Surgery for Low Back Pain

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Low Back Pain: Where Does the Pain Come From?

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Introduction

Patients with low back pain constitute a common patient group and can be divided into the acute group where the pain may be severe but short standing, and the group where the pain continues for a longer time and often influences many aspects of life. Patients with persisting low back pain, lasting longer than 3 months, are usually referred to as chronic [4], but perhaps a better expression for the condition is long-lasting low back pain (LLBP). One reason for using long-lasting instead of chronic is that in low back pain, as in conditions known to follow the patient for the rest of his/her life, a well-defined test does not set a precise diagnosis (compare with classic chronic diseases such as diabetes, heart failure and rheumatoid arthritis). Patients with LLBP suffer from more or less well-defined conditions that involve different anatomical structures and pathways in the pain system, and only 10–15% of patients with low back pain get a specific diagnosis [17].

There is a rapid ongoing development in surgical implants and surgical techniques, as well as suggested non-surgical treatment methods, for patients with low back pain. However, the lack of instruments to set a precise diagnose and/or identify the pain foci in many of these patients still remains. There are probably multiple reasons for the somewhat slow development of diagnostics compared to the rapid development in the treatment area. One reason for this might be the anatomy of the spinal structures with multiple flexible parts; another, the complexity of the nervous system where pain may arise from a direct influence of the peripheral and/or central nervous system as well as the stimulation of nociceptors located in different spinal structures. Hence, the slow development of diagnostic tools may also be caused by the fact that research in the area of diagnostics for lumbar pain is not only difficult and time consuming, but also not economically supported to the same extent as new treatment methods, where the economical potential can be defined more easily in a business perspective.

In this article possible pain sources for acute and chronic low back pain, as well as existing diagnostic tools to support or reject possible pain foci, are described. Further, the nervous system response and modulation mechanisms in response to long-standing pain, as well as psychological/personality factors influencing pain experiences, are discussed.

Intervertebral Discs

Intervertebral discs are today considered as the main pain foci in patients with long-standing or chronic low back pain. The disc is the largest mobile part of the three-joint system building a motion segment in the spine (one motion segment defined as two vertebrates with connecting disc and bilateral facet joints). The highest shear and fibre strains of the disc have been demonstrated to occur posterolaterally in response to combined movements [37]. It is, therefore, not surprising that disc deterioration often is seen at the posterior part of the disc as a posterolateral or central disc herniation, a disc bulging, or by an increased fluid content at the posterior border of the disc in MRI (high intensity zone, HIZ).

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Patients with disc herniations often report preceding low back pain before the onset of sciatic pain. This pain experience is suggested to be caused by stimulation of nerve endings in the annulus fibrosus due to the annular tear.

In parallel with investigations on mechanical problems in the spine, different inflammatory and signalling substances have been suggested to be of importance in the development and persistence of back pain. A number of experimental studies have demonstrated negative effects of disc tissue, and in particular, nucleus pulposus (NP) on nerve roots. NP can reduce spinal nerve root conduction velocity [32], induce nerve fibre degeneration, increase nerve fibre discharges [40], attract inflammatory cells [31] and induce increased intraneural capillary permeability [12]. Pro-inflammatory factors, which include cytokines (e.g. TNF and various interleukins), have been demonstrated to be present in disc herniation tissue [2]. High levels of pro-inflammatory mediators (IL-6 and IL-8) have also been found in disc tissue from patients considered to have discogenic low back pain undergoing fusion surgery [11].

In non-degenerated discs the presence of nerve fibres are detected in the absolute outer layers of the annulus fibrosus [33, 36]. These nerve fibres have been demonstrated to be both substance P-, calcitonin-gene-related peptide- (CGRP-) and vasoactive intestinal polypeptide- (VIP)- immunoreactive [25]. Nerve impulses signalling sensory information from the intervertebral disc have in animal studies been demonstrated to be conducted through the sinuvertebral nerve into rami communicantes to sensory neurons in more cranially located dorsal root ganglia.

