Differential development of sensory hypersensitivity and a measure of spinal cord hyperexcitability following whiplash injury

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1. Introduction

Persistent pain and disability develops in up to 50% of those who experience a whiplash injury [6] and it is this group who incur substantial costs [24]. The reasons for the transition to chronicity for some injured people is not clear but data from a recent systematic review indicate that most recovery occurs in the early 2–3 month period after injury, after which time the condition plateaus [19]. A greater understanding of processes within this early period post-injury will be important in order to develop strategies to prevent the transition to chronicity.

We have previously shown that widespread sensory hypersensitivity (decreased pain thresholds to mechanical and cold stimuli) is present from within a few weeks of injury and is associated with poor functional recovery [35,36]. Consistent with these findings, Kasch et al. [20] found that reduced cold pressor pain tolerance is also a predictor of non-recovery. These phenomena are generally thought to reflect augmented central pain processing mechanisms [8]. One drawback of the measures used in these studies is that the pain threshold data relies on the participants’ voluntary responses. Potentially responses may be influenced by factors such as misinterpretation of the examiner’s requests or even secondary gain.

In view of this, recent investigations of chronic whiplash have utilized the nociceptive flexion reflex (NFR), a spinal reflex where threshold muscle reflex activity is measured following electrical stimulation to the sural nerve at the ankle. The NFR is a measure of spinal cord excitability that (unlike pain threshold responses) does not rely on a cognitive response from participants [10]. Decreased NFR thresholds have been shown to be present in chronic WAD [2,37] and are indicative of spinal cord hyperexcitability. NFR responses in acute WAD or their association with the transition to chronic pain have never been investigated.

The aims of this study were to: (1) determine the temporal development of NFR responses following whiplash injury; (2) to compare the development of NFR responses and pain thresholds following whiplash injury; and (3) to identify predictors of persistent spinal cord hyperexcitability (NFR responses) at 6 months post-injury.

Widespread sensory hypersensitivity is present in acute whiplash and is associated with poor recovery. Decreased nociceptive flexion reflex (NFR) thresholds (spinal cord hyperexcitability) are a feature of chronic whiplash but have not been investigated in the acute to chronic injury stage. This study compared the temporal development of sensory hypersensitivity and NFR responses from soon after injury to either recovery or to transition to chronicity. It also aimed to identify predictors of persistent spinal cord hyperexcitability. Pressure and cold pain thresholds, NFR responses (threshold and pain VAS) were prospectively measured in 62 participants at <3 weeks, 3 and 6 months post whiplash injury and in 22 healthy controls on two occasions a month apart. Pain levels and psychological distress (GHQ-28; IES) were measured at baseline. Whiplash participants were classified at 6 months post-injury using the Neck Disability Index; recovered (≤8%), mild pain and disability (10–28%) or moderate/severe pain and disability (≥30%). All whiplash groups demonstrated spinal cord hyperexcitability (lowered NFR thresholds) at 3 weeks post-injury. This hyperexcitability persisted in those with moderate/severe symptoms at 6 months but resolved in those who recovered or reported lesser symptoms at 6 months. In contrast generalized sensory hypersensitivity (pressure and cold) was only ever present in those with persistent moderate/severe symptoms and remained unchanged throughout the study period. This suggests different mechanisms underlie sensory hypersensitivity and NFR responses. In multivariate analyses only initial NDI scores (p = 0.003) were a unique predictor of persistent spinal cord hyperexcitability indicating possible ongoing peripheral nociception following whiplash injury.
2. Methods

2.1. Study design

A prospective longitudinal design was used to study persons who sustained a whiplash injury from within 3 weeks of injury and followed to 3 and 6 months post-injury. An asymptomatic control group was assessed twice, a month apart.

2.2. Participants

Sixty two volunteers (36 females, mean age 36.1 ± 13.13 years) reporting neck pain as a result of a motor vehicle crash (MVC) and 22 asymptomatic volunteers (14 females, mean age 40.1 ± 13.6 years) participated in the study. The participants with whiplash were recruited via hospital accident and emergency departments, primary care practices (medical and physiotherapy) and from advertisement within radio and print media. They were eligible if they met the Quebec Task Force Classification of WAD II or III [33]. Subjects were excluded if they were WAD IV (fracture dislocation), experienced concussion, loss of consciousness or head injury as a result of the accident and if they reported a previous history of whiplash, neck pain or headaches that required treatment or if they had ever been diagnosed with tension-type headache or migraine. The asymptomatic control groups was recruited from the general community from print media advertisement and were included provided they had never experienced any prior pain or trauma to the cervical spine, head or upper quadrant. Ethical clearance for this study was granted by the Institutional Medical Research Ethics Committee.

