

Masterclass

A pain neuromatrix approach to patients with chronic pain

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SUMMARY. This paper presents an approach to rehabilitation of pain patients. The fundamental principles of the approach are (i) pain is an output of the brain that is produced whenever the brain concludes that body tissue is in danger and action is required, and (ii) pain is a multisystem output that is produced when an individual-specific cortical pain neuromatrix is activated. When pain becomes chronic, the efficacy of the pain neuromatrix is strengthened via nociceptive and non-nociceptive mechanisms, which means that less input, both nociceptive and non-nociceptive, is required to produce pain. The clinical approach focuses on decreasing all inputs that imply that body tissue is in danger and then on activating components of the pain neuromatrix without activating its output. Rehabilitation progresses to increase exposure to threatening input across sensory and non-sensory domains.

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INTRODUCTION

Pain and movement are the primary currencies in manual therapy. Most patients present for therapy because they are in pain and most therapies incorporate movement into assessment, diagnosis, aetiology and management. Indeed, many therapies attempt to restore movement in the hope that pain will automatically get better as movement improves. However, from aetiological and therapeutic perspectives, it is difficult to determine the chicken and the egg: is pain caused by abnormal movement or is abnormal movement caused by pain? The current paper presents a model for management in which pain and changes in motor control are considered two dimensions of a multidimensional output of the 'pain neuromatrix'. The model is based on the author's interpretation of the current thought across the pain sciences. The theoretical background for this model is presented and the implications for assessment and intervention are discussed.

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It is important to note that there are assumptions underlying the present approach that have not yet been validated and this paper is occasionally speculative for the sake of clinical relevance. There are no illusions that one approach provides the panacea for persistent pain. Rather, it is hoped that the astute reader will take from this work those aspects that can be integrated with their clinical experience and approach in order to promote better outcomes in a population for which success is elusive.

BACKGROUND

A fundamental principle of this approach is that pain is produced by the brain when it perceives that danger to body tissue exists and that action is required. All dimensions of pain serve to promote this objective. Thus pain is a multiple system output, not just 'an unpleasant sensory and emotional experience associated with actual or potential damage...' (Merskey & Bogduk 1994). The principle that pain is primarily aimed at action is not novel — it was proposed by Patrick Wall a decade ago (Wall 1994) — however, it is yet to gain widespread acceptance. This is somewhat surprising because the notion that pain is a reliable informant of what is actually happening in the tissues is no longer tenable. There

are many factors that are important in determining what hurts for whom and when, for example context, company, competitive stimuli, meaning. Discussion of those factors is not appropriate here. However, their undeniable impact points clearly to (a) the complexity of pain as a multiple system mechanism, and (b) the simplicity of its modus operandi: the body is in danger and action is required.

Each dimension of pain is consistent with an immediate relevance to survival. Experientially, pain is unpleasant (Merskey & Bogduk 1994) and demands attention (Eccleston & Crombez 1999). Pain reduces cortical processing capacity (Derbyshire et al. 1998), slows decision making (Crombez et al. 1996), and increases cognitive error rate (Buckelew et al. 1986). Not surprisingly, chronic pain patients often report being forgetful and easily distracted (Jamison et al. 1988; Parmelee et al. 1993; Schnurr & MacDonald 1995). During pain, immune activity is modified (Watkins & Maier 2000), hypothalamus–pituitary–adrenal axes and sympathetic nervous system activity is altered (Melzack 1999), reproductive system function is reduced (Negro-Vilar 1993; Rivier 1995) and visuomotor systems are activated (Price 2000). Thus, pain is the cortical output of highest priority.

Motor output as a dimension of pain varies according to the task at hand, but generally serves to both promote escape and limit provocation of the painful part. Limb muscle studies have shown that to this end, muscle pain is associated with increased and decreased activity of the agonist muscle and antagonist muscles, respectively (Lund et al. 1991; Graven-Nielsen et al. 1997). Trunk muscle studies have shown that pain is associated with reduced modulation of muscle activity during dynamic movements (Arendt-Nielsen et al. 1996; Zedka et al. 1999) and reduced and increased activity of deep and superficial trunk muscles, respectively, during single limb movements (Hodges et al. 2003). This type of response is consistent with splinting the trunk and/or preparing the body for torque production in the limbs. Taken together, the data suggest that motor output changes associated with pain are predominantly (although not exclusively) driven by higher centres.

What is the pain neuromatrix?

