Management strategies for chronic pain

D M Justins

This article on management strategies for chronic pain will not reveal new drugs, new techniques, new nerve blocks, and new methods of neuroablation. Unfortunately improved understanding of the pathophysiology of pain has not been reflected by major improvements in the clinical management of many categories of chronic pain, and difficulties remain for a significant group of patients, many of whom may be seen by rheumatologists.

One major advance has been the adoption of a biopsychosocial perspective, and the accompanying article by Large emphasises that the psychological dimensions of chronic pain become more important with increasing chronicity, whether or not organic disease is present. This realisation has led to the development of psychological management strategies and, when appropriate, has allowed treatment to be directed away from the traditional search for a "cause and a cure". Macrae *et al* have stated that today's major challenge is changing doctors' perception of pain and methods of treatment, not just among pain clinicians but in the wider medical community.¹

Evidence based medicine

The modern reader might be amused by 19th century pain management strategies such as metallotherapy, blue light rays, audioanalgesia, and mesmerism. What will the next century make of some of the methods in use in the 1990s when treatments are still being introduced into practice before adequate evaluation? Initial case reports advertise methods which gain a firm foothold in the mythology of pain management before controlled trials are conducted.

To justify continued use of any treatment we must have positive evidence of a positive effect derived from randomised controlled trials or from systematic reviews of published reports. Clinicians who manage chronic pain need to accept that validity should be based on scientific rigour rather than on the reputation or seniority of authors. There is an urgent need to improve the methods by which surgery and high technology methods are evaluated.²

The Cochrane Collaboration has proposed a scheme to prepare and maintain systematic, up to date reviews of all the relevant randomised controlled trials in particular medical fields.³ Sackett *et al* have suggested guidelines for criti-

cal appraisal of published reports so as to distinguish useful from useless or even harmful treatment.⁴ The randomised controlled trial is a cornerstone of critical appraisal.⁴ Application of these guidelines to pain management reveals a paucity of evidence for many popular treatments at a time when the formulation of everything from individual therapy to broad health policy should be based on the evidence of scientific research.

The relevance of much of the published research in chronic pain is greatly diminished by the failure to use standard entry and outcome criteria. The IASP classification of chronic pain provides standard descriptions of chronic pain syndromes and standard definitions of pain terms.⁵ Even in the clinical setting the selection of optimal therapy is impeded by the difficulties in assessing outcome.⁶

Despite all these shortcomings in the hard science of pain management, there are many practitioners who feel that the often desperate state of these patients is enough to justify the use of treatments on a trial and error basis for individual patients. Research into the management of chronic pain is not particularly easy. Many patients with chronic pain have complex symptomatology and are receiving multimodal therapy, so it may be difficult to design double blind, placebo controlled trials to disentangle the effect on the symptoms of the different components of the therapy. This is not to suggest that we should not try!

Appropriateness of care

Assessing the appropriateness of various strategies is a major problem in the management of chronic pain.⁷ Management may sometimes be unhelpful or even harmful. Pither and Nicholas examined 89 patients attending a pain management programme and reported that 66% were receiving inappropriate analgesics, 54% were given inappropriate sedatives and tranquillisers, 60% had been overinvestigated, 70% had been given inappropriate explanations of their pain, 61% had been encouraged to rest, and 40% had been referred to a psychiatrist.⁸

Chronic pain is a complex multidimensional problem and there is not a single pathophysiological mechanism. All chronic pains are not the same. Patients with the same apparent cause can present with very different pains and

Pain Management
Centre, St Thomas
Hospital, London
SE1 7EH,
United Kingdom
D M Justins

Correspondence to:
Dr D M Justins.

Accepted for publication
8 September 1995

Table 1 General principles for chronic pain management

Use a biopsychosocial model rather than a traditional disease model.
Individualise management.
Patient participation in decision making and management is essential.
Multimodal therapy is necessary in most cases.
Use concurrent rather than sequential application of multimodal therapy.
Disability and psychological dysfunction may need special attention.
Choose therapy which has been subjected to critical appraisal.

with very different cognitive and behavioural responses. Lack of understanding of all the mechanisms renders selection of treatments more difficult, but some general principles are suggested in table 1.

Prevention of chronic pain

A small trial reported by Bach *et al* in 1988 provided a tantalising suggestion that preoperative epidural blocks could prevent the development of postamputation pains.⁹ Jahangiri *et al* have shown that perioperative epidural infusion of diamorphine, clonidine, and bupivacaine reduces the incidence of phantom pain after amputation.¹⁰ The possible benefits of “pre-emptive therapy” are not confined to postsurgical pains. Early rehabilitation prevents the development of chronic disability following an episode of acute back pain.¹¹ The use of sympathetic blocks within two months of an attack of acute herpes zoster has been claimed to reduce the incidence of post-herpetic neuralgia. Because of the natural history of back pain and post-herpetic neuralgia large multicentre trials are needed to resolve these questions.

Assessment

Before they reach a pain specialist most patients with chronic pain have already been extensively and expensively investigated so it is often desirable to stop further fruitless searches for a “cause” of the pain.

Quantifying the subjective experience of pain is difficult but distress and functional disability can be measured more easily.¹² Various devices have been used to measure or assess pain and there has recently been interest in the use of dynamometric techniques such as isokinetic measurements of spinal movements, but the validity of this approach requires rigorous evaluation.¹³ There is uncertainty about the validity of many of the other methods of assessment and further research is needed in the whole field. Main *et al* have argued for simpler and more focused types of psychological tests when assessing pain and disability.¹⁴ It is important to distinguish between disability and impairment. As many as one third of chronic pain patients will have no objective findings of somatic disease.¹⁵ There is evidence that compensation issues may interfere with evaluation and treatment of chronic pain patients, who often remain disabled after the litigation is concluded.¹⁶

Intravenous tests can be used to aid diagnosis and selection of treatment.^{17 18} Lignocaine blocks sodium channels and so could suppress

ectopic impulses in damaged nerves. Phentolamine is an α adrenergic receptor blocker so could, in theory, be effective in sympathetically maintained pain. Fentanyl, a μ -opioid receptor agonist, can reveal whether the pain is opioid sensitive when long term opioid treatment is being considered.

