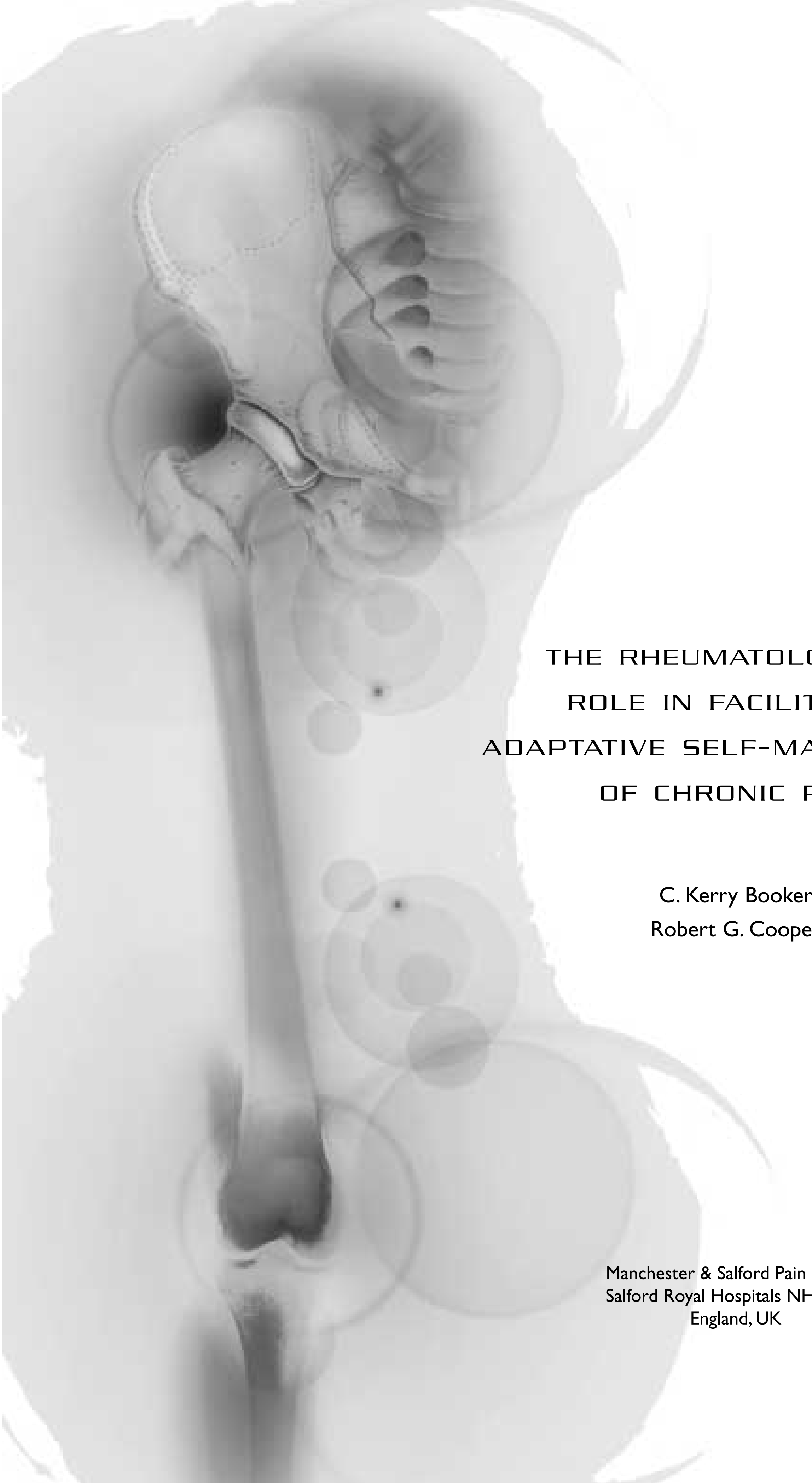

THE RHEUMATOLOGIST'S ROLE IN FACILITATING ADAPTATIVE SELF-MANAGEMENT OF CHRONIC PAIN

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Introduction

The majority of patients newly referred to a rheumatologist have musculoskeletal pain as their main complaint. Since it is impossible to prioritise all such patients as urgent problems, a routine appointment is usually issued. In view of the waiting time to routinely see a consultant rheumatologist in the UK, the majority of patients will have developed chronic pain (i.e. that lasting longer than 3 months) by the time they attend their hospital appointment. A considerable proportion of such individuals will thus have also developed pain-related disability when first seen. Such a combination of pain and pain-related dysfunction comes at a great cost to the affected individual, their family and indeed the Nation, because of health and welfare costs and work loss. Whilst a proportion of patients with chronic pain are found to have a potentially curable or partially treatable disease (e.g. hypothyroidism, connective tissue disease, statin-induced myositis etc.) others are are found to have conditions that are easily diagnosed but that are not amenable to cure (e.g. degenerative disc disease, fibromyalgia etc.) It is this last group of patients that take up a great deal of a rheumatologist's time and that are sometimes labelled as «heart-sink» patients. For the primary care physician the situation can be even worse and such patients are often referred to a specialist in order to alleviate the pressure of the patient's demands. Given the substantial human and economic costs of the foregoing situation, it is clear that a more effective way of dealing with these unfortunate individuals is of great importance.

Drawing upon our experience in an inter-disciplinary pain management centre (see Main & Spanswick, 2000), we will here elaborate our argument for a change in the way that primary care physicians and rheumatologists manage chronic pain patients to effect better outcomes. Although we will use chronic low back pain as a model, the argument applies to all patients with chronic pain.