In degenerated discs nerve endings have been found to extend into deeper layers of the annulus fibrosus [15, 27] and even into the NP [34]. The nerve fibres have been detected both in the anterior and the posterior parts of disc specimens following vascularized granulation tissue [25, 34]. The stimulation of these nerve endings may correlate with the dull chronic ache, exacerbated by the mechanical load of the spine, that is experienced by chronic low back pain patients and is often referred to as discogenic pain.

The main diagnostic tool today to detect disc degeneration is magnetic resonance imaging (MRI) where a number of signs as a decrease in water content, decreased disc height, disc bulging and/or indirect signs as vertebrae oedema can be detected. However, disc degeneration changes seen by MRI investigations can also be seen at high frequency in asymptomatic individuals [6, 7, 21].

Another tool that is widely used and debated is discography. The mechanism of discography involves the theory of increasing the intradiscal pressure for stimulation of mechanical nociceptors in the annulus fibrosus. Based on this assumption, discography has been suggested to be a tool for evaluating pain characteristics and the precise level of pain generation. However, concordant pain during a discography is not always combined with a fissured and ruptured disc on discography/CT discography [28] and discography has not conclusively been demonstrated to be helpful to increase the result of spinal fusions in chronic low back patients [13, 14].

Another way to use discography is to look at the decrease in pain after local anaesthetics are injected; however, studies in this field are not conclusive.

Facet Joints

In the normal capsule of the facet joint both sensory and autonomic nerve fibres have been detected, and thus, the facet joint capsule has a structural basis for pain perception [38]. As in all joints, osteoarthrosis of the facet joint may occur and is more common in patients with disc degeneration. An inflammatory reaction is common in joints with osteoarthrosis and may stimulate nociceptors. Also mechanosensors may be influenced if the joint destruction leads to changes in the mobility of the joint such as in degenerative spondylolisthesis. Facet joint injections are sometimes used in elderly patients with facet joint osteoarthrosis to decrease low back pain with a minimal procedure. Measurement of nitric oxide has been performed in other osteoarthritic joints such as the knee joint and temporomandibular joint, and a relationship between NO and osteoarthrosis, as well as pain, has been observed [23, 39]. Recently, increased concentration of NO in, or in close relation to, the facet joints was also demonstrated in patients with facet joint osteoarthritis and low back pain [8].

If measurement of inflammatory markers or pain markers can be used as diagnostic tools to diagnose pain originating from the facet joints or some other part of a painful spinal segment is not yet clear.

Muscles

Most muscles are well innervated and changes in their normal function may contribute to the pain experience both in acute and long-standing low back pain.

In acute low back pain the muscle spasm is often extensive and has been suggested to be the main reason for the, often quite severe, pain that may hold back these patients from almost all movements the first day(s). However, if the muscle response in acute low back pain is a primary or a secondary event remains unclear.

The activation patterns for the trunk muscles (both abdominal and lumbar) have been demonstrated to be changed in patients with chronic low back pain in both experimental and clinical studies [16, 22]. If this, in concordance with the spasm in acute pain, is a response aiming to stabilize a degenerated spinal segment by decreasing movement and pain (pain-adaptation model) or if the changed muscle function contributes to the pain (pain-spasm-pain model) is, however, unclear [41].

Ligaments

Nerve fibres have been detected in the posterior-longitudinal ligament (PLL) [25], but not in some of the other ligaments such as the ligamentum flavum. The disc and the PLL have a close anatomical relationship, and it is reasonable to believe that a gradual loss of disc height causing bulging of the posterior part of the disc will influence the PLL and thus initiate stimulation of nociceptors in the PLL. This may be caused by stretching or by chemical factors released from the disc. However, little is known of the role of PLL and other ligaments in pain signalling and no diagnostic tools to look at these structures in vivo in regard to pain signalling exist.

Vertebraes

Nociceptors have been demonstrated to be present in bone structures also. Compression fractures in the spine are a common cause of pain in the spine in older and/or osteoporotic patients. These can occur without trauma and can be visualized with x-ray, CT or MR scans.