In the current therapeutic approach, the pain neuromatrix is that combination of cortical mechanisms that when activated produce pain. The term is taken from Melzack's 'Neuromatrix theory' (1996), but also acknowledges progress in imaging studies that identify neuroanatomical correlates of pain (the 'Pain matrix' — including anterior cingulate cortex (ACC), insular cortex, thalamus and sensorimotor cortex).

The conceptual view of the pain neuromatrix

Although the brain is a remarkable information processor, it is probably unable to create an experience instantaneously based on incoming sensory information, even though this is commonly assumed to be the case. Rather, the brain probably produces a common output that extends to awareness and motor tasks, much in the manner proposed by Hebb (1949) and Melzack (1996). Those theories conceptualize a network of cells (termed 'the neuro-matrix' by Melzack), determined genetically and/or on the basis of sensory input, that produces a constant perceptual and motor output, such that both outputs can be activated by a single input, or in some cases no inputs at all. To illustrate this, consider sitting in a train while the adjacent train begins to move. In this situation, visual cues alone are often sufficient to produce the experience of moving and a postural response appropriate to that experience, even though there is no corroborative vestibular or proprioceptive input.

The neuroanatomical pain matrix

Imaging studies demonstrate that there is no single 'pain centre'. Many cortical areas can be activated during pain and wide variability exists within and between individuals (see Ingvar and Hsieh 1999 for review). However, some cortical areas are involved more often than others. These structures are known as the 'pain matrix' and provide a neuroanatomical reference for the current therapeutic model.

Most studies report activity in the ACC during pain (e.g. Ingvar & Hsieh 1999; Creac'h et al. 2000; Apkarian et al. 2001; Bantick et al. 2002), although most imaging studies not investigating pain also report ACC activity. Across studies, the middle portion of the ACC is thought to be important for deciding 'what should I do?', such that it can be considered an action centre (the ACC has been termed the limbic-motor cortex, Craig 2002). In pain studies, the ACC is considered to serve to (i) establish an emotional valence of pain, and (ii) coordinate the selection and planning of an appropriate behavioural/motor response strategy (Price 2000). Similar ACC activity has been reported during non-nociceptive but biologically threatening events such as anticipated pain (Sawamoto et al. 2000) and anxiety (Kimbrell et al. 1999; Osuch et al. 2000) and the ACC is chronically active in chronic pain patients (Hsieh et al. 1995). Other key areas include the thalamus (Bushnell & Duncan 1989), anterior insula, prefrontal and posterior parietal cortices (Ingvar & Hsieh 1999). These areas are implicated in the affective-emotional and motoric dimensions of pain and together can be considered primary substrates of the pain neuromatrix (Fig. 1).

In terms of the therapeutic approach presented here, the exact neuroanatomy of the pain neuro-

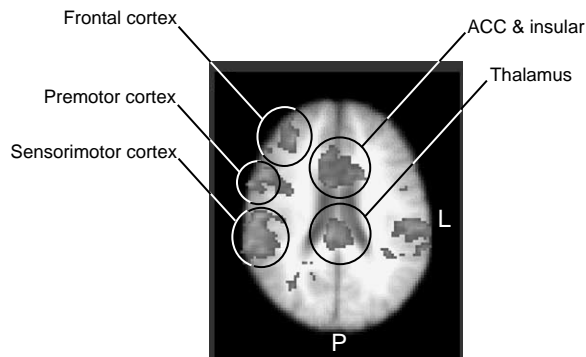


Fig. 1—Functional magnetic resonance image (axial view, multiple slices) of brain activity during painful thermal stimulation (57°C) of the left hand. Components of the pain matrix are circled — thalamus, anterior cingulate cortex (ACC), insular, frontal, premotor and primary sensory and motor cortices. Note that the image is left-right inverted such that L marks the left cortical hemisphere. P denotes posterior.

matrix is not important. Rather, the above studies suggest cortical regions that are likely to be involved in what is ultimately an individual-specific pain neuromatrix.

The virtual body

A critical component of the current approach is that pain is experienced in the body image that is held by the brain, labelled here the 'virtual body'. Although this notion seems at first glance to be counter-intuitive, this is only because the virtual body is so effective — the phantom experiences of an amputated limb is a profound case in point. Phantom experience has been discussed at length by Melzack (Melzack 1989; 1990; 1996; Melzack et al. 1997). Neuroanatomically, the primary somatosensory homunculus is the most well-known spatial representation of the internal and external physical environment (Deiber et al. 1991; Grafton et al. 1992; Grafton et al. 1996), but there are other representations as well. The dorsal insular cortex is proposed to contain representation of the *physiological condition* of the entire body (Craig 2002). This proposal is based firstly on studies that show activity of the dorsal insular during homeostatic mechanisms (including pain), and secondly on studies that show its connection almost exclusively with small-diameter afferents. It is noteworthy in this regard that small diameter afferents, although conventionally called nociceptors, are more accurately considered *interoceptors* — they detect changes in the body tissue¹ (MacIver & Tanelian 1992; Carlton et al. 2001; Cook & McCleskey 2002).

