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New South Wales
**State Spinal Cord
Injury Service**

Managing Pain for Adults with Spinal Cord Injury

Targetting Health
Professionals



**MOTOR ACCIDENTS
AUTHORITY**



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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 (Widerström-Noga et al., 2002; Putzke et al., 2002; Rintala et al., 1998). The reported prevalence of pain after SCI varies widely 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 with one third of these people reporting severe pain (Störmer et al., 1997; Siddall et al., 1999; Siddall et al., 2003).

Like other types of chronic pain, pain following SCI is best considered within a biopsychosocial framework. This means that assessment and treatment take into account the various factors that may be contributing to the person's pain, including biological (such as level and extent of neurological impairment), psychological (including mood and cognition) and environmental factors (such as responses of significant others).

CLASSIFICATION

There are a number of different types of pain that are commonly seen in persons with a 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 proposed a classification system (Siddall et al., 2002). In a similar fashion to 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 (eg. carpal tunnel syndrome).

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

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
	Below-level	Spinal cord trauma/ischaemia

MECHANISMS

Despite increased research interest over the last decade, our understanding of the pathogenesis and biological mechanisms underlying pain after SCI still remains quite limited. While psychosocial factors seldom give rise to pain in isolation (so-called “psychogenic” pain), they universally influence the pain perception and behaviour and are important contributors to the pain experience.

Biological Mechanisms

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. By contrast, the mechanisms underlying neuropathic pain are poorly understood. 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. However, the site of abnormal activity may vary with different neuropathic pain types.

Neuropathic pain arising from peripheral structures, as occurs with compressive neuropathies, is similar to other types of peripheral neuropathic pain and is thought to be due to abnormal impulses arising from damaged nerve structures. At-level and below-level neuropathic pains are more specifically related to spinal cord injury and a number of possible mechanisms have been proposed to account for these pain types. These include abnormal activity of neurons within the spinal cord close to the site of damage, as well as more rostral changes in the thalamus and cortex that seem to occur as a secondary consequence of spinal cord damage (Vierck et al., 2000) (refer to Figure 1).

Pathophysiological changes include the following:

1. Direct damage to the spinal cord

This results in activation of inflammatory mediators, glia 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.