2.3. Pain threshold measures

Pressure pain thresholds (PPTs) were measured using a pressure algometer with a probe size of 1 cm² and application rate of 40 kPa/s (Somedic AB, Farsta, Sweden) over the C5 spinous process and bilaterally over the median nerve at the elbow and over the muscle belly of tibialis anterior. Participants were requested to push a button when the sensation changed from one of pressure alone to one of pressure and pain [5]. Triplicate recordings were taken at each site and the mean values used for analysis.

Cold pain thresholds were measured over the mid to lower regions of the cervical spine using the Thermostat system (Somedic AB, Farsta, Sweden). The thermode was preset to 30 °C with the rate of temperature change being 1 °C/s. Participants were asked to push a patient-controlled switch when the cold sensation first became painful [16]. Triplicate recordings were taken and the mean values used for analysis.

2.4. Nociceptive flexion reflex (NFR)

The NFR was measured on the participants’ right side. Participants lay prone, with the right ankle supported by pillows to maintain the right knee in a position of approximately 30° flexion. Bipolar Ag–AgCl surface electrodes were placed over the sural nerve, inferior to the lateral malleolus to convey the electrical stimulus. A second pair of Ag–AgCl surface electrodes was placed over the right biceps femoris muscle proximal to the musculo-tendinous junction to measure electromyographic (EMG) reflex responses. The electrical stimulus consisted of a train of five 1 ms square wave pulses (perceived as a single stimulus) with a 5 ms pulse interval delivered by a DS7A High Voltage Constant Current Stimulator (Digitimer, UK). Stimulation intensity began at 2 mA and progressed in 2 mA increments until an NFR was elicited. This stimulation intensity was defined as the NFR threshold. A computer program was written which delivered the stimulus at random intervals between 4 and 8 s after activation. Each stimulus was rated for pain intensity on a 10 cm visual analogue scale (VAS) with 0 indicating no pain and 10 the worst pain possible. In the event that a VAS of 8 or greater was recorded without an NFR being elicited the procedure was abandoned. The EMG signal was acquired with a Neurolog NL900D system (Digitimer, UK) at a sampling frequency of 2500 Hz.

A computer program was written (MATLAB 7.0; Mathworks Inc., USA) that determined the presence of a NFR. A reflex response was defined as per recent recommendations using the standardized peak (NFR interval peak z score) EMG activity from biceps femoris [27]. The NFR interval peak z score is the NFR interval peak (EMG activity 90–150 ms post stimulation interval) – baseline mean (60 ms prior to stimulation)/baseline SD. Rhudy and France [27] suggest a NFR interval peak z score of greater that 10.32 be used to define a reflex response. The 90–150 ms interval was chosen as it avoids possible contamination by low threshold cutaneous flexor reflex, startle reactions and voluntary movements [12]. The two measures taken from this test were (1) the current intensity required to elicit a reflex response and (2) pain intensity (VAS) at this current intensity.

2.5. Questionnaires

2.5.1. Visual analogue scale (VAS)

Participants were asked to rate their level of pain over the last 24 h on a 10 cm VAS scale where 0 = no pain and 10 = worst pain imaginable.

2.5.2. Neck Disability Index

The NDI consists of 10 items addressing functional activities such as personal care, lifting, reading, work, driving, sleeping and recreational activities as well as pain intensity, concentration and headache [42]. There are six potential responses for each item ranging from no disability (0) to total disability (10). The overall score (out of 100) is calculated by totaling the responses of each individual item and multiplying by two. A higher score indicates greater pain and disability [42]. The NDI is a valid, reliable and responsive measure of neck pain and disability [25] and has been frequently used in research of whiplash [34,38].

2.5.3. General Health Questionnaire-28 (GHQ-28)

The General Health Questionnaire-28 (GHQ-28) is a 28-item measure of emotional distress in medical settings [13].

