



Managing patients with **neuropathic pain**


Neuropathic pain results from a lesion or disease affecting the somatosensory system. There are a range of causes of neuropathic pain including diabetes, surgery, multiple sclerosis, stroke, herpes zoster, cancer and chemotherapy; diagnosis can be challenging. A patient history and clinical examination focusing on sensory, motor or autonomic changes is the starting point of any investigation. The management of neuropathic pain aims to improve the patient's quality of life if symptom resolution is not possible. Tricyclic antidepressants, gabapentin, either alone or in combination, and carbamazepine for trigeminal neuralgia or diabetic polyneuropathy are appropriate options for treating most types of neuropathic pain in primary care. Alternative medicines, e.g. valproate or lamotrigine may be trialled, however, there is limited evidence of effectiveness in patients with neuropathic pain.

KEY PRACTICE POINTS:

- The key to investigating neuropathic pain is a patient history and clinical examination focusing on the presence and distribution of any sensory, motor or autonomic changes
- Where appropriate, investigate the underlying cause of pain, acknowledging that in some cases a definite cause may not be identified
- Treatment should focus on reducing the effect pain has on independence and wellbeing so patients can continue with their daily life
- Appropriate medicines to prescribe in primary care are:
 - Tricyclic antidepressants, e.g. amitriptyline or nortriptyline (unapproved indications)
 - Gabapentin, with Special Authority approval
 - Carbamazepine for patients with trigeminal neuralgia
 - Capsaicin cream (0.075%) for patients with localised cutaneous pain, subsidised with prescription endorsement
- Referral to a pain clinic may be beneficial at any point in treatment if the patient experiences severe pain or if their pain is causing significant disruption to their life; access and referral criteria may vary throughout the country

Neuropathic pain arises from damage to the sensory system

Patients with neuropathic pain often experience burning, shooting, stabbing or electric sensations or pain that has these qualities.¹ The pain may be constant, in response to triggers, or occur intermittently with no obvious cause. Long-term neuropathic pain can cause central sensitisation, resulting in an exaggerated pain in response to mildly painful stimuli (hyperalgesia) or pain in response to innocuous stimuli (allodynia).

 For further information on central sensitisation and nerve fibre hyperexcitability in chronic pain, see: "Understanding chronic pain" BPJ 70 (Sep, 2015).

Neuropathic pain is becoming more common

The number of people in New Zealand with neuropathic pain is unknown; worldwide it is estimated to affect 7% of people. The prevalence of neuropathic pain is predicted to rise due to an ageing population, increased cancer survival and chemotherapy-induced neuropathy, and an increased prevalence of diabetic neuropathy.²

Pain may be central or peripheral in origin

Neuropathic pain is referred to as peripheral or central depending on the location of the lesion(s) causing symptoms.³ Patients may have elements of both, and some conditions, such as multiple sclerosis, can cause central and/or peripheral neuropathic pain (Table 1).³

Neuropathic pain is challenging for clinicians and patients

Clinical judgement is especially important when evaluating patients suspected of having neuropathic pain because of the diverse array of potential causes. Investigations will not help all patients as a definite cause may never be identified, while

some may require multiple investigations before a diagnosis is reached. Treatments for neuropathic pain are relatively ineffective. Medicines often reduce, but do not eliminate, pain in approximately half of treated patients.⁶ Trialling different medicines or combinations of medicines may be necessary.

Treat the patient, not the condition

For some patients neuropathic pain may be temporary, but for others it will become part of their life. Untreated or undertreated neuropathic pain can lead to a reduced quality of life, psychological distress, problems with sleep, lethargy and difficulty completing daily activities. Even with optimal treatment patients are likely to continue to experience some pain. Repeated consultations, uncertainty about their condition and ongoing investigations can contribute to patient anxiety and exacerbate pain. The inconvenience of multiple appointments and ineffectiveness of medicines can increase frustration, e.g.^{6,7}

"There must be some other medication that can fix the pain... I need to get the pain sorted so I can get back to work."

"[My General Practitioner] sent me to a neurologist at the hospital and... all he did was increase my pain medication."