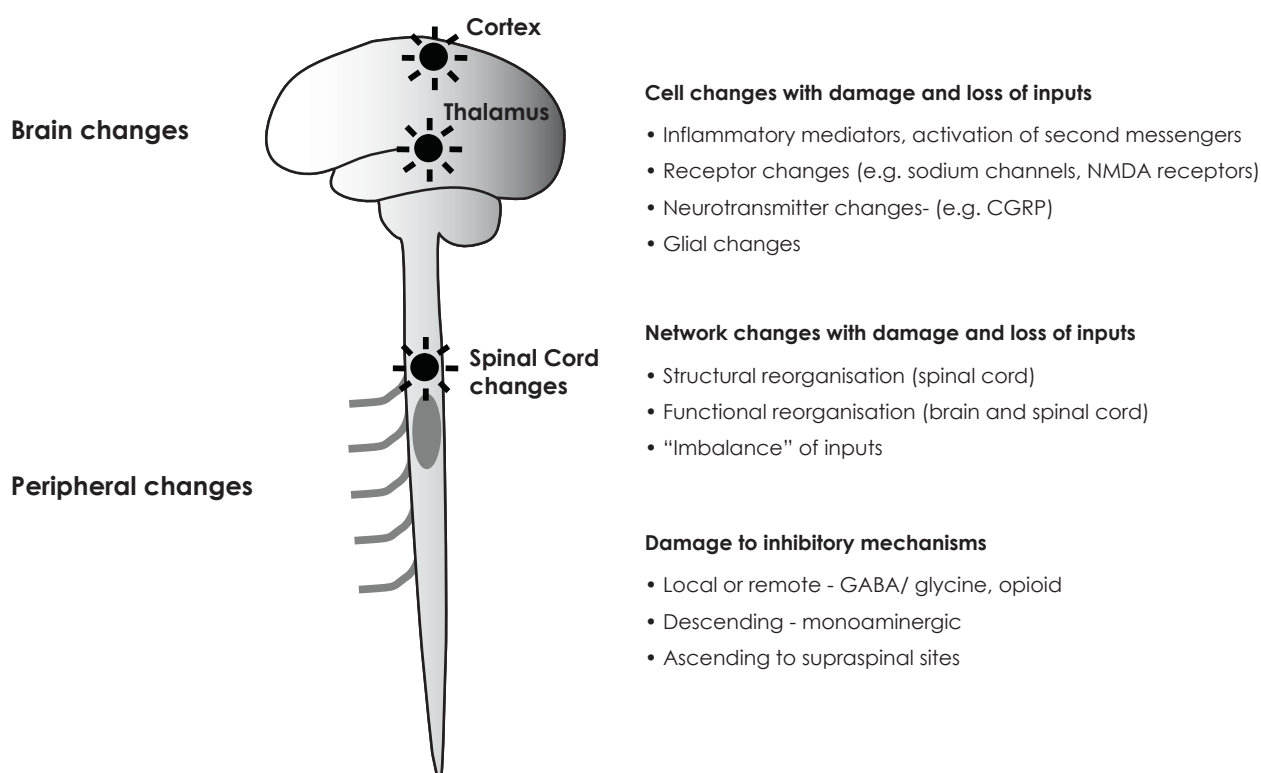
2. Damage to inhibitory mechanisms

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 changes 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. It has also been demonstrated that brain changes occur with abnormal activity in the thalamus and reorganisation of the somatosensory cortex as the brain attempts to adapt to the loss of inputs.

