International Association for the Study of Pain®

Technical Corner from *IASP Newsletter*
2000 - 3

This section, edited by Michael C. Rowbotham, MD, and Annika Malmberg, PhD, presents timely topics in pain research and treatment.

Pain Following Spinal Cord Injury: Clinical Features, Prevalence, and Taxonomy

Philip J. Siddall,[a] Robert P. Yezierski,[b] and John D. Loeser[c]

[a]Pain Management and Research Centre, University of Sydney, Royal North Shore Hospital, Sydney, Australia; [b]The Miami Project, University of Miami, Florida, USA; [c]Department of Neurological Surgery, University of Washington, Seattle, Washington, USA

Pain is a debilitating accompaniment of spinal cord injury (SCI) that imposes a major burden on patients who have already suffered substantial emotional and physical trauma. Although loss of function is considered the most significant consequence of SCI, pain has a direct bearing on the ability of those with such injuries to regain their optimal level of activity. Results from a postal survey in Britain indicate that for 11% of those responding, it was pain rather than loss of function that prevented them from working [31]. The impact of pain following spinal cord injury is also demonstrated by a report that 37% of SCI patients with high thoracic and cervical lesions and 23% of SCI patients with low thoracic or lumbosacral lesions would be willing to trade pain relief for loss of bladder, bowel, or sexual function [26].

Although SCI pain is a well-recognized problem, there is great disparity in its reported prevalence. In addition, a wide variety of terms and systems are used to classify SCI pain types [32]. This article will review the epidemiology and clinical features of SCI pain and discuss the different classification systems that have been used to define it. We introduce a taxonomy developed by the Task Force on Pain following Spinal Cord Injury of the International Association for the Study of Pain (IASP).

Epidemiology

Prevalence/incidence of SCI pain. Many studies have reported the prevalence of pain in patients with SCI. A summary of results from 10 of these studies indicates that an average of 69% of such patients experience pain and that nearly one-third of those in pain rate their pain as severe [3]. Several more recent studies have confirmed this estimate: a study of 901 patients demonstrated a prevalence of chronic pain of 66% [36], a postal

survey documented a prevalence of 66% [12], and two longitudinal studies revealed a prevalence of 64% at 6 months [34] and 63% at 12 months following discharge from acute hospitalization [17].

The average reported figure for the prevalence of chronic SCI pain is about 65%, with around one-third of those affected reporting severe pain. However, individual studies vary widely. In a review by Bonica [3], prevalence figures varied between 34% [24] and 90% [4], and the percentage of patients in severe pain ranged from 12% [26] to 30% [4]. Similar variation is seen in more recent studies, including a report that incidence of pain during in-patient rehabilitation following SCI was as high as 90% [27].

Relationship of pain to other variables. Although pain is a relatively common problem for SCI patients [3,21,25], the contributing factors are unclear. Several factors may be important, including spinal cord level of injury, cause of injury, completeness of SCI, and psychosocial issues.

Suggestions that injuries at various spinal cord levels, including cervical [14] and thoracolumbar [9] levels and the conus medullaris and cauda equina [4,6,25], are most likely to be associated with pain lack supporting evidence. Damage to the spinal cord from gunshot wounds is also claimed to be more likely to result in pain [25,29]. Clinical observations that neuropathic pain is more common in people with incomplete lesions [2,8] have been supported by findings at autopsy [16].

Specific subtypes of pain may be more common with particular types of injuries. Several case reports have documented spontaneous and evoked segmental neuropathic pain following cervical central cord injuries [11,15,22], with patients often reporting burning pain and hyperesthesia in the arms and hands. The association between incomplete cervical injuries and segmental hyperesthesia is supported by a prospective, longitudinal study in which the only significant association between any physical factor and pain was that allodynia was more common in patients with cervical central cord lesions [34]. Despite these indications of a relationship between physical factors and pain following SCI, other studies have found no significant relationship between the presence or severity of pain and the level or completeness of SCI [28,36,37] and have implicated psychosocial rather than physical factors. Chronic pain is associated with depressive symptoms and greater perceived stress [30]. Cluster analysis of data obtained from questionnaires revealed a relationship between pain, spasticity, "abnormal nonpainful sensations," and sadness [39].

