

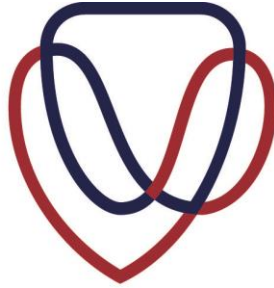


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**The efficacy of pain neuroscience education in combination with cognitive-
targeted exercise therapy in total joint arthroplasty: A randomised
controlled trial**

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*Submitted in fulfilment of the requirements in respect of the Master's degree
M.Sc Physiotherapy in the Department of Physiotherapy
In the Faculty of Health Sciences
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Declaration

I, Ruan Mockè, declare that the Master's Degree research dissertation that I herewith submit for the Master's Degree qualification M.Sc Physiotherapy at the University of the Free State is my independent work, and that I have not previously submitted it for a qualification at another institution of higher education.

A handwritten signature in black ink, appearing to read 'R. Mockè', written over a dotted line.

R. Mockè

16th day of January 2018

Abstract

Introduction: Identification of factors influencing pain and functional impairment have been studied due to the phenomenon of chronic post-surgically pain. Evidence that chronic pain is present in an unsatisfactory high percentage of individuals that undergo total joint arthroplasty (TJA), have directed this study. High levels of catastrophising and kinesiophobia is also present in individuals that undergo TJA. It needs to be established what the outcome of TJA will be if these factors are addressed as part of a standardised physiotherapy rehabilitation program (SPRP) at 12 weeks post-surgery.

Aim: The aim of this study was to evaluate the effect of pain neuroscience education (PNE) with a SPRP, in combination with cognitive-targeted exercise therapy (CTET), compared to the effect of PNE with a SPRP alone on the pain, physical function, pain catastrophising and FOM in patients undergoing TJA.

Methodology: A total of 19 individuals participated in this study. The participants were stratified into total hip arthroplasty (THA) and total knee arthroplasty (TKA) subgroups where after they were randomly grouped into a control group (n=9), and an intervention group (n=10). The data was analysed using the repeated measures analysis of variance (ANOVA). The individuals were assessed pre-surgery, on hospital discharge, six weeks and again at 12 weeks post-surgery. The intervention procedure, CTET, was administered prior to surgery and six weeks post-surgery.

Results: All outcome measures for pain, physical function, pain catastrophising and FOM had improved in the control and intervention group when baseline scores were compared to 12 weeks post-surgery. The research findings indicate that supplementing PNE and a SPRP with CTET could clinically assist in improved pain severity, pain interference and physical function as well as reduction in rumination and helplessness within the first 12 weeks post-surgery. Comparing the control and intervention group with one another showed no statistical significant difference in improvement in any outcome measure at any stage during this study.

Conclusion: PNE with a SPRP, in combination with CTET did not show a statistical significant difference in results compared to PNE with a SPRP alone on the pain, physical function, pain catastrophising and FOM within the first 12 weeks post TJA surgery. CTET may however be beneficial to improve certain aspects of pain, physical function and pain catastrophising within 12 weeks post-surgery.

Keywords: pain, chronic, arthroplasty, neuroscience, education, cognitive-targeted, fear-avoidance, function, catastrophising, physiotherapy, rehabilitation

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List of Abbreviations

Repeated measures analysis of variance (ANOVA)p.57	Osteoarthritis (OA) p. 1
Brief Pain Inventory (BPI) p. 5	Pain Catastrophising Scale (PCS) p. 5
Cognitive-targeted exercise therapy (CTET) p. 3	Pain neuroscience education (PNE) p. 2
Confidence interval (CI) p. 89	Specific, Measurable, Achievable, Realistic and Time-targeted Principal (SMART)p. 98
Diabetes mellitus type II (DM II)p. 107	Standard physiotherapy rehabilitation protocol (SPRP) p.5
Fear of movement (FOM) p. 22	Tampa Scale of Kinesiophobia (TSK-13) p. 5
Functional magnetic resonance imaging (fMRI) p. 74	Total hip arthroplasty (THA) p. 2
International Association for the Study of Pain (IASP) p.16	Total joint arthroplasty (TJA) p. 1
Intra-class correlation co-efficiency (ICC) p. 86	Total knee arthroplasty (TKA) p. 2
Magnetic resonance imaging (MRI) p. 46	Visual analogue scale (VAS) p. 30
Namibian Association of Medical Aid Funds (NAMAF) p. 84	Western Ontario and McMaster Universities Arthritis Index (WOMAC)p. 5

Operational definitions

Allodynia:

A non-noxious stimulus that is interpreted by the individual as a painful experience due to neural sensitisation (Woolf, 2011).

Chronic pain:

For the purpose of this study chronic pain will be defined as pain at or after 12 weeks post-surgery equal to or greater than three out of 10 (IASP, 2013; Piscitelli, Iolascon, Innocenti, Civinini, Rubinacci, Muratore, D'Arienzo, Leali, Carossino and Brandi, 2013).

Cognitive targeted exercise therapy:

Exercises or actions that are performed with the specific aim to reduce the participants' experienced fear of movement regarding a reasonable action (Moseley, 2003a).

Default mode network:

The term refers to the brain areas that are active when a person is not partaking in any task or form of mental exercise (Callard and Margulies, 2014).

Fear of movement:

In this study fear of movement, or kinesiophobia, refers to the irrational fear of a specific action or activity under normal circumstances which the participant should be able to perform even with a measure of effort or discomfort (Vlaeyen, Seelen, Peters, Jong, Aretz, Beisiegel and Weber, 1999).

Hyperalgesia:

A noxious stimulus that is experienced more intensely than what the stimulus would normally provoke (Woolf, 2011).

Injury:

The theoretical definition of injury, as described by Haddon (1980) (as cited in Langley & Brenner 2004), has long been considered the most comprehensive to describe causes and pathologies related to the term injury, “*namely that injury refers to damage to the body produced by energy exchanges that have relatively sudden discernible effects*” (Langley and Brenner, 2004).

Neuro plasticity:

The ability of the neural structures to alter themselves by reorganisation or through adapting function to develop effective processing for altered demands (Nudo, 2013).

Neural sensitisation:

Abnormal heightened sensitivity of nociceptive neurons to a noxious stimuli in combination with or without a nociceptive response to non-noxious stimuli (IASP, 2013) .

Neuro-signature/Neurotag:

The distinctive pattern of the brain areas at a particular time that are active during the individual’s pain experience from a specific form of stimulus or during the experience of a specific type of pain (Louw and Puentedura, 2014).

Pain catastrophising:

Pain catastrophising is characterised as the inclination to magnify the threat value of a pain stimulus combined with the feeling of helplessness in the painful situation, leading to a relative inability to inhibit pain-related thoughts in anticipation of, during or following the painful encounter (Grosen, Vase, Pilegaard, Pfeiffer-Jensen and Drewes, 2014).

Pain neuromatrix:

A general term that refers to all the brain areas that are active during the individual’s pain experience, irrespective of stimulus or the type of pain (Louw and Puentedura, 2014).

Pain neuroscience education:

The education of the participants in pain biology and pain physiology (Louw, Butler, Diener and Puenteadura, 2013).

Rumination:

The abnormal focus of an individual's attention on the sensation of pain that leads to catastrophisation over the pain experience (Forsythe, Dunbar, Hennigar, Sullivan and Gross, 2008).

Total joint arthroplasty:

The removal of the two articulate surfaces of a synovial joint and replacing them with artificial surfaces through surgery by an orthopaedic surgeon (Kahn, Soheili and Schwarzkopf, 2013). In this study two types of total joint arthroplasty are represented and referred to, namely total hip and total knee arthroplasty.

1 Chapter 1: Introduction

1.1 Background

Total joint arthroplasty (TJA) is the surgical intervention where the joint surfaces of a symptomatic pathological joint are replaced by a structural implant that mimics normal joint functions. TJA is the preferred treatment for individuals suffering from severe levels of osteoarthritis (OA) (Kahn *et al.*, 2013). OA is the most common form of arthritis, attributed to use and loading activities, with pain being the dominant complaint arising from this chronic condition (Valdes, Suokas, Doherty, Jenkins, Doherty and Rodkey, 2014). Although most individuals experience pain relief after TJA, 10 -34 % of individuals that undergo total knee arthroplasty (TKA) and 10% of individuals that undergo total hip arthroplasty (THA) are still left with chronic pain that can be described as severe between three and 43 months post-surgery (Beswick, Wylde, Gooberman-Hill, Blom and Dieppe, 2012; IASP, 2013).

The benefits of pre-operative physiotherapy exercises have been shown to improve in-hospital outcome measures relating to pain, physical function and muscle strength in both THA and TKA patients (Kuster, 2002; Rooks, Huang, Bierbaum, Bolus, Rubano, Connolly, Alpert, Iversen and Katz, 2006). Physiotherapy rehabilitation after TJA is widely supported due to the favourable results obtained by exercise and pain management strategies (Artz, Elvers, Lowe, Sackley, Jepson and Beswick, 2015). Physiotherapists' rehabilitation before and after TJA traditionally focuses on the biomechanical model, with special attention to neuromuscular training (Naylor, Harmer, Fransen, Crosbie and Innes, 2006).

Historically, pre-surgical education strategies have been implemented in a biomedical format with the aim to decrease post-surgery difficulties that could be experienced by individuals after TJA (Louw, Diener, Butler and Puentedura, 2012). A systematic review by Louw *et al.* (2012) found that most patient education consisted of information with regards to pre-admission procedures, the surgical procedure, anatomy and pathology of the arthritic joint, normal joint function, surgical complications, contra-indications, exercise, gait education, as well as milestones and pain education that related to pharmacological and non-pharmacological pain management. A biomedical approach to patient education before surgery has not shown to be hugely effective in improving post-surgery pain levels in individuals who underwent both TKA and THA (Louw, Diener, Butler and Puentedura, 2013)

Factors that facilitate acute pain to develop into chronic pain includes levels of acute pain, catastrophising, fear of pain, anxiety, depression and disuse (Vlaeyen and Linton, 2012). Understanding the origin and function of pain, as well as how the brain has the ability to alter the perception of pain, could assist the patient to cope better in the pre- and post-surgery stages (Louw, Diener, Butler and Puentedura, 2011). Certain aspects of pain catastrophising, such as the continual thought process around the pain being experienced, seem to be the most positively affected by pain neuroscience education (PNE) (Van Oosterwijck, Nijs, Meeus, Truijen, Craps, Van den Keybus and Paul, 2011). Furthermore, PNE has led to an improved pain perception in a wide variety of different musculoskeletal conditions (Puentedura and Louw, 2012; Louw, Diener, Landers and Puentedura, 2014). Interventions targeting pain, in the form of PNE, have thus been implemented with positive results (Van Oosterwijck *et al.*, 2011).

Apart from pain relief, functional abilities improved when pain catastrophising had been attended to (Leung, 2012). Pain catastrophising, on the other hand improves with improvement in pain and function, indicating a circular pattern of influence on one another (Wylde, Dieppe, Hewlett and Learmonth, 2007). Reduction in levels of pain has shown a positive influence on pain-related fear of movement (FOM) in individuals suffering from chronic pain conditions (Doménech, Sanchis-Alfonso and Espejo, 2014). These results confirm a clinical model by Vlaeyen and Linton (2000) demonstrating the effects of fear avoidance in chronic pain. The role of cognitive-targeted exercise therapy (CTET) in exercising a patient into performing a fear-avoiding activity has been suggested (Nijs, Paul van Wilgen, Van Oosterwijck, van Ittersum and Meeus, 2011; Vibe Fersum, O'Sullivan, Skouen, Smith and Kvåle, 2013)

CTET aims to expose the individual to a feared action or movement, without any danger involved, to cause memory formation through increased nerve impulse strength of the correct movement by use of stress hormones such as cortisol (Nijs, Torres-cueco, Wilgen, Girbés, Struyf, Roussel, Oosterwijck, Kuppens, Vanderweeën, Hermans, Beckwée, Voogt, Clark and Moloney, 2014). PNE is considered to be a precondition for CTET, to stimulate critical thinking and expose perceived beliefs regarding pain (de Jong, Vlaeyen, de Gelder and Patijn, 2011). The number of neuromuscular-based CTET exercise repetitions are not influenced by pain symptoms, but rather focus on a time-based format regarding patient voiced goals (Nijs, Roussel, Paul van Wilgen, Köke and Smeets, 2013). Gaining insight by questioning the patient regarding the CTET exercises highlights cognitive pain factors such as beliefs, fear-avoidance behaviours, catastrophising, hypervigilance, anxiety, stress and maladaptive coping strategies (Vibe Fersum *et al.*, 2013).

1.2 Problem statement

The aim of TJA is to replace the OA joint that is considered to be the cause of the pain. However, some individuals still suffer from moderate to severe pain after elective TJA for three months and longer. These patients thus have chronic pain although the current best evidence protocol for the management of a severe osteoarthritic knee or hip joints - TJA with standard physiotherapy rehabilitation protocol (SPRP) - have been applied. The value of adding interventions, such as PNE and CTET, to the current best evidence protocol for the management of a severe osteoarthritic hip or knee joints have not been investigated.

1.3 Research question

Does CTET, in combination with a SPRP, including PNE in patients receiving TJA, have an improved outcome on patients' pain, function, pain catastrophising and FOM compared to a SPRP and PNE alone?

1.4 Aim of the study

The aim of this study was to determine the efficacy of a SPRP, including PNE, compared to a SPRP, including PNE and CTET, on pain, function, pain catastrophising and FOM in patients undergoing TJA.

1.5 Research objectives

The specific objectives of this research study within the targeted population were to determine the efficacy of a SPRP, including PNE, compared to a SPRP, including PNE and CTET, on:

- pain as measured by the Brief Pain Inventory questionnaire (BPI)
- physical function as measured by the Western Ontario and McMaster Universities Arthritis Index (WOMAC)
- pain catastrophising utilising the pain catastrophising scale (PCS).
- fear of movement utilising the Tampa scale for Kinesiophobia (TSK-13).

1.6 Significance and justification of research

Chronic pain is a huge economic burden on society (Phillips, 2009). This study contributes to research in the field of prevention of chronic pain in TJA. In doing so it may lessen the economic burden caused by chronic pain.

The effect of chronic pain on the individual is even more profound, as it causes suffering and has a negative impact on quality of life (Phillips, 2009). This study contributes to research in the field of prevention of chronic pain in TJA. In doing so it may lessen the suffering an individual experiences and enhance his or her quality of life.

Physiotherapists play a critical role in the rehabilitation of individuals undergoing TJA, since physiotherapists specialise in the rehabilitation of individuals undergoing an orthopaedic procedure (Ministry of Health, 1976). The findings of this study provide valuable information to enhance standard physiotherapy rehabilitation in patients undergoing TJA. The findings of this study provide information on the value of CTET and PNE in the current best evidence protocol for TJA.

The findings of this study guide me as physiotherapist, as to how I can add value to the quality of the lives, of the patients I rehabilitate after TJA.

1.7 Outline of the research dissertation

This research study is presented following the outline below:

1. Introduction

In Chapter 1, this chapter, an overview of the research document is given.

2. Literature review

Chapter 2 provides a summary of current available evidence on the topic of research.

3. Research methodology

In Chapter 3 the study design and research methodology are described in detail.

4. Results

Chapter 4 provides a detailed explanation of the results of this study.

5. Discussion of results, limitations, recommendations and conclusion

In Chapter 5 the results are discussed and the final conclusion stated.

2 Chapter 2: Literature review

This chapter consists of a summary of relevant research and evidence regarding TJA, exercise rehabilitation after TJA, pain, function, FOM and catastrophising in TJA patients. Furthermore, CTET and PNE are discussed by reviewing the significant literature on these topics. Literature in this chapter ranges from 1987 to 2017.

2.1 Search strategy

Research on the applicable topics was conducted by means of online search engines during the period of January 2015 to November 2017. The literature search was limited to English publications. References cited in the identified articles were also searched for possible inclusion in the literature review.

The search engines included are:

- Pub Med,
- MEDLINE,
- PEDro,
- Science Direct
- National Centre for Biotechnology Information
- Pain Research Forum

Keywords that were regarded as important for the literature search included:

- “pain neuroscience education”
- “pain physiology”
- “asymptomatic, radiological examinations”
- “asymptomatic, pain”
- “cortisol, pain”
- “psychological factors, pain”
- “cognitive-targeted exercise therapy, pain”
- “osteoarthritis, pain”
- “total joint arthroplasty, pain”
- “fear, total knee arthroplasty”
- “fear, total hip arthroplasty”
- “catastrophising, total joint arthroplasty”
- “catastrophising, total knee arthroplasty”
- “catastrophising, total hip arthroplasty”
- “cognitive-targeted exercise therapy, total knee arthroplasty”
- “cognitive-targeted exercise therapy, total hip arthroplasty”
- “standard physiotherapy rehabilitation, pre-TJA”
- “standard physiotherapy rehabilitation, post-TJA”

2.2 Total joint arthroplasty

OA is one of the most common painful, chronic, bone diseases experienced by the general public today (Anderson and Loeser, 2010). Even with current drug and conservative therapy, TJA is generally the most effective solution to severe OA changes in hip and knee joints for the elderly (Valeberg, Høvik and Gjeilo, 2016). The need for TJA has increased dramatically during the last decade in developed countries. This trend is expected to continue as the general population ages and the availability of orthopaedic services increases (Nations, 2013). Internationally the number of THA is 131 procedures for every 100 000 individuals and for TKA 156 procedures for every 100 000 individuals, with women being more prone to undergo surgery (Kurtz, Roder, Lau, Ong, Widmer, Maravic, Gomez-Barrena, Pina, Manno and Geesink, 2007; Kurtz, Roder, Ong, Lau, Widmer, Maravic, Gomez-Barrena, Pina, Manno and Geesink, 2011)

Two groups of prosthesis namely a fixed-bearing and a rotating-platform is used regularly in individuals undergoing TKA (Hanusch, Lou, Warriner, Hui and Gregg, 2010). A polyethylene femoral interface articulates with a metal, often a cobalt chromium alloy, tibial tray (Ajwani and Charalambous, 2016). The main theoretical benefit of the rotating-platform is to reduce prosthesis wear and to reduce movement of the implant due to facilitating normal tibia rotation during gait. No statistical significant difference in these two types TKA prosthesis regarding pain and function have been found (Hanusch *et al.*, 2010; Tjørnild, Søballe, Hansen, Holm and Stilling, 2015; Ajwani and Charalambous, 2016).

THA prosthesis varies with regard to a cemented or un-cemented design for both the femoral and acetabular components (Baker, McMurtry, Chuter, Port and Anderson, 2010). Several articulate surfaces can be used by an orthopaedic surgeon. A metal femoral head, usually cobalt-chrome, on a polyethylene acetabular liner, or a metal-on-metal approach can be used. Furthermore, both the femoral head and acetabulum can be replaced with a ceramic prosthesis, or a ceramic-on-polyethylene prosthesis may be used. The posterolateral surgical approach is the most common approach for individuals undergoing THA. Advantages include that the abductor muscle mechanisms are kept intact (Baker *et al.*, 2010). This approach also gives the surgeon excellent visibility of the femur and acetabulum. Although, higher dislocation rates have been found when compared to other less common approaches (Colas, Allalou, Poichotte, Piriou, Dray-Spira and Zureik, 2017).

2.3 Standard physiotherapy rehabilitation in TJA

2.3.1 Pre- surgery physiotherapy rehabilitation for TJA

Physiotherapy rehabilitation prior to surgery has been proposed to try and reduce unfavourable outcomes including chronic pain and impaired physical function in individuals after TKA and THA (Wang, Lee, Zhang, Moodie, Cheng and Martin, 2016). Individuals undergoing THA show greater improvement with regards to pain and function with a physiotherapy-based neuromuscular training program prior to surgery than individuals undergoing TKA (Gill and McBurney, 2013). Pre-surgery physiotherapy rehabilitation has been reported to have superior improvements in pain and function within the first month after surgery when compared to non-intervention groups (Wang *et al.*, 2016). Several studies have confirmed that pre-surgery rehabilitation should be administered to individuals prior to TKA and THA due to the benefits in pain and physical function (Wallis and Taylor, 2011; Gill and McBurney, 2013; Mak, Fransen, Jennings, March, Mittal and Harris, 2014).

Physiotherapy rehabilitation for individuals undergoing TKA and THA generally focuses on improving strength and range of motion of the affected area, improving general biomechanics and includes exercises to improve balance prior to surgery (Bistolfi, Bistolfi, Federico, Carnino, Gaido, Rold, Magistroni, Actis and Massazza, 2016). Therefore, improvement in function has been achieved through greater affected leg strength, achieving equilibrium between leg strengths and improving functional daily tasks prior to surgery (Bistolfi *et al.*, 2016).

2.3.2 Post-surgery physiotherapy rehabilitation for TJA

Acute pain perception and functional abilities in individuals undergoing TKA and THA surgery have been reported to influence patient future satisfaction regarding the surgery (Bistolfi *et al.*, 2016). Physiotherapy rehabilitation after surgery has been associated with earlier returns to functional abilities, earlier return to full weight-bearing and increased walking distances (Mistry, Elmallah, Bhave, Chughtai, Cherian, McGinn, Harwin and Mont, 2016). In-hospital physiotherapy have been reported to play an important role with regards to improved function in individuals with either THA and TKA (Artz *et al.*, 2015). Data regarding the long-term benefits of physiotherapy rehabilitation in individuals after TKA and THA is however lacking according to several systematic reviews (Mak *et al.*, 2014; Bistolfi *et al.*, 2016). Although, delayed physiotherapy rehabilitation started 2 months post-surgery have not been reported to be superior to minimal physiotherapy rehabilitation (Kauppila, Kyllönen, Ohtonen, Hämäläinen, Mikkonen, Laine, Siira, Mäki-Heikkilä, Sintonen, Leppilähti and Arokoski, 2010). Early in-hospital and out-patient physiotherapy rehabilitation exercises are thus recommended (Artz *et al.*, 2015).

Physiotherapy rehabilitation after surgery includes neuromuscular strengthening exercises for both the upper and lower limb, gait retraining with and without an assistive device, techniques aimed at improving ROM and biomechanical correction exercises (Artz *et al.*, 2015). Techniques that improve the individual's knee ROM should be initiated as early as possible after TKA to decrease the possibility of the individual to have a stiff knee, which in turn decrease functionality (Mockford, Thompson, Humphreys and Beverland, 2008).

SPRP in combined with other forms of rehabilitation have also been tested in individuals undergoing TKA (Piva, Gil, Almeida, DiGioia, Levison, Fitzgerald and Fitzgerald, 2010; Fung, Ho, Shaffer, Chung and Gomez, 2012). Combining a balance specific exercise program to a SPRP, compared to a SPRP, have not indicated a statistical improvement in function, pain and ROM in individuals undergoing TKA (Piva *et al.*, 2010). It has been reported that hydrotherapy is not superior to a SPRP in decreasing pain and improving function in individuals that underwent TKA (Harmer, Naylor, Crosbie and Russell, 2009). Interestingly ergometer cycling have been suggested to improve health-related quality of life in individuals undergoing THA, but is not advised for individuals undergoing TKA (Liebs, Herzberg, Rüter, Haasters, Russlies and Hassenpflug, 2010).

2.4 Pain in OA and TJA

2.4.1 Pain in osteoarthritis

TJA is a common intervention in people suffering from hip and knee OA (OA) (Lensen, van Steyn, Crijns, Waltjé, Roox, Geesink, van den Brandt and De Bie, 2008). OA has been defined as a disease of the cartilaginous tissue, accompanied by symptoms of hyperalgesia, that is found predominantly in progressively aged individuals (Li, Kim, van Wijnen and Im, 2011). The notion that OA is joint-specific and localised has been regarded as incomplete, following evidence of altered, neurological symptoms found in disassociated sites, both unilaterally and contra-laterally, to the OA (Gwilym, Pollard and Carr, 2008). The need has been described to identify the subgroup of patients suffering from OA, who portray neural sensitised features, in order to obtain an accurate diagnosis and tailor-made, treatment protocol (Thakur, Dickenson and Baron, 2014).

A recent study utilised data from 386 participants who had undergone a 26-week conservative, chronic disease management program due to hip and knee OA (Eyles, Mills, Lucas, Williams, Makovey, Teoh and Hunter, 2016). The aim of the study was to find predictive mechanisms for the worsening of a participant's pain symptoms. The values on the Western Ontario and McMaster Universities Osteoarthritis Index Global score (WOMAC) and a self-administered scale, focusing on the general health of the treated limb, were employed to measure the deterioration of the patient's condition. The study was not able to find any exact variables that could contribute to why a participant had unsuccessful pain management. However, interestingly, individuals who were already on a TJA waiting list were found to be most likely to have unsuccessful pain management when combining the scores of their WOMAC and the outcomes of the general health of the treated limb. A possible link to a dysfunctional, body-self-perception, due to a belief that surgery is necessary, might have been a reason for the surgery list predictor (Eyles *et al.*, 2016) (also see 2.7.3).

These results obtained by Eyles *et al.* (2016) support the notion that the search for a successful management strategy for individuals, suffering from OA, should not solely focus on the intensity of the pain (Cedraschi, Delézay, Marty, Berenbaum, Bouhassira, Henrotin, Laroche and Perrot, 2013; Eyles *et al.*, 2016). Furthermore, it seems to be supported even in basic physiology, as peripheral, neurological mechanisms involved in the production of noxious stimuli (free axonal endings) in knee OA are not found in cartilaginous bone (Perrot, O'Brien, Breivik and Grossman, 2015). Free axonal endings are, however, found in the synovium, periosteum bone and tendons around the typical synovial joint (Perrot *et al.*, 2015). OA-associated, physiological changes of the joints include loss of the articular cartilage proteoglycan, progressive, articular cartilage degeneration, bony growths and subchondral bone thickening (Poulet, de Souza, Knights, Gentry, Wilson, Bevan, Chang and Pitsillides, 2014). These physiological joint changes could, therefore, be a source of peripheral neuro-plastic changes that cause the pain associated with OA (Perrot *et al.*, 2015).

When knee OA was chemically induced in rats; medication prescribed to treat chronic central neuro-sensitised pain, Amitriptyline (anti-depressant) and Gabapentin (anti-convulsant), showed a greater efficacy to decrease knee pain after 14 days than Naproxen, that is commonly administered to treat inflammatory pain due to arthritis (Ivanavicius, Ball, Heapy, Westwood, Murray and Read, 2007). The efficacy of these medications in treatment of OA, traditionally used for treatment of central neural structures, opens the door for further research in the involvement of higher functioning, neural structures in the pain experience. The pre-frontal limbic areas, which include the amygdala and medial pre-frontal cortex, that relate to individuals' emotional assessment of themselves, were reported to be actively engaged during spontaneous pain episodes in chronic knee OA (Parks, Geha, Baliki, Katz, Schnitzer and Apkarian, 2011).

2.4.2 Pain in TJA

After TJA, some patients experience a substantial amount of pain and psychological distress (Apfelbaum, Chen, Mehta and Gan, 2003). Furthermore, a large number of surgical patients still experience pain long after soft tissue repair should have taken place (Beswick *et al.*, 2012). This is confirmed by a systematic review by Beswick *et al.* (2012) indicating that the percentage of people with unfavourably long-term pain outcome ranges from 7% to 23% after THA, and 10% to as high as 34% after TKA (Beswick *et al.*, 2012). Furthermore, 44% of individuals that underwent TKA and 27% of individuals that underwent THA state that they experience continual post-surgical pain of any severity, with 15% of individuals that underwent TKA and 6% of individuals that underwent THA in the United Kingdom reporting severe chronic pain (Wylde, Hewlett, Learmonth and Dieppe, 2011). It has also been stated that pain levels are similar in individuals undergoing uni-compartmental and TKA (Lenguerrand, Wylde, Gooberman-Hill, Sayers, Brunton, Beswick, Dieppe and Blom, 2016). This therefore hints to the fact that the amount of tissue damage or repair does not directly correlate to pain severity.

Dissatisfaction and increased complaints of pain have manifested in younger TKA individuals with low income (Barrack, Ruh, Chen, Lombardi, Berend, Parvizi, Della Valle, Hamilton, Nunley and Nunley, 2014). Ethnicity plays a role in TJA surgery outcome measures as well, with African-American individuals recording more pain, less function and a greater risk of prolonged hospital stays (Ibrahim, 2010). Furthermore, smoking and uncontrolled diabetes mellitus appear to coincide with the worst outcomes in individuals undergoing TKA and individuals undergoing THA (DeFroda, Rubin and Jenkins, 2016). The importance of vascular flow on tissue healing has to be considered when taking these two factors into account.

2.5 Function in OA and TJA

2.5.1 OA in disuse and disability

Several studies have indicated the leading effect OA has in disability in adults (Covinsky, Lindquist, Dunlop, Gill and Yelin, 2008; McDonough and Jette, 2010). Greater OA severity has been linked to greater disability, although greater OA has also been linked to more advanced age, which in itself could cause decrease functionality (Sadosky, Bushmakin, Cappelleri and Lionberger, 2010). Interestingly, individuals with bilateral OA of their knees has similar results in perception of functionality questionnaires and performance testing to individuals who were only affected unilaterally with OA (Marmon, Zeni, Snyder-Mackler and Snyder-Mackler, 2013). Research has identified factors that play a role in decreasing the disability rate in the first 3 years after being diagnosed with OA. These factors include greater muscle strength, increased mental health, good self-efficacy, social network and support, and greater frequency of aerobic exercise (van Dijk, Dekker, Veenhof and van den Ende, 2006).

When considering the epidemiology of depression in persons diagnosed with OA, close to 20% of individuals experience this psychological phenomenon (Sharma, Kudesia, Shi and Gandhi, 2016). Symptomatic OA severity has been positively correlated to increase rates of depression in older adults (Kirkness, McAdam-Marx, Unni, Young, Ye, Chandran, Peters and Asche, 2012). Increased levels of pain in a OA affected joint has been reported to be a predictor of prospected disability and depression in American geriatric patients (Hawker, Gignac, Badley, Davis, French, Li, Perruccio, Power, Sale and Lou, 2011).

2.5.2 TJA in disuse and disability

The highest prevalence of THA is among individuals ranging in age from 60 to 84 years; of TKA in individuals ranging from 50 to 80 years of age (Judge, Welton, Sandhu and Ben-Shlomo, 2010; Foran, 2015). The demand for TJA in younger individuals are increasing, with an estimated 50% of individuals to be younger than 65 years old by 2030, and thus active in the workforce (Kurtz, Lau, Ong, Zhao, Kelly and Bozic, 2009). Most individuals who were employed prior to undergoing TKA and THA return to work, with the type of work influencing the return-to-work date (Tilbury, Schaasberg, Plevier, Fiocco, Nelissen and Vliet Vlieland, 2014). The average time for return to work for both individuals that underwent THA and TKA are 12.5 and 12.9 weeks after surgery respectively (Tilbury *et al.*, 2014). It is important however to know that between 14 – 19% of these individuals work up to 15 hours less per week than prior to surgery (Tilbury *et al.*, 2014).

Poor pre-surgery functional abilities have been indicated as correlating with the worst functional outcomes in individuals after TKA surgery (Sancheti, Sancheti, Shyam, Joshi, Patil and Jain, 2013; Manrique, Gomez and Parvizi, 2014). Increased pain perception by the patient after surgery may influence early in-patient management by the physiotherapist through the patient's avoidance or refusal of early mobilisation, and this can lead to secondary health problems (Pearse, Caldwell, Lockwood and Hollard, 2007). Unmet pre-surgical expectations have been found in some research to be a reason for depression (Lopez-Olivo, Landon, Siff, Edelstein, Pak, Kallen, Stanley, Zhang, Robinson and Suarez-Almazor, 2011). Although, conflicting evidence regarding the influence depression has on outcome measures such as functionality can be found in literature (Vissers, Bussmann, Verhaar, Busschbach, Bierma-Zeinstra and Reijman, 2012). It has however been established that an improvement of patients' mobility with reduction in pain will leave them more satisfied with their TJA procedure (Sullivan, Tanzer, Reardon, Amirault, Dunbar and Stanish, 2011).

2.5.3 Stress chemicals in disuse and disability in TJA

Serotonin has been thought to play an intricate role in pain control by descending pain inhibition (Bardin, 2011). Besides the influence on pain, serotonin has been linked to psychological conditions, such as depression and anxiety (Müller and Jacobs, 2010). Depletion of the availability of tryptophan for serotonin synthesis has been thought to be a driving force behind depression, anxiety and pain (Maes, Galecki, Chang and Berk, 2011). Dispensing of antidepressants and antipsychotics for chronic pain is effective in serotonin modulation (Hannibal and Bishop, 2014). Altered spinal nociception processing has been attributed to a faulty descending serotonin system which could lead to hyperalgesia after noxious tissue or nerve damage (Feng, Ming and Yu-Xia, 2012).

Exercise has been reported to positively affect serotonin production by the brain (Young, 2007). For this reason exercise has formed part of the national treatment regime guidelines for persons suffering from depression (National Institute for Health and Care Excellence, 2016). Evidence exists that exercise induced analgesia in TKA and THA individuals are present, even though the individuals have an increase in pain severity, thus confirming the role of neuromuscular exercise to aid in pain management following surgery (Kosek, Roos, Ageberg and Nilsson, 2013).

2.6 Catastrophising in TJA

2.6.1 Psychological factors in pain catastrophising

Pain catastrophising has been well associated with pain and disability in patients suffering from pain (Peters, Vlaeyen and Weber, 2005; Sullivan, Lynch and Clark, 2005). The type of surgery an individual undergoes does not play an important role in predicting whether pain may become chronic, but psychological factors, which include anxiety and catastrophising, do (Masselin-Dubois, Attal, Fletcher, Jayr, Albi, Fermanian, Bouhassira, Baudic, Kleef, Darzi, Athanasiou, Lantéri-Minet, Laurent, Mick, Serrie, Valade and Vicaut, 2013). Chronic pain, after an invasive procedure of which the primary outcome should be pain relief, adds to the psychological distress experienced by patients (Woolhead, Donovan and Dieppe, 2005; Wylde *et al.*, 2007). Furthermore, chronic pain and lasting psychological distress can be caused by neural remodelling and sensitisation ('plasticity') as a result of brief intervals of acute pain (Blacher, 1987; Carr and Goudas, 1999).