The rationale for a change in practice

Providing excellence in the diagnosis and treatment of exotic inflammatory and connective tissue diseases is perhaps the major motivating factor behind an individuals' decision to specialise in clinical rheumatology. Referral practices dictate however, that a large proportion of referred musculoskeletal patients do not have a clearly identifiable inflammatory disease. In our own teaching hospital, 36% of new patients attending rheumatology clinics have chronic back pain and about 18% have fibromyalgia. Given the limited success of traditional biomedical treatment of these conditions, it is reasonable to conclude that we apparently have little to offer about half of all patients attending specialist rheumatology centres.

Patients with rheumatological disease are also at risk of poor self-management due to psychological distress and/or inadequate attention to treatment recommendations. Evidence provided by research into treatment compliance in patients with rheumatoid arthritis, demonstrates that adherence to the prescribed medication regimen is generally low (Brus et al., 1997), but worse in busy middle-aged adults than in the elderly (Parks et al., 1999). Newly diagnosed RA patients, who demonstrated frequent use of passive pain coping strategies of worry and activity avoidance and a restricted social network, were likely to demonstrate a significant decline in mobility and self-

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-care after one year which could not be accounted for by disease activity (Evers et al., 1998). Functional disability in the first year of the disease predicts work loss at two-year follow-up (Eberhardt et al., 1993), poor prognosis at ten years (Sherrer et al., 1986) and future mortality at 15- and 20--year follow-up (Corbett et al., 1993).

These facts emphasise that technical expertise in itself is insufficient in changing the patient's healthcare behaviour. Patient's behaviour that is reinforced by short term rewards (e.g. avoiding pain by avoiding activity), but longer term costs (e.g. joint stiffness, cardio-vascular and musculo--skeletal deconditioning), needs to be supplanted by practices that may have short term costs (i.e. increased discomfort), but longer term advantages. We venture to suggest that an important component of clinical excellence is the ability to motivate change toward adaptive healthcare behaviour. This requires the rheumatologist to subvert practices that carry a significant risk of producing iatrogenic distress and to systematically identify and address barriers to therapeutic compliance. However this task is by no means simple. It requires a firm understanding of the biological, psychological and social processes which conspire to transmute the nature of the biomedical mechanisms over time, as well as the nature of illness presentation within the (social) context of the consulting room.

The size of the problem

The last two decades have witnessed a remarkable increase in the number of people seeking treatment for a variety of painful syndromes (Waddell, 1998). For instance, in England from 1985 to 1993, the annual number of general practitioner (GP) consultations for low back pain (LBP), doubled to four million (9.2% of all adults in Britain attended their GP on average 1.6 times), representing 4% of all GP visits. During this time hospital referrals for LBP rose from 0.3 million to 1.6 million, and the number having surgery increased from 11,000 to 24,000. Accordingly, the number of working days lost annually through LBP-related sickness and disability, increased from 25 to around 100 million between 1979 and 1995. Musculoskeletal diseases accounted for 30% of sickness and invalidity days in the year 1994--1995, all other musculoskeletal incapacities totalled 15.6%, and back incapacities, accounted for 14.4% of sick certification in that year. One of the most alarming aspects of this health-care trend in back pain is that while the prospects of returning to work following a one month lay-off are around 80%, this figure falls to less than 50% at six months of pain persistence, and worsens thereafter to below 5% at 2 years. A similar picture of symptom persistence and pain-related disability is seen in many other chronic pain syndromes, (e.g. fibromyalgia).

Structural pathology in chronic low back pain

The search for simple mechanisms to explain persistent pain in the lower back has been frus-trated by recent demonstrations that people can have significant evidence of spinal pathology without ever experiencing pain. Boos et. al. (1995) matched patients on age, gender and occupational risk factors. Disc herniations were found in 96% of patients and 76% of controls (severe herniations 35% vs. 13%, respectively), but the proportion of patients with neural compromise was significantly higher than controls (83% vs. 22%; of which 54% and 4% respectively, had major nerve root compression). The relevance of the size of herniation relative to canal width, the potential role of neurochemical irritation, neural innervation of herniated discs or the relevance of intra-neural and extra-neural fibrosis following surgery has yet to be clearly established. Unfortunately, it is unlikely that such pathology can adequately explain the various types of pain pathology. Indeed, it is estimated that up to 85% of low back pain patients are left without a definitive diagnosis.

Factors associated with favourable outcome of lumbar surgery

A recent prospective longitudinal study of the outcome of lumbar surgery has evaluated the contribution that clinical, morphological, psychosocial and work-related factors play in successful intervention (Schade et al., 1999). MRI--identified alterations (nerve root compromise, disc extrusions) were significant predictors of pain relief and improvements in disability. In contrast, patients with minor disc protrusions without neural compromise had more pain six months after surgery, than those with major disc herniations and neural compromise. Importantly, failure to return to work was associated not with clinical or morphological findings but was solely attributed to the experience of depression and occupational work stress. Greenough (1999) has reviewed the indications for surgery in LBP together with outcomes for specific clinical and morphological presentations.

Factors associated with the transition from acute to chronic pain

Most patients with a «first time» episode of LBP will have suffered minor muscular or ligamentous injuries that are self-limiting and improve rapidly. These are dealt with readily and appropriately in the primary care setting. Nachemson (1982) estimated that 90% of cases remit naturally within an average of four weeks, or 12 weeks at the latest. The process of remission, however, has been challenged and alternatively described as ongoing pain with recurrent acute exacerbations (Croft et al., 1997). Moreover, the characterisation of those individuals who go on to become chronic sufferers has received closer analysis.