Classification of Pain Types

The wide disparity in reported prevalence of SCI pain and in the relationship of such pain to various other factors may be due to the inherent variability in study design (postal survey, interviews, time following injury, severity of pain, etc.). However, the inconsistency may also stem from the lack of a clear consensus on which types of pain to include and how to classify them. The need for a comprehensive taxonomy of SCI pain has often been cited. The lack of a universal classification system is a major issue that has

hindered effective communication in both research and treatment of this problem. One of the aims of the IASP Task Force on Pain following SCI has been to develop a taxonomy that can be used generally by practitioners and researchers in this field.

Why a new taxonomy? Several classification systems have been used in the past by various authors, but the lack of a universally recognized system has meant little consistency of terminology. Although there are some similarities among studies, many refer to types of pain not included by others. Furthermore, although several broad categories can be identified that have features in common, the terminology used to describe them varies. This lack of consistency in inclusion criteria and terminology has had negative implications for both SCI research and the evaluation of treatments.

Epidemiological studies. Epidemiological studies have been hampered by lack of a universally approved taxonomy. This problem was highlighted by Bonica [3], who found widely disparate reports of the prevalence of SCI pain. Although this disparity may be partly due to methodological issues regarding data collection, it is likely to be mainly due to the variability in the definition and classification of pain types.

Basic research. The need for a consistent taxonomy is apparent in basic research. Numerous animal models of neuropathic pain following SCI present slightly different features [7,13,20,35,38,40]. These models are used to investigate the pathophysiological mechanisms responsible for the development of pain following SCI and to identify therapeutic targets for novel treatment strategies. However, if there are several distinct types of pain following SCI, a clear, universally accepted taxonomy is necessary to identify consistently the different types of pain that are being investigated. Such a system will enable more effective application of research findings to the clinical setting.

Clinical research. Clinical treatment studies and patient management also have been hampered by the lack of a consistent taxonomy. SCI treatment studies often rely on descriptors to indicate pain type, but use ambiguous terms that impede interpretation of results. Even when a particular taxonomy is used, discrepancy across studies can often make comparison and application of the findings difficult. A standardized taxonomy will enhance communication among clinicians and enable more accurate evaluations of specific therapies for different types of pain associated with SCI.

Desirable characteristics for a taxonomy. The IASP Task Force on Pain following SCI has identified a number of characteristics that are desirable in any new classification system. A taxonomy should be comprehensive and able to incorporate any type of pain observed within the defined framework; it should also be systematic and provide a logical structure or framework that is consistent with clinical use and with current concepts of pain types and terminology. It should make classification more "user friendly." To the extent possible, it should be based on mechanisms so that it provides a rational approach to research, assessment, and treatment. Lastly, if a taxonomy is to be useful, it must be consistently applied by those who use it.

Pain Types

In order to develop a taxonomy for SCI that meets the criteria described above, we had to address several issues. A comprehensive system must include most, if not all, types of pain that are generally recognized as being associated with SCI. The terms that have been used in the past are too numerous to list here. Many of them use different words to describe the same types of pain. A summary of the different types of pain associated with SCI is presented below.

Mechanical instability of the spine. This musculoskeletal pain is due to disruption of ligaments or fracture of bones with resultant instability, although it does not require an underlying SCI. It may date from the time of injury or, rarely, may develop later. Pain occurs in the region of the spine and may radiate toward the extremities, but it is not radicular. The condition is characterized by movement of osseous structures in abnormal planes or by abnormal range of motion and is therefore related to position, is increased by activity, and is relieved by immobilization. Radiographs or computerized tomography (CT) or magnetic resonance (MR) imaging will help demonstrate such instability. The pain is usually relieved by opiates and nonsteroidal anti-inflammatory drugs (NSAIDs). Immobilization pending spontaneous healing or surgical fusion is an effective treatment in almost all patients.

Muscle spasm pain. Muscle spasm pain is observed in some cases of complete and incomplete SCI. It usually starts well after the injury and is best relieved by alleviating the muscle spasms. Analgesics are rarely helpful.

Secondary overuse or pressure syndromes occur in normally innervated regions and may be related to overuse. Pain is felt in proximal muscles or in regions such as the shoulder, as in the case of rotator cuff tendonitis. The onset of pain may be delayed for months or many years after injury. The pain is described as aching and regional. It worsens with the use of the affected joint or body part and may be modified by attention to environmental factors such as posture and proper use of wheelchairs. The pain may also be relieved by rest, NSAIDs, and opiates. Nerve compression syndromes may be associated with problems such as carpal tunnel syndrome and can be documented by electrical and MR diagnostic studies. The pain may be relieved by modification of environmental factors, surgical decompression, or use of orthopedic appliances.

