Chronic pain syndromes in the emergency department: Identifying guidelines for management

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Abstract

Objectives: To explore current literature on chronic pain syndromes and develop ED recommendations for the management and minimalization of chronic non-cancer pain.

Methods: A focused literature review.

Results: Chronic pain is a common presentation to the ED but is poorly understood and managed. Research into the psychophysiology of chronic pain shows that there are definite changes in the receptive and processing pathways in patients suffering chronic pain syndromes. Evidence shows the effectiveness of early recognition with multimodal treatment, however high level evidence is lacking. All experts recommend balanced drug therapy, cognitive and behavioural interventions. Certain interventions are appropriate to the ED setting.

Conclusions: Emergency Medicine lacks a cohesive, informed strategy for management of chronic pain. The proposed guidelines represent the first step toward establishing consistency in the management of patients with chronic pain syndromes. Further work needs to be undertaken at a national level in developing evidence based guidelines.

Key words: emergency treatment, pain and chronic disease.

Chronic pain: Introduction

Pain is a common reason for presentation to the ED. Emergency physicians deal with acute pain generously, using the wide range of therapeutic options at their disposal. Likewise, terminal cancer patients with pain are treated with utmost sympathy. In contrast, for a number of reasons, physicians may feel a sense of anger and helplessness when confronted with chronic unexplained pain. First, chronic pain sufferers are difficult to differentiate from narcotic seekers, particularly after hours with limited time available. Second, they may be suffering from an intercurrent acute illness or stress. Most have often undergone extensive investigations, have multiple health care professionals involved, and rarely have a prewritten health plan available. Finally, the mechanisms of chronic pain are poorly understood and this
may engender a sense of disbelief in emergency physicians.

Research demonstrates that chronic pain is prevalent in Western Society (Table 1) Cordell et al. recorded pain as the ‘chief complaint for visit’ in 52% of 1665 consecutive ED visits.1 Of these, 38% were identified as having specific chronic pain syndromes. This equates to almost one fifth of emergency visits in his study.

The purpose of this literature review is to summarize the recent advances in the management of chronic pain syndromes. I would like to change the perspective of the ED physician. Differentiating the various phenomena such as pseudoaddiction, neuropathic pain, complex regional pain, visceral, somatoform and chronic unexplained pain has little clinical advantage to the busy ED physician but the common ‘rescue medication options’ are vital. In this article, I will review current emergency department research and non-emergency department research, and then propose a practical guide to ED care of chronic pain. Background research into neurophysiological advances is written up as a separate occasional report.

### Methods

Literature was searched via Medline, Cochrane, Cinahl, and PsycINFO from 1996 to 2004, under ‘chronic pain and emergency management’. Medline from 1996 was searched for ‘chronic pain and prevention’, ‘chronic pain and emergency’, ‘breakthrough pain’, and ‘chronic pain’. This was extended back to 1966 under ‘chronic pain and emergency’. Bibliographies were manually searched for older keynotes articles. Four major emergency medicine textbooks (two Australian), the Australian Therapeutic Guidelines (Analgesia) and eMedicine Clinical Knowledge Base were likewise scanned. Articles focusing on acute pain, cancer or non-cancer terminal pain were discarded.

Background research into the advances in understanding of pathophysiology of chronic pain used ‘chronic pain’ reviews from Medline searched from 1996.

This is summarized in a separate article, although certain references are used in both articles.

Relevant research has been grouped and reported under headings of

1. Emergency medicine research in chronic pain management
2. Non-emergency research – treatment, risk identification and rationalizing referral and investigation.

Guidelines for the ED are proposed from the above research.

### Table 1. Studies quantifying prevalence of chronic pain syndromes in adults

<table>
<thead>
<tr>
<th>Author</th>
<th>Prevalence (estimated percentage of general population)</th>
<th>Sample size</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nielson29 (quotes Millar42)</td>
<td>17% —</td>
<td>—</td>
</tr>
<tr>
<td>Schochat34†</td>
<td>13.5% 3174</td>
<td>—</td>
</tr>
<tr>
<td>Papageorgiou44†</td>
<td>10–11% 1386</td>
<td>—</td>
</tr>
<tr>
<td>Elliot45</td>
<td>46.5% 3605</td>
<td>—</td>
</tr>
<tr>
<td>Singh and Patel13</td>
<td>35% —</td>
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†These studies surveyed only chronic widespread pain, excluding regional pain syndromes.

