A classification and treatment protocol for low back disorders Part 3 – Functional restoration for intervertebral disc related disorders

Jon Joseph Ford, Andrew John Hahne, Alexander Ying Pui Chan, Luke Desmond Surkitt

Musculoskeletal Research Centre, Faculty of Health Sciences, La Trobe University, Bundoora, Victoria, Australia

Background: It has been widely recommended that clinical trials on people with low back disorders (LBDs) should have a greater focus on subgroup specific treatment in order to increase the likelihood of clinically meaningful effects being demonstrated. Functional restoration is a treatment approach that has demonstrated some evidence of effectiveness in subacute and chronic LBDs. However, studies to date have not used a clearly defined and appropriately detailed clinical protocol or applied the treatment to homogenous pathoanatomical based subgroups.

Objectives: This paper presents a detailed classification and treatment protocol for people with LBDs and clinical features thought to be indicative of non-reducible discogenic pain or disc herniation with associated radiculopathy.

Discussion: A pathoanatomical interpretation of traditional functional restoration, classification principles and particular treatment components in the clinical protocol are discussed.

Conclusion: The described clinical protocol will be used in the Specific Treatment for Problems of the Spine trials comparing specific physiotherapy to evidence-based advice.

Keywords: Back pain, Classification, Functional restoration, Exercise, Lumbar intervertebral disc, Subgroup

Introduction

This is the third in a series of papers describing a clinical protocol for the classification and specific treatment of low back disorders (LBDs). The protocol was developed for the Specific Treatment of Problems of the Spine (STOPS) trials.¹ Low back disorders are a prevalent and costly burden to society and the individual.²⁻⁵ There is minimal evidence supporting physiotherapy treatment as an effective strategy in dealing with this problem.^{6,7} Randomized controlled trials (RCTs) evaluating treatment specific to identified LBD subgroups have the potential for providing stronger evidence supporting the effectiveness of physiotherapy.^{7–10} There are a variety of approaches to developing and validating LBD subgroups^{8,11} with each method having significant limitations.^{8,12,13} Parts 1 and 2 of this series described and provided a justification for our approach to classification based on identifying pathoanatomical subgroups of people with subacute LBDs.14,15 The described method involved the refinement of clinical

methods in widespread current clinical use within the context of the best available research evidence in a manner consistent with evidence-based principles.¹⁶ In this third part of the series, a clinical protocol will be described for subgroups of LBD related to the lumbar intervertebral disc.

Reducible discogenic pain (RDP) is an LBD subgroup with specific clinical features including a positive response to specific movements and postures, commonly called mechanical loading strategies (MLSs).^{14,17} The term 'reducible' refers to the intradiscal phenomenon of displaced and symptom provoking nucleus pulposus material being reduced by MLSs to a more central and less symptom provoking position.^{17,18} Irreducible or non-reducible discogenic pain (NRDP) has been proposed as an additional LBD subgroup where clinical features of RDP are present, in the absence of a positive response to MLSs.^{17–19} There have been no clinical trials on the effectiveness of conservative treatment for people with NRDP.

Disc herniation involves a localized displacement of intervertebral disc material beyond the normal margins of the disc space.²⁰ When a disc herniation damages a spinal nerve root via mechanical and/or

Correspondence to: Jon Joseph Ford, Group Leader, Low Back Research Team, Musculoskeletal Research Centre, Faculty of Health Sciences, La Trobe University, Bundoora, Victoria 3085, Australia. Email: j.ford@ latrobe.edu.au

chemical irritation, radicular leg pain and/or clinical signs of radiculopathy (impaired reflexes, sensation or strength) are commonly observed.^{21,22} While various surgical and conservative treatments have been proposed for the management of disc herniation with associated radiculopathy (DHR), it is not clear which conservative treatments are most effective.²³

The term functional restoration (FR) was first coined by Mayer who defined it as 'a multimodal pain management program that employs a comprehensive cognitive-behavioural treatment orientation to help patients better cope with, and manage, their pain... while undergoing the sports medicine physical approach to correct functional deficits.²⁴ (p. 483) Based on recent systematic reviews,^{25,26} the key features of FR include:

- an overall aim to restore reasonable capacity for activities of daily living including work;
- negotiation of meaningful goals at program commencement;
- development of graded exercise and non-exercise based activity schedules;
- the graded exercise program approximating functional tasks in a safe and supervised clinical environment to increase psychological and physical tolerances;
- a focus on increasing strength, flexibility and cardiovascular fitness;
- a cognitive-behavioural approach to address psychosocial barriers to achieving goals.

Despite promising results from earlier studies,²⁷ there is currently a lack of consistent evidence supporting the effectiveness of FR programs for subacute and chronic LBDs.²⁵ There is some evidence that multidisciplinary FR may be effective in improving pain and activity capabilities in chronic LBDs.²⁸ The effect of single discipline FR has not been evaluated in a current systematic review although the results from some individual trials seem promising.^{29–32}

Within and between trial heterogeneity may be responsible for the varied results to date.^{25,28} Many of the principles of FR are equally applicable to people with NRDP and DHR as to those with non-specific LBDs. However, based on injury severity, complexity and a poorer prognostic outlook, NRDP and DHR represent subgroups of LBDs that are likely to require a modified FR approach with a greater focus on the pathoanatomical barriers to recovery. The purpose of the third part in this series is to present a clinical protocol for people with NRDP and DHR. This classification and treatment approach was developed for use in the STOPS trials on a population of people with non-compensable, subacute LBDs classified as having NRDP or DHR.

Method

The STOPS trials protocol and methodology has been described elsewhere¹ and adheres to accepted

guidelines for conducting RCTs.^{33–36} The key features of the STOPS trials were:

- classification of potential participants into one of five subgroups at baseline assessment. Two of the subgroups were NRDP and DHR;
- consenting participants being randomly allocated to either subgroup specific physiotherapy (10 sessions over 10 weeks) or evidence-based advice (2 sessions over 10 weeks);
- treatment in both physiotherapy and advice groups being specific to the relevant subgroup;
- separate trials being completed for each subgroup (including NRDP and DHR);
- follow-up of participants at 5 weeks, 10 weeks, 26 weeks and 12 months.

Classification of non-reducible discogenic pain

The nomenclature for NRDP is predicated on the definition of RDP. The classification of RDP has been described in Part 2 of this series¹⁴ and is supported by extensive research on biological plausibility,^{37,38} concurrent validity,³⁹ predictive validity⁴⁰ and effective-ness in classification based RCTs.⁴¹ Large multi-disciplinary surveys⁴² as well as an expert panel using the Delphi Technique⁴³ have identified the features that practitioners and researchers believe are important in the classification of RDP. In the context of this literature, RDP can be conceptualized as a painful annular tear where the position of the nucleus pulposus can be influenced by MLSs to ease symptoms and promote more rapid recovery.

Non-reducible discogenic pain is defined by the features of RDP in the absence of a positive response to MLSs. The concept of discogenic pain not responsive to standard treatments was initially proposed by Crock⁴⁴ who described a chemically mediated painful degradation of the intervertebral disc which he called internal disc disruption. On the basis of clinical impression and an evaluation of the literature, Crock postulated that traumatic damage to the intervertebral disc and vertebral end plate could cause an irritant substance to drain into the spinal canal and/or vertebral body with subsequent initiation of an auto-immune response. This response was hypothesized as causing an internal process of disc degradation/disruption leading to annular tearing and irritation of the free nerve endings in the outer third of the annulus fibrosus. Radiological findings indicative of this process included positive lumbar discography in conjunction with normal X-ray and computerized tomography scan findings. Additional clinical features included intractable back pain and leg pain, generalized and pain inhibited loss of lumbar movement, significant disability, depression and normal neurological findings. Since Crock's initial descriptions further research has been published supporting such pathophysiological processes rendering a lumbar intervertebral disc as symptomatic.37,45

The clinical features and mechanisms of NRDP have also explored in a recent international Delphi survey of 21 expert physiotherapists.⁴³ The experts reached consensus on a number of clinical features indicative of NRDP including worsening of symptoms in response to certain MLSs, constant pain, symptoms being difficult to control, positive discography findings and an increase in symptoms with most lumbar movements. The mechanisms underpinning NRDP described by the expert physiotherapists were similar to the descriptions of Crock for internal disc disruption as well as other researchers^{46–48} and focused on the presence of inflammation within an annular tear as the most likely cause of non-responsiveness to MLSs.

Evidence also exists suggesting symptomatic discs may be rendered non-responsive to treatment due to an increased density and/or chemical sensitization of nociceptive afferents in the degenerated annulus fibrosis.^{38,49,50} Typically MLSs involve lumbar extension^{14,51} which compresses the posterior annulus fibrosis.³⁷ It is plausible that an inflamed posterior annular tear, particularly in the presence of a greater density of nociceptive afferents, would most likely be aggravated, and certainly not improved by such movements or postures. These proposed clinical features and mechanisms of NRDP are consistent with the clinical observations and basic science interpretations of other experts.^{17,19,38}

A variety of approaches for the development and validation of LBD subgroups/classification systems have been recommended,^{9,17,19,39} each one having significant limitations.⁸ As discussed in Part 1 of this series, there are no acceptable gold/reference standards with which to fully validate the clinical features of LBD subgroups^{8,52,53} including NRDP. There is however, a convergence of evidence^{12–14} supporting the validity of NRDP as a subgroup of LBDs based on the validity of RDP, clinical observations on NRDP from multiple sources including a formal expert panel of physiotherapists, and potential mechanisms underpinning NRDP.

Of particular relevance is our clinical observation, supported by pathoanatomical mechanisms, that NRDP is a potential subgroup of LBDs resistant to standard treatment approaches, including the application of MLSs and generic FR programs. The identification of LBD subgroups with a poorer prognosis and subsequent evaluation of the effectiveness of specific treatment has been described as a high research priority.⁵⁴ Non-reducible discogenic pain was therefore included as one of the subgroups in the STOPS trials for which the effectiveness of specific FR was evaluated.

Classification of disc herniation with associated radiculopathy

The identification of DHR as a pathoanatomical subgroup of LBDs can be traced back to initial

descriptions of surgical discectomy and the associated reduction in leg symptoms.^{55–57} Radicular disorders such as DHR are still recognized as a distinct LBD subgroup in almost all clinical practice guidelines.⁵⁸ Identification of DHR is assisted by the use of advanced imaging techniques such as magnetic resonance imaging and computerized tomography however reliance on imaging alone can be misleading due to high rates of asymptomatic disc herniations^{59,60} and correlation with clinical features is therefore recommended.^{59,61} Clinical features thought to be indicative of DHR include radicular leg pain and/or parasthesia, reproduction of leg symptoms on provocative neurodynamic testing (e.g. straight leg raise or prone knee flexion testing) and radicular signs (segmental sensory, motor and reflex deficits comparable with the level of disc herniation).^{58,62} Experimental and clinical studies suggest that disc herniation can cause mechanical and/or chemical irritation of a nerve root resulting in these clinical features.^{21,22}

Support for the validity of DHR as a subgroup of LBDs can be found in clinical studies demonstrating increased severity of symptoms⁶³ and a poorer prognosis^{63,64} in people with DHR compared to other LBDs. In addition, treatments that target the pathoanatomical mechanisms underpinning DHR have been shown to be effective, including discectomy,⁶⁵ epidural steroid injections⁶⁶ and selective nerve root injections.⁶⁷

Despite this evidence, there remains some dispute over the key features of DHR.^{61,68} However, as mentioned above, the complete validation of any LBD subgroup is a difficult challenge due to inherent complexities as well as an absence of suitable gold/reference standards. Given the significant convergence of evidence^{12–14} in the research literature, the recognition of DHR as a subgroup responsive to invasive treatment that has stood the test of time for over 50 years, and poorer prognosis with conservative treatments, it seems reasonable to include DHR as a subgroup in the STOPS trials.

Classification criteria for non-reducible discogenic pain and disc herniation with associated radiculopathy

As part of the STOPS trial protocol¹ only participants who satisfied the NRDP or DHR eligibility criteria at baseline assessment were included in the trial. A specifically designed MICROSOFT EXCEL (2008) spreadsheet was developed to ensure reproducible determination of subgroup membership according to the clinical data entered at baseline assessment. A detailed description of the baseline assessment method can be found elsewhere.¹

To be classified into the DHR subgroup, participants had to have clinical evidence of radiculopathy and radiological evidence of disc herniation as recommended in recent guidelines.⁶¹ Radiculopathy was defined as satisfying both of the following criteria.

