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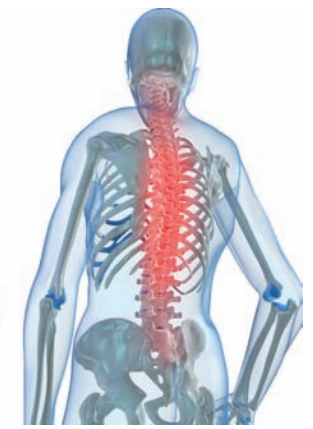

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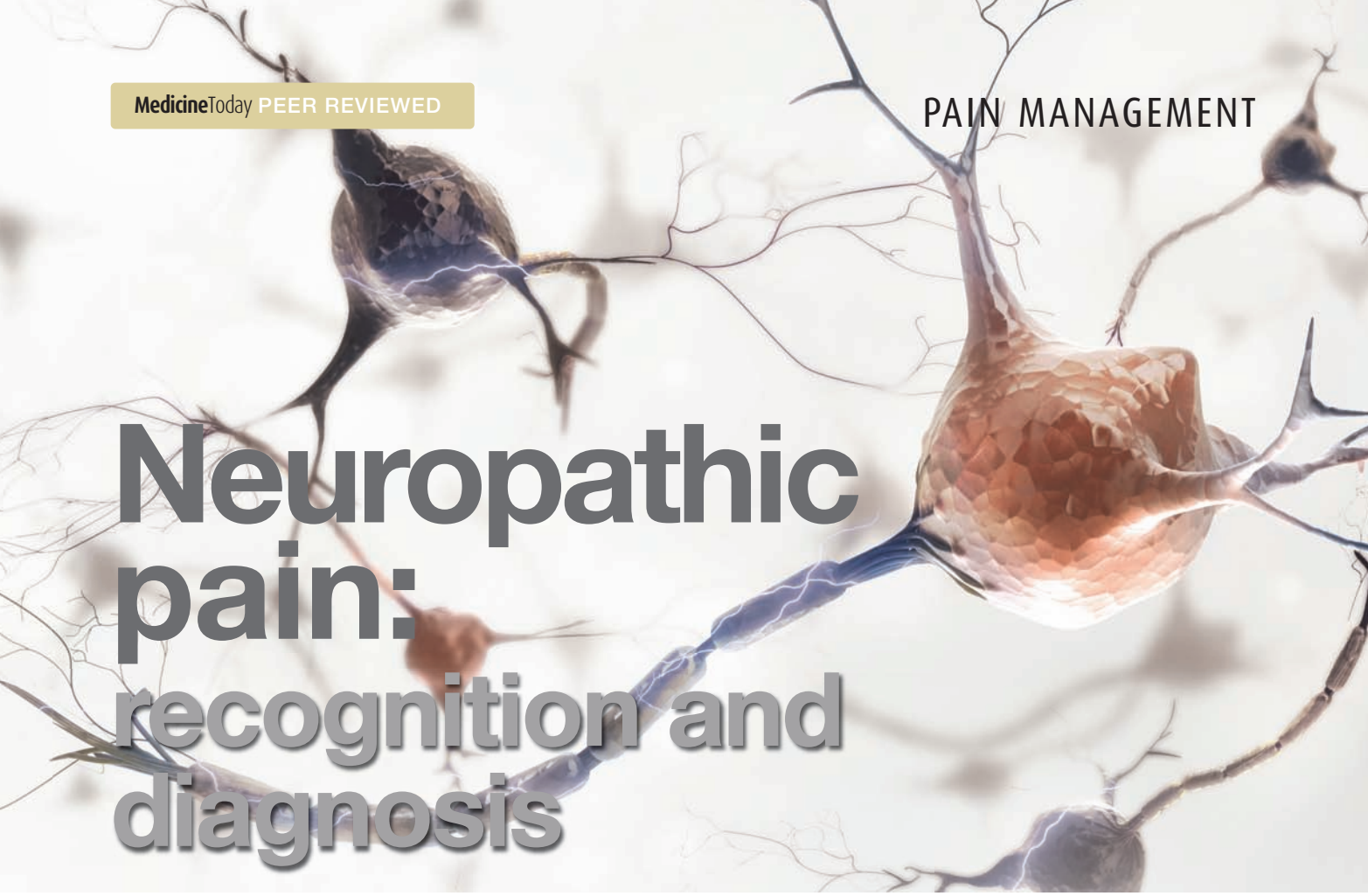

Neuropathic pain: recognition and diagnosis

GUY BASHFORD MB BS, Dip MSM, FAFRM, FFPMANZCA

Despite the complex pathophysiology of neuropathic pain, diagnosis is usually straightforward, relying on negative and positive symptoms and signs in the presence of a condition that can damage the somatosensory system.

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Neuropathic pain is a common cause of persistent pain in the general practice population. It is defined as 'pain arising as a direct consequence of a lesion or disease affecting the somatosensory system'.[1] This replaces the less strict definition used in the past – pain initiated or caused by a primary lesion or dysfunction in the nervous system.

In a significant proportion of patients, neuropathic pain arises when disease or trauma affects the sensory peripheral nerves or somatosensory regions of the spinal cord or brain. The most common causes of neuropathic pain and their prevalence are shown in the Table.

Most neuropathic pain is due to ectopic nerve activity in damaged peripheral nerves and complex neuroplastic changes occurring in the spinal cord and brain in response to the damaged somatosensory nervous system – termed central sensitisation. Similar central sensitisation is seen in conditions such as complex regional pain syndrome type I and fibromyalgia, but without evidence for an underlying nerve lesion.

Nociceptive pain, in contrast, is caused by tissue damage or potentially tissue-damaging stimuli. However, even in seemingly straightforward cases of nociceptive pain, such as in osteoarthritis of the knee, persistent pain often leads to central nervous system changes that blur the correlation between the severity of tissue damage and the pain perceived by the individual. In addition, neuropathic pain is not a diagnosis of exclusion and often coexists with nociceptive pain.

Patients with neuropathic pain have, on average, greater levels of distress and psychosocial comorbidities than those with nociceptive pain.[2] This is likely to be due in part to delayed diagnosis and to inaccurate psychiatric or nociceptive labels being used to explain the patient's symptoms.

It is important to identify neuropathic pain for the following reasons:

- to avoid frustrating and potentially dangerous diagnostic efforts seeking a nonexistent source of nociceptive pain
- to help the patient and their family understand the basis of their pain as a first step to managing it
- to provide appropriate treatment, as neuropathic pain responds to pharmacological and nonpharmacological therapies largely different from those used for nociceptive pain; for example, neuropathic pain is usually refractory to simple analgesics but may respond to tricyclic antidepressants such as amitriptyline, serotonin and noradrenaline reuptake inhibitors (e.g. venlafaxine, duloxetine) and certain antiepileptic drugs (e.g. gabapentin, pregabalin).

Management of neuropathic pain will be discussed in the second part of this article commencing on page 7 of this collection.

TABLE. COMMON CAUSES AND PREVALENCE OF NEUROPATHIC PAIN

Cause of pain	Prevalence (per 100,000)
Sciatica (a mixed pain)	2100
Diabetic peripheral neuropathic pain	600
Postherpetic neuralgia	500
Cancer-related neuropathic pain	200
Spinal cord impairment	120
Multiple sclerosis	51
Phantom limb pain	50
Central poststroke pain	30
Trigeminal neuralgia	15

RECOGNITION OF NEUROPATHIC PAIN IN GENERAL PRACTICE

The prevalence of neuropathic pain is around 7 to 8% in the general population; however, it is higher among people who have conditions that damage the peripheral or central somatosensory nervous system.[3,4] In diabetes and stroke, a minority of patients will develop neuropathic pain (around 11% and 8%, respectively), while in other, less common, conditions such as multiple sclerosis and spinal cord injury, the prevalence of neuropathic pain may be much higher (over 50%). Postherpetic neuralgia is another well-recognised cause of neuropathic pain.

Postsurgical neuropathic pain is potentially the most common form of neuropathic pain, although its prevalence in the general population is unclear. Some types of surgery result in neuropathic pain in almost 50% of patients (e.g. breast, hernia and thoracotomy procedures). Other common procedures such as total knee arthroplasty can lead to persistent neuropathic pain in 10 to 15% of cases. The understandable desire of GPs and surgeons to identify complications of surgery can enormously delay diagnosis of neuropathic pain and lead to inappropriate treatments.

In some disease states, such as stroke, the onset of neuropathic pain can be delayed and sensory deficits can be subtle, making diagnosis difficult. In people with diabetes, neuropathic pain due to polyneuropathy must be distinguished from vascular, musculoskeletal and infective causes of pain.

HISTORY AND PHYSICAL EXAMINATION

The complicated pathophysiology of neuropathic pain can lead doctors to believe diagnosis is more difficult than it is in reality. The first step is recognising the presence of a condition in which the peripheral or central nervous system may have been damaged and where underlying tissue damage is unlikely to be causing the pain. Relatively straightforward history taking and physical examination are then likely to provide the diagnosis.

History taking should seek negative and positive symptoms:

- negative symptoms reflect reduced sensation and are present in a distribution corresponding to the proposed peripheral or central nervous system lesion; these symptoms may be subtle (e.g. reduced peripheral sensation in people with diabetes, reduced light touch, hot or cold sensation in patients with stroke, and reduced sensation to one side of a surgical scar)
- positive symptoms include:
  - spontaneous pain (pain arising without a stimulus)
  - hyperalgesia (increased pain sensitivity to a nociceptive or painful stimulus)
  - allodynia (pain in response to a non-nociceptive stimulus).

Because of the involvement of the central nervous system in the development of positive symptoms such as allodynia and hyperalgesia, these symptoms are not always confined to the distribution of the damaged peripheral nerve.

DN4 SCREENING QUESTIONNAIRE FOR NEUROPATHIC PAIN[7]

Each characteristic or symptom is checked yes or no, and each yes answer is scored one point, giving a score out of 10. A score of 4 or more is diagnostic of neuropathic pain.

Interview of the patient

1. **Does the pain have one or more of the following characteristics?**
- Burning
- Painful cold
- Electric shocks

2. **Is the pain associated with one or more of the following symptoms in the same area?**
- Tingling
- Pins and needles
- Numbness
- Itching

Examination of the patient

3. **Is the pain located in an area where the physical examination reveals one or more of the following characteristics?**
- Reduced touch sensation
- Reduced pinprick sensation

4. **In the painful area, can the pain be caused or increased by:**
- Brushing?

A history of pain evoked by light touch such as clothes or bedclothes, an abnormal response to hot or cold sensations or spontaneous pain in an area of numbness suggest neuropathic pain. Another characteristic clinical feature is summation, where repetitive stimulation evokes progressively worsening pain. Paraesthesias (e.g. tingling or the sensation of ants crawling under the skin) are more common in patients with neuropathic pain than in those with nociceptive pain.

Complex neurological examinations are often undertaken in research settings but are usually not necessary for the diagnosis of neuropathic pain in general practice. The physical examination should include an attempt to identify an area of reduced sensation in an anatomical area consistent with the presumed underlying neurological disease or trauma. Examples include stocking sensory loss in a person with diabetes and numbness to one side of a surgical scar. Allodynia can be elicited by the stroke of a brush. In some circumstances, GPs may wish to test with other stimuli, such as cold or heat, but this is rarely necessary for diagnosis.

The likelihood of neuropathic pain can be graded using an algorithm outlined by Treede and colleagues.[1] In the presence of pain with a distinct neuroanatomically plausible distribution and a history suggesting a relevant lesion or disease, neuropathic pain is considered:
- definite if both the distribution of the pain and the relevant lesion have been confirmed by at least one test (which may include clinical sensory examination for distribution)
- probable if either the distribution or the lesion has been confirmed by at least one test
- possible if neither the distribution nor the lesion has been confirmed by a test.

SCREENING TOOLS

Several screening tools for neuropathic pain are recommended in recent guidelines and can be of use to GPs.[5,6] The simplest of these are:
- the DN4 (Douleur Neuropathique-4) questionnaire, which gives a score out of 10 after two simple historical questions have been answered and two simple physical examination techniques performed.[7] It formalises the recommended history and physical examination for GPs (see the box on this page)
- Pain Detect, which is a self-report questionnaire (available online at http://www.virtualmedicalcentre.com/calc_pfizer_pain_detect.asp).

As with all forms of persistent pain, it is important for GPs to regularly assess the psychosocial aspects of the patient's pain as well as the biological components. The Brief Pain Inventory can assist with this.[8]

OTHER INVESTIGATIONS

Radiological or electrophysiological testing may provide additional evidence for a disease or lesion involving the somatosensory nervous system (e.g. a thalamic stroke). However, it is important to remember that standard nerve conduction studies are relatively insensitive for detecting A-delta or C-fibre dysfunction. Damage to these small sensory fibres is a frequent cause of neuropathic pain.

At a research level, functional neuroimaging (positron emission tomography and functional MRI) provides enormous insight into the neuroplastic nature of pain and is likely to help accelerate the development of more effective treatments for neuropathic pain.

SUMMARY

- Neuropathic pain is a common cause of persistent pain in the general practice population and results from damage to the peripheral or central parts of the somatosensory nervous system. Some of the central pathophysiology of neuropathic pain is shared with other types of persistent pain.
- Nociceptive causes of pain should also be considered, but neuropathic pain is not a diagnosis of exclusion and often both types of pain are present in the same patient.
- Diagnosis of neuropathic pain is important as it helps patients to understand that they are suffering from real and distressing symptoms in the absence of tissue damage and allows early treatment with

appropriate pharmacological and nonpharmacological therapies.

- It is important to identify individuals with disease processes that are likely to lead to neuropathic pain, including persistent postsurgical pain.
- Screening tools such as DN4 and Pain Detect can be of assistance to GPs.
- History taking and physical examination need not be complicated; they involve seeking negative and positive sensory symptoms and signs consistent with a sensory lesion and the resulting pain.
- Patients with neuropathic pain have, on average, greater distress and psychosocial comorbidity than those with nociceptive pain. MT

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Neuropathic pain

A guide to clinical management

GUY BASHFORD MB BS, Dip MSM, FAFRM, FFPMANZCA

Treatment selection for neuropathic pain depends as much on comorbid sleep disturbance, depression, anxiety and particularly in the elderly, medical comorbidities as on the pain itself.

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Most neuropathic pain in the community goes unrecognised and untreated. Even when patients and their general practitioners recognise neuropathic pain – defined as 'pain arising as a direct consequence of a lesion or disease affecting the somatosensory system'[1] – they often adopt a nihilistic attitude to treatment. Many of the medications commonly used for nociceptive pain (pain caused by tissue damage or potentially tissue-damaging stimuli) are not effective in neuropathic pain. Likewise, many physical approaches for managing nociceptive pain may not be appropriate for neuropathic pain. Research into treatments for neuropathic pain has, however, expanded rapidly in the past decade, and there is growing consensus about management of this type of pain.

