Pain, the Tissues and the Nervous System: A conceptual model

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Key Words

Pain, biopsychosocial aspects of pain, stress.

Summary

This paper challenges current clinical models and systems for assessing and managing on-going pain states to incorporate a broader biological and therapeutic framework. Included is an acceptance of the current criticisms made towards a purely tissue based/modality based paradigm for pain treatment. The mature organism model proposed is presented as a workable conceptual starting block for incorporating mechanisms of pain into the broad science of stress biology and the biopsychosocial model of pain.

Introduction

The principal aim of this paper is to introduce a biologically based model that it is hoped will:

1. Help clinicians link together many of the complex issues and mechanisms involved in pain problems.

2. Help clinicians see the inadequacy of site-specific diagnosis and of passive treatment approaches that target these sites with the tendency to over-focus on the behaviour and intensity of pain and the therapeutic quest for its relief. This is especially so for the large numbers of on-going pain states that physiotherapists encounter.

3. Provide a model which helps explain to patients the nature and consequences of pain and the positive options for recovery.

The overwhelming message coming from respected researchers, clinicians and writers on the way forward with 'pain' is for patient empowerment via education and active rehabilitation of function rather than over-reliance on passive therapies (eg Harding and Williams, 1995; Klaber Moffett and Richardson, 1997; Loeser, 1996; Waddell, 1996; Zusman, 1997a, b). The literature also recommends that this approach should be augmented with the recognition, assessment and adequate therapeutic focus on psychosocial factors that have been repeatedly shown to have great predictive value for chronicity and therapeutic outcome (eg Caudill, 1995; Cohen and Campbell, 1996; Gatchel and Turk, 1996; Hildebrandt *et al*, 1997; Main and Watson, 1995; Waddell *et al*, 1993). Physiotherapists are being bluntly urged to change their approaches and rationales. For example: 'The emphasis of physical therapy for non-specific low back pain should change from symptomatic methods, which have shown to be ineffective, to early activation and restoration of function, as in all other musculoskeletal conditions. This requires a fundamental shift in physical therapy practice and resources' (Waddell, 1996).

Clinicians involved in the diagnosis and management of all benign pain states have two major problems. First, evidence of pathological changes in tissues underlying the painful area and in tissues which can refer pain to the area is often lacking. Secondly, there is a large body of evidence demonstrating tissue pathology in the absence of pain (eg Boden *et al*, 1990; Deyo and Phillips, 1996; Haldeman, 1990; Jensen *et al*, 1994; Melzack and Wall, 1996; Simms, 1996).

Unfortunately physiotherapists have rarely questioned this paradox and have persevered with highly skilled physical tissue analyses aimed at validating the tissues and peripheral nerve trunks and roots as definitive sources of ongoing pain in the great majority of patient presentations (eg see Boyling and Palastanga, 1994). This is hardly surprising, considering that the only alternative status for on-going pain states that medicine cannot fit within a tissue abnormality/disease based construct, is the unsavoury and unproductive 'psychogenic pain' label. So often the implied message for the patient is that if nothing can be found, nothing can be wrong and the patient is therefore to blame (Morris, 1991). Thankfully, the purely psychogenic origin of pain theory has been widely criticised and challenged (Gamsa, 1994; Melzack and Wall, 1996) and is one which many physiotherapists have been naturally unwilling to accept in any case.

It is not surprising that in our attempts to help our patients, legitimise their pain and find something for them, that we have plunged into greater and greater depths of skilled tissue testing and focused analyses of the behaviour of pain. A fundamental reasoning error may be made by labelling a tissue as faulty on the basis that passive manual testing can reproduce the patient's pain. The reasoning error is to assume that a sensitive tissue evoking pain on mechanical testing is responsible for the pain rather than a

reflection of the sensitised state of the nervous system (see Cohen, 1995, 1996; Loeser, 1991).