- 1. Pain, paraesthesia, or numbness below the knee (for L3/4, L4/5, or L5/S1 herniations) or in the anterior thigh (for L1/2, L2/3 or L3/4 herniations). Bilateral symptoms were allowable provided they were worse on one side.
- AND
- 2. At least one of the following tests positive:
 - (i) positive provocative neurodynamic testing including straight leg raise (for L3/4, L4/5, L5/S1 herniations) or prone knee flexion test (for L1/2, L2/3 or L3/4 herniations) defined by at least one of the participant's usual leg symptoms being reproduced in the affected leg at any angle of elevation of either leg (i.e. standard or crossed straight leg raise);
 - (ii) a deficit on reflex testing including ankle jerk (for L4/L5 or L5/S1 herniations) or knee jerk (for L2/3 or L3/4 herniations) defined as being absent or reduced on the affected side compared to the non-affected side;
 - (iii) a deficit on sensory testing at the anterior thigh for L1 and L2 nerve roots, medial aspect of the knee for L3, medial aspect of foot for L4, dorsum of foot for L5 and lateral aspect of the foot for S1. A deficit was defined as reduced sensation on the affected side compared to the non-affected side involving the segment of the exiting nerve root corresponding to the level of disc herniation on imaging or one of the two next descending nerve roots. For example, a positive sensory test for an L4/5 herniation would need a deficit in at least one of the L4, L5 or S1 nerve roots;
 - (iv) a deficit on motor testing the strength of the hip flexors for L1 and L2 nerve roots, knee extensor strength for L3, ankle dorsiflexor strength for L4, extensor hallucis longus strength for L5 and ankle evertor or calf strength for S1. A deficit was defined using the same method of nerve root determination as for sensory testing.

Radiological evidence of a lumbar disc herniation was based on the radiologist reports of CT or MRI scans. The reported results had to satisfy the following criteria.

- 1. A description using specific language of either a lumbar disc herniation, protrusion, extrusion, sequestration or prolapse in at least one of the lumbo-sacral segments. The use of the term disc bulge alone was insufficient.
- 2. A description of a posterior (or central), posterolateral (or para-central), or lateral disc herniation. For postero-lateral or lateral herniations the direction of the laterality had to correlate with the side of primary leg symptoms. In these cases nerve root contact was <u>not</u> required. For posterior herniations, there had to be at least nerve root contact, of any degree, on the side of primary leg symptoms, or bilateral nerve root contact. Contact with the theca or thecal sac alone was insufficient.
- 3. Participants with any non-disc related causes of radiculopathy including spondylolisthesis, anterolisthesis, retrolisthesis, tumours, osteomyelitis, Paget's disease and canal/foraminal stenosis attributed to bony structures, ligamentous structures, or cysts were

not included. These criteria were generic exclusion criteria for each of the STOPS trials but are described here as an important radiological consideration for DHR.

To be classified as NRDP participants had to have four out of a possible nine clinical features of discogenic pain as determined in a recent expert physiotherapy panel.⁴³ The features included: (i) the presence of lumbar pain+leg symptoms, (ii) symptoms being aggravated by prolonged sitting, (iii) symptoms being aggravated by lifting, (iv) symptoms being aggravated by forward bending, (v) symptoms being aggravated by sit to stand, (vi) symptoms being aggravated by cough/sneeze, (vii) history of working in a job with heavy manual handling, (viii) the mechanism of injury being associated with flexion/ rotation and/or compression loading, and (ix) symptoms much worse the next morning or day after onset of injury. In addition, participants were required to not have any of the features from other pathoanatomical subgroups in the STOPS trials including:

- disc herniation with associated radiculopathy as described above;
- reducible discogenic pain defined as positively responding, by way of improved range of motion or symptoms, to a variety of MLSs including sustained or repeated extension ± lateral movements;¹⁴
- zygapophyseal joint pain defined as having at least 3 of the following 4 features: unilateral symptoms, a regular compression pattern,^{69,70} comparable palpatory findings or a positive response to palpatory assessment of the comparable palpatory finding.¹⁵

The subgroups of NRDP and DHR both have the lumbar intervertebral disc as the primary cause of pain and activity limitation. Disc herniation with associated radiculopathy can also be considered as a progression from NRDP where a symptomatic annular tear has extended further into, with or without penetration of, the outer annular wall resulting in focal herniation and nerve root irritation.³⁷ Both subgroups have inflammation and nerve related facptors as a likely driver of greater severity, comlexity and poorer responsiveness to standard methods of treatment, including generic FR. Because of these commonalities, both subgroups received an FR program focusing on treatment strategies specific to the pathoanatomical mechanisms of symptom generation.

Treatment protocol

The NRDP and DHR treatment protocols used in the STOPS trials were based on the principles of standard FR,²⁴ FR specific to DHR,^{71,72} specific motor control training,⁷³ postural self-management principles to assist in disc tissue healing,¹⁸ a conservative approach to exercise progression to avoid exacerbation of significant pathology³⁸ and education regarding recovery time-frames and the management of inflammation.⁴⁶ The operational detail in the protocols was derived from clinical training programs developed by the principle

author (JF) based on an extensive review of the literature and his 20 year experience as a Musculoskeletal Physiotherapist providing treatment to patients and clinical mentoring for physiotherapists. In addition, 13 physiotherapists who were working with and had been trained by the principle author participated in a one day forum to refine the NRDP and DHR clinical methods.

Any physiotherapy treatment should be applied in a personalized manner using clinical reasoning principles; however, such skills are difficult to define and teach, particularly in complex cases.74,75 Treatment integrity issues have also been identified in clinical trials evaluating the effectiveness of complex treatment programs.⁷⁶ The STOPS treatment protocol therefore had a focus on structured processes to ensure adequate and reproducible clinical decision making across all physiotherapists and trial participants. The algorithmic nature of the protocol allowed each participant to receive treatment personalized to their individual presentation. Sufficient scope was also provided in the protocol for the physiotherapist to modify the treatment based on their interpretation of the clinical presentation.

Session 1 treatment

Details regarding the trial physiotherapists as well as the training and mentoring program have been described in Part 1 of this series.¹⁵ Physiotherapists had a number of resources to assist in provision of the treatment protocol including a 240 page treatment manual, a comprehensive baseline assessment

Table	1	Clinical	notes	content	for	Session	1
	-	••		••••••			

report completed when determining eligibility for the trial, a series of professionally produced participant information sheets and a blank copy of the clinical notes specifically designed for recording progress and clinical decision making in each treatment session. The clinical notes were structured using specific written cues to ensure all essential components of the treatment protocol were adhered to whilst allowing the physiotherapist some flexibility to select treatment techniques and rates of progression based on individual participant presentation. A summary of the content of the clinical notes for Session 1 is outlined in Table 1.

Although based on principles of FR, the treatment protocol had key modifications based on the mechanisms underpinning NRDP and DHR. In Session 1, the assessment of inflammation was an important example of this principle. Inflammation is rarely considered in RCTs of conservative treatment⁷⁷ despite being a precaution for common, mechanically based interventions.^{78,79} Given the proposed importance of inflammation in NRDP and DHR, if the participant was assessed in Session 1 as having mild inflammatory symptoms, they were referred to their local pharmacy to discuss non-prescription NSAIDs. For moderate or severe inflammatory features, or if over the counter NSAIDs failed to have an effect, the participant was referred to their medical practitioner with a letter requesting consideration of prescription NSAIDs. In case with severe and non-responsive inflammation, oral corticosteroids were discussed

Treatment protocol component	Rationale			
Session 1 assessment				
Review information from baseline assessment Complete body chart and history Gather asterisks from subjective and physical examination Determine whether clinical evidence exists of inflammation (At least 2 of constant symptoms, getting out of bed at night due to the pain, early morning symptoms >60 minutes) In the absence of inflammation, assess for relevant MLSs and reassess asterisks.	To gather and interpret information relevant to treatment planning and for reassessment of the participant's response to treatment Given the potential importance of inflammation in NRDP and DHR assessment of clinical evidence of inflammation is necessary ⁷⁹ Given the variable nature of complex LBDs, it is reasonable to reassess response to MLSs, despite this having been conducted on baseline assessment			
Session 1 treatment				
Explanation and information sheets regarding NRDP/DHR treatment options, treatment timeframes and recovery expectations. Open questions to the participant regarding understanding the explanation and level of engagement with the proposed treatment plan	Engaging the participant with the treatment process is critical to effective specific treatment ^{74,195}			
Lumbar taping in a neutral spine position and provision of posture information sheet	Improved posture will minimize stress to the disc and facilitate tissue healing ^{82,196}			
Management of inflammation, if applicable, including	Relative rest from aggravating postures/activities,			
provision of a specific information sheet recommending postural management, pharmacy consultation regarding suitable NSAIDs* and walking program short of pain onset	NSAIDs* and subclinical activity in a neutral spine position may prevent an excessive and counter-productive inflammatory response ³⁸			
in the absence of inflammation, and if responsive to MLSs, provide appropriate specific treatment ¹⁴	Disc related problems can variably respond to MLSs. Should responsiveness be demonstrated, a trial application of specific treatment should occur ⁷⁹			
A general emphasis on self-management rather than passive treatment approaches	Self-management is important in conditions with slower recovery timeframes ⁸⁹⁻⁹¹			

Note: *NSAIDs=non-steroidal anti-inflammatory drugs.

with the medical practitioner as an option. Although there is only limited evidence on the effectiveness of oral steroids in DHR, trials have not been conducted on homogenous subgroups based on accepted clinical features of inflammation.^{77,80} As such, consideration was given to oral steroids in specific cases where clear clinical features of uncontrolled inflammation were noted.

Walking was used as a specific strategy to manage inflammation in Session 1. The trial physiotherapists prescribed a walking program 2–4 times per day at an intensity and duration that did not worsen symptoms. Repetitive and submaximal movement creates rhythmic and multi-planar movement of the lumbar spine that facilitates disc nutrition^{38,81,82} and potential removal of inflammatory by-products within and around the lumbar disc.³⁸ In subsequent sessions, and as the clinical features of inflammation reduced, the duration of walking was increased up to 30 minutes and frequency reduced to a level that was manageable with regards to the time commitments of the participant.

Based on the biomechanical and pathoanatomical mechanisms of the lumbar intervertebral disc, the maintenance of a neutral spine position in participants with NRDP and DHR was seen to be particularly important in the treatment protocol.^{82,83} The use of lumbar taping and education supported by a postural information sheet was therefore a mandatory component of the treatment protocol for both subgroups. The taping protocol has been described in Part 2 of this series¹⁴ and consisted of a physiotherapist applied hypoallergenic liquid skin barrier, hypoallergenic tape and finally rigid strapping tape. A decision making algorithm was provided to physiotherapists regarding when to wean the participant from the tape based on degree and rate of improvement in symptoms.¹⁴ The postural information sheet emphasized the maintenance of a neutral lumbar position particularly for activities involving sustained or repeated flexion (e.g. sitting, vacuuming), or manual handling (e.g. lifting, pushing, pulling). A commercially available lumbar roll or rolled-up towel was recommended for participants who spent a significant proportion of their day in prolonged sitting. Regular breaks from sitting were also emphasized in accordance with the literature suggesting that there is no one ideal posture and that regular alteration of sitting position is important for optimal health of the intervertebral disc.82

It has been proposed that postural information is a valuable treatment strategy for people with LBDs^{82,84} and is commonly used by physiotherapists to educate this population.⁸⁵ However, consistent with the classification approach of the STOPS trials, postural information was only routinely provided in

subgroups with disc related problems based on the adverse impact of flexed postures on disc mechanics. In Part 1 of this series, a subgroup of participants likely to respond to manual therapy and having clinical features indicative of zygapophyseal joint pain was described. Given these features included lumbar extension and ipsilateral lateral flexion as primary aggravating factors, postural information was not considered mandatory for that subgroup. In Part 4 of this series, a subgroup of LBDs with neurophysiological and/or psychosocial factors as primary barriers to recovery will be described. In such a population, information about posture may be counterproductive with potential for reinforcement of fear avoidance beliefs.^{86,87} The selective provision of postural information within the STOPS trials was another example of specific treatment being applied based on clearly defined classification principles.