APPROACH TO MANAGEMENT

The first step in the management of neuropathic pain is recognising and diagnosing its presence; this was discussed in an article in the May 2013 issue of *Medicine Today*.[2] Common causes of neuropathic pain include sciatica, surgical nerve damage, diabetic neuropathy and postherpetic neuralgia. The diagnosis of neuropathic pain allows a concerted effort to educate the patient and their family about this pain. Without this education, distressing symptoms will likely be attributed to primary nociceptive or physical causes.

Not surprisingly, psychological and social comorbidities are generally more common in patients with neuropathic pain than in those with nociceptive pain. For instance, spontaneous pain is extremely common in neuropathic pain and is likely to disturb sleep. Selecting appropriate pharmacological and nonpharmacological management approaches for an individual depends as

TABLE. TREATMENT OF NEUROPATHIC PAIN: RECOMMENDATIONS OF RECENT GUIDELINES*

Medication	Neuropathic Pain Special Interest Group (2007)[4]	European Federation of Neurological Societies (2010)[5]
Tricyclic antidepressants	First line	First line
Alpha-2-delta ligands (gabapentin, pregabalin)	First line	First line
SNRIs (duloxetine, venlafaxine)	First line for painful polyneuropathy	First line for painful polyneuropathy
Topical lignocaine	First line for localised peripheral neuropathy	First line for postherpetic neuralgia if small area of pain/allodynia
Opioid analgesics	Second line except in selected third-line circumstances	Second to third line for painful polyneuropathy, postherpetic neuralgia and central pain
Tramadol	Second line except in selected circumstances	Second to third line for painful polyneuropathy, postherpetic neuralgia

ABBREVIATION: SNRI = serotonin and noradrenaline reuptake inhibitor.
* Excluding treatment of trigeminal neuralgia and sciatica.

much on comorbidities such as sleep disturbance, depression and anxiety as on the pain. It is also influenced by the presence of other medical conditions, particularly in the elderly. It is therefore imperative that comorbidities are identified and their severity assessed before deciding on a specific drug or nondrug treatment approach for neuropathic pain.

DRUG TREATMENTS FOR NEUROPATHIC PAIN

Of the approximately 200 randomised controlled trials on neuropathic pain, around 60% were published within the past five years, and there is increasing agreement between evidence-informed treatment recommendations.[3-5] Recent guidelines for the treatment of neuropathic pain are summarised in the Table.

However, it must be recognised that the effects of proven medications are often disappointing, and their use at scientifically proven therapeutic doses may be limited by adverse effects. Complete resolution of neuropathic pain is very rare. Nevertheless, a 30% reduction in pain is likely to improve quality of life. It is important to ensure patients have realistic expectations before initiating treatment. They should understand that even with the best proven medications, only a quarter to a third of patients achieve pain relief of 50% or more.

Ineffective or unproven medications

Nearly all patients presenting with neuropathic pain will have tried paracetamol or an NSAID for their symptoms. There is no evidence that paracetamol is effective in neuropathic pain, except in combination with tramadol. There is also no evidence supporting the use of NSAIDs in neuropathic pain states.

Antidepressants

There is much evidence supporting the use of antidepressants with combined serotonin and noradrenaline reuptake inhibitory effects in neuropathic pain. Tricyclic antidepressants have been a mainstay of treatment for decades. There is level 1 evidence of their effectiveness in a variety of peripheral pain states (e.g. painful peripheral neuropathy and postherpetic neuralgia) and, to a lesser extent, central pain states (central spinal cord injury and poststroke pain).

The Neuropathic Pain Special Interest Group of the International Association for the Study of Pain recommends a tricyclic antidepressant dose of 25 to 100 mg, where appropriate and safe.[4] The group also recommends a six to eight-week trial, which should include two weeks at the maximum dose. Achieving a therapeutic dose is often limited by side effects and safety, particularly in the elderly and those with cardiac or neurological comorbidities. The group recommends screening ECGs in all patients older than 40 years.

Side effects of tricyclic antidepressants are shown in the box on page 9. As these drugs cause sedation, with careful titration they can potentially improve sleep without causing troubling daytime drowsiness.

Serotonin and noradrenaline reuptake inhibitors (SNRIs; duloxetine and venlafaxine) have a safer and more tolerable side effect profile than a tricyclic antidepressant. Use of duloxetine (60 mg daily)

is supported by level 1 evidence in patients with diabetic peripheral neuropathic pain. In many cases it is a safer alternative than a tricyclic antidepressant, especially where depression is significant and a full antidepressant dose is required. There is evidence of efficacy for venlafaxine at higher doses (150 to 225 mg daily), at which levels noradrenaline reuptake inhibitor activity becomes apparent.

The selective serotonin reuptake inhibitors have much less evidence of effectiveness in neuropathic pain than the tricyclic antidepressants or SNRIs.

Alpha-2-delta ligands

The anticonvulsants gabapentin and pregabalin have both been studied widely in patients with neuropathic pain and, along with tricyclic antidepressants, represent first-line options for the treatment of this type of pain. They are believed to act through binding to the alpha-2-delta subunits of calcium channels in the central nervous system, reducing the influx of calcium into neurons and in turn decreasing neurotransmitter release.

Gabapentin and pregabalin are relatively safe alternatives to antidepressants, with their main side effects being dose-related drowsiness or dizziness and, less frequently, peripheral oedema and weight gain. As with tricyclic antidepressants, careful titration of these sedating drugs can improve sleep without causing daytime drowsiness. These agents have generally been demonstrated to be anxiolytic but not antidepressant in study groups with neuropathic pain.

Other anticonvulsants

Carbamazepine remains the first-line treatment for the relatively rare condition of trigeminal neuralgia but is not generally used for other forms of neuropathic pain. The evidence is equivocal for its efficacy in the more common forms of neuropathic pain, and patients require careful blood monitoring for the development of blood dyscrasias and hepatotoxicity. The evidence is likewise mixed for the efficacy of sodium valproate in neuropathic pain. Lamotrigine is occasionally used for neuropathic pain and this is supported by some trial evidence; its major adverse effects are serious dermatological conditions such as Stevens–Johnson syndrome.

Opioids

Traditionally, neuropathic pain has been considered resistant to treatment with opioids. In fact, randomised controlled trials show that the number needed to treat (NNT) with opioids in neuropathic pain is quite similar to the NNT with tricyclic antidepressants and also to the NNT with opioids in trials on nociceptive pain. In spite of this, with the exception of some forms of acute neuropathic pain and cancer pain, opioids are generally recommended as third-line agents because of the frequency of adverse effects and uncertainty about their long-term efficacy.[4,5]

There is trial evidence supporting the use of tramadol, which is both an SNRI and a weak mu opioid receptor agonist, in a number of neuropathic pain states. It may be recommended as a second- or third-line treatment for patients who are not taking, and are not likely to later require, an antidepressant as part of pain management.

Topical treatments

Lignocaine transdermal patches have level 1 evidence of effectiveness in relatively localised forms of neuropathic pain such as postherpetic neuralgia. Capsaicin cream has limited evidence of effectiveness and is not commonly tolerated in the longer term.

Combination therapies

With the recommended treatments requiring an average of three people to be treated to reduce one patient's pain by 50%, it is clear that many patients will require serial trials or a combination trial of proven medications. Theoretically, combining drugs with different mechanisms of action and different adverse effects has the potential to provide better pain relief with fewer side effects. Results of recent trials have been promising, with combinations of antidepressants, alpha-2-delta ligands and opioids reducing both side effects and NNTs.[6]

COMMON SIDE EFFECTS OF FIRST-LINE ORAL DRUGS FOR NEUROPATHIC PAIN

Tricyclic antidepressants
- Drowsiness
- Confusion
- Dry mouth
- Orthostatic hypotension
- Weight gain
- Urinary retention
- Cardiac arrhythmia

Serotonin and noradrenaline reuptake inhibitors
- Nausea
- Dry mouth
- Drowsiness
- Sexual dysfunction

Alpha-2-delta ligands
- Dizziness
- Drowsiness
- Peripheral oedema
- Weight gain

NONPHARMACOLOGICAL APPROACHES

When the response to proven medications is not satisfactory then adopting a broader rehabilitation approach is likely to be preferable to trials of high doses or combinations of unproven medications. Few randomised controlled trials have assessed nonpharmacological approaches to neuropathic pain, in contrast to osteoarthritic and nonspecific low back pain.

Cognitive behavioural therapies and especially pain management groups have

excellent face validity, and should be considered whenever psychosocial comorbidities are significant or when responses to pharmacological approaches are unsatisfactory. Australian physiotherapists have been at the forefront of research on the use of guided motor imagery, including mirror box therapy, with NNTs in complex regional pain syndrome being similar to the NNTs with the far more expensive and invasive option of spinal cord stimulation.

SUMMARY

- Research into treatments for neuropathic pain has expanded rapidly in the past decade, and there is growing consensus about its management.
- Management should include education of patients and their families about neuropathic pain and how it differs from nociceptive pain.
- Selection of first-line medications for treatment of neuropathic pain should take into account the presence and severity of the common comorbidities of sleep disturbance, depression and anxiety, and also, particularly in the elderly, medical comorbidities.
- Tricyclic antidepressants, SNRI antidepressants (duloxetine and venlafaxine) and alpha-2-delta ligands (gabapentin and pregabalin) are first-line choices for most types of neuropathic pain, with tramadol and opioids being second- or third-line options. Topical lignocaine patches are a first-line choice for localised neuropathic pain.
- If single agents are not sufficiently effective or cause intolerable side effects at high doses then judicious combination therapy using drugs with different mechanisms of action and largely different side effects may be an appropriate therapeutic alternative.
- If the response to proven medications is unsatisfactory then adopting a broader rehabilitation approach is preferable to trials of high doses or combinations of unproven medications. MT

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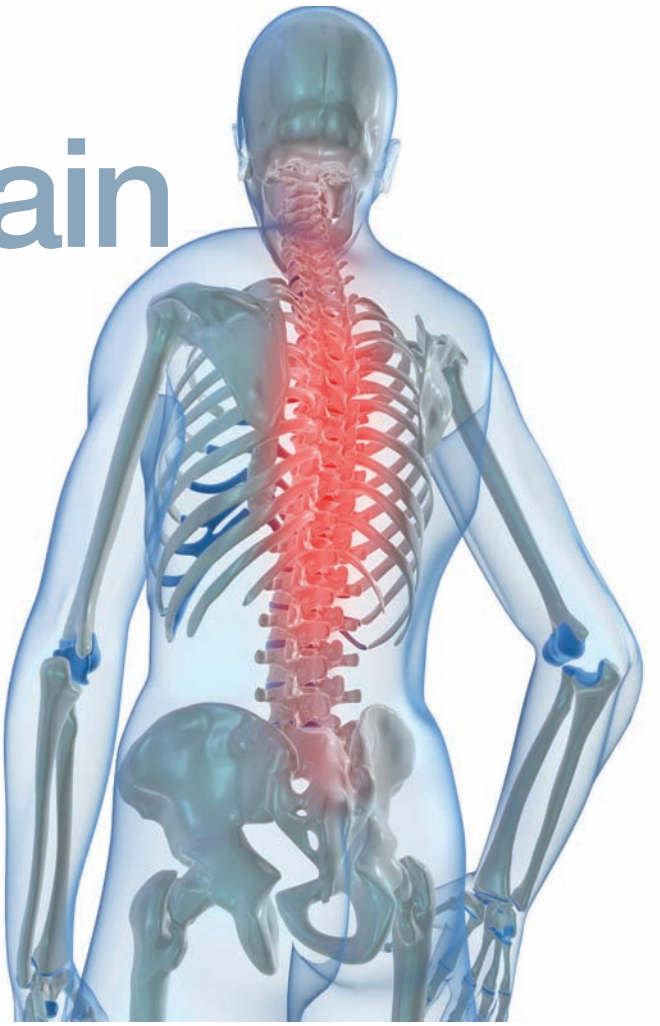
Regional pain syndrome

a disorder of pain sensitisation



GEOFFREY O. LITTLEJOHN MB BS(Hons), MD, MPH, FRACP, FRCP(Edin), FAFRM

Early recognition of regional pain syndrome as a disorder of pain sensitisation is needed so patients can be treated effectively.

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Regional pain syndrome is a common pain syndrome that affects different areas of the body. Typical terms used to describe this syndrome include whiplash, repetitive strain syndrome or nonspecific low back pain. It is often triggered by a musculoskeletal injury or disease. Delayed or missed diagnosis is common and has an adverse impact on patient outcome. This article reviews regional pain syndrome and discusses it in the context of related pain syndromes of fibromyalgia and complex regional pain syndrome.

PAIN IN MUSCULOSKELETAL INJURY

Acute pain within the various components of the musculoskeletal system is a cardinal symptom of the pathophysiological processes involved with tissue damage, disease or dysfunction. Immediate activation of the inbuilt hard-wired innate pain system serves a primary protective function.[1] These pain pathways are intimately linked to withdrawal reflexes, removing the structure from danger. Shortly thereafter, pain responses in the brain relating specifically to the tissue damage allow for protective responses and therefore healing to occur.

The combination of the particular process involved, be it injury, inflammation or degeneration, and the specific muscle– tendon unit affected results in predictable clinical features of different disorders. These range from enthesitis or tenosynovitis to muscle strain. The healing and resolution of the symptoms of most common painful musculoskeletal disorders will usually take place over days to weeks. However, the intensity and duration of the nociceptor stimulation is

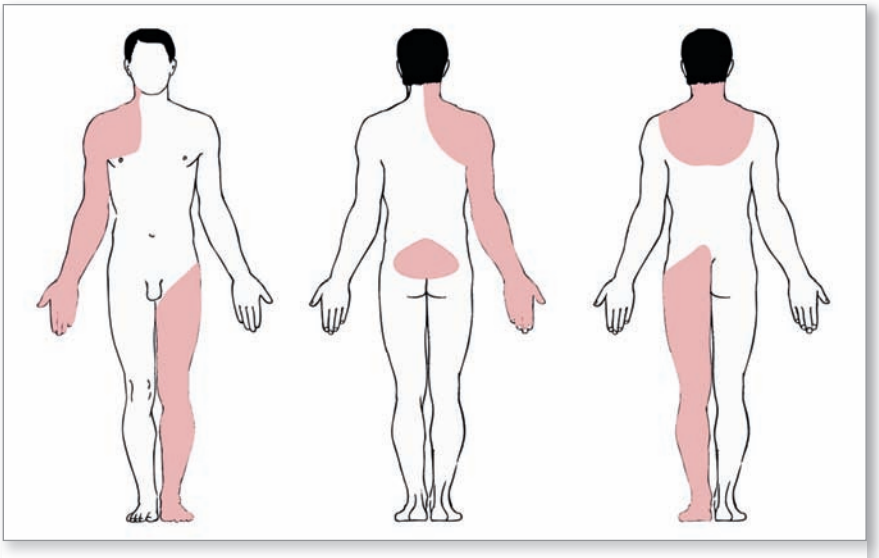

Figure 1. Common locations of regional pain syndrome.

