

Ministry of Healthcare of Ukraine
Poltava State Medical University

**GUIDELINES
FOR STUDENTS
INDEPENDENT WORK
IN THE PRACTICAL CLASSES PREPARING**

Academic discipline

Module

Content module

Study subject

Course

Faculty

Internal medicine

Current practice of internal medicine

Management of the patients with main symptoms and syndromes in rheumatology clinic

Management of the patient with pain in the back and limbs

VI

of foreign students training

1.The aims of the training course:

To Know:

1. Differential diagnosis of pain in the back.
2. Plan examination, additional laboratory and instrumental methods of examination
3. Tactic of patients curation.
4. Existing standards of treatment.
5. Primary and secondary prevention.
6. Weather and performance

To be able to:

- Conduct surveys and examination of the patients with major rheumatic syndromes
- To use the major invasive and non-invasive diagnostic techniques in rheumatology, to determine the indications and contraindications for their conduct, possible complications
- Identify different options for the course and complications of rheumatic diseases
- draft survey of patients with rheumatic diseases
- carry out differential diagnosis, justify and formulate diagnoses for major rheumatic syndromes based on laboratory analysis and test tool
- prescribe treatment, determine prognosis, to conduct primary and secondary prevention in rheumatic diseases
- to interpret laboratory indices in rheumatic diseases
- Demonstrate knowledge of moral principles

The contents of topic:

Pathophysiology of Back Pain

Introduction

Back pain (BP) is the most expensive benign condition in industrialized countries and the most common cause of activity limitation in persons younger than 45 years. It is defined as pain that persists longer than 12 weeks and is often attributed to degenerative or traumatic conditions of the spine. Fibrositis, inflammatory spondyloarthropathy, and metabolic bone conditions are also cited as causes. Although acute BP has a favorable prognosis, the effect of chronic BP and its related disability on society is tremendous. Unlike acute BP, chronic BP serves no biologic purpose. However, it is a disorder that evolves in a complex milieu influenced by endogenous and exogenous factors, and it alters the individual's productivity to an extent beyond what the initiating pathologic dysfunction would have.

Background

Approximately 80% of Americans experience BP 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 BP. BP is second only to the common cold as a cause of lost work time; it is the fifth most frequent cause for hospitalization and the third most common reason to undergo a surgical procedure. Productivity losses from chronic BP approach \$28 billion annually in the United States.

BP is defined as chronic after 3 months because most normal connective tissues heal within 6-12 weeks unless pathoanatomic instability persists. A slowed rate of tissue repair in the relatively avascular intervertebral disk may impair the resolution of chronic BP.

Traumatic or degenerative conditions of the spine are the most common causes of chronic BP. Although disk protrusion and herniation have been popularized as causes of BP and sciatica, asymptomatic disk herniations on CT and MRI are common. Furthermore, the relationship between the extent of disk protrusion and the degree of clinical symptoms is not clear. A strictly mechanical or pathoanatomic explanation for BP and sciatica has proved inadequate; therefore, the role of biochemical and inflammatory factors remains under investigation. In fact, this failure of the pathologic model to predict back pain often leads to an ironic predicament for the patient with BP. If diagnostic studies are unrevealing of a structural cause, physicians and patients alike question whether the pain has a psychologic, rather than physical, cause. Physical and nonphysical factors,

interwoven in a complex fashion, influence the transition from acute to chronic BP. The identification of all contributing physical and nonphysical factors enables the treating physician to enact a comprehensive approach with the best likelihood for success.

Pathophysiology

Degenerative cascade

The lumbar spine forms the caudal flexible portion of an axial structure that supports the head, upper extremities, and internal organs over a bipedal stance. The sacrum forms the foundation of the spine through which it articulates with the sacroiliac joints to the pelvis. 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 and had considerable mobility in both the sagittal and coronal planes. The bony vertebrae act as specialized structures to transmit loads through the spine. Parallel lamellae of highly vascularized cancellous bone form trabeculae, which are oriented along lines of biomechanical stress and encapsulated in a cortical shell. Vertebral bodies progressively enlarge in cross-sectional area because gravitational loads increase from cephalic to caudal segments. Bony projections from the lumbar vertebra, including the transverse processes and spinous processes, maintain ligamentous and muscular connections to the segments above and below them.

The intervertebral disk is 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.

The nucleus is composed of proteoglycan megamolecules can imbibe water to a capacity approximately 250% of their weight. 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 4 decades to approximately 65%. Nutrition to the inner annulus fibrosis and nucleus pulposus depends on the diffusion of water and small molecular substances across the vertebral endplates because only the outer third of the annulus receives blood supply from the epidural space.

