Case Report

Quantitative sensory testing changes in the successful management of chronic low back pain

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Individuals with chronic low back pain (LBP) represent a significant percentage of patients in physical therapy practice. The clinical pattern often includes diffuse pain and a variety of sensory complaints, making categorization difficult and leading to diagnoses such as non-specific LBP. Objective measures of sensory changes through quantitative sensory testing may help identify central sensitization of nociceptive pathways in this population. Identification of these somatosensory changes may contribute to clinical decision making and patient management. The purpose of this case report is to present objective evaluation findings, including altered somatosensation, in a patient with a 2-year history of LBP, and to describe changes in function and quantitative sensory testing with successful management.

Keywords: Quantitative sensory testing, Low back pain, Hypoesthesia, Hyperalgesia, Central sensitization

Introduction

Back pain is the most common chronic painful condition, with the annual prevalence in the US ranging from 15 to 45%.¹ In simplest terms, chronic pain has been defined as pain persisting past the healing phase following injury,² yet identifying this endpoint can be difficult. While a number of pathological conditions may give rise to low back pain (LBP), 85% of this population is classified as having non-specific LBP.³ Clinical prediction rules have been developed, utilizing clusters of clinical findings to direct patients with LBP into treatment categories such as manipulation,⁴ stabilization,⁵ or traction.⁶ However, others have proposed that patients with chronic pain should be categorized using a pain mechanism-based classification, suggesting that recognition of clinical patterns of altered somatosensation may help identify and 'diagnose' the stage of pain processing.^{7,8} Quantitative sensory testing (QST) has been recognized as one means of identifying altered nociceptive processing.⁹

Central sensitization is defined as an increased responsiveness of nociceptive neurons in the central nervous system to normal or subthreshold afferent input leading to hyperalgesia.^{10,11} Several studies

have reported signs and symptoms of nociceptive central sensitization in chronic LBP, including QST changes.^{12–15} Persistent noxious afferent input may cause altered nociceptive processing, which may evolve into more lasting changes at the spinal and supraspinal levels.^{16,17}

Alterations in QST may present as heightened responses, such as hyperalgesia to pressure algometry, or as diminished responses, such as hypoesthesia to light touch. Interestingly, hypoesthesia to mechanical and vibration stimuli has been demonstrated concomitantly with hyperalgesia.^{18,19} Likewise, elevated vibrotactile thresholds have been demonstrated in response to experimental pain²⁰ and in patients with painful articular disorders.^{19,21} While these sensory deficits (hypoesthesia) and increased pain sensitivity (hyperalgesia) are believed to be mediated by distinct neurophysiological mechanisms,²² both are triggered by nociceptive input and considered to be centrally mediated, either at spinal and/or supraspinal levels. Clinically, this may result in functional changes. Individuals with chronic back pain often demonstrate reduced proprioceptive acuity in the lumbar region²³ and find subtle movements of their pelvis and back more difficult to perform than people without back pain.^{17,24} Management of chronic LBP is often impairment-based with treatment goals directed towards decreasing pain, increasing strength, and normalizing somatosensory and functional deficits.²⁵ However, few studies

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Figure 1 Pain diagram at initial evaluation.

have described how sensory findings may change in relation to treatment outcomes.

The purpose of this case report was to describe how objective findings changed over 14 physical therapy visits (10 weeks) in a patient with chronic LBP, following a treatment regimen which included both thrust and non-thrust manipulation and in the latter stages, a specific motor control exercise program. The case highlights how unsuccessful management triggered QST measures, thereby generating an alternative and more successful treatment plan.

Case Description

The patient was a 33-year-old female graduate student that presented with a 2-year history of LBP and bilateral lower extremity pain. The pain was located bilaterally (R>L) at the posterior superior iliac spine (PSIS) and posterior mid-thighs (Fig. 1). This pain varied from 2/10 to 9/10 on the numeric pain rating scale (NRPS) depending on activity level. Numbness on the plantar surface of both first toes and dorsal aspect of her right first and second toes was also reported.

The patient reported her first experience of symptoms 2 years previously while at an amusement park. After exiting a roller coaster, she had a sharp onset of pain that radiated into the right lower extremity and resolved immediately. Several days later, she noticed a progressive onset of burning and fatigue bilaterally at the PSISs and posterior mid-thighs. She denied any prior history of back, leg, or foot pain.