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

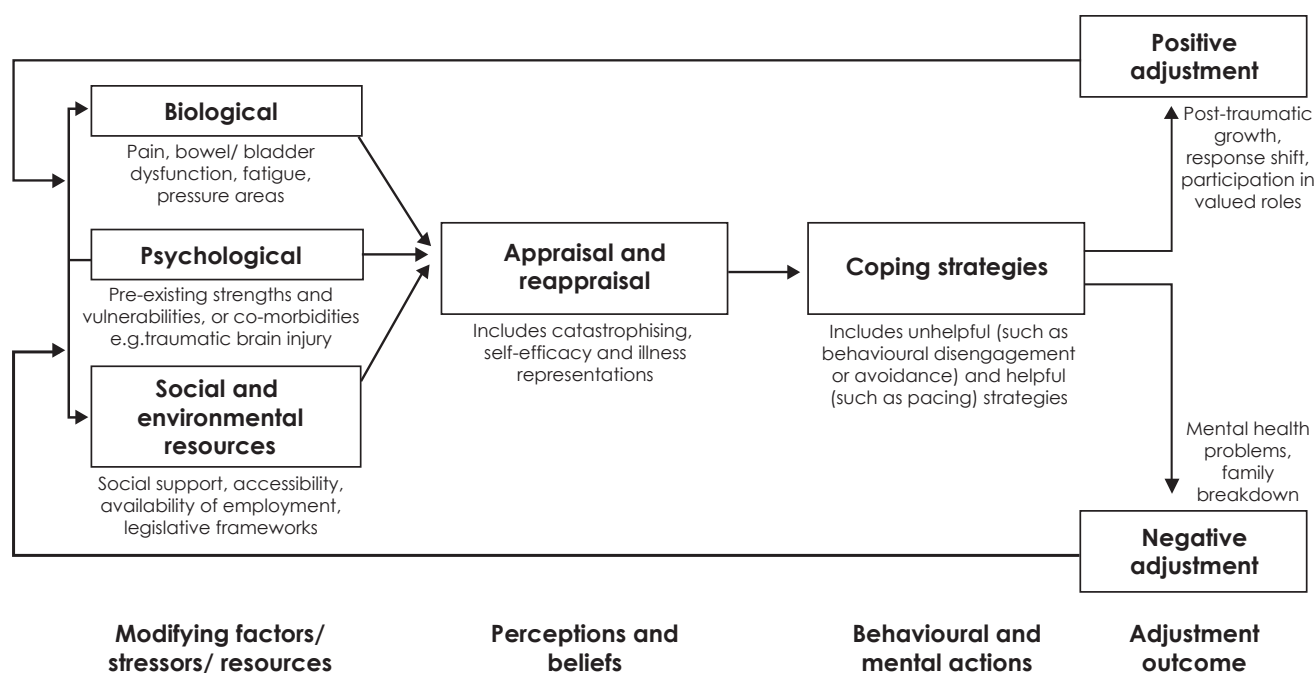


Psychosocial Mechanisms

Research into the psychosocial mechanisms in SCI pain has burgeoned in the past five years. Important concepts from the general pain literature, particularly cognitions such as pain catastrophizing (focusing upon negative aspects of pain, such as future disability) and pain self-efficacy (the individual's perceived ability to manage despite the pain), have increasingly been investigated. The evidence to date suggests that these mechanisms are significant in influencing the level of psychological distress and level of physical disability associated with these pain conditions (Nicholson Perry et al., in press; Raichle et al., 2007; Wollaars et al., 2007). In addition, other factors, such as SCI-related self-efficacy, also appear to have important relationships with mood and disability among those with pain (Middleton et al., 2007).

Social support has been acknowledged widely as having a significant impact upon the ability of individuals with a range of medical conditions to acquire acceptable quality of life. There is some evidence that particular types of responses by significant others can moderate responses to pain, such as solicitousness (Giardino et al., 2003). Consequently, it has been suggested that adjustment to SCI pain should be considered within a more general biopsychosocial model of SCI rather than a separate pain model (Nicholson Perry et al., in press).

Figure 2 Biopsychosocial Model – Spinal cord injury adjustment model
(after Middleton & Craig, in press)



DIAGNOSIS AND ASSESSMENT

Given the broad range of mechanisms that impact upon the development and maintenance of pain following SCI, a multidimensional approach to assessment and diagnosis is required. It is recommended that a full review of the patient with a SCI is conducted if one has not otherwise been conducted in the past twelve months. This will help to identify general issues, for example, in relation to seating or equipment, which may be contributing to the pain problems. Recent efforts have assisted in developing an accurate, biological mechanism-based diagnosis and classification of SCI pain (Siddall et al., 2002).

Identifying the type of pain provides a basis for further assessment, investigation and treatment. Siddall and Middleton (2006) described an algorithm based on the IASP Classification and existing levels of evidence to help guide clinical decision-making. Unfortunately, for many of the treatments currently used for SCI pain there is limited evidence of efficacy, particularly when the pain is neuropathic in nature.

Clinical assessment of pain following SCI involves:

1. History

A detailed history should be taken describing the pain type, onset and distribution, exacerbating and relieving factors, including relationship to posture and functional activity, such as transfers, etc.

2. Examination

Performing a comprehensive physical 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 proposed pain classification system, 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 in type.

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 (ie. cystic cavitation of the spinal cord extending above and/or below lesion). This should be considered in someone with the onset or progression of neuropathic pain and deterioration in motor and/or sensory function.

3. Psychosocial Assessment

Screening of psychosocial factors contributing to disability and distress is important at this stage. Ideally, a full assessment should be undertaken by a suitably trained psychosocial professional, although some screening of mood may be done through the use of questionnaires, such as the Hospital Anxiety and Depression Scale (Zigmond & Snaith, 1983), and screening questions in the initial phase:

- Does the pain affect your ability to take part in daily activities, including sleep?
- Does the pain affect your mood?
- When the pain is bad, what do you do, what goes through your mind and what happens to your mood? (Screen for suicidal ideation at such times)
- Do you use pain medications (or other substances such as alcohol) on a prn basis to manage your pain? Do you take more than the recommended dose?
- What do you understand is causing your pain, and what the future holds? (Listen for evidence of catastrophic thinking about the pain).

