Pain

- Pain is not a simple sensation produced by ‘pain receptors’ but is an interpretation of the activity of many receptors, which produce the multidimensional perception of pain.
- Signal detection theory can be used for distinguishing true analgesics, which modify $d^*$, from placebos and suggestion which modify $beta$.
- The Gate Control Theory says that pain occurs when the transmission (T) cells in lamina 5 of the dorsal horns of the spinal cord are stimulated by small (S) fibre activity. Pain can be stopped by inhibition of the T cells by large (L) fibre activity via the Substantia Gelatinosa (SG).
- Anxiety increases pain due to autonomic arousal activating small fibres, whereas stress can reduce pain, as in Stress Analgesia, via opiate mechanisms in the peri-aqueductal grey matter.
- Neurophysiological techniques for pain reduction, such as Dorsal Root Stimulation and Transcutaneous Nerve Stimulation, act by stimulating L fibres and SG cells.
- Psychological techniques for pain reduction act either by reducing S fibre input, as in Relaxation Therapy and Biofeedback, or by producing Cognitive Coping Skills by Imaginative Inattention or Somatization.

Pain is probably the commonest of symptoms and analgesics the commonest of treatments. Pain though is a complex phenomenon with physiological and psychological components. It is not a simple sensation, but is better regarded as a perception or an interpretation, and as a response or set of behaviours. Its physiology is complicated, but directly relevant to the psychology. ‘Pain receptors’, receptors whose activity would cause pain, do not exist, although particular receptors are especially important: there is Specialization but not Specificity.

Pain is difficult to describe. Unlike heat or pressure which vary only in degree, pain conventionally has three dimensions: Sensory, the particular quality (pricking, scalding, aching, etc.); Affective, the emotions produced (sickening, exhausting, frightening, etc.); and Evaluative, assessing its amount (discomfort, distressing, excruciating,
etc.). For research these components can be measured by the MCGILL PAIN QUESTIONNAIRE, and hence by appropriate scaling techniques we can say that, say, a STRONG pain is twice as bad as a MODERATE pain, and an EXCRUTIATING pain four times as bad as a strong pain. PAIN THRESHOLD, the mildest stimulus identifiable as pain, is distinguished from PAIN TOLERANCE, the worst stimulus which can be borne for a reasonable amount of time; the threshold is influenced mainly by physiological factors and the tolerance by psychological factors. Pains also vary in time; the acute, fast or phasic component of pain immediately after an injury has a different quality to the slower, chronic, or tonic component, the ‘real’ pain, which occurs a second or more after the fast pain (so that after hitting a thumb with a hammer there is a moment of detachment and realization before the onset of real pain). Social and cultural influences also affect pain; thus I may report less pain when having blood taken in a venepuncture demonstration in front of several students, than if nervous and on a trolley awaiting an operation. Such measurement problems are avoided in the laboratory by using a signal detection technique (see Chapter 2). Subjects indicate on a standardized scale the amount of pain experienced when infra-red energy is shone on a patch of black India ink painted on the forearm. Statistical analysis separates changes in ‘beta’ or threshold (a ‘stiff upper lip’, stoically denying the pain) from changes in $d'$ or pain intensity. Aspirin and Entonox (a mixture of nitrous oxide and oxygen) can be shown to be true analgesics, increasing $d'$ (sensitivity), whereas placebos or suggestion act by changing beta (the criterion for reporting pain), and hence are not true analgesics. The influence of social demands and expectations must make one wary in interpreting results for which such precise analysis is not available.

The philosopher Descartes (1596–1650) produced a commonsensical theory of pain (Fig. 20.1): ‘pain receptors’ are stimulated (by fire, in this case) and cause alarm bells to ring in the head, which are sensed as pain. However, such a simple model cannot account for many pain phenomena:

i. Pain occurs in the PHANTOM LIMBS that follow amputation.

ii. Paraplegic patients describe pains below the level of their spinal transection.

iii. Pain can be the result of damage to spinal cord or brain.

iv. Pain can be relieved by COUNTER-IRRITATION (irritating the skin by blistering or scarification), or by simple procedures such as rubbing.

v. Gross tissue damage need not be accompanied by pain. At the Anzio beach head in the Second World War many severely wounded soldiers reported little or no pain.

vi. Minor stimuli, such as the lightest of touch, can result in an exquisite pain (HYPERAESTHESIA) in CAUSALGIA, which develops after damage to peripheral nerves and neuroma formation.
None of these problems should occur if the Cartesian model is correct; either a connection is present or it is not, and the system is activated by tissue damage or it is not. No other condition is possible.

The brain contains no 'pain centre', and no area of cortex is specialized for it (unlike the other sensory modalities). Pain also differs from other senses in having powerful descending influences which modify transmission from the spinal cord; stimulation of peri-aqueductal grey matter produces a deep stimulation-induced analgesia, under which surgery is possible without anaesthetic.