1. Emergency medicine research in chronic pain management

The search of medical literature databases suggested little input from emergency physicians into the management of chronic pain in their departments. There were no prospective trials; all were reviews or expert opinion articles. In 1996, Afilalo, Canettes and Ducharme searched emergency medicine literature for any studies on pain. One textbook, two chapters in other texts, and 60 articles were found. These were mostly clinical research, based around particular painful conditions. They concluded that emergency physicians should do more research in early pain control.

Review of emergency medicine journals specifically for chronic pain research, reveals little else since 1996. Ten relevant references are summarized below.

In 1996, MacLeod and Swanson wrote up an extensive but promising system for managing chronic pain in Calgary, Canada, however, neither researcher has reported on the outcome of their intervention in emergency literature. Interestingly, they audited 1882 non-serious headache presentations and estimated that only 29 patients were likely to be drug seeking. Along with Ducharme, they argue that the label of ‘drug-seeking’ is too often applied, narcotics are withheld and the patient either treated too conservatively, or overinvestigated. Assuming all to be drug seekers adversely affects the majority who present with genuine chronic pain. Stereotypic ‘drug-seeking’ behaviours that disappear with adequate
pain management — narcotic or otherwise — is termed 'pseudoaddiction'.

In a series of review articles entitled ‘Orthopaedic pitfalls in the ED’, Perron and associates note that chronic pain may well follow misdiagnosis of scaphoid fractures, lunate and perilunate injuries, Lisfranc injuries and osteomyelitis. They recommend early diagnosis and appropriate treatment of the acute injury.

One article by Ferrari, in the Emergency Medicine Journal is excellent. It is the only article that acknowledges and tries to address the cognitive aspects of chronic pain. It describes prevention of chronic pain after whiplash, using education and reassurance to encourage more healthy behaviours post injury.

Lummus and Thompson, in Emergency Medicine Clinics of North America, review the management of prostatitis, and its occasional progression to a chronic pain focus. Suggested strategies for treatment of non-inflammatory, chronic prostatitis include diazepam and baclofen, antiadrenergics and psychotherapy. The nature of the underlying abnormality is not addressed in any detail.

The Audio-Digest of Emergency Medicine devoted one session to Professor Prager, lecturing on chronic pain management, in June 2003. He outlined neurostructural changes, the addiction/pseudoaddiction/dependence quandary and the need for multimodal therapy. There was no emphasis on cognitive or behavioural interventions.

Ducharme reviewed acute pain and pain control in 2000. Of the 18 pages, one is devoted to chronic pain and sets out very important general guidelines for approach to these patients. He emphasizes the difficulties in identification of these patients, and the complexities involved with the chronic pain. Opioids may be required, an exacerbating physical cause should be excluded, depression should be treated and the physician must explain the limitations of the ED with empathy. Very truly, he concludes that ‘Pain control is optimized by increasing our understanding of the neurobiology of pain combined with interaction with the patient: science and knowledge combined with empathy and humanity’.

Singh and Patel, in their June 2004 update in eMedicine, give a well rounded general summary of chronic pain and management principles and some medications but unfortunately stop short of a concrete plan or guidelines.

Our paper is the forerunner of original research commencing in our department this year.

2. Non-emergency medicine studies on chronic pain management

Studies were chosen if they outlined strategies that could be of relevance to the ED. They can be divided into three categories.

1. Those examining treatment modalities
2. Early identification of chronic pain sufferers and those at risk
3. Rationalizing referral or investigation via screening.

These articles are listed by descending level of evidence.

2.1 Treatment modalities
Management of chronic pain centres on drug therapy, physiotherapy, rehabilitation and psychological interventions.

There are two Cochrane Reviews on chronic pain management; one on the efficacy of anticonvulsant drugs and one on the efficacy of psychological therapies for the management of chronic and recurrent pain in children and adolescents. The first concluded that carbamazepine could be recommended for trigeminal neuralgia but that there was not a strong evidence base for other treatments. Gabapentin warranted further study and might prove effective. Neither induced significant harm. The study in adolescents found that there was very good evidence that psychological treatments were effective in reducing severity and frequency of chronic headache in children and adolescents.

