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Preliminary investigation of the mechanisms underlying the effects of manipulation: exploration of a multi-variate model including spinal stiffness, multifidus recruitment, and clinical findings

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Abstract

Study Design—Prospective case series.

Objective—Examine spinal stiffness in subjects with low back pain (LBP) receiving spinal manipulative therapy (SMT), evaluate associations between stiffness characteristics and clinical outcome, and explore a multi-variate model of SMT mechanisms as related to effects on stiffness, lumbar multifidus (LM) recruitment and status on a clinical prediction rule (CPR) for SMT outcomes.

Summary of Background Data—Mechanisms underlying the clinical effects of SMT are poorly understood. Many explanations have been proposed, but few studies have related potential mechanisms to clinical outcomes or considered multiple mechanisms concurrently.

Methods—Subjects with LBP were treated with 2 SMT sessions over 1 week. CPR status was assessed at baseline. Clinical outcome was based on the Oswestry disability index (ODI). Mechanized indentation measures of spinal stiffness and ultrasonic measures of LM recruitment were taken before and after each SMT, and after 1 week. Global and terminal stiffness were

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calculated. Multivariate regression was used to evaluate the relationship between stiffness variables and percentage ODI improvement. Zero-order correlations among stiffness variables, LM recruitment changes, CPR status, and clinical outcome were examined. Path analysis was used to evaluate a multi-variate model of SMT effects.

Results—Forty-eight subjects (54% female) had complete stiffness data. Significant immediate decreases in global and terminal stiffness occurred post-SMT regardless of outcome. ODI improvement was related to greater immediate decrease in global stiffness (p=0.025), and less initial terminal stiffness (p=0.01). Zero-order correlations and path analysis supported a multi-variate model suggesting clinical outcome of SMT is mediated by improvements in LM recruitment and immediate decrease in global stiffness. Initial terminal stiffness and CPR status may relate to outcome though their relationship with LM recruitment.

Conclusions—The underlying mechanisms explaining the benefits of SMT appear to be multi-factorial. Both spinal stiffness characteristics and LM recruitment changes appear to play a role.

Introduction

High velocity, low-amplitude spinal manipulative therapy (SMT) is an intervention for low back pain (LBP) supported by several systematic reviews and practice guidelines.[1-3] Clinical characteristics identifying patients with LBP likely to benefit from SMT have been described in a clinical prediction rule (CPR),[4-6] however underlying mechanisms mediating outcomes of SMT are unknown. Research has identified a variety of physiologic effects of SMT,[7-13] but previous studies have focused on single effects in isolation, often with asymptomatic subjects. Studies involving patients with LBP have rarely related SMT effects to outcomes. Thus it remains difficult to determine how SMT's varied effects relate to each other, and which may explain clinical outcome.

Benefits of SMT are often attributed to its impact on spinal stiffness,[14-19] yet SMT's effect stiffness, and associations between these effects and outcomes are unclear. An SMT force has been shown to stimulate peripheral afferents, altering central nervous system (CNS) input, and enhancing motoneuron excitability.[8,20,21] These findings suggest the effectiveness of SMT could relate to a mechanical impact on spinal stiffness and subsequent neurophysiologic consequences facilitating muscle activity.[22] The lumbar multifidus (LM) may be a specific muscle benefitting from post-SMT facilitation. The LM has an important role in spinal control.[23,24] Both animal and human studies support reflex LM inhibition as a consequence of LBP,[25-28] which may increase risk of persistent or recurrent symptoms.[23,29] Increased LM recruitment after SMT has been reported,[30-32] but the relationship of LM facilitation to concurrent stiffness measures and a patient's CPR status in explaining clinical outcomes has not been examined.

Given the above, purposes of this study were; 1) examine spinal stiffness in subjects with LBP receiving SMT and evaluate associations between stiffness characteristics and clinical outcome, and 2) explore the validity of a model linking SMT's effects to CPR status, spinal stiffness and LM muscle recruitment.

