CASE REPORT

Use of an abbreviated neuroscience education approach in the treatment of chronic low back pain: A case report

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ABSTRACT

Chronic low back pain (CLBP) remains prevalent in society, and conservative treatment strategies appear to have little effect. It is proposed that patients with CLBP may have altered cognition and increased fear, which impacts their ability to move, perform exercise, and partake in activities of daily living. Neuroscience education (NE) aims to change a patient’s cognition regarding their pain state, which may result in decreased fear, ultimately resulting in confrontation of pain barriers and a resumption of normal activities. A 64-year-old female with history of CLBP was the patient for this case report. A physical examination, the Numeric Pain Rating Scale (NPRS), Oswestry Disability Index (ODI), Fear-Avoidance Beliefs Questionnaire (FABQ), and Zung Depression Scale were assessed during her initial physical therapy visit, immediately after her first physical therapy session, and at 7-month follow-up. Treatment consisted of an abbreviated NE approach, exercises (range of motion, stretches, and cardiovascular), and aquatic therapy. She attended twice a week for 4 weeks, or 8 visits total. Pre-NE, the patient reported NPRS = 9/10; ODI = 54%; FABQ-W = 25/42, FABQ-PA = 20/24, and Zung = 58. Immediately following the 75-minute evaluation and NE session, the patient reported improvement in all four outcome measures, most notably a reduction in the FABQ-W score to 2/42 and the FABQ-PA to 1/24. At a 7-month follow-up, all outcome measures continued to be improved. NE aimed at decreasing fear associated with movement may be a valuable adjunct to movement-based therapy, such as exercise, for patients with CLBP.

INTRODUCTION

Epidemiological data suggest that chronic, widespread, nonspecific musculoskeletal pain is on the rise, especially in the area of chronic low back pain (CLBP), adding to the ever-increasing costs of health care (Magni et al, 1993; McMahon and Koltzenburg, 2005). Current research indicates that very few of the interventions that have proven successful in acute LBP are effective for patients with CLBP (Bonifazi et al, 2006; Carville et al, 2008; Goldenberg, Burckhardt, and Crofford, 2004; Rooks et al, 2007).

The lack of clinically effective interventions presents significant challenges and frustration to both patients and physical therapists.

Education has long been an intervention used by physical therapists (PTs) to help alleviate the disability associated with low back pain (LBP) (Brox et al, 2008; Engers et al, 2008; Heymans et al, 2005; Liddle, Gracey, and Baxter, 2007). There are a number of studies that have investigated the effectiveness of patient education on pain and disability (Louv, Louw, and Crous, 2009), with outcomes ranging from “poor” (Cohen et al, 1994; Gross, Aker, Goldsmith, and Peloso, 2000) to “excellent” (Udermann et al, 2004). Most educational programs used in musculoskeletal care rely on anatomical and biomechanical models for addressing pain (Brox et al, 2008; Butler and Moseley, 2003; Maier-Riehle and Harter, 2001; Moseley, 2003b; Moseley, 2004).
which has not only shown limited efficacy (Brox et al, 2008; Butler and Moseley, 2003; Koes, van Tulder, van der Windt, and Bouter, 1994; Maier-Riehle and Harter, 2001; Waddell, 2004) but may actually increase patient fears and negatively impact their outcomes (Hirsch and Liebert, 1998; Maier-Riehle and Harter, 2001; Nachemson, 1992; Poirauveau et al, 2006). An example of such an anatomical and biomechanical education program is the traditional Back School, which teaches patients about the structure and function of the spine as well as body mechanics and correct lifting methods (Maier-Riehle and Harter, 2001). Back Schools have come under scrutiny, showing limited effectiveness in decreasing pain and disability in patients with spinal disorders (Koes et al, 1994; Maier-Riehle and Harter, 2001). A common shortcoming of these anatomical and biomechanical approaches is that they typically do not include information about pain processing, neuroscience (Moseley, 2003b; Moseley, 2002), or psychosocial issues, the latter of which has been shown to be a strong predictor of long-term disability and chronic pain (Fritz, George, and Delitto, 2001). Psychologically based models of education for LBP, termed cognitive behavioral therapy (CBT) (Brox et al, 2008; Johnson et al, 2007) attempt to address some of the psychosocial issues related to LBP. CBT aims to reassure patients and address fears related to movement, pathology, and function. Systematic reviews have shown that the outcomes of CBT are similar to non-CBT-based treatment in patients with LBP (Brox et al, 2008; Johnson et al, 2007).

Recent research has been investigating the use of neuroscience education in decreasing pain and disability in patients with LBP (Moseley, 2002; Moseley, 2003a; Moseley, 2003c; Moseley, 2004; Moseley, 2005; Moseley, Nicholas, and Hodges, 2004). Neuroscience education (NE) is best described as a form of CBT, aimed at increasing a patient’s knowledge and understanding of pain, thus reducing fear associated with musculoskeletal injury. CBT typically uses psychological counseling aimed at reducing fear and anxiety. Although NE also aims to reduce a patient’s fear and anxiety, the message focuses on simply explaining the neurobiology that underpins the patient’s chronic pain state. Pain is a powerful motivating force that guides medical care as well as treatment seeking behaviors in patients (Bernard and Wright, 2004; Mortimer and Ahlberg, 2003). Patients are interested in knowing more about pain (Louw, Louw, and Crous, 2009), and studies have shown that health care professionals usually underestimate a patient’s ability to understand “complex” issues such as the neurophysiology of pain (Moseley, 2003c). NE sessions are designed to present detailed information on the neurobiology of pain along with the physiology of the nervous system in an easy-to-understand format that includes pictures, examples, and metaphors (Moseley, 2003c). The details of this approach are described elsewhere and expanded upon in this case report. The pain science educational sessions described in the literature may last between 2 and 3 hours and may include individualized education or group sessions (Butler and Moseley, 2003; Moseley, 2003a; Moseley, 2004; Moseley, 2005).

