structure to the vertebral bodies at its rim, to the longitudinal ligaments anteriorly and posteriorly, and to the hyaline cartilage end plates superiorly and inferiorly. The cartilage end plates in turn lock into the osseous vertebral endplates via the calcified cartilage with few, if any, collagen fibres crossing the boundary".*

2.3.2.2 Annulus Fibrosis

The construction of the annulus fibrosis consists of around 15-25 concentric rings (or Lamellae) (Marchand & Ahmed, 1990) with the collagen fibres being organized in a parallel fashion with each lamella (Prithvi Raj, 2008). The fibre orientation is around 60 degrees to the vertical axis which alternates left and right of the axis in each adjacent lamellae (Prithvi Raj, 2008).

Elastin fibres are located between the lamellae. These fibres are suggested to assist the disc in returning to its' original position after either flexion and/or extension. Because the elastin fibres run radially from one lamella to the next, they also bind the lamella together (Yu *et al.*, 2002). The cells of the outer region of the annulus are aligned parallel to the collagen fibres, are elongated and thin and tend to be fibroblast-like, but tend to be more oval towards to inner portion of the annulus (Prithvi Raj, 2008).

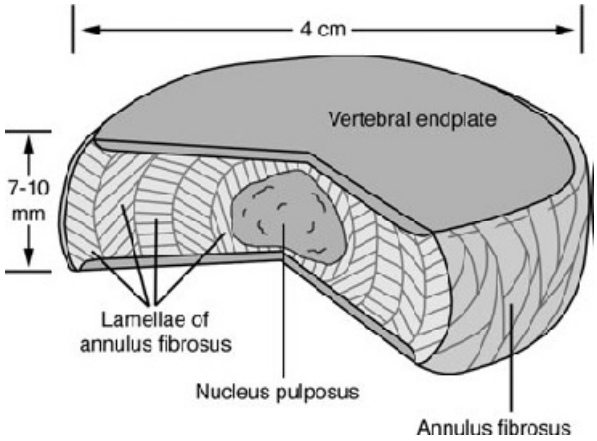

Figure 2.5: The Structure of an Intervertebral Disc (Prithvi Raj, 2008)

2.3.2.3 The Endplate

This morphologically distinct region consists of a thin layer, usually 1 mm thick, consisting of hyaline cartilage which interfaces the disc and the intervertebral body (Prithvi Raj, 2008). The collagen fibres within it run horizontal and parallel to the vertebral bodies, with the fibres continuing into the disc (Roberts *et al.*, 1989).

2.3.2.4 Properties and Function of the Intervertebral Discs

McGill (2002) reported that the intervertebral disc acts as a whole to allow for about six degrees of motion between the vertebra by exhibiting hydostatic structure properties. The annulus and the nucleus work together when the spine is subjected to bending and compression to supporting compressive forces by pressurizing the nucleus and thus applying a hydraulic force to the end plates vertically and laterally to the annulus. This will cause an outward bulging by the annulus collagen fibres and causes them to tense.

Roberts *et al.* (2006) reported that normal intervertebral discs contain an extracellular matrix that is responsible for about 1% of the total volume of the disc. This matrix is interspersed by a small number of cells, which are believed to consist of at least two phenotypical distinct populations (Chelberg *et al.*, 1995).

Roberts *et al.* (2006:11) reported that: "...*the cells are morphologically different. Those in the annulus fibrosis and cartilage endplates are more elongated and fibroblast-like compared to those of the nucleus pulposus, which are more rounded*

or oval and chondrocyte-like, sometimes with a capsule around them." The cells seem to be simple in appearance, but might be much more complex in function, with long thin cell processes which could be able in sensing mechanical strain (Errington *et al*., 1998).

Roberts *et al.* (2006: 11) also reports that: "...*in addition, these two populations behave differently, such as in their response to applied loads or in synthesizing different matrix molecules when grown in culture."* Nucleus pulposus cells are commonly produced only type-2 collagen in alginated beads, while annulus fibrosus cells produce both type -1 and type-2 (Chelberg *et al*., 1995).

The normal, healthy adult disc does contain a limited number of nerves and blood vessels, but these are limited only to the outer few millimetres of the annulus fibrosus. There are also a number of mechanoreceptors present, which demonstrates a similar morphology of Golgi tendon organs, some Ruffini receptors and an ever smaller amount of pacinian corpuscles (Roberts *et al.,* 1995).

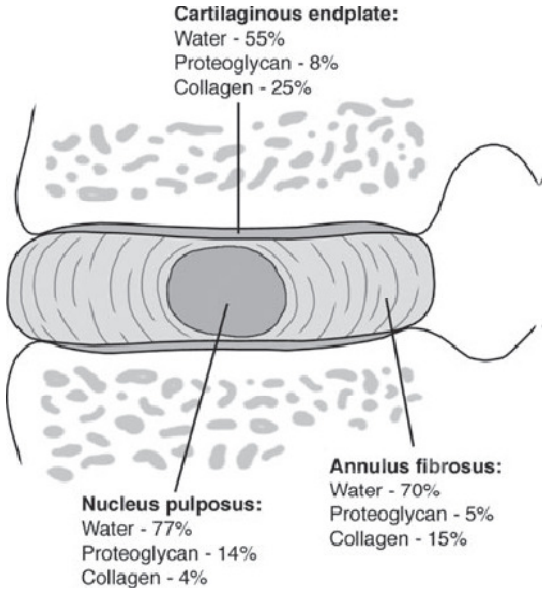

Figure 2.6: The Composition of the Intervertebral Disc Structures
(Prithvi Raj, 2008)

The main function attributed to the intervertebral disc is a mechanical function, in that the loads from body weight and muscle activity are transmitted through the vertebral column (Prithvi Raj, 2008).

2.3.3 Movements of the Vertebral Column

Kendall *et al.* (1993:23) reported that: "*Vertebral articulations include the bilateral synovial joints of the vertebral arches where the inferior facets of one vertebra articulate with the superior facets of the adjacent vertebra, and the fibrous joints between successive vertebral bodies united by intervertebral fibrocartilaginous discs. Movement between two adjacent vertebrae is slight and determined by the slope of the articular facets and the flexibility of the intervertebral discs. The range of motion of the column as a whole, however, is considerable and the movements permitted are flexion, extension, lateral flexion and rotation.*"

Motion of the vertebral column is provided by the intervertebtal discs, which includes flexion (forward and backwards) as well as rotation (Prithvi Raj, 2008). The ability of the vertebral column to move differs in the thoracic and the lumbar region. In the lumbar region, the range of motion for greater flexion, extension and lateral bending ability about the three axis of motion, while the thoracic region demonstrates greater ability to rotate (McGill, 2002).

2.3.4 The Model of Spinal Stability and Instability

Stability can be defined as "...*the ability to maintain intervertebral and global torso equilibrium despite the presence of small mechanical disturbances and/or small neuromuscular control errors*" (Granata & England, 2006:E271).

Instability of the lumbar spine in those with chronic low back pain has been identified as a significant causative factor for pain (Friberg, 1987). This type of lumbar instability can be thought of as a specific region of laxity around the neutral resting position of the spinal neutral zone (Panjabi, 1992). This also includes an inability of the stabilizing mechanism as a whole to maintain the neutral zone of the vertebral column within physiological limits, which means that there is no deformity of any structures, any neurological deficit or dehabilitating pain (Panjabi, 1992).

The stability of the spine can be compromised by a decrease in motion segmental stiffness by as little as 10% (Gardener-Morse *et al.*, 1995). This stability is provided by osteoligamentous structures and muscles since the spinal structure has to cope with large amounts of pressure due to complex loading patterns in all three dimensions simultaneously, and if unprotected, the vertebral column is susceptible to injury (Arokoski *et al.*, 2004). The ability of the vertebral column to generate stability and be decreased by injury and chronic mechanical derangement (Oxland & Panjabi, 1992).

Trunk muscles must compensate during normal motion by changing their typical activation pattern to maintain stability (Panjabi, 1992). To achieve this, the muscles of the lumbar and abdominal regions must have to precisely control motion by generating enough muscle tension and correct timing to optimizing loading on the spine and to avoid overload injury (Bergmark, 1989a; Crisco & Panjabi, 1991; Callaghan & McGill, 1995; Gardner-Morse *et al.*, 1995; Kaigle *et al.*, 1995; Cholewicki & McGill, 1996; McGill, 1997; Gardner-Morse & Stokes, 1998).

Hasegawa *et al.* (2008) reported that the concept of lumbar stability is difficult to define objectively. Radiological evaluation remains unclear and controversial, since radiological evaluations are performed regularly on degenerative lumber spines and clear diagnoses are unclear and undefined (Knutsson, 1944; Morgan & King, 1957; Lindahl, 1966; Nachemson *et al.*, 1979; Dupuis *et al.*, 1985; Frymoyer & Selby, 1985; Dvorak *et al.*, 1991; Iguchi *et al.*, 2003).

Biplanar, cineradiographic and fluoroscopic measurements provide some additional information on disordered motion patterns (Stokes *et al.*, 1981; Pearcy *et al.*, 1985; Kanayama *et al.*, 1996; Okawa *et al.*, 1998; Harada *et al.*, 2000; Takayanagi *et al.*, 2001). These dynamic approaches cannot be used to draw a biomechanical conclusion about instability, because no information about the load deformation relationship can be determined from the images (Hasegawa *et al.*, 2008). Hasegawa *et al.* (2008) reported that measurement techniques such as biplanar, cineradiographic or fluoroscopic measurements provide some additional information on chaotic motion. Unfortunately, no biomechanical conclusion or diagnosis can be

made about instability because no information about the load deformation mechanisms can be concluded from these dynamic approaches.

Correct levels of muscle stiffness cannot alone verify biomechanical segmental properties of the spine, as other variables, including measurement of the neutral zone are needed to make accurate assessments (Panjabi, 1992). It has been suggested that up to about 20-30% of all reported chronic low back pain problems demonstrates verified lumbar segmental instability (O'Sullivan, 2000; Beith *et al.*, 2001). Spinal stability or instability can be an indicator of the ability of a person to activate and perform a co-contraction of the deep local muscle system (O'Sullivan, 2000; Beith *et al.*, 2001).

Granata and England, (2006: E271) states that research indicates that three subsystems contribute to spinal stability as initially proposed by Panjabi (1992):

- The passive contributions from the spinal ligaments, discs, and bone.
- The steady-state active muscle recruitment contribution to spinal stability.
- Neural feedback system that includes active and voluntary responses.

2.3.5 The Passive Stabilising Structures of the Spine: Bone, Ligaments and Fascia

When the spine is in a position of neutral lordosis, e.g. neither being in flexion or extension, only muscles are responsible for mechanical support for the spine, but as the spine is flexed and rotates, the passive structures of the spine are also stressed and the forces on these tissues changes the injury mechanics (McGill, 2002). Spinal ligaments are an important spinal stabilizer, and they have been suggested as possessing an important proprioceptive role in spinal mechanics (Solomonow *et al.*, 2000).

In vitro, under compressive loads of about 90N, the osteoligamentous lumbar spine becomes mechanical unstable, and this load is less than the weight of the human upper body (Crisco *et al.*, 1992). Too much load and stress on the joints and ligaments of the spine will result if the muscle is inadequate (Gracovetsky *et al.*, 1985; Panjabi, 1992; Saal, 1992).

McGill (2002: 49) stated that: "*Both the disc and the vertebrae deform while supporting spinal loads. Under excessive compressive loading, the bulging of the endplates into the vertebral bodies also causes radial stresses in the endplate sufficient to cause fracture in a stellate pattern".* When these fractures in the endplates are big enough, the liquid from the nucleus can move into the fracture spaces into the vertebral body (McGill, 1997). It can happen that a local area under the endplate can collapse to form the classic Scmorl's node injury, which has been described as an injury associated with compression of the spine when there is limited motion of the spine.

McGill (2002) reported that fasica of the lumbodorsal region has attachment sites on the posterior superior iliac spines as well as on the spinous processes, and some of these fascial connections cross the midline, which has been implicated in force transmission by completing a brace-like structure around the abdomen. This structure has been researched, and it has been found that it may function more as an extensor muscle retinaculum rather than being an active extensor due to the well-developed collagen fibre construction (Bogduk & Macintosh, 1984). Unstable behaviour and possible tissue injury is thus prevent by this mechanism due to muscles activation from the abdominal wall and the latissimus dorsi adding tension to the fasica and thus stiffness to the spine (McGill, 2002).

2.3.6 Dynamic Spine Stability: The Muscles Supporting theSpine

The concept of different trunk muscles playing different roles in the provision of dynamic stability to the spine was proposed by Bergmark (1989a). It has hypothesised the presence of two muscle systems in the maintenance of spinal stability.

The global muscle system consists of muscles that provide trunk stability without being directly attached to it. These include the muscles of the rectus abdominis, external oblique and the thoracic portion of the lumbar iliocostalis, which are large torque-producing muscles which are not capable of having direct influence on the spine, but provide excellent spinal stability (Bergmark, 1989a).

The other important group of muscles is the so-called local muscle system, which is responsible for providing stability to the segmental portions of the spine and directly controls the lumbar segments. This is due to the fact that this group of muscles attaches directly to the lumbar vertebrae and includes the multifidus, transversus abdominis and the posterior fibres of the internal oblique (Bergmark, 1989a).

The local muscle group (transversus abdominis, internal oblique and the multifidus) have been reported to be tonically active during upright postures and during active spinal motion (Oddsson & Thorstensson, 1990; Cresswell *et al.*, 1992). The transversus abdominis is considers one of the moist important trunk stabilizers, because it has been found that it seems to be tonically active regardless of the position of the trunk, loading of the spine or direction of movement (Cresswell *et al.*, 1992).

Leinonen *et al.* (2003) reported that in order to prevent low back injuries, it is important for correct muscle control and movement sensation to be of working order. The erector spinae group serves an important function, as they seem to have a greater mechanical advantage in some portions of the muscle than other due to multiple attachment sites, and this is important for maintaining an upright posture of the trunk (Bogduk, 1991; Bogduk *et al.*, 1992).

Because the erector spinae are attached to the lumbar vertebrae and directly extend the lumbar spine, they have been reported to be very important from an anatomical point of view (Sung *et al.*, 2008). This serves as an effective lever arm for performing back extension due to its attachment to the spinous processes of the lumbar vertebrae, and this portion of the erector spinae contributes about 20% of the lumbar spine extension moment at L4 and L5 (Bogduk *et al.*, 1992).