2.5.4. Impact of Events Scale (IES)

The Impact of Events Scale (IES) is a 15-item questionnaire that measures current subjective stress related to a specific life event [15].

2.6. Procedure

All questionnaires and physical measures (sensory tests, NFR) were measured at baseline (≤ 3 weeks post-injury). At subsequent assessments (3 and 6 months post-injury), the NDI and physical measures (sensory tests, NFR) were taken. The participants with whiplash first completed the NDI and then testing was undertaken in the following order: PPT, cold pain thresholds, NFR testing. The same examiner performed all physical tests and was blind to the questionnaire results.

Only the physical tests were performed on the asymptomatic control participants.

2.7. Sample size calculations

Sample size calculations were based on the between group differences in cold and pressure pain thresholds reported in our
previous studies [35,36]. For the NFR data we used pilot data of previously tested 10 participants. The variable with the smallest effect size (0.69 at 80% power and p < 0.05) was PPT measured at Tibialis Anterior indicating that 17 participants/group were required.

2.8. Data analysis

The participants with whiplash with were classified into one of three groups based on results of the NDI at 6 months post-injury. The groups were recovered (NDI: <8%), mild pain and disability (NDI: 10–28%) and moderate/severe pain and disability (NDI: ≥30%) [23,41].

A repeated measures mixed model analysis of variance (ANOVA) with a between subjects factor of Group (four levels: asymptomatic, recovered, mild, moderate/severe) and a within subjects factor of Time (three levels: ≤3 weeks, 3 and 6 months post-injury) was performed for pain threshold and NFR data. Gender was used as a covariate in these analyses. Differences between the groups were analysed with a priori contrasts. Significance was set at p < 0.05.

To determine potential baseline variables associated with NFR threshold at 3 months post-injury, stepwise regression analysis was performed. Baseline variables (VAS, NDI, PPT, cold pain threshold, GHQ-28, IES) entered into the analysis were those with a significant correlation of p < 0.1 with Pearson’s correlation analysis. For the regression analysis, significance was set at p < 0.05.

SPSS 17.0 was used for all analyses.

3. Results

3.1. Participant details

Sixty-seven participants with acute whiplash volunteered to participate. Of these, A NFR could not be elicited from three participants due to cessation of the test when VAS pain scores reached greater than 8/10 (predetermined cut-off). These participants were excluded from further participation in the study. Of the remaining 64 participants, two withdrew from the study after the 3 month assessment, thus the remaining 62 participants completed all three assessments.

The mean (SD) time post accident was 14 ± 6 days. In addition to neck pain, 76% of participants reported headache, 48% upper limb symptoms and 45% dizziness that they attributed to their neck pain. Details of the pain, disability and other questionnaire data for the sample are provided in Table 1. After the accident, the onset of pain was immediate in 34% of subjects, occurred within 24 h in 46% and after 24 h in 20%. Forty-six percent of collisions were rear impact, 22% were front on impact, 21% were combined and 11% were side impact. For ethical reasons treatments could not be withheld from participants. Participants were able to seek whatever treatment they desired with or without any direction or comment from the investigators. At each time point participants were asked of the nature of any treatments received. The most common form of treatment was physiotherapy which was received by 60% of participants – 58% of the recovered group, 60% of the milder group and 60% of the moderate/severe group. Seventy-five percent (n = 47) of the whiplash participants reported taking medication for their condition. Of these, 23 took NSAIDS (7 of the recovered group, 7 of the mild group and 9 of the moderate severe group); 20 took simple analgesics (6 of the recovered group, 5 of the mild group and 9 of the moderate/severe group) and 4 took weak opioid medication (2 in each of the mild and moderate/severe groups). Sixty-one percent of subjects filed a compensation claim.

3.2. Participants classification at 6 months post-injury

Forty percent of the participants with whiplash reported recovery by 6 months post-injury, 28% reported persistent mild pain and disability and 32% persistent moderate/severe pain and disability based on NDI scores at 6 months. The gender distribution, age and baseline scores of VAS, NDI, IES and GHQ-28 are depicted in Table 1.