Nerve root entrapment may result in lancinating, burning, or stabbing pain in the distribution of a single nerve root, although the pain may be bilateral. The pain occurs at the level of spinal trauma and is usually present from the time of injury. If the affected nerve root contributes to the brachial or lumbosacral plexus, there may be electromyographic and evoked potential abnormalities. Often radiographic, CT, or MR scans reveal compression of a nerve root in the intervertebral foramen by a bone or disk, although the same findings can sometimes be seen in the absence of pain. If the pain is associated with vertebral instability, it can often be relieved by stabilization. The pain may be relieved by opiates and/or by neuropathic pain-relieving drugs. If there is bone or disk material in the foramen, surgical decompression is usually effective.

Pain arising from damage to the cauda equina is a type of nerve root pain with a burning quality that affects the legs, feet, perineum, genitals, and rectum. Although this condition is sometimes considered to be pain of spinal cord origin, it may become important to distinguish cauda equina pain as a neuropathic pain of peripheral nerve origin, given the development of new treatment agents that are directed specifically at peripheral neuropathic pain conditions.

Segmental deafferentation pain. Neuropathic pain often occurs at the border of normal sensation and anesthetic skin and is referred to as girdle, border, or transitional zone pain. The pain occurs within a band of two to four segments and can be unilateral or bilateral and circumferential. Segmental deafferentation pain is often associated with allodynia and hyperalgesia in the painful region. It usually develops during the first few months after injury. The pain does not usually respond to opiates, but may respond to neuropathic pain-relieving medications. It also may be relieved by a number of interventions including epidural or somatic root blocks, dorsal root entry zone (DREZ) lesions, dorsal rhizotomy, spinal cord stimulation, or distal cordectomy to raise the sensory level to the top of the painful region.

Spinal cord injury pain. This type of pain is perceived more diffusely in anesthetic regions below the level of injury and is usually bilateral. It is often referred to as deafferentation pain, dysesthetic pain, or central dysesthesia syndrome. Common descriptors are burning, tingling, numbness, aching, and throbbing. The pain is usually constant and is unrelated to position or activity, but may worsen with concurrent infections. This type of pain is the most difficult to treat and usually responds poorly to opiates. However, it may be relieved by neuropathic pain-relieving drugs. It also may respond to intrathecal opiates and clonidine when the systemic route is ineffective. Such pain does not usually respond to cordectomy or any other ablative procedure and only rarely responds to spinal cord or brain stimulation.

Syringomyelia. A syrinx (an abnormal cavity in the spinal cord) may have a delayed onset, sometimes developing years after the causative injury. Syringomyelia presents with ascending neurologic deficits and pain, often with a region of reduced pain and temperature sensation above a partial or complete lesion. It is diagnosed by MR scan and requires surgical treatment.

Visceral pain. Visceral pain usually has delayed onset following SCI and is indicated by burning, cramping, and constant but fluctuating pain in the abdomen. It may be due to normal afferent input via the sympathetic or vagal nerves in paraplegics and via the vagus nerve in tetraplegics [18,19]. Visceral pain is often poorly defined and may occur in the absence of any demonstrable visceral pathology.

Cognitive, affective, and environmental factors may be superimposed upon any of the above conditions and can be major factors in disability and great impediments to successful rehabilitation. Repeated references in the literature attest to the role of such factors in the genesis of pain behaviors and pain-associated disability following SCI.

These factors can be addressed by psychological management strategies, regardless of the type of pain.

A System for Classification

The above section provides a comprehensive list of the different types of pain that are commonly seen following SCI. However, as mentioned above, one of the desirable characteristics of a taxonomy is that it should be systematic. Several systems have been proposed based on location, descriptors, and pathology, or a mixture of these factors [1,5,10,33]. In common with the IASP taxonomy [23], some of these systems have several tiers to allow further definition. Dividing a taxonomy into tiers provides a structure that may aid clinical assessment, identification of mechanisms, and treatment. Our proposed taxonomy organizes the pain types described above into a three-tiered structure. Each tier defines the affected structure and the pathology responsible for the pain.

The first, very broad tier simply divides pain into nociceptive and neuropathic types. These categories are usually distinguishable to a degree of certainty on the basis of location and patient descriptors and have the strongest implications for any management approaches. Nociceptive pain is usually described as dull, aching, and cramping, and occurs in a region of sensory preservation. Neuropathic pain is usually described as sharp, shooting, electric, or burning, and occurs in a region of sensory disturbance, i.e., increased or decreased sensibility. The second tier provides further definition of these broad pain types and provides further direction for treatment. Nociceptive pain is divided into musculoskeletal and visceral pain types, and neuropathic pain is divided into "above-level," "at-level," and "below-level" types, where "level" refers to the level of the spinal cord that was injured. The second tier of SCI pain types is found in Table 1.

