

Re: "Interrater reliability of a new classification scheme for patients with neural low back-related leg pain" Schäfer A et al. *J Man Manip Ther* 2009;17:109–116.

We read with interest the papers by Schäfer and colleagues¹ on the classification of patients with low back and leg pain and the associated study by Walsh & Hall² on psychological differences between subgroups within this classification scheme. We agree that the time has come for clinicians to seriously consider that leg pain associated with low back pain (LBP) is not a homogenous subgroup unto itself and consideration should be given as to whether patient signs and symptoms have a dominant neural or musculoskeletal component.

These papers raise some interesting questions on which we would like to comment. In the paper by Schäfer and colleagues¹ we do not believe the criteria used by the authors for the "central sensitization" and "peripheral nerve sensitization" subgroups differentiates sufficiently between these categories. Schäfer et al¹ rely on the Leeds Assessment of Neuropathic Signs and Symptoms (LANSS) with a score of 12 or more to place patients in the central sensitization category and a series of neural provocation signs combined with palpation to place patients in the peripheral nerve sensitization category.

Our understanding of the LANSS is that it was developed to identify patients that may have pain of *neuropathic* origin³ and we do not agree with Schäfer et al¹ that a score over 12 necessarily indicates that *central sensitization* is the dominant symptom mechanism. Spontaneous pain, burning pain, and the presence of hyperalgesia screened for in the LANSS are not exclusively found in individuals with central sensitization and have also been at-

tributed to *peripheral nerve sensitization*⁴. In a validation study of the self administered LANSS, Bennett et al⁵ used patients of varying "peripheral nerve" diagnoses including diabetic neuropathy and nerve entrapment. Therefore, while a high score on the LANSS *may* be indicative of central sensitization, we feel this tool is not sensitive enough, nor originally intended for, diagnosis of this pain type exclusively.

We also contend that the clinical signs and tests used to classify patients by authors as having a peripheral nerve sensitization could also be attributed to patients who have in fact central sensitization. Positive neural tissue provocation tests such as the straight leg raise, prone knee flexion and their pain provocative variants could also be attributed to central sensitization. Sterling et al⁶ proposed that positive responses to upper limb neural provocation tests in a whiplash cohort could represent a manifestation of the flexor withdrawal response due to increased excitability of the central nervous system. Positive responses to analogous tests of the lower extremity (e.g. straight leg raise) may represent nerve trunk sensitivity as the authors claim, but could also be explained by the mechanism described by Sterling et al⁶.

The other criterion that Schäfer et al¹ use to classify patients as peripheral nerve sensitization is pain on palpation of nerve trunks. It is questionable whether or not this finding assists in meaningful differentiation between the two pain mechanisms as elicited tenderness on palpation could be an area of secondary hyperalgesia and hence be attributed to part of a central sensitization pain mechanism just

as easily as peripheral sensitization as the authors propose⁷. Therefore we believe that neural provocation tests and palpation by themselves lack the sufficient sensitivity to discriminate between these two types of pain mechanisms.

Perhaps it is this difficulty in differentiation that in part explains the surprising subgroup differences that were reported by Walsh & Hall². The authors report that the peripheral sensitization subgroup had significantly higher Oswestry Disability, pain, anxiety and Fear Avoidance Belief Questionnaire scores than all the other subgroups described by Schäfer et al¹. We share their surprise at these findings and agree with the expectation that these measures would be significantly higher in the centralization subgroup. We feel these findings serve to underscore our point that the criteria for the described classification scheme do not clearly differentiate between the subgroups per the proposed mechanisms.

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REFERENCES

1. Schäfer A, Hall TM, Lüdtke K, Mallwitz J, Briffa N. Interrater reliability of a new classification scheme for patients with neural low back-related leg pain. *J Man Manip Ther* 2009;17:109–116.
2. Walsh J, Hall T. Classification of low back-

- related leg pain: Do subgroups differ in disability and psychological factors? *J Man Manip Ther* 2009;17:118-123.
3. Bennett M. The LANSS pain scale: the Leeds assessment of neuropathic symptoms and signs. *Pain* 2001;92:147-157.
 4. Sandkühler J. Models and mechanisms of hyperalgesia and allodynia. *Physio Rev* 2009;89:707-758.
 5. Bennett MI, Smith BH, Torrance N, Potter J. The S-LANSS score for identifying pain of predominantly neuropathic origin: validation for use in clinical and postal research. *J Pain* 2005;6:149-158.
 6. Sterling M, Treleaven J, Jull G. Responses to a clinical test of mechanical provocation of nerve tissue in whiplash associated disorder. *Man Ther* 2002;7:89-94.
 7. Treede RD, Rolke R, Andrews K, Magerl W. Pain elicited by blunt pressure: neurobiological basis and clinical relevance. *Pain* 2002;98:236-240.