Studies using NE have been shown to decrease fear and change patients’ perceptions of pain by effecting immediate improvements in patients’ attitudes about pain (Moseley, 2003b), pain cognition and physical performance (Moseley, 2004), and pressure pain thresholds during physical tasks (Moseley, Nicholas, and Hodges, 2004). It has also been shown to improve outcomes following therapeutic exercise (Moseley, 2002) while significantly reducing the widespread brain activity characteristic of a chronic pain experience (Moseley, 2005). Many of these results are long-lasting, with some research showing lasting changes out to a 1-year follow-up (Moseley, 2002; Moseley, 2003a; Oliveira, Gevirtz, and Hubbard, 2006).

A proposed explanation for the noted improvements following NE is that the patient “re-conceptualizes” their problem in such a way that it leads to increased confidence and activity levels. It has been shown that altered pain beliefs are directly associated with altered movement performance, even in the absence of physical treatments such as manual therapy or exercise (Moseley, 2004). This implies that motor performance may be directly limited by pain beliefs, which would be consistent with clinical observation (Moseley, 2005). In contrast to fear-avoidance beliefs, which focus on altered processing due to fear (including fear of pain), pain beliefs refer to a patient’s beliefs and knowledge of pain (i.e., how pain works) (Moseley, 2005).

The purpose of this case report is threefold. First, the report aims to show how an NE session can be applied in a time frame that is clinically reasonable (approximately 1 hour). Second, it aims to describe the clinical reasoning process used in determining that an NE session was needed for this patient. Third, the exact content of NE sessions are poorly described, and this case report aims to provide clinicians with a concise and effective way to deliver this intervention to a patient with central sensitization, using strategies, metaphors, and descriptions.

**CASE DESCRIPTION**

This patient was seen at Sports Rehabilitation and Physical Therapy Associates in Overland Park, KS.
The patient signed a consent form and was enrolled in a single case study on neuroscience education for chronic low back pain.

History

The patient was a 64-year-old female who presented to physical therapy with a 3-year history of CLBP. She had previously worked as a registered nurse but had not been working because of her persistent pain. She reported that the onset of her LBP had been insidious, with no known injury or cause. The LBP, and resultant disability, progressively increased to the point where she stopped working approximately 18 months prior to the initial physical therapy examination. She was never placed on disability nor received workman’s compensation. Since the onset of her LBP, she had been treated by numerous health care providers, including physical therapists, chiropractors, massage therapists, acupuncturists, spine surgeons, her general practitioner, various pain management specialists, psychologists, a neurologist, and a physiatrist. The most common treatments received included therapeutic exercise (various forms), massage, chiropractic adjustments, physical modalities, epidural steroid injections, education about her back pain, relaxation exercises, and various medications, including pain medications and muscle relaxants. At the time of her initial physical therapy visit, she reported that she was taking Hydrocodone (7.5 mg Hydrocodone Bitartrate and 500 mg Acetaminophen every 4–5 hours), Oxycontin (10 mg every 4 hours), Skelaxin (800 mg 3–4 times per day), and Celebrex (100 mg 2 times per day). These medications offered only short-term relief and did not result in any long-term improvements in her symptoms. Her most recent treatments included a series of three epidural steroid injections and a multilevel radiofrequency nerve ablation, which had not eased her pain. She was then referred to the primary author for the evaluation and treatment of her CLBP.

During the subjective history, the patient reported that her general health was good and she had no other orthopedic injuries or concerns about other joints. Prior to the initial physical therapy evaluation, the patient completed a medical screening questionnaire (MSQ) to screen for potential red and yellow flags that may require further workup and/or referral. The MSQ includes a review of systems, with special questions designed to screen for organ system involvement based on subjective symptoms perceived by the patient. The MSQ was reviewed by the primary therapist with the patient and did not reveal any red flags at the time of the initial evaluation.

Over the previous 18 months, she had steadily decreased her activities of daily living (ADL) due to pain and an increased fear of pain. Of particular concern to her was her inability to clean her house. She reported increased pain with vacuuming more than half a room at a time. If she vacuumed more than half a room, her back pain would increase considerably (“flare”) to where she needed to take extra pain medication, lie down, and usually was unable to do any further housework. She reported that she enjoyed exercising (“working out”) but had decreased her walking distance and frequency due to her CLBP. At the time of the initial examination, she reported that she was unable to stand at a stove and cook a meal and was unable to sit at a desk for more than 30 minutes. The pattern of her pain was assessed by having her complete a body chart as seen in Figure 1, which was then discussed. The examining therapist did not find any red flags when she was questioned about her pain pattern, and it appeared to be consistent with that of a patient with central sensitization in that pain was widespread (diffuse area). She rated her current pain as 9 out of 10 on the Numeric Pain Rating Scale (NPRS) (Cleland, Childs, and Whitman, 2008; Moseley, 2002). The patient reported that her easing factors included lying down on either side (left or right), frequent periods of sitting down, or taking her medication. Her most recent MRI (2005) revealed multilevel “bulging” discs (most notably at L2/3, L4/5, and L5/S1) and multilevel degenerative disc disease—findings that,

![Pattern of the patient's pain.](image)
based on the interview, created a lot of anxiety for the patient. The patient’s goals for attending physical therapy were to return to part-time work (2–3 half days per week in her husband’s office), be able to stand in the kitchen and cook a whole meal, clean her house, and to return to her regular exercise program, which included aerobics, walking, and yoga.