This study represents an important preliminary step in advancing understanding of the underlying mechanisms of SMT's effects. Importantly, it is the first to examine longitudinal effects of SMT on stiffness, measured with validated techniques, and relate these effects to outcomes in subjects with LBP. Additionally, it is the first study to concurrently examine SMT's effects on 3 key variables; stiffness, LM recruitment, and CPR status, and relate these effects to each other, and clinical outcome.

Materials and Methods

Subjects

Subjects were recruited from physical therapy clinics and community-based advertisements. Inclusion criteria were LBP with or without leg symptoms, age 19-60 years, and Oswestry disability index (ODI) ≥ 20%. Exclusion criteria were signs of nerve root compression (e.g., positive straight leg raise, etc.), inability to lie prone and supine for ≥ 20 minutes, SMT within the past 4 weeks, diagnosis of osteoporosis, prior lumbosacral surgery, or any findings suggestive of non-musculoskeletal LBP (infection, cancer, etc.). We evaluated the 5 CPR clinical characteristics,[5] and recruited subjects likely to either receive pronounced benefit, or little to no benefit based on CPR status (table 1).[4] Subjects with 4 or 5 characteristics were included as likely SMT responders. Subjects with 2 or fewer characteristics were included as likely non-responders based on previously-identified likelihood ratios for predicting SMT response.[5] Subjects with 3 characteristics were excluded because predicting response in these subjects is uncertain.[5] The study was approved by the Institutional Review Board at the University of Utah and Brooke Army Medical Center. All subjects provided informed consent.

Measurements

Subjects attended 3 sessions. Session 1 included completion of questionnaires, a standardized clinical examination, and pre- and post-SMT spinal stiffness and LM assessments. Subjects returned for session 2, 3-4 days after the first. Session 2 involved administration of the questionnaires and a second SMT intervention with pre- and post-stiffness and LM assessments. Session 3 occurred after another 3-4 days and included the questionnaires, clinical examination, and final stiffness and LM assessments (figure 1).

Demographic information was recorded at session 1, and subjects completed questionnaires at the beginning of each session. A modified version of the ODI, with documented reliability and responsiveness, to measure LBP-related disability.[33] A 0-10 numeric pain rating assessed pain intensity. Ratings for current, best and worst pain during the past 24 hours were averaged.[34] A pain body drawing documented symptom location over the past 24 hours.[35] The 7-item work (FABQW), and 4-item physical activity (FABQPA) subscales of the Fear Avoidance Beliefs Questionnaire[36] measured subjects' beliefs about the impact of physical activity and work on their LBP. The clinical examination included the CPR criteria (table 1).

Spinal stiffness was assessed using a mechanized indentation instrument similar to those described previously.[37] The instrument consists of a motorized indentation probe with a compressive-tension load cell (Entran, Fairfield, NJ) supported by an external frame. Probe displacement is measured by a linear variable differential transformer (Honeywell International Inc., Morristown, NJ). Signals from the load cell and transformer are collected by customized LABview software (National Instruments, Austin, TX) at a collection rate of 200 Hz. Calibration procedures and reliability for stiffness measures made using similar procedures are detailed elsewhere.[38]

Spinal stiffness was assessed with the subject prone (figure 2). The examiner manually identified and marked the L3 spinous process as the indentation site. The L3 level was used because motion at the L3/L4 segment is less likely to be painful, and does not differ from L4/L5 motion[39] the level from which LM measures were taken. Studies show the effects of SMT are not segment-specific,[40-42] therefore post-SMT changes were expected to be reflected at the L3/L4 segment. The subject was instructed to inhale and exhale comfortably, then hold their breath at the end of exhalation for approximately 5 seconds during indentation. Indentation involved advancement of the probe from a pre-load of 5 N to a final

load of 60 N. The 60 N load was maintained for 1 second; then the probe automatically raised. Three indentations were performed at each assessment, with mean values used for analysis.

Indentation data (force and displacement) were used to calculate stiffness. Slope of the force displacement curve between 5 N and 60 N was calculated as global stiffness (GS), representing stiffness of the underlying tissues throughout indentation. Terminal stiffness (TS) was calculated as the ratio between the applied maximal force and resultant maximal displacement (N/mm), representing stiffness at the end of indentation.