Where the answers to such questions reveal a marked impact upon activity and mood (particularly associations with suicidal ideation), use of pain contingent or harmful levels of pain medications or other substances, pain contingent activity levels (particularly the boom and bust cycle, where individuals increase their activity levels during better pain periods, followed by restricted activity during flare ups of pain), or catastrophic thinking about the pain, then a full assessment is indicated.

Aspects to be explored in a full assessment include:

- Adjustment to the injury and pain (including tendency to catastrophic thinking)
- Self-efficacy (for both the pain and injury)
- Behavioural responses to the injury and pain (including adaptive and maladaptive behaviours)
- Mood (including presence of depression, anxiety or post-trauma symptoms)
- Impaired cognition (due to head injury or other factors)
- Co-morbid psychological disorders (including psychiatric conditions or drug and alcohol dependency)
- Social factors (including social support, as well as significant others responses to pain and injury).

MANAGEMENT

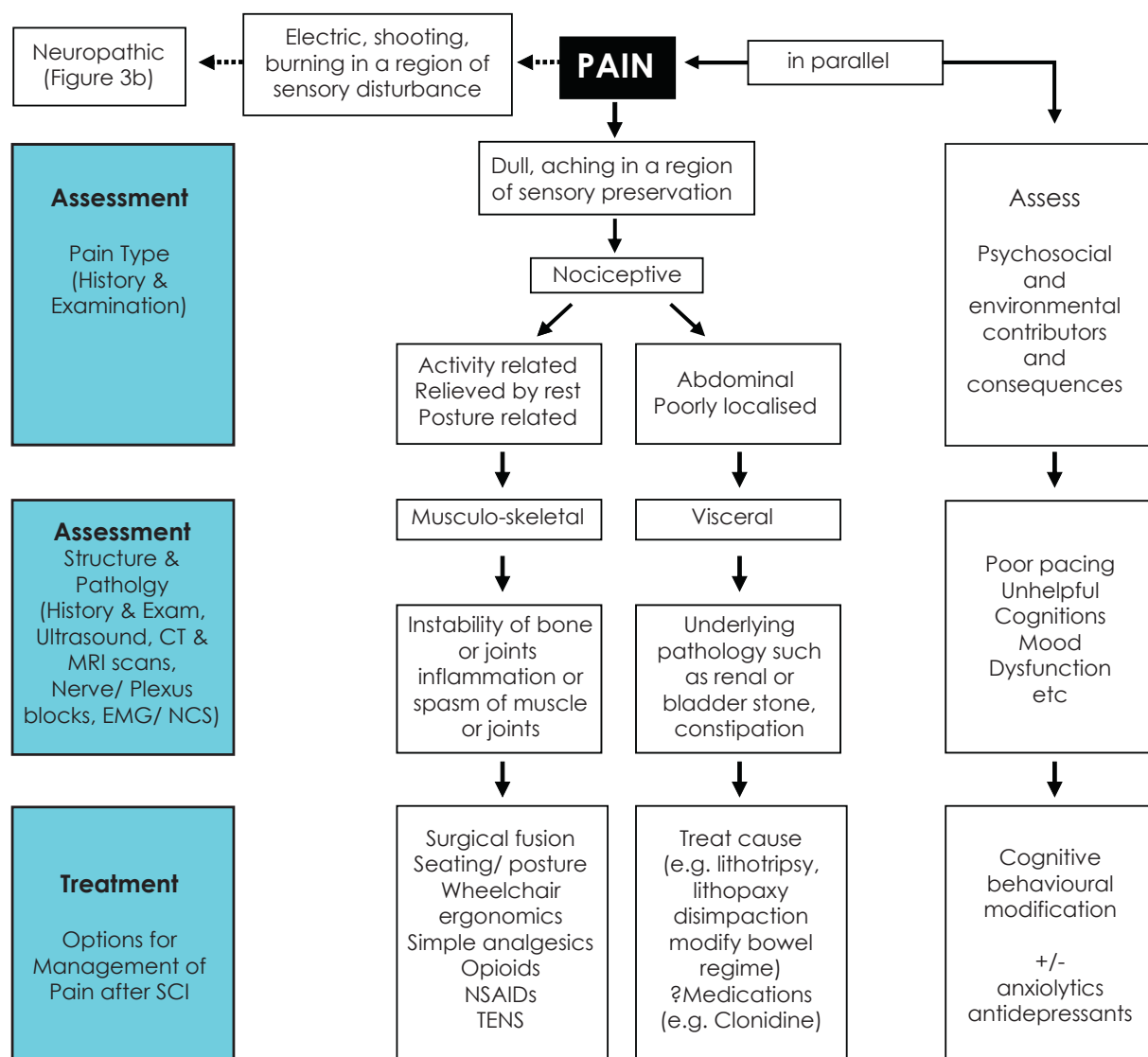
Successful management of pain depends on the accurate identification of factors that may be generating or modifying pain perception (see Figure 2 above) and using strategies that effectively target these factors. Management of chronic pain syndromes following SCI proves very difficult and unfortunately is often only partially effective. As already mentioned, when treating chronic pain, it is essential to comprehensively evaluate the type/s of pain 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. Treatable underlying pathology, such as local nerve root compression or post-traumatic syringomyelia (with expanding syrinx formation) must be excluded.

