INTRODUCTION

Although traumatic SCI results in a number of serious impairments including paralysis, sensory loss, and neurogenic bowel/bladder function, perhaps no SCI-associated condition is more vexing to the treating physiatrist than chronic pain. Some of these SCI-related impairments can be accommodated with compensatory strategies, whereas chronic pain, especially neuropathic pain associated with injury to the spinal cord, remains quite recalcitrant. In addition to the expected challenges in treating any chronic pain condition, treatment of SCI-related pain has the difficulty of disruption of normal neural pathways that subserve pain transmission and attenuation. This article attempts to describe the classification, epidemiology, evaluation methods, and treatment strategies for this serious pain syndrome.

CLASSIFICATION

Before 2000, there was no consistent approach to the classification of SCI-related chronic pain. This variability was described by Hicken and colleagues\(^1\) during a review in 2002 in which 29 distinct schemes were described with potentially confusing and inconsistent terminology. By 2008, 3 classification systems emerged as the leading systems based on their utility, comprehensiveness, validity, and reliability. These schemes included the Cardenas classification,\(^2\) the taxonomy of the International
Association for the Study of Pain,\textsuperscript{5} and the Bryce-Ragnarsson classification.\textsuperscript{4} Through the leadership of Bryce, a unified system was created and published in 2011. The International Spinal Cord Injury Pain Classification (ISCIP) has been adopted by many leading SCI and pain professional associations throughout the world.\textsuperscript{5} This classification is visually depicted in Fig. 1.

Given the probable ubiquity of the ISCIP classification, some commentary on this approach is warranted. The first tier of this system is divided into nociceptive, neuropathic, other, and unknown categories. The distinction between the nociceptive and neuropathic categories is certainly approximate because the treatment approaches to these syndromes are often vastly different. As discussed later in this article, nociceptive pain can often be addressed by classic physiatric techniques (in the case of musculoskeletal pain) or other medical interventions (in visceral and other nociceptive pain). This fact is in contradistinction to neuropathic pain in which many treatment approaches are either pharmacologic or interventional. The ISCIP classification also demonstrates the continued difficulties of even expert clinicians and scientists to categorize every single pain condition associated with the SCI population, as demonstrated by the other and unknown categorizations. The reliability of the ISCIP

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<tr>
<th>Tier 1: pain type</th>
<th>Tier 2: pain subtype</th>
<th>Tier 3: primary pain source and/or pathology (write or type in)</th>
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<tr>
<td>Nociceptive pain</td>
<td>Musculoskeletal pain</td>
<td>e.g., glenohumeral arthritis, lateral epicondylitis, comminuted femur fracture, quadratus lumborum muscle spasm</td>
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<td>Visceral pain</td>
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<td>e.g., myocardial infarction, abdominal pain due to bowel impaction, cholecystitis</td>
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<tr>
<td>Other nociceptive pain</td>
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<td>e.g., autonomic dysreflexia headache, migraine headache, surgical skin incision</td>
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<td>Neuropathic pain</td>
<td>At-level SCI pain</td>
<td>e.g., spinal cord compression, nerve root compression, cauda equina compression</td>
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<td>Below-level SCI pain</td>
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<td>e.g., spinal cord ischemia, spinal cord compression</td>
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<td>Other neuropathic pain</td>
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<td>e.g., carpal tunnel syndrome, trigeminal neuralgia, diabetic polyneuropathy</td>
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<td>Other pain</td>
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<td>e.g., fibromyalgia, complex regional pain syndrome type i, interstitial cystitis, irritable bowel syndrome</td>
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<td>Unknown pain</td>
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classification has undergone initial testing using a clinical vignette approach by clinicians experienced with SCI who received minimal training in use of the system. The correctness levels varied from 65% to 85% based on the degree of correlation strictness for the various responses. This result confirms the difficulty in assessment and classification of SCI-associated pain.