A structured abstract from the NHS centre for Reviews and Dissemination looked at Kingery’s critical review of controlled clinical trials for peripheral neuropathic pain (FNP) and complex regional pain syndromes (CRPS). For FNP they found consistent support for the use of tricyclic antidepressants, intravenous and topical lignocaine, intravenous ketamine, carbamazepine and topical aspirin. Intravenous morphine was likely to be effective, while oral NSAID were thought to be probably ineffective. Magnesium, propranolol, lorazepam, phenolamine and oral codeine were found to be ineffective. For CRPS, the only treatment consistently beneficial was oral corticosteroids. Guanethidine and reserpine regional blocks were found to be ineffective. For both PNP and CRPS, clonidine was probably effective.

Dworkin et al., in Archives of Neurology, reviewed randomized controlled trials on management of neuropathic pain and recommended five first line medications for neuropathic pain. Each one could be used as initial treatment or added on, providing that drug interactions
and patient susceptibility were considered. The recommended treatments were gabapentin, 5% lignocaine patch, opioid analgesics, tramadol and tricyclic antidepressants. An important statement made was ‘concerns about causing a substance abuse disorder when there is no history of one do not justify refraining from using opioid analgesics in patients with chronic neuropathic pain’.

Hocking and Cousins, in an evidence based review of ketamine in chronic pain management, conclude that there is limited level two evidence for the efficacy of ketamine in specific situations but in view of side-effects they can only recommend it as a ‘third line’ drug, ‘where standard analgesic options have failed’.16

Nielson and Weir systematically reviewed literature on the effectiveness of biopsychosocial and multimodal approaches to treatment of chronic musculoskeletal pain, primarily back pain.22 They concluded that there was level two evidence in support of the cognitive-behavioural and behavioural arms of biopsychosocial management for chronic low back pain and other musculoskeletal pain for up to 12 months.

In two separate original research papers, Hering et al.23 and Srikitthachorn et al.24 looked at serotonin levels in patients with chronic analgesic-induced headache. In single crossover studies, they showed that serotonin levels were decreased during long-term simple analgesic use (predominantly paracetamol), and that serotonin levels increased again after abrupt discontinuation of simple analgesics, with significant improvement in pain score, and headache-free days. They used NSAIDs while withholding paracetamol.

A single prospective double blind trial of 106 conservatively treated wrist fractures showed that post injury vitamin C (500 mg) was associated with less occurrence of Reflex Sympathetic Dystrophy.25

In one single-blinded prospective comparison of 200 chronic pain patients, randomized to receiving either ketorolac and chlorpromazine, or meperidine and promethazine, Mehl-Madrona26 found that the regimes were equally effective, with side-effects of respiratory depression with meperidine and dizziness with chlorpromazine. However it is important to note that the ketorolac/chlorpromazine doses were double that used commonly in Australia, while the meperidine was half that routinely used.

A randomized double blinded placebo-controlled cross-over study of 20 chronic pain patients used intranasal ketamine for episodes of breakthrough pain.24 There were no major side-effects. Researchers found significant relief of breakthrough pain and no use of regular rescue medication. It is notable that 65% of the placebo group also responded.

Two prospective outcome studies of multidisciplinary pain clinics (Robbins et al.25 — 201 patients and Hubbard et al.26 — 50 patients) found that specific, intensive, multimodal programmes improved outcomes for chronic pain patients compared to unstructured or incomplete care. Brief descriptions of programmes outlined on average 35 sessions with different therapists (Robbins) or a course averaging 8 weeks with four then two sessions of various therapies per week (Hubbard). There were no allusions to single visit or rescue modalities relevant to an ED.

An expert panel report by Stanton-Hicks et al.27 on an updated interdisciplinary clinical pathway for CRPS, continues to emphasize that psychological interventions and rehabilitation have as much importance as pharmacological treatments. They recommend tricyclic antidepressants, gabapentin, and early short course oral steroids. NSAIDs, opioids, calcitonin, alpha-1 adrenergic antagonists, and other antidepressants can be tried on an individual basis. Implantable therapies can be effective provided screening is careful. Trial of transdermal clonidine is suggested.

Of interest, they propose a central neuroimmune disorder as the cause of CRPS. There are no specific recommendations for ED management.

In another report on assessment of pain prior to implantable therapies, Prager outlines the types of implantable modalities, from peripheral nerve stimulators, central stimulators to spinal and epidural catheters for drug administration.28 His flowchart of therapeutic modalities begins with NSAIDs and simple analgesics, progresses through physical therapies with transcutaneous endoneural stimulation (TENS), alternative therapies, corrective surgery if warranted, then long-term systemic opioids, neuromodulation and neuroablation. There are no specific recommendations for ED management; however, questions from his screening process could be incorporated into ED assessment.