Different virtual bodies may dominate experience at different times. For example, Andre et al. (2001)

¹An excellent example of the distinction between nociceptors and interoceptors was described by Vallbo et al. 1999 when they observed that C fibres are exquisitely sensitive to slow, weak mechanical stimuli that evoke sensual touch.

demonstrated temporary non-painful and movable phantom limbs in amputees after vestibular caloric stimulation (inserting 20°C water into one ear), regardless of whether, prior to stimulation, they had no phantom, a painful phantom or a dysmorphic phantom. Those authors concluded that sudden vestibular stimulation activated a stable and intact virtual limb in order to provide the postural frame of reference on which to base a postural response to the perturbation. That is, caloric stimulation caused the virtual body to be overridden by a relatively permanent counterpart. However, in the current discussion, the incumbent virtual body is important for the very reason that it is continually updated by sensory input. Therefore, it may be an important part of the pain neuromatrix because it provides a neural substrate for allocating pain an anatomical reference. In light of this, treatment aimed at reducing pain is aimed at the virtual body, albeit commonly (but not exclusively) accessed through the corresponding body tissues.

The virtual body is also indispensable for the maintenance of coherent motor commands, including the coordination of postural and movement responses. The central nervous system (CNS) produces motor commands on the basis of (i) the predicted requirements of the movement including the predicted disturbance to stability, and (ii) the perceived current position, movement and stability of the body, such that postural control commands are effected prior to movement (Belen'kii et al. 1967; Bouisset & Zattara 1987). Thus, the virtual body provides a common platform from which experiential and motoric dimensions of pain can be launched, which make it an important consideration for clinicians interested in pain and movement.

Added complexity of chronic pain — effects on activity of the pain neuromatrix

Two inter-dependent mechanisms can contribute to chronicity — nociceptive (including humoral or immune-related dysfunction that stimulates nociceptive structures and body tissues) and non-nociceptive (cognitive-evaluative) mechanisms. In either case, there is an increase in the conviction of the CNS that body tissue is in danger and, therefore, there is an increase in activity of the pain neuromatrix.

When pain persists, both the nociceptive system and the virtual body undergo profound changes, which increase sensitivity to noxious as well as non-noxious input and corrupt the integrity of motor output. Review of the changes that occur is beyond the scope of this paper; however, potent changes occur peripherally and centrally. Alterations of wide dynamic range second-order nociceptors are particularly relevant (Doubell et al. 1999; Mannion & Woolf 2000) because these second-order nociceptors

dominate ascending connections to the brain areas identified as key components of the pain neuromatrix (see Price, 2000 for review). Further, reorganization of primary sensory and motor cortices, which also occurs with chronic pain (Flor et al. 1998), probably has even more profound effects across pain dimensions.

Discussion of cognitive–evaluative mechanisms associated with chronicity is also beyond the scope of this paper. It is sufficient to highlight the importance of beliefs and attitudes that emphasise the threat value of pain (see Gatchel & Turk, 1999 for review). The threat value of pain is an important predictor of its unpleasantness, but more importantly it is of obvious relevance to the underlying principle of the current approach — that pain is produced by the brain when it perceives that danger to body tissue exists and that action is required. Finally, it is thought that alterations in both nociceptive and cognitive–evaluative input can lead to modification of the shape and properties of the virtual body, such that motor and postural responses become variable and inaccurate.

The effect of chronicity — implications for the pain neuromatrix approach to chronic pain

The current approach proposes that the primary effect of chronicity is enhanced synaptic efficacy (broadly speaking, the sensitivity) of the pain neuromatrix, such that less input is required for activation. This proposal is able to explain many clinical phenomena in the chronic pain patient group, e.g. dynamic functional allodynia (pain during movements that would not normally be painful), or pain elicited by seeing another person perform a painful movement. The main point is that smaller and seemingly less relevant inputs are sufficient to activate the neuromatrix and thus produce pain. Herein lies the primary challenges of this patient group: an unclear relationship between pain and tissue input; difficult-to-predict flare-ups; poor tolerance of normal therapeutic approaches; problems with physical and functional upgrading; difficulty generalizing gains to other activities.

