5

Pain

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Introduction

The main thrust of this chapter is to help the student understand the nature of pain and realize that the diagnosis of pain states is far from being an accurate and agreed upon science. Recent advances in our understanding of pain are powerfully challenging much of current medical practice. This includes the part played by physiotherapy and other closely allied professions that deal with noninvasive forms of pain therapy and management. If physiotherapy can take on pain, understand it more clearly and what it means for the individual and society, then the profession's future place in its management will be assured a very significant role.

Chapter Objectives

After reading this chapter you should be able to:

1 Understand and see the weaknesses of the terms organic/nonorganic.
2 Discuss and understand the three pain dimensions: the sensory dimension, the affective dimension and the cognitive dimension. The

student should be able to relate the pain dimensions to all pain experiences and understand that pain's primary purpose is to influence 'behaviour'.

3 Integrate pain into basic concepts of stress biology:
   (a) Normal pain is generally an adaptive perceptual message generated by the brain in response to tissue damage. Pain does not necessarily occur at the time of injury.
   (b) Ongoing pain is adaptive if it serves a protective function but maladaptive if healing is complete and the symptoms are way out of proportion to the stimuli that provoked it.
   (c) Pain alters psychological/mental function and this markedly alters brain output systems.
4 Discuss the current controversies underlying the established pain pathways and the misguided notion that there is a single pain perception centre in the brain.
5 Appreciate the relevance of the pain dimensions to acute and chronic pain.
6 Understand the concept of dysfunction/altered function and relate it to:
   (a) The patient in pain;
   (b) The weakness of many modern therapy models.
7 Understand that there are multiple physiological mechanisms underlying the perception of any given pain and that the major ones in current clinical use by some physiotherapists are:
   (a) Nociceptive mechanisms;
   (b) Peripheral neurogenic mechanisms;
   (c) Central mechanisms;
   (d) Output mechanisms – sympathetic, motor, neuroendocrine;
   (e) Affective/cognitive mechanisms.
8 Understand the biological principles underlying the pain mechanisms and relate the processes to clinical presentations.
9 Appreciate that there are many controversial issues in ascribing pain solely to a particular tissue or structure.
10 Understand the terms 'allodynia' and hyperalgesia and the difficulties of their clinical interpretation.
11 Understand the weaknesses of therapeutic approaches to pain that involve targeting a presumed 'source' of the pain and that use wholly passive techniques.

Organic and Nonorganic Pain?

The notion that pain is a pure sensation akin to taste and smell has received much derision in recent times (Wall, 1989; Melzack and Wall, 1996). Most of us tend to think of pain as an unpleasant, distressing sensation that originates in traumatized tissues and courses its way along neural pathways to the brain and consciousness. Thus, the amount of pain perceived fits with the amount of damage done and the pain happily recedes in direct relation to the pace of healing.

The problem is that our clinics and departments are full of patients who have ongoing pain with no clear trauma or disease process, or who have suffered trauma but the pain continues on long after a reasonable healing period. Often there is a huge discrepancy between the amount of pain perceived and evidence of any reasonable tissue abnormality with which to equate it. Time and again this very discrepancy has promoted the adoption of a two-tier diagnostic model that divides patients into 'organic', where observable pathology equates with the pain, and 'nonorganic'

or 'psychosomatic' where no such relationship exists (Waddell *et al.*, 1980; Waddell and Turk, 1992; Long, 1995). Put simply this often equates to: organic = 'we believe you, have an operation/pill/manipulation to fix it'; and nonorganic = 'you are making it up, you need to see the psychologist/psychiatrist'.

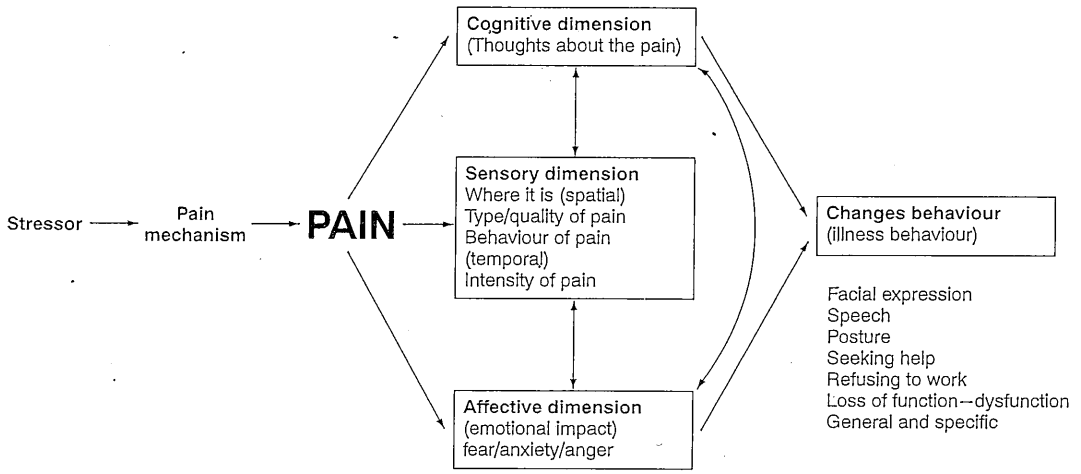
Since it is frequently noted that in only 15–25% of patients with low back pain an accurate diagnosis can be established (Nachemson and Bigos, 1984; Spitzer and LeBlanc, 1987; Deyo *et al.*, 1992), the rather unsettling conclusion is that medicine is directly or indirectly denying the honesty of the vast majority of patients (Loeser, 1991). The tragedy of this attitude combined with the inadequacies of current diagnosis for pain, is that patients can become disillusioned and unhappy, often angry, and enter a rather forlorn pilgrimage that takes them from specialist to specialist and from therapy to therapy. They are given multiple diagnosis and get multiple forms of advice (Ochoa *et al.*, 1994) that add to the confusion and which may be a major factor in making their problem a lot worse (Morris, 1991; Loeser and Sullivan, 1995).

The Three Dimensions of Pain: Sensory, Cognitive and Affective

One of the key ways to understand pain and the patient's experience of pain is to view it in three interrelated dimensions (Melzack and Casey, 1968; Melzack, 1986) and never as a single 'sensory' one (Figure 5.1).

Unless we are under fairly severe physical threat or extremely focused on something, most of us, if we twist our ankle or sprain a finger, generally feel some pain at the time of the incident which goes on for some time afterwards. We very quickly become aware of where the pain is coming from, how intense the pain is, how the pain is behaving over time and the type or quality of the pain. This is the 'sensory' dimension of pain and the dimension which most physiotherapy assessments focus

Figure 5.1 Three dimensions of pain (see text for details). (Reproduced, with permission, from Butler and Gifford 1998 The Dynamic Nervous System. NOI Press, Adelaide)



on when filling in our body charts and asking questions during the subjective examination. However, pain to a lesser or greater degree alters the way we think, the 'cognitive' dimension of pain, and the way we feel, the emotional or 'affective' dimension of pain.

Thoughts (the *cognitive dimension*) might involve some assessment of how bad the damage is, what to do about it and alterations of planning for work and recreation, for example. Individual thoughts vary greatly, for example: 'I better seek help from my nearest physiotherapist/acupuncturist/healer'; 'I better go to bed for two weeks'; 'I'll ignore it and get on with my plans and see how it goes'; 'I think I have broken it, I'm not going to move until the ambulance arrives'; 'Last time I did this it took 4 months: this isn't looking good at all'; 'Oh great I can take a week off work'. Even in acute pain there is a great variability in individual thought processes that may have marked repercussions on outcome. A simple cognitive spectrum has at one end a stoical attitude that ignores the problem and makes light of it and at the other extreme the catastrophizer who just sees the downside of it all and appears, at least to some, to rather revel in the drama.

The *affective dimension* of pain recognizes that for every pain we have, an emotional reaction is expressed that is fundamental to the pain experience and not just a reaction to the sensory appreciation of pain (Chapman, 1995). If you strike a dog it will either yelp and run, or bare its teeth and possibly even bite you. In biological terms threat and the pain message generally produce very powerful aversive behaviours that adaptively serve to protect the threatened organism. In human terms (and probably in higher vertebrates too; Dawkins, 1993), threat is strongly associated with aversive *feelings* of fear and anger which promote adaptive behaviours that are ultimately protective in function and powerfully serve to enhance individual and species survival (Gray, 1987; Panksepp *et al.*, 1991; Chapman, 1995). The immediate discomfort of a twisted ankle is likely to produce some sort of emotional response like anger, mild annoyance, worry and anxiety. Most responses to pain involve some kind of unpleasant emotion that is encompassed in the terms 'psychological distress' (Main *et al.*, 1992) and 'suffering' (Cassell, 1991).

A simple spectrum way of viewing often complex affective dimensions is a scale of emotional impact that has at one end modest psychological distress and at the other extreme a status of clinical depression. Thus, a twisted ankle may invoke mild concern or moderate anxiety while a sudden sciatic pain for no apparent reason may be far more distressing. Ongoing severe pain combined with marked disability and loss of function may be a severe test of an individual's coping capability and may easily lead to ongoing maladaptive emotional states. Just as thoughts about pain alter our behaviour so too do the emotions compel action – we shout, we moan, we grimace, we chastise, we change our posture often in quite dramatic ways, all of which conveys powerful meaning to others in that it summons assistance and support, in particular from those who are closely related. For instance, witness the powerful support given by a parent to their injured and emotional child.

Figure 5.1 illustrates the three pain dimensions and highlights their main objective, that is, to change our behaviour in order to promote restoration of function (MacLean, 1990). What we are ultimately looking at is a coping strategy that is as unique to an individual as his or her physical features are. Biologically, the injury message can be viewed as a signal of threatened homeostasis that kickstarts

adaptive *physiological* and *behavioural* coping mechanisms in order to promote survival.

Behavioural changes involve alterations in movement patterns and great vigilance for the part concerned. The idea is to care for the part that is injured and hurting as well as to keep others who are likely to damage it further well away from it. Thus the pain of a twisted ankle may compel limping and restricted range of movement as well as extreme guarding involving verbal and physical demonstration to nearby others. So often a patient can happily touch an acutely injured area themselves, but note the change of facial expression, the protective reactions and the enhanced tissue sensitivity when under physical examination, especially when the examiner has done little to gain the trust of the patient. Everyone's behaviour in a given situation is a unique interaction of innate reactions modified by the experiences our upbringing and culture imposes upon us (Gray, 1987; Gross, 1992). Everyone behaves differently and we must be prepared to try and adapt to each individual.

Pain and Stress Biology

While the three dimensions of pain are seen as part and parcel of every pain experience it should also be clear that each dimension must interact with the others (Figure 5.1). For example, negative or 'unhelpful' thoughts about the injury and the pain promote unhelpful emotional responses that then promote the arousal of the autonomic and neuroendocrine axes which in turn promote noxious sensory responses from the tissues and neurones responsible for the mechanisms of the pain (Figure 5.2).