The patient reported 7/10 pain with sitting >30 minutes, particularly when driving, which would resolve to 2/10 with 5–10 minutes of movement or lying prone. She had difficulty ambulating >1 mile

due to pain (4/10) with gradual onset of burning and fatigue in both lower extremities and feet. Her regular fitness and running program were limited by pain. She stated that she slept through the night without waking, and was always able to go to sleep regardless of the presence of pain. Symptoms were better in the morning compared to evening depending upon her workload. She denied weight loss, bowel or bladder dysfunction, saddle anesthesia or stumbling while walking.

Several radiographic imaging studies were completed in the 6 months following the injury. Initial radiographs and computed tomography scan of the lumbar spine were both judged as unremarkable. Magnetic resonance imaging (MRI) of the lumbar spine was deemed essentially normal, noting early degenerative change of the L4–L5 disc, and very minor posterior disc bulge at L4–L5 and L5–S1 without neural compromise.

Further MRI studies of the brain, cervical, and thoracic spine to rule out multiple sclerosis or cord compression were unremarkable. A lower extremity nerve conduction velocity study, as well as complete blood count and urinalysis, was deemed negative. She was placed on a trial of Pregabalin, Gabapentin, and Naproxen, all without success. Four visits of physical therapy at another facility including soft tissue massage and general core strengthening exercises of pelvic tilts and general theraball exercises did not change symptoms.

She noted a progressive worsening of pain since onset, with the numbness in the bilateral great toes progressively increasing over the past year. The patient denied any past medical or surgical history. Her goals were to drive without pain and to return to a regular fitness routine.

Examination

The patient had a lean body type with a body mass index of 17.7. In standing, she demonstrated a normal lumbar lordosis and neutral pelvis. Examination findings may be found in Table 1. Movement testing of the lumbar spine revealed no aberrant movement during active flexion and extension; she was able to return to standing erect without difficulty.

Prone extension was limited to 75%, and with repeated movement of 10 repetitions, only central discomfort at the L5–S1 level was reported without PSIS or lower extremity symptoms (with exception of the toe numbness) upon reassessment in standing. Hip muscle weakness was found bilaterally with manual muscle testing, revealing 4-/5 for both the hip extensors and abductors. Sacroiliac joint (SIJ) pain provocation testing as described by Laslett *et al.*²⁶ was positive on only two of five tests, decreasing the

likelihood of SIJ dysfunction. Hypomobility was demonstrated with central and right unilateral posterior–anterior (PA) movement at L5–S1 and L4–L5, which reproduced her bilateral PSIS pain to 3/10.²⁷ Reliability of these assessments has been deemed as fair, though percentage agreement among examiners is high and these measurements have been utilized in determining those who will respond to lumbar manipulation.^{28–31} The patient scored 30% on the Modified Oswestry Low Back Pain Disability Questionnaire (MODQ) placing her at moderate disability level.³² The MODQ has been well established regarding reliability and validity in the outcomes of treatment for LBP.³² Other outcomes measures utilized included NPRS, the global rating of change (GROC), and the patient's report in functional improvement. The NPRS has been shown to be a reliable and responsive outcome measure for individuals experiencing LBP with a minimal clinically important difference (MCID) of 2 points.³³ Similarly, the 11-point GROC has also been shown to be responsive with an MCID of 2 points.³⁴

At completion of the initial evaluation, loss of range of motion and pain were deemed as her main impairments. A discogenic source of symptoms with possible mild nerve root irritation was hypothesized,³⁵ due to aggravation of symptoms with repeated lumbar flexion and ease of symptoms with repeated lumbar extension, in addition to the hypomobility found with passive joint accessory testing. She was issued a home program including prone and standing extension exercises. Prognosis was good given her age, health, relative insight into her disorder, and response to repeated extension, though chronicity and inability to reproduce or change distal lower extremity symptoms were taken into consideration. Given the hypomobility of the lumbar spine and failure to respond to core strength in previous physical therapy, clinical lumbar instability was judged to be unlikely. Although chronic, no further sensory

examination was initiated because the symptoms were considered mechanical in nature. Planned treatment included lumbar extension exercises, modification of sitting posture, and thrust manipulation of the lumbar spine if necessary.