THE CLINICAL APPROACH — DESENSITISATION AND GRADED ACTIVATION OF COMPONENTS OF THE PAIN NEUROMATRIX

The aim of the current approach is to utilize functional components of the individual-specific pain neuromatrix that are appropriate for movement and that are consistent with the normal aims of treatment, without reinforcing the synaptic efficacy of the neuromatrix. There are three aspects of the approach: (i) reduction of threatening input so as to reduce

activity of the pain neuromatrix and thereby reduce its efficacy, (ii) targeted activation of specific *components* of the pain neuromatrix without activating the neuromatrix, and (iii) upgrading physical and functional tolerance by exposure to threatening inputs across sensory and non-sensory domains.

1. Reduction of threatening input

Reduction of threatening input — nociceptive mechanisms

Where possible, nociceptive mechanisms that contribute to threatening information should be treated. Obviously, treatment will depend on sound tissue and neural examination techniques and selection of appropriate therapeutic strategies. Broadly speaking, any strategy that has an inhibitory effect on nociceptive input is probably appropriate in the short term unless it simultaneously activates non-nociceptive threatening inputs. For example, although manual therapy strategies may activate endogenous inhibitory mechanisms, (e.g. Vicenzino et al. 1998), treatment may be delivered in such a context as to reinforce to the patient that there is something wrong in their tissues (i.e. the conviction that the body is in danger). Comprehensive appraisal of the mechanisms of various therapeutic strategies lies beyond the expertise of the current author and is not appropriate here.

Reduction of threatening input — non-nociceptive mechanisms — education

The main objective of education is to decrease the threat value associated with pain by increasing the patient's understanding of human physiology. Our group has conducted several studies to evaluate the effect of such education as a way of altering beliefs and attitudes about the meaning of pain (e.g. Moseley 2002; 2003b). The education material is outlined in Table 1 and presented in detail in Butler and Moseley (2003). It includes high-level pain physiology information. Contrary to popular opinion among health professionals, patients are able to understand complex physiology if the information is presented appropriately. In fact, after education, patients appear to have a better understanding of pain physiology than most health professionals, excepting those who have participated in education themselves, or who have specialist training in pain sciences (Moseley 2003a).

Pain physiology education differs from popular education strategies (for low back pain), which have focussed on anatomy and physiology of the lumbar spine. Such a focus is thought to have limited clinical effect (Cohen et al. 1994; Triano et al. 1995) although for a contrary view, see Waddell and Burton (2000). More recently, education about cognitive and behavioural responses, given prior to injury or after an

Table 1. Material presented in education about pain physiology

The neuron	Modality-specific receptors, axon, terminal bouton
The action potential (message)	All or nothing, post-synaptic membrane potential, propagation, orthodromic and antidromic flow
The synapse	Neurotransmitters, inhibitory and facilitatory input, chemically driven ion channels, ion channel synthesis and absorption
Primary nociceptors (danger receptors)	Respond to danger
Second order nociceptors (danger messenger nerves)	Sum of inhibitory and facilitatory input, interneurons from non-nociceptive fibres (normally inhibitory), project to many parts of the brain
Brain output dependent on total perception of danger	All information that is relevant to that decision is considered — thoughts, memories, beliefs, explanatory model, consequences
Descending input	Inhibitory and facilitatory — also dependent on above
Primary nociceptor state-dependent functioning	Potentialiation and summation, ectopic pacemakers, dorsal root ganglion, neurogenic inflammation, allodynia and hyperalgesia
Second order nociceptor state-dependent functioning	Potentialiation, active blocking of ion channels, increased receptor synthesis, sprouting, activation by endocrine mediators

initial episode, reduces chronic *disability*, although the effect on chronic *pain* is not known (Symonds et al. 1995; Burton et al. 1999). There are limited data that support the efficacy of such education with patients who already have chronic pain. Anecdotally, this may be because the response that is encouraged in such programs is counterintuitive for patients because their understanding of pain is based on a structural-pathology model (i.e. that pain is analogous to injury). Pain physiology education targets this limitation by aiming to reconceptualize the underlying physiological problem of a patient's pain, on the assumption that an appropriate cognitive and behavioural/motor response will follow. This is sensible according to principles of 'deep learning', in which information is retained and understood and applied to problems at hand (Sandberg & Barnard 1997). In contrast, 'superficial' or 'surface' learning is that in which information is remembered but not understood or integrated with attitudes and beliefs (Evans & Honour 1997). To this effect, it is important to note that deep learning is facilitated when the learner is motivated (Sankaran 2001) and when the information presented is made personally relevant (Moreno & Mayer 2000), both of which are promoted by the method of education used here.