The relationship of spasticity to pain is complex. Spasticity can limit the range of motion about a joint and result in musculoskeletal pain. Reduction of spasticity may reduce the pain associated with biomechanical pain. However, as noted above, SCI can also produce neuropathic pain. Modulation of spasticity may not be effective in reducing neuropathic pain. It is also relevant to note that some interventions to treat spasticity may also modulate the pain transmission pathways. In addition, the sensory loss in many patients with SCI, especially those with American Spinal Injury Association impairment scale A neurologic levels, may eliminate or substantially reduce the pain responses associated with noxious events. Increased spasticity may be the only harbinger of these events.

EPIDEMIOLOGY

Given the classification ambiguity of chronic SCI-related pain described above, attempts at epidemiology can be problematic. Other potential confounders include oversampling because patients may have more than one pain syndrome, adequate pain description, criteria used for chronicity and severity, traumatic versus nontraumatic differentiation, and appropriate inclusion/exclusion criteria. Dijkers and colleagues executed a review consisting of 42 articles that described the epidemiology of this clinical problem. A wide variance was noted in the literature, with prevalence of SCI-associated pain in the literature varying from 26% to 96% without an apparent clustering around any group of percentages. The quality of the individual study did not seem to significantly influence the reported prevalence rate. More detailed analysis failed to demonstrate appreciable difference in SCI-associated pain prevalence related to gender, injury completeness, or paraplegia versus tetraplegia. Some individual studies have reported trends for these demographic items, but when viewed from the perspective of the entirety of the medical literature, these trends disappear. Pain conditions among individuals with SCI are generally stable over time. Emergence or dramatic change in a chronic pain condition is worthy of new evaluative approach.

EVALUATION

The approach to SCI pain should commence in a manner similar to all chronic pain conditions—history, physical examination, and judicious use of diagnostic testing. Information should be obtained regarding the patient’s initial SCI including date, mechanism of injury, associated injuries such as long bone and visceral trauma, description of vertebral column stabilization procedures, and comorbidities of the acute hospitalization and rehabilitation phase of injury. Descriptors should be attained regarding pain history, including time of onset from initial injury, time course, pain location, intensity and quantity, alleviating and aggravating factors, past evaluations, treatments (including effectiveness), and pharmacologic assessment. Inquiry into the presence or change in upper motor neuron signs, such as clonus or spasticity, is reasonable. Functional, occupational, and recreational history should be acquired for 2 reasons. First, these activities may contribute to the development of pain (e.g., development of shoulder pain in a wheelchair athlete). Second, the degree of pain interference with these activities will allow the clinician to judge the functional impact of the
patient’s pain condition. Some degree of psychological assessment is warranted with exploration into possible depression, anxiety, personality disorders, concomitant brain injury, substance use, and cognitive impairment. In selected cases, a more formal psychological assessment, including psychometric testing, by either a psychologist or a psychiatrist may be appropriate. Last, the patient should be queried as to what diagnostic tests have been undertaken previously.

Although not an absolute “red flag,” emergence of below-level pain after years or even decades from the initial SCI should be viewed as a concerning sign. For above-level pain syndromes, the typical elements of history should be queried as for the non-SCI patient with added elements that are pertinent to the patient with SCI. A reasonable example of this approach is a patient with SCI who presents with a suspected carpal tunnel syndrome. The patient should be asked questions about sensory symptoms and their distribution with the added elements of wheelchair, crutch, or cane use because use of these devices could be a contributing factor to a suspected median neuropathy. Particular attention should be paid to treatment failures. Some patients may have been exposed to a particular agent but were not given sufficient time or dose that would reasonably be expected to result in a therapeutic response. Other patients may have discontinued use of a medication because of intolerable adverse effects. If these 2 scenarios are present, insufficient response might be overcome with either a rechallenge of prior medications or use of adjuvant therapies to manage side effects.