The strength of the tissue basis of pain construct can be further reinforced by the application of apparently successful techniques and interventions to the 'blameworthy' tissues. For example, successfully relieving pain by mobilising, manipulating or injecting the cervical zygapophyseal joints of a patient who complains of neck and arm pains can be seen as validating the targeted joint as being the source of the problem. This logical application of reasoning unfortunately omits a broad understanding of the effects of factors such as:

- The patient/therapist interaction (Klaber Moffett and Richardson, 1997) and other non-specific treatment effects that generally get lumped together with the rather abused term 'placebo'. This includes such things as the provider's attitude towards the treatment and towards the patient (warmth, interest, empathy); the faith of the patient in the treatment; the reputation, expense or impressiveness of the procedure; and suggestibility (Deyo, 1993; Miller, 1989; Whitney and Von Korff, 1992).
- The natural regression to the mean of an ongoing condition. This is a statistical concept which suggests that patients tend to seek treatment when their symptoms are most extreme, and that left to their own devices, most will subsequently return towards some average or more typical level of pain (Deyo, 1993; Whitney and Von Korff, 1992).

The weakness of the tissue based model for diagnosing and treating on-going pain has been highlighted because there is powerful evidence that it does not help and that it may actually be making matters worse (eg see Loeser, 1996; Zusman, 1997a, b). The tide of current opinion is urging us to adopt an attitude that views pain as a dynamic entity whose mechanisms shift and change over time and that must be considered from a more broadly based biopsychosocial perspective (Feuerstein and Beattie, 1995; Turk, 1996; Waddell *et al*, 1993).

Thus, a consideration of the repercussions of pain and its mechanisms in terms of physical deconditioning in concert with the patients' attention, thoughts, feelings, knowledge and beliefs, are currently viewed as vital to an expanded perspective on the management of all pain states (eg see Campbell, 1996; Gatchel and Turk, 1996; Gifford and Butler, 1997; Harding and Williams, 1995). For physiotherapy, integrating matters of the mind/brain has never been a comfortable issue when the problems we deal with are so plainly physical in the opinion of our patients.

The Mature Organism Model

The mature organism model (MOM) shown in figure 1 has been developed as a teaching tool to help clinicians and patients reach a broader understanding of pain, appreciate it in a biological and psychosocial context and hence manage it better. A fundamental feature of the model is the placement of pain in the discipline of stress biology. That is, the sensation of pain is seen as a perceptual component of the stress response whose prime adaptive purpose is to alter our behaviour in order to enhance the processes of recovery and chances of survival. Stress biology and the stress response broadly considers the systems and responses concerned with maintaining homoeostasis. Pain and our reaction to it needs to be integrated into the broad discipline of stress biology.

A 'body' is a very sophisticated vehicle that carries and looks after our genes. All 'bodies' can be viewed as a survival machines in which the genetic material lives that enables them to replicate (Dawkins, 1989, 1996). All higher animals have bodies that consist of a number of organs and tissue systems whose activities are integrated via the central nervous system (CNS). If we analyse ourselves in biological terms, it helps if the CNS/brain is viewed as the central scrutinising centre – or the stress response co-