Having followed 117 patients from initial onset through to six months post-injury, Philips and Grant (1991b) found that 40% of their sample continued to complain of pain at six months. These patients were characterised as rating their acute pain as more intense, of a more aversive sensory and affective quality, to be more avoidant of activity, and having higher scores on depression, anxiety and sickness impact than patients whose pain resolved. The best prediction of chronic status was made at 3 months when 80% of cases were correctly classified. This finding raises the possibility that chronic pain sufferers have failed to resolve or attenuate patterns of behaviour and subjective experiences that are present at the acute stage.

The views expressed in a recent series of articles support the need to evaluate acute pain intensity but propose that factors known to be important in chronic pain management should also form part of the equation (Dworkin, 1997a; Atkinson et al., 1997; Katz, 1997; Linton, 1997; and, Dworkin, 1997b). In particular, fear and avoidance of pain, maladaptive coping styles and strategies, somatic anxiety and depression, the impact of acute pain on disability in terms of health care utilisation, and the physical, social and occupational consequences should be evaluated.[1] Whilst this leaves open the question of the relative causal contribution of patho-anatomical and psychological factors in the transition to chronicity, it provides support for careful assessment and intervention at the earliest opportunity.

Indeed the New Zealand government now require the primary care physician to screen for individuals at high risk of disability on «psychosocial yellow flags» at the initial consultation, with reviews at seven days, four and six weeks following back injury (Kendall, 1997). In the UK, the Clinical Standards and Advisory Group (CSAG, 1994) and The Royal College of General Practitioners (1996), have made similar recommendations. Much of the advice on appropriate early self-management of LBP is contained in a patient booklet called "The Back Book" (Roland et. al., 1996). Details of all of these guidelines and issues relating to their implementation receive excellent coverage in Waddell (1998).

Because clinical management and communication with the patient requires a sound understanding of the biomedical mechanisms of pain, we will outline current knowledge, beginning with normal pain transmission.

Mechanisms of pain transmission

Melzack and Wall's (1965) description of their «gate control model» of pain ended the formal adherence to notions of a one-way transmission of nociceptive impulses from the periphery or organ to the brain. In doing so, it challenged a view of pain as the direct and invariant relationship between stimulus and response that had changed little since espoused by Descartes some three centuries earlier.

The model described how the transmission of nociceptive impulses following their arrival in the dorsal horn of the spinal cord was subject to

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[1] Physical assessment of the pain patient is fraught with conceptual and technical difficulties. Pain report is, after all, a communication of the perceptual experience of nociception resulting from the interaction of neurophysiological, biochemical, biomechanical, cognitive and behavioural factors. These, in turn, come under the influence of prior experience of/or beliefs about pain and illness, coping style and strategies, peer dynamics and socio-economic factors. Thus, the evaluation of *impairment* (i.e. abnormality or loss of pathological, anatomical, or physiological structure), *functional limitation* (i.e. the manifestation of impairment which results in an inability or restriction of ability to perform those functions and activities which are considered normal for that person's age and gender), or *disability* (i.e. the inability of the individual to perform a specific role that is normal for him/her as a result of impairment and functional limitation) is inevitably a biopsychosocial process.

modification as a result of two factors: 1) the convergent effects of other peripheral afferents, which may exacerbate or diminish the effects of the nociceptive message, and 2) the presence of central control processes which interact with sensory-discriminative, motivational-affective and cognitive-evaluative systems allowing for the inhibition or excitation of ascending impulses (closing or opening of pain gate, respectively). This spinal gating mechanism that resides in the substantia gelatinosa of the dorsal horn, therefore, is influenced by peripheral afferent activity, as well as efferent neural impulses from the brain. The major contribution of the gate control theory of pain is its exposition of the role of the central nervous system in marshalling multiple neurophysiological and psychological systems essential to understanding pain perception and puzzling clinical problems. The details underpinning the theory provided a framework within which to understand findings such as the influence of summation and patterning of impulses on pain transmission. The postulated mechanisms of pain processing, following from the gate control theory, have remained remarkably robust and have required few modifications in light of advances in molecular biology, pharmacology, anatomy, electrophysiology and neuroimaging research (Besson , 1999; Loeser & Melzack, 1999).

