

# Rural Spinal Cord Injury Project

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## MANAGING PAIN

for adults with Spinal Cord Injury



Targeting Health Professionals

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# MANAGING PAIN FOLLOWING SPINAL CORD INJURY

Pain is a common complication after spinal cord injury (SCI), which can significantly impact upon a person's functional ability and independence, psychological well-being, ability to return to work and quality of life. The reported prevalence of SCI pain varies between 34-90%, due to differences in study design, and definitions of pain types and severity. However, most studies indicate that around two-thirds of people with spinal cord injury suffer from chronic pain (Bonica, 1991; Siddall et al., 1999). Similarly, the epidemiological factors involved in the development of SCI pain are not clear, as various authors debate such effects as level of injury (eg. tetraplegia versus paraplegia), type of injury and extent of neurological impairment (complete versus incomplete lesion) (Davidoff et al., 1987; Nashold, 1991). Some studies have also suggested a relationship between chronic spinal cord injury pain and psychosocial variables and these always need to be considered as a contributing factor (Richards et al., 1980; Summers et al., 1991).

## CLASSIFICATION

There are a number of different types of pain that are commonly seen following spinal cord injury. Classification of these pain types has always been somewhat problematic due to considerable uncertainty about the underlying mechanisms and systems involved and a wide variety of terms have been used in describing the same type of pain. In an attempt to standardise nomenclature the International Association for the Study of Pain recently proposed a classification system (Siddall et al., 2002). As with other types of chronic pain, this is first divided into *nociceptive* (pain arising from somatic or visceral structures) and *neuropathic* (pain arising from nerve structures including the spinal cord and brain). The system then identifies five common types of pain seen following SCI, including:

1. *Musculoskeletal pain* arising from bones, joints, ligaments and muscles either in the acute post injury phase or with chronic overuse;
2. *Visceral pain* arising from disturbances to bladder, bowel or other visceral function;
3. *At-level neuropathic pain*, sometimes described as endzone or borderzone, which is a band of burning, electric or shooting pain and hypersensitivity in the dermatomes close to the level of injury; and
4. *Below-level neuropathic pain*, referring to pain with the same burning, shooting, electric qualities as the previous type of pain but it is located diffusely below the level of injury usually bilaterally in the buttocks and legs.
5. The remaining category (*above-level neuropathic pain*) is not exclusive to spinal cord injury but includes several types of neuropathic pain that are commonly seen such as complex regional pain syndromes (often referred to as reflex sympathetic dystrophy or causalgia) and compressive neuropathies.

This classification attempts to identify most of the pain types commonly seen with the aim of providing direction for treatment.

