

# Introduction to pain management

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The theoretical foundations of modern interdisciplinary pain management are presented. The influence of Melzack and Wall's gate control theory of pain on the interaction between physiological and psychological processes cannot be overstated. In Chapter 2 the nature of psychological factors is considered. An understanding of behavioural and cognitive factors is at the heart of modern pain management. In Chapter 3, the influence of cultural, subcultural and social factors on the perception of pain, treatment seeking and response to treatment are considered. The role of social learning mechanisms is also discussed. In Chapter 4, the influence of economic and occupational factors on pain and disability is viewed from the standpoints of treatment seeking and as potential obstacles to recovery. In Chapter 5, biomedical, psychological and socioeconomic factors, in conjunction with the important influence of iatrogenic misunderstandings and distress, are integrated into a model of disability. Finally, in chapter 6 the historical origins of pain management are traced.



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# 1

## **Models of pain**

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### **INTRODUCTION: THE NATURE OF PAIN—A PERPETUAL PUZZLE**

The nature of pain has puzzled humanity for centuries. As a personal experience pain has an immediacy and impact. This experience seems difficult to capture in words, yet such is the power of its impact that it could be considered that pain can be described adequately only in picture or metaphor. For centuries there has been a sustained attempt to develop a philosophical and scientific understanding of it. Many different models of pain have been offered and it is sometimes difficult to appreciate the extent to which our thinking is constrained by the prevailing conceptual models, or *Zeitgeist*. In this chapter an attempt will be made to view the nature of pain from a historical perspective through the eyes of influential thinkers from a variety of disciplines. Interestingly, although theoretical models are a product of their time, they can cast a long shadow. Contemporary conceptualisations about the nature of pain echo thinking from the past.

Any scientific understanding of the nature of pain requires consideration of fundamental physiology, psychology and the relationship between mind and body. Such an analysis requires consideration also of the use of language and the nature of scientific discourse. An overview of earlier theories about pain is offered to identify their influences on contemporary thinking. We are indebted to Bonica (1990) for much of the historical source material.

**Table 1.1** Models of pain and their origins I: pre-Cartesian

<i>Epoch</i>	<i>Key figures</i>	<i>Pain sources and mechanisms</i>
<i>Primitive</i>		Magical fluids; exorcism; sorcery; women healers; medicine men
<i>Ancient Egyptian</i>		Influences of gods; spirits of dead; network of 'metu'; object intrusion
<i>Ancient India</i>		Frustration of desires; all joy/pain in heart
<i>Ancient China</i>		Yin–Yang balance; vital energy (Qi); network of 14 meridians; acupuncture
<i>Ancient Greece</i> (6th–4th BC)	Alcmaeon Anaxagoras Hippocrates Plato	Brain centre of sensation All sensations associated with pain Four humours; pain as deficit/excess Pain from both peripheral sensation and heart; pain/pleasure affect whole body; pain as a 'passion of the soul'
	Aristotle	Five senses; brain no direct function in sensation; pain as increased sensitivity of every sensation; caused by vital heat
<i>Ancient Rome</i> (3rd BC)	Galen	Central and peripheral nervous system; brain as centre of sensibility Three classes: soft nerves with 'psychic pneuma' (sensory), hard nerves (motor) and pain nerves
<i>Middle Ages</i> (10th AD)	Avicenna	Five 'external' and five 'internal' senses (in brain); 15 different types of pain (due to humoral changes)
	Magnus Mondino da Vinci	Sensation in anterior cerebral ventricle Brain seat of sensation with power to cool heart Brain centre of sensation; nerves tubular; spinal cord as a conductor

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## THEORIES OF PAIN

### Pre-Cartesian

Although any contemporary model of pain will include both physiological and psychological factors, early theories of pain were very different (Table 1.1). Early civilisations offered a variety of explanations for pain and attributed it to such factors as religious influences of gods, the intrusion of magical fluids, the frustration of desires and deficiency or excess in the circulation of Qi.

The early Greeks gave more specific consideration to the nature of pain. Plato believed that the heart and liver were the centres for appreciation of all sensation, and that pain arose not only from peripheral sensation but as an emotional response in the soul, which resided in the heart.

Aristotle believed that the brain had no direct function in sensory processes. This view was not universally held. Nerves had been described during the Renaissance and, indeed, there was an understanding that these were connected to the brain and spinal cord. They were thought to be responsible for other sensations that were felt in the brain and not associated with pain at all. Pain due to external injuries was thought to be due to the entry of evil spirits. There was even less understanding of pain from internal or visceral causes. It was frequently attributed to the influence of evil spirits or the gods.

Hippocrates considered that pain was a consequence of deficiencies or excesses in the flow of one of the four fluids or humours (blood, phlegm, yellow bile or black bile). Galen in contrast clearly established the anatomy of the

cranial and spinal nerves. He distinguished three types of nerve: 'soft' nerves, 'hard' nerves and pain nerves. He also considered that the centre of sensibility was the brain.

Nonetheless Aristotle's theories still had considerable influence. For a long time pain was still considered to be an emotion or sensation experienced in the heart or an effect possibly of the entry of evil spirits. The brain was thought to play no part in the experience of pain. Indeed, the controversy over whether pain should be regarded as a sensation or as an emotion has continued to the present day and led to an overstated dichotomy between sensory and emotional factors. Descartes' new theory was, therefore, a massive leap in the understanding of the mechanism of pain, and drew significant criticism from his contemporaries at the time.

### Cartesian model

Descartes' explanation of pain (Descartes 1664) needs to be understood against the background of his philosophy. He attempted to show that humans consisted of an earthly machine (*machine de terre*) inhabited by and governed by a rational soul (*ame raissonnable*). He tried to explain how blood, itself derived from food, gave rise to animal spirits by means of which the special earthly machine, the brain with its nerves, carried out the behests of the rational soul. The spirits dilated the brain, thus enabling it to receive the impressions of external objects, and flowed from the brain along the nerves into the muscles, thus enabling the nerves to serve as 'the organs of the external senses'. Thus animal spirits constituted a very subtle fluid amenable to the physical laws governing fluids, and the nerves were hollow tubes along which the spirits flowed in a wholly mechanical manner (Foster 1901). The nerves were not merely hollow tubes, however, but contained also delicate threads which spread all over the body from their origins at the internal surface of the brain and served as organs of sense. These threads were easily set in motion by the objects of the senses and at the same instant pulled upon the parts of the brain from which they originated.