The level of pain intensity has been reported to be affected negatively by catastrophising in people undergoing TKA and lumbar fusions (Roth, Tripp, Harrison, Sullivan and Carson, 2007; Papaioannou, Skapinakis, Damigos, Mavreas, Broumas and Palgimesi, 2009). The role of catastrophising in subjectively maintaining higher pain levels in post-surgical patients, including THA and TKA, have been confirmed in research (Khan, Ahmed, Blakeway, Skapinakis, Nihoyannopoulos, Macleod, Sevdalis, Ashrafian, Platt, Darzi and Athanasiou, 2011). Two possibilities arise from the literature, namely that pain catastrophising directly leads to greater chronic post-surgical pain or that it is indirectly involved in the transition of acute pain to chronic pain by initiating fear and hypervigilance (Kremer, Granot, Yarnitsky, Crispel, Fadel, Best and Nir, 2013).

Pain catastrophising has been divided into two subgroups, namely situational and dispositional catastrophising (Campbell, Kronfli, Buenaver, Smith, Berna, Haythornthwaite and Edwards, 2010). Dispositional catastrophising is defined as the remembrance of catastrophising incidents, while situational catastrophising refers to catastrophising that is measured during or directly after the administration of noxious stimulation (Turner, Mancl and Aaron, 2004). Interestingly, situational catastrophising indicates a significantly higher correlation to experimental pain responses than dispositional catastrophising (Dixon, Thorn and Ward, 2004; Campbell *et al.*, 2010). Consequently, situational measurement of pain-related catastrophising may have greater accuracy, and may be more significantly related to the person's experience of pain than dispositional methods, which rely on the memory of how an individual responded, in general, to the noxious situation (Campbell *et al.*, 2010). In a situational procedure study, Masselin-Dubois *et al.* (2013) found pain magnification, one of the dimensions of catastrophising and measured by the pain catastrophising scale, to be an independent predictor of chronic pain intensity, irrespective of the surgical procedure the individual had undergone (Masselin-Dubois *et al.*, 2013).

Pain catastrophising is a precursor to pain-related fear, indicating that an increase in pain catastrophising is directly associated with an increase in fear (Vlaeyen, Timmermans, Rodriguez, Crombez, van Horne, Ayers, Albert and Wellens, 2004; Leeuw, Goossens, Linton, Crombez, Boersma and Vlaeyen, 2007). Fear, specifically kinesiophobia, is characterised by the unjustifiable, unfounded and debilitating fear of moving the body or body part, due to a sense of susceptibility to a painful injury or re-injury (Milenković, Kocić, Balov, Stojanović, Savić and Ivanović, 2015). Kinesiophobia and its relation to pain with certain activities were replicated by thinking of the action, whilst no noxious stimulation was given (Tucker, Larsson, Oknelid and Hodges, 2012). Reducing kinesiophobia and catastrophising have proven to improve pain levels in individuals with anterior knee pain (Doménech *et al.*, 2014). Several studies have confirmed that FOM can be used to predict the transition from acute to chronic lower-back pain (Buer and Linton, 2002; Boersma, Linton, Overmeer, Jansson, Vlaeyen and de Jong, 2004).

2.7 Fear of movement in TJA

2.7.1 Development of Fear of pain

A positive correlation between fear and pain and disability has been found in many conditions, including chronic pain conditions such as OA (Moseley, Nicholas and Hodges, 2004; Meeus, Nijs, Van Oosterwijck, Van Alsenoy and Truijen, 2010). Vlaeyen and Linton (2000) pioneered the fear-avoidance model in an attempt to describe how factors, such as pain disability, affective distress and physical disuse of the affected area, occur due to constant avoidance behaviours as a result of fear (Vlaeyen and Linton, 2000). The fear-avoidance model sets injury as the originator of the two dissimilar pathways that can be followed. Although patients may experience pain without a cause or injury, known as idiopathic pain, they can also have an injury without any pain (Louw and Puentedura, 2013). Therefore, injury as the originator of the fear-avoidance model has been replaced by an emotional or physical issue by Louw and Puentedura (2013).

It has been stated that exposure to a traumatic incident, which includes TJA, is not a prerequisite to the development of a fear for such an event or action (Hermans, Craske, Mineka and Lovibond, 2006). An individual can apply a preventative measure to a perceived feared exposure and this is known as avoidance. Continual avoidance through disuse of the affected area or limb has shown to decrease the cortical representation, motor maps in the brain (Lissek, Wilimzig, Stude, Pleger, Kalisch, Maier, Peters, Nicolas, Tegenthoff and Dinse, 2009). Apart from the development of muscle atrophy, disuse negatively affects the contractile properties of the involved skeletal muscle (Seki, Taniguchi and Narusawa, 2001). Thus avoidance does not only impair the individual's performance of the movement or task due to muscle weakness, but also impairs his or her motor task processing due to neuroplastic changes in the brain (Karni, Meyer, Rey-Hipolito, Jezzard, Adams, Turner and Ungerleider, 1998; Barton and Morris, 2003). Neuroplastic changes confirmed in literature where individuals who did not perform physical exercise after THA or TKA have decreased functional abilities (Buhagiar, Naylor, Harris, Xuan, Kohler, Wright and Fortunato, 2013)

According to Vlaeyen and Linton (2000), in their review of the fear-avoidance model, pain intensity was not considered a driving factor in avoidance or disability. Pain intensity, however, seems to be a driving factor in functional disability and, along with a previous history of lower-back pain, it appears to be the best predictor for future back pain (Sieben, Portegijs, Vlaeyen and Knottnerus, 2005; Sieben, Vlaeyen, Portegijs, Verbunt, van Riet-Rutgers, Kester, Von Korff, Arntz and Knottnerus, 2005). It has now, however, been established that acute pain intensity and the level of fear are the primary indicators of chronic pain development (Louw and Puentedura, 2013). Pain-related fear and anxiety can be described as the fear that materialises when stimuli that are associated with pain are seen as the primary risk, by the individual (Leeuw, Houben, Severeijns, Picavet, Schouten and Vlaeyen, 2007).

When acute pain is believed to not involve great risk, individuals are prone to carry on with general daily activities, promoting healing. Conversely, when the pain is catastrophically interpreted, or rather misinterpreted, it can be the beginning of a vicious cycle (Leeuw, Houben, *et al.*, 2007). The perceived threatening nature of the acute pain is thus a key factor in fear development. Critically, it has been stated that the correct understanding by the individual of his or her condition causes decreased pain and decreased disability (Virani, Ferrari and Russell, 2001). This supports the concept of the fear-avoidance model which shows that dysfunctional interpretations and limited knowledge concerning the individual's condition give rise to pain-related fear, resulting in related safety-seeking behaviours, such as avoidance and hypervigilance (Van Damme, Crombez and Eccleston, 2004; Leeuw, Houben, *et al.*, 2007). Therefore, proper knowledge of the nature of pain and pain physiology is a vital component of this study.

2.7.2 Defensive motivation factors in the fear of pain

Navratilova and Porreca (2014) describe pain as a driving force that encourages motivation and learning, with the main incentive being relief from the painful state (Navratilova and Porreca, 2014). A positive correlation has been found between the reward of relief from chronic pain and motivation behaviours (King, Vera-Portocarrero, Gutierrez, Vanderah, Dussor, Lai, Fields and Porreca, 2009). The cortico-limbic region is activated both during an individual's expectation of a noxious event and the expectation of relief from the noxious event (Wager, Rilling, Smith, Sokolik, Casey, Davidson, Kosslyn, Rose and Cohen, 2004). These activated brain areas include the thalamus, the insula and the anterior cingulate cortex. Researchers have found that the pain experience can be altered cognitively by using descending, endogenous modulatory systems (Bushnell, Čeko and Low, 2013). Therefore, interventions that focus on cognitive recruitment have the possibility to curb chronic pain conditions.

The acute pain experience motivates an individual to acquire pain relief effects through medication or relief-associated actions, even if increased benefit from these responses are not present (Gandhi, Becker and Schweinhardt, 2013). This maladaptive, coping strategy could lead to a reduced motivation to complete or initiate new goal-directed tasks found in chronic pain sufferers (Schwartz, Temkin, Jurado, Lim, Heifets, Polepalli and Malenka, 2014).

Areas that have shown activation during reward-guided learning and decision-making include, amongst others, the anterior-cingulate cortex (ACC) (Rushworth, Noonan and Boorman, 2011). The ACC accumulates information regarding the reward of a specific action; in pain patients the reward will be pain relief through a specific action. This learned, pain relief action will then be selected in subsequent painful situations (Navratilova and Porreca, 2014). Thus, even maladaptive actions for the acquisition of pain relief will then be repeated and thus be established as a coping strategy (Navratilova and Porreca, 2014). An obsessive persistence to gain control over chronic pain by an individual or family members through assorted interventions has the contra-effect of preventing the individual from adapting successfully to a painful situation without assistance (Lauwerier, Van Damme, Goubert, Paemeleire, Devulder and Crombez, 2012). Thus, the multiple, failed attempts to obtain pain relief may not only de-motivate an individual, but also hinder the patient from forming strong coping techniques when in pain (Gandhi *et al.*, 2013; Navratilova and Porreca, 2014).

2.7.3 Threat perception factors in the fear of pain

It has been reported that attention towards or away from a noxious stimuli changes neuronal responses in the dorsal horn at the spinal cord level (Sprenger, Eippert, Finsterbusch, Bingel, Rose and Buchel, 2012). Neuroplastic changes are vital to enhance or suppress pain from developing from acute to chronic. Research has reported markedly decreased pain ratings in participants while undergoing a pain stimulus when listening to their favourite music, compared to the control group who were not exposed to music (Dobek, Beynon, Bosma and Stroman, 2014). Additionally, the participants who were exposed to the music showed activation of the descending pain modulation pathways in the brain and the spinal cord (Dobek *et al.*, 2014). Therefore, changing the focus of an individual from the intensity of the pain or disability through acquired knowledge and improved functional abilities can cause positive neuroplastic changes in the peripheral neural tissues (Dobek *et al.*, 2014) (also see 2.11).

Disruption of a normal body-self-perception is recognised as an attributing factor in chronic, neuropathic pain syndromes, such as reflex sympathetic dystrophy (Lewis and Schweinhardt, 2012). A painful stimulus that occurs while the individual is looking at the body part has been reported to correlate with a decreased pain score (Longo, Betti, Aglioti and Haggard, 2009). Interestingly, the greatest visual analgesic effect with a painful stimulus has been found to be when the individual was looking at his own hand while it was placed in a crossed-arms position (Valentini, Koch and Aglioti, 2015). Furthermore, Lewis and Schweinhardt (2012) found that participants with reflex-sympathetic dystrophy had decreased touch perception abilities, using two-point discrimination thresholds (Lewis and Schweinhardt, 2012). These studies, therefore, support the notion that acquiring the correct perception by an individual of his or her own body and the state of the effected physiological tissues, should be incorporated in the management of a chronic pain patient (Lewis and Schweinhardt, 2012; Valentini *et al.*, 2015).

2.8 Biomedical education prior to surgery

Pre-surgical information booklets have been widely used with positive results, to increase knowledge regarding the nature of the operation, contra-indications and rehabilitation post-surgery (Eschalier, Descamps, Pereira, Vaillant-Roussel, Girard, Boisgard and Coudeyre, 2017). The use of interactive information DVD's have been reported to decrease patient length of hospital stay after surgery (Yoon, Nellans, Gellar, Kim, Jacobs and Macaulay, 2010).

In a systematic review Louw *et al.* (2013) showed that education prior to TJA was primarily given on a one-to-one basis by physiotherapists. The content of the physiotherapy educational sessions revolved around educating the individual about normal and pathological anatomical structures, surgical intervention, post-surgery rehabilitation along with milestones and contra-indications, as well as pain management with regards to medication and non-medicinal options. Implementing pre-surgical education can vary from six weeks to one day prior to THA and TKA surgery, with the prevailing timeframe being between two to four weeks prior to surgery with a duration of 20 - 40 minutes for an education session (McDonald, Page, Beringer, Wasiak and Sprowson, 2014).

According to a Cochrane review education prior to TKA and THA surgery may not have superior results when compared to standard care, which includes pharmaceutical pain management and exercises, with regards to pain, function and health-related quality of life (McDonald *et al.*, 2014). Factors that include length of hospital stay indicate individuals that underwent TKA is affected more positively by pre-surgery education than individuals that underwent THA. Due to TKA having an increased risk of chronic pain after surgery, a greater focus on this subgroup may influence this result (Lenguerrand *et al.*, 2016). Anxiety prior to surgery has also been positively affected by pre-surgical education which could have a positive effect on the individuals coping ability (McDonald *et al.*, 2014).

2.9 Biopsychosocial model

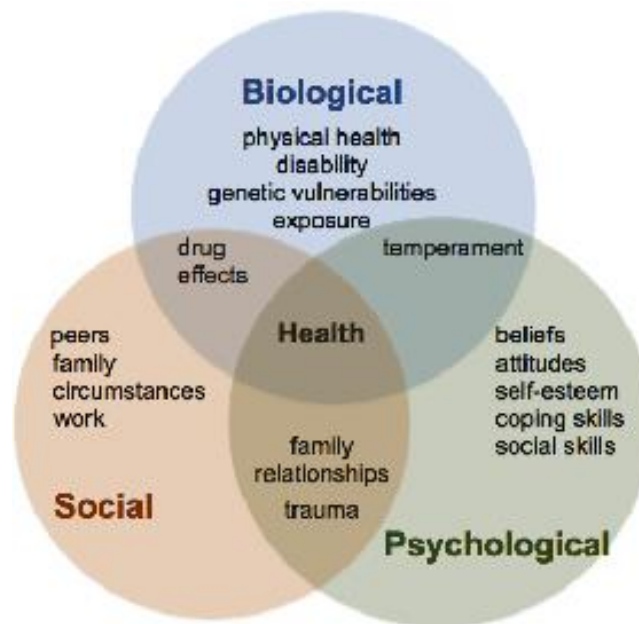


Figure 2-1: Illustration of the Biopsychosocial model – Borrel-Carrió, Suchman and Epstein (2004)

The biopsychosocial model was first introduced in 1977 by physician, Dr George L. Engel, and psychiatrist, Dr John Romano (Borrell-Carrió, Suchman and Epstein, 2004) (see Figure 2-1). The biopsychosocial model methodically regards biological, psychological, as well as social, factors and the relations between these subsystems in improving health, illness and health care delivery for patients (see Figure 2-1). The biological subsystem defines the involvement of anatomy of diseases as a whole, and the effect of these on the patient's biological functioning. The psychological subsystem deals with the results of psychodynamic factors like motivation and personality on the experience of the response to ill health, while the social subsystem involves the cultural, environmental and familial influences on the expression and familiarity caused by ill health (Dogar, 2007).

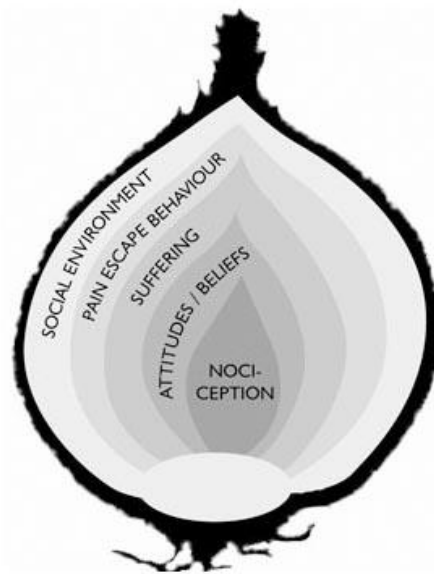


Figure 2-2: Illustration of the Onion skin model of pain – Waddell (1998)

The onion skin model is another way to describe the factors that are involved in the pain experience visually (Waddell, 1998) (see Figure 2-2). With the onion cut in half, each skin layer represents a factor that is involved in eliciting a painful experience. The five layers represent, from central to lateral, a) nociception, b) attitude and beliefs, c) suffering, d) pain escape behaviours and e) social environment (Loeser, 2000).

The onion skin model has been supplemented with the orchestra model (Butler and Moseley, 2003). The orchestra model represents the virtual body, neuromatrix and neurotag as working in combination with processes in tissues and danger messaging processes. An example of this is the central, nociception onion skin activating an alarm response, like an orchestra playing a tune. The tune is represented by the neurotag involved in the pain experience. When an orchestra can only repeat one tune, the recurrent neurotag becomes a stronger reaction, and the ability to react in a different way decreases. The neurotag then becomes the dominant adaptive or maladaptive reaction to the specific stimulus. These ignition cues from various onion skin layer factors, as well as fear, damaged tissues and inaccurate neural information, add to the knowledge that, even though pain is a central processing event, it manifests itself in anatomical and biological ways (Butler and Moseley, 2003).

It can be argued that, as each patient and his or her condition varies, the list of factors that constitutes the biopsychosocial approach to their situation may vary (Jull and Sterling, 2009). The binding factor to this approach is that what the patient thinks, feels and believes about his or her condition will impact the evaluation, treatment and prognosis significantly (Vlaeyen, Kole-Snijders, Boeren and van Eek, 1995).

2.10 Pain neuroscience education

The increase in awareness regarding the influence of psychological factors on pain has led to studies on how to equip patients with coping skills prior to surgery (Riddle, Keefe, Nay, McKee, Attarian and Jensen, 2011; Somers, Blumenthal, Guilak, Kraus, Schmitt, Babyak, Craighead, Caldwell, Rice, McKee, Shelby, Campbell, Pells, Sims, LaCaille, Huebner, Rejeski and Keefe, 2012; Tsui, Day, Thorn, Rubin, Alexander and Jones, 2012). It has been stated that physiotherapists are able to incorporate coping skills into their regular, treatment protocols with good results (Bennell, Egerton, Bills, Gale, Kolt, Bunker, Hunter, Brand, Forbes, Harris and Hinman, 2012). Effectively utilising the brain's capabilities in the management of pain perception creates the capacity for every person to self-manage their pain. PNE aims at increasing patients' knowledge and understanding of the physiology of pain, thus reducing fear associated with a musculoskeletal injury (Louw *et al.*, 2011).

Due to the ability of the brain to inhibit or excite pain perception, natural reasoning necessitates the education of patients who experience pain. Given the complexity of pain neurophysiology, the initial thinking was that it would not be feasible to educate the patient about understanding the pain neuromatrix. Moseley (2003a) reveals that this is not the case, and that the patient can establish a constructive idea of pain through this biopsychosocial approach when educated appropriately (Moseley, 2003a).

Educating the patient regarding the complex biopsychosocial response to pain is primarily done on a one-to-one basis by a physiotherapist (Louw and Puentedura, 2013). PNE revolves around explaining the role of different mechanisms around injury and factors influencing the individual's pain experience. Anatomical structures of neurons, including action potentials and synapses along with nociception and nociception pathways are explained in this education. Furthermore, peripheral and central sensitisation along with spinal inhibition and facilitation are also covered by PNE (Louw *et al.*, 2011). The detrimental effects of avoidance and disuse as a maladaptive coping mechanism are stressed as to improve the individual's self-management of their pain. Important factors that include catastrophising, FOM and anxiety with regards to their influence on pain are however not covered directly in PNE (also see 2.6). Considering the existing evidence regarding the influences of these factors on pain, an active intervention targeting catastrophising, FOM and anxiety is deemed necessary.

In a systematic review by Louw *et al.* (2011), PNE proved to be effective in decreasing chronic pain, as well as functional limitations and catastrophising (Louw *et al.*, 2011). Due to the acknowledgement of the origin of the pain by the participant, instantaneous cognitive effects take place. The results are shown in the immediate effect on the patients' attitude to pain and their improvement in physical tasks and pain perception (Moseley *et al.*, 2004). In the study conducted by Moseley *et al.* (2004), the physical effects of PNE on pain were seen in the neural tension tests of participants with chronic lower-back pain, which included the straight-leg raise and forward bending tests. Similar results were generated in lumbar and chronic, spinal pain patients (Louw *et al.*, 2011; Dolphens, Nijs, Cagnie, Meeus, Roussel, Kregel, Malfliet, Vanderstraeten and Danneels, 2014). Added benefits of the biopsychosocial approach include a decreased need for recurrent visits to the doctor and additional tests after surgery (Louw *et al.*, 2014).

Although the content of PNE has been standardised, the duration and frequency in current literature varies greatly. Although initial PNE sessions lasted up to four hours, the recent trend in literature is for shorter 30 minute sessions (Louw, Nijs and Puentedura, 2017). The effects of PNE in combination with other interventions have reported promising results. One study has stated that PNE alone was superior to combining it with an exercise program for immediate pain reduction, although at three months the superior effects were negated (Ryan, Gray, Newton and Granat, 2010). The ability of PNE to have an immediate effect not only on pain perception but also on neural tension has been demonstrated in using a straight leg raise and brachial plexus, provocation tests (Moseley *et al.*, 2004; Van Oosterwijck *et al.*, 2011; Louw, Diener and Puentedura, 2015).

Due to the negative impact of catastrophising on an individual's pain perception, PNE studies have often included it as a secondary outcome in the form of the PCS (Moseley *et al.*, 2004; Meeus *et al.*, 2010; Van Oosterwijck *et al.*, 2011). Research regarding the effects of PNE on OA conditions of the hip and knee is sparse. A recent study on individuals suffering from knee OA found that PNE significantly affected pain catastrophising and FOM after surgery, in a positive way (Lluch, Dueñas, Falla, Baert, Meeus, Sánchez-Frutos and Nijs, 2017). Other central sensitisation factors however did not show a difference between PNE and a biomedical approach to education.

The rumination subscale of the PCS shows to benefit most from PNE (Meeus *et al.*, 2010) (also see 3.2.3.4). One research paper evaluating education prior to surgery indicated favourable results to improve pain experiences in the systematic review done by Louw *et al.* (2013). The researcher therefore added a pain management subsection, which included an overview of pain and possible causes of pain, into the general educational session for the intervention group (Louw, Diener, *et al.*, 2013).

A handful of studies focused on the effects of PNE on fear by using the TSK-13 (Meeus *et al.*, 2010; Ryan *et al.*, 2010; Van Oosterwijck *et al.*, 2011) (also see 3.2.3.5). Encouraging results have been found by Van Oosterwijck *et al.* (2011) which show that PNE significantly reduces pain-related fear in chronic, whiplash patients. In chronic lower-back pain and chronic fatigue syndrome, no statistically significant decrease in FOM or fear of (re)injury has been measured (Meeus *et al.*, 2010; Ryan *et al.*, 2010). In a recent study on chronic lower-back pain, lumbar instability was found to predict which individuals suffered from fear in the intervention group, whereas in the control group it was catastrophising and self-efficacy (Pérez-Fernández, Lerma-Lara, Ferrer-Peña, Gil-Martínez, López-de-Uralde-Villanueva, Paris-Aleman, Beltrán-Alacreu and La Touche, 2015). No studies could be found after extensive searching in literature regarding the impact of PNE on fear in TKA and THA individuals by the researcher.

The sensitivity to fear is linked to functional restrictions in individuals suffering from chronic musculoskeletal pain (de Jong *et al.*, 2011). In a case report, a young female patient with lower-back pain had significant reduction in her psychological distress factors, including fear avoidance, by combining PNE with other physical treatment modalities (Zimney, Louw and Puentedura, 2014). PNE can thus possibly be employed in combination with other treatment modalities to diminish fear. The positive effects of one-on-one PNE education has been shown to last up to 12 months in chronic low back pain individuals (Moseley, 2003b). The lasting effects of PNE when combined with another treatment modality has been proven by several randomised control trials (Louw and Puentedura, 2013). Due to the lasting effects of PNE, preventative PNE has come under consideration to assist in deterring maladaptive coping strategies and sensitisation (Nijs *et al.*, 2013)

2.11 Cognitive-targeted exercise therapy

CTET aims to decrease fear by gradual exposure to the activity that reproduces the participant's fear. Although the pain neuromatrix involves numerous areas in the brain, the 'emotional' areas in the brain are the amygdala and anterior cingulate cortex. The key issues relating to fear and pain are the negative emotions and pain-related memory role of the amygdala (Li, Wang, Chen, Zhang and Wan, 2013). The ability of the body to remember pain areas and fear, has been reported to correlate with nociceptive sensitisation and prolonged, pain perception (Flor, Knost and Birbaumer, 1997; Crombez, Vlaeyen, Heuts and Lysens, 1999). The ability to provide a detailed and effective, CTET program requires a systematic approach by the physiotherapist (Butler and Moseley, 2003)

The skill to communicate evidence-based pain neurophysiology in a clear manner complements the treatment approaches physiotherapists have during one-on-one consultations. Furthermore, academic study material written by physiotherapists for teaching PNE are available, along with post-graduate short courses (Louw and Puentedura, 2013). Lastly, physiotherapists have an in-depth knowledge surrounding exercises, which includes neuro-muscular training that is deemed necessary to complete a CTET program (Nijs, Girbés, Lundberg, Malfliet and Sterling, 2014). Physiotherapy fills the crucial role for applied CTET when considering the requirements necessary to successfully provide CTET. Understanding pain physiology, the pain neuromatrix and pain mechanisms, including catastrophising and fear, forms the basis for the requirements to recognise and address pain sensitisation in individuals (Butler and Moseley, 2013).

The notion that the brain has a specific area dedicated to pain and pain perception has been negated by researchers doing cerebral lobotomies (Reiman, 2016) (see Figure 2-3). Functional magnetic resonance imaging (fMRI) has been vital in discovering the numerous areas in the brain involved in the pain experience (Moseley, 2003a; Flor, 2012). These areas have collectively been combined to formulate the pain neuromatrix (Louw and Puentedura, 2014).

The novel idea of a pain neuromatrix was encouraged by four conclusions deduced from phantom limb pain observations (Melzack, 2001). These conclusions were made because of the fact that, firstly, phantom limbs were described as actual, and it may be deduced that what the body experiences as normal is sub-served by the same brain processing as the phantom limb. As the processing acts on, and changes due to, input from the body, it could also act and change separate from these inputs. The second conclusion describes that information normally experienced from the body could be experienced in absence of these bodily inputs; thus, the information was dependent on neural networks in the brain. Melzack (2001) suggests that stimuli or input from the body elicits activity in the neural networks but does not create it. Thirdly, the autonomous identification of the body can only be attributed to central processing, and not to the peripheral nervous system or the spinal cord. The fourth conclusion was that this acknowledgement of self, apart from other persons or environments, was a result of genetic specification, but could be altered by a person's experience.

Melzack (2001) further describes four components derived from the four conclusions stated above. The components of this theoretical nervous system network are the body-self neuromatrix, the cyclic order of processing and synthesis in which a neuro-signature is produced in the neuromatrix, the living neural core which transduces the neuro-signatures into awareness and the establishment of an active neuromatrix that provides a reaction pattern to obtain the desired effect (Melzack, 2001). A typical neurotag has been constructed by Butler and Moseley (2003) (see Figure 2-3). Areas involved in this typical neurotag are the premotor/motor cortex, the cingulate cortex, the prefrontal cortex, the amygdala, the sensory cortex, the hypothalamus/thalamus, the cerebellum, the hippocampus and the spinal cord (Butler and Moseley, 2003).

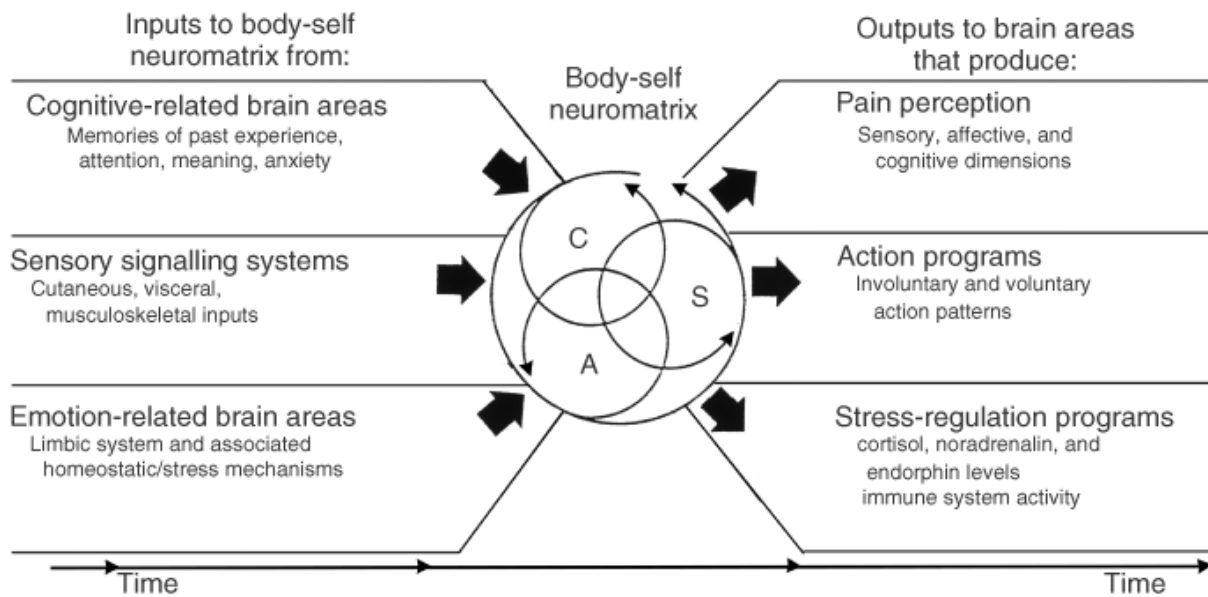


Figure 2-3: Illustration of the pain neuromatrix – Butler and Moseley (2003)

C: Cognitive neuromodule, A: Affective neuromodule, S: Sensory neuromodule

A three point approach is used in CTET when considering the pain neuromatrix, and includes graded activity (Macedo, Smeets, Maher, Latimer and McAuley, 2010), graded exposure *in vivo* (de Jong, Vangronsveld, Peters, Goossens, Onghena, Bulté and Vlaeyen, 2008) and self-control-based interventions, such as acceptance and commitment therapy (Wicksell, Olsson and Hayes, 2010). During beneficial and positive treatments and actions, the amygdala activity, as well as the activity of the somatosensory cortex and the insula, decreases (Schmid, Theysohn, Gaß, Benson, Gramsch, Forsting, Gizewski and Elsenbruch, 2013). Schmid *et al.* (2013) thus validate the fact that positive, secure and safe movement reduces fear, as well as the emotional components such as catastrophising produced by fear.

In a randomised control trial, conducted by Vibe Fersum *et al.* (2013), CTET proved superior to traditional, manual therapy in the pain management of non-specific, chronic lower-back pain (Vibe Fersum *et al.*, 2013). The manual therapy included the mobilisation and manipulation of the spine and pelvis, as well as exercise therapy, which included isolated, deep abdominal, contraction exercises. All outcome measures, including pain-related fear, showed a marked improvement in the individuals' chronic lower-back pain due to CTET. Encouraging results in pain and fear improvement were also found in a recent study on an individual who had undergone lumbar, spine surgery (Louw *et al.*, 2015). It has also been stated that CTET exercise are beneficial, as far as upper limb function and quality of life are concerned, to individuals who have had an upper, motor-neuron incident, such as a stroke (Lee, Bae, Jeon and Kim, 2015).

High levels of FOM and catastrophising have been found in TKA individuals (Sullivan, Tanzer, Stanish, Fallaha, Keefe and Dunbar, 2009). Functional exercises aimed at reducing FOM were found to have superior results in pain, kinesiophobia and health-related quality of life when compared to general advice to TKA individuals to continue being active (Monticone, Ferrante, Rocca, Salvaderi, Fiorentini, Restelli and Foti, 2013).

2.12 Conclusion

Patients commonly undergo TJA surgery due to pain and chronic, degenerative changes in joints (Dieppe, Basler, Chard, Croft, Dixon, Hurley, Lohmander and Raspe, 1999). Reasoning, according to the biomechanical model, will render surgery and musculoskeletal rehabilitation as sufficient interventions. Unfortunately, this is insufficient for the patient who has already undergone sensitisation due to chronic pain. As discussed, the neurophysiological changes experienced by prolonged periods of pain theoretically do not reorganise when the affected joint is replaced. Risk factors that lead to a state of chronic pain include acute pain levels, previous pain experiences and attitudes or fear toward pain (Denk, McMahon and Tracey, 2014). Acute pain, pain memory and the knowledge that no nociceptive input is needed to experience pain, drive the need for acute and sub-acute, post-surgical interventions to decrease the prevalence of chronic, post-surgical pain through cognitive strategies (Li *et al.*, 2013).

The positive results from numerous studies on the effects of PNE on not only pain, but also physical and psychological conditions, have made it an essential tool in pain management practice (Louw *et al.*, 2011; Dolphens *et al.*, 2014). CTET seeks to diminish fear associated with movement, and catastrophising directly and has thus far reported promising results (Vibe Fersum *et al.*, 2013). Due to the fact that fear and catastrophising have the ability to cause acute pain to develop into chronic pain, CTET is seen as a vital key in unlocking successful, chronic pain prevention and management.

3 Chapter 3: Research design and methodology

This chapter describes the research methodology employed in this study. It includes the study design, the study population, sample size, inclusion and exclusion criteria, as well as outcome measures used. A detailed discussion of the data collection procedure is also included. Further discussions focus on the ethical considerations, to which the researcher adhered, and possible methodological errors that were identified.

3.1 Study design

The study design was a quantitative, stratified, randomised controlled trial. A randomised, controlled trial was selected as it was deemed best to answer the research question. The design has the ability to determine the effect of the intervention on the participants by comparing the intervention group with the control group (Joubert, Bam and Cronje, 2008).

3.2 Study participants

3.2.1 Study population

The study population consisted of all consenting patients undergoing unilateral primary THA or TKA for severe OA of the hip and knee joint respectively, in Swakopmund, Namibia.

3.2.2 Study sample and sample size

All participants in this study population were residing either in Walvis Bay, Wlotzkasbaken, Henties Bay or Swakopmund, Namibia. These study participants were referred to the Swakopmund Medi-clinic for TJA surgery. The combined total number of TKA and THA done in Swakopmund as acquired from the Namibian Association of Medical Aid Funds (NAMAF) were 14 in 2013, 23 in 2014 and 22 in 2015. The researcher aimed at attaining as many study participants as possible within a one-year time constraint to complete the study. A sample size of 20 participants were thus required and deemed reasonable, taking into account the number of individuals undergoing THA and TKA over the previous three years in Swakopmund. At the end of 2016 a total of 26 individuals underwent TJA in Swakopmund, with 23 individuals meeting the inclusion criteria.