The erector spinae portion in the thoracic area consists of the thoracic components of the longissimus thoracis and iliocostalis lumborum and have been found to also generate moment arms across the L4-L5 joint due to fact that they also cross the lumbar spine (McGill & Norman, 1986; Bogduk *et al.*, 1992). The muscles have a stabilizing effect by surround the injured joint segment and thus reduce sudden

kinematic behaviour, especially in the neutral region of the spine where muscles are under reduced tension (Kaigle *et al.*, 1995).

Intra-abdominal and abdominal spring force is created during spinal stability which is achieved with trunk flexor extensor muscle activation (Panjabi *et al.*, 1989). In patients with low back pain, the alteration in function of these muscles causes a change in function as compared to healthy controls without back pain (Hides *et al.*, 1996; Hodges & Richardson, 1996). This dysfunction presents itself clinically as an imbalance between the local and global muscle systems (Bergmark, 1989b). This then leads to a theoretical reduction of the deep local muscle systems' ability to maintain effective stability and control lumbar stability (Goldby *et al.*, 2006).

The lumbar extensor muscles that stabilize the spine of those with back pain have been found to be impaired in terms of their function and co-ordination (Magnusson *et al.*, 1996; Wilder *et al.*, 1996; Hodges & Richardson, 1999; Leinonen *et al.*, 2001). Function-wise there is also a reduction, as it has been found that those with low back pain demonstrates an increase in reaction time during and after sudden load transmission as compared to healthy controls (Magnusson *et al.*, 1996; Wilder *et al.*, 1996). Those with low back pain demonstrate hostomorpic and structural changes to the type-2 muscle fibres. These muscle fibres have been reported to show atrophy due to disuse and deconditioing (Parkkola *et al.*, 1993a; Ng *et al.*, 1998; Mannion, 1999; Mannion *et al.*, 2000).

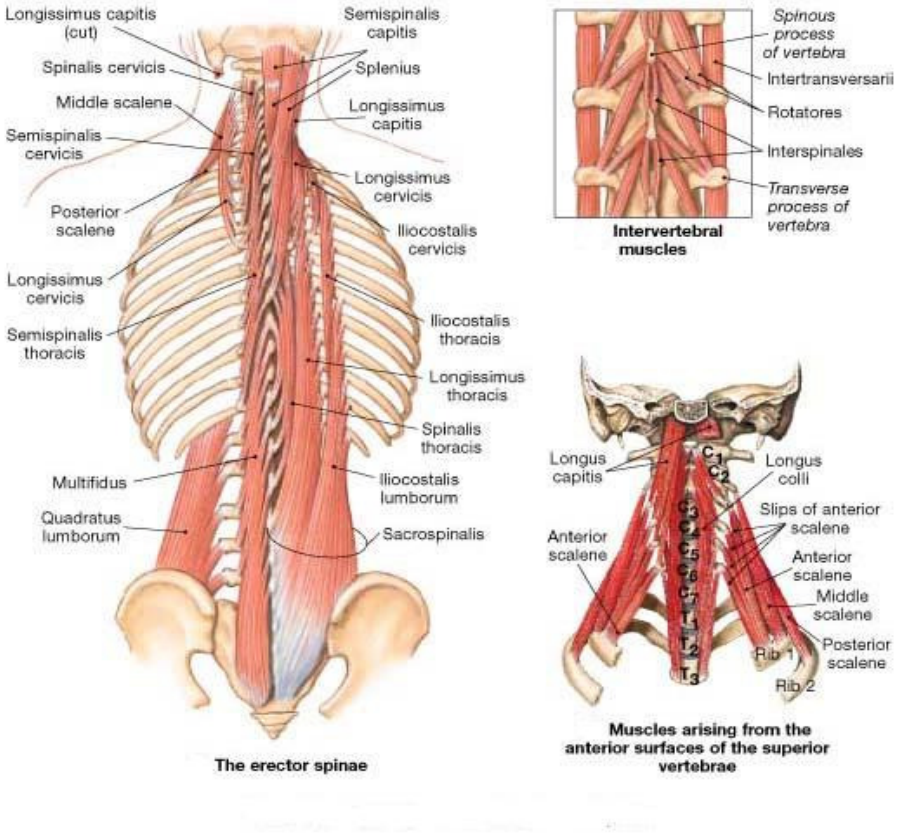

Figure 2.7: The Muscles of the Spine (Martini, 1998)

Leinonen *et al.* (2003) reports that the proprioceptive information of body movements originates from the muscle spindles, Golgi tendon organs, joint receptors and cutaneous receptors (Rothwell, 1994; Schmidt & Lee, 1998). Upper (Hodges & Richardson, 1996; Hodges *et al.,* 1999; Hodges *et al.*, 2001) and lower (Hodges & Richardson, 1998) limb movements cause trunk muscle activation via a feed-forward mechanism. When certain trunk muscles activate, they prepare to potentially bear load and to maintain postural stability.

The muscles responsible for this movement include the transversus abdominis muscle and the transversospinal muscles. It has been found that these muscles seem to activate shortly after perturbation. More significantly, it has been suggested by others that they seem to activate shortly before the muscles that are responsible for gross limb movement (Belen'kii *et al.*, 1967; Cordo & Nashner, 1982; Friedli *et al.*, 1988; Zattara & Bouisset, 1988; Aruin & Latash, 1995; Hodges & Richardson, 1996; Hodges *et al.*, 1999; Hodges *et al.*, 2001).

It has been proposed that possible faults, even only small faults in the proprioception and position sense ability can lead to tissue overload and injury. Insufficient activation can lead to abnormal loading across the surfaces of joints and can lead to early degenerative disease (Gross, 1987; Cholewicki & McGill, 1992; Forwell & Carnahan, 1996).

Movement of the limbs causes reactive forces that are imposed on the spine which are equal in the magnitude but opposite in the direction of those forces that are responsible for the movement (Belen'kii *et al.*, 1967; Cordo & Nashner, 1982; Bouisset & Zattara, 1987; Crisco & Panjabi, 1991). It has been proposed that the body anticipates limb movement and implements direction specific strategies to control reactive forces and to prepare the body for perturbation (Aruin & Latash, 1995; Moseley *et al.*, 2002). It has also been found that the transversus abdominis isn't influenced by the direction of any reactive moments, and is considered to be responsible in contributing to spine stiffness (Hodges & Richardson, 1997).

Due to the importance of the activity of the transversus abdominis, Richardson *et al.* (2002) has reported that the concept has become the basis of the specific exercise treatment techniques (Richardson *et al.*, 1999). The ability to co-contract the transversus abdominis and the lumbar multifidus independently of the other larger trunk muscles has become the goal of many exercise regiments. This exercise is based on evidence of the stability roles of the different implicated muscles (Goel *et al.*, 1993; Kaigle *et al.*, 1995) as well as on evidence that the transversus abdominis seems to functions independently of the other abdominal muscles (Hodges & Richardson, 1997). A very low level of muscular activation is actually performed during this action (Richardson *et al.*, 1999). Progression of treatment has consisted, in principle, of increasing the patient's ability at performing this independent deep muscle contractions action while minimising the contribution of the other trunk muscles.

Reasons have been proposed for the motivation to limit the activation of lumbar stability exclusively to the transversus abdominis in the form of lumbar paraspinal electromyography which has been used to estimate extensor force generation and

spinal muscle compression forces during lifting activities (Örtengren *et al.*, 1981; Dolan & Adams, 1993). It has been found that high levels of muscle activity could lead to unfavourable compressive forces acting on the spine, which in theory can cause possible injury (Callaghan *et al.*, 1998; McGill, 1998).

It has been reported that contraction of the muscles of the low back needs only to be about 25% of the maximal voluntary contraction (MVC) to provide optimal stiffness (Cresswell *et al.*, 1994). Others have reported that to improve muscle performance, contractions needs only to be around 30-40% of the MVC (Richardson *et al.*, 1999). Thus, the therapeutic exercises selected for a rehabilitation programme must be carefully selected as to not increase the risk of injury on the low back but be sufficient to increase the performance of the muscles.

2.3.6.1 Muscles Involved in Spinal Stability

Production of high force at fast speeds as well as the provision of stability and postural control is indicative of the functional capacity of the muscles of the low back, and these functions are dependent on anatomical, physiological, biomechanical and neural variables (Thorstensson & Carlson, 1987; Lieber, 1992; Enoka, 1994).

Muscle fibre composition, cross-sectional area, fibre length, pennation angle and muscle mass are some of the anatomical and physiological variables that are considered important. Important biomechanical variables are moment arm through which the muscle acts, velocity of the muscle contraction as well as the type. Compartmentalisation and recruitment strategies are considered neural parameters (Lieber, 1992; Enoka, 1994).

Ng *et al.* (1998) reported that anatomical, physiological, biomechanical and neural variables in combination results in the diverse functions among individual muscles. But another factor, namely muscle fibre composition is one of the most important elements that is indicative of the functional capacity of a specific muscle (Johnson *et al.*, 1973; Thorstensson & Carlson, 1987; Rome *et al.*, 1988).

The lumbar region consists of the iliocostalis lumborum, longissimus lumborum and the multifidus (from lateral to medial), which forms the paraspinal muscle group of

the lumbar region (Hyun *et al.*, 2007). To control posture and to stabilize joints has been identified as the primary function of these muscles (Bajek *et al.*, 2000).

Richardson *et al.* (2002: 399) reported that: "*The local muscle system includes deep muscles such as the transversus abdominis and the lumbar multifidus that are attached to the lumbar vertebrae and sacrum and are capable of directly controlling the lumbar segments".*

The next layer of muscles has been called the global muscle system. This group of muscles consists of muscles such as the external oblique and the erector spinae, which are bigger muscles and are located more towards to surface of the body and are more responsible for producing and controlling trunk movements (Bergmark, 1989a). The trunk basically consists of back extensor muscles that extend the trunk, lateral flexors that bend it sideways and the anterior abdominal group that flexes the trunk. All of these muscles have a trunk stabilizing function, but the extensor group has been recognized as being the most important (Kendall *et al.*, 1993).

Richardson *et al.* (2002) reported that conventional exercises are generally designed to increase the strength of the global muscle system. A more specific exercise approach is thus recommended to target the local muscle system in order to improve the dynamic stability function in order to provide muscle stiffness to support the spinal segments during functional postures and activities.

Different muscles exert types of force production on the trunk. In the upward direction, the erector spinae and the quadratus lumborum are primarily responsible for force generation. In the upward and anterior direction, the rectus abdominis and external oblique are responsible. The gluteus maximus and the hamstring muscle group generates force in a downward posterior direction, while the hip flexors (which includes the tensor fasciae latae, rectus femoris and sartorius) pulls downward and anteriorly (Kendall *et al.*, 1993).

Kendall *et al.* (1993:70) stated that: "*The low back muscles act with the hip flexors (especially the psoas with its direct pull from the lumbar spine to the femur) to tilt the pelvis down and forward (anterior tilt). They are opposed in action by the combined*

pull of the anterior abdominals pulling up anteriorly, and the hamstrings and gluteus maximus pulling down posteriorly to level the pelvis from a position of anterior tilt. Hip abductors on one side and lateral trunk muscles on the other side combine in action to tilt the pelvis laterally: right abductors (gluteus minimus and medius) pull downward on the right side of the pelvis as left lateral trunk muscles pull upward on the left side, and vice versa. These actions are assisted by hip adductors on the same side as the lateral trunk muscles."

It has been previously reported that the transversus abdominis, which is very important in contributing to spinal stability, shows strong abnormal motor control abilities in those with low back pain (Hodges & Richardson, 1996; Hodges & Richardson, 1998). Changes in multifidus composition has, which could affect the deep portion of the muscle, has also been observed (Rantanen *et al.*, 1993; Hides *et al.*, 1996; Moseley *et al.*, 2002).

The deep fibres of the multifidus and the transversus abdominis are controlled very similarly in healthy subjects without low back pain. Both of these muscles can contribute to the control of intervertebral motion, but the body needs all of the trunk muscles to stabilize the spine. Since both the deep multifidus and the transversus abdominis seem not to be specific to any direction of movement or movement from limbs, they are considered to be the most important spinal stabilizers (Richardson *et al.*, 1999; Moseley *et al.*, 2002).

Training of the transversus abdominis and multifidus muscles is believed to be an important component in the rehabilitation of patients with low back pain (Hides *et al.*, 1996; Hodges & Richardson, 1996; O'Sullivan *et al.*, 1997; O'Sullivan *et al.*, 1998; Hides *et al.*, 2001; Richardson *et al.*, 2002). The simultaneous contraction of the deep abdominal muscles and the lumbar multifidus can enhance the segmental stability of the spine during functional tasks and maintaining neutral spine postures by providing a type of dynamic corset for the lumbar spine, enhancing thus its stability irrespective of the position of the spine (Aspden, 1992).

The skeletal muscles of the body have been divided into two subcategories. Type 1 fibres have been described as being slow-twitch fibres. Type 2a and Type 2b are

described as being fast-twitch fibres. These distinctly different muscle fibres also show different reactions to mechanical loading and will be described in detail in the next section (Brooke & Kaiser, 1970; Billeter *et al.*, 1980; Herbison *et al.*, 1982; Yoshihara *et al.*, 2001).

Ng *et al.* (1998: 390) reported that: "*Type 1 fibres are slow twitch fibres that possess properties of high oxidative, low glycolytic capacity and are relatively resistant to fatigue. Type 2b fibres are most commonly known as fast twitch fibres with the characteristics of low oxidative, high glycolytic capacity and are prone to fatigue. Type 2a fibres possess features in between 1 and 2b fibres."* Generally, type 2 fibres are larger in cross-sectional area (Enoka, 1994) and produce greater force (Rothstein, 1982; Jones *et al.*, 1989) than type 1 fibres. It has also been reported by others that type 2 fibres are on average larger than type 1 fibres in the limb muscles (Polger *et al.*, 1973). However, it has been shown that type 1 fibres in the lumbar multifidus muscle are much larger that the type 2 fibres found in the same muscle (Bagnall *et al.*, 1984; Mattila *et al.*, 1986; Yoshihara *et al.*, 2001).

Ng *et al.* (1998) reported that Type 1 muscle fibres predominate in tonic muscles and tend to assume a postural stability, sustained contraction and endurance activities. In contrast, Type 2 muscle fibres have fast twitch properties and are involved in high speed, forceful movements of short duration (Johnson *et al.*, 1973; McCafferty & Horvath, 1977; Herbison *et al.*, 1982). They occupy a relatively larger area in muscles responsible for fast movements (Johnson *et al.*, 1973) and appear to dominate in non-postural muscles such as the triceps brachii (Johnson *et al.*, 1973).