Pathophysiology of pain

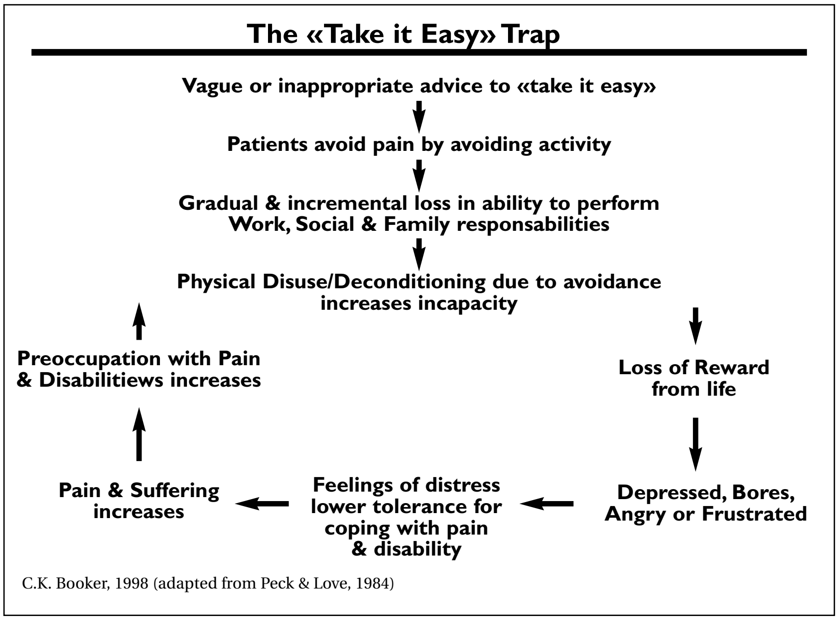
Unfortunately it is not uncommon for pathological pain processes to persist as a result of chronic inflammatory conditions, peripheral neural and soft tissue damage (sometimes apparently trivial in nature), and damage due to central trauma. The painful state may be characterised by a prolonged experience of pain following brief stimulation, in the lowering of pain threshold (allodynia), the magnified response to noxious stimulation (hyperalgesia), and the spread of pain and hyperalgesia in non-dermatomal distributions, and in uninjured tissue (referred pain and secondary hyperalgesia). However, persistent pain following normal healing and the cessation of the normal inflammatory response cannot be fully explained by a description of local and peripheral biochemical activation. Hopkin's (1997) concise review of research on pain mechanisms noted that chronic or inflammatory pain can sensitise the nervous system leading to stimulation of chemical, functional and structural changes that «prime the pain processing pump». It does this by lowering thresholds of pain-sensing neurons and a concomitant release of growth factors and neurotransmitters that act to reinforce the pain message. Consequently, increasing effort has been made to understand the potential contribution for central neural plasticity in the maintenance of the pain response (see Coderre et al., 1993 for an excellent early review). Such research is throwing light on seemingly anomalous clinical findings. Examples include, referred pain and secondary hyperalgesia to the site of previous injuries (leading to hypothesised neural 'pain memory'), phantom pain and the spread of CNS receptive fields following limb amputation. The benefits of pre-emptive opioid use prior to surgery, which blocks the surgery-induced afferent barrage and/or its central consequences, can also be explained by Gate Control theory. Readers interested in the exciting developments involving the Hypothalamic – Pituitary - Adrenal Axis (HPA), neuroendocrine and immune interactions in pain and rheumatic conditions should consult the following texts (Chikanza, 1996; Melzack, 1999; Bijlsma et.al., 1999; Watkins & Maier, 2000). Neurosciences research, therefore, has and is underlining the complexity and plasticity of the peripheral and central nervous and consigns notions that structural anatomy is the only legitimate cause of pain sensation to the medical history books. Unfortunately, the layperson and some medical colleagues still believe in Cartesian dualism. That is the belief that pain is caused either by organic or psychological means; ergo if structural anatomy is normal, then the cause must be psychogenic. Whilst the layperson cannot be blamed for their medical ignorance, informed practitioners who act on this belief should reflect upon their own emotional response to difficult patients so as to guard against the understandable, but unhelpful, tendency to assign the «psychogenic label». Secondary psychological reactions to intractable pain are to be expected, as the patient may be unable to maintain adaptive coping strategies when faced with such an ongoing and aversive stressor.

Characteristics of acute versus chronic pain presentations

Presentation at the acute stage is characterised by a marked reduction in activity, verbal and facial expressions of pain, frustration and medication-

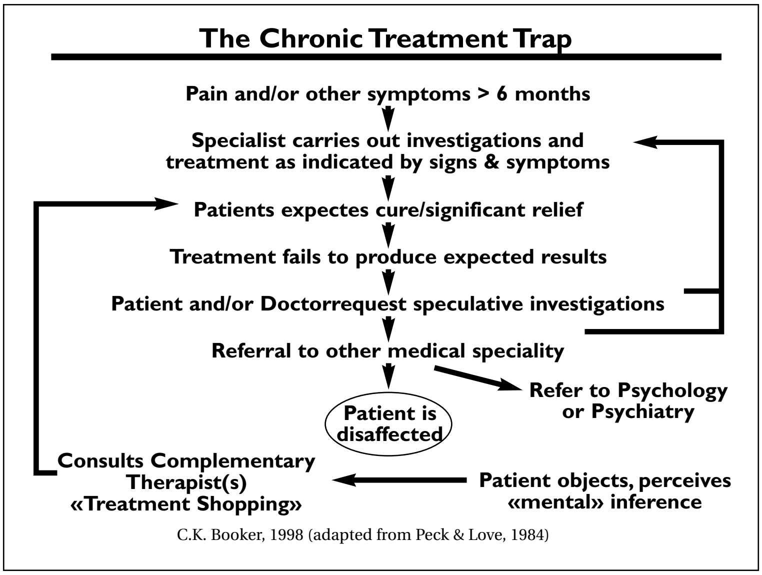
-taking which are considered to be normal and acceptable ramifications of acute suffering (Philips and Grant, 1991a). The behaviour of the chronic intractable benign pain (CIBP) patient presenting to tertiary care centres may or may not differ in its magnitude, but the degree of familial disruption is more evident.

The primary feature that distinguishes the acute sufferer from the CIBP patient, is that of an ongoing pain problem that cannot at the time of presentation be causally connected with any active patho-physiological or patho-anatomical process. Guided by the prescription of «common-sense» advice to 'take it easy' or to 'let pain be your guide' affected patients fail to mobilise in the days following acute injury. Burdened by the fear that an increase in pain signifies bodily harm and tissue damage, the individual becomes increasingly inactive and avoidant of all but sedentary activities. Physical deconditioning characterised by a clinically significant reduction in cardiovascular fitness, muscle strength and endurance, and in extreme cases, joint stiffness (esp. lumbar facet joints), a loss of muscle bulk (e.g. atrophy of abdominal flexor and trunk extensor) and bone demineralisation ensues. This takes place in concert with a progressive increase in psychosocial dysfunction (Figure 1). A history of repeat specialist consultations and generally ineffective medical or surgical interventions unfolds as the pain generalises from the original site. The patient's dissatisfaction with healthcare professionals and the healthcare system can culminate in conflict and hostility with each «failed» treatment or communication [see Figure 2]. Further passive attempts to control pain by escalating the amount and potency of analgesia, is not only ineffectual but also counter-productive as the pain relief habituates leaving CNS disturbance, lability of mood, and feelings of worthlessness and hopelessness [Figure 3]. Eventually, as illness behaviour fails to elicit a benevolent response, the scene is set for interpersonal conflict with family and friends [Figure 4]. Subsequent loss of social and occupational responsibi-



C.K. Booker, 1998 (adapted from Peck & Love, 1984)

Figura 1



C.K. Booker, 1998 (adapted from Peck & Love, 1984)

Figura 2

lities and status combined with psychosocial withdrawal heralds profound demoralisation, resentment and sometimes, clinical depression. Patients who have descended to this level of dysfunction are effectively «prisoners of pain».