SELECTED COMMONLY USED NAMES FOR REGIONAL PAIN SYNDROME BY REGION

Upper quadrant
- Repetitive strain injury/repetitive strain syndrome
- Cumulative trauma disorder
- Work-related upper limb pain
- Diffuse upper limb disorder

Cervical
- Whiplash-associated disorder (and variants)

Lumbar
- Low back pain
- Nonspecific low back pain

important because it may significantly modulate the healing time.

Continued nociceptor stimulation, such as through ongoing injury, can lead to a change in the stimulation threshold in the neurones that receive the pain message in the dorsal horn of the spinal cord. This important neurobiological process is called sensitisation and because it involves brain and spinal cord processes of the central nervous system the term central sensitisation is applied. In this situation, otherwise innocuous sensory inputs that have links to the sensitised dorsal horn pain-transmitting cells, particularly those coming from mechano-receptor A-beta fibres, will have their input translated into pain sensations. Therefore, with central sensitisation, touch and movement in the region of the injury will be painful. Through other mechanisms, there is regional spread of pain beyond the injured tissue.

Additionally, other disease processes, particularly inflammation, can not only activate the peripheral nociceptor itself but also increase its sensitivity to minor stimuli, known as peripheral sensitisation.

The above effects of peripheral and central sensitisation are normal and to some degree are to be expected after musculoskeletal injury or inflammation. However, the dorsal horn pain transmission centre, where the peripheral nociceptive input is first processed, may be further affected by a more potent control system that links to the brain. The brain has powerful downward control over processing in the dorsal horn and change in this 'pain brake' will augment, amplify and extend any pain response.

Emotional distress has a controlling effect on this process and can drive a series of events that result in central sensitisation with altered 'downstream' musculoskeletal symptoms and function. Under these circumstances, chronic pain states or pain syndromes may arise and particularly may involve the musculoskeletal tissues.[2]

PAIN SYNDROME

A pain syndrome is a predictable and characteristic collection of symptoms and clinical signs, predominately pain, for which there is no identifiable primary nociceptive cause (that is, tissue damage). In other words, there is no identifiable local or proximal tissue pathology that causes the ongoing pain. Pain syndromes involving the musculoskeletal system occur because of the process of sensitisation described above.

REGIONAL PAIN SYNDROME

The term regional pain syndrome denotes a characteristic set of clinical features that are localised to one region of the musculoskeletal system, most commonly the low neck or back or the upper or lower quadrant (Figure 1). Regional pain syndrome falls within a spectrum of disorders that includes complex regional pain syndrome and fibromyalgia syndrome.

Nomenclature and descriptors

It should be noted that regionalised musculoskeletal disorders that have a defined mechanism or pathophysiology, such as injury, strain, inflammation or degeneration, are not pain syndromes. The term 'regional pain syndrome' is descriptive only and used in preference to other terms that inappropriately attempt to link the syndrome to a putative cause (see the box on this page).

There are no validated classification or diagnostic criteria for regional pain syndrome, but practical clinical criteria

can be used to help in its diagnosis (see the box on this page).[3] The 2010 American College of Rheumatology diagnostic criteria for fibromyalgia encompass regional pain syndrome if the patient has more than three pain regions and very high rates of poor sleep, fatigue, cognitive change and related symptoms.[4]

Although many criteria for chronic pain syndromes require symptom duration of three months to ensure that tissue-damage contributions are minimised, for regional pain syndrome the presence of the defined clinical features denotes the syndrome as being present. Early identification is therefore emphasised as an important part of optimal management of affected patients.

Regional pain syndrome is characterised by regional pain and tenderness. It is usually triggered by an injury to a component of the muscle–tendon unit of the neck, shoulder, upper limb or back, for example supraspinatus tendinitis, forearm muscle strain or strain of a pain-sensitive structure of various types in the low neck or back. The tissue-damage component of the initial injury usually resolves but regionalised pain, from the spine to a periphery, may persist.

The pain may be described as constantly aching, dull or burning with short-lived sharper lancinating episodes. The pain is usually aggravated by activity, constrained posture of the affected part, weather or emotional changes. The pain is often accompanied by non-neuroanatomical dysaesthesia, such as sensations of numbness, tingling, pins and needles, and glove and stocking sensation changes. These symptoms are common sources of inappropriate investigation and treatment. The proximal spine is often tight, as are muscles in the region of pain. There may be subtle swelling of the forearm or hand and discolouration of the periphery, such as blotchy red or white palms.

Examination shows no evidence of a unifying tissue-damaging lesion to explain all symptoms. There is no muscle wasting and the neurological examination is normal. The key clinical sign is pain on gentle palpation in the whole region involved; some regions (not only muscle) will be more sensitive than others, usually termed tender points, but all regions are abnormally tender.

Other bodily regions not involved in the regional pain syndrome are not abnormally tender. The spine and shoulder/hip girdle may be tight but intrinsic movements are normal. Dermatographia, where a low-level stimulus such as stroking with the fingernail will cause a brisk and exaggerated wheal and flare response in related paraspinal areas, is common.

Distinct from the tender areas and tender points, indicating regional lowering of the pain threshold, there may be some specifically sensitive palpable tight muscle bands known as trigger points, especially in the region of the spine, shoulder girdle or upper forearm.

The differential diagnosis should include consideration of a single lesion that could cause the clinical features – for example, spinal pathology. Many patients with an injury to an upper limb muscle–tendon unit will develop superadded regional pain and tenderness, for example, to the whole upper quadrant. Therefore, quite commonly an injury can coexist with regional pain syndrome, making management of affected patients complex. This situation often occurs in the context of injury at work or a motor vehicle accident when an otherwise straightforward musculoskeletal injury occurs in the context of considerable psychosocial stress.

Workers' compensation, litigation and disability issues force extreme and unique pressures onto the pain axis, promoting change in the brain control of the spinal pain system. This results in amplification of otherwise innocuous sensory inputs, including those from mechanoreceptors. When these inputs come from deep spinal structures there is consequent amplification of normal low-level referred regionalised pain and tenderness. Approaches to the management of patients with chronic pain in this environment are subject to vested interest and confusion. Different outcomes and pathways in patients with the same initial problem will occur according to multiple subtle factors.

The essential pathophysiology of regional pain syndrome, therefore, is sensitisation of pain-related neural pathways in the spinal cord and brain.[5] This in turn relates to decreased tonic control of pathways from the brain that otherwise inhibit spinal cord pain-related inputs. It is the emotion-influenced parts of the brain, such as the anterior cingular and insular cortexes, that have potent control over this pathway and provide the link between emotional distress and the development of regional pain syndrome. Regional pain syndrome is therefore a 'top-down' amplification of regional pain and other sensory systems. It may be

PRACTICAL CLINICAL CRITERIA FOR REGIONAL PAIN SYNDROME

Essential features

- Regional pain
- Regional allodynia (abnormal tenderness to light touch), including areas of focal tenderness (tender points) within the region
- Clinical features not neuroanatomical, involved region consistently links to spine
- Significant emotional distress

Common features

- Sensory dysfunction (dysaesthesia)
- Muscle dysfunction (tightness, trigger points)
- Spine dysfunction (stiffness, referred sensory symptoms)

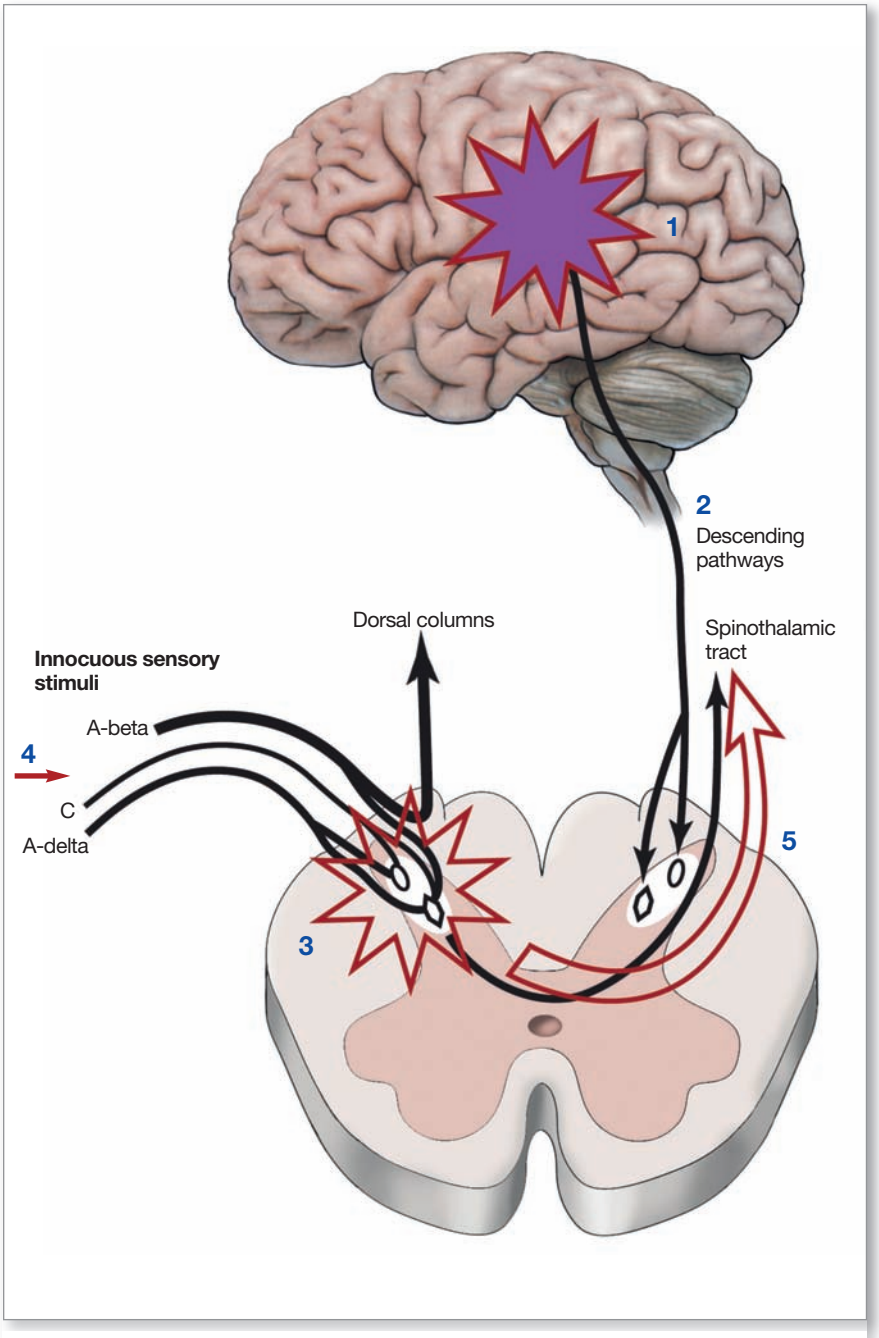

Figure 2. Mechanism of ongoing pain in regional pain syndrome. Central emotion-related brain areas (1) change the function of the descending pain-control system (2) causing sensitisation of deeply placed pain-related projection neurons in the spinal cord (3). Sensory stimuli have important connections to this region (3) and thereby innocuous stimuli (4) can gain 'access' to the pain system with muscle/joint movement now becoming painful (5). A-beta fibres are mechanoreceptors and C and A-delta fibres are nociceptive fibres. Red star shape indicates sensitisation. Red arrow indicates pain.

triggered by local injury but there is no significant ongoing primary peripheral source of nociception causing the syndrome (Figure 2).[6]

COMPLEX REGIONAL PAIN SYNDROME

Complex regional pain syndrome shares many features with regional pain syndrome.[3,7] The term 'complex' denotes a condition that usually has more significant peripheral clinical features such as swelling, colour change or sweating, and with possibly more severe soft tissue tenderness. There is generally less proximal abnormality. In addition, very localised regions, such as the patella or digits, may be involved. No direct regional link to the spine is necessary, although it is common.

Different terms have been used to describe complex regional pain syndrome at different times, usually reflecting the current understanding of the perceived mechanisms of the disorder at the time. Currently, complex regional pain syndrome type I (generally previously known as reflex sympathetic dystrophy) is used for the most common type (90%) and type II (previously known as causalgia) if nerve injury is involved (10%). Criteria for the diagnosis of complex regional pain syndrome include combinations of characteristic symptoms and signs (see the box on page 15).

Patients with regional pain syndrome affecting the upper quadrant will often also fulfill criteria for complex regional pain syndrome. In other regions, such as the neck or low back, the distinction in criteria between regional pain syndrome and complex regional pain syndrome is clearer. The overlap between regional pain syndrome and fibromyalgia, with its widespread pain, and complex regional pain syndrome, with its more intense local symptoms, is indicative of all these conditions being due to the common process of sensitisation of the pain-related nervous system. Therefore, although the names differ, the underlying process is

similar, as is the management of affected patients.

REGIONAL PAIN SYNDROME AND OTHER IMPORTANT MUSCULOSKELETAL PAIN SYNDROMES

Fibromyalgia is more common than regional pain syndrome, affecting 2 to 4% of people. It is a syndrome of widespread pain and tenderness occurring in patients with emotional distress, poor sleep, increased fatigue, altered cognition and enhanced sensory systems. Many patients with fibromyalgia are prone to regional pain syndrome and likewise many with regional pain syndrome will develop fibromyalgia.

Myofascial pain syndrome is a common disorder of muscles characterised by tightening and bunching of muscle fibres and it is associated with exquisite pain on palpation of so-called trigger points.[9] This may cause altered movement or pain. The problem is usually present in the mid-belly of the muscle and usually occurs after injury or with overuse or postural strain. Many patients with regional pain syndrome also have myofascial pain syndrome in muscles affected by the regional pain process and need to have this treated in conjunction with the regional pain syndrome. Ergonomic considerations and physical therapies are the mainstay of treatment but these therapies are not effective if the patient with regional pain syndrome is not managed appropriately.

Spinal referred pain may be both a contributor to and a consequence of regional pain syndrome. Patients with tight spinal regions may need treatment with stretching, strengthening or relaxation programs to diminish symptoms. Imaging of patients with regional pain syndrome will show the usual 30% or more abnormalities with MRI, CT or plain x-ray, usually unrelated to the clinical presentation. In the context of regional pain syndrome features, extreme care should be taken if considering invasive procedures, such as nerve blocks, denervation, surgery or protracted physical therapies, because regional pain syndrome is not responsive and will usually be aggravated by these approaches.

TYPICAL DIAGNOSTIC CRITERIA FOR COMPLEX REGIONAL PAIN SYNDROME[8]

Continuing pain, disproportionate to any inciting event, and no other diagnosis to better explain signs and symptoms.
Plus at least one symptom in three of the following categories and one sign in two or more of the following categories:

- Sensory: hyperaesthesia, hyperalgesia (to pinprick), allodynia to light touch, deep somatic pressure and/or joint movement.
- Vasomotor: temperature asymmetry, skin colour changes
- Sudomotor/oedema: oedema, sweating changes
- Motor/trophic decreased range of motion and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair, nail, skin).