Table 1. Tier Two Groupings of Pain Related to Spinal Cord Injury

Term	Distinguishing Features
Musculoskeletal	Dull, aching, movement-related, eased by rest, responsive to opioids and NSAIDs Located in musculoskeletal structures
Visceral	Dull, cramping Located in abdominal region with preserved innervation Also includes dysreflexic headache (vascular)
Neuropathic	Sharp, shooting, burning, electric abnormal responsiveness (hyperesthesia, hyperalgesia)
Above level	Located in the region of sensory preservation
At level	Located in segmental pattern at the level of injury
Below level	Located diffusely below the level of injury

The third tier of classification provides further refinement in terms of a specific structure and pathology and therefore more closely identifies a possible mechanism as well as

implications for treatment. For example, musculoskeletal pain includes pain that may be due to muscle spasm, bone trauma, or inflammation around a joint. At-level neuropathic pain includes pain due to nerve root damage (including damage to the cauda equina), syringomyelia, or spinal cord trauma.

Psychological factors are not included in the taxonomy as a type of pain. Cognitive, affective, and environmental factors are often superimposed upon any of the pain types following SCI and should always be considered as contributing factors. However, the task force believes that psychological factors should be considered as part of any pain syndrome and not as a specific pain type.

What terms should be used to identify these pain types? Once the items to be included are identified, the next step is to decide on terminology. This choice is influenced by several factors including previous use, familiarity, "correctness," and the system that is to be used. In the tier one division, the terms *nociceptive* and *neuropathic* are recommended. This familiar "first pass" classification is in common clinical usage and simplifies assessment and treatment. Nociceptive pain is pain arising from stimulation of somatic or visceral nociceptors. The term *neuropathic* is suggested, rather than *neurogenic,* to be consistent with the IASP taxonomy [23], which limits the use of *neurogenic* to pain due to a transitory perturbation of the nervous system.

In the second tier, nociceptive pain is divided into *musculoskeletal* and *visceral* pains, terms that also are in common use. The division of neuropathic pain into types based on site permits more detailed definition. Site of pain relative to the level of injury may be the only available option for further division, based on our current understanding of mechanisms. The pathology (spinal cord damage) associated with neuropathic SCI pain appears to be the same in patients with "at-level" or "below-level" pain, but the clinical presentation is different. The most identifiable characteristic of these two types of pain is their location. SCI pain or dysesthetic pain is located diffusely below the level of injury. On the other hand, pains arising from the nerve root, segmental deafferentation, and syringomyelia are located in dermatomes adjacent to the level of injury. Therefore, if it is difficult to distinguish these types of pain on the basis of specific pathology, we suggest simply referring to them as *at-level* or *below-level* neuropathic pain.

The primary objective within the third tier is to identify a specific structure and pathology that may be generating pain. To help meet this objective, we have avoided using syndromes as terminology.

Use of the Proposed Taxonomy

The three tiers (Table 2) provide a structure for grouping different types of pain and provide some direction in assessment and management of SCI pain. While the first tier provides a general direction in assessment and treatment, we anticipate that most SCI pains will be classified at least at the second tier (musculoskeletal pain; visceral pain; and above-level, at-level, and below-level neuropathic pain).

Table 2. Proposed Classification of Pain Related to Spinal Cord Injury

Broad Type (Tier 1)	Broad System (Tier 2)	Specific Structures/Pathology (Tier 3)
Nociceptive	Musculoskeletal	Bone, joint, muscle trauma or inflammation. Mechanical instability. Muscle spasm. Secondary overuse syndromes
	Visceral	Renal calculus, bowel, sphincter dysfunction, etc. Dysreflexic headache
Neuropathic	Above level	Compressive mononeuropathies. Complex regional pain syndromes
	At level	Nerve root compression (including cauda equina) Syringomyelia Spinal cord trauma/ischaemia (transitional zone, etc.) Dual level cord and root trauma (double lesion syndrome)
	Below level	Spinal cord trauma/ischemia (central dysesthesia syndrome, etc.)