As part of the STOPS trial protocol,¹ MLSs were tested at baseline assessment. Participants classified with DHR could potentially have been responsive to MLSs at baseline, however NDRP participants were by definition non-responsive. Mechanical loading strategies were however reassessed for both groups in the early stages of the treatment program, particularly if inflammation present at baseline was successfully treated during the program. The resolution of inflammation in disc related problems has the potential to increase the likelihood of responsiveness to MLSs.⁷⁹ If a positive response to MLSs was observed on reassessment in the early sessions, directional preference management according to the STOPS clinical protocol for RDP¹⁴ was trialled. The approach of continual assessment and reassessment demonstrates the emphasis within the STOPS trials protocols of clinical reasoning principles⁷⁴ as well as a participant specific and algorithmic approach to treatment provision.

A key principle of the treatment protocol for NRDP and DHR was the exclusion of any 'passive' treatment; that is, modalities or manual therapy delivered by the physiotherapist to alleviate pain.⁸⁸ There is a significant rationale, based on the pathoanatomy of intervertebral disc healing³⁸ as well as outcome studies,^{63,64} that people with NRDP or DHR have a slower rate of recovery. In such conditions, the use of passive treatment strategies has been hypothesized as falsely reinforcing patient expectations of rapid recovery, where in fact a longer period of self-managed rehabilitation is required.^{89–91} In addition, passive treatment in a condition with a slower recovery time has the potential risk of participants developing a treatment dependence on short term symptomatic relief.⁹² Such a dependency was not desirable within the context of a 10 week physiotherapy program as part of the STOPS trials.

Other aspects of Session 1 assessment and treatment as described in Table 1 have been described in Part 1 of this series.¹⁵

Sessions 2–10

The timing of Sessions 2–10 was determined by the trial physiotherapist; however, a general recommendation was made for treatment to be more frequent, approaching twice weekly, in the early stages of the program. This enabled adequate engagement of the participant with the concepts underpinning the treatment and ensured correct application of those principles during the required between session exercise and self-management program. A summary of the content of the clinical notes for Session 2 is outlined in Table 2.

When reviewing response to Session 1 treatment at the beginning of Session 2 the primary focus was not on symptomatic improvement, as rapid between session changes was not consistent with the healing process of the lumbar intervertebral disc.³⁸ Rather the trial physiotherapist was predominantly concerned with any increase in symptoms and associated causal factors such as social/recreational activity beyond the tolerance of the disc and/or psychosocial factors influencing the participant's perception of response to treatment. This evaluation if required, was conducted based on information gained from detailed subjective and physical examination including reassessment of key asterisks (measures used for the purpose of reassessing the participant's response to the treatment strategies).

For Session 2 treatment, key explanations/information sheets from Session 1 were reinforced and inflammation was assessed and managed as required. The trial physiotherapist also enquired regarding the impact of the LBD on work participation. Any work issues identified were discussed using a problem solving approach as part of the treatment program.

A goal setting information sheet was also discussed with the participant. Collaborative identification of goals was seen as an important part of the treatment protocol to maximize participant motivation and engagement with the treatment process, particularly in relation to exercise compliance. The trial physiotherapist encouraged the participant to identify four SMART (specific, measurable, attainable, realistic, and timely) goals^{93,94} based on meaningful activities. The trial physiotherapist then identified exercise based goals comparable to the participant identified activity goals. An explanation was given to the participant as to how achieving the exercise goals would increase the likelihood of achieving activity goals. From Session 2 onwards, exercise and activity

Treatment protocol component	Rationale		
Session 2-10 assessment			
Participant report on progress following Session 1. Detailed questioning regarding possible causes of worsening symptoms following Session 1 if applicable	To assist in determination of between session treatment effect. Detailed questioning conducted, to differentiate treatment effect from other factors (e.g. social/recreational activity)		
If the participant reported a perceived increase in pain, reassessment of Session 1 asterisks from the subjective and physical examination	To confirm whether the participant was genuinely worse compared with Session 1		
Follow-up on presence of inflammation	To review inflammatory status and the need for ongoing management		
Follow-up on compliance with between session exercise Questioning regarding any work issues as a result of the LBD	To continue the process of encouraging and evaluating participant engagement with the treatment program The STOPS trials excluded participants with a compensation claim but managing related work incapacity, if relevant, remained an important focus		
Session 2–10 treatment			
Briefly review explanations and information sheets regarding NRDP/DHR, treatment options, treatment timeframes and recovery expectations	Repeat explanation ensured engagement of the participant with the treatment program and enabled further questions to be asked		
Ongoing management of inflammation if applicable Collaborative setting of participant goals	As per Session 1 rationale To align the FR program content with goals that were meaningful for the participant, thereby increasing treatment effectiveness and participant motivation ¹⁹⁷		
Provision of additional information sheets on posture, pacing, relaxation, sleep management and pain management strategies, as required Manage participant's perceived increase in pain, if appropriate	Self-management strategies and specific advice are an important component of any treatment regime. ⁸⁷ The postural and pacing information sheets were mandatory Specific management of perceived increases in pain was an important process for settling exacerbations and improving self-management skills ⁸⁷		
In the absence of inflammation and responsiveness to MLSs, commencement/progression of specific motor control training.	Specific motor control in disc related problems is important to restore normal biomechanics, facilitate recovery and minimize recurrence ⁷³		

Table 2 Clinical notes content for Sessions 2–10

goals were reviewed with the participant every fortnight and positive reinforcement of progress provided as well as further explanation as required.

Additional participant information sheets were provided and explained as required including posture (described in Session 1), pacing, relaxation, sleep management and pain management strategies. The pacing information sheet educated participants regarding finding a balance between under and over activity relative to the severity of the injury. Introduction to the relaxation information sheet was recommended for participants who had more severe activity limitations or in cases with high anxiety subscale scores on the baseline Orebro Musculoskeletal Pain Questionnaire.95 The information sheet provided guidance on a range of relaxation methods with specific instruction on a breathing based technique.⁹⁶ People with LBDs commonly have associated poor sleep habits,97,98 and in relevant participants an information sheet was provided with explanation on practical strategies to improve sleep including sleep routines and body positioning. Information sheets on strategies for self-management of pain were also provided outlining the use of medications for pain management as well as other strategies including ice, heat and exercise.^{87,99}

It is common for people with LBDs to have fluctuating symptom levels particularly when recovery is slow.¹⁰⁰ Adequate self-management strategies were seen as critical in the NRDP/DHR treatment protocol to facilitate tissue healing in the poorly vascularized lumbar intervertebral disc.³⁸ Management of a perceived increase in pain was therefore an important component of the treatment program and was facilitated by the physiotherapists according to Fig. 1.

Managing a participant's perceived increase in pain within an FR program can be a challenging clinical reasoning exercise, given the multitude of potential causal factors. As part of the process outlined in Fig. 1, participants reporting a perceived increase in pain in Sessions 2-9 were carefully questioned by the trial physiotherapist regarding the potential causes. This included tracking of symptom intensity from the previous session until the increase in pain was first noted. In cases where no identifiable cause for the increased symptoms was ascertained, or where there were no changes from the previous session on reassessment of key asterisks, brief reassurance was provided and the treatment program continued. This explanation was consistent with current evidence-based advice, aimed to minimize development of fear avoidance beliefs^{86,101} and was predicated on the assessment that despite the participant's perception, no significant exacerbation of the NRDP or DHR had in fact occurred.

Conversely if the perceived increase in pain was assessed as being a significant exacerbation, the trial physiotherapist educated the participant on selfmanagement strategies using a specific 'increase in pain' information sheet. The information included reassurance that the increase in pain was temporary, encouragement to learn from the causal factor identified to prevent future exacerbations, detail on self-management strategies, encouragement to continue exercising as able and advice to return to preexacerbation levels of exercise as soon as possible.⁸⁷ A review of the above described posture and pacing information sheets was also provided. Despite this explanation being based on pathoanatomical principles, efforts to minimize the development of fear avoidance beliefs were still employed.

Related to participant perception of increases in pain were expectations regarding improvement in pain versus improvement in activity levels. Most participants had an adequate understanding of timeframes for pain to improve from the initial participant explanation. However, some required specific additional education regarding realistic expectations for improvement in pain given the slow recovery rates for NRDP and DHR. This involved an explanation that improvements in activity capabilities were likely to occur earlier than improvements in pain, and that such a change was an indication that progress towards recovery was being made.

Specific motor control training

All participants commenced specific motor control training once symptoms of inflammation were controlled, and relevant directional preference management had been adequately trialled and/or completed. Training of specific motor control is not a standard component of FR.²⁴ However, based on the potential benefit of a 'protective corset' on slowly recovering pathologies such as NRDP and DHR,^{38,71,73} we integrated a comprehensive and specific motor control training program with an FR approach.

The principles of specific motor control training have been well described;^{73,102} however, it was our clinical experience that a large amount of variability in clinical application existed between physiotherapists. In addition, specific motor control training is commonly reported as a difficult concept to efficiently teach in people with LBDs.^{103–105} On this basis, a series of clinical decision making algorithms, based on established protocols,^{73,102} was developed for use by the trial physiotherapists.

The goal of the specific motor control program was to retrain the core muscles of the lumbar spine, comprising transverses abdominis, lumbar multifidus and the pelvic floor, to maintain a tonic and automatic contraction^{106,107} at less than 30% of maximum voluntary contraction^{108,109} in daily activities. In most cases, this required initial training in



Figure 1 Clinical decision making with participant perception of an increase in pain. *ADL=activities of daily living.

non-weight bearing positions using a lower abdominal drawing in manoeuvre which has been shown to selectively activate transversus abdominis.¹¹⁰ Lumbar multifidus and the pelvic floor muscles, including pubococcygeus, have been shown to co-contract with transverses abdominis to provide a 'corset' for the lumbo-pelvic area^{111,112} and the treatment protocol aimed to achieve such a result associated with the lower abdominal drawing in manoeuvre. The treatment protocol initially focused on quality of movement and precise isolation of the relevant core muscles which has been shown to be important in restoring normal motor control in people with LBDs.^{108,109} Once adequate motor control of the core muscles was achieved in non-weight bearing positions, subsequent progression to functional activities was made.^{73,110} Importantly this progression

involved integration of the global muscles of the spine with the core muscles during specific functional exercises as well as during strength training of the trunk.¹⁰⁴ There is emerging evidence that functional retraining of normal lumbo-pelvic kinematics can improve motor control and clinical outcomes¹¹³ and these methods were also used to provide specific participant feedback during functional motor control exercises.

There is an overlap between assessment and treatment of motor control in the lumbar spine and these processes are summarized in Fig. 2 for non-weight bearing positions.

Adequate relaxation of the global muscles, such as rectus abdominis, external obliques and erector spinae, was required before attempting to contract the core stability muscles. Patients with maladaptive



Figure 2 Initial non-weight bearing specific motor control training strategies. *ASIS=anterior superior iliac spine; ^MVC=maximal voluntary contraction.

motor control strategies commonly demonstrate a dominance of the global muscles during functional tasks and at rest.^{107,114} In retraining a normal motor control pattern, adequate relaxation was seen as an important first step in inhibiting tone of the global muscles, thereby allowing a more isolated contraction of the core muscles.⁷³ In attaining a relaxed state, a neutral spine position was also encouraged as this appears to improve the activation of the deep abdominal core muscles.^{115,116}

An instruction to 'draw the lower abdomen in towards the spine' was used consistent with the developers of the abdominal drawing in method.⁷³ In addition to these standard instructions, we added the terms 'slowly' and 'gently' to emphasize the submaximal nature of the contraction.¹¹⁷ Tactile cues to the lower abdomen were used in conjunction with verbal cues to provide additional emphasis on a lower rather than more general drawing in of the abdomen.⁷³ Nonweight bearing positions were used initially in the position where best activation of transverses abdominis was observed;^{73,110} however, side lying was recommended as the optimal position for initial retraining¹¹⁰ due to ease of obtaining optimal relaxation of the global muscles and an improved length tension relationship in transverses abdominis compared to other positions (e.g. supine or crook lying).