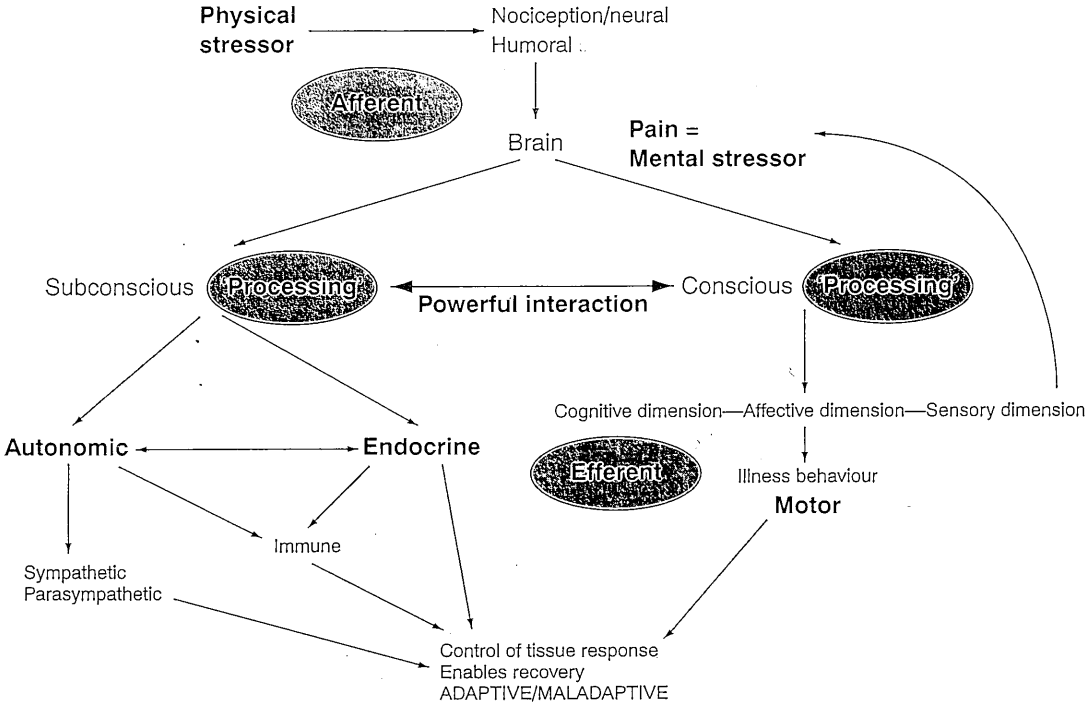
Figure 5.2 is an attempt to simplify the main input, or afferent systems, and the main output, or efferent systems, involved in the central 'processing' of stressors associated with pain and to demonstrate their powerful interactions. The term 'stressor' is used in the sense of any real or perceived threat to homeostasis, and 'stress reaction' or 'stress response' as the body's adaptive counterresponse to it (Levine and Holger, 1991). Stressors, like a twisted ankle, may be seen as resulting from environmental factors outside the body, i.e. exogenous stressors, or, like some disease processes, resulting from endogenous factors that originate from within the body. Endogenous stressors must include mental/cognitive 'stress' since negative thoughts and emotions very powerfully influence the activity of the stress response (Goldberger and Shlomo, 1993; Sapolsky, 1994).

Witness how you feel when you see something disturbing on the television, or think of something that has upset you in the past, or have ongoing unexplained pain. Typical signals of anxiety like pounding of the heart, a queasy stomach and a loss of appetite are powerful examples of how the 'psyche' can influence the body – this is termed a 'psychosomatic' reaction in the stress literature (Weiner, 1991). Note how the proper biological use of this term is *not* in the derogatory sense that it is so common.

The crucial message here is that our thoughts and feelings strongly influence biological processes in the tissues of the body. This has powerful repercussions for the way in which we manage the patient in pain, especially chronic pain where negative/unhelpful thoughts and feelings often dominate the patient's lives.

At the present time it seems that the scientific disciplines of stress and pain biology have hardly met each other, yet the links are fairly clear and

Figure 5.2 The afferent and efferent systems related to physical stressors. (Reproduced, with permission, from Butler and Gifford 1998 The Dynamic Nervous System. NOI Press, Adelaide)



help greatly in providing a much needed reinterpretation of our thoughts about pain and pain management.

Let us overview the afferent and efferent systems using the sprained ankle as an example. The primary stressor consists of the physical forces acting on the ankle joint and its associated structures. The first adaptive reaction is flexor withdrawal response brought on by an impressively quick and complex sensory/afferent – motor/efferent reflex neural response (see Chapter 3). At the same time tissue trauma afferent messages are relayed via nociceptive systems to the brain whose processing may invoke a conscious appreciation of the stressor (as pain) as well as activating complex 'subconscious' brain compartments that invoke appropriate outputs to counter the stressor.

The three major output systems are:

1 The autonomic (sympathetic and parasympathetic nervous systems);
2 The neuroendocrine systems;
3 The motor systems.

All three are influenced by the way we are thinking and feeling. The degree to which these systems are activated vary greatly and depend to a large extent on the degree of perceived threat. Thus, their activation is greatly enhanced if the twisted ankle occurred at the same time as a loud and unusual noise compared to just quietly walking along a street. This again emphasizes the powerful link between the way we are thinking and feeling –

the mind/our cognition – and the activity of systems that powerfully influence the tissues of the body. These two situations also serve to demonstrate how the stress response may activate the very powerful stress-induced analgesic systems. Injury in the presence of acute threat (such as at the same time as a very loud noise) may not produce any feeling of pain at the time, as pain would merely hinder any physical activity needed to escape the threat (McCubbin, 1993; Blank, 1994; Fields and Basbaum, 1994).

Note how input to the brian involves two routes, one via the nervous system which is fast (and hence evolutionary advanced), and the other more primitive route via the bloodstream (humoral) which is far slower. Damaged tissues produce chemical messengers like the cytokines and other inflammatory mediators that are thought to communicate with the brain via the bloodstream (De Souza, 1993; Rivier, 1993; Udelsman and Holbrook, 1994; Watkins *et al.*, 1995). Input about injury also involves the ears, eyes and occasionally smell, and their afferent pathways to the brain.

Nociceptive pathways or wiring diagrams have been described in great detail and the reader is directed elsewhere for fuller information (see Willis, 1985; Charman, 1994). However, there are two interesting aspects that arise from the pioneering work of Ronald Melzack (Melzack and Casey, 1968) and the comments of his long time associate, Patrick Wall (Wall, 1996a). Melzack and Casey (1968) assigned two separate anatomical pathways to the sensory and affective dimensions of pain; that is, the lateral neospinothalamic (neo = biologically 'new' and hence fast) pathway that relays from the nociceptor terminal synapses in the dorsal horn, across the cord and ascends to nuclei in the thalamus before being relayed on to the somatosensory projection areas of the cortex. This pathway was assigned to the sensory dimension of pain described earlier.

The affective dimension was assigned to a more medial multisynaptic pathway that courses from the dorsal horn up to the brainstem reticular formation and on to the limbic nuclei that perfuse the brainstem and areas of the more primitive cortex. These areas of the brain are powerfully linked to centres involved in emotional feelings and to the activation of their associated reflex behaviours (Damasio, 1995). In parallel there are also links to seats of sympathetic efferent activity, like the locus coeruleus nucleus in the brainstem, and to seats of endocrine hormone activity via the hypothalamus and its links to the pituitary gland (Chrousos and Gold, 1992; Johnson *et al.*, 1992; Valentino *et al.*, 1993). These subconscious nuclei/regions of the brain not only output to the body but are also capable of exerting a remarkably global influence on brain activity in general. There are thus very powerful links to and from higher centres associated with consciousness.

The key concept to hold on to is that any form of threat activates conscious and subconscious systems that are bidirectionally linked by known pathways and that each can influence the other (Chapman, 1995). Changes in behaviour and tissue repair processes require a coordinated physiological response to be successful.

The second issue, raised frequently by Wall (see Wall, 1996a, b), relates to the problems of viewing the nervous system and brain as a computer-like creature that is given its wiring diagram during development and from then on never changes. Wall tends to balk at the concept of dedicated pathways and regions in the brain (Wall, 1996b), like one each for sensory and affective component of pain. No one has managed to perform a

surgical lesion or focal application of a drug to a specific tract or area of the brain that can produce an isolated loss of affect or sensation with regard to pain (Wall, 1996a). Further, neurosurgical lesions along presumed pain pathways only produce temporary relief that is later followed by the ability to again generate a pain state. Tragically, cutting the so-called pain pathway or destroying a presumed pain 'centre' in the brain, just does not work and may often make the pain worse later on (Melzack and Wall, 1996). What this highlights is the complexity of the 'thing' we call pain and that there is not a simple dedicated pain pathway and pain appreciation centre. We are really looking at a hugely distributed system that involves the integration of many subsystems whose ultimate goal is the coordinated restoration of a homeostatic equilibrium sufficient to allow survival.

Basically it looks as if the whole of the brain is involved in pain. This makes sense if one views the brain as the primary 'stress control centre' and that injury and pain require the activation of many systems in order to promote survival. In viewing the pain as a component of a stress system/response, it is worth drawing attention to the fact that pain itself is a stressor, and on its own, without any necessary tissue damage or nociceptive activity (think of things like ongoing migraine headaches here), will activate a stress response to a greater or lesser degree depending on the significance our thoughts give to the pain.

The stress response thus has two components that are influential in determining its activity, a mental component and physical component, and they are inseparable (Figure 5.3).

Figure 5.3 Adaptive and maladaptive stress responses. (Reproduced, with permission, from Butler and Gifford 1998 The Dynamic Nervous System. NOI Press, Adelaide)

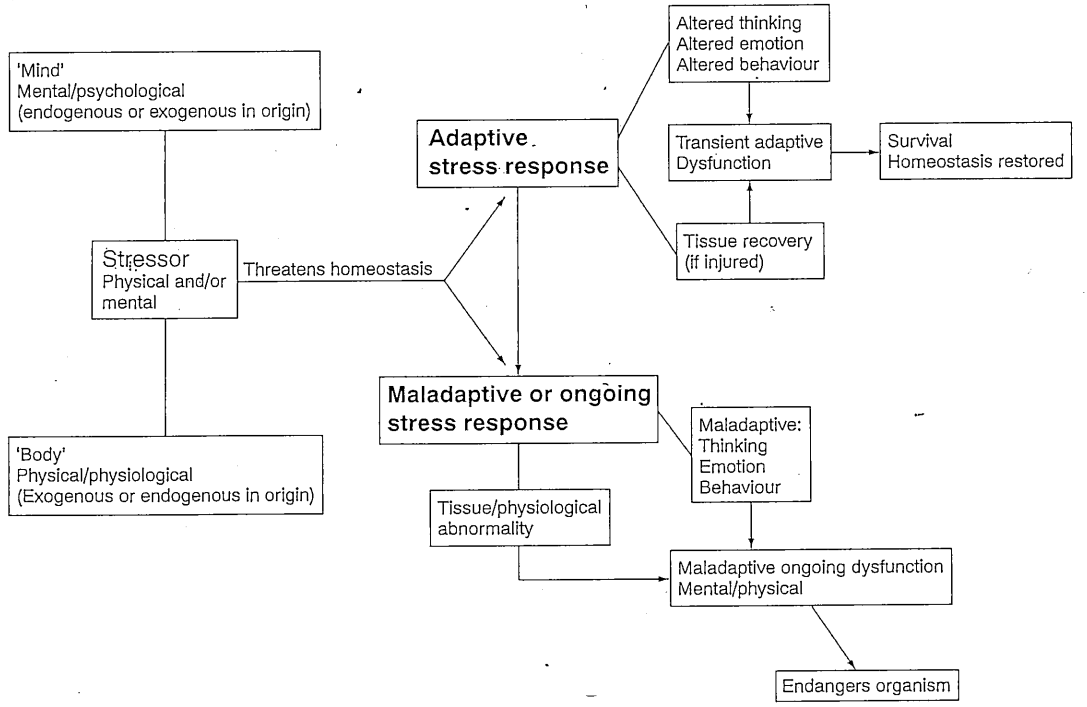
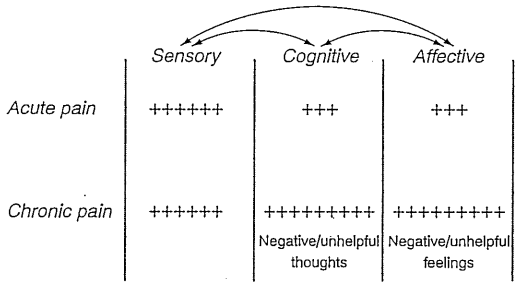


Figure 5.4 Demonstrating the relative importance of the dimensions of pain in the acute and chronic situation. All dimensions are variable. (Reproduced, with permission, from Butler and Gifford 1998 The Dynamic Nervous System. NOI Press, Adelaide)



Before discussing Figure 5.3, the reader's attention is drawn to Figure 5.4 because it highlights some of the great differences between acute and chronic pain in relation to the three dimensions of pain discussed. In acute pain the sensory dimension is fairly dominant – patients in acute pain visit our clinics and describe the pain in precise terms, can often easily identify physical factors that make it better or worse and in addition simply and quickly state their feelings and thoughts about it if given the opportunity. By contrast, in chronic pain the dominance of cognitive and emotional dimensions is profound when compared to the acute problem (Figure 5.4). Chronic pain sufferers can often keep talking for hours about the problems that they encounter, in themselves, in their family, at work and with medicine and their management. If you ask a chronic pain patient if they are upset or angry – the answer is invariably yes (see Fernandez and Turk (1995) for an excellent review).