Visits 1–5 (0–3 weeks from initial examination)

Thrust and non-thrust manipulations were added during subsequent physical therapy sessions without any overall improvement in symptoms. Utilized interventions are summarized:

1. soft tissue massage to paraspinals;
2. central and unilateral PA oscillatory mobilization of L4, L5, and SIJ (grades III and IV);²⁷
3. passive physiological intervertebral movements into rotation at L5/S1; right and left (grade IV);²⁷
4. high-velocity low-amplitude rotational thrust — L5/S1 right/left
5. home exercise program including:
 - prone and standing lumbar extension;
 - supine lumbar rotation bilaterally;
 - lumbar support for sitting and driving.

Due to her lack of response to mobility-based treatments, trunk strength was reassessed during her fifth visit. The prone plank test on elbows and knees was examined as a global core strength assessment.³⁶ She could maintain this position for 88 seconds, though it reproduced her back and leg pain minimally 30 seconds following testing. She was issued core strength exercises including prone plank holds, prone bent knee lifts, and supine bridging with alternate straight leg raises.

Visits 6–7 (4–5 weeks from initial examination)

The patient reported worsening symptoms with performance of core and hip strengthening exercises, noting immediate aggravation of bilateral PSIS and posterior mid-thigh pain with sitting or driving and increased pain. These exercises were discontinued at visit 6. The MODQ and GROC were unchanged. Her pain levels on the NPRS continued to be significant, with maximal levels of 9/10. Functional limitations were unchanged from the initial evaluation.

Table 1 Examination findings at initial visit

Range of motion	Limitation (degrees)	Symptoms
Lumbar flexion	None	Increased with repeated
Lumbar extension	30	Central discomfort
Lumbar lateral flexion	None	None
Lumbar rotation	None	None
Extension quadrant	None	Central discomfort
Hip flexion/extension	None	None
Hip internal/external rotation	L: 40/40; R: 60/30	None
Hip abduction/adduction	None	
Neurological screening (lower extremity)		
Mechanical detection (light touch)	Right ↓ R first and second digits	Left No deficit
Myotomes (L2–S2)	No deficit	No deficit
Deep tendon reflexes patella/Achilles	Intact	Intact
Babinski reflex	Negative	Negative
Straight leg raise test	Negative	Negative
Slump test	Negative	Negative

Figure 2 Quantitative sensory measures at visit 6 (A) and final session (B; visit 14). Gray area indicates vibratory sensation loss centrally and unilaterally; bars indicate two-point discrimination measures. At visit 6, vibration perception was absent at S1 and S2 centrally; unilateral loss was noted on the right at L5, and on the right and left of S1. Two-point discrimination was increased at L4, L5, S1, and S2. These measures became similar to measures above and below following 4 weeks of treatment. Vibratory sensation was intact at all segments with the exception of L5 at final collection.

Due to the chronic nature of her symptoms, QST measures were undertaken to investigate the possibility of central sensitization of the nociceptive pathways. QST measures selected were pressure pain threshold (PPT), two-point discrimination (TPD), and vibratory sensation. TPD testing was completed with an aesthesiometer on the left and right sides of the back at each segmental level.¹⁷ The testing method is described by Moberg³⁷ where the two points of the instrument are applied to the skin until blanching occurs. The subject reports if one or two points are felt. Distance between points was increased by 5-mm increments, and consistent identification of three trials of a given measure was recorded (Fig. 2).

Vibratory sensation testing was completed with two identical 128-Hz tuning forks at L1–S4 levels over the spinous process and 2 cm lateral on the right and left. Two tuning forks were used to avoid auditory cues to the patient. Either a vibrating or non-vibrating fork was applied to the skin; presence or non-presence of vibration sensation was queried (Fig. 2).

Pressure pain threshold was examined with an algometer over the right and left facet joints as

described by Vanderweeen *et al.*³⁸ (Table 2). Briefly, a JTech Commander pressure algometer (1.0 cm² tip; JTech Medical, Salt Lake City, UT, USA) was applied at a perpendicular angle at a rate of 50 kPa/s until the patient reported the change from pressure sensation to pain. PPT results at the symptomatic levels of the lumbar spine are similar to previously reported values in this patient population.³⁹