Inclusion criteria:

- Patients had to give informed consent after receiving information regarding the study.
- Patients had to receive unilateral primary joint replacement due to severe OA resulting in chronic pain in either the knee or hip joint.
- Participants had to be literate in either Afrikaans or English.
- The participants had to be available from three to six weeks prior to surgery to permit time for the intervention.
- Participants had to be older than 18 years.
- Participants had to come from the three collaborating orthopaedic surgeons performing surgery in Swakopmund Medi-Clinic in Namibia.
- The participants' primary physicians had to give medical clearance to the researcher to ensure all individuals had the cardiac and pulmonary ability to undergo a SPRP.

Exclusion criteria:

- Patients with any medical condition in which moderate levels of exercise was contraindicated (i.e. uncontrolled metabolic conditions).
- Any current or previous neurological condition that influenced the patients' ability to understand or to learn new information.
- Self-reported, psychiatric history, such as schizophrenia.
- Neuropathic conditions, such as Parkinson's disease, multiple sclerosis or stroke.
- Patients undergoing bilateral TJA.
- Patients undergoing primary TKA or THA not due to OA
- Patients undergoing revision TKA or THA
- An extended, out-of-town absence/vacation during the duration of the study, either prior to or after surgery.

3.2.3 Outcome measures

The outcome measures utilised at the different intervals during the study were structured and evidence-based tools commonly used in the literature (see Table 3-1)

Table 3-1: Overview of Outcome measures

Assessment Tool	Type	Tested
Socio-demographic questionnaire	Self-administered questionnaire	Socio-demographic information
Brief pain inventory	Self-administered questionnaire	Pain
WOMAC	Self-administered questionnaire	Functional status
Pain Catastrophising Scale	Self-administered questionnaire	Catastrophising
TSK-13 Scale	Self-administered questionnaire	Kinesiophobia

Table 3-2: Allotment of research duties

Procedure	Administrator
Cognitive-targeted exercise therapy	Researcher
Pain neuroscience education	Researcher
Pre- and post-operative rehabilitation	Local physiotherapist
Outcome measure testing	Self-administered questionnaires completion done by participant

The outcome measures used in this study were the following:

3.2.3.1 Socio-demographic questionnaire

The aim of this self-administered questionnaire, as designed by the researcher, was to obtain socio-demographic information from the participants. This included their age, gender, ethnicity, marital status, educational level and lifestyle information found in literature to possibly affect objectives of this study [see Appendix 5]. The information obtained by the socio-demographic questionnaire was verified against admission to hospital information which proved the information accurate and comparable. The socio-demographic questionnaire was pre-tested during the pilot study.

3.2.3.2 Brief Pain Inventory

The BPI is a short, self-administered questionnaire that was originally developed for use in patients experiencing pain due to cancer (Keller, Bann, Dodd, Schein, Mendoza and Cleeland, 2004). The BPI has however been validated for chronic conditions such as OA and low back pain, as well as for acute conditions that include pain post-surgery (Cleeland, 2009). The BPI have been linguistically validated for Afrikaans, Zulu, Xhosa, Dutch and German (Cleeland, 2016).

The BPI measures both pain intensity (severity) and the impact of pain on function, mood and social situations (interference) [see Appendix 7]. For severity scoring, the BPI assesses pain at its 'worst', 'least', 'average' and 'now'. Interference is recorded by measuring how much pain has interfered with seven daily activities. These include general activity, walking, work, mood, enjoyment of life, relations with others and sleep (Cleeland, 2009). The BPI is interpreted with questions three to six measuring severity and question nine (A – G) measuring interference. Questions one, two, seven and eight do not form part of the BPI calculations (Cleeland, 2009). The minimum total BPI score obtainable will be 0 and the maximum 110. The greater the value, the more the participant is suffering from pain.

The BPI has been reported to be a valid measurement instrument when determining the level of pain in individuals undergoing THA and TKA (Kapstad, Rokne and Stavem, 2010; Høvik, Winther, Foss and Gjeilo, 2016). When using the BPI in individuals undergoing TKA, a positive relationship between pain, function and even pain due to other conditions or factors not associated with the surgery have been found (Winterboer, Wittig-Wells, Higgins and Cumming, 2013). Internal consistency, regarding pain and its impact, indicated a value greater than 0.80 using the Cronbach's alpha in individuals undergoing THA. The validity and reliability in participants suffering from non-cancer pain showed good results with coefficient alphas measuring greater than 0.70 (Keller *et al.*, 2004). The BPI has also been found to have good construct, predictive and convergence validity for individuals suffering from OA (Mendoza, Mayne, Rublee and Cleeland, 2006). Good construct validity (Cronbach's $\alpha = 0.88$) was found when compared to the WOMAC and SF-36 in individuals undergoing THA (Kapstad *et al.*, 2010).

Non-completion of more than one answer was considered inadequate for statistical purposes or for the purpose of this study. Questions regarding medication were marked not applicable whenever the participant had stopped using pain medication; however, these questions were not taken into consideration when calculating pain severity or pain interference (Cleeland, 2009).

3.2.3.3 Western Ontario and McMaster Universities Arthritis Index

The WOMAC (Western Ontario and McMaster Universities) Arthritis Index (Bellamy, Buchanan, Goldsmith, Campbell and Stitt, 1988) for Pain, Stiffness and Physical Function is a self-administered questionnaire. The total WOMAC questionnaire is an instrument that has been validated to assess the physical function status in hip and knee OA individuals (Wolfe, 1999). Furthermore, the WOMAC has been validated for individuals undergoing TKA and has been validated across 17 different cultures, including South Africa, and 80 different languages, including Afrikaans (Dowsey and Choong, 2013).

The WOMAC consists of 24 items divided into 3 subscales, namely pain, stiffness and physical function [see Appendix 6]. The test questions are scored on a scale of 0 – 4, which correspond to the following: None (0), Mild (1), Moderate (2), Severe (3), and Extreme (4). The scores for each subscale are summarised, with a possible score range of 0 – 20 for Pain, 0 – 8 for Stiffness, and 0 – 68 for Physical Function. Higher scores on the WOMAC indicate worse pain, stiffness and functional limitations. A total WOMAC score out of a possible 96 – a sum of all the subscales – was used in this research.

The WOMAC was used in this research as it is a valid instrument for the measurement of the functional status in both TJA and chronic degenerative conditions, which include OA (Kahn *et al.*, 2013). The WOMAC test-retest reliability is satisfactory with regards to individuals suffering from OA with intra-class correlation coefficients (ICC) of .86, .68, and .89, respectively (Salaffi, Leardini, Canesi, Mannoni, Fioravanti, Caporali, Lapadula and Punzi, 2003). Intra-class correlation coefficients are reflective of random as well as systematic differences in assessed scores. The validity has been established through correlation with similar health related quality of life and functional status questionnaires namely the SF-36, the Lequesne algofunctional index and global health index in hip and knee OA individuals (Salaffi *et al.*, 2003; Santos, Andraus, Pires-Oliveira, Fernandes, Frâncica, Poli-Frederico, Fernandes, Santos, Andraus, Pires-Oliveira, Fernandes, Frâncica, Poli-Frederico and Fernandes, 2015). The WOMAC has high inter-correlation validity between subgroup ($p < 0.0001$) with the correlation between physical function and pain being the highest with a Spearman correlation of 0.824.

The minimum, clinical difference in compared WOMAC questionnaires that can be regarded as a clinical meaningful difference estimate has been described as an overall improved score of greater than 20 (Dowsey and Choong, 2013).

Non-completion of more than one answer was considered inadequate for statistical purposes or for the purposes of this study. Physical function questions were rated the maximum score if the participant had not done the activity on completing the WOMAC questionnaire, such as “Getting in and out of bath”, upon discharge from hospital.

3.2.3.4 Pain Catastrophising Scale

The PCS (Sullivan, 1995) is a 13-item, self-administered instrument that asks the participants to reflect on past painful experiences, and indicate the degree to which they experienced each of 13 thoughts or feelings when experiencing pain [see Appendix 8]. The PCS has been linguistically validated in more than 20 languages and a South African specific SA-PCS conducted on Afrikaans, English and Xhosa speaking individuals suffering from fibromyalgia have shown good validity (Morris, Grimmer-Somers, Louw and Sullivan, 2012).

Pain catastrophising experiences are indicated on a five-point scale with the end points (0), not at all, and (4), all the time. The PCS yields a total score out of 52 of the three subscale scores assessing rumination, magnification and helplessness. This is as follows:

- Rumination: Sum of items 8, 9, 10, 11
- Magnification: Sum of items 6, 7, 13
- Helplessness: Sum of items 1, 2, 3, 4, 5, 12

Rumination relates to the inability of the participant to stop thinking of the painful area or condition and thus this subsection aims to evaluate the attention the participant places on their condition. Increased catastrophising attention has been found to relate to increased pain-outcomes in numerous studies, including OA and post-surgery patients (Keefe, Kashikar-Zuck, Opiteck, Hage, Dalrymple and Blumenthal, 1996; Khan *et al.*, 2011). Magnification of pain relates to the participants fear of the seriousness of their condition currently and prospectively. In a study on TKA individuals magnification had shown to be a self-determining predictor of the intensity of chronic pain (Masselin-Dubois *et al.*, 2013). Helplessness relates to the inability of the participant to cope with their painful experience or condition. Improving coping skills in individuals undergoing TKA have shown to improve catastrophising and pain outcomes (Riddle *et al.*, 2011).

The PCS has been used to predict the measure of catastrophising in people developing chronic pain. Pain catastrophising was found a reliable indicator for pain to become chronic in individuals who had TKA (Burns, Ritvo, Ferguson, Clarke, Seltzer and Katz, 2015). The PCS has shown very good α -index scores of .87 and satisfactory internal consistency (range = .60 – .87) (Osman, Barrios, Kopper, Hauptmann, Jones and O’Neill, 1997). Construct validity has been found in relation to the fear avoidance believes questionnaire ($\rho = 0.34$) and the Hopkins symptom check list-25 ($\rho = 0.56$) using the Spearman’s ρ in individuals assessing the cross-cultural validity of the PCS (Fernandes, Storheim, Lochting and Grotle, 2012).

Research at the Centre for Research on Pain, Disability and Social Integration in Montreal, indicates that a total PCS score of 30 represents a clinically relevant level of catastrophising. A total PCS score of 30 also corresponds with the 75th percentile of the distribution of PCS scores in clinic samples of chronic pain patients (Sullivan, 1995). For the purpose of this research study, a total PCS score of 30 or above was considered as catastrophising. Non-completion of more than one answer was considered inadequate for statistical purposes in this study.

3.2.3.5 Tampa Scale

The Tampa Scale (TSK-13) (Burwinkle, Robinson and Turk, 2005) assesses kinesiophobia in participants [see Appendix 9]. The TSK-13 has been linguistically validated in multiple languages, including German, Afrikaans, English and Xhosa (Morris *et al.*, 2012).

The TSK is a 17-item, self-report checklist using a 4-point Likert scale as a measure of FOM and injury or re-injury. The Likert scale uses scores of individual items from 1 (strongly disagree) to 4 (strongly agree). Reversed scoring must be used for items 4, 8, 12 and 16. Several studies have consistently found poor loading of the reverse-scored items, and it has been recommended that these items be dropped from the questionnaire (Houben, Leeuw, Vlaeyen, Goubert and Picavet, 2005). Therefore, the reversed-scored items were not reported in the current study (TSK-13). For the purpose of this study, an added question separate from the TSK-13 was handed out. This question asked participants to document their most feared or most avoided, normal, everyday activity in a descending format from most feared to worrying.

The TSK-13 was used by Ryan *et al.* (2010) to compare the outcome of pain education with pain education and exercise classes in patients suffering from lower-back pain (Ryan *et al.*, 2010). Short term results favoured the pain education group compared to the group who received exercise classes. The TSK-13 has an internal consistency score range from α -index (.70 – .83.) and test-retest reliability ranges from $r(s) = 0.64$ to 0.80 (Swinkels-Meewisse, Swinkels, Verbeek, Vlaeyen and Oostendorp, 2003). The TSK-13 has proven to have moderate validity when compared to the fear avoidance believes questionnaire ($\rho = 0.59$) (Swinkels-Meewisse *et al.*, 2003).

The TSK-13 measurement index was determined by the value obtained from a minimum score of 13 to the maximum of 52. Participants who had a final score of greater than 37 were classed as having a high FOM and participants who scored 37 or lower were classed as having a low FOM (Dolphens *et al.*, 2014). Non-completion of more than one answer was considered inadequate for statistical purposes for the use in this study.

3.3 Pilot study

A pilot study was performed to identify and eliminate any potential problems and test the study procedure prior to the main study. Two participants were included in the pilot study. These participants resided in Swakopmund, Namibia, and two independent orthopaedic surgeons were requested to recruit these participants. Of the two participants, one was randomly assigned to the control group and the other to the intervention group. Signed informed consent was obtained before the pilot study commenced. The procedure, as explained in Figure 3-1, was followed during the pilot study to assess the feasibility of the proposed study procedure for the main study.

A local physiotherapist performed the SPRP according to pre-approved, musculoskeletal guidelines [see Appendix 10 and Appendix 11]. The PNE was provided separately to the individuals in the intervention and the control group by means of flashcards [see Appendix 12]. The participants' ability to understand and their willingness to ask questions were assessed. The CTET sessions of the intervention group took place at the individual's home. This was done to establish any factors that might influence consistency in the application of this protocol. The researcher decided not to use the local physiotherapist venue as to keep the physiotherapist blinded as to whom received CTET.

The results of the outcome measures were scored and charted to establish any gaps in the measurement or measurement scoring. After completion of the pilot study, no changes were made to the outcome measures, other than grammatical corrections or study procedures; therefore, the data of the study participants were included in the main research study. The grammatical changes made to the outcome measure forms were submitted to the Ethics Committee of the University of the Free State for approval.

3.4 Data collection and intervention procedure

This study commenced in December 2015 after all relevant approval was obtained [see Appendix 16 and Appendix 17]. The data collection concluded in October 2016.

The procedure was that the researcher was contacted by the orthopaedic surgeons once an individual was identified for THA or TKA. On referral from the orthopaedic surgeon, the patient was contacted telephonically to arrange a meeting to discuss the prospective study. The meetings took place either at the participant's home or at the participant's local physiotherapy practice. The meeting included a discussion in lay terms about the study.

Appendix 1 and Appendix 3] and consent [see Appendix 2 and Appendix 4] form was given to every potential participant to inform him or her about the study and the relevant, ethical considerations. Only patients who met the inclusion criteria and had completed the consent form took part in the study and received an appointment time for the baseline testing process. The baseline testing included the socio-demographic questionnaire as well as all the outcome measures (see 3.2.3, Table 3-1 and Table 3-2).

Stratified randomisation was employed to form two gender specific subgroups with regards to the TKA and THA participants. In order to prevent bias, the randomised process ensured that all participants had the same probability to be allocated to either the control or intervention group. The randomised list of who will be placed into the control or intervention group was computer-generated by the Department of Biostatistics at the University of the Free State. The list included a stratified description regarding the gender, as either V (female) or M (male), the joint receiving the arthroplasty, as either K (TKA) or H (THA) as well as the rank number for the TJA considering the gender. An example would be VH03, describing the third female undergoing THA. The participants did not undergo TJA surgery simultaneously but rather over a period of several months. Randomly the number one or two would follow the stratified description. The number one or two would indicate whether the participant would be allocated to the control or intervention group. The definition of this one or two would also differ for different stratified groups. For instance, one could indicate the control group for female THA individuals, while one on the randomisation list for male TKA could indicate the intervention group. This was done to prevent any selection bias. Furthermore, the researcher was the only person besides Biostatistics of the UFS that had access to the randomisation list.

The study procedure consisted of a three-week, exercise rehabilitation program focusing on the musculoskeletal system prior to surgery and continuing six weeks after surgery for all participants. A SPRP for THA and TKA adapted from Maxey was Magnusson (2007) was used (Maxey and Magnusson, 2007) [see Appendix 10 and Appendix 11]. The standard, musculoskeletal pre-rehabilitation was performed by the participants' local, qualified physiotherapist. Local physiotherapists who were involved in the implementation of the SPRP for joint arthroplasty [see Appendix 10 and Appendix 11] were blinded to the group to which the patient had been randomised in order to prevent bias influence or possible interference through altering exercises. The SPRP included lower limb and upper limb strength training, ROM exercises for knee mobility, gait retraining and cardiovascular exercises [see Appendix 10 and Appendix 11].

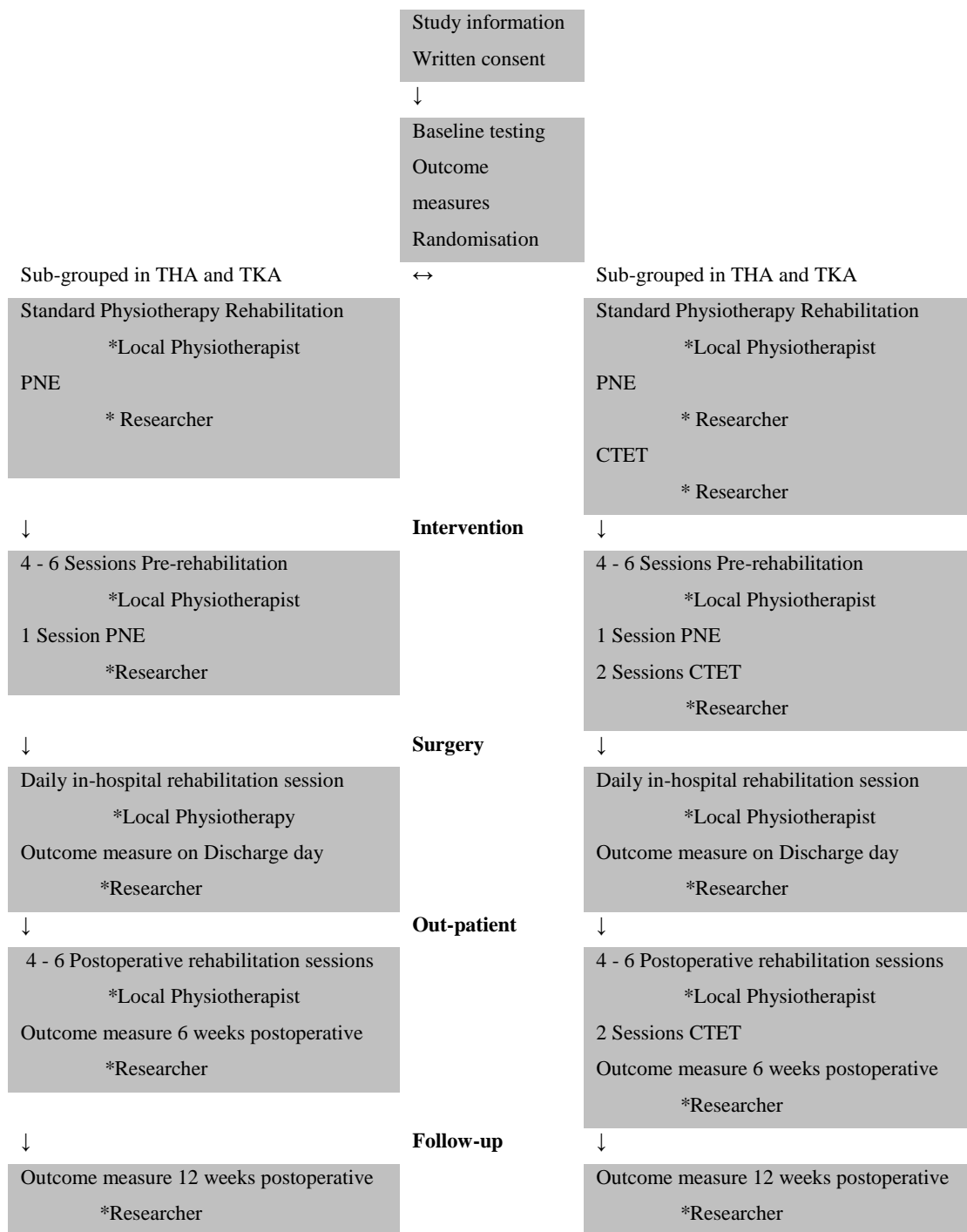
PNE [see Appendix 12] session was administered prior to surgery, using flashcards, at their home or at their local physiotherapist practice (see Table 3-2). An information pamphlet regarding PNE was handed-out to the participant to read at home [see Appendix 14]. The researcher undertook the role of pain neuroscience educator. Participants attended one individual one-to-one session prior to surgery, where evidence-based pain physiology, as well as central and peripheral sensitisation mechanisms were explained [see Appendix 12]. The PNE consisted of peripheral mechanisms involved in processing the noxious stimuli in either a manner that inhibits or excites the neurons. Furthermore, second-order neuron involvement in modulating the noxious perception as well as central mechanisms in mal-adaptive coping strategies were explained. The influence of different endocrine hormones in the chronic pain process was also explained through illustrations and practical examples [see Appendix 13]. After the session, time for questions were allocated. These included questions that the participants had around the application of PNE knowledge on their current condition. Participants were encouraged to ask questions during the course of the study by contacting the researcher. Each participant received a pamphlet, relating to the topics covered, serving as a self-study tool only, to read through at home [see Appendix 14].

CTET [see Appendix 15] were given to the intervention group by the researcher prior to surgery and at the six week post-surgical point (see Table 3-2 and Figure 3-1). The exercises for the intervention group consisted of graded, exposure exercises that aimed at improving the participant's confidence in a previously avoided action, due to fear, and thus also increasing the participant's knowledge of his or her abilities. The participants in the intervention group were required to attend their two 40-minute, CTET classes prior to surgery, but only after they had received the PNE. The CTET classes were held on two separate days, not more than three days apart at the individuals home. In the first session, the role of fear in the pain neuromatrix was explained in more detail [see Appendix 15]. The concept of time-contingent performance of exercises was compared with pain-guided and pain-barrier concepts. Goal setting took place according to the SMART Principle (Specific, Measurable, Achievable, Realistic and Time-targeted) for each participant individually, according to their TSK-13 score and the added question on the TSK-13 scale regarding feared or avoided activities. Cognitive-targeted exercises were then initiated.

The second visit focused solely on cognitive-targeted, exercises [see Appendix 15]. During the exercises, continual questioning and discussion monitored any fear of performing the exercise (see Figure 3-1). The progression of exercises was towards the participant's most fearful activity, as established by the added question to the TSK-13 scale, regarding fear or avoidance. Initiating the progressive exercises was preceded by 'motor imagery' of the activity. The final stage of the exercise therapy consisted of training the individual in his or her feared movement. Two cognitive-targeted exercise sessions were conducted prior to the 6-week, post-surgical period on two separate days [see Appendix 15]. As each participant's fears differed, every individual was trained regarding his or her uniquely feared movement, and only when the participant had reached this stage in the rehabilitation process was the CTET regarded as complete.

The overall study was thus conducted over a 15 week period for all participants. Outcome measures were repeated after surgery upon discharge from the hospital, as out-patients at six weeks and 12 weeks post-surgery (see Table 3-2). A diagram of the study procedure is shown in Figure 3-1.

Figure 3-1: Assessment and study procedure



3.5 Measurement and methodology errors

Although all the local physiotherapists adhered to a SPRP [see Appendix 10 and Appendix 11], variation might have occurred. A participant might have presented in a way that warranted added exercises. It was however expected that any deviation from the SPRP should be recorded. No deviations or addition of exercises were recorded by the physiotherapists. Withholding rehabilitation of the musculoskeletal system from participants who require additional attention is considered unethical. Attained improvement in individuals' outcome measures might be due to added exercises by the local physiotherapist to the SPRP and not by the CTET itself. The physical improvements were thus compared to TSK-13 scores to confirm any results.

The sampled group was only representative of a small population serviced by four towns in Namibia. Unfortunately, these four towns were the only communities in the Erongo region that had access to orthopaedic surgeons in Swakopmund, Walvis Bay, Wlotzkasbaken and Henties Bay. Only the Medi-clinic private hospital was used – thus the study sample only includes individuals able to pay privately or have a private medical aid, and therefore does not include state patients. There are however no other hospitals, whether private or state that do THA and TKA surgery in this region that covers 63,539 km².

Taking into account the level of pain physiology explained to the participants through the PNE, the reliability could have been affected by the participant not understanding the neuroscience behind pain. The psychological standpoint of, “the pain is in your head” could have been wrongly and negatively deduced. To decrease the possibility, PNE sessions were based on literature as advised by Moseley (2003), as well as Louw and Puentedura (2013). If the participant was unable to understand the pain education, it would have been clearly portrayed by the revised pain neurophysiology questionnaire administered after the education session (Moseley, 2003a; Louw and Puentedura, 2013)

Kinesiophobia specific exercises may cause increased fear for the participant before activity initiation (Wicksell *et al.*, 2010). To prevent this, the participants were only exposed to exercises to which they verbally consented, without danger, and were monitored by frequent and specific questioning regarding distress levels during the exercises. If any tension, relating to fear, persisted after the training had ceased, the participant's orthopaedic surgeon and general practitioner would be contacted.

The use of neurological or pain medication outside the prescribed time frame can be a possible threat to the validity of the results of this study. Provisional measures to counteract this included the information given and consent form signed by the participant [see

Appendix 1, Appendix 3, Appendix 2 and Appendix 4]. As part of the Brief Pain Inventory, the participants had to divulge the medications they were currently using.

The dependant variables for this study were the pain and functional levels, catastrophising and FOM status of the participants, with the independent variable being CTET. Confounding variables were SPRP, PNE and the TJA surgery.

Drop-outs could have influenced the study results; thus, prevention measures, such as that all participants understood the length of the study and that all participants were from Swakopmund or the surrounding towns namely Walvis Bay (35km), Wlotzkasbaken (32km) or Henties Bay (70km), were implemented. The study also ran in accordance with the normal rehabilitation and follow-up time frames of the orthopaedic surgeons to motivate compliance to the study procedures.

3.6 Ethical aspects

3.6.1 Ethics committee

This study protocol was approved by the Faculty of Health Sciences Research Ethics Committee, University of the Free State (ECUFS 172/2015), to ensure compliance with the ethical standards required to perform a clinical trial.

3.6.2 Informed consent

The participants had the right to decide whether or not to participate in the study and, additionally, to withdraw from the study at any time. Before consent was obtained, the participants received an information document [see Appendix 1 and Appendix 3] which explained the whole study procedure, and time for questions was allowed so that the potential participants could clarify any uncertainties. Thereafter all the willing participants signed the consent form [see Appendix 2 and Appendix 4], indicating that they understood the scope of the study and wanted to participate.

Due to the nature of the study, approval was also acquired from the private hospital and all orthopaedic surgeons involved [see Appendix 16 and Appendix 17]. All the participants were treated fairly and with the required respect by the researcher. To ensure high ethical standards, confidentiality was ensured by numbering the questionnaires and not collecting any identifiable information. The participants' right to privacy was also protected by not revealing their identities in the report. No participant under the age of 18 years was involved in this study, and the participants were assigned a numerical code to maintain confidentiality at all times. No personal data regarding medical aid or private institutions were used in public.

If the study intervention proves to have statistically and clinically significant, positive findings, it will be offered to future individuals undergoing THA or TKA surgery.

3.7 Data analysis

Baseline data were analysed in order to determine descriptive statistics for the different outcome measures for the control and intervention group. Comparability of the groups before the intervention was studied with the One-sample Kolmogorov-Smirnov test, for normality, and independent samples t-test, for comparing groups before the intervention took place. In testing for similarity the significance were set with a p-value equal to 0.05. In the case of both the Levene's test of variance and the t-test for equality of means a value greater than $p=0.05$ were denoted as having similarity. Associations between baseline parameters were examined to determine normality and difference. Due to the small sample size of this study, calculation of the assumption of normality was deemed important to calculate if the data has a normal distribution. Normal distribution is considered when the data tends to be centred to form a bell curve on a distribution graph, thus not bias towards the left or right. Normality testing is considered a pre-requisite to prove the validity of results presented, as it determines which statistical tests can be used. Non-parametric tests for instance do not rely on assumptions about the shape or form of the distribution from which the data were drawn, where parametric tests assume normal distribution of data. Parametric data where possible will be used as the level of accuracy is greater than non-parametric data. Furthermore, independence was not tested as the control and intervention group did not depend on each other, and thus were ipso facto independent.

Analysis of variance is used to determine the variation that exists between two mean results. Where normality and differences were found, possible changes in the outcome measures in response to the intervention was examined between the two groups by using repeated measures analysis of variance (ANOVA) with the parametric Greenhouse-Geisser test, with the intervention serving as the between-subjects factor. Within-subject results were not described as it did not assist the researcher to answer the research question of this study.

Regression analysis is used in statistics to determine if a predictor variable is effective in predicting an outcome variable. Therefore, regression analyses were used to determine the predictor, group membership, for the intervention outcome, and in particular which variable had a significant prediction for the intervention outcome. For all statistical tests, the significance level was set at 0.05, and 95% confidence intervals will be calculated for outcome measures with normal distributions. The format of ANOVA for regression is outlined in Appendix 21. Only when statistical significance is shown by the ANOVA for regression group result a multiple comparisons table will be shown that includes all stages of testing indicating statistical significance at each stage. The software SAS 9.4 and SPSS version 24 was utilised for analysis by the Department of Biostatistics, University of the Free State and Dr. M. A. E. Muller from the University of Namibia.

4 Chapter 4: Results Chapter

4.1 Introduction

This chapter contains the results obtained during this study which are, where applicable, illustrated by the use of tables. Firstly, the socio-demographic information results will be presented. Thereafter, the results obtained from outcome measures measuring pain, physical function, pain catastrophising and fear of movement, will be discussed.

4.2 Socio-demographic information

The socio-demographic information (see Table 4-1) regarding individuals that participated in this study was obtained by a socio-demographic questionnaire [see Appendix 5]. A total of 19 out of a possible 20 participants (95%) completed the study. One participant did not undergo surgery after being diagnosed with Hepatitis B.

Table 4-1 Socio-demographic information

	Control group (n=9)	Intervention group (n=10)
Gender	9 (47.4%)	10 (52.6%)
Male	4 (44%)	5 (50%)
TKA	2	3
THA	2	2
Female	5 (56%)	5 (50%)
TKA	3	3
THA	2	2
Age		
Median	54	63.5
Minimum	44	43
Maximum	75	85
Employment		
Pensioner	6 (67%)	5 (50%)
Employed	3 (33%)	5 (50%)
Unemployed	0	0
Ethnicity		
Caucasian	8 (89%)	8 (80%)
Coloured	1 (11%)	2 (20%)
Marital status		
Married	8 (89%)	8 (80%)
Widow	0	1 (10%)
Widower	0	0
Divorced	1 (11%)	1 (10%)
Risk factors		
Smoker	2 (22%)	2 (20%)
Diabetes	1 (11%)	3 (30%)
Heart conditions	0	0
Cholesterol	2 (22%)	2 (20%)

The participants were divided into two groups, namely a control (n=9) and an intervention group (n=10). In the control group, four participants were male (44%), whereas the intervention group consisted of five males (50%). The median age for the control group (54 years) was lower than that of the intervention group (63.5 years). The eldest participant in this study was 85 years of age and allocated to the intervention group. The minimum ages of both groups were similar (see Table 4-1) [see Appendix 5].

More of the participants in the control group (66.7%) than in the intervention group (50%) were employed. The majority of participants (89%) in this study were Caucasian. With regards to marital status the control (89%) and intervention (80%) groups were similar (see Table 4-1).

In both the control (22%) and intervention (20%) groups only two participants were smokers. When considering lifestyle diseases, 11% of participants in the control group suffered from diabetes mellitus type II (DM II) compared to 30% in the intervention group. In both groups the participants who had DM II were on medication. Of the participants in both the control (22%) and intervention (20%) groups only two participants presented with a lipid disorder (increased levels of blood cholesterol). The participants who had a lipid disorder were on medication, and their blood cholesterol levels were within normal parameters (see Table 4-1).

4.3 Pain measured pre- and post-arthroplasty

The BPI [see Appendix 7] was used to determine the participants' pain severity, and interference of pain on their functioning throughout the course of the study.

To be able to establish the ability to use parametric or non-parametric testing methods to describe the descriptive statistics, normality was tested at baseline stage using the Kolmogorov-Smirnov test (see Table 4-2).

The Kolmogorov-Smirnov test shows that at the baseline stage pain severity has a p-value 0.177 and for the baseline stage of pain interference p- value 0.200, which are both more than significance set at p=0.05 (see Table 4-2). Thus, the null hypothesis, that the data is normally distributed, cannot be rejected. The researcher proceeded to use parametric testing. Parametric testing has a greater degree of confidence compared to non-parametric testing.

Table 4-2 BPI – Test for normality at Baseline stage for both control and intervention group for BPI

One-Sample Kolmogorov-Smirnov Test BPI			
		Pain severity (n=19)	Pain interference (n=19)
		Baseline	Baseline
Normal Parameters^{a,b}	Mean	13.63	31.16
	Std. Deviation	7.380	13.997
Test Statistic		0.166	0.129
Asymp. Sig. (2-tailed) (p-value)		0.177^c	0.200^{c,d}

a. Test distribution is Normal.

b. Calculated from data.

c. Lilliefors Significance Correction.

d. This is a lower bound of the true significance.

* Statistically significant (p<0.05), Asymp. Sig. = Asymptotic Significance

The control and intervention group were tested at baseline using the independent sample t-test to ensure the two groups were the same with regard to pain severity and pain interference during this initial testing stage. The hypothesis tested was that the two groups had no difference at baseline. Table 4-3 represents the baseline descriptive statistics used for the independent samples t-test shown in Table 4-3 4-4.