Repeated eccentric and torsional loading and recurrent microtrauma results in circumferential and radial tears in annular fibers. Some annular tears may cause endplate separation, which results in additional loss of nuclear nutrition and hydration. Coalescence of circumferential tears into radial tears may allow nuclear material to migrate out of the annular containment into the epidural space and cause nerve root compression or irritation.

Throughout youth (at least the first 2 decades), 80-90% of the weight of the trijoint complex of the lumbar spine is transmitted across the posterior third of the disk; however, as disk height decreases and the biomechanical axis of loading shifts posteriorly, the posterior articulations (ie, facet joints) bear greater percentages of the weight distribution. Bone growth (osteophytes) compensates for this increased biomechanical stress to stabilize the trijoint complex.

Over time, hypertrophy of the facets and bony overgrowth of the vertebral endplates contribute to progressive foraminal and central canal narrowing. In addition to relative thickening of the ligament flavum and disk herniation, these changes contribute to reduction of the anteroposterior canal diameter and foraminal patency with neural compression. Spinal stenosis reaches a peak later in life and may produce radicular, myelopathic, or vascular syndromes such as pseudoclaudication and spinal cord ischemia.

BP is most common in the early stages of disk degeneration, in what Kirkaldy-Willis called the stabilization phase. Impaired healing of the intervertebral disk due to its poor and peripheral blood supply serving only the external third of the outer annulus has been introduced as a possible explanation for the divergent behavior of this structure, which can produce chronic nociception.

Also, elucidation of biochemical changes that may sensitize the disk and other structures capable of nociception within the trijoint construct may contribute to this discrepancy.

Types of Pain

Diskogenic pain

Many studies have demonstrated that the intervertebral disk and other structures of the spinal motion segment can cause pain.

Kuslich et al used regional anesthesia in 193 patients who were about to undergo lumbar decompressive surgery for disk herniation or spinal stenosis.¹ Pain was elicited by using blunt surgical instruments or an electrical current of low voltage in 30% of patients who had stimulation of the paracentral annulus fibrosis and in 15% with stimulation of the central annulus fibrosis. 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 in the absence of nonphysical or operant influences.

In 1987, Mooney proposed that this BP chronicity was best explained by a tissue component of the spine that obeyed physiologic rules different from those of other connective tissues in the body.² This divergent behavior is best illustrated in the intervertebral disk with its composition of large, unique, water-imbibing proteoglycan molecules. During adulthood, these large molecules break into small molecules that bind less water than the original molecule. Repair by means of proteoglycan synthesis is slow. Fissuring and disruption of annular lamellae further exacerbate molecular breakdown and dehydration of the disk. Arterial blood supply to the peripheral one third of the outer annulus is meager and inadequate to prevent subsequent internal degeneration.

The annulus and nucleus pulposus are similarly compromised, as they receive nutrition only by means of diffusion through adjacent vertebral endplates. Although sluggish healing of the intervertebral disk may partially account for the tendency of a spinal lesion to lead to chronicity, a direct concordance between structural degeneration and spinal pain does not exist.

Recent elucidation of biochemical behaviors and neurophysiologic factors affecting the disk and other regional pain-sensitive tissues may account for this discrepancy. In humans, painful disks have a lower pH than that of nonpainful disks. Also, experimental lowering of the pH in animal models induced pain-related behaviors and hyperalgesia. Diskography of canine disks that were normally or experimentally deformed seemed to show increased concentrations of neuropeptides, such as substance P (SP), calcitonin gene-related peptide (CGRP), and vasoactive intestinal peptide (VIP) in the dorsal root ganglion (DRG), implicating their possible role in the transmission or the modulation of pain. SP probably modulates initial nociceptive signals received in the gray matter of the dorsal spinal cord.

Somatostatin is another neuropeptide in dense concentration the dorsal gray of the spinal cord. Somatostatin is released from the DRG after noxious thermal stimulation and likely plays a role in pain transmission and in producing neurogenic inflammation. Therefore, the release of neuropeptides like SP, VIP, and CGRP may occur in response to noxious biochemical forces and environmental factors (eg, biomechanical stress, microtrauma, vibration), stimulating the synthesis of inflammatory agents (eg, cytokines, prostaglandin E₂) and degradative enzymes (eg, proteases, collagenase). These factors cause progressive deterioration of the motion segment structures, especially the intervertebral disk.

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. Afferent nociceptors in nerve roots may be sensitive to various proinflammatory mediators, which are inhibited by corticosteroids, such as prostanoids produced from arachidonic acid and released from cell membrane phospholipids by PLA₂.