MANAGEMENT OF REGIONAL PAIN SYNDROME

There is limited evidence-based literature on therapy for regional pain syndrome, due to classification and logistic issues. Many patients with regional pain syndrome are involved in medicolegal deliberations, which makes high quality studies difficult to perform.

Once established, regional pain syndrome can be difficult to manage, hence prevention and early diagnosis remain priorities. Accurate diagnosis, a label specifically incorporating the term 'pain syndrome' and careful education are essential for effective management. The patient needs to understand that the pain is from changes in pain control and not due to a tissue abnormality in the symptomatic region. Of utmost importance is the need to exclude or treat other conditions that might coexist with or mimic the features of regional pain syndrome.

MANAGEMENT PRINCIPLES FOR PATIENTS WITH REGIONAL PAIN SYNDROME

Diagnosis and education

- Consider regional pain syndrome as a possible diagnosis in high-risk situations (i.e. work or motor vehicle injuries)
- Ensure accurate diagnosis
- Identify any unresolved nociceptive stimulus but avoid unnecessary investigation
- Provide careful explanation of pain syndrome and indicate expected good outcome

Physical management

- Encourage activity and involve a physical therapist
- Avoid passive physical therapies
- Plan resumption of normal activities

Pharmacological treatment

- Provide neuroactive medication (low-dose tricyclic antidepressants, serotonin–noradrenaline reuptake inhibitors and alpha-2-delta ligands)
- Provide analgesia: simple (e.g. paracetamol, which might be ineffective) or complex (e.g. opioids, which are often unhelpful for sensitisation pain)

Psychological strategies

- Identify and manage psychosocial stressors
- Address social predicaments and involve a psychological therapist
- Use a patient-centred approach

Having done this, it is also essential to avoid excessive investigation.

The principles of management of patients with regional pain syndrome are essentially the same as those of other similar pain syndromes, particularly fibromyalgia.[10] Regional pain syndrome differs, however, in that an initial triggering injury might be taken by the healthcare professional and patient alike to be the continuing source of the pain, which can result in expensive, frustrating and illogical management, with poor outcomes. These predicaments often lead to emphasis on medicolegal deliberations rather than effective self-management-derived outcomes.

Common management strategies are based on the four principles of education, exercise, mechanism-targeted drug therapy and psychological treatments (see the box on page 15). These same principles also apply to the related pain syndrome of complex regional pain syndrome, where other interventions may be required (this is beyond the scope of this article). Patients need to know that they have a potentially reversible problem; they should understand the concept of sensitisation as a mechanism of their pain and recognise the input of societal constraints and personal reactions to their significant life predicaments as potent stressors and amplifiers of pain-related mechanisms.

The positive effects of activity, particularly aerobic fitness, on sensitisation, muscle stretch and regional muscle symptoms must be emphasised. Activity avoidance due to fear of further injury or aggravation holds many people back.

Medications shown to modulate pain in this setting include tricyclic antidepressants and related dual serotonin–noradrenaline reuptake inhibitors, such as duloxetine (all used off label). Other effective drugs include those modulating alpha-2-delta ligands, such as pregabalin and gabapentin (used off label). Opioid medications can interfere with positive psychological drive and its use must be carefully considered in patients with this disorder. As in fibromyalgia, many patients do not respond to opioids because the endogenous system is already fully activated.

Psychological treatments are the mainstay of management and will range from commonsense explanations and pragmatic strategies, usually delivered using a team approach, through to complex cognitive behaviour programs in the minority of cases. The goal is to educate and upskill the patient to continue self-management. Gaining control of the situation for the patient is critical in reducing the emotional distress that drives the altered pain-control pathways. However, other powerful forces, such as controlling health professionals and the requirements of the legal safety net system, may negate this.

Outcomes vary but improvement is expected in most affected patients. The patients have to take an active role in their own management and extrinsic stressors need to be minimised.

CONCLUSION

Regional pain syndrome is a pain disorder that is well-characterised and has a high-impact. Early diagnosis is aided by anticipation of pain sensitisation in high-risk situations, such as work-related injuries or traffic accidents, where safety net deliberations and disputes are common, and in psychologically vulnerable persons. Patient outcomes are significantly improved when the disorder is recognised early and treated as a disorder of pain sensitisation rather than one of peripheral nociception. MT

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Pain in older people

Often unrecognised and undertreated

BENNY KATZ FRACP, FFPMANZCA

Pain is not a normal part of ageing and it is often unreported, unrecognised and undertreated in older patients. Appropriate treatment is required to ensure this population is managed effectively.

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Although pain is one of the most common presenting symptoms of older people attending their GPs, many studies have found that pain is often unreported by older individuals; it is also often unrecognised by health professionals and is, therefore, undertreated.[1] The most vulnerable individuals, such as those with cognitive impairment or in residential care facilities, are at greatest risk. The extent of this problem will increase as the population ages.

The prevalence of pain increases with advancing age. About one in three community dwelling older people report experiencing persistent pain of more than three months' duration,[2] and more than 50% report experiencing severe pain following surgery or trauma.[3] Pain should not be considered a normal part of ageing; its high prevalence is a consequence of the increased burden of pathology. Major causes of pain include arthritis, particularly of the lower limbs, spinal disease, trauma and surgery. There is also an increased prevalence of cancer-related pain and neuropathic pain – for example, postherpetic neuralgia and painful diabetic peripheral neuropathy.

PAIN IS OFTEN UNREPORTED, UNRECOGNISED AND UNDERTREATED

Older individuals may consider persistent pain a normal part of the ageing process and therefore not report it. Doctors often enquire about the presence of pain in specific sites such as the chest or back, without asking an open-ended question about whether the patient has any pain or discomfort and allowing adequate time for a response. Some individuals will deny the presence of pain yet report unpleasant discomforts such as aching joints or tingling in their feet. Cognitive impairment and communication difficulties increase the risk of pain going unrecognised.

Age-related changes in pain perception must be considered. Some types of pain such as headache and chest pain become less prevalent with age and may not be a feature of conditions usually associated with pain in younger individuals. It may not be a major symptom of myocardial infarction in older people, for instance. An older person presenting for no apparent reason with failure to walk should have a hip fracture excluded, even in the absence of pain. Experimental studies reveal an increase in stimulus intensity is required before older individuals report pain; however, once pain is experienced it is not tolerated as well as in younger people.[4,5]

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TRANSIENT, ACUTE AND PERSISTENT PAIN

Most pain is transient and mild, and perhaps an inevitable part of life. Acute pain serves a biological role, warning of tissue damage and protecting the individual from further injury – for example, by using a fractured limb. In patients with acute pain, treatment is focused on the cause of the pain, together with temporary symptomatic relief. Pain usually settles over a short period of time as healing takes place.

Pain lasting beyond the usual tissue healing time, often defined as pain lasting more than three months, is referred to as chronic or persistent pain, Careful evaluation is required to ascertain why the pain has persisted and to ensure that an important diagnosis has not been overlooked. Concurrent medical conditions and their treatments may limit the patient's treatment options leading to persistence of pain. Patient preference may preclude pain-relieving interventions, such as joint replacement surgery. Some painful conditions such as fibromyalgia and neuropathic pain may be refractory to conventional analgesia. Mood disorders should be considered when assessing an older person with persistent pain. Additionally, patient assessment should ascertain the impact pain is having on daily activities, mood, sleep, social function and quality of life.

FOCUSING ON THE PAIN

Once curative options have been excluded, the goal of management shifts to symptom control. The pain and its impact on the patient are considered as important as the underlying cause of the pain. Mood disturbance and functional limitation may be more amenable to therapy than the pain.

A frank discussion with the patient is required, covering the nature of the pain, treatment options and prognosis. Even if it is unrealistic to hope for a cure, the patient should not be left feeling hopeless. Patients often feel reassured being told their pain is real and not imagined, that they do not have cancer and that support is available even if a cure is not. Avoid sending mixed messages, by ordering further investigations, specialist opinions or trialling a new medication. A patient is unlikely to move forward if they are waiting for an elusive cure. Chronic pain should be considered as a condition in its own right, not simply a symptom of underlying pathology.

The focus should move from the cause and severity of the pain to the impact that the pain is having on the patient's mood and function. A successful pain management program may result in the person still reporting the same level of pain but being able to achieve more before being stopped by the pain. Treatment goals should be negotiated with the patient who must consider the potential benefit of any new therapeutic approach versus the burden of that intervention and potential side effects. Many patients who feel they can cope with ongoing pain decide not to pursue further treatments, whereas others will elect to continue with a pain management approach.

MANAGEMENT

Analgesic medications are usually first-line treatment for people with pain. This may have been initiated by the individual or prescribed by a doctor. Most patients will have tried at least one analgesic before seeking professional help. Failure to respond to treatment may be the result of taking the wrong medication, taking the wrong dose or an inadequate duration of therapy.

Older individuals tend to experience more adverse effects from their medications. This may be avoided by starting at a lower dose, with gentle dose escalation according to patient response. Predictable side effects such as opioid-induced constipation should be treated pre-emptively.

If pain has not been adequately controlled with medications, a combination of pharmacological and nonpharmacological approaches tends to be more effective than escalation of medication use.

A detailed discussion of the treatment options of pain is beyond the scope of this article; however, some points need to be made about the treatment of older individuals with pain. Most guidelines recommend the use of paracetamol as the first step in the management of pain in older people.[6] This recommendation is based on its availability, cost, efficacy and side effect profile. Generally, no dose reduction is required for older people, apart from the very frail, poorly nourished, alcohol misusers or those with liver disease. Many patients find the large tablet size together with the need for frequent dosing to be inconvenient. Co-administration of paracetamol with other analgesics may enable dose reduction of the second analgesic.

NSAIDs and COX-2 inhibitors

Second-line analgesics include NSAIDs, selective cyclooxygenase (COX)-2 inhibitors, codeine and tramadol. NSAIDs and COX-2 inhibitors offer convenient dosing, but are associated with serious adverse reactions, particularly in older individuals. Special caution is required in patients with peptic ulcer disease, hypertension, cardiac failure or renal impairment.

The gastrointestinal advantage of using a COX-2 inhibitor is lost when it is co-prescribed with low-dose aspirin. Concomitant use of diuretics and ACE inhibitors with NSAIDs increases the risk of renal impairment. However, in selected individuals the benefit may still outweigh the risk. If used, NSAIDs should be taken at the lowest dose for the shortest period possible. A safer option for inflammatory arthritic conditions may be a low dose of prednisolone.

Paracetamol and codeine

There are multiple preparations available combining paracetamol with codeine. The analgesic effect of codeine is thought to be mediated through its transformation to morphine by cytochrome CYP2D6. About 8% of Caucasians and 2% of Asians are genetically deficient in CYP2D6 and obtain little pain relief from codeine.[7] In addition, a number of commonly

prescribed drugs, such as haloperidol, amitriptyline, fluvoxamine, fluoxetine and paroxetine, inhibit CYP2D6.

The side effect profile of paracetamol and codeine combinations is similar to that of other opioids and, in particular, constipation and cognitive effects can be problematic in the elderly. Although codeine remains widely used, there is an increasing trend to avoid codeine in favour of low-dose opioids such as oxycodone.

Tramadol

The efficacy of tramadol 100 mg and paracetamol 1000 mg/codeine 60 mg are equivalent. Tramadol has an affinity for the mu opioid receptor and inhibits the uptake of serotonin and noradrenaline, giving it a role in treating both nociceptive and neuropathic pain. Its usefulness for older people is limited by its high potential for adverse side effects and drug interactions, so dose reduction is required for the elderly.

The most common adverse effects are nausea, vomiting, dizziness, constipation, sweating, tiredness and headaches. A serotonin syndrome may be precipitated by tramadol itself, but is particularly likely when it is used with other serotonergic drugs, such as selective serotonin reuptake inhibitors, tricyclic antidepressants and monoamine oxidase inhibitors. Features of a serotonin syndrome include confusion, agitation, fever, sweating, ataxia, hyperreflexia, myoclonus and diarrhoea.

Opioid analgesics

There has been an increasing trend for the use of strong opioid analgesics such as morphine, oxycodone, buprenorphine and fentanyl for the management of persistent pain in older people. Often only small doses are required, and, in general, paracetamol should be continued to minimise the dose of opioid needed (see the box on this page). There is evidence for the efficacy, of opioid analgesics for treating patients with chronic pain conditions, including those of neuropathic origin;[8] however, these studies have tended to be of short duration with little evidence for their long-term efficacy, particularly in older people. The mean decrease in pain intensity in these studies is in the order of 30% with about 80% of patients experiencing at least one adverse event such as constipation, nausea and somnolence. The prescriber must ensure compliance with legal requirements and regularly review the patient to ensure ongoing treatment is of benefit.

A preparation containing oxycodone and naloxone is now available in Australia. The naloxone component reduces, but does not eliminate, opioid-induced constipation. Not all patients will benefit, but it appears useful for patients experiencing opioid-induced constipation despite standard measures.[9]

Transdermal analgesic patches offer the convenience of infrequent dosing and reduced burden of tablets. Buprenorphine and fentanyl transdermal patches are not suitable for the management of acute pain or where rapid dose titration is required. Buprenorphine transdermal patches may be used to initiate opioid therapy for persistent pain as an alternative to codeine, tramadol and low-dose oxycodone and morphine. The buprenorphine patch 5 µg/hour is approximately equivalent to morphine 10 mg oral over 24 hours.[10] In contrast, fentanyl transdermal patches are very potent. The fentanyl 12 µg/hour patch is approximately equivalent to

PRINCIPLES OF OPIOID THERAPY FOR OLDER PEOPLE WITH CHRONIC NONCANCER PAIN[6,8]

- Explore all other treatment options, including physical and psychological treatments, before starting opioid therapy
- Continue paracetamol use
- Explain and obtain agreement for the following:
  - expectation of pain reduction rather than elimination
  - adverse effects are common, but usually manageable
  - need for monitoring and adherence
  - withdrawal of drugs if there is nonadherence or no significant improvement over four weeks
- Avoid use of immediate-release and parenteral opioids
- Appropriate first-line opioid therapies are:
  - controlled-release morphine: starting dose, 15 mg/day; minimum interval between dose increase, three days; maximum recommended dose, 100 mg/day; seek specialist input if considering higher doses
  - controlled-release oxycodone: starting dose, 5 mg/day; minimum interval between dose increase, three days; maximum recommended dose, 80 mg/day; seek specialist input if considering higher doses
  - buprenorphine patches: starting dose, 5 µg/hour; minimum interval between dose increase, seven days; maximum recommended dose, 20 µg/hour; seek specialist input if considering higher doses
  - a test dose of immediate-release oral morphine or oxycodone may be appropriate in frail older people before initiating sustained-release preparations
- Opioids that are not appropriate for first-line therapy are:
  - hydromorphone
  - fentanyl patches
  - methadone

morphine 50 mg orally over 24 hours. This is too high for a starting dose, so fentanyl patches should not be commenced in patients who are not already stabilised on strong opioid analgesia.