3.3. Pain thresholds

3.3.1. Pressure pain thresholds

There was a significant main effect for Group (p < 0.05) for all test sites. There was a significant effect for Time (p = 0.03) and a significant interaction effect for Group × Time for the cervical spine site (C5) (p = 0.02) but there was no Time effect (p > 0.05) nor Group interaction for the median nerve (p = 0.11) or tibialis anterior sites (p = 0.89).

The whiplash group with moderate/severe symptoms at 6 months showed lower PPTs at all sites when compared with controls and the other two whiplash groups. PPTs of this group did not significantly change over the study period and remained less than all other groups at 3 months post-injury. The recovered and mild pain groups showed lower PPTs at the cervical spine site (C5) than control participants at entry into the study. However, both these groups improved over time and by 6 months post-injury were no longer different from control subjects (Table 2). There was an effect of gender at all sites (p < 0.02) with females having lower PPTs than males.

3.3.2. Cold pain thresholds

There was a significant main effect for Group (p = 0.0001) but no effect for Time (p = 0.12) nor any interaction between Group × Time (p = 0.95). Cold pain thresholds of the whiplash group with moderate/severe symptoms at 6 months post-injury were higher than the other two whiplash groups and controls at both time points (Fig. 1). There was a significant effect for gender (p = 0.02) with females having lower cold pain thresholds.

3.4. NFR responses

For NFR threshold, there was a significant main effect of Group (p = 0.04), no effect for Time (p = 0.12) but a significant Group × Time interaction (p = 0.05). At the initial assessment point, all

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### Table 1

<table>
<thead>
<tr>
<th>Group</th>
<th>N</th>
<th>Age (years) Mean ± SD</th>
<th>Sex (%) female</th>
<th>VAS Mean ± SD</th>
<th>NDI Mean ± SD</th>
<th>IES Mean ± SD</th>
<th>GHQ-28 mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recovered &lt;8% NDI</td>
<td>25</td>
<td>31.9 ± 12.9</td>
<td>60.8</td>
<td>2.7 ± 1.6</td>
<td>4.9 ± 5.5</td>
<td>26 ± 17.2</td>
<td>26.6 ± 11.6</td>
</tr>
<tr>
<td>Milder pain/disability 10–28% NDI</td>
<td>17</td>
<td>37 ± 11.8</td>
<td>50</td>
<td>3.1 ± 1.5</td>
<td>16.5 ± 9.9</td>
<td>19.9 ± 17</td>
<td>29.3 ± 12.6</td>
</tr>
<tr>
<td>Moderate/severe pain/disability &gt;30% NDI</td>
<td>20</td>
<td>40 ± 13.9</td>
<td>72</td>
<td>5.1 ± 1.7</td>
<td>39.3 ± 17.5</td>
<td>30.2 ± 17.4</td>
<td>45.2 ± 8.4</td>
</tr>
<tr>
<td>Total Whiplash group</td>
<td></td>
<td>36 ± 13</td>
<td>58</td>
<td>3.6 ± 1.9</td>
<td>29.7 ± 16.9</td>
<td>25.8 ± 17</td>
<td>33 ± 14.2</td>
</tr>
<tr>
<td>Controls</td>
<td>22</td>
<td>40 ± 12.6</td>
<td>63.6</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</table>
the whiplash groups showed lower NFR thresholds compared to controls (all p < 0.01) but with no difference between the three whiplash groups (p > 0.2). The NFR threshold of the whiplash groups who recovered or had milder symptoms at 6 months increased and by 3 and 6 months were no different from controls (Fig. 2). There was no effect of gender (p = 0.34) on NFR thresholds. For pain at NFR threshold, there was no main effect for Group (p = 0.69) or Time (p = 0.4) nor any Group x Time interaction (p = 0.35; Table 2). There was no effect of gender (p = 0.69) on pain at NFR threshold.

3.5. Prediction of NFR threshold at 6 months post-injury

Variables that showed a correlation significance of p ≤ 0.1 with NFR threshold at 6 months included: initial pain intensity (VAS) (r = −0.24, p = 0.06); initial pain and disability (NDI) (r = −0.4, p = 0.001) and cold pain threshold (r = −0.34; p = 0.008). When these variables were entered into the multiple linear regression analysis, initial NDI scores were the only significant predictor of NFR threshold at 3 months and explained 24% of the variance of this measure (Table 3).