Questionnaires

Prior to the patient being further evaluated for physical therapy services, she was asked to complete questionnaires pertaining to pain, disability, fear-avoidance beliefs, and depression. These questionnaires were chosen on the basis of her early communication with the primary therapist prior to setting up the appointment and based on her CLBP history. These same questionnaires were completed by the patient immediately following the NE session (75 minutes later) and at 7-month follow-up. The four self-report questionnaires used for this case report included: 1) Numeric Pain Rating Scale (NPRS), 2) Oswestry Disability Index (ODI), 3) Fear-Avoidance Beliefs Questionnaire (FABQ), and 4) Zung Depression Scale.

The NPRS is an 11-point scale (range from 0 to 10) that is used to measure pain intensity. The scale is anchored on the left with the phrase “No Pain” and on the right with the phrase “Worst Imaginable Pain.” The NPRS was used to rate the patient’s current level of pain and the worst and least amount of pain in the last 24 hours. The average of the three ratings was used to represent the patient’s level of pain. The NPRS is a valid and reliable measure of pain that has been used in randomized controlled trials for NE and spinal pain (Cleland, Childs, and Whitman, 2008; Moseley, 2002; Moseley, 2003a; Moseley, 2005).

The ODI is a 10-item questionnaire that assesses different aspects of physical function. Each item is scored from 0 to 5, with higher values representing greater disability. The total score is multiplied by 2 and expressed as a percentage. The ODI has been shown to be a valid and reliable measure of disability related to LBP (Deyo et al, 1998; Fritz and Irrgang, 2001; Hakkinen et al, 2007).

The FABQ is a 16-item questionnaire that was designed to quantify fear and avoidance beliefs in individuals with LBP. The FABQ has two subscales: 1) a seven-item scale to measure fear-avoidance beliefs about work and 2) a four-item scale to measure fear-avoidance beliefs about physical activity. Each item is scored from 0 to 6 with possible scores ranging between 0 and 24 and 0 and 4 for the physical activity and work subscales, respectively, with higher scores representing an increase in fear-avoidance beliefs. The FABQ has demonstrated acceptable levels of reliability and validity in previous LBP studies (Cleland, Fritz, and Childs, 2008; Grotle, Vollestad, and Brox, 2006; Poiraudeau et al, 2006; Waddell et al, 1993).

The Zung Self-Rating Depression Scale is a short self-administered 20-item survey to quantify the depressed status of a patient and has been used in measuring depression related to spinal disorders (Ronnberg et al, 2007; Zung, 1965). Zung Depression Scale scores range from 20 to 80, with a score of 55 or higher suggesting possible depression (Zung, 1965).

Physical examination

A lower quarter screening examination was performed, aimed at identifying any potential red flags and nervous system involvement. The lower quarter screen included a neuromuscular examination of the lower extremities consisting of myotome strength, sensation along dermatomes and peripheral nerves, and deep tendon reflexes of the lower extremities (Butler, 1991; Butler, 2000). The screening examination did not identify any abnormal findings. Adjacent peripheral joints (hip and knee) and spinal regions (cervical and thoracic) were subjected to quick active range of motion (ROM) assessments in all planes. The adjacent joints and spinal regions, although “sensitive” to movement, did not reveal significant ROM deficits or exacerbation of her primary complaint of widespread LBP.

The rest of the physical examination was focused, because the therapist felt that including too many tests and measures would likely yield little useful data and may have exacerbated the patient’s pain state. This type of examination is consistent with what has been proposed in the physical examination of patients with chronic pain (Butler, 2000). Nattrass et al (1999) stated that there was no evidence for a relationship between impairments and disability in CLBP and that it is “illogical” to evaluate impairment in CLBP patients using a spinal range of motion model when aiming to measure disability. The patient presented with signs and symptoms associated with central sensitivity (see diagnosis section). The presentation of central sensitivity with a generalized up-regulation of the central nervous system (CNS) (secondary hyperalgesia) influences the results of physical examinations and limits the validity of tests and measures aimed at finding specific physical dysfunction (Butler, 2000; Smart, Blake, Staines, and Doody, 2010). Identifying specific physical impairments may have limited value when examining patients with widespread and prolonged pain (Butler, 2000; Fernandez-de-las-Penas et al, 2009; Moseley, 2003b).
The tests and measures section of the physical examination began with active movement testing. The therapist deemed this important because it was critical to assess the quantity and quality of functional movement present, as well as any aberrant movements, as this finding has been purported to predict success with lumbar stabilization exercises (Hicks, Fritz, Delitto, and McGill, 2005). The patient was instructed to "bend forward while keeping your knees straight and to stop whenever you feel like you need to stop." It is important to point out here that the patient was not asked to bend forward until pain onset, pain increase or pain limit, and in fact the word "pain" was not used during testing instructions. The patient was only able to achieve 10 degrees of flexion, as measured by a single inclinometer. Fritz, Whitman, and Childs (2005) reported that single inclinometer measurements of lumbar flexion and extension had intraclass correlation coefficients (ICC) of 0.60 and 0.61 for interrater reliability, respectively. During the forward bend, the patient reported that she was afraid to move beyond the 10 degrees for fear her pain would get worse. When questioned about "her pain," the therapist asked the patient to relate the pain she was fearful of reproducing to her body chart. She related that she was afraid that further forward flexion would bring on the "entire" pain area depicted on the body chart. Because of the expressed anxiety about active movement, no further active lumbar ROM movements were performed to avoid exacerbating the patient's condition.