**Figure 1.1** Descartes' model of pain From Waddell *The Back Pain Revolution* 1998, reproduced with permission.

Descartes offered the example of a foot coming into contact with fire (Fig. 1.1):

If for example the fire comes near the foot, the minute particles of this fire, which as you know have a great velocity, have the power to set in motion the spot of skin of the foot which they touch, and by this means pulling upon the delicate thread which is attached to the spot of the skin, they open up at the same instant the pore against which the delicate thread ends, just as pulling at one end of a rope one makes to strike at the same instant a bell which hangs on the other end. (Descartes 1664, translated by Foster 1901, p. 265).

As Foster points out, Descartes' theory required these nerves to have physical properties for which he had no evidence.

Descartes offered a dualistic view of mind and body. The body essentially was a machine whose workings could be explained by the laws of nature. The 'rational soul' was the 'conductor of the orchestra'. Descartes never really satisfactorily resolved the relationship between the two. There certainly does not seem to be any central 'processing' of the information, although it is consistent with the notion of summation.

### The Cartesian legacy

In understanding pain mechanisms, there have been two major assumptions that have been inherited from Descartes: first that of a one-to-

one relationship between the amount of damage (or nociception) and the pain experienced, and secondly the separation of mind and body.

The model of a one-to-one relationship between the amount of tissue damage and the amount of pain experienced has attractiveness, in that it seems to be consistent with the everyday experience of acute pain. It has to be remembered of course that pain is first and foremost a biological warning signal. Pain is necessary to protect us from damage. There are well-recorded instances of children with an insensitivity to pain who unknowingly injure themselves. They are unable, for example, to experience pain from internal disease and need to have their temperature monitored regularly in order to alert their carers to the possibility of internal pathology. They do not have a normal life expectancy. Thus the function of pain in alerting us to actual or potential tissue damage is extremely important. We are programmed to react rapidly to pain. Sudden pain produces an instinctive withdrawal response. We attend immediately to it. We attempt to escape from the source of pain and we try to protect the injured tissue from further damage. The pain is giving us important information about the source, nature and intensity of the pain. On occasions, such information may be of life-threatening importance. Avoidance of further painful experiences will necessarily avoid damage, aid healing and return to the 'normal' state. The assumption that a higher level of pain is indicative of more serious physical damage (Fig. 1.2) is therefore useful, but an *accurate* appraisal of the pain is not necessary.

Most patients have little understanding of the complexity of the neurophysiology and anatomy of nociceptive pathways and pain experience and indeed do not require such knowledge. They are unlikely to have experienced pain in the past without some injury or cause. The concept of pain experience as *not* directly associated with the amount of tissue damage is alien and illogical to them. Perhaps we should not be surprised. For unless the pain persists there is usually no need for a patient to understand pain mechanisms. Furthermore, the medical profession and allied disciplines will, to a large extent, have reinforced



**Figure 1.2** The patient's model of pain. This model of pain demonstrates a direct relationship between stimulus (the pressure on the T bar) and the amount of pain (the brightness of the electric bulb). It implies information travelling in one direction only, and does not allow for any modulation of the stimulus. This model implies: that a small stimulus may not cause pain, a large stimulus will always cause pain and when pain is sensed there is always damage causing it. Stopping the stimulus is the way of stopping the pain. Cutting the wire is the only other alternative.

this model of pain and damage. Rest is usually prescribed for any painful injury, along with pain killers and other treatments. Certainly patients are advised not to move something if it hurts. The message 'let the pain be your guide' is commonly given to patients recovering from an acute injury, reinforcing the notion of pain being a direct measure of tissue damage.

A legacy of the 'mind-body split' has been the conceptualisation of pain as being *either* physical *or* psychological. Patients' general defensiveness about considering anything from a psychological viewpoint has made contemporary pain management, which accepts an interaction between physical and psychological factors, difficult for patients to accept. (Many patients view discussions about 'non-physical' influences on pain with disbelief, suspicion and sometimes downright hostility.) Attribution to a psychological influence on the perception of pain by a professional may be taken by the patient as synonymous with an implication of some sort of mental illness, a suggestion that the pain is imaginary or even that the patient is malingering.

In conclusion, the Cartesian theory represented a significant advance on its predecessors, in postulating a mechanism of pain transmission from the periphery of the body to higher centres in the brain, but every theory has its limitations.

**Table 1.2** Models of pain and their origins II: 17th–mid 20th century

Epoch	Key figures	Pain sources and mechanisms
<i>17th and 18th centuries</i>		
1628	Harvey	Heart as site of pain
1664	Descartes	Body as a machine; nerves as tubes containing threads; pineal gland and ventricles as reservoirs of animal spirits; spirits flow along tubes; bell-rope mechanism
1794	Darwin (E.)	Pain as 'phase of unpleasantness'; intensity
<i>19th century</i>		
1827	Bell	Dorsal and ventral roots
1839	Muller	Special nerve energies
1859	Schiff	Specificity (sensory) theory
1894	von Frey	Pain as fourth cutaneous modality
1895	Erb	Intensity (summation) theory
1895	Goldscheider	Skin sensory input summated at dorsal horn; spinal 'summation path'
1895	Marshall	Pain as an affect
1895	Strong	Pain includes original sensation plus psychic reaction
<i>20th century</i>		
1900	Sherrington	Pain having both sensory and affective dimensions
1920	Head	Pain centre in the thalamus
1932	Nafe	Spatial and temporal patterning of impulses
1943	Livingston	Central summation theory; T cells critical
1951	Gerard	Nerve lesions and 'synchronously firing pools'
1952	Hardy	4th theory—primary perception and secondary reaction
1955	Sinclair	Peripheral pattern theory
1959	Nordenboos	Sensory interaction theory

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It was unable to explain clinical phenomena such as phantom pain (where pain is felt in a part of the body that has been amputated), the absence of pain in the presence of injury (such as battle wounds that are noticed only when the threat to life has passed), or the persistence of pain beyond tissue-healing time (as in chronic pain conditions). Finally it could be argued that its assumptions of a one-to-one relationship between nociception and pain experience, and its dualistic separation of mind and body, have been particular hindrances to the development of an adequate theory of pain.