Adjuvant agents

A number of drugs that are not primarily analgesics have been found to have a beneficial role in pain management, particularly in patients with neuropathic pain. These adjuvant agents, or co-analgesics, include tricyclic antidepressants such as amitriptyline (off-label use), serotinergic-noradrenergic reuptake inhibitors such as duloxetine (indicated for the treatment of diabetic peripheral neuropathic pain) and the antiepileptic agents gabapentin and pregabalin (both indicated for the treatment of neuropathic pain). Selective serotonin uptake inhibitors have not proven to be efficacious in this role.[11]

The advantage of newer agents, such as gabapentin, pregabalin and duloxetine is that they are better tolerated, though not necessarily more effective. Most studies define a positive response as a 50% reduction in pain from baseline, but a lesser reduction may be considered worthwhile by the patient. These drugs rarely eliminate pain. Attempting to achieve total eradication of neuropathic pain is likely to result in intolerable side effects.

Nonpharmacological approaches

There is a wide range of nonpharmacological approaches for pain that may be used individually or as an adjunct to pharmacotherapy.[12] The use of a walking aid may ease the burden on a weight-bearing joint, and domiciliary services can perform domestic and gardening tasks that aggravate the pain. Simple measures such as massage and hot packs are very popular. Exercise training including strengthening and balance exercises have general health benefits in addition to any improvement in pain. Transcutaneous electrical nerve stimulation may prove helpful and can be used for many hours per day.

Psychological strategies such as cognitive behavioural therapy, relaxation, hypnosis and guided imagery are worth considering. Increasing social activity may shift the patient's focus away from the pain. Complementary and alternative therapies are increasingly being used in western societies. Most clinical trials support the safety of these therapies rather than offering evidence of their efficacy. Trials of chondroitin and glucosamine have yielded mixed results.[13] The potential for adverse interactions of dietary supplements and herbal therapies with prescription medications needs to be considered. Patients may not inform their doctor about the use of these medications unless specifically asked.

Patients who continue to be troubled by pain despite standard treatments should be referred to a multidisciplinary pain clinic or pain specialist.

CONCLUSION

Effective management of persistent pain in older people poses special challenges often best managed using a multidisciplinary approach. The goals should be modified to suit the needs of the older person. Although the goals may be different from those of a younger person, the benefit to the older individual may be as great. MT

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MedicineToday PEER REVIEWED

Pain management in inflammatory arthritis

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Many people with inflammatory arthritis continue to experience high levels of pain even when receiving effective treatments. Chronic pain is a complex biological process that cannot be easily remedied with a single pill. A multimodal approach is required.

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Inflammatory arthritis (IA) affects up to 3% of the population and is characterised by pain, stiffness, loss of function and impaired quality of life.[1] It comprises diseases such as rheumatoid arthritis (Figure 1), psoriatic arthritis, reactive arthritis and spondyloarthropathies, and when treatment is successful IA is a satisfying condition to treat. There are many examples of the debilitated patient with polyarthritis who is revived with a pulse of steroid. Instant trust and often a smile can follow. Unfortunately, this is not always the case and for doctors and patients it can be frustrating when treatment fails to live up to the high expectations.

Some extraordinary developments in targeted biological therapy for patients with IA have occurred over the past decade. Indeed, for many patients with IA, when these therapies work, they really do work well. However, unfortunately the reality is that biological agents are costly, not all patients qualify for a rebate on the PBS, not all patients respond to treatment and those that do respond may still have ongoing pain. Despite these significant advances in treatment, it should

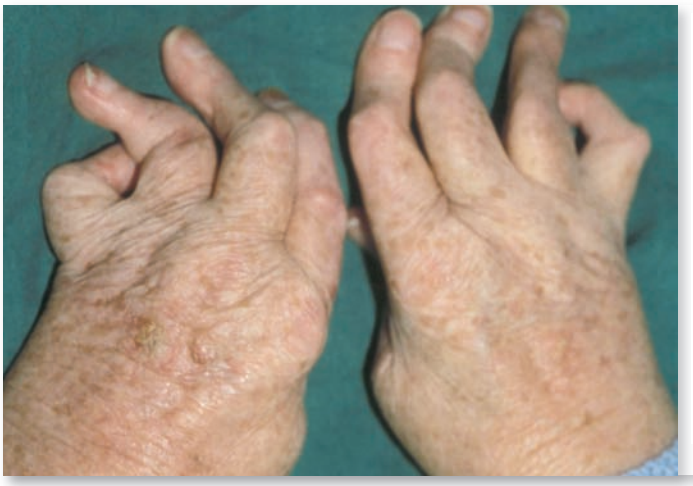

Figure 1. Deforming rheumatoid arthritis.

come as no surprise that patients with IA are reported to perceive pain as their predominant impairment[2] and pain management as one of their highest priorities.[3]

A case study of a woman with persistent knee pain is given in the box on this page.

CAUSES OF CHRONIC JOINT PAIN

One of the reasons for the lack of effective pain management is the paucity in our knowledge of what actually causes joint pain. The aetiology of pain in patients with IA is multifactorial and often

CASE STUDY. A 78-YEAR-OLD WOMAN COMPLAINING OF PERSISTENT KNEE PAIN

Case scenario

A 78-year-old woman with a 30-year history of erosive, deforming rheumatoid arthritis (RA) presented complaining of persistent pain in her right knee. She was known to have severe secondary osteoarthritis (Figure 2) but was not a suitable candidate for joint replacement therapy. The pain had been worsening for the past six months, was localised to the anteromedial aspect of the knee and was associated with mild swelling. She described the pain as a deep ache that was worse with weight-bearing and had now begun to limit her activities and disturb her sleep. The pain did not feel like her usual arthritis flare pain, she felt systemically well, had only 10 minutes of early morning stiffness and her other joints remained stable. She was taking methotrexate 20 mg weekly, folic acid 5 mg weekly, prednisone 5 mg daily and celecoxib 200 mg daily. On examination she had a body mass index of 31 kg/m^2 and walked unaided with an antalgic gait and had widespread tenderness to minimal pressure. There was a valgus deformity of the right knee with quadriceps wasting, a small effusion, medial joint line tenderness and marked crepitus.

Commentary: The initial differential diagnoses are quite broad and include osteoarthritis progression, RA flare, septic arthritis, crystal arthritis, stress fracture, avascular necrosis (AVN) and referred pain from the hip or back. This woman's history is more suggestive of mechanical rather than inflammatory pain; however, with mild swelling an inflammatory component to her symptoms needs to be excluded. A key piece of information is that this pain is different to the patient's usual RA flare pain, so RA is unlikely to be the cause. Although she feels systemically well, the history of progressive pain that is disturbing her sleep is a 'red flag' for more serious causes to be excluded. AVN, osteomyelitis and stress fracture should all be considered when the pain severity seems out of proportion to the clinical findings. Examination of the hip and back for irritability and reproduction of the knee symptoms can be helpful in identifying the source of the pain. The patient is not currently taking any paracetamol and this should be added early to her usual medications. Her widespread tenderness and poor sleep also suggest that a component of fibromyalgia may be present. Preliminary investigations should include measurement of baseline erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) level to assess disease activity and a plain anteroposterior and lateral weight-bearing x-ray of the knees.

Case continued

Investigations in this woman showed the disease activity to be mildly elevated but stable with an ESR of 29 and a CRP level of 6.7.

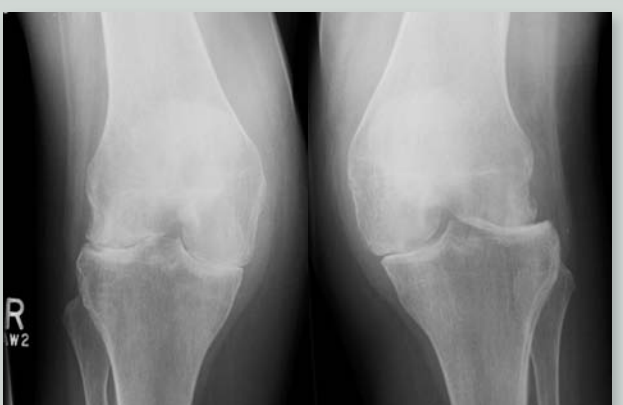

Figure 2. X-rays of the knees showing severe secondary osteoarthritis.

difficult to assess. In the past decade, we have seen extraordinary advances in the understanding of pain mechanisms at the molecular level. However, there is still a long way to go. We know that pain receptors are located throughout the joint, having been identified in the capsule, ligaments, menisci, periosteum and subchondral bone. Therefore, damage at any of these sites can manifest as 'joint pain'.

Simplistically, to guide treatment, we often think of pain mechanisms as being inflammatory, mechanical, neuropathic or a combination of these. Recent evidence shows, however, that there is actually a complex interplay between the neural and immune systems, and that inflammatory cytokines influence both peripheral and central pain pathways.[4] As a consequence, many patients with IA manifest a generalised increase in pain perception and, in effect, become 'pain sensitised'. Untreated and undertreated pain also has a negative impact on sleep and mood, which are in turn associated with greater pain.[5] Hence, the patient becomes trapped in a self-perpetuating downward cycle that can be difficult to break.

BARRIERS TO PAIN MANAGEMENT

There are several barriers to pain management outlined in the box on page 24. An awareness of these is the first step to improving pain management in patients. Regardless of the underlying pathological process, the clinical evaluation of pain is always a challenge. There are many examples of patients with similar disease duration and severity who manifest very different degrees and phenotypes of pain. This is most likely explained by the concept that the experience of pain is uniquely influenced by factors other than the primary pathology, such as

CASE STUDY (continued)

There was no fracture or chondrocalcinosis (to suggest calcium pyrophosphate dihydrate disease) seen on the x-ray; however, there were severe tricompartmental osteoarthritic changes.

Commentary: The low-grade elevation in inflammatory markers needs to be interpreted in the context of the patient's disease, and input from her usual rheumatologist should be sought regarding any changes to the patient's disease-modifying antirheumatic drugs or the need for an MRI. An MRI is useful if there is a high suspicion of stress fracture, AVN, osteomyelitis or ongoing low-grade synovitis. A joint aspirate is always useful diagnostically and therapeutic cortisone injections can also be performed at the same time (if there is no concern regarding infection).

Case continued

The synovial fluid analysis showed a noninflammatory cell infiltrate with 1000 polymorphonuclear leucocytes, no crystals and was Gram stain and culture negative. An MRI showed no active synovitis, stress fracture, osteomyelitis or AVN. There was significant bone marrow oedema consistent with her severe osteoarthritis.

Commentary: These results are most consistent with pain occurring secondarily to osteoarthritis. Being a single joint, the best treatment option would be an intra-articular corticosteroid injection. However, this will be of temporary benefit and without definitive treatment (joint replacement) the patient is likely to have ongoing pain. Nonpharmacological strategies including education, walking aids, heat packs, transcutaneous electrical nerve stimulation (TENS) and gentle exercise to aid muscle strength and weight loss will be helpful. Bracing may also improve stability of the joint and input from a physiotherapist and occupational therapist should be sought. Glucosamine and fish oil may also be considered but are unlikely to make a significant difference.

Case continued

The patient returned one month later and stated that her knee pain was moderately improved but she still had widespread pain and was not sleeping. Despite the improvement in her knee pain, she was reluctant to exercise, had become withdrawn and was feeling tired all the time. She had been taking paracetamol 3 g daily and celecoxib 200 mg daily. There was no evidence of inflammation in her knee but she had marked quadriceps wasting. She wanted to know what else she could do for pain relief.

Commentary: In addition to her knee pain she has symptoms that are suggestive of fibromyalgia and it is likely her pain threshold has been further reduced by her poor sleep and possible coexistent low mood. Stronger opioid analgesics are unlikely to improve these symptoms and should be avoided. The patient's beliefs about the cause of her pain and her reluctance to exercise should be explored further. A gentle exercise and quadriceps-strengthening program may improve her muscle mass, joint stability, falls risk and possibly sleep. Regular review with the patient and referral to a physiotherapist for a modified exercise program with achievable goals may reduce some of the patient's anxiety regarding exercise. Having a plan of what to do if her pain gets worse (e.g. use of a TENS machine, hot and cold packs or codeine [for knee pain]) would also be useful. After ensuring that the patient's vitamin D level was optimised, in addition to exercise, a trial of a low-dose antidepressant for her fibromyalgia (off-label use) could also be considered. If her mood symptoms were significant and no progress was made, consideration of input from a psychologist or psychiatrist may be warranted.

BARRIERS TO EFFECTIVE PAIN MANAGEMENT

Patient
- Reluctance to report pain to physicians
- Reluctance to take more 'pills'
- Lack of education regarding available pain therapies
- Fear of side effects or drug addiction
- Fear of masking the disease
- Compromised cognitive function secondary to certain pain medications

Physician
- Focused on disease management, not pain
- Inadequate training and knowledge of pain management
- Inadequate assessment of pain
- Time constraints of a busy practice
- Concern about scrutiny from regulatory agencies

Healthcare system
- Low priority of pain management
- Cost of medications
- Limited access to allied health services
- Lack of pain management clinics

IMPORTANT ELEMENTS OF THE PAIN HISTORY

- Characteristics of the pain
- Impact of the pain on the patient (e.g. physical, psychosocial, role functioning, work, etc)
- Prior treatments for the pain
- Physical functioning
- Mood and psychological well-being
- Sleep and energy levels
- Premorbid and comorbid medical and psychiatric conditions
- Comprehensive medication history
- Social support structures

psychological status, past pain experience, cultural background, environment and genetics of the individual. In the end, pain evaluation often requires the clinician to make a clinical judgement taking into account all these factors in a limited time frame, and without an objective test or laboratory measure. Important aspects of the pain history that should be explored are shown in the box on this page.

APPROACH TO PAIN MANAGEMENT

It is important to remember that the goals of treatment are not only to reduce pain, but also to improve function and quality of life. A patient-centred integrated approach involving the GP, rheumatologist and other allied health professionals is therefore likely to offer the best overall outcome. Patients with IA should have a chronic disease care plan to enable them to access a Medicare rebate for these additional services.

When considering an overall treatment strategy many aspects such as the patient's underlying disease, pain characteristics, age, comorbidities, social supports, coping strategies and health beliefs should be taken into account. Factors that will affect the likelihood of compliance, such as patient preference, cost of medications, the frequency and complexity of the regimen, route of administration and tolerability of the regimen, should also be considered. A combination of both pharmacological and nonpharmacological approaches usually offers the best opportunity for therapeutic success (see the boxes on page 2 s5).