It is critical that patients understand the material that is presented — care should be taken to use various graphics (including whiteboards, hand-drawn pictures, personalized manuals), and accessible metaphors in order to achieve this objective. Education can be time consuming but we have demonstrated an effect, albeit smaller, with group programs (Moseley 2003b).

Targeting the explanatory model

Based on the underlying principle of the current work, the way that a patient explains their pain ('explanatory model') is an important consideration in targeting education and planning therapy. What story does the patient tell about their pain? Evaluation of the explanatory model is possible through

direct questioning — 'What in your body do you think is causing your pain?' Most commonly, the explanatory model held by a patient is based on a structural-pathology model because that is the dominant model in the medical and lay arenas. However, the structural-pathology model is often not appropriate. Focus on a structural label for pain may actually heighten attention on the pain, emphasize the vulnerability of the body to damage and increase patients' health care consumption (Jones et al. 1988; Nachemson 1992; Hirsch & Liebert, 1998).

There are three key points to consider in modification of the explanatory model. First, care should be taken to present currently accurate information rather than an explanation that has been diluted for the sake of simplicity, to save time or to avoid a confrontation with the patient. This means that on the basis of the information that they have been presented, the patient should be unable to support an inappropriate or indefensible explanatory model. Second, the information presented should offer the patient an alternative explanatory model that is supported by the currently accurate information about human physiology. Third, the information must be presented in a manner that is respectful of the patient and acknowledges their suffering. At first glance this seems a moot point. However, the negative stigma associated with chronic pain is remarkably pervasive despite the fact that the basis of the stigma is naïve.

In summary, to be effective with education, the therapist must (i) be an expert and be perceived as such by the patient, and (ii) be respectful and compassionate and be perceived as such by the patient.

2. Activation of components of the individual's pain neuromatrix

Management framework

The following framework for management is presented to patients (Fig. 2). It represents a practical response to the pain physiology education and

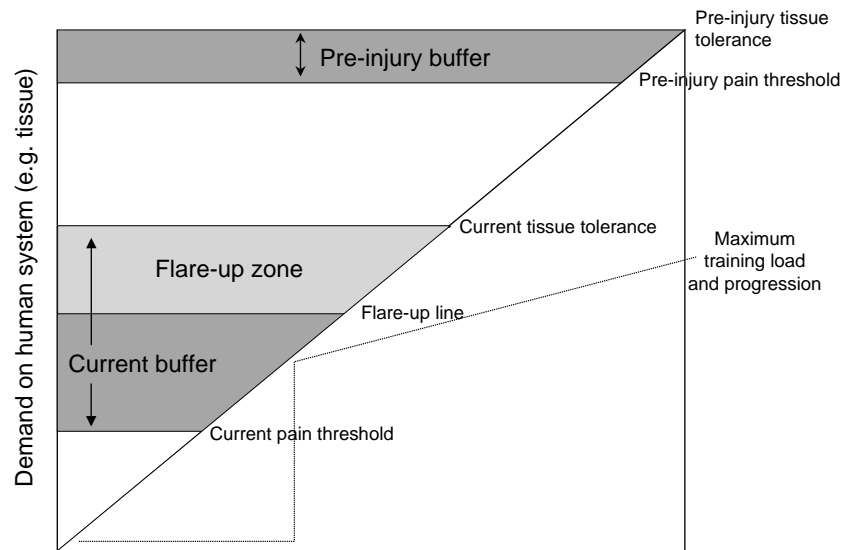


Fig. 2—A management framework that is presented to patients. The *y*-axis represents the demand on a particular human system (usually the musculoskeletal system) that is imparted by a certain activity or combination of activities. The pre-injury and current buffer conceptualise the protective gap between onset of pain and tissue damage. Note that in chronic pain, by virtue of enhanced synaptic efficacy of the pain matrix, the buffer has been expanded. This is an effective protective strategy. The training load (dashed line) begins below the flare-up line and progresses conservatively. The flare-up line and tissue tolerance gradually increase in line with progression of the training load.

incorporates an understanding of pain as the multiple system output of a sensitized pain neuromatrix. Prior to the onset of pain, there was a maximum amount of an activity that could be performed before tissue would in some way yield. This is termed the ‘previous tissue tolerance’. There was a reliable protective mechanism, mediated primarily by the sensory nerves within body tissue, that would usually activate the pain neuromatrix. However, three main effects occur after injury and with chronic pain: (i) reduction in tissue tolerance, by virtue of altered tissue properties, deconditioning and disuse, (ii) reduction in the activation threshold of the peripheral nociceptors such that the integrity of the tissue-based protective mechanisms is maintained, (iii) increase in and diversification of threatening input, mediated by increased sensitivity of the higher order nociceptive structures (spinal cord and brain) and/or cognitive–evaluative factors associated with threat, such that the pain neuromatrix is activated at low levels of threatening input.