The great mistake is to view pain, especially chronic pain, from the perspective of an isolated sensory dimension that sees all pain as an adaptive warning that damage is being done.

Much chronic pain is largely maladaptive and results in far-reaching disability. What it really needs is a rehabilitative approach whose primary aim is to restore physical function and at the same time addresses both the cognitive and affective dimensions of the patient's disorder (see Chapter 15; Gatchel and Turk, 1996; Turk *et al.*, 1983).

Briefly, this means educating the patient about the underlying mechanisms of their pain (see Chapter 15) and the influences their thoughts, emotions, attitudes and physical behaviour can have on it. Having then established a sound basis of knowledge that the patient can understand and relate to, the phase of 'mental', behavioural and physical rehabilitation can go ahead.

Figure 5.3 represents two hypothetical routes that can be taken as a result of some sort of event that can be deemed stressful and may include pain as one of the mental or psychological stressors. The two routes are:

1 The adaptive one, where function and homeostasis are happily restored;
2 The maladaptive one, where an ongoing problem occurs and which is ultimately a threat to the viability of the organism.

The adaptive stress-response route is the one most of us take when confronted with minor injuries and aches and pains. However, it is common for some maladaptive issues to creep in, especially with regard to our thoughts and feelings.

Pain and Altered Function: 'Dysfunction'

The interaction of our thoughts and feelings about a problem combine to promote altered behaviour while tissue recovery proceeds. During

this time we generally adopt a state of adaptive or maladaptive altered function which for the purposes here is loosely described as 'dysfunction' and divided into three subcategories (see Butler and Gifford, 1998). It is the author's view that along with pathobiological mechanisms (see below), dysfunction should be added and integrated into the five hypothesis categories put forward in the clinical reasoning model proposed by Jones (Butler, 1991; Jones, 1992) and further developed to include the pain mechanisms suggested by Butler (Butler, 1994). (Clinical reasoning is discussed at length in Chapter 6, but see also the proposals in Butler and Gifford, 1998.)

The three subcategories proposed are:

1 General physical dysfunction;
2 Specific physical dysfunction;
3 Psychological/mental dysfunction.

General Physical Dysfunction

This refers to limping, hobbling, stereotyped patterns of movement and posture, difficulty/inability to perform simple tasks like negotiate stairs, sit comfortably etc. It is anything a good subjective and objective functional physiotherapy assessment reveals and which by the World Health Organization definition would be termed 'disability' (WHO, 1980).

Specific Physical Dysfunction

This includes:

- a loss of range of joints, muscles and nerves as a result of increased tissue sensitivity, mechanical block/tightness, pure spasm, fear or a combination of these;
- Weakness due to neurological deficit, disuse, pain inhibition, fear and so forth;
- Symptoms/abnormal responses, e.g. excessive tissue tenderness to palpation (allodynia, hyperalgesia, see below), pain provoked at end of range, pain provoked in specific test positions;
- Instability/muscle imbalance;
- 'Other', e.g. deformity, leg length discrepancy, tissue thickening, tissue thinning/wasting.

This is the area where physiotherapy excels, i.e. finding specific dysfunction. However, we should be cautious and a little wary of adopting what may be a pseudo-diagnostic approach. Pain has generated many very enthusiastic specialists in specific techniques of treatment and analysis. Manual therapy is one such approach. If you look closely, there are subspecialities within manual therapy – some practitioners focus on the cranial sutures, others on reflex areas on the feet, or the sacroiliac joint position, or muscle imbalance and tightness, for example. The list could spread to include electrotherapy and many different surgical approaches. The statement 'if you look you will find' should alert us to the dangers of dogmatic single-model approaches to pain states. This is especially true in the analysis of specific physical dysfunction in chronic pain (see Loeser, 1991).

It is important to note that we are all full of dysfunctions whether or not we are in pain. If we are in pain it is easy to find something wrong relevant to a precise tissue model but which may not be relevant at all to the patient's pain state. While everyone wants to know what is wrong, there are dangers from those who obsessively bias their models.

Pain science forces the broadening of our views about pain states. A good example is a patient with a 4-month-old sciatica who goes to see five or six different therapists and comes away from each with a different diagnosis and different

advice. He may well have 'cranial suture abnormalities', 'leg length discrepancies' and 'sacro-iliac upslip', problems with 'lymph drainage', loss of 'passive accessory movements' in his lumbar spine, 'adverse neural tension', 'muscle imbalance', and so on. But this is just a list of specific, and rather equivocal, physical dysfunctions which may or may not be relevant to the underlying pathobiological mechanisms that are giving rise to the pain state.

Psychological / Mental Dysfunction

This category of dysfunction recognizes the importance of the cognitive and affective dimensions of pain and their fundamental role in the production of suffering and maladaptive physical dysfunction. Current stress biology and the relatively new scientific disciplines like psychoneuroimmunology are demonstrating and emphasizing the powerful links between negative or unhelpful thoughts and feelings and negative tissue physiological effects (Ader *et al.*, 1991). If we can helpfully change patients' beliefs, thoughts and feelings about their problems the recovery will be vitally enhanced. Although traditionally this area is the domain of clinical psychology, physiotherapists who specialize in managing pain can with guidance and proper training effectively help these components of many patients' disorders. If pain is multidimensional then so should its management be – whether we are dealing with chronic or acute pain. The majority of patients in pain need more information about their problem and what to do about it. A good deal of patients' needs are met when they are given understandable answers to the following fundamental questions (see Butler and Gifford (1998) for full discussion):

1 What is wrong with me?
2 How long will it take to get better?
3 Is there anything that I can do to help?
4 Is there anything that you can do to me or give me that will help me?

Pathobiological Mechanisms: Pain Mechanisms

In the rest of this chapter the focus will be on some of the pathophysiological or pathobiological mechanisms that can give rise to pain and produce physical and psychological / mental dysfunction. This is important since most current diagnostic systems in medicine and physiotherapy are based on ill-founded anatomic or mechanistic labels that focus the patient's and clinician's attention on a damaged structure which is in need of some kind of passive therapy or passive intervention to fix it (Loeser, 1991; Loeser and Sullivan, 1995). As noted earlier there is little pathological / structural / anatomical evidence to be found that equates with the degree of pain in the great majority of patients suffering with ongoing pain. There is also plenty of evidence to show the continued presence of apparently blameworthy anatomical abnormality long after the resolution of pain (for example see Garfin *et al.*, 1991). Current pain biology draws our attention to the need for an inclusion and analysis of pain mechanisms into a diagnostic clinical reasoning hypothesis.

The refreshing aspect of this approach is that the inclusion of maladaptive central nervous system afferent and efferent processing as a means of generating pain independently of any tissue or peripheral nerve abnormality, means that many of our patients' more atypical pains and pain behaviours can more easily be explained. This is

helpful to those of us who deal with them and hence to the poor patient who up to now has been largely denied a reasonable organic basis for their very real pain. At long last many complex regional pain syndromes that have previously been considered as hysterical 'conversion' disorders are now rightly being considered as neurological disease states (Janig, 1996). The answers to understanding pain surely lie in a far broader understanding of pathobiological mechanisms that emphasize the significance of the central nervous system rather than the anatomical aberrations of its target tissues.

Pain Mechanisms

There are five recognized pain mechanism categories in the clinical reasoning model used by some manual therapy systems today (Butler, 1994; Jones, 1995).

These are:

1 Nociceptive mechanism;
2 Peripheral neurogenic mechanism;
3 Central mechanism;
4 Sympathetic/motor mechanism;
5 Affective mechanism.

Each term relates to a physiological process that can give rise to pain jointly or in isolation. Some mechanisms stand out as being far more dominant in a pain presentation than others.

NOCICEPTIVE MECHANISM

This mechanism represents pain at its physiologically simplest. The pain is coming from roughly where it is felt, or at least from where tissue damage and inflammation are stimulating and provoking nociceptors to transmit impulses that produce the perception of pain in the brain. Prevent the nociceptive messages from reaching the central nervous system by inhibiting inflammation, or by blocking the nerves supplying the injured area and the pain slowly or rapidly vanishes. Nociceptors are specialized afferent neurones of two basic types, fast myelinated Aδ fibres and smaller, slower unmyleinated C fibres (Fields, 1987). It is the Aδ fibres that are responsible for the flexor withdrawal reflex, the C fibres are much too slow. In fact, if one were to stick a pin in the foot of a horse a C fibre would relay the event to the spinal cord in about 8 seconds (Wall, 1989) – an event which is much too slow to prevent injury.

In the normal state nociceptors will only fire if noxious or near noxious stimuli are presented to them. They are thus said to be 'high threshold' afferents. This means that if a therapist performs an end-range stretch on a normal tissue, nociceptors will begin to fire and the recipient will start to frown and become ever more vigilant as the therapist slowly pushes harder and harder. In most people, the amount of discomfort/pain is consistent and in proportion to the force. Some people are obviously more sensitive than others but there is a recognizable limit to what one would expect under normal conditions before thinking that something may be wrong. It could be useful to put people on a pain-response scale with feeble at one extreme and stoical at the other – *so long as it does not colour your view of that person.* Appreciate that everyone is different because their sensitivity is a result of interaction of genes and environment.

A further consistent feature of normal nociception is that once the pressure is released the noxious sensation immediately diminishes. Nociceptors in this non-injury, or 'physiological', state only fire at a set point, increase their firing as the stimulus gets more noxious and stop firing very quickly once the stimulus terminates (Woolf,

1991). But, while what we feel does equate with nociceptor activity in many situations, there are times when nociceptor activity may be intense, yet we feel no pain at all. How often have you noticed being covered in bruises yet were unaware of the exact incident that caused the injury? Lack of pain at the time of injury is surprisingly common – it depends on whether or not the central nervous system is in a pain-permitting mode (Woolf, 1994), or put another way, whether the *pain gates* are being held firmly closed by powerful inhibitory currents (Melzack and Wall, 1965, 1996). Our central nervous system contains incredibly powerful pain-inhibiting circuitry that is kickstarted in threatening circumstances or when our attention is focused elsewhere (Fields and Basbaum, 1994).