Neurodynamic testing followed, which included the straight leg raise (SLR) test (Butler, 1991; Butler, 2000) on the left and the right, to the first point of resistance. Her SLR averaged 70 degrees on both legs (goniometer measurement – average for three measurements), with reported symptoms of "pulling" in both her legs, left equal to right. The "pulling" in her legs was not her primary complaint and did not correlate with the pain pattern outlined on the body chart. Slump testing (Butler, 1991; Butler, 2000) was limited due to her inability to "slouch" without pain and anxiety. A modified slump was performed in a slightly slumped position but revealed decreased knee extension of -30° from full extension bilaterally, with a sensation of "pulling" in the leg and low back. The modified slump test elicited her primary pain complaint of back and leg pain, and this did correlate with the pain pattern outlined on the body chart.

**Diagnosis**

Although the patient presented with a complex pain diagram, the primary therapist concluded that the patient’s signs and symptoms were consistent with a chronic pain state with central sensitization. "Central sensitization" is defined as a condition in which peripheral noxious inputs into the central nervous system (CNS) lead to an increased excitability where the response to normal inputs is greatly enhanced (Merskey and Bogduk, 1994; Smart, Blake, Staines, and Doody, 2010). This is not to imply that the symptoms were non-organic, but rather a complex neurophysiological process was in play whereby the CNS had become "hyper-excitiable" to input received from the periphery (Butler, 2000; Woolf, 2007).

Although no standardized or validated screening tools exist to identify patients with central sensitivity, a number of theoretical clinical decision-making strategies exist based on the recognition of characteristic signs and symptoms (Butler, 2000; Lidbeck, 2002). The patient in this case report demonstrated several of the signs and symptoms that are typically associated with central sensitivity including: a high FABQ score; diffuse area of pain as seen in her body diagram in Figure 1; spontaneous pain ("it comes on when it wants to"); pain associated with emotional disturbances and maladaptive cognitions; history of failed treatments; and diffuse/non-anatomic areas of pain and tenderness on palpation (Table 1) (George and Zeppieri, 2009).

It is important to note that at the initial evaluation, the patient presented with a score of 58 on the Zung Depression Scale, and most people with depression are reported to score between 50 and 69 (Romera et al, 2008). Because this score was elevated and would have suggested the potential need for a referral to a psychological health care provider, the primary therapist discussed this with the patient. The patient related a history of treatment for depression by a psychologist. It was suggested to the patient that she may benefit from further examination by a psychological health care provider, but she declined, because she reported that her previous treatment (3 months prior) had not helped. The patient felt that her “depression” was caused by her pain and limited function, and she was hoping that physical therapy might be able to improve her CLBP. The therapist and the patient agreed to monitor these symptoms and proceed with physical therapy. If her depressive symptoms worsened, she would be referred to a psychological health care provider.

**Intervention**

Upon completion of the examination, the treating therapist decided that the patient would benefit from an NE session, because the patient’s clinical presentation fit the clinical presentation of central
sensitization (Butler, 2000; Butler and Moseley, 2003; McMahon and Koltzenburg, 2005; Woolf, 2007) for which NE has been shown to be a useful intervention (Moseley, 2003a; Moseley, 2004, Moseley, 2005; G. L. Moseley, Nicholas, and Hodges, 2004). On the basis of the patient’s high level of fear, especially related to spinal movements, it was decided that physical therapy interventions would focus more on a CBT approach addressing her underlying fear of movement, because manual therapy treatments aimed at improving physical impairments were not likely to succeed. Previous sessions in physical therapy had used various forms of exercise suggesting a graded exercise or graded exposure program may have limitations, because the patient had already “failed” with these approaches. By using NE and having her gain a better understanding of her pain, especially the increased sensitivity of her nervous system, it was argued the patient would not only be able to move better but be more inclined to participate and follow through with other active movement components of the intervention. NE was also chosen as opposed to other educational models such as Back School, which spends some time and detail on describing anatomical and/or biomechanical models of pain, which is contrary to the NE approach of deemphasizing anatomical and/or biomechanical findings.

The material that was presented was based on current knowledge of the neurophysiology of pain according to Wall and Melzack (McMahon and Koltzenburg, 2005) and the Explain Pain book written by Butler and Moseley (2003). The NE lasted 45 minutes in a private room and was administered by a physical therapist familiar with NE. The content of the NE session is listed in Appendix 1. The NE was accompanied by the use of pictures, examples, and metaphors (Butler and Moseley, 2003; Moseley, 2007).

The initial session (examination and NE) was subsequently followed by a series of seven physical therapy visits, which included additional NE and other treatment strategies that have been reported to improve outcomes in patients with chronic pain, such as aquatic therapy (Assis et al, 2006), cardiovascular exercise (Bonifazi et al, 2006; Brosseau et al, 2008a; Busch et al, 2007; Carville et al, 2008; Goldenberg, Burckhardt, and Crofford, 2004; Gowans et al, 2001; Rooks et al, 2007; Sim and Adams, 2002), and strengthening (Brosseau et al, 2008b; Valkeinen et al, 2009). The therapeutic activities are listed in Table 2. In essence, following the initial NE educational session, the subsequent seven sessions comprised a CBT graded exercise/exposure approach. The patient completed a total of eight sessions of physical therapy over the course of 4 weeks. The dosage and frequency were chosen on the basis of a previous study using NE (Moseley, 2002) as well as clinical experience. During the 4 weeks of physical therapy, the patient was advised to continue with the use of her medication as prescribed by her physician. Any questions regarding her taking, not taking, or changing medication were referred to her physician. The patient was asked to inform the primary therapist of any changes (increase and decrease) in the use of medication.