NONPHARMACOLOGICAL STRATEGIES

Education

Patient education is an essential part of the pain management program and gives patients a sense of control over their situation. All educational activities should be sensitive to culture, ethnicity and the values and beliefs of individual patients and their families. Validating the patient's pain, setting realistic goals (e.g. reducing rather than completely removing pain), addressing the patient's fears and misconceptions, and having an action plan for 'bad days' can reduce anxiety levels and the frequency of subsequent medical reviews. Written information to explain the diagnosis, cause(s) of pain and reinforce the management strategy is also very helpful. Patient information sheets are available on the Australian Rheumatology Association website and are a useful patient resource.[6]

Exercise

Exercise is particularly useful in patients who have muscle wasting, are overweight or have fibromyalgia. With regular exercise, muscle tone is maintained, musculoskeletal structures are stabilised and regenerative processes are stimulated. Additional benefits include the production of natural analgesics (endogenous opioids), improved mental health and sleep, weight loss and reduced cardiovascular risk. Although concerns about causing further damage to joints are often voiced, patients should be reassured that provided trauma is avoided, physical activity within reasonable limits will not cause harm. For painful joints, nonweight-bearing activities such as hydrotherapy and cycling are useful starting points and a physiotherapist should be involved when possible.

Counselling/cognitive behavioural therapy

Chronic pain is often associated with psychological disorders such as anxiety and depression. These patients should be identified, treated and referred for further evaluation when necessary. For many patients, counselling and regular review by their GP may be all that is required. However, if this fails, cognitive behavioural therapy (CBT) can be a useful adjunctive management tool. CBT can be broadly defined as interventions that

change behaviour, thoughts or feelings to help patients experience less distress and enjoy more satisfying and productive daily lives. Patients learn to identify and change dysfunctional beliefs and attitudes that adversely affect their ability to cope with pain. Interestingly, CBT and stress management training have been shown to decrease inflammation as well as pain, which may also help with control of the underlying disease.

Other

Other nonpharmacological interventions frequently used by patients include joint assistive devices, hot and cold therapy, ultrasound, relaxation, meditation, hypnosis, acupuncture, massage and transcutaneous electrical nerve stimulation (TENS). Although these interventions still require formal evaluation in high-quality trials, they appear to have favourable risk–benefit profiles. Smoking is associated with erosive joint disease and a decrease in response to disease-modifying anti-rheumatic drugs (DMARDs), so facilitating smoking cessation will aid control of the disease and resultant articular pain.

PHARMACOLOGICAL STRATEGIES

The first question to address when treating the patient with IA is whether there is an inflammatory component to the pain. For the patient with a clear flare of disease with tender swollen joints, early morning stiffness and raised inflammatory markers (erythrocyte sedimentation rate [ESR] and C-reactive protein [CRP] level), short-term courses of oral corticosteroids or NSAIDs can be useful. Similarly, intra-articular corticosteroids can be used by experienced clinicians to treat individual joints of patients with IA.

In the setting of long-standing inflammatory disease, the presence of active inflammation can be more difficult to assess. Established articular changes with associated structural deformity make the assessment of synovitis more difficult. Doppler ultrasound has recently also shown that the ESR and CRP level may not be sensitive enough to detect persistent low-grade disease activity. After consideration of a patient's comorbidities and discussion with their treating rheumatologist, sometimes a short course of a corticosteroid (seven to 10 days) can help to rule out ongoing active inflammation as a contributor to the patient's pain. It must be emphasised, however, that systemic glucocorticoids are not generally recommended for the routine management of pain in patients with IA (in the absence of signs and symptoms of inflammation). The choice, dose and duration of immunosuppressive therapy needs to be individualised to the patient and should be discussed with their treating rheumatologist.

Available in a standard or concentrated preparation, fish oil possesses anti-inflammatory properties and may be a useful adjunctive therapy in patients with persistent inflammation. However, to achieve this, current evidence suggests that fish oil needs to be taken in high doses (≥2.7 g of omega-3 [eicosapentaenoic acid plus docosahexaenoic acid] daily),

TREATMENT OF PATIENTS WITH A SUSPECTED INFLAMMATORY CAUSE OF PAIN

An inflammatory cause of pain is suspected in patients who present with early morning stiffness, increased inflammatory markers and joint swelling.

- Send the patient for rheumatologist review
- Trial the use of paracetamol and/or an NSAID
- If this is unsuccessful, trial a different NSAID
- Try a short trial (7 to 10 days) of low-dose prednisone
- Consider using an alternative disease-modifying agent

TREATMENT OF PATIENTS WITH RESIDUAL NONINFLAMMATORY PAIN: A MULTIMODAL APPROACH

Nonpharmacological agents

- Patient education
- Fish oil ≥2.7 g/day
- Glucosamine 1500 mg plus chondroitin sulfate 800 mg
- Vitamin D_3 if levels <80 nmol/L
- Massage/acupuncture
- Exercise program/hydrotherapy
- Orthoses/splints
- Transcutaneous electrical nerve stimulation (TENS) machine
- Psychological support cognitive behavioural therapy
- Relaxation

Pharmacological agents

Mild pain

- Paracetamol 2 to 4 g/day*
- Topical or oral NSAID†

Moderate pain

- Consider adding codeine or tramadol

Severe pain

- Consider adding transdermal buprenorphine, oxycodone, transdermal fentanyl or morphine

Adjuvant agents

- Topical capsaicin 0.025%
- If component of fibromyalgia present, consider use of amitriptyline‡, duloxetine‡, gabapentin‡ or pregabalin‡ (off-label uses)
- If neuropathic pain present, consider use of amitriptyline‡ (off-label use), duloxetine (off-label use)‡§, gabapentin‡ or pregabalin‡
- If depression or anxiety is significant, suggest psychiatric review regarding optimal agent

* Reduce dose in elderly, caution in patients with liver disease.
† Caution in patients with cardiovascular, renal or gastrointestinal disease.
‡ Watch for interaction with tramadol.
§ TGA approved for diabetic peripheral neuropathic pain.

TABLE. PERSISTENT PAIN IN INFLAMMATORY ARTHRITIS: ANALGESIC OPTIONS

Agent	Starting doses	Limitations
Simple analgesics		
Paracetamol	2 to 4 g/day	Generally safe, but reduce dose in elderly or patients with liver or renal disease; watch total dosage with combination pills
Glucosamine 1500 mg plus chondroitin sulfate 800 mg	1 tablet/day	High cost; limited data regarding efficacy
Fish oil	2.7 g/day or more	Bad taste; high cost; limited data regarding efficacy; care needed in patients with bleeding disorders or taking blood-thinning medications
Weak opioids		
Codeine	30 to 60 mg/day	Causes nausea and constipation
Tramadol	50 to 100 mg/day	Causes dizziness, nausea and constipation; care needed when coprescribed with selective serotonin reuptake inhibitors
Strong opioids		
Oxycodone	2.5 to 5 mg	Cause dizziness, headaches, nausea, constipation, respiratory depression and somnolence; have the potential for tolerance and addiction
Buprenorphine	5 mg patch weekly	
Morphine	10 mg/day	
Fentanyl patch	12.5 µg/hour every three days	
Adjuvants		
Capsaicin 0.025%, topical	Apply four times a day	Causes local burning and skin irritation
Amitriptyline*	10 to 25 mg/day	Causes dizziness, somnolence, dry mouth and constipation
Fluoxetine*	20 mg/day	Causes nausea, somnolence, dry mouth and sexual dysfunction
Duloxetine†	30 to 60 mg/day	Causes dizziness, somnolence and dry mouth
Gabapentin‡	300 to 900 mg/day	High cost; causes dizziness, somnolence, swelling and ataxia
Pregabalin‡	150 mg/day	Causes dizziness, somnolence, swelling and ataxia

* Off-label use.
† Off-label use unless used for the treatment of patients with diabetic peripheral neuropathic pain.
‡ Off-label use unless used for the treatment of patients with neuropathic pain; gabapentin RPBS-listed and pregabalin PBS-listed for refractory neuropathic pain.

which are often not well tolerated. Doses of more than 7 g of omega-3 fats per day may increase a patient's risk of bleeding.

After the inflammatory component of IA has been minimised, treatment goals shift to being similar to those for patients with secondary osteoarthritis. In the patient with persistent pain despite having well-controlled IA, a general stepwise approach is used. A summary of the agents and their common side effects to be considered is shown in the Table. In general, use of more than one drug with the same mode of action is likely to increase the risk of adverse effects and should be avoided. Severe vitamin D deficiency is also known to cause arthralgias and myalgias and deficiency should be treated as a reversible cause of pain (aiming for vitamin D levels of more than 80 nmol/L). It should be noted that not all patients with pain are responsive to pharmacological treatments; once a treatment has been initiated, it is important to monitor its efficacy to justify continuation of that treatment.

Mild pain

Depending on the individual, paracetamol, NSAIDs or a combination of both should be considered before using more potent analgesics. The daily dose of paracetamol should not exceed 4 g, and should be reduced if the patient is elderly or has a history of liver disease. Several selective and nonselective NSAIDs are available and switching agents should be considered if insufficient analgesia is achieved after a few weeks. This is because the failure of one NSAID does not predict failure of all NSAIDs. The combination of these agents with methotrexate is generally considered to be safe.

It is well known that the use of selective and nonselective NSAIDs are associated with an increased risk of cardiovascular events in the general population. Naproxen appears to have the least potential to increase the risk of cardiovascular disease, although there are limited data available that evaluate the magnitude of this risk in patients with IA. As a guide, in patients with IA and pre-existing gastrointestinal, hypertension, cardiovascular or renal disease, paracetamol should be used as first-line treatment. If required, NSAIDs including coxibs should be used with caution at the lowest effective

KEY POINTS FOR GPS

- Pain management is a high priority for patients with inflammatory arthritis.
- A multimodal approach should be tailored to an individual patient's needs.
- All patients with inflammatory arthritis and chronic pain should have an action plan for 'bad days'.
- Systemic glucocorticoids are not recommended for the management of pain in the absence of inflammation.
- Patients should be reviewed regularly and ineffective therapies discontinued if therapeutic goals are not achieved.

dose and for the shortest time, monitoring for adverse events carefully. If NSAIDs are needed in patients with high gastrointestinal risk, the use of selective COX-2 inhibitors is preferential and the concurrent prescription of a proton pump inhibitor is also recommended.

Moderate pain

If paracetamol and NSAIDs provide inadequate relief, a weak opioid such as codeine or tramadol may be added. They combine favourably with paracetamol; however, use is limited by side effects such as dizziness, nausea and constipation. It should be remembered that 5 to 10% of the population are slow metabolisers, and are unable to synthesise enough morphine from codeine or convert tramadol to its active metabolite to produce an analgesic effect. Care should be taken to avoid the concomitant use of selective serotonin reuptake inhibitors and tramadol.

Severe pain

The role of long-term opioid therapy for patients with persistent nonmalignant pain continues to be controversial. However, it is reasonable to consider the use of these drugs in patients in whom the above combined treatment strategies are contraindicated or have failed and there is pain-related impairment of function and quality of life. An example would be a patient with refractory pain who requires, but is not a candidate for, joint replacement surgery. Stronger agents to consider include transdermal buprenorphine, oxycodone and occasionally transdermal fentanyl or morphine. Concerns regarding tolerance, addiction and the commonly reported side effects of constipation, nausea and somnolence limit the use of opioids. If used, a laxative should be coprescribed, although a newer preparation combining oxycodone plus naloxone is now available and has been designed to reduce opioid-induced constipation.

Safe and effective prescribing of opioids on a long-term basis requires skills in both opioid pharmacotherapy and risk assessment and management. Patients receiving these agents should be regularly reassessed for the attainment of therapeutic goals, adverse effects and responsible medication use. A contract with the patient regarding a definitive trial period should be discussed and a gradual withdrawal plan instigated if the aspired benefits have not been achieved.

Adjuvant agents

Adjuvant agents are not recommended as analgesic options in isolation, but can be considered at any time as part of a comprehensive pain management strategy. Despite a lack of data in patients with IA, if there is a significant element of either fibromyalgia or neuropathy (where there is good efficacy data), it is reasonable to trial an antidepressant , such as amitriptyline (off-label use) or duloxetine (off-label use unless the patient has diabetic neuropathic pain) or one of the newer, more costly neuromodulating agents, such as gabapentin or pregabalin (Table). A low dose should be initiated and slowly increased according to efficacy and adverse effects, knowing that therapeutic benefits are often slow to develop. For patients with troublesome joints, topical capsaicin may be tried, although about 30% of patients are unable to tolerate the local burning sensation. There is no evidence to support the use of muscle relaxants as analgesics in patients with IA.

CONCLUSION

Pain management is a high priority for patients with IA and even small decreases in the severity of pain can positively influence a patient's well-being (see the key points box on this page). Currently, there are no guidelines because of a lack of published studies in this patient population. For patients with persistent joint pain, a multidisciplinary approach combining education, nonpharmacological and pharmacological interventions should be tailored to the individual's risk–benefit profile. MT

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Managing chronic pelvic pain in girls and women

SUSAN EVANS FRANZCOG, FFPMANZCA

Women with chronic pelvic pain have often experienced pain since adolescence, with self-doubt, difficulties in personal development, relationships and sexual confidence. The management approach outlined in this article can help to improve outcomes for these patients.

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When a woman presents with a long list of pelvic pain complaints, it's easy to feel overwhelmed, especially as time is often limited. Frequently, the patient has experienced pain for years before making the decision that 'something must be done', and presents to her doctor distressed and desperate.

Women with chronic pelvic pain have often experienced pain since adolescence, with self-doubt, difficulties in personal development, relationships, sexual confidence, educational achievement and financial opportunities. Their pain is a taboo topic with embarrassing gender, fertility and sexual issues, not suitable for easy conversation with family or friends. When the pain is severe, affected women often stay home isolated and distressed.

This article outlines an approach to the management of women with chronic pelvic pain, which can lead to improved outcomes for affected patients.

AN OVERALL CONCEPT OF PELVIC PAIN

Chronic pelvic pain can be considered as having three components, as described below.

- **Pelvic causes of pain.** Although there may now be several organs involved, ask the patient how the problem started in the first place. For example, a woman may have had dysmenorrhoea as a teenager then over time developed an irritable bowel, overactive bladder or painful pelvic muscles.
- **The chronic pain condition.** This is sensitisation of the nerve pathways that transmit pain, sometimes called neuropathic pain or central sensitisation. Once anyone has pain of some kind on most days for more than six months, chronic pain is likely. This is best considered as a medical condition requiring treatment. The patient will recognise her pain is more complex than before, with a mix of pain symptoms, anxiety, low mood, poor sleep and fatigue.
- **The brains' adaptation to chronic pain.** The brain constantly adapts to change and repeated episodes of pain

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change the way the brain works. Hypervigilance and withdrawal from activities are common as she becomes concerned with the potential pain implication of every movement or sensation.

MANAGEMENT

Making a list of each pain symptom at a patient's first presentation helps delineate the clinical picture and makes it easy to reflect on positive progress at a later date.

At the first visit:

- focus on the symptom that bothers her most
- address her individual fears
- initiate treatment for the chronic pain condition
- encourage self-management and learning
- make a follow-up appointment to retain her trust.

At the follow-up visit:

- reflect on the positive progress made
- review treatments for chronic pain
- support lifestyle change, including exercise
- initiate treatment for symptoms that remain, including headache
- plan regular review visits. Symptoms will vary over time and episodic flares of pain respond well to prompt management review.