A role for rheumatology in the treatment of chronic low back pain

During the initial assessments, the time honoured processes of history taking, examination and initiating appropriate investigations will be undertaken by the rheumatologist to confirm a diagnosis in the belief that a therapy specifically targeted at the cause of the symptoms will produce the best outcome. This does hold true for certain types of LBP. For example, where spinal inflammation is the cause of stiffness and pain in ankylosing spondylitis, non-steroidal agents and physiotherapy are clearly effective; and in malignant vertebral body pain, radiotherapy may be very effective in the short term. Unfortunately, this systematic diagnosis-based approach to directing therapy in chronic LBP is unsatisfactory for a number of reasons (Grönblad & Cooper, 1999). Appropriate investigations, including multiple and complex radiology, may be normal or demonstrate multiple lesions potentially capable of causing symptoms but without pinpointing the actual culprit. Good examples of the latter include degenerative disc disease, which is frequently present radiologically at multiple segmental levels, and spinal instability, which could theoretically cause pain through stretching of local neurological and/or soft tissues, but which often occurs in association with degenerative disc and facet joint changes at contiguous or multiple spinal levels. Under these circumstances it is not possible to target therapy or to have confidence in the outcome of proposed therapies.

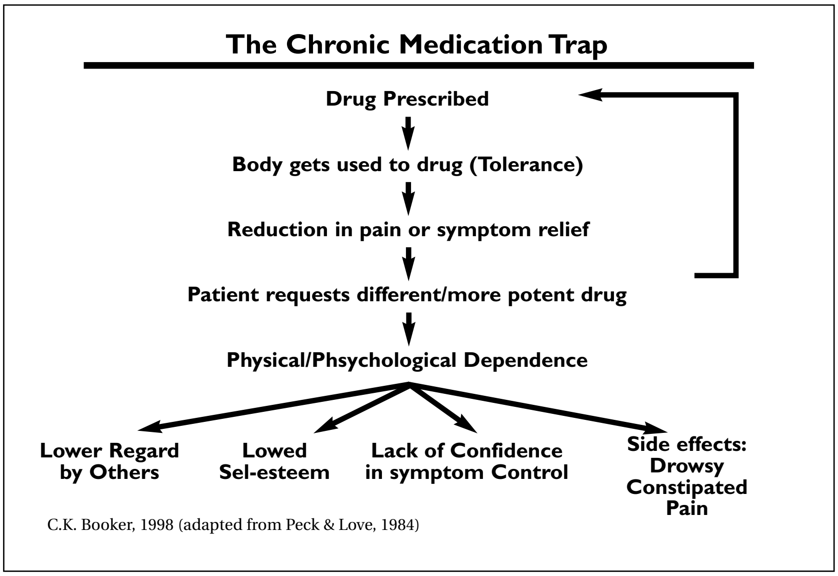


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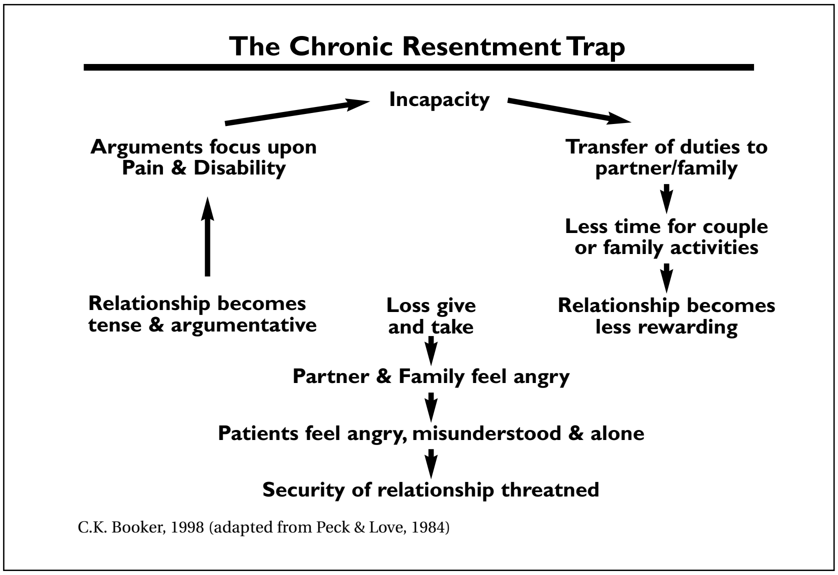
Figura 3



C.K. Booker, 1998 (adapted from Peck & Love, 1984)

Figura 4

Worse still, where the cause of symptoms is clearly elucidated, such as in classic clinically and radiologically matched disc herniation, and where there would be a reasonable consensus amongst rheumatologists and spinal surgeons regarding the optimal treatment, the clinical outcome may still be uncertain. Thus, when severe compressive neurological complications do not force early decompressive surgery, conservative therapy appears the correct option. This allows nature sufficient time to normalise local tissue disruption and hence to settle the symptoms. Various studies have shown that outcome at 12 – 18 months is as good (in about 80% of cases) whether or not surgery is undertaken (Waddell, 1998). When signs and symptoms show no evidence of improving despite a reasonable trial of conservative therapy (which most would agree should last 3 –6 months) and surgery is therefore undertaken, about 10% of operated patients will subsequently redevelop their low back pain – and especially their radicular symptoms. In patients suffering such low back and radicular pain, these and the associated disability, may be particularly severe. The trend for these patients is for repeat attendances, and a rotation to the other major musculo-skeletal specialities of orthopaedics, neurosurgery and pain management.