Multifidus function was assessed ultrasonically (Sonosite Inc. Bothell, WA) with a 60 mm, 5 MHz curvilinear array. Thickness of the LM at L4/5 on the subject's more symptomatic side was quantified during submaximal contraction using a protocol with documented reliability.[43] Contraction was elicited by the prone subject holding a 1-2 kg weight and lifting the contralateral arm approximately 5 cm, resulting in approximately 30% maximal voluntary LM contraction.[44] Image acquisition was performed 3 times. Measures were averaged to reduce variability.[45] Images were measured offline using Image J software (Wayne Rasband, National Institutes of Health, USA). Contracted thickness was measured as the distance between the posterior-most portion of the facet joint and the fascial plane between the muscle and subcutaneous tissue (figure 3).[46] Recruitment was calculated as $(\text{thickness}_{\text{initial}} - \text{thickness}_{\text{final}}) / \text{thickness}_{\text{initial}}$.

Stiffness and LM assessments were performed 5 times (figure 1). The first 2 assessments occurred at session 1, before (SS1) and after (SS2) SMT to evaluate immediate changes. The third (SS3) and fourth (SS4) assessments were performed before and after SMT during session 2. Final assessment (SS5) during session 3 examined sustained stiffness and LM changes.

Intervention

A physical therapist or chiropractor provided SMT during sessions 1 and 2. We used a supine SMT technique described in detail elsewhere (figure 4).[4,5] The technique provides a posterior-inferior thrust at the subject's pelvis. Thrusts were applied to each side of the pelvis during each session.

Data Analysis

Analyzes were performed using SPSS and AMOS version 17 (SPSS Inc, Chicago, IL). Descriptive statistics were calculated for stiffness variables (GS and TS) at each assessment. Within- and between-sessions stiffness comparisons were made using paired t-tests.

Associations between stiffness and clinical outcomes were examined using stepwise hierarchical linear regression. Percentage ODI improvement from baseline to session 3 $((\text{ODI}_{\text{initial}} - \text{ODI}_{\text{final}}) / \text{ODI}_{\text{initial}} * 100\%)$ was the dependent variable. Demographic characteristics (age, sex, BMI) and CPR status were entered in step 1 as control variables. Initial GS and TS, and immediate (SS1 to SS2) and sustained (SS1 to SS5) GS and TS changes were considered for stepwise entry in step 2 to evaluate if stiffness characteristics explained outcome beyond the control variables. Stiffness changes were calculated relative to the SS1 value: $(\text{stiffness}_{\text{initial}} - \text{stiffness}_{\text{final}}) / \text{stiffnessnitial}$. Significance <0.05 was required to enter the model. Significance ≥ 0.10 removed a variable.

A theoretical model linking clinical outcomes from SMT to spinal stiffness, LM recruitment, and CPR status was explored with path analyses. The initial model (figure 5) was developed from theorized relationships and results from this and other studies,[5,31] based on the following hypotheses: 1) outcome is directly and independently explained by CPR status,

initial TS, and immediate GS change, 2) CPR status, initial TS and immediate GS change also explain outcome based on their influence on LM recruitment.

Zero-order (i.e., bivariate) correlations between model variables were examined. Path coefficients and overall model fit were tested using maximum likelihood estimates. Path coefficients are standardized regression β weights indicating the direct effect of one model variable on another. Overall fit was assessed with recommended measures.[47,48] The χ^2 tested the null hypothesis of no difference between the model and sample data. Because χ^2 is sensitive to sample size we divided by degrees of freedom and considered $\chi^2/df < 2.0$ to represent a well-fitting model.[49] Root mean square error of approximation (RMSEA) estimated model fit and parsimony compared to a perfect model. Values <0.6 indicate excellent model fit. Comparative fit index (CFI) assessed improvement in fit comparing the tested model to a theoretical model with no relationship between variables. Values >0.95 signify good fit. Adjusted goodness-of-fit index (AGFI) estimated the proportion of variance of the sample covariance matrix accounted for by the tested model adjusted for degrees of freedom, penalizing non-parsimonious (i.e., inefficient) models. Values >0.85 indicate good fit. After testing the initial model, paths and variables with the largest p-values were trimmed sequentially to create a more parsimonious model. Parameter estimates and fit indices were recalculated after each elimination. Trimming was stopped once further elimination resulted in diminished model fit.

