AUTHOR RESPONSE

We would like to thank O'Hearn and colleagues for their interest in our paper and the stimulating questions they raised. We believe however that some of the concerns raised regarding the validity of the classification system are based on misinterpretation of our previous paper[1].

First of all, the classification system for patients with low back related leg pain classifies patients in four distinct, hierarchical categories[1,2]. The classification system is considered hierarchical because if patients fit the criteria for the first category they are assigned to this category regardless of whether they meet criteria further down the hierarchy. Subjects with a score of 12 or higher on the LANSS scale[3] were classified as Central Sensitization (CS). When there was no evidence of Central Sensitization, but negative symptoms such as hypoesthesia, muscle weakness or hyporeflexia were present, categorization of Denervation (D) was made. Of the remaining subjects, those with positive nerve provocation tests were classified as Peripheral Nerve Sensitization (PNS). Subjects with no suggestion of neural involvement were classified as Musculoskeletal (M). This implies that groups CS or D may include patients with signs and symptoms indicative of PNS. However central pain mechanisms in group CS and mechanisms responsible for loss of conduction in group D are considered dominant, as they may persist even when the peripheral trigger has resolved[4]. It is widely acknowledged that neuropathic pain can rarely be attributed to a single mechanism[5]. Where mixed mechanisms may be contributing, clinical judgment is required to establish the predominant mechanism[6] to provide a foundation for clinical reasoning and treatment. We believe that the classification system has the potential to aid clinical judgment in this regard.

We agree with O'Hearn that positive nerve provocation tests may not be exclusively a sign of PNS, but that also central mechanisms could elicit these signs as demonstrated by Sterling et al[7]. However, a strength of our classification system is that none of the sub-groups are defined by a single clinical feature. Patients in group PNS are characterized not only by the presence of three positive nerve provocation tests (Positive straight leg raise or prone knee bend test, positive trunk flexion test in standing and positive nerve palpation) but also by the absence of symptoms and signs indicative of central sensitization (LANSS < 12). In this way, patients with dominant central pain mechanisms are excluded and patients with relatively "pure" peripheral nerve sensitization remain in group PNS.

As O'Hearn stated, the LANSS questionnaire was designed to detect pain of predominantly neuropathic origin. However, items within the LANSS scale are primarily concerned with identifying positive features of neuropathic pain, such as hyperalgesia and allodynia, which are hall mark signs for central pain mechanisms[8-10]. Also, consensus exists that central sensitization is one of the main mechanisms contributing to neuropathic pain[11-13]. Consequently, we named the group "Central Sensitization". The term Central Sensitization is simply a label. In retrospect this label conjures belief that subjects thus classified have purely mechanisms of central sensitization but this is not the case. We have therefore considered changing the group label in future publications. Furthermore we acknowledge that the LANSS scale is not the most appropriate instrument to detect central sensitization. The use of the LANSS scale is a first step, until other, more sensitive and specific instruments are developed.

We believe it is misleading to interpret scores obtained from the Oswestry Disability Questionnaire, and pain, anxiety and Fear Avoidance Belief Questionnaire in the study of Walsh & Hall[14] as evidence of central sensitization in group PNS. We reiterate that subjects in group CS were classified first and had higher levels of hallmark features of central sensitization, in contrast to lower levels in subjects classified in group PNS. As Walsh & Hall[14] point out, higher levels of disability and fear of movement in group PNS may be caused by neural tissue mechanosensitivity, present in all subjects in group PNS but to a lesser extent in other groups.

We would like to point out that further refinement of the classification system of low back related leg pain is required. We hope that our papers stimulate further research in this area.

REFERENCES

1. Schäfer A, Hall T, Lüdtke K, Mallwitz J, Briffa K. Interrater reliability of a new classification system for patients with low back related leg pain. *J Man Manip Ther* 2009;17:109–117.
2. Schäfer A, Hall T, Briffa K. Classification of low back-related leg pain—A proposed

patho-mechanism-based approach. *Man Ther* 2009;14:222–230.
3. Bennett M. The LANSS Pain Scale: the Leeds assessment of neuropathic symptoms and signs. *Pain* 2001;92:147–157.
4. Woolf CJ, Salter MW. Neuronal plasticity: Increasing the gain in pain. *Science* 2000; 288:1765–1768.
5. Finnerup NB, Otto M, McQuay HJ, Jensen TS, Sindrup SH. Algorithm for neuropathic pain treatment: An evidence based proposal. *Pain* 2005;118:289–305.
6. Bennett MI, Smith BH, Torrance N, Lee AJ. Can pain can be more or less neuropathic? Comparison of symptom assessment tools with ratings of certainty by clinicians. *Pain* 2006;122:289–294.
7. 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.
8. Treede RD. Peripheral and central mechnisms of cutaneous hyperalgesia. *Prog Neurobiol* 1992;38:397–421.
9. Magerl W, Fuchs PN, Meyer RA, Treede RD. Roles of capsaicin-insensitive nociceptors in cutaneous pain and secondary hyperalgesia. *Brain* 2001;124:1754–1764.
10. Treede RD, Handwerker HO, Baumgärtner U, Meyer RA, Magerl W. Hyperalgesia and Allodynia: Taxonomy, Assessment, and Mechanisms. In: K Brune and Handwerker HO, eds. In: *Hyperalgesia: Molecular mechanisms and Clinical Implications*. Seattle: IASP Press, 2004;3–15.
11. Jensen TS, Baron R. Translation of symptoms and signs into mechanisms in neuropathic pain. *Pain* 2003;102:1–8.
12. Woolf CJ, Mannion RJ. Neuropathic pain: Aetiology, symptoms, mechanisms, and management. *Lancet* 1999;353:1959–1964.
13. Dickenson AH, Bee LA. Neurobiological mechanisms of neuropathic pain and its treatment. In: J Castro-Lopes, Raja S, and Schmelz M, eds. In: *Pain 2008 An Updated Review*. Seattle: IASP press, 2008; 277–286.
14. Walsh J, Hall TM. Classification of low-back related leg pain—Do subgroups differ in disability and psychosocial factors? *J Man Manip Ther* 2009;17(2):118–123.

Alex Schafer PT, PhD
Toby Hall MSC, PhD Cand
Kerstin Lüudtke, MSC
Joachim Mallwitz, MD
Noelle K. Briffa, PhD