Although previous studies using NE included manual therapy, it was decided not to include manual therapy as a primary treatment approach in this case, based on several factors. First, with a primary clinical presentation of central sensitivity, it was decided that a manual therapy approach focusing

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**TABLE 1** List of signs and symptoms typically observed with central sensitization and found to be present in this patient.

<table>
<thead>
<tr>
<th>Signs and symptoms</th>
<th>Reference</th>
<th>Present in this patient</th>
</tr>
</thead>
<tbody>
<tr>
<td>“Hyper-excitable” to input from the periphery</td>
<td>(Butler, 2000; Woolf, 2007)</td>
<td>✓</td>
</tr>
<tr>
<td>High FABQ score</td>
<td>(Cleland, Fritz &amp; Childs, 2008; Nijs, Van Houdenhove &amp; Oostendorp, 2010)</td>
<td>✓</td>
</tr>
<tr>
<td>Diffuse area of pain</td>
<td>(Butler, 2000; George &amp; Zeppieri, 2009)</td>
<td>✓</td>
</tr>
<tr>
<td>Spontaneous pain</td>
<td>(Fernandez-de-las-Penas et al, 2009; Nijs, Van Houdenhove &amp; Oostendorp, 2010)</td>
<td>✓</td>
</tr>
<tr>
<td>Pain associated with emotional disturbances and maladaptive cognitions</td>
<td>(Nijs, Van Houdenhove &amp; Oostendorp, 2010)</td>
<td>✓</td>
</tr>
<tr>
<td>History of failed treatments</td>
<td>(Butler, 2000; Meeus &amp; Nijs, 2007)</td>
<td>✓</td>
</tr>
<tr>
<td>Visiting many health care providers</td>
<td>(Butler, 2000; Meeus &amp; Nijs, 2007)</td>
<td>✓</td>
</tr>
<tr>
<td>Diffuse/non-anatomic areas of pain and tenderness on palpation</td>
<td>(Fernandez-de-las-Penas et al, 2009; Nijs, Van Houdenhove &amp; Oostendorp, 2010)</td>
<td>✓</td>
</tr>
<tr>
<td>Depression</td>
<td>(Romera et al, 2008)</td>
<td>✓</td>
</tr>
<tr>
<td>Latency</td>
<td>(Butler, 2000; Fernandez-de-las-Penas et al, 2009; Nijs, Van Houdenhove &amp; Oostendorp, 2010)</td>
<td>✓</td>
</tr>
</tbody>
</table>

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TABLE 2 Description of subsequent therapy sessions (2 times per week, 4 weeks).

Subsequent therapy visits (for the therapist)

- In the first session, the patient underwent a subjective examination, objective examination and an introductory neuroscience educational session (described above).
- In the therapy sessions that followed, the therapy sessions consisted of:
  - Continued neuroscience education. Before starting the exercise sessions, the patient received 5 minutes of formal education, which was used to answer questions, address goals, and address concerns or to ask the patient to explain her understanding of certain aspects of her pain, e.g., nerve sensitivity. The neuroscience education was also reinforced during exercises, manual therapy, and/or modalities.
  - The patient engaged in cardiovascular exercise – starting with treadmill walking and later progressed to the elliptical trainer. For both treadmill and elliptical the speed was steadily increased to help increase heart rate.
  - Since the neuroscience education discusses issues related to the nervous system, a series of neural mobilization exercises were used for the upper extremities, lower extremities and trunk, e.g., slump sliders.
  - Basic trunk mobility exercises and stretches, e.g., single-knee to chest, trunk rotations, prayer stretch, piriformis stretch.
  - Strengthening exercises with a focus on higher repetitions using lighter weight (dumbbells, TherabandTM) and focusing on timed repetitions (clocks) and over time work on increasing the time of the sessions (endurance).
  - Aquatic therapy. The patient also attended a 30-minute aquatic therapy session in which she performed exercises focusing on trunk mobility, “unloading,” and strengthening.
  - The final part usually consisted of a modality (usually heat) for 10 minutes in which time the patient is asked to focus on relaxation, breathing, and reflection on her treatment plan and goals. This was also used as a time for the therapist to provide a summary of goals, progress, and plans associated with the next therapy session and home exercise program.
  - Home exercise program (HEP): The HEP was a continuation of the therapy session with focus on the exercises performed in the therapy sessions (neural glides, trunk exercises, light weights, and walking program.

On passive joint movements would not be effective in helping this patient with widespread pain. Second, a biomechanical approach contradicts the neuroscience model, which deemphasizes a specific tissue or joint and focuses on the central sensitization. Third, the patient had seen several other manual therapy providers, including chiropractors, but improvements were transient and may have actually contributed to the fear-avoidance beliefs by focusing the patient on joint and tissue “faults.” Finally, the therapist’s goal was to help the patient become less dependent on health care professionals and focus more on independent management and active approaches such as exercise.

Upon completion of the initial NE session (75 minutes, which included the examination) the patient related that she felt she had a much better understanding of her current condition. At this time, she was asked to complete the same questionnaires and once again perform active forward flexion. The patient was discharged from PT after eight sessions with a complete home exercise program (HEP). The HEP consisted of basic stretches, neural tissue mobilization, and a walking program. The primary therapist kept in contact with the patient by monthly phone calls to monitor progress and answer questions. After 7 months, the patient returned to PT for a follow-up assessment.