Paravertebral muscles function to control posture and to stabilize joints, which are the responsibility of the type 1 slow twitch fibres as they predominate in these muscles. They are suited to this kind of task since they are fatigue resistant (Fidler *et al.*, 1975; Thorstensson & Carlson, 1987). These muscles have been shown to be active in almost every kind of normal activity performed daily, but they are rarely ever fully contracted, regardless of modern lifestyle requirements (Bajek *et al.*, 2000).

Ng *et al.* (1998) reported that in general, 54-73% type 1 fibres are found in longissimus, iliocostalis and multifidus muscles of healthy subjects. Those with low

back problems tend to show changes in percentages. Longissimus is between 54-68%, 47-68% in multifidus and 68-69% in erector spinae muscles respectively.

A greater percentage of type 1 muscle fibres in the back muscles could possibly be explained as a postural control function. This type of activity will require endurance capacity to maintain, especially in an upright position against gravity which has to be maintained (Ng *et al.*, 1998). Due to a functional difference between the lumbar and thoracic portions of the muscles in postural control, a higher percentage of type 1 fibres have been identified in the thoracic portion of the muscles (Širca & Kostevc, 1985).

Ng *et al.* (1998) wrote that due to the S-shape of the spinal column, the line of gravity is more anterior to the rotational axis of the intervertebral joints in the thoracic vertebrae than in the lumbar spinal area. This implies that much larger flexion moments are generated at the thoracic levels (Joseph & McColl, 1961).

An investigation of the relevant muscles involved is of clinical importance, because it has been suggested that there is a functional differentiation between individual back muscles in the production of torque and the provision of stability (Thorstensson & Carlson, 1987; Panjabi *et al.*, 1989).

2.3.6.1.1 Multifidus

The multifidus muscle has been identified as being primarily involved in providing the required stiffness for the lumbar spine, and is consequently regarded as the most important of the back extensor muscles (Wilke *et al.*, 1995). Observations made with electromyographic measurements have suggested that the multifidus extends the length of the spine (Floyd & Silver, 1951; Joseph & McColl, 1961; Morris *et al.*, 1962; Donisch & Basmajian, 1972).

According to Kendall *et al.* (1993):
Multifidi
"Origin: *In the sacral region, posterior surface of the sacrum, medial surface of posterior superior iliac spine and the posterior sacroiliac ligaments as well as through the transverse processes of the L5 through to the C4 vertebrae.*

Insertion: *The muscle spans two to four vertebrae and inserts into the spinous processes of an above vertebrae."*

Moseley *et al.* (2002) reported that the greatest amount of activity in the multifidus muscle has been shown by surface EMG measurements to be during rotation and extension of the vertebral column or during resistance of lumbar flexion (Floyd & Silver, 1951; Joseph & McColl, 1961: Arokoski *et al.*, 1999). Direction specific activity during standing trunk movements and limb movements in prone subjects have been demonstrated in intramuscular EMG studies (Morris *et al.*, 1962; Pauly, 1966). It has to be noted that it is however, impractical to use surface EMG measurements to measure the deep fibres of the multifidus (Moseley *et al.*, 2002).

The lumbar multifidus has been identified as a major contributor to stabilization and lumbar spine control (Moseley *et al.*, 2002). Various types of studies, such as biomechanical models and in vitro studies have identified a function of maintaining spinal stiffness and intervertebral motion control in all planes of motion, especially the sagital and frontal planes of motion (Panjabi *et al.*, 1989; Crisco & Panjabi, 1991; Wilke *et al.*, 1995; Moseley *et al.*, 2002).

The multifidus has been suggested to contribute to rotation of the spine as well as maintaining postural integrity (Morris *et al.*, 1962; Donisch & Basmajian, 1972; Dofferhof & Vink, 1985; Kalimo *et al.*, 1989). The maintaining of postural control and in the end segmental integrity seems to be dependent on the ability of the muscles to respond during changes in structural integrity, has been shown by studies in vivo from porcine models that spinal motion has to be controlled by the multifidus after induced instability by ligamentous disruption (Kaigle *et al.*, 1995).

Moseley *et al.* (2002) reported that the multifidus has five fascicles that arise from the spinous process and lamina of each lumbar vertebra and descends in a caudolateral direction (Macintosh *et al.,* 1986). The most superficial fibres of each fascicle cross up to five segments and attach caudally to the ilia and sacrum. The deep fibres attach from the inferior border of a lamina and from the inferior edge of the spinous process (Macintosh *et al.*, 1986; Bogduk, 1997). They cross a minimum of two segments to insert into a mamillary process and facet joint capsule (Macintosh

et al., 1986; Bogduk, 1997). These are the deepest muscle fibres in the lumbar spine because there are no rotation muscles in this deep region (Bogduk, 1997). Biomechanically, the superficial fibres are more distant from the centres of lumbar vertebra rotation and have an effective moment arm for extension of the lumbar spine and control of lumbar lordosis (Macintosh & Bogduk, 1986). In contrast, the deep fibres are near the centre of lumbar vertebra rotation, and have a limited ability to assist in extending the spine (Panjabi *et al.*, 1989). The moment arm of this muscle is small, but it may exert its effects throughout the range of spine motion without compromise from its length-tension relation (McGill, 1991). Many of the trunk muscles are suited structure-wise to control the spine orientation, and most have a limited ability to control intervertebral shear and torsion (Panjabi *et al.*, 1989; Bogduk, 1997). The deep fibres of the multifidus are ideally placed to control these motions through intervertebral compression forces (Moseley *et al.*, 2002). The proximity of the deep multifidus to the centre of rotation means that it produces compression with minimal movement torque and thus minimal pressure on the spine (Panjabi *et al.*, 1989; Kaigle *et al.,* 1995).

It has also been reported that the control of shear forces and intersegmental motion by the deep fibres of the multifidus can occur irrespective of spine motion or internal and external forces (Moseley *et al.*, 2002). This has led to argument that the multifidus increases spinal stability and stiffness through tonic activity (Cholewicki *et al.*, 1997). Various states of activity have been suggested by various authors. Continued activity has been suggested by Valencia & Munro (1985) and Wolf *et al.* (1989). Donisch & Basmajian (1972) suggested phasic activity, and Morris *et al.* (1962) and Pauly (1966) suggested silent activity patterns.

Moseley *et al.* (2002: E34) reported that: "*One explanation for the functional distinction between the deep and superficial fibres of the multifidus may lie in the control of intervertebral shear and torsion through compressive force between segments. Because of their force vectors, the more superficial trunk muscles exert greater torque. If these muscles were recruited to control torsion and shear by increasing compression, then the component of their output that produces torque would need to be controlled. The resultant co-activation would result in an excessive energy and compressive 'cost'. In contrast, the deep trunk muscles exert minimal*

torque, which means that they can produce segmental compression with less resultant energy 'cost'."

It is further suggested that the neuromuscular system can control individual segments of the spine, by a mechanism by which the segmental attachments of the deep multifidus provides flexibility (Panjabi *et al.*, 1989). The multifidus muscle is further able to support the spine by maintaining the alignment of the trunk against reactive forces that produces flexion by exhibiting anticipatory contractions during upper limb flexion, but not during extension. This provides evidence that the multifidus acts to control forces that attempts to create flexion rather than extension (Hodges & Richardson, 1996).

Another argument for multifidus as a postural control muscle can be related to the fibre type composition of both the deep and superficial multifidus, in that most of the fibres of the deep multifidus are of the slow-twitch fatigue resistant fibres (Sirca & Kostevc, 1985; Jorgensen *et al.*, 1993; Bajek *et al.*, 2000). The multifidus does contain a number of type 2a and 2b fibres that are smaller than the type 1 fibres. They are not resistant fatigue and are required for fast and power contractions. This greater number of fatigue resistant type 1 fibres has added to the suggestion of the multifidus as a muscle of postural control (Bajek *et al.*, 2000).

The multifidus has also been reported to poses a muscle spindle density that is quite low (Amonoo-Kuofi, 1983). It has been suggested that patients that have undergone surgery for disc herniation show definite changes in the multifidus muscle, which includes selective type 2 fibre atrophy, and internal structure abnormalities which are non-disease specific changes to type 1 fibre which appear 'moth-eaten'. This could be caused by such factors as muscle disease and disuse (Rantanen *et al.*, 1993).

This type of finding has added to the suggestion that type 2 fibres of the back muscles are exposed to insufficient workloads by people in a modern day setting, which is much more sedentary, and consequently the loads are insufficient to maintain the normal size and strength of the muscle fibres (Rantanen *et al.*, 1993). The fact that the type 2 fast twitch fibres have been found to be smaller and less in percentage has been suggested to be a result of modern lifestyles, in which powerful

contractions and heavy instant demands that would require fast twitch activation occurs less frequently to stimulation the retention of muscle size and strength (Bajek *et al.*, 2000). This has also lead to the suggestion that a higher intensity of exercise can be of benefit for those with low back pain (Mayer *et al.*, 1985).

Moseley *et al.* (2002) reported that the stability of the spine is challenged during arm movements and this causes different activations of the deep and superficial fibres of the multifidus. The superficial fibres seem to control spine orientation and the deep fibres control intersegmental motion, while the erector spinae group and the superficial and lateral fibres of the multifidus are activated earlier relative to movements such as deltoid flexion.

Moseley *et al.* (2002) reported that activity that is specific to direction is matched to the direction of reactive forces caused by limb movement and connected to the spinal orientation control (Cordo & Nashner, 1982; Aruin & Latash, 1995; Hodges & Richardson, 1997) and the displacement of the centre of mass (Aruin & Latash, 1995).

A non-direction specific pattern of activity is shown by the deep fibres of the multifidus and the transverses abdominis (Hodges & Richardson, 1997). The deep fibres of the multifidus can act in both directions with repeated arm movements, although force applications on the spine are in the opposite direction (Moseley *et al.*, 2002).

During repetitive movement about the 90 degree flexion, it seems that the deep multifidus becomes phasic. With control during reactive forces, it would appear that when the reactive forces are aligned more vertically, the contribution of the deep multifidus is reduced (Moseley *et al.*, 2002).

The multifidus is attributed to be more active during extension based movements and counteracting reactive forces in the sagital plane when compared to the iliocostalis lumborum, which has the responsibility to regulate forces more in the frontal plane (Ng *et al.*, 1997). This has been confirmed during back extension

testing in which the multifidus will be more active during EMG extension testing and consequently shows higher rates of fatigue (Ng *et al.*, 1996; Ng *et al.*, 1997).

2.3.6.1.2 Transversus Abdominis

According to Kendall *et al.* (1993: 151):

Transversus abdominis

*"**Origin**: Inner surfaces of cartilages of lower six ribs, interdigitating with the diaphragm; thoracolumbar fascia; anterior three fourths of internal lip of iliac crest; and lateral one third of inguinal ligament.*

***Insertion**: Linea alba by means of a broad aponeurosis, the pubic crest and pectin pubis.*

***Action:** Acts like a girdle to flatten the abdominal wall and compress the abdominal viscera; upper portion helps to decrease the infrasternal angle of the ribs as in expiration. This muscle has no action in lateral trunk flexion except that it acts to compress the viscera and stabilize the linea alba, thereby permitting better action by anterolateral trunk muscles."*

The contraction of the transversus abdominis causes an increase in intra-abdominal pressure and also causes tension generation in the thoracolumbar fascia. It has been reported in the past that this action causes a decrease in spinal loading by creating a trunk extensor moment (Grillner *et al.*, 1978; Gracovetsky *et al.*, 1985).

It has been speculated that contraction of the transversus abdominis could possibly enhance the stabilization potential of the spine, but there has been uncertainty of the direct mechanism involved (Tesh *et al.*, 1987; Cresswell *et al.*, 1992; McGill & Norman, 1993; Richardson *et al.*, 1999). There has been reported that the combination of transversus abdominis activation along with oblique activity will elevate intra-abdominal pressure and could as a consequence assist in spinal stabilization (Cresswell *et al.*, 1994).

Activation of the transversus abdominis can also act to stabilize the sacroiliac joint (Richardson *et al.*, 2002). Due to the corset-like structure of the transversus abdominis, the muscle can effectively flatten the abdominal wall and compress the viscera, which can lead to the anterolateral trunk muscles functioning more

effectively and to theoretically stabilize the spine (Farfan, 1973; McGill & Norman, 1993). This kind of functionality has made the transversus abdominins a focus of specific exercise training to manage back pain (Richardson *et al.*, 2002).

To be able to increase the stability of the spine to function during different postures and movements, the creation of a pressurised visceral cavity anterior to the spine can result in the production of a force against the apex of the lordotic curve in the lumbar spine, and assist in producing lumbar stability (Aspden, 1989). Also, translation and rotational motion of the spine can also be limited through lateral tension through the transverse processes.

Previous research has suggested that the activation of the transversus abdominis precedes external loads applied to the body and consequently it activates before the load is applied to the body. The transversus abdominis also activates before the other trunk muscles do (Cresswell *et al.*, 1994). Hodges and Richardson reported that the central nervous system initiates anticipatory contractions of the muscles of the trunk in expectance of possible movement and load application to the body.

Hodges and Richardson (1996) further reported that this type of anticipatory response due to possible external loading is more of a gross general muscular response. The body can only be sure of the magnitude and direction of the external load once it is applied to the body and then only apply the correct muscular response.

Displacement of the centre of mass occurs with limb movement and dynamic forces are transmitted to the body by the inertial reactions between segments (Friedli *et al.*, 1988). To be able to account for these changes the central nervous system needs to pre-programme the activation of the transversus abdominis and the other trunk muscles to act in anticipation of movement to precisely act to oppose the perturbing force acting on the body (Bouisset & Zattara, 1981). It has been reported that this type of pre-programming in activating postural muscles is activated as part of the motor command for movement of a limb or it is parallel with motor commands (Bouisset & Zattara, 1981; Horak *et al.*, 1984).

Rehabilitation has recently been focusing on the role of the transversus abdominis in the contribution to spinal stability. There are several muscles that have been identified to play a role in stabilization, but evidence has suggested the critical role of the transversus abdominis can thus have recommended more focus in a rehabilitation setting (Aspden, 1992; Cresswell *et al.*, 1994; Hodges & Richardson, 1996).

The transversus abdominis has been reported to be the primary muscle involved in creating and sustaining of intra-abdominal pressure, and has been established as the first of the stabilizing muscles to contract before movement of the body is initiated (Cresswell *et al.*, 1992; Cresswell & Thorstensson, 1994; Hodges & Richardson, 1996; Hodges & Richardson, 1997). Since the transversus abdominis contracts before the other stabilizing muscles, a delay in the activation of the transversus abdominis due to a deficit in motor control has been linked to those with low back pain (Hodges & Richardson, 1996).