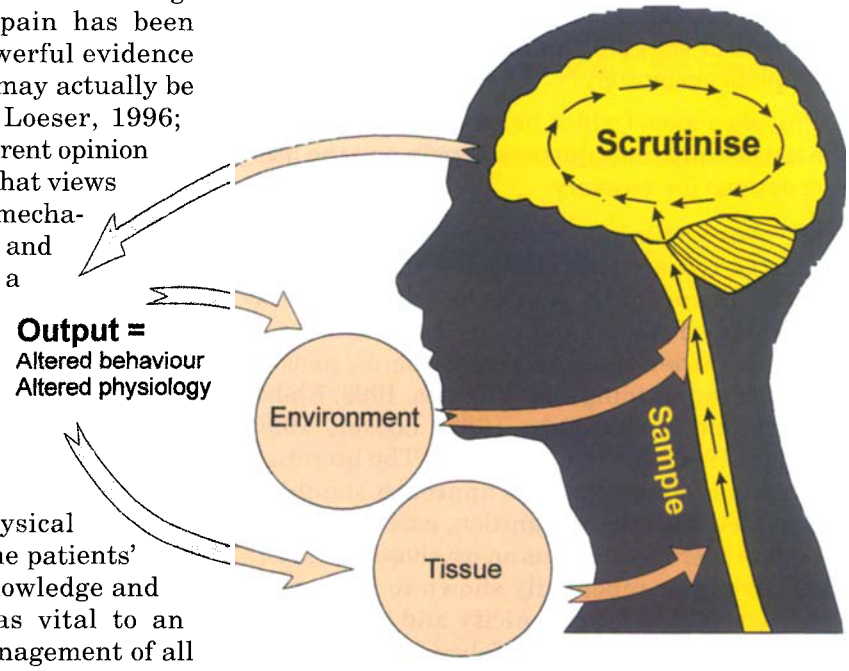

Fig 1: Staying alive – homoeostatis: the mature organism model. This figure represents the fundamental pathways into and out of the brain/CNS that are required for bodily survival. Details of each component are discussed in the text

ordination centre, that continually samples (consciously and 'unconsciously') the outside environment, its own body and relevant past experiences (the brain samples from 'itself') – and then 'outputs', or responds on what it finds to the best advantage for its body and the vital genes it contains (fig 1). Outputs/responses can be broadly divided into:

- Overt behavioural responses. For example, when injured we demonstrate subsets of illness behaviours (Fordyce, 1984; Pilowsky, 1995a, b; Waddell *et al*, 1993), which may be useful (hence adaptive) or of no use whatsoever (hence maladaptive). The spectrum of behavioural actions runs from such disparate processes as producing a food-seeking response when levels of vital energy diminish, to the complex behaviours associated with acute injury and many on-going pain states. Behaviours can be considered as motor responses that alter movements and postures as well as producing the outward expressions of an emotional state (Holstege *et al*, 1996). Thus language and facial expression do much to convey feelings such as anger, fear or anxiety.
- The less obvious but highly complex physiological processes that are a necessary response to environmental and bodily changes/injuries. These also allow a chosen behaviour to occur or are the result of the behaviour.

The biological systems that may be involved in producing the behavioural or physiological response to any given threat to our homoeostasis include the somatic motor system, the autonomic nervous system (sympathetic, parasympathetic and enteric divisions), the neuroendocrine system and the immune system. Thus, physical injury may alter the activity of all these systems.

The young organism is naive, it has a relatively 'empty' CNS/brain in terms of environmental and physiological experiences (but see Mithen, 1996, and Plotkin, 1994). As the organism matures its CNS/brain 'fills up' with mindful and physiological experiences on which it can draw to aid in its quest for independent survival and reproductive success. In these terms, maturity is about getting to know the environment and learning how to act within it to bodily and genetic advantage. The maturation process is also about the brain getting to know its own survival machine and how to use it and look after it. Simply, this can be regarded as a progression from naiveté, where the CNS/brain houses only a few innate but vital sample-scrutinise-action pathways, to maturity, where layers of sample-scrutinise-action experiences are imprinted into the system and can be drawn upon later, if needed. Thus, as the organism matures it slowly gets 'filled' with meaningful new interconnections and pathways that can be considered the biological representations of past experience (Kandel *et al*, 1995). Physiological and environmental experiences are thus stored as implicit or explicit memories during learning and are capable of being recalled or remembered when needed (Gross, 1996; Rose, 1992). Along with physical maturation, learning, memory and recall in the broad body-based physiological as well as cognitive sense, are the fundamental biological processes that take the naive organism from being grossly dependent on its parents, to being fully independent.