Outcomes

Immediate effects

Two of the outcomes measures did not show any apparent change. These were the NPRS and the ODI. The FABQ, however, dropped substantially. The FABQ-W score dropped from 25/42 to 2/42, and the FABQ-PA dropped from a 20/24 to 1/24 after the NE session. The standard errors of measurement (SEM) for the FABQ-PA and FABQ-W have been reported as 2.0 and 2.4, respectively (George, Valencia, and Beneciuk, 2010). Furthermore, the minimal detectable change (MDC) for the FABQ-PA and FABQ-W were reported as 5.4 and 6.8, respectively (George, Valencia, and Beneciuk, 2010). Active forward flexion (with the same verbal instructions given as before) improved dramatically from 10 degrees to 72 degrees immediately following NE. The patient was not asked to rate her pain during the active forward flexion, because the NE session was designed to “de-focus” pain (i.e., knowing there would be pain, but the meaning of the pain [not signaling damage] had been changed) (Moseley, 2003b; Moseley, 2007). The Zung Depression Scale dropped from a score of 58 to 48 after the NE.

Long-Term Effects

The initial plan was to have the patient complete the questionnaires after 4 weeks of physical therapy. Unfortunately, after 4 weeks, the patient called and cancelled her appointment due to a death in the family. At that stage, the patient reported that she felt that her HEP, progressive increase in function, and increased “understanding” of her LBP were beneficial and she planned to continue with her home
program. After 3 months, the primary therapist called the patient to assess her progress, at which time she continued to express satisfaction with her progress. At 7-months after initial visit, the patient called and was still reporting progress, and was willing to return for a formal reevaluation.

At the 7-month follow-up, the patient subjectively reported substantial progress in regards to her ability to perform housework (now able to vacuum two rooms comfortably without a “flare”; able to stand 60 minutes to cook a meal), exercise (walking 4–5 times a week for 30 minutes at a time; and returned to a step-aerobics class), office work (able to return to 3 half days as a receptionist; and be able to sit at a desk 30 minutes at a time and not have a “flare”), and general feeling of well-being. Her general sense of well-being seemed to correlate well with her outcomes measures: Her FABQ-W had risen to 12/42 (initial 25/42, immediate posttreatment 2/42), and her FABQ-PA was 6/24 (initial 20/24, immediate posttreatment 1/24), both of which were still reduced compared to her initial scores 7 months before. Her ODI improved from an initial score of 54% to 40%, whereas her Zung Depression Scale had decreased from a 58 at the initial examination to a 38 at the 7-month follow-up. Her NPRS was still a 7/10 but was reduced from a 9/10 at the initial examination. The patient’s forward flexion was still markedly improved from the initial measurement (10 degrees) to 70 degrees, which was only slightly less than her measurement following the pain science education. The results of her questionnaires and active forward flexion are illustrated in Figure 2. Her new body chart image revealed a “fist sized” area of “discomfort” over the L4/5 spinal region only.

DISCUSSION

The NE session resulted in an immediate change in the patient’s scores and self-report questionnaires, most noticeably the FABQ. Her active forward flexion also showed notable improvement. The physical therapy program combining the NE session with various forms of exercise and therapeutic activities resulted in clinically meaningful changes in pain, function, depression, and active movement.

Although one cannot presume cause and effect from a single case report, the findings suggest that NE may have contributed to an immediate and long-term reduction in disability, fear-avoidance beliefs and depressive feelings as well as an increase in function and physical movement in a patient with CLBP. Although care should be taken when interpreting data from a single case report, the results from this clinical episode suggests that the current findings are important. Our goals for this case report were to present NE in a more clinically manageable time frame (45-minute session vs. 3 hours); demonstrate a clinical reasoning process in which the clinician can make an informed decision for when to apply NE in patient management; and describe in detail the exact content of an abbreviated NE session aimed at explaining the chronic pain state in a non-threatening way to a patient with central sensitivity. This case report may provide clinicians with insights into the clinical application of the latest neuroscience research, and our findings suggest that altering a patient’s thoughts and beliefs regarding CLBP may lead to immediate improvements in function and mobility.

FIGURE 2 Results for pain, disability, fear-avoidance beliefs, depression, and range of motion.
The findings of this case report are consistent with previous research, which have shown that NE results in altered pain cognition along with an increased ability to perform physical tasks (Moseley 2005; Moseley, Nicholas, and Hodges, 2004). In contrast to our patient’s outcomes, subjects in the Moseley, Nicholas, and Hodges (2004) study did not report improvements in perceived disability, whereas our patient reported improvements in her ODI at 7 months. It should be noted that although the patient’s ODI decreased from 54% to 40% and the NRS decreased from 9/10 to 7/10 at the 7-month follow-up, these scores still indicate moderate levels of pain and disability. This may be due to the fact that other interventions were used and may suggest that a multimodal approach involving pain science education and formal physical therapy may maximize outcomes. Although it is not clear why NE may lead to the changes described above, it is hypothesized that this form of education “de-threatens” the situation for the patient (Moseley, 2005). NE should be seen as a form of CBT because it addresses issues related to the nervous system and “how pain works.” It may be that when a patient has a more realistic understanding of his/her pain, he/she becomes less “afraid” of their pain, which leads to a more normalized perception of pain and improved physical performance.

Several functional MRI (fMRI) studies have shown that areas such as the motor and premotor areas are involved in processing pain (Fior, 2000; Moseley, 2005; Tsao and Hodges, 2007). It could be argued that the “de-threatening” of pain may lead the motor area (and other areas) to become less involved in processing pain, and once freed from contributing to a chronic pain response, may now be able to once again focus on their primary functions. In the present case, the most pronounced immediate change occurred with the FABQ. The patient’s initial FABQ-W and FABQ-PA were very high, and these scores dropped sharply following her NE session. It is hypothesized that the educational session may have reduced her fear associated with movement, which allowed her to function at a higher level. This finding is consistent with previous research (George, Fritz, Bialosky, and Donald, 2003; Hodges and Moseley, 2003; Moseley, 2002; Moseley, 2005; Moseley and Hodges, 2002; Moseley, Nicholas, and Hodges, 2004).