In observing the participant response, the primary outcomes indicative of an adequate and submaximal transverses abdominis contraction were a 2–3 cm isolated inward movement of the abdomen approximately 3 cm above the pubic symphysis and a palpable slow and co-ordinated change in tone from a 'soft' feel in the relaxed state to a 'spongy' feel at submaximal contraction.¹¹⁷ These palpatory findings provided the physiotherapist with information additional to observation regarding the submaximal nature of the contraction.¹¹⁷ The trial physiotherapist concurrently palpated adjacent to the L3-L5 spinous processes to assess for co-contraction of lumbar multifidus, in the process identifying whether specific multifidus retraining was also required to achieve normal motor control. Well documented substitution strategies⁷³ were also monitored, and participant feedback provided, to ensure the observed drawing in of the lower abdomen and the palpatory findings were not the result of activity from the global muscles, in particular internal obliques.

For assessment of transverses abdominis the processes described in Fig. 2 were carried out with participant feedback from the trial physiotherapist for 2–3 repetitions. Participants then commenced specific motor control training, in the position of highest functional demand where correct contraction of the core muscles could be achieved. This allowed motor control training in a position specific to the participant's capabilities and where improvement could be attained with between session practice. In order to ensure that trial physiotherapists did not inadvertently select a starting position where correct motor control during between session practice would be unattainable, side lying was generally recommended for participants where specific motor control was poor or inconsistent.

In the event of the participant not being able to engage transverses abdominis in any position with the processes described in Fig. 2, a range of additional facilitation strategies were attempted by the physiotherapist (Fig. 3). These methods were also used for participants who had good control of transverses abdominis but poor control of lumbar multifidus and/or poor awareness of pelvic floor activation.

In participants where transverses abdominis was difficult to isolate, activation was facilitated by an initial focus on the pelvic floor and/or lumbar multifidus.^{73,118} In such cases the strategies listed in Fig. 3 were applied and co-contraction of transverses abdominis was concurrently monitored. In the event of co-contraction occurring, the participant was encouraged to focus on awareness of simultaneous activation of transversus abdominis as well as the pelvic floor and/or lumbar multifidus. The pelvic floor instructions aimed to illustrate the anatomy of the region and provide guidance in performing a submaximal isotonic contraction. The multifidus instructions aimed to provide guidance in performing a submaximal isometric contraction. If necessary



Figure 3 Activation and facilitation of transverses abdominis, lumbar multifidus and pelvic floor motor control.

multifidus was facilitated by provision of kinaesthetic feedback to the participant with an initial isotonic contraction, followed by an attempt to transfer this awareness to the required isometric contraction.

During all specific motor control training the participant was encouraged to develop a kinaesthetic awareness of the correct motor pattern. This was important in order for the participant to have some form of proprioceptive feedback regarding correct performance of the exercises when practicing between sessions.⁷³ Subsequently, during the treatment program, adequate kinaesthetic awareness of normal motor control was also required for transference into more functional and demanding exercises/activities. Due to the effect of even low force postural

perturbation in initiating maladaptive motor patterns^{119–121} participants were instructed not to self-palpate as means of providing feedback on exercise performance until more consistent specific motor control skills were demonstrated. It was our clinical experience that self-palpation also focused the participant on tone rather than the primary goal of an isolated drawing in of the lower abdomen.

Once an appropriate motor control strategy, exercise and starting position had been identified, a detailed information sheet was provided and explained to the participant. The content included information on the anatomy and normal function of the core muscles, the general principle of progressing specific motor control training from non-weight



Figure 4 Dosage and progression of specific motor control training.

bearing to functional activities, and instructions for between session practice. The trial physiotherapist documented an appropriate dosage regime on the information sheet according to the principles outlined in Fig. 4. Over the following sessions the trial physiotherapist, working with the participant, aimed to achieve a tonic contraction of the core muscles, during walking for two minutes before progressing to more advanced functional training.

Functional restoration including functional motor control training

Once adequate motor control had been demonstrated in walking, the participant commenced an FR program with an emphasis on functional motor control. In keeping with the principles of FR, a graded exercise program based on the functional requirements of the participant activity goals set in Session 2 was recommended. Each exercise was completed with tonic control of the core muscles in a correct motor control pattern. Key components of this program included:

- supervised functional exercise and specific motor control training at least weekly for at least 3 weeks in the clinic gym;
- a focus on the quality of functional movement including facilitation of correct posture and lumbo-pelvic kinematics;
- concurrent between session functional exercise and specific motor control training at least 5 times a week for 15–45 minutes;

- participant documentation of exercise compliance using an exercise diary;
- regular participant/physiotherapist review of activity and exercise based goals with positive reinforcement of progress made;
- a planned progression towards independence after completion of the 10 treatment sessions with the provision of a medium and long term exercise plan.

Within the STOPS trials, the described components of FR were provided for all participants. However, in the subgroups of lumbar zygapophyseal joint pain and RDP,^{14,15} FR was generally provided in the second half of the treatment program to suit the relative importance and time priorities necessitated by other specific treatment components. Table 3 describes the phases of treatment with the timeframes and session frequency for FR in the NRDP and DHR subgroups of the STOPS trials.

A series of information sheets were provided to participants outlining exercises that could be selected by the trial physiotherapist for inclusion in the FR program. Included in the information sheets was a photo and description of the exercise as well as space for the trial physiotherapist to document appropriate dosage and goals. Exercises commonly used by the trial physiotherapists included: walking, step-ups (with a variable height step), alternate dumbbell bicep curls, alternate dumbbell forward raises, dumbbell lateral raises, ¹/₂ lunges, ¹/₂ squats, isotonic trunk extension to neutral over a fitball, abdominal **Lifting light weights** will build up your core stability in preparation for heavier lifting, gardening and housework. Hold a dumbbell in each hand with your palm facing forward and arms straight. Without moving your upper arm bend your left elbow and curl the dumbbell up toward the shoulder. Lower the left and curl the right arm. Perform the movement with good control of the trunk and arms.





Starting dosage: times per day lots of repetitions Goal in days/weeks times per day

repetitions

lots of

Figure 5 Example information sheet detail for bicep curls.

crunches in supine and lifting practice using a weighted box. An example of the information sheet detail for bicep curls is provided in Fig. 5.

The trial physiotherapists were provided with a range of typical starting dosages for basic functional exercises with estimations of goal dosages for the end of the treatment program (Fig. 6). Initial dosages were selected based on the participant presentation incorporating principles of greater severity of pain/activity limitation necessitating lower dosage, higher level of function required for normal daily activities necessitating higher end program dosages (i.e. higher rate of progression) and lower levels of core motor control necessitating a lower initial dosage.

All exercises were progressed by the addition of external resistance using free weights. Progression of resistance was conservatively managed by the trial physiotherapists to minimize the risk of participant exacerbation. Generally progression was made by increasing repetitions of the exercise from $3 \times 3-8$ up to 3×15 , followed by a progression to a heavier free weight with a reduction in repetitions to $3 \times 3-8$ (depending on participant response), followed by a progression of again increasing repetitions to 3×15 . The participant was taught by the physiotherapist how to progress the resistance program without exacerbation using this dosage cycle.

Given the nature of the participants' pathology every effort was made to be cautious with progression of exercises until the response of the participant was established and adequate education in self-management principles attained. Participant perceived increases in pain were dealt with by the methods described in Fig. 1, and used to further assist in the effective learning of self-management principles.

Table 3 Functional restoration timeframes and session frequencies for NRDP and DHR

Phase 1: Preparation for FR

- Weeks 1-2 (2 sessions per week)
- Participant explanations
- Management of inflammation and directional preference (if applicable)
- Commencement of non-weight bearing specific motor control training unless adequate control demonstrated in functional positions/activity

Phase 2: FR establishment

- Weeks 2–3 (1–2 sessions per week)
- · Commencement of supervised functional exercises
- · Ongoing participant explanation of Phase 1 information sheets
- Additional education regarding specific issues as required

Phase 3: FR progression

- Weeks 3–6 (1 session per week)
- · Greater focus on increasing exercise based on functional activity goals
- · Additional education regarding specific issues as required

Phase 4: Transfer to independence

- Weeks 7 to 10 (1 session per fortnight)
- Review of progress and positive reinforcement of gains made
- Strategies for independent progression of exercises
- · Preparation for treatment completion and long term exercise/self-management



Figure 6 Functional restoration starting and estimated goal dosages. *T=treadmill, S=step-ups, **W=upper limb resistance exercises.

Transitioning to an independent functional restoration program

As part of the STOPS trials, the participant was required to become independent from physiotherapy in 10 sessions. Towards the end of the treatment program, the transition towards independence involved a reduction in treatment frequency and attempts to further develop participant skills in problem solving, selfmanagement and appropriate progression of exercises. Inherent in this process was the trial physiotherapist and participant understanding that the time required to achieve full restoration of function and maximum reduction in symptoms could be 3-6 months after completion of the treatment program. Participants were provided with a discharge information sheet that outlined expectations regarding recovery timeframes and specific detail on the exercise program required to achieve the participant's activity goals. Ongoing regular exercise was recommended at home or in a gym. An example of an exercise progression towards achieving a participant goal of running after completion of the treatment program is provided in Table 4.

If despite the FR program, the participant reported a lack of progress, or required repeated education on recovery expectations and/or perceived increases in pain, consideration was given to changing the treatment model. In such participants at five weeks into the treatment program a repeat Orebro Musculoskeletal Pain Questionnaire score was obtained and if scored at over 105/210,95 the treatment focus shifted away from a pathoanatomical emphasis. An FR program continued as the treatment method but with the emphasis on increased neural sensitivity^{122,123} as the primary basis for symptoms and activity limitations, rather than pathoanatomical mechanisms. The trial physiotherapists provided this education using cognitive-behavioural principles.96 There is evidence supporting this approach in subacute LBDs with suspected psychosocial factors.¹²⁴ Full details of this treatment program will be provided in Part 4 of this series of papers.

Discussion

A detailed clinical protocol has been presented for people with subacute, non-compensable LBDs

Table 4 Example home/gym progression for a participant wishing to return to running

Participant's primary goal was to run 40 km per week, but had not run for 4 months before commencing treatment. At the end of the 10 week STOPS FR program the participant was walking on treadmill at 6.0 kph for 10 minutes.

Recommended home/gym program following discharge from the STOPS FR program (3-5 times per week)

1. Increase endurance by increasing the treadmill dosage to 6.0 kph for 20 minutes

- 2. Consideration of additional exercise to evaluate/condition to higher stress on the disc before running. A step machine can evaluate capacity for the increased pelvic tilt and greater single leg stance ground reaction force involved in running. Mini-trampoline running can be similarly used
- Commence gentle treadmill running using arm support on the rails and an initial speed of 8–10 kph. Commence at a dosage of 10 minute walk, 1 minute run, and 9 minute walk. Gradually increase the proportion of running relative to walking in the 20 minute total duration of treadmill exercise
- 4. Once running for 20 minutes at slow speed is achieved, progress treadmill running to desired recreational speed
- 5. Progress to road running for 20 minutes at desired recreational speed
- 6. Progress duration of running until goal of 40 km per week achieved

classified into the subgroups of NRDP and DHR who participated in the STOPS trials. We believe the protocol is reproducible, generalizable and developed on the best available evidence in combination with the clinical principles of FR. In developing the protocol, key features of FR were adapted for participants with NRDP or DHR based on pathoanatomical mechanisms. The following discussion provides a rationale for these variations from a standard FR approach. Some may criticise the 'non-empirical' judgemental processes required in preparing such a protocol. However, the use of patient oriented clinical judgement combined with the latest research literature is consistent with an evidence-based approach¹⁶ and we believe reflects best practice.

Classification of LBDs is a complex exercise⁸ and there is a lack of consensus on the most appropriate methodological model for developing and validating classification systems.^{8,125} This conundrum is made more difficult by the absence of adequate gold/ reference standards for the diagnosis of LBDs.^{8,126} The classification approach used to develop the clinical features of NRDP and DHR have been described and justified in Parts 1 and 2 of this series.^{14,15} The clinical features of NRDP and DHR as described in this clinical protocol are supported by an extensive literature on biological plausibility,^{37,38} researchers,¹⁷ expert physiotherapy panels,⁴³ clinical experts^{18,71} and large multi-disciplinary surveys of practitioners.42 Based on principles of convergence of validity^{12–14} we believe our classification system for NRDP and DHR is reasonable for use in a clinical trial.