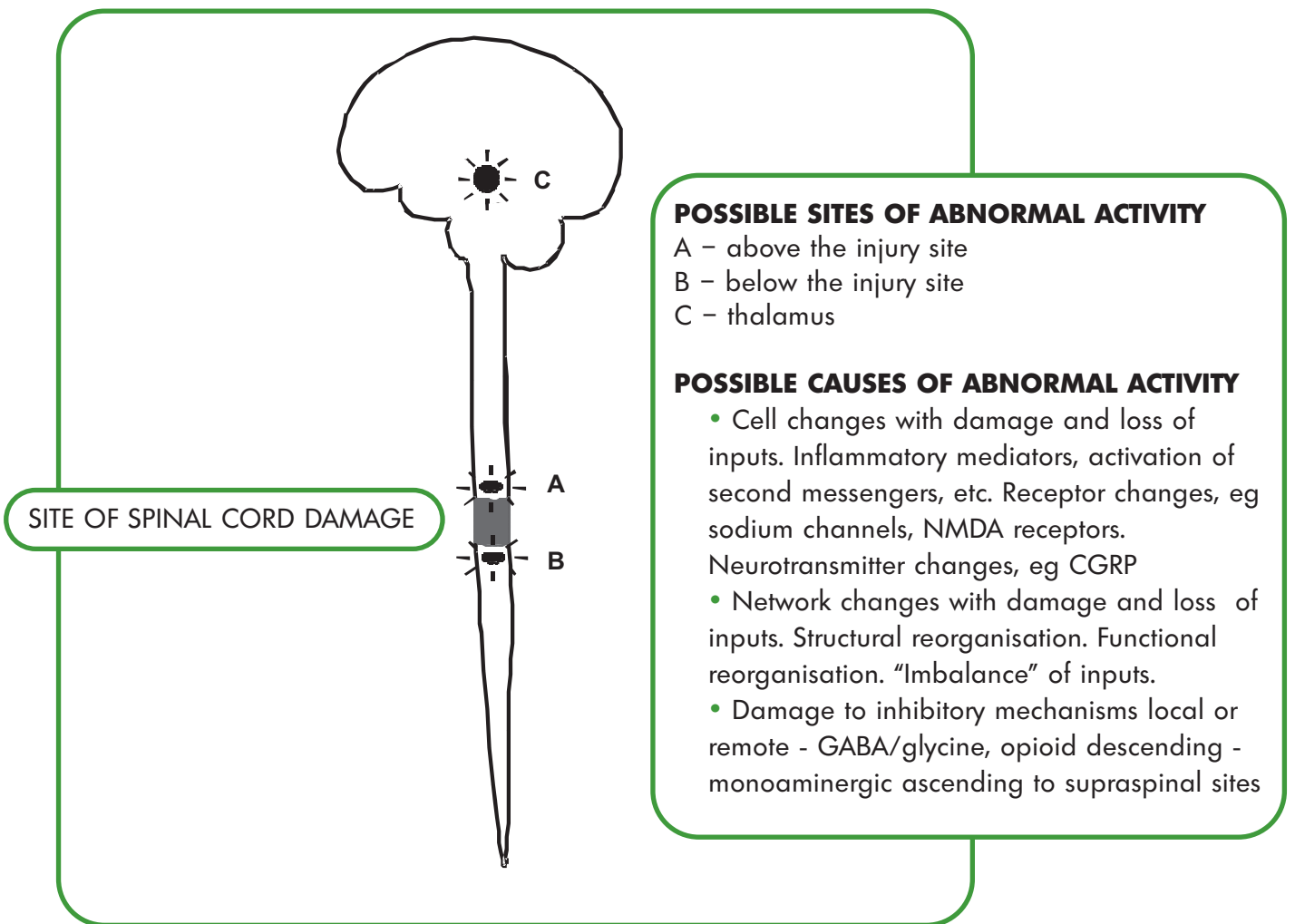
**TABLE 1** International Association for the Study of Pain: SCI Pain Classification

<b>BROAD TYPE (Tier one)</b>	<b>BROAD SYSTEM (Tier two)</b>	<b>SPECIFIC STRUCTURES/PATHOLOGY (Tier three)</b>
<b>Nociceptive</b>	<b>Musculoskeletal</b>	<b>Bone, joint, muscle trauma or inflammation</b> <b>Mechanical instability</b> <b>Muscle spasm</b> <b>Secondary overuse syndromes</b>
	<b>Visceral</b>	<b>eg Renal calculus, bowel, sphincter dysfunction, etc</b>
<b>Neuropathic</b>	<b>(above-level)</b>	<b>Compressive mononeuropathies</b> <b>Complex regional pain syndromes</b>
	<b>(at-level)</b>	<b>Nerve root compression (including cauda equina)</b> <b>Syringomyelia</b> <b>Spinal cord trauma/ischaemia (Endzone, borderzone, etc)</b>
	<b>(below-level)</b>	<b>Spinal cord trauma/ischaemia</b>

## **MECHANISMS**

Despite increased research interest over the last decade, our understanding of the pathogenesis and mechanisms underlying pain after SCI still remains quite limited. The mechanisms underlying musculoskeletal pain and visceral pain are better understood and in common with other types of nociceptive pain are basically due to increased inputs arising from damaged and inflamed structures. The mechanisms underlying neuropathic pain however are poorly understood. Neuropathic pain arising from peripheral structures, as occurs with compressive neuropathies, is similar to other types of peripheral neuropathic pain and is presumably due to abnormal impulses arising from damaged nerve structures.

At-level and below-level neuropathic pain are more specifically related to spinal cord injury and a number of possible mechanisms have been proposed to account for these pain types. Broadly, neuropathic pain arises from abnormal activity in pain pathways. This means that spontaneous activity of neurons may give rise to pain or increased responsiveness of neurons may give rise to hypersensitivity to touch or other stimuli. The changes responsible for the increase in nerve activity occur at both a spinal and supraspinal level (Vierck et al., 2000) (refer to Figure 1).



**FIGURE 1** Diagram summarising possible mechanisms for pain and supposed areas of abnormal activity in the brain and spinal cord following SCI.