Eventually patients may feel they have become a burden to health professionals, e.g.:⁷

"I just think the General Practitioner gets sick of trying, he doesn't know what to do with me... he's sick of me."

A proactive approach to care can help

Neuropathic pain can place a strain on the doctor-patient relationship. Explain the difficulties early to avoid unrealistic expectations, e.g. that multiple consultations and trials of medicines are normal. Use language which implies management, rather than cure, unless there is an expectation that cure is possible.⁶ Encourage patients to return if they are still experiencing pain after a new medicine has been initiated.

Table 1: Conditions associated with central or peripheral neuropathic pain.³⁻⁵

Causes of central neuropathic pain	Causes of peripheral neuropathic pain
<ul style="list-style-type: none">■ Multiple sclerosis■ Stroke■ Traumatic brain injury■ Trigeminal neuralgia■ Cerebral vascular malformations	<ul style="list-style-type: none">■ Cancer and chemotherapy■ Diabetic polyneuropathy■ Hereditary neuropathies■ Multiple sclerosis■ Post-herpetic neuralgia■ Radiculopathies■ Surgery or amputation■ Trauma

Assessment of patients with suspected neuropathic pain

A history and focused clinical examination to assess for sensory, motor or autonomic abnormalities are central to the investigation of neuropathic pain. Consider other possible causes of pain and whether the timing of the pain coincides with significant events in the patient's history.

Are there positive or negative sensory, autonomic or motor signs in the same area? E.g. the presence of:^{8,9}


- Sensory signs
 - Allodynia
 - Hyperalgesia
 - Dysaesthesia (unpleasant sensation)
 - Hypoaesthesia (decreased sensitivity to stimulation)
 - Loss of proprioception
- Motor signs
 - Weakness
 - Absent reflexes
- Autonomic signs
 - Changes in skin colour due to vasodilation or vasoconstriction
 - Sweating

Patient history

When taking the patient's history a zero to ten scoring system can help to score pain severity.¹⁰ This information can be later used to assess the efficacy of medicines and aid dose titration. Specific features suggestive of neuropathic pain include:¹⁰

- Autonomic signs, e.g. changes in skin colour or temperature, sweating or heart rate changes
- Motor signs, e.g. areas of weakness, cramps, or spasms
- A family history of conditions associated with neuropathic pain, e.g. diabetes or multiple sclerosis or hereditary polyneuropathies such as Charcot-Marie-Tooth disease

Questionnaires can help determine whether a patient's pain is neuropathic. These are used in conjunction with clinical examination as they fail to identify 10–20% of patients with neuropathic pain.¹¹ One example of a questionnaire is the Leeds Assessment of Neuropathic Symptoms and Signs (LANSS). This diagnostic tool was designed to distinguish neuropathic from non-neuropathic pain. A LANSS score of 12 or more has a sensitivity ranging from 82–91% and specificity ranging from 80–94% when compared with diagnosis in a pain clinic.^{11,12}


 See "The S-LANSS Pain Score" for an example of a validated and self-administered questionnaire (S-LANSS).

Clinical examination

The clinical examination includes assessment of changes in sensory nerve function. For example, soft touch using cotton wool, sharp touch by pinprick, vibration using a tuning fork, cold and warmth sensation (see: "Sensory nerves and the sensations they carry").^{10,11}

Allodynia to light touch, cold and heat makes a diagnosis of neuropathic pain more likely, whereas allodynia to pressure occurs with the same frequency in patients with neuropathic or non-neuropathic pain.¹¹

Identify and document the borders of any area of altered sensation and compare results with the contralateral side of the body.¹¹ The precise distribution of dermatomes can vary between individuals.¹³

 A figure showing common boundaries of dermatomes, is available from: emedicine.medscape.com/article/1878388-overview#a2

Some patients may not have any identifiable changes in sensory or autonomic function, i.e. only pain with no findings on clinical examination and sensory testing.

Consider if findings are consistent with common causes of neuropathic pain

Lesions localised in a plexus are often asymmetric with involvement of multiple dermatomes, while lesions localised to one nerve root are asymmetric with a dermatomal pattern of sensory symptoms. Longer nerve fibres are typically affected first in diabetic and hereditary polyneuropathy or chemotherapy-induced neuropathy, resulting in symmetrical symptoms and signs in the distal limbs.