Further assessment of lumbar stabilizing musculature was also performed. Review of the patient's MRI results revealed increased fatty deposition in the multifidi bilaterally at the L5 level, which is consistent with findings in subjects with chronic LBP.⁴⁰ The prone instability test was negative. Transverse abdominus (TA) and multifidus muscle contractions were then assessed utilizing the 'drawing-in' maneuver described by Teyhen *et al.*⁴¹ This was found to preferentially activate the TA with minimal changes in the internal oblique.⁴² Both muscles were found to demonstrate decreased strength and impaired coordination of contraction on the right in comparison to the left side. This was without symptom reproduction. A spinal stabilization program was initiated with specific emphasis on pain-free performance and progression of exercise. This consisted of supine individual and co-contraction of TA and multifidus with 5-second holds in hook lying. Training of these specific muscles has been demonstrated to improve function and decrease pain in subjects with LBP.^{43–45} Continued use of lumbar support with sitting and driving was recommended.

Visits 8–14

Over the course of a month, the patient was trained in a specific spinal stabilization program emphasizing activation of the right TA and multifidus. The task

Table 2 Pressure pain measures at initial (visit 6) and final (visit 14) sessions

	Initial		Post	
	L	R	L	R
Pressure pain threshold (kPa)				
L1	646.0	679.1	682.6	682.6
L2	682.6	744.6	751.5	758.4
L3	579.2	703.3	765.3	765.3
L4	600.5	664.0	703.3	765.3
L5	585.4	703.3	676.4	737.7
S1	618.5	581.9	633.6	648.8
S2	588.1	388.2	615.7	612.3
S3	464.0	382.0	539.9	436.4
S4	445.4	415.1	412.3	345.4

was progressed from greater to less stable postures and from static to increasingly complex movements⁴⁶ including postures of supine hook lying, sitting, quadruped with alternating upper extremity lifts, and quadruped with alternating lower extremity lifts. All were performed in a pain-free manner. Towards the end of these sessions, the patient was re-introduced to regular cardiovascular exercise via the elliptical or stationary bicycle 2–3 times per week without symptom aggravation. The patient noted that her pain had decreased in intensity to no greater than a 2–3/10 and was located mostly at the right PSIS, with less buttock and posterior thigh symptoms. She stated that the pain was less frequent while sitting, driving, and standing. She noted that she was able to obtain temporary relief from symptoms with performance of spinal stabilization exercises while seated for longer periods. On session 14, pain on NPRS was rated at 0/10 and 3/10 at worst over the past week, which met the MCID. A two-point improvement in her MODQ score (26%) remained below the MCID, even though she reported functional improvements such as walking for over 2 hours several times/week and the ability to sit for prolonged periods. She reported a GROC score of +4 (moderately better). QST was also reassessed (Table 2 and Fig. 2). The patient was able to consistently identify the sensation of vibration at all levels centrally and unilaterally with the exception of L5 on the right. TPD had improved along with increased core strength, coordination and report of pain. PPTs had overall increased as well, indicating less hyperalgesia.

Discussion

The patient in this case presented with subjective complaints, imaging studies, and physical examination findings, including significant hypomobility consistent with discogenic pain. A clinical prediction rule for predicting failure with stabilization treatment has identified four factors — fear avoidance beliefs questionnaire physical activity subscale score of <9, lack of aberrant movement with lumbar flexion, lack of lumbar hypermobility with PA testing, and a negative prone instability test.⁵ Presence of 2/4 of these factors was associated with a negative likelihood ratio of 0.18, indicating that the patient may be more likely to benefit from an alternative treatment approach. Given that she presented with three predictors, it would appear that she would be unlikely to respond to a spinal stabilization program.

Conversely, a clinical prediction rule for predicting success with spinal stabilization has also been proposed.⁵ The variables identified were age <40 years, average straight leg raising >91 degrees, a positive prone instability test, and aberrant movement

present with lumbar flexion. In this case, the patient was positive for only one of these factors (age <40 years) yet was still successful with the utilization of a spinal stabilization program.

Given the patient's initial presentation, it was hypothesized that she would benefit from lumbar mobilization and manipulation, as well as extension-based exercise. Yet little progress was made following 6 weeks of manual therapy treatment even though symptom provocation was not reported. With addition of higher level core strength exercises, her symptoms were aggravated, even though she was able to complete them consistently and at the level of healthy controls.