In 1965 Melzack and Wall proposed their gate control theory (Fig. 20.2), which accounts for many troublesome aspects of pain. The transmission (T) cells (which are probably in lamina 5 of the dorsal horns of the spinal cord) project centrally, and are the input to an action system; when stimulated beyond a threshold they produce pain perception and its behavioural manifestations. Small, non-myelinated peripheral nerve fibres have excitatory projections onto the T-cell, and also onto cells in the substantia gelatinosa (SG) of the spinal cord, which in turn activate the T cell. Painful stimuli cause small (S) fibre activity and activate the T cells. Even when S fibre activity has ceased, the T cell continues to be activated by the SG cell, which is 'turned on', and pain therefore lasts longer than the stimulus itself. Large fibre inputs (L) also stimulate the T cell (as when light touch produces
causalgia) but also cause *inhibition* of T cells via the SG cells. L fibre stimulation (as in rubbing, or applying warmth or counter-irritation) therefore decreases T cell activity. T cell activity results from a balance between large and small fibre activity. In causalgia and phantom limb pain, neuroma formation produces excess small fibre activity, and hence pain. The entire gate system, of SG and T cells, can also be controlled from above; direct inhibition from peri-aqueductal grey matter closes the gate, and cognitive control can either close the gate, resulting in lack of pain after severe damage (as in the Anzio soldiers, who were so happy to leave the battlefield alive that their wounds seemed insignificant), or open the gate and produce increased pain sensitivity in anxious or neurotic patients. The model also explains the difference between the fast and slow pains, reflecting the different properties of fast (L) and slow (S) fibres.

The gate control theory is exciting in providing a coherent explanation of diverse phenomena, and in showing how psychological and cognitive influences can directly influence neurophysiology. Such psychological influences are manifold, and only a few examples can be given. Anxiety is a frequent accompaniment to pain, through several mechanisms. Firstly, anxiety increases autonomic arousal, which activates S fibres, and partially opens the gate to the T-cells. Secondly, events normally causing pain, such as punishment or
illness, are associated with anxiety, and the learned association of anxiety and pain then causes anticipation of pain, so that cognitive control opens the T-cell gate in expectation of a painful stimulus. Finally, minor symptoms, which occur all the time (a mild ache in the knee, for instance), and for which there is often an immediate explanation ('I banged the knee yesterday') can become exaggerated in the absence of an explanation ('perhaps this is bone cancer'), which produces anxiety, and the dull ache then becomes a mild pain, generating further anxiety, and more support for the sinister interpretation, producing more anxiety, more pain, etc.

Stress appears to have opposite effects to anxiety, reducing pain (STRESS ANALGESIA), probably via the peri-aqueductal grey matter. This makes good biological sense, since in an emergency pain could impair an organism's coping ability. In animals, experimental analgesia can be induced by repeated unavoidable foot shocks. There are at least two mechanisms, one involving the pituitary-adrenal system (probably based on endogenous adrenal medullary opiate-like peptides), and the other involving a non-opiate system. Stress analgesia can also be conditioned, as animals placed in a chamber where previously they have received foot shock analgesia show pain suppression without foot shocks; this suppression depends upon internal opiates, being blocked by naloxone, and showing cross-tolerance with morphine.

Pain control is central to medical practice and many techniques are available. Drugs are discussed at length in textbooks of pharmacology. However, drugs do not always produce adequate pain relief, or else in severe pain can only provide relief at doses that impair consciousness to a degree the patient finds unacceptable. Non-pharmacological techniques are available, many derived from the gate control theory. Surgical techniques, such as TRACTOTOMY, sever the spino-thalamic tract interrupting the T-cell output to the brain. Electrophysiological techniques use implanted electrodes to stimulate directly the dorsal spinal roots, or the dorsal columns, to decrease T-cell activity by stimulating L fibres. Less invasive, but also effective, is TRANSCUTANEOUS NERVE STIMULATION, in which electrical currents stimulate large peripheral nerve fibres directly, producing an immediate and continuing analgesia lasting several hours after stimulation, presumably due to SG cells being 'turned on'.

Effective psychological techniques use two major approaches, reduction of small fibre input and direct cognitive closure of the gate. Pains such as headache are associated with increased frontalis muscle tension producing excessive small fibre input. Tension, and hence autonomic S fibre activity, is reduced indirectly by RELAXATION THERAPY, which reduces all muscle activity, or directly by BIOFEEDBACK, in which electrodes over the muscle provide feedback about muscle tension and the patient learns to reduce activity in the specific muscle. Both techniques are highly effective when used properly. An alternative
Technique is to encourage COGNITIVE COPING SKILLS, so the patient learns to cope with their pain, to ignore it, and thus for the gate to close, pain actually to diminish. Several methods are used: in IMAGINATIVE ATTENTION, patients are trained to ignore pain by concentrating on her vivid images, such as being at a party, on the beach, or lying a hot soothing bath; in SOMATIZATION the patient focuses attention the pain in a remote manner, treating it as external to himself, in academic, intellectual fashion. Such techniques positively exploitence mechanisms, and are undoubtedly effective. Finally, psychological, drug, and stimulation treatments are combined in MULTIPLE THERAPY, the combination being more effective than the individual components.

The gate control theory has revolutionized medical approaches to in, emphasizing that pain is both a perception and a response, and once under psychological control, albeit within a biological context.