So far it has been suggested that nociceptors only fire in response to noxious events in the tissues. This is not strictly true, since a small percentage do fire a little all the time and in parallel with other fibres (Schmidt *et al.*, 1994). This includes the very large myelinated, fast-conducting Aβ fibres. Aβ fibres, in the physiological state, send impulses that relate to non-noxious stimuli such as joint position, pressure and stretch on tissues and so forth. They inform us of our body's position and its actions, and provide the sensory information necessary for an adequate body image (Melzack, 1991) (see Chapter 3). Thus, there is a constant background barrage of impulses from the tissues of the body involved in locomotion which includes a modicum of nociceptive activity.

It has been found that in the knee joint of a normal resting cat there is a continuous afferent neural activity of about 1800 impulses every 30 seconds. When the joint is slowly moved in mid-range this increases to 4400 impulses every 30 seconds (see Schmidt *et al.* (1994) for an excellent overview). If the joint is then experimentally inflamed to mimic a nociceptive event, there is a quite remarkable increase in afferent activity. At rest, the barrage increases to 11 100 impulses every 30 seconds and when gently moved it increases to a 30 900 impulse rate. Here we have a seven-fold increase in afferent activity with gentle movement, so no wonder we don't want to move an acutely inflamed joint! Hanesch *et al.* (1992) have noted a 100-fold increase in some fibres. It appears that the afferent fibre population, in particular the nociceptors, actually change their response properties in the presence of injury. There is a dynamic or 'plastic' change in their function which is the result of chemical activity at the site of injury (Perl, 1992; Levine and Taiwo, 1994; Meyer *et al.*, 1994). What is interesting is when observed over time there is a steady increase of activity in parallel with the growing inflammatory chemical cascade.

Practical Example

Think about some of the features of a twisted ankle. After the immediate pain of injury subsides there is a period of modest constant ache in the absence of any clear stimulus, which often steadily builds up over the following few hours and into the night. Some tissues give very little pain until the next morning. Ongoing background ache is probably the result of the ongoing afferent fibre barrage dramatically increasing. Nociceptors – Aδ and C fibres, as well as Aβ fibres – increase their spontaneous firing rate in the presence of inflammation (Schmidt et al., 1994). Further, many nociceptors that were silent before the injury now wake up and begin firing too (McMahon and Koltzenburg, 1990). Some fibres take several

hours to wake up and keep increasing their activity for many hours after this (Schaible and Schmidt, 1988). This all fits nicely with the fairly predictable time course of pain in relation to ligament, muscle and tendon injury. The fact that some injuries may not become painfully apparent until the next day may be due to such factors as their poor metabolic turnover (slow to inflame and swell), lack of a good blood supply, a feeble nerve supply (think of a disc's innervation) and one which contains mainly those of the sleeping variety. An injured disc may react very slowly, may be injured in an area that has no nerve or blood supply and those nerves which do eventually get to know about the injury may take a long while to wake up (Gifford, 1995). Additionally, a slow build-up of fluid may in part account for increasing discomfort over time (See Gifford (1995) for an overview of possible disc pain mechanisms). Constant background aching may not always be a feature, but check and double check with the acute pain patient, for as is so often the case, the background ache may be of little concern compared to the horrible sharp pain experienced with pressure and movement.

Over a similar time-frame the ankle becomes acutely sensitive to touch, and minor movements that would not normally hurt become acutely painful. The quality of pain, in contrast to the ache just described, is sharp in nature and quite often it continues for some moments after the stimulus has stopped. This clinically reactive state is beautifully mirrored by the dynamic changes in responsiveness of damaged tissue nociceptor populations that have been observed in the laboratory (Handwerker and Reeh, 1991; Meyer et al., 1994). Nociceptors which typically only fired at a set high threshold in the non-injury state, now fire at considerably lower thresholds, and when they are stimulated they fire far more as well as tending to go on firing long after the stimulus is removed (Woolf, 1991; McMahon and Koltzenburg, 1994).

Hyperalgesia

Tenderness and increased sensitivity to mechanical testing for most of us is a sure sign that the area and underlying tissues that produce the tenderness are injured in some way. This is especially true if the tenderness can easily be associated with some injuring activity. Tissue tenderness is generally termed 'hyperalgesia'. However, the International Association for the Study of Pain (IASP) defines hyperalgesia as 'an increased response to a stimulus which is normally painful' (Merskey and Bogduk, 1994). Broadly, this means that if you twist your ankle and it hurts, and then later twist it again, it hurts a great deal more than it did the first time.

Clinical Examples of Hyperalgesia

With a non-injured tissue, in physiotherapy terms, if you perform a firm unilateral postero-anterior pressure in the L5–S1 region of a normal back, it is likely to produce some local discomfort. If the tissues are in a hyperalgesic state, due to some form of injury somewhere, and you repeat the procedure with exactly the same force, an increased response will occur – i.e. pain rather than mere discomfort.

Another example would be the early normal perception of discomfort when a physiotherapist passively tests a SLR, upper limb tension test or simple wrist extension into the first few degrees of resistance. If the tissues being gently mechanically stressed in these tests were hyperalgesic, this modest awareness of onset of discomfort would be signalled with increased intensity as pain.

Allodynia

Unfortunately the term hyperalgesia is rarely used in this strict IASP definitional sense in much of the pain literature. It is additionally used to encompass pain that is produced in response to a stimulus which does not normally provoke pain, for which the IASP uses the term 'Allodynia' (Merskey and Bogduk, 1994). Thus, as already described, if you twist your ankle, some hours later movements and tests that would not normally cause pain will now start to do so. Modest attempts at inversion will hurt, very gentle palpation of the area will elicit pain and so on.

Clinical Examples of Allodynia

In the examples used above, unilateral postero-anterior pressures over L5–S1 will hurt with very gentle pressures, and straight leg raise, upper limb tension testing and wrist extension will begin to hurt very early in range where no symptoms would normally be elicited. Extreme examples of allodynia can be seen in ghastly neuropathic (peripheral neurogenic) conditions like trigeminal neuralgia and Herpes Zoster (shingles), where pain can be elicted by gently blowing on the skin in the affected area.

Although the two terms *hyperalgesia,* and *allodynia* may initially be a bit confusing we must all persevere as they need to become the words of choice in physiotherapy assessment of pain.

It is probably most useful to view hyperalgesia as an umbrella term to refer to enhanced sensitivity in general (Campbell *et al.*, 1993).

Primary and Secondary Hyperalgesia

Hyperalgesia, enhanced sensitivity of tissues, may be an honest reflection of underlying tissue damage. 'The damage is where it hurts when I press on it or mechanically stress it or test it in some way' is a justifiable statement in many acute injury states, especially if the tenderness is in an area that fits with the injury history. Hyperalgesia at the site of injury is termed *primary hyperalgesia.* The best way of understanding this is to think of the exaggerated response as a 'true positive' – the thing that hurts is damaged or diseased in some way, it is the tissue responsible for this patient's pain.

It seems easy, but the overzealous readiness of most of us to jump to a conclusion of 'this is the tissue at fault because it is tender when I press on it and it produces pain when I mechanically stress it with this test and this test' may be one of the biggest clinical errors to be unmasked by pain science.

It is becoming more obvious that many tissues that hurt when they are physically stressed may be perfectly normal (Wall, 1993; Quintner and Cohen, 1994; Cohen, 1995). This phenomenon of normal tissues being abnormally mechanically

sensitive is termed *secondary hyperalgesia*. In contrast to primary hyperalgesia, here we are clearly dealing with a 'false positive' in terms of the tissues under test. Thus, a positive upper limb tension test in a patient with a chronic repetitive strain injury or a chronic whiplash may not be a reflection of anything wrong at all with the nerves being tested, it is just that *normal inputs* to the central nervous system induced by the test are processed in terms of pain rather than innocuous sensation. The source of the mechanism for this type of sensitivity is in the circuitry of the central nervous system – not in the tissues under duress (Dubner, 1991a; Dubner and Basbaum, 1994; Price *et al.*, 1994).

The brunt of the understanding of this biologial mechanism is that the huge injury and inflammatory related increase in impulse delivery to the dorsal horn of the spinal cord (the afferent barrage) is thought to be largely responsible for setting up sensitivity changes in second-order neurones that relay rostrally. It turns out that specific populations of second-order dorsal horn cells (nociceptive specific (NS) cells), which normally only respond to impulses arriving via nociceptors (Aδ and C fibres), actually change their response characteristics to becoming responsive to innocuous Aβ fibre input (Cook *et al.*, 1987). Further, cells that normally respond to both nociceptive and Aβ input (wide dynamic range (WDR) cells) now increase their sensitivity so as to fire far more dramatically and for far longer (Dubner and Basbaum, 1994). This is quite dramatic 'plasticity', because what in effect has happened is that the wiring and circuitry normally dedicated to the transmission of innocuous peripheral sensations has now become diverted into a pain transmitting one, and that dedicated to noxious information transmission has been upregulated to a highly sensitive status.

The view that the nervous system is a hard-wired unchanging computer-like organ has to be revised. As will be further discussed, the nervous system has an in-built and remarkable flexibility that widely accounts for our ability to adapt to the ever changing circumstances our lives and the environment we live in impose upon it.

Inflammation and Nociception

Inflammation is currently regarded as one of the key initial processes leading to increases in sensitivity and the clinical pain state (Levine and Taiwo, 1994). It is not simple, but it needs our attention in the sense that it should be seen as a benign event that, apart from frequently leading to pain, is vital in initiating the recovery of damaged tissues and setting the stage from which the later phases of healing evolve. Inflammation has to be seen as an adaptive response and therefore something which should be tolerated better perhaps. Chronic inflammation and pathological inflammation are maladaptive and a different matter in that they may adversely contribute to tissue damage and the general well-being of the patient.

Inflammation can be seen as having two major components, one that involves chemicals released from the terminals of nerve fibres in the damaged area, *the neurogenic component*, and the rest, *the non-neurogenic component*.

The non-neurogenic components of inflammation broadly encompass:

1 The first aid/damage limitation aspects that include the clotting and laying down of fibrin lattices.
2 The physical activity of cells of the immune system, such as the polymorphonuclear leukocytes and macrophages involved in removing 'debris' and any threatening micro-organisms.

Importantly, the immune system is not only involved in fighting invading organisms but also in initiating and controlling healing, and in chemically signalling to the central nervous system (Abbas *et al.*, 1994). For instance, mast cells at the site of injury release chemical messengers called cytokines into the tissues as well as into the general circulation. Cytokines, such as the interleukins, are thought to be transported to the brain via the circulation and help mobilize systems involved in the control of inflammation. They also appear to have a role to play in producing hyperalgesia – both at the nerve terminals and in the central nervous system (Watkins *et al.*, 1995).

3 The flow of fluid into the area, so-called plasma extravasation that gives rise to oedema (Coderre *et al.*, 1989, 1991).

4 The release of chemicals from cell membranes that have been damaged and from specialized inflammatory cells themselves. The prostaglandins and leukotrienes are important chemicals that are produced as the result of cell wall breakdown with the subsequent release of membrane phospholipids (Pettipher *et al.*, 1992). Phospholipids are the precursors to a complex cascade of chemical reactions that result in leukotriene and prostaglandin formation. The whole cascade is inhibited by corticosteroids. Non-steroidal anti-inflammatory drugs only inhibit the so-called cyclo-oxygenase pathways that give rise to the prostaglandins (Velo and Franco, 1993).

5 The release of chemicals synthesized within specialized inflammatory cells and from plasma, for example mast cell degranulation and release of histamine, or the infiltration of fibrin from plasma (Rang *et al.*, 1991, 1994).