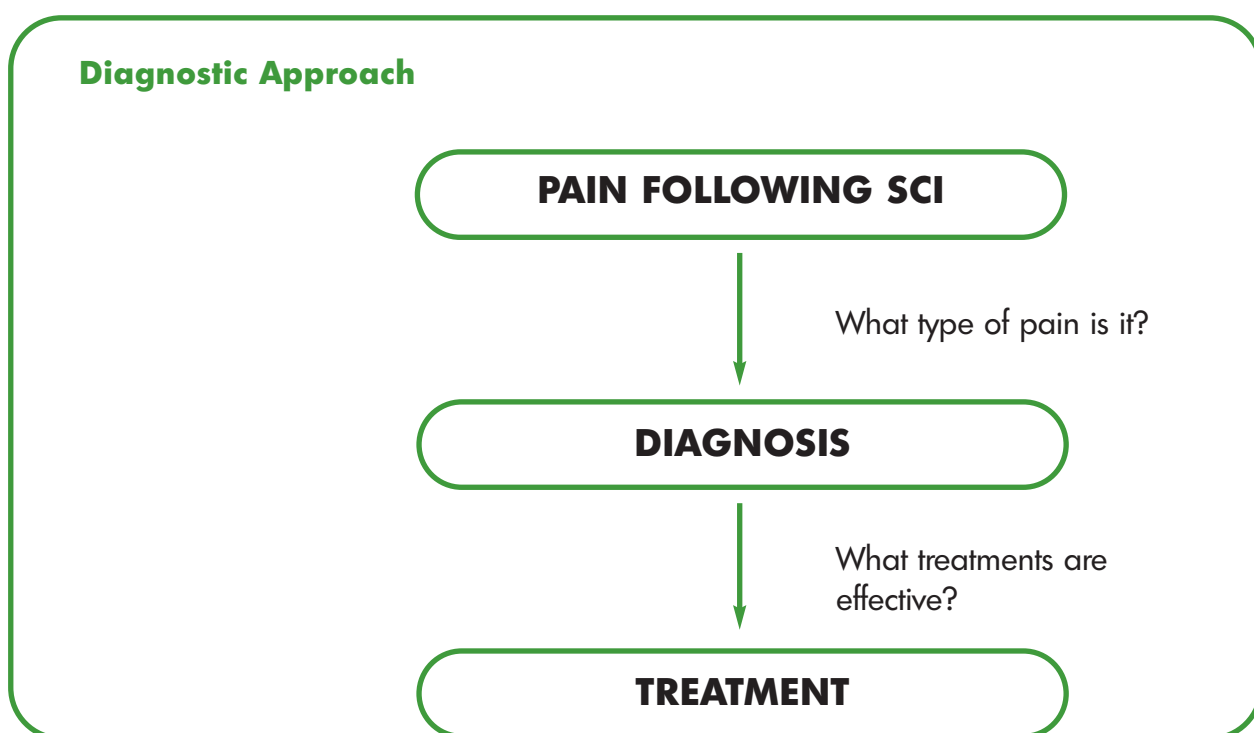
**These changes include:**

1. *Direct damage to the spinal cord.* This results in activation of inflammatory mediators and neuronal second messengers. Activation of these mediators and second messengers may in turn induce receptor changes (increased expression or enhanced responsiveness) and changes in the release of neurotransmitters that may transmit pain. All of these local changes may give rise to an “irritated focus” in the spinal cord near the level of injury. This “irritated focus” may generate pain spontaneously or may serve as a damaged amplifier that distorts and amplifies incoming messages from the periphery.

2. *Damage to inhibitory mechanisms.* Damage to the spinal cord also results in damage to the normal inhibitory mechanisms that serve to block out pain. This can occur following local damage to inhibitory neurons or with interruption to inhibitory pathways descending from the brain to the spinal cord. It has also been suggested that under normal circumstances there is a balance of inhibitory and excitatory inputs and that preferential damage to inputs that dampen pain (for example, light touch transmitted through the dorsal columns) may result in an “imbalance” and the generation of pain.

3. *Network change with damage and loss of inputs.* Damage to the spinal cord also leads to attempts to reorganise nerve pathways. Although limited, attempts to reconnect nerves at a spinal level may result in faulty “rewiring” that gives rise to pain. In this scenario, messages that normally travel along touch pathways are “hotwired” into pain pathways so that touch is felt as pain. Even if physical “rewiring” does not occur, the nervous system may attempt to compensate for lost inputs from damaged areas. This may involve activation of latent pathways in the spinal cord that again may tap into pain perception.