Rehabilitation principles should underpin any pain management programme, with the overall objective being to increase self-efficacy and promote greater activity and participation. Goals for treatment should be developed collaboratively with the individual prior to making management recommendations, in order to ensure the most effective multi-modal approach. Goals may be set in a range of domains, including (but not exclusively) activities of daily living, physical fitness and endurance, vocational activities, recreational activities, relationships and family, and mood.

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 SCI 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.

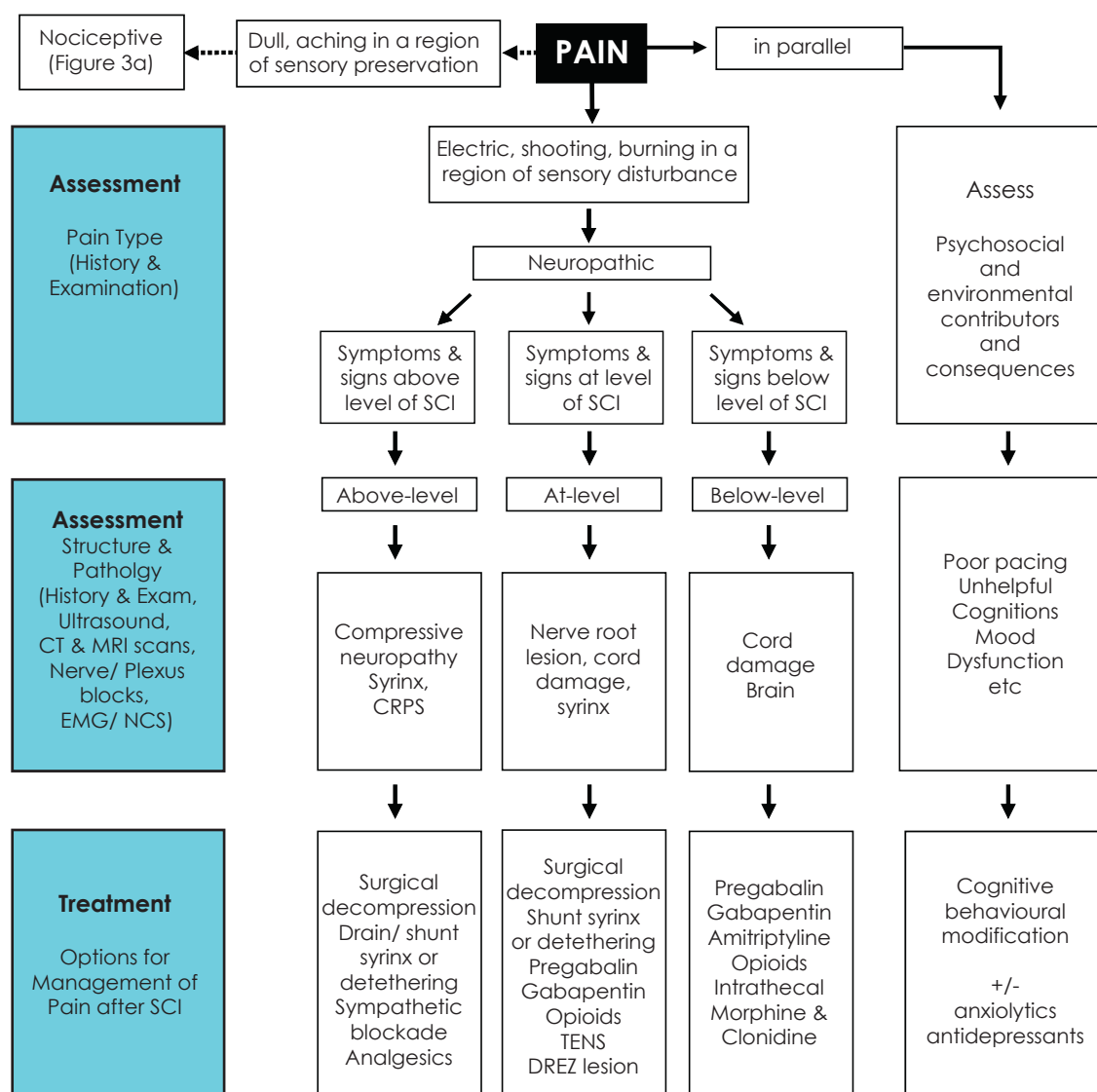
Visceral pain requires specific attention to the presumed source of pain. Urinary tract infections and calculi need to be treated appropriately. Bowel related pain may respond to simple measures such as change in diet or bowel regime, but may also require further assistance from a spinal specialist.