### Post-Cartesian developments

Two major physiological theories of pain became the object of research in the 19th century (Table 1.2). Descartes had developed the concept

of a pain pathway linking the periphery of the body with higher centres in the brain. It led to the specificity theory, in which pain was considered to be a *specific sensation* independent of other sensations. According to the intensive (summation) theory, however, touch was experienced as a painful sensation only when it reached a certain threshold (following intense stimulation). Both these theories and their later derivatives were essentially physiological in nature and the *perception* of pain was not specifically addressed. They offered a relatively simple relationship between tissue damage and pain perception. The sensory system responsible for mediating pain was regarded as relatively rigid and straightforward in that any tissue damage initiated a sequence of neural events that inevitably produced pain. An unfortunate inference from this simplistic view was that pain was fixed

solely by characteristics of the noxious stimulus and that the nociceptive system functioned primarily as a passive relay mechanism. Neither of the theories could explain either pain in the absence of tissue damage or variation in pain between individuals with (apparently) the same amount of tissue damage. The latter tended to be attributed to long term personality characteristics rather than be seen as a feature of the psychological impact of the pain itself.

We now know that pain perception depends on complex neural interactions where impulses generated by tissue damage are modified both by ascending systems activated by innocuous stimuli and by descending pain-suppressing systems activated by various environmental and psychological factors.

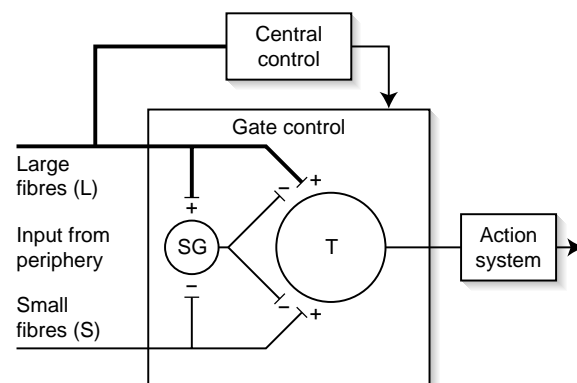
With the addition of the later sensory interaction theory (postulating a fast and a slow system of pain transmission), the foundation of the gate control theory (GCT) (Melzack & Wall 1965) was laid.

## The gate control theory

In 1965 Melzack & Wall proposed the original gate theory of pain, or GCT-I (Fig. 1.3). This theory marked a turning point in our understanding of pain (Table 1.3). It was of landmark importance in two respects: first in terms of the mechanisms of the transmission and modulation of nociceptive signals, and secondly in terms of its recognition of pain as a psychophysiological phenomenon resulting from the interaction between physiological and psychological events.

The GCT incorporated physiological specialisation, central summation patterning and modulation of input by psychological and other factors into a new model that has formed much of the contemporary psychology of pain.

The GCT postulated spinal 'gates', in the dorsal horn at each segmental level in the spinal cord, determining which of the competing impulses (pain, heat or touch) was transmitted at any particular moment. Successful transmission through the gate was affected not only by the



**Figure 1.3** Gate control theory I (GCT-I). L, the large diameter fibres. S, the small diameter fibres. The fibres project to the substantia gelatinosa (SG) and first central transmission (T) cells. The inhibitory effect exerted by the SG on the afferent fibre terminals is increased by activity in L fibres and decreased by activity in S fibres. The central control trigger is represented by a line running from the large fibre system to the central control mechanisms; these mechanisms, in turn, project back to the gate control system. The T cells project to the action system. (+, excitation, -, inhibition.) From Melzack & Wall 1965, p. 971, reproduced with permission.

**Table 1.3** Models of pain and their origins III: since the gate control theory

Epoch	Key figures	Pain sources & mechanisms
1962, 1965	Melzack & Wall	GCT-I: competition at dorsal horn for transmission
1966	Mendell	Wind-up
1968	Melzack & Casey	Ascending and descending influences; motivational, cognitive and behavioural aspects of pain experience
1983	Travell	Myogenic pain theory
1991	Jones	Limbic system mapping (PET)
1991	Dubner	Neuroplasticity
1994	Flor	Psychophysiological mechanisms
1996	Ohrbach	Stress-hyperactivity theory
1996	Melzack	Neuromatrix theory

intensity of the stimulation, and competing local stimuli, but also by descending impulses from the higher central nervous system. In contrast to the model of Descartes, the transmission of information about events in the periphery is not a simple one-way system. There is continual modulation of information from the periphery—in the gate theory's case, nociception.

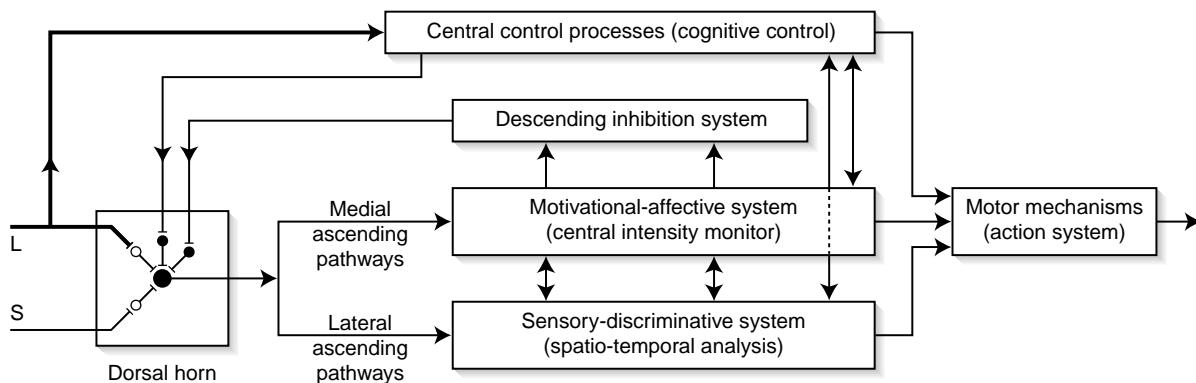
Figure 1.3 is a schematic representation of the sensory input at each segmental level in the dorsal horn of the spinal cord. The large fibres (L) and the small fibres (S) project into the substantia gelatinosa (SG) and the first transmission cells (T). It can be seen from the figure that input from large fibres will *reduce* the amount of nociceptive traffic, whereas input from the smaller fibres will *increase* nociceptive traffic going further up to the action system.