Diagnostic nerve blocks have been used but caution is advised in interpreting the results because of the high incidence of false positive and false negative responses. The placebo response may account for many false positives. Local anaesthetic blocks may also be poor prognostic indicators of the response to surgical or chemical neuroablation.¹⁹

Medication

The multiplicity of neurochemicals and receptors types involved in nociception suggest many potential targets and these are discussed in the accompanying article by Dray. The advances in the basic sciences promised exciting new targeted analgesics but these new weapons have not materialised in clinical practice. The standard analgesics are still variations on willowbark (non-steroidal anti-inflammatory drugs, NSAIDs) and poppy (opioids). Of the coanalgesic drugs, amitriptyline, an old antidepressant, and carbamazepine, an old anticonvulsant, are still market leaders.

Opioids

The long term use of opioids in patients with opioid sensitive non-malignant pain remains controversial.^{20 21} The use of opioids should only be considered if other medication has failed and proper trials (for example, intravenous tests) indicate an unequivocal response to the opioid.²² Opioids may be appropriate in a patient with intractable pain following spinal cord injury but inappropriate in a patient with atypical facial pain or a history of drug abuse. Side effects and dependence will occur in most patients and tolerance may develop. Psychological dependence is claimed to be unusual in patients with ongoing opioid sensitive pain.²⁰

Incident pain due to bone secondaries and neuropathic pain often responds poorly or not at all to opioids. Nerve damage may result in a loss of opioid receptors and produce neurochemicals which interfere with opioid receptors. McQuay *et al* described a technique for assessing opioid sensitivity of chronic pain of different aetiologies using patient controlled analgesia.²³ Nociceptive pain responded better but some patients with neuropathic pain still experienced benefit. Rowbothom *et al* showed that some patients with post-herpetic neuralgia will obtain relief when given opioids.²⁴

Another controversy relates to morphine metabolites. In some patients the metabolism of morphine produces an unusually high proportion of morphine 3-glucuronide compared to morphine 6-glucuronide. The 6-glucuronide is a potent analgesic but the 3-glucuronide is inactive or even antagonistic, so that opioid unresponsiveness may result. This has been called paradoxical pain. Methadone is not metabolised in the same way and

may be the drug of choice for these patients.²⁵ The actual existence of this phenomenon is disputed by others.²⁶ Paradoxical hyperalgesia has been reported in a patient on very high epidural morphine dose.²⁷

New routes of administration are being tried. Transdermal fentanyl offers an option for cancer pain. Butorphanol is marketed in the USA in a nasal spray for the relief of migraine. New sustained release preparations of morphine and dihydrocodeine have been developed.²⁸ The morphine preparation contains polymer coated sustained release pellets. Tramadol is a new μ -opioid agonist with additional action on monoamine reuptake and results of thorough trials are awaited.

Some centres still promote the use of spinal opioids for chronic non-malignant pain but there is major uncertainty about the appropriateness of the technique. Yaksh has reviewed the pharmacology.²⁹ Abram states that chronic non-malignant pain is more difficult to control with spinal opioids than cancer pain.³⁰ The suggested indications in cancer pain are tolerance, unreliable absorption by normal routes, unacceptable side effects, and the need for drug combinations such as opioid and local anaesthetic to control neuropathic pain or pathological fracture.^{27 31}

Ambulatory infusion devices have allowed cancer patients greater mobility. Other medication such as an antiemetic can be included. The infusion may be subcutaneous, intravenous, spinal, or intraventricular.³²

The potential neuropsychiatric toxicity of the opioids and other medication in cancer patients has become better appreciated. Psychostimulants such as methylphenidate have been used as adjuvant analgesics. The drugs may potentiate opioid analgesics, counteract opioid induced sedation or cognitive impairment, and thus allow dose escalation in difficult pain problems.³³

Non-steroidal anti-inflammatory drugs

Rheumatologists are familiar with the NSAIDs but pain clinics find little application for these drugs. NSAIDs may be useful in chronic musculoskeletal pains but the increased risk of peptic ulcer is well known. Langman *et al* concluded that ibuprofen and diclofenac produced the lowest risk of gastric bleeding.³⁴ In a preliminary study Vanos *et al* report on intravenous regional blocks using ketorolac for reflex sympathetic dystrophy.³⁵

Coanalgesic or adjuvant medication

Many other drugs have been claimed to be effective in chronic pain but critical appraisal of the evidence is not convincing. Portenoy has reviewed adjuvant/coanalgesic medication for chronic pain.³⁶

Tricyclic antidepressants are effective for neuropathic pain, central pain, atypical facial pain, tension headache, and some musculoskeletal pains.³⁷ The drugs act by enhancement of noradrenergic and serotonergic mechanisms. The analgesic effect is separate from the antidepressant effect and occurs with much lower doses. McQuay *et al* showed a dose

response for analgesia and side effects which was unrelated to mood elevation.³⁸ Amitriptyline, desipramine, and maprotiline have been subjected to controlled trials in postherpetic neuralgia and painful diabetic neuropathy. Leijon and Boivie found that amitriptyline was superior to placebo and carbamazepine in central poststroke pain.³⁹ Dothiepin has been used in atypical facial pain.

The anticonvulsants are useful for trigeminal neuralgia, diabetic neuropathy, and other conditions in which neural damage manifests as epileptiform bursts of spontaneous lightning or shooting pain. Carbamazepine and phenytoin block sodium channels and so depress excitatory transmission. Oxcarbazepine has been used for trigeminal neuralgia.⁴⁰

Clonazepam, alprazolam, and clobazam are claimed to be helpful in certain neuropathic pains but there is little convincing evidence of major inherent analgesic activity for the benzodiazepines. Intrathecal midazolam may act on GABA (γ amino butyric acid) receptors and has been used for cancer pain.

Rowbotham *et al* showed that intravenous lignocaine was effective in post-herpetic neuralgia.⁴¹ In a randomised controlled trial in neuropathic cancer pain, Bruera *et al* were unable to demonstrate any analgesic effect of this technique.⁴² Uncertainty remains over the value of intravenous local anaesthetics in central pain syndromes. Antiarrhythmia drugs such as mexiletine and flecainide are used for neuropathic pain.⁴³

Phenoxybenzamine, phentolamine, and prazosin, as well as corticosteroids and many other drugs, have been recommended for sympathetically maintained pain.⁴⁴ Guanethidine and bretylium are used in an intravenous regional technique. Adrenergic agonists such as clonidine and medetomidine have been used in a range of different pain conditions.