Figure 3a Summary of recommended diagnostic and treatment approach for Nociceptive pain



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

Figure 3b Summary of recommended diagnostic and treatment approach for Neuropathic pain



Pharmacological Management

Nociceptive

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 as a major side effect in patients with SCI, who already have slow colonic motility, as well as potential for developing physical dependence and tolerance, must be carefully considered. 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.

Neuropathic

As described above, analgesics are usually insufficient to control neuropathic pain and should be used in conjunction with adjuvant medications (ie. anticonvulsant, tricyclic antidepressant; see dosing schedules below). For both chronic at-level (radicular or segmental) and below-level types of neuropathic pain, first-line treatment with either gabapentin or pregabalin is now recommended. Unfortunately in Australia, these two medications are not currently PBS listed and therefore for many patients the cost is prohibitive. Commonly used older anticonvulsants such as sodium valproate have been shown to be no more effective than placebo in randomised controlled trials. Carbamazepine has been shown effective in combination with amitriptyline in treatment of other types of neuropathic pain, but has not been studied in SCI pain. Because of their different modes of action, it may be more effective to combine a tricyclic antidepressant, such as amitriptyline or nortriptyline, or alternatively a weak opioid, such as tramadol, with an anticonvulsant. Due to concerns about serotonergic syndrome, the combination of a tricyclic antidepressant and tramadol should be avoided. 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.

Use of strong opioids, such as oxycodone, morphine, fentanyl and methadone may provide some benefit in treatment of SCI neuropathic pain but is still somewhat controversial because of the generally poor long term response and problems with dose escalation, dependence and side effects. Of all types of SCI pain, neuropathic pain remains the least well understood and provides the greatest challenge to treatment, with only one in three people experiencing greater than fifty per cent reduction in pain with biomedical interventions.