Research suggests that proinflammatory cytokines may also contribute to diskogenic pain by sensitizing nociceptors and their effect on disk degeneration by suppressing proteoglycan synthesis

and increased diskal matrix degradation. Cytokines are produced in response to neural injury in the CNS and may play a role in spinal neural hypersensitization and chronic neuropathic pain. Cytokines known to play a role in nociception include nerve growth factor, interleukin (IL)-1, IL-6, IL-10, and tumor necrosis factor-alpha (TNF-alpha).

Corticosteroids can inhibit activity of TNF-alpha, which induces IL-1 and prostaglandin E₂ production. Once released, these substances contribute to early and late effects of the inflammatory process and stimulate nociception. A nociceptive role for nitric oxide (NO) in diskogenic pain syndromes is under investigation. NO levels are elevated in human disk herniations and when the hydrostatic pressure of the disk is increased due to biomechanical stressors. NO inhibits proteoglycan synthesis in cells in the nucleus pulposus, leading to proteoglycan loss, reduced water content, and disk degeneration.

Neurotransmitters and biochemical factors may sensitize neural elements in the motion segment so that the normal biomechanical stresses induced by previously asymptomatic movements or lifting tasks cause pain. Furthermore, injury and the consequential neurochemical cascade may initiate the degenerative and inflammatory changes described above, which mediate additional biochemical and morphologic changes and which modify or prolong the pain stimulus. Whether the biochemical changes that occur with disk degeneration are the consequence or cause of these painful conditions is unclear. However, chemical and inflammatory factors may create the environmental substrata through which biochemical stress and forces cause axial or limb pain with various characteristics and degrees.

Radicular pain

The pathophysiology of spinal nerve-root or radicular pain is unclear. Proposed etiologies include neural compression with axonal dysfunction, ischemia, inflammation, and biochemical influences. Spinal nerve roots have unique properties that may explain their proclivity to produce symptoms. Unlike peripheral nerves, spinal nerve roots lack a well-developed intraneural blood-nerve barrier, a lack which makes them more susceptible to symptomatic compression injury than peripheral nerves and vulnerable to endoneural edema formation.

Increased vascular permeability caused by mechanical nerve-root compression can induce endoneural edema. Furthermore, elevated endoneural fluid pressure due to intraneural edema can impede capillary blood flow and may cause intraneural fibrosis. Also, spinal nerve roots receive approximately 58% of their nutrition from surrounding CSF. Perineural fibrosis, which interferes with CSF-mediated nutrition, renders the nerve roots hyperesthetic and sensitive to compressive forces.

Research has elucidated several vascular mechanisms that can produce nerve-root dysfunction. Experimental nerve-root compression showed that venous blood flow can be stopped at low pressures, ie, 5-10 mm Hg. The occlusion pressure for radicular arterioles is substantially higher than this, approximating the mean arterial blood pressure and showing a correlation with systolic blood pressure; this factor increases the potential for venous stasis.

Some investigators postulate that venous then capillary stasis causes congestion that, in turn, may induce symptomatic nerve-root syndromes. Nerve-root ischemia or venous stasis may also generate pathologic biochemical changes that cause pain, unlike the progressive sensory then motor dysfunction typically seen with peripheral nerve compression. Studies of ischemia experimentally induced with low-pressure nerve-root compression demonstrated that compensatory nutrition from CSF diffusion is probably inadequate when epidural inflammation or fibrosis is present. Rapid onset of neural and vascular compromise is more likely than slow or gradual mechanical deformity to produce symptomatic radiculopathy.

Research has revealed other possible causative mechanisms for symptomatic radiculopathy. A 1987 animal study showed that autologous nucleus pulposus placed in the epidural space of dogs produced a marked epidural inflammatory reaction that did not occur in the comparison group, which received saline injections. Similar studies have shown myelin and axonal injury to nerve roots, as well as, reduced nerve conduction velocities exposed to autologous nucleus pulposus. Recent studies have demonstrated that experimental radicular exposure to degenerative nucleus

pulposus and annulus fibrosis does not produce the same dysfunctional nerve-root changes. Cells of the nucleus pulposus can induce local neural dysfunction and generate algogenic agents, such as metalloprotease (eg, collagenase, gelatinase), as well as IL-6 and prostaglandin-E₂.

Other biochemical substances, including TNF, have been implicated as causes. TNF increases vascular permeability and appears to be capable of inducing neuropathic pain. When injected into nerve fascicles, TNF produces changes similar to those seen when nerve roots are exposed to the nucleus pulposus. In addition, a still-unanswered issue is whether an autoimmune response occurs when nucleus pulposus is exposed to the systemic circulation because it is sequestered by the annulus fibrosis and because the immune system may not be recognized as normal. Indeed, research to date suggests that the cause of symptomatic radiculopathy is multifactorial and more complex than just neural dysfunction due to structural impingement.

