CHAPTER 43

Overview of Low Back Pain Disorders

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Pain originating from the spine usually manifests as pain in the low back and neck, and infrequently as pain in the upper lumbar and mid back areas. Spinal pain (SP) can be grouped into three broad categories: acute pain when the pain duration is between 2 to 4 weeks; subacute pain when the pain persists for up to 12 weeks; and chronic pain, when the pain continues for more than 12 weeks. Chronic SP could be further categorized as persistent or recurrent pain.

EPIDEMIOLOGY

Although acute SP is frequently self-limiting, chronic SP is often persistent and recurring in character. Almost 30% of the patients with acute onset low back pain (LBP) will progress to develop chronic LBP that is typically recalcitrant to available treatments.[1,2] This fact is evidenced by the decreased likelihood of return to work with increasing SP duration. The workers who were off work for 6 months with LBP had lifetime return to work rate of only 50%; this rate further dropped to 25% for workers who were off work for 1 year, and to less than 5% for those who were off work for 2 years.[3] Despite the availability of a wide array of treatment choices to SP patients offered by both conventional and other approaches, morbidity from SP has continued to rise sharply, satisfaction among SP patients has remained low, and SP has remained the most prevalent cause of pain and disability in advanced industrialized nations.[1,2] In addition to its chronic unrelenting nature, chronic SP patients are also prone to psychosocial, behavioral, and substance abuse– and disability-related issues. Chronic SP therefore poses substantial challenges to individuals, their families, and the community as a whole.

The epidemiologic studies of chronic SP are approximative by nature, because the conditions causing SP in general are nonhomogenous and are often inadequately defined. The lifetime incidence of LBP is therefore reported variably, ranging from 14% to as high as 90%.[1,2,4] Acute LBP has been ranked as the fifth most common reason for all physician visits; in a given year almost 50% of adults will have LBP.[5] The financial and socioeconomic impact of SP to society is also colossal. For instance, the direct costs of health care for LBP disorders in the United States have been estimated at over $20 billion annually, whereas the indirect cost estimates are even higher, at over $50 billion annually.[6,7] In the United States, LBP has been cited as the most prevalent reason for lost work time, workers' compensation claims, and early social security disability.[8]

RISK FACTORS

The risk factors associated with SP have been classified into three broad categories: biomechanical, psychosocial, and personal. The biomechanical risk factors are determined by spinal