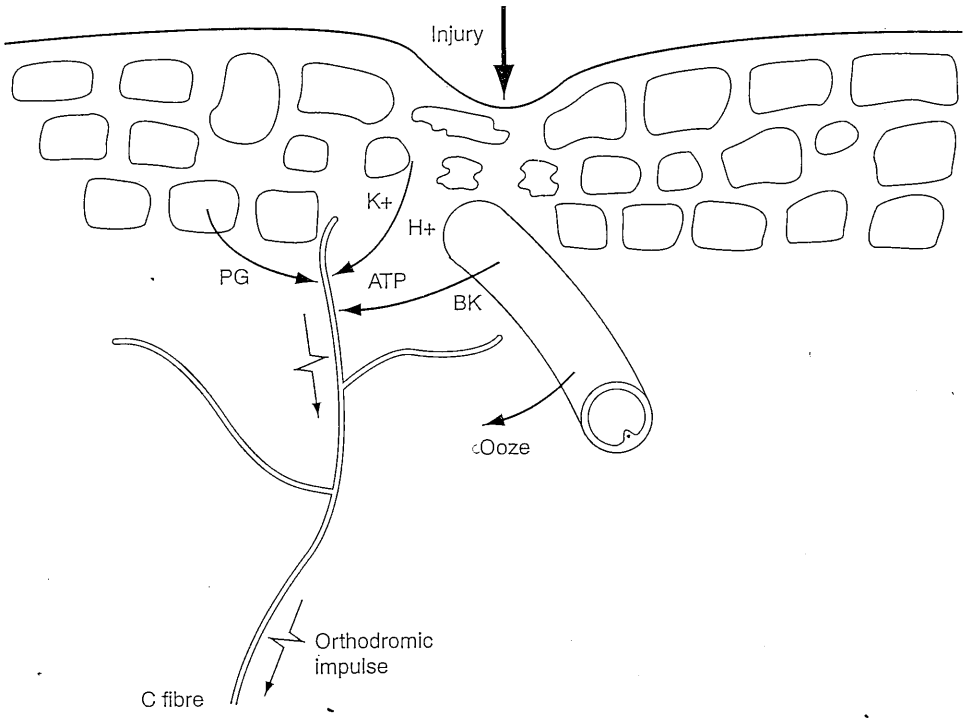
The neurogenic components of inflammation can be described as follows.

Neurones are traditionally thought of as solely having an impulse-conducting function. Recently there has been an upsurge in interest in the secretory function of peripheral nerve fibres, in particular the C fibres (Levine *et al.*, 1988; Sluka *et al.*, 1995). As already mentioned, C fibres are unmyelinated slow conductors and, at face value, pretty useless at warning of acute injury. Thankfully Aδ fibres fulfil this function.

The question has arisen as to what all the C fibres are for. In some cutaneous nerves over 90% of the afferent fibres found are C fibres (Melzack and Wall, 1996). It appears that their major role is to help maintain the health of the tissues they supply. C fibres actually sample the tissues, take up chemicals, actively transport them to their cell bodies in the dorsal root ganglia where appropriate responses are instigated and relayed back to the tissues (Moskowitz and Cutrer, 1994). If a tissue is damaged the nucleus in the cell body and cells in the dorsal horn of the spinal cord gets to know about it via these slow chemical channels of communication as well as via impulse activity (Donnerer *et al.*, 1992). Not only do they get to know, they also do something about it. C fibres become highly active in secreting chemicals in damaged tissues. They add to the inflammatory soup that is already forming and influence the secretion of chemicals, the local circulation and plasma extravasation (Levine *et al.*, 1986a).

The active chemicals that C fibres secrete are neuropeptides such as substance P and CGRP (calcitonin gene related peptide) (Walsh *et al.*, 1992). C fibres release neuropeptides experimentally when impulses travel down their axons and dendrites in the 'wrong direction' – so-called antidromic impulses. These impulses may be

Figure 5.5 Simplification of injury events leading to activation of a C fibre. Direct activation by intense pressure and consequent cell damage. Cell damage leads to release of potassium (K+) and hydrogen (H+) ions, adenosine triphosphate (ATP) and protaglandins (PG). Bradykinin (BK) is released into the area via the plasma 'kinin' system. (Adapted from Fields, HL, 1987, *Pain*, McGraw-Hill, New York, with permission.)

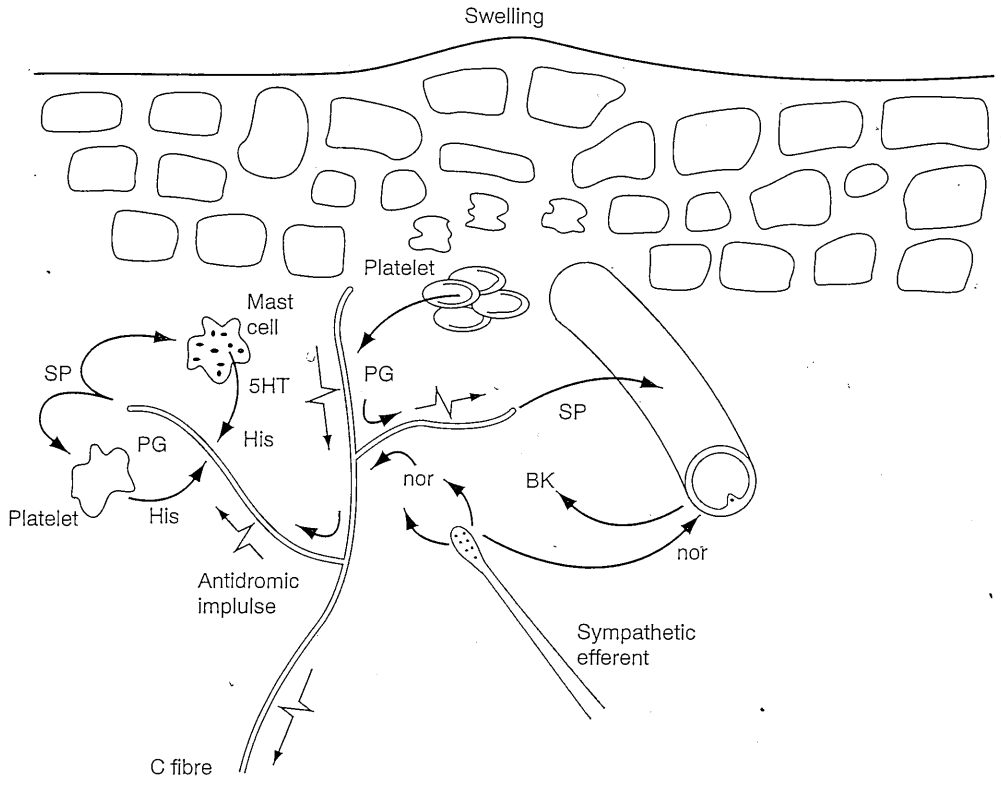


propagated from the dorsal horn terminals of the C fibres as well as locally where orthodromic (correct direction) impulses travelling proximally along dendrites may actually pass back down an adjacent dendrite in the 'wrong' direction (Sluka *et al.*, 1995).

Sympathetic postganglionic fibres, in addition to their motor function on the smooth muscle of blood vessels, also have a secretory role when tissues are injured and when we are under stress. They are known to release noradrenaline (norepinephrine, USA), which enhances the vascular response and may cause pain if the nociceptors become sensitized to it (Janig and McLachlan, 1994).

So the combined effect of neurogenic and non-neurogenic components seems to be a rather daunting and massively angry soup of noxious chemicals. Figures 5.5 and 5.6 highlight a few important aspects. The injuring force damages the tissues and causes nociceptive nerve impulses to be transmitted. Prostaglandins, hydrogen ions (inflammatory soup is acidic), potassium ions and adenosine triphosphate are among the early chemicals released from the damaged tissues and bradykinin and fluid enters the area from the circulation (Fields, 1987; Levine and Taiwo, 1994). All these chemicals are known to have sensitizing effects on nociceptors, in other words they lower

Figure 5.6 Simplification of some of the post-injury events leading to C fibre sensitization and propagation of impulses. Antidromic impulses lead to release of neuropeptide substance P (SP). Substance P produces vascular effects and facilitates 5HT and histamine (His) release from mast cells and prostaglandin and histamine release from platelets. Sympathetic efferent terminals secrete norepinephrine (nor) which only acts to excite C fibres directly if alpha-L-adrenergic receptors have been expressed. Norepinephrine acts to enhance the vascular response in 'normal' inflammation. (Adapted from Fields, HL, 1987, *Pain*, Mcgraw-Hill, New York, with permission.)



their thresholds and thus become more sensitive, i.e. they account for primary hyperalgesia.

Some of the chemicals, like bradykinin, promote firing of nociceptors – hence ongoing ache/awareness. As time goes on, the soup becomes more complex, mast cells and platelets appear and are activated to release further sensitizing and pain-producing chemicals; the sympathetic efferents secrete noradrenaline, the C fibres secrete substance P and CGRP and the whole area becomes a swollen sensitized mass of interacting chemicals. Most of the literature cited focuses on the rather explosive and self-enhancing nature of inflammation. Little is mentioned about mechanisms that actually inhibit and control the events.

PERIPHERAL NEUROGENIC MECHANISM

Pain can derive from damage to peripheral nerve trunks and roots, hence the neuralgias, sciatica, brachialgias and many nerve entrapment syndromes that are well documented. The discussion here involves a consideration of nerve fibre (neurone) injury alone, not the consequences of injury

to nerve trunk and root connective tissue (see Chapter 4).

Normal afferent neurones basically report what is happening in the tissues they innervate – the target tissues. An afferent sensory neurone is thus seen as an independent sensory channel. Impulses start at the nerve end (encoded), then travel along the axon (transmission) into the dorsal horn of the spinal cord to form the first synapse with second-order neurones and interneurones. The impulse 'message' is normally contained within the fibre. If anything goes wrong at any level along this pathway, abnormal impulse discharges, in abnormal patterns, which may begin in abnormal places along the axon, will give rise to the input of false and rather strange information (Devor and Rappaport, 1990; Devor, 1994). When nerve fibres are injured and responding like this the patient tends to perceive rather odd pain and symptoms that are in odd places. Symptoms often appear to have a mind of their own and are therefore rather worrying to the patient. Further, the pain generated can be out of all proportion to events that caused it, and it can be very nasty, unremitting and very hard to alleviate (Tanelian and Victory, 1995).

If impulses start somewhere along an axon the source of this abnormal impulse activity is said to be 'ectopic'. This is easily demonstrated if you briskly tap the ulnar nerve at the elbow and get a shower of pins and needles in your little finger. Thus, ectopic impulses can be demonstrated on a normal nerve, but as a generalization, normal nerve fibres are relatively insensitive and we go about our daily lives quite unaware that our nerves are being stretched, pinched and distorted in some way all the time. The only region of a nerve which is known to have enhanced mechanosensitivity under normal conditions is the dorsal root ganglion (Devor and Rappaport, 1990; Wall and Devor, 1983).

The pathophysiological mechanisms which can lead to ectopic impulse generator sites in nerve fibres have already been discussed (Chapter 4). It is highly likely that many of us house injured and regrowing nerve fibres that are of no consequence whatsoever. The fact that some of us succumb to peripheral neurogenic pain and symptoms may be simply down to the expression and installation of protein receptors and ion channels in the regrowing and damaged cell walls of nerve fibres which then render them mechanically and chemically hypersensitive (Devor *et al.*, 1994; Devor, 1995).

In order for normal afferent nerve fibre terminals in target tissues to be mechanically sensitive they have to contain active receptors in their cell walls that are sensitive to mechanical forces. A normal fibre does not contain large populations of active mechanoreceptors along the length of its axon, nor does it contain large populations of ion channels other than those essential for impulse propagation.