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

	Nociceptive pain (generally musculoskeletal)	Neuropathic pain
Step 1	Paracetamol, NSAIDs	Adjuvants (anticonvulsants, TCAs)
Step 2	Tramadol	Tramadol
Step 3	Buprenorphine, morphine, oxycodone, fentanyl	Buprenorphine, oxycodone, methadone, fentanyl

Opioids

A number of opioids are available as slow release preparation. Tramadol may be considered as a first step if simple analgesics fail to provide sufficient relief. A number of other slow release opioids such as buprenorphine, morphine, oxycodone, methadone and fentanyl patches are also available and can be considered depending on the individual situation. Tolerance and dose escalation and side effects such as constipation are always a concern in the patient using long-term opioids. Referral to a Pain Clinic may be desirable if long term administration of opioids is being considered. 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.

Table 3 Commonly prescribed medications for neuropathic spinal cord injury pain

Medication Class	Notes on Use	Precautions and side effects ¹
Simple analgesics Paracetamol (1g tds to qid) NSAIDs	For nociceptive pain For nociceptive pain but may have some effect on neuropathic pain.	Be cautious of use in sodium restriction, phenylketonuria, hepatic & renal impairment. Be cautious of use in heart failure, uncontrolled hypertension, asthma, surgical procedures, history of peptic ulcer, G6PD deficiency, renal or hepatic impairment, bleeding diathesis & elderly.
Tricyclic Antidepressants Amitriptyline (10-75mg nocte) Nortriptyline (10-75 mg nocte)	Adjunctive medication for use in neuropathic pain. Monitor for possible effects on bladder (ie. (urinary retention in persons voiding by reflex) and bowel function (constipation).	Can provoke cardiac dysrhythmias and contraindicated where significant conduction abnormalities. Be cautious using in persons with coronary heart disease, orthostatic hypotension, prostatic hypertrophy, closed angle glaucoma, hyperthyroidism, epilepsy, use of other serotonergic drugs, hepatic impairment, elderly. Anticholinergic side effects are common and include dry mouth, blurred vision, orthostatic hypotension, urinary retention, constipation, and sedation.
Anticonvulsants Gabapentin (300 – 1200 mg tds) Pregabalin (75 - 300 mg bd) Carbamazepine (200 mg bd - 400mg tds)	Regarded as first line treatment of neuropathic pain following SCI. First line treatment. Recent RCT provided strongest evidence for efficacy in treatment of neuropathic SCI pain. Effective in trigeminal neuralgia but controlled studies in SCI pain are lacking.	Be cautious of use in renal impairment. Common side effects include fatigue, sedation, dizziness, ataxia, visual disturbance, amnesia, abnormal thinking, vasodilatation, peripheral oedema, dry mouth, weight increase, rash. Renal impairment requires dosage reduction. Common side effects include dizziness, drowsiness, visual disturbance (including blurred vision, diplopia), ataxia, dysarthria, lethargy, memory impairment, euphoria, tremor, weight gain, constipation, dry mouth, peripheral oedema, facial oedema. Be cautious of use if taking clozapine, hepatic impairment. Common side effects include drowsiness, ataxia, dizziness, blurred vision, diplopia, headache, rash, dry mouth, abdominal pain, nausea, vomiting, anorexia, diarrhoea, constipation, hyponatraemia, leucopaenia, thrombocytopaenia, increased liver enzymes.
Opioids Tramadol, Buprenorphine, Oxycontin, Methadone, Morphine, Fentanyl	There are no studies that have specifically shown oral opioid analgesics effective in SCI pain	Careful consideration of issues such as sedation, constipation, abuse, dependence and tolerance before prescribing should occur.

¹Information summarised from Australian Medicines Handbook. Please refer to medication product information for full details, including contraindications, before prescribing.

Other treatments

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, however, a satisfactory oral equivalent does not exist. Medications such as oral mexiletine may be tried because of its relationship to the local anaesthetics, but unfortunately is often not very effective. Like the anticonvulsants, local anaesthetics probably act by dampening central aberrant neuronal activity.

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, but only after psychosocial factors have been fully assessed and appropriately managed. Ablative surgical procedures generally have a very limited role in management, apart from DREZ lesions (performed by radio frequency or laser coagulation) for treatment of at-level neuropathic pain.