Deficiencies in vitamins B12 or E, or rare copper deficiencies, can cause combined motor and sensory symptoms and signs. Consider the timing of symptom onset and whether they are acute or slowly deteriorating; deteriorating function may indicate an ongoing process such as diabetes or hereditary polyneuropathy.⁸

Laboratory tests which can assist diagnosis

In patients where an aetiology is not clear initial tests include:⁴

- Complete blood count
- C-reactive protein
- HbA_{1c} and/or fasting blood glucose
- Vitamin B12
- Folate
- Liver function tests
- Renal function tests
- Thyroid stimulating hormone
- Serum protein electrophoresis

The S-LANSS Pain Score

1. In the area where you have pain, do you also have “pins and needles”, tingling or prickling sensations?	
<input type="checkbox"/> NO – I don’t get these sensations	0
<input type="checkbox"/> YES – I get these sensations	5
2. Does the painful area change colour (perhaps look mottled or more red) when the pain is particularly bad?	
<input type="checkbox"/> NO – The pain does not affect the colour of my skin	0
<input type="checkbox"/> YES – I have noticed that the pain does make my skin look different from normal.	5
3. Does your pain make the affected skin abnormally sensitive to touch? Getting unpleasant sensations or pain when lightly stroking the skin might describe this.	
<input type="checkbox"/> NO – The pain does not make my skin abnormally sensitive to touch.	0
<input type="checkbox"/> YES – My skin in that area is particularly sensitive to touch.	3
4. Does your pain come on suddenly and in bursts for no apparent reason when you are completely still? Words like “electric shocks”, jumping and bursting might describe this.	
<input type="checkbox"/> NO – My pain doesn’t really feel like this.	0
<input type="checkbox"/> YES – I get these sensations often.	2
5. In the area where you have pain, does your skin feel unusually hot like a burning pain?	
<input type="checkbox"/> NO – I don’t have burning pain	0
<input type="checkbox"/> YES – I get burning pain often	1
6. Gently rub the painful area with your index finger and then rub a non-painful area (for example, an area of skin further away or on the opposite side from the painful area). How does this rubbing feel in the painful area?	
<input type="checkbox"/> The painful area feels no different from the non-painful area	0
<input type="checkbox"/> I feel discomfort, like pins and needles, tingling or burning in the painful area that is different from the non-painful area.	5
7. Gently press on the painful area with your finger tip and then gently press in the same way onto a non-painful area (the same non-painful area that you chose in the last question). How does this feel in the painful area?	
<input type="checkbox"/> The painful area does not feel different from the non-painful area.	0
<input type="checkbox"/> I feel numbness or tenderness in the painful area that is different from the non-painful area.	3
Total score:	

Scoring a score of 12 or more suggests pain of predominantly neuropathic origin

Source: Bennett, M *et al* J Pain, Vol 6, No 3 March, 2005 pp 149–158 The S-LANSS Score for Identifying Pain of Predominantly Neuropathic Origin: Validation for Use in Clinical and Postal Research.

Testing to identify the location of nerve damage

Investigations beyond the clinical examination and routine laboratory tests are considered on a case-by-case basis.¹⁰

Additional testing following consultation with a neurologist or pain specialist may include:⁹

- Nerve conduction studies, electromyography or laser-evoked potentials
- MRI or CT scan for assessment of stroke or radiological evidence of nerve compression
- Genetic testing to identify hereditary polyneuropathies if the patient has a family history
- Nerve biopsy to identify inflammatory diseases, after less invasive investigations have been undertaken¹⁷
- Skin biopsy can detect small fibre sensory neuropathies, e.g. diabetic polyneuropathy and should be taken from the distal leg, e.g. 10 cm above the external malleolus.¹⁶

Treating pain and reducing its effect on the patient

Successful treatment of long-term pain includes keeping patients active and engaged in their daily life.