Although the focus of this case was to report on the immediate and long-term outcomes for a patient with CLBP following NE, it is important to acknowledge that this patient also received aquatic therapy, cardiovascular exercise, and strengthening exercises, which have been shown to be effective interventions for patients with centralized pain states. We contend that the interaction of exercise and NE is very important. The educational sessions aimed at reinforcing the idea that her tissues had healed but had become “sensitive” and “deconditioned” secondary to the central sensitization. Exercises were described and prescribed by using the same NE wording. For example, as she performed exercises she was constantly reminded that any pain and/or discomfort were more associated with the fact that her nerves had become “sensitive.” Aerobic exercise education was focused on teaching her that aerobic exercises increase blood flow and oxygen to tissues, thus making them healthier. Aerobic exercises also would “flush out” chemicals such as adrenaline and cortisol, which are thought to make nerves more sensitive (Geiss et al, 2005). The patient was also educated on how gradual exposure to discomfort would allow her to not only increase her activities, but improve her “pain tolerance” level (graded exposure). During all of the exercises, the patient was presented with a central and recurring theme: the pain she was experiencing was not indication of tissue injury, but rather sensitivity.

Chronic pain rates are increasing, with data now showing that almost one in every five Americans are experiencing a chronic pain state (Butler and Moseley, 2003; Magni et al, 1993; McMahon and Koltzenburg, 2005), and physical therapists, with their extensive knowledge of anatomy, physiology, neuroscience, and exercise are ideally equipped to manage patients with chronic pain. This case report adds to the current literature suggesting that NE should be considered in the treatment of patients with CLBP (Moseley, 2002; Moseley, 2003a; Moseley, 2003c; Moseley, 2004; Moseley, 2005; Moseley, Nicholas and Hodges, 2004). Finally, this case report illustrates that an abbreviated session of NE can be as effective as the 2- to 3-hour sessions used in research studies (Moseley, 2005), which are not “practical” in clinical practice.

**CONCLUSION**

This case report describes the immediate and long-term results of NE on a patient with a 3-year history of CLBP. The results suggest that NE may help to reduce disability, fear of movement, and depression while increasing active spinal ROM. These outcomes were evident immediately after the first session and were maintained at a 7-month follow-up. The immediate and long-term changes concur with similar studies using NE for patients with CLBP.

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APPENDIX 1. DESCRIPTION OF THE NEUROSCIENCE EDUCATION (PATIENT LANGUAGE)

The Peripheral Nervous System

- Your nervous system is a continuous system. This explains how low back pain can spread and become leg, upper back and neck pain over time.
- The nervous system is an alarm system – designed to warn you of danger. For example a sprained ankle or thumbstuck. Warning you about danger is normal, part of survival.
- Nerves have electrical current inside them and “buzz” all the time. This is normal. (Drawing of electrical activity in a nerve)
- When nerves become excited they have more activity (electricity)
- There is a threshold – when the electricity reaches this, your nerves “fire” and sends a message – probably a danger message.
- With persistent pain and issues such as failed treatment, different (or no) explanations of your pain, fear, stress, job and family issues the nerves “wake up” and “buzz” at a higher level – closer to the threshold of firing. Now, just a little stress, fear, movement or thought can raise the activity to the level needed to make your nerves “fire”.
- Although unpleasant, this “waking up” is a normal part of the nervous system’s alarm function.
- Inside nerves there are sensors. These sensors are there to tell you about your environment. Unfortunately, we now know that when pain persists and your nerves become “sensitive” they can become sensitive to odd things like temperature, stress, anxiety, movement or pressure to name a few. This is normal. As you will see later we can calm nerves down so they don’t become so sensitive.

Take home message: (for the patient)

- Let’s summarize: In your case, you developed back pain and based on many issues (pain, treatment, explanations, job and family) the nerves in your back have become very sensitive, but because your nervous system is one large, attached system that connects your low back to your hip, legs, neck and shoulders, the “system” wakes up. The good news is that we can explain this and the more you understand about this, the more your nerves will calm down.

Practical: (for the therapist)

- Show the patient a picture of a body nervous system
- Draw a graph with nerve activity, time and action potential
- Palpate the ulnar nerve at the cubital tunnel and tibial nerve at the posterior tarsal tunnel. Increased sensitivity to palpation should be tied back to the discussion of nerve sensitization

The Central Nervous System and Neuromatrix

- With the sensitized (and easily firing) nerves in your body, the central nervous system and your brain cannot help but get interested (excited). Again – this is normal.
- To understand pain you need to understand the brain.
  - There are no pain areas in the brain.
  - When you experience pain, pain uses multiple areas in the brain.
  - These areas start communicating with each other.
  - Over years of having pain, these maps become more entrenched and therefore difficult to get rid of.
  - The good news: These maps can be changed. The more you understand about your pain and have less fear, these maps lose their power. Where a map would get ignited and result in pain, a map can actually be engaged and NOT have you experience pain – since the meaning of the experience is different. You understand it more.

- Let’s apply this to your back. Let’s examine you before and after this educational session:

  ■ Before education: Pain is a response to a threat. During your evaluation you were asked to bend over. You bent down a little and said “it hurts”. Because you are afraid of bending over, the nerves in your back fire a lot quicker. Remember – they are more sensitive than normal. These danger messages are sent straight to the brain and the brain processes the danger messages. You run your pain map and because forward bending is a threat (of giving out, hurting, the unknown, etc.) your brain’s summary of the threat is pain. Needless to say – you stop and say “it hurts.” This is normal.