The diagram also shows the influence of 'central control' (at the dorsal horn). This central control is initiated by input from the large fibres and feeds back into the 'gate control system'. These large fibres carry specific information about the nature and location of the stimulus. They conduct rapidly, not only potentially setting the sensitivity of the cortical neurons but

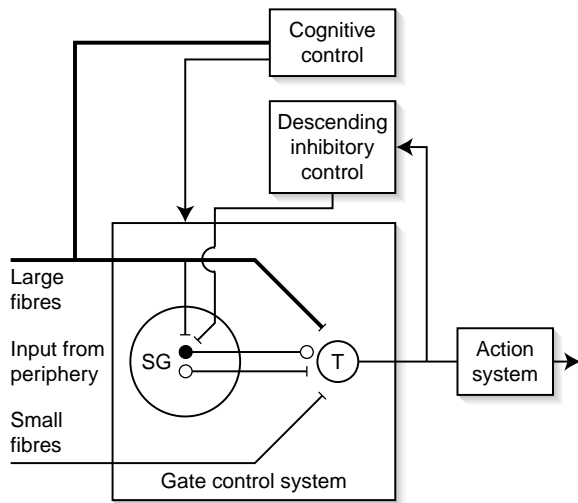
also influencing the sensory input in the gate itself. This may occur not only at the level of the original input but also at other levels.

This rapid transmission allows the brain to identify, evaluate and modulate input before the action system is activated by the T cells. The final common pathway is provided through the 'T' cell, which, when the output from this exceeds a critical level, sends information further on to the 'action system'. The 'action system' describes those areas of the brain responsible for the behavioural responses to pain and the 'experience' of pain itself.

Soon after the first publication the theory was expanded (Melzack & Casey 1968) to describe the neural system beyond the gate (Fig. 1.4). The expanded theory suggested different systems for the motivational, affective and cognitive aspects of pain experience. The neospinothalamic projection serves to process *sensory discriminative* information (location, intensity and duration). The information passing through the paleospinothalamic system activates the reticular and limbic areas, giving rise to the unpleasant affect and aversive (*motivational*) drive, which ultimately produces action. The higher centres in the brain



**Figure 1.4** Gate control theory II (GCT-IIb). The output of the T cell in the dorsal horn projects to the sensory–discriminative system via the lateral ascending system and to the motivational–affective system via the medial ascending system. The central control 'trigger', composed of the dorsal column and the dorsolateral projection systems, is represented by a heavy line running from the large fibre system to the central control processes, which take place in the brain. These in turn project back to the dorsal horn as well as to the sensory–discriminative and motivational–affective systems. Added to the scheme of Melzack & Casey is the brainstem inhibitory control system activated by impulses in the medial descending system, and which provides descending control on the dorsal horn. Moreover, there is much interaction between the motivational–affective and the sensory–discriminative systems as indicated by the arrows. The net effect of all of these interacting systems is activation of the motor (action) system. Modified from Melzack & Casey, *The Skin Senses* edited by DR Kenshalo 1968. Courtesy of Charles C. Thomas, Publisher Ltd, Springfield, Illinois, USA.



**Figure 1.5** Gate control theory II (GCT-II). The new model includes excitatory (white circle) and inhibitory (black circle) links from the substantia gelatinosa (SG) to the transmission (T) cells as well as descending inhibitory control from brainstem systems. The round knob at the end of the inhibitory link implies that its actions may be presynaptic, postsynaptic, or both. All connections are excitatory, except the inhibitory link from SG to T cell. Modified from Melzack R & Wall PD 1983, reproduced with permission of Penguin Books Ltd.

(neocortical) influence the sensory–discriminative and the motivational–affective systems having evaluated the input in the light of past experience.

In 1983 Melzack & Wall modified the GCT model in the light of new information. The model retained the excitatory and inhibitory links from the substantia gelatinosa and added the expanded version of the central controls as described by Melzack & Casey (1968) (Fig. 1.5).

### Further developments from the GCT

Much work has been done over the last 30 years to tease out the details of Melzack & Wall's theory. The general framework of the theory remains intact. Much more is now known about the complexity of the neurophysiology and neuroanatomy of the pain pathways, in particular the mechanisms of ongoing transmission of nociception that take place in the dorsal horn of the spinal cord. Anatomical pathways,

neurotransmitters and gene expression have all been described. Perhaps the most important discovery is the 'plasticity' of the nervous system by Dubner (1997)—in other words, the ability of the nervous system to change its sensitivity following tissue injury or nerve injury. Both central and peripheral sensitisation may occur following injury.

### Peripheral sensitisation

Tissue injury is heralded by electrical activity of nociceptors. However, there is also evidence of increasing sensitivity of nociceptors at the site of injury, following injury. In addition there is also evidence of 'silent' nociceptors, which are normally dormant and become active only following tissue injury.

### Central sensitisation

The increased neuronal barrage appears to lead to functional changes in the spinal cord and brain that contribute to hyperalgesia and spontaneous pain. This appears to be mediated by excitatory amino acids such as glutamate and aspartate at *N*-methyl-D-aspartate (NMDA) receptor sites. This in turn is enhanced by neuropeptides such as substance P (SP), calcitonin gene-related peptide (CGRP) and dynorphin.

