Chronic Nonspecific Back Pain

Why is chronic back pain so important?

Chronic nonspecific back pain is very common. Few of us never have back pain; most people have periodic back pain and some have chronic back pain. Chronic back pain is mostly located in the lumbosacral and posterior neck region.

In industrialized countries, low back pain (LBP) is the most common cause of activity limitation in persons younger than 45 years. It is defined as pain in the low back that persists longer than 12 weeks. Although acute LBP has a favorable prognosis, the effect of chronic LBP and its related disability on society is tremendous. For example, approximately 80% of Americans experience LBP during their lifetime. An estimated 15–20% develop protracted pain, and approximately 2–8% have chronic pain. Every year, 3–4% of the population is temporarily disabled, and 1% of the working-age population is disabled totally and permanently, because of LBP. It is estimated that the costs of LBP approach $30 billion annually in the United States.

Why is the “6-week rule” so important?

Most normal connective tissues heal within 6–12 weeks unless instability or malignant or inflammatory tissue destruction is present. Therefore, in any prolonged back pain, these pain etiologies should be ruled out. Pain that radiates to the legs in a radicular pattern should be thoroughly investigated, especially if sensory or motor deficits are noted in the patient

When is periodic back pain “normal” and chronic back pain “not normal”?

The lumbar spine can support heavy loads in relationship to its cross-sectional area. It resists anterior gravitational movement by maintaining lordosis in a neutral posture. Unlike the thoracic spine, the lumbar spine is unsupported laterally. The intervertebral disks are composed of the outer annulus fibrosis and the inner nucleus pulposus. The outer portion of the annulus inserts into the vertebral body and accommodates nociceptors and proprioceptive nerve

endings. The inner portion of the annulus encapsulates the nucleus, providing the disk with extra strength during compression.

The nucleus pulposus of a healthy intervertebral disk constitutes two-thirds of the surface area of the disk and supports more than 70% of the compressive load. Until the third decade of life, the gel of the inner nucleus pulposus is composed of approximately 90% water; however, the water content gradually diminishes over the next four decades to approximately 65%. Until the third decade of life, approximately 85% of the weight is transmitted across the disk. However, as disk height decreases and the biomechanical axis of loading shifts posteriorly, the posterior articulations (facet joints) bear a greater percentage of the weight distribution. Bone growth compensates for this increased biomechanical stress to stabilize the trijoint complex.

Therefore, to some extent, hypertrophy of the facets and bony overgrowth of the vertebral endplates constitute a normal physiological reaction to the age-dependent degeneration of the disks to stabilize the spine. Only in patients with inadequate “self-stabilization” do these changes contribute to progressive foraminal and central canal narrowing. Spinal stenosis reaches a peak later in life and may produce radicular, myelopathic, or vascular syndromes such as pseudoclaudication and spinal cord ischemia. LBP is most common in the early stages of disk degeneration and “self-stabilization.”

What types of pain may be identified?

Specific pain

Back pain that lasts longer than 3 weeks with major functional impairment should be thoroughly evaluated to identify serious causes, inflammation, instability, or local compression. It has to be repeated that generally the proportion of back pain patients with specific pain is rather low (around 5%). On the one hand, the pain causes mentioned above should never be overlooked, but on the other hand, over interpretation of radiographic results should be avoided. As a rule of thumb, unrelenting pain at rest should suggest a serious cause may exist. Imaging studies and blood workup are usually mandatory in these cases and in cases of progressive neurologic deficit, too. Other historical, behavioral, and clinical signs that should

alert the physician to a non-mechanical etiology will require diagnostic evaluation. Evidence for specific back pain might be the following diagnostic “red flags”:

- Colicky pain or pain associated with visceral function (or dysfunction).
- History of cancer or fatigue, or both, and weight loss.
- Fever or immunosuppressed status.
- History of older age and osteoporosis (with increased risk of fractures).
- Progressive neurological impairment or bowel and/or bladder dysfunction.
- Severe morning stiffness as primary complaint.