A limitation of the case report is that QST was not performed until the sixth visit. Through QST, it was demonstrated that the patient had objective findings of both hyperalgesia (PPT) and hypoesthesia (vibration and TPD) in the area of the lumbar spine. Although seemingly paradoxical, both have been identified as potential consequences of central sensitization of nociceptive pathways, yet may be mediated by different mechanisms.²² Furthermore, these nociceptive changes may occur at a spinal and/or supraspinal level.^{20,22} With regard to hypoesthesia, deficits in vibration perception were found, consistent with previous QST studies in chronic LBP.^{47,48} TPD thresholds in this patient were higher on the more symptomatic side, although both sides were increased as compared to asymptomatic levels. These findings correspond with previous studies in persons with LBP,^{17,24} in which altered TPD was identified and associated with a poorer performance in a set of lumbopelvic motor control tasks²⁴ and distorted body perception.¹⁷ This was attributed, at least in part, to cortical reorganization. The cause/effect relationship between pain and altered motor control is complex and likely bidirectional. Hyperalgesia was objectively demonstrated through algometric measures at the lumbar spine. While some studies in this patient population have demonstrated signs of widespread hyperalgesia through algometric measures at a remote site such as the hand, these measures were not taken in this patient, which is a limitation of this case report. Nevertheless, regional hyperalgesia, as was found in this case, and widespread hyperalgesia may simply be progressive stages in central sensitization.³⁹

Thrust and non-thrust manipulations were not effective in modulating pain, nor was gross core strengthening in this patient. Instead, a program of specific, non-provocative, graduated strengthening of deep stabilizers led to a decrease in symptoms. Specific training of deep stabilizers may be critical in the management of chronic LBP, as has been suggested previously.⁴⁴ Alternatively, directed exercise

which does not further facilitate the hyperexcitable nociceptive pathways (i.e., the flare response) may be a critical element in the management of LBP.

Noteworthy in this case was the finding that QST measures correspondingly normalized as pain diminished and the patient's report of function improved. Measures of hypoesthesia improved and (Fig. 2), likewise, PPT increased at most segments (see Table 2). QST may serve as an important objective outcome measure in painful musculoskeletal conditions.⁷ In contrast, MODQ scores changed little over the course of treatment in this case. Previous research has questioned the responsiveness of the MODQ⁴⁹ while other work has substantiated it.⁵⁰ Future studies investigating the relationship between sensory changes and functional impairment in persons with chronic pain may clarify underlying mechanisms and better direct management strategies.