Table 4-3 BPI - Baseline descriptive results for the two groups

Group statistics for similarity at baseline					
	Group	n	Mean	Std. Deviation	Std. Error Mean
Pain interference	Control	9	26.78	11.830	3.943
	Intervention	10	35.10	15.198	4.806
Pain severity	Control	9	12.00	4.183	1.394
	Intervention	10	15.10	9.398	2.972

Both p-values in the t-test for equality of means (0.204 and 0.376) were greater than 0.05, thus at baseline the control and intervention group did not differ significantly on pain interference or pain severity (see Table 4-4). Both Levene's test for equality of variances (0.768 and 0.092) were greater than $p=0.05$, thus also indicating similarity between groups. Thus, the data was found to be normally distributed and similar at baseline for pain severity and pain interference (see Table 4-2 and Table 4-4).

Table 4-4 BPI – Test for similarity in control and intervention group at baseline

Independent Samples Test at Baseline										
		Levene's Test for Equality of Variances		t-test for Equality of Means						
		F	Sig.	t	Df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
									Lower	Upper
Pain interference	Equal variances assumed	0.090	0.768	-1.320	17	0.204	-8.322	6.302	-21.619	4.975
Pain severity	Equal variances assumed	3.191	0.092	-0.910	17	0.376	-3.100	3.407	-10.289	4.089

F= Variation between sample variances , Sig.= Significance (p-value), t= Inferential statistic, df= Degrees of freedom

Possible changes in the pain severity and pain interference sub-sections in response to the intervention were examined between the two groups by using repeated measures ANOVA with the Greenhouse-Geisser test. The parametric data is described by use of means and standard deviation in the BPI sub-sections and the total BPI descriptive scores will include minimum and maximum values as well (see Table 4-5 and Table 4-6)

The mean pain severity of participants in both the control and intervention groups increased from baseline to hospital discharge and decreased from hospital discharge to 12 weeks post-surgery. The intervention group showed a decrease in pain severity from baseline at all stages until 12 weeks post-surgery. The total pain severity for the control and intervention group (n=19) indicated a slight increase in mean from baseline to hospital discharge, although the pain severity decreased to 12 weeks post-surgery. In the control group the mean pain severity decreased from baseline to 12 weeks post-surgery, from 12 to 4.44, and in the intervention group from 15.10 to 2.00 (see Table 4-5). The total combined pain severity for the control and intervention group (n=19) decreased from 13.63 at baseline to 3.16 at 12 weeks post-surgery.

Pain interference on function scores showed similar trends to the pain severity scores decreasing from baseline to 12 weeks post-surgery (see Table 4-5). In the control group the mean pain interference (26.78) increased from baseline to hospital discharge (31.44) and decreased from hospital discharge to 12 weeks post-surgery (6.56). In the intervention group the median pain interference decreased from baseline (35.10) to hospital discharge (32.30), only decreasing from baseline to 12 weeks post-surgery (3.60). The intervention group however had a higher mean at baseline (35.10) than the control group (26.78). The total pain interference for the control and intervention group (n=19) indicated a slight increase in mean from baseline to hospital discharge, although the pain interference decreased to 12 weeks post-surgery (see Table 4-5).

Table 4-5 BPI – Pain severity and pain interference on function descriptive results and effect size

	Pain severity						Pain interference					
Stage	Control group (n=9)		Intervention group (n=10)		Total (n=19)		Control group (n=9)		Intervention group (n=10)		Total (n=19)	
	Mean	Standard deviation	Mean	Standard deviation	Mean	Standard deviation	Mean	Standard deviation	Mean	Standard deviation	Mean	Standard deviation
Baseline	12.00	4.183	15.10	9.398	13.63	7.380	26.78	11.830	35.10	15.198	31.16	13.997
Hospital discharge	13.78	5.995	13.80	5.473	13.79	5.564	31.44	11.577	32.30	8.920	31.89	9.977
Six weeks post-surgery	7.11	4.167	5.30	3.093	6.16	3.655	14.22	10.010	12.40	10.844	13.26	10.208
Twelve weeks post-surgery	4.44	2.920	2.00	1.944	3.16	2.693	6.56	4.187	3.60	5.103	5.00	4.807
Effect size between groups	*0.002						*0.008					

*Effect size closer to 1 indicates a more important effect of the independent variable

The total BPI mean scores for the control group indicates an increase in pain from baseline to hospital discharge, where after the scores decrease to 12 weeks post-surgery. The intervention group however shows a decrease in total BPI mean scores from baseline to 12 weeks post-surgery with no increase at any stage of testing. The intervention group has a higher mean score (50.20) at baseline than the control group (38.78). Furthermore, the intervention group also has a lower mean total BPI score at 12 weeks post-surgery (5.60) than the control group (11.00). The total BPI descriptive scores for the control and intervention group are shown in Table 4-6.

Table 4-6 BPI -Total descriptive results per subdivision for both groups

Total BPI								
Stage	Control group (n=9)				Intervention group (n=10)			
	Minimum	Maximum	Mean	Standard deviation	Minimum	Maximum	Mean	Standard deviation
Baseline	11	58	38.78	14.193	20	105	50.20	24.008
Hospital discharge	10	62	45.22	14.805	31	76	46.10	13.127
Six weeks post-surgery	12	51	21.33	11.937	1	44	17.70	13.760
Twelve weeks post-surgery	4	23	11.00	6.910	0	21	5.60	6.883

To determine if the changes in pain as measured by the BPI had any statistical significance a test for between-subject effects was done, with the intervention serving as the between-subjects factor (see Table 4-7 and Table 4-8). The regression analysis output is shown in Table 4-7. The intervention did not show significance for Group in the regression ($p=0.865$) and thus no statistical significant difference were observed in pain severity between the control and intervention group at any stage during the study.

Table 4-7 BPI – Pain severity between groups results

Tests of Between-Subjects Effects					
Pain severity					
Source	Type III Sum of Squares	Df	Mean Square	F	Sig. (p-value)
Group	1.521	1	1.521	0.030	0.865

F= Variation between sample means , Sig.= Significance, df= Degrees of freedom

The regression analysis output is shown in Table 4-8. The intervention did not show significance for Group in the regression ($p=0.711$) and thus no statistical significant difference were observed in pain interference between the control and intervention groups at any stage during the study.

Table 4-8 BPI - Pain interference between groups results

Tests of Between-Subjects Effects					
Pain interference					
Source	Type III Sum of Squares	df	Mean Square	F	Sig. (p-value)
Group	22.926	1	22.926	0.142	0.711

Both the control and intervention group reported significant improvement from baseline to six weeks post-surgery in pain severity when the data was compared within each group (see Table 4-9). This statistical significant improvement continued when baseline results were compared to 12 weeks post-surgery results for pain severity. Furthermore, both groups reported statistical significant improvements in pain severity between six weeks post-surgery and 12 weeks post-surgery when comparing within group data (see Table 4-9).

The control and intervention group both indicated to have statistical significant improvement in pain interference when baseline and 12 weeks post-surgery results were compared within groups (see Table 4-9). Again, pain interference was found to be statistical significantly improved when six weeks post-surgery scores were compared with 12 weeks post-surgery (see Table 4-9).

Table 4-9 BPI – Within group results control and intervention group

Paired Samples Test			
Pain interference			
Time period	Group	Pain severity	Pain interference
Baseline vs. Hospital Discharge	Control group	0.493	0.483
	Intervention group	0.650	0.628
Baseline vs. Six weeks post-surgery	Control group	*0.038	0.102
	Intervention group	*0.002	0.628
Baseline vs. Twelve weeks post-surgery	Control group	*0.003	*0.004
	Intervention group	*0.001	*0.001
Six weeks post-surgery vs. Twelve weeks post-surgery	Control group	*0.007	*0.018
	Intervention group	*0.001	*0.001

Significant p-value (p<0.05)

4.4 Physical functional measured pre- and post-arthroplasty

The overall score of the WOMAC questionnaire [Appendix 6] was used to determine the participants' functional status throughout this study.

To be able to establish the ability to use parametric or non-parametric testing methods to describe the descriptive statistics, normality was tested. Testing for normality in the WOMAC questionnaire results were done using the Kolmogorov-Smirnov test (see Table 4-10). The hypothesis tested is that the data is normally distributed.

The Kolmogorov-Smirnov test showed that the total WOMAC scores at baseline had a p-value of 0.200, which is greater than significance set at $p=0.05$ and thus the data was normally distributed.

Table 4-10 WOMAC – Test for normality (both control and intervention group)

One-Sample Kolmogorov-Smirnov Test WOMAC		
		Baseline (n=19)
Normal Parameters ^{a,b}	Mean	49.68
	Std. Deviation	20.388
	Test Statistic	0.086
Asymp. Sig. (2-tailed) (p-value)		0.200^{c,d}

a. Test distribution is Normal.

b. Calculated from data.

c. Lilliefors Significance Correction.

d. This is a lower bound of the true significance.

Asymp. Sig. = Asymptotic Significance

The control and intervention group were tested for similarity at baseline using the independent samples t-test to ensure the two groups were the same during this initial testing stage. The hypothesis tested was that the two groups had no difference at baseline. The baseline descriptive statistics used for the independent samples t-test are shown in Table 4-11.

Table 4-11 WOMAC - Baseline descriptive results

Group statistics for similarity at baseline Total WOMAC (n=19)				
Group	n	Mean	Std. Deviation	Std. Error Mean
Control	9	39.11	13.824	4.608
Intervention	10	56.50	21.173	6.695

Although the sub-scales of the WOMAC were not used separately in this study, the sub-scales were calculated independently to determine if the scores obtained within the WOMAC was similar at baseline between groups.

All the p-values of the independent t-tests were smaller than 0.05, except for pain ($p=0.155$) thus at baseline the control and intervention group were not the same on stiffness, function and the total WOMAC scores (see Table 4-12).

Table 4-12 WOMAC - Test for similarity in control and intervention group at baseline

Independent Samples Test at Baseline WOMAC										
		Levene's Test for Equality of Variances		t-test for Equality of Means						
		F	Sig.	T	df	Sig. (2-tailed) (p-value)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
									Lower	Upper
Pain	Equal variances assumed	0.305	0.588	-1.487	17	0.155	-2.767	1.861	-6.693	1.160
Stiffness	Equal variances assumed	7.635	*0.013	-2.373	17	*0.030	-2.367	.997	-4.471	-.262
Function	Equal variances assumed	0.005	0.944	-2.398	17	*0.028	-14.744	6.149	-27.717	-1.772
Total WOMAC	Equal variances assumed	0.634	0.437	-2.381	17	*0.029	-19.878	8.347	-37.488	-2.267

*Statistical significant (p<0.05), F= Variation between sample variances , Sig.= Significance, t= Inferential statistic, df= Degrees of freedom

Possible changes in the pain severity and pain interference sub-sections in response to the intervention were examined between the two groups by using repeated measures ANOVA with the Greenhouse-Geisser test (see Table 4-14). The parametric data is described by use of minimum, maximum, means and standard deviation (see Table 4-13).

The mean WOMAC score in the control group increased from baseline (39.22) to hospital discharge (54.22) and decreased from hospital discharge to 12 weeks post-surgery (18.00). The intervention group showed a decrease in their WOMAC score from baseline (59.10) at all stages until 12 weeks post-surgery (9.10) (see Table 4-13). The total combined WOMAC for the control and intervention group (n=19) indicate an increase in mean between baseline (49.68) and hospital discharge (54.89). There was a consistent decrease in mean total combined WOMAC scores from hospital discharge to 12 weeks post-surgery (13.32) (see Table 4-13).

Table 4-13 WOMAC- Descriptive results for both groups as well as the total combined score and effect size

WOMAC descriptive scores (Maximum 96)												
Stage	Control group (n=9)				Intervention group (n=10)				Total combined WOMAC score (n=19)			
	Minimum	Maximum	Mean	Standard deviation	Minimum	Maximum	Mean	Standard deviation	Minimum	Maximum	Mean	Standard deviation
Baseline	16	59	39.22	15.865	32	95	59.10	19.991	16	95	49.68	20.388
Hospital discharge	38	74	54.22	11.508	47	74	55.50	8.784	38	74	54.89	9.893
Six weeks post-surgery	9	44	28.00	11.467	16	40	27.20	7.927	9	44	27.58	9.489
Twelve weeks post-surgery	4	38	18.00	12.114	5	14	9.10	3.755	4	38	13.32	9.650
Effect size between groups									*0.160			

*Effect size closer to 1 indicates a more important effect of the independent variable

To determine if the changes in physical function as measured by the WOMAC questionnaire had any statistical significance a test for between-subject effects was done, with the intervention serving as the between-subjects factor (see Table 4-14).

The regression analysis output is shown in Table 4-14. The intervention did not show significance for Group in the regression ($p=0.467$) and thus no statistical significant difference was observed in physical function between the control and intervention group at any stage during the study.

Table 4-14 WOMAC – Between group results

Tests of Between-Subjects Effects					
Total WOMAC					
Source	Type III Sum of Squares	df	Mean Square	F	Sig. (p-value)
Group	155.404	1	155.404	0.553	0.467

F= Variation between sample means, Sig.= Significance, t= Inferential statistic, df= Degrees of freedom

Both the control and intervention group had statistical significant improvement within their groups in their total WOMAC scores from baseline to six and 12 weeks post-surgery (see Table 4-15). The intervention group however had a substantially low p-value of 0.001 indicating a very strong statistical significant score. The intervention group also indicated to have a statistical significant difference in results between six and 12 weeks post-surgery (see Table 4-15).

Table 4-15 WOMAC – Within group results for control and intervention group

Paired Samples Test			
Total WOMAC			
Time period	Group	n	Sig. (2-tailed)
Baseline vs. Hospital Discharge	Control group	n=9	0.520
	Intervention group	n=10	0.618
Baseline vs. Six weeks post-surgery	Control group	n=9	*0.042
	Intervention group	n=10	*0.004
Baseline vs. Twelve weeks post-surgery	Control group	n=9	*0.018
	Intervention group	n=10	*0.001
Six weeks post-surgery vs. Twelve weeks post-surgery	Control group	n=9	0.062
	Intervention group	n=10	*0.002

*Significant p-value (p<0.05)

4.5 Catastrophising about pain pre-and post-arthroplasty

Catastrophising about pain was tested using the PCS (Sullivan, 1995), which consists of three sub-sections, namely rumination, magnification and helplessness [see Appendix 8].

To be able to establish the ability to use parametric or non-parametric testing methods to describe the descriptive statistics, normality was tested. Testing for normality for the PCS results were done using the Kolmogorov-Smirnov test (see Table 4-16). The hypothesis tested is that the data is normally distributed.

The Kolmogorov-Smirnov test shows that at all three subsections of the PCS have p-values greater than 0.05 (see Table 4-16). The data for the two groups were thus normally distributed.

Table 4-16 PCS – Test for normality at baseline for control and intervention group

One-Sample Kolmogorov-Smirnov Test PCS				
Baseline				
		Helplessness	Magnification	Rumination
Normal Parameters ^{a,b}	Mean	9.74	4.32	7.63
	Std. Deviation	7.901	3.606	5.737
Test Statistic		0.169	0.137	0.150
Asymp. Sig. (2-tailed) (p-value)		0.159^c	0.200^{c,d}	0.200^{c,d}

a. Test distribution is Normal.

b. Calculated from data.

c. Lilliefors Significance Correction.

d. This is a lower bound of the true significance.

* Statistically significant (p<0.05), Asymp. Sig. = Asymptotic Significance

The control and intervention group were tested at baseline for similarity using the independent sample t-test to ensure the two groups were the same with regard to helplessness, magnification and rumination during this initial testing stage. The hypothesis tested was that the two groups had no differences at baseline. Table 4-15 represents the baseline descriptive statistics used for the independent samples t-test shown in Table 4-17.

Table 4-17 PCS - Baseline descriptive results for the two groups

Group statistics for similarity at baseline					
	Group	n	Mean	Std. Deviation	Std. Error Mean
Helplessness	Control	9	9.33	7.348	2.449
	Intervention	10	10.0	8.749	2.767
Magnification	Control	9	4.22	3.598	1.199
	Intervention	10	4.40	3.806	1.204
Rumination	Control	9	5.78	4.944	1.648
	Intervention	10	9.30	6.129	1.938

The p-values for the t-tests for helplessness (0.840), magnification (0.918) and rumination (0.189) were greater than 0.05, thus at baseline the control and intervention group did not differ significantly on helplessness, magnification and rumination at the baseline stage (see Table 4-18). Furthermore, Levene’s test for equality of variances (0.0261, 0.973 and 0.191) were greater than $p=0.05$, thus also indicating similarity between groups.

Table 4-18 PCS – Test for similarity in control and intervention group at baseline

Independent Samples Test at Baseline (n=19)										
		Levene's Test for Equality of Variances		t-test for Equality of Means						
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
									Lower	Upper
Helplessness	Equal variances assumed	1.353	0.261	-1.320	17	0.840	-0.767	3.731	-8.638	7.105
Magnification	Equal variances assumed	0.001	0.973	-0.104	17	0.918	-0.178	1.705	-3.774	3.418
Rumination	Equal variances assumed	1.854	0.191	-1.368	17	0.189	-3.522	2.574	-8.954	1.909

F= Variation between sample variances , Sig.= Significance, t= Inferential statistic, df= Degrees of freedom

Possible changes in the pain severity and pain interference sub-sections in response to the intervention were examined between the two groups by using repeated measures ANOVA with the Greenhouse-Geisser test. The parametric data is described by use of means and standard deviation in the PCS sub-sections. (Table 4-617).

The mean score in the control group for both the helplessness and magnification sub-sections improved from baseline (helplessness 9.33 and magnification 4.22) to hospital discharge (helplessness 4.22 and magnification 2.33) with rumination (5.78) scoring the same during the same time period. All sub-sections progressively decreased from hospital discharge to 12 weeks post-surgery (helplessness 0.89, magnification 0.44, rumination 1.78). The intervention group showed a decrease in their all the sub-sections from baseline (helplessness 10.10, magnification 4.40 and rumination 9.30) at all stages until 12 weeks post-surgery (helplessness 0.30, magnification 0.20 and rumination 0.90).

Table 4-19 PCS – Descriptive results for both groups at the different stages and effect size

	Helplessness				Magnification				Rumination			
Stage	Control group (n=9)		Intervention group (n=10)		Control group (n=9)		Intervention group (n=10)		Control group (n=9)		Intervention group (n=10)	
	Mean	Standard deviation	Mean	Standard deviation	Mean	Standard deviation	Mean	Standard deviation	Mean	Standard deviation	Mean	Standard deviation
Baseline	9.33	7.348	10.10	8.749	4.22	3.598	4.40	3.806	5.78	4.944	9.30	6.129
Hospital discharge	5.44	5.790	5.70	5.187	2.33	2.958	2.00	1.826	5.78	4.177	7.30	4.762
Six weeks post-surgery	2.44	3.358	3.10	4.383	1.00	0.707	1.20	1.989	3.22	2.682	3.50	3.779
Twelve weeks post-surgery	0.89	1.269	0.30	0.675	0.44	0.726	0.20	0.632	1.78	2.167	0.90	1.595
Effect size between groups	*0.001				*0.001				*0.043			

*Effect size closer to 1 indicates a more important effect of the independent variable

To determine if the changes in helplessness, magnification and rumination as measured by the PCS had any statistical significance a test for between-subject effects was done, with the intervention serving as the between-subjects factor (see Table 4-20, Table 4-21 and Table 4-23).

The regression analysis output is shown in Table 4-718. The intervention did not show significance for Group in the regression ($p=0.876$) and thus no statistical significant difference were observed in helplessness between the control and intervention group at any stage during the study.

Table 4-20 PCS – Helplessness between groups results

Tests of Between-Subjects Effects					
Helplessness					
Source	Type III Sum of Squares	df	Mean Square	F	Sig. (p-value)
Group	1.404	1	1.404	0.025	0.876

F= Variation between sample means , Sig.= Significance, df= Degrees of freedom

The regression analysis output is shown in Table 4-8. The intervention did not show significance for Group in the regression ($p=0.951$) and thus no statistical significant difference were observed in magnification between the control and intervention groups at any stage during the study.

Table 4-21 PCS – Magnification between groups results

Tests of Between-Subjects Effects					
Magnification					
Source	Type III Sum of Squares	df	Mean Square	F	Sig. (p-value)
Group	0.047	1	0.047	0.004	0.951

The regression analysis output is shown in Table 4-820. The intervention did not show significance for Group in the regression ($p=0.397$) and thus no statistical significant difference were observed in rumination between the control and intervention groups at any stage during the study.

Table 4-20 PCS – Rumination between groups results

Tests of Between-Subjects Effects					
Rumination					
Source	Type III Sum of Squares	df	Mean Square	F	Sig. (p-value)
Group	23.393	1	23.393	0.756	0.397

When the groups were compared with themselves, the intervention group indicated an statistical significant improvement from baseline to six weeks post-surgery, as well as from baseline to 12 weeks post-surgery for rumination (see Table 4-22). Furthermore, there was a statistical significant improvement in rumination scores from six weeks post-surgery to 12 weeks post-surgery for the intervention group when results were compared within the group. At no stage did the control group show a statistical significant improvement in results for rumination (see Table 4-22).

Both the control and intervention group reported statistical significant improvements in magnification from baseline to six weeks post-surgery as well as from baseline to 12 weeks post-surgery (see Table 4-22). The intervention group was the only group found to have a statistical significant improvement in magnification between baseline and hospital discharge. The control did show a statistical significant improvement in magnification scores from six weeks post-surgery to 12 weeks post-surgery (see Table 4-22).

Both the control and intervention group showed statistical significant improvement in baseline helplessness scores compared to both six weeks post-surgery as well as 12 weeks post-surgery scores when the groups were compared from within (see Table 4-22). The intervention group was the only group which indicated to have a statistical significant improvement in helplessness from six weeks post-surgery to 12 weeks post-surgery.

Table 4-22 PCS – Within group result control and intervention group

Paired Samples Test					
PCS					
Time period	Group	n	Rumination	Magnification	Helplessness
Baseline vs. Hospital Discharge	Control group	n=9	1.000	0.142	0.152
	Intervention group	n=10	0.343	*0.039	0.076
Baseline vs. Six weeks post-surgery	Control group	n=9	0.262	*0.017	*0.036
	Intervention group	n=10	*0.006	*0.004	*0.008
Baseline vs. Twelve weeks post-surgery	Control group	n=9	0.073	*0.009	*0.007
	Intervention group	n=10	*0.001	*0.004	*0.005
Six weeks post-surgery vs. Twelve weeks post-surgery	Control group	n=9	0.083	*0.013	0.154
	Intervention group	n=10	*0.028	0.063	*0.050

*Significant p-value (p<0.05)

4.6 Fear of movement pre-and post-arthroplasty

Participants' FOM was assessed by the TSK-13 (Burwinkle *et al.*, 2005) [see Appendix 9].

To be able to establish the ability to use parametric or non-parametric testing methods to describe the descriptive statistics, normality was tested at baseline level. Testing for normality for the PCS results were done using the Kolmogorov-Smirnov test (see Table 4-23).

The Kolmogorov-Smirnov test showed that the TSK-13 scale for kinesiophobia scores at baseline had a p-value = 0.2 and the scores are thus normally distributed at the baseline stage of testing (see Table 4-23).

Table 4-23 TSK-13 – Test for normality at baseline for the control and intervention group

One-Sample Kolmogorov-Smirnov Test TSK-13		
		Baseline
Normal Parameters ^{a,b}	Mean	34.79
	Std. Deviation	8.046
Test Statistic		0.155
Asymp. Sig. (2-tailed) (p-value)		0.200^{c,d}

a. Test distribution is Normal.

b. Calculated from data.

c. Lilliefors Significance Correction.

d. This is a lower bound of the true significance.

* Statistically significant ($p < 0.05$), Asymp. Sig. = Asymptotic Significance

The control and intervention group were tested at baseline for similarity using the independent samples t-test to ensure the two groups were the same during this initial testing stage. The hypothesis tested was that the two groups had no difference at baseline. The baseline descriptive statistics used are shown in Table 4-24.

Table 4-24 TSK-13 – Baseline descriptive results

Group statistics for similarity at baseline Total TSK-13 (n=19)				
Group	n	Mean	Std. Deviation	Std. Error Mean
Control	9	34.44	8.141	2.714
Intervention	10	35.10	8.386	2.652

There was no difference between the control and intervention group with regard to TSK-13 at the baseline stage ($p=0.865$) (see Table 4-25). Furthermore, Levene’s test for equality of variances (0.484) were greater than $p=0.05$, thus also indicating similarity between groups.

Table 4-25 TSK-13 - Test for similarity in control and intervention group at baseline

Independent Samples Test at Baseline TSK-13										
		Levene's Test for Equality of Variances		t-test for Equality of Means						
		F	Sig.	t	Df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
									Lower	Upper
Baseline	Equal variances assumed	0.484	0.496	-0.172	17	0.865	-0.656	3.801	-8.674	7.363

F= Variation between sample variances , Sig.= Significance, t= Inferential statistic, df= Degrees of freedom

Possible changes in the pain severity and pain interference sub-sections in response to the intervention were examined between the two groups by using repeated measures ANOVA with the Greenhouse-Geisser test. The parametric data is described by use of means and standard deviation (see Table 4-26).

The mean scores for FOM according to the TSK-13 scale decreased from baseline to 12 weeks post-surgery in both the control and the intervention group. The control group TSK-13 scores decreased from 34.44 to 23.78, while the intervention group decreased from 35.10 to 23.00 (See Table 4-26). The total TSK-13 scale scores decreased consistently from baseline (34.79) to 12 weeks post-surgery (23.37).

Table 4-26 TSK-13 – Total descriptive results and effect size

TSK-13 descriptive scores (Maximum 52)												
Stage	Control group (n=9)				Intervention group (n=10)				Total TSK-13 score (n=19)			
	Min	Max	Mean	Standard deviation	Min	Max	Mean	Standard deviation	Min	Max	Mean	Standard deviation
Baseline	22	49	34.44	8.141	24	50	35.10	8.386	22	50	34.79	8.046
Hospital discharge	20	45	29.11	7.390	21	40	28.70	6.038	20	45	28.89	6.523
Six weeks post-surgery	14	37	24.11	8.433	18	33	25.30	5.034	14	37	24.74	6.682
Twelve weeks post-surgery	13	30	23.78	5.890	17	27	23.00	3.432	13	27	23.37	4.633
Effect size between groups									*0.001			

*Effect size closer to 1 indicates a more important effect of the independent variable

To determine if the changes in FOM as measured by the TSK-13 scale had any statistical significance a test for between-subject effects was done, with the intervention serving as the between-subjects factor (see Table 4-27).

The regression analysis output is shown in Table 4-27. Group was not significant in the model thus the intervention did not show significance in the regression ($p=0.950$) and thus no statistical significant difference were observed in FOM between the control and intervention groups at any stage during the study.

Table 4-27 TSK-13 – Between group results

Tests of Between-Subjects Effects					
TSK-13					
Source	Type III Sum of Squares	df	Mean Square	F	Sig (p-value).
group	0.509	1	0.509	0.004	0.950

F= Variation between sample means , Sig.= Significance, t= Inferential statistic, df= Degrees of freedom

The control and the intervention group had statistical significant decreased FOM when baseline scores were compared to six weeks post-surgery, as well as 12 weeks post-surgery scores (see Table 4-28). The control group indicated a statistical significant improvement in FOM from baseline to hospital discharge.

Table 4-28 TSK-13 – Within group results control and intervention group

Paired Samples Test			
TSK-13			
Time period	Group	n	p-value
Baseline vs. Hospital Discharge	Control group	n=9	*0.044
	Intervention group	n=10	0.058
Baseline vs. Six weeks post-surgery	Control group	n=9	*0.001
	Intervention group	n=10	*0.007
Baseline vs. Twelve weeks post-surgery	Control group	n=9	*0.001
	Intervention group	n=10	*0.001
Six weeks post-surgery vs. Twelve weeks post-surgery	Control group	n=9	0.816
	Intervention group	n=10	0.051

*Significant p-value (p<0.05)

Table 4-29 Summary of study data

Summary of study data													
Measures	n (%)	Control				Intervention				P- value			
		Base (SD)	HD (SD)	6Wk (SD)	12Wk (SD)	Base (SD)	HD (SD)	6Wk (SD)	12Wk (SD)	Base (SD)	HD (SD)	6Wk (SD)	12Wk (SD)
Demographic													
Age		54				63.5							
Gender	52.6% female 47.4% male												
Risk factors													
Smoking	21%	2				2							
Diabetes	21%	1				3							
Heart condition	0%	0				0							
Cholesterol	21%	2				2							
Employment													
Pensioner	58%	6				5							
Employed	42%	3				5							
Unemployed	0%	0				0							

Ethnicity										
Caucasian	84%		8			8				
Coloured	16%		1			2				
Marital status										
Married	84%		8			8				
Widow	5%		0			1				
Widower	0%		0			0				
Divorced	11%		1			1				
Pain (110)		38.78	45.22	21.33	11.00	50.2	46.10	17.70	5.60	
Pain severity (40)		12.00	13.78	7.11	4.44	15.00	13.80	5.30	2.00	0.865
Pain interference (70)		26.78	31.44	14.22	6.56	35.10	32.30	12.40	3.60	0.711
Physical function (96)		39.22	54.22	28.00	18.00	59.10	55.50	27.20	9.10	0.467
Catastrophising (52)										
Rumination (16)		5.78	5.78	3.22	1.78	9.30	7.30	3.50	0.90	0.397
Magnification (12)		4.22	2.33	1.00	0.44	4.40	2.00	1.20	0.20	0.951
Helplessness (24)		9.33	5.44	2.44	0.89	10.10	5.70	3.10	0.30	0.876
Fear of movement (52)		34.44	29.11	24.11	23.78	35.10	28.70	25.30	23.00	0.950

SD (Standard deviation); Base (Baseline); HD (Hospital Discharge); 6Wk (Six weeks post-surgery); 12Wk (Twelve weeks post-surgery)

5 Chapter 5: Discussion

5.1 Introduction

In this chapter the results obtained by this stratified randomised controlled trial regarding the efficacy of a SPRP, including PNE, compared to a SPRP including PNE and CTET on pain, function, FOM and pain catastrophising in patients undergoing TJA, will be discussed in full. The discussion of the results will follow the outline used in the results chapter. The findings will be discussed in relation to current related literature to indicate their relevance. Possible limitations of this study will also be discussed. Furthermore, a conclusion will be given with regards to the findings of the study, as well as recommendations for future study in this field.

5.2 Socio-demographic information regarding individuals that underwent TKA and THA

The socio-demographic questionnaire demonstrated that more females than males participated in the study (see Table 4-1). In an international survey by Kurtz *et al.* (2011) women constitute between 50% – 67% of patients who undergo THA. The representation of females in the study (see Table 4-1) falls within the normal representation found in literature (Beaupre, Lier, Davies and Johnston, 2004; Kurtz *et al.*, 2011). In a study seeking to establish the effects of education and exercise on functional abilities and quality of life prior to primary TKA, females were represented by 54.5% of the study population (Beaupre *et al.*, 2004). Which is also similar to the representation of females in this study.

In individuals suffering from OA, females have shown to experience more pain and pain catastrophising than males (Keefe, Lefebvre, Egert, Affleck, Sullivan and Caldwell, 2000). More specifically rumination, thinking about pain, and helplessness, an inability to cope with pain, has been found to be the components of catastrophising that are increased in females (Sullivan, Tripp and Santor, 2000). Due to the representation of females in this study, results obtained are comparable to literature with regards to gender and gender discrepancies in pain, rumination and helplessness.

The female representation within the TKA and THA subgroups in the control and intervention group should be considered even though the study is comparable to literature. Females were represented equally in the control and intervention group with regards to TKA and THA subgroups (see Table 4-1). Therefore, both the control and intervention group were affected equally, with regards to pain and pain catastrophising, due to the number of female participants in this study. Furthermore, male participants were equally distributed within the THA subgroup in this study. The study did however have one more male participant in the intervention group than in the control group when considering the TKA subgroup. The intervention group would then benefit from possible lower pain, rumination and helplessness scores due to a higher male representation (Keefe *et al.*, 2000; Sullivan *et al.*, 2000).

In this study the individuals that underwent THA ranged in age from 43 – 85 years of age, while those that underwent TKA ranged from 49 – 69 years of age (see Table 4-1). Judge *et al.* (2010) found the individuals in the greatest need of THA to be between ages 60 – 84 years of age, with a more recent study by Foran (2015) showing the age group that undergoes the most TKA as between 50 – 80 years of age. The median age of the intervention and control group fell within this age range for both the TKA and THA subgroups (see Table 4-1). The THA group however was represented by younger individuals than what is indicated by literature namely 43 and 44 years of age. The population group that undergo THA are decreasing in age according to a recent study, with 25% of individuals that undergo THA aged between 15 – 45 years of age in Scandinavian countries (Kurtz *et al.*, 2009; Artama, Skyttä, Huhtala, Leino, Kuitunen and Eskelinen, 2016). Up to 21% of individuals that undergo THA are younger than 55 years of age in South Africa (de Vos, Schepers and Ngcelwane, 2016). Both these young individuals fell within the male THA subgroup, although separated within the intervention (43 year old) and the control group (44 year old). The distribution therefore affects the control and intervention group results in an equal manner and could thus not be a determining factor in differences of findings.

The control group was represented mostly by individuals receiving pension (67%). The intervention group had an equal number of actively employed and individuals receiving pension (see Table 4-1). Barrack *et al.* (2014) indicate that individuals with low income have worse results regarding satisfaction and functional limitations when undergoing TKA. Similar results with regards to worse pain and functional outcomes in THA were obtained in individuals who were unemployed (Schäfer, Krummenauer, Mettelsiefen, Kirschner and Günther, 2010). The income status of the individuals did not form part of the socio-demographic questionnaire of this study, although all the individuals had medical aid or paid for the TJA privately. Thus, all individuals underwent the TJA with sufficient financial support to cover the hospital and rehabilitation expenses. None of the participants were unemployed in this study. In the study done by Schäfer *et al.* (2010) 54% of the individuals were pensioners compared to 57.80% of individuals in this study (see Table 4-1). Schäfer *et al.* (2010) did not show that persons receiving pension had an increased risk of not responding favourably to THA. Therefore, the high presentation of individuals receiving pension could not have influenced the study results.