Transversus abdominis has been found to contract prior to limb movement regardless of the direction of movement in those without low back pain (Hodges & Richardson, 1997), and it has been established that in those with low back pain, that the activation of transversus abdominis is delayed significantly (Hodges & Richardson, 1996). The effect of this decrease in motor control ability will be a lack of proper control of stability against forces acting on the spine and maintaining normal stability mechanics during normal movement (Hodges & Richardson, 1996; Richardson *et al.*, 2002).

2.3.6.1.3 Internal and External Obliques

According to Kendall *et al.* (1993: 148):

"External Oblique, Anterior Fibres

***Origin**: External surface of ribs five through eight interdigitating with serratus anterior.*

***Insertion**: Into a broad, flat aponeurosis, terminating in the linea alba, a tendinous raphe which extends from the xiphoid.*

***Action**: Acting bilaterally, the anterior fibres flex the vertebral column approximating the thorax and pelvis anteriorly, support and compress the abdominal viscera,*

depress the thorax and assist in respiration. Acting unilaterally with the anterior fibres of the internal oblique on the opposite side, the anterior fibres of the external oblique rotate the vertebral column, bringing the thorax forward (when the pelvis is fixed), or the pelvis backwards (when the thorax is fixed)."

According to Kendall *et al.* (1993: 148):
"*External oblique, lateral fibres*
***Origin**: External surface of ninth rib, interdigitating with the serratus anterior; and external surface of the 10th, 11th and 12th ribs, interdigitating with the latissimus dorsi.*
***Insertion**: As the inguinal ligament, into anterior superior spine and pubic tubercle, and into the external lip of anterior one half of the iliac crest.*
***Action**: Acting bilaterally, the lateral fibres of the external oblique flex the vertebral column, with major influence on the lumbar spine, tilting the pelvis posteriorly. Acting unilaterally with the lateral fibres of the internal oblique on the same side, these fibres of the external oblique laterally flex the vertebral column, approximating the thorax and iliac crest. These external oblique fibres also act with the internal oblique on the opposite side to rotate the vertebral column.* "

According to Kendall *et al.* (1993: 149):
"Internal oblique lower anterior fibres
***Origin**: Lateral two thirds of the inguinal ligament, and short attachment on iliac crest near anterior superior spine.*
***Insertion**: With transversus abdominis into crest of pubis, medial part of pectineal line, and into linea alba by means of an aponeurosis.*
***Action**: The lower anterior fibres compress and support the lower abdominal viscera in conjunction with the transversus abdominis."*

According to Kendall *et al.* (1993: 149):
"*Internal oblique upper anterior fibres*
***Origin**: Anterior one-third of intermediate line of the iliac crest.*
***Insertion**: Linea alba by means of aponeurosis.*
***Action:** Acting bilaterally, the upper anterior fibres flex the vertebral column, approximating the thorax and pelvis anteriorly, supporting and compress the abdominal viscera, depressing the thorax and assist in respiration. Acting*

unilaterally, in conjunction with the anterior fibres of the external oblique on the opposite side, the upper anterior fibres of the internal oblique rotates the vertebral column, bringing the thorax backward (when the pelvis is fixed), or the pelvis forward (when the thorax is fixed)."

According to Kendall *et al.* (1993: 149):
"*Internal oblique, lateral fibres*
***Origin**: Middle one third of the intermediate line of iliac crest and thoracolumbar fascia.*
***Insertion**: Inferior borders of 10th, 11th and 12th ribs and linea alba by means of aponeurosis.*
***Action**: Acting bilaterally, the lateral fibres flex the vertebral column, approximating the thorax and pelvis anteriorly, and depress the thorax. Acting unilaterally with the lateral fibres of the external oblique on the same side, these fibres of the internal oblique laterally flex the vertebral column, approximating the thorax and pelvis. These fibres also with the external oblique on the opposite side to rotate the vertebral column."*

Kendall *et al.* (1993) reported that significant weakness in the both the internal and external obliques can lead to functional as well as postural problems. Functional postural changes such as kypohosis, scoloiosis and swayback postures along with changes such as an inability to flex the spine laterally as well as an anterior pelvic tilt in the standing position results due to a weakness in these muscles. Respiratory inefficiency and an inability the support the abdominal viscera can also result.

The obliques have also been identified as being important in the overall maintenance of spinal integrity. They provide lateral support and also play a role in stabilization by activating when the spine is placed under axial compression (Juker *et al.*, 1998). They have also been attributed to play a role in lateral bending and torso twisting (McGill, 1991).

In very similar fashion to the transversus abdominis, the internal and external obliques also seem to be able to anticipate upper limb movement and initiate contractions in advance of the movement (Belen'kii *et al.*, 1967; Bouisset & Zattara,

1981; Zattara & Bouisset, 1988; Aruin & Latash, 1995). Previous EMG research has reported that higher recruitment and co-activation of the internal and external oblique occurs during asymmetric tasks (Lavender *et al.*, 1992; Granata & Marras, 1993) such as combined sagital and twisting motions. Activation seems to be less prominent when movement occurs in the midsagital plane only (Lavender *et al.*, 1992; Granata & Marras, 1993; Granada & England, 2006).

It has been reported that an optimal movement strategy may allow variability in redundant, task-irrelevant dimensions, as in the kinematic variability in the transverse plane during midsagital movement (Todorov & Jordan, 2002). Increased control is required during simultaneous movements such as combined movement in the sagittal and transverse planes, as this imposes higher levels of load on the body (Todorov & Jordan, 2002).

2.3.6.1.4 Quadratus Lumborum

According to Kendall *et al.* (1993: 143):

"*Quadratus lumborum*

***Origin**: Iliolumbar ligament. Occasionally from upper borders of transverse processes of lower three or four lumbar vertebrae.*

***Insertion:** Inferior border of last rib and transverse processes of upper four lumbar vertebrae.*

***Action:** Assist in extension, laterally flexes the lumbar vertebral column, and depresses the last rib. Bilaterally, acting together with the diaphragm and fixes the last two ribs during respiration."*

The quadratus lumborum can play a significant role in local lateral buttressing (McGill, 2002). It has been reported that the quadratus lumborum (QL) is a very effective lateral stabilizer due to its attachments to the lumbar vertebrae. During compressive loading on the spine, the first mode of buckling is lateral, and this will makes the QL very effective in maintaining lateral stability of the spine (Lucas & Bresler, 1961; McGill, 2002).

To further support the idea of the QL being a stabilizer, it has been reported that the muscle seems to contract isometrically during spinal motion and hardly changes length during spinal motion (McGill, 1991; McGill, 2002).

2.3.6.1.5 Erector Spinae (Extensor Group)

The main muscles of the erector spinae group include the longissimus, illiocostalis lumborum and multifidus groups (Kendall *et al.*, 1993; McGill, 2002). However, due to the high importance of the multifidus, it was discussed in its own segment and will not be addressed here.

According to Kendall *et al.* (1993: 138):
"*Erector spinae group*
Origin: *Common origin from anterior surface of broad tendon attached to the medial crest of sacrum, spinous processes of lumbar and 11th and 12th thoracic vertebrae, posterior part of medial lip of the iliac crest, supraspinous ligament and lateral crests of sacrum.*
Insertion*: By tendons into the inferior borders of angles of lower six or seven ribs."*

The pars thoracis components of these muscles attach to the ribs and vertebral components. They have relatively short contractile fibres with long tendons that run parallel to the spine to their origins on the posterior surface of the sacrum and medial border of the iliac crests (McGill, 2002). This group of muscles possess an effective moment arm to generate force due to the fibres being located underneath the fasica, and they thus produce an effective moment arm with the minimum amount of compressive force being subjected to the spine (McGill, 2002).

The lumbar and thoracic portions of these muscles have been partitioned in the past into the longissimus thoracis pars lumborum and pars thoracis, and into iliocostalis lumborum pars lumborum and pars thoracis (Bogduk, 1980). These two functional groups (pars lumborum, which attach to lumbar vertebrae and pars thoracis, which attach to thoracic vertebrae) show quite a number of differences. The lumbar and thoracic sections show differences in muscle fibre composition, in that the lumbar section shows an even distribution of type 1 and type 2 fibres, while the thoracic

section demonstrates a composition of about 75% slow twitch type 1 fibre dominance (Sirca & Kostevc, 1985).

2.3.6.1.6 Gluteus Maximus, Gluteus Medius and GluteusMinimus

According to Kendall *et al.* (1993: 226):

"*Gluteus maximus*

Origin: *Posterior gluteal line of ilium and portion of bone superior and posterior to it, posterior surface of lower part of sacrum, side of coccyx, aponeurosis of erector spinae, sacrotuberous ligament, and gluteal aponeurosis.*

Insertion: *Larger proximal portion and superficial fibres of distal portion of muscle into iliotibial tract of fascia lata. Deep fibres of distal portion into gluteal tuberosity of femur.*

Action: *Extends, laterally rotates, and lower fibres assists in adduction of the hip joint. The upper fibres assist in abduction. Through its insertion into the iliotibial tract, helps to stabilize the knee in extension."*

According to Kendall *et al.* (1993: 221):

"*Gluteus medius*

Origin: *External surface of ilium between iliac crest and posterior gluteal line dorsally, and anterior gluteal line ventrally, gluteal aponeurosis.*

Insertion: *Oblique ridge on lateral surface of greater trochanter of femur.*

Action: *Abducts the hip joint. The anterior fibres rotate medially rotate and may assist in flexion of hip joint; the posterior fibres laterally rotate and may assist in extension."*

According to Kendall *et al.* (1993: 220):

"*Gluteus minimus*

Origin*: External surface of ilium between anterior and inferior gluteal lines, and inferior gluteal lines, and margin of greater sciatic notch.*

Insertion: *Anterior border of greater trochanter of femur, and hip joint capsule.*

Action: *Abducts, medially rotates, and may assist in flexion of the hip joint."*

2.3.6.1.7 Rotatores and Intertransversarii

The small rotatores muscles have been described as having a role of creating axial twisting torque, while the intertransversarii is thought to assist in lateral flexion. These muscles are very small and have small cross-sectional areas and moment arms, and their contribution to movement has been questioned due to their ability to generate only a few Newtons worth of force. It is believed that they could serve another function, possibly in assisting with stabilization rather than movement (McGill, 2002).

Some believe that these muscles could possibly serve as length transducers or vertebral position sensors in the spinal proprioception system. This is based on the evidence that these groups of muscles are well-supplied in muscle spindles, about 4.5-7.3 times the amount than the multifidus contains (Nitz & Peck, 1986; McGill, 2002).

2.3.6.1.8 Rectus Abdominis

According to Kendall *et al.* (1993: 147):

"*Rectus abdominis*

***Origin**: Pubic crest and symphysis.*

***Insertion**: Costal cartilages of the fifth, sixth and seventh ribs, and xiphoid process of sternum.*

***Action:** Flexes the vertebral column by approximating the thorax and pelvis anteriorly. With the pelvis fixed, the thorax will move towards the pelvis; with the thorax fixed, the pelvis will move towards the thorax."*

Due to the continues 'loop' nature of the rectus abdominis, the muscle also serves to function as a transmission for lateral forces from the oblique muscles. Intermuscular tendons and fascia prevents the rectus from being torn apart by these lateral forces (Porterfield & DeRosa, 1998).

Kendall *et al.* (1993) suggested that weakness of this muscle will result in a decrease in the ability to flex the vertebral column. In the supine position it will be difficult to raise the shoulders and head off the group, while in the standing position, an anterior pelvic tilt will be permitted, which will increase the lumbar lordosis. A patient

presenting with these specific weaknesses may complain of pain across the low back. This is described as fatigue early on and later as an ache which may or may not progress to being acutely painful. Pain is usually worse at the end of the day and is relieved by recumbency such as a night's sleep (Kendall *et al*., 1993).

In a very similar manner as the transversus abdominis, studies have demonstrated anticipatory contractions in the rectus abdominis and erector spinae before upper limb movement, especially before humeral flexion. This suggests that these muscles also function in an anticipatory manner towards stabilization (Friedli *et al*., 1988; Zattara & Bouisset, 1988; Aruin & Latash, 1995).

On a side note, research has suggested that all sections of the rectus abdominis activate at similar levels during flexion torque generation. The so-called upper and low concept of rectus abdominis tends to function as one muscular unit, and the distinction between upper and lower rectus does not seem to exist (Lehman & McGill, 2001).

2.3.6.1.9 Latissimus Dorsi

According to Kendall *et al.* (1993: 279):

"*Latissimus dorsi*

Origin: *Spinous processes of last six thoracic vertebrae, last three or four ribs, thorough the thoracolumbar fascia from the lumbar and sacral vertebrae and posterior one third of external lip of the iliac crest, a slip from the inferior angle of the scapula.*

Insertion: *Intertubercular groove of the humerus.*

Action: *With the origin fixed, medially rotates, adducts, and extends the shoulder joint. By continued action, depresses the shoulder girdle, and assists in lateral flexion of the trunk. With the insertion, fixed assists in tilting the pelvis anteriorly and laterally. Acting bilaterally, this muscle assists in hyperextending the spine and anteriorly tilting the pelvis, or in flexing the spine, depending upon its relation to the axes of motion.*

Kendall *et al.* (1993: 279) further states: "*Weakness interferes with activities that involve adduction of the arm toward the body or the body toward the arm. The strength of lateral trunk flexion is diminished."*

McGill (2002) adds that the latissimus dorsi has a potential lumbar stabilization function. Due to its origin at the lumbar spinous processes and its insertion on the humerus, it creates a lumbar extensor moment and stability, allowing it to be active during pulling and lifting motions, which has implications for how it is trained for functional motion patterns.

2.3.6.2 Neuromuscular Stabilization and Postural Control

Biomechanical models have attempted to explain the contributions for various factors to the potential energy of the musculoskeletal system. These factors include muscle recruitment, spinal posture and external load (Bergmark, 1989a; Gardner-Morse *et al.*, 1995; Granata & Wilson, 2001). This is important because static stability is achieved when the equilibrium posture of the spine is also a state of minimum potential energy (Thompson & Hunt, 1984).

During static postural tasks the neuromuscular response to a kinematic perturbation will cause the system to return toward the equilibrium state (Peterka, 2003). Granata and Wilson (2006) stated that stability can be estimated from the time dependant behavior of kinematic variance. When a disturbance to the state of equilibrium has occurred, the neuromuscular control system seeks to maintain postural stability by actively working to return the system to an even equilibrium state (Peterka, 2003). A state of equilibrium can be observed when kinematics that has been measured are attracted towards the static posture equilibrium (Collins & DeLuca, 1993). The system will be returned to a normal state when a kinematic disturbance occurs during static postural tasks (Peterka, 2003).