Despite a lack of understanding of all the various physiological processes underlying the development and maintenance of pain following SCI, recent efforts have assisted accurate, mechanisms-based diagnosis and classification of SCI pain (Siddall et al., 2002), providing better direction for treatment. A number of treatments have recently become available that have demonstrated improved efficacy in the management of pain following SCI.



The first step in assessing pain following SCI is to take a detailed history and perform a thorough physical examination.

1. History should include a description of pain type, onset and distribution, exacerbating and relieving factors, including relationship to functional activity such as transfers etc.

2. Clinical examination is essential and should include sensory, motor and reflex testing to classify the level and degree of neurological lesion using ASIA standards (Maynard et al, 1997). As indicated by the pain classification, the first step is to determine whether the pain is nociceptive or neuropathic in nature. This is largely dependent on pain description (nociceptive: dull, cramping, aching, worse with movement or related to visceral function, localised tenderness, located in the region of sensory preservation; neuropathic: shooting, electric, burning, unrelated to activity, numbness or hypersensitivity to touch, located in the region of sensory disturbance).

The pain can then be classified more specifically as visceral or musculoskeletal or above, at or below-level neuropathic. A careful history and clinical examination provides the basis for subsequent focussed investigations, including imaging and/or electrodiagnostic testing or other special procedures. Neuropathic pain may be indicative of new spinal pathology with development of a syrinx. This should be considered in someone with the onset or progression of neuropathic pain and deterioration in motor and/or sensory function.

## MANAGEMENT

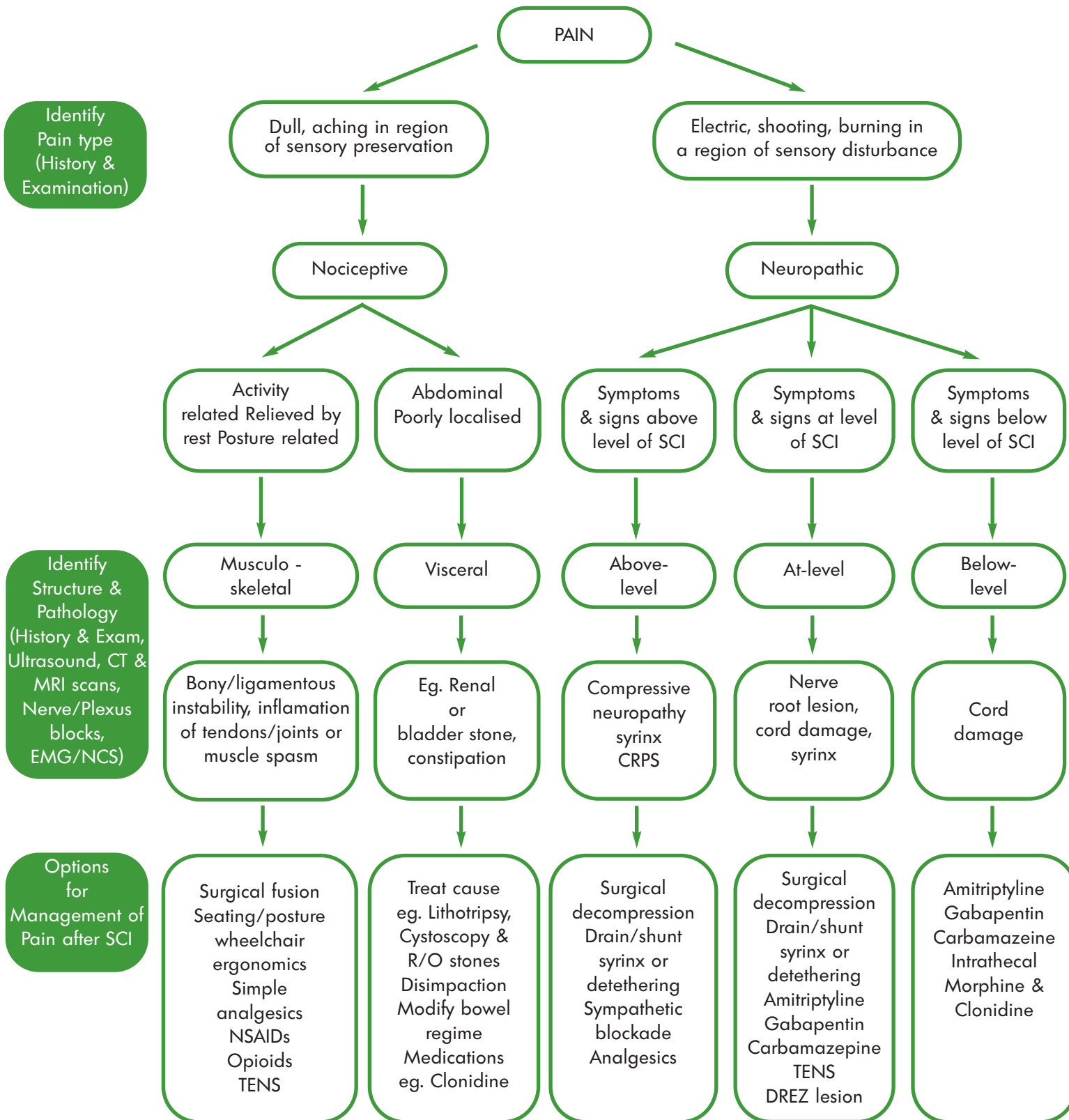
Successful management of pain depends on the accurate identification of factors that may be generating or modifying pain perception and using strategies that effectively target these factors. Management of chronic pain syndromes following SCI proves very difficult and unfortunately is often only partially effective. When treating chronic pain it is essential to comprehensively evaluate the types of pain described (such as musculoskeletal, visceral or neuropathic elements) and psychosocial factors contributing with emphasis on functional capabilities, behavioural responses to pain, adjustment to disability and degree of motivation. This is of great importance when selecting an appropriate combination of pharmacological, physical, psychological and other treatment approaches.