Consider the first few moments of an acute injury (figure 2). Here, the CNS/brain receives information about the environmental conditions via the sensory organs and about the state of its tissues via sensory neural (eg nociceptor) and humoral (circulatory) pathways. It then proceeds to scrutinise the incoming information in order to provide an appropriate response. This may or may not involve the perception of pain. Part of the CNS/brain response/output may be to prevent nociceptive messages from impinging on consciousness (Fields and Basbaum, 1989, 1994). For instance, an injured person whose life is under severe threat is unlikely to feel any pain. The issues of fight or flight take priority over the perception of pain and its concomitant illness

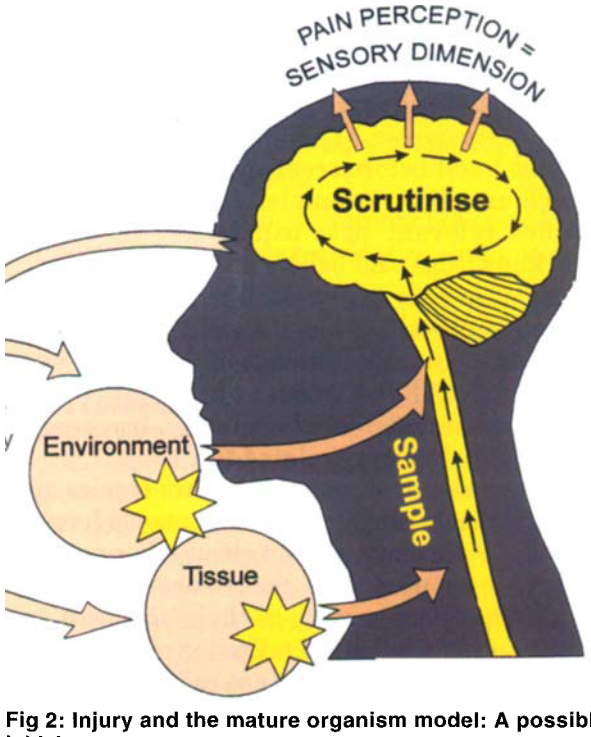

Fig 2: Injury and the mature organism model: A possible initial stage

behaviour (Gray, 1987). Thus, whether we feel pain or not is very variable and largely a product of the circumstances of the injury as assessed by our brain (eg see Beecher, 1946; Blank, 1994; Melzack *et al*, 1982).

Acute adaptive pain signals threat. Its major purpose, in parallel with the biologically linked emotional reactions like fear and anger, is to motivate and bring about an alteration in our behaviour in order to further our chances of recovery and survival (MacLean, 1990; Wall, 1979). Thus, acute pain from injury and the classic instantaneous behaviour patterns that are found across all cultures may be viewed as being 'adaptive', biologically well ingrained in our systems, and hence difficult to modify consciously. Later on, pain helps us to be become physically vigilant and avoid use of the injured part, our whimpering and distress attracts support from others, and our general demeanour demands care and respect from anyone venturing too close without undue care (Walters, 1994). Pain adaptively drives recuperative behaviour (Wall, 1979).

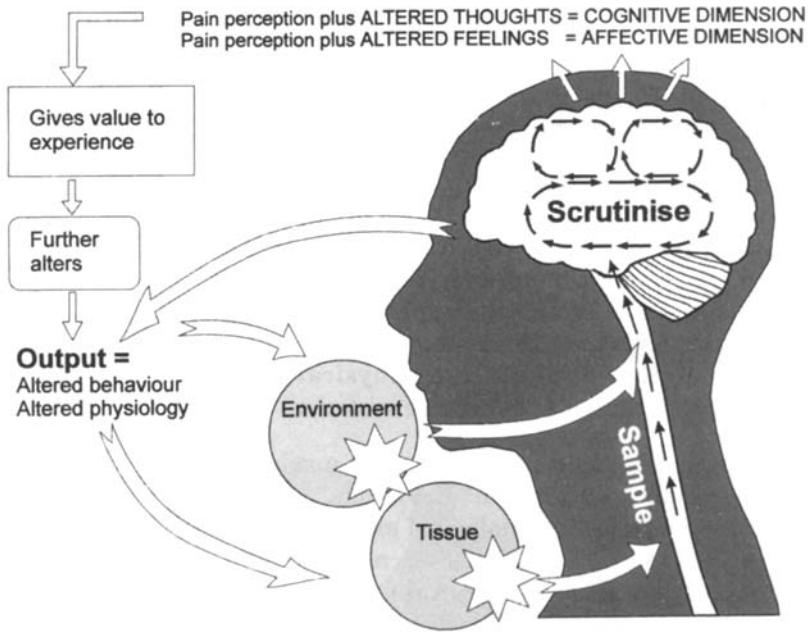

Fig 3: Injury and the mature organism model, showing the brain sampling itself and how the contents of our brains that represent such attributes as experience, beliefs and culture will influence the output system activity