Complex dynamic tasks are an effective way to indicate neuromuscular deficits in those with low back pain (Radebold *et al.*, 2001). It is assumed that the kinematics of each dynamic movement is the same as all the others during flexion-extension movements (Dingwell & Cusumano, 2000).

Granata and England (2006: E271) reported that: "*Kinematic variances about this target trajectory are the manifestations of stochastic disturbances and control errors during the movement process. Neuromuscular response to the kinematic*

perturbations will cause the movement dynamics to be attracted towards the target trajectory". Granata and England (2006) also report that when the sum of the exponents is negative, the system is stable. This is because the rate of expansion is lower than the rate of contraction.

The procedure of postural control is very complex, as it involves an integration of sensory and motor function (Leinonen *et al.*, 2003). This is the result of proprioception, which has been described as the sensation of the position, effort and movement at a joint which is associated with muscle contraction or the timing of muscle contraction, and is derived from receptors in muscles, joints and the skin (Gandevia *et al.*, 1992).

Receptors in various tissues play different roles in the proprioception depending on the position at a given joint (Swinkels & Dolan, 1998). Muscle spindles are believed to be activated through the whole physiological range of motion, while joint receptors are only believed to be activated near the end of the range of motion (Burgess *et al.*, 1992). Injury to a joint caused by instability in peripheral joints has been reported to be because of an inability of the joint to be accurately controlled due to a lack of position sense (Forwell & Carnahan, 1996).

This lack of proprioceptive control causes protective muscular contractions to occur too late because of a delay in neuromuscular reflex activation. This can then lead to excessive movement which will place abnormal loading on joint surfaces that can lead to pain and articular damage (Forwell & Carnahan, 1996). Injured joints have shown a reduced level of proprioception (O'Sullivan *et al.*, 2002).

It has been reported previously that lumbar stenosis patients tend to be more dependent on motor control rather than on sensory conduction between the lower limbs and the central nervous system during single leg standing tests (Leinonen *et al.*, 2002). During single leg standing tests, a greater dependency is observed to be on motor control than on sensory conduct in those with lumbar stenosis (Leinonen *et al.*, 2002). It seems that proximal body segments trigger postural reactions rather than lower leg proprioception (Bloem *et al.*, 2000).

Even before body disturbance occurs which are predictable, the body initiates protective lower limb muscle activations before the initiation of the disturbance occurs (Belen'kii *et al.*, 1967). It is therefore possible to assume that in addition to lower leg function, postural control seems to be related to information processing.

Feedback errors may result in the impairment of perception of lumbar movement resulting from sensory loss, deficits in information processing or a combination of these factors (Leinonen *et al.*, 2003). Muscle spindle activity has been reported to be of great importance, since a decrease in muscle spindle input has been found to decrease in those with lumbar pain. Muscle spindle input also seems to be important to ensure the correct positioning of the lumbosacral spine (Taimela *et al.*, 1999; Brumagne *et al.*, 2000).

It has been reported that soft tissue containing nociceptors and proprioceptors are injured in the process of sudden unexpected movements such as slips and falls due to an overreaction of the neuromuscular system (Lavender *et al.*, 1993). It has also been found that the generation of muscle force that is used to stabilize the spine is very often several times larger than the external load and the body weight combined (Radebold *et al.*, 2000).

Much larger forces placed on the spine is probably due to these much larger muscle forces that are responsible for most compressive and shear forces. Under static conditions, peak muscle forces tend to increase greatly under sudden loading conditions, and these forces are even more pronounced under extreme and sudden loading (Marras *et al.*, 1987; Lavender *et al.*, 1989; Lavender *et al.*, 1993).

It has been shown that the local muscle system is much more vulnerable to dysfunction due to chronic low back pain and instability due to neuromuscular system changes. Specific dysfunctions have been shown in the multifidus as well as in the deep abdominal muscles in those with chronic low back pain (Biedermann *et al.*, 1991; Lindgren *et al.*, 1993; Hides *et al.*, 1994; Hides *et al.*, 1996; Hodges & Richardson, 1996).

These types of changes seem to result in changes in the synergistic control between the different trunk muscles (Grabiner *et al.*, 1992; Edgerton *et al.*, 1996; Hodges & Richardson, 1996). It has consequently been found that in those with chronic low back pain, the global muscle system seems to substitute or even dominate over the impaired function of the local muscle system (O'Sullivan *et al.*, 1997).

Research has found that the requirement to control lateral flexion moment of the trunk in the direction of movement with upper limb abduction is accompanied by consistent early activation of the internal and external oblique, the rectus abdominis and the lumbar multifidus (Hodges & Richardson, 1996). This activation is not simply a general increase in background muscle activity, but is related specifically to an anticipated perturbation due to variation in time onset of each of the trunk muscles with different movement in various directions (Hodges & Richardson, 1996).

There are several factors that can contribute to this situation. More required neuromuscular effort is needed when moment increases with moment velocity to control and adjust kinematic disturbances (Granata & England, 2006). Along with an increase in trunk velocity and acceleration there is also an increase in torso muscle activity and co-contraction (Dolan & Adams, 1993; Marras & Mirka, 1993). Large motor unit activation is required to modulate the muscle forces when the activity of muscles is high, and this will automatically limit fine motor control during fast paced movement (Dolan & Adams, 1993; Marras & Mirka, 1993).

Fast dynamic movements reduce the time allowed for corrections by the neuromuscular system. This suggests an increased delay in the active requirement and neural feedback which is relative to the trajectory of the movement, and a delay in feedback is suggested as a factor that causes a decrease in stability in control systems (Ogata, 2002). It is suggested that higher kinematic errors may be expected when movement is fast, as suggested by the Fitt law of motor control (Fitts, 1954).

2.3.6.3 The Role and Application of Stabilisation

Leinonen *et al.* (2003: 842) reported that: "*Protection from injury requires an ability of the body to anticipate events and to make suitable muscular responses. The appropriate proprioceptive information from trunk and lower limbs, as well as*

functional motor control of the trunk and lower limbs is essential in the maintenance of postural stability".

Stabilization refers to the ability of the low back to maintain a position that is referred to as the neutral zone, and it has been suggested that those with low back pain demonstrates difficulty in achieving and maintaining this position for any length of time. This has been suggested to be caused by a possible discrepancy in proprioception (Lam *et al.*, 1989).

The position that is referred to as the position of neutral spine has been defined as a spinal position between end-range flexion and end-range extension (O'Sullivan *et al.*, 2003). A lack of neutral zone maintenance has been reported in those with a clinical diagnosis of lumbar segmental instability (Fritz *et al.*, 1998; O'Sullivan, 2000). A suggested dysfunction in stabilization muscles in the lumbar spine has led to the suggestion that this lack of position sense in the neutral zone of motion can lead to a problem in maintaining the neutral zone (Fritz *et al.*, 1998).

Stabilization has been suggested to be performed by two recognized techniques: bracing and the draw-in. Bracing involves a general isometric contraction of all the abdominal muscles, while the draw-in involves a more specific contraction of the transversus abdominis which involved the individual drawing in the abdominal wall independently of the other large trunk muscles (Richardson *et al.*, 2002). The drawing in maneuver is a more favoured technique used in lumbar stabilization exercise programmes (O'Sullivan *et al.*, 1998; Richardson *et al.*, 2002).

A contraction of the transversus abdominis has been demonstrated with the use of real-time ultrasound during a drawing in of the abdominal wall (Richardson *et al.*, 2002). Stabilizing of the trunk prior to limb movement is the goal of this technique, as it will cause an isolated contraction of the transversus abdominis as well as the multifidus, which will facilitate movement (Teyhen *et al.*, 2005).

The drawing in maneuver has been reported to activate the transversus abdominis preferentially to the internal and external obliques, which show little change with this

maneuver. This would seem to justify the use of this technique in the use of low back pain rehabilitation programmes (Teyhen *et al.*, 2005).

Also, during the draw-in pattern it has been reported that the multifidus muscle contracts along with the transversus abdominis (Richardson *et al.*, 1999). Richardson *et al.* (2002: 401) explained further that: "*The brace pattern was a general contraction of all the abdominal muscles, involving the individual performing an isometric bracing action. Real-time ultrasound imaging of a relaxed abdominal wall and during a brace of the abdominal wall demonstrates contraction of all the abdominal muscles. Surface EMG of both the oblique abdominal muscles and the erector spinae muscles demonstrated higher values during the abdominal bracing contraction than for the draw-in pattern".*

It has been suggested that focus on minimal co-activation of the global muscle group and more focus on the isometric training of the deep abdominal muscles and multifidus should form the focus in the early stages of rehabilitation exercise programmes (O'Sullivan *et al.*, 1997). These contractions involve only a low level of the MVC (maximal voluntary contraction) are very specific and require a high level of patient compliance, but are very difficult to perform, due to the dominant substitution of other trunk synergistic muscles such as the rectus abdominis, external oblique, as well as the long back extensor muscles. The control of breathing also complicates the issue (O'Sullivan *et al.*, 1997).

During in vivo research, it has been suggested that only low levels of muscle contraction are required to achieve stability (Cholewicke & McGill, 1996). This is in line with the suggestion that strength training alone does not achieve proper motor learning and control, but depends more on pattering and inhibition of motor neurons. This will require selective inhibition of unnecessary muscular activity and the activation of additional motor units, which has to become a skilled learned by the individual (Basmajian, 1977; Edgerton *et al.*, 1996).

It has been suggested that the deep multifidus should be contracted independently of the global muscle group, namely the internal and external obliques and the rectus abdominis muscles (Magnusson *et al.*, 1996). The external oblique is also activated

during many therapeutic exercises simultaneously with the paraspinal muscles at the L5 level. Thus, it can be concluded that it is difficult to contract the lumbar paraspinal muscles (local stabilisers) independently from the external oblique (global stabilisers) during therapeutic exercises, as shown by surface EMG measurements (Arokoski *et al.*, 2004). However, in general, the activity of the abdominal muscles, especially the rectus abdominis, is lower than in the paraspinal muscles. This is an indication that load is mostly targeted at the paraspinal muscles during therapeutic exercises (Arokoski *et al.*, 2004).

Whole body exercise programmes are not recommended initially for those with low back pain, but exercises should rather focus on the activating of the transversus abdominis by means of precise self-bracing (using the drawing-in technique) techniques, which should occur independently of the other global abdominal muscles (Richardson *et al.,* 2002: Rasmussen-Barr *et al.*, 2009).

Other researchers have also advocated the training of muscular stabilization of the spine (Saal & Saal, 1989; Jull & Richardson, 1994). Thus, rehabilitation focuses on preferential activation of the deep trunk muscles (transversus abdominis and multifidus) during active movement. This has been theorised to improve the stability of the lumbar spine and found to decrease symptoms associated with low back pain significantly (Saal, 1990; O'Sullivan *et al.*, 1997; Danneels *et al.*, 2001; Richardson *et al.*, 2002).

2.4 Recommended Treatment Modalities for Low Back Pain

Both pharmacologic and conservative treatments exist as safe and effective treatments for low back pain (Joranson *et al.*, 2002). Conservative treatment remains the preferred method when treating those with low back pain (Shirado *et al.*, 2005). Low back pain has been shown to be different in each case, and it is thus impossible to expect that all will benefit from a single treatment procedure only, and the success in treating low back pain is to identify different subgroups of low back pain patients of having a high probability of achieving success with specific interventions (Cleland *et al.*, 2005). A treatment that recognizes the non-medical factors involved in back pain has been called for, such as a biopsychosocial approach to low back pain (Fordyce, 1995).

Returning patients to normal levels of activity and reassuring them on the necessity of this has been suggested to be more of a priority for primary care physicians than only focusing on diagnostic studies and specialty referrals in improving the situation of those with low back pain (Waddell, 1996). Better long-term outcomes have not been achieved by repeat visits, diagnostic testing and specialist referrals but have added more to the cost of care than achieving better results (Sundararajan *et al.*, 1998; Carey *et al.*, 1999).

Further episodes of back pain incidences, occurrences, duration and work absence due to back pain can reportedly be prevented by means of physical exercise (Burton, 2005). Several guidelines recommend exercise therapy for chronic low back pain (Spitzer *et al.*, 1987; Albright, 2001; Hayden *et al.*, 2005; Krismer & Van Tulder, 2007). Chronic low back pain seems to share a close relationship with impaired trunk muscle function (Shirado *et al.*, 1992; Shirado *et al.*, 1995a; Ito *et al.*, 1996). To strengthen and improve flexibility is the main purpose of therapeutic exercise programmes (Shirado *et al.*, 1995b).

Stabilization training is used extensively in the rehabilitation of low back pain, and this type of training is different from general exercises by being more body specific and requiring more attention and precision from the patient involved (Bergmark, 1989a). Stabilization has shown to be very effective for low back pain, and it is because of this that it has been recommended that treatments for low back pain must be scientifically proven of its effectiveness (Richardson *et al.*, 2002).

2.4.1 Acute Low Back Pain and its Necessity for Exercise Treatment

Of all the cases of acute low back pain, about 80-90% of these cases will recover within 2-6 weeks of onset with or without treatment (Kendall *et al.*, 1993; Wright *et al.*, 2005). It has been reported that most patients with acute low back pain improve rapidly over a period of one month in terms of pain reduction, disability and return to work status, and after three months, all variables reaches a plateau and remains constant over a 1 year period (Pengel *et al.*, 2003).

Approximately 30% of those with low back pain in the primary health setting are pain free after one month and 60% are pain free after their first consultation. But, it has been reported that about 60% will experience one or more recurrent episodes in the subsequent year and 20-25% will still report a significant impact on their functional status due to low back pain (Von Korff & Saunders, 1996; Van den Hoogen *et al.*, 1997; Croft *et al.*, 1998).

When patients do recover spontaneous it is less likely that they will require treatment. Treatment might not improve the possibility of a successful outcome and might even cause a worsening of the situation by prolonging medical consumption, the duration of the disease and disability (Faas *et al.*, 1993; Sinclair *et al.*, 1997). Activities will be limited to a large degree, but this restriction should not be guided by pain but rather by time, and early return to activities is recommended (Simmonds & Dreisinger, 2003).

It has been suggested that an active rehabilitation programme has to be started as soon as possible (Wright *et al.*, 2005). Mayer *et al.* (2005) recommend a combination of direction-specific exercises along with a low-level heat wrap for return to functional activities. Recommendations state that exercise intervention programmes have to be started in the so-called sub-acute stage of low back pain, which is about between four weeks to three months. The expected spontaneous recovery of acute low back pain and the early intervention have to be taken into account during the acute phase of low back pain (Elders *et al.*, 2000; Karjalainen *et al.*, 2003b; Staal *et al.*, 2003; Hlobil *et al.*, 2005).