Avoiding iatrogenic distress

Having had their hopes of a cure dashed on several occasions, and holding the belief that they have been given contradictory diagnoses, scepticism becomes a common characteristic among such chronic pain sufferers (Kouyanou et. al., 1998). Frequently more than one appointment is thus required to persuade patients of their practitioner's sincerity and accuracy of diagnosis. Moreover, whilst identifying those at high risk of chronicity or further functional decline is important, it is quite another matter to persuade such patients that further specialist referral and/or investigations are unnecessary, and that a return to activities whilst still in pain is the optimal way forward. Even the most diligent primary or hospital-based clinician can wilt under sustained pressure, from such classic 'heart-sink' patients, and initiate further unnecessary tests or treatments. Three general strategies can aid the clinician in warding off inappropriate requests and guiding the patient forward:

1) be aware of the antecedents and consequences of one's own actions;

2) have a game plan based on the above, rather than acting reactively;

3) encourage the patient to acknowledge what they may already suspect to be the consequences of 'treatment shopping'. Table 1 summarises this approach. The issues listed in the 'solutions' section of Table 1 have become a core component of most pain consultants' roles, but this often takes several lengthy sessions to accomplish. Rehabilitation without re-conceptualisation is doomed to failure.

Barriers to progress

There are various issues over which the clinician can exert significant beneficial influence. As noted earlier, psychosocial factors are better than any biomedical measure at predicting general clinical progress, pain self-report, degree of disability and return to work, in cohorts of LBP sufferers. The relationship between chronic pain and disability, therefore, is mediated by a complex interaction of many factors, all of which constitute barriers to clinical improvement. Significant disability is not inevitable; but the greater the number of barriers present, the greater the potential for maladaptive, secondary psychological responses to ongoing pain. Table 2 summarises many of the key barriers acting in everyday general and hospital-based clinical practice. These barriers need to be acknowledged by patients and addressed systematically. Research has consistently confirmed the potency of misplaced beliefs, attitudes and feared expectations as barriers to change, and as a motivational factor in behavioural avoidance (Jensen et al., 1999; Arnstein et al., 1999). The fact that the patient's family often share these misattributions, and may act as advocates for caution, has also been shown to inhibit adaptive behaviour (Romano et al., 1991). Education and attitude change must therefore address the concerns of influential family members and attempts be made to recruit them as allies for treatment compliance. Ultimately, the promotion of a change toward adaptive healthcare behaviour is the shared responsibility of the patient, their family and their practitioner.

Facilitating information exchange

Eliciting and untangling such issues is time-consuming. During the initial consultation the clinician must avoid being overly didactic or prescriptive. One of the main complaints patients make is that their views were not sought, and this tends to be borne out by research (Ley, 1982). As a rule of thumb some simple guidelines for good doctor-patient communication provide a basis for elici-

Table 1. ABC'S of iatrogenic distress in benign pain conditions

Antecedents:

- Failure to acknowledge previous failed treatment / investigations.
- Adherence to the acute rather than chronic model of pain.
- Susceptibility to patient pressure/desire to help when action not indicated.
- Unsure as to how to manage distressed or dependent patient.

Behaviour:

- Allow short-term strategy (due to time constraints) to become the norm.
- Knowingly advocate investigations/interventions of questionable value.
- Prescribe alternative (similar) medication when not clinically indicated.
- Perform invasive procedure to portray 'active management'.
- Admit to ward on basis of vague management plan.
- Avoid addressing cure vs. self-management issues.
- Make 'unparsimonious' referral to other specialist.

Consequences:

- Reinforces 'practitioner active/patient passive' model.
- Reinforces initial dependence and hero-worship.
- Attracts solicitous/unscheduled appointments or requests.
- Patient begins to think that one is making 'stabs in the dark'.
- Referral to other speciality interpreted as 'buck-passing'.
- Increasing number of interventions increases risk of complications.
- Appropriate rehabilitation is delayed.
- Patient and clinician can feel disaffected, cynical, angry or hopeless.
- End up in situation one was hoping to avoid in first instance.

Solutions:

- Screen for high risk on barriers to progress (especially psychosocial yellow flags).
- Arrange structured medical counselling sessions (treatment conditional).
- Elicit beliefs about cause/effect of physical and psychosocial issues.
- Address fears, expectations, beliefs re: diagnosis/prognosis.
- Avoid using misleading medical jargon.
- Explain significance of findings vis-à-vis structural safety/other illness.
- Explain (imperfect) association of signs & symptoms (e.g. referred pain).
- Be honest about limits of scientific knowledge re cause of chronic pain.
- Explain rational-deductive basis of treatment decision-making.
- Do not use 'scare stories' to dissuade from treatment.
- Encourage informed collaborative decision-making.
- Encourage early focus on functional goals not symptom relief goal.
- Guide towards a self-management approach.
- Facilitate a biopsychosocial re-conceptualisation.
- Agree plans for new episodes or flare-ups (draw up 'emergency card').
- Don't let your actions be driven by sympathy or avoidance.
- Refer to inter-disciplinary pain centre if patient continues to plead 'out of desperation' for non-efficacious treatment.

ting the patient's misattributions, and lay the foundations for treatment compliance (see Figure 5). The practitioner should enquire (listen) into the patient's explanation of the physical and psychosocial effects of illness (symptoms) together with the perceived cause, taking care to establish what the patient has done (actions) to alleviate the symptomatology. Next, the practitioner should explain the presumed biomedical cause of the condition together with the potential (short and long term) effects of the condition across physical and psychosocial domains. Then, explain his/her course of intervention (action) and establish the patient's understanding of the relevant issues. Finally, the clinician should check that the patient understands and agrees with the approach recommended and establish the patient's intention to comply. The promotion of a change in adaptive healthcare behaviour is, therefore, the shared responsibility of both patient and practitioner.