Nonspecific Pain

Evidence for nonspecific back pain might be the following diagnostic “red flags” (nonorganic signs and symptoms):

- Dissociation between verbal and nonverbal pain behaviors.
- Use of affective pain descriptions.
- Little pain modulation, with continuous high pain intensity.
- Compensable cause of injury, out of work, seeking disability (conflict of interest between compensation and wanting to be cured).
- Signs of depression (having difficulty falling asleep, waking up early in the morning, loss of interest, and loss of energy and drive, especially in the fi rst half of the day) and anxiety (continuous worrying and restlessness).
- Psychoactive drug requests.
- History of repeated failed surgical or medical treatments.

Diskogenic pain

Many studies have demonstrated that the intervertebral disk and other structures of the spinal motion segment can cause pain. However, it is unclear why mechanical back pain syndromes commonly become chronic, with pain persisting beyond the normal healing period for most soft-tissue or joint injuries. Inflammatory factors may be responsible for pain in some cases, in

which epidural steroid injections provide relief. Corticosteroids inhibit the production of arachidonic acid and its metabolites (prostaglandins and leukotrienes), inhibiting phospholipase A2 (PLA2) activity. Levels of PLA2, which plays a role in inflammation, are elevated in surgically extracted samples of human herniated disks. Furthermore, PLA2 may play a dual role, inciting disk degeneration and sensitizing annular nerve fibers.

Radicular pain

Surprisingly, the pathophysiology of radicular pain is unclear. Likely etiologies include nerve compression because of foraminal stenosis, ischemia, and inflammation. Often, the cause of radiculopathy is multifactorial and more complex than neural dysfunction due to structural impingement. In clinical practice, structural impairment is usually considered to be responsible, if inflammation is found. Therefore local epidural, often para-radicular, steroid injections are used for therapy, although their long-term effect is rather questionable.

Facet-joint pain

The superior and inferior articular processes of adjacent vertebral laminae form the facet or zygapophyseal joints. They share compressive forces with the intervertebral disk. After trauma or with inflammation they may react with pain signaling, joint stiff ness, and degeneration. Interestingly, there is no strong relation between radiographic imaging results and pain; therefore, the diagnosis is strictly clinical (pain radiating to the buttocks and dorsal aspects of the upper limb, provoked by retroflexion of the back and/or rotation). Unfortunately, long-term effects of local steroid injections into the joint or into the vicinity as well as electrical ablation of the nerves innervating the joints (“medium bundle block”) have failed to demonstrate long-term effects.

Sacroiliac pain

The sacroiliac joint receives its primary innervation from the dorsal rami of the first four sacral nerves. Arthrography or injection of irritant solutions into the sacroiliac joint provokes pain with variable local and referred pain patterns into regions of the buttock, lower lumbar area, lower

extremity, and groin. Certain maneuvers may provoke typical pain, too. Local blocks sometimes accelerate recovery and facilitate physical therapy.

Muscular pain

Muscular pain is most often the cause of chronic back pain. Pain receptors in the muscles are sensitive to a variety of mechanical stimuli and to biomechanical overload. Anxiety and depressive disorders often play an important role in sustaining muscular pain due to the "arousal reaction," with a continuous increase of muscular tension. Muscular pain may be described as "myofascial pain," if muscles are in a contracted state, with increased tone and stiff ness, and contain trigger points (small, tender nodules that are identified on palpation of the muscles, with radiation into localized reference zones). In most patients myofascial pain is the result of a combination of factors: the "arousal reaction," direct or indirect trauma, exposure to cumulative and repetitive strain, postural dysfunction, and physical deconditioning.

On the cellular level, it is presumed that abnormal and persistently increased acetylcholine release at the neuromuscular junction generates sustained muscle contraction and a continuous reverberating cycle. If muscular back pain does not resolve within a few weeks (usually 6 weeks is seen to be crucial), it should be seen as a complex disease with physiological ("biological"), psychological, and psychosocial influences (according to the biopsychosocial model of chronic pain evolution). Therefore, when local therapies alone fail to give long-term pain relief, a major diagnostic and therapeutic workup including physical, psychosocial, and neuropsychological aspects ("multimodal therapy") may be needed.

If adequate therapy is delayed over several months with a trial of unimodal therapies, such as analgesics or injections only, long-term positive effects of multimodal therapeutic approaches become unlikely or very limited.