Physical Management

Physiotherapy and occupational therapy interventions may be necessary to improve fitness, posture and overuse syndromes, in particular. Physical treatments including exercise and hydrotherapy programs, postural re-education, retraining activities of daily living such as transfers and mobility, wheelchair and seating adjustments, modifying lifestyle and possible other physical modalities are often helpful in managing pain resulting from a mechanical cause. Assessment by an experienced physiotherapist and/or occupational therapist is recommended as part of a team approach.

Psychosocial Management

Significant psychological co-morbidities (such as psychiatric diagnosis, traumatic brain injury or drug and alcohol dependence) are likely to interfere with the optimal management of pain and therefore require separate assessment and management, and must be included in the treatment plan. Detection of general adjustment issues relating to the injury, rather than exclusively to the pain, may indicate that the outcome of pain treatment will be compromised if this is not addressed. Thus, referral to appropriate support services for further assessment and management is warranted in this situation. Social factors may also need to be addressed. This may include education of significant others regarding management of the pain or referral for relationship work. Specific cognitive behavioural pain management interventions have been shown to offer benefit to those participating in relation to mood, interference with daily activities and catastrophic thinking (Nicholson Perry et al., submitted; Norrbrink Budh et al., 2006). Although there has been no investigation of these interventions delivered on an individual basis, they could be adapted for use in the context of individual treatment (refer to table 4).

Self-management Strategies

Strategies that health professionals can suggest to clients that will contribute to reducing their pain-related disability and distress include:

- Maintaining a regular pattern of activity despite the pain, rather than falling into a 'boom and bust' cycle which depends on pain levels
- Breaking activities into manageable chunks and planning ahead for regular rest breaks, rather than pushing on until pain becomes unbearable
- Planning ahead and prioritising activities so that the person with SCI can achieve the things that are most valuable to them – not forgetting to prioritise enjoyable activities!
- Establishing a regular pattern of medication use, rather than only taking it when pain levels become high
- Developing a plan for dealing with days when the pain is worse, which can be shared with family and carers so that they can remind the person with SCI about what they were planning to do
- Trying not to panic! In contrast to acute pain conditions, most persistent pain in the context of a spinal cord injury is NOT an indication that there is anything wrong, but is more likely to reflect central nervous system changes.

Further details are available in the book **Manage Your Pain** by Associate Professor Michael Nicholas and colleagues.

Table 4 Components of cognitive behavioural pain management

Component	Content
Education regarding pain	Information regarding underlying pain mechanisms relevant to chronic pain, such as central sensitization and limitations of medical treatment for chronic pain. Additional information specific to understanding of spinal cord injury–related pain.
Goal-setting	Standard collaborative goal-setting, emphasizing breaking down long-term goals (including return to work) into smaller short-term goals that are challenging but achievable to enhance a sense of mastery and provide reinforcement for efforts.
Activity pacing	Based on goals, identification of problematic positions or tasks. Establishment of a baseline by measuring current tolerance to activities, followed by adoption of quota-based daily goals for each activity with systematic upgrading over a period of time.
Relaxation	Applied relaxation to enhance coping, reduce muscle tension and improve sleep.
Desensitization	Exposure to pain by consciously focusing attention on pain until anxiety levels decrease.
Functional exercise	After baseline levels are identified by the patient as tolerable, implement a whole of body reconditioning exercise programme with associated functional goals and graduated increase in intensity and duration over time.
Stretch	General daily stretch programme, with emphasis on integration of stretch into daily activities and other self-management tasks such as pressure relief.
Cognitive therapy	Identification of thoughts regarding pain and its management, and the challenging of those considered unhelpful, such as catastrophizing.
Medication reduction	Rationalization of medications with gradual reduction where possible, monitoring for negative functional impact and trade-off between efficacy and unwanted side-effect profile.
Flare-up management & relapse prevention	Explanation of concept of flare-up as an exacerbation of chronic pain, and development of a plan to manage it or other situations likely to trigger relapse. Careful exploration of how to differentiate general chronic pain flare-up from more sinister causes of increased pain in context of SCI, such as syringomyelia.

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