Facet-joint pain

The superior and inferior articular processes of adjacent vertebral laminae form the facet or zygapophyseal joints, which are paired diarthrodial synovial articulations that share compressive loads and other biomechanical forces with the intervertebral disk. Like other synovial joints, the facets react to trauma and inflammation by manifesting pain, stiffness, and dysfunction with secondary muscle spasm leading to joint stiffness and degeneration. This process is borne out, as previously described, through the degenerative cascade of the trijoint complex. Numerous radiologic and histologic studies have shown that diskal and facet degeneration are linked and that, over time, degeneration of the segment leads to osteoarthritis of the facets.

Studies of provocative intra-articular injection techniques demonstrated local and referred pain into the head and upper extremities from cervical facets, into the upper midback and chest wall from thoracic facets, and into the lower extremity from the lumbar facets. The fibrous capsule of the facet joint contains encapsulated, unencapsulated, and free nerve endings.

Immunohistochemical studies have demonstrated nerve fibers containing neuropeptides that mediate and modulate nociception (eg, SP, CGRP, VIP). SP nerve fibers have been found in subchondral bone and degenerative lumbar facets subjected to aging and cumulative biomechanical loading. In fact, SP levels are correlated with the severity of joint arthritis. The infusion of SP into joints with mild disease reportedly accelerated the degenerative process. Furthermore, these chemicals and inflammatory mediators have been linked to proteolytic and collagenolytic enzymes that cause degradation of the cartilaginous matrix and osteoarthritis. Therefore, evidence of nociceptive afferents and the presence of algogenic neuropeptides, such as SP and CGRP, in facets and periarticular tissues support a role for these structures as spinal pain generators. Clinical research has demonstrated facet pain in 54-67% of patients with neck pain, 48% of patients with thoracic pain, and 15-45% of patients with BP.

Sacroiliac pain

The sacroiliac joint is a diarthrodial synovial joint that 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. Determined by using variety of blocking techniques, the reported prevalences of sacroiliac pain have been widely variable (2-30%) in patients evaluated for chronic BP.

Muscular pain

Pain receptors in muscle are sensitive to a variety of mechanical stimuli, including pressure, pinching, cutting, and stretching. Pain and injury occur when the musculotendinous contractual unit is exposed to single or recurrent episodes of biomechanical overloading. Injured muscles are usually abnormally shortened, with increased tone and tension due to spasm or overcontraction. Injured muscles often meet diagnostic criteria for myofascial pain (MP) syndrome, a condition that Drs Janet Travell and David Simons originally described.

MP is characterized by muscles that are in a shortened or contracted state, with increased tone and stiffness, and that contain trigger points (TrPs). TrPs are tender, firm, 3- to 6-mm nodules that are identified on palpation of the muscles. TrP palpation provokes radiating, aching pain into localized

reference zones. Mechanical stimulation of the taut band, a hyperirritable spot in the TrP, by needling or rapid transverse pressure often elicits a localized muscle twitch. Sometimes, TrP palpation can elicit a jump sign, an involuntary reflex or flinching disproportionate to the palpatory pressure applied. MP may become symptomatic as a result of direct or indirect trauma, exposure to cumulative and repetitive strain, postural dysfunction and physical deconditioning. MP can occur at the site of tissue damage or due to radicular and other neuropathic disorders at sites where pain is referred. Muscles affected by neuropathic pain may be injured due to prolonged spasm, mechanical overload, or metabolic and nutritional shortfalls. The pathogenesis of MP and TrPs remains unproven. To date, research suggests that myofascial dysfunction with characteristic TrPs is a spinal segmental reflex disorder. Animal studies showed that TrPs can be abolished by transecting efferent motor nerves or infusing of lidocaine; however, spinal transection above the level of segmental innervation of a TrP-containing muscle does not alter the TrP response. Simons postulates that abnormal, persistently increased, and excessive acetylcholine release at the neuromuscular junction generates sustained muscle contraction and a continuous reverberating cycle. This cycle has been postulated to result in painful and dysfunctional extrafusal muscle contraction that forms the basis for MP and possibly the actual structural substrate of the TrP.