Managing the pelvic causes of pain

One of the reasons that women with pelvic pain find it difficult to get comprehensive care is that so many different organs can be affected. The pelvis not only includes the uterus and ovaries, but also the bowel, bladder, pelvic muscles (pelvic floor, obturator internus, piriformis), peripheral nerves (e.g. pudendal, ilioinguinal) and vulvovaginal skin, often with some endometriosis.

A common mix of problems might be a history of endometriosis (which may no longer be present), uterine dysmenorrhoea, pelvic muscle spasm (pain on one side, pain with movement or exercise, generalised ache or painful intercourse), an overactive bladder (frequency, nocturia, urgency) and an irritable bowel. Headaches, including menstrual headaches and a background lower grade chronic headache with a migraine style, are common. Treatment of the pelvic organ pain component involves minimising pain from each of the organs involved.

Dysmenorrhoea

If endometriosis or dysmenorrhoea is present, the aim of treatment is to minimise the number of menstrual periods, minimise the amount of bleeding and maintain a progestogenic environment. This improves symptoms and may reduce both recurrent endometriosis and worsening pain. Endometriosis is a 'progesterone resistant' condition and oestrogen stimulates certain pain cytokines. Progestogen treatment reduces symptoms and may reduce episodes of recurrent endometriosis.

Suitable options for management with progestogen are outlined below.

- A levonorgestrel intrauterine device. This should be inserted just after a menstrual period. In women whose menstrual periods have been irregular, taking the oral contraceptive pill for one to two cycles prior to insertion prepares the endometrium and may reduce postinsertion bleeding. Offer the option of insertion as a day-surgery procedure to nulliparous women or those concerned that insertion may be overly painful, and recommend the use of NSAIDs for cramps postinsertion.
  A levonorgestrel device provides the lowest hormone dose option and provides contraception for five years, but may only provide pain relief for three to four years.
- A progestogen-dominant monophasic oral contraceptive pill used continuously to minimise the number of periods.
- Norethisterone 5 mg/day or dienogest 2 mg/day (the latter is not available in Australia) used continuously.
- An etonogestrel implant.

Useful combinations if pain persists or bleeding is troublesome include:

- a levonorgestrel intrauterine device and either norethisterone 5 mg daily or an etonogestrel implant
- a levonorgestrel intrauterine device and a progestogen-dominant monophasic oral contraceptive pill used continuously. (A pill-free week with continuous use of the pill may still be needed every two to three months to avoid irregular bleeding.)

For severe cases of chronic pelvic pain, a gonadotrophin-releasing hormone analogue, such as a goserelin implant or nafarelin spray, together with continuous combined hormone replacement to achieve amenorrhoea may be required.

Hysterectomy treats dysmenorrhoea well when fertility is no longer required but should not be considered as a cure for chronic pelvic pain. Dysmenorrhoea is often only one part of the chronic pain picture. To doctors dysmenorrhoea indicates either endometriosis or uterine pain; however, to women, dysmenorrhoea is the entire menstrual pain experience, so may also include pelvic muscle spasm, bowel pain, menstrual migraine or premenstrual syndrome.

Bladder symptoms of frequency, nocturia and urgency

Although bladder symptoms of frequency, nocturia and urgency can be due to a range of conditions, painful bladder syndrome (including interstitial cystitis) is common in women with chronic pelvic pain. Flares resemble urinary tract infections but with a negative urine culture.

Management includes the following.

- Excluding diet triggers (e.g. citrus fruits, fizzy drinks,

caffeine, artificial sweeteners).
- Using amitriptyline, oxybutynin or solifenacin. Amitriptyline, if tolerated, has the added advantage of helping with sleep, headaches, the chronic pain condition, some pelvic muscle pains and some irritable bowel symptoms. A small dose of amitriptyline with additional oxybutynin or solifenacin can be taken if higher doses of amitriptyline are not tolerated.
- Drinking enough, but not too much, water (about 1.5 to 2 L).
- Managing 'flares' by drinking 500 mL water mixed with one teaspoon of bicarbonate soda or two urinary tract alkalising powder sachets, then 250 mL water every 20 minutes for a few hours.

Antibiotics should be taken only if infection is proven. Providing the patient with a request form for urine culture to use if symptoms flare provides security that a urine infection will not be missed.

Dyspareunia

Dyspareunia can be particularly distressing for women because it affects their relationship with their partner. Management depends on which conditions are present, as described below.

- **Recurrent thrush.** Regular use of fluconazole (e.g. 200 mg once weekly until settled then monthly just before a period) is effective.
  A private script is cost effective.
- **Vulvar vestibulodynia.** Touching the opening of the vagina near the Bartholin's glands with a Q-tip elicits pain even if no abnormality can be seen. Use of 2% amitriptyline in a vaginal cream twice daily is often effective. Exclude thrush by taking frequent swabs.
- **Pelvic muscle spasm**. To assess for tightness and tenderness, use one finger to gently palpate the pelvic floor muscles just inside the vagina. Intercourse, insertion of tampons and smear tests are painful for affected patients, both at the time and afterwards. The patient may experience a sudden stabbing pain up the vagina or bowel, or difficulty initiating a void despite strong urge. An experienced pelvic physio - therapist who understands that these muscles require 'downtraining' and not strengthening is essential. Unfortunately, this expertise is uncommon. As a guide, pelvic physiotherapy should not aggravate the patient's pain. Other treatments include low-dose amitriptyline, listening to a pelvic muscle relaxation CD daily, botulinum toxin injected as a day-surgery procedure, and optimisation of bladder and bowel function.

Pain on one side

Although women may have chronic pain every day, it is a severe exacerbation of this pain that often prompts urgent emergency department or general practice visits. Unfortunately, when the ultrasound (with pain on insertion and movement of the vaginal probe), blood and pregnancy test results, and sometimes laparoscopy, are normal, the patient is sent away without a diagnosis.

Although ovulation pain (in women not taking the oral contraceptive pill), appendicitis and ectopic pregnancy (positive pregnancy test) are all possible, the most common cause of pelvic pain on one side is spasm of the muscle obturator internus, which lies inside the hips on the pelvic side wall, usually in the context of a flare of background neuropathic pain. A typical history for obturator internus spasm includes pain on one side, sometimes bilateral, which has the following features: comes on suddenly; is worse with exercise or movement; may be worse on standing or sitting for a long time; may radiate into the anterior thigh; often wakes the patient at night; is worse during a menstrual period or during intercourse; is associated with an overactive bladder; and is helped by a heat pack but poorly helped by use of narcotics. The pain is often described as 'ovary pain'. Although obturator internus spasm may improve for some weeks after surgery, possibly due to the medications used or a change in routine, it usually recurs once normal activities resume.

The diagnosis of obturator internus spasm can be confirmed with one finger vaginal examination just inside the vagina directed out laterally to the pelvic sidewall. The muscle will be tight and tender and may be hypertrophied. Palpating along the muscle with the pad of the forefinger reproduces the pain. (Use your right forefinger for the patient's right side and your left forefinger for her left side.) Management is similar to that of pelvic muscle pain with intercourse, but botulinum toxin is especially effective. Avoidance of pilates or other 'core-strength' exercises until pain improves is advisable.

Common additional features of pelvic muscle/girdle dysfunction include a tender lower back, tenderness across the iliac crests (gluteus medius tendonitis), tenderness laterally (trochanteric bursitis), or pain down the back of the leg that may mimic sciatica (piriformis syndrome).

Irritable bowel – bloating, constipation, cramps and diarrhoea

Although most women with chronic pelvic pain have irritable bowel symptoms, only a few have endometriosis invading the bowel wall. Inflammatory bowel disease is possible, but the more common situation is food intolerance with sensitisation of the bowel as part of the chronic pain condition. Common food intolerances include:

- fructose-releasing foods (fructans), such as wheat, onions, apples, corn syrup
- lactose

- galactans, such as in beans, brussel sprouts
- polyols, such as artificial sweeteners
- fatty foods.

The patient may already have found a gluten-free diet beneficial. Although few women with pelvic pain have coeliac disease, a gluten-free diet excludes wheat (a common source of fructan) and therefore treats both coeliac disease and fructose malabsorption.[1] Bowel sensitisation, especially bloating, often improves with use of low-dose amitriptyline (5 to 10 mg taken in the early evening).

Pudendal neuralgia

Pudendal neuralgia causes pain in the 'saddle' area on sitting, anywhere from the clitoris to the anal area. It may be unilateral or bilateral, is usually a burning or sharp 'electric' feeling and may be associated with increased clitoral arousal.

Management includes:

- avoiding activities that compress the nerve, such as cycling
- using a 'u-shaped' foam cushion (with the front and centre area cut out when sitting)
- using pelvic physiotherapy downtraining techniques to relax and lengthen the pelvic muscles to take pressure off the nerve and ceasing straining of the bowels and bladder
- using botulinum toxin to relax pelvic muscles and pudendal nerve blocks.

MRI of the pelvis and lumbosacral spine should be carried out to exclude tumours, pudendal entrapment and Tarlov cysts.

The chronic pain condition – 'central sensitisation'

Although managing peripheral pains is useful, once pain of some kind is present on most days, it is essential to manage the chronic pain condition. This is as true of chronic back pain and postinjury pain as it is of pelvic pain.

Common symptoms of the chronic pain condition include:

- bloated or burning pains
- normal sensations felt as pain (allodynia); even the partner's hand on the patient's abdomen may be unpleasant
- painful sensations becoming more painful (hyperalgesia)
- pain felt over a larger area when severe (wind-up pain)
- poor sleep, fatigue, anxiety and low mood
- nausea, dizziness or sweating.

Treatment of the chronic pain component involves use of medications and lifestyle changes to manage central and peripheral nerve pathways. Treatment includes the following.

- An explanation that the nerve pathways have physically changed and become sensitised; although a cure is unlikely, substantial improvement can certainly be achieved.
- Neuropathic medications, such as:
  - low-dose amitriptyline (start at 5 mg three hours before bed and increase slowly to 5 to 25 mg daily).
  - pregabalin (start at 25 mg every night and increase slowly as tolerated)
  - duloxetine (start with 30 mg in the morning with food, then 60 mg every morning after two weeks; 15 mg [half the contents of a capsule] should be used initially in those sensitive to medications)
  - other neuropathic medications with assistance from a pain specialist.
- Regular exercise. This should be considered essential for pain management. Exercise can be considered the best nondrug treatment for pain. Even if the patient has become completely inactive, a 10-minute walk daily is not unreasonable and can be increased slowly. Too much exercise suddenly results in more pain the next day. Pilates-style core-strength exercise should be avoided until the pain improves because this can aggravate pelvic muscle pain. Exercises such as walking, dancing or gentle team sports are preferred.
- Avoid getting overtired. Tired nerves are more irritable and poor sleep frequently precedes a bad pain day.
- Activities enjoyed by the patient with strong personal motivating factors. This may include playing with her children, sport or craft. These activities are often continued even with some pain present.
- Acupuncture.

Narcotic analgesics should be avoided if possible because chronic pelvic pain is not short-term pain and dependence is common. Although narcotics can help pain in the short term, we now know that long-term use sensitises nerve pathways even further and may worsen chronic pain. Reducing the dose of narcotics later is difficult and it is common to find that within a short time frame the patient has just as much pain as before but now with an added analgesic dependence. Narcotics are often relatively ineffective in both neuropathic and pelvic muscle spasm pain. The role of the nervous system in chronic pelvic pain is explained further elsewhere.[2]

The brain and adaption to pain

Women with pelvic pain fear that they will not be believed or will be told 'it's all in your head'. Pelvic pain is often described as an embarrassing pain, with social, sexual, fertility, gender and employment stigma concerns. Managing pelvic pain requires sensitivity to the patient's needs, while building her self-esteem and confidence in her ability to self-manage the pain.

Medical care is particularly effective where:

- There is reassurance that the patient's medical care team believe in her pain and will take her pain seriously.
- The patient's fears are addressed. Ascertain what it is that worries her most about the pain. Fear of undiagnosed cancer or a life-threatening illness can usually be allayed with a smear test and ultrasound. Fear of infertility can be

addressed individually, and fear of worsening pain can be allayed by reassurance that the patient's health care team will work with her to manage the pain.
- A pain psychologist, if available, is part of the multidisciplinary team. Frequently there are life factors contributing to the overall pain experience. This can include a history of sexual assault. Although most women with pelvic pain have not been sexually assaulted, if this is present management is more complex.
- The patient is encouraged to remain active. Even on days with pain, the patient can be reassured that she is not in danger and that there are still positive things she can achieve. Although becoming overtired is not helpful, giving up work rarely improves pain. It is best to keep active and keep moving. The physiotherapy adage of 'motion is lotion' remains true.
- There are positive cultural, personal and family attitudes towards to the patient's pelvis. A negative view of the pelvis as being dirty, shameful or troublesome allows the pain to become an emotional as well as a physical event.

Role of laparoscopy

Laparoscopy is an excellent tool for removing endometriosis or the uterus in older women with dysmenorrhoea. However, repeated laparoscopies risk exacerbation of central sensitisation and surgical complications. Without specific indications for repeat laparoscopy, nonsurgical options are preferred in treating women with pelvic pain, at least in the first instance.

When considering laparoscopic findings, the following should be taken into account:
- an abnormality found at laparoscopy may or may not be the major cause of the patient's pain; there are many causes of pain that cannot be seen at laparoscopy and endometriosis is only one aspect of chronic pelvic pain
- there is no correlation between the amount of endometriosis found and the severity of pain
- there is a correlation between the amount of endometriosis found and the likelihood of infertility
- surgery includes time off work with rest, use of anaesthetic (neuropathic) and muscle relaxant medications and a higher priority placed on the patient's wellbeing by her family; these can all provide short-term improvement without addressing the underlying cause of pain
- photographs of what is found can be used positively to show the patient that most of her pelvis is normal, or that her ovaries are normal and fertility is unlikely to be severely affected.

If there is pain on most days or different types of pain, laparoscopy alone is unlikely to be sufficient to treat the pain.

Managing pain flares

Even when pain has been well managed, episodes of increased pain (flares), whereby symptoms worsen for weeks or months, are common and should not be considered as treatment failure. Effective management of pain flares are listed below.
- A review of medications. Frequently, neuropathic medications have been ceased when the patient is well, with symptoms recurring over subsequent weeks or months. Recommencing these or trialling an alternative medication is often beneficial.
- An increase in the dose of progestogen medication, which might include early replacement of the levonorgestrel intrauterine device or adding norethisterone 5 mg daily.
- Review of activity and exercise. A recent increase in inactivity or core-strength training may aggravate pelvic muscle pain. Frequently, management of this aspect of the patient's pain has been overlooked.
- Consideration of new symptoms, such as a newly painful bladder, newly painful intercourse or an episode of vaginal candida infection.