Pain assessment should have a component of patient self-report. These measures supplement information obtained during the clinical interview and provide a means of evaluating success or failure of treatment strategies. The most commonly used measure of pain, for all types of pain, is the numerical rating scale (NRS). An NRS includes a range of numbers, generally starting from 0 (eg, 0–10 or 0–100), which is anchored to descriptors, for example, no pain at the lowest extreme of the range and worst pain imaginable at the highest extreme. Several studies have established the NRS as a reliable measure of pain intensity. Another typical measure of pain intensity is a visual analog scale, which consists of a line (horizontal or vertical) anchored at either extremes with no pain on one end and another extreme (eg, worst pain imaginable) on the other end. Respondents are instructed to draw a small line that intersects with the scale at the point that represents their pain intensity. The measured distance (eg, in millimeters) from the no pain anchor to the recorded mark represents the subject’s pain intensity. Typically, a clinically meaningful change in pain intensity is approximately a 33% decrement in visual analog score (VAS).

Beyond pain intensity, it is reasonable to attempt assessment of pain according to daily activities. Of note, the Initiative on Methods, Measurement, and Pain Assessment in Clinical Trials (IMMPACT) group recommended that measures of pain severity, physical functioning, and emotional functioning be included in all clinical trials of chronic pain interventions. The impact of pain on physical functioning may be obtained through a number by pain interference scales such as the Graded Chronic Pain Disability scale, the Brief Pain Inventory, and the Multidimensional Pain Inventory. These scales have demonstrated reasonable reliability and validity in SCI populations.

Many patients with chronic pain disorders and SCI have comorbid psychological disorders including depression, anxiety, anger, psychosis, eating disorders, substance dependence, cognitive impairment, and personality disorders. The physician should inquire about the existence and severity of these behavioral problems. Cotreatment with mental health professionals is often warranted for more in-depth neuropsychological assessments. Examples of standardized psychological assessments used
in these populations include the Beck Depression Inventory and the Patient Health Questionnaire. The latter has demonstrated validity in the SCI population.\textsuperscript{14}

The physical examination of the individual with SCI-associated pain should start with the International Standards for Neurologic Classification of Spinal Cord Injury neurologic evaluation.\textsuperscript{15} This examination is supplemented by further neurologic testing including reflex testing, assessment of other sensory abnormalities (allodynia, hyperalgesia, and hyperpathia), and evaluation of muscle overactivity (spasms, spasticity, and clonus). Focal examination of a particular pain area would then proceed as a neuromusculoskeletal approach used for pain complaints in all populations. Items to be included are inspection, palpation, active and passive range of motion, and provocative maneuvers. Observation of wheelchair propulsion, posture, and gait may be appropriate in selected patients. Appropriate comfort and fit of assistive devices (cane, walker, and crutch) and orthotic devices should be undertaken if these equipment seem to contribute to the pain syndrome. A survey of mood, behavior, personality, and cognition is certainly reasonable.

Regarding diagnostic testing, above-level syndromes can be evaluated in a manner parallel to the non-SCI patient. Conditions associated with at-level and below-level lesions are more challenging. Imaging of the site of initial spinal region should be considered in these circumstances. Potential examples of pain generators that might be detected include segmental instability or compression about the site of injury, spinal nerve impingement, orthopedic hardware loosening, fluid collections, and syringomyelia. Discussion with the interpreting radiologist is recommended to assist with the choice of imaging modalities. Potential discussion points could include interference of hardware, the need for radiographic contrast (intravenous gadolinium for magnetic resonance imaging [MRI], subarachnoid ionic contrast for computed tomographic [CT] myelogram, etc), and the differentiation of acute from chronic changes. Given the possible unreliability of abdominal/pelvic examinations in an insensate patient, imaging may also be warranted if visceral pain is suspected. In addition to the traditional MRI and CT modalities, specialized techniques may be warranted for potential pain generator relative to neurogenic bowel and bladder (ie, colonoscopy, cystoscopy, urodynamics testing, etc). Triple-phase bone scanning could be appropriate for evaluation of unsuspected fractures or complex regional pain syndrome.\textsuperscript{16}

Judicious use of laboratory testing follows a parallel pathway for above-level syndromes and a surveillance approach for at-level and below-level syndromes. Care must be taken with regard to interpretation so as not to “over read” the importance of particular abnormality. An example of this pitfall would be to attribute asymptomatic urinary bacterial colonization as the sole cause of visceral pain. Potential laboratory tests in this population might include a complete blood cell count, erythrocyte sedimentation rate/C-reactive protein levels (to trend an inflammatory process such as abscess), and hormonal assessment (including pregnancy testing). Subtherapeutic vitamin B\textsubscript{12} and Vitamin D levels\textsuperscript{18} have been implicated in neuronal dysfunction in SCI and represent potentially reversible abnormalities.