Results

Fifty-one subjects were recruited; 1 dropped out after session 1. Baseline characteristics (n=50) are outlined in table 2. Twenty-one subjects (42.0%) were likely SMT responders based on CPR status (table 1), and 28 (56.0%) were likely non-responders. One subject with 3 characteristics was incorrectly enrolled. This subject was included as a likely non-responder. Significant ODI improvement occurred at each follow-up session (table 3).

Two subjects had incomplete stiffness data due to technical errors. Stiffness values are presented for 48 subjects. Significant immediate changes in GS and TS occurred with SMT during session 1, and in TS with SMT during session 2. No sustained stiffness changes occurred from session 1 to session 2 or 3 (table 3). Stiffness characteristics contributed to the explanation of outcome beyond control variables (table 4). Initial TS and immediate GS change entered the model. Regression coefficients indicated less initial TS and greater immediate GS reduction were associated with greater ODI improvement.

Significant zero-order correlations were found between initial TS and immediate (r = -0.29) and sustained change (r = -0.34) in LM recruitment, indicating less initial TS was associated greater increase in recruitment. Immediate change in LM recruitment was associated with CPR status (r =0.30) indicating being a likely responder correlated with greater immediate increase in LM recruitment. Significant correlations existed between LM recruitment change at each measurement (table 5). The initial model (figure 5) fit the data (χ^2/df =1.6, p =0.11), however additional indicators revealed opportunities to improve parsimony and fit. A trimmed model (figure 6) removing paths between CPR status, initial TS and immediate GS change; and removing the intermediary LM recruitment change improved fit and parsimony (χ^2/df = 0.86, p =0.55). The amount of explained variance in percent ODI improvement was low (R^2 =0.11 and 0.10 respectively).

Discussion

Using mechanized spinal stiffness assessments, we identified stiffness characteristics that related to clinical outcome with SMT. Specifically, less initial TS and greater immediate

reduction in GS explained variation in ODI improvement over 1-week. We used these results and previous research to examine a model of SMT's effects as related its ability to provide a mechanical force able to impact stiffness and facilitate LM recruitment in subjects with clinical characteristics fitting the CPR. Path analyzes suggested the effects of SMT may be mediated by both immediate GS changes and enhancement in LM recruitment, with initial TS and CPR status influencing the ability to improve LM recruitment with SMT. This study was the first to examine multiple potential SMT mechanisms concurrently. Although our models fit the data, they explained a relatively small percentage of variability in outcome, indicating an opportunity to refine and expand models in future research.

Our model of SMT effects was based on recent studies suggesting the mechanical stimulus of SMT may result in CNS changes, including post-synaptic alpha motorneuron and/or corticomotoneuron facilitation, and improved cortical somatosensory integration.[8,50-52] Our prior research identified post-SMT facilitation of the LM, and related this effect to SMT outcomes.[30,31] Our model also included clinical characteristics we previously identified as a CPR for predicting likelihood of clinical benefit with SMT.[4,5] The CPR characteristics introduce additional factors including fear-avoidance beliefs, symptom duration and location to our models. Baseline CPR status and TS were significantly related to immediate post-SMT LM recruitment change in our initial model, but were not correlated directly with each other or outcome. This may indicate the relationship between these factors and outcome is mediated through LM recruitment changes. Paths between immediate GS change and both immediate LM recruitment change and outcome approached significance,(p =0.09) possibly supporting the hypothesis that effective SMT impacts both stiffness and LM recruitment. Immediate improvements in LM recruitment were highly related to recruitment changes at 1-week follow-up, when no SMT was provided, suggesting this facilitation is not transient, and may be enhanced by combining SMT with exercise interventions designed to promote LM function. Considering emerging evidence the role LM dysfunction in persistent or recurrent LBP,[23,29] facilitation of LM recruitment may be an important aspect of the mechanism of effect underlying SMT.

