The Physiology of Deep, Somatic Pain

By Nikolai Bogduk

This is part one of Nickolai Bogduk's article exploring our knowledge of pain physiology.

Contemporary knowledge about pain physiology is dominated by cutaneous pain, neuroma pain and neuropathic pain. The reason for this is understandable. The skin provides a target that can be stimulated in a controlled manner using a variety of stimuli – touch, pin-prick, heat, and applications of chemicals, both in experimental animals and in human volunteers. Cutaneous pain can be studied without invading the organism. Neuromas and nerve injuries can be induced at selected and desired sites and provide a known and isolatable source of nociception. Phenomena such as cutaneous hyperalgesia and receptive fields can be readily mapped because they are distributed across only a two-dimensional surface.

The irony is that epidemiologically, cutaneous pain, neuroma, and neuropathic pain are relatively uncommon. Far more common is deep, somatic pain, otherwise referred to as musculoskeletal pain for the reason that, to the patient, the pain seems to arise in muscles, bones or joints; it is felt deeply and definitely not in the skin.

For something as common as musculoskeletal pain, knowledge of its physiology is meagre compared to that of cutaneous pain. Although research into cutaneous pain has been critical in elucidating nociceptive pathways and control mechanisms, and although these principles might be applied to musculoskeletal pain, unless they have been explicitly demonstrated to apply, the possibility remains that different and distinctive processes might apply to musculoskeletal pain.

Certain obvious differences are immediately evident. Skin is exteroceptive, designed to respond to external physical stimuli such as heat and touch. Teleologically, there is no reason for deep somatic structures to be heat nociceptive in the same way as skin. It can be construed that the purpose of cutaneous nociception is to avoid or escape external, threatening stimuli; deep somatic pain cannot be escaped.

Since deep tissues lack touch transduction, there is no reason to expect they exhibit Aβ allodynia.

The Legacy

The history of research into musculoskeletal pain can be depicted graphically in three time lines. The earliest studies can be classified as clinical experimental studies, in which pain phenomena were studied in normal, human volunteers. These were then followed by anatomical studies, which pursued the histological substrates of deep, somatic pain. The youngest style of research has been animal experiments in which nociception from musculoskeletal tissues, as opposed to skin, has been studied. Each of these streams of research commenced at various times during the twentieth century, and has continued into the present time.

Another dimension of musculoskeletal pain research has been the target structure. Clinical studies have focused largely on

Much of our present understanding of the phenomenology of musculoskeletal pain can be traced to the work of Kellgren in the late 1930s. In an effort to understand musculoskeletal pain in patients he explored how deep somatic pain might be elicited in normal volunteers, where it was perceived, what it felt like, and what other features were associated with it.

Kellgren's first study was on referred pain from muscle. He demonstrated that noxious stimulation of muscle, with injections of hypertonic saline, produced pain that was diffuse and perceived remote from the site of stimulation. Moreover, in the limbs, muscle pain tended to be perceived towards the joint upon which the muscle acted. Stimulation of axial and paraxial muscles produced pain anteriorly in the trunk or abdomen or into the upper or lower limb.

Kellgren's most lasting and penetrating contribution, however, was in the study of spinal referred pain.

In an era when disc prolapse had just been discovered and spinal pain was ascribed to nerve root compression, Kellgren ventured a competing paradigm.
He showed that noxious stimulation of the interspinous ligaments, by injection of hypertonic saline, could produce referred pain in remote areas.\(^2\) Stimulation of thoracic ligaments produced pain in the posterior and anterior chest wall. Stimulation of cervical and lumbar ligaments produced pain in the respective limbs.

Kellgren’s experiment was not intended to demonstrate that interspinous ligaments were the source of back pain and neck pain. Rather, they established several principles:

1. Spinal pain could arise from noxious stimulation of intrinsic structures of the vertebral column.
2. Such stimulation produced referred pain in the trunk and limbs.
3. Referred pain could be produced by mechanisms other than nerve root irritation.
4. This referred pain was not neuralgic in nature, in that it was not shooting, burning or stabbing in quality, and not associated with numbness or paraesthesiae in the skin; rather, it was dull and aching in quality, diffuse and hard to localise in distribution, and perceived deeply, in which respects it resembled the complaints of many patients.
5. In order to distinguish this type of referred pain from pain caused by nerve root irritation or pain arising from viscera, it could be referred to as somatic referred pain. That term specified that the source of pain was in the somatic tissues of the body as opposed to viscera or nerves.
6. Somatic referred pain followed a segmental distribution that was not dermatomal in nature. Stimulation of successively lower spinal segments produced pain in successively more caudal regions of the body wall or limbs, but these regions did not correspond to the known dermatomes of the body. Kellgren believed this pattern to reflect a segmental pattern of innervation of deep tissues.

Kellgren’s report and interpretation were not well accepted, because they ran contrary to prevailing wisdom that referred pain must be caused by root irritation. Sinclair et al\(^3\) tried to reproduce Kellgren’s experiment and failed to produce referred pain to the limbs. They argued against his interpretations and submitted that his injections must have inadvertently stimulated nerve roots. In a contemporary essay on referred pain Sinclair and associates\(^4\) argued that referred pain was due to axonal branching in the periphery, and involved antidromic propagation of impulses to the referred zone, which then triggered pain in that zone, which was then propagated orthodromically back along the same nerve.

However, Kellgren’s observations were subsequently reproduced by Hockaday and Whitty\(^5\) and by Whitty and Willison,\(^6\) although the frequency and extent of referred pain to the limbs that they encountered was not as dramatic as that reported by Kellgren. Full corroboration was provided by Feinstein et al\(^7\) who published maps of referred pain that resembled those of Kellgren in extent but not in exact location.

In a short but inordinately influential paper Inman and Saunders\(^8\) firmly consolidated the concept of deep, somatic referred pain. The paper presented little information on methods beyond stating that deep somatic tissues – periosteum, ligaments, bone, joints and muscles, throughout the body were noxiously stimulated by scratching with a needle, drilling with a wire, or by injections of formic acid or six per cent saline; it presented no quantitative data; but it assertively declared profound results. The sensitivity of deep somatic tissues was ranked in the order – periosteum > ligament > joint capsule > tendon > fascia > muscle. Most influentially, the paper depicted maps of the dermatomes, the myotomes, and the sclerotomes of the body, in order to contrast their patterns. Dermatomes are the regions of skin innervated by individual spinal nerves, and myotomes are the regions of muscle innervated by a given spinal nerve. Sclerotomes were presented as the regions of bones, joints and ligaments purportedly innervated by the same spinal cord segment. The latter were declared to be the basis for somatic referred pain, and have been repeatedly quoted in the literature since. This paper was influential because it declared an attractive concept but its influence was inordinate because the maps of sclerotomes that it provided were idealised and not based on published quantitative data. The consistency of patterns of referred pain was not stipulated.

In 1950, Kellgren left the spine, and together with Samuel\(^9\) studied the knee joint. In normal volunteers they explored the sensitivity of different structures in the knee with a needle introduced through anaesthetised skin; in patients undergoing arthrotomy they studied the sensitivity of synovium; but in a dramatic experiment they opened the knee of Samuel in order to explore the sensitivity of the synovial membrane across its entire extent. They found the fibrous structures: ligaments and capsule to be nociceptive to mechanical and chemical stimulation, but the synovial membrane was largely insensitive to pin-prick, crushing with forceps, and...
chemical stimulation, except on a few occasions in isolated areas near the upper border of the patella and towards the sides of the joint.

Part two of this article will be featured in Massage Therapists Summer 2006 issue.

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References