Chronic pain: time for epidemiology

In calling for an epidemiological study of chronic pain, Dr Smith and his colleagues (April 1996 *JRSM*, p 181) identify two major difficulties: case definition and case identification. Clues to both are available when the neurophysiology of pain in the peripheral nervous system is reviewed.

'Pain' is a generic word. Wall[1] has described pain as a general reaction-pattern of three distinct behavioural phases—immediate, acute and chronic. Each phase may exist independently, or in combination with the others. Since these phases are separate physiological entities rather than facets of a single reaction, they must be defined and identified in any study.

Pain does not always signal tissue damage. Medical diagnosis traditionally regards pain as signals of actual or threatened tissue damage and equates it with the immediate phase, or nociception, via injury-sensitive A-delta and C fibres. This concept of pain is reinforced by the International Association for the Study of Pain's (IASP) definition of 'an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described by the patient in terms of such damage'.

The puzzle of pain. Doctors usually have no difficulty in recognizing pain that is caused by injury (for example, a fracture) or inflammation (such as rheumatoid arthritis), but they are perplexed by chronic pain conditions that have no apparent signs of tissue damage (such as headache, backache or 'tennis elbow'). Bewilderment arises because, in the absence of tissue injury or inflammation, perceived pain does not come from pain signals but is the consequence of abnormal function within the nervous system[2] when non-painful signals are overstated and misperceived as painful ones.

Peripheral neuropathy. This is a common functional disorder, since the spinal nerve root is particularly vulnerable to pressure, stretch and angulation (radiculopathy). In neuropathy, exteroreceptors and their pathways become excessively sensitive (or supersensitive[3]) and can give rise to neuropathic pain. When there is no injury or inflammation, chronic pain is almost invariably associated with motor, sensory, autonomic and trophic signs of peripheral neuropathy[3,4].

The shortened muscle syndrome. Peripheral neuropathy invariably affects the musculoskeletal system causing muscle contracture and shortening. Many chronic myofascial pain syndromes (from Achilles tendonitis to tennis elbow) are caused by the relentless pull of shortened muscles onto tendons and their attachments[5]. Muscle shortening across a joint can cause arthralgia, and eventually degeneration of the joint. Shortening of paraspinal muscles can compress a disc. This leads to a vicious circle. Pressure on a nerve root causes radiculopathy; this leads to pain and shortening in muscles; and shortening in paraspinal muscles further compresses the nerve root. At present, many musculoskeletal pain syndromes that rightly belong to the shortened muscle category are incorrectly labelled according to their various locations (for example, tennis 'elbow' is a condition that relates to the cervical spine).

Epiphenomenon of neuropathy. Confusion exists in the understanding of chronic pain because neuropathic pain is commonly misconstrued as ongoing nociception. Neuropathic pain does not signal tissue damage and nociceptors are not stimulated; pain is, instead, an epiphenomenon of neuropathy (just as fever is a feature of infection). Abnormally sensitive groups I and II proprioceptive fibres are probably responsible for deep muscle pain[6]. Since muscle shortening is not revealed by any imaging test, a unique and invaluable investigative technique is to dry-needle the muscle with a fine, flexible needle (as used in acupuncture)[7]. It is the only technique that can reveal deep muscle contracture.

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