The following points should be considered when developing a treatment plan:^{1, 18}

- Sleep deprivation can exacerbate pain: more than 70% of patients with long-term pain are reported to have problems with sleep
- Can the patient carry out daily tasks, e.g. dressing or washing? Patients with trigeminal neuralgia may have difficulty eating and weight gain from reduced activity can lead to further limitations in mobility in some patients.

- Participation in family, work and social life
- Depression or anxiety may develop due to long-term pain or clinical uncertainty


Establish specific goals

Develop goals of care with the patient that are relevant to the patient and achievable, e.g. being able to walk the dog by the next consultation.

Non-pharmacological approaches to reducing pain

Discuss “sleep hygiene” techniques with patients who have disturbed sleep. This emphasises reserving the bed and bedroom for sleeping and sex. Advise patients to avoid watching television or using electronic devices in the bedroom and to leave the room if they are awake for longer than 15 minutes, returning only when tired enough to sleep. Other advice includes avoiding stimulants or diuretics, including alcohol close to bed-time and keeping a regular sleep routine, even during weekends.

Tricyclic antidepressants, taken in the evening, may provide the dual benefit of pain relief and sedation overnight (see: “The pharmacological treatment of neuropathic pain”).

 For further information on sleep hygiene, see: “Managing insomnia”, BPJ 14 (Jun, 2008)

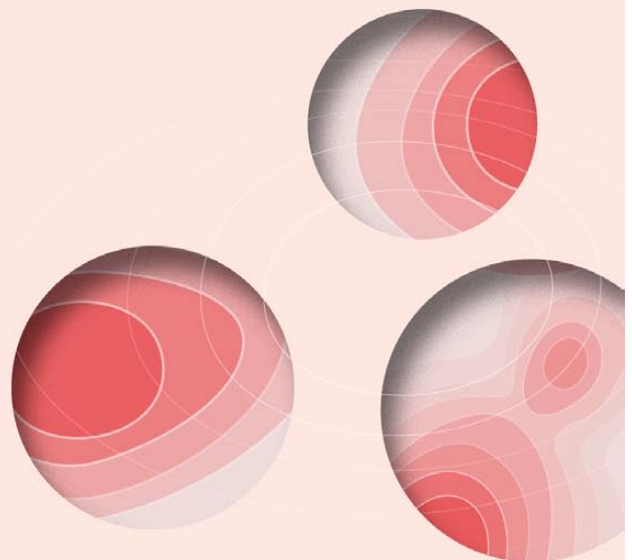
Staying active and engaged is a priority

If patients withdraw from their normal activities, discuss the reasons and consider whether any medicines require alteration. Some patients may benefit from consultation with an occupational therapist, physiotherapist or counsellor. Patients with low activity levels may benefit from supervised exercise or a Green Prescription.

Sensory nerves and the sensations they carry

Sensory nerve fibres which can be affected in patients with neuropathic pain include:^{14, 15}

- Unmyelinated (small) C fibres:
 - Conduct pain and temperature sensations
 - Cause unpleasant, indefinite, burning sensations
- Myelinated (larger) A cutaneous fibres:
 - A β fibres – conduct touch and pressure sensation, but may also contribute to noxious pain sensations in neuropathic pain
 - A δ fibres – conduct cold, touch and pain sensations, thought to convey immediate, well-localised pricking pain



Persistent pain is a strong risk factor for falls in older patients; exercise improves strength and balance.¹⁹ Improving mobility to maintain independence and social participation will be a goal of care for many patients.

Planning can help patients fulfil their usual activities

People with long-term pain often tire quickly. Suggest patients devote time to important tasks early in the day or when they have the most energy; this may lead to a greater sense of control over their condition.

Support groups help patients connect and learn from others

Advice and information for patients about how to manage and live with long-term pain is available from:

- Self-management courses through a range of Primary Health Organisations, see: www.healthnavigator.org.nz/healthy-living/self-care/programmes-courses/stanford-self-management-programme/
- Arthritis New Zealand: www.arthritis.org.nz/information/treatment-management/living-a-healthy-life/
- National Health Service (NHS) Choices: www.nhs.uk/Livewell/Pain/Pages/Painhome.aspx
- The Pain Toolkit, pain self management tools: www.paintoolkit.org/tools
- New Zealand Pain Society: www.nzps.org.nz

Consider if support devices are necessary

Some patients may benefit from assistive devices, such as toilet and shower rails. Occupational therapists may be able to organise subsidised installations of these supports for select patients. Patients at risk of falls may benefit from a medical alarm.