In this study, four participants (two in each of the milder and moderate/severe groups) took weak opioid medication at some point during the study. In view of the potential effect of opioid medication on NFR thresholds [1], the analyses were rerun with these participants removed. The results were no different; therefore the full data set including these four participants is presented.

4. Discussion

The present study examined the temporal development of central hyperexcitability via NFR responses and sensory hypersensitivity following acute whiplash injury to the period of persistent symptom development. It also examined physical and psychological factors associated with persistent central hyperexcitability. The findings of the study are consistent with previous data demonstrating the early presence of cold and widespread mechanical hyperalgesia in whiplash-injured individuals who go on to develop persistent moderate to severe pain and disability levels [35] with these phenomena not present at any time point in those who recover fully or well after the injury [35]. In contrast central hyperexcitability evidenced by decreased NFR thresholds was present in all whiplash groups in the acute injury stage but persisted only in those with poor functional recovery (moderate or greater pain and disability). Initial high levels of pain and disability was the only significant contributor to persistent central hyperexcitability 6 months post-injury.

Previous studies have utilized the NFR to demonstrate spinal cord hyperexcitability in chronic musculoskeletal and functional pain conditions such as chronic whiplash, fibromyalgia and tension headache [2,9,21]. However, the present study is the first to demonstrate the presence of spinal cord hyperexcitability in an acute musculoskeletal pain condition. NFR thresholds were measured in the lower limb of patients with whiplash (neck pain) thus indicating that the spinal cord hyperexcitability is generalized and may be as a result of disturbed descending modulatory mechanisms. When compared to asymptomatic controls, all whiplash groups were hyperexcitable in the acute (3 weeks) stage of injury irrespective of initial levels of pain and disability or eventual recovery. The mean (±SD) NDI scores of the total sample at the 3 week assessment point were 29.73 ± 16.9 (mild to moderate levels of pain and disability [23]) indicating that a severe injury (in terms of pain and disability levels) is not required in order to induce spinal cord hyperexcitability. These findings are perhaps not unexpected as animal studies have clearly demonstrated profound plastic changes in the spinal cord following tissue damage and ensuing nociceptive input [7]. However in acute whiplash, definitive evidence of tissue damage to peripheral structures is rarely found [30], although this may be more as a result of the failure of available diagnostic methods to adequately identify specific lesions or injuries [32]. The findings of the current study may indicate that peripheral nociception is present following whiplash injury and this may be due to the presence of a peripheral pathology of some kind, the specific nature of which cannot be determined from this study.

NFR responses were no different from asymptomatic controls by 3 months post-injury in the participants with whiplash who fully recovered or reported only persistent mild pain and disability indicating the resolution of spinal cord hyperexcitability in these groups. In contrast, the whiplash group with persistent moderate to severe pain and disability continued to manifest spinal cord hyperexcitability at both 3 and 6 months post-injury. Central hyperexcitability that occurs following tissue injury and ensuing peripheral nociceptive input has been shown to normalize following elimination of such input [14]. It is likely that decreasing pain and disability levels reported by recovering patients reflect decreasing peripheral nociceptive input as injured structures heal and this allows the spinal cord to become less excitable. In those with persistent moderate to severe symptoms, it suggests ongoing nociceptive input presumably from structures that have not healed. Indirect evidence of possible unresolved peripheral lesions in whiplash has been provided from cadaveric studies [39] and clinical studies using MRI [18] and radiofrequency neurotomy of zygapophysial joints [22]. Alternatively it is possible that healing of peripheral structures has occurred in the group with poor recovery and that other processes may be responsible for maintaining the spinal cord hyperexcitability. Animal research has shown the occurrence of potentially irreversible changes in the CNS including death of inhibitory interneurons and gene expression [2] although these findings are yet to be verified in human studies. It is possible that spinal cord plastic changes persist after the resolution of tissue damage but why such irreversible changes would occur only in the whiplash group with poor recovery is perplexing.