History, Physical Examination, and Causes

History

In most cases, chronic BP has been investigated with appropriate physician evaluation and perhaps imaging studies. Characterization of the pain as mechanical is a primary goal when a history is obtained from a patient with chronic BP and sciatica. Mechanical or activity-related spinal pain is most often aggravated by static loading of the spine (eg, prolonged sitting or standing), long-lever activities (eg, vacuuming or working with the arms elevated and away from the body), and levered postures (eg, forward bending of the lumbar spine). Pain is reduced when multidirectional forces balance the spine (eg, walking or constantly changing positions) and when the spine is unloaded (eg, reclining). Patients with mechanical BP often prefer to lie still in bed, whereas those with a vascular or visceral cause are often found writhing in pain, unable to find a comfortable position. Unrelenting pain at rest should suggest a serious cause, such as cancer or infection. Imaging studies and blood workup are usually mandatory in these cases and in cases of progressive neurologic deficit. Other historical, behavioral, and clinical signs that should alert the physician to a nonmechanical etiology requiring diagnostic evaluation are outlined below.

Diagnostic red flags

- Pain unrelieved by rest or any postural modification
- Pain unchanged despite treatment for 2-4 weeks
- Writhing pain behavior
- Colicky pain or pain associated with a visceral function
- Known or previous cancer
- Fever or immunosuppressed status
- High risk for fracture (eg, older age, osteoporosis)
- Associated malaise, fatigue, or weight loss
- Progressive neurological impairment
- Bowel or bladder dysfunction
- Severe morning stiffness as primary complaint
- Patients unable to ambulate or care for self

Nonphysiologic or implausible descriptions of pain may provide clues that operate; other psychosocial influences coexist.

Physical examination

Physical examination is important to confirm a mechanical or benign cause for the patient's BP. Observations of verbal and nonverbal behaviors suggesting symptom magnification should be noted. Inspection of the spine requires the patient to disrobe. Open-back gowns give the physician only 1 view of the spine; therefore, swimming attire is often appropriate for complete, 360°

inspection. Leg-length discrepancy and pelvic obliquity, scoliosis, postural dysfunction with forward head and shoulders, or accentuated kyphosis should be noted. Physicians' preferences vary in regard to the importance of testing range of motion; however, just asking the patient to bend forward often enables the most worthwhile observations.

The patient is asked to drop his or her head and shoulders forward and then move slowly into forward bending. Normal forward bending is revealed when the patient recruits from each cephalic segment to the level below, and so on, progressing from the cervical spine through the thoracic and lumbar region, where flexion of the hips completes the excursion into full flexion. Patients with clinically significant mechanical back pain or lumbar segmental instability usually stop cephalic-to-caudal segmental recruitment on reaching the thoracolumbar junction, or sometimes the involved lumbar level. To continue forward bending, they then contract their lumbar muscles to brace the mechanically compromised segment and then continue recruitment in a reverse direction, beginning with motion through the hips, then proceeding cephalad, level to level, completing the excursion of the spine to the erect posture.

In cases of severe mechanical back pain and segmental instability with regional muscular spasm, the patient often reports an inability to perform any flexion below a thoracic spinal level. Any soft-tissue abnormalities and tenderness to palpation should be recorded. Palpation of lumbar paraspinal, buttock, and other regional muscles should be performed early in the examination. The examiner should palpate and note areas with superficial and deep-muscle spasm, and he or she should identify TrPs and small, tender nodules in a muscle that elicit characteristic regional referred pain.

Dissociation of physical findings from physiologic or anatomic principles is the key in patients in whom psychological factors are suspected to be influential. Examples of this phenomenon include nondermatomal patterns of sensory loss, nonphysiologic demonstration of weakness (give-way weakness when not caused by pain, or ratchety weakness related to simultaneous agonist and antagonist muscular contraction), and dissociation between the lumbar spinal movements found during history taking or counseling sessions from movements observed during examination.

Assessment of Waddell signs have been popularized as a physical-examination technique to identify patients who have nonorganic or psychogenic embellishment of their pain syndrome. Examination techniques that Waddell proposed consist of simulated rotation of the hips *en masse* with the lumbar spine without allowing for spinal rotation; this maneuver normally does not cause pain. Another is the application of light pressure on the head, which should also be painless. Likewise, gentle effleurage of superficial tissues is unlikely to cause pain. Other techniques are striking dissociation between sitting and supine straight leg raising and the demonstration of nonphysiologic weakness and sensory deficits by the patient, as mentioned already.

Straight leg raising with the patient supine should produce ipsilateral leg pain between 10° and 60° to be declared positive. Straight leg raising that produces pain in the opposite leg carries a high probability of disk herniation, and investigation should be considered, especially if neurologic evidence for radiculopathy is present. Nonspecific complaints, overtly excessive pain behavior, patient contraction of antagonist muscles that limit the examiner's testing, or tightness of buttock and hamstring muscles are commonly mistaken for positive results on straight leg raising. Reverse straight leg raising may elicit symptoms of pain by inducing neural tension on irritated or compressed nerve roots in the mid-to-upper lumbar region. In addition, this maneuver helps the astute physician identify tightness of the iliopsoas muscle, which commonly contributes to chronic lumbar discomfort.