**Musculoskeletal pain** may respond quite well to simple analgesics and opioids. Non-steroidal anti-inflammatory medications may also be used but caution needs to be exercised, as gastric symptoms may be masked in people with higher lesions. Changes in posture, exercises, adjustments to wheelchairs and seating, hydrotherapy programs and other forms of physical treatment modalities may be helpful in treating pain that is arising from a mechanical source.

**Visceral pain** requires specific attention to the presumed source of pain. Urinary tract infections and calculi need to be treated appropriately. Bowel related pain may respond to simple measures such as change in diet or bowel regime, but may also require further assistance from a spinal unit.

**Neuropathic pain** responds poorly to most available treatments including opioids. The drugs that have been demonstrated to be most effective are the tricyclic antidepressants and anticonvulsants. Tricyclic antidepressants work by increasing the available amounts of the inhibitory transmitters serotonin and noradrenaline. Anticonvulsants work by dampening abnormal neuronal activity in peripheral nerves and the central nervous system.

FIGURE 2 Summary of recommended diagnostic and treatment approach



## GUIDELINES FOR PHARMACOLOGICAL MANAGEMENT

Once a diagnosis has been formulated and the type of pain identified pharmacological management may be instituted depending on the needs and desires of the patient. As indicated above, simple analgesics, NSAIDs and opioids are more likely to be effective in the treatment of musculoskeletal pain, although opioids may be used in those with neuropathic pain. Visceral pain may be treated with analgesics although only after investigation for pathology that may be amenable to other treatment (renal calculi, bladder infection, bowel dysfunction, etc).

Opioids are generally considered when simple measures fail to relieve the pain, although problems with constipation need to be considered as a major side effect in patients with SCI. As a general principle short acting and injectable opioids should be avoided. If long term treatment is being considered patients should be placed on a slow release formulation to reduce dose escalation and to provide more stable analgesia. This may be done after titration with a standard immediate release preparation of the same drug to determine analgesic requirements. Morphine has been available as a slow release preparation for some time. Both tramadol and oxycodone are also now available in slow release preparations and it has been suggested that both may be more effective in people with neuropathic pain. Referral to a Pain Clinic may be desirable if long term administration of opioids is being considered. Tolerance and dose escalation are always a concern in the patient using long term opioids. This needs to be discussed early with the patient and clear limits need to be established and agreed upon when prescribing. Addressing psychological factors is also important so that opioids are being used for analgesia rather than the treatment of distress.

As a guide the following analgesic “ladder” (Table 2) may be used for treatment of pain following spinal cord injury.

TABLE 2 Graduated process for analgesic prescription

	<b>Nociceptive pain</b> (generally musculoskeletal)	<b>Neuropathic pain</b>
<b>Step 1</b>	Paracetamol, NSAIDs	
<b>Step 2</b>	Tramadol	Tramadol
<b>Step 3</b>	Morphine, oxycodone	Oxycodone

As described above, analgesics are usually insufficient to control neuropathic pain and should be used in conjunction with adjuvant medications (tricyclic antidepressant and/or anticonvulsant; see dosing schedules below). A tricyclic antidepressant may be very helpful if there is a burning component to the pain.