The Caucasian ethnic group had the highest representation in this study (84%) (see Table 4-1). This is supported by literature that shows that Caucasian individuals are up to 66% more likely to undergo TJA than other racial groups (Ibrahim, 2010). There are however no literature available regarding the ethnic representation of TJA in South Africa. Caucasian individuals have shown to have improved pain and function scores after TJA (Lavernia, Alcerro, Contreras and Rossi, 2011). The results obtained in this study regarding pain and physical function could, therefore, be positively influenced by the high representation of Caucasian individuals. Both groups however had the same number of Caucasian individuals in each group and could thus not be considered a cause for any differences in results obtained in this study.

Married individuals were in the majority, with both the control and intervention group having eight such individuals (see Table 4-1). It has been shown that married individuals respond more positively to THA surgery when compared to single and widowed individuals (Schäfer *et al.*, 2010). This could be a factor in the positive results obtained in this study. However, because the number of married individuals were equal in the control and intervention group it would not be a deciding factor when the results are compared between groups.

A small percentage of the individuals in this study population were smokers and suffered from metabolic conditions (see Table 4-1). Both smoking and DM II have been linked to decrease rates of wound healing in general (Brem and Tomic-Canic, 2007; McDaniel and Browning, 2014). Although in this study, in the control group and intervention group the participants who had DM II were on medication and their DM II levels after fasting were under 120mg/dl. Of the participants presenting with a lipid disorder (increased levels of blood cholesterol), all were on medication and their blood cholesterol levels were under 159mg/dl when tested, thus also under control and within normal parameters. DeFroda *et al.* (2016) indicate that uncontrolled diabetes and smoking yield poorer results in individuals who undergo TJA (DeFroda *et al.*, 2016). This could have had an influence on the results found in this study; however, the control group and intervention group were similar in this regard and, therefore, smoking and metabolic conditions could not be determining factors regarding the differences in the findings.

5.3 Discussion on comparing the control and intervention group

5.3.1 Comparing the control group versus the intervention group for pain pre- and post-arthroplasty as measured by the Brief Pain Inventory (BPI)

A description of the BPI, its reliability and validity can be found in 3.2.3.2.

At 12 weeks post-surgery the minimum score of the BPI for the control group indicated that all of the individuals had some form of pain at this stage (see Table 4-6). In contrast, the minimum score for the BPI of the intervention group at 12 weeks post-surgery indicates that at least one participant was pain-free at this stage (see Table 4-6). The results of a study done by Wylde *et al.* (2011) indicate that 44% of TKA individuals and 27% of THA individuals have some form of chronic pain 41 months after surgery. This was a large study of 1294 participants who underwent TKA or THA surgery, followed by a complete physiotherapy rehabilitation programme through the Avon Orthopaedic centre conducted by Wylde *et al.* (2011). Pinto *et al.* (2013) found that at four to six months after surgery, 66.7% of THA and 88.6% of TKA participants experienced some form of pain. Pinto *et al.* (2013) aimed in their study to assess the combined influence of demographics along with clinical and psychological risk factors, which included pain catastrophising and anxiety, on 124 THA and TKA individuals. In their study all TKA and THA individuals underwent a physiotherapy rehabilitation programme.

It is however important to assess the intensity of pain experienced by individuals at 12 weeks post-surgery in this study due to the definition of chronic pain being pain present for 12 weeks or longer with an intensity of three out of 10 (IASP, 2013; Piscitelli *et al.*, 2013). Wylde *et al.* (2011) indicated that 15% of TKA and 6% of THA patients still experience severe levels of chronic pain 3 to 4 years after surgery. No severe or even moderate levels of pain were found in any individual in this study, measured at 12 weeks post-surgery. These 12 weeks post-surgery results, compared with results from Wylde *et al.* (2011) and Pinto *et al.* (2013), indicates that both the control and intervention group in the current study obtained superior results when compared to similar research studies. The positive results obtained in this study is further confirmed by comparing results by Beswick *et al.* (2012), who showed that between 7% and 23% of THA and 10% to 34% of TKA individuals had unfavourably long-term pain outcomes. Unfavourable outcomes were assigned to persons who had moderate-to-severe and severe pain levels. Both Pinto *et al.* (2013) and Wylde *et al.* (2011) incorporated physiotherapy rehabilitation into their study. The superior results obtained during this study in BPI scores could be advocated to PNE. Pre-surgery education greatly improves the individual's ability to acquire positive coping strategies (Schwartz *et al.*, 2014).

There was however no statistical significant difference in pain severity or pain interference between the control and intervention group at any stage during this study (see Table 4-7 and Table 4-8). However, the positive results in pain reduction in both groups at 12 weeks post-surgery highlights the effectiveness of the intervention group programme (CTET, SPRP and PNE) as well as the control group programme (SPRP and PNE). The results also confirm why TJA is generally the most effective solution to chronic pain due to severe osteoarthritic changes in hip and knee joints for the elderly and why physiotherapy rehabilitation is considered highly important after TJA (Mistry *et al.*, 2016; Valeberg *et al.*, 2016). The results corroborate why educating the patient, both pre- and post-surgically, regarding pain neuroscience, as far as biopsychosocial factors are concerned, could be developed as an effective tool to decrease pain, anxiety and catastrophising (Louw *et al.*, 2011).

The ability of CTET to reduce fear has shown a positive response in decreasing nociception sensitisation and the perception of ongoing pain (Crombez *et al.*, 1999). Monticone *et al.* (2013) found that exercises focusing on FOM had positive effects in decreasing pain in participants undergoing TKA (Monticone *et al.*, 2013). Both PNE and CTET assists the individual to improve body-self perception which has been directly correlated to decreased pain perception (Eyles *et al.*, 2016). The improved coping strategies not only decreases with PNE, but positive coping strategies improves motivation and thus adherence to rehabilitation (Schwartz *et al.*, 2014). A SPRP including, PNE and CTET, however did not have a superior outcome on pain compared to a SPRP, including PNE, on individuals undergoing TJA.

Clinically this result compels the physiotherapist to re-asses a purely neuromuscular approach to TJA rehabilitation, both pre- and post-surgery. Furthermore, a dedicated approach to assist the individual with education regarding pain and pain physiology should be incorporated into current rehabilitation protocols prior or after TJA to assist in improving post-surgical pain as well as improve coping strategies. PNE is a cost effective tool to incorporate, not only as it does not acquire any additional medical equipment for the practitioner, but also because studies have shown that it leads to less doctor visits and additional testing post-surgery (Louw, Diener, *et al.*, 2013).

5.3.2 Comparing the control group versus the intervention group for physical function pre- and post-arthroplasty as measured by the Western Ontario and McMaster Universities Arthritis Index (WOMAC)

A description of the WOMAC, its reliability and validity can be found in 3.2.3.3.

The control group improved with 21.22 from baseline to 12 weeks post-surgery, thus resulting in a clinically meaningful difference (see Table 4-13). The intervention group improved with 50.00 from baseline to 12 weeks post-surgery, resulting in an even greater clinically meaningful difference. At six weeks post-surgery the intervention group already showed to have a clinically meaningful difference in their scores with 31.90 (see Table 4-13). Dowsey and Choong (2013) states that an overall improved WOMAC score, with greater than 20, indicates a clinically meaningful difference in the WOMAC questionnaire. Papakostidou *et al.* (2012) showed a clinically meaningful difference between baseline and 12 weeks post-surgery with a difference of 27.00. Decreased physical functional abilities prior to surgery have been directly correlated to decreased function post-surgery (Sancheti *et al.*, 2013). As the intervention group had such a dramatic improvement in function the effects of CTET cannot be overlooked (see Table 4-15). CTET improves self-efficacy which shows to improve physical function. Furthermore, CTET aims to establish normal motor tasks, which improves motor maps in the brain, and therefore positively affects functional abilities (Barton and Morris, 2003).

There was however no statistical significant difference in total WOMAC scores between the control and intervention group at any stage during this study (see Table 4-14). The positive results in physical function improvement in both groups at 12 weeks post-surgery emphasise the effectiveness of a SPRP and PNE control group programme as well as a CTET, SPRP and PNE intervention group programme on patients' physical function after receiving TJA. The results also confirm why TJA is generally the most effective solution to chronic pain due to severe osteoarthritic changes in hip and knee joints and why physiotherapy rehabilitation is considered highly important after TJA (Mistry *et al.*, 2016; Valeberg *et al.*, 2016).

The intervention, of a SPRP, including PNE and CTET, did not indicate to have an improved outcome on physical function compared to a SPRP, including PNE, on individuals undergoing TJA at 12 weeks post-surgery.

Clinically this result compels the physiotherapist to include patient education regarding pain and pain physiology along with their neuromuscular approach to TJA rehabilitation, both pre- and post-surgery. This approach could assist in improving post-surgical physical function results due to decreased FOM and increased muscle strength. The possibility exists that individuals suffering from high levels of disuse or disability prior to surgery may benefit from CTET. As CTET improves functional strength due to individuals doing activities previously avoided, physical function could improve (van Dijk *et al.*, 2006).

5.3.3 Comparing the control group versus the intervention group for pain catastrophising as measured by the Pain Catastrophising Scale (PCS)

A description of the PCS, its reliability and validity can be found in 3.2.3.4.

At 12 weeks post-surgery the minimum score of the PCS for the control group indicated that at least one individual had no catastrophising at all. The intervention group at the same stage also had the same minimum PCS score. The intervention group however also had a median score that indicated at least 50% of the individuals in the intervention group had no catastrophising at all. When the recorded results of Forsythe *et al.* (2008) are compared before surgery (9.80) and at 12 weeks post-surgery (10.00), the scores are very similar. The positive results in pain catastrophising improvement in both groups at 12 weeks post-surgery underscores the effectiveness of a SPRP, including PNE as well as a SPRP, including PNE and CTET in patients' pain catastrophising after receiving TJA.

Both the control and intervention group were characterised as being non-catastrophising at 12 weeks post-surgery. There was however no statistical significant difference in pain catastrophising between the control and intervention group at any stage during this study. The results however suggest that educating patients, both pre- and post-surgery, regarding pain neuroscience, as far as biopsychosocial factors are concerned, may be an effective tool to decrease pain catastrophising (Louw *et al.*, 2011). The intervention programme (SPRP, PNE and CTET) did not lead to improved physical function outcomes compared to the control programme (SPRP and PNE) in individuals undergoing TJA.

Clinically this result compels the physiotherapist to add information regarding pain and pain physiology to their neuromuscular TJA rehabilitation to improve pain catastrophising, both pre- and post-surgery. It should however be considered that the results have improved the catastrophising levels of already low catastrophising individuals. The effects of the control (SPRP and PNE) or intervention (CTET, SPRP and PNE) programme could be different, whether more beneficial or less, in individuals with high catastrophising scores. Additionally, the implementation of CTET, prior to surgery and in the sub-acute stage, could have led to improved functional strength due to participation in activities previously feared, which leads to greater functionality and positively affects pain catastrophising (Meeus *et al.*, 2010).

5.3.4 Comparing of the control group versus the intervention groups FOM pre- and post-surgery as measured by the Tampa Scale for Kinesiophobia (TSK-13)

The participants' FOM was assessed by the TSK-13. No persistent tension was noted after the CTET training had ceased. A description of the TSK-13, its reliability and validity can be found in 3.2.3.5.

No significant difference was found when comparing the control to the intervention group at the different stages of the study (see Table 4-27). Even though the baseline scores for FOM were low for both groups, they were still comparable to studies that involved TJA and physiotherapy intervention. Van Oosterwick *et al.* (2011) found that PNE had a positive effect on pain, disability and catastrophising, but not FOM after only one education session in people suffering from chronic whiplash disorders (Van Oosterwijck *et al.*, 2011). Ryan *et al.* (2010) did not find PNE, combined with physiotherapy exercises, to have superior results in decreasing FOM when compared to physiotherapy exercise alone for chronic low back pain (Ryan *et al.*, 2010). The positive results in reduction in FOM in both groups at 12 weeks post-surgery compared to literature highlights the effectiveness of both a SPRP, including PNE as well as a SPRP, including PNE and CTET on patients' FOM after TJA. The intervention, a SPRP, including PNE and CTET, however did not have a profound effect outcome on FOM compared to a SPRP, including PNE on individuals undergoing TJA.

Clinically this result requires that the physiotherapist combine neuromuscular TJA rehabilitation with education on pain and pain physiology to improve FOM, both pre- and post-surgery. This study did not find sufficient support to implement CTET for individuals that have low levels of FOM prior to surgery

5.4 Discussion of the control group and intervention group

5.4.1 Control and intervention group's pain pre- and post-arthroplasty as measured by the BPI

Although the mean pain severity score of the control group is lower at baseline than the intervention group, both groups have lower scores before surgery than that reported by literature of 17.9 and 17.8 out of a possible 40 (Pereira, Meleiro, Correia, Fonseca, Pereira, Meleiro, Correia and Fonseca, 2016; Valeberg *et al.*, 2016) (see Table 4-5). Valeberg *et al.* (2016) tested 71 individuals for pain and pain sensitivity before and after TKA. Similar pain results to Valeberg *et al.* (2016) were found prior to surgery (17.8) in a study that tested the possible influence of two different commonly used anaesthetics administered during TKA and THA surgery (Pereira *et al.*, 2016). The slightly lower mean pain results at baseline in this study could possibly indicate that pain severity might not be the primary reason for undergoing TJA for this study population. Furthermore, pain severity might be less due to low baseline pain catastrophising and FOM in the study individuals, which both have been linked to negatively impact pain perception (Masselin-Dubois *et al.*, 2013).

The baseline score for the intervention group was the highest recorded mean pain severity score throughout the study, whereas the hospital discharge score for the control group was recorded as the highest (see Table 4-5). Interestingly the mean hospital discharge scores for both groups were almost exactly the same, indicating the same level of pain severity post-surgery after in-hospital physiotherapy rehabilitation were experienced by the study sample.

The mean pain severity scores obtained at 12 weeks post-surgery were higher for the control group than the intervention group (see Table 4-5). Both these scores are less than that obtained by Valeberg *et al.* (2016). The lowest score obtained by Pereira *et al.* (2016) was in the group receiving neuraxial anaesthesia, which had a mean pain severity score of 11.5 at 24 weeks post-surgery. In the study by Pereira *et al.* (2016) the participants only received rehabilitation after surgery, while in hospital. Thus, difference in pain severity results from this study compared to Pereira *et al.* (2016) could possibly be attributed to a combination of active physiotherapy rehabilitation, PNE and techniques aimed at decreasing catastrophising and FOM that Pereira *et al.* (2016) did not incorporate. Understanding the reason for experiencing pain and the physiological process has been reported to reduce pain perception in individuals (Schwartz *et al.*, 2014). Furthermore, the chronic pain phenomenon could have been averted by the management of the acute pain experience through early in-hospital physiotherapy (Rooks *et al.*, 2006).

The pain interference mean score for the intervention group was higher at baseline than that of the control group (see Table 4-5). Both the control and intervention group had lower mean pain interference scores than 37.39 recorded by Pereira *et al.* (2016). Although the control group had a lower mean pain interference score compared to the 30.2 described in literature for individuals undergoing TKA (Valeberg *et al.*, 2016). The high pain interference baseline results for the intervention group could indicate that pain interference may possibly be one of the driving factors in the decision of the intervention group to undergo TJA. Due to the link of greater OA severity to greater functional disability, and greater OA changes are found in older individuals, the higher mean age of the intervention group could be the reason why the baseline score for how much pain interferes with daily activities are higher than that of the control group (Sadosky *et al.*, 2010) (see Table 4-1). An increased pain interference baseline score would then according to literature have a negative impact on the individuals physical function scores (Sancheti *et al.*, 2013).

Again, the baseline score for the intervention group was the highest recorded mean pain interference score throughout the study, whereas the hospital discharge score for the control group was recorded as the highest (see Table 4-5). Valeberg *et al.* (2016) had a mean pain interference score of 17.1 at eight weeks post-surgery. Pereira *et al.* (2016) had a mean pain interference score of 23.56, 24 weeks after TKA and THA. Both these results for pain interference demonstrated in literature are a lot higher than found in both the control and intervention group at 12 weeks post-surgery (see Table 4-5). Again, as with the pain severity the intervention group had a lower six week and 12 week post-surgery result for pain interference (see Table 4-5). This study confirms what has been previously reported that physiotherapy assists in decreasing pain and improving function after TJA (Rooks *et al.*, 2006).

The combined scores of the pain severity and pain interference scores at baseline for the control group were lower than the 48.10 out of a possible 110 obtained by Valeberg *et al.* (2016) (see Table 4-6). The intervention group however had a higher combined mean score prior to surgery than Valeberg *et al.* (2016), while still lower than 55.19 recorded by Pereira *et al.* (2016). This baseline value for both the control and intervention group constituted mild pain according to Wylde *et al.* (2011). Wylde *et al.* (2011) recommends that a pain score of 1 – 50 indicates mild pain, 51 – 75 moderate pain and 76 – 100 severe pain.

From baseline the total BPI score increased at hospital discharge then decreased thereafter for the control group, while the intervention group consistently decreased from baseline to 12 weeks post-surgery (see Table 4-6). The total BPI scores at hospital discharge were very similar for the control and intervention group. The similarity of these results possibly indicate that CTET has a minimal effect on pain severity and pain interference in the acute phase post TJA. All individuals, although receiving pain medication to manage their pain, had to sit, walk and climb a staircase in the exact same timeframe after undergoing TJA, under physiotherapy supervision. The possibility thus exists that the SPRP for in-hospital rehabilitation that focuses on advancing physical function in a controlled but rapid time-based progression mimic the principles of CTET.

Furthermore, the mean scores at 12 weeks post-surgery for the control and intervention group are less than the 24 out of a possible 110 obtained by Valeberg *et al.* (2016) and less than 35.06 obtained by Pereira *et al.* (2016) (see Table 4-6). The intervention group had a lower total BPI score at 12 weeks post-surgery than the control group, although both groups had scores that constitute mild pain according to Wylde *et al.* (2011). Valeberg *et al.* (2016) only exposed their participants to physiotherapy rehabilitation after TKA in-hospital where after they were given a home exercise program to follow on their own. This short-term exposure to supervised physiotherapy rehabilitation along with an unsupervised home exercise program could explain the difference in overall BPI results after surgery. It should however be remembered that Valeberg *et al.* (2016) only tested TKA, who experience greater pain levels post-surgery than THA, and the score was obtained at eight weeks post-surgery (see 2.4.2 Pain in TJA). Pereira *et al.* (2016) had a mean total BPI score of 35.06 at 24 weeks after TJA, that again could be explained by the limited physiotherapy rehabilitation after surgery. The results confirm the positive effects of a SPRP, including PNE as well as a SPRP, including PNE and CTET on the pain that patients experience undergoing TJA. Although no differences were reported with regards to statistical differences found during within group results, the effect of CTET is limited for pain during the first 12 weeks post-surgery.

5.4.2 Control and intervention group's physical function pre- and post-arthroplasty as measured by the WOMAC

The intervention group had a mean WOMAC score that was marginally higher at baseline testing than results found in literature of 58.44 (Nilsson, Petersson, Roos and Lohmander, 2003) (see Table 4-13). The control group had a mean WOMAC score that was substantially lower at baseline testing than results found by Nilsson *et al.* (2003) and Papakostidou *et al.* (2012). Nilsson *et al.* (2003) studied 219 patients who underwent primary THA, including physiotherapy rehabilitation, due to OA, to assess the relation between patient self-reported WOMAC and Short form 36 outcomes. Another study evaluating self-administered outcomes, which included the WOMAC questionnaire, to determine quality of life after TKA had a baseline score of 54.8 (Papakostidou, Dailiana, Papapolychroniou, Liaropoulos, Zintzaras, Karachalios and Malizos, 2012). Papakostidou *et al.* (2012) only required the TKA individuals to undergo in-hospital physiotherapy rehabilitation.

The control group had an increase in mean WOMAC scores from baseline to hospital discharge and the intervention group had an improved result during the same period. Considering the changes from baseline to hospital discharge in the mean WOMAC score for the control and intervention group, the mean values were very similar (see Table 4-13). The similarity in in-hospital scores again highlights the effectiveness of in-hospital physiotherapy after TJA.

The mean WOMAC score obtained at 12 weeks post-surgery was considerably lower for the intervention group compared to the control group out of a maximum of 96 (see Table 4-13). At 12 weeks post-surgery Papakostidou *et al.* (2012) showed a WOMAC score of 27.8. The intervention group score obtained by this study at 12 weeks post-surgery is less than Papakostidou *et al.* (2012) at 12 months post-surgery (10.1) and Nilsson *et al.* (2003) at 3.6 years post-surgery (23.04). Neither Papakostidou *et al.* (2012) nor Nilsson *et al.* (2003) included strategies to influence pain catastrophising or FOM in their studies. It should be considered that the control and intervention group were dissimilar at baseline with regards to function and stiffness. The intervention group had worse function and stiffness scores at baseline, thus the improvements obtained up to 12 weeks post-surgery indicates the effectiveness of CTET as an intervention. The results thus confirm the positive effects of intervention programme (CTET, SPRP and PNE) as well as the control group programme (SPRP and PNE), on physical function in patients undergoing TJA.

5.4.3 Control and intervention group's pain catastrophising pre- and post-arthroplasty as measured by the PCS

The control group had a considerable lower median rumination score than the intervention group at baseline testing (see Table 4-17). Whilst the intervention group had a higher score, the control group had the same median rumination score at baseline as found in literature of 5.0 (Forsythe *et al.*, 2008). Forsythe *et al.* (2008) tested 55 individuals undergoing TKA and aimed to determine the influence of pain catastrophising on the development of chronic post-surgery pain. Although, the median rumination score decreased in the intervention group, it still scored higher than control group at hospital discharge. As rumination relates to the inability of the participant to stop thinking of their painful condition, this result at hospital discharge hints that PNE in combination with CTET could cause a hypervigilant response to rationalise the acute pain experience and indirectly cause greater attention thereon. It should be considered that the intervention group did show a decrease in rumination score from baseline to hospital discharge that the control group did not accomplish (see Table 4-17).

The median rumination obtained at 12 weeks post-surgery were noticeably lower in both the control and intervention group when compared to the 4.3 out of a maximum of 16 achieved by Forsythe *et al.* (2008) (see Table 4-19). A study done by Van Oosterwijck *et al.* (2011) found that PNE only significantly improved the rumination subsection of the PCS and not the total PCS score. Considering the result found by Van Oosterwijck *et al.* (2011), PNE in combination with CTET could together cause the more favourable results obtained by the intervention group at 12 weeks post-surgery (see Table 4-19). The intervention group was the only group that indicated to have statistical significant differences in results for rumination. These results were for baseline compared to six weeks post-surgery as well as 12 weeks post-surgery. There was a statistical significant difference in results from six weeks to 12 weeks post-surgery as well (see Table 4-22). Furthermore, pain catastrophising has been reported to be a predictor for chronic pain (Vlaeyen and Linton, 2012). Thus, the lack of severe levels of chronic pain perception as mentioned before by the intervention group indicates the influence of CTET on pain catastrophising, especially rumination.

The magnification median score for the control and intervention group at baseline were exactly the same (see Table 4-19). This baseline magnification median score is however higher than 1.40 found in literature for individuals undergoing TKA (Forsythe *et al.*, 2008). Furthermore, this baseline score was the highest median score for magnification throughout the study for the control and intervention group. Magnification relates to the fear of the seriousness the participant has with regards to their condition. The intervention group was the only group that indicated a statistical significant decrease in magnification between baseline and hospital discharge (see Table 4-22). This could indicate the value of implementing CTET prior to surgery to decrease the fear of the seriousness of the individuals condition.

Both the control and intervention group showed consistent improvement in their median magnification scores throughout the study (see Table 4-19). Forsythe *et al.* (2008) had a magnification score of 1.8 at 12 weeks post-surgery, thus not showing an improvement when compared to their baseline scores. At six weeks post-surgery for the intervention group, and at 12 weeks post-surgery the median magnification score for the control and intervention group indicated that, at least 50% of the participants had no pain catastrophising magnification (see Table 4-19). As pain magnification has been reported to predict chronic pain intensity, this decrease in magnification could help explain the moderate levels of pain experienced by both groups. This marked improvement in magnification emphasise the encouraging effects of the intervention programme (CTET, SPRP and PNE) as well as the control group programme (SPRP and PNE), on individuals ability to establish a coping strategy that decreases the fear of the seriousness of their condition when undergoing TJA.

The helplessness median score for the control and intervention group at baseline were higher than the 3.40 recorded by Forsythe *et al.* (2008) out of a possible 24 (see Table 4-19). Even though the intervention group had a lower baseline median helplessness score, the control group had the lowest median score at hospital discharge in this study (see Table 4-19). Helplessness relates to the inability of the participant to cope with their painful experience. The failure to achieve the expected low levels of pain and increased level of function in the acute phase after TJA could explain the higher score at this testing stage in the intervention group.

At 12 weeks post-surgery both the control and intervention group had lower helplessness scores than the 3.6 recorded by Forsythe *et al.* (2008). The intervention group had the lowest score and when taking in consideration the median score more than 50% of the participants in the intervention group had no pain catastrophising helplessness at 12 weeks post-surgery (see Table 4-19). This marked in relation to Forsythe *et al.* (2008) in helplessness underlines the positive effects of the intervention programme that includes CTET, SPRP and PNE as well as the control programme of a SPRP and PNE, on individuals ability to establish a positive coping strategy when undergoing TJA.

The combined scores for rumination, magnification and helplessness scores at baseline were higher in the intervention group than in the control group (see Table 4-19). These baseline values constitute as non-catastrophising according to Sullivan (1995). Sullivan (1995) recommended that catastrophising be considered when a score of 30 or higher is obtained on the PCS. These baseline scores are however higher than the 9.80 found by Forsythe *et al.* (2008). The pre-surgery PCS result for a study studying the association between pain catastrophising and functional abilities on both THA and TKA were higher at 24.48 (Hayashi, Kako, Suzuki, Hattori, Fukuyasu, Sato, Kadono, Sakai, Hasegawa and Nishida, 2017). Hayashi *et al.* (2017) unfortunately only tested their individuals up to two weeks post-surgery, they obtained a score of 19.48. It is important however to highlight that both the control and intervention group had lower median total PCS scores at hospital discharge than Hayashi *et al.* (2017) at two weeks post-surgery. Although the PCS scores for both groups at baseline are within the non-catastrophising range, they were consistently higher at baseline than found in literature except the control rumination at baseline which was equal to Forsythe *et al.* (2008). This result brings into question the percentage representation of high catastrophising individuals undergoing TKA and THA. The researcher could not find such relevant data in literature after a thorough search.

From baseline the total PCS scores consistently decreased in both the control and intervention group to 12 weeks post-surgery (see Table 4-19). The median total PCS scores for both groups also constitute as non-catastrophising according to Sullivan (1995). The scores recorded by both groups at 12 weeks post-surgery are less than the 10.00 out of a possible 52 obtained by Forsythe *et al.* (2008). The effects of CTET in the acute stage post-surgery did not show extremely beneficial results when compared to the control group. The encouraging effects of CTET in the sub-acute stage is however evident in the results with regards to rumination. The results thus confirm the positive effects of a SPRP, including PNE as well as a SPRP, including PNE and CTET, on pain catastrophising in patients undergoing TJA.

5.4.4 Control and intervention group's FOM pre- and post-arthroplasty as measured by the TSK-13

The control and intervention group had similar mean TSK-13 scores at baseline testing (see Table 4-26). The results are very similar to results found in literature of 34.40 (Monticone *et al.*, 2013). Monticone *et al.* (2013) tested 110 individuals undergoing TKA with a physiotherapy home-based exercise program to establish the effects thereof on FOM. 104 individuals that underwent THA and had three out of 10 or less pain after 12 weeks had a pre-surgery TSK-13 score of 35.00 (Erlenwein, Muller, Falla, Przemeczek, Pfingsten, Budde, Quintel and Petzke, 2017). Erlenwein *et al.* (2017) compared the prevalence of chronic pain after surgery when the definition of chronic pain includes persons with less than three out of 10 pain with those with pain greater than three. The results are however higher than the 30.8 obtained by a study on 282 participants to determine the frequency and associated factors to falling prior to THA and TKA (Hill, Wee, Margelis, Menz, Bartlett, Bergman, McMahon, Hare and Levinger, 2016).

The hospital discharge mean TSK-13 scores for both groups were also similar to each other (Table 4-26). This result indicates that CTET might not have any benefits on the acute FOM of participants after TJA. The mean TSK-13 obtained at 12 weeks post-surgery were very similar in the control and intervention group, although the scores were higher than 20.10 recorded by Monticone *et al.* (2013) who only re-tested the participants at 24 weeks post-surgery.

The FOM level obtained at 12 weeks post-surgery in both groups are described as low (Dolphens *et al.*, 2014). Dolphens *et al.* (2014) recommended that scores greater than 37 be classified as having high levels of FOM. Scores obtained of 37 or lower is recommended to be described as having low levels of FOM. The maximum TSK-13 mean scores achieved at 12 weeks post-surgery in both the control and intervention group also fall within the definition of having a low level of FOM (Dolphens *et al.*, 2014). The mean score at 24 weeks post-surgery for Erlenwein *et al.* (2017) was 30.00 in individuals with pain less or equal to three out of 10. Furthermore, the minimum scores of obtained by the control and intervention group at 12 weeks post-surgery are the lowest possible score obtainable for the TSK-13 (see Table 4-26). Even though this study had baseline values that equates to low FOM, the individuals scored similar or more than relevant studies in literature. This again brings into question the number of individuals undergoing TJA that have high levels of FOM. The researcher was unable after a thorough search of literature obtain a representative percentage of high FOM individuals undergoing TJA.

It has been reported in literature that lower levels of pain coincides with lower levels of FOM (Vlaeyen and Linton, 2012). FOM is also negatively affected by pain catastrophising (Masselin-Dubois *et al.*, 2013). Thus, the low levels of pain and pain catastrophising obtained at 12 weeks post-surgery obtained in this study had a positive influence on FOM levels of the individuals. The results confirm the encouraging effects of an intervention programme (CTET, SPRP and PNE) as well as the control programme (SPRP and PNE) on FOM in patients undergoing TJA.

6 Chapter 6: Limitations, recommendations and conclusion

6.1 Conclusion

The control group programme (SPRP and PNE) were effective in improving pain, function, FOM and pain catastrophising in patients undergoing TJA. The intervention group programme (CTET, SPRP and PNE) indicated to be beneficial in decreasing pain catastrophising in patients undergoing TJA.

There was no statistical significant improvement in pain, function, FOM and pain catastrophising in patients undergoing TJA, between the control group receiving a SPRP, including PNE and the intervention group receiving a SPRP, including PNE and CTET.

Key findings of this study are:

- A SPRP administered under physiotherapy supervision, including a single one-on-one PNE session prior to surgery are effective in limiting the occurrence of moderate and severe post-surgical pain in individuals undergoing TKA and THA within 12 weeks post-surgery.
- A SPRP administered under physiotherapy supervision, including a single one-on-one PNE session prior to surgery are effective in substantially improving the physical function of individuals undergoing TKA and THA within 12 weeks post-surgery.
- A SPRP administered under physiotherapy supervision, including a single one-on-one PNE session prior to surgery are effective in limiting the occurrence of pain catastrophising, especially magnification in individuals undergoing TKA and THA at 12 weeks post-surgery.
- A SPRP administered under physiotherapy supervision, including a single one-on-one PNE session prior to surgery are just as effective as a SPRP, including PNE and CTET in limiting the occurrence of FOM in individuals undergoing TKA and THA 12 weeks post-surgery.

- The possibility exists that supplementing a SPRP, administered under physiotherapy supervision, including a single one-on-one PNE session prior to surgery with supervised CTET could assist the individual undergoing TJA to a greater extent with pain catastrophising, especially rumination and helplessness within the first 12 weeks post-surgery.

Due to the low scoring in pain catastrophising and FOM, even though they were similar or higher than those of relevant studies in literature causes the researcher to consider not only the representation of these individuals in TJA in literature, but also the effects the magnitude of these two psychological conditions have on individuals undergoing TJA. Two possible trends of thought are that either a small presence of pain catastrophising or FOM need to be present to help facilitate acute pain to become chronic, or these two conditions are not the primary force behind chronic pain after TJA.

The benefits of physiotherapy involvement in TJA are confirmed in this study. The physiotherapists expertise in neuromuscular training, the on-on-one nature of the rehabilitation process as well as the knowledge of pain physiology places the physiotherapist in a prominent role both in the acute and sub-acute phase after TJA. Furthermore, this study confirms the physiotherapists ability to safely administer fear-based exercises such as CTET, that may benefit selective individuals undergoing TJA.

6.2 Limitations

Literature on the effects of PNE on TKA and THA participants is sparse. The positive effects of PNE on pain perception are, however, well documented.

This study comprised an economically and ethnic-biased, small study population of 19 individuals. The small population, although representative of the region, is vulnerable to extreme scores. Only a private hospital was used to sample participants for this study, and when one keeps in mind that the literature indicates that individuals with a lower income have worse functional results, the study participants could have been prone to better results.

The CTET was often dissimilar between individuals in the control group as the feared movements differed. This caused the researcher to follow a general guideline in the progression of activities instead of a fixed protocol that could be re-tested by means of the same methodology employed in this study.

The study did not take into consideration, or test for, any mood disorders, such as depression (Wylde *et al.*, 2011). Multiple studies have shown a relationship between pain and depression, as well as anxiety disorder and pain levels (Means-Christensen, Roy-Byrne, Sherbourne, Craske and Stein, 2008).

The individual's self-efficacy, which is described as the individual's perceived ability to continue normal, everyday activities despite experiencing pain symptoms, was also not tested. Self-efficacy has been shown to be a greater mediator of perceived pain than kinesiophobia (Costa, Maher, McAuley, Hancock and Smeets, 2011).

The participants in his study were rehabilitated by four different physiotherapists, which could have had an impact on the research results. These physiotherapists, however, were trained in the application of a SPRP for THA and TKA to initiation of this study in order to diminish the possibility that the rehabilitation might have had an impact on the study results.

There may be differences between the intervention and the control groups regarding cardio-pulmonary functioning or exercise tolerance, although the influence was decreased by stratifying THA and TKA individuals before randomly appointing them into a group.