Neurologic evaluation is performed to determine the presence or absence and levels of radiculopathy or myelopathy. Anatomic localization is determined by muscle and reflex testing combined with historical information obtained during the interview and the absence of neurologic symptoms or signs that implicate cerebral or brainstem involvement. Consistent myotomal weakness and sensory findings that at least seem to coincide with segmental radiculopathy or polyradiculopathies should not be ignored.

The neurologist should identify syndromes of the lower motor neurons versus the upper motor neurons and the level of spinal dysfunction. Hyperreflexia in caudal spinal levels may change to

reduced or absent reflexes in the upper extremities, determining radicular or spinal-cord localization of dysfunction. Rectal examination is indicated in patients in whom myelopathy, especially cauda equina syndrome, is a diagnostic concern. Tone of the anal sphincter; presence or absence of an anal wink; and correlation with motor, sensory, and reflex findings are appropriate in these cases.

Causes

Epidemiologic data suggest that risk factors include cigarette smoking; morbid obesity; occupations that require repetitive lifting, especially in forward bending and twisting positions, particularly when lifting requirements exceed the worker's physical capacity; and exposure to vibration caused by motor vehicles or industrial machinery. Studies indicate that smoking is most likely to be a risk factor for BP in people with jobs that require heavy physical exertion. Fitness may be correlated with recovery and return to work after BP; however, in prospective studies controlled for age, isometric lifting strength and degree of cardiovascular fitness were not predictive of back injury. Occupational risk factors are difficult to define because exposures to specific causative influences are unclear, mechanisms of injury may be confusing, and supporting research findings are variable and conflicting for most environmental risks. Furthermore, job dissatisfaction, work conditions, legal and social factors, financial stressors, and emotional circumstances heavily influence back disability. Although many experts agree that heavy physical work, lifting, prolonged static work postures, simultaneous bending and twisting, and exposure to vibration may contribute to back injuries, the medical literature provides conflicting support for most of these proposed risk factors. Extreme height and morbid obesity may predispose an individual to back pain. However, research studies have not clearly demonstrated that height, weight, or body build are directly related to the risk of back injury. Weakness of the trunk extensor muscles, compared with flexor strength, may be a risk factor for sciatica.

When BP persists beyond 3 months, into the chronic phase, appropriate clinical and diagnostic information supporting a benign or mechanical cause should be accrued, if it has not been already. Also, prompt physician evaluation, including reasonable radiographic, laboratory, and electrophysiologic testing, is indicated in patients with persistent severe neurologic deficit, intractable limb pain, suspected systemic illness, or changes in bowel or bladder control. The spectra of mechanical (or activity-related) and nonmechanical causes of BP are outlined below.

Mechanical or activity-related causes of BP

- Diskal and segmental degeneration - May include facet arthropathy from osteoarthritis
- Myofascial, muscle spasm, or other soft-tissue injury and/or disorders
- Disk herniation - May include radiculopathy
- Radiographic spinal instability with possible fracture or spondylolisthesis - May be due to trauma or degeneration
- Fracture of bony vertebral body or trijoint complex - May not reveal overt radiographic instability
- Spinal canal or lateral recess stenosis
- Arachnoiditis, including postoperative scarring

Differential diagnosis can include many neurological and systemic disorders, as well as referred pain from viscera or other skeletal structures such as the hip.

Disorders that may be associated with nonmechanical BP

- Neurologic syndromes
 - Myelopathy from intrinsic or extrinsic processes
 - Lumbosacral plexopathy, especially from diabetes
 - Neuropathy, including inflammatory demyelinating type (ie, Guillain-Barré syndrome)
 - Mononeuropathy, including causalgia
 - Myopathy, including myositis and metabolic causes
 - Dystonia, truncal or generalized central pain syndrome
- Systemic disorders
 - Primary or metastatic neoplasm, including myeloma

- Osseous, diskal, or epidural infection
- Inflammatory spondyloarthropathy
- Metabolic bone disease, including osteoporosis
- Vascular disorders such as atherosclerosis or vasculitis
- Referred pain
 - Gastrointestinal disorders
 - Genitourinary disorders, including nephrolithiasis, prostatitis, and pyelonephritis
 - Gynecologic disorders, including ectopic pregnancy and pelvic inflammatory disease
 - Abdominal aortic aneurysm
 - Hip pathology