References

- 1 Manchikanti L, Singh V, Datta S, Cohen SP, Hirsch JA. Comprehensive review of epidemiology, scope, and impact of spinal pain. *Pain Physician* 2009;12:E35–70.
- 2 Apkarian AV, Baliki M, Geha P. Towards a theory of chronic pain. *Prog Neurobiol* 2009;87:81–97.
- 3 Indahl A, Velund L, Reikeras O. Good prognosis for low back pain when left untampered. A randomized clinical trial. *Spine (Phila Pa 1976)* 1995;20:473–7.
- 4 Childs JD, Fritz JM, Flynn TW, Irrgang JJ, Johnson KK, Majkowski GR, et al. A clinical prediction rule to identify patients with low back pain most likely to benefit from spinal manipulation: a validation study. *Ann Intern Med* 2004;141:920–8.
- 5 Hicks GE, Fritz JM, Delitto A, McGill SM. Preliminary development of a clinical prediction rule for determining which patients with low back pain will respond to a stabilization exercise program. *Arch Phys Med Rehabil* 2005;86:1753–62.
- 6 Cai C, Pua YH, Lim KC. A clinical prediction rule for classifying patients with low back pain who demonstrate short-term improvement with mechanical lumbar traction. *Eur Spine J* 2009;18:554–61.
- 7 Courtney CA, Kavchak AE, Lowry CD, O'Hearn MA. Interpreting joint pain: quantitative sensory testing in musculoskeletal management. *J Orthop Sports Phys Ther* 2010;40:818–825.
- 8 Woolf CJ, Bennett GJ, Doherty M, Dubner R, Kidd B, Koltzenburg M, et al. Towards a mechanism-based classification of pain? *Pain* 1998;77:227–9.
- 9 Arendt-Nielsen L, Yarnitsky D. Experimental and clinical applications of quantitative sensory testing applied to skin, muscles and viscera. *J Pain* 2009;10:556–72.
- 10 Sandkuhler J. Models and mechanisms of hyperalgesia and allodynia. *Physiol Rev* 2009;89:707–58.
- 11 International Association for the Study of Pain. IASP taxonomy [document on the Internet]. 2008. Available from: www.iasp-pain.org/AM/Template.cfm?Section=Pain_Defi...isplay.cfm&ContentID=1728.
- 12 O'Neill S, Manniche C, Graven-Nielsen T, Arendt-Nielsen L. Generalized deep-tissue hyperalgesia in patients with chronic low-back pain. *Eur J Pain* 2007;11:415–20.
- 13 Schliessbach J, Arendt-Nielsen L, Heini P, Curatolo M. The role of central hypersensitivity in the determination of intradiscal mechanical hyperalgesia in discogenic pain. *Pain Med* 2010;11:701–8.
- 14 Giesbrecht RJ, Battie MC. A comparison of pressure pain detection thresholds in people with chronic low back pain and volunteers without pain. *Phys Ther* 2005;85:1085–92.
- 15 Giesecke T, Gracely RH, Grant MA, Nachemson A, Petzke F, Williams DA, et al. Evidence of augmented central pain processing in idiopathic chronic low back pain. *Arthritis Rheum* 2004;50:613–23.
- 16 Graven-Nielsen T, Arendt-Nielsen L. Peripheral and central sensitization in musculoskeletal pain disorders: an experimental approach. *Curr Rheumatol Rep* 2002;4:313–21.
- 17 Moseley GL. I can't find it! Distorted body image and tactile dysfunction in patients with chronic back pain. *Pain* 2008;140:239–43.
- 18 Hendiani JA, Westlund KN, Lawand N, Goel N, Lisse J, McNearney T. Mechanical sensation and pain thresholds in patients with chronic arthropathies. *J Pain* 2003;4:203–11.
- 19 Shakoor N, Agrawal A, Block JA. Reduced lower extremity vibratory perception in osteoarthritis of the knee. *Arthritis Rheum* 2008;59:117–21.
- 20 Apkarian AV, Stea RA, Bolanowski SJ. Heat-induced pain diminishes vibrotactile perception: a touch gate. *Somatosens Mot Res* 1994;11:259–67.
- 21 Hollins M, Sigurdsson A, Fillingim L, Goble AK. Vibrotactile threshold is elevated in temporomandibular disorders. *Pain* 1996;67:89–96.
- 22 Geber C, Magerl W, Fondel R, Fecher M, Rolke R, Vogt T, et al. Numbness in clinical and experimental pain — a cross-sectional study exploring the mechanisms of reduced tactile function. *Pain* 2008;139:73–81.
- 23 Lee AS, Cholewicki J, Reeves NP, Zazulak BT, Mysliwiec LW. Comparison of trunk proprioception between patients with low back pain and healthy controls. *Arch Phys Med Rehabil* 2010;91:1327–31.
- 24 Luomajoki H, Moseley GL. Tactile acuity and lumbopelvic motor control in patients with back pain and healthy controls. *Br J Sports Med* 2011;45:437–40.
- 25 Kent P, Mjosund HL, Petersen DH. Does targeting manual therapy and/or exercise improve patient outcomes in nonspecific low back pain? A systematic review. *BMC Med* 2010;8:22.
- 26 Laslett M, Aprill CN, McDonald B, Young SB. Diagnosis of sacroiliac joint pain: validity of individual provocation tests and composites of tests. *Man Ther* 2005;10:207–18.
- 27 Maitland GD, Hengeveld E, Banks K, English K. Vertebral manipulation. 7th ed. Edinburgh: Elsevier; 2005. p. 369–71.
- 28 Binkley J, Stratford PW, Gill C. Interrater reliability of lumbar accessory motion mobility testing. *Phys Ther* 1995;75:786–92.
- 29 Flynn T, Fritz J, Whitman J, Wainner R, Magel J, Rendeiro D, et al. A clinical prediction rule for classifying patients with low back pain who demonstrate short-term improvement with spinal manipulation. *Spine (Phila Pa 1976)* 2002;27:2835–43.
- 30 Maher C, Adams R. Reliability of pain and stiffness assessments in clinical manual lumbar spine examination. *Phys Ther* 1994;74:801–9.
- 31 Hicks GE, Fritz JM, Delitto A, Mishock J. Interrater reliability of clinical examination measures for identification of lumbar segmental instability. *Arch Phys Med Rehabil* 2003;84:1858–64.
- 32 Davidson M. Rasch analysis of three versions of the Oswestry Disability Questionnaire. *Man Ther* 2008;13:222–31.
- 33 Childs JD, Piva SR, Fritz JM. Responsiveness of the numeric pain rating scale in patients with low back pain. *Spine (Phila Pa 1976)* 2005;30:1331–4.
- 34 Costa LO, Maher CG, Latimer J, Ferreira PH, Ferreira ML, Pozzi GC, et al. Clinimetric testing of three self-report outcome measures for low back pain patients in Brazil: which one is the best? *Spine (Phila Pa 1976)* 2008;33:2459–63.
- 35 O'Neill CW, Kurgansky ME, Derby R, Ryan DP. Disc stimulation and patterns of referred pain. *Spine (Phila Pa 1976)* 2002;27:2776–81.
- 36 Cowley PM, Fitzgerald S, Sottung K, Swensen T. Age, weight, and the front abdominal power test as predictors of isokinetic trunk strength and work in young men and women. *J Strength Cond Res* 2009;23:915–25.
- 37 Moberg E. Two-point discrimination test. A valuable part of hand surgical rehabilitation, e.g. in tetraplegia. *Scand J Rehabil Med* 1990;22:127–34.
- 38 Vanderweelen L, Oostendorp RA, Vaes P, Duquet W. Pressure algometry in manual therapy. *Man Ther* 1996;1:258–65.
- 39 Binderup A, Arendt-Nielsen L, Madeleine P. Pressure pain sensitivity maps of the neck-shoulder and the low back regions in men and women. *BMC Musculoskelet Disord* 2010;11:234.
- 40 Kjaer P, Bendix T, Sorensen JS, Korsholm L, Leboeuf-Yde C. Are MRI-defined fat infiltrations in the multifidus muscles associated with low back pain? *BMC Med* 2007;5:2.
- 41 Teyhen DS, Miltenberger CE, Deiters HM, Del Toro YM, Pulliam JN, Childs JD, et al. The use of ultrasound imaging of the abdominal drawing-in maneuver in subjects with low back pain. *J Orthop Sports Phys Ther* 2005;35:346–55.