Recent systematic reviews suggest that FR may have long-term benefits although the effect sizes appear to be small.^{25,26} These findings contrast with an earlier high quality review on a smaller number of RCTs that showed larger effects.²⁷ The potential for sample and treatment heterogeneity within and between RCTs has been well described^{7–10} and preliminary evidence exists showing more consistent research findings when a classification approach is adopted.^{41,127,128} However, there are no RCTs that evaluate the effectiveness of physiotherapy FR applied to a specific population of people with LBDs defined by pathoanatomical mechanisms. The STOPS trials are an attempt to address this gap in the literature.

The value of LBD treatment protocols based on pathoanatomical mechanisms has been discussed in Parts 1 and 2 of this series of papers.^{14,15} Treatment with inadequate consideration of pathoanatomical mechanisms has a risk of being ineffective or harmful.^{8,38,129} Our clinical protocol attempted to adapt the principles of FR in order to specifically address the mechanisms of likely importance in NRDP and DHR. Underlying this approach was the premise that symptoms of participants

with NRDP or DHR would typically be more severe and/or complex as well as take longer to recover compared with other pathoanatomical LBD subgroups. Non-reducible discogenic pain by definition is less responsive to MLSs and is therefore less likely to recover as quickly. Both NRDP and DHR are presumed to involve painful annular tears into the innervated outer third of the intervertebral disc. Although some connective tissue healing does take place in the annulus, it occurs slowly due to poor vascularity.38,50,130 Disc herniation with associated radiculopathy involves the additional complication of mechanical ± chemical irritation of the relevant nerve root.²¹ Studies have shown that people with DHR, compared to other LBDs, typically have higher levels of pain and activity limitation at baseline as well as a poorer prognosis over time.^{63,64} Based on the clinical experience of our research group in combination with the literature on mechanisms underpinning discogenic problems, a generic FR approach that does not account for the specific needs of NRDP and DHR is not likely to be successful.

Inflammatory processes have been demonstrated in a wide range of studies on disc related LBDs.^{48,49} Evidence of inflammatory processes such as macrophage infiltration, and an increase in pro-inflammatory substances such as interleukins, tumour necrosis factor-alpha, and nerve growth factor have been found in discogenic pain^{57,131–136} and disc herniation.^{137–139} The presence of untreated inflammation is acknowledged as a significant barrier to physiotherapy treatment^{78,79} and may also impede specific motor control training. Despite this evidence, we are unaware of any studies on FR that have systematically attempted to identify and manage inflammation, particularly when targeting disc related LBDs. In the STOPS trials for NRDP and DHR, participants were assessed for clinical features of inflammation as determined by clinical experts, 78,79,140 a multi-disciplinary survey of practitioners 77 and preliminary evidence of concurrent validity.^{141,142} An escalating anti-inflammatory treatment regime was incorporated into the protocol depending on the presence and severity of the inflammatory symptoms. In keeping with the algorithmic approach of the STOPS trials, if the participant did not have the features of inflammation, or did not respond to specific management, the anti-inflammatory regime was deemed not appropriate for use.

A commonly described mechanism underpinning the treatment effect of FR is the reversal of the 'deconditioning syndrome' that is hypothesized as arising from catastrophizing and fear avoidance of painful activities.^{24,143} Based on operant conditioning principles for chronic pain,¹⁴⁴ graded activity has been recommended as a treatment for fear avoidance and deconditioning in individuals with LBDs.⁷⁶ Graded activity involves the identification of specific functional goals, development of exercises that relate to the goals, and planning of exercise progression in predetermined increments until the functional goals are achieved.¹⁴⁵ Importantly, increases in exercise dosage is time rather than pain contingent and patient reports of catastrophic fears regarding exercise are addressed by challenging the patient's presumed counter-productive beliefs.^{76,146,147}

The validity of the fear avoidance model and the deconditioning syndrome has been challenged, particularly when applied in a generic manner to non-specific populations.^{148,149} A more specific approach to addressing catastrophizing, fear avoidance and deconditioning with graded activity based on individual biomedical, psychosocial and neurophysiological characteristics has been recommended in the literature.^{76,147,150,151}

Given the severity, complexity and possible inflammatory nature of NRDP and DHR, the use of time contingent progressions in exercise for all participants, irrespective of pain response has the potential to cause deterioration in the participant's pain and activity capabilities. The STOPS FR program for NRDP and DHR therefore incorporated an algorithmic and participant specific approach to exercise progression. The trial physiotherapist aimed to improve deconditioning in participants where this was likely to have been a problem (i.e. those with very low daily activity levels). However, progression in the exercise program was contingent upon the session to session response in pain and activity as well as participant goals. In the event of a perceived increase in pain a detailed decision making algorithm was used to determine whether this increase was due to a significant pathoanatomical based exacerbation. Specific questioning was also employed assessing contributing factors to the increase in pain including the exercise program, other social/ recreational factors and catastrophizing/fear avoidance beliefs. On the basis of this assessment, the trial physiotherapist intervened to address the identified issues using a variety of methods. It is important to recognize that the population recruited for the STOPS trials in the NRDP and DHR subgroups were less likely to have psychosocial factors as the primary driver of their LBDs due to the classification methodologies used and the exclusion of participants with chronic or compensable LBDs. In this population, we would argue that our modified approach to FR and graded activity was appropriate. Our clinical experience and the literature on discogenic problems would also suggest that an inflexible graded activity approach would have exacerbated a significant proportion of the participants.

Specific motor control training as part of the treatment protocol was based on the principles developed by Richardson and colleagues.⁷³ Extensive research has been published on the importance of normal motor control in the lumbar spine including but not limited to:

- an anatomical and biomechanical suitability of the core muscles for providing stability to structures in the lumbar spine;^{152–161}
- feed forward mechanisms in people without an LBD resulting in 'pre-setting' of the core muscles in anticipation of postural perturbation;^{162–164}
- contraction of core muscles independent of direction of trunk forces and movements;^{165,166}
- maladaptive differences between people with and without LBDs in terms of altered feed forward mechanisms,^{106,119–121} reduced core muscle cross sectional size,^{167,168} increased global muscle activity in certain subgroups¹²⁸ and altered cortical representation of motor patterns.¹⁶⁹

This substantial literature has lead to the hypothesis that correcting maladaptive motor control is an important component in the rehabilitation of LBDs and prevention of recurrence.^{114,170} This hypothesis is strengthened based on clinical data showing a reversal of the maladaptive changes observed in people with LBDs,^{108–110,171–173} which is not achieved by less specific exercise such as abdominal bracing or general exercise.^{174,175} In addition, RCTs have shown significant improvement for pain and activity in participants receiving specific motor control training compared to usual care^{104,176–178} with larger effects demonstrable when homogenous subgroups are recruited.¹⁷⁹ Recent controversy around specific motor control training^{180,181} has been refuted based on an insufficient consideration of this expansive literature as a whole.¹⁷⁹

As part of a reproducible and generalizable clinical protocol, a number of key decisions were made regarding specific motor control training. One such aspect was the preferred starting position of side lying rather than standard positions of prone or supine/crook lying for initial training of the core muscles.⁷³ Side lying was recommended as the optimal starting position to trial physiotherapists on the basis of the position:

- providing maximal support to participants thereby optimizing relaxation of the global muscles;
- allowing the abdomen to relax and extend anteriorly in as a precursor to abdominal drawing in thereby improving the length tension relationship within transversus abdominis, providing greater resistance to the desired muscle action, increasing the potential distance of in drawing movement and as such resulting in improved proprioceptive feedback to the participant;¹⁸²
- allowing the trial physiotherapist to easily assess and provide feedback on co-contraction of lumbar multifidus and transversus abdominis.¹¹⁷

In addition, there is an absence of literature demonstrating superiority of other starting positions to side lying for facilitating specific motor control training. In the event of inadequate contraction of the core muscles in side lying other positions were attempted consistent with current research^{183–185} and original recommendations.⁷³

Within the context of a rehabilitation program for NRDP and DHR, adequate specific motor control in non-weight bearing positions was an essential prerequisite for commencement of FR. In general, participants did not progress to higher level FR until adequate motor control in less challenging positions was demonstrated. This is consistent with other high quality RCTs on the effectiveness of core stability in a specific LBD population,¹⁸⁶ samples of non-specific LBDs^{187–189} and clinical descriptions of the specific motor control method.⁷³

Based on preliminary evidence, some have suggested that the core muscles can be activated by relatively non-specific functional activity,¹⁹⁰ or by using apparatus such as fitballs or Pilates devices in the absence of specific motor control training.¹⁹¹ This hypothesis is yet to be adequately validated. In the population of NRDP or DHR, we deemed it more appropriate to initially focus on isolated control of the core muscles to maximize the likelihood of correct motor control during subsequent functional loading. Other clinical researchers have emphasized the importance of correct kinematics in the lumbo-pelvic region as part of a motor control program.^{19,113} The STOPS clinical protocol incorporated these methods in the early stages of the treatment program, particularly once participants had demonstrated satisfactory motor control in non-weight bearing positions.

The specific motor control approach to the rehabilitation of LBDs has been criticized for an insufficient focus on the global muscles' role in trunk stability and normal function.^{181,192} The STOPS treatment protocol firstly identified and corrected maladaptive motor control patterns and with subsequent exercise progression into high level functional activities, thereby appropriately integrated core and global muscle function. Global muscle strength is important in many functional activities, particularly the erector spinae in lifting,³⁷ and the treatment protocol therefore recommended that the physiotherapist specifically strengthen these muscle groups in conjunction with core muscle control.

A number of key educational strategies were provided to participants depending on their presentation. Much of the educational content described in the protocol around establishing realistic expectations, goal setting, managing perceived increases in pain and pacing could be regarded as a cognitive-behavioural approach. Our protocol demonstrates that a psychosocial model that challenges counter-productive beliefs can co-exist with treatment based on pathoanatomical mechanisms. In this way, the STOPS clinical protocol utilizes a true biopsychosocial approach,¹²⁹ incorporating both biomedical as well as psychosocial factors as determinants of clinical decision making.¹⁹³

The protocol was highly specific but also algorithmic, ensuring participants did not receive a 'one size fits all' approach. Such decision making processes, based on the response of the participant to particular treatment strategies, are consistent with the clinical practice of experienced practitioners⁷⁴ and are recommended as best practice.¹⁹⁴ The protocol aimed to provide clear structure for clinical decision making, however, on close inspection the trial physiotherapist also had many options to exercise their own judgement in the provision of treatment. We believe the protocol establishes a high standard of treatment whilst allowing personalization of the program to the participant's LBDs with sufficient flexibility allowing additional practitioner decision making.

The described clinical protocol adheres to the essential principles of FR, and with modifications made to increase specificity to disc related disorders, will allow the STOPS trials to be replicable by future studies. The clear description of the classification and treatment protocol will also enable physiotherapists in clinical practice to make an informed choice regarding modification of the described methods to best suit their own patient population. The results of the STOPS trials will therefore be more likely generalizable to physiotherapists in clinical practice.

Summary

A clinical protocol for the classification and specific treatment of LBDs subgroups with criteria indicative of NRDP and DHR has been presented. This protocol is being used in the STOPS trials evaluating the effectiveness of specific physiotherapy. Should the trials demonstrate significant and clinically meaningful effects, the protocol will be useful for physiotherapists and researchers wanting to replicate the classification and treatment approach in clinical and RCT settings.

References

- 1 Hahne AJ, Ford JJ, Surkitt LD, Richards MC, Chan AY, Thompson SL, *et al.* Specific treatment of problems of the spine (STOPS): design of a randomised controlled trial comparing specific physiotherapy versus advice for people with subacute low back disorders. BMC Musculoskeletal Disord 2011;**12**:104.
- 2 Dagenais S, Caro J, Haldeman S. A systematic review of low back pain cost of illness studies in the United States and internationally. Spine J 2008;8(1):8–20.
- 3 Walker B. The prevalence of low back pain: a systematic review of the literature from 1966 to 1998. J Spinal Disord 2000;**13**(3):205–17.