Previous studies examining stiffness and SMT have relied on manual assessments with notoriously poor validity and reliability.[53-56] We used mechanized stiffness assessments and found significant immediate reductions post-SMT regardless of outcome. This result contradicts previous studies that failed to identify an immediate effect of non-thrust mobilization on stiffness,[57-59] possibly highlighting different mechanical effects of thrust versus non-thrust techniques. We did not find sustained stiffness changes from the first to the final session, contradicting studies reporting stiffness reductions across a treatment episode.[60,61] This may be due to our short follow-up time or low SMT dosage.

Changes that occur with SMT should be examined in relation to clinical outcome in order to distinguish underlying mechanisms from unrelated epiphenomena. Only one prior study[60] examined the relationship between stiffness changes and outcome, reporting no correlation between outcome and stiffness change across 8 weeks of treatment for all subjects in their trial, or within groups receiving different treatments, including non-thrust mobilization.[60] We found a significant relationship between immediate post-SMT stiffness decrease and clinical outcome. This finding may suggest a relationship between stiffness change and outcome for thrust SMT that is not present if non-thrust mobilization techniques are employed. This result may also indicate the most relevant stiffness variable for future research is the immediate post-SMT response, not sustained change over time.

We quantified two measures of spinal stiffness (GS and TS). Our results suggest differential effects of SMT on each. Both stiffness measures take into account peak force and resulting displacement of the indentation process, but differ in that GS reflects the rate of change in

applied force and resulting displacement during the entire indentation, while TS represent only the peak applied force and peak resulting displacement. The significance of an immediate decrease in GS, but not TS, in explaining clinical outcome may suggest stiffness was altered by SMT at points during indentation, but total peak displacement was unchanged. The significance of less initial TS, but not GS, may indicate SMT is more effective in patients able to achieve a requisite degree of peak displacement for a given load. Subjects unable to achieve this displacement may be less responsive to SMT, at least at the dosage in this study. Prior studies have examined the effects of SMT on stiffness and muscle recruitment as separate hypotheses.

Prior research has identified an association between greater pre-treatment stiffness based on manual assessment and clinical outcomes with SMT.[5,62] This study found an association between less pre-treatment TS and SMT outcome. This apparent contradiction may relate to the validity of manual stiffness assessments. Despite efforts at standardization, clinicians base manual stiffness assessments on varied constructs, including both quality and quantity of motion.[63] Previous associations between manual judgments and favorable SMT outcomes may have occurred because clinicians were identifying stiffness based on a construct other than aspects of the force-displacement curve on which the mechanized assessments are based. Manual judgments could relate to SMT outcome, but may identify a construct other than actual mechanical stiffness.

This study had several limitations. Our SMT protocol was brief, and may not reflect the dosage or duration typical of clinical practice. We did not evaluate long-term clinical outcomes. The short-term responses of the subjects may not be indicative of long-term outcomes. We did not include asymptomatic subjects, making it difficult to relate stiffness characteristics among symptomatic subjects to normative values. Our sample size was small, particularly for the path analyses. Our results should be considered preliminary, and our theoretical model requires replication and likely revisions and expansion. Clarifying the potential clinical relevance of changes in stiffness and LM recruitment will require additional research.

Understanding mechanisms of effect of SMT has been identified as a research priority with the potential to enhance the development and delivery of more effective SMT procedures.[22, 64] This study provides important advances in understanding hypothesized relationships between SMT and spinal stiffness, and is the first to concurrently evaluate stiffness, LM recruitment, and CPR status, relating these factors to clinical outcomes. We believe future research will need to model the multi-factorial nature proposed by contemporary theories of SMT effects in order to identify relevant mechanisms.

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Figure 1.
Time line for sessions and measurements in the study.