When a nerve is injured there may be loss of continuity of many individual fibres with subsequent Wallerian degeneration distally accompanied by attempted regrowth of the proximal axon (Devor and Rappaport, 1990). If a fibre is damaged but remains in continuity there may be loss of myelin and modest disruption of the axon membrane.

In any region of nerve damage there is usually a massive proliferation of Schwann cells, fibroblasts and macrophages into the area (Devor *et al.*, 1994; Wong and Crumley, 1995). Subsequent to this is the release of bioactive molecules like nerve growth factor that are absorbed by the axons and transported to their cell bodies in the dorsal

root ganglion where the production of membrane receptors and ion channels is upregulated (Devor *et al.*, 1994). These receptors and channels are protein molecules which are actively transported back to the damaged areas and installed in the membranes, hence changing their sensitivity (Devor *et al.*, 1994). Receptors may be mechanoreceptors (stretch-activated ion channels) making the area more mechanically sensitive, but may also be adrenoreceptors (sensitive to adrenaline and noradrenaline) or receptors that are sensitive to hypoxia or inflammatory chemicals. Thus the area can not only become more sensitive to movement – stretches and pressures – it can also become more sensitive to chemicals like adrenaline and noradrenaline that arrive via the general circulation or are secreted from sympathetic nerve terminals in the area.

Additionally, these sites of damage can start to produce sustained impulse volleys that are *self-generated,* may go on and on or come in continuously repeating bursts and waves, and which are thought largely responsible for some of the devastating ongoing and uncontrollable pains that nerve injury sometimes produces. These sites are termed 'ectopic pacemaker sites' because they have this independent ability to produce ongoing trains of impulses (Devor, 1994). Clinically all this stands for ongoing pain which waxes and wanes for no apparent reason. Pain that is produced in response to movement may continue on and on long after the movement is stopped. This type of pain may be influenced by fluctuations of adrenaline and noradrenaline secretion due to mental and physical stress, and it is pain that is often of bizarre qualities which happens in odd places with strange referrals.

It is often a great relief for the patient to have this knowledge explained to them in simple terms, just because pain that comes and goes for no apparent reason is worrying to most people. For example, neurogenic pain that gets worse at rest, or during the night may be due to a nerve's abnormal sensitivity resulting from ischaemia/hypoxia – blood pressure is lower at night and the circulation is relatively sluggish. Getting up, moving around, shaking a hand that has carpal tunnel syndrome may often be enough to restore peace to a nerve that has started firing as result of building ischaemia. This sort of information is helpful for the patient and focuses us on the need to include strategies that improve circulation to the area.

There are a few important issues.

1 Nerve injury that develops ectopic pacemaker ability will barrage the central nervous system with massive impulse activity. This has far-reaching implications for possibly permanent changes in central cell sensitivity and 'rewiring' of the nervous system (see central mechanisms below) (Woolf *et al.*, 1992; Woolf and Doubell, 1994).
2 This activity is not mandatory in every nerve injury. In the laboratory, rats can be bred that have a high or low propensity to developing pacemaker capability, some nerves are more vulnerable than others and some sites on individual nerves are more prone than others (Devor and Raber, 1990). The genetic aspect is intriguing, especially if you consider that the receptors and ion channels are produced as a result of specific genes in the cell bodies of the affected nerve being 'switched on' to express the proteins required. Some of us may be innately more prone to being 'switched on' than others.
3 Ectopic pacemaker capability may take a while to occur after injury. For instance, Aδ fibres that are injured may remain totally silent for

the first day or two after injury, but then slowly increase their spontaneous activity over the following two weeks. C fibres tend to increase activity as the A fibres decrease theirs (Devor, 1994). Think of the unfortunate whiplash patient who for the first few days feels stiff and sore but some days or weeks later develops weird symptoms in odd places. Not only is it strange and worrying to the patient, it also tends to be seen as the first signs of malingering by many health professionals. It is hardly surprising that people with odd neurogenic pain develop a fear of moving and adapt to movement patterns and postures in strange and unnatural ways, but they are often confronted with undertones of disbelief and frustration by those who have to deal with them. It should also be appreciated that lesions to nerves that are capable of producing such devastating pains are best seen as pathophysiological in nature; the anatomical evidence for them is at a molecular level on the membranes of individual neurones, something that cannot be picked up on radiographs, modern imaging techniques or nerve conduction studies.

CENTRAL MECHANISMS

Some mention has already been made of plastic changes in the response properties of dorsal horn cells when they are subjected to nociceptive-derived impulse barrages. These cells clearly shift their properties to an enhanced excitability state in order to accommodate damaging events in the periphery (Woolf, 1994). The adaptive value of this is seen in terms of increased and spreading pain as well as a spread of tenderness in order to promote tissue protective behaviour during the first stages of healing when tissues are at their weakest and most vulnerable. A corollary of this is that enhanced sensivity should drift back to normal as healing progresses.

What is intriguing is that in some circumstances increased excitability may remain long after healing has taken place and that ongoing pain – generated by abnormal and spontaneous discharges of dorsal horn neurones, and tissue hypersensitivity (wholly secondary hyperalgesia), as a result of normal input from the periphery activating these hyperactive cells – may be a major mechanism in many chronic pain states. While the pain and tenderness are blatantly perceived in the tissues, the dominant mechanism that is creating the pain and sensitivity state is in the central nervous system (Wall, 1988; Pennisi, 1996).

Unfortunately, hyperexcitable dorsal horn cells cannot be blamed for all chronic pain states. Although its understanding is crucial to new concepts about pain, it has to be taken as just one tiny fraction of the many possibilities the central nervous system holds. Knowledge of the dorsal horn is gratefully accepted. It would be very handy indeed for the many who suffer chronic and disabling pain to blame a small population of highly excitable dorsal horn cells as being responsible for producing an ongoing illusion that the peripheral tissues are still in a damaged state. Unfortunately the complexity of the CNS is unlikely to allow such a simple concept to be much more than a late twentieth century notion that will ultimately be superseded as yet more knowledge of the brain unfolds. However, it is worth speculating with regard to possible repercussions of central mechanisms for chronic pain states and their management. What we are really looking for are analogies and metaphors that help us to understand and explain the crazy chronic pain states to our patients and to then give them confidence that their tissues are no longer primarily respon-

sible. Confidence in being able to work through pain, work *with the pain*, or nudge *slowly* into it without fear, is one of the most powerful ways of starting chronic pain rehabilitation (see Chapter 15).

This concept of prolonged 'hyperexcitability' can be taken a stage further if one considers phantom limb pain. If a patient who has ongoing pain without having had an amputation then decides to have an amputation in a radical effort to be rid of the pain, the exact same pain would unfortunately remain (or return a short while later) in the form of a phantom (see Dielissen *et al.*, 1995). In effect, what this is saying is that any ongoing nociceptive input into the central nervous system may give rise to an 'imprint', 'memory' or 'central representation' of the pain. Thus, this pain 'imprint' or 'memory' may ultimately be unresponsive to anything therapeutic that is done to the tissues where the pain originated and where the pain and tenderness are still felt, or that is done to the peripheral nervous system that innervates the tissues that are painful – even an amputation. Clearly the mechanism responsible for the pain has moved from a nociceptive tissue dominant one, to one being housed in central nervous system pathways. Chronic pain sufferers are mutilated daily by operations that purport to correct 'relevant' anatomical abnormalities, that denervate the affected zones, that block the pain pathway etc., but careful scrutiny shows that the outcomes, although initially very good, are in reality very poor (see Melzack and Wall, 1996, p. 222; Wall, 1996b). The repercussions of this for physiotherapy pain treatments are clear.

The concept of a somatosensory memory for pain in amputees and spinal cord injured patients who suffer ongoing pain has been put forward by Katz and Melzack (1990). It seems likely that this concept may be relevant to many chronic pain sufferers who have not undergone any form of amputation or surgery. At the synaptic level, the biological processes involved in memory are considered in terms of an increasing and strengthening 'bond' between chains of neurones (Dudai, 1989; Rose, 1992). If one considers that one neurone may synapse with up to 20 000 others in the central nervous system it is quite easy to see that many of the 10 million million or more synapses (Rose, 1992) and possible pathways may be quite ineffective or 'silent' and that there are huge potentials for new unique pathways forming that have specific meaning. Thus, when something new happens, such as an ongoing pain experience, previously dormant synapses are woken up and new pathways are formed, weakly at first, but as the process is repeated during 'learning', the synaptic efficacy becomes ever stronger until eventually permanent synaptic relationships are formed. This is a situation considered to be analogous with the establishment of a long-term memory (Pockett, 1995). In short-term memory links are strengthened but only short-lived (Rose, 1992; Kandel *et al.*, 1995). The biological and biochemical processes involved in synaptic efficacy are very complex but crucially involve the pre-synaptic neurone being able to have greater and greater influence on the post-synaptic neurone. In other words, the post-synaptic, neurone becomes more excitable in relationship to the special messages received presynaptically.

During injury, this is the very state of the relationship between the primary afferent fibres arriving from the peripheral tissues and the second-order dorsal horn cells that relay up the spinal cord to the brainstem and higher centres. Similar processes occur at higher level synapses in the brain and go on to include enhanced efferent (output)

response patterns due to whole input–output circuits becoming more effective (Dougherty and Lenz, 1994; Flor and Turk, 1996; Galea and Darian-Smith, 1995). Thus we get enhanced motor responses in the form of antalgic postures, protective movement patterns and reflexes and emotionally generated feelings and behavioural responses (crying, anger, frustration etc.), as well as enhanced sympathetic and neuroendocrine activity via output from the limbic and reticular formation relays (Chapman, 1995).

Since there are such stereotyped responses to most acute injuries it does suggest that some pathways may be innately ready-to-run. Indeed, any threat to an organism requires a sophisticated and well coordinated reaction and recovery operation. However, if inputs carry on for long enough in conditions where normal inhibitory influences fail to keep a check on neurone excitability, these well established input–output pathways may run amok – maladaptively providing abnormal inputs and outputs that may help maintain abnormal and often unique physical and psychological/mental dysfunctions (for expanded discussion, see Butler and Gifford, 1998).

If this analogy with memory is true it suggests that chronic pain, its behaviour and the physically reactive movement patterns we so often see, are stubbornly implanted 'habits' in the nervous system. Further, they would require immense efforts to overcome, perhaps equivalent to an 'unlearning' or 'relearning' process. It may help if one considers how relatively easy it is to influence acute pain or ongoing nociceptive pain with drugs, yet how difficult it is to alter the pain of many chronic sufferers with drugs or anything else. It perhaps could be likened to finding a drug that could ablate long-term memories with the proviso that we remain conscious.

In order to understand some of the conditions that give rise to ongoing excitability it is necessary to journey back to the dorsal horn cells.

Synaptic efficacy and enhanced excitability, like the biology of the peripheral neurogenic mechanisms, is all about receptors. In particular, the barrage of impulses in concert with increased central delivery of neuropeptides by primary afferent C fibres is thought largely responsible for the observed changes in the second-order neurones of the dorsal horn (Pockett, 1995; Yaksh and Malmberg, 1994). Thus, dormant receptors become effective and the cells' nuclear machinery is activated to produce more receptors that are transported to and then planted in the dendritic cell wall. Luckily, the excitability of these second-order cells does not inexorably tumble on – there are inhibitory currents and neurochemicals that control and dampen down these events (gate control – see Melzack and Wall, 1996). Thus lifting of inhibitory currents, or the loss of pertinent inhibitory neurones may have far-reaching consequences. Three clinically relevant examples of how changes in inhibitory checks may lead to abnormally enhanced second order cell sensitivity are given below. All would theoretically interact.