- 4 Loney P, Stratford P. The prevalence of low back pain in adults: a methodological review of the literature. Phys Ther 1999;**79**(4):384–96.
- 5 Ehrlich GE. Low back pain. Bull World Health Organ 2003;81(9):671-6.
- 6 van Tulder MW, Koes B, Malmivaara A. Outcome of noninvasive treatment modalities on back pain: an evidence-based review. Eur Spine J 2006;**15**(Suppl 1):S64–81.
- 7 van Middelkoop M, Rubinstein SM, Verhagen AP, Ostelo RW, Koes BW, van Tulder MW. Exercise therapy for chronic nonspecific low-back pain. Best Pract Res Clin Rheumatol 2010;24(2):193–204.
- 8 Ford J, Story I, O'Sullivan P, McMeeken J. Classification systems for low back pain: a review of the methodology for development and validation. Phys Ther Rev 2007;**12**:33–42.
- 9 Fritz J, Cleland J, Childs J. Subgrouping patients with low back pain: evolution of a classification approach to physical therapy. J Orthop Sports Phys Ther 2007;37(6):290–302.
- 10 Fersum K, Dankaerts W, O'Sullivan P, Maes J, Skouen J, Bjordal J, *et al.* Integration of subclassification strategies in randomised controlled clinical trials evaluating manual therapy treatment and exercise therapy for non-specific chronic low back pain: a systematic review. Br J Sports Med 2010;**44**:1054–62.
- 11 Riddle D. Classification and low back pain: a review of the literature and critical analysis of selected systems. Phys Ther 1998;78:708-37.
- 12 George S, Delitto A. Clinical examination variables discriminate among treatment-based classification groups: a study of construct validity in patients with acute low back pain. Phys Ther 2005;85(4):306–14.
- 13 Reitsma JB, Rutjes AWS, Khan KS, Coomarasamy A, Bossuyt PM. A review of solutions for diagnostic accuracy studies with an imperfect or missing reference standard. J Clin Epidemiol 2009;62(8):797–806.
- 14 Ford J, Surkitt L, Hahne A. A classification and treatment protocol for low back disorders part 2: directional preference management for reducible discogenic pain. Phys Ther Rev 2011;16:423–37.
- 15 Ford J, Thompson S, Hahne A. A classification and treatment protocol for low back disorders. Part 1: specific manual therapy. Phys Ther Rev 2011;16:172–81.
- 16 Sackett D, Straus S, Richardson W, Rosenberg W, Haynes R. Evidence-based medicine. London: Churchill Livingstone; 2000.
- 17 Petersen T, Laslett M, Thorsen H, Manniche C, Ekdahl C, Jacobsen S. Diagnostic classification of non-specific low back pain. A new system integrating patho-anatomic and clinical categories. Physiother Theory Pract 2003;19:213–37.
- 18 McKenzie R. The lumbar spine: Mechanical diagnosis and therapy. 1st ed. Waikanae: Spinal Publication; 1981.
- 19 O'Sullivan P. Diagnosis and classification of chronic low back pain disorders: maladaptive movement and motor control impairments as underlying mechanism. Man Ther 2005; 10(4):242–55.
- 20 Fardon D, Milette P. Nomenclature and classification of lumbar disc pathology: recommendations of the Combined Task Forces of the North American Spine Society, American Society of Spine Radiology, and American Society of Neuroradiology. Spine 2001;26(5):E93–E113.
- 21 Bogduk N. On the definitions and physiology of back pain, referred pain, and radicular pain. Pain 2009;147:17–9.
- 22 Rhee J, Schaufele M, Abdu W. Radiculopathy and the herniated lumbar disc. Controversies regarding pathophysiology and management. J Bone Joint Surg Am 2006;88(9):2070– 80.
- 23 Hahne A, Ford J, McMeeken J. Conservative management of lumbar disc herniation with associated radiculopathy: a systematic review. Spine 2010;35(11):E488–E504.
- 24 Mayer TG, Gatchel RJ, Kishino N, Keeley J, Capra P, Mayer H, et al. Objective assessment of spine function following industrial injury; a prospective study with comparison group and one-year follow up. Spine 1985;10(6):482–93.
- 25 Physical conditioning programs for improving work outcomes in workers with back pain [database on the Internet]. The Cochrane Collaboration. 2010.
- 26 Poiraudeau S, Rannou F, Revel M. Functional restoration programs for low back pain: a systematic review. Annales de Réadaptation et de Médecine Physique 2007;50(6):425–9.
- 27 Schonstein E, Kenny D, Keating J, Koes B, Herbert RD. Physical conditioning programs for workers with back and

neck pain: a Cochrane systematic review. Spine 2003; 28(19):E391-5.

- 28 Guzmán J, Esmail R, Karjalainen K, Malmivaara A, Irvin E, Bombardier C. Multidisciplinary rehabilitation for chronic low back pain: systematic review. BMJ 2001;322:1511–6.
- 29 Kankaanpaa M, Taimela S, Airaksinen O, Hanninen O. The efficacy of active rehabilitation in chronic low back pain: effect on pain intensity, self-experienced disability, and lumbar fatigability. Spine 1999;24(10):1034–42.
- 30 Pengel L, Refshauge K, Maher C, Nicholas M, Herbert R, McNair P. Physiotherapist-directed exercise, advice, or both for subacute low back pain: a randomized trial. Ann Intern Med 2007;146:787–96.
- 31 Lindström I, Öhlund C, Wallin L, Peterson L, Nachemson A. Mobility, strength, and fitness after a graded activity program for patients with subacute low back pain. Spine 1992;17:641–9.
- 32 Friedrich M, Gittler G, Halberstadt Y, Cermak T, Heiller I. Combined exercise and motivation program: effect on the compliance and level of disability of patients with chronic low back pain: a randomized controlled trial. Arch Phys Med Rehabil 1998;**79**(5):475–87.
- 33 Furlan A, Pennick V, Bombardier C, van Tulder M. Updated method guidelines for systematic reviews in the Cochrane Back Review Group. Spine 2009;34:1929–1941.
- 34 Maher C, Sherrington C, Herbert R, Moseley A, Elkins M. Reliability of the PEDRO scale for rating quality of randomized controlled trials. Phys Ther 2003;83(8):713–21.
- 35 Moher D, Hopewell S, Schulz KF, Montori V, Gøtzsche PC, Devereaux PJ, et al. CONSORT 2010 explanation and elaboration: updated guidelines for reporting parallel group randomised trial. BMJ 2010;340:c869.
- 36 de Morton N. The PEDro scale is a valid measure of the methodological quality of clinical trials: a demographic study. Aust J Physiother 2009;55:129–33.
- 37 Bogduk N. Clinical anatomy of the lumbar spine and sacrum. 4th ed. New York: Churchill Livingstone; 2005.
- 38 Adams MA, Stefanakis M, Dolan P. Healing of a painful intervertebral disc should not be confused with reversing disc degeneration: implications for physical therapies for discogenic back pain. Clin Biomech 2010;25(10):961–71.
- 39 Hancock M, Maher C, Latimer J, Spindler M, McAuley J, Laslett M, *et al.* Systematic review of tests to identify the disc, SIJ or facet joint as the source of low back pain. Eur Spine J 2007;**16**:1539–50.
- 40 Aina A, May S, Clare H. The centralization phenomenon of spinal symptoms: a systematic review. Man Ther 2004; 9(3):134-43.
- 41 Long A, Donelson R, Fung T. Does it matter which exercise? A randomised control trial of exercise for low back pain. Spine 2004;**29**(23):2593–602.
- 42 Kent P, Keating J. Classification in nonspecific low back pain: what methods do primary care clinicians currently use? Spine 2005;**30**(12):1433–40.
- 43 Chan A, Ford J, McMeeken J, Wilde V. The indicators of non-reducible discogenic pain: an international expert panel. Submitted for publication. 2011.
- 44 Crock H. Internal disc disruption: a challenge to disc prolapse fifty years on. Spine 1986;11:650–3.
- 45 Peng B, Hou S, Wu W, Zhang C, Yang Y. The pathogenesis and clinical significance of a high-intensity zone (HIZ) of lumbar intervertebral disc on MR imaging in the patient with discogenic low back pain. Eur Spine J 2006;15(5):583–7.
- 46 Saal J. The role of inflammation in lumbar pain. Spine 1995;20:1821–7.
- 47 Hurri H, Karppinen J. Discogenic pain. Pain 2004;112(3):225– 8.
- 48 Ross J. Non-mechanical inflammatory causes of back pain: current concepts. Skeletal Radiol 2006;**35**(7):485–7.
- 49 García-Cosamalón J, Del Valle M, Calavia M, García-Suárez O, López-Muñiz A, Otero J, *et al.* Intervertebral disc, sensory nerves and neurotrophins: who is who in discogenic pain? J Anat 2010;**217**(1):1–15.
- 50 Beattie P. Current understanding of lumbar intervertebral disc degeneration: a review with emphasis upon etiology, pathophysiology, and lumbar magnetic resonance imaging findings. J Orthop Sports Phys Ther 2008;38(6):329–40.
- 51 Donelson R. Rapidly reversible low back pain. Hanover: SelfCare First, LLC; 2007.
- 52 Carragee E, Haldeman S, Hurwitz E. The pyrite standard: the Midas touch in the diagnosis of axial pain syndromes. Spine J 2007;**7**:27–31.

- 53 Saal J. General principles of diagnostic testing as related to painful lumbar spine disorders: a critical appraisal of current diagnostic techniques. Spine 2002;**27**(22):2538–45.
- 54 Foster N, Dziedzic K, Windt D, Fritz J, Hay E. Research priorities for non-pharmacological therapies for common musculoskeletal problems: nationally and internationally agreed recommendations. BMC Musculoskeletal Disord 2009;10(1):3.
- 55 Dandy W. Loose cartilage from intervertebral disk simulating tumor of the spinal cord. Arch Surg 1929;**19**(4):4–8.
- 56 Mixter W, Barr J. Rupture of the intervertebral disc with involvement of the spinal canal. N Engl J Med 1934;211:210– 4.
- 57 Weiler C, Nerlich A, Bachmeier B, Boos N. Expression and distribution of tumor necrosis factor alpha in human lumbar intervertebral discs: a study in surgical specimen and autopsy controls. Spine 2005;30(1):44–53.
- 58 Koes B, van Tulder M, Lin C, Macedo L, McAuley J, Maher C. An updated overview of clinical guidelines for the management of non-specific low back pain in primary care. Eur Spine J 2010;19(12):2075–94.
- 59 Herzog R. The radiologic assessment for a lumbar disc herniation. Spine 1996;21(24S):19S-38S.
- 60 Jarvik J, Deyo R. Diagnostic evaluation of low back pain with emphasis on imaging. Ann Intern Med 2002;137(7):586–97.
- 61 Genevay S, Atlas S, Katz J. Variation in eligibility criteria from studies of radiculopathy due to a herniated disc and of neurogenic claudication due to lumbar spinal stenosis. A structured literature review. Spine 2010;**35**(7):803–11.
- 62 Koes B, van Tulder M, Peul W. Diagnosis and treatment of sciatica. BMJ 2007;334(7607):1313–7.
- 63 Atlas S, Deyo R, Patrick D, Convery K, Keller R, Singer D. The Quebec Task Force Classification for Spinal Disorders and the severity, treatment, and outcomes of sciatica and lumbar spinal stenosis. Spine 1996;**21**(24):2885–92.
- 64 Heymans M, Ford J, McMeeken J, Chan A, de Vet H, van Mechelen W. Exploring the contribution of patient-reported and clinician based variables for the prediction of low back work status. J Occup Rehabil 2007;**17**:383–97.
- 65 Gibson J, Waddell G. Surgical interventions for lumbar disc prolapse: updated Cochrane Review. Spine 2007;32(16):1735– 47.
- 66 Armon C, Argoff C, Samuels J, Backonja M. Assessment: use of epidural steroid injections to treat radicular lumbosacral pain: report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology. Neurology 2007;**68**:723–9.
- 67 DePalma M, Bhargava A, Slipman C. A critical appraisal of the evidence for selective nerve root injection in the treatment of lumbosacral radiculopathy. Arch Phys Med Rehabil 2005;86(7):1477–83.
- 68 van der Windt D, Simons E, Riphagen I, Ammendolia C, Verhagen A, Laslett M, *et al.* Physical examination for lumbar radiculopathy due to disc herniation in patients with low-back pain. Cochrane Database Syst Rev 2010;2:CD007431.
- 69 Edwards B. Manual of combined movements. 1st ed. Edinburgh: Churchill Livingstone; 1992.
- 70 McCarthy C. Spinal manipulative thrust technique using combined movement theory. Man Ther 2001;6(4):197–204.
- 71 Saal J, Saal J. Nonoperative treatment of herniated lumbar intervertebral disc with radiculopathy: an outcome study. Spine 1989;14(4):431–7.
- 72 Hahne A, Ford J. Functional restoration for a chronic lumbar disk extrusion with associated radiculopathy. Phys Ther 2006;86(12):1668–80.
- 73 Richardson C, Jull G, Hodges P. Therapeutic exercise for lumbopelvic stabilisation: a motor control approach for the treatment and prevention of low back pain. Edinburgh: Churchill Livingstone; 2004.
- 74 Jones M, Rivett D. Introduction to clinical reasoning. In: Jones M, Rivett D, editors. Principles of clinical reasoning in manual therapy. Oxford: Butterworth-Heinemann; 2004.
- 75 Petty N, Morley M. Clinical expertise: learning together through observed practice. Man Ther 2009;**14**(5):461–2.
- 76 Pincus T, Smeets R, Simmonds M, Sullivan M. The fear avoidance model disentangled: improving the clinical utility of the fear avoidance model. Clin J Pain 2010;26(9):739–46.
- 77 Walker B, Williamson O. Mechanical or inflammatory low back pain. What are the potential signs and symptoms? Man Ther 2009;14(3):314–20.