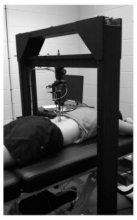

Figure 2.
Mechanized indentation instrument used for spinal stiffness assessments.

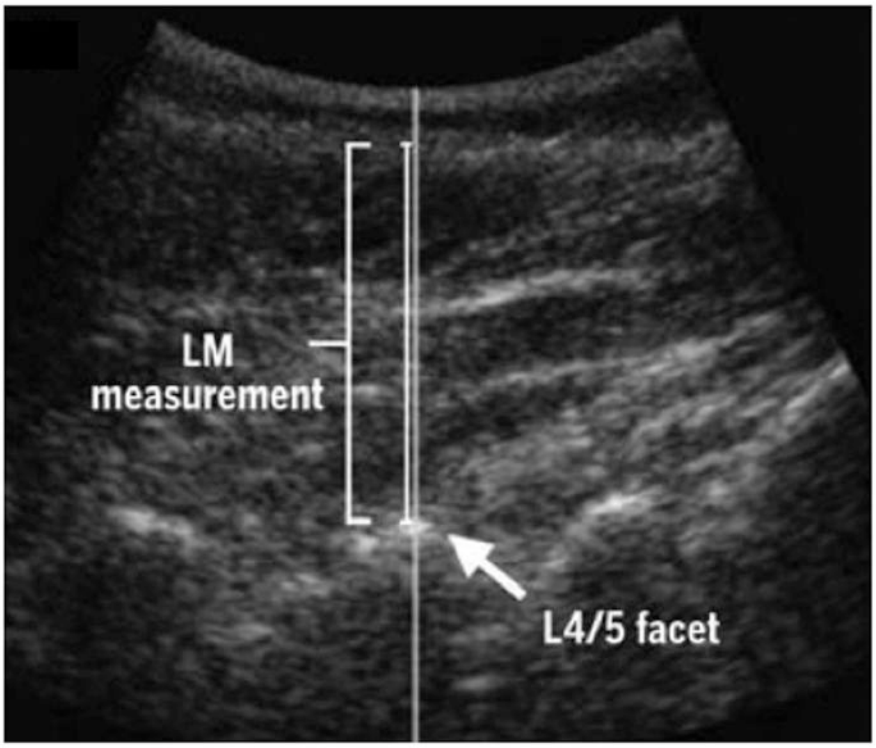

Figure 3.
Ultrasound image of the lumbar multifidus (LM) during a submaximal contraction using a contralateral arm raise. Contracted thickness was measured between the posterior-most portion of the L4/5 facet joint and the plane between the muscle and subcutaneous tissue.

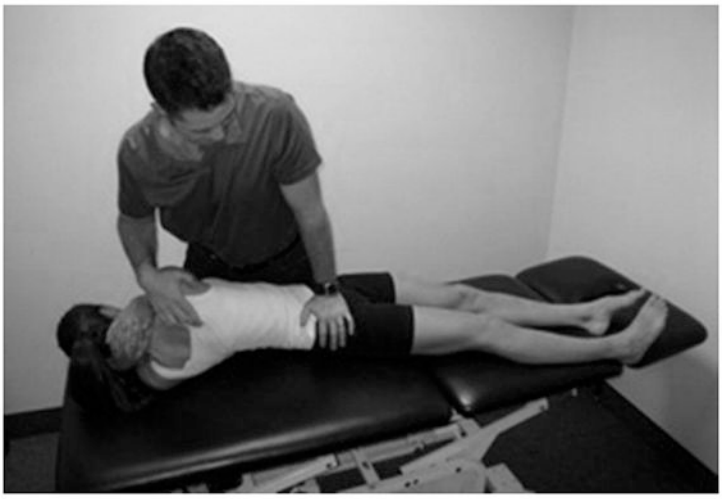

Figure 4.
Spinal manipulation technique. Each side of the subject's pelvis was manipulated at each session.