In addition to sensitisation other changes occur in the spinal cord that lead to amplification of peripheral signals. There is an increase in the receptive field of wide dynamic response (WDR) cells in the dorsal horn following peripheral tissue injury. WDR neurons respond not only to input from primary nociceptive afferents but also to low threshold mechano receptive primary afferents (touch and other non-painful sensory input). The enlarged receptive field together with peripheral and central sensitisation may lead not only to amplification of nociception but also to potentially painful experience from non-nociceptive stimuli.

For a detailed exposition of recent advances in the understanding of pain physiology the reader is directed to other writings (Dubner 1997, Price, Mao & Mayer 1997).

Technological advances in brain imaging (Roland 1993) have permitted investigation into the relationship between regional cerebral blood flow (rCBF) and neuronal synaptic activity. Development of functional magnetic resonance imaging (fMRI) has allowed the rapid detection of magnetic field changes with spatial localisation using position emission tomography (PET) scanning. Recent research has identified differences in the extent to which parts of the limbic system are activated in different patients in response to the same experimental pain stimulus. Investigation of attentional mechanisms and the role of memory of pain are being actively researched. (It seems, for example, that painful memories can themselves act as pain stimuli and lead to the experience of pain in the absence of any new pain stimulus.)

In a sense much of the research into physiological and psychological factors has been developed along separate or parallel tracks. More recently, the development of pain-associated incapacity has been investigated from a *dynamic* point of view and can be viewed as an attempt to 'put the mind and the body back together again'.

The GCT has also been important from the *psychological* point of view. It has offered a testable model of how psychological factors could activate descending pain-inhibitory systems, influence nociceptive processing and thereby modulate pain. It offered a way of integrating concepts of pain behaviour, both as a *response* to pain and as behaviour that could come under environmental control. The theory has stimulated interest into beliefs, appraisal and fears about pain and pain-related coping strategies. It has encouraged the investigation of the nature of pain-associated disability and led to the development biopsychosocial models, which have attempted a wide integration of physical, psychological and social perspectives. The importance of psychological influences on the perception of pain has been increasingly recognised. Experimental studies into pain threshold and tolerance have highlighted the importance of fatigue, depression and fear. Several research groups are currently investigating the role of attentional mechanisms and memory. Studies

in phantom pain have led to the formulation of the recent neuromatrix theory (Melzack 1996), which has stimulated much debate into cognitive representation of sensation. Finally, the link between psychology and physiotherapy has led research away from the myogenic pain theory of Travell & Simons (1993) to the investigation of psychophysiological responses to pain (Flor & Birbaumer 1994) and stress-hyperactivity theory (Ohrbach & McCall 1996).

## MODELS OF PAIN AND MODELS OF ILLNESS

There are similarities between the Cartesian model of pain and the traditional disease model of illness in their assumption of a direct one-to-one relationship between physical signs of disease and accompanying symptoms.

In 1858 Virchow in his model of cellular pathology established the basis of the medical model of illness and disease that has influenced medical treatment for the last century. In arriving at a diagnosis and treatment, the physician proceeded through a number of stages. On the basis of the patient's presenting symptoms (complaints), the doctor investigated the presence of associated physical signs. Having identified treatable pathology and arrived at a diagnosis, treatment was then prescribed with the expectations that the disease would be cured, the physical signs would disappear and the patient would no longer be troubled by the presenting symptoms. Consider, for example, patients who present with ear pain. Doctors will inquire further about the nature of the ear pain and consider a range of possible causes for the pain. They will then examine the patient to confirm their provisional diagnosis. Doctors will see an inflamed ear drum and infer that the patient has an infection that is causing the pain. They will prescribe antibiotics to cure the infection and both doctors and patients expect the symptoms (the pain) and the signs (the redness of the ear drum) to improve. In other words, an explanation for the symptoms and signs has been found. By treating the cause of the symptoms and signs it is expected that they will be cured.

This model is dependent, however, on there being a close relationship between symptoms, signs and disease. It does not take into account the variability of the patient's response to illness and in the self-report of symptoms. Despite these shortcomings, the model works well for most acute medical and surgical conditions.

## LIMITATIONS OF THE TRADITIONAL MEDICAL MODEL FOR THE TREATMENT OF PAIN

### Syndromes and diagnoses

Medicine has categorised disease and illness in order to aid diagnosis and subsequent treatment. Textbooks abound with 'clinical syndromes'. These indicate a number of different symptoms and physical signs, which when present should lead the doctor to reach a specific diagnosis. The 'diagnosis' may then be further confirmed or refuted by performing specific tests. The results of these tests may be included in the description of the 'clinical syndrome'.

It is not necessary, usually, to have all the recognised symptoms, signs or test results to make a diagnosis of the clinical syndrome. The observations may be given different emphasis so that some features of the syndrome may be more heavily weighted than others with the result that sometimes patients may be given a diagnosis on very little evidence. Furthermore, many of the symptoms, signs and even test results may be found also in other 'clinical syndromes' (the diagnosis of 'complex regional pain syndrome' (CRPS) is a good example of this), thus making a specific diagnosis more difficult and highly subjective. In some cases it may not be considered possible to confirm the diagnosis until the response to treatment is known. In such situations it is difficult to have full confidence in the accuracy of the initial diagnostic process.

The variations detected in the types, number and severity of symptoms, signs, tests and even responses to treatment are usually attributed to variations in the disease process rather than in the patient. If the level of 'complaints' made by

the patient is high, this is usually attributed to long term personality characteristics and is often ignored. Failure to respond to treatment following 'diagnosis' usually leads to re-evaluation to assess the diagnosis. This will usually entail at least further investigations in the hope of 'turning up something'. It may also lead to repeated attempts at treatment that unfortunately have a high chance of failure. This in turn may lead to different diagnoses from doctor to doctor, which is very confusing for patients.

If patients cannot be classified into a known syndrome or diagnosis and fail to improve following several trials of treatment, they may end up with the label of 'hysteria', 'hypochondriasis' or 'functional overlay'. The difficulty may be to do more with limitations of the classification system than with the nature of the patient's response to illness. Nonetheless, some patients are left with no clear diagnosis and, having failed to respond to treatment, are discharged without the prospect for any further treatment and told to 'learn to live with it'. It can be seen how strict adherence to the disease model of illness can leave patients without an adequate explanation for their pain, may offer no prospect of helpful treatment and may lead to the suggestion that their fundamental problem is one of mental attitude or psychiatric disorder.