1 There are continuous (tonic) descending inhibitory currents going on all the time which can be hugely influenced by our attention (Melzack and Wall, 1996). What is also known is that this descending inhibitory current is actually enhanced during and immediately after tissue injury (Schaible and Grubb, 1993). It seems that there is a reflex pathway that tries to adaptively limit the hyperexcitability and the consequent expansion of receptive fields of second-order neurones. It could be argued that these inhibitory currents may be lifted or overridden by

consciously over-focusing attention on pain (see Butler and Gifford, 1998).

While everyone does tend to focus on pain there are circumstances when excessive attention may occur. Consider the situation of a keyboard operator who begins to get discomfort and odd pins and needles in her arm. She knows of others in the same office who have been off work for months, she gets slightly anxious, she discusses it with them, the bosses increase the work load, there are financial worries and other pressures at home, she consults the doctor who listens quietly but is unhelpful and not overly concerned, and so on.

There are plenty of situations in modern life that can be translated into cumulative and rather malevolent, adverse biological reactions. Note that the intention here is to speculate on the mechanisms, not insist, as none of this has been tested. However, the concept of pain as a memory and the issues of enhancing of a pain imprint by maladaptively focusing on a problem, have received some interest and support from Wall (1995b), a pain scientist, and Rose (1995), a memory scientist. Importantly, some people may simply be born with, and/or develop, via the mental and physical rough and tumble of life, relatively weak inhibitory controls.

2 Inhibitory interneurones can actually be killed by large afferent barrages from damaged tissues and nerves in animal experiments where descending inhibitory currents are lifted (see Dubner and Basbaum, 1994). Massive and ongoing volleys of C fibre impulse activity cause a marked build-up of the excitatory amino acid neurotransmitter chemicals glutamate and aspartate. Both have been shown to cause neurone death and it is believed that dorsal horn inhibitory interneurones are particularly vulnerable (Dubner, 1991a, b, 1992; Dubner and Basbaum, 1994). Loss of inhibition means loss of control of excitability of second-order neurones and hence the threat of chronic pain.

The important clinical element here is that barrages from ectopic pacemaker sites in damaged nerves are particularly spiteful since they generate such massive barrages that go on for such a long time. Additionally, nerve damage invariably causes some death of afferent fibres, and the central nervous system, realizing that it has lost sensory input, upregulates its sensitivity in an attempt to seek out and recover the missing input. In some cases damaged neurones may actually grow new dendritic sprouts that wander into neighbouring dorsal horn zones and make further and often inappropriate synapses (Shortland and Woolf, 1993; Woolf and Doubell, 1994). The potential for ongoing pain, misinformation and abnormal processing is almost frightening.

3 Melzack and Wall's pain gate theory (Melzack and Wall, 1965) very strongly considered the inhibitory influence of Aβ fibres on second-order neurones (see Melzack and Casey, 1968; Melzack and Wall, 1996). It was the basis on which transcutaneous nerve stimulation (TENS) was developed for pain control. Modern pain science, as we have seen, has shown that Aβ fibre input can *enhance* pain once a dorsal horn cell has become sensitized. However, there is no doubt that Aβ input in the early stages of injury does inhibit and hence reduce potential for enhanced excitability. Any peripheral loss of Aβ fibres may therefore be an important factor. Aβ fibres, being large, myelinated and fast conducting, are metabolically demanding and therefore very prone to

damage and degeneration in conditions that enhance peripheral nerve ischaemic conditions (see Chapter 4). Minor and ongoing peripheral nerve damage that causes loss of Aβ fibres and hence its central inhibitory function may be a contributing factor in the injury-induced prolonged enhanced sensitivity state of dorsal horn cells.

Maladaptive central mechanism recognition in chronic pain is as yet only an assumption; there is no physical diagnostic test that unequivocally identifies it and there probably won't be for a long time. In fact, most standard clinical diagnostic tests reveal little (see Ochoa *et al.*, 1994) despite the fact that our physical assessments can be very lengthy when pain response is focused on. The key to understanding central mechanisms and many chronic pains is to view the problem as a hyperalgesic one – excessive abnormal and ongoing hypersensitivity of tissues such as muscle, joint, nerve, skin etc; remember the 'If you look you will find' statement earlier. There are often abnormal movement patterns and gross restrictions in ranges if pain is focused on, yet if you really look there is little evidence of true mechanically blocked loss of range any more than in most normal people.

Below are some of the features of pain related to a maladaptive central mechanism (Butler and Gifford, 1998).

- There is lack of symptom consistency.
- Symptoms do not fit within the normal boundaries set out in textbooks.
- Symptoms are often weird.
- All examining movements tend to hurt. It is rare for the patient to report a decrease in pain with examining movements.
- Patients have excessive 'reactivity' or inappropriate reactivity. In manual therapy terms they are often designated as having a highly irritable pain state and the pain treated with great respect. Unfortunately, focusing on the pain like this for therapeutic reasons may well be a factor in enhancing the pain for the patient.
- They have atypical movement patterns – compare the movement patterns of a typical acute or subacute shoulder with a chronic maladaptive one for example; often there are odd writhing movements of the body, odd shakes and contortions of the arm with plenty of facial grimacing, yet patients are quite capable of normal movement patterns when distracted from the pain.
- Response to treatment is unpredictable – one treatment may help immensely, but repeat the exact treatment again another time and the pain gets much worse.
- The patients are not happy, are often angry and have many maladaptive thoughts and emotions about their lives, their bodies, their medical management and the society and workplace they are in.

The central pain mechanisms category provides us and the patient with a developing organic basis with which to explain much maladaptive or 'enigmatic' pain (Pennisi, 1996). The description and stance taken here is my own interpretation of modern pain and memory science that many of my patients have found immensely helpful in understanding, validating and coming to terms with their chronic pain problems (see Butler and Gifford, 1998 for fuller discussion).

SYMPATHETIC AND MOTOR MECHANISMS: EFFERENT/OUTPUT MECHANISMS

The sympathetic nervous system (SNS) has received a mass of attention with respect to pain states that are said to be maintained by or

dependent on its activity, for example causalgia, reflex sympathetic dystrophy, sympathetically maintained pain (SMP), algodystrophy and many more. The classic justification for pinning blame on the sympathetic nervous system has always been the successful alleviation of symptoms following sympathetic block techniques (Wallin *et al.*, 1976, but see Bonica, 1990).

The truth of the matter is that there is no key set of signs and symptoms that enable one to distinguish whether or not a given pain state is 'sympathetically maintained'. It is rather a hit and miss affair. Thus a patient may have a classic 'sympathetically maintained' pattern – of ongoing pain in the distal extremity of an arm or leg, obvious oedema, cold extremities and discolouration of the skin, marked hyperalgesia/allodynia and perhaps dramatic joint loss of range and stiffness – yet be totally unresponsive to a sympathetic block technique. This has led to the current use of the term 'SIP, or sympathetically independent pain (Campbell *et al.*, 1994), to help classify a group of pain patients who have an apparently 'sympathetic' syndrome and yet are not helped by sympathetic blocking techniques.

Classifying these complex pain presentations using presumed pathology as the guiding principle was extremely difficult and has recently been acknowledged as being unworkable (Janig, 1996; Stanton-Hicks *et al.*, 1995). The current proposal is that the term 'Complex regional pain syndrome' (CRPS) Types I and II now be used (Janig and Stanton-Hicks, 1996). These two categories broadly encompass all pain syndromes which have similar features to those mentioned above but which may or may not have a component that is relieved by sympathectomy or sympathetic block:

1 Type I encompasses those that are sympathetic, dystrophy-like and are initiated by some sort of noxious event.
2 The Type II grouping is more causalgia-like in that the constellation of symptoms develops after a nerve injury (see Janig and Stanton-Hicks, 1996; Stanton-Hicks *et al.*, 1995).

Many of the chronic pain syndromes seen by physiotherapists, such as chronic whiplash pain and 'repetitive strain injury', at times, can come under this classification. To think of them in terms of simple aberrations of the sympathetic nervous system is a serious oversimplification. Ongoing pain states have multiple sources and multiple pathobiological mechanisms. Sometimes the SNS just happens to play a part that is amenable to invasive or pharmaceutical therapy.

The SNS needs to be viewed as a compartment of a complex system, albeit relatively primitive, that helps integrate general and specific responses to challenges to our homeostasis.

Its function as part of the autonomic nervous system is paramount to survival and it therefore has to be activated in any pain state. Nociception, and hence pain, is thus a kick-starter of the sympathetic nervous system in terms of general survival responses, e.g. increased heart rate and a more alert brain, as well as more tissue-specific responses, e.g. local vasolidation and chemical influences on inflammation in tissues (Levine *et al.*, 1986b).

The SNS responses are hugely influenced by the way we are thinking – think about a nice cup of coffee and you will start salivating, dream about a romantic night out with a friend and all sorts of amazing things start happening, be slightly anxious about something and your body starts to think it's being physically threatened and so forth. The autonomic nervous system responds to tissue input as well as conscious input. This is a

very powerful mind–body, body–mind link, and it is an area which we must acknowledge in every single dealing with our patients. If you can influence the way a person is thinking and feeling you may influence physiology in the tissues and nerves that serve the body.

The sympathetic nervous system is involved in all pain states. For instance:

- It moderates the local circulation and chemical environment.
- It innervates lymphoid and thymus tissue and therefore plays a part in controlling the immune responses (Arnason, 1993; Smith and Cuzner, 1994).
- It helps regulate endocrine hormone functions such as those that control the release of the powerful anti-inflammatory corticosterones via pathways to the hypothalamus and pituitary (Chrousos and Gold, 1992; Valentino *et al.*, 1993).

Pain, healing, recovery and our general organ responses are all under the influence of the SNS. The fact that a pain may be sympathetically maintained focuses on the fact that sympathetic secretions of adrenaline and noradrenaline can:

1 Initiate nociceptive impulses from the terminals of nociceptors that have become sensitized to these chemicals (Sato *et al.*, 1993); and/or
2 Initiate impulses from damaged nerve axons and/or regrowing endbulbs of nerve fibres that have undergone Wallerian degeneration and become similarly sensitized (Devor, 1994). (These are the ectopic sites mentioned earlier.)
3 It is also known that dorsal root gangion cell bodies increase their sensitivity to adrenaline when their axons are damaged (Wall and Devor, 1983) or there is chronic inflammation persisting at their nerve terminals (see Janig and McLachlan, 1994).

The SNS to the limbs and nonvisceral tissues of the body does not contain any sensory fibres – it only contains sensory fibres from visceral nerves. There are no sympathetic afferents in the somatic nerves. Thus, sympathetically maintained pain has to be a result of the secretory, efferent function of this system (Walker and Nulsen, 1948). Post-ganglionic sympathetic fibres innervate most tissues of the body. They travel in somatic nerves or accompany vascular plexi and have two basic functions:

1 Control of smooth muscle and hence peripheral circulation;
2 Secretory – secretion of noradrenaline and prostaglandins.

It is important to incorporate the knowledge that the adrenal medulla powerfully secretes the catecholamines adrenaline and noradrenaline and that this is controlled by SNS activity (Sapolsky, 1994). Thus any increase in anxiety, any emotional response or any excitement will powerfully influence plasma and hence tissue levels of these catecholamines.