Psychosocial factors that may influence BP chronicity and disability

- Compensable injury
- Somatoform pain disorder
- Psychiatric syndromes, including delusional pain
- Drug seeking
- Abusive relationships
- Seeking disability or out-of-work status

Diagnostic Strategies

As indicated in the last section, unrelenting pain at rest should generate suspicion for cancer or infection. The appropriate imaging study is mandatory in these cases and in cases of progressive neurologic deficit. Plain anteroposterior and lateral lumbar spine radiographs are indicated for patients older than 50 years and for those with pain at rest, a history of serious trauma, or other potential conditions (eg, cancer, fracture, metabolic bone disease, infection, inflammatory arthropathy). The yield for discovering a serious condition with radiography outside these parameters is minimal, and the cost savings are substantial.

When BP and sciatica persist into the subacute phase (pain lasting 6-12 wk), appropriate consultation and diagnostic imaging should be considered. Referring the patient to a physician with expertise in spinal disorders may be the most appropriate procedure for initial evaluation rather than relying on expensive diagnostic testing.

CT scanning is an effective diagnostic study when the spinal and neurologic levels are clear and bony pathology is suspected.

MRI is most useful when exact spinal and neurologic levels are unclear, when a pathologic condition of the spinal cord or soft tissues is suspected, when postoperative disk herniation is possible, or when an underlying infectious or neoplastic cause is suspected.

Myelography is useful to elucidate nerve-root pathology, particularly in patients with previous lumbar spinal surgery or with a metal fixation device in place. CT myelography provides accurate visual definition to elucidate neural compression or arachnoiditis when patients have undergone several spinal operations and when surgery is considered for the treatment of foraminal and spinal-canal stenosis.

When leg pain predominates and imaging studies provide ambiguous information, clarification may be gained by performing electromyography (EMG), somatosensory evoked potential (SSEP) testing, or selective nerve root blocks. When the cause of sciatica is related to neural compression by bony or soft-tissue structures in the spinal canal, surgical consultation should be considered. If diagnostic information is inadequate to explain the degree of neurologic deficit, pain, and disability, multidisciplinary evaluation may provide insight into perpetuating physical and psychosocial factors

Treatment

Emergency Department Care

If new neurologic deficits are noted accompanied by bowel or bladder dysfunction one should suspect cauda equina syndrome. This is a true emergency, and emergency imaging is mandated. MRI is the preferred imaging modality in this situation. If cauda equina syndrome is strongly

suspected, the practitioner should consider giving dexamethasone without delay to prevent further loss of neurologic function while pursuing confirmatory testing.

Conservative therapy is the mainstay of treatment, as even those with true sciatica generally respond. Ultimately, only 2% of patients with sciatica and 4-6% of patients with true disc herniation require surgery. Conservative therapy traditionally includes the following:

- Bed rest, once the cornerstone of treatment, is no longer widely recommended.
 - A growing body of evidence suggests that even brief bed rest is not necessary except in patients with true sciatica. In this case, the supine position decreases pressure on the spinal cord itself, and is useful for the first 2-3 days.
 - Early mobilization with gentle range of motion and strengthening exercises are recommended for patients with nonsciatic back pain.
 - Early return to work on light duty or restricted activity lead to better long-term outcomes.
- Pharmacologic therapy involves both anti-inflammatory medication and muscle relaxants.
 - Narcotics may be used initially to gain relief, but their long-term use is associated with increased functional impairment.
 - Steroids, while highly recommended by some practitioners, lack prospective confirmation of their value. Some physicians may prescribe a single burst or short course of oral steroids, which can be beneficial, particularly in those with a significant degree of inflammation.
 - Epidural steroid injection may also bring significant short-term relief, but this treatment is not without adverse effects and has not been shown to provide lasting benefit.
- Unless the patient is allergic to the medicine or it is otherwise contraindicated, severe low back pain can be improved significantly with a combination of nonsteroidal anti-inflammatory drugs (NSAIDs) and muscle relaxants.
- Use of hot or cold compresses has never been proven scientifically to speed symptom resolution, but some patients may experience brief relief.
- Gentle flexion/extension exercises are helpful.
- Spinal traction is ineffective.

Evidence-based clinical practice guidelines from the American Pain Society (APS) for patients with chronic low back pain describe the use of interventional diagnostic tests and therapies, surgeries, and interdisciplinary rehabilitation.