### Failure to recognise the importance of variability in response to pain

There is a large variation of self-report of symptoms with (apparently) the same pathology and recent research has demonstrated large variation in complaints of pain with specific pathology. In one study (Croft, personal communication) only 57% of patients with demonstrable major arthritis of the hip joint as identified by X-ray complained of pain. A recent magnetic resonance imaging (MRI) study of the lumbar spine found that 76% of asymptomatic volunteers demonstrated a disc herniation at one or more levels (Boos et al 1995). There is clearly no direct relationship between disc abnormalities and pain. The reasons for this are not fully understood, but the data suggest that the disc bulge is often not

the pain source. Without doubt psychological factors are important influences on the perception of pain, decision to consult and response to treatment, but it is clear that the physical basis of pain is still inadequately understood. Recent research suggests that ascription of all patient variability to the 'mental realm', in the sense of discrediting it, would appear to be extremely unwise; yet this has been an inevitable consequence of overstrict adherence to the narrow medical model.

### Reconceptualisation of reaction to pain and illness

Since the 1920s, and particularly between 1940 and 1960, psychologists have been developing a specific interest in the determinants of human behaviour. Experimental studies (initially on animals) have led to a science of human behaviour in which the *context* in which a behaviour is observed is considered to be of major importance. In understanding the behaviour, sociologists articulated concepts such as the 'sick role' to describe certain patterns of response to disease and incapacity. Terms such as 'consulting behaviour' (currently in vogue in the context of health economics) entered professional parlance. Studies of psychological disorder meanwhile identified functional and dysfunctional responses to the stresses and strains of life. A range of psychological features are associated with both the experience of pain and the reaction to it (Ch. 2). Recognition that understanding a patient's pain behaviour, thoughts and feelings are necessary to understand the patient's pain has led to a move from the disease model of pain to an illness model that recognises the importance of physical and psychological factors in pain. Further considerations of consulting behaviour, reaction to illness and socioeconomic factors have led to biopsychosocial models of disability (Ch. 5) (Waddell 1992) and multidimensional approaches to treatment (as evidenced by interdisciplinary pain management programmes).

Acceptance of the complexity of the nature of pain with adoption of newer theoretic models is of more than intellectual importance. Models

affect clinical practice, as will be illustrated in the following three cases. Simply put, the bio-psychosocial model represents an alternative to the disease model of illness and pain.

### FROM THEORETICAL MODELS TO CLINICAL PRACTICE

Patients demonstrate a wide range of responses to acute pain (ranging from the understated or stoical to the highly florid and dramatic). In most cases of acute pain, the cause of pain will be clear, the diagnosis is unambiguous and the only issue of concern will be matching up the analgesic strategy with the patient's particular response. Marked pain behaviour may or may not accompany the pain experience. Usually, particularly with episodes of acute pain, treating the cause of the pain successfully will also have a significant effect on the 'pain behaviour' and the latter becomes of little concern. Analgesia may be effective even when behaviour is quite bizarre. This may be taken to confirm the view that all pain behaviour, however bizarre, is secondary to nociception and consequently that targeting treatment solely at the nociceptive source is all that is required to manage both the pain and the consequent behaviour. If the treatment of the pain does not change the pain behaviour then the pain is often regarded as psychological or imagined. Although this may not be made explicit to the patient, the patient often picks up such implications from the behaviour and manner of the treating staff.

In order to emphasise the importance of the interpretation of pain behaviour, we have posed two theoretical questions about pain behaviour and offered some alternative answers, which are further illustrated by case examples.

Each strategy represents a way of understanding the patient's needs and reflects the model of pain adopted by the clinician.

Read Case studies 1.1–1.3. Which answer do you consider to be appropriate in each case? Consider each option in terms of its recognition of pain and distress and whether a narrow medical or a cognitive-behavioural approach is being adopted.

**Case study 1.1** Straightforward case of uncontrollable pain behaviour that stops as soon as an epidural is working

Mrs G. was 32 years old and having her first baby. She had been admitted during the night having started in labour. Her contraction pains had increased steadily over the early morning, but she was still early on in labour. She became increasingly distressed by her pain and by mid morning was literally screaming with pain. She became very restless and difficult for the midwives to manage. Clinical examinations were virtually impossible and she began to become progressively agitated. She became abusive, shouting and swearing. She would not let anyone near her to perform a vaginal examination to assess progress. She started throwing the Entonox around saying it was useless but would not listen to any instructions that would enable her to use it more successfully.

With much difficulty an epidural was successfully given. Mrs G.'s behaviour changed rapidly as the epidural became effective. Assessment was completed and there appeared to be no obstetric problems or cause for alarm. She apologised for her outburst and her labour continued uneventfully.

**Case study 1.1**

Although pregnancy is not normally painful, delivery usually is. Large variation in the pain of labour is well recognised. Some mothers appear to experience little or no pain whereas others have a considerable amount of pain and require significant intervention with various analgesic techniques.

If there are problems with delivery, such as obstructed labour, most if not all clinicians would expect the mother to experience more pain than normal. In fact an increase in the complaint of pain is one of the features that is specifically observed as an indication of possible trouble. An increase in pain complaints and pain behaviour (rolling around, restlessness, etc.) will lead the carers to make further investigations.

Pain behaviour during delivery can be quite bizarre even if there are no major problems with delivery. Such behaviour can make the management of even the most straightforward delivery almost impossible. The mother may become so agitated and restless that it may be impossible to carry out a proper evaluation. The marked pain behaviour is frequently attributed to culture, personality type, low pain threshold or anxiety.

However, it is well recognised that if the 'pain signals' are prevented from getting to the brain the bizarre behaviour as well as the pain will usually stop, rendering the patient much easier to assess and manage. Mothers certainly like epidural analgesia because it is generally effective and preferable to alternatives, but perhaps the most important reason for the rise of epidural analgesia is that the midwives like it. It not only makes even the most 'difficult' mother much more easy to deliver but more importantly it simplifies assessment and monitoring of progress. Any other complaints can be attributed to the patient's psyche rather than nociception.