The limited time available to complete the study limited the duration of post-surgical testing to 12 weeks. The minimum duration for conditions to be considered chronic were however achieved at 12 weeks post-surgery.

6.3 Recommendations

Further studies should involve a greater and more diverse number of study participants. With an increased number of study participants, a third group that only undergoes a SPRP can be added to quantify the effects of PNE and a SPRP as well as a SPRP, PNE and CTET more successfully.

Inclusion of self-efficacy questionnaires can shed light on individuals' ability to continue with their everyday activities in the presence of pain, thus assisting the pain catastrophising questionnaire to bridge the gap of pain and avoidance-causing disabilities.

Future studies should consider longer follow-up times to not only increase the availability of physiotherapy interventions over a longer period of time, but also to assess the long-term effects of both PNE and CTET on individuals undergoing TJA.

Stratifying individuals into comparable, fearful activity groups can elicit a better understanding of the effects of CTET on pain, physical function, catastrophising and FOM in individuals undergoing TJA.

Inclusion of statistics regarding the compliance to the SPRP and home exercise programs should be incorporated in future studies. This will enable the researcher to give a better explanation of the results with regards to function.

Future studies could implement blood test results, such as determining the level of cortisol prior to when a movement or task, of which the individual is fearful, is performed and again afterwards, or comparing pre-surgical cortisol levels with those at 12 weeks post-surgery.

Inclusion of individuals that have high pain catastrophising and FOM baseline scores should be included in future studies to assess the potency of PNE as well as CTET on this demographic group undergoing TJA.

Gathering data regarding the percentage of individuals suffering from high levels of pain catastrophising as well as FOM that undergo TJA should be considered.

7 References

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8 Appendices

8.1 Appendix 1

Participant's Information Leaflet (English)

**PARTICIPANT'S INFORMATION LEAFLET AND INFORMED
CONSENT**

Researcher:

Ruan Mockè

Department of Physiotherapy

University of Free State

Dear Participant

**The efficacy of pain neuroscience education in combination with cognitive-
targeted exercise therapy in total joint arthroplasty: A randomised
controlled trial**

INTRODUCTION

I am Ruan Mockè, and as part of my Masters degree in Physiotherapy you are invited to participate in a research project on the efficacy of pain neuroscience education with cognitive-targeted exercise therapy in total joint arthroplasty.

Participation is voluntary and this document gives information to help you to decide whether you want to take part in this study. Before you agree you should fully understand what is involved. If you do not understand the information or have any other questions, do not hesitate to ask. You should not agree to take part unless you fully understand what is expected of you.

WHAT IS THE PURPOSE OF THIS STUDY?

The purpose of our study is to determine whether specific cognitive-targeted exercises (exercises that focus on certain behavioural patterns) along with education about pain and pain physiology can improve the outcome of your total hip or knee replacement.

The plausibility of improved outcomes due to the study after your joint replacement surgery includes both pain and functional improvements that could further support your quality of life.

WHAT IS THE DURATION OF THIS STUDY?

The study will not prolong or shorten the rehabilitation regime associated with your total hip or knee replacement. The study will also not prolong your hospital stay. The appropriate questionnaires and tests will take approximately one (1) hour and will be performed by the researcher. These questionnaires and tests will be repeated four (4) times. Furthermore there will be educational classes which will be approximately forty (40) minute sessions.

All results of the tests and questionnaires will be handled confidentially. You will receive a participant code for the duration of the trial, so you do not have to write your name on the questionnaires.

Assistance will be available to help you with the questionnaire and explain any terms you have difficulty with.

HAS THE STUDY RECEIVED ETHICAL APPROVAL?

This clinical study protocol was submitted to the Faculty of Health Sciences Research Ethics Committee, University of the Free State and the study will only be conducted once written approval has been granted (ECUFS number 172/2015). The study has been structured in accordance with the Declaration of Helsinki (last revised 2012-2013), which deals with the recommendations guiding doctors in biomedical research involving human/subjects. A copy of the Declaration may be obtained from the investigator should you wish to review it. The contact number of the Ethics committee's secretariat is +27(0) 51 401 7794/5 if you have any queries about this research study.

WHAT ARE YOUR RIGHTS AS A PARTICIPANT IN THIS STUDY?

Your participation in this study is voluntary. You can refuse to participate or withdraw at any time. As you do not write your name on the questionnaire, you give us the information anonymously. Once you have given the questionnaire back to us, you cannot recall the information thereon. You will also not be identified as a participant in any publication that comes from this study. If you are interested in the outcome of the study, you can contact the researcher after the study at 064 40 5056.

MAY ANY OF THESE STUDY PROCEDURES RESULT IN DISCOMFORT OR INCONVENIENCE?

Cognitive-targeted exercise therapy will involve physical activity. The aim of this type of exercise is not to improve your cardiovascular fitness, but rather to improve activities you have been having problems with as of late. These activities might seem difficult initially and care will be taken to gradually increase them in difficulty. The normal effects of exercise are an increased heart rate, increased respiration rate, sweating and increased oxygen and nutrient supply to your muscles.

If, in the unlikely event that these effects cause severe discomfort to you, the researchers will immediately inform both your medical doctor and orthopaedic surgeon for management of your symptoms.

PROCEDURE TO BE FOLLOWED

After you have read this leaflet, time will be given for discussion, clarification of consent and answering of questions by the researcher. You will then be asked to sign this informed consent form. Thereafter you will be issued with an appointment date to administer the baseline (first) set of tests and questionnaires. Information regarding your Body Mass Index (length and weight) will be obtained from your orthopaedic surgeon. The questionnaires and tests do not involve any invasive procedures or any painful activities. The questionnaires will evaluate your level of pain, functional ability, psychological distress and your knowledge of pain physiology. Completion of the questionnaires will require of you to choose suitable descriptive words from a given list, which best describes the question stated. The physical tests will involve you sitting and standing up to walk 3 metres and return to sitting. Another physical test will assess your walking style and ability.

You will be expected to expose your hip and knee during the physical tests. Please inform us if you are uncomfortable with any physical test or exercise.

All participants will receive normal rehabilitation and exercises from your local physiotherapist. Participants will also need to attend an one hour (1) session on the topic of pain and pain physiology. Some participants will be contacted telephonically to schedule two exercise sessions with the researcher which will last approximately forty (40) minutes. This will take place before your scheduled operation.

On discharge out of the hospital you will be required to repeat the same tests and questionnaires as during the first session. You will receive your normal out-patient rehabilitation from your local physiotherapist. Some participants will again be contacted telephonically to schedule two exercise sessions of forty (40) minutes each with the researcher.

At six (6) weeks after your surgery you will repeat all the questionnaires and tests.

The same procedure with the tests and questionnaires will then be repeated at twelve (12) weeks after your operation.

All participants that will be contacted telephonically, for the additional exercises, will be asked not to divulge this to any other participant or their local physiotherapist. All local physiotherapists will be informed on the possibility and nature of the additional exercises.

ARE THERE ANY WARNINGS OR RESTRICTIONS CONCERNING MY PARTICIPATION IN THIS STUDY?

We require you not to drink excessive alcohol, take pain medication or smoke for three hours prior to filling out the questionnaires and doing the physical tests, as this may affect the outcome.

You can only partake in the study if you have had pain in your knee or hip for three (3) months or longer.

No financial remunerations will be required or given for the possible extra scheduled exercises given by the researcher during this study.

The study will involve no physical risk other than the risk involved with normal total joint arthroplasty surgeries. Psychological stress involved with some exercises could cause tension, although participant monitoring and verbal consent will be continual during these exercises.

If the participant chooses to withdraw from the study their post-surgical rehabilitation will continue as normal by their local physiotherapist.

CONFIDENTIALITY

All information obtained during the course of this study is strictly confidential. Data may be published in scientific journals but will not include any information which could identify you as a participant in this study. Any information uncovered regarding your test results or state of health obtained as a result of your participation in this study will be held in strict confidence. You will be informed of any finding of importance to your health or continued participation in this study but this information will not be disclosed to any third party in addition to the ones mentioned above without your written permission. The only exception to this rule will be cases in which a law exists compelling us to report individuals infected with communicable diseases. In this case, you will be informed of our intent to disclose such information to the authorized state agency.

8.2 Appendix 2

Informed Consent (English)

Researcher:

Ruan Mockè

The efficacy of pain neuroscience education in combination with cognitive-targeted exercise therapy in total joint arthroplasty: A randomised controlled trial

INFORMED CONSENT

- I hereby confirm that I have been informed by the researcher about the nature, conduct, benefits and risks involved in this clinical study. I have also received, read and understood the above written information (Patient Information Leaflet and Informed Consent) regarding the clinical study.
- I am aware that the results of this study will be documented anonymously in a study report.
- I am aware that information regarding my Body Mass Index (height and weight) will be gathered from my orthopaedic surgeon.
- I may, at any stage, without prejudice, withdraw my consent and participation in the study. I have had sufficient opportunity to ask questions and declare myself prepared to participate in the study.

Patient's name _____

(Please print)

Patient's signature _____ Date _____

I, the researcher, herewith confirm that the above patient has been informed fully about the nature, conduct and risks of the above mentioned study.

Investigator's name (1) _____

(Please print)

Investigator's signature _____ Date _____

I sincerely appreciate your help.

8.3 Appendix 3

Inligtingsbrosjyre (Afrikaans)

**INLIGTINGSBROSJURE AAN DEELNEMERS EN INGELIGTE
TOESTEMMINGSVORM**

Navorsers:

Ruan Mocke

**Departement Fisioterapie
Universiteit van die Vrystaat**

Geagte Deelnemer

“The efficacy of pain neuroscience education in combination with cognitive-targeted exercise therapy in total joint arthroplasty: A randomised controlled trial.”

INLEIDING

My naam is Ruan Mocke, en as deel van my Meestersgraad in Fisioterapie, nooi ek u om deel te neem aan my navorsingsprojek. Die projek handel oor die doeltreffendheid van onderrig in pyn-neurowetenskap saam met kognitief-gebaseerde oefeningsterapie vir pasiente met totale gewrigsvervangings.

Deelname aan die studie is vrywillig en hierdie dokument verskaf inligting om u te help besluit of u wil deelneem aan die studie. Voor u instem om deel te neem aan die studie, is dit belangrik dat u verstaan wat die studie behels. Indien u enige van die inligting wat deur die dokument gegee word nie ten volle verstaan nie, of indien u vrae het, moet u nie huiwer om my te kontak nie. Dit is belangrik dat u die prosedure ten volle verstaan alvorens u toestem om deel te neem.

WAT IS DIE DOEL VAN DIE STUDIE?

Die studie beoog om te bepaal of spesifieke kognitief-gebaseerde oefeninge (oefeninge wat fokus op bewuste gedragspatrone) saam met onderrig in aspekte van die pyn en pynfisiologie gebied, die uitkoms van 'n totale gewrigvervanging kan verbeter.

HOE SAL DIE STUDIE VERLOOP?

Die studie sal nie die normale rehabilitasie wat gepaardgaan met u totale gewrigsvervanging, bespoedig of verleng nie. Die studie sal ook nie u hospitaalverblyf enigsins verleng nie. Die vraelyste en fisiese toetse wat hierdie studie vereis, sal ongeveer een (1) uur neem om te voltooi onder toesig van die navorser. Die vraelyste en fisiese toetse sal vier (4) keer herhaal word tydens die verloop van u rehabilitasie. Verder sal daar van u verlang word om twee onderrigssessies van ongeveer veertig (40) minute by te woon.

Alle inligting wat verkry word uit die toetse en vraelyste sal vertroulik en anoniem hanteer word. U sal 'n deelnamekode ontvang wat tydens die verloop van die studie gebruik sal word in plaas van u naam. U naam sal dus nêrens op die vraelyste verskyn nie. Die navorser sal teenwoordig wees tydens die invul van die vraelyste om enige verdere vrae te hanteer.

IS DIE STUDIE GOEDGEKEUR DEUR 'N ETIEKKOMMITEE?

Die kliniese studie is onderhewig aan goedkeuring deur die Etekkomitee, Fakulteit Gesondheidswetenskappe van die Universiteit van die Vrystaat. Die studie sal dus slegs geïmplementeer word indien goedkeuring verkry word (ECUFS nommer 172/2015). Die studie is opgestel in ooreenstemming met die riglyne van die Verklaring van Helsinki (soos hersien in 2012-2013). Die verklaring beskryf die riglyne en aanbevelings aan dokters en biomediese navorsers wat n studie met menslike kandidate uitvoer.

Indien u enige navrae het oor die navorsingstudie, kan u die Etekkomitee se sekretariaat kontak by telefoonnommer +27(0) 51 401 7794/5.

WAT IS U REGTE AS DEELNEMER TYDENS DIE STUDIE?

U deelname aan die studie bly vrywillig. U kan op enige stadium deelname aan die studie weier of onttrek. Aangesien u naam nie op die vraelyste sal verskyn nie, sal die inligting wat daarop verskyn anoniem bly. Na u die vraelyste ingehandig het by die navorser, sal u egter nie die inligting wat daarop verskyn kan onttrek nie. U sal nie geïdentifiseer kan word as 'n deelnemer aan die studie wanneer die studie gepubliseer word nie. Indien u belangstel in die uitkoms van die studie, kan u die navorser kontak na afloop van die studie by 064 40 5056.

SAL ENIGE VAN DIE PROSEDURES TYDENS DIE STUDIE ONGEMAK VEROORSAAK?

Kognitief-gebaseerde oefeningsterapie behels fisiese oefening. Die doel van die tipe oefening is egter nie om u kardiovaskulêre fiksheid te verbeter nie, maar fokus eerder op aktiwiteite wat u onlangs moeilik gevind het om uit te voer. Alle oefeninge sal stelselmatig progresseer van maklik na moeilik soos u verbeter en sterker word. Die fisiologiese effekte van oefening behels 'n verhoogde harttempo, verhoogde afskeiding van sweet, verhoogde asemhalingstempo en verhoogde suurstof- en bloedtoevoer na die spiere in die liggaam. In die onwaarskynlike geval dat u wel erge ongemak ervaar tydens die studie, sal beide u mediese dokter en ortopediese chirurg gekontak word.

WAT IS DIE PROSEDURE WAT GEVOLG MOET WORD?

Nadat u die brosjure volledig deurgegaan het, sal die navorser enige vrae oor die inhoud van die studie beantwoord. Daarna sal u gevra word om die ingeligte toestemmingsvorm te teken. Hierna sal daar 'n afspraak met u gemaak word waartydens die aanvanklike stel vrae en toetse deurgewerk sal word. Inligting aangaande u "Body Mass Index" (gewig en lengte) sal verkry word vanaf u ortopediese chirurg. Die vraelyste en toetse behels geen indringende of pynlike prosedures nie. Die vraelyste sal u vlak van pyn, funksionele beperkinge, psigologiese stressors en kennis van pynfisiologie evalueer. Daar sal bv. van u verwag word om sekere woorde uit 'n lys te kies wat u antwoord die beste beskryf. Een van die fisiese toetse wat van u verlang sal word is byvoorbeeld om van 'n sittende posisie op te staan, drie (3) meter te loop, en dan weer terug te keer na die sittende posisie. 'n Ander fisiese toets sal u loop-vermoë en u looppatroon evalueer.

Daar sal van u verwag word om u heup en knie te ontbloot tydens sommige van die fisiese toetsings. Indien u ongemaklikheid ervaar tydens enige van die toetsings, moet u die navorser dadelik in kennis stel.

Alle deelnemers sal hulle normale rehabilitasie-oefeninge ontvang van hul plaaslike fisioterapeut. Verder sal deelnemers een onderrigssessie van ongeveer 'n uur aangaande pyn en pynfisiologie ontvang. Slegs sommige deelnemers sal telefonies gekontak word om twee oefeningsessie van ongeveer 40 minute te skeduleer wat deur die navorser uitgevoer sal word. Bogenoemde sal alles plaasvind voor u operasieprosedure.

Met ontslag uit die hospitaal sal daar van u verlang word om dieselfde vraelyste en fisiese toetsings te ondergaan soos tydens u eerste kontakssessie. U sal voortgaan met u normale rehabilitasie soos gegee deur u plaaslike fisioterapeut. Weereens sal sommige deelnemers telefonies gekontak word om twee addisionele oefensessies van veertig minute by te woon wat deur die navorser behartig sal word.

Ses (6) weke na u operasie sal u die vraelyste en fisiese toetse herhaal. Die proses sal weer herhaal word twaalf (12) weke na chirurgie. U plaaslike fisioterapeut sal vooraf ingelig word oor wat die studie behels. Die deelnemers wat gekontak word vir addisionele oefeninge, sal gevra word om dit nie te deel met mede-deelnemers of die plaaslike fisioterapeut nie.

U sal op geen manier benadeel word in terme van standaard fisioterapeutiese behandeling nie, indien u sou onttrek van die studie.

IS DAAR ENIGE WAARSKUWINGS OF VOORSKRIFTE BETREFFENDE U DEELNAME AAN DIE STUDIE?

U kan slegs deelneem aan die studie indien u pyn in die knie of heup reeds vir drie maande of langer ondervind.

Daar sal van u verlang word om nie, vir ten minste 3 ure voor die vraelyste invul of fisiese toetse uitvoer, alkohol in oormaat te drink, of 'n oormaat pyntablette in te neem nie.

Geen finansiële bydrae sal verlang of gegee word vir die moontlike addisionele oefeningssessiestydens die verloop van die studie nie.

Die studie sal geen risiko's anders as wat normaalweg tydens totale gewrigsvervanging chirurgie van toepassing is byvoeg nie. Psigologiese spanning wat moontlik ervaar kan word tydens sommige van die oefeninge sal tot 'n minimum gehou word deur konstante instruksies deur die navorser en verbale toestemming vanaf u tydens die oefeninge.

VERTROULIKHEID

Alle inligting wat versamel word tydens die studie is vertroulik. Data kan moontlik gepubliseer word in 'n wetenskaplike joernaal, maar geen inligting wat u as deelnemer kan identifiseer, sal gebruik word nie. Die inligting aangaande u toetsresultate en gesondheid wat tydens die studie verkry word sal streng vertroulik hanteer word. U sal in kennis gestel word indien enige inligting verkry word wat belangrik is vir u gesondheid. Geen derde party, behalwe u orthopediese chirurg, sal in kennis gestel word sonder u skriftelike toestemming nie. Die enigste uitsondering is wanneer die navorser volgens wet die individu moet rapporteer oor gevaarlike oordraagbare siektes. In so 'n geval sal u in kennis gestel word van die navorser se voorneme om die inligting te openbaar aan die betrokke instansie.

8.4 Appendix 4

Ingeligte Toestemming (Afrikaans)

Navorser: Ruan Mocke

“The efficacy of pain neuroscience education in combination with cognitive-targeted exercise therapy in total joint arthroplasty: A randomised controlled trial.”

INGELIGTE TOESTEMMING

- Hiermee bevestig ek dat ek ingelig is deur die navorser oor die aspekte, gedragskodes, voordele en risikos aangaande die kliniese studie. Ek het die bostaande brosjure aangaande die kliniese studie gelees en verstaan (Deelnemer inligtings brosjure en ingeligte toestemming) aangaande die kliniese studie.
- Ek is bewus daarvan dat die resultate van die studies anoniem gedokumenteer word in ‘n kliniese verslag.
- Ek is bewus daarvan dat ek op enige stadium tydens die studie my toestemming mag terugtrek.
- Ek is bewus dat inligting aangaande my "Body Mass Index" (lengte en gewig) vanaf my ortopediese chirurg verkry sal word.
- Ek het voldoende tyd gehad om vrae te vra en ek verklaar myself gewillig om aan die studie deel te neem.

Deelnemer naam: _____

(Drukskrif asseblief)

Deelnemer handtekening _____

Datum _____

Ek, die navorser, bevestig hiermee dat die bostaande deelnemer volledig ingelig is aangaande die kliniese studie.

Navorser _____

Navorser handtekening _____ Datum _____

Ek waardeer u hulp opreg.

8.5 Appendix 5

Socio-demographic Questionnaire

Date: _____

Code: _____

Socio-demographic questionnaire

ED EDEX

B

Office use

Please complete the questions by giving the correct answer to each.

1 Age

Please complete the following questions by marking the correct answer with an X.

2 Gender

Male	Female
------	--------

3 Pensioner

Yes	No
-----	----

4 Ethnicity

Black	Caucasian	Asian	Other
-------	-----------	-------	-------

5 Marital Status

Single	Married	Divorced	Widow	Widower
--------	---------	----------	-------	---------

6 Education level

Unschoolled	Junior School	High School	University	Post - Graduate
-------------	---------------	-------------	------------	-----------------

7 Smoker

Yes	No
-----	----

If **Yes** how many per day 8 Do you consume alcohol

Yes	No
-----	----

If **Yes** how many glasses per week 9 Do you suffer from Diabetes

Yes	No
-----	----

10 Do you suffer from Cholesterol

Yes	No
-----	----

11 Have you been diagnosed with any vascular problems?

Heart Disease	Impaired Circulation	None
---------------	----------------------	------

Researcher: Ruan Mocke

8.6 Appendix 6

WOMAC Osteoarthritis Questionnaire

WOMAC Osteoarthritis Questionnaire

ED **EDEX**
B/H/ 6/12

1. The following questions concern the amount of pain you are currently experiencing in your knee/hip. For each situation, please enter the amount of pain you have experienced in the past 48 hours.

	None	Mild	Moderate	Severe	Extreme
A. Walking on a flat surface	0	1	2	3	4
B. Going up or down stairs	0	1	2	3	4
C. At night while in bed	0	1	2	3	4
D. Sitting or lying	0	1	2	3	4
E. Standing upright	0	1	2	3	4

For office use only

2. Pain the last 48hrs?

	None	Mild	Moderate	Severe	Extreme
A. Right knee/hip	0	1	2	3	4
B. Left knee/hip	0	1	2	3	4

3. How severe is your stiffness after first awakening in the morning?

	None	Mild	Moderate	Severe	Extreme
	0	1	2	3	4

4. How severe is your stiffness after sitting, lying, or resting later in the day?

	None	Mild	Moderate	Severe	Extreme
	0	1	2	3	4

5. The following questions concern your physical function. By this we mean your ability to move around & to look after yourself. For each of the following activities, please indicate the degree of difficulty you have experienced in the last 48 hours, in your knees.

What degree of difficulty do you have with:

	None	Mild	Moderate	Severe	Extreme
A. Descending (going down) stairs	0	1	2	3	4
B. Ascending (going up) stairs	0	1	2	3	4
C. Rising from sitting	0	1	2	3	4
D. Standing	0	1	2	3	4
E. Bending to floor	0	1	2	3	4
F. Walking on a flat surface	0	1	2	3	4
G. Getting in/out of car	0	1	2	3	4
H. Going shopping	0	1	2	3	4
I. Putting on socks/stockings	0	1	2	3	4
J. Rising from bed	0	1	2	3	4
K. Taking off socks/stockings	0	1	2	3	4
L. Lying in bed	0	1	2	3	4
M. Getting in/out of bath	0	1	2	3	4
N. Sitting	0	1	2	3	4
O. Getting on/off toilet	0	1	2	3	4
P. Heavy domestic duties (mowing the lawn, lifting heavy grocery bags)	0	1	2	3	4
Q. Light domestic duties (such as tidying a room, dusting, cooking)	0	1	2	3	4

8.7 Appendix 7

Brief Pain Inventory (Short Form)

Appendix 7

Researcher: Ruan Mockè

Date:
Code:

Brief Pain Inventory (Short Form)

ED **EDEX**
B/H/ 6/12

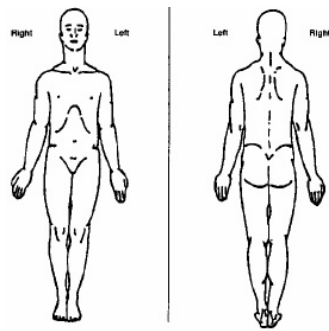
1. Throughout our lives, most of us have had pain from time to time (such as minor headaches, sprains & toothaches). Have you had pain other than these everyday kinds of pain today?

Yes	No
-----	----

Office use

--

2. On the diagram, shade in the areas where you feel pain. Put an X on the area that hurts the most.



3. Please rate your pain by marking the box beside the number that best describes your pain at its **worst** in the last 24 hours.

0	1	2	3	4	5	6	7	8	9	10
---	---	---	---	---	---	---	---	---	---	----

No Pain

Pain as bad as you can imagine

--

4. Please rate your pain by marking the box beside the number that best describes your pain at its **least** in the last 24 hours.

0	1	2	3	4	5	6	7	8	9	10
---	---	---	---	---	---	---	---	---	---	----

No Pain

Pain as bad as you can imagine

--

5. Please rate your pain by marking the box beside the number that best describes your pain on the **average**.

0	1	2	3	4	5	6	7	8	9	10
---	---	---	---	---	---	---	---	---	---	----

No Pain

Pain as bad as you can imagine

--

6. Please rate your pain by marking the box beside the number that tells how much pain you have **right now**.

0	1	2	3	4	5	6	7	8	9	10
---	---	---	---	---	---	---	---	---	---	----

No Pain

Pain as bad as you can imagine

--

7. What treatments or medications are you receiving for your pain?

8. In the last 24 hours, how much relief have pain treatments or medications provided? Please mark the box below the percentage that most shows how much **relief** you have received.

0%	10%	20%	30%	40%	50%	60%	70%	80%	90%	100%
----	-----	-----	-----	-----	-----	-----	-----	-----	-----	------

No Relief

Complete Relief

9. Mark the box beside the number that describes how, during the past 24 hours, pain has interfered with your:

A. General Activity

0	1	2	3	4	5	6	7	8	9	10
---	---	---	---	---	---	---	---	---	---	----

Does not interfere

Completely interferes

B. Mood

0	1	2	3	4	5	6	7	8	9	10
---	---	---	---	---	---	---	---	---	---	----

Does not interfere

Completely interferes

C. Walking ability

0	1	2	3	4	5	6	7	8	9	10
---	---	---	---	---	---	---	---	---	---	----

Does not interfere

Completely interferes

D. Normal Work (includes both work outside the home and housework)

0	1	2	3	4	5	6	7	8	9	10
---	---	---	---	---	---	---	---	---	---	----

Does not interfere

Completely interferes

E. Relations with other people

0	1	2	3	4	5	6	7	8	9	10
---	---	---	---	---	---	---	---	---	---	----

Does not interfere

Completely interferes

F. Sleep

0	1	2	3	4	5	6	7	8	9	10
---	---	---	---	---	---	---	---	---	---	----

Does not interfere

Completely interferes

G. Enjoyment of life

0	1	2	3	4	5	6	7	8	9	10
---	---	---	---	---	---	---	---	---	---	----

Does not interfere

Completely interferes

(Keller *et al.*, 2004)

8.8 Appendix 8

Pain Catastrophising Scale

Appendix 8

PC Scale

Code: _____

Date: _____

Researcher: Ruan Mockè

ED **EDEX**
B/H/ 6/12

Everyone experiences painful situations at some point in their lives. Such experiences may include headaches, tooth pain, joint or muscle pain. People are often exposed to situations that may cause pain such as illness, injury, dental procedures or surgery.

Instructions:

We are interested in the types of thoughts and feelings that you have when you are in pain. Listed below are thirteen statements describing different thoughts and feelings that may be associated with pain. Using the following scale, please indicate the degree to which you have these thoughts and feelings when you are experiencing pain.

Meaning	Rating
Not at all	0
To a slight degree	1
To a moderate degree	2
To a great degree	3
All the time	4

Statement	When I'm in pain ...	Rating	Office use only
1	I worry all the time about whether the pain will end.		<input type="text"/>
2	I feel I can't go on.		<input type="text"/>
3	It's terrible and I think it's never going to get any better		<input type="text"/>
4	It's awful and I feel that it overwhelms me.		<input type="text"/>
5	I feel I can't stand it anymore		<input type="text"/>
6	I become afraid that the pain will get worse.		<input type="text"/>
7	I keep thinking of other painful events		<input type="text"/>
8	I anxiously want the pain to go away		<input type="text"/>
9	I can't seem to keep it out of my mind		<input type="text"/>
10	I keep thinking about how much it hurts.		<input type="text"/>
11	I keep thinking about how badly I want the pain to stop		<input type="text"/>
12	There's nothing I can do to reduce the intensity of the pain		<input type="text"/>
13	I wonder whether something serious may happen.		<input type="text"/>

(Sullivan, 1995)

8.9 Appendix 9

Tampa Scale for Kinesiophobia

Tampa Scale for Kinesiophobia

Code: _____

Date: _____

Descriptions:

1 = strongly disagree

2 = disagree

3 = agree

4 = strongly agree

ED EDEX

B/H/ 6/12

Office use only

1. I'm afraid that I might injury myself if I exercise	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input type="checkbox"/>
2. If I were to try to overcome it, my pain would increase	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input type="checkbox"/>
3. My body is telling me I have something dangerously wrong	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input checked="" type="checkbox"/>
4. My pain would probably be relieved if I were to exercise	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input type="checkbox"/>
5. People aren't taking my medical condition seriously enough	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input type="checkbox"/>
6. My accident has put my body at risk for the rest of my life	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input type="checkbox"/>
7. Pain always means I have injured my body	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input checked="" type="checkbox"/>
8. Just because something aggravates my pain does not mean it is dangerous	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input type="checkbox"/>
9. I am afraid that I might injure myself accidentally	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input type="checkbox"/>
10. Simply being careful that I do not make any unnecessary movements is the safest thing I can do to prevent my pain from worsening	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input type="checkbox"/>
11. I wouldn't have this much pain if there weren't something potentially dangerous going on in my body	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input type="checkbox"/>
12. Although my condition is painful, I would be better off if I were physically active	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input checked="" type="checkbox"/>
13. Pain lets me know when to stop exercising so that I don't injure myself	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input type="checkbox"/>
14. It's really not safe for a person with a condition like mine to be physically active	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input type="checkbox"/>
15. I can't do all the things normal people do because it's too easy for me to get injured	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input type="checkbox"/>
16. Even though something is causing me a lot of pain, I don't think it's actually dangerous	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input checked="" type="checkbox"/>
17. No one should have to exercise when he/she is in pain	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input type="checkbox"/>

(Burwinkle *et al.*, 2005)

*What actions are you most fearful of doing, or which activities do you avoid during your everyday activities? _____

*Not part of the Tampa Scale of Kinesiophobia Questionnaire

8.10 Appendix 10

Standardised Musculoskeletal Rehabilitation Program (Hip)

Total Hip Arthroplasty

Pre-rehabilitation

GOALS FOR THE PERIOD: Improve strength and balance of lower extremities

Exercises when indicated

1. Closed-chain exercises (progression to gym equipment and inclined sled): step-ups, mini-squats, heel raises, SLRs, and hip abduction 3 x 15
2. Pool therapy (30 minutes)
3. Treadmill (as part of gym program) 5 – 60 minute walk
4. Heel cord stretches 3 x 30 seconds

Days 1-2 (in hospital)

GOALS FOR THE PERIOD: Protect healing tissues, prevent postoperative complications, and improve volitional control of involved lower extremity (LE)

Precautions

Provide patient education regarding total hip arthroplasty precautions
Evaluate cough effort for effectivity and productivity

Isometric Exercises when indicated

1. Gluteal sets 5 x 5 second hold (3 times daily)
2. Quadriceps sets 15 x 5 second hold (3 times daily)

Active Range of Motion (AROM) Exercises when indicated

3. Ankle pumps 1 minute (5 times daily)

Day 2 Mobility when indicated

4. Mobilise patient to sitting 30 minutes (in appropriate chair)
5. Transfer training (Supine-to-sitting, sitting-standing-sitting)
6. Gait training 5 – 15 meters

Days 3-7 (in hospital)

GOALS FOR THE PERIOD: Improve lower-extremity (LE) and upper-extremity (UE) strength

AROM Exercises when indicated (initiated in supine – progressed to in standing)

1. Heel slides 3 x 12 (3 times daily)
2. Hip abduction 3 x 12 (3 times daily)
3. Terminal knee extension 15 x 5 second hold (3 times daily)

Resistive Exercises when indicated (Seated)

4. Resisted shoulder internal rotation (IR) and external rotation (ER) with Theraband 3 x 12 (3 times daily)
5. Shoulder depressions and triceps dips while seated 15 x 5 second hold (3 times daily)

Mobility Exercises when indicated

6. Initiate stair training when indicated x 3
7. Initiate car transfer training x 3
8. Object pick-up training x 3
9. Progressive gait training 15 – 50 meters

Weeks 1-6 (after discharge to home setting or as appropriate in interim setting)

GOALS FOR THE PERIOD: Improve strength and balance of lower extremities; promote return to activities and hobbies as indicated

Exercises when indicated

1. Closed-chain exercises (progression to gym equipment and inclined sled): step-ups, mini-squats, heel raises, SLRs, and hip abduction 3 x 15
2. Pool therapy when indicated by surgeon (30 minutes)
3. Treadmill (as part of gym program) 5 – 60 minute walk
4. Heel cord stretches 3 x 30 seconds

(Maxey and Magnusson, 2007)

8.11 Appendix 11

Standardised Musculoskeletal Rehabilitation Program (Knee)

Total Knee Arthroplasty

Pre-rehabilitation

GOALS FOR THE PERIOD: Improve strength and balance of lower extremities

Exercises when indicated

1. Closed-chain exercises (progression to gym equipment and inclined sled): step-ups, mini-squats, heel raises, SLRs, and hip abduction 3 x 15
2. Pool therapy (30 minutes)
3. Treadmill (as part of gym program) 5 – 60 minute walk
4. Heel cord stretches 3 x 30 seconds

Days 1-2 (in hospital)

GOALS FOR THE PERIOD: Protect healing tissues, prevent postoperative complications, and improve volitional control of involved lower extremity (LE)

Precautions

Provide patient education regarding total knee arthroplasty precautions
Evaluate cough effort for effectivity and productivity

Isometric Exercises when indicated

1. Gluteal sets 5 x 5 second hold (3 times daily)
2. Quadriceps sets 15 x 5 second hold (3 times daily)

Active Range of Motion (AROM) Exercises when indicated

3. Ankle pumps (5 times daily)

Day 2 Mobility when indicated

4. Mobilise patient to sitting 30 minutes (in appropriate chair)
5. Transfer training (Supine-to-sitting, sitting-standing-sitting)
6. Gait training 5 – 15 meters
7. Passive knee flexion 10 x 5 second hold

Days 3-7 (in hospital)

GOALS FOR THE PERIOD: Improve lower-extremity (LE) and upper-extremity (UE) strength

AROM Exercises when indicated (initiated in supine – progressed to in standing)

1. Heel slides 3 x 12 (3 times daily)
2. Hip abduction 3 x 12 (3 times daily)
3. Terminal knee extension 15 x 5 second hold (3 times daily)
4. Knee flexion 15 x 5 second hold (3 times daily)

Resistive Exercises when indicated (Seated)

5. Resisted shoulder internal rotation (IR) and external rotation (ER) with Theraband 3 x 12 (3 times daily)
6. Shoulder depressions and triceps dips while seated 15 x 5 second hold (3 times daily)

Mobility Exercises when indicated

7. Initiate stair training when indicated x 3
8. Initiate car transfer training x 3
9. Object pick-up training x 3
10. Progressive gait training 15 – 50 meters
11. Knee flexion to 80° (Seated)

Weeks 1-6 (after discharge to home setting or as appropriate in interim setting)

GOALS FOR THE PERIOD: Improve strength and balance of lower extremities; promote return to activities and hobbies as indicated

Exercises when indicated

1. Closed-chain exercises (progression to gym equipment and inclined sled): step-ups, mini-squats, heel raises, SLRs, and hip abduction 3 x 15
2. Treadmill (as part of gym program) 5 – 60 minute walk
3. Heel cord stretches 3 x 30 seconds

(Maxey and Magnusson, 2007)

8.12 Appendix 12

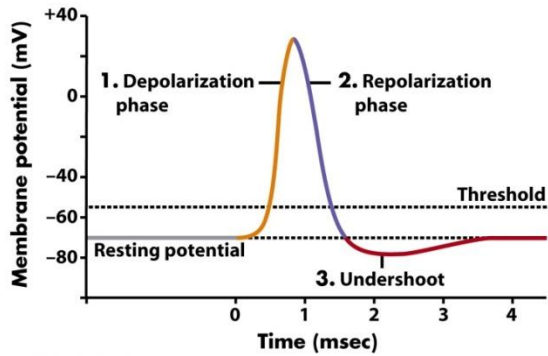
Pain Neuroscience Education Flashcards

The following flashcards were used to explain the physiology of pain through pain neuroscience education.