Amitriptyline is used most frequently with the usual recommended dose between 25-75mg as a night time dose, however, some patients report drowsiness and an initial dose of 10 mg nocte may be preferable if there is a concern about side effects. Although there are no studies specifically looking at spinal cord injury pain, the selective serotonin reuptake inhibitors (SSRIs) are generally less effective in treating neuropathic pain than the tricyclic antidepressants.

Anticonvulsants are also effective in the management of neuropathic pain. Traditionally, they are used for at-level neuropathic (radicular or segmental) pain, particularly where there is a “sharp, shooting” component. However, they may also be used for the treatment of below-level neuropathic pain. Of the anticonvulsants, Carbamazepine has traditionally been preferred. It can be used alone in standard anticonvulsant doses (100 mg/day increasing to a maximum of 400 mg tds).

More recently, reports suggest the effectiveness of Gabapentin in treating intractable neuropathic pain and anecdotally it has been used with some success in the treatment of neuropathic pain following spinal cord injury (300 mg/day increasing to a usual maximum of 2400 mg/day). Unfortunately in Australia, it is not PBS listed and therefore in many patients the cost is prohibitive. Because of their different modes of action, it may be more effective to combine a tricyclic antidepressant and an anticonvulsant.

**TABLE 3** Commonly prescribed medications for neuropathic spinal cord injury pain

Drug Name	Usual Dosage	Side Effects & Precautions
Amitriptyline (Endep, Tryptanol)	10-75mg nocte	Dry mouth, blurred vision, confusion, nausea, vomiting, abdominal cramps
Carbamazepine (Tegretol)	200mg bd- 400mg tds	Drowsiness, nausea, dizziness, ataxia, diplopia, hypersensitivity rash, liver dysfunction, bone marrow toxicity
Gabapentin (Neurontin)	300-800mg tds	Dizziness, drowsiness, ataxia

A number of other drugs and techniques have been used with varying degrees of success. Subcutaneous or intravenous infusion of local anaesthetics such as lignocaine may be helpful for the treatment of neuropathic pain in the acute setting or as a diagnostic procedure. If successful, oral Mexiletine may be helpful because of its relationship to the local anaesthetics. Like the anticonvulsants, local anaesthetics probably act by dampening central aberrant neuronal activity. Other techniques, which have proved helpful in some cases, include anaesthetic blockade at various sites including sympathetic, epidural or spinal blockades.

Intrathecal administration of baclofen is effective in patients with poorly controlled spasms and spasm-related pain. Intrathecal administration of clonidine and morphine via an implanted pump has been demonstrated to be effective in some people with neuropathic pain following SCI. Intrathecal drug administration may be an alternative if patients have severe pain or spasm that fails to respond to other approaches.

In the past, surgical procedures have included sympathectomy, dorsal rhizotomy, cordotomy and cordectomy. These procedures are rarely, if ever, indicated now.

## **PHYSICAL AND PSYCHOLOGICAL MANAGEMENT**

The importance of physical and psychological management must not be underestimated. The principle of being “occupied” is important. Physical treatments including exercise and hydrotherapy programs, postural re-education, wheelchair and seating adjustments and possibly other physical modalities are often helpful in managing pain resulting from a mechanical cause.

It should never be forgotten that pain is a complex phenomenon and that emotional, behavioural and environmental factors may contribute to the experience of pain.

Following a spinal cord injury, the individual has to adjust to dramatic changes in functional independence, lifestyle, relationships, vocation and self-image. Pain can play an important role in the adjustment to spinal cord injury depending on the person’s pre-injury style of coping with stress and the way in which clinicians and therapists react to the pain problem.

Pain may be an expression of the patient’s difficulty in adjustment. Therefore, attention should always be paid to psychological factors with the use of cognitive-behavioural techniques aimed at physical and mental “reactivation” and strategies such as relaxation and distraction.

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## RURAL SPINAL CORD INJURY PROJECT

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This document was published as a fact sheet for the Rural Spinal Cord Injury Project (RSCIP), a pilot healthcare program for people with spinal cord injuries (SCI) conducted within New South Wales. It is not a stand alone resource but part of a series of eight fact sheets produced by specialists to fulfil the educational components of the project.

All recommendations are for spinal patients as a group. Individual therapeutic decisions must be made by combining the recommendations with clinical judgement, including a detailed knowledge of the individual patient's unique risks and medical history, as well as the resources available. This document is published as a guide only and does not take the place of advice from your regular health professional and /or medical practitioner.