- Practice guidelines for nonradicular pain
 - Interdisciplinary rehabilitation emphasizing cognitive-behavioral approaches should be considered for patients who do not respond to usual interventions.
 - Provocative discography (injecting material into a disc nucleus in an attempt to reproduce the patient's typical pain) is not recommended.
 - Facet joint corticosteroid injection, prolotherapy (repeated injections of irritant material to stimulate an inflammatory response), and intradiscal corticosteroid injection are not recommended.
 - Persistent disabling symptoms and degenerative spinal changes should prompt discussion and shared decision-making regarding surgery or interdisciplinary rehabilitation (evidence is insufficient to weigh the risks and benefits of vertebral disc replacement in these patients).
- Practice guidelines for persistent radiculopathy
 - For patients with herniated discs, the use of epidural steroid injection should be discussed.
 - For patients with herniated discs and disabling leg pain from spinal stenosis, surgical options should be discussed.
 - For patients with persistent pain after surgery, the risks and benefits of spinal cord stimulation should be discussed.

Consultations

- ED consultation with a specialist is necessary for patients who present with acute cauda equina syndrome, demonstrate intractable pain, have evidence of a serious etiology (eg, epidural abscess, tumor), or where a social situation makes hospitalization necessary.
- Whether orthopedic or neurosurgical consultation is chosen depends on local custom and resources.
- Other medical consultation may be needed if the cause of back pain is not mechanical.

Medication

The goal of pharmacotherapy is to reduce pain and inflammation.

Nonsteroidal anti-inflammatory agents (NSAIDs)

NSAIDs are most commonly used to relieve mild to moderate pain. Although the effectiveness of NSAIDs tends to be patient specific, ibuprofen is usually the DOC for initial therapy. Other options include flurbiprofen, ketoprofen, and naproxen.

Ibuprofen (Ibuprin, Advil, Motrin)

DOC to treat mild to moderate pain if no contraindications exist.

Inhibits inflammatory reactions and pain by decreasing prostaglandin synthesis. **Adult - 600 mg PO tid**

Ketoprofen (Oruvail, Orudis, Actron)

For relief of mild to moderate pain and inflammation.

Small dosages initially are indicated in patients who are small or elderly and in those with renal or liver disease. Doses over 75 mg do not increase therapeutic effects. Administer high doses with caution, and closely observe patient for response.

25-50 mg PO q6-8h prn; not to exceed 300 mg/d

Naproxen (Anaprox, Naprelan, and Naprosyn)

For relief of mild to moderate pain; inhibits inflammatory reactions and pain by decreasing activity of cyclooxygenase, which results in a decrease of prostaglandin synthesis.

500 mg PO initial, followed by 250 mg q6-8h; not to exceed 1.25 g/d

Muscle relaxants

These agents reduce tonic somatic motor activity of the muscle.

Carisoprodol (Soma)

Short-acting medication that may have depressant effects at spinal cord level.

Skeletal muscle relaxants have modest short-term benefit as adjunctive therapy for nociceptive pain associated with muscle strains and, used intermittently, for diffuse and certain regional chronic pain syndromes. Long-term improvement over placebo has not been established.

350 mg PO tid/qid

Cyclobenzaprine (Flexeril)

Skeletal muscle relaxant that acts centrally and reduces motor activity of tonic somatic origins influencing both alpha and gamma motor neurons.

Structurally related to tricyclic antidepressants and thus carries some of the same liabilities.

10 mg PO tid with a range of 20-40 mg/d in divided doses; not to exceed 60 mg/d

Analgesics

Pain control is essential to ensure patient comfort, to promote pulmonary toilet, and to aid physical therapy regimens. Many analgesics have sedating properties that benefit patients who have sustained injuries.

Acetaminophen (Tylenol, Panadol, Aspirin Free Anacin)

DOC for pain in patients with documented hypersensitivity to aspirin or NSAIDs, in those with upper GI disease, or in those who are taking oral anticoagulants.

325-650 mg PO q4-6h or 1000 mg tid/qid; not to exceed 4 g/d

Acetaminophen and codeine (Tylenol #3)

A drug combination indicated for the treatment of mild to moderate pain.

30-60 mg/dose based on codeine content PO q4-6h or 1-2 tabs q4h; not to exceed 12 tabs/d

Hydrocodone bitartrate and acetaminophen (Vicodin ES)

A drug combination indicated for the relief of moderate to severe pain.1-

2 tab or cap PO q4-6h pm

Deterrence/Prevention

- Back muscle strengthening exercises have value in preventing future episodes of low back strain.
- Weight loss in overweight patients results in less strain on back muscles.
- Practicing proper lifting techniques results in less back strain.
- General overall improvement of physical conditioning can decrease low back pain exacerbations.

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Composed by Prikhodko N.P.