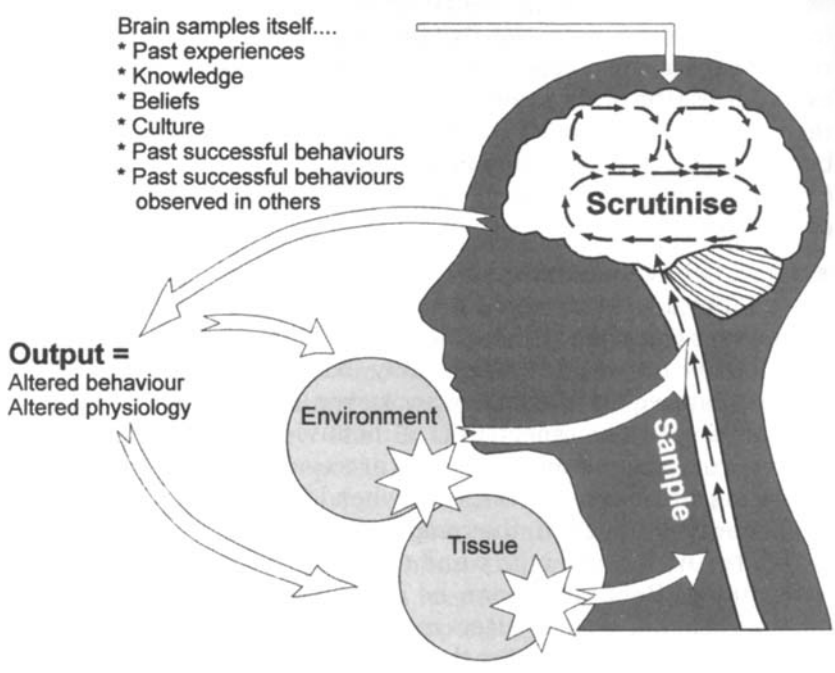

Fig 4: Injury and mature organism model: As a result of tissue sampling, environment sampling and self sampling, the brain/CNS produces appropriate thoughts and feelings. These perceptual 'outputs' of the brain give value to the injury experience and hence further influence the activity of the physiological and behaviourally related output systems involved in survival and recovery

Figure 3 adds another component to the model that introduces the possibility of a degree of flexibility of response. The brain samples itself before creating a behaviour. For example, it samples relevant past experience, knowledge and beliefs and mixes this in with its appraisal of the current situation. This sampling includes knowledge of past successful behaviours in similar situations, as well as successful behaviours related to us or observed in others. Adventure stories and the rather sickening desire many people have to investigate accidents or read about other people's mishaps may well have great survival advantages! What people store in their brains' filing cabinets of experience is a reflection of the culture and society in which they were raised, and their relative age and life experiences. A mature organism has a large number of behavioural strategies to choose from. It is worth reflecting that as a result of the great variety of options provided by modern complex societies the more difficult it becomes for individuals to make a secure choice – doubt promotes anxiety. The message is that along with the powerful effects resulting from the inputs being sampled and reflexly scrutinised from the damaged tissues, our current thoughts and feelings about the situa-

tion that we find ourselves in, as well as the thoughts and attitudes of those around us, will all have a marked influence on the degree of pain, our illness behaviour and the level of suffering (see Fordyce, 1986; Jensen *et al*, 1991; Skevington, 1995; Turk, 1996).