Initially higher pain and disability levels (NDI) were the only independent predictor of deceased NFR thresholds at 6 months post-injury. If it is assumed that more intense (and possibly ongoing) nociceptive input is associated with higher reported pain and disability then this finding would support animal studies showing
that more intense or sustained nociceptive input is associated with more profound and long lasting central changes [43]. Additionally patient responses on the NDI may also reflect levels of psychological distress and such distress may influence spinal nociceptive processes. However neither scores on the GHQ-28 (general distress) nor the IES (posttraumatic stress symptoms) predicted persistent central hyperexcitability at 6 months post-injury. Previous data have demonstrated that the psychological substrate of catastrophisation is not associated with NFR thresholds in healthy volunteers [11,28] and in patients [37]. Although the present study did not specifically measure catastrophisation, our data also indicate that psychological factors do not affect spinal processing of nociceptive stimuli. Clinically, this suggests that interventions aimed at decreasing early levels of pain and disability may assist in preventing the maintenance of spinal cord hyperexcitability.

Previous studies have demonstrated the presence of sensory hypersensitivity in the form of lowered pressure and thermal pain thresholds and less cold pain tolerance in the early acute stages following whiplash injury in those with poor recovery but does not occur in those who recover well [20,35]. It has been suggested that the sensory hypersensitivity is indicative of augmented central pain processes and that these processes may be unique to those with poor recovery [35]. It is therefore somewhat surprising that in the current study, decreased NFR thresholds were found in all whiplash groups irrespective of recovery. The differential presence of decreased NFR thresholds and sensory hypersensitivity between whiplash sub-groups suggests that different mechanisms underlie these features. It is recognized the NFR is a measure of spinal nociceptive processes [31]. Widespread decreased pressure pain thresholds have been proposed to reflect a loss of descending inhibition [40] and as such may be more indicative of supraspinal processes. Cold hyperalgesia is emerging as an important factor in WAD. It has been shown to differentiate whiplash sub-groups [26] and to be predictive of poor recovery [36] and non-responsiveness to standard physical interventions [17]. The mechanisms underlying cold hyperalgesia are unclear but proposed mechanisms include sympathetic nervous system dysfunction [44]; changes in properties of peripheral cold-specific fibre terminals following injury [4] and chronic stress [3]. There appears to be some relationship between cold hyperalgesia and psychological distress in both acute [29] and chronic WAD [37]. As both cold hyperalgesia and widespread mechanical hyperalgesia more clearly differentiated, from an early post-injury stage, those who go onto develop chronic pain; it would seem that processes addi-

### Table 3

<table>
<thead>
<tr>
<th>Estimate</th>
<th>Standard error</th>
<th>t value</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Intercept)</td>
<td>12.55</td>
<td>1.13</td>
<td>11.14</td>
</tr>
<tr>
<td>Initial NDI</td>
<td>-0.105</td>
<td>0.034</td>
<td>-3.057</td>
</tr>
<tr>
<td>Cold pain threshold</td>
<td>-0.118</td>
<td>0.071</td>
<td>-1.66</td>
</tr>
<tr>
<td>VAS</td>
<td>0.014</td>
<td>0.363</td>
<td>0.038</td>
</tr>
</tbody>
</table>

Residual standard error: 4.17 on 57 df.
Multiple $R^2$: 0.24, adjusted $R^2$: 0.17.
F-statistic: 9.34 on 1 and 57 df, $p$ value: 0.003.

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![Fig. 1. Cold pain thresholds (mean ± SE) for all groups (controls, recovered, mild pain and disability, moderate/severe pain and disability) over time (<3 weeks, 3 and 6 months post whiplash injury).](image1.png)

![Fig. 2. NFR threshold (mean ± SE) for all groups (controls, recovered, mild pain and disability, moderate/severe pain and disability) over time (<3 weeks, 3 and 6 months post whiplash injury).](image2.png)
tional to spinal cord hyperexcitability contribute to this transition. It is important that future research explore such potential processes.

In summary, whiplash injury induces spinal cord hyperexcitability irrespective of initial symptom levels but this persists only in those with ongoing moderate to severe symptoms. In contrast cold hyperalgesia and widespread pressure hyperalgesia are only ever present in those who develop persistent moderate/severe symptoms. This infers that mechanisms other than spinal cord excitability underlie the sensory changes. Initial pain and disability levels were the only independent predictor of persistent spinal cord hyper excitability and suggest possible ongoing peripheral nociception in those with poor recovery.

Conflict of interest

The author has no conflicts of interest related to this manuscript.

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