**MANAGEMENT**

**Nonpharmacologic**

A generalized exercise program in the form of global strength training, cardiovascular training, or recreational physical activities has the potential to be beneficial for several SCI-related conditions (eg, spasticity, muscle atrophy, bone health), but its effect on global pain in this population has not been greatly satisfactory. Animal studies have suggested that antinociceptive behaviors can be reduced with weeks of exercise
training. Extrapolation from these experiments to the human condition has not been straightforward. Some human trials suggest that a long-term exercise program can attenuate global pain complaints, but these effects may not persist if regular exercise is discontinued. More targeted exercise programs for specific pain complaints have a much higher likelihood of success. The best example of this approach is seen with shoulder pain in paraplegic individuals.

In addition to generalized and specified exercise programs, referral to physical or occupational therapy may be appropriate for the patient with SCI with musculoskeletal pain in an effort to address biomechanical abnormalities that can be associated with mobility aids. Modification of orthotics, canes, walkers, crutches, and wheelchairs has the potential to influence detrimental ergonomics. Perhaps the best example of this intervention is adjustment of rear wheel of a manual wheelchair in an effort to modify the forces about the shoulder that can occur as a result of wheelchair propulsion.

Acupuncture is popular for both the general and SCI populations. In 1997, a report from the National Institutes of Health supported the use of acupuncture for certain conditions, including pain. Survey assessments have reported that between 15% and 35% of individuals with SCI have tried acupuncture for pain relief with a variable degree of effectiveness. One retrospective review reported that two-thirds of patients treated with acupuncture for below-level neuropathic pain found it effective. Support for acupuncture from prospective studies is limited. Nayak and colleagues reported that approximately half of the patients who received 15 sessions of this modality experienced a clinically meaningful reduction in pain. This study suggested that acupuncture may be more effective in individuals with incomplete injuries or musculoskeletal pain when compared with complete injuries or neuropathic pain. Dyson and colleagues reported that both acupuncture and sham acupuncture groups had reductions in pain ratings when exposed in a double-blind manner.

Pharmacologic

At present, there is only one medication that currently has US Food and Drug Administration (FDA) indication for SCI-associated pain. Pregabalin is a structural derivative of the inhibitory neurotransmitter γ-aminobutyric acid. Pregabalin is an alpha-2-delta ligand that has analgesic, anticonvulsant, anxiolytic, and sleep-modulating activities. Pregabalin binds potently to the alpha-2-delta subunit of voltage-gated calcium channels. It is hypothesized that this binding reduces the influx of calcium into hyperexcited neuron, which in turn results in a reduction in the release of several neurotransmitters, including glutamate, noradrenaline, serotonin, dopamine, and substance P. Siddall and colleagues randomized 137 patients with SCI to either placebo (67 patients) or flexible dosing of pregabalin (70 patients). Roughly half of the patients had complete injuries. The dosing ranged from 150 to 600 mg/d in 2 divided doses. The mean baseline pain score was 6.54 in the pregabalin group and 6.73 in the placebo group. The mean endpoint pain score was lower in the active treatment group (4.62) compared with that in the placebo group (6.27; \( P < .001 \)), with efficacy observed as early as week 1 and maintained for the duration of the study (12 weeks). The average pregabalin dose after the 3-week stabilization phase was 460 mg/d. During this trial, pregabalin was associated with improvements in disturbed sleep and anxiety. The most common adverse events were mild or moderate, typically transient, somnolence and dizziness. Edema was reported in 20% of the pregabalin patients, which resulted in 3 discontinuance episodes, compared to 6% in the placebo group, which resulted in 2 discontinuance episodes. Cardenas and colleagues executed a similar study with 230 patients with 108 patients receiving active drug and 112 patients receiving placebo. This study had a longer duration (16 weeks). Approximately half of the patients
had complete injuries. Pregabalin-treated patients experienced a nearly 2-point improvement on a 10-point NRS scale in the intensity of SCI-related pain during the previous 24 hours compared to baseline levels. Similarly, the patients experienced an improvement in disturbed sleep and sleep interference. The average daily dose of pregabalin was 410 mg/d during the dose maintenance period and 357 mg/d over the full treatment period. The most frequent treatment-related adverse effects included somnolence, dizziness, edema, dry mouth, fatigue, and blurred vision. Most adverse effects were mild to moderate in severity and transient in nature. Treatment-emergent peripheral edema was reported in 13.4% of pregabalin-treated patients in this study, resulting in 1 discontinuation. The incidence of edema in this study, as well as the previous study, was comparable with the reported incidence of other neuropathic pain conditions including diabetic peripheral neuropathy and postherpetic neuralgia. Thus, patients with SCI do not seem more susceptible to developing peripheral edema in response to pregabalin than patients with other neuropathic pain conditions.