We hope that in many SCI patients, it will be possible to identify the structure and pathology (third tier) responsible for the generation of pain. We believe that more specific identification of structures and pathology will further define mechanisms and allow more effective treatment. We do not intend that the terminology in all three tiers should be used together clinically, but that the tier with the highest degree of definition should be used. For example, pain due to muscle spasm should be referred to as muscle spasm pain rather than as nociceptive musculoskeletal/muscle spasm pain. If it is not possible to accurately identify a structure, then a second-tier term such as *visceral* or *at-level* neuropathic pain can be used.

Conclusions

Pain is a common problem that has a major impact on patients with SCI. Several types of pain may occur, which fall broadly into nociceptive and neuropathic pain categories. These can be divided into musculoskeletal pain; visceral pain; and above-level, at-level, and below-level neuropathic pains. As a last step, they can be further classified depending on the specific structures and pathology involved. The IASP Task Force on Pain following SCI offers this taxonomy in the hope that it will result in better communication that will ultimately improve both research and treatment. We present this taxonomy with the hope that emerging knowledge about the clinical characteristics and mechanisms of pain following SCI will further improve the classification of such pain.

Acknowledgments

We acknowledge the helpful contributions from members of the IASP Task Force on Pain following Spinal Cord Injury and specifically Drs. Aleksandar Berić, Frederick Lenz, Charles Vierck, Eva Widerström-Noga, and Kim Burchiel, as well as the helpful suggestions and comments of Prof. Michael Cousins and Drs. David Taylor, James Middleton, Sue Rutkowski, William Donovan, and Elliot Roth.

References:

1. Berić A. Spinal cord damage: injury. In: Wall PD, Melzack R (Eds). Textbook of Pain. London: Churchill Livingstone, 1999, pp 915-927.
2. Berić A, Dimitrijevic MR, Lindblom U. Central dysesthesia syndrome in spinal cord injury patients. Pain 1988; 34:109-116.
3. Bonica JJ. Introduction: semantic, epidemiologic, and educational issues. In: Casey KL (Ed). Pain and Central Nervous System Disease: The Central Pain Syndromes. New York: Raven Press, 1991, pp 13-29.
4. Botterell EH, Callaghan JC, Jousse AT. Pain in paraplegia: clinical management and surgical treatment. Proc R Soc Med 1953; 47:281-288.
5. Bryce TN, Ragnarsson KT. Pain after spinal cord injury. Phys Med Rehabil Clin N Am 2000; 11:157-168.
6. Burke DC. Pain in paraplegia. Paraplegia 1973; 10:297-313.
7. Christensen MD, Everhart AW, Pickelman JT, Hulsebosch CE. Mechanical and thermal allodynia in chronic central pain following spinal cord injury. Pain 1996; 68:97-107.
8. Davidoff G, Roth E, Guarracini M, Sliwa J, Yarkony G. Function-limiting dysesthetic pain syndrome among traumatic spinal cord injury patients: a cross-sectional study. Pain 1987; 29:39-48.
9. Davis L, Martin J. Studies upon spinal cord injuries. J Neurosurg 1947; 4:483-491.
10. Donovan WH, Dimitrijevic MR, Dahm L, Dimitrijevic M. Neurophysiological approaches to chronic pain following spinal cord injury. Paraplegia 1982; 20:135-146.
11. Felling A. Central pain in spinal cord lesions. Proc R Soc Med 1937; 31:39-48.
12. Fenollosa P, Pallares J, Cervera J, et al. Chronic pain in the spinal cord injured: statistical approach and pharmacological treatment. Paraplegia 1993; 31:722-729.
13. Hao JX, Xu XJ, Aldskogius H, Seiger A, Wiesenfeld-Hallin Z. Allodynia-like effects in rat after ischaemic spinal cord injury photochemically induced by laser irradiation. Pain 1991; 45:175-185.
14. Holmes G. Pain of central origin. In: Anonymous. Contributions to Medical and Biological Research, Vol. 1. New York: P.B. Hoeber, 1919, pp 235-246.
15. Hopkins A, Rudge P. Hyperpathia in the central cervical cord syndrome. J Neurol Neurosurg Psychiatry 1973; 36:637-642.
16. Kakulas BA, Smith E, Gaekwad U, Kaelan C, Jacobsen PF. The neuropathology of pain and abnormal sensations in human spinal cord injury derived from the clinicopathological data base at the Royal Perth Hospital. In: Dimitrijevic MR, Wall PID, Lindblom U (Eds). Altered Sensation and Pain. Recent Achievements in Restorative Neurology, Vol. 3. Basel: Karger, 1990, pp 37-41.
17. Kennedy P, Frankel H, Gardner B, Nuseibeh I. Factors associated with acute and chronic pain following traumatic spinal cord injuries. Spinal Cord 1997; 35:814-817.
18. Komisaruk BR, Gerdes CA, Whipple B. 'Complete' spinal cord injury does not block perceptual responses to genital self-stimulation in women. Arch Neurol 1997; 54:1513-1520.
19. Kuhn RA. Functional capacity of the isolated human spinal cord. Brain 1950; 73:1-51.
20. Levitt M, Levitt JH. The deafferentation syndrome in monkeys: dysaesthesias of spinal origin. Pain 1981; 10:129-147.
21. Mariano AJ. Chronic pain and spinal cord injury. Clin J Pain 1992; 8:87-92.
22. Maroon JC. 'Burning hands' in football spinal cord injuries. JAMA 1977; 238:2049-2051.
23. Merskey H, Bogduk N. Classification of Chronic Pain, 2nd ed. Seattle: IASP Press, 1994.
24. Munro D. Two-year end-results in the total rehabilitation of veterans with spinal cord and cauda-equina injuries. N Engl J Med 1950; 242:1-10.
25. Nashold BS. Paraplegia and pain. In: Nashold BS, Ovelmen-Levitt J (Eds). Deafferentation Pain Syndromes: Pathophysiology and Treatment. New York: Raven Press, 1991, pp 301-319.