Assessment — determining the baseline

In the current context, ‘baseline’ is the extent to which the components of the pain neuromatrix can be utilized *without* activating the whole neuromatrix. That is, what level of nociceptive input (e.g. how many, how often, how demanding an exercise or functional task?) or other input (e.g. how threatening from a non-nociceptive perspective?) can occur without activating the pain neuromatrix? Determination of the baseline is more difficult with chronic pain than with acute or sub-acute problems, by virtue of the increase and diversification of threatening inputs.

Assessment consists of determining motor and functional baselines.

Motor baselines

Clinical assessment of motor strategies can involve those tests with which the therapist is most comfortable and competent (e.g. assessment of deep trunk muscle function or one-legged standing balance), however the tests may require more conservative baselines than is required in acute or sub-acute problems. Motor strategies can provide important insight into what inputs are likely to activate the pain neuromatrix. According to the theoretical basis outlined earlier, particular note should be made of the relative activation of torque producing and non-torque producing (so-called ‘stabilizing’) muscles. Broadly speaking, this is a consideration common to numerous body areas, for example, deep versus superficial trunk muscle activation in back pain (O’Sullivan et al. 1997; Richardson et al. 1999), vastus medialis obliquus versus other quadriceps muscles in knee pain (Voight and Wieder, 1991; Cowan et al. 2002), deep cervical flexors versus superficial neck muscles in neck pain (Jull et al. 1999).

In a portion of cases the baseline will be relatively normal. In many cases, assessment will involve excursion from this normal baseline in a multi-dimensional manner. Fig. 3 outlines the options. If the patient can easily obtain a normal baseline, the new baseline can be determined by progressively making the task more threatening. This may involve increasing the physical demand of the task — increase the speed, amplitude and duration of the movement. Alternatively or in addition, it may involve modifying the context or environment, increase the implications

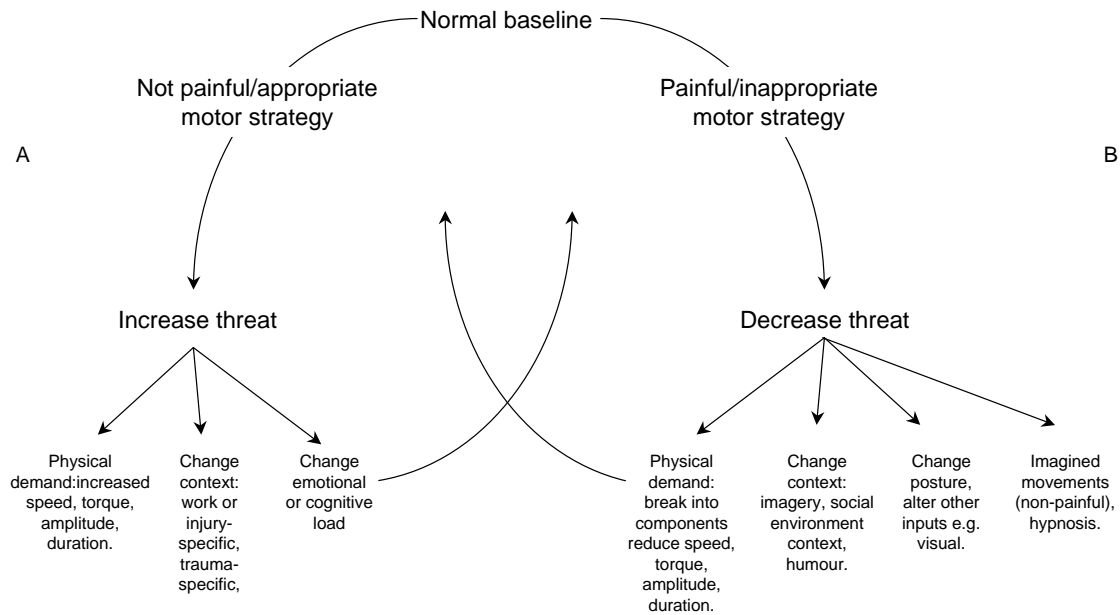


Fig. 3—Options for determining the baseline and planning progression. (A) If the normal baseline level is performed pain-free (functional task) or using an appropriate motor strategy (motor task), then the aim is to increase threat via physical, contextual changes or emotional/cognitive load. (B) If the normal baseline level elicits pain (functional task) or an inappropriate motor strategy (motor task), then the aim is to decrease threat, via similar means.

of poor performance (e.g. ‘someone of your ability should have no problem performing this’), or perform the same movement from a different postural reference or within a different emotional context.