The reader may start to see that through knowledge of this mechanism, there are multiple facets to take into account when considering the often bizarre patterns of many pain states. The difficulties do not stop here; for instance, it has recently been shown that commensurate with the fact that pain mechanisms change and move with time (Butler and Gifford, 1998; Melzack and Wall, 1996), SMP may with time become SIP (Torebjork *et al.*, 1995). Thus, even though the symptoms for the patient feel the same, the mechanism may change over time. In some patients SMP may thus be a transient phenomenon. This is certainly

the case in the animal models used by pain scientists (Wall, 1995a).

Similar to the peripheral neurogenic and central mechanisms discussed, the common feature is sensitivity due to the presence of relevant receptors. In SMP the receptor thought responsible for enhanced catecholamine sensitivity in damaged nerve fibres and in nociceptors, is called the alpha-2 adrenoreceptor (Bennet and Roberts, 1996). If it is absent there is no sensitivity; if present there is potential for a pain state. It rather begs the question, 'why shouldn't the DNA that is ultimately responsible for producing these receptor proteins be able to shut down its maladaptive production of these malevolent little chemicals . . . and does our multidimensional therapeutic interaction with our patients help bring this about?' Maybe?

The current literature on SMP makes fascinating reading and is as good as any starting point to get into the science of pain in relation to those chronic pain patients we try to 'fix' in our day-to-day dealings with patients (see for example, Janig and Stanton-Hicks, 1996).

Lastly, it is important to consider all efferent systems in the maintenance of any given pain state: somatic motor, neuroendocrine and even the immune system can be considered here (see Butler and Gifford, 1998).

Muscle itself can be a primary source of pain if it has been injured, but this has to be considered in terms of a nociceptive pain mechanism. In terms of pain and all pain mechanisms generally, there has to be a highly efficient link of afferent input – to central processing, to rapid efferent output, in order to provide an appropriate response. Clearly, screwing up one's face and screaming, tears, jumping out of the way, running away, limping, antalgic postures, abnormal movement patterns, getting into a hot bath, and going to the doctor are all motor reactions in response to pain. Prolonged maladaptive muscle activity should be considered as a mechanism that can add to the discomfort of the tissues and hence an increased nociceptive drive (Ohrbach and McCall, 1996).

The following is an interesting example of how pain behaves and the sorts of indirect influences that there can be on its behaviour. Herta Flor and colleagues (Flor *et al.*, 1991, 1992; Birbaumer *et al.*, 1995), using electromyographic (EMG) measures, have shown that subgroups of chronic pain patients show markedly increased muscular activity and tension when they are in pain and when they are exposed to personally relevant stressful situations. The increase in muscular response was found to be localized to the site of pain and maintained for a prolonged period when compared to healthy controls. They also noted that patients with pain exhibit a reduced capacity to consciously perceive and voluntarily regulate their levels of muscular tension. In any pain state, whatever the mechanism, there is likely to be an increased muscle response which may well add to the barrage of afferent impulses that help maintain the pain state, the often multiple pain mechanisms responsible, and the levels of perceived pain (see Ohrbach and McCall, 1996, and Flor and Turk, 1996, for excellent critical overview of current theories).

AFFECTIVE MECHANISMS

This pain mechanism 'hypothesis category' was introduced by Butler (1994) for manual therapy and was an attempt to incorporate the notion that 'affect' or emotion influences our perception of pain and that this is a vital consideration in all pain states. The reader should take into account that bringing the emotions and the brain into the

'hard-wired' and strongly tissue-based manual therapy philosophy back in 1993–1994 was quite a bold step by Butler.

There are many problems with misguided day-to-day clinical application of psychological and psychiatric theory and pain states, in particular when a patient's thoughts and feelings are over-focused on *as the reason for their pain problems.* The stance proffered here is that:

1 Low or depressed mood and other maladaptive alterations in psychological function that are commonly found in ongoing pain states are largely *the result of the pain state* rather than the cause of pain (see psychological/mental dysfunction earlier in chapter and see Chapter 15). (For a review of psychological factors in chronic pain see Gamsa, 1994a, b, and Banks and Kerns, 1996.) Patients happily accept this; what they cannot accept, and what most rational investigative science cannot accept either, is that their emotions are causative or blameworthy for their pain state from the very beginning (for good overview see Mendelson, 1995; Banks and Kerns, 1996).
2 Low or depressed mood and other maladaptive alterations in psychological function powerfully influence the health of the body and the perception of pain.

The affective 'pain mechanism' still stands and is in use today to help manual therapy clinicians in their evaluation of factors involved in their patients pain states. It is by no means perfect for two reasons:

1 The affective 'pain' mechanism, in isolation, implies that the emotions are a *primary* source of pain. This is obviously dangerous in the evaluation of pain that is 'physical' in character, in history and in nature. However, to most open-minded people, it is reasonable to link pain with emotions like sadness, grief, anger, disgust, extreme anxiety, and even love, for it can 'physically' and 'mentally' hurt when you are deeply emotional (see Cassell, 1991; Damasio, 1995; Morris, 1991, for example). Problems arise when a psychological component is used and viewed in terms that disparage and suggest hysteria or even dishonesty and malingering.
2 By only using the word 'affective' it unfortunately omits the 'cognitive' dimensions and factors discussed earlier. Thoughts influence feelings and the interaction of thoughts and feelings influence the perception of pain and the health of the body.

It may be a wise and open-minded step to rename this category 'psychological/mental processing mechanisms' and leave it at that (see Butler and Gifford, 1998).

A Proposal Of How We Should Be Thinking?

Pain is a regular companion to impaired movement. It follows that in order to understand fully the altered movement patterns and loss of range that accompany pain, we need to understand pain in its broadest sense.

Hopefully the reader will realize that pain diagnosis and the management of the patient in pain is not that easy and therefore being unable to help or understand a patient in pain should not be attributed to personal failure by the therapist. We have to face many difficulties, not just in advancing our knowledge about pain, but more importantly in making it useful to the patients we see. In this chapter an attempt has been made to highlight the importance of the cognitive and emotional dimensions of pain as well as attempt

to demystify some of the complexities of pain mechanism biology.

One message should be that the continuing search for a passive technique or therapy for pain relief may need re-evaluating. It certainly seems as if the current medical model, that evaluates a disorder in terms of a 'disease' and then targets a therapeutic intervention on it, is not living up to its promise for many patients in pain (Loeser and Sullivan, 1995).

The great irony of this latter part of the twentieth century is that we now know more about pain than ever before, yet the problem is getting markedly worse. More and more people are *complaining* about ongoing pain and medicine is clearly losing the battle to contain it (CSAG, 1994; Fordyce, 1995; Morris, 1991).

On physiotherapy courses, the question that so often arises is, 'we have identified the mechanism and the source of the problem, now what technique do we use to fix it?' The effect of any successful therapeutic intervention involves a complex interaction of components like personal interaction, security, trust, warmth, interest, empathy, knowledge, faith in the therapist and the technique, expectations, therapist reputation, expense, touch, novelty, technique and the impressiveness of the technique, exercise, planning of goals and coping strategies, restoration of range and strength and so forth. A given patient may respond powerfully to a specific technique done by a specific practitioner. The same technique given in exactly the same physical way by anyone else may be quite impotent. This does not denigrate the power of the physical technique if it is seen as an important physical part that may be essential to the whole atmosphere of 'therapy' or restoration of function. As a Gestalt psychologist would put it, the key is that the whole is not the same as the sum of its individual parts, it is something far greater. Thus, trying to evaluate or scientifically prove the power of a technique on its own, without including the whole atmosphere of 'therapy', is problematic.

The recent Clinical Standards Advisory Group that investigated the current management of back pain recommended an 'increased role and resources for physical therapy for back pain, but this is contingent on resources being used to provide interventions of proven value' (CSAG, 1994, p. 46). Consider how we currently 'prove' the value of an intervention – it is often by attempting, to the best of our scientific ability, to cut out the most powerful dimensions of human suffering – the mental dimensions and their so-called placebo effect. Perhaps what we should be proving is the power of the whole and encouraging therapists to recognize that the atmosphere they create during therapy (including what they physically do) is biological and is the key to successful outcome. It is important with your patients to use the physical and communicative techniques which you feel most comfortable and confident with for that particular patient. It may be with a bias to manual therapy, electrotherapy or simply explaining, advising, setting goals and giving a simple exercise. I imagine that they are the ones that would be shown to be scientifically effective for me with that particular patient, but they could be a total waste of time for the next therapist with the same patient.

Measuring Success in Therapeutic Pain Relief

The last message is that we must be more aware of how we measure our therapy successes and perhaps question whether the fact that the patient

walked out with less pain and more range is a real advance or just a timely positive blip that polishes our egos yet is of no real advantage to the overall functional result. What we perceive we are doing may be quite different from reality. For instance, Howe *et al.* (1985) tabulated surgical results for the same patients, using different definitions of satisfactory results. By some definitions the success rate was 50%; by other definitions, almost 100%. The same could probably just as easily apply to physiotherapy interventions if they were measured in different ways and scrutinized. Ultimately, successful outcome has to be seen in terms of restoration of mental and physical function and the re-establishment of a positive role in the family and society – and not wholly contingent on the relief of pain.

Currently there is an underlying and very powerful body of literature that requires us to pay less attention to pain focused and pain relief directed therapy and more to functional recovery and active rehabilitation, especially where problems are beyond the early acute stage (see Fordyce, 1995). What is largely criticized, and is the most important message, is that if patients perceive that they are attending therapy *for it to cure them*, if they have surrendered to the authority and power of medicine/physiotherapy, then we may be inadvertently reinforcing their problem (Pither and Nicholas, 1991) and helping it to become chronic. Physiotherapy is all about providing information, restoring confidence and restoring function by empowering the patient in an optimistic and happy atmosphere.

Conclusions and Key Points

1 Pain is a perception. In this sense its source is always in the brain (Backonja, 1996).
2 Pain is multidimensional; it is more than just hurt, it alters the way we think and feel, it changes our behaviour and it alters our lives and the lives of those around us.
3 Clinicians are urged to have a better understanding of pain mechanisms and to bring the knowledge into the clinic. There are many patients who have pain that their physiotherapists, their doctors and their family and friends cannot understand. The patient is left with a bewildering condition whose validity is often tacitly and cruelly challenged by those around them and society in general. If some steps can be taken to improve our understading of pain, and our ability to diagnose and explain it to our patients, we will surely emerge with a more constructive approach to its management than we have at present.
4 Much of the current literature on pain is upholding the principles of rehabilitation proposed by the psychology driven cognitive–behavioural approach to pain states (see Chapter 15) (Gatchel and Turk, 1996; Turk *et al.*, 1983). The integration of these principles into the current physiotherapeutic-driven physical assessment and management skills used in primary care holds considerable promise for the future of the patient with pain (Butler and Gifford, 1998).
5 This chapter, although critical of some current therapy systems, is not suggesting we reject anything physiotherapy has so far evolved. However, it is stongly demanding (as pain science is of us: see Loeser and Sullivan, 1995 and Waddell, 1996) that we question what we

now have in order to move forward. Physiotherapy must continue to develop approaches empowered by hindsight in tandem with open-minded speculation based on up-to-date scientific knowledge. Good science is about making hypotheses but at the same time framing them so they are open to being challenged and capable of being disproved. Contrast this with a 'pseudoscience' approach where the systems and hypotheses are often framed so that they are invulnerable to any experiment that offers a prospect of disproof and where sceptical scrutiny or criticism is often powerfully opposed (see Sagan, 1997).

Further Reading

Butler, DS, Gifford, LS (1998) *The Dynamic Nervous System.* NOI Press, Adelaide. This book represents a distillation and integration of the biology of pain and stress into a clinical model for physiotherapists. It takes many of the theoretical and clinical issues raised here and in the chapter on neurodynamics to a greater breadth and depth and has large practical sections and clinical examples.

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