The flashcards consisted of a visual explanation (front), which was supported by a verbal explanation (information on the back)

Front

1



Back

- 400 individual nerves, 72km of nerves.
- All connected
- Like alarm systems
 - When threshold is reached message is sent to brain
- Rusted nail?
 - Yes, tetanus, take nail out etc.
- So alarm system goes off and sends message to spinal cord and ultimately the brain
- Explain nerve as a high jump athlete
 - All or nothing
- Increased resting potential

2

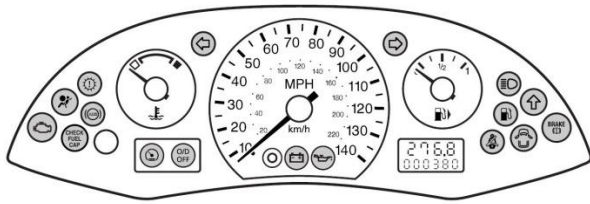


- Christmas tree
 - Lights are on sending message its Christmas
 - Abnormal time of year for Christmas lights
- Pain is normal



- 25% persons alarm system rests only slightly under firing level, instead of returning to rest.
 - Extra sensitive nerves
- This is not abnormal but impedes on activities and function
- Driving example
- Since you have pain now – the same activity you can only do 5 minutes
 - Initially normal –
 - Prolonged sensitive nerves most probable cause in persistent pain
- Another example – sensitive security beams

4



- When pain develops your nerves increase in sensitivity to protect you.
- Most sensors change over every 2-3 days
- influenced by your understanding of pain.
- Recognising stimuli that increase your sensitivity and acting to decrease their effect.
- Gentle movement
- Oxygen in area
- Relaxation – deep breathing
- Knowledge helps
 - Cold wind blows

5



- Working in the garden
 - Attention
- Pain but no tissue damage
- Many patients pain started without an injury.
- Pain can be developed due to emotional overload or stress.

6



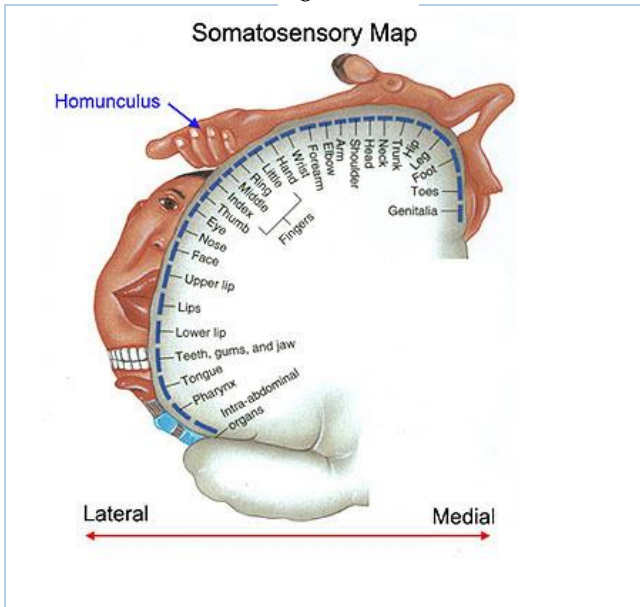
- All areas that are painful have extra sensitive nerves.
- Nerves pass from sight to spinal cord to brain sending information so something can be done/decided.
- The brain makes the decision – rub the area, go to doctor etc.
- Over analyzed.
- Example - Think of a large office building – top floor is CEO and downward individual divisions (body parts).

7



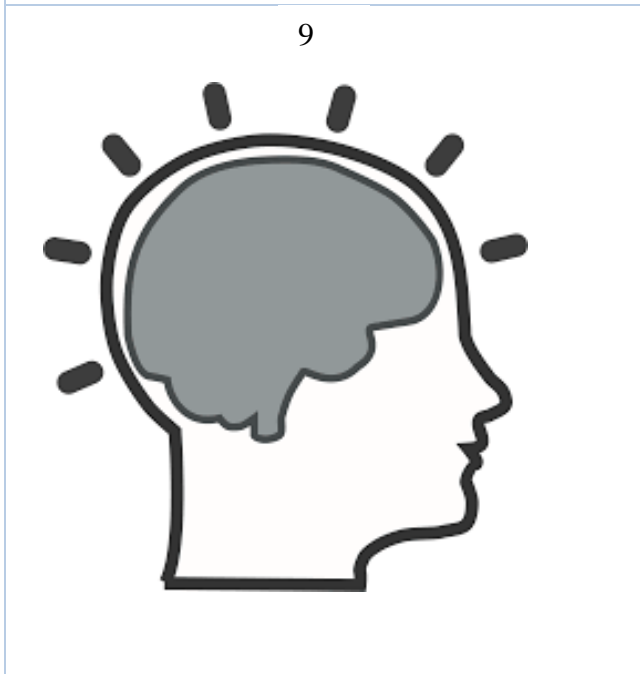
- House alarm goes off
- When your nerves fire-up in the area of injury
- Alarm goes off continually
- Immune molecules through your body.

8



- Example: eyes closed – finger to nose and finger.
- Healthy people have clear well defined maps
- Loss of physical body part
- Gentle, easy, repetitive movement and understanding pain is helpful in defining body parts on the maps.

9



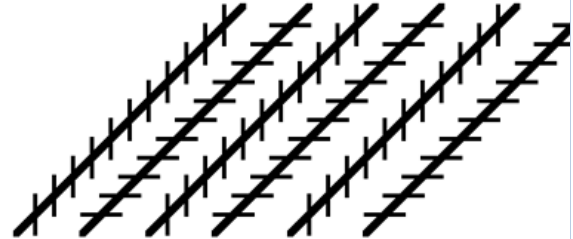
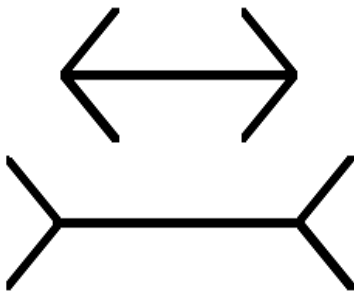
- Meeting to discuss if pain needs to be experienced or not.
- Airline map in the back of the airline magazine
- No brain no pain.

10



- Example: Dirt road on farm.
- Constant pain, anxiety and fear
- The message moves with greater efficiency.
- Thus the pain you experience is not an indication of how bad the injury is.
- Nerves that fire together wire together.



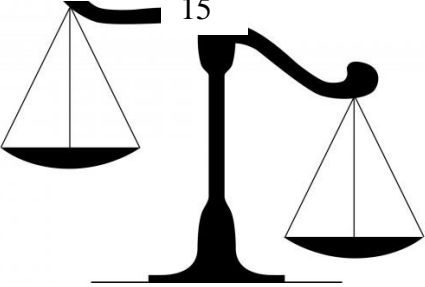
11



12



- The brain drug cabinet.
- Example: Soldiers who have been shot and don't feel pain.
- Wet brain (tap over brain).
- Decreased numbing chemicals to get more information about the area.
- Is this dry brain permanent – n o- it can be changed.
- Knowledge, Aerobic exercise, Medication, Foods

<p>13</p> 	<ul style="list-style-type: none"> ○ Ever wondered why people recover so differently after the same incident ○ Example: Ankle sprains ○ Environment ○ stress hormones ○ Environments can also impact positively
<p>14</p> 	<ul style="list-style-type: none"> ○ Example: Ankle sprains- sometime become purple and swollen ○ These chemicals cause arteries to open up more-swelling, warmth, redness. Also immune molecules to investigate. ○ More stress – more ○ Stay positive after surgery!
<p>15</p> 	<ul style="list-style-type: none"> ○ If a lion jumps into the room. ○ Big muscle made to move make you move. ○ Breathing increases – digesting your lunch isn't important. ○ Pain is represented as a lion in your body – a threat ○ Temporary versus constant

(Louw and Puentedura, 2013)

8.13 Appendix 13

Pain Neuroscience Education Guideline for Researcher use only

Pain Neuroscience education for patients (Louw and Puentedura, 2013)

1. Alarm system, nails in foot and Christmas trees

1.1 Aim

Nerve education

1.2 Intervention

- 400 individual nerves, 72km of nerves.
- All nerves are connected via the brain
- Nerves have electricity going through them, similar to alarm systems
 - When threshold (action potential) is reached message is sent to brain
- If you step on a rusted nail, would you want to know about it?
 - Yes to protect yourself whether it is to get a tetanus shot, removing the nail etc.
- Alarm system goes off and sends message to spinal cord and ultimately the brain
 - Thereafter, the alarm system resets to be able to send info on other issues
- Christmas tree
 - Lights are on sending message its Christmas
 - After Christmas husband comes down stairs and sees tree is on and its a week after Christmas
 - Put off those lights – after a couple of commands you realise it isn't Christmas anymore and put it away till next year.

1.3 Images to use

- Action potentials
- Foot onto rusted nail
- Christmas tree
- Full body nervous system

2. Waking up the alarm system

2.1 Aim

Pain is not related to health of tissues, rather to increased nerve sensitivity.

Injuries will heal, thus pain is more related to sensitive nervous system.

2.2 Intervention

- Recap 1.1
- 25% of peoples alarm systems get activated after threat, but the alarm system rests only slightly under firing level, instead of returning to rest.
 - Extra sensitive nerves
- This is not abnormal but impedes on activities and function.
- Usually after, for instance two hours of driving the nerves start activating saying:”I am tired, I have a sore bum” etc.
 - After climbing out the alarm resets for another two hours
- Since you have pain – the same activity could become uncomfortable again after 5 minutes
 - Initially normal – but tissue heals- sensitive nerves most probable cause in persistent pain
- Another example – to sensitive motion detector beams that are set off by leaves cats etc.

2.3 Questions

1. How do healthcare providers know the alarm system is extra sensitive?
2. Why did nerves stay so extra sensitive and not calm down?
3. What can be done to calm the nerves?

1. How do healthcare providers know the alarm system is extra sensitive?

This is a common question that arises, especially if other tests, radiological or blood tests, comes back showing nothing. Listen to your own story – you’ve slowed down, you’ve become sensitive to movement, pressure etc.

Sensitive nerves are picked up by using physical movement tests. Positive reactions to medications such as anti-depressants and membrane stabilisers indicate sensitive nerves.

2. Why did nerves stay so extra sensitive and not calm down?

This is a common question that arises, especially if the patient has come into contact with a person that has a similar condition, yet feels different.

Extra sensitive nerves are normal – everyone gets them, some calm down quicker than others. Some external factors influence the calming if they are present during your pain. These factors include failed treatments, family issues, levels of fear, job concerns and different pain explanations to name a few.

After trying numerous different approaches you worry that nothing is working. The brain has no intention then to calm you sensitive nerves down due to your concerns.

3. What can be done to calm the nerves?

Knowledge about why the nerves have stayed extra sensitive helps to calm the pain down. To calm the nerves down blood flow, oxygen and medication is needed. Research shows persons that understand pain decreases nerve sensitivity.

Blood flow and oxygen can be improved with aerobic exercise, there is no need to do the comrades to achieve this.

Medication: Pharmaceutical drugs prescribed by your doctor

We will talk about a very powerful drug. The brain produces the most potent medicine known on the planet, helping people survive severe injuries while experiencing little or no pain.

The release of this medicine can be described as a wet brain with plenty of juice – whereas persons with persistent pain have dry brains. Aerobic exercises helps for a wetting the brain, and the knowledge of pain wets the brain.

2.3 Images to use

- Brain with flags – blank so patient can fill in own stressors

3. Nerve sensors

3.1 Aim

Nerves have the biological capacity to become sensitive to non-noxious stimuli – example cold weather. Pain has its bases on the perception of threat.

3.2 Intervention

Your body's alarm system is very complicated, an indication thereof is sensitivity to cold for instance.

Inside nerves are sensors, called receptors, which protect and inform you of changes in your environment.

Temperature:

- Sensors for changes in temperature
- Sensitive to cold weather

Stress:

- Sensors sensitive to stress chemicals flowing in your blood.
 - The more stressed, anxious, nervous or upset the more you will feel pain.

Blood flow:

- Sensors sensitive to the amount of blood around your tissues.
- Sitting too long and decrease blood flow – sensors activate and nerves become sensitive

Movement and pressure:

- Sensors sensitive to movement and pressure
- Movement of an injured area may cause pain

Immunity:

- When sick or have flu many immune molecules.
- The same when injured – immune response.
- Immune molecules can make you ache

When pain develops your nerves increase in sensitivity to protect you.

The good news is that these nerves change, and can change for the better. Most sensors get replaced by new sensors every 2-3 days. The amount of sensors that is replaced is heavily influenced by your understanding of pain. Recognising stimuli that increase your sensitivity and acting to decrease their effect helps to decrease your nerve's sensitivity.

- Gentle movement
- Oxygen in area
- Relaxation – deep breathing

The knowledge helps when the cold wind blows over a recent surgical area that would make the nerves sensitive – the brain will know its only cold wind – no reason to sound the alarm – the effect thereof will decrease over time.

3.3 Images to use

Car dashboard with warning lights

4. Speeding buses hurt more than ankle sprains

4.1 Aim

Injury and pain is not synonymous with each other.

Pain is an output of the brain and not an input.

4.2 Intervention

If you sprained your ankle would it hurt? All will probably say yes.

What if you sprain your ankle as you cross the road? And you see out of the corner of your eye a speeding bus coming towards you. Does the ankle still hurt? No

The pain in the ankle will cause you to fall down – the brain rather says: "Get out of here!"

When you are working in the garden – don't you sometimes see that there is blood on your leg/arm and you don't know when it happened? The danger of the scrape was overruled by the joy of the garden, sunshine or labour.

*Image of a man's head with a nail in.

The nail penetrated the gentleman's head four years prior to the x-ray being taken. Busy construction site – not realising nail went into his head.

Many people have pain but no tissue damage can be found. Tissues don't have to be injured before pain can be experienced. As the brain evaluates all threats, whether stress, anxiety or tissue damage to make a decision to defend you, sometimes using pain. Many patients pain started without an injury. Pain can also be developed due to emotional overload or stress.

4.3 Images to use

Ankle sprain and speeding bus weight

X-ray with nail in head

5. CEO making big decisions

5.1 Aim

Explain concept of central sensitisation, increase in widespread sensitisation and action potential windup. Explanation of the plasticity of the central nervous system.

5.2 Intervention

All areas that are painful have extra sensitive nerves. Nerves pass from the noxious sight to the spinal cord, then to the brain. The sent information is then processed and an appropriate response is then implemented. The decision the brain makes varies from rubbing the area, going to the doctor or panicking.

When pain persists, and there are many concerns around the area, the brain will analyse the incoming messages very closely. An example of a modern day over reaction is when you press x on your computer keyboard and “xxxxxxxxxxxxxxxxxxxxx” appears on the monitor.

Think of a large office building, the CEO as at the top floor, with another division of the company on every floor under the top floor. This is an illustration of how your brain and the body parts work. Normally only monthly reports are sent to the CEO. When there is trouble in a specific division, the CEO wants daily reports, until the problem is resolved (extra sensitive nerves).

When the report is on the CEO desk, it gets analysed with a magnifying glass for the best action to protect the company. If the technology division is in trouble – the CEO will want more information from the technology division. Thus your body may feel pain in areas associated with the area where nerves are sensitive, thus making them sensitive.

5.3 Images

Large office block with CEO on top.

Action potential windup – indicating amplification in information.

6. Nosy Neighbours

6.1 Aim

Explaining how pain can develop by spreading neural sensitisation. Pain in remote areas is more due to spreading sensitisation than injury.

6.2 Intervention

If your house alarm goes off your neighbours will be curious why, they might be concerned if you are ok, and some may come and see if all is in order.

When your nerves fire-up in the area of injury – nerves in other areas react in the same way as your neighbours.

If your alarm goes off continually your neighbours might get aggravated or angry. The same with nerves in remote areas. These nerves may become sensitive, resulting in remote aches and pains.

If the problem persists the police is phoned and asked to intervene. Likewise your body releases immune molecules through your body. The police will go door-to-door, thus nerve to nerve to make sure all is well or to get statements. The presence of the police makes the situation more tense. Remember that “old crime areas”, previous surgery areas will be checked by the police, which in return may result in agitated nerves.

6.3 Images to use

Body chart for drawing spreading pain.

Police knocking at the door.

7. Little people in your head

7.1 Aim

Explain somatosensory cortex to patients and the allocation of body parts.

Explain use it or lose it principle and thus promote movement.

Explain CRPS and phantom limb pain.

7.2 Intervention

Have you wondered how people experience pain in a body part that doesn't exist?

Several years ago scientist stimulated various body parts in animals and then in humans and found something interesting. They found a body mass in your brain representing each part of your body.

Experiment: eyes closed – one finger to your nose and then again to the finger on the other hand. This technique is based on the map in your brain.

Healthy people have clear well defined maps, whereas pain causes fuzziness (out of focus) of the maps. When the body part is painful it is moved less. Movement is necessary to keep these maps sharp. Movement is thus a key component in recovery.

Gentle, easy, repetitive movement and understanding pain is helpful in defining body parts on the maps. In situations where it's too painful to move, you can close your eyes and imagine you are moving the body part. These maps are thus fed by movements especially in repetitive movements in sport for example a golfers swing.

So if you lose a body part, the map is still there, but due to non-movement the map becomes fuzzy. The reaction of the brain then is to cause pain in that area to protect it. This is called phantom limb pain. As long as the brain is confused it will cause pain. Knowledge why you hurt helps to calm stressful and anxious nerves.

Visualisation, recognition of left and right, mirror and watching other people move helps to restore these maps.

7.3 Images to use

Somatosensory homunculus.

Mirror box images. Visual illusions used by Moseley (2003).

8. Airlines are flying through your head

8.1 Aim

Multiple brain areas are involved in nociception shown by a neurosignature

Other factors associated with pain can increase or initiate pain for example, memory, words and vision. The pain neural signature impacts function of other brain areas like memory, motor control, and concentration

8.2 Intervention

For many years people thought there was only a single area in brain dedicated to pain interpretation, but if it was so easy we could cut it out and all the person's pain would be gone.

Research has shown that multiple areas are involved. These areas connect and form a map in all pain experiences whether it is back pain, leg pain or head pain.

These areas are:

- **Sensation:** the brain tells you where you are experiencing sensation including pain.
- **Movement:** An area that plans, coordinate and execute movement is activated to protect you.
- **Focus and concentration:** Can become over active.
- **Fear:** emotional areas of brain especially in pain that's poorly understood. Fear, fear of injury, fear of movement are characteristics of the over sensitive emotional brain
- **Memory:** recall previous injury and implement strategies
- **Motivation:** area used to process pain instead of motivate
- **Stress responses:** specialised brain areas for stress. Controls release of adrenaline and cortisol – protect you. Also control sleep, appetite, body weight and body temperature

These areas discuss the appropriate action. It is like a meeting to discuss if pain needs to be experienced or not.

The map is almost like the airline map in the back of the airline magazine. As people fly different airplanes. So everybody's destination is the same but the pathway may differ.

I am not saying the pain is in your head (you are making it up), but I'm saying the pain is in your head – no brain no pain.

8.2 Images to use

Blank brain picture – draw in airlines or map
fMRI or neuromatrix

9. Driving the same road over and over and over...

9.1 Aim

Understand brain processing of pain – living in pain strengthens pathways and heightens sensitivity.

Explain why previous tasks were not painful and now are – and why so little activity causes pain.

9.2 Intervention

With the map – as pain heals the map slowly fades – people leave the meeting.

The longer the pain continues the more the map is used - dirt road on farm. If the traffic becomes great the municipality will decide to grade the road – causing you to be able to drive faster. The same with pain – initially it is a new single track road – the as it is used more it later becomes graded and pain is experienced faster. If pain, anxiety and fear constantly use the road it will be paved and permanently be placed on the country map.

If you put it into the pain perspective – you may have noticed you could walk for an hour before experiencing pain – that has now started to decrease. Another part is because the road has become paved – the message moves with greater efficiency.

Thus the pain you experience is not an indication of how bad the injury is. The brain pathway has just become better in interpreting and sending the messages quicker. Nerves that fire together wire together.

It is now believed that increase neural sensitivity causes persistent pain. Good news – there are ways to decrease this sensitivity.

9.3 Images to use

Blank brain and pathways

10. Believing everything you see

10.1 Aim

Pain is a response based on perception of threat- key word perception. Many patients have predisposed ideas about bulging disks and arthritis.

Show radiological findings with relation to pain.

10.2 Intervention

Pain is the process where the brain, based on everything it knows about your current situation, makes a conscious decision to defend you.

So let's take a look at the way your brain processes information regarding to what it knows

Show: two lines – which one is longer?

They are the same length – but why does the one look longer? Your brain uses its pre-knowledge of shapes and sizes and makes it a logical choice – even if it's wrong.

For example –if you have developed leg pain after this surgery – due to your previous knowledge and previous encounters, maybe the internet even you will experience the “same pain” – even though the structures have been removed.

10.3 Images to use

Visual illusions

11. Wet and dry brains

11.1 Aim

Let patients understand how the brain modulates pain. Descending inhibits danger messages, facilitation – increased sensitivity- central sensitisation may occur. Give patients strategies to help modulate pain.

11.2 Intervention

If you bump your toe it hurts. How long does it hurt? A few seconds and then it eases...how? The brain drug cabinet. It helps with survival and also normal. A lot of patients in the emergency rooms with little pain have greater injuries. Soldiers who have been shot and don't feel pain.

The process of helping with pain is called a wet brain (tap over brain). Thus chemicals can be flushed down to help manage pain. Long term pain unfortunately affects this drug cabinet negatively.

As the brain becomes more worried about the area it takes away the numbing chemicals to get more information about the area. Causing a dry brain (also a reason for increased sensitivity in activities).

Is this dry brain permanent – n o- it can be changed.

Knowledge: a lot of research shows more understanding lessens the pain and fear. Less fear and greater understanding will wet your brain.

Aerobic exercise: “runners high”. 10 min of moderate exercise calms the nerves more from the brain.

Medication: some patients with chronic pain get anti-depressants. Not because your Dr thinks you're depressed- but it helps open your medicine cabinet in the brain.

Foods: this is a loaded box – not going to sit on this. “Comfort food” comforts.

11.3 Images to use

Wet and dry brain.

X-rays with significant injury with little pain.

12. Injuries occur in a vacuum, right?

12.1 Aim

Environmental issues influence pain - good or bad.

12.2 Intervention

Ever wondered why people recover so differently after the same incident, ankle sprain, and joint replacement? There are a lot of factors, but a big one is the environment around the injury.

Sprains happen when you are happy or sad, or employed or out of money or happily married or going through a divorce...research shown the environment has a big impact on the degree of pain experienced. If you hurt yourself in a stressful time there is already stress hormones throughout the body- causing increase sensitivity quicker and possibly slower calming down. So environments can also impact positively.

Research shown children playing contact sport experiences less pain later in life. A bruise in rugby is a badge of honour after the match.

Demolition derby drivers. 1/3 of people from car accidents report long lasting pain. In a recent study demolition drivers – trying to write-off each other's cars. Get into 52 accidents per event – average events 30 – that's over 1500 accidents causing neck and body jolting, many crashes being at 70km/hr. – less than 10% experience long lasting pain.

The injuries stay real – the just see it as part of the activity, except the injury, and prepare for the next event. Less stressful environment.

12.3 Images to use

Demolition derby drivers.

People going through pain to finish event.

13. Inflammatory soup

13.1 Aim

Explain persistent inflammation, retrograde depolarisation as well as thoughts contributing to inflammation and neural sensitivity. Especially post-surgery patients.

13.2 Intervention

Ankle sprains- sometime become purple and swollen. The body releases chemicals after ligament strain to cause swelling and stiffness – so you don't walk on it. This is normal and the start of the healing process. Nerves in the area also release chemicals to aid in the defence.

These chemicals cause arteries to open up more-swelling, warmth, redness. Also immune molecules (nosy neighbours) to investigate.

So the more you stress about the ankle, the possibility of it staying swollen for longer is greater.

Stay positive after surgery!

13.3 Images to use

Swollen ankle

14. When Lions attack

14.1 Aim

Teach patient the stress response and the various protective mechanisms – pain is only one protective mechanism.

Biological understanding for sleep, mood swings and fatigue.

Broader sense understands CRPS and fibromyalgia, irritable bowel syndrome.

14.2 Intervention

If a lion jumps into the room. What will you do? Take a nap? Correct your posture? No – you have some key systems that help you develop the right stress response for survival. Adrenalin will pump through your body making it ready to react. Big muscle made to move make you move.

When a lion attacks you might say a few choice words. Breathing increases – digesting your lunch isn't important. So your pain is represented as a lion in your body – a threat. In order to defend you your brain will decide on a survival mechanism. The threats may be long: job, money, nothing helps, there is no hope etc.

What if the lion gets caught by a game ranger? When the lion is removed the body returns to normal. The system is thus only temporarily – not to run for prolonged periods of time. What happens if the threat stays for long periods? Thus a roaring lion every day.

Issues occur:

Sore muscles: Blood goes to strong muscles and not to small muscles – less stability – more pain?

Mood swings: stress hormones, cortisol, increase mood swings

Appetite changes: cortisol changes food intake and appetite.

Weight gain: cortisol effects the hypothalamus regulates hunger. Patients in pain move less.

Sensitive nerves: while the threat is there the nerves stay sensitive.

Sleep disturbances: Key areas of the brain used with restorative sleep are busy processing pain.

Posture issues: The brain doesn't worry about posture with a lion in the room.

Irritable bowel: stress pulls blood away from the GI tract – making the tract work harder.

Low libido: Sex is not on the top of your list when a lion is in the room

Fatigue: Stress chemicals cause fatigue

Focus and concentration issues: high levels cortisol change concentration abilities.

Depression: Cortisol is a leading cause in depression.

The good news is the tissues can be made a lot less sensitive. Muscles and so heal within days or weeks not months or years. Thus it's not the tissue that's the issue but the nerve. Stability is important – as they get less blood due to big muscles moving more blood. You have many systems in use to protect you – that's why you may have different issues going on. Especially if you are stressed, fatigued, weak and sensitive. The more you know about your pain and why you are experienced it – the more the lion becomes a cub. Thus less stress responses.

14.3 Images to use

A lion in a room

8.14 Appendix 14

Pain Neuroscience Education Pamphlet



Pain Neuroscience Pamphlet

-
- *Why do I hurt even though I injured myself so long ago?*
 - *Why is it every time the weather changes my pain seems to be affected?*
 - *Why does it seem that my pain has a mind of its own?*
 - *Is there anything I can do to help myself so that I can heal quicker?*
-



The good, the bad and the Christmas tree

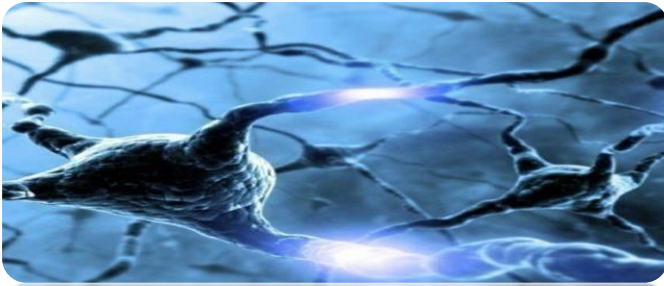
When you step on a rusty nail – pain is good.

It gives you the opportunity to realise you have injured yourself and take appropriate action. If you never felt pain from stepping on the rusty nail, you could find yourself with a severely infected foot a couple of hours later.

When pain becomes chronic – pain is bad.

Pain lasting beyond the normal timeframe for repair of the tissue involved falls into this category.

Thus pain at the appropriate time for the appropriate duration is what we are considering. For instance putting up a Christmas tree in December might be appropriate, but still having it up in the month of May is abnormal.



Speeding busses delay pain



The Boss – The Cops

Acquiring bruises without remembering how you got them is rather common. Ask any person that has spent an afternoon sprucing up their garden themselves. Scratches, scrapes and bumps can be found on your body, most of them happened unnoticed.

The eagerness and earnest you have to apply your green thumb into every flower bed causes certain information to be regarded as unnecessary to complete the task at hand.

The same applies to spraining your ankle when you cross the road, and at the same instant see a speeding bus hurtling towards you. Your brain decides – “get out of here” – instead of focusing on the ankle. Sure you will probably feel your ankle on the sidewalk, but that means your body considers your ankle more important than the sidewalk.

Consider your brain to be the CEO of your body.

As when in a company something happens that affects productivity, the CEO is notified and he decides what the appropriate steps to take are. He does this by acquiring constant information of the situation. Thus similarly the brain wants information from the site of injury, putting the nerves in the area on alert

The immune system steps into action in acute pain situations to protect your body from potential risk.

Later on, the inflammation causing abilities of the immune system can cause excessive inflammation. The immune system also goes to familiar sites where injuries have taken place before. This is why chronic pain sufferers commonly have multiple pain sites.



Stress hormones and Oil leaks

As mentioned before with an ankle sprain and speeding bus – the environment where you injure yourself plays a big role in your pain experience.

Injuring yourself whilst already in a stressful state, leads to an increased in stress hormone release in your body and causes nerves to become sensitive quicker. Thus a stressed CEO takes longer to calm down after a disturbance than a CEO at peace.

Constant stress causes a depletion of hormones that help regulate and modulate your pain experience.

Research suggests that one third of persons that are in motor vehicle accidents have chronic neck pain thereafter. Thus one accident equals a 30% chance of prolonged neck pain. Only 2,5% of demolition derby drivers that were in 1500 accidents had persistent neck pain. These drivers expect a crash and they don't stress about it.



The lion in the room

Chronic pain becomes like a lion in the room.

When a lion jumps into the room, a person does not worry about their posture before they run, or their breakfast being digested before they run, their heart rate goes up etc. Similar physiological responses are seen in chronic pain sufferers.

To tame the lion one needs to understand it:

Pain is a protective mechanism, thus if pain has surpassed normal healing timeframes the brain is overprotective of the area.

The brain seeks constant information to oversee the situation. This information bombardment causes nerve sensitivity that leads to increase pain experiences.

As your brain determines pain perception, gaining knowledge of why you have pain and doing appropriate graded exercises to assist in producing pain modulating anti-stress hormones can help you with your pain.

(Louw and Puentedura, 2013)

8.15 Appendix 15

Cognitive-targeted exercise therapy guidelines for Researcher use only

Cognitive-targeted exercise therapy

Goal setting SMART

Specific:

The specific aim was to assist the patient to do the movement they avoided or refrained from doing on a daily basis before surgery (see Table 16).

Examples of fear avoided actions in study individuals are:

- Climbing of the staircase at home
- Picking-up objects from the ground
- Using the toilet (rising from position)
- Walking long distances

Measurable:

Progression in exercise exposure was made measurable as follows:

- Non-weight bearing on stable surfaces
 - Examples:
 - Supine with participant pushing 65cm Physioball against the wall with legs
 - Supine with hips 90°, participant touches lateral ankle
 - Supine with cycling with legs in air
- Weight bearing on stable surfaces
 - Forward lunge progressed to weighted forward lunge
 - Wall squats progressed to free standing squats
 - One leg balance
- Functional activity on stable surfaces
 - Step climbing, 5cm to 25cm high
 - Golfers lift
 - Sit to stand 65cm high chair to 45 cm high chair
 - Step climbing as used in Chester step test format

The measurable outcome was the ability to perform the feared or avoided movement without hesitation (see Table 16)

Achievable:

- Actions that were a contra-indication for post total knee or hip arthroplasty was not considered for inclusion into the training regime
- Activities that would need longer than 12 weeks strengthening to achieve was not considered achievable in this study

Realistic:

- All activities had to be reasonable for a participant of that age and health level
- Activities that were contra-indicated for total hip or knee arthroplasty was considered unrealistic to achieve in this study

Time-targeted:

- Session one (prior to surgery): The role of the pain neuromatrix was explained in more detail. Explanation of motor imagery was explained. Completion of non-weight bearing stable surface cognitive targeted exercise therapy.
- Session two, three and four (Prior to surgery and six weeks post-surgery): Graded exposure of cognitive-targeted exercise therapy from weight bearing exercise on stable surfaces to functional feared activities on stable surfaces (see Table 15)

Pain neuromatrix explained

The pain neuromatrix was explained as through the extract of this studies literature review below:

“The novel idea of the pain neuromatrix was encouraged by four conclusions deduced from phantom limb pain observations (Melzack 2001). These conclusions were made because of the fact that, firstly, phantom limbs were described as actual and it may be deduced that what the body experiences as normal is sub-served by the same brain processing as the phantom limb. As the processing acts on and changes due to input from the body, they could also act and change separate from these inputs. The second conclusion described that information we normally experience from the body can be experienced in absence of these bodily inputs, thus the information is dependent on neural networks in the brain. Melzack (2001) suggested that stimuli or input from the body elicit activity in the neural networks but do not create them. Thirdly, the autonomous identification of the body can only be attributed to central processing, and not to the peripheral nervous system or the spinal cord. The fourth conclusion was that this acknowledgement of self, apart from other persons or environments, is a result of genetic specification, but could be altered by a person’s experience.”