For example, a patient with chronic back pain is able to perform a near perfect voluntary abdominal muscle task in standing. Assessment of muscle activity while this patient imagines bending forward to pick up a box might reveal a loss of quality of contraction, for example marked activation of the superficial abdominal muscles/long back extensors. This finding implies that forward bending is a threatening movement and identifies success at this task as a suitable short-term goal. Threatening stimuli can be work-specific (e.g. work environment), context-specific (e.g. sitting in car) and posture-specific (that is, the postural frame of reference from which a movement is occurring).

For many patients, the changes in nociceptive and cognitive–evaluative inputs are such that the baseline is difficult to find because conventional baselines are painful (i.e. they activate the pain neuromatrix). In this situation, the aim is to utilize as much of the pain neuromatrix as possible without activating it. That is, to reduce the threat associated with the task (Fig. 3b). This may involve breaking the movement down, performing imaginary movements (that are imagined pain-free), or changing the postural reference or context of the movement. Can the task be modified in order to make it non-threatening? How can components of the movement/task be performed in a manner that does not activate the pain neuromatrix? For example, if minimal cervical rotation in sitting is painful, try cervical rotation in standing, imagined

cervical rotation in sitting, performing the movement in the dark (i.e. removing visual stimuli that may add to the threat), or try imagery in which the patient is turning their head toward a cool breeze on a hot day (see Table 2). These strategies activate motor mechanisms that would normally activate the pain neuromatrix but do so in a manner that is explicitly non-threatening and therefore less likely to activate the pain neuromatrix.

Functional baselines

Functional baselines are effectively determined by the patient, however persistent assistance from the therapist is required. The functional baseline is determined by the flare-up line presented in Fig. 2, which is the point at which the patient notices a marked increase in pain that persists for more than a few minutes. Patients invariably relate to the term flare-up and recognize it as a period of severe pain which is often accompanied by incapacity, inability to sleep, nausea and vomiting, and ‘drastic’ treatment options. Flare-ups need not be this severe and, anecdotally at least, are less severe if the patient has gained an accurate understanding of human physiology as it relates to his/her pain. However, the prevalence of this idea of a flare-up probably reflects in part that patients find it difficult to determine their own flare-up line. Physiologically, the flare-up line may coincide with activation of tissue-initiated nociception, although there are no data that evaluate this possibility.

Persistent and skilled questioning should suffice to identify the baseline of any functional task. For

Table 2. Examples of multidimensional decrease and increase in threat

Activity	Flare-up line	Mon	Tue	Wed	Thu	Fri	Sat	Sun
Deep neck muscles/head tilts	10 × 10	5 × 5	7 × 5	9 × 5	8 × 6	9 × 6	10 × 6	9 × 7
Hnging washing	10 items	4 items	5	6	7	8	9	10
Typing at desk	8 min	4 min	4.30	5	5.30	6	6.30	7

example, an interaction between therapist and patient;

Therapist: 'How long can you walk before you flare-up?'

Patient: 'I can walk for 30 min but I pay for it the next day'

Therapist: 'Can you walk for 20 min without flaring up?'

Patient: 'No, but I have'

Therapist: 'Can you walk for 10 min without flaring up?'

Patient: 'Probably not — definitely not up hills'

Therapist: '5 min on a flat surface?'

Patient: 'Probably'

Therapist: '3 min on a flat surface?'

Patient: 'Definitely'

Thus, for this patient the baseline for walking is 3 min on a flat surface. This process of questioning, although laborious, is usually critical in order to get a clearly defined baseline and to impart an understanding to the patient of what is meant by 'baseline'. The patient is usually able to undertake this process on their own once several activities have been discussed and baselines have been defined. It is critical that the agreed baseline is recorded, preferably in the patient's diary.

Progression — a little a lot

Fig. 3a also provides a framework within which motor and functional tasks can be progressed. Once a baseline is identified, a goal is established. When that goal is achieved, it becomes the new baseline. Different issues are pertinent to progression of motor and functional tasks. In either case, progression must be conservative but continual — small, frequent increments in the training load. This can be frustrating for patients and therapists alike, however, it is important to both increase tolerance and avoid flare-ups: 'persistence and patience' is a good theme. Generally speaking, patients respond to this if they can understand the rationale, which is presented both as part of the education and in an ongoing way.