Intravenous calcitonin has been used for phantom limb pain. Naloxone has been claimed to be effective in central pain syndromes but this claim is debated. Calcium channel blockers such as nifedipine and diltiazem have been suggested for peripheral neuropathic pain. The bisphosphonate clodronate produced a beneficial effect in a randomised trial in patients with painful skeletal metastases resulting from breast cancer.⁴⁵

Baclofen, a GABA agonist agent, is also used for neuropathic lancinating pain and by continuous intrathecal infusion in the management of painful spastic paresis in patients with spinal cord injury or multiple sclerosis.⁴⁶

Ketamine is an NMDA (*N*-methyl-*D*-aspartate) receptor antagonist which has been used in the treatment of neuropathic pain.⁴⁷ Stannard and Porter reported successful use of ketamine in three patients suffering from phantom limb pain.⁴⁸

Watson has reviewed the use of topical capsaicin in a variety of conditions and he concludes that it is generally not satisfactory as sole treatment although in some cases it may serve as an adjuvant analgesic.⁴⁹ Capsaicin selectively depletes substance P from terminal

afferents. The burning sensation produced by the capsaicin has frustrated efforts to conduct double blind trials.

Neural blockade in management of chronic pain

Anaesthetists originally became involved in pain management because they performed nerve blocks but now it is thought that the rationale for attacks on the peripheral "wiring" ignores the complex multidimensional pathophysiology of chronic pain. The substances that have been injected include local anaesthetics, steroids, glycerine, and neurolytic solutions such as phenol or alcohol. Physical methods use cryolesions or radiofrequency thermocoagulation. Glycerol suppresses experimental neuroma activity, as does topical corticosteroid.⁵⁰ Intracutaneous or subcutaneous injections of sterile water have been claimed to give good results for whiplash and musculoskeletal trigger point pain syndromes but convincing evidence is lacking. Sclerosant solutions are claimed to destroy nerves and stimulate the formation of scar tissue. Sclerosant injections are beloved by some rheumatologists but not used by any other group.

The management of chronic pain might benefit from an ultra long acting local anaesthetic. Current research efforts are aimed at modifying existing drugs to yield new release mechanisms. Release may be delayed by storing the local anaesthetic in a lipid solution. Sustained or prolonged release may come from pellets containing local anaesthetic and biodegradable polymers, lipid encapsulated local anaesthetic, or microencapsulated local anaesthetic in crystalline form. Korsten *et al* reported on the use of a series of injections of *n*-butyl-*p*-aminobenzoate (a congener of benzocaine) in 12 patients with intractable cancer pain.⁵¹ Once established the sensory block lasted until death in 10 patients—up to 133 days—and was not associated with motor block. The mode of action is not clear but further investigation would seem warranted. In a randomised controlled trial in patients with cancer pain, King *et al* showed prolongation of the anaesthetic effect of epidural bupivacaine up to a mean value of about 12 hours when mixed with 50% glycerin. When given alone glycerin 50% did not produce any analgesic effect.⁵²

There are hundreds of published reports of the use of epidural injections for low back pain but few randomised controlled trials, so we do not possess conclusive scientific evidence about the efficacy or the indications. The prevailing impression is that epidural steroids are beneficial for painful radiculopathy where there may be both a mechanical and an inflammatory component.⁵³ The factors said to be associated with failure of lumbar steroids include: being unemployed because of pain, duration of symptoms over six months, primary diagnosis that is not radiculopathy, and smoking.

There is debate over the safety of epidural steroids. The data sheet for depot methylpred-

nisolone states in bold type in two places that the preparation must not be given by the intrathecal route and there is little dispute about this. The data sheet also states that the preparation is not recommended for the epidural route. The controversy was precipitated by Nelson in 1988, who claimed that the polyethylene glycol in the carrier vehicle could penetrate to the subarachnoid space through the arachnoid villi.⁵⁴ In support he quoted a single case report which described localised dural fibrosis in a patient with lumbar disc disease who had received epidural steroids. Cause and effect are not established by this case and there is little supporting evidence. Cicala *et al* did not detect any significant difference from controls in the tissue reaction when a single dose of depot methylprednisolone was injected into the epidural space of a rabbit.⁵⁵ Abram *et al* failed to show any histological evidence of neurotoxicity following four repeated injections of intrathecal triamcinolone at five day intervals in rats.⁵⁶ It is, however, acknowledged that intrathecal depot methylprednisolone injection is potentially harmful and Nelson claims that epidural injection is dangerous because of the risk of inadvertent dural puncture and subsequent subarachnoid injection of the steroid. Some rheumatologists do not realise that the dural sac may extend to S4 and be easily penetrated by a needle inserted through the sacral hiatus. An authoritative report⁵⁷ from the National Health and Medical Research Council of Australia addressed the question of epidural steroids in great detail and concluded that there was no evidence to support the contention that this route of administration was dangerous.

Facet joint injections and facet joint denervation continue in the repertoire of many pain practitioners, although the justification for these techniques is sometimes based on fairly woolly anatomical thinking.⁵⁸ Local anaesthetic blocks of the medial branch of the dorsal rami are claimed to be specific for the diagnosis of cervical zygapophyseal joint pain.⁵⁹ Other authors have failed to show any convincing link between diagnostic lumbar facet joint blocks and the outcome of either surgical fusion or non-operative treatment.⁶⁰

Paravertebral nerve root blocks are used in cases with uniradicular pain. Many practitioners do not realise that there will be significant epidural spread in about 70% of cases following paravertebral injection so no conclusions should be made about the diagnosis on the basis of the response to an injection.⁶¹

Chemical lumbar sympathectomy is an effective treatment for rest pain and skin ulceration due to occlusive vascular disease in the leg but of much less benefit for claudication. It is difficult to predict outcome before the block. Endoscopic transthoracic sympathectomy of the upper limb is a safe and effective alternative to open cervical sympathectomy in the management of hyperhidrosis as well as vasospastic conditions and sympathetically maintained pain.⁶²

Coeliac plexus block is widely used for pain due to pancreatic cancer but pain from chronic

pancreatitis does not respond as well to coeliac plexus block.⁶³ The use of computerised tomography scanning has been claimed to improve the safety and effectiveness of the block.⁶⁴ Kirvela *et al* describe the advantages of using ultrasonic guidance for lumbar sympathetic and coeliac plexus block.⁶⁵ Davies reviewed 2730 neurolytic coeliac plexus blocks and concluded that major complications (paraplegia with or without loss of sphincter function) happened once per 683 blocks.⁶⁶

Bilateral chemical sympathectomy has been claimed to be an effective treatment for rectal tenesmoid pain due to pelvic carcinoma.⁶⁷ Superior hypogastric plexus block for pelvic cancer pain targets the sympathetic plexus which lies anterior to the sacral promontory.⁶⁸ Block of the ganglion impar has been used for perineal pain in cancer.