Nielson reported strategies to reduce chronic pain, breaking them into primary injury prevention (essentially a workplace health and safety function) secondary prevention (recognition of a subacute phase with high pain scores and the option of early intervention), and tertiary prevention – the damage control phase.29 He states that ‘both biological and psychological foundation for long-term persistent pain is in place within hours of injury’. This last statement attaches great importance to our ED management of acute injury. He recommends early and appropriate acute pain manage-
ment which must include adequate explanations to patients. A flat denial of significant disease is ineffective. There must be a positive explanation of symptoms.

2.2 Identifying at-risk and chronic pain patients

Pincus et al. systematically reviewed six of 25 papers investigating psychological factors that predicted chronicity/disability in prospective cohorts of low back pain. The authors found persistence of symptoms more likely among patients experiencing psychological distress (e.g. worry, fear) or depressed mood, and among patients who reported many somatic symptoms. To a lesser extent, they found that patients who coped with the injury by ‘catastrophising’ were at risk of chronic pain.

Hasenbring, in ‘Nervous System Plasticity and Chronic Pain’ summarizes the research on the various coping strategies used to address acute and chronic pain. Including several of her own studies that measure subjective pain ratings during and after noxious input, she reports that three common strategies were overexpressed in chronic pain patients. Catastrophizing the incident, suppressing the incident and self distraction were all associated with chronic pain. Suppression was a particularly common strategy as it increased immediate pain tolerance, but appeared to be followed by a rebound effect. She suggests that this is partly due to the subject persisting in the painful task and over-taxing the damaged region. Self distraction helped somewhat in acute pain but seemed to lose efficacy in patients with chronic pain. Sensory monitoring was associated with lower pain scores and less chronic pain.

An observational study by Kouyanou et al. of 125 patients attending a chronic pain clinic found that iatrogenic factors including over-investigation, inappropriate advice, misdiagnosis, over treatment and inappropriate prescription of medication were common among patients with chronic pain. They suggest that these factors may be causative, rather than effect.

In a small prospective observational study, Dunbar followed chronic pain patients to determine if any social factors and behaviours could predict the development of narcotic abuse. His findings reinforce the experience of most emergency physicians, documenting that multiple visits, multiple allergies, dose escalation, concurrent polysubstance abuse and poor social support are risks for narcotic addiction.

2.3 Screening and rational referral

In expert reports Mayou and Farmer, Nielson, Prager, Kroenke all strongly recommend the biopsychosocial approach that plots the patient’s risk factors on a grid against precipitating, predisposing and perpetuating factors. (Table 2) This also helps to define areas in which the physician might intervene, but seems somewhat lengthy for a screening ED assessment.

Mayou and Farmer reviewed functional somatic syndromes (including chronic pain) and symptoms. They plot the number of somatic symptoms reported by the patient against the likelihood of psychiatric morbidity. In a reassuring graph, they illustrate that five or more somatic symptoms have a greater than 50% association with psychiatric condition. Fifteen or more somatic complaints had a 100% chance of psychiatric comorbidity. These patients are less likely to require urgent after hours investigation, and may benefit most from psychiatric referral.

Kroenke looks at the prevalence of somatic symptoms (including chronic pain) which remain medically unexplained, quoting studies with figures similar to Mayou. He suggests a simple scheme for predicting an anxiety disorder using risk factors of;
1. Stress in the preceding week
2. Symptom count greater than five
3. Self rated health as low
4. Symptom reported as severe.

Non-organic aetiology is even more likely when the patient is a frequent health care user, and also if the physician rates the encounter as ‘difficult’.

Psychiatric referral is recommended by Kroenke but must be prefaced by careful explanation. The authors do describe the role of non-specialist treatment, but it is directed at a family physician. Their advice is generalized and difficult to conceive within an ED. They emphasize the importance of correct information, sim-

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<th>K.B.</th>
<th>Predisposing factors</th>
<th>Precipitating factors</th>
<th>Perpetuating factors</th>
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<tbody>
<tr>
<td>Biological</td>
<td>Low IQ</td>
<td>Minor fall</td>
<td>Increasing obesity</td>
</tr>
<tr>
<td>Psychological</td>
<td>Family history of depression</td>
<td>Stressed about pending court case</td>
<td>Poor coping skills</td>
</tr>
<tr>
<td>Social</td>
<td>Abusive parent</td>
<td>Lost job</td>
<td>Increasing debt after losing job</td>
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ple behavioural plans, anxiety management, diaries, graded exercise and antidepressants even in the absence of depression.