Low back pain can also be caused by muscle spasms. Kendall *et al.* (1993: 333) reported that: "*Muscle spasm is an involuntary contractions of a muscle or of a segment within a muscle that occurs as a result of painful nerve stimulation. Irritation from root, plexus or peripheral nerve branch level will tend to cause spasm of a number of muscles, while spasm due to irritation of the nerve endings within a muscle may be limited to the muscle involved, or may be widespread due to reflex pain mechanisms.*"

Kendall *et al.* (1993) further reported that protective spasm may occur secondary to injury of underlying structures such as ligament or bone. It then acts as a protection mechanism often following a back injury. This prevents movement and further irritation of the injured structure. The treatment of these types of reactive muscle spasms should ideally form part of any treatment programme.

The summary of the recommendation comes down to the guidelines that recommend that when a person first represents with acute low back pain, they first have to be examined for the so-called 'red flags', which are indications of serious underlying pathology (Koes *et al.*, 2001). These guidelines for red flags include weakness, particularly if localized in one area such as the leg; pain and/or difficulty controlling the bladder; numbness or tingling in the feet, legs or groin; severe, disabling or night pain; serious pain and a history of cancer or intravenous drug use; pain that does not subside within a couple of days; pain in the abdomen, as well as fever and weight loss along with back pain (Burton *et al.*, 2004). If the patient doesn't present with any red flags, current recommendations state that they should be advised to continue or gradually resume their activities of daily living (Waddell *et al.*, 1997; Koes *et al.*, 2001; Waddell, 2004).

Beyond this, further recommendations state that treatment has to be delayed until the patient has been away from work for at least 4-6 weeks. This is only to prevent the slip into chronicity, as many patients will recover spontaneously from an episode of acute low back pain (Frank *et al.*, 1996).

2.4.2 Recommended Treatment for Chronic Low Back Pain

Most other treatments for chronic low back pain have been reported to be only moderate in effect (Bogduk, 2004). One of the most effective and most recommended treatments for chronic low back pain is exercise (Koes *et al.*, 1991; Nordin & Campello, 1999; Van der Velde & Mierau, 2000; Friedrich *et al.*, 2005). The only problem is that the effects of exercise programmes have sometimes reported to be small and no form have been reported to be supreme over others (Van Tulder *et al.*, 2000; Arokoski *et al.*, 2004; Liddle *et al.*, 2004; Hayden *et al.*, 2005).

Several factors determine the success of therapeutic exercise programmes. Suggestions have indicated that exercise needs to be designed for the type and stage of the particular disorder to be successful (Nuvuaga & Nwuga, 1985; Deyo *et al.*, 1990; Graves *et al.*, 1990; Manniche *et al.*, 1991; Erhard *et al.*, 1994). The intensity and the execution of the technique have to be correct as well (Mitchell & Carmen, 1990; Kohles *et al.*, 1990; Manniche *et al.*, 1991; Tucci *et al.*, 1992). Regular and consistent performance of the therapeutic exercises will ensure full benefits. All prescribed sessions have to be attended and exercise intensity has to be maintained in the form of a home programme when the active intervention has ended (Graves *et al.*, 1990; Stankovic & Johnell, 1990; Manniche *et al.*, 1993; Saur *et al.*, 1996).

No evidence currently supports the use of mode of exercise over another (Burton, 2005). This could in part be due to the natural histories of low back pain not being reported properly. Specific exercise modalities that have been scientifically validated to improve low back pain are difficult to establish, since there are differences in methodology of research that make specific regiments difficult to decide on (Simmonds & Dreisinger, 2003).

A large degree of heterogeneity in terms of content has been used in physical exercise interventions (Kool *et al.*, 2004). Poorly designed studies have also contributed to the effectiveness of the role that exercise plays in the prevention of low back injuries (Simmonds & Dreisinger, 2003).

The biomedical approach has always been the favoured approach when managing chronic low back pain (Meyer, 2007). This approach uses a two-point perspective plan when pain is present. The underlying pathology is identified and localized. Secondly, the pain is removed with an appropriate cure or remedy (Vlaeyen & Morley, 2005).

The biopsychosocial approach views pain as a dynamic interaction between physical, psychological and social factors. More realistic treatment goals for patients include:

- The reduction, mostly not elimination, of pain (decreased self-reported pain scores)
- Improvement in physical and social function such as increased range of motion, standing and walking
- Improvement of vocational/disability status such as return to work and start job training
- Improvement of general functional status such as increased activities of daily living, social recreational activities and domestic activities
- Improvement in mood and associated symptoms such as sleeping patterns
- Increased self-management of pain, and development of active coping style and self-management skills
- Reduction or elimination of opiate and sedative-hypnotic medications
- Reduction in utilization of medical services such as reduced medical procedures, inpatient admissions and outpatient visits
- Modifying sensory input by medications and/or therapeutic modalities
- Addressing misunderstandings about the meaning of pain and associated anxieties towards the pain

(Ashburn & Staats, 1999; Sanders *et al.*, 1999; Simmonds & Dreisinger, 2003; Sanders *et al.*, 2005).

The use of the multidisciplinary/interdisciplinary approach has been receiving increasing support. This treatment model uses multiple therapies in a coordinated manner which employs active interaction and a common philosophy that encourages active involvement from the patient in the pain rehabilitation programme (Ashburn & Rice, 1998; Ashburn & Staats, 1999; Karjalainen *et al.*, 2001; Karjalainen *et al.*, 2003a; Rome *et al.*, 2004).

Kääpä *et al.* (2006: 371) reported that the goal of these treatment programmes is to: "*...to provide accurate information about back pain, lend attitudes favourable towards self-care, reduce fears and worries, assist patients in developing personalised action plans to manage their back pain, and to improve functional outcomes. In brief, the aim of the multidisciplinary rehabilitation is to provide effective coping strategies*

despite of persisting bothersome pain." The pain rehabilitation programme may be defined as rehabilitation which primarily includes the dimension of physical rehabilitation and at least one of the dimensions that includes either the psychological, social or occupational dimensions(Guzman *et al.*, 2001).

This mode of treatment has been shown to produces the best outcomes in those with chronic low back pain. It places emphasis on functional restoration and functional capacity and develops coping strategies (Linton *et al.*, 2000; Caraggee, 2005). This type of approach enables patients to cope in the long-term by providing them with a type of self-management strategy (Rasmussen-Barr *et al.*, 2009). It has been reported that it is essential to address the psychosocial factors when treating musculoskeletal dysfunctions, otherwise treatment will not be as effective (Geisser *et al.*, 2005).

It has been debated that during the treatment of chronic low back pain if the primary focus should be on the reduction of pain. Pain is unpleasant for the involved person, but other major consequences of chronic low back pain such as restricted functioning, disability and work absenteeism are also long-term problems that need addressing (Staal *et al.*, 2003; Staal *et al.*, 2005). Patients also have to be recommended to stay active as long as possible (Deyo, 1996; Buchbinder *et al.*, 2001).

It has been recommended that treatment shouldn't focus primarily on pain, but rather on the consequences of pain, such as a loss of function, physical inactivity and being absent from work (Staal *et al.*, 2005). These goals are considered more important to treat, rather than pain itself. It is recommended that the reduction of pain should not been regarded as a primary goal of treatment. These other goals should rather be actively pursued, even if not reduction in pain levels occur (Sullivan, 2004). It has been suggested that the relief in pain found in studies using exercise intervention might be the result of the natural course of low back pain and not the result of the treatment programme (Shirado *et al.*, 2005).

The relief from pain has been reported not to be necessary to return to work (Lindstrom *et al.*, 1995; Crombez *et al.*, 1999; Van Tulder *et al.*, 2000). Low backpain

has a high rate of recurrence, and a life free of pain might be an unrealistic expectation, which as to be communicated to the patient in a way that they understand (Staal *et al.*, 2005).

Recommendations from research suggests that current goals should also focus on the eliminating of the use of unproven technologies or non-indicated medication, because chronic pain patients are vulnerable and at an increased risk due to their condition. Any treatment should be as conservative as possible and has to try and protect them from dangerous side-effects (Sanders *et al.*, 2005). Professionals trained especially in pain rehabilitation and management need to provide services and co-ordinated care across the various disciplines in order to achieve as many treatment goals as possible. Wherever possible, interaction should also exist between professionals, patients and their families to provide a social support network for the patient (Sanders *et al.*, 2005).

Care based on patient conditions and needs in an outpatient setting is also supported by the academic literature (Sanders *et al.*, 2005). It is recommended that a total of 20 treatment sessions per patient with chronic pain be used to match the recommendations of outcome based treatment studies (Sanders & Brena, 1993).

Advice to resume activities of daily living and work are reported to be without additional risk for aggravating back problems, since it has been demonstrated that exercise does not adversely affect the spine (Staal *et al.*, 2005). Less work absence has been reported in those who are advised to resume activities as compared to controlled treatments (Indahl *et al.*, 1998; Hagen *et al.*, 2000).

Adverse effects on work retention or current episodes of back pain have been reported to not be aggravated by a return to work and an absence of work restrictions (Hall *et al.*, 1994; Hiebert *et al.*, 2003). With current evidence, it appears that a return to work and normal activities is not associated with an increased risk for further episodes of low back pain (Staal *et al.*, 2005).

Bed rest for low back pain has generated conflicting evidence, since it is often prescribed for low back pain. Current evidence suggests that bed rest should only be

used in case of severe acute low back pain, and for nothing more than two days. Bed rest is not recommended for chronic low back pain (Brodke & Ritter, 2005).

2.4.3 The Role of Exercise as a Treatment Modality

For chronic low back pain, exercise has shown to be a beneficial form of treatment (Van Tulder *et al.*, 2004). Evidence suggests that prescribed exercise does not increase the return rate of back pain in those with a history of chronic low back pain (Staal *et al.*, 2005). A significant decrease has been shown in those with a history of recurrent low back pain when prescribed exercises has been used as treatment modality (Donchin *et al.*, 1990; Soukup *et al.*, 1999; Hides *et al.*, 2001). It has been shown that when a treatment programme is medically supervised, exercise is more effective than usual care (Hurwitz *et al.*, 2002).

In those who are working full-time with recurrent low back pain and disability, it has been reported that a reduction in short-term and long-term disability as well as a reduction in short-term pain can be achieved with remedial exercise programmes (Rasmussen-Barr *et al.*, 2009). When prescribed exercise has been used, no study has found an increase in frequency of back problems associated with exercise programmes, and other studies have found no effect on recurrence rates (Staal *et al.*, 2005).

During the treatment of chronic low back pain it has been stated that back training programmes are effective treatment for the reduction of disability and the improvement of physical function (Abenhaim *et al.*, 2000). Active physical rehabilitation is now extensively prescribed as a treatment for chronic low back pain (Arokoski *et al.*, 2004). Exercise therapy can reduce pain intensity, alleviate functional disability, and improve back extension strength and endurance (Manniche *et al.*, 1991; Taimela & Härkäpää, 1996; O'Sullivan *et al.*, 1997; Kankaanpää *et al.*, 1999).

Rainville *et al.* (2000) reported that exercise can have a multitude of beneficial effects. An altering of pain attitudes and beliefs as well as an improvement of pain intensity and disability through a desensitization of fears can concerns are possible psychological benefits. Therapeutic benefits include the improvement of physical

function that is impaired by chronic pain. The prevention of work related fatigue and muscle pain are important factors than needs to be prevented, and this can be achieved by sufficient levels of muscle strength and good physical capacity (Oldervoll *et al.*, 2001). Cognitive intervention and exercise seem to help patients overcome their psychological barriers to pain and be more physically active (Keller *et al.*, 2003) as well as having a positive effect on patients' ability to cope with pain (Arnold, 2008). It has also been recommended that exercise programmes should contain functional exercises in which both the local and global muscles work together (Bergmark, 1989a). This has been formerly described to be important in an exercise protocol (Kavcic *et al.*, 2004).

An increase in cross-sectional area along with increases in muscle strength has been previously reported in stabilising muscles (Parkkola *et al.*, 1992; Takemasa *et al.*, 1995; Mannion *et al.*, 2001a). Exercise therapy has been shown to be more effective than general practitioners providing usual care. Exercise therapy and conventional physiotherapy (a combination of hot packs, massage, traction, mobilisation, short-wave diathermy, ultrasound, stretching, flexibility and coordination exercises, and electrotherapy) are equally effective for the treatment of chronic low back pain (Van Tulder *et al.*, 2000).

Irrespective of the type of exercise compared, studies have reported that exercise programmes containing active exercises are equally effective in those with chronic low back pain (Bentsen *et al.*,1997; Ljunggren *et al.*, 1997; Mannion *et al.*, 1999; Bendix *et al.*, 2000). The effect of controlling negative pain behaviour relating to chronic back pain by exercise treatment is that intensive exercise programmes manages to make the patient expand the limits of their physical functioning and thus provides them with a feeling of pain control (Petersen *et al.*, 2002).

A decrease in the repeat of low back pain episodes by primary medical intervention doesn't seem to be sufficient. Results from long-term studies suggest that exercise treatment that is specific to low back pain and the recommencement of everyday activities has suggested to be more effective than medical treatment alone (Hides *et al.*, 2001). However, there is considerable variation in active physical treatment programmes for low back pain patients, both with respect to their duration and their

physical intensity. There also appears to be no direct dose-response relationship (Arokoski *et al.*, 2004).

Research findings indicate that the focus of therapy should be on helping patients learn awareness of body mechanics and dynamic posture; initiation and activation of a long-term exercise programme to gradually increase fitness, strength, co-ordination, a range of flexibility and motion; postural and muscle balance; specific physical coping strategies, as well as preventing debilitation caused by inactivity (Harris & Susman, 2002; Simmonds & Dreisinger, 2003; Liddle *et al.*, 2004).

Treatment modalities such as transcutaneous electrical nerve stimulation (TENS), ultrasound, heat and ice are regarded as secondary treatment options for those with chronic low back pain, and these should only be used if they assist in the ability of the patient to increase fitness, strength and range of motion (Schonstein *et al.*, 2003; Sluka & Walsh, 2003; Jousset *et al.*, 2004). Independently applied exercise and physical management programmes have to be set as long-term goals for the patient to be able to do when the active treatment has been successfully completed (Sanders *et al.*, 2005).