Intravenous regional sympathetic block (Bier's block) has enjoyed great popularity for many years. The effects of bretylium and guanethidine on peripheral sympathetic mechanisms were recorded as long ago as 1963.⁶⁹ There is a lack of evidence from methodologically sound randomised controlled trials to support the contention that intravenous guanethidine is of great value in so called reflex sympathetic dystrophy.^{70 71}

Interpleural bupivacaine has been used for post-thoracotomy cancer pain and chronic pancreatitis.⁷² Pleural injection of phenol has been suggested for pain associated with oesophageal cancer. Kirvela and Antila carried out a retrospective review of 281 thoracic paravertebral blocks performed for chronic postoperative pain and suggest that it is a reliable and safe technique for unilateral pain.⁷³

Stimulation produced analgesia

Acupuncture and TENS (transcutaneous electrical nerve stimulation) are widely used for chronic pain but the clinical response is variable and unpredictable. Some patients do gain significant benefit without serious adverse effects.⁷⁴ Headaches, musculoskeletal disorders, and some neuropathic pains may respond well to these techniques. Transcutaneous electrical spinal stimulation is a new technique which awaits proper evaluation.

In a randomised controlled trial Vincent compared true and sham acupuncture in migraine and showed benefits for true acupuncture.⁷⁵ Deluze *et al* reported a three weeks double blind randomised trial of electroacupuncture in fibromyalgia.⁷⁶ The results showed a significant improvement in the active treatment group.

Deafferentation pain may respond to neurostimulation. Pain following peripheral nerve injury may respond to peripheral nerve stimulation in about 51% of cases.⁷⁷ Peripheral nerve repair is helpful for others.⁷⁸ It is now obvious that pain following peripheral nerve injury does not respond regularly or reliably to sympathetic blockade.

In some centres there is currently great enthusiasm for spinal cord stimulation which has been claimed to help arachnoiditis,

postlaminectomy syndrome, peripheral vascular disease, peripheral nerve injury, postamputation pain, painful peripheral neuropathy, spinal cord injury, and intractable angina. The exact mode of action of spinal cord stimulation remains a mystery. The benefit in peripheral vascular disease and intractable angina may relate to physiological changes quite separate from pain mechanisms.⁷⁹ Gybels concludes that spinal cord stimulation is not a simple intervention but a time consuming and rather expensive technique.⁷⁷ Deep brain stimulation is generally applied through an electrode implanted in the periventricular grey matter of the posterior end of the third ventricle or in the sensory thalamus, but why electrical stimulation of the brain suppresses chronic pain is not well understood.

Neurosurgery

The realisation that cutting the nervous system anywhere is rarely the answer to chronic pain has led to a reduction in the use of neuroablative surgery.⁸⁰ Modern techniques such as dorsal root entry zone lesioning are indicated in conditions such as brachial plexus avulsion. Stuart and Crammond reported on 273 patients who underwent percutaneous cervical cordotomy for pain of malignant origin.⁸¹ They concluded that this remains a valuable technique, particularly for patients with lung cancer and mesothelioma. Gybels agrees that the operation is useful for unilateral pain in the extremities and trunk but observes that percutaneous anterolateral cordotomy is not an easy procedure and requires continuing practice in order to maintain consistently good results.⁷⁷ Selective posterior rhizotomy may be considered for Pancoast's syndrome. Spinal surgery should always be considered in the treatment of metastatic back pain, and pain resulting from bony metastases should be managed with operative immobilisation where instability exists.^{82 83}

Physical therapy

The Clinical Standard Advisory Group Committee on Back Pain (in the United Kingdom) has published an epidemiology review and management guidelines for low back pain which highlight the need for active rehabilitation within the first six weeks of onset in order to prevent long term pain and disability.^{84 85}

Koes *et al*,⁸⁶ in a randomised trial for patients with persistent back and neck pain, reported that manipulative therapy and physiotherapy were superior to general practitioner or placebo treatment at one year follow up, but another study found manipulation only useful for acute episodes of back pain and of very little benefit in chronic back pain.⁸⁷

Frost *et al* have conducted a randomised controlled trial to evaluate a fitness programme for patients with chronic low back pain and they showed that moderately disabled patients with chronic low back pain who attend a back-school and fitness programme benefit more in the short and long term than patients who attend a backschool and exercise independ-

ently at home.⁸⁸ There were significant differences between the groups in measures of disability, self efficacy, and walking distance, as well as in the sensory and affective components of pain. This trial shows the need to assess a wide range of measures and not just pain alone.⁸⁹ In another study 87% of 116 patients who participated in a functional restoration programme were working at two year follow up compared with only 41% of the 72 patients in the untreated control group.¹¹ Rehabilitation programmes can be vital even for cases with irreversible damage such as avulsion lesions of the brachial plexus.⁹⁰

Psychological techniques

Many patients with chronic pain have been in the traditional medical system for a long time. The expectation of doctor and patient is that the history, examination, and investigation will lead to a diagnosis which will result in a specific treatment and cure. The traditional disease model works for acute pain but not for chronic pain, and relief is not forthcoming. The patient experiences increasing helplessness, hopelessness and dependence, depression, social withdrawal, and family breakdown which may all be superimposed on any pre-existing psychological problems.

Cognitive behavioural approaches aim to achieve the best possible level of physical and emotional function within the confines of the condition. It is important to recognise that not all pain behaviour is inappropriate or "pathological". The patients who will benefit from psychological approaches are those in whom the behaviours are exaggerated or clearly inappropriate.⁹¹ Pain reduction is not an aim but may follow the improvement in function. Treatment involves behaviour modification, physical rehabilitation, medication reduction, education, cognitive therapy, group discussion, relaxation techniques, family therapy, and individual psychological support. It is important to decrease catastrophising and to increase perceived patient control over pain. Participation of the spouse or family is vital.