Figure 4 highlights the importance that the current thoughts and feelings a person suffering pain may have on the outputs of the brain. Most therapeutic approaches usually consider pain in a single *sensory dimension*. That is, the perception of where the pain is located, the quality and type of pain, its intensity and the way it behaves over time. However, pain has been considered in terms of three dimensions for many years (see Melzack, 1986; Melzack and Casey, 1968): That is, the *sensory dimension* as described; the *cognitive dimension,* which recognises that pain alters our thoughts, and the *affective dimension* recognising that for every pain we have there is some kind of emotional reaction. Following injury, or any noxious event, people's feelings or emotions and thoughts about their situation will change. Injury and the consequent pain may produce powerful aversive feelings of fear, anxiety or increasing anger (see Chapman, 1995; Fernandez and Turk, 1995; Panksepp *et al*, 1991).

The realisation of injury and the perception of discomfort also produces thoughts about the incident. For example, some patients may start to allocate blame, others may show concern for their immediate management or their prior plans.

These three dimensions represent three levels of integrated higher neural processing relating to consciousness that are ultimately responsible for an individual's behaviour pattern. Clearly, thoughts and feelings about a given situation are the fundamental processes that give it value. Value means that individuals (or their brain/CNS) view the experience as important, that something must be done, and that the experience is worth giving attention to, focusing on (Wright, 1994), and remembering for possible survival stratagies in the future.

Emotions are vital to providing experience with value and motivation (Damasio, 1995; Melzack and Casey, 1968) and are largely determined by our thoughts and beliefs as well as being reflexly triggered in novel and unexpected situations (LeDoux, 1993, 1994). Emotional centres in the limbic brain are powerfully linked to the areas of the major brain output systems – for example, the neuroendocrine system via the hypothalamus and pituitary glands; the sympathetic system via the hypothalamus and locus ceruleus in the brain stem and the somatic motor system via the motor cortex (Brown, 1994; Chapman, 1995; Chrousos and Gold, 1992; Chrousos *et al*, 1995). The powerful links between the neuroendocrine and sympathetic systems and the immune system are also well recognised (see below). The important clinical implication is that if we can positively change the way people feel emotionally, by for instance changing their knowledge and beliefs about their problems or situations, we can beneficially change activity in the output systems (Bandura *et al*, 1987; Bandura *et al*, 1985; O'Leary *et al*, 1988). This does not just mean bringing about changes in observable behaviour but also changes in autonomic, neuroendocrine and immune activity. The organic basis of 'mind over matter' is very much a scientific reality (for excellent overviews see Martin, 1997; Sapolsky, 1994; Sternberg and Gold, 1997).

Integrating Pain Mechanisms into the Mature Organism Model

The need to integrate an analysis of pain mechanisms into standard physiotherapy clinical reasoning processes and diagnosis has been proposed (Butler, 1994; Gifford and Butler, 1997). Mechanisms of pain consider the underlying biological processes involved either in creating the sensation of pain or in augmenting it. Three types of mechanism can be identified, relating to the sample-scrutinise-act/respond loop proposed in the MOM (Gifford, 1998):

- Mechanisms relating to CNS 'input' or, in terms of the MOM, to the CNS/brain sampling systems, ie nociceptive, peripheral neurogenic, humoral and immune (via circulation). The environment is also sampled via the major sense organs.
- Mechanisms relating to processing/scrutinising within the CNS/brain, ie central pain mechanisms/altered central processing and the influence of thoughts and feelings on pain perception and the outputs of the brain.
- Mechanisms relating to output of the CNS/brain: autonomic, motor, neuroendocrine and immune.

A fundamental concept that has sprung from the scientific unravelling of chronic pain neurobiology is that pain mechanisms move with time (see Gifford, 1997; Gifford and Butler, 1997). However, in viewing the issues relating to pain mechanisms from the perspective of the MOM it is apparent that in any situation, acute or chronic, physiological processes involving the tissues, the activity of the sampling systems (eg nociceptor firing), the brain scrutinising systems, and all the output systems, will have a role to play. Even though tissue damage and subsequent nociceptor activity can be seen as a dominant mechanism in acute pain it should be appreciated that psychosocial factors have a powerful role in deter-

mining and modifying the implicit physiological outputs and explicit behavioural patterns that are such an important part in recovery.