This 'model' reinforces the idea that behavioural and other responses to pain are only secondary so that if the source of the pain is identified and isolated from the brain then not only should the pain get better but so also should the bizarre behaviour. All efforts should be directed towards identifying the source of pain and eliminating the pain. There is no need to tackle the pain behaviour directly. Both objectives may be reached simply by concentrating on finding and treating the cause of the pain.

**Case study 1.2** Marked pain behaviour that is not helped by an epidural

Mrs H. was 28 years old and was making very slow progress in labour. Her previous labour had also been difficult. She complained of a lot of pain, but this had not responded well to the usual pethidine. The pain had reached unbearable levels and she pleaded for something to be done. An epidural was given. The siting of the epidural, however, was difficult and painful and Mrs H. asked for a consultant to be brought to complete it. Not only was a consultant not available but unfortunately the epidural did not work perfectly. She continued to complain, demanded to know why it hadn't worked and requested that something further be done.

Mrs H. became even more agitated and started to complain about the soreness of the drip site, the epidural catheter in her back and a number of other things. The staff gradually became disaffected with her and began to ignore her complaints, giving them progressively less credence.

Unfortunately, when Mrs H. later became extremely distressed, the staff dismissed her pain complaints as being 'rather exaggerated'. It transpired that she was in obstructed labour and in fact needed an emergency caesarean section.

### Case study 1.2

This case illustrates that, even though some pain behaviour may be considered as not directly due to nociception, there is little doctors and others can do since the excessive pain behaviour is considered to result from factors outside their control (such as the patient's cultural background, pain threshold or mental make-up). Such ascriptions can of course simply illustrate prejudice on the part of the staff.

The treating doctors and nurses may become increasingly dismissive in their attitude to the patient's complaints and miss important organic causes of increased pain. Patients may feel the need to impress the doctor even more to take their pain complaints seriously. This may be reflected in more florid pain behaviour.

**Case study 1.3** A patient with profound pain behaviour and little to find in the way of objective signs of nociception

Mr A. presented with chronic low back pain and a high level of disability. There were no positive neurological findings to suggest a prolapsed intervertebral disc or any major structural pathology. Apart from guarded movements and some behavioural responses to examination there was little else to find. Thorough physical examination and a review of health did not reveal any other problems. X-rays of lumbar spine were reviewed and a diagnosis of degenerative spinal disease was made. The degeneration was considered to be the principal cause of his pain.

Mr A. was very angry as he had been told in the past that there was nothing wrong with him. He was convinced that he had a serious problem with his back. He could walk only short distances with the aid of two walking sticks and spent most of his day resting because any activity caused intense pain. He had undergone a number of treatments without any relief. His doctors had given up on him.

### Case study 1.3

The narrow medical model is particularly unhelpful in assisting the management of patients with pain that is persistent, intractable or unexplained. In such situations, there seems to be even less association between patients' complaints, their

behaviour and the level of 'nociceptive' input. However, doctors and others frequently persist none the less with the narrow medical model (whether out of prejudice, laziness or simply habit). Such patients are often treated by most doctors and therapists as if there is a nociceptive cause even if there is no evidence to support this. Treatment may be instigated on the basis that there *may be* a nociceptive source, however unlikely.

Despite the lack of evidence of a link between degenerative spine disease and pain (as reported above), patients are often told that their pain is due to degenerative disease or disc bulges. They are given an explanation that reinforces their view of a one-to-one relationship between physical damage and pain. (They may then be unresponsive to pain management with its emphasis on self-help.)

When no identifiable cause can be found, doctors may change the way in which they relate to the patient. Patients' behaviour may become the focus of attention. If there happens to be a compensation claim, then exaggeration, faking and malingering will be considered. If patients

#### Q Why do some patients exhibit more pain behaviour than others?

- A
1. They may be frightened of the pain, and emphasise their discomfort lest it is underappreciated (and under treated).
  2. They may have suffered poorly controlled pain in the past.
  3. It may be part of their style.
  4. They may have learned to behave like this.

#### Q How do clinicians respond to pain behaviour and why?

- A
1. They may disregard the behaviour as 'random noise', simply ignore it and continue to treat with analgesia.
  2. They may seek to alleviate the distress and ignore the requests for analgesia.
  3. They may seek to alleviate the distress as part of their overall management.
  4. They may give the patient inadequate treatment and reject them.

weep or intimate they cannot cope then psychiatric illness, depression or personal inadequacy are often inferred. The pain may be dismissed as unworthy of attention or even regarded as imaginary. Most patients will identify such changes in perspective even if it is not communicated directly to them by the way in which they are treated. As one might expect, this serves only to increase the level of distress. Patients may find themselves being discharged with little more in the way of help than being told that they 'will have to live with it'. Unfortunately they are frequently given little advice on *how* to 'live with it'.

## DEVELOPMENT OF IATROGENIC DISTRESS AND DYSFUNCTION

Chronic pain and pain-associated incapacity can become progressively distressing for patients. The level of distress can be made even worse by the way in which they are managed by their doctor.

### Influence of symptom presentation on the treating doctor

Without a definitive diagnosis it is difficult for doctors to form a clear treatment plan. Doctors often do not know what to do with the patient and this may lead them to adopt a number of strategies:

- further or repeated investigation on the assumption that something 'has been missed'
- referral to another/different pain specialist
- a trial of treatment on the 'assumed' best bet diagnosis
- communication in body language, if not explicitly, that the problem is psychiatric and possibly even referral to psychiatry
- no further offering of assistance.

The very fact that the patients' symptoms and signs do not match with the traditional model of the illness can have a subtle effect on the way

that doctors communicate with and manage them. Firstly it colours doctors' beliefs about the veracity of the complaints. Doctors will tend to be dismissive of 'non-organic' findings. They will tend to ignore them at best or regard them as signs of malingering, faking, secondary gain or psychiatric problems at worst. Such 'behavioural findings' are often described as 'bizarre'.