Easily remembered phrases are useful in this regard — e.g. 'every day you do more than you did yesterday, but not much more'.

Progression diary

Compliance is a major barrier to chronic pain rehabilitation (see Nicholas 1995 for review). Most often, patients exceed the flare-up line and become entrenched on a boom-bust cycle of increasing disability and dysfunction. Providing patients with a sound rationale as to why a flare-up should be avoided counters this, at least in part. However, in order to balance progression with avoidance of flare-ups, it is often necessary to keep a progression diary, in which the motor and functional training load is well documented and planned in advance. The aim of a progression diary (see Table 3) is to map out the increments of motor and functional training and to ensure that patients *do not exceed the training load*. This is critical because patients will often, having completed the functional goal of a given day, be feeling relatively pain-free and satisfied with their progress, and decide to do just a little more. Anecdotally, exceeding the training load in this manner, which often leads to flare-up, which in turn is poorly managed by virtue of an inaccurate understanding of the physiology underlying the flare-up, is the most common reason for failure in management.

Training alarm

Much functional and motor training is defined by time. Because distraction remains the most effective means of endogenous analgesia, it is both an ally and enemy in management because patients are at risk of exceeding the training load, simply because they do not realize they have done so. For example, consider the patient who has as a pre-planned training load of sitting for 14 min. The patient enjoys reading and uses reading as an effective distraction. However, the patient is sufficiently distracted that they read for 20 min at which time they have quite rapid onset of pain that leads to a flare-up. Alarm clocks are

Table 3. Example of a progression diary, applied to functional and motor tasks

Target	Change context	Change posture	Recruit premotor and motor no movement	Alter sensory input	Change emotional state
To decrease threat	Rotate while talking with friends	Rotate in standing/lying/side lying	Imagine pain-free rotation	Rotate with eyes closed	Rotate while listening to favourite music
To increase threat	Rotate in car	Rotate with arms	Imagine rotating	Rotate while eating	Rotate when angry

invaluable in preventing this scenario and are easy to use. In the previous example, by setting the alarm to 14 min the patient is able to benefit from distraction by reading but not exceed the training load.

Progression of motor tasks

Each time a new baseline is established, progression should involve exposure to more threatening inputs in a multidimensional manner. If the increment of progression according to physical demand is too large, it is possible to progress in other dimensions, for example, perform the motor task under stressful conditions, in more threatening emotional states, or while performing a cognitive task. Table 2 provides examples of multidimensional increases in threat associated with rehabilitation of the patient with cervical spine pain. This strategy is consistent with the theoretical goal of activating components of the neural network without triggering it to produce pain.

Progression of work

Often the success of a return to work trial is prevented by the patient exceeding a suitable training load, usually because the increment in physical load is too large. We have had greater success in the chronic pain patient group by pursuing more conservative training loads that are increased more often and with smaller increments. For example, M was a patient who presented with 4 year history of debilitating arm pain initiated and aggravated by keyboard work. She had failed two previous attempts to return to work that began with three 4 hour(h) shifts (12 h) per week. On both occasions she did not complete 2 weeks of work. In our revised progression M began with 15 min at work and incremented initially daily in 5 min blocks. Importantly, she went to work every-day. She successfully graduated to five 3-h shifts (15 h) per week by the 12th week of the program and proceeded to full time work duties in ~6 months. At first glance, this seems like a laborious return to work program. However, it was both quicker and cheaper (in terms of time off work) than previous attempts.

SUMMARY AND CONCLUSION

The current paper has presented the background, guiding principles and clinical approach to treating chronic pain using a pain neuromatrix approach. The approach is founded on key principles — that pain is a multiple system output that is activated by an individual-specific pain neuromatrix; that the pain neuromatrix is activated whenever the brain concludes that body tissue is in danger and action is required; and that pain is allocated an anatomical reference in the virtual body, upon which coherent motor output is also dependent. When pain persists,

there is (i) an increase and diversification of threatening inputs such that the pain neuromatrix can be activated by all manner of threatening stimuli, nociceptive (including immune and endocrine-driven mechanisms) and cognitive–evaluative, and (ii) alterations in the morphology and behaviour of the virtual body, thus further altering motor output.

The therapeutic aspects of the approach focus on reducing the sensitivity and activity of the pain neuromatrix, via reduction of threat. The key components are education about human physiology and a systematic approach to identification and progression of motoric and functional baselines across sensory and non-sensory domains.

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