26. Nepomuceno C, Fine PR, Richards JS, et al. Pain in patients with spinal cord injury. Arch Phys Med Rehabil 1979; 60:605-609.
27. New P. A survey of pain during rehabilitation after acute spinal cord injury. Spinal Cord 1997; 35:658-663.
28. Richards JS, Meredith RL, Nepomuceno C, Fine PR, Bennett G. Psycho-social aspects of chronic pain in spinal cord injury. Pain 1980; 8:355-366.
29. Richards JS, Stover SL, Jaworski T. Effect of bullet removal on subsequent pain in persons with spinal cord injury secondary to gunshot wound. J Neurosurg 1990; 73:401-404.
30. Rintala D, Loubser PG, Castro J, Hart KA, Fuhrer MJ. Chronic pain in a community based sample of men with spinal cord injury: prevalence, severity, and relationship with impairment, disability, handicap, and subjective well-being. Arch Phys Med Rehabil 1999; 79:604-614.
31. Rose M, Robinson JE, Ells P, Cole JID. Pain following spinal cord injury: results from a postal survey. Pain 1988; 34:101-102.
32. Roth EJ. Pain in spinal cord injury. In: Yarkony GM (Ed). Spinal Cord Injury: Medical and Rehabilitation Management. Gaithersburg, MD: Aspen, 1994, pp 141-158.
33. Siddall PJ, Taylor DA, Couaft WIJ. Classification of pain following spinal cord injury. Spinal Cord 1997; 35:69-75.
34. Siddall PJ, Taylor DA, McClelland JM, Rutkowski SB, Cousins MJ. Pain report and the relationship of pain to physical factors in the first six months following spinal cord injury. Pain 1999; 81:187-197.
35. Siddall PJ, Xu CL, Cousins MJ. Allodynia following traumatic spinal cord injury in the rat. Neuroreport 1995; 6:1241-1244.
36. Störmer S, Gerner HJ, Gröninger W, et al. Chronic pain/dysaesthesiae in spinal cord injury patients: results of a multicentre study. Spinal Cord 1997; 35:446-451.
37. Summers JD, Rapoff MA, Varghese G, Porter K, Palmer RE. Psychosocial factors in chronic spinal cord injury pain. Pain 1991; 47:183-189.
38. Vierck CJ, Light AR. Effects of combined hemotoxic and anterolateral spinal lesions on nociceptive sensitivity. Pain 1999; 83:447-457.
39. Widerström-Noga EG, Felipe-Cuervo E, Broton JG, Duncan RC, Yezierski RP. Perceived difficulty in dealing with consequences of spinal cord injury. Arch Phys Med Rehabil 1999; 80:580-586.
40. Yezierski RP, Liu S, Ruenes GL, Kajander KJ, Brewer KL. Excitotoxic spinal cord injury: behavioural and morphological characteristics of a central pain model. Pain 1998; 75:141-155.

Correspondence to: Dr. P.J. Siddall, Pain Management and Research Centre, Royal North Shore Hospital, St Leonards, NSW 2065, Australia. Tel.: +61-2-9926-6387, +61-2-9926-6548; email: phils@med.usyd.edu.au.