Outcome studies which analyse pain, function, psychology, and economics suggest that these programmes are effective.⁹² Williams *et al* showed increased activity, decreased medication, and decreased reported pain experience.⁹³ Patients are more likely to return to work and to normal day to day activity. Quality of life improves. Peters *et al* have shown the significant cost benefits of a pain management programme in returning patients to useful employment.⁹⁴ There is a need to improve pretreatment assessment so as to match patients to the appropriate treatment package. There are other questions. What is the optimal time point for intervention? What bits of the programme are important? How do we manage patients resistant to treatment? How do we ensure adherence to the programme? As the programme increases in complexity then compliance falls off dramatically!

Management strategies for specific problems

Postamputation pain syndromes including phantom pain and stump pain are very common but there is no reliable treatment.⁹⁵ This is a classic example of a chronic pain syndrome where attacks on the periphery are often fruitless, suggesting that the pathophysiological disturbance has migrated centrally although the initial injury was clearly peripheral.

Chronic pelvic pain is a very common complaint and these patients may even be referred to rheumatologists. The treatment of chronic pelvic pain without obvious pathology remains unsatisfactory but psychological techniques have benefited some.⁹⁶

There are currently some "fashionable conditions" which worry rheumatologists and pain clinicians. Patients with the chronic fatigue syndrome often complain of widespread muscle pain. Management is difficult and a substantial proportion of patients may remain functionally impaired.⁹⁷ The very existence of repetitive strain injury is challenged.⁹⁸ Several workers dispute the existence of fibromyalgia as a distinct disease entity and suggest that the tender points which are a crucial part of the diagnosis of fibromyalgia are a measure of general distress and, although related to pain, are associated separately with fatigue and depression.^{99 100}

Recent reviews have highlighted the complexity of chronic low back pain.^{101 102} The new IASP classification⁵ emphasises the important distinction between spinal pain (with or without referred pain) and radicular pain. The term "sciatica" is an anachronism and should not be used. Multimodal management which is focused upon rehabilitation is often the most appropriate strategy. Surgery, especially repeat surgery, should only be used for definite indications. Remember the maxim "Never operate on pain, only operate on pathology".

Osteoporosis is a frequent cause of pain and disability, especially in older women. The bisphosphonates may aid in the prevention and treatment of osteoporosis, although the effect on fracture rate is uncertain.¹⁰³ The management of the incident pain which results from fractures of an osteoporotic spine is still a major problem and is often unsatisfactory in older patients who may be intolerant of powerful medication and corsets while being incapable of regaining muscle strength.

The term "whiplash" has been replaced by acceleration deceleration injury of the neck or cervical sprain injury.¹⁰⁴ Pain will persist for over six months in about 25% of cases and it is likely that by this time psychological, behavioural, and economic factors may complicate the original injury. No single treatment can be recommended.

There are 20 000 patients in the United Kingdom with spinal cord injury, and it has been estimated that 50% suffer from persistent pain which in many cases is difficult to manage. Pain is more common after incomplete injuries to the cord and is typically neuropathic but it may also be due to associated musculoskeletal

Table 2 Complex regional pain syndrome - type I

Diagnostic criteria

- 1 Initiating noxious event or cause of immobilisation.
- 2 Continuing pain, allodynia, or hyperalgesia - pain is disproportionate to inciting event.
- 3 At some time - oedema, changes in skin blood flow, abnormal sudomotor activity.
- 4 No other condition to account for the degree of pain and dysfunction.

Criteria 2 - 4 must be satisfied.

Table 3 Complex regional pain syndrome - type II

Diagnostic criteria

- 1 Continuing pain, allodynia, or hyperalgesia after a nerve injury.
- 2 At some time - oedema, changes in skin blood flow, abnormal sudomotor activity.
- 3 No other condition to account for the degree of pain and dysfunction.

All three criteria must be satisfied.

problems such as vertebral instability. In general a multidisciplinary rehabilitation oriented approach, including psychological techniques, is most likely to benefit these patients.¹⁰⁵

The use of the term "reflex sympathetic dystrophy" (RSD) was regarded as very unsatisfactory since many of the cases to which it was applied either did not show any signs of sympathetic disturbance and dystrophy or even if they did, they failed to respond to sympathetic block. A new term "complex regional pain syndrome" (CRPS) type I should replace RSD⁵ (table 2). Causalgia is CRPS type II (table 3). Sympathetically maintained pain (pain relieved by a specific sympathetic block) may be found in association with either of these syndromes. Irrespective of the name, management remains difficult in many cases. Physical therapy may be the most important treatment. Other treatments include somatic nerve blocks, plexus or epidural infusions, sympathetic nerve blocks, intravenous regional blocks, surgical sympathectomy, TENS and spinal cord stimulation, and a vast array of drugs including corticosteroids, tricyclics, prazosin, phenoxymethylamine, nifedipine, labetalol, propranolol, clonidine, and topical capsaicin.⁴⁴ Intra-articular injections of steroids and local anaesthetic have been used by some practitioners when joints have become grossly restricted.

Conclusion

Current management strategies produce satisfactory relief for many patients, but chronic pain will remain intractable in a significant proportion. Even these intractable cases can be offered help following the recognition of the multidimensional nature of chronic pain and of the need to provide specific services to manage these patients. The biopsychosocial model is of more relevance than the traditional medical model, and rehabilitation, rather than cure, may be a more realistic goal for many patients. In the future we may see more specific

treatments based on an improved understanding of the specific pathophysiology of different pain syndromes but for the moment there are no "magic bullets".