The process of change

The assumption that the provision of information is sufficient to effect treatment compliance ignores what has been learned about attempts to change maladaptive behaviour. Prochaska and Di Clemente's (1992) Stages of Change model posits that the transition from engaging in maladaptive behaviour (e.g. drug misuse or physical inactivity) to adaptive behaviour, proceeds through a number of stages, in a cyclical fashion (pre-contemplation → contemplation → preparation → action → maintenance → relapse → pre-contemplation etc.).

Table 2. Barriers to Clinical Progress in Chronic Benign Pain

Medical
- Co-morbidity - serious and/or progressive disease
  - syndrome (e.g. irritable bowel syndrome) or illness with systemic symptomatology
- Substance abuse (prescribed or illicit)
- Intervention for treatable pathology denied because patient is 'distressed'
- Investigations: incomplete; awaiting specialist consultation/treatment
- Patient is still treatment-shopping (or being urged to by partner)

Mood
- Anger, anxiety, fear of pain or (re)injury, depression, estrangement

Misattribution/Misdirection
- Belief that pain is synonymous with serious disease or ongoing damage
- Belief that improvement in functional capacity is impossible without pain relief
- Belief that structural/pathological decline is inevitable
- Belief that rest is the best remedy
- Belief that if pain is not associated with observable pathology it is 'imaginary'
- Belief that failure to find a cure for pain is a result of misdiagnosis
- Interpret medical terms (e.g. degenerative disc disease) catastrophically
- Being told that an operation is needed, but is too dangerous

Motivation
- Lack of insight/foresight into consequences of behaviour
- Inability to meet work demands (lack of support/job stress or dissatisfaction)
- Attempt to avoid increase in pain/damage by avoiding activity
- Avoidance of undesired role responsibility
- Attempt to control or appease significant other
- Frank socio-economic presentation (ongoing medico-legal claim is not proof)
- Significant investment of effort/money in obtaining aids and appliances
- Overwhelming passivity

Magnification
- Heightened somatic awareness (due to selective attention)
- Associated health anxiety
- Extreme emotional/behavioural response in presence of subtle clinical signs
- Excessive guarding response to examination due to fear of being hurt
- Attempt to 'show' how bad the pain can be on 'bad day'
- Barrier complex (a combination of components from each section)

Malingering (mutually inclusive features)
- i) Gross inconsistency between symptom report and clinical signs
- ii) Absence of distress against background of significant disability/pain behaviour
- iii) Explains ii) as being due to stoical forbearance but shows no autonomic arousal to physical challenge
- iv) Poor employment record; and,
- v) Indication of significant economic gain as a result of disabled status.

Thus, the individual who does not believe their behaviour to be problematic, or who acknowledges the possible contraindications but does not wish to change, is considered to occupy the *pre-contemplative* stage. Faced with such a patient, the clinician's task is to increase the patient's awareness of the risks associated with their behaviour. Progress to the *contemplation* stage entails a cost/benefit analysis, based upon accurate information and expectations and with active support given to strengthening their confidence in being able to exercise change. The latter means that not only must the patient believe they have the requisite skill and physical ability to comply with recommended goals (a sense of self-efficacy), but also that they have an accurate understanding of the consequences of these actions (accurate outcome expectations). For intellectual acceptance to lead to action, the *preparatory* stage must enable the setting of relevant, attainable and meaningful goals and an ongoing commitment to overcome barriers to progress. The *action* stage (lasting about 6 months) allows for the implementation of goals, problem-solving analysis following goal failure, and further planning. Adherence to this change in lifestyle for up to one year is termed the *maintenance* stage, and is facilitated by the practitioner's commitment to actively reviewing goal attainment and reinforcing the efforts taken. The next stage is *relapse,* followed by feelings of helplessness or despair and resistance to re-institute adaptive behaviour (pre-contemplation stage).

Research in heroin, smoking and eating disorders shows that the individual who attempts to

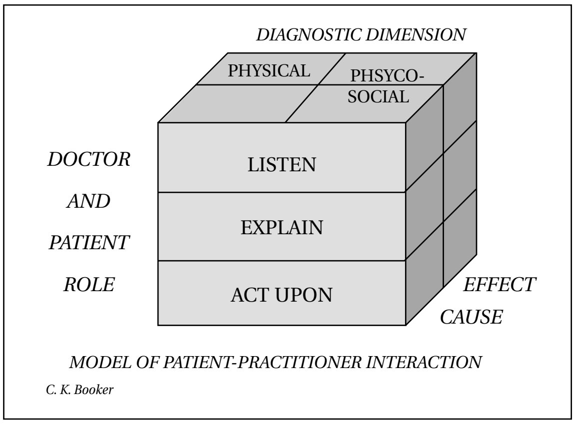


Figure 5

change, cycles through these stages between four and seven times before attaining permanent change toward adaptive behaviour. The practitioner's role here is to (empathetically and without attributing blame), encourage a review of reasons for relapse, a review of factors underpinning a desire for change, and to regroup allies who have previously acted as facilitators of change. Change is therefore a *process* and not an *event*, and failure of the pain patient to adhere to initial prescriptions for painful activity, to reduce inappropriate medication practices, and to return to social interaction will inevitably follow a similar pattern. The patient who persists in the practice of potentially harmful behaviours is probably «stuck» in a helpless state, borne out of frustration with it's multiple health-related daily hassles, discomforts and disadvantages. Shackled by the view that they can effect little or no control over their situation, and by an over-reliance of short term coping strategies instead of those based on a realisation of long term consequences, these beliefs and expectations become self-fulfilling prophesies. Part of the clinician's role, therefore, is to help such patients to become 'unstuck'!