In the chronic pain situation, similar sampling-scrutinising-output analysis applies, it is just that the pathobiological pain mechanism focus shifts from the tissue and nociceptor mechanisms in the periphery to focus more on maladaptive and widespread reactivity and sensitivity of the whole sampling-scrutinising-output systems (Gifford, 1997a, 1998, Gifford and Butler, 1997). It is fundamental that we begin to accept that the status of the tissues as a 'source' of the pain, even though still reactive to mechanical testing, is far less relevant as time goes on. The following issues are seen to be important considerations in the analysis and management of on-going pain.

Alterations in Tissues and Tissue Sampling Systems

1. The relevance of perceived and/or clinically extracted abnormalities in tissues that are painful or that may traditionally refer to the area of pain must be questioned. Abnormalities in tissue sensitivity, anatomy and mechanics are best analysed in terms of physical dysfunction that may or may not be relevant to the pain condition but may be well worth addressing in the overall management of the individual (Gifford, 1997; Gifford and Butler, 1997). Physiotherapists must confront the problem of 'if you look you will find' when assessing tissues in on-going pain states (Gifford, 1997; Gifford and Butler, 1997; Loeser, 1991). This means that clinicians will always find something wrong in the tissues, but it may not be relevant to the patient's problem and focusing on it may divorce the patient from the full extent of the problem.

2. Maladaptive on-going sensitivity/activity of the sensory nerve terminals in the tissues. There may be little wrong with many chronically sensitive tissues.

3. Maladaptive on-going sensitivity of peripheral nerve trunks and their contained neurones (Devor, 1994).

Alterations in Processing in the CNS/brain

1. Maladaptive processing in the CNS/brain and the potential for a pain memory (see Basbaum, 1996; Gifford, 1997; Gifford and Butler, 1997; Katz and Melzack, 1990; Melzack, 1996). The important concept here is that the neural perceptual correlates of a specific pain and its associated emotional content may become imprinted in unique CNS pathways in ways not unlike those thought to produce long-term memory (Lenz *et al*, 1997; Pockett, 1995). The fact that once an experience is 'imprinted' it may be very hard to remove (Connolly and Tully, 1996) sheds some light on the reasons why so many therapies and surgical procedures for relieving chronic pain have such poor outcomes (Deyo and Phillips, 1996; Melzack and Wall, 1996; Wall, 1996). This also highlights the need to shift the therapeutic focus from pain relief to functional restoration.

2. The unhelpful or maladaptive thoughts and feelings a patient may have occur not only as a result of on-going pain and the increasing loss of function, but also due to less tangible aspects such as mismanagement by medicine and other primary care clinicians. This includes physiotherapists, osteopaths and chiropractors (Deyo, 1993; Deyo and Phillips, 1996; Loeser, 1991; Loeser and Sullivan, 1995; Pither and Nicholas, 1991; Zusman, 1997a) as well as the effects of family, work and society in general (Nicholas, 1996; Skevington, 1995; Turk, 1996).

Altered CNS/brain Outputs

The possible maladaptive CNS/brain outputs are produced as a result of the ongoing unpleasantness and general negative psychological and physiological aspects associated with ongoing pain (Agnati *et al*, 1991; Crofford and Demitrack, 1996; Stratakis and Chrousis, 1995). These include:

1. *Neuroendocrine.* For example, chronic perturbations of stress hormone systems may slow or blunt tissue recovery responses and enhance a patient's sensitivity to pain (Devor, 1994; Pennisi, 1997; Sapolsky, 1994; Sheps *et al*, 1989, 1990; Vander et al, 1990; Weiner, 1991).

2. *Raised autonomic tone levels.* Increased sympathetic tone is a common component of a pain state, especially if the pain is perceived in a worrying, threatening or emotionally charged way (Chapman, 1995).