In patients with low back pain and disc degeneration, changes in the vertebrae are also often noticed in MRI. Signal changes in the bone marrow of the vertebral body adjacent to a degenerated disc are called Modic changes and are suggested to be oedema caused by micro fractures or inflammatory changes [3]. Exactly how this influence nociceptors is unclear; however, some correlations between Modic changes and pain symptoms have been described [24, 26].

Nervous System Involvement and Adaptation

Free nerve endings present in various spine structures respond to mechanical pressure/deformation and chemical stimuli just as in other organs. The pain impulses are conducted through myelinated A delta and unmyelinated C fibres to the dorsal root ganglion and continues via the spinothalamic tract to the thalamus and gives rise to the pain experience when reaching the somatosensory cortex.

Inflammatory substances from a deteriorated disc or from facet joint arthrosis may influence nerve roots and DRG, as well as nociceptors in different surrounding structures. Biochemical and mechanical factors may also act together to increase direct negative effects on nerve roots. Nerve tissue damage may also by itself increase inflammation by stimulation of macrophage infiltration and increasing the number of activated T cells, which may add to the pain [1, 29]. Several biomarkers associated to pain and/or neurotransmission have been studied in CSF and serum in patients with chronic low back pain and also in patients with sciatica [5, 9, 10, 18]. However, no clear diagnostic help has been demonstrated by the use of biomarkers in patients with low back pain.

When handling pain patients, one always has to bear in mind that pain perception is a subjective experience. The function of pain perception is primarily the detection of tissue damage, a mechanism extremely important for the survival of the individual, but may also cause major clinical problems. In response to stimulation of free nerve endings, the somatosensory system may increase its sensitivity resulting in a

non-functional way to respond – normally innocuous stimuli result in an amplified response (peripheral sensitization).

Pain impulses may also be modulated at higher centres, both at the spinal and the supraspinal level (central hyperexcitibiability). The first possible level for impulse modulation is the DRG. The changed magnitude of perceived pain is often referred to as neural plasticity and is considered to play a critical role in the evolution of chronic pain.

Upregulation of chemokines within the nervous system, which can be released by astrocytes or microglia, may also contribute to pain modulation and the development of chronic pain [1]. Augmented central pain processing has been demonstrated in chronic low back pain patients with fMRI [19]. Hyperalgesia and increased neural activity measured by fMRI after thumbnail pressure were seen in this patient group when compared to controls. Chronic low back pain patients have also been demonstrated to have brain chemistry alterations demonstrated by proton magnetic resonance spectroscopy. A reduction of *N*-acetyl aspartate and glucose has been found in dorsolateral prefrontal cortex in these patients [20].

The way people "think" about chronic low back pain has also been suggested to influence movements, and it has been demonstrated that pain physiology education can markedly alter brain activity, registered by fMRI, during performance of a specific task [30].

The Psychosocial Aspects of Chronic Pain

Since pain is a subjective experience, it can, as with most experiences, be affected by psychosocial factors. Low back pain patients with certain psychological characteristics such as pain-related anxiety and low acceptance of pain have been demonstrated to be less sensitive to treatment [35]. On the other hand, long-standing severe pain may also affect a person psychologically and it is, therefore, difficult to ascertain the role psychological factors play in the development of chronic pain. However, most authors agree that psychosocial factors contribute to the individual perception of long-standing pain and coping with it.

Summary

In summary, many structures in the spine can theoretically contribute to acute low back pain as well as long-standing low back pain. The intervertebral disc, the facet joints and the muscle are the most likely local actors for initiation and maintenance of low back pain (both acute and long-standing).

There are mechanical as well as biological rationales behind the theory that the disc is a tissue of major interest in low back pain. However, when it comes to diagnostics, investigations/test(s) to detect disc degeneration do exist, but are still inconclusive in pointing out a certain disc as the pain foci. As for the facet joints, ligaments and the vertebras, still less is known regarding their role in low back pain patients. In patients with low back pain, changed activity of the muscles localized around the spine is common; however, whether this is a secondary response or not is less clear. When the complexity of the nervous system and psychological factors is added, the need for more research and better diagnostic tools in this patient group becomes obvious.

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