Other non-pharmacological approaches have limited evidence of efficacy

Interventions such as acupuncture or transcutaneous electrical nerve stimulation (TENS) may be useful for patients who have ongoing pain despite medication.²⁰ There have been few clinical trials, however, assessing their efficacy or comparing their effects with pharmacological treatments.

Psychological approaches, such as cognitive-behavioural therapy (CBT), may help patients adapt to living with pain, however, there is little evidence to suggest they reduce neuropathic pain.

The pharmacological treatment of neuropathic pain

The medicines recommended for the initial treatment of neuropathic pain include (see Table 2 for dosing):^{1,21}

- TCAs, such as amitriptyline and nortriptyline – fully subsidised, unapproved indication. Amitriptyline is the TCA with the strongest evidence of effectiveness and is favoured in some international guidelines.^{1,21} However, nortriptyline has less anticholinergic activity than amitriptyline and less adverse effects in elderly patients.^{22,23}
- Gabapentin – fully subsidised with Special Authority approval for patients diagnosed with neuropathic pain. The related medicine pregabalin has a similar efficacy and adverse effect profile as gabapentin, and is available unsubsidised. Be mindful that co-administration of morphine can increase levels of gabapentin and dosing may need to be adjusted.²⁴
- Carbamazepine – initial treatment for patients with trigeminal neuralgia or diabetic polyneuropathy; fully subsidised.

These medicines have evidence of efficacy and are first or second-line treatments for neuropathic pain;^{21,25} guidelines do not favour one initial medicine over another.^{1,21} For most neuropathic pain medicine efficacy is not dependent on the underlying cause, therefore, potential adverse effects may dictate the choice of treatment, e.g. patients with central neuropathic pain may be less tolerant of medicines with CNS adverse effects.²¹ The patient's need for analgesia may fluctuate and regular follow-up is important. For patients with central-post stroke pain there is evidence that medicines are less effective.²⁶

Tricyclic antidepressants, gabapentin, either alone or in combination, and carbamazepine (for trigeminal neuralgia and diabetic neuropathy) are appropriate options for treating most types of neuropathic pain in primary care. Alternative anticonvulsant medicines, e.g. valproate or lamotrigine may be trialled in primary care, however, there is limited evidence of effectiveness in patients with neuropathic pain.

The role of non-steroidal anti-inflammatory drugs (NSAIDs) is unclear.

NSAIDs are not included as treatment options in neuropathic pain guidelines.^{1,27} However, there is insufficient evidence to conclude that they are ineffective, and in practice NSAIDs are often used by patients with neuropathic pain.²⁸ It is possible that NSAIDs have an effect in patients with mild pain or pain with an inflammatory component or they may have a placebo effect in some patients.²⁸

Other antidepressants have a limited role in the treatment of neuropathic pain.

TCAs are preferred over selective noradrenaline reuptake inhibitors (SNRIs) or selective serotonin reuptake inhibitors (SSRIs) for patients with neuropathic pain. Venlafaxine is the only SNRI available in New Zealand which has evidence of efficacy in the treatment of neuropathic

Table 2: Possible first line medicines for the treatment of neuropathic pain.^{1,21,29}