- 78 Maitland G, Hengeveld E, Banks K, English K, editors. Maitland's vertebral manipulation. 7th ed. Philadelphia, PA: Elsevier; 2005.
- 79 McKenzie R, May S. The lumbar spine: mechanical diagnosis and therapy. 2nd ed. Orthopaedic Physical Therapy Products. Waikanae: Spinal Publications (NZ) Ltd; 2003.
- 80 Levin J, Smuck M. Radiculopathy from herniation of the nucleus pulposus: 2. The role of corticosteroids. J Back Musculoskeletal Rehabil 2007;20:103–13.
- 81 Hendrick P, Te Wake A, Tikkisetty A, Wulff L, Yap C, Milosavljevic S. The effectiveness of walking as an intervention for low back pain: a systematic review. Eur Spine J 2010;19:1613–20.
- 82 Pynt J, Higgs J, Mackey M. Seeking the optimal posture of the seated lumbar spine. Physiother Theory Pract 2001;17:5–21.
- 83 Holm S, Nachemson A. Variations in the nutrition of the canine intervertebral disc induced by motion. Spine 1983;**8**:866–74.
- 84 Todd A, Bennett A, Christie C. Physical implications of prolonged sitting in a confined posture-a literature review. J Ergonom Soc South Africa 2007;**19**(2):7–21.
- 85 Poitras S, Blais R, Swaine B, Rossignol M. Management of work-related low back pain: a population-based survey of physical therapists. Phys Ther 2005;85:1168–81.
- 86 Main C, Foster N, Buchbinder R. How important are back pain beliefs and expectations for satisfactory recovery from back pain? Best Pract Res Clin Rheumatol 2010;24(2):205–17.
- 87 Blyth F, March L, Nicholas M, Cousins M. Self-management of chronic pain: a population-based study. Pain 2005; 113(3):285–92.
- 88 Jette A, Delitto A. Physical therapy treatment choices for musculoskeletal impairments. Phys Ther 1997;77(2):145–54.
- 89 Abenhaim L, Rossignol M, Valat J, Nordin M, Avouac B, Blotman F, *et al.* The role of activity in the therapeutic management of back pain. Spine 2000;**25**(4):1S–33S.
- 90 Moffett J, McLean S. The role of physiotherapy in the management of non-specific back pain and neck pain. Rheumatology 2006;45:371–8.
- 91 Liddle S, Gracey J, Baxter G. Advice for the management of low back pain: a systematic review of randomised controlled trials. Man Ther 2007;12:310–27.
- 92 Pincus T, Vogel S, Breen A, Foster N, Underwood M. Persistent back pain – why do physical therapy clinicians continue treatment? A mixed methods study of chiropractors, osteopaths and physiotherapists. Eur J Pain 2006;10(1):67–76.
- 93 Lazarus A. Reality check: is your behavior aligned with organizational goals? Phys Exec 2004;30:5–8.
- 94 Doran G. There's a S.M.A.R.T. way to write management's goals and objectives. Manage Rev 1981;70(11):35–6.
- 95 Hockings R, McAuley J, Maher C. A systematic review of the predictive ability of the Orebro Musculoskeletal Pain Questionnaire. Spine 2008;33(15):E494–E500.
- 96 Nicholas M, Molloy A, Tonkin L, Beeston L. Manage your pain; practical and positive ways of adapting to chronic pain. Sydney: Harper Collins Publishers; 2006.
- 97 Naughton F, Ashworth P, Skevington S. Does sleep quality predict pain-related disability in chronic pain patients? The mediating roles of depression and pain severity. Pain 2007;**127**:243–52.
- 98 Kelly G, Blake C, Power C, O'Keeffe D, Fullen B. The association between chronic low back pain and sleep a systematic review. Clin J Pain 2011;27(2):169–81.
- 99 von Korff M, Barlow W, Cherkin D, Deyo R. Effects of practice style in managing back pain. Ann Intern Med 1994;**121**(3):187–95.
- 100 Hayden J, Dunn K, van der Windt D, Shaw W. What is the prognosis of back pain? Best Pract Res Clin Rheumatol 2010;24:167–79.
- 101 Indahl A, Velund L, Reikeraas O. Good prognosis for low back pain when left untampered. Spine 1995;20(4):473–7.
- 102 O'Sullivan P. Lumbar segmental 'instability': clinical presentation and specific stabilizing exercise management. Man Ther 2000;**5**(1):2–12.
- 103 Henry S, Westervelt K. The use of real-time ultrasound feedback in teaching abdominal hollowing exercises to healthy subjects. J Orthop Sports Phys Ther 2005;35(6):338–45.
- 104 Standaert C, Weinstein S, Rumpeltes J. Evidence-informed management of chronic low back pain with lumbar stabilization exercises. Spine J 2008;8(1):114–20.
- 105 Hides J, Stanton W, Freke M, Wilson S, McMahon S, Richardson C. MRI study of the size, symmetry and function

of the trunk muscles among elite cricketers with and without low back pain. Br J Sports Med 2008;**42**(10):809–13.

- 106 Ferreira P, Ferreira M, Hodges P. Changes in recruitment of the abdominal muscles in people with low back pain: ultrasound measurement of muscle activity. Spine 2004; 29(22):2560–6.
- 107 Hides J, Belavy D, Cassar L, Williams M, Wilson S, Richardson C. Altered response of the anterolateral abdominal muscles to simulated weight-bearing in subjects with low back pain. Eur Spine J 2009;18(3):410–8.
- 108 Tsao H, Hodges P. Immediate changes in feedforward postural adjustments following voluntary motor training. Exp Brain Res 2007;181(4):537–46.
- 109 Tsao H, Druitt T, Schollum T, Hodges P. Motor training of the lumbar paraspinal muscles induces immediate changes in motor coordination in patients with recurrent low back pain. J Pain 2010;11(11):1120–8.
- 110 Hides J, Stanton W, Wilson S, Freke M, McMahon S, Sims K. Retraining motor control of abdominal muscles among elite cricketers with low back pain. Scand J Med Sci Sports 2010;20(6):834-42.
- 111 Sapsford R. Contraction of the pelvic floor muscles during abdominal maneuvers. Arch Phys Med Rehabil 2001; 82(8):1081–8.
- 112 Neumann P, Gill V. Pelvic floor and abdominal muscle interaction: EMG activity and intra- abdominal pressure. Int Urogynecol J Pelvic Floor Dysfunct 2002;13:125–32.
- 113 Dankaerts W, O'Sullivan P. The validity of O'Sullivan's classification system (CS) for a sub-group of NS-CLBP with motor control impairment (MCI): Overview of a series of studies and review of the literature. Man Ther 2011;16(1):9– 14.
- 114 Hodges P, Moseley G. Pain and motor control of the lumbopelvic region: effect and possible mechanisms. J Electromyogr Kinesiol 2003;13(4):361–70.
- 115 Reeve A, Dilley A. Effects of posture on the thickness of transversus abdominis in pain-free subjects. Man Ther 2009;14(6):679–84.
- 116 O'Sullivan P, Dankaerts W, Burnett A, Farrell G, Jefford E, Naylor C, *et al.* Effect of different upright sitting postures on spinal-pelvic curvature and trunk muscle activation in a painfree population. Spine 2006;**31**(19):E707–12.
- 117 Hides J, Scott Q, Jull G, Richardson C. A clinical palpation test to check the activation of the deep stabilizing muscles of the lumbar spine. Int Sport Med J 2000;1(4).
- 118 Critchley D. Instructing pelvic floor contraction facilitates transversus abdominis thickness increase during low-abdominal hollowing. Physiother Res Int 2002;7(2):65–75.
- 119 Hodges P, Richardson C. Inefficient muscular stabilisation of the lumbar spine associated with low back pain: a motor control evaluation of transversus abdominis. Spine 1996; 21:2640–50.
- 120 Hodges P, Richardson, CA. Delayed postural contraction of transversus abdominis in low back pain associated with movement of the lower limb. J Spinal Disord 1998;11(1):46– 56.
- 121 MacDonald D, Moseley G, Hodges P. Why do some patients keep hurting their back? Evidence of ongoing back muscle dysfunction during remission from recurrent back pain. Pain 2009;142(3):183–8.
- 122 Latremoliere A, Woolf C. Central sensitization: a generator of pain hypersensitivity by central neural plasticity. J Pain 2009;10(9):895–926.
- 123 Nijs J, Van Houdenhove B, Oostendorp RA. Recognition of central sensitization in patients with musculoskeletal pain: application of pain neurophysiology in manual therapy practice. Man Ther 2010;15(2):135–41.
- 124 Bunzli S, Gillham D, Esterman A. Physiotherapy-provided operant conditioning in the management of low back pain disability: a systematic review. Physiother Res Int 2011;16(1):4–19.
- 125 Kent P, Keating J, Buchbinder R. Searching for a conceptual framework for nonspecific low back pain. Man Ther 2009;14:387–96.
- 126 Carragee E, Hannibal M. Diagnostic evaluation of low back pain. Orthop Clin North Am 2004;**35**(1):7–16.
- 127 Dankaerts W, O'Sullivan P, Burnett A, Straker L. Differences in sitting postures are associated with nonspecific chronic low back pain disorders when patients are subclassified. Spine 2006;**31**(6):698–704.