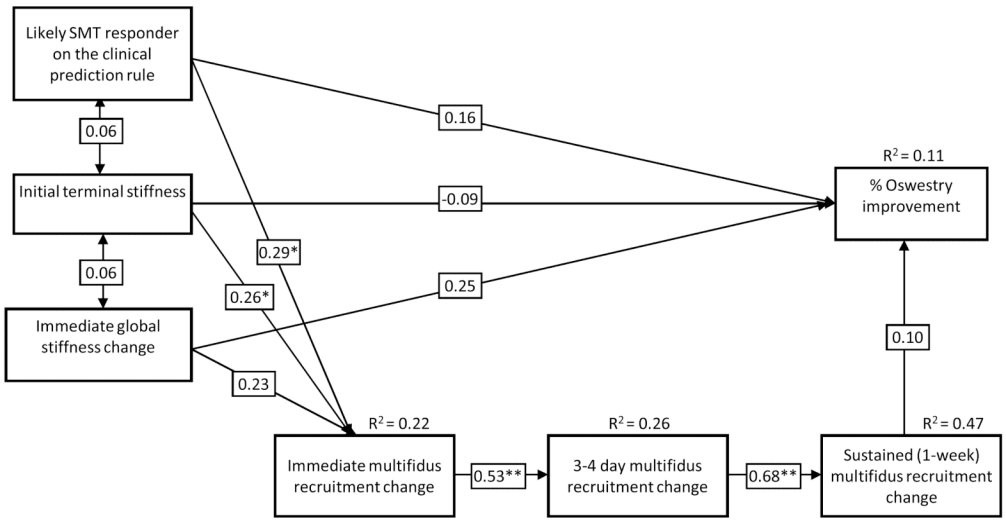

Figure 5.

Path analysis and output from initial theoretical model. Direct standardized regression coefficients between variables are shown with each arrow.(* p <0.05, ** p <0.001) The R^2 values represent the explained variance accounted for by the variables linked in the model. (χ^2 / df = 1.6, p = 0.11, RMSEA = 0.12, CFI = 0.89, AGFI = 0.76)

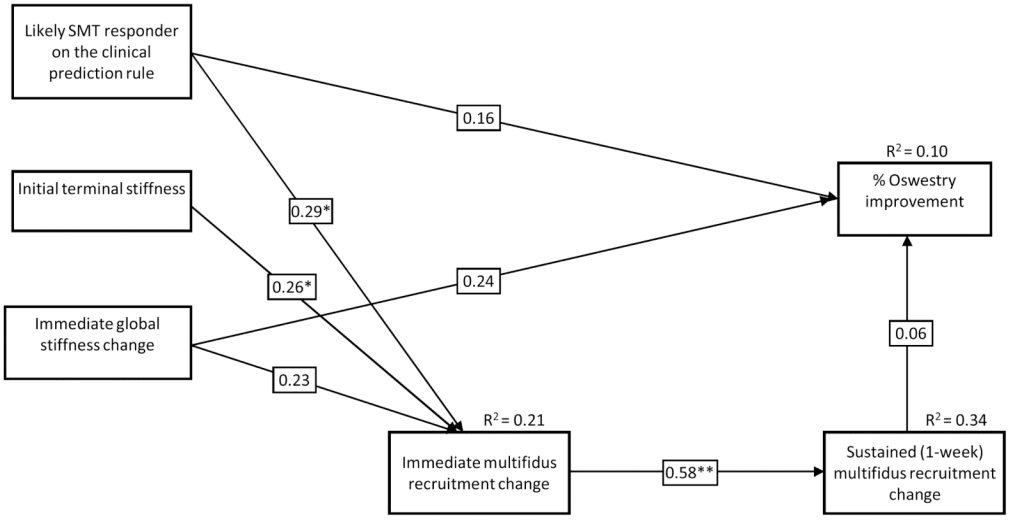

Figure 6.