  ■ Understanding pain better: You now realize that the danger messages you got during the forward bending are not a signal of tissue injury or damage. Your back may be sore, sensitive or even deconditioned, but not broken. Tissues heal. Now we ask you to bend forward – you get danger messages, but the danger messages run up to the brain, runs the map, but you are not threatened by this. You now understand that hurt does not equal harm. The map still runs. The map still gives you a sum result, but it may not be pain or if it is, the pain is better understood and thus, less threatening. You can therefore go ahead and bend further.

Take home message: (for the patient)

- Pain is a response by the brain based on the perceived threat. Your pain is real. The pain you experience though may not be a true reflection of your tissues.
- Your whole brain is involved in processing the pain

Practical: (for the therapist)

- Show the patient a functional MRI (fMRI) scan. A good example is the fMRI case study by Moseley which shows both a pre-neuroscience fMRI (lit up/lots of activity) and then post-neuroscience fMRI showing the brain “calmed down”

Output Systems

- With all this “input” there has to be consequences. The brain will call upon systems to defend you – again quite normal.

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The lion metaphor: If a lion jumps into this room right now, what would you do? You will call upon systems to defend you—such as your heart (beating faster), breathing (faster and shallower), muscles (use big muscles to run/hit), shift blood (to areas you need and away from other areas you don’t need right now, for example muscles dealing with posture and stability), immune system (you can worry about the scratchy throat tomorrow when the lion is gone), systems healing tissues (do that tomorrow), sleep (not while there’s a lion in the room), language and more.

These systems are very well designed to work for short times while there’s a threat and once the threat (the lion) goes away, everything returns back to normal.

Here’s the big issue: With persistent pain, issues at home, no/little explanation of your pain, failed treatment, stress, etc. you basically have a huge lion in your life—day in and day out.

These systems have been turned on for years. So what happens? Muscles dealing with posture get sore, tired, fatigued and sensitive. Your breathing is shallow allowing for less blood and oxygen, again making tissues sore, tired and sensitive. You’re in a constant stress mode. You have problems with sleep, getting aches/flu easier and so forth.

Stress chemicals. For your body to protect you, it uses stress chemicals. Two main stress chemicals are adrenaline and cortisol. They shunt blood, get you breathing faster, be more vigilant and more. The problem is that over time they cause your tissues to become sore, sensitive and fatigued. Prolonged high levels of these have also been linked to issues such as memory changes, mood swings, appetite changes, weight gain, sleep disturbances and depression.

Take home message: (for the patient)

- These systems are there to protect you. It is normal. However, these systems need to be restored to normal resting levels. Why are they active? Threat. Every time you experience pain and not understand it (which we already discussed) your systems will get activated. Can you now see why understanding your pain better can help? Basically the large lion (big threat) becomes a small lion cub which is less threatening and you need not call on these systems too much to protect you.

Practical: (for the therapist)

- Have ready a single piece of paper with a lion picture in the center. Present it to the patient and ask what they would do if a lion came into the room. Write down all the systems you identify in this patient as having been activated to protect her.
- Tie it back to the introduction: With tissues now more sore, fatigued and sensitive, the nerves will become even more sensitive, thus a vicious cycle develops.

Treatment Options

- Hopefully by now you have a better (different) understanding of why you still hurt. But how do we treat it?
  - Explanation. The studies show that the more you understand about your pain the better you will do. Hopefully, with the discussion we’ve had thus far, you will realize why this will help. Understanding causes your nerves to “buzz down.”
    - Calming them down.
  - Aerobic exercise. Aerobic exercise helps pump blood and oxygen through your body. This will allow your fatigued tissues to become less fatigued. Also, studies have shown that exercise also calm nerves down, which is a big focus of our treatment. Aerobic exercise also helps with depression, mood, sleep, memory and appetite.
    - Stress chemicals: Aerobic exercise literally “burns” the stress chemicals out of your system. Imagine running a marathon—in the first few miles adrenaline is flowing and you probably think you could “win it all.” As the race progresses, adrenaline levels decrease and you know the rest.
    - Medication. This is the realm of your physician, but realize medications are also used in calming nerves down—such as anti-seizure medication and anti-depressants.

Take home message: (for the patient)

- There are many ways we can make your tissues healthier, better conditioned and less sensitive. Since they’re not injured, they don’t need to be “fixed”
- What if you experience pain during exercise/therapy? By now you should realize that the pain you feel is not necessarily a true reflection of your tissues. As you participate in therapy (hands-on and various forms of exercise) you most likely will experience pain. Your tissues have become so sensitive over the years; it will take a while to calm them down. They are basically “telling you” what the exercise is doing. As you stretch or work on some tissue, the nerves are still so sensitive that they will keep sending danger messages, but hopefully by now you understand this and the brain produces chemicals to dampen those danger messages down. The good news is that you can (and will) over time increase activity and function as the pain steadily decreases.

Practical: (for the therapist)

- It is imperative that the patient develop a greater understanding and appreciation of how therapy may benefit him/her.
- There is no magic exercise or technique, but rather by understanding better how pain works and how therapy can help, the pain process can be reversed and the patient will increase his/her function.

Subsequent therapy visits (for the therapist)

- In the first session, the patient underwent a subjective examination, objective examination and an introductory neuroscience educational session.
- In the therapy sessions to follow, the therapist should:
  - Use any available time (during exercise, manual therapy, etc.) to reinforce the neuroscience message
  - Have the patient explain back to you their understanding of the sections above
  - Focus on cardiovascular exercise
  - Develop a home exercise program
  - Work on setting goals for their job, ADL’s, exercise, recreation and social interaction
  - Set goals for therapy—especially prognosis