Management of chronic pain in persons with neurological disability

A multidisciplinary team is essential in the management of neurological disability.

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Ed Baalbergen’s interests are rehabilitation of the disabled, with a special interest in patients with spinal cord injury and stroke.

The field of rehabilitation medicine is one that has been recognised as a specialist field in Western society for many decades. Formal neurorehabilitation following an insult to the central nervous system that results in significant disability is essential, not only to regain maximal functionality, but also to restore dignity and improve the quality of life of the sufferer. In addition, patients who have been afforded the benefit of formal rehabilitation are less likely to return to hospital with complications related to their disability.

In South Africa, formal neurorehabilitation is a relatively new discipline and many specialists are beginning to appreciate the importance of this field of medicine. On the whole, South African rehabilitation units function in much the same manner as those in the rest of the world, utilising the expertise of an interdisciplinary team approach (Fig. 1).

The team approach ensures that patients are rehabilitated holistically and that all aspects of their disability are addressed, including psychological needs.

It is important to understand the various types of pain that may present in the disabled patient. Chronic pain is complex, and successful long-term treatment can be especially difficult. A sound understanding of the various types of pain is therefore essential. Broadly speaking, pain can be divided into:

- nociceptive pain
- neuropathic pain
- a combination of the two.

Nociceptive pain

Commonly, nociceptive pain is caused by the activation of nociceptive sensory axons in the somatic or visceral tissues, usually by a noxious or painful stimulus. Normally the pain is localised, finite and subsides when the causative stimuli subside, but its course can be chronic.

Examples of chronic nociceptive pain commonly seen in the disabled patient include:

- pain from a mechanical source such as discitis
- osteoarthritis
- contractures, etc.

If the pain is somatic it is often described as throbbing, stabbing, and gnawing, or sharp and localised. Visceral nociceptive pain, on the other hand, is usually poorly localised and, in addition to the characteristics described above, it is often diffuse and can be cramping and colicky.

Making an accurate diagnosis of visceral or somatic pain in the disabled can be a challenge in itself – depending on the neurological lesion site, localisation of the pain is poor and the usual signs and symptoms may be difficult to elicit.
Pain is a debilitating accompaniment that imposes a significant burden on individuals who have already suffered substantial emotional and physical trauma.

Neuropathic pain

Neuropathic pain is caused by the primary lesion in the peripheral or central nervous system leading to chronic nerve damage and changes within the neural pathway. This type of pain is usually chronic in nature and, unlike nociceptive pain, does not subside. It is ongoing and is usually present in the absence of a stimulus, although its intensity may wax and wane. Patients describe the pain in many ways – tingling, burning, shock like, or shooting. To treat this type of pain effectively, the practitioner must first distinguish between central and peripheral neuropathic pain syndromes, as treatment modalities/options differ.

Central neuropathic pain syndromes include pain after stroke or spinal cord injury, or pain associated with multiple sclerosis.

Peripheral neuropathic pain syndromes include:

- complex regional pain syndromes
- post-traumatic nerve injury
- radiculopathy
- diabetic and HIV-related neuropathies
- neuralgias related to herpes infections
- trigeminal neuralgias
- phantom limb pain.

Both central and peripheral neuropathic pain can present with a particular sign or symptom or even a combination of symptoms and signs. These include pain due to:

- a non-noxious stimulus (allodynia)
- loss or decreased sensation in an affected part (anaesthesia and hypoaesthesia)
- dysesthesias, which are abnormal unpleasant sensations either evoked or spontaneous
- hyperalgias, which are exaggerated responses to noxious stimuli
- paraesthesia (pins and needles) or phantom pain, which involves the perception of pain in a site that no longer exists.

Although nociceptive pain often presents in the disabled community, the commonest form of pain that plagues the disabled patient is that of neuropathic pain, and the diagnosis and management of neuropathic pain syndromes is challenging.