- 1 Macrae WA, Davies HTO, Crombie IK. Pain: paradigms and treatments. *Pain* 1992;49:289-91.
- 2 Large R, Peters J. A critical appraisal of outcome of multidisciplinary pain clinic treatments. In: Bond MR, Charlton JE, Woolf CJ, eds. *Proceedings of the VIIth World Congress on Pain*. Amsterdam: Elsevier, 1991:417-27.
- 3 Chalmers I, Haynes B. Reporting, updating, and correcting systematic reviews of the effects of health care. *BMJ* 1994;309:862-5.
- 4 Sackett DL, Haynes RB, Guyatt GH, Tugwell P. *Clinical epidemiology: a basic science for clinical medicine*, 2nd ed. Boston: Little Brown, 1991:193.
- 5 Merskey H, Bogduk N. *Classification of chronic pain: descriptions of chronic pain syndromes and definitions of pain terms*, 2nd ed. Seattle: IASP Press, 1994.
- 6 Turk DC, Rudy TE, Sorkin BA. Neglected topics in chronic pain treatment outcome studies: determination of success. *Pain* 1993;53:3-16.
- 7 Hicks NR. Some observations on attempts to measure appropriateness of care. *BMJ* 1994;309:730-3.
- 8 Pither CE, Nicholas MK. The identification of iatrogenic factors in the development of chronic pain syndromes: abnormal treatment behaviour? In: Bond MR, Charlton JE, Woolf CJ, eds. *Proceedings of the VIIth World Congress on Pain*. Amsterdam: Elsevier, 1991:429-34.
- 9 Bach S, Noreng MF, Tjelliden NU. Phantom limb pain in amputees during the first 12 months following limb amputation after preoperative lumbar epidural blockade. *Pain* 1988;33:297-301.
- 10 Jahangiri M, Bradley JWP, Jayatunga AP, Dark CH. Prevention of phantom pain after major lower limb amputation by epidural infusion of diamorphine, clonidine and bupivacaine. *Ann R Coll Surg Engl* 1994;76:324-6.
- 11 Mayer TG, Gatchel RJ, Mayer H, Kishino ND, Keeley J, Mooney V. A prospective two year study of functional restoration in industrial low back injury: an objective assessment procedure. *JAMA* 1987;258:1763-7.
- 12 Waddell G, Pilowsky I, Bond MR. Clinical assessment and interpretation of abnormal illness behaviour in low back pain. *Pain* 1989;39:41-53.
- 13 Jayson MV. Trauma, back pain, malingering, and compensation. *BMJ* 1993;305:7-8.
- 14 Main CJ, Evans PJD, Whitehead RC. An investigation of personality structure and other psychological features in patients presenting with low-back pain: a critique of the MMPI. In: Bond MR, Charlton JE, Woolf CJ, eds. *Proceedings of the VIIth World Congress on Pain*. Amsterdam: Elsevier, 1991:207-17.
- 15 Rosomoff HL, Fishbain DA, Goldberg M, Santana R, Steele Rosomoff R. Physical findings in patients with chronic intractable benign pain of the neck and or back. *Pain* 1989;37:279-87.
- 16 Mendelson G. Compensation and chronic pain. *Pain* 1992;48:121-3.
- 17 Raja S, Treede R, Davis K, Campbell J. Systemic alpha-adrenergic blockade with phentolamine: a diagnostic test for sympathetically maintained pain. *Anesthesiology* 1991;74:691-8.
- 18 Arner S. Intravenous phentolamine test: diagnostic and prognostic use in reflex sympathetic dystrophy. *Pain* 1991;46:17-22.
- 19 Boas RA. Nerve blocks in the diagnosis of low back pain. *Neurosurg Clin* 1991;2:807-16.
- 20 Portenoy RK, Foley KM. Chronic use of opioid analgesics in non-malignant pain: report of 38 cases. *Pain* 1986;25:171-86.
- 21 Wall PD. Neuropathic pain. *Pain* 1990;43:267-8.
- 22 Jadad AR, Carroll D, Glynn CJ, Moore RA, McQuay HJ. Morphine responsiveness in chronic pain: double blind randomised cross over study with PCA. *Lancet* 1992;339:1367-71.
- 23 McQuay HJ, Jadad AR, Carroll D, Faura C, Glynn CJ, Moore RA, et al. Opioid sensitivity of chronic pain: a patient-controlled analgesia method. *Anaesthesia* 1992;47:757-67.
- 24 Rowbotham MC, Reisner-Keller LA, Fields HL. Both intravenous lidocaine and morphine reduce the pain of postherpetic neuralgia. *Neurology* 1991;41:1024-8.
- 25 Bowsher D. Paradoxical pain. *BMJ* 1993;306:473.
- 26 Davis CL, Hardy JR. Palliative care. *BMJ* 1994;308:1359-62.
- 27 Hogan Q, Haddox JD, Abram S, Weissman D, Taylor ML, Janjan N. Epidural opiates and local anaesthetics for the management of cancer pain. *Pain* 1991;46:271-9.
- 28 Gourlay GK, Plummer JL, Cherry DA, Onley MM. A comparison of Kapanol (a new sustained release morphine formulation), MST Continus, and morphine solution in cancer patients: pharmacokinetic aspects of morphine and morphine metabolites. In: Gebhart GF, Hammond DL, Jensen TS, eds. *Proceedings of the 7th World Congress on Pain*. Seattle: IASP Press, 1994:631-43.
- 29 Yaksh TL. The spinal pharmacology of acutely and chronically administered opioids. *J Pain Symptom Manage* 1992;7:356-61.
- 30 Abram SE. Advances in chronic pain management since gate control. *Regional Anesth* 1993;18:66-81.