CONCLUSION

Pelvic pain truly is a 'hidden epidemic'.[3] A GP with an interest in this area is in a good position to manage most chronic pelvic pain issues. MT

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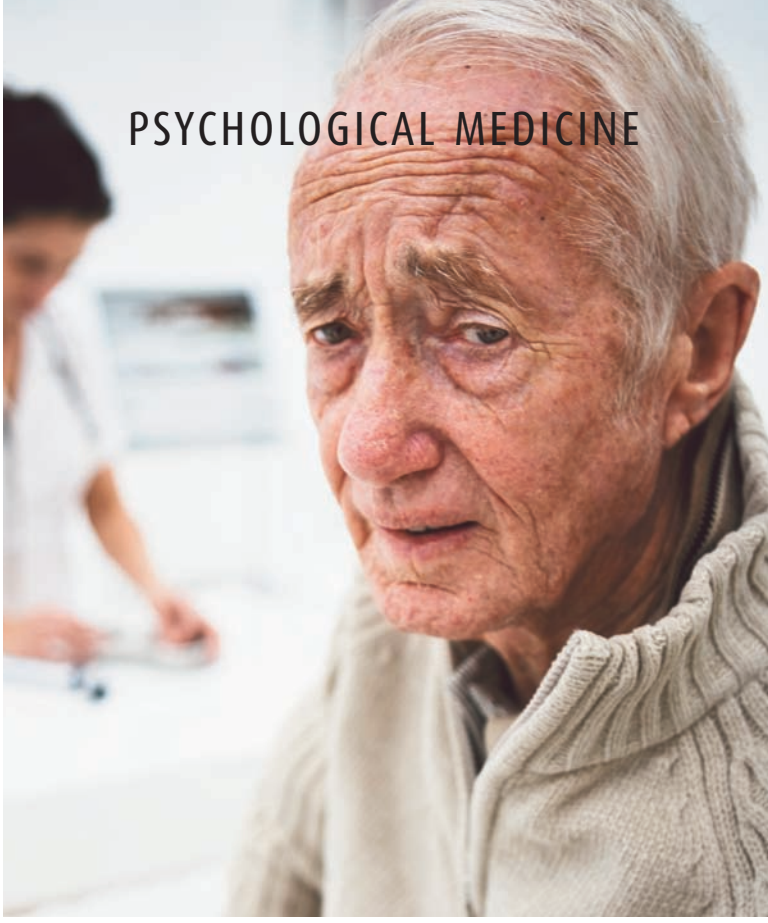

Common psychiatric issues in chronic pain

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Chronic pain is a major health problem, commonly accompanied and aggravated by emotional difficulties. If we look for, recognise and treat co-occurring psychiatric issues, we may improve outcomes for many patients.

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Nearly one in five Australians suffers from chronic or persisting pain, and the prevalence is expected to increase as our population ages. Whatever the original cause of an individual's pain, it is often a sensitised nervous system rather than structural tissue damage that is most strongly correlated with persistence of the pain. The concepts of neuroplasticity and central sensitisation are increasingly used to explain how such persistence develops.[1] Persistent pain is commonly linked to emotional distress and serious mental health problems.[2] Such comorbid psychiatric conditions, which are often unrecognised, may interfere with a patient's response to treatment and make successful pain management more difficult. If these psychiatric conditions are recognised and treated and function improves, it should make for a happier patient (and a happier doctor).

Much of the research on chronic pain and psychiatric problems has been focused on patients attending pain clinics. By the time patients are referred to a pain clinic, their pain is often highly disabling, complex and difficult to manage, and significant psychiatric comorbidity is commonly present. It is likely that they have already seen a range of clinicians. However, the persistent pain in patients attending their GP may be just as complex. Although many patients have milder emotional responses to their pain, these are still significant – and treatable.

It is important to recognise that patients with chronic pain are a heterogeneous group, not only in the physical origins of their pain but also in their psychological responses to it. Every patient is different, with his or her own coping styles, skills and vulnerabilities. Nonetheless, it is worth looking for groups within this population. From a psychiatric perspective, several common

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TALKING WITH PATIENTS ABOUT CHRONIC PAIN: USEFUL CONCEPTS

- Chronic pain is very different to acute pain – it is much more complex. What works for acute pain may not work for chronic pain, and vice versa.
- In chronic pain, changes have developed in the nervous system (central sensitisation).
- 'Neuroplasticity' is not just part of explaining how chronic pain develops. It is also a reason for optimism to explain why behavioural, 'psychological' treatment can help.
- Mind and body are not separate disconnected entities; rather, they are aspects of the whole person. Both constructs include a range of factors that can aggravate or reduce pain levels.
- A rehabilitation approach is more likely to work than persisting in looking for 'the cure'.
- It's not just the level of pain that is important, it's also about how well the individual is functioning.

comorbid conditions can be described, each of which may vary in severity from a minor presentation to a full-blown psychiatric disorder. The main issues to consider are depression, anxiety, somatisation, substance dependence and difficulties related to certain personality traits. These are not exclusive categories. For example, a patient with chronic pain may have an anxiety disorder plus a tendency to somatise and a dependency on opioids.

Patients with persistent pain who have straightforward depression or anxiety are usually open about describing their emotions and their symptoms, and they can be dealt with at a primary or secondary care level. It is when the picture becomes complicated by somatisation or substance dependence or when problems develop in the doctor–patient relationship that referral to a multidisciplinary pain centre is indicated.

A brief list of concepts that are often helpful when communicating with patients about their chronic pain is provided in the box on this page.

DEPRESSION

Research shows that 10% to 40% of patients with chronic pain have major depression.[3] In addition, many patients have chronic dysthymia, and many more just experience a lot of sadness. Depression is essentially about loss, and people with persisting pain have often experienced a cascade of losses: in their health, their self-image (reduced strength or competence), and their role in the family and their job, to name a few.

There is evidence that patients with coexisting chronic pain and depression have a poorer prognosis and response to treatment than patients with either condition alone.[4] Chronic pain and depression both require active treatment. We should not expect (as many patients do) that fixing the pain will make the depression go away.

GPs who are interested and comfortable in a counselling role are often best placed to treat such a patient – they have an existing relationship that can be continued over a long term. Patients with depression need support in talking about their feelings, especially about their losses and ways of coping with these.

There is a spectrum of intensity for depressed mood. The further that a patient's mood state is towards the major depression end of the spectrum, the more likely it is that antidepressants will be necessary. If the patient's pain is neuropathic and low-dose tricyclics (e.g. amitriptyline 20 mg) are already useful, increasing the dose into the usual antidepressant range (say, amitriptyline 100 mg or more) may be effective, assuming that the patient can tolerate any side effects and is not at risk of taking an overdose. SNRI antidepressants also appear to have analgesic properties that are distinct from their antidepressant effects. Two reviews have shown this for duloxetine and venlafaxine;[2,5] to date, there is little evidence about desvenlafaxine. So in the treatment of patients with both persistent pain and major depression SNRIs have an advantage over SSRIs, which do not appear to have these analgesic properties.

The decision to refer a patient to a psychiatrist, psychologist or counsellor will depend on where the depression sits on the major depression–sadness spectrum. Experience in helping patients with chronic pain is often more relevant than the clinician's label. Good communication between the GP and other health professionals is important because the GP will remain central to long-term management.

ANXIETY

Research indicates that between 15% and 30% of patients with chronic pain have an anxiety disorder, such as generalised anxiety disorder, panic disorder or post-traumatic stress disorder (PTSD).[2,6] Depression and anxiety often co-occur, together with chronic pain.

PTSD is increasingly recognised as accompanying chronic pain that originated in a physical injury that was emotionally traumatic, such as an assault or motor vehicle accident. In a study of patients who developed chronic pain following severe accidental injury, PTSD symptoms and other psychological factors were the strongest predictors of persisting pain three years later.[7]

Short of having an anxiety disorder, many patients with persistent pain experience some degree of anxiety. Both anxiety and pain can have physiological effects, such as increased muscle tension, that aggravate the pain. A person who worries that physical activities will increase the pain and also that pain means further damage may become very avoidant. Whereas depression is basically about losses that have already occurred, anxiety is more

apprehension about what might happen.

The GP is often the best person to deal with a combination of persisting pain and anxiety. Patients need to have their fears listened to and not have the conversation cut short with a prescription.

Cognitive behavioural therapy (CBT) encompasses a number of strategies to help people manage both anxiety and pain.[7] Such strategies include goal setting, problem-solving, pacing of activity, relaxation and the challenging of unhelpful beliefs.[8]

More recently, there has been interest in acceptance commitment therapy, which uses some elements of CBT but focuses more on encouraging the patient to accept their pain, usually via mindfulness meditation. Training in mindfulness often involves teaching patients to sit, relax and focus on their breath, and then being aware of and accepting their pain without reacting to it with distress.[9,10] In this context, acceptance means accepting that the pain is present right now, not accepting that it will never change.[8]

The usefulness of medications for mild to moderate anxiety is limited. Many drugs, such as benzodiazepines and analgesics, (and alcohol) are effective short-term anxiolytics, but they may do more harm than good in people with chronic conditions. Antidepressants may help to reduce anxiety, with less risk of tolerance, dose escalation and dependence.

Referral to a psychologist for CBT may be considered for a patient who has significant anxiety, especially if the anxiety is accompanied by avoidance of activities that threaten to increase pain or anxiety or by a high level of catastrophising. The treatment of PTSD, whether severe or subclinical, is under debate. There is little strong evidence that standard counselling or psychotherapy achieves much. It seems more likely that PTSD requires specific, focused treatment. This may include approaches such as exposure, relaxation and guided imagery by a practitioner with experience in the use of such techniques.[11]

SOMATISATION

Somatisation is more a process than a diagnosis. A somatising patient has medically unexplained somatic symptoms that may be driven or aggravated by underlying emotional distress, but the patient is not open to considering this possibility. Extreme somatising behaviour can result in somatisation disorder.

There may be cultural, language, educational or emotional reasons for somatisation. In addition, multiple visits to doctors who are much more attentive to physical complaints than emotional ones may reinforce somatising behaviour. There is also the stigma of 'mental illness'. Many people still believe that mind and body are separate entities, that only physical treatments can help 'real' pain and that psychological treatment is for 'imaginary' pain. But it is not 'either…or' in persistent pain, it is 'not only…but also'.

A patient who is very resistant to considering emotional factors or psychological approaches to helping cope with their pain is unlikely to agree to a referral to a psychologist, much less to a psychiatrist. In discussing referral, it helps to address the mind–body split, and a patient's fear that the GP disbelieves his or her symptoms. It may be helpful to explain that you wish to refer the patient to a practitioner who is an expert in helping people cope with and better manage their pain. A multidisciplinary pain centre is often more acceptable to the somatising patient. In the centre, the various personnel, from pain specialist to nurse practitioner, can work together with the patient, and it will be emphasised that the treatment package routinely addresses the whole range of physical and psychological factors.

SUBSTANCE DEPENDENCE

In terms of the potential for substance dependence, opioids are the obvious drugs of concern for patients with persistent pain. Expert opinions differ about the value of opioids in the long term and for whom they are suitable. Many people start taking opioids for acute pain but keep taking them after the pain has become persistent. In 2005, a large US study found that 3% of the population were taking prescribed opioids and that 45% of those had sufficient symptoms to be diagnosed with common mental health disorders (major depression, dysthymia and anxiety disorders), although very few of these disorders were being treated.[12] So when considering the use of opioids for a patient with chronic pain, first screening for and treating depression and anxiety is very important.

The subject of opioids in chronic pain is a topic in itself. One of the many ways in which acute pain and chronic pain are different is their response to opioid treatment: acute pain may often respond well to opioids but the evidence for long-term efficacy of opioids in chronic pain is much less convincing. The issues involved in prescribing opioids for chronic pain are discussed in a recent article in *Australian Prescriber* (and available online).[13]

Patients with chronic pain are at risk of developing dependence on other analgesics, benzodiazepines and alcohol, which initially relieve pain, insomnia and anxiety. However, once dependent, patients may be even less motivated or able to use psychological approaches to pain management.

There is also a group of patients who, while not physiologically dependent on a drug, have a fixed belief that the only real treatment for their pain must be medication (chemical coping) or surgery. For them, the pain is purely physical and will only respond to such 'real medicine' (see the section on somatisation above). This mind–body split may have been reinforced by their experiences with doctors. Such somatising patients are at high risk of developing a substance dependence.

In difficult cases, referral of the patient to a drug health clinician or pain clinic can be considered. At a tertiary level, pain clinics benefit greatly from close liaison with a drug health unit. Clear

communication between treating clinicians about treatment goals and dosage regimens is required, but it is essential that a single clinician be responsible for prescribing an individual's medication.

DIFFICULTIES RELATED TO PERSONALITY TRAITS

Personality traits are enduring patterns of relating to others and coping with the environment. Persisting pain can interfere with coping. For instance, the athlete for whom running is central becomes irritable and stressed without his regular exercise. As the pain goes on and on, with repeated experiences of frustration, disappointments, unhappy consultations and failed treatments, his range of coping strategies will narrow and his vulnerabilities will increase. The patient's interactions with doctors will become more difficult. This is an understandable development, rather than the result of a pre-existing personality disorder.

The diagnosis of personality disorder is occasionally useful but it is often arbitrary and unreliable. However, having an idea of a patient's personality traits, whether dependent or cynical, workaholic or avoidant, helps in developing an effective relationship in which the patient feels more heard and understood. It pays to focus on your patients' strengths, not just their weaknesses (they may well do that themselves).

Issues of personality style, or traits, and personality disorders relevant to general practice were discussed in a recent article in *Medicine Today*.[14] The author of that article noted that assessing the patient's personality style can help in predicting his or her response to the illness and compliance with treatment, and how the patient may relate to the doctor. In turn, this should help the doctor decide how to relate to the patient. To deal with the problem of imposing categories of personality disorders onto what are essentially dimensions, the author proposed a two-tier model: tier 1 describes the personality style, and tier 2 describes the degree of dysfunction along a spectrum (from minor trait to clear disorder).

Personality styles or personality disorders are mainly visible in an individual's manner of relating to the other people in his or her life. Patients with chronic illness who interact regularly with clinicians are more likely to become entrenched in patterns of transactions that may be uncomfortable for both.

A relationship is a two-person thing. The patient's relationship with healthcare workers is often crucial to successful management. Rather than just thinking 'difficult patient', the clinician can try to assess an individual's personality style and consider the kind of interaction that has developed between them. Increasing difficulty in the doctor–patient relationship is a strong indication for referral to a multidisciplinary pain centre, or perhaps to a psychiatrist if the patient will accept this.

FINAL POINTS

- Psychiatric conditions commonly accompany chronic pain and are often unrecognised.
- Comorbidity often worsens disability and reduces the likelihood of a good response to treatment.
- Depression is the best studied of the psychiatric conditions that commonly accompany chronic pain. The other main co-occurring issues are anxiety, somatisation, substance dependence and personality issues that contribute to difficulties in the doctor–patient relationship.
- Depression and anxiety are usually best treated at the primary care level.
- Chronic pain becomes harder to deal with when somatisation or substance dependence is present and when problems in the doctor–patient relationship develop. Management in these cases more often requires collaboration at a tertiary care level, such as a multidisciplinary pain centre. MT

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