Furthermore, the concepts within the body-self neuromatrix were explained by use of the figure A provided.

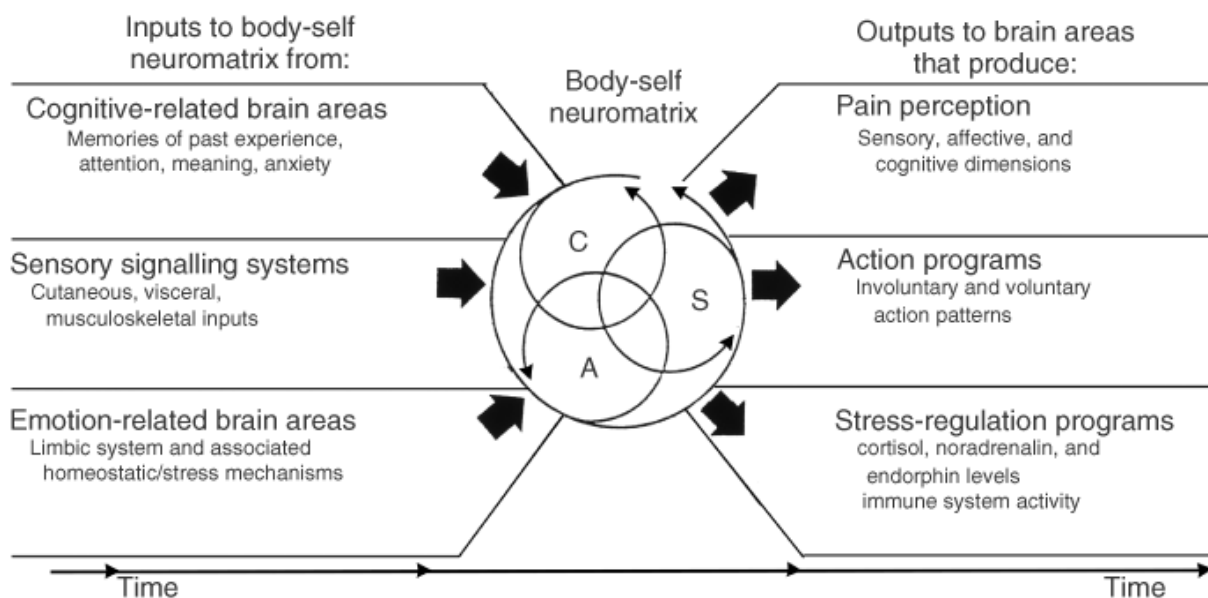


Figure A: Body-self neuromatrix

Graded activity

Graded activity aims to reinforce healthy behaviours and increase functional activities. The graded activity did not avoid painful activities, although the patient was also not allowed to “work through” the pain.

Graded exposure in vivo

Graded exposure focused on the added question, regarding feared or avoided movements, on the patients TSK-13 Scale. The exposure started with movements of the body parts as used in their feared movement. Thereafter, the exposure increases to more difficult and precise movements associated with activities the participant feared or avoided. Evaluation of the perception of the participant with regards to the exposure and its consequences was assessed before each progression. At that time any irrational and counterproductive beliefs were addressed with the aim to decrease the participants anxiety associated with the activity. The final step in graded exposure exercises was to complete the feared or avoided activity with confidence (see Table 16).

Self-control based interventions

The participant was urged to change behaviours regarding compensational postures and actions despite beliefs or thoughts that the action or posture is normal. The participant was also urged to improve their quality of life through movements, actions and activities instead of being hypervigilant in pain symptom reduction or activity avoidance.

Feared movement

Constant questioning throughout the implementation of cognitive-targeted exercise therapy was used to determine the amount of fear and anxiety the participant had, and to reassure the participant that they are safe and the environment is controlled.

Table 15 - CTET sessions

Session 1 (Prior to surgery)	Session 2 (Prior to surgery)	Session 3 (Six weeks post-surgery)	Session 4 (Six weeks post-surgery)
Pain neuromatrix explained	Non-weight bearing CTET on stable surfaces review	Weight bearing exercises on stable surface review	Functional feared/avoided activities initiated
Motor imagery explained	Motor imagery of graded exposure Cognitive-targeted exercise	Motor imagery of graded exposure Cognitive-targeted exercise	Motor imagery of graded exposure Cognitive-targeted exercise
Non-weight bearing CTET initiated on stable surface	Weight bearing exercises on stable surface	Functional feared/avoided activities initiated	Complete exposure to feared/avoided activity

Table 16 – Graded exposure examples

Feared/Avoided activity	Non-weight bearing on stable surfaces	Weight bearing on stable surfaces	Functional activities
Staircase climbing	Supine with participant pushing 65cm ball against wall	Forward lunge progressed to weighted forward lunge Wall squats progressed to free standing squats One leg balance	Step climbing progressed from 5cm to 25cm high step
Object pick-up from ground	Supine with hips flexed 90°, participant crunches and touches lateral ankle	Forward lunge progressed to weighted forward lunge Wall squats progressed to free standing squats One leg balance	Golfers lift progressed to semi squat pick-up
Rising from low seat	Supine with participant pushing 65cm ball against wall	Wall squats progressed to free standing squats	Sit-to-stand from 65cm high chair progressed to 45cm high chair Step climbing as used in Chester step test format
Walking distances	Supine with participant air-cycling with supported back	Forward lunge progressed to weighted forward lunge	12-minute walk test Step climbing as used in Chester step test format

(Macedo *et al.*, 2010; Louw and Puentedura, 2014)

8.16 Appendix 16

Consent from Medi-Clinic Swakopmund

06 October 2015

Mr R Mocke
PO Box 8433
Swakopmund
Namibia

E-mail: ruan.mocke.rm@gmail.com

cc: Jacqueline Groenewald
jacqueline.groenewald@mediclinic.co.za

Dear Ruan

**THE EFFICACY OF NEUROSCIENCE EDUCATION IN COMBINATION WITH COGNITIVE-TARGETED
EXERCISE THERAPY IN TOTAL JOINT ARTHROPLASTY: A RANDOMISED CONTROL TRIAL**

Please be advised that Mediclinic hereby approves the application for the above-mentioned research,
provided you obtain the necessary consent of the orthopaedic surgeons.

Yours sincerely,


ESTELLE COUSTAS
NURSING EXECUTIVE

8.17 Appendix 17

Consent from Orthopaedic Surgeons

Dr. A. Skinner

M.B. Ch.B. (U.S.) M. Med. (U.O.V.S)

Orthopaedic Surgeon

Practice No.: NAMAF 028 000 0011991

21 October 2015

TO WHOM IT MAY CONCERN

RE: APPROVAL TO CONDUCT RESEARCH STUDY

I, Alex Skinner hereby give permission to Ruan Mocke to conduct a research study on my arthroplasty patients entitled "THE EFFICACY OF PAIN NEUROSCIENCE EDUCATION IN COMBINATION WITH COGNITIVE-TARGET EXERCISE THERAPY IN TOTAL JOINT ARTHROPLASTY: A RANDOMISED CONTROLLED TRIAL.

For any queries please do not hesitate to contact me.

Yours faithfully



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Date: 17-9-2015

Mr R. Mockè
Swakopmund

Dear Sir

Regarding reseach project and patients in my care

Thank you for the opportunity to contribute to your research project.

Without any reservation, I give my consent for patients under my care to be included in your research if or when they qualify.

I look forward to see your data and the conclusion.

Regards



André van Niekerk



Dr. J.J. Viviers
Orthopaedic Surgeon
MB ChB (Pret) MMed (Orthop) (UOFS)
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TO WHOM IT MAY CONCERN

Re: Research Study conducted by Ruan Mocke

I have been contacted by Ruan Mocke regarding his research on patients undergoing total hip and knee replacements at Swakopmund Mediclinic. I have read the summary of the study and I am happy for my patients to be involved should they choose to do so.

Kind regards

Kota Viviers

22-9-2015

CONFIDENTIAL

8.18 Appendix 18

Ethics approval

IRB nr 0006240
REC Reference nr 230408-011
IORG0005187
FWA00012784

20 October 2015

MR R MOCKE
c/o MRS K BODENSTEIN
DEPARTMENT OF PHYSIOTHERAPY
CR DE WET BUILDING
UFS

Dear Mr Mocke

ECUFS NR 172/2015

MR R MOCKE

DEPARTMENT OF PHYSIOTHERAPY

PROJECT TITLE: THE EFFICACY OF PAIN NEUROSCIENCE EDUCATION IN COMBINATION WITH COGNITIVE-TARGETED EXERCISE THERAPY IN TOTAL JOINT ARTHROPLASTY: A RANDOMISED CONTROLLED TRIAL

1. You are hereby kindly informed that, at the meeting held on 15 October 2015, the Ethics Committee approved the above project after all conditions were met.
2. Any amendment, extension or other modifications to the protocol must be submitted to the Ethics Committee for approval.
3. A progress report should be submitted within one year of approval of long term studies and a final report at completion of both short term and long term studies.
4. Kindly use the ECUFS NR as reference in correspondence to the Ethics Committee Secretariat.
5. The Ethics Committee functions in compliance with, but not limited to, the following documents and guidelines: The SA National Health Act, No. 61 of 2003; Ethics in Health Research: Principles, Structures and Processes (2015); SA GCP(2006); Declaration of Helsinki; The Belmont Report; The US Office of Human Research Protections 45 CFR 461 (for non-exempt research with human participants conducted or supported by the US Department of Health and Human Services- (HHS), 21 CFR 50, 21 CFR 56; CIOMS, ICH-GCP E6 Sections 1-4; The International Conference on Harmonization and Technical Requirements for Registration of Pharmaceuticals for Human Use (ICH Tripartite), Guidelines of the SA Medicines Control Council as well as Laws and Regulations with regard to the Control of Medicines, Constitution of the Ethics Committee of the Faculty of Health Sciences.

Yours faithfully


DR SM LF GRANGE
CHAIR: ETHICS COMMITTEE

Cc Mrs Karen Bodenstein



8.19 Appendix 19

Language editor letter

Word Trust

Prof. T. C. Smit
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22 May 2017

To whom it may concern

I, the undersigned, hereby acknowledge that I edited and proof read the following M.Sc thesis for language and typographical correctness:

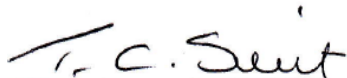
The efficacy of pain neuroscience education in combination with cognitive-targeted exercise therapy in total joint arthroplasty: A randomised controlled trial

Ruan Mockè

I have indicated the areas in the thesis to which attention should be paid. All textual changes made to this thesis after the date above are not covered by the editing and proof reading.

I trust that my advice was accepted and that these corrections and changes were executed as suggested.

Sincerely



Prof. T. C. Smit

PhD (Cognitive Linguistics); MA (TESOL); BA Hons. (TESOL); BA Hons. (English Literature); Post-Graduate Diploma in Special Education (Remedial Teaching); Post-Graduate Diploma (Secondary Teaching); BA (Languages)

8.20 Appendix 20

Raw data

Table 8-1 BPI Questions relating to pain intensity control group descriptive values

BPI Question 3,4,5 and 6												
Stage	Control group (n=9)											
	Question (Description)	Total persons										
Pain/10		0/10	1/10	2/10	3/10	4/10	5/10	6/10	7/10	8/10	9/10	10
Baseline	3 (Worst pain)	0	0	0	2	1	2	0	2	2	0	0
	4 (Least pain)	2	4	1	2	0	0	0	0	0	0	0
	5 (Average pain)	0	0	2	3	2	2	0	0	0	0	0
	6 (Current pain)	1	3	2	2	1	0	0	0	0	0	0
Hospital discharge	3 (Worst pain)	0	0	1	1	0	0	1	4	1	1	0
	4 (Least pain)	4	1	2	1	1	0	0	0	0	0	0
	5 (Average pain)	0	1	1	3	1	2	1	0	0	0	0
	6 (Current pain)	1	2	1	0	2	1	2	0	0	0	0
Six weeks post-surgery	3 (Worst pain)	0	0	3	3	0	0	2	1	0	0	0
	4 (Least pain)	6	2	1	0	0	0	0	0	0	0	0

	5 (Average pain)	0	4	2	1	1	1	0	0	0	0	0
	6 (Current pain)	4	4	1	0	0	0	0	0	0	0	0
	3 (Worst pain)	0	0	6	1	2	0	0	0	0	0	0
Twelve weeks post-surgery	4 (Least pain)	7	2	0	0	0	0	0	0	0	0	0
	5 (Average pain)	0	6	1	1	0	0	0	0	0	0	0
	6 (Current pain)	7	1	0	1	0	0	0	0	0	0	0

Table 8-2 BPI Questions relating to pain intensity intervention group descriptive values

BPI Q3,4,5,6												
Stage	Intervention group (n=10)											
	Question (Description)	Total persons										
Pain/10		0/10	1/10	2/10	3/10	4/10	5/10	6/10	7/10	8/10	9/10	10
Baseline	3 (Worst pain)	0	0	0	1	2	1	4	0	1	1	0
	4 (Least pain)	3	1	1	3	1	0	0	0	0	1	0
	5 (Average pain)	0	1	2	1	0	3	1	1	0	1	0
	6 (Current pain)	4	1	1	2	0	0	1	0	0	1	0
Hospital discharge	3 (Worst pain)	0	0	0	0	1	5	1	1	1	1	0
	4 (Least pain)	3	1	3	1	0	2	0	0	0	0	0
	5 (Average pain)	0	0	0	6	0	2	1	1	0	0	0
	6 (Current pain)	2	2	2	2	0	0	1	0	0	0	0
Six weeks post-surgery	3 (Worst pain)	0	2	3	3	2	0	0	0	0	0	0
	4 (Least pain)	6	2	2	0	0	0	0	0	0	0	0

	5 (Average pain)	1	6	1	2	0	0	0	0	0	0	0
	6 (Current pain)	5	2	3	0	0	0	0	0	0	0	0
	Twelve weeks post-surgery	3 (Worst pain)	2	4	4	0	0	0	0	0	0	0
4 (Least pain)		9	1	0	0	0	0	0	0	0	0	0
5 (Average pain)		7	3	0	0	0	0	0	0	0	0	0
6 (Current pain)		7	2	1	0	0	0	0	0	0	0	0

Table 8-3 BPI Questions relating to pain interference control group descriptive values

Pain interference												
Stage	Control group (n=9)											
	Question (Description)	Total persons										
Pain/10		0/10	1/10	2/10	3/10	4/10	5/10	6/10	7/10	8/10	9/10	10
Baseline	9A (General Activity)	0	2	1	0	1	1	1	2	1	0	0
	9B (Mood)	1	2	1	1	1	0	2	1	0	0	0
	9C (Walking ability)	0	2	1	0	1	1	2	0	1	0	1
	9D (Normal work)	0	1	1	1	1	1	2	1	1	0	0
	9E (People relations)	1	1	3	2	2	0	0	0	0	0	0
	9F (Sleep)	1	1	3	1	0	1	0	0	1	0	1
	9G (Enjoyment of life)	0	2	2	2	1	0	0	1	1	0	0
	Hospital discharge	9A (General Activity)	0	1	0	0	0	0	0	2	4	1
9B (Mood)		1	2	2	1	1	1	0	1	0	0	0
9C (Walking		0	1	1	0	0	0	3	2	1	1	0

	ability)												
	9D (Normal work)	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	
	9E (People relations)	1	3	2	1	0	2	0	0	0	0	0	
	9F (Sleep)	0	2	2	1	1	1	1	0	1	0	0	
	9G (Enjoyment of life)	1	1	2	1	2	2	0	0	0	0	0	
Six weeks post-surgery	9A (General Activity)	0	1	3	2	3	0	0	0	0	0	0	
	9B (Mood)	3	2	2	1	0	1	0	0	0	0	0	
	9C (Walking ability)	0	4	2	2	0	1	0	0	0	0	0	
	9D (Normal work)	0	4	2	2	0	0	0	1	0	0	0	
	9E (People relations)	4	3	2	0	0	0	0	0	0	0	0	
	9F (Sleep)	1	3	1	2	1	0	0	0	0	1	0	
	9G (Enjoyment of life)	1	4	2	1	0	0	0	1	0	0	0	
	Twelve weeks post-	9A (General Activity)	1	2	4	2	0	0	0	0	0	0	0

surgery	9B (Mood)	5	3	1	0	0	0	0	0	0	0	0
	9C (Walking ability)	3	4	2	0	0	0	0	0	0	0	0
	9D (Normal work)	1	5	2	1	0	0	0	0	0	0	0
	9E (People relations)	5	3	1	0	0	0	0	0	0	0	0
	9F (Sleep)	3	3	2	1	0	0	0	0	0	0	0
	9G (Enjoyment of life)	7	1	1	0	0	0	0	0	0	0	0

Table 8-4 BPI Questions relating to pain intensity intervention group descriptive values

Pain interference												
Stage	Intervention group (n=10)											
	Question (Description)	Total persons										
Pain/10		0/10	1/10	2/10	3/10	4/10	5/10	6/10	7/10	8/10	9/10	10
Baseline	9A (General Activity)	0	0	0	0	3	1	0	3	2	0	1
	9B (Mood)	1	2	2	1	1	0	0	1	0	2	0
	9C (Walking ability)	0	0	1	0	1	0	2	3	1	0	2
	9D (Normal work)	0	0	0	2	0	2	2	0	3	0	1
	9E (People relations)	4	2	1	1	0	0	0	1	0	0	1
	9F (Sleep)	2	1	2	1	0	0	0	0	2	1	1
	9G (Enjoyment of life)	0	1	2	0	1	0	2	2	1	0	1
	Hospital discharge	9A (General Activity)	0	0	1	1	0	1	4	2	1	0
9B (Mood)		0	4	2	0	0	1	2	0	1	0	0
9C (Walking ability)		0	0	0	1	2	1	4	1	1	0	0

	9D (Normal work)	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
	9E (People relations)	0	5	3	0	0	1	0	1	0	0	0
	9F (Sleep)	0	1	0	2	0	0	3	0	4	0	0
	9G (Enjoyment of life)	0	2	3	0	2	1	0	2	0	0	0
Six weeks post-surgery	9A (General Activity)	2	1	3	1	2	0	1	0	0	0	0
	9B (Mood)	5	1	2	1	1	0	0	0	0	0	0
	9C (Walking ability)	3	2	3	0	1	0	1	0	0	0	0
	9D (Normal work)	2	1	5	0	0	1	1	0	0	0	0
	9E (People relations)	6	3	0	0	0	1	0	0	0	0	0
	9F (Sleep)	2	2	2	1	2	0	0	1	0	0	0
	9G (Enjoyment of life)	3	2	3	1	0	1	0	0	0	0	0
	Twelve weeks post-surgery	9A (General Activity)	5	2	3	0	0	0	0	0	0	0
9B (Mood)		8	1	1	0	0	0	0	0	0	0	0

9C (Walking ability)	7	1	2	0	0	0	0	0	0	0	0	0	0
9D (Normal work)	5	3	2	0	0	0	0	0	0	0	0	0	0
9E (People relations)	9	1	0	0	0	0	0	0	0	0	0	0	0
9F (Sleep)	5	4	0	1	0	0	0	0	0	0	0	0	0
9G (Enjoyment of life)	8	0	2	1	0	0	0	0	0	0	0	0	0

Table 8-5 PCS Questions relating to catastrophising control group descriptive values

Total PCS														
Control group (n=9)														
Linkert Scale 0 - 4														
Stage	Linkert Scale	1 (H)	2 (H)	3 (H)	4 (H)	5 (H)	6 (M)	7 (M)	8 (R)	9 (R)	10 (R)	11 (R)	12 (H)	13 (M)
Baseline	0	1	2	3	4	2	2	4	2	4	4	3	3	3
	1	2	3	3	3	3	3	1	1	2	3	2	2	3
	2	2	0	1	0	1	0	4	1	2	1	2	2	2
	3	3	4	0	1	2	1	0	3	0	0	1	2	0
	4	1	0	2	1	1	3	0	2	1	1	1	0	1
Hospital discharge	0	5	4	3	6	5	5	6	1	5	4	3	3	4
	1	1	4	4	0	2	1	1	1	1	2	2	3	3
	2	0	1	2	2	2	3	1	2	1	1	2	0	1

	3	3	0	0	1	0	0	1	4	2	2	2	3	1
	4	0	0	0	0	0	0	0	1	0	0	0	0	0
Six weeks post-surgery	0	5	7	6	6	8	4	8	2	5	6	4	6	7
	1	4	1	1	3	0	4	1	3	1	2	4	2	2
	2	0	1	2	0	1	1	0	3	3	1	1	0	0
	3	0	0	0	0	0	0	0	1	0	0	0	1	0
	4	0	0	0	0	0	0	0	0	0	0	0	0	0
Twelve weeks post-surgery	0	7	8	7	9	9	6	9	5	6	6	7	8	9
	1	2	0	1	0	0	2	0	1	3	3	1	1	0
	2	0	1	1	0	0	1	0	3	0	0	1	0	0
	3	0	0	0	0	0	0	0	0	0	0	0	0	0
	4	0	0	0	0	0	0	0	0	0	0	0	0	0

Helplessness (H); Magnification (M); Rumination (R)

Table 8-6 PCS Questions relating to catastrophising intervention group descriptive values

Total PCS														
Intervention group (n=10)														
Linkert Scale 0 - 4														
Stage	Linkert Scale	1 (H)	2 (H)	3 (H)	4 (H)	5 (H)	6 (M)	7 (M)	8 (R)	9 (R)	10 (R)	11 (R)	12 (H)	13 (M)
Baseline	0	2	3	3	3	3	2	6	1	2	3	1	3	3
	1	5	2	3	3	3	2	1	2	2	2	2	3	2
	2	2	0	0	0	0	2	1	1	2	1	1	1	3
	3	0	4	1	1	1	3	1	1	1	2	1	1	1
	4	1	1	3	3	3	1	1	5	3	2	5	2	1
Hospital discharge	0	3	5	5	4	5	6	5	0	3	4	1	3	7
	1	2	4	4	3	4	2	3	3	2	2	2	3	1
	2	4	1	1	1	0	2	1	0	2	3	3	2	2
	3	1	0	0	2	1	0	0	4	3	1	3	1	0
	4	0	0	0	0	0	0	1	3	0	0	1	1	0
Six weeks post-surgery	0	4	9	7	7	9	6	8	4	5	7	4	6	8
	1	3	0	1	2	1	2	1	2	3	2	3	3	1
	2	1	1	1	1	0	2	1	2	2	1	2	1	1
	3	1	0	1	0	0	0	0	1	0	0	0	0	0
	4	1	0	0	0	0	0	0	1	0	0	1	0	0
Twelve weeks post-surgery	0	8	10	10	10	9	9	10	6	8	9	9	10	9
	1	2	0	0	0	1	1	0	3	2	1	1	0	1
	2	0	0	0	0	0	0	0	1	0	0	0	0	0

	3	0	0	0	0	0	0	0	0	0	0	0	0	0
	4	0	0	0	0	0	0	0	0	0	0	0	0	0

Helplessness (H); Magnification (M); Rumination (R)

Table 8-7 TSK-13- Questions relating to FOM control group descriptive values

Total TSK-13														
Control group (n=9)														
Linkert Scale 1 – 4														
Stage	Linkert Scale	1	2	3	5	6	7	9	10	11	13	14	15	17
Baseline	1	3	2	1	2	2	0	0	0	2	0	4	2	3
	2	1	2	5	5	1	4	1	2	4	2	2	1	1
	3	5	3	1	2	6	3	4	4	2	5	1	3	3
	4	0	2	2	0	0	2	4	3	1	2	2	3	2
Hospital discharge	1	2	2	5	6	4	3	1	6	2	0	2	3	3
	2	4	5	2	2	5	3	1	3	4	1	4	0	4
	3	2	2	1	1	0	3	4	0	3	5	1	5	1
	4	1	0	1	0	0	0	3	0	0	3	2	1	1
Six weeks post-surgery	1	5	6	6	7	8	3	2	3	3	1	5	4	2
	2	2	1	2	2	0	3	3	3	4	4	3	1	6
	3	2	0	1	0	1	3	4	2	2	2	1	2	1
	4	0	2	0	0	0	0	0	1	0	2	0	2	0
Twelve weeks post-surgery	1	5	3	3	4	5	4	4	2	2	2	3	2	3
	2	3	4	3	5	3	4	4	4	6	2	6	5	4
	3	1	2	3	0	1	1	1	3	1	5	0	2	2
	4	0	0	0	0	0	0	0	0	0	0	0	0	0

Table 8-8 TSK-13- Questions relating to FOM intervention group descriptive values

Total TSK-13														
Intervention group (n=10)														
Linkert Scale 1 - 4														
Stage	Linkert Scale	1	2	3	5	6	7	9	10	11	13	14	15	17
Baseline	1	3	1	1	6	4	1	1	1	3	0	1	0	2
	2	4	1	3	2	2	2	1	1	3	1	3	2	4
	3	0	5	1	0	0	4	4	5	1	7	3	6	3
	4	3	3	5	2	4	3	4	3	3	2	3	2	1
Hospital discharge	1	1	1	6	6	5	5	0	1	4	0	4	1	1
	2	7	7	3	3	3	2	1	2	3	2	5	3	4
	3	2	2	1	1	2	3	6	4	2	6	1	4	4
	4	0	0	0	0	0	0	3	3	1	2	0	2	1
Six weeks post-surgery	1	3	3	4	6	4	3	1	1	3	2	3	1	5
	2	5	7	5	3	6	4	5	6	6	1	7	7	3
	3	2	0	1	1	0	1	4	3	0	6	0	1	1
	4	0	0	0	0	0	2	0	0	1	1	0	1	1
Twelve weeks post-surgery	1	6	5	3	6	5	4	2	3	5	1	6	4	2
	2	4	5	6	3	5	4	7	6	5	5	4	4	6
	3	0	0	1	1	0	2	1	1	0	3	0	1	2
	4	0	0	0	0	0	0	0	0	0	1	0	1	0

8.21 Appendix 21

Regression of ANOVA format and calculations

ANOVA for Regression

Analysis of Variance (ANOVA) consists of calculations that provide information about levels of variability within a regression model and form a basis for tests of significance. The basic regression line concept, $\text{DATA} = \text{FIT} + \text{RESIDUAL}$, is rewritten as follows:

$$(y_i - \bar{y}) = (\hat{y}_i - \bar{y}) + (y_i - \hat{y}_i).$$

The first term is the total variation in the response y , the second term is the variation in mean response, and the third term is the residual value. Squaring each of these terms and adding over all of the n observations gives the equation

$$\sum (y_i - \bar{y})^2 = \sum (\hat{y}_i - \bar{y})^2 + \sum (y_i - \hat{y}_i)^2.$$

This equation may also be written as $\text{SST} = \text{SSM} + \text{SSE}$, where SS is notation for *sum of squares* and T, M, and E are notation for *total*, *model*, and *error*, respectively.

The square of the sample correlation is equal to the ratio of the model sum of squares to the total sum of squares: $r^2 = \text{SSM}/\text{SST}$.

This formalizes the interpretation of r^2 as explaining the fraction of variability in the data explained by the regression model.

The sample variance s_y^2 is equal to $\sum (y_i - \bar{y})^2 / (n - 1) = \text{SST}/\text{DFT}$, the total sum of squares divided by the total degrees of freedom (DFT).

For simple linear regression, the MSM (mean square model) = $\sum (\hat{y}_i - \bar{y})^2 / (1) = \text{SSM}/\text{DFM}$, since the simple linear regression model has one explanatory variable x .

The corresponding MSE (mean square error) = $\sum (y_i - \hat{y}_i)^2 / (n - 2) = \text{SSE}/\text{DFE}$, the estimate of the variance about the population regression line (σ^2).

ANOVA calculations are displayed in an *analysis of variance table*, which has the following format for simple linear regression:

Source	Degrees of Freedom	Sum of squares	Mean Square	F
Model	1	$\sum (\hat{y}_i - \bar{y})^2$	SSM/DFM	
Error	$n - 2$	$\sum (y_i - \hat{y}_i)^2$	SSE/DFE	
Total	$n - 1$	$\sum (y_i - \bar{y})^2$	SST/DFT	

The "F" column provides a statistic for testing the hypothesis that

$$\beta_1 \neq 0$$

against the null hypothesis that $\beta_1 = 0$.

The test statistic is the ratio MSM/MSE, the mean square model term divided by the mean square error term. When the MSM term is large relative to the MSE term, then the ratio is large and there is evidence against the null hypothesis.

For simple linear regression, the statistic MSM/MSE has an F distribution with degrees of freedom (DFM, DFE) = (1, $n - 2$).

Example

The dataset "Healthy Breakfast" contains, among other variables, the *Consumer Reports* ratings of 77 cereals and the number of grams of sugar contained in each serving. (*Data source: Free publication available in many grocery stores. Dataset available through the Statlib Data and Story Library (DASL)*)

Considering "Sugars" as the explanatory variable and "Rating" as the response variable generated the following regression line:

Rating = 59.3 - 2.40 Sugars (see Inference in Linear Regression for more information about this example). The "Analysis of Variance" portion of the MINITAB output is shown below. The degrees of freedom are provided in the "DF" column, the calculated sum of squares terms are provided in the "SS" column, and the mean square terms are provided in the "MS" column.

Analysis of Variance

Source	DF	SS	MS	F	P
Regression	1	8654.7	8654.7	102.35	0.000
Error	75	6342.1	84.6		
Total	76	14996.8			

In the ANOVA table for the "Healthy Breakfast" example, the F statistic is equal to $8654.7/84.6 = 102.35$. The distribution is $F(1, 75)$, and the probability of observing a value greater than or equal to 102.35 is less than 0.001. There is strong evidence that β_1 is not equal to zero.

The r^2 term is equal to 0.577, indicating that 57.7% of the variability in the response is explained by the explanatory variable.

ANOVA for Multiple Linear Regression

Multiple linear regression attempts to fit a regression line for a response variable using more than one explanatory variable. The ANOVA calculations for multiple regression are nearly identical to the calculations for simple linear regression, except that the degrees of freedom are adjusted to reflect the number of explanatory variables included in the model.

For p explanatory variables, the model degrees of freedom (DFM) are equal to p , the error degrees of freedom (DFE) are equal to $(n - p - 1)$, and the total degrees of freedom (DFT) are equal to $(n - 1)$, the sum of DFM and DFE.

The corresponding ANOVA table is shown below:

Source	Degrees of Freedom	Sum of squares	Mean Square	F
Model	p	$\sum (\hat{y}_i - \bar{y})^2$		SSM/DFM
		MSM/MSE		
Error	$n - p - 1$	$\sum (y_i - \hat{y}_i)^2$		SSE/DFE
Total	$n - 1$	$\sum (y_i - \bar{y})^2$		SST/DFT

In multiple regression, the test statistic MSM/MSE has an $F(p, n - p - 1)$

distribution.

The null hypothesis states that $\beta_1 = \beta_2 = \dots = \beta_p = 0$, and the alternative hypothesis simply states that *at least one* of the parameters $\beta_j \neq 0, j = 1, 2, \dots, p$. Large values of the test statistic provide evidence against the null hypothesis.

Note: The F test does not indicate which of the parameters $\beta_j \neq$

is not equal to zero, only that at least one of them is linearly related to the response variable.

The ratio $SSM/SST = R^2$ is known as the *squared multiple correlation coefficient*. This value is the proportion of the variation in the response variable that is explained by the response variables. The square root of R^2 is called the *multiple correlation coefficient*, the correlation between the observations y_i and the fitted values \hat{y}_i .

Example

The "Healthy Breakfast" dataset contains, among other variables, the *Consumer Reports* ratings of 77 cereals, the number of grams of sugar contained in each serving, and the number of grams of fat contained in each serving. (*Data source: Free publication available in many grocery stores. Dataset available through the Statlib Data and Story Library (DASL).*)

As a simple linear regression model, we previously considered "Sugars" as the explanatory variable and "Rating" as the response variable. How do the ANOVA results change when "FAT" is added as a second explanatory variable?

The regression line generated by the inclusion of "Sugars" and "Fat" is the following:

Rating = 61.1 - 2.21 Sugars - 3.07 Fat (see Multiple Linear Regression for more information about this example).

The "Analysis of Variance" portion of the MINITAB output is shown below. The degrees of freedom are provided in the "DF" column, the calculated sum of squares terms are provided in the "SS" column, and the mean square terms are provided in the "MS" column.

Analysis of Variance

Source	DF	SS	MS	F	P
Regression	2	9325.3	4662.6	60.84	0.000
Error	74	5671.5	76.6		
Total	76	14996.8			

Source	DF	Seq SS
Sugars	1	8654.7
Fat	1	670.5

The mean square error term is smaller with "Fat" included, indicating less deviation between the observed and fitted values. The P -value for the F test statistic is less than 0.001, providing strong evidence against the null hypothesis. The squared multiple correlation $R^2 = SSM/SST = 9325.3/14996.8 = 0.622$, indicating that 62.2% of the variability in the "Ratings" variable is explained by the "Sugars" and "Fat" variables. This is an improvement over the simple linear model including only the "Sugars" variable.

(Yale University, 1997)