# PAIN – PART I

## *Fionn McKee SMTO*

*Part I of this article on 'Pain' deals with the physiology and anatomy of pain. In Issue 28 Fionn will continue with the more practical aspects of pain that Massage Therapists should appreciate, in light of the theory of the 'pain gate' model.*

### INTRODUCTION

*As each individual is unique, so is the pain experience. As Sir William Osler said, 'It is not nearly as important what illness a patient has, as what patient has the illness'.*

Pain, a component of the somatosensory system, is defined by the International Association for the Study of Pain as *'an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage'.*

The perception of pain serves as a defense system to maintain homeostasis, warning of injury that should be avoided and/or treated. Injured limbs actually inhibit voluntary movement to promote necessary healing processes. So essential is the painful response that those individuals born with congenital pain insensitivity do not react to pain, often resulting in severe, permanent tissue damage, and even premature death.

### GATE CONTROL THEORY

The 'Gate Control Theory of Pain' (from the psychologist, Ronald Melzack, & biologist, Patrick Wall, 1965) is one of the two most widely accepted theoretical conceptualisations of pain, the other being the 'Parallel Processing Model of Pain Distress' (Leventhal & Everhart, 1979). Gate control theory is based on the following propositions:

- The transmission of nerve impulses from afferent fibres, i.e. from the point of stimulation, to the spinal cord transmission (T) cells is modulated by a spinal gating mechanism in the dorsal horn.
- In some circumstances, the brain activates efferent fibres that influence the afferent transmission of pain sensations.
- The spinal gating mechanism is influenced by the relative amount of activity in large-diameter (L) and small-diameter (S) fibres: activity in large fibres tends to inhibit transmission (close the gate) while small-fibre activity tends to facilitate transmission (open the gate).
- The spinal gating mechanism is influence by nerve impulses that descend from the brain.
- A specialised system of large-diameter, rapidly conducting fibres (the Central Control Trigger) activates selective cognitive/neural processes that then influence, by way of descending fibres, the modulating properties of the spinal gating mechanism.
- When the output of the spinal cord transmission (T) cells exceeds a critical level, it activates the Action System – those neural areas that underlie the complex, sequential patterns of behaviour and experience, characteristic of pain.

The pain gate theory states that stimulation of faster nerve fibres (Type A beta which carry non-pain information) inhibits or creates a 'gate' in the dorsal horn cells of the substantia gelatinosa, inhibiting nerve transmission from the slower pain nerve fibres (Type C).

### THE ANATOMY AND PHYSIOLOGY OF PAIN

To experience any sensation, the following must be intact:

- sensory receptors
- sensory-conveying organs
- sense-interpreting centres in the brain
- associative memory centres in the brain

What is happening during a painful experience? Two classes of pain have been discovered: nociceptive pain and neuropathic pain. Neuropathic pain involves only direct injury to nerves in both the central and peripheral nervous systems.

Tissues containing specialised sensory receptors, called nociceptors, are activated by 'noxious' stimuli and have been discovered to exist in almost all multicellular animals on Earth, as well as in some bacteria. The signal for pain starts in one of the millions of receptors called nociceptors.

Nociceptors are most abundant in superficial areas of the skin, joint capsules, inside the periostea of bones, and around vessel walls. These are in all tissues and organs except the CNS. There can be as many as 1,300 in one square inch of skin.

Although nociceptors are involved with pain perception, stimulation of a nociceptor does not invariably result in a painful response. Unlike other sensory receptors, nociceptors become increasingly sensitive with continued stimulation. Damaged tissue releases prostaglandins and leukotrienes, chemicals that sensitise nociceptors. These nociceptors respond to stimuli that would not normally be interpreted as painful (this is sometimes called allodonia).

For example, sunburn pain is exacerbated by a gentle touch or breeze due to the hyperactivity of the nociceptors. Aspirin and similar drugs reduce sensitisation by inhibiting the production of prostaglandins.

Three types of nociceptors have been classified: A delta (I and II), medium-diameter cell bodies with lightly myelinated axons, and C fibres that have small-diameter cell bodies and non-myelinated axons. The myelinated A delta fibres conduct impulses faster (90 m/s) and thus communicate with the brain sooner that the C fibres (0.5 m/s). The sensory receptors of C and the A delta fibres are simply their bare nerve endings.

Further categorisation identifies nociceptors as mechanical, thermal, and polymodal. Mechanical nociceptors involve the activation of A delta, conducting at 5-30 m/s, fibres during the experience of intensive pressure. Likewise, thermal nociceptors are activated by extreme hot or cold temperatures (>45$^{o}$C or <5$^{o}$C), and also involve thinly myelinated A delta fibres.

Polymodal nociceptors are activated by high intensity mechanical, chemical, and thermal stimuli, involving non-myelinated C fibres. All classes of nociceptors are present on skin and tissues, and work together in forming the pain response. For example, one may initially experience a feeling of 'sharp' pain when hitting the thumb with a hammer, proceeded by a prolonging 'aching'. The first pain occurs when A delta fibres transmit information from mechanical and thermal nociceptors to the brain. C fibres transmitting polymodal nociceptors and responsible for the later, prolonged, aching experience.

A delta and C fibres travel through the pain gate and synapse with other nerve fibres in the marginal layer (lamina I) and the substantia gelatinosa (lamina II) of the superficial dorsal horn. The fibres then release a neurotransmitter, substance P, into the synaptic cleft, sending an impulse up to the thalamus, giving us a vague consciousness of pain.

The thalamus then sends two signals; one to the cerebral cortex and one back to the original location of pain to inhibit nociceptors from transmitting further, unnecessary pain impulses. The signaled cerebral cortex considers the tissue damage and transmits messages to both the limbic system (memory, learning and emotion regions) and the autonomic nervous system (ANS). The limbic system functions to increase or subdue pain by controlling the individual's emotional responses. Blood flow, pulse rate, and breathing are moderated by the ANS, therefore ANS assistance helps to provide an ideal environment for damaged tissue restoration.

Chemical messages transmitted by hormones also influence conduction of pain signals to the brain. The previously mentioned prostaglandins increase the frequency of impulses in addition to sensitising nociceptor nerve endings. Substance P stimulates nociceptors at the site of injury to intensify the incidence of pain sensation. For pain reduction, the hormones serotonin and norepinephrine are released to promote the liberation of endorphins by nociceptors. Thus, by the release of hormones both an individual's pain experience and perception are altered. This leaves additional room for variation in such experience among people, *which will be discussed in OTMS 28.*

As already mentioned, A delta and C fibres travel through the 'pain gate' before any further signal transmission occurs. The 'pain gate' is positioned in the dorsal horn at the base of the spinal cord. It serves to screen nerve impulses; the majority of impulses from a certain class of fibre are permitted to travel through the spinal cord and to the brain. For example, if touch/pressure impulses (A delta I fibres) outnumber painful impulses (A delta II fibres), then only the touch/pressure impulses are received by the brain.

This explains why massaging a pain site tends to reduce the perception of pain. By applying pressure to a very localised site, pain signals will not successfully pass through the 'pain gate' and hence little or no pain is perceived by the individual.

***Fionn McKee has recently completed the Scottish Massage Schools' Remedial and Sports Massage Diploma course in Edinburgh and is currently practising in Glasgow. He can be contacted by calling 0141 429 7204.***