It has been suggested that the focus of exercise treatment in those with chronic low back pain should be on the local muscle system and the performing of specific stabilizing exercises (O'Sullivan *et al.*, 1997). The rationale for performing specific stabilizing exercises is that the repeated voluntary activation of the specific muscles induces plastic changes in the nervous system. This leads to a modification of the automatic recruitment of the trained muscle while performing functional tasks (Van Vliet & Heneghan, 2006; Tsao & Hodges, 2008).

It has been reported that beneficial effects in relieving pain and disability in those with chronic low back pain and decreasing recurrence rate after acute episodes has been achieved by performing exercises that promotes the independent contraction of the transversely orientated abdominal muscles along with the multifidus (O'Sullivan *et al.*, 1997; Richardson *et al.*, 1999; Hides *et al.*, 2001). Unfortunately, the size of the cross-sectional area of the multifidus muscle is not influenced by stability exercises and much more intensive functional exercises are needed to restore the

size of the multifidus muscle in those with chronic low back pain (Danneels *et al.*, 2001). This adds to the hypothesis that more intensive exercises are needed to restore the stabilisation muscles.

It has been previously reported that reductions in feed-forward control mechanisms of the trunk muscles have been observed in those with chronic low back pain (Hodges, 2001; Leinonen *et al.*, 2001). Observations in patients with low back pain have shown abdominal trunk muscle activation during upper (Hodges & Richardson, 1996; Hodges & Richardson, 1999) and lower limb movements (Hodges & Richardson, 1998).These muscle activations have also been observed during expected and unexpected upper limb and trunk loading movements (Magnusson *et al.*, 1996; Wilder *et al.*, 1996; Radebold *et al.*, 2000; Radebold *et al.*, 2001).

These functions appear to be improvable with active rehabilitation (Luoto *et al.*, 1996; Magnusson *et al.*, 1996; Wilder *et al.*, 1996). General exercises and advice to stay active have been shown to be beneficial for those with chronic low back pain (Maher *et al.*, 1999), In recent times, more specifically directed exercises for the spinal muscles in addition to general exercises have been recommended for those with chronic low back pain (Richardson *et al.*, 2002). Muscles that are associated with lumbar-pelvic stability have been targeted more frequently in with the aim of developing more effective and efficient exercise programmes for low back pain (Richardson *et al.*, 1999).

In conclusion it can be said that specific trunk muscle exercise programmes are aimed at restoring the structural and functional impairments that result from the effects of chronic low back pain (Kaser *et al.*, 2001; Mannion *et al.*, 2001a).

2.4.4 The Use of Exercise Intervention in Chronic Low Back Pain

A gain in muscle strength due to neural drive improvement will be the first effect of an exercise programme (Komi, 1986; Frontera *et al.*, 1988; Jones *et al.*, 1989; McCartney *et al.*, 1995). Next, hypertrophy of the muscle fibres will occur due to an increase in density (Jones *et al.*, 1989; Kadi, 2000). This will be followed by an increase in the cross-sectional area of the muscle (Keller *et al.*, 2003). In patients with chronic low back pain, selective muscle hypertrophy will occur after three

months of strength training. Research reports increases of Type 2 fibres in the multifidus with no change observed in the size of the Type 1 fibres (Rissanen *et al.*, 1995).

Pain may be moderated because of a relative reduction of physical load at work because of improved muscle strength (Oldervoll *et al.*, 2001). Muscle strength may increase with strength training, but it has been reported that at the L3-L4 level, the cross-sectional area and density remains the same (Parkkola *et al.*, 1992; Mannion *et al.*, 2000; Danneels *et al.*, 2001; Keller *et al.*, 2003). Density increases have been reported to be as much as 13% at the T12-L1 level, but the cross-sectional area in these muscles remains the same after training (Frontera *et al.*, 1988).

It is not clear whether specific modes of exercise, such as flexion, extension or strength training exercises are more effective than another (Van Tulder *et al.* 2000). The typical exercises that are tested and recommended by research include a combination of stretching, strengthening and unloaded movement exercises (Slade & Keating, 2006). Some studies uses home exercises along with formal supervised exercises programmes (Arokoski *et al.*, 2004). The present study did not use any form of unsupervised home exercises. Arokoski *et al.* (2004) reported that subjects in their study were given exercises to perform on their own at their homes. They found that most subjects exercised insufficiently at home when not being supervised.

It has been shown that the reduction of pain and disability during active physical rehabilitation is strongly dependent on a decrease in psychological distress and fear avoidance (Mannion *et al.*, 2001b). Even though pain and fear avoidance behaviours were addressed by explaining and motivating the subjects, there was still a lack of compliance from the subjects to perform the exercises at home (Arokoski *et al.*, 2004). Rasmussen-Barr *et al.* (2009) found no improvement in fear avoidance even when exercise treatments were supervised.

It has been suggested that the lumbar paraspinal muscles are both aerobic and anaerobic during therapeutic exercises when measured using surface EMG (Åstrand & Rodahl, 1991). The highest level of paraspinal electromyographic activity has been reported to be during exercises that involves lifting the hips up to a bridge position

when supine and during bilateral leg extensions in the prone position. The same authors found that exercises involving hyperextension of the back from the prone position are not the only exercises that can activate the lumbar paraspinal muscles (Arokoski *et al.*, 2004). An increase in muscle activity is produced when extra load is generated, such as holding additional weights or unbalanced limb movements (Arokoski *et al.*, 1999; Arokoski *et al.*, 2001).

On its' own, low back pain will not affect the exercise response, but sitting or standing positions may worsen pain and the patient may be prevented from performing at recommended exercise intensities or even cause a variation in effort. Patients should thus perform a variety of exercises in different positions and limitations should be identified as soon as possible (Simmonds & Dreisinger, 2003).

Petersen *et al.* (2002) compared McKenzie training and intensive strengthening exercises on chronic low back pain subjects in an outpatient-based clinic. The McKenzie group received standard McKenzie-based therapy, while the strengthening group performed their exercises in a group setting while being supervised, consisting of six subjects at a time (Petersen *et al.*, 2002).

A session began with 5-10 minutes on a stationary cycle succeeded by low-intensity warm-up exercises for 10 minutes of 10 repetitions of low resistance exercises for the lumbopelvic muscles in flexion, extension and rotation. This was then followed by intensive dynamic strengthening training that was performed in flexion and extension (Petersen *et al.*, 2002). The authors chose this type of training because it was shown to be effective in the treatment of chronic low back pain, as conducted by Manniche *et al.* (1988). Repetitions were progressed and the programme was conducted for eight weeks with two sessions per week. The authors showed improvements in both groups, but no statistically significant difference between the two groups (Petersen *et al.*, 2002).

Arokoski *et al.* (2004) reported that as measured by surface EMG, a prolong holding of the paraspinal muscles during certain exercises appears to sufficiently activate these muscles in the re-education phase. They also found that certain exercises should be added later in the rehabilitation programme when greater muscle loads

can then be tolerated. In order not to risk further injury, exercises that cause the least amount of strain should be done in the beginning of the programme (Arokoski *et al.*, 2004).

It has been suggested that training the lumbar muscles for endurance by means of longer programmes and lower effort seem much more preferable over pure strength and power training (Biering-Sorensen, 1984; Luoto *et al.*, 1995; McGill, 1998; McGill, 2002). Dynamic endurance training should be encouraged over static endurance training (Moffroid, 1997).

Sherman *et al.* (2005) compared the effect of yoga, exercise and just reading a self-care booklet to establish which was more effective. Their study lasted for 12 weeks and subjects attended weekly supervised classes, as well as exercising at home unsupervised. Their yoga was a traditional style in which the exercises were designed to be safe for those with low back pain.

Their exercise group included strengthening exercises for leg, hip, abdominal and back muscles. These were increased in terms of repetitions performed over the course of 12 weeks (Sherman *et al.*, 2005). Both the yoga and exercise groups performed their programmes for 75 minutes at a time. The study reported that the yoga group reported superior outcomes compared to the exercise group, but these were neither statistically nor clinically significant. The authors reported that yoga exercise might be beneficial for those with chronic low back pain, not because of exercise features alone, but rather through its benefits of linking physical movements with mental focus.

From a physical perspective, popular lore persists that yoga increases flexibility and strength, tones muscles and releases muscle tension (Sherman *et al.*, 2005). Several studies of patients with low back pain found that yoga increased hip flexion (Williams *et al.*, 2003), and spinal and hamstring flexibility (Baldwin, 1999; Galantino *et al.*, 2004). The authors of the study consider their form of yoga safe for persons with chronic low back pain (Sherman *et al.*, 2005).

It has been suggested that a concern for the use of exercise as a treatment would probably manifest itself in the long-term, and not in the form of any short-term adverse effects (Staal *et al.*, 2005). However, research has suggested that lower rate of recurrence of low back pain and reduced work absenteeism has been reported where regular exercise habits have been followed over a 14-month period, with regards to prescribed exercise regiments compared to those who show poor compliance (Taimela *et al.*, 2000).

As has been reported, exercise remains an effective method for treating chronic low back pain, but compliance to maintain a prescribed regiment of regular therapeutic exercise has been problematic, and has been reported by several authors (Martin *et al.*, 1984; Reilly *et al.*, 1989; Sluijs & Knibbe, 1991). Research reported has suggested that up to two-thirds of patients show poor compliance with exercise, which is especially relevant with unsupervised home training (Reilly *et al.*, 1989; Sluijs & Knibbe, 1991; Sluijs *et al.*, 1993; Nelson *et al.*, 1995).

It has been reported by Oldervoll *et al.* (2001) that the attendance rate in their research was 77% in their strength promotion group and 81% in their endurance training group. Grønningsæter *et al.* (1992) reported an attendance rate of 80% among women and 76% among men. Their participants were offered training during paid working hours. The training in the Oldervoll *et al.* (2001) study took place just before or after work hours, being very similar to the present study.

It has been reported that attendance for exercise sessions seems to be more related to intrinsic motivation factors rather than exercise sessions taking place within or outside of working hours (Oldervoll *et al.*, 2001). According to Robinson & Rodgers (1994) the completion of training depends on physical factors such as motivation, education and knowledge of and belief in the beneficial effects of physical activity on health, weight and mental health.

It has been suggested however, that clinical outcome is not necessarily associated with exercise compliance (Sluijs *et al.*, 1993). Observed result seems to suggest that valid objective adherence protocols towards exercise recommendations just simply do not exist (Friedrich *et al.*, 2005). Valid and reliable tools to assess the degree of

patient compliance has been shown to be lacking by several authors (Deyo, 1982; Faas *et al.*, 1995; Pfingsten *et al.*, 1997). Overstating of compliance and not reporting noncompliance can be attributing factors to patients performing worse on their outcome (Friedrich *et al.*, 2005).

In summary, dropout rates seem to be high in exercise intervention studies. Geisser *et al.* (2005) reported a dropout rate of 28% and Koes *et al.* (1996) reports dropout rates of greater than 20%. Petersen *et al.* (2002) reported a dropout rate of 30%. Past research has indicated that there is a high rate of non-compliance with exercise. Around 50% of subjects in supervised studies will drop-out within six months (Dishman, 1991). It has been reported that this phenomenon is not uncommon in studies of chronic low back pain patients even in an outpatient setting (Bentsen *et al.*, 1997; Keel *et al.*, 1998; Snook *et al.*, 1998).

2.4.5 Conservative vs. Aggressive Exercise Treatments

Conservative rehabilitation programmes for low back pain have always been effective. For example, previous authors had subjects performing gentle co-activation exercises of the multifidus and transverses abdominis muscles with real-time ultrasound feedback imaging. These subjects had significantly fewer recurrences than those performing no exercise (Richardson *et al.*, 1999; Hides *et al.*, 2001).

Pain and disability measurements show to be stable over a one-year period of time after an aggressive exercise-based rehabilitation programme was completed and high levels of compliance was shown with recommended exercises following the intervention programme (Hartigan *et al.*, 2000). Intensive exercise programmes have also shown to have large effect on short-term pain and function as compared to other treatments (Manniche *et al.*, 1991; Johannsen *et al.*, 1995; Petersen *et al.*, 2002). Ostelo *et al.* (2003) also reported that intensive exercise programmes were more effective on functional status and faster return to work in first-time lumbar disc surgery patients.

Exercise does seem to affect the spine in any sort of negative way. Even the aggressive and more intensive exercises performed by elite athletes shown no more negative effects that in non-athletes. Low back pain seems to occur less frequently in athletes and no greater frequency of sciatica is reported in athletes as compared to healthy controls (Videman *et al.*, 1995).

2.4.5.1 Does Aggressive Exercise Rehabilitation Play a Role in Managing ChronicLow Back Pain?

The issue of more aggressive exercise regimes for the treatment of chronic low back pain remains controversial. Goldby *et al.* (2006) reported that rehabilitation programmes show efficacy in patients with chronic low back pain but they often include universal aerobic or strenuous exercise regiments (Van Tulder *et al.*, 1997; Maher *et al.*, 1999; Van Tulder *et al.*, 2000; Furlan *et al.*, 2001; Mior, 2001).

Little importance is attached to the use of aerobic capacity in itself for the management of musculoskeletal pain (Grønningsæter *et al.*, 1992). A combination of aerobic exercises along with strength developing activities is used in most physical exercise intervention studies, but bias has been shown towards aerobic activities and thus the relative importance regarding the use of these two regiments remains unclear (Oldervoll, 2001). Increased aerobic activity has not shown to be a crucial mechanism in the reduction of low back pain (Oldervoll, 2001). Goldby *et al.* (2006) used exercises for only four muscles and focused on implementing the contractions achieved by the exercises into everyday postures and positions. Significant improvements with this regime from pre-test to post-test were shown in their research. The results were ascribed to ‘immeasurable physiological effects’, was well as factors such as peer support, patient empowerment and self-treatment, which has also been described by Long *et al.* (1996).

2.5 Ergonomics: The Key to Protecting the Spine

Mechanical circumstances that have been identified as key factors in causing low back injuries are sudden loading incidents such as trips, slips, falls and bending and twisting while lifting (Frymoyer *et al.*, 1983; Kelsey *et al.*, 1984; Bigos *et al.*, 1986; Omino & Hayashi, 1992). These types of incidences accidently arise during recreational activities. A higher rate however has been identified among professional

personnel such as nurses handling patients. Most of them will recover with six weeks of an acute injury but some may become chronic (Radebold *et al.*, 2000).

2.5.1 The Role of Ergonomic Modification and Risk Factor Prevention

The concept of risk prevention is poorly understood and documented inconsistently, but it is important and highly relevant to understand the relationship (Burton, 2005). A previous history of low back pain has been identified as being the most powerful indicator of future episodes of low back pain (Hestbaek *et al.*, 2003b).