- 31 Du Pen S, Kharasch ED, Williams A, Petersen DG, Sloan DC, Hasche-Klunder H, *et al.* Chronic epidural bupivacaine-opioid infusion in intractable cancer pain. *Pain* 1992;49:293-300.
- 32 Bruera E. Ambulatory infusion devices in the continuing care of patients with advanced disease. *J Pain Symptom Manage* 1990;5:287-96.
- 33 Bruera E, Watanabe S. Psychostimulants as adjuvant analgesics. *J Pain Symptom Manage* 1994;9:412-5.
- 34 Langman MJS, Weil J, Wainwright P, Lawson DH, Rawlins MD, Logan RFA, *et al.* Risks of peptic ulcer associated with individual non-steroidal anti-inflammatory drugs. *Lancet* 1994;343:1075-8.
- 35 Vanos DN, Ramamurthy S, Hoffman J. Intravenous regional block using ketorolac: preliminary results in the treatment of reflex sympathetic dystrophy. *Anesth Analges* 1992;74:139-41.
- 36 Portenoy RK. Adjuvant analgesics in pain management. In: Doyle D, Hanks GWC, MacDonald N, eds. *Oxford textbook of palliative medicine*. Oxford: Oxford University Press, 1993:187-203.
- 37 Watson CPN. Antidepressant drugs as adjuvant analgesics. *J Pain Symptom Manage* 1994;9:392-405.
- 38 McQuay HJ, Carroll D, Glynn CJ. Dose-response for analgesic effect of amitriptyline in chronic pain. *Anaesthesia* 1993;48:281-5.
- 39 Leijon G, Boivie J. Central post-stroke pain - a controlled trial of amitriptyline and carbamazepine. *Pain* 1989;36:27-36.
- 40 Zakrzewska JM, Patsalos PN. Oxcarbazepine: a new drug in the management of intractable trigeminal neuralgia. *J Neurol Neurosurg Psychiatry* 1989;52:472-6.
- 41 Rowbotham MC, Reisner-Keller LA, Fields HL. Both intravenous lidocaine and morphine reduce the pain of postherpetic neuralgia. *Neurology* 1991;41:1024-8.
- 42 Bruera E, Ripamonti C, Brenneis C, Macmillan K, Hanson J. A randomised double-blind crossover trial of intravenous lidocaine in the treatment of neuropathic cancer pain. *J Pain Symptom Manage* 1992;7:138-40.
- 43 Glazer S, Portenoy RK. Systemic local anesthetics in pain control. *J Pain Symptom Manage* 1991;6:30-9.
- 44 Charlton JE. Reflex sympathetic dystrophy. Non-invasive methods of treatment. In: Stanton-Hicks M, Janig W, Boas RA, eds. *Reflex sympathetic dystrophy*. Boston: Kluwer, 1990:151-64.
- 45 Patterson AHG, Powles TJ, Kanis JA, McCloskey E, Hanson J, Ashley S. Double blind controlled trial of oral clodronate in patients with bone metastases from breast cancer. *J Clin Oncol* 1993;11:59-65.
- 46 Penn RD, Savoy SM, Corcos D, *et al.* Intrathecal baclofen for severe spinal spasticity. *N Engl J Med* 1989;320:1517-21.
- 47 Dickenson AH. NMDA receptor antagonists as analgesics. In: Fields HL, Liebeskind JC, eds. *Progress in pain research and management*, vol 1. Seattle: IASP Press, 1994:173-87.
- 48 Stannard CF, Porter GE. Ketamine hydrochloride in the treatment of phantom limb pain. *Pain* 1993;54:227-30.
- 49 Watson CPN. Topical capsaicin as an adjuvant analgesic. *J Pain Symptom Manage* 1994;9:425-33.
- 50 Devor M, Govrin-Lippmann R, Raber P. Corticosteroids suppress ectopic neuronal discharge originating in experimental neuromas. *Pain* 1985;22:127-37.
- 51 Korsten HHM, Ackerman EW, Grouls RJ, *et al.* Long-lasting epidural sensory blockade by n-butyl-p-aminobenzoate in the terminally ill intractable cancer pain patient. *Anesthesiology* 1991;75:950-60.
- 52 King H-K, Xiao C-S, Wooten DJ. Prolongation of epidural bupivacaine analgesia with glycerin. *Can Anesth Soc J* 1993;40:431-4.
- 53 Rowlington JC. Epidural steroids. Do they have a place in management? *American Pain Society Journal* 1994;3:20-7.
- 54 Nelson DA. Dangers from methylprednisolone acetate therapy by intraspinal injection. *Arch Neurol* 1988;45:804-6.
- 55 Cicala RS, Turner R, Moran E, *et al.* Methylprednisolone acetate does not cause inflammatory changes in the epidural space. *Anesthesiology* 1990;72:556-8.
- 56 Abram SE, Marsala M, Yaksh TL. Analgesic and neurotoxic effects of intrathecal steroids in rats [abstr]. *Anesthesiology* 1992;77:A810.
- 57 National Health and Medical Research Council. *Epidural use of steroids in the management of back pain and sciatica of spinal origin*. Canberra: NHMRC, 1994.
- 58 Stolker RJ, Vervest ACM, Groen GJ. The management of spinal pain by blockades: a review. *Pain* 1994;58:1-20.
- 59 Barnsley L, Bogduk N. Medial branch blocks are specific for the diagnosis of cervical zygapophysial joint pain. *Regional Anesth* 1993;18:343-50.
- 60 Esses SI, Moro JK. The value of facet joint blocks in patient selection for lumbar fusion. *Spine* 1993;18:185-90.
- 61 Purcell-Jones G, Pither CE, Justins DM. Paravertebral somatic nerve block. A clinical and radiographic study. *Anesth Analges* 1989;68:32-9.
- 62 Byrne J, Walsh TN, Hederman WP. Endoscopic transthoracic electrocautery of the sympathetic chain for palmar and axillary hyperhidrosis. *Br J Surg* 1990;77:1040-9.
- 63 Mercadante S. Celiac plexus block versus analgesics in pancreatic cancer pain. *Pain* 1993;52:182-92.
- 64 Brown DL, Moore DC. The use of neurolytic celiac plexus block for pancreatic cancer: anatomy and technique. *J Pain Symptom Manage* 1988;3:206-9.
- 65 Kirvela O, Svedstrom E, Lundblom N. Ultrasonic guidance of lumbar sympathetic and coeliac plexus block. A new technique. *Regional Anesth* 1992;17:43-6.
- 66 Davies DD. Incidence of major complications of neurolytic coeliac plexus block. *J R Soc Med* 1993;86:264-6.
- 67 Bristow A, Foster JMG. Lumbar sympathectomy in the management of rectal tenosmoid pain. *Ann R Coll Surg Engl* 1988;70:38-9.