3. *Diminished immune responses* that relate to negative emotional states (Dunn, 1995; Felten, 1991; Kiecolt-Glaser and Glaser, 1991). The immune system is innervated by the sympathetic nervous system (Arnason, 1993; Watkins, 1994) and is negatively influenced by it and the neuroendocrine system during emotionally stressful states (Dunn, 1995; Martin, 1997; Stein and Miller, 1993; Stein *et al*, 1991).

4. *Abnormal movement patterns* and unhelpful general-health behaviours that include the detrimental tissue consequences of prolonged lack of use (eg see Bortz, 1984; Buckwalter, 1995; Troup and Videman, 1989; Twomey and Taylor, 1984).

5. *Alterations in normal descending inhibitory currents* that are involved in the gating of incoming sensory information (Melzack and Wall,

1996). For example, individuals who are in pain, and especially those without an adequate explanation or understanding of their pain, may well focus unduly on it and thus maintain habitually open pain 'gates' which would otherwise be held closed (Gifford, 1997; Gifford and Butler, 1997; Klaber-Moffett and Richardson, 1995). Thus focusing, repeating and giving attention/value to an experience can promote learning and altered CNS/brain processing (Byl and Melnick, 1997).

Implications for Clinicians

Many issues have already been raised. It is hoped that the MOM provides a broader perspective on the analysis of pain states and also appeals to clinicians to give attention to psychosocial components (Turk, 1996; Waddell *et al*, 1993). In this respect, many aspects of the cognitive-behavioural model for chronic pain are worthy of our attention (see Harding, 1997; Harding and Williams, 1995) and it has been demonstrated to be effective (Flor *et al*, 1992). The following points are also pertinent:

- It should be recognised that the perception of a problem and the outputs of the brain that direct our behaviour are not solely determined by tissue damage and physical dysfunction. Maladaptive alterations in peripheral and CNS processing can account for on-going pain and enhanced sensitivity states in tissues that may have never been injured or in those that have been injured but have healed as completely as possible.

- We need to appreciate fully that negative psychological states limit the efficiency of physiological CNS/brain output systems that govern tissue health, control recovery and maintain homoeostasis. It is becoming ever clearer that positive psychological states combined with healthy lifestyles and behaviour have the opposite effects (eg Felten, 1991; Martin, 1997).

- Many on-going pain sufferers have not had their problems properly validated by medicine. For example, work related upper limb disorders (WRULDS) and whiplash are labels that are not generally respected and, like myalgic encephalomyelitis (ME), are not nice to have. Lack of 'physical' validation is the cause of much suffering. This is supported by the title of an article in the journal *Spine*: 'If you have to prove you are ill, you can't get well' (Hadler, 1996). The point is that there is a need for more efficient diagnostic strategies (Deyo and Phillips, 1996) and skills. Labelling specific tissues as responsible for on-going pain states holds physiotherapy in the traditional medical disease model that perpetuates the public's belief that if something is physically wrong it can be fixed. The integration of the clinical analysis of mechanisms of pain (Gifford and Butler, 1997) within the conceptual model proposed is possibly a good place to start the re-education process – for us, and ultimately for our patients.

Education about pain that includes the modification of commonly held 'abnormal structure/mechanics' related beliefs about pain is seen as vital to successful rehabilitation and outcome (eg see Hildebrandt *et al*, 1997). The MOM drawing may be a useful tool to use in helping explain the nature of pain and its repercussions on the individual. Many patients accept that pain alters their mood states and that this causes them to adopt behaviours and activities that are largely determined by the pain and which are not conducive to general health or the health of the tissues where the pain is felt. On-going pain states are best explained to patients in terms of an altered sensitivity state as a result of altered information processing throughout the system, and not solely a result of damaged and degenerating tissues. This helps patients accept the notion that hurt does not necessarily equate with harm – which leads on to the positive message that carefully graded increases in physical activity mean stronger and healthier tissues. This is reinforced when patients achieve improved physical function. By contrast, continued focus on a tissue as the pain source reinforces fear of movement and activity, the need to be constantly vigilant for pain and the desire for increasingly expensive passive therapeutic interventions that are yet to demonstrate convincing efficacy (eg see Waddell, 1996; Waddell *et al*, 1993).

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