Before pregabalin release and also at present, gabapentin remains commonly used. Similar to pregabalin, gabapentin is active at voltage-gated calcium channels. This agent has been considered effective in SCI-associated neuropathic pain in several smaller studies. Levendoglu and colleagues reported that gabapentin was more effective than placebo in a crossover study involving 20 patients with paraplegia with neuropathic pain that had been present for more than 6 months. Tai and colleagues conducted a prospective, randomized, double-blind, crossover study on 7 patients who had for more than 30 days postinjury SCI-related pain. This study found a significant reduction in “unpleasant feelings” with gabapentin compared to placebo. Reduction in pain intensity and burning pain trended toward significance, whereas no differences were observed for other pain descriptors. To and colleagues performed a retrospective chart review of 44 patients with SCI-related neuropathic pain examining the effectiveness of gabapentin. About 76% of these subjects reported a reduction in pain intensity. The mean pretreatment VAS was 8.86, which decreased to 4.13 after 6 months of treatment. Last, Putzke and colleagues examined the use of gabapentin in this population with a longitudinal observational study on 27 patients. This group observed a relatively high discontinuation rate (6/27 or 22%). Of the remaining 21 patients, 14 (67%) reported a greater than 2-point reduction in VAS at 6 months. It is reasonable to conclude that gabapentin can be effective in neuropathic pain in SCI-associated neuropathic pain. A recent study of intrathecal gabapentin failed to demonstrate any benefit in an unselected chronic pain population despite promising results from animal data, particularly with neuropathic pain. Although pregabalin has US FDA approval for SCI-related pain, there is no head-to-head trial comparing the effectiveness of gabapentin with that of pregabalin. The affordability of gabapentin may make it the more desirable choice. Many insurers require a trial of gabapentin before approving pregabalin.

The use of antidepressants for below-level neuropathic pain SCI pain has a longstanding tradition. The substantial benefit of tricyclic antidepressants (TCAs) in neuropathic pain has led to this use. Perhaps the most commonly used agent is amitriptyline. There are conflicting results in the medical literature with some studies demonstrating efficacy and other studies demonstrating descriptive minimal efficacy in SCI-associated pain. One comparison trial described a therapeutic benefit of amitriptyline over gabapentin. The so-called second-generation TCAs (ie, secondary amines such as nortriptyline, desipramine, and protriptyline) are preferred because analgesic efficacy is equivalent and tolerability is better compared to those of first-generation TCAs (ie, tertiary amines such as amitriptyline, clomipramine, and
doxepin). All TCAs are considered to have a ceiling effect. Thus, once a therapeutic effect is achieved, further dosing increases should be avoided in order to minimize adverse effects.42