Path analysis and output from the trimmed model. Direct standardized regression coefficients between variables are shown with each arrow.(* p <0.05, ** p <0.001) The R^2 values represent the explained variance accounted for by the variables linked in the model. (χ^2 / df = 0.86, p = 0.55, RMSEA = 0.0, CFI = 1.0, AGFI = 0.88)

Table 1

Clinical Prediction Rule criteria for identifying likely responders to spinal manipulative therapy.[5]

Criterion	Measurement Procedure	Definition of positive
1. Duration of current episode of low back pain	Self-report	Less than 16 days
2. Extent of distal symptoms	Pain Drawing	Not having symptoms distal to the knee
3. Fear-avoidance beliefs	Fear-Avoidance Beliefs Questionnaire work subscale	Less than 19 points
4. Lumbar spinal stiffness	Manual postero-anterior stiffness assessment	At least one lumbar segment judged to be hypomobile
5. Hip internal rotation range of motion	Prone assessment using an inclinometer	At least one hip with greater than 35° of range of motion

Table 2

Baseline Subject Characteristics (n=50). (Numbers represent mean (± standard deviation) unless otherwise indicated)

Characteristic	
Age	33.3 (± 12.9) years
Sex	54.0% female
Body Mass Index (kg/m^2)	27.0 (± 6.1)
Prior Episodes of Low Back Pain	88.0% yes
Duration of Symptoms in Current Low Back Pain Episode	median =186 days (range: 2 days - 24 years)
Distribution of Symptoms	26.0% symptoms distal to the knee
Numeric Pain Rating	4.9 (± 1.6)
Fear-Avoidance Beliefs Questionnaire –Physical Activity Sub scale	14.4 (± 4.4)
Fear-Avoidance Beliefs Questionnaire –Work Sub scale	15.4 (± 8.9)
Oswestry Disability Index	32.2 (± 11.9)
Likely Manipulation Responder (at least 4 clinical prediction rule criteria)	42.0% yes

Table 3

Stiffness measurements and Oswestry scores at each measurement session (n=48). Values represent mean (standard deviation).

		Oswestry Score	Global Stiffness (N)	Terminal Stiffness (N/mm)
Session 1:	SSI	32.2 (11.9)	5.6 (1.6)	4.3 (1.8)
	SS2		5.4 (1.7)	3.9 (1.4)
	Change from SS1 to SS2		0.12 (0.39)	0.39 (1.2)
	p value		0.034	0.027
Session 2:	SS3	26.2 (14.1)	5.5 (1.5)	4.3 (1.5)
	Change from SSI to SS3	5.8 (9.5)	0.022 (0.78)	0.0009 (1.3)
	p value	<0.001	0.85	1.0
	SS4		5.4 (1.6)	4.0 (1.3)
	Change from SS3 to SS4		0.084 (0.55)	0.34 (0.63)
	p value		0.30	0.001
Session 3:	SS5	23.9 (11.4)	5.4 (1.5)	4.1 (1.2)
	Change from SS1 to SS5	8.1 (11.4)	0.26 (1.5)	0.12 (0.81)
	p value	<0.001	0.31	0.23

Table 4

Results of stepwise hierarchical linear regression with percentage change in Oswestry as the dependent variable (n=48).

Variables Entered	Adjusted R^2	Significance of R^2 Change	Standardized Beta Coefficient (Final Model)	Significance of Beta Coefficient
Step 1:	0.076	0.13		
age			0.21	0.19
male sex			0.09	0.53
BMI			-0.61	0.002
Met CPR			0.10	0.65
Step 2:	0.14	0.05		
Immediate change in global stiffness			0.34	0.024
Step 3:	0.21	0.047		
Initial terminal stiffness			-0.32	0.047

Table 5

Zero-order correlations among variables considered for the theoretical models (* p <0.05, **p <0.001).

	2	3	4	5	6	7
1. Likely SMT responder based on clinical criteria	0.062	.003	0.30*	0.034	0.26	0.18
2. Initial terminal stiffness		0.061	-0.29*	-0.25	-0.34*	-0.038
3. Immediate change in global stiffness			-0.25	-0.080	0.038	0.24
4. Immediate change in multifidus recruitment				0.53**	0.59**	-0.013
5. 3-4 day change in multifidus recruitment					0.69**	0.006
6. 1-week change in multifidus recruitment						-0.096
7. Percentage Oswestry improvement						