- 42 Teyhen DS, Rieger JL, Westrick RB, Miller AC, Molloy JM, Childs JD. Changes in deep abdominal muscle thickness during common trunk-strengthening exercises using ultrasound imaging. *J Orthop Sports Phys Ther* 2008;38:596–605.
- 43 Ferreira ML, Ferreira PH, Latimer J, Herbert RD, Hodges PW, Jennings MD, *et al.* Comparison of general exercise, motor control exercise and spinal manipulative therapy for chronic low back pain: A randomized trial. *Pain* 2007;131:31–7.
- 44 Rasmussen-Barr E, Ang B, Arvidsson I, Nilsson-Wikmar L. Graded exercise for recurrent low-back pain: a randomized, controlled trial with 6-, 12-, and 36-month follow-ups. *Spine (Phila Pa 1976)* 2009;34:221–8.
- 45 Hides JA, Jull GA, Richardson CA. Long-term effects of specific stabilizing exercises for first-episode low back pain. *Spine (Phila Pa 1976)* 2001;26:E243–8.
- 46 Gentile AM. A working model of skill acquisition with applications to teaching. *Quest* 1972;17:3–23.
- 47 Blumenstiel K, Gerhardt A, Rolke R, Bieber C, Tesarz J, Friederich HC, *et al.* Quantitative Sensory Testing Profiles in Chronic Back Pain Are Distinct From Those in Fibromyalgia. *Clin J Pain* 2011; Epub ahead of print.
- 48 Freynhagen R, Rolke R, Baron R, Tölle TR, Rutjes AK, Schu S, *et al.* Pseudoradicular and radicular low-back pain — a disease continuum rather than different entities? Answers from quantitative sensory testing. *Pain* 2008;135:65–74.
- 49 Wilson HD, Mayer TG, Gatchel RJ. The lack of association between changes in functional outcomes and work retention in a chronic disabling occupational spinal disorder population: implications for the minimum clinically important difference. *Spine (Phila Pa 1976)* 2011;36:474–80.
- 50 Frost H, Lamb SE, Stewart-Brown S. Responsiveness of a patient specific outcome measure compared with the Oswestry Disability Index v2.1 and Roland and Morris Disability Questionnaire for patients with subacute and chronic low back pain. *Spine (Phila Pa 1976)* 2008;33:2450–7.