- 68 Plancarte R, Amescua C, Patt RB, Aldrete JA. Superior hypogastric plexus block for pelvic cancer pain. *Anesthesiology* 1990;73:236-9.
- 69 Cooper CJ, Fewings JD, Hodge RL, Whelan RF. Effects of bretylium and guanethidine on human hand and forearm vessels and on their sensitivity to noradrenaline. *Br J Pharmacol* 1963;21:165-73.
- 70 Blanchard J, Ramamurthy S, Walsh N, Hoffman J, Schoenfeld L. Intravenous regional sympatholysis: a double blind comparison of guanethidine, reserpine, and normal saline. *J Pain Symptom Manage* 1990;5:357-61.
- 71 Jadad AR, Carroll D, Glynn CJ, McQuay HJ. Intravenous regional sympathetic blockade for pain relief in reflex sympathetic dystrophy: a systematic review and a randomised, double-blind crossover study. *J Pain Symptom Manage* 1995;10:13-20.
- 72 Fineman SP. Long-term post-thoracotomy cancer pain management with interpleural bupivacaine. *Anesthes Analges* 1989;68:694-7.
- 73 Kirvela O, Antila H. Thoracic paravertebral block in chronic postoperative pain. *Regional Anesth* 1992;17:348-50.
- 74 Johnson MI, Ashton CH, Thompson JW. An in depth study of long term users of transcutaneous electrical nerve stimulation (TENS). Implications for clinical use of TENS. *Pain* 1991;44:221-9.
- 75 Vincent CA. A controlled trial of the treatment of migraine by acupuncture. *Clin J Pain* 1989;5:305-12.
- 76 Deluze C, Bosia L, Zirbs A, Chantraine A, Vischer TL. Electroacupuncture in fibromyalgia: results of a controlled trial. *BMJ* 1992;305:1249-52.
- 77 Gybels JM. Indications for the use of neurosurgical techniques in pain control. In: Bond MR, Charlton JE, Woolf CJ, eds. *Proceedings of the Vth World Congress on Pain*. Amsterdam: Elsevier, 1991:475-82.
- 78 Campbell JN, Raja SN, Meyer RA. Painful sequelae of nerve injury. In: Dubner R, Gebhart GF, Bond MR, eds. *Pain research and clinical management*, vol 3. Amsterdam: Elsevier, 1988:135-43.
- 79 Tallis R, Jacobs M, Miles JB. Spinal cord stimulation in peripheral vascular disease. *Br J Neurosurg* 1992;6:101-5.
- 80 Tasker RR. Management of nociceptive, deafferentation and central pain by surgical intervention. In: Fields HL, ed. *Pain syndromes in neurology*. London: Butterworth, 1990:143-200.
- 81 Stuart G, Crammond T. Role of percutaneous cervical cordotomy for pain of malignant origin. *Med J Aust* 1993;158:667-70.
- 82 Fallon MT, O'Neill WM. Spinal surgery in the treatment of metastatic back pain. *Palliative Med* 1993;7:235-8.
- 83 Chan D, Carter SR, Grimer RJ, Sneath RS. Endoprosthetic replacement for bone metastases. *Ann R Coll Surg Engl* 1992;74:13-18.
- 84 Clinical Standard Advisory Group. *Back pain*. London: HMSO, 1994.
- 85 Clinical Standard Advisory Group. *Epidemiology review: the epidemiology and cost of back pain*. London: HMSO, 1994.
- 86 Koes BW, Bouter LM, van Mameren H, *et al.* Randomised clinical trial of manipulative therapy and physiotherapy for persistent back and neck complaints: results of one year follow up. *BMJ* 1992;304:601-5.
- 87 Meade TW, Dyer S, Browne W, Townsend J, Frank AO. Low back pain of mechanical origin: randomised comparison of chiropractic and hospital outpatient treatment. *BMJ* 1990;300:1431-7.
- 88 Frost H, Klaber Moffett JA, Moser JS, Fairbank JCT. Randomised controlled trial for evaluation of fitness programme for patients with chronic low back pain. *BMJ* 1994;310:151-4.
- 89 Waddell G. Clinical assessment of lumbar impairment. *Clin Orthop Res* 1987;221:110-20.
- 90 Wynn Parry CB. Pain in avulsion lesions of the brachial plexus. *Pain* 1980;9:41-53.
- 91 Fordyce WE. Learning processes in pain. In: Sternbach RA, ed. *The psychology of pain*. New York: Raven Press, 1986:49-65.
- 92 Flor H, Fydrich T, Turk DC. Efficacy of multidisciplinary pain treatment centres: a meta-analytic review. *Pain* 1992;49:221-30.
- 93 Williams ACdeC, Nicholas MK, Richardson PH, *et al.* Evaluation of a cognitive behavioural programme for rehabilitating patients with chronic pain. *Br J Gen Pract* 1993;43:513-8.
- 94 Peters J, Large RG, Elkind G. Follow up results from a randomised controlled trial evaluating in- and outpatient pain management programmes. *Pain* 1992;50:41-50.
- 95 Davis RW. Phantom sensation, phantom pain and stump pain. *Arch Phys Med Rehabil* 1993;74:79-91.
- 96 Steege JF, Stout AL, Somkuti SG. Chronic pelvic pain in women: toward an integrative model. *Obstet Gynecol Surv* 1993;48:95-110.
- 97 Wilson A, Hiskie I, Lloyd A, Hadzi-Pavlovic D, Boughton C, Dwyer J, *et al.* Longitudinal study of outcome of chronic fatigue syndrome. *BMJ* 1994;308:756-9.
- 98 Barton NJ, Hooper G, Noble J, Steel WM. Occupational causes of disorders in the upper limb. *BMJ* 1992;304:309-11.
- 99 Croft P, Schollum J, Silman A. Population study of tender point counts and pain as evidence of fibromyalgia. *BMJ* 1994;309:696-9.
- 100 Cohen ML, Quinter JL. Fibromyalgia syndrome, a problem of tautology. *Lancet* 1993;342:906-9.

- 101 Frank A. Low back pain. *BMJ* 1993;**306**:901-9.
- 102 Jayson MIV. Mechanisms underlying chronic back pain. *BMJ* 1994;**309**:681-2.
- 103 Compston JE. The therapeutic use of bisphosphonates. *BMJ* 1994;**309**:711-5.
- 104 Barnsley L, Lord S, Bogduk N. Whiplash injury. *Pain* 1994;**58**:283-307.
- 105 Boivie J. Central pain. In: Wall PD, Melzack R, eds. *Textbook of pain*, 3rd ed. Edinburgh: Churchill Livingstone, 1994:871-902.

ARD

Annals of the Rheumatic Diseases

invite you to visit our stand # 640
at the 60th ACR meeting!

For more details, contact Ruth Straube on:
+44 (0) 171 383 6486

AMERICAN COLLEGE OF RHEUMATOLOGY
ASSOCIATION OF RHEUMATOLOGY
HEALTH PROFESSIONALS