Other important factors that have also been identified as being important indicators of risk are heavy physical work, frequent bending, twisting, lifting, pulling/pushing, repetitive work, static postures, vibrations and obesity, which stresses the disc endplates and facet joints. Smoking has also been identified as an important risk factor due to the reduced oxygen to the spinal structures (Andersson, 1997; Jansen *et al.*, 2002; Laursen & Scibye, 2002; Leboeuf-Yde, 2004).

Other factors such as rapid work pace, repetitive motion patterns, insufficient recovery time, heavy lifting, non-neutral body postures (either dynamic or static), mechanical pressure concentrations, vibration (both segmental and whole-body) and low temperatures have been identified as possible ergonomic risk factors in occupational settings that can be related to incidences of low back pain (Punnett *et al.*, 2005).

Rotation has been identified as having a greater risk factor than forward bending, and this risk increases when rotation is added to other postures (Prado-Leon *et al.*, 2005). A lack of activity has also been identified as being important, as an inactive spine or even an overactive spine performing high loads of physical activity are believed to be at a disadvantage. A U-shaped curve is believed to exist, where sedentary work and hard work are both perceived to be harmful. An occupation that requires some movement in combination with lighter tasks are believed to be better for the lumbar spine (Leboeuf-Yde, 2004). From a biological point of view, sufficient mechanical loading is needed to increase the strength of the soft tissue, but too much loading will result in tissue breakdown (McGill, 2002).

It has been suggested that static postures can contribute significantly to back pain and that the risk is associated with the maintaining of mild trunk flexion (between 21-45 degrees) (Punnett *et al.,* 1991). The risk is further increased when the trunk is twisted for more than 45 degrees or flexed laterally more than 20 degrees (Punnett *et al.*, 1991).

Prolonged seated postures especially followed by immediate lifting of heavy objects have also be identified as a risk factor (Van Vuuren *et al.*, 2005). Sitting for long periods of time also causes creeping changes in the posterior ligaments as well as in the position of the nucleus within the annulus (Adams & Hutton, 1988; McKenzie, 1979). It has been shown that only half of the intervertebral joint stiffness is regained in two minutes after 20 minutes of full flexion and some joint laxity remains after 30 minutes (McGill & Brown, 1992). It is thus considered that it is a risk factor for the development of low back pain in some occupation that lifting is required to be done with a so-called 'unstable back' (Van Vuuren *et al.*, 2005).

Low back injury risk is shown to be related to the dynamic rate of movement during repetitive trunk flexion (Marras *et al.*, 1995a). As reported earlier, evidence seems to suggest that when trunk movements include non-sagital movement components, the risk of low back injury is further increased (Fathallah *et al.*, 1998). During fast paced movements, there is a higher spinal load applied due to the influence of muscle recruitment and co-contraction of the spinal muscles which causes a load higher than with slower movements (Granata & Marras, 1995).

Analysis of lifting exertions suggests that co-contraction may be recruited, in part, to augment spinal stability (Cholewicki *et al.*, 1997; Gardner-Morse & Stokes, 1998; Granata & Orishimo, 2001). Dynamic trunk flexion could carry the risk of reduced spinal stability with increased spinal compression during dynamic trunk flexion (Granata & England, 2006). Van Vuuren *et al.* (2005) reported no increased risk during bending only, but found a much higher risk when torso flexion and twisting are combined in an occupational setting.

Psychosocial risk indicators include distress, depression, beliefs, job dissatisfaction and mental stress at work (Andersson, 1997; Hoogendoorn *et al.*, 2000; Linton, 2000).

One of the potential risks of physical activity and exercise in cases of chronic low back pain might be that it could be counter-productive to increase physical activity during episodes of pain, as physical activity involves increased biomechanical loading, which might worsen the condition of damaged spinal structures (Staal *et al.*, 2005). There is inherent risk for back injury and pain with all human activities, including exercise and work.

For the development of back pain or disc degeneration, exercise and sports participation for those without low back pain has shown no major risk factors for the development of back disorders (Staal *et al.*, 2005). Studies involving children (Harreby *et al.*, 1997), college students (Cahmak *et al.*, 2004) and adults (Suni *et al.*, 1998; Croft *et al.*, 1999) have reported that regular exercise seems to maintain healthy back status and leads to lower risks for the development of new episodes.

Exercise and sports participation have been argued not to be significant risk factors for low back problems, as a lack of participation has been reported as a risk factor for the progression of lumbar disc degeneration (Elfering *et al.*, 2002). Even among workers with jobs that require lifting over 5 000 kg per shift personal fitness level and frequency of physical activities have a positive effect on reducing the incidence of back pain compared to co-worker with much lower activity and fitness levels (Stevenson *et al.*, 2001).

It has thus been reported that prescribed exercises for those with low back pain can be relatively safe without adding difficult to deal with risk for additional injury or pain since exercise does not seem to cause an increase in risk of back pain in the asymptomatic population if the exercises are prescribed correctly (Staal *et al.*, 2005).

2.5.2 Specific Task Modification: Occupational Risk Factor Management

An occupationally related accident or activity is often blamed for damaging spinal structures and causing low back pain (Staal *et al.*, 2005). It is then when concern

becomes legitimate that prescribed physical demanding exercises, daily activities and work may cause further damage to the spine and lead to increased symptoms when the notion of cause and effect is extended into a medical setting and specific causes are explored. This could explain why restrictive recommendations for work and activities to those with chronic low back pain are provided by health care providers to manage pain and symptoms (Rainville *et al.*, 2000).

McGill (2002) recommends the following guidelines for the stages of patient progression for low back pain rehabilitation:

1. Identify and remove exacerbating activities.
2. Record in a journal the state of the low back throughout the day.
3. Develop spine position awareness.
4. Begin appropriate spine rehabilitation exercises and abdominal stabilisation.
5. Develop muscular endurance.
6. Transfer these to daily activities.

Ergonomic stressor prevention has shown good potential for disease reduction, but interventions into these types of interventions have not yet been widely implemented (Punnett *et al.*, 2005). The removal of these stressor has been hypothesized to remove back pain or at least reduce its effects (Frank *et al.*, 1996; Marras *et al.*, 2000). The major types of stressors that have been identified to be removed includes the redesign of workstations to eliminate the need for bending and twisting, installation of material or patient hoists and other lifting devices, greater variety of work tasks to avoid repetitive loading of the same body tissue and structure, and improved mechanical isolation to reduce whole-body vibration transmission (Frank *et al.*, 1996; Marras *et al.*, 2000).

2.5.3 The Back School Concept: The Role of Research and its Application

The back school concept boils down to education. It can be described as a group intervention, conducted or supervised by a paramedical therapist or a medical specialist, consisting of both an educational/skills programme and exercise intervention programmes (Heymans *et al.*, 2004). The purpose of this type of programme is in educating the patient regarding the nature of the low back pain and

disorder, and also assists them to form positive and active attitudes as well as placing emphasis on correct body mechanics and partaking in prescribed physical exercises (Hall & Iceton, 1983; Cohen *et al.*, 1994; Turner, 1996).

For those suffering from chronic low back pain, back schools have been shown to be effective in the occupational setting (Tulder *et al.*, 2001). Friedrich *et al.* (2005) used an exercise programme in conjunction with a motivational programme to treat patients suffering from chronic low back pain. Their motivational programme included extensive counselling and information strategies (such as reinforcing the internal locus of control, patient problem solving, emphasising the importance of exercise), using positive reinforcement techniques when compliance was given to exercise, signing a contract agreeing to participate in the exercise programme as well as patients reporting on the exercises that they perform each day.

They found that the combination of exercise and the motivational programme was significantly more effective than exercise alone in decreasing pain and disability, and increasing the degree of working ability (Friedrich *et al.*, 2005). This finding is explained by the effect on long-term success in that the combined exercise and motivational programme provides the patient with a ready set of tools that are retrievable even after treatment termination. It would support the patient in dealing with the multifaceted psychosocial phenomenon of chronic low back pain (Friedrich *et al.*, 2005).

It has to be explained to the patient that factors that maintain pain can be different from the factors that causes it, thus validating their pain (Meyer, 2007). For the general population, biopsychosocial principles from information and education should be given to the patient, for it has shown that it improves back beliefs and can have a positive influence on health and vocational outcomes (Burton, 2005). The intensity of the pain experienced by the patient may be increased by fear avoidance beliefs and catastrophysing (Meyer, 2007). The beliefs that patients have about their fear of pain and injury must be targeted in the early phases of the pain development, for their beliefs are important factors contributing to long-term disability and work loss (Waddell *et al.*, 1993; Picavet *et al.*, 2002).

According to Meyer (2007) certain myths about chronic pain exist that have to be expelled as soon as possible. These myths include

- Search long enough, and you will find the cause and the cure.
- Abnormal scan results validate and explain the pain.
- Only organic pain is real.
- You have to learn to live with it.
- Let the pain be your guide – rest when it hurts.
- Pain is equal to tissue damage (McIndoe, 1994).

For patients with chronic pain, the need for behavioural and psychological treatment in the interdisciplinary rehabilitation setting has been backed up with strong evidence from the literature (Sanders *et al.*, 1999; Dworkin & Breitbart, 2004; Keefe *et al.*, 2004). A patient will need psychological/behavioural treatment if significant levels of depression and anxiety are present, along with pharmacological intervention for symptoms if needed. When present, psychological/psychiatric conditions such as post-traumatic stress disorder and social adjustment issues should also receive treatment they present symptoms (Sanders *et al.*, 2005). Access to stress management training, cognitive behavioural therapy, operant therapy and biofeedback should also be available as the condition of the patient requires it (Astin, 2004).

Low intensity 'Swedish Back School' principles have shown to not be as effective as higher intensity back schools in occupational settings (Heymans *et al.*, 2005). A reduction in pain and less frequent episodes have been demonstrated by higher intensity back schools (Heymans *et al.*, 2006).

The low intensity back school based on the Swedish principle is determined by its application. This usually consists of four group sessions once a week for four consecutive weeks (Heymans *et al.*, 2006). Each session is divided into an educational (30 minutes) and a practical part (90 minutes), and guided by written information and a standardised exercise programme (Heymans *et al.*, 2006).

Subjects receive information regarding coping with back pain in their work settings as well as the work setting itself. The exercise part comprises a standardised exercise programme of strength training and home exercises. This involves progressive resistance training as well as functional exercises (Heymans *et al.*, 2006).

High intensity back school in the Heymans *et al.* (2006) study was conducted twice a week over eight weeks. It consisted of 16 sessions, each lasting an hour where the principles of cognitive behavioural therapy were applied throughout the programme (Vlaeyen *et al.*, 1995). Work simulating and strength training exercises were performed during subsequent sessions with a gradual increase in resistance (Heymans *et al.*, 2006). However, it has been reported that workers treated in low intensity back school groups return to work faster and were absent from work for fewer days than compared to high intensity back schools (Indahl *et al.*, 1995; Indahl *et al.*, 1998; Heymans *et al.*, 2006).

A beneficial effect on work absence has been reported by others in the form of high intensity graded active intervention that contained high intensity back school , but effects have been reported as appearing slow (Staal *et al.*, 2004). It has been reported that for patients in an occupational setting that suffer from chronic low back pain, intensive intervention programmes show effective results (Guzman *et al.*, 2001; Schonstein *et al.*, 2003).

It is also recommended that high intensity back schools utilising both an educational/skills programme and exercise may be used for those patients with recurrent and persistent pain (Burton, 2005).

Goldby *et al.* (2006) used subjects suffering from chronic low back pain over a time period of 10 weeks and randomised them into three groups. Their first group received a stabilisation exercise programme; the second group manual physiotherapy and their third group only received education in the form of a back school. The latter was their control group. All three their groups received the back school. However, the control group showed the least improvement in overall scores

compared to the other groups. They showed significant improvement in pre-test scores.

It may then be argued that the back school is not effective only on its own; it needs to be combined with other treatment modalities to achieve an optimal effect.

2.6 Research Problem

As stated by McGill (2002) most of the exercises prescribed for chronic low back pain are of single modal only and they have not assessed the impact of progressive treatment methods. Thus, research on this subject has been found to be limited.

Conservative treatment may be classified into three phases: primary, secondary and tertiary rehabilitation (Mayer *et al.*, 2001). The first phase is the acute management phase, which is treated for 0-12 weeks after onset and can be considered as the primary phase (Shirado *et al.*, 2005). Next, physical deconditioning has to be prevented by preventing chronic disability in the secondary phase of management. The tertiary phase involves the prevention of permanent disability for those who already suffer the effects from chronic disability by a full interdisciplinary team (Mayer *et al.*, 2005). Either secondary or tertiary rehabilitation should ideally be used on patients that present with chronic low back pain (Shirado *et al.*, 2005).

Irrespective of the type of exercises that are used, it has been shown that treatment programmes that contain active exercises are similarly effective in treating those with chronic low back pain, as shown by several studies. A method for controlling pain and inhibiting negative pain behaviour that is associated with pain is suggested to be in the form of intensive exercise programmes. These programmes are hypothesized to make the patients expand the limits of their physical functioning (Petersen *et al.*, 2002).

In a well-controlledrandomised trial Petersen *et al.* (2002) compared the effect of McKenzie therapy to intensive strength training in the treatment of chronic low back pain. They concluded that the McKenzie method and intensive strength training seemed to be equally effective in the treatment of chronic low back pain.

It has been suggested however, that there exists too little research into popular exercise techniques like the McKenzie technique to validate its' use as a treatment option. It has thus been recommended that the optimal intensity, frequency, duration and specific types of exercises be further investigated to validate their use in the clinical setting as well as in the academic literature (Hildebrandt *et al.*, 2004).

A barrier has been reported in the accurate replication of many interventions in that programmes used in research have been described completely. To have the details that would enable replication of interventions used will advance the science of exercise prescription (Slade & Keating, 2006). Uncertainty exists whether anyone type of exercise regimen (such as flexion, extension, and/or strengthening exercises) is more effective that others (Van Tulder *et al.*, 2000).

The aim of this study will then be to solve the problem by comparing conservative treatment methods to more progressive-aggressive multimodal treatment methods in the form of remedial exercise along with cognitive behavioural techniques in the form of the back school approach. The aim of the study will then be to investigate the two forms of treatment methods in the form of remedial exercises along with cognitive behavioural techniques in the form of the back school approach and to then compare their effects.
A high intensity back school approach as well as a low intensity back school approach will be used for this study.
The student will attempt to answer the following questions through his research:

- How effective are progressive-aggressive exercises versus more traditional exercise in the treatment of chronic non-specific low back pain?
- What exercises will be effective and how effective will they be when progressed?
- How will a more aggressive approach influence the outcomes compared to the more traditional approach to remedial exercise therapy?