- 128 Dankaerts W, O'Sullivan P, Burnett A, Straker L. Altered patterns of superficial trunk muscle activation during sitting in nonspecific chronic low back pain patients: importance of subclassification. Spine 2006;31(17):2017–23.
- 129 Waddell G. The back pain revolution. Edinburgh: Churchill Livingstone; 2002.
- 130 Adams M, Roughley P. What is intervertebral disc degeneration, and what causes it? Spine 2006;**31**(18):2151–61.
- 131 Purmessur D, Freemont A, Hoyland J. Expression and regulation of neurotrophins in the nondegenerate and degenerate human intervertebral disc. Arthritis Res Ther 2008;10(4):R99.
- 132 Freemont A, Watkins A, Le Maitre C, Baird P, Jeziorska M, Knight M, et al. Nerve growth factor expression and innervation of the painful intervertebral disc. J Pathol 2002;197(3):286–92.
- 133 Le Maitre C, Freemont A, Hoyland J. The role of interleukin-1 in the pathogenesis of human intervertebral disc degeneration. Arthritis Res Ther 2005;7(4):R732–45.
- 134 Lee S, Moon C, Sul D, Lee J, Bae M, Hong Y, *et al.* Comparison of growth factor and cytokine expression in patients with degenerated disc disease and herniated nucleus pulposus. Clin Biochem 2009;**42**(15):1504–11.
- 135 Peng B, Hao J, Hou S, Wu W, Jiang D, Fu X, et al. Possible pathogenesis of painful intervertebral disc degeneration. Spine 2006;**31**:560–566.
- 136 Le Maitre C, Hoyland J, Freemont A. Catabolic cytokine expression in degenerate and herniated human intervertebral discs: IL-1beta and TNFalpha expression profile. Arthritis Res Ther 2007;9(4):R77.
- 137 Ahn S, Park H, Byun W, Ahn M, Bae J, Jang S, et al. Comparison of the clinical outcomes and natural morphologic changes between sequestered and large central extruded disc herniation. Yonsei Med J 2002;43(3):283–90.
- 138 Habtemariam A, Gronglad M, Virri J, Seitsala S, Karaharju E. A comparative immunohistochemical study of inflammatory cells in acute-stage and chronic-stage disc herniations. Spine 1998;23:2159–66.
- 139 Virri J, Grönblad M, Seitsalo S, Habtemariam A, Kääpä E, Karaharju E. Comparison of the prevalence of inflammatory cells in subtypes of disc herniations and associations with straight leg raising. Spine 2001;26(21):2311–5.
- 140 Grassi W, De Angelis R, Lamanna G, Cervini C. The clinical features of rheumatoid arthritis. Eur J Radiol 1998;27:S18– S24.
- 141 Rudwaleit M, Metter A, Listing J, Sieper J, Braun J. Inflammatory back pain in ankylosing spondylitis: a reassessment of the clinical history for application as classification and diagnostic criteria. Arthritis Rheum 2006;54(2):569–78.
- 142 Földes K, Balint P, Gaal M, Buchanan W, Balint G. Nocturnal pain correlates with effusions in diseased hips. J Rheumatol 1992;19(11):1756–8.
- 143 Vlaeyen J, Linton S. Fear-avoidance and its consequences in chronic musculoskeletal pain: a state of the art. Pain 2000;85:317–32.
- 144 Fordyce W, Fowler R, Lehmann J, De Lateur B, Sand P, Trieschmann R. Operant conditioning in the treatment of chronic pain. Arch Phys Med Rehabil 1973;54(9):399–408.
- 145 George S, Bialosky J, Fritz J. Physical therapist management of a patient with acute low back pain and elevated fearavoidance beliefs. Phys Ther 2004;84:538–49.
- 146 George S, Zeppieri G. Physical therapy utilization of graded exposure for patients with low back pain. J Orthop Sports Phys Ther 2009;**39**(7):496–505.
- 147 Staal J, Hlobil H, Koke A, Twisk J, Smid T, van Mechelen W. Graded activity for workers with low back pain: who benefits most and how does it work? Arthritis Rheum 2008;59(5):642– 9.
- 148 Verbunt J, Smeets R, Wittink H. Cause or effect? Deconditioning and chronic low back pain. Pain 2010; 149:428–30.
- 149 Hasenbring M, Verbunt J. Fear-avoidance and endurancerelated responses to pain: new models of behavior and their consequences for clinical practice. Clin J Pain 2010;26(9):747– 53.
- 150 Vlaeyen J, Morley S. Cognitive-behavioral treatments for chronic pain what works for whom? Clin J Pain 2005;21:1–8.
- 151 van der Windt D, Hay E, Jellema P, Main C. Psychosocial interventions for low back pain in primary care: lessons learned from recent trials. Spine 2008;**33**:81–9.

- 152 Barker P, Briggs, CA, Bogeski, G. Tensile transmission across the lumbar fasciae in embalmed cadavers: effects of tension to various muscular attachments. Spine 2004;**29**:129–38.
- 153 Barker P, Guggenheimer KT, Grkovic I, Briggs CA, Jones DC, Thomas CD, et al. Effects of tensioning the lumbar fascia on segmental stiffness during flexion and extension. Spine 2006;**31**(4):387–405.
- 154 Barker P, Briggs C. Attachments of the posterior layer of lumbar fascia. Spine 1999;24(17):1757–64.
- 155 Panjabi M. The stabilizing system of the spine: Part I: function, dysfucntion, adaptation and enhancement. J Spinal Disord 1992;5:383–9.
- 156 Wilke H, Wolf S, Claes L, Arand M, Wiesend A. Stability increase on the lumbar spine with different muscle groups. Spine 1995;20(2):192–8.
- 157 MacIntosh J, Bogduk N. The biomechanics of the lumbar multifidus. Clin Biomech 1986;1:205–13.
- 158 Kaigle A, Holm S, Mansson T. Experimental instability in the lumbar spine. Spine 1995;20(4):421–30.
- 159 Hodges P, Eriksson M, Shirley D, Gandevia S. Intraabdominal pressure increases stiffness of the lumbar spine. J Biomech 2005;38(9):1873–80.
- 160 Hodges P, Cresswell, AG, Daggfeldt, K, Thorstensson, A. In vivo measurements of the effect of intra-abdominal pressure on the human spine. J Biomech 2001;34:347–353.
- 161 Hodges P, Kaigle Holm A, Holm S, Ekström L, Cresswell A, Hansson T, *et al.* Intervertebral stiffness of the spine is increased by evoked contraction of transversus abdominis and the diaphragm: in vivo porcine studies. Spine 2003; 28(23):2594–601.
- 162 Cresswell A, Oddsson, L, Thorstensson, A. The influence of sudden perturbations on trunk muscle activity and intraabdominal pressure while standing. Exp Brain Res 1994;98:336–41.
- 163 Hodges P, Richardson C. Feedforward contraction of transversus abdominis is not influenced by the direction of arm movement. Exp Brain Res 1997;114:362–70.
- 164 Hodges P, Richardson C. Contraction of the abdominal muscles associated with movement of the lower limb. Phys Ther 1997;77(2):132–44.
- 165 Cresswell A, Grundstrom H, Thorstensson A. Observations on intra-abdominal pressure and patterns of abdominal intramuscular activity in man. Acta Phys Scand 1992;144:409–18.
 166 Hodges P, Cresswell A, Thorstensson A. Preparatory trunk
- 166 Hodges P, Cresswell A, Thorstensson A. Preparatory trunk motion accompanies rapid upper limb movement. Exp Brain Res 1999;124:69–79.
- 167 Hides J, Lambrecht G, Richardson C, Stanton W, Armbrecht G, Pruett C, et al. The effects of rehabilitation on the muscles of the trunk following prolonged bed rest. Eur Spine J 2011;20(5):808– 18.
- 168 Dickx N, Cagnie B, Parlevliet T, Lavens A, Danneels L. The effect of unilateral muscle pain on recruitment of the lumbar multifidus during automatic contraction. An experimental pain study. Man Ther 2010;15(4):364–9.
- 169 Tsao H, Galea M, Hodges P. Reorganization of the motor cortex is associated with postural control deficits in recurrent low back pain. Brain 2008;**131**(8):2161–71.
- 170 Richardson C, Hides J, Wilson S, Stanton W, Snijders C. Lumbo-pelvic joint protection against antigravity forces: motor control and segmental stiffness assessed with magnetic resonance imaging. J Gravit Physiol 2004;11(2):P119–22.
- 171 Tsao H, Hodges P. Persistence of improvements in postural strategies following motor control training in people with recurrent low back pain. J Electromyogr Kinesiol 2008; 18(4):559–67.
- 172 Tsao H, Galea M, Hodges P. Driving plasticity in the motor cortex in recurrent low back pain. Eur J Pain 2010;14(8):832– 9.
- 173 Vasseljen O, Fladmark A. Abdominal muscle contraction thickness and function after specific and general exercises: a randomized controlled trial in chronic low back pain patients. Man Ther 2010;15(5):482–9.
- 174 Ferreira P, Ferreira M, Maher C, Refshauge K, Herbert R, Hodges P. Changes in recruitment of transversus abdominis correlate with disability in people with chronic low back pain. Br J Sports Med 2009;44(16):1166–72.
- 175 Hall L, Tsao H, MacDonald D, Coppieters M, Hodges P. Immediate effects of co-contraction training on motor control

of the trunk muscles in people with recurrent low back pain. J Electromyogr Kinesiol 2009;**19**(5):763–73.

- 176 Ferreira P, Ferreira M, Maher C, Herbert R, Refshauge K. Specific stabilisation exercise for spinal and pelvic pain: a systematic review. Aust J Physiother 2006;52(2):79–88.
- 177 Rackwitz B, de Bie R, Limm H, von Garnier K, Ewert T, Stucki G. Segmental stabilizing exercises and low back pain. What is the evidence? A systematic review of randomized controlled trials. Clin Rehabil 2006;20(7):553–67.
- 178 Macedo L, Maher C, Latimer J, McAuley J. Motor control exercise for persistent, nonspecific low back pain: a systematic review. Phys Ther 2009;89(1):9–25.
- 179 Hodges P. Transversus abdominis: a different view of the elephant. Br J Sports Med 2008;42(12):941-4.
- 180 Allison G, Morris S. Transversus abdominis and core stability: has the pendulum swung? Br J Sports Med 2008;42(11):630–1.
- 181 McGill SM. Low back disorders: evidence-based prevention and rehabilitation. 2nd ed. Illinois: Human Kinetics Publishers; 2008.
- 182 Kelly M, Tan B, Thompson J, Carroll S, Follington M, Arndt A, *et al.* Healthy adults can more easily elevate the pelvic floor in standing than in crook-lying: an experimental study. Aust J Physiother 2007;53:187–91.
- 183 Mew R. Comparison of changes in abdominal muscle thickness between standing and crook lying during active abdominal hollowing using ultrasound imaging. Man Ther 2009;14(6):690–5.
- 184 Chanthapetch P, Kanlayanaphotporn R, Gaogasigam C, Chiradejnant A. Abdominal muscle activity during abdominal hollowing in four starting positions. Man Ther 2009; 14(6):642–6.
- 185 Urquhart D, Hodges P, Allen T, Story I. Abdominal muscle recruitment during a range of voluntary exercises. Man Ther 2005;10:144–53.
- 186 O'Sullivan P, Twomey L, Allison G. Evaluation of specific stabilizing exercise in the treatment of chronic low back pain with radiologic diagnosis of spondylolysis or spondylolisthesis. Spine 1997;22(24):2959–67.
- 187 Cairns MC, Foster NE, Wright C. Randomized controlled trial of specific spinal stabilization exercises and conventional physiotherapy for recurrent low back pain. Spine 2006; 31(19):E670–81.
- 188 Costa L, Maher CG, Latimer J, Hodges PW, Herbert RD, Refshauge KM, et al. Motor control exercise for chronic low back pain: a randomized placebo-controlled trial. Phys Ther 2009;89(12):1275.
- 189 Ferreira M, Ferreira P, Latimer J, Herbert R, Hodges P, Jennings M, et al. Comparison of general exercise, motor control exercise and spinal manipulative therapy for chronic low back pain: a randomized trial. Pain 2007;(1–2):31–7.
- 190 McGalliard M, Dedrick G, Brismee J, Cook C, Apte G, Sizer P. Changes in transversus abdominis thickness with use of the abdominal drawing-in maneuver during a functional task. Phys Med Rehabil 2010;**2**:187–94.
- 191 Endleman I, Critchley D. Transversus abdominis and obliquus internus activity during pilates exercises: measurement with ultrasound scanning. Arch Phys Med Rehabil 2008; 89(11):2205–12.
- 192 van Dieen J, Cholewicki J, Radebold A. Trunk muscle recruitment patterns in patients with low back pain enhance the stability of the lumbar spine. Spine 2003;**28**:834–41.
- 193 Gatchel R, Turk D. Criticisms of the biopsychosocial model in spine care creating and then attacking a straw person. Spine 2008;33(25):2831–6.
- 194 Dagenais S, Tricco AC, Haldeman S. Synthesis of recommendations for the assessment and management of low back pain from recent clinical practice guidelines. Spine J 2010; 10(6):514–29.
- 195 Slade S, Molloy E, Keating J. 'Listen to me, tell me': a qualitative study of partnership in care for people with nonspecific chronic low back pain. Clin Rehabil 2009;23:270–80.
- 196 Harrison D, Harrison S, Croft A, Harrison D, Troyanovich S. Sitting biomechanics part I: review of the literature. J Manipulative Phys Ther 1999;22(9):594–609.
- 197 Sowden M, Hatch A, Gray S, Coombs J. Can four key psychosocial risk factors for chronic pain and disability (Yellow Flags) be modified by a pain management programme? A pilot study. Physiotherapy 2006;92:43–9.