Doctors, having made certain judgements about patients and their illnesses, begin (unwittingly) to communicate this to the patient. This is done in the way they talk to the patient, their (the doctor's) non-verbal behaviour and sometimes what they explicitly say to the patient. Often doctors are not aware that they communicate anything to the patient, but our experience has shown that most patients who we have seen for assessment for pain management feel that it has been implied by their previous doctors that 'it is all in the mind'. This, of course, colours how patients interact with their doctors.

### Effect on the patient of the doctor's 'difficulties'

The fact that the doctor appears not to make any sense of the physical findings is quickly perceived by the patient, who is then likely to lose confidence in the doctor. From the patient's point of view the doctor does not understand their problem or does not know enough about it. This in turn colours the way the patient communicates with the doctor. If patients suspect that their doctor do not believe them they may be reluctant to disclose more worrying symptoms. Furthermore, the patient will be less likely to accept the doctor's formulation or recommendations.

Unfortunately patients may have no one else to turn to. If doctors order some more physical tests in the unremitting search for a purely physical explanation of patients' problems, then they will tend to go along with it, hoping that something will be found. Usually such hopes are dashed, the patient becomes even more distressed and the doctor-patient relationship is

significantly damaged. This experience may be repeated with other doctors until patients then begin to lose faith in all doctors. They have become damaged and distressed by the process of seeking treatment. Their pain persists and they may believe they have to try even harder to persuade specialists that their pain is not 'all in the mind'. Unfortunately, the attempt to convince them often enhances their pain behaviour and therefore makes it even more likely for other doctors to be dismissive, especially if the patient has been previously described in their case notes as showing 'functional overlay'.

It can be seen how an attempt to understand chronic pain in terms of the narrow medical model not only fails to do justice to the complexity of the problem, but can be harmful to the patient and, in many cases, is probably responsible for a considerable proportion of the distress associated with chronic pain problems.

## CONCLUSION

In so far as we can tell, pain has always been an aspect of the human condition, but its precise

characterisation has been elusive. It has been regarded both as a sense and as an emotion, but prior to the time of Descartes there does not seem to have been any clear recognition that there was central processing of pain signals in the brain. The role of sensory transmission and central registration in the brain of pain signals was recognised by Descartes, but the 'divorce' of the mind from the body, which was explicit in his Cartesian dualism, posed considerable difficulties in explaining the relationship between the experience of pain and underlying physiology. The advent of Melzack & Wall's gate control theory of pain in 1965, however, has led to developments in both the physiology and the psychology of pain such that modern pain management requires consideration not only of the perception of pain, but of the reaction to it (whether behaviourally, cognitively or emotionally). The move from unidimensional explanations to multidimensional perspectives offers not only a formidable intellectual challenge but also a wonderful opportunity to improve our understanding of chronic pain and its management.

## KEY POINTS

- Chronic pain differs from acute pain and has to be understood in terms of a complex interplay between physical and psychological features.
- The narrow disease model needs to be replaced with a broader illness model.
- Decision making and management are coloured by the doctor's own implicit model of pain.
- Inconsistencies in diagnosis and prescription of ineffective or ineffectual treatments may profoundly distress patients, thereby confounding their management.
- Adequate formulation of the patient's problem requires a multidimensional perspective.
- Chronic pain patients require illness management not just disease management.
- Proper management of chronic pain requires recognition of the interplay between physical and psychological factors and the socioeconomic context in which the consultation occurs.

## REFERENCES

- Bonica J J 1990 History of pain concepts and therapies. In: Bonica J J (ed) *The management of pain*, 2nd edn. Lea & Febiger, New York, ch 1
- Boos N, Rieder R, Schade V, Spratt K F, Semmer N, Aebi M 1995 The diagnostic accuracy of magnetic resonance imaging, work perception and psychosocial factors in identifying symptomatic disc herniations. *Spine* 20:2613–2625
- Descartes R 1664 *L'homme*. E Angot, Paris
- Dubner R 1997 Neural basis of persistent pain: sensory specialisation, sensory modulation and neuronal plasticity. In: Jensen T S, Turner J A, Wiesenfeld–Hallin Z (eds) *Progress in pain research and management (Proceedings of the 8th World Congress on Pain)* vol. 8. IASP Press, Seattle, pp 243–257
- Flor H, Birbaumer N 1994 Focus article: acquisition of chronic pain. Psychophysiological mechanisms. *APS Journal (Pain Forum)* 3(2):119–127
- Foster M 1901 *Lectures on the history of physiology during the 16th, 17th and 18th centuries*. Cambridge University Press, Cambridge
- Melzack R 1996 Focus article: gate control theory: on the evolution of pain concepts. *Pain Forum* 5:128–138
- Melzack R, Casey K L 1968 Sensory, motivational and central control determinants of pain. In: Kenshalo D R (ed). *The skin senses*. Charles C Thomas, Springfield IL, pp 423–439
- Melzack R, Wall P D 1965 Pain mechanisms: a new theory. *Science* 150:971–979
- Melzack R, Wall P D 1983 *The challenge of pain*. Basic Books, New York
- Ohrbach R, McCall W D 1996 The stress–hyperactivity–pain theory of myogenic pain. Proposal for a revised theory. *Pain Forum* 5:51–66
- Price D D, Mao J, Mayer D J 1997 Central consequences of persistent pain states. In: Jensen T S, Turner J A, Wiesenfeld–Hallin Z (eds) *Progress in pain research and management (Proceedings of the 8th World Congress on Pain)* vol 8. IASP Press, Seattle, pp. 155–184
- Roland P E 1993 *Brain activation*. Wiley-Liss, New York
- Travell J G, Symond D G 1993 *Myofascial pain and dysfunction: the trigger point manual*. Williams & Wilkins, Baltimore MD
- Virchow R 1858 *Cellular pathologie in ihrer Begründung auf physiologische und pathologische*. A Hirschwald, Berlin
- Waddell G 1992 Biopsychosocial analysis of low back pain. *Clinics in Rheumatology* 6:523–557
- Waddell G 1998 *The Back Pain Revolution*. Churchill Livingstone, New York, USA