The most recent additions to antidepressant use for chronic pain are the dual serotonin and norepinephrine reuptake inhibitors. Medications in this class include duloxetine, milnacipran, and desvenlafaxine. Pain modulation seems to be independent of their antidepressant properties. Duloxetine, the first medication approved for use in the United States within this class, has FDA indication for chronic musculoskeletal pain, fibromyalgia, and diabetic neuropathy. A small trial of duloxetine for central neuropathic pain caused by either stroke or SCI failed to show a reduction in pain intensity but did demonstrate changes in other aspects of these chronic pain syndromes, including allodynia.43 There are no reports of using either milnacipran or venlafaxine for chronic SCI-associated pain. Several serotonin-norepinephrine reuptake inhibitors are in various stages of clinical development for a wide variety of indications.44

Opioid medications have been suggested as reasonable options for chronic nociceptive and perhaps neuropathic pain. Perhaps, no other decision in medicine causes more anxiety than prescribing opiates for patients with chronic, noncancer pain. Concerns over diversion, misuse, dependence, addiction, monitoring, and cost can make the analysis of using chronic opiate therapy troublesome for even experienced clinicians.45 In the patient with SCI, concerns over the potential exacerbation of neurogenic bowel because of opioid-related constipation makes this decision even more challenging. There are several new strategies for the management of opioid-related constipation including peripheral opioid receptor antagonists and prokinetic agents.46 A review of the use of opioids in neuropathic pain suggested clinical efficacy of this medication class for long-term use. It is relevant to note that this review has a large preponderance of peripheral-based neuropathic pain (diabetic neuropathy or postherpetic neuropathy), but some subjects with SCI were included.47 There are several developments within the opioid class medications that may be of specific interest to physiatrists treating SCI-related pain. Tapentadol is a centrally acting analgesic with dual mechanisms of action—agonist activity at the mu opioid receptor and inhibition of norepinephrine reuptake. A potential therapeutic advantage of this agent is its utility in neuropathic pain. This benefit has been observed with both low–back pain with a neuropathic pain component as well as diabetic peripheral neuropathy.48,49 There are no specific reports on the use of this agent in SCI. In addition, this medication may also have therapeutic advantages over other opiates including a lower incidence of withdrawal symptoms as well as decreased frequency of gastrointestinal side effects. However, because of the activity that this agent has with monoamine metabolism, there is a potential to exacerbate or precipitate serotonin syndrome.50 Another dual-acting product is tramadol, which is a combination of a serotonin and noradrenaline reuptake inhibitor and a mu opioid agonist. This medication is noteworthy because its mechanism of action is distinct from those of other opioids. Tramadol has been shown to demonstrate benefit in osteoarthritis, fibromyalgia, and neuropathic pain; however, there is insufficient evidence to definitely define tramadol as more effective compared with other opioids.51 A small, randomized controlled trial in SCI-related neuropathic pain demonstrated a positive response to this medication.52

The relationship between pain and spasticity is complex. Reduction of spasticity may reduce the pain associated with biomechanical pain. Modulation of spasticity may not be effective in reducing neuropathic pain.7 There are several oral medications that can accomplish spasticity reduction including baclofen, tizanidine, diazepam, and dantrolene. Of particular interest, tizanidine has a dual mechanism of action: an
$\alpha_2$-adrenergic agonism at the spinal level and an influence on descending noradrenergic pathways. It is this latter mechanism that may be of particular interest in the management of SCI-related pain. Similarly, botulinum toxins have the potential to reduce muscle overactivity in a focally directed manner. Abobotulinum toxin A has formal FDA indication for adult, upper extremity spasticity after stroke and brain injury, although there are ongoing clinical trials for the other preparations and indications. Over and above their antispasticity activities, botulinum toxins have the capacity to be antinociceptive. However, there are no formal studies examining the effects of botulinum toxin on SCI-related pain independent of their spasticity reduction properties.