A large percentage of patients who have suffered a neurological insult resulting in significant disability will suffer from some form of neuropathic pain – for example, it is said that up to 50% of patients with spinal cord injuries suffer from some form of neuropathic pain.¹ Much work has been done in an attempt to define and understand the pathophysiology of neuropathic pain syndromes and this has given practitioners a greater understanding of this complication and treatment options.

Mechanisms of nociceptive and neuropathic pain

Four mechanisms are thought to be responsible for neuropathic pain:

- **Central sensitisation.** In central neuropathic pain there appears to be central sensitisation,²,³ where it is thought that sustained painful stimuli result in spinal sensitisation. This can be modulated at the presynaptic central neuron which utilises the calcium channels. This has important treatment implications and can assist in the choice of specific pharmacological options to treat this pain.

- **Central disinhibition.** With central disinhibition² of pain, the inhibitory neurotransmitters such as GABA and glycine are involved and serotonergic and noradrenergic neurons mediate the descending inhibitory influences.

- **Dysregulation of sodium channels.** It is thought that there is dysregulation of the sodium channels at the dorsal nerve root ganglion, resulting in hyperexcitability. Calcium channels are also thought to play a role here.²,³

- **Peripheral sensitisation.** There appears to be evidence of a degree of peripheral sensitisation² to pain stimuli.

The latter two are major mechanisms that have been identified in peripheral neuropathic pain.

Based on this information it is then possible to direct therapy according to the mechanism/s of pain identified.

Management options

Having identified whether the patient is experiencing neuropathic pain or nociceptive pain or a combination of the two, one can more logically focus on a specific therapy. It is important to set realistic goals for patients with chronic pain.

Clinicians need to explain to patients (and the primary caregiver) that the pain they are experiencing cannot necessarily be cured, but in the vast majority of patients it can at least be partially relieved.

A spectrum of treatment modalities should be offered from physical therapies, psychotherapeutic input such as visualisation techniques, etc. to pharmacological interventions and finally surgical options. The importance of the involvement of the interdisciplinary team in the holistic treatment of chronic pain cannot be overemphasised. Physical therapists often employ transcutaneous electrical nerve stimulation (TENS) to assist with pain relief and in some cases this may be sufficient. Exercise, ultrasound therapy, massage and cross-friction techniques as well as dry-needling acupuncture and acupuncture are also valuable tools in our armamentarium. These physical treatment modalities, in addition to a sound psychotherapeutic evaluation to assist with cognitive behavioural therapy, should all be offered and employed as a first line in persons with neurological disability and chronic pain.

Most patients, however, will also benefit from pharmacological interventions. The treatment of nociceptive pain has been well documented. In patients presenting with mild to moderate pain it is accepted practice to always start with simple analgesics (paracetamol) in adequate doses. Compound analgesics can always be added or substituted and the use of the non-steroidal anti-inflammatory drugs (aspirin, diclofenac, coxibs, etc.) forms the basis of treatment of nociceptive pain. Should these measures not suffice, a weak opioid (codeine or tramadol) with or without a non-opioid can be tried. Moderate to severe pain can be treated safely and effectively with fentanyl.
Neuropathic pain is more complex and difficult to treat; the successful treatment of this type of pain depends on early diagnosis, identification of possible mechanisms and the use of correct drugs. In addition to alternative therapeutic approaches already mentioned. The mechanisms thought to be responsible for neuropathic pain involve a wide range of neurophysiological, neurochemical and neuroanatomical changes – a wide range of drugs is fortunately available to treat this type of pain.

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Pain arising due to central sensitisation responds to drugs acting on the calcium channels at the presynaptic central neuron (opioids and NMDA receptor antagonists). Commonly gabapentin is used effectively. The drug has the advantage of being a relatively ‘clean’ drug with relatively few side-effects. A newer drug (pregabalin) is a relatively ‘clean’ drug with relatively few side-effects. A newer drug (pregabalin) is currently not available in South Africa but will hopefully become available in the near future. It is used widely in Europe, and is said to be some 30% more effective in reducing pain than gabapentin.