Proposed guidelines for emergency department management of chronic pain

In the absence of consensus statements or management guidelines in the literature, I open for debate the following guidelines for management of chronic pain syndromes in the ED.

Principles

1. Definitive treatment of chronic pain is not the role of the ED.
2. ED management includes rescue medication, rational investigation and referral, given in an environment which does not exacerbate chronic pain.
3. Pain should be treated aggressively, promptly and appropriately. Narcotics should not be withheld without definite evidence of addiction.16,17
4. A focused search for precipitants (organic and psychologic) should be based on the individual’s risk profile.2 A thorough physical examination is itself reassuring.10,28
5. Emphasize that severe pain does not reliably predict tissue damage, but address concerns and avoid simplistic reassurance or denial of disease.10,32,34
6. Emergency physicians must continue to gather best available evidence based management protocols from other specialty areas.
7. This field needs more original clinical research.

Recognition of chronic pain

8. Expect exaggerated avoidance, emotion, allodynia and non-dermatomal spread of pain.30,36 These are NOT signs of malingering.
9. Autonomic manifestations are common and do not guarantee new organic pathology nor drug withdrawal.30,37
10. Multiple somatic complaints are common.4,34
11. Those at risk of developing chronic pain are those who are frightened or worried at the outset.10,31,35 They tend to rate the initial pain as severe38 they catastrophize, suppress or try to distract themselves,31 and may have a pre-existing depression or other painful condition.39
12. Behaviour AFTER adequate pain relief best differentiates addiction from pseudoaddiction.2,30

Drug therapies

13. Rescue therapy should be multimodal, using narcotics, non-steroidal anti-inflammatory drugs, ketamine (all routes but watch dose), tramadol, paracetamol and local anaesthetics.16–18,24,29
14. If a longer term change is unavoidable, manipulate the five first line medications recommended for neuropathic pain. They are additive, side-effects permitting. They are opioids, tramadol, gabapentin, tricyclic antidepressants and 5% lignocaine patches (not available yet in Australia).17
15. Carbamazepine is recommended in trigeminal neuralgia.14
16. Short course oral steroids can be used for chronic regional pain syndromes, if considered safe.27
17. Vitamin C will do no harm and may assist acute injury, specifically Colles fracture.25

Non-drug therapies

18. Advise against prolonged immobilization10,32 — early return to altered work duty has the better outcome.10 Exercise should be graded.29
19. Where available, ensure referral of patient to multimodal pain clinic.25,36 Failing this, utilize individual psychology, psychiatric and physiotherapy services. Urgent social work referral may provide education on correct coping strategies.
20. Minimize stress for the patient, circulating catecholamines can stimulate the altered neuroplastic adrenoreceptor expression.40
21. Tabulation of the patient’s biopsychosocial-precipitant matrix is time-consuming but can reassure the patient and reveal further therapeutic opportunities.10,30,34
22. A care path approach could speed management, with the patient filling out a questionnaire. The plan should include an information booklet on coping strategies and a follow up letter to the general practitioner detailing tests and treatment in the ED. Individualized care plans should be requested from primary care giver or pain clinic.

Conclusions

With limited time and resources, recognition of the chronic pain syndrome patient in the ED remains very
difficult. Many patients are afraid to admit their diagnosis. The magic ‘Pain-o-meter’ does not yet exist, and patients know that acute pain receives a far more sympathetic reception.

Balanced, detailed recommendations are available, yet lacking specificity for the demands, opportunities and limitations of a busy emergency department.

Emergency physicians need to differentiate chronic pain breakthroughs from acute intermittent pain and drug addiction. Ideally, emergency physicians should recognize those at risk of chronic pain, forestall the syndrome’s beginnings, and rationally exclude icteric illness. Furthermore, the approach should link evidence based medicine with the time limitations in emergency practice.

A better understanding of chronic pain, and knowledge of current management practices may reduce clinician angst, lead to more consistent, less prejudicial interventions for patients presenting with chronic pain syndromes to emergency departments. In our modern society with high prevalence of chronic pain, further work needs to be undertaken in developing consensus statements and management guidelines.

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Competing interests

None declared.

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References