Medicinal marijuana and synthetic cannabinoids represent intriguing pharmacologic choices for the management of SCI-associated pain. Cannabis contains 60 or more cannabinoids, the most abundant of which are delta-9-tetrahydrocannabinol (THC) and cannabidiol (CBD). Rintala and colleagues executed a small study with dronabinol on SCI-related neuropathic pain. This agent is a pure isomer of THC. This investigation failed to demonstrate a significant difference in pain intensity compared with an active control. Sativex is a cannabis extract that contains THC + CBD in a fixed ratio, delivered as an oromucosal spray. Sativex has indication for multiple-sclerosis-related spasticity in several countries but not in the United States. A recent study of neuropathic pain associated with multiple sclerosis failed to demonstrate significant differences during the double-blind phase of this trial. There are no specific reports on the use of this agent in SCI-associated pain. There is an ongoing clinical trial examining the use of vaporized cannabis in SCI pain. There are no formal studies examining the use of medicinal marijuana in this patient population despite Cardenas and Jensen reporting that up to 37% of patients with SCI have used marijuana for pain reduction purposes.

Nicotine has been reported to exacerbate SCI-related pain with abstinence resulting in relief. One recent study examined the effect of nicotine in a randomized, placebo-controlled crossover design on the subtypes of SCI-related pain (neuropathic, musculoskeletal, and mixed pain) among smokers and nonsmokers. This study involved 42 subjects of whom two-thirds had paraplegia. Nonsmokers with SCI showed a reduction in mixed forms of pain after nicotine exposure, whereas smokers with SCI reported increase in pain for both mixed and neuropathic pain. This study suggests differential effects on SCI-related pain for smokers and nonsmokers. This observation potentially offers some insight into the mechanisms of SCI-associated pain as well as supports the suggestion of smoking cessation in some patients with SCI.

### Interventional

Spinal cord stimulation is defined as posterior epidural stimulation of the dorsal columns. The proposed mechanisms of action of this therapy involve the gate theory of pain, enhancement of parasympathetic activity, inhibition of sympathetic activity, upregulation of descending inhibitory pathways, and downregulation of ascending pain pathways. There have been many case reports documenting both success and failure of this technology for this pain syndrome. Shaw has presented a meeting abstract in which 12 patients with SCI received dorsal column stimulation for treatment of pain. The patients with complete injuries had variable success; however, none of them experienced paresthesias at stimulation above their injury. In the incompletely injured patients, paresthesias were experienced at varying levels of stimulation intensity. The patients with incomplete SCI had a higher degree of pain relief than those with complete SCI; however, 1 patient with complete SCI, who had been injured for less than 2 years, had complete relief of pain. Lagauche and colleagues have studied the use of vaporized cannabis in SCI pain.
executed a review of this modality in SCI-related pain and failed to find a consistently positive therapeutic effect.

Intrathecal drug delivery provides direct administration of therapeutic agents to the subarachnoid space where they have enhanced access to receptor sites. Intrathecal baclofen is a well-established technique for reduction of spasticity associated with SCI. To the extent that spasticity is related to musculoskeletal pain, this technique has the capacity to attenuate pain in this population. However, the use of intrathecal baclofen as a pure pain-modulating agent is limited. The utility of more traditional intrathecal analgesic agents has not been overwhelmingly successful. Combination therapy with baclofen and clonidine, morphine and clonidine, baclofen and morphine, baclofen and ziconotide, as well as hydromorphone and ziconotide have resulted in varying degrees of success. Intrathecal gabapentin failed to demonstrate a therapeutic effect in a generalized pain population.

A particularly interesting, albeit experimental, neuromodulation approach to SCI-associated pain is oscillating field stimulation. A human trial of a low-voltage, alternating polarity device was undertaken to assess the possibility of this therapy, causing substantive neurologic recovery as suspected from animal studies. Pain was assessed during this trial to insure that this device did not cause pain. Somewhat surprisingly, use of this device was associated with a rather dramatic reduction in pain. After the 15-week treatment phase, VAS scores improved from a mean of 8 to a mean of 2 six months after treatment had been discontinued. No neuropathic pain was reported in any patient. The status of this device is uncertain until a larger, multicenter trial is undertaken.

SUMMARY

SCI pain is clearly a challenging pain syndrome. Each element of this review (classification, epidemiology, evaluation, and management) has demonstrated limitations. Further investigation by clinicians and researchers in both the SCI and pain communities is warranted in an effort to further delineate the nature of this problem and create more effective treatment strategies. Physiatrists are uniquely positioned to participate in this process and should engage in this endeavor whenever possible.

REFERENCES