Medicine	Fully subsidised formulations	Recommended dosing
<p>Tricyclic antidepressants (amitriptyline, nortriptyline; unapproved indication)</p>	<p>Amitriptyline:</p> <ul style="list-style-type: none"> ■ 10 mg, 25 mg, 50 mg tablets <p>Nortriptyline:</p> <ul style="list-style-type: none"> ■ 10 mg, 25 mg tablets 	<ul style="list-style-type: none"> ■ Begin with 10 mg, daily, taken at night ■ Increase gradually to up to 75 mg, daily, taken at night ■ Occasionally, higher doses may be required; supervision from a neurologist or pain clinic recommended
	<p>Gabapentin Capsules:</p> <ul style="list-style-type: none"> ■ 100 mg, 300 mg, 400 mg 	<ul style="list-style-type: none"> ■ Begin with 300 mg, taken either once daily or three times daily ■ Increase every two or three days by 300 mg, daily, to at least 1200 mg, daily, divided so that capsules are taken three times during the day ■ Maximum dose 3600 mg, daily ■ Lower doses should be used in patients with renal impairment
<p>Carbamazepine first-line for the treatment of trigeminal neuralgia</p>	<p>Immediate-release tablets:</p> <ul style="list-style-type: none"> ■ 200 mg and 400 mg ■ Tablets may be halved <p>Extended-release tablets:</p> <ul style="list-style-type: none"> ■ 200 mg and 400 mg ■ Tablets may be halved, but not crushed <p>Oral liquid:</p> <ul style="list-style-type: none"> ■ 100 mg/5 mL 	<ul style="list-style-type: none"> ■ Begin with 100 mg, once or twice daily ■ Increase every two weeks in 100–200 mg increments; 1200 mg is usually sufficient ■ For immediate-release tablets divide dose so that tablets are taken three or four times during the day ■ For extended-release tablets divide dose so that tablets are taken twice during the day ■ Slightly higher doses of extended release tablets may be required for equivalent effect

* For a full list of adverse effects, contraindications and cautions, see: www.nzf.org.nz

† NNT defined as at least moderate pain relief (30–50% reduction in pain); NNH defined as the number of patients needed to treat for one patient to discontinue medicine due to adverse effects

Time of onset of pain relief	Adverse effects and cautions*	Approximate number-needed-to-treat (NNT) [†]	Approximate number-needed-to-harm (NNH) [†]
<ul style="list-style-type: none"> ■ Dosing of tricyclic antidepressants needs to increase over a course of weeks ■ Some patients may gain adequate pain relief at lower doses used within first two weeks, others may require up to four weeks until adequate dosing and pain relief is achieved 	<ul style="list-style-type: none"> ■ Drowsiness ■ Anxiety ■ Agitation ■ Confusion ■ Constipation ■ Dizziness ■ Use with caution in patients with cardiovascular disease (may cause QT prolongation and arrhythmia), diabetes (potential changes in weight and glucose control), epilepsy or urinary retention and patients at risk of angle-closure glaucoma 	4	13
<ul style="list-style-type: none"> ■ Effects may be felt within one to two weeks, with maximal relief occurring over four to eight weeks^{32, 33} 	<ul style="list-style-type: none"> ■ Dizziness ■ Headache ■ Drowsiness ■ Gastrointestinal effects such as nausea, vomiting, diarrhoea, constipation and abdominal pain ■ Lower limb oedema ■ Cough ■ Neurological effects such as anxiety and nervousness, confusion, hostility, and abnormal thoughts 	6	26
<ul style="list-style-type: none"> ■ Can provide relief from trigeminal neuralgia within a few days³¹ 	<ul style="list-style-type: none"> ■ Nausea ■ Vomiting ■ Constipation ■ Dry mouth ■ Drowsiness ■ Fatigue ■ More serious adverse effects include hyper- or hypotension, aggression, hallucinations and hepatic adverse effects ■ Carbamazepine should be used with caution in a range of patients. It is a strong inducer of P450 enzymes, can reduce the efficacy of oral contraceptives, and can cause drug-induced hypersensitivity reactions 	2	3