Most of the recommendations outlined to this article require a re-organisation of existing resources and an appreciation of the process by which change is either frustrated or promoted. Acknowledgement of the benefits of this approach counsel a change (at the earliest opportunity) in clinical practice from one of a passive periodical review, to one built upon the tenets of sharing responsibility in order to encourage active self-management and attainment of mutually agreed goals. In the medium term, the number of inappropriate repeat appointments is reduced and the initial costs of spending more time in aiding reconceptualisation, are balanced by more appropriate health-care utilisation. The feelings of helplessness and frustration experienced by the clinician are also minimised. Guidelines for providing information to the patient following the medical examination are summarised in Table 3.

The management of complex cases by inter-disciplinary pain centres.

For patients who are highly distressed and disabled a referral to an inter-disciplinary pain centre may be indicated since the ability of any individual clinician to effect change is severely limited in such cases. A range of pain specialists staff inter-disciplinary pain centres such as the Manchester & Salford Pain Centre (MSPC). The MSPC has 4 consultants in pain management and anaesthesia, one consultant rheumatologist and one consultant neurosurgeon, 4 physical therapists, 4 clinical psychologists and 2 nurses. A range of services is provided including post-operative and palliative care, neurostimulation, standard analgesic treatment and individual and group pain management programmes. The latter provides

Table 3. Issues addressed during patient feedback session

- Explain the meaning of signs, symptoms and test results
- Correct misconceptions (e.g. pain not due to inflammation/damage)
- Discuss potential for change in symptoms and functioning
- Explain possible consequences of excessive inactivity/rest/behavioural avoidance
- Discuss drug-taking practice
- Discuss requests for further medical treatment/ /investigation
- Enquire as to perceived consequences of functional improvement on medico-legal or welfare claims
- Obtain views about perceived beneficial or adverse effects of intervention
- Ensure patient is willing to make personal commitment to change

skills based upon a cognitive-behavioural self-management approach to groups of 10-12 chronic pain sufferers on a daily basis over a 3-week period, with follow-up at 1, 3 and 6 months post-programme. Table 4 lists the key objectives of pain management programmes. The rationale, treatment and assessment approach is described in a recent book written by MSPC staff (Main & Spanswick, 2000). Details of a structured psychological interview and the assessment process are also covered in another recent article by the first author (Booker, 1999). Meta-analysis of treatment outcome for inter-disciplinary management (Flor, Fydrich & Turk, 1992) indicate that, compared to uni-professional care, there is a significant reduction in medication use (65% vs 21%, respectively), healthcare use (35% vs 4%), pain behaviour (62% vs 0%) and an increased proportion of return to work (68% vs 36%) and physical function improvements (53% vs 13%) following inter-disciplinary intervention. A more recent meta-analysis of randomised controlled trials of cognitive-behavioural/behavioural therapy confirms the efficacy of these approaches (Morley, Eccleston & Williams, 1999). Inter-disciplinary pain management interventions for rheumatoid arthritis patients have also been shown to have positive outcomes (Parker et. al., 1988; Keefe & Horn, 1993).

Table 4. Pain Management Programme Objectives

- Reduce tendency to engage in cycles of under- and over-activity (learn to pace activity).
- Reduce time spent resting or lying down.
- Reduce level of pain behaviour.
- Reduce, eliminate or optimise use of pain-related medication.
- Eliminate use of unnecessary aids such as wheelchairs, sticks, collars or corsets.
- Reduce dependence on partner/family vis-à-vis activities of daily living.
- Reduce inappropriate health-care use.
- Reduce the likelihood of relapse by teaching problem-solving techniques.
- Increase physical strength, stamina and flexibility.
- Increase tolerance for sitting, standing and walking activities.
- Improve strategies for coping with 'bad days'.
- Improve sleep by changing behavioural routines.
- Improve mood by training in relaxation and cognitive-behavioural techniques.
- Improve understanding of chronic pain.
- Maintain changes in lifestyle by continually reviewing personal goals.
- Return to work or vocational rehabilitation where appropriate.

Summary

When confronted with a patient whose pain problem appears to be becoming more chronic and therefore potentially disabling, family practitioners and hospital doctors alike need to recognise the potential risk of maladaptive biopsychosocial and iatrogenic factors. When thorough medical examinations and investigations are normal or unhelpful, it should not be assumed that the individual is either imagining their symptoms or seeking secondary gain. Chronic pain without discernible cause, or where the cause is not dangerous and not amenable to a curative approach, is the rule rather than the exception in chronic back pain patients. Secondary psychological distress and disability can be prevented by accurate and honest communication, correcting patient's attributions about intractable pain and addressing misplaced fears of activity-related tissue inflammation and damage. The initial increase in consultation time is eventually offset by fewer review appointments and reduced healthcare usage, not to mention job satisfaction. Ultimately, the establishment of specialist inter-disciplinary teams is needed for complex cases that consume a huge amount of healthcare resources with little apparent improvement in health status.

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