Antidepressants such as serotonin and noradrenaline re-uptake inhibitors (SNRIs), selective serotonin re-uptake inhibitors (SSRIs), tricyclic antidepressants (TCA) and opioids can be used to modulate pain arising through central disinhibition as the therapeutic effect of the SNRIs and GABA agonists is probably due to enhancement of inhibitory mechanisms.

The importance of the involvement of the interdisciplinary team in the holistic treatment of chronic pain cannot be overemphasised.

Pain thought to arise from peripheral sensitisation can occasionally be treated effectively by means of local therapies. These include the application of capsaicin ointment or creams to the affected areas.

The various options for the treatment of neuropathic pain are listed in Table 1.

Surgical options

For the minority of patients who do not respond to these measures, surgical options can be explored. The implantation of programmable medication pumps delivering various drugs such as morphine, ketamine, mazine or a combination of these has been used successfully in patients with intractable neuropathic pain. In addition, the implantation of dorsal column nerve stimulators has been used with some measure of success. Candidates for these options should be carefully evaluated by the team as these devices are expensive and the surgery undertaken is not without risks.

Chronic pain

patches (Durogesic), or other potent opioids such as slow-release morphine.

One should always keep in mind the potential side-effects of these drugs – pharmacological management should aim to prevent symptoms, reduce pain severity or frequency, improve functioning and overall quality of life and minimise side-effects.

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The TCAs (amitriptyline) have been shown to be the most effective in this class. However, it must be said that the efficacy of any one pharmacological agent may differ for different types of neuropathic pain and hence if one class of drug does not work effectively, substitute classes may be tried.

In peripheral pain syndromes, where the mechanisms are due to dysregulation of sodium channels, sodium channel agonists such as anticonvulsants can be used to limit repetitive firing of the sodium channels and thereby reduce pain. These drugs include phenytoin, carbamazepine and valproic acid.

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The various options for the treatment of neuropathic pain are listed in Table 1.

Table I. Neuropathic pain – pharmacotherapy options

<table>
<thead>
<tr>
<th>Drug</th>
<th>Starting dose</th>
<th>Dose adjustments</th>
<th>Effective ranges</th>
</tr>
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<tbody>
<tr>
<td>Gabapentin (Neurontin)</td>
<td>100 - 300 mg/day</td>
<td>300 mg every 3 days</td>
<td>300 - 1800 mg tds</td>
</tr>
<tr>
<td>Lamotrigine (Lamictin)</td>
<td>25 mg/day</td>
<td>25 - 50 mg every 2 weeks</td>
<td>250 mg bd</td>
</tr>
<tr>
<td>Carbamazepine (Tegretol)</td>
<td>100 - 200 mg bd</td>
<td>100 - 200 mg every 2 - 3 days</td>
<td>200 - 400 mg tds</td>
</tr>
<tr>
<td>Tricyclic antidepressants (Amitriptyline)</td>
<td>25 mg/day</td>
<td>25 mg weekly</td>
<td>25 - 150 mg/day</td>
</tr>
<tr>
<td>Venlafaxine (Efexor)</td>
<td>37.5 mg/day</td>
<td>37.5 mg every 3 days</td>
<td>75 - 150 mg bd</td>
</tr>
<tr>
<td>Tramadol</td>
<td>25 - 50 mg bd or 100 mg SR daily</td>
<td>50 - 100 mg every 3 days</td>
<td>50 - 100 mg qid or 150 mg SR daily</td>
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Local alcohol injections into the stellate ganglion, for example in patients with complex regional pain syndromes, also have a role to play. Referral to a pain clinic is then required.

References


In a nutshell

- The treatment of chronic pain in the disabled requires the utilisation of an interdisciplinary team approach, as the treatment is complex.
- Chronic pain comprises nociceptive pain, neuropathic pain or a combination of the two.
- Treatment regimens utilise a combination of physical therapies, psychotherapeutic modalities and pharmacotherapeutic interventions.
- Complete resolution of pain is usually not possible – realistic goals must be set.
- The pharmacological regimen should be individualised for each patient.
- Combination therapy of one or more agents may result in a synergistic effect.