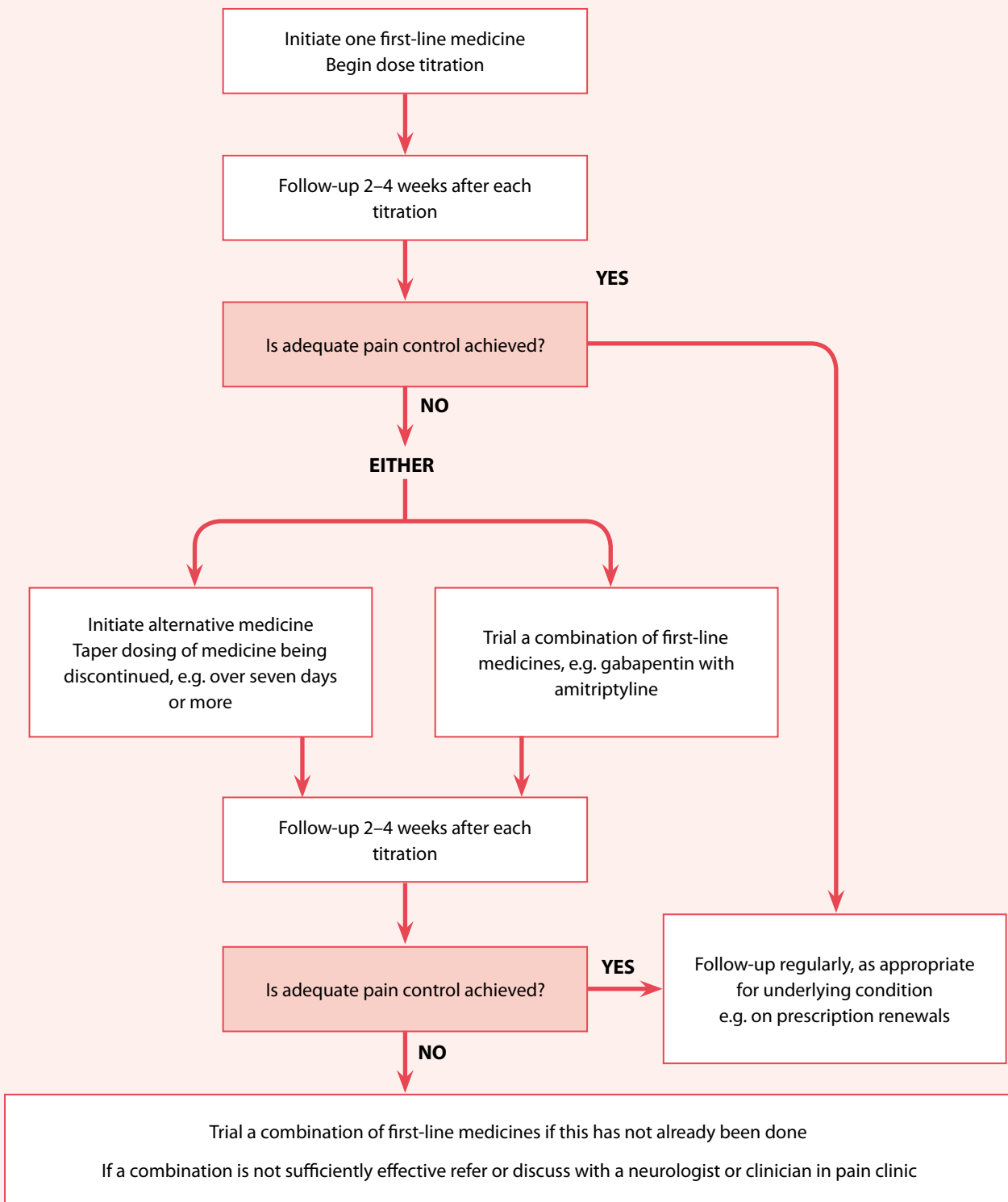


Figure 1. Optimising analgesia for neuropathic pain, except trigeminal neuralgia.^{1,5}

pain.²⁹ Consultation with a neurologist or clinician in a pain clinic is recommended before using venlafaxine as a treatment for patients who have not responded sufficiently to initial treatment. Duloxetine is recommended as a potential first-line treatment for neuropathic pain by international guidelines,¹ however, this medicine is not available in New Zealand.

There is currently no evidence that SSRIs are effective in the treatment of neuropathic pain.²¹

Opioids are reserved for patients with severe neuropathic pain due to the potential adverse effects, including dependency.^{2, 21} Discussion with a clinician experienced in treating pain is recommended before prescribing opioid-based medicines for patients with neuropathic pain that is not controlled by other approaches.

Topical treatment of cutaneous neuropathic pain

Topical capsaicin cream (0.075%) is a treatment option for patients with localised, cutaneous neuropathic pain.²¹ It is fully subsidised by prescription endorsement for patients diagnosed with post-herpetic neuralgia or diabetic peripheral neuropathy. Capsaicin cream produces a burning sensation on application; patients should wash hands immediately after application and avoid transferring the product to eyes or mucous membranes.²⁹

Lidocaine 2.5% + prilocaine 2.5% patches (unsubsidised) may be a second-line topical analgesic for patients who do not tolerate capsaicin cream.²¹


Initiating and optimising treatment


For most analgesics, dosing should start low and be titrated upwards. Practice points for optimising treatment for neuropathic pain are shown in Figure 1. Treatment success is measured by subjective assessment of the patient's level of pain, sleep disruption and ability to function in daily life.

If sufficient control is not obtained with the first medicine trialled, switch to another of the recommended initial medicines, e.g. from a TCA to gabapentin, or use a combination of medicines. If medicines are switched, gradually reduce the dose of the medicine being withdrawn, e.g. over seven days or more, while the new medicine is initiated in order to provide continuous treatment. If medicines are combined, aim for a combination of medicines that have different analgesic mechanisms.³⁰ Combination treatment with a TCA and gabapentin provides better outcomes, at lower doses, without additional adverse effects, than either of these medicines alone in higher doses. For example, nortriptyline 50 mg, daily, with gabapentin 2000 mg, daily in divided doses, provided better pain relief in one study than nortriptyline 60 mg, daily or gabapentin 2250 mg, daily.³⁰

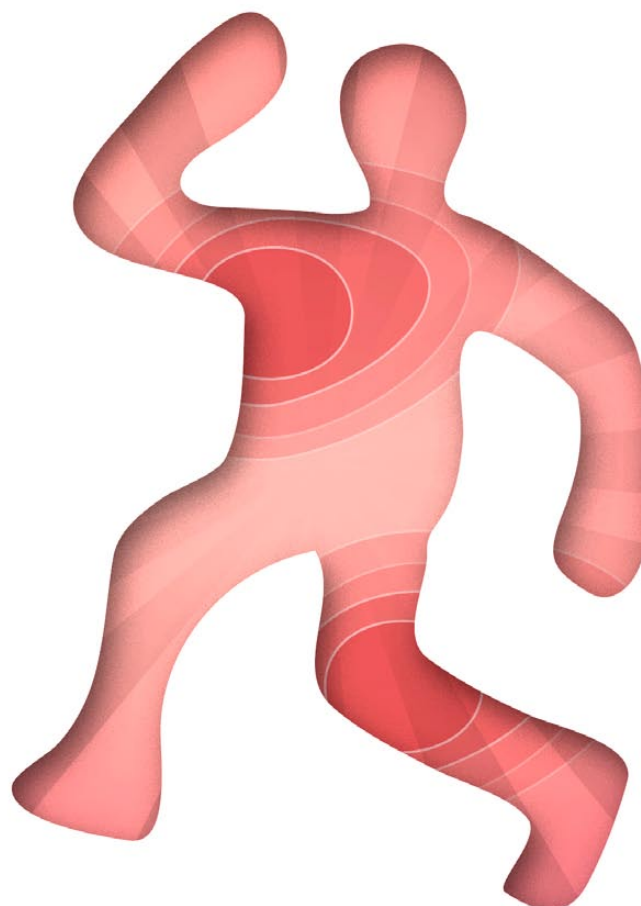
Treating trigeminal neuralgia

Carbamazepine should be the initial treatment trialled for patients with trigeminal neuralgia.¹ If carbamazepine is ineffective, consider consulting with a neurologist or pain clinic as there is little evidence from clinical trials to guide prescribing of other medicines.^{1, 31} Lamotrigine or baclofen (unapproved indications) have been suggested as potential second-line treatment options for patients with trigeminal neuralgia.³¹

 **Best practice tip:** Consider strategies for reducing “piles of pills” for patients with long-term pain, e.g. more frequent dispensings during an analgesic trial. Patients with chronic pain may have comorbidities and be taking a number of medicines; consider whether they are eligible for the Long Term Condition service to simplify their medicine regimen.

 For further information, see: “Piles of pills: Prescribing appropriate quantities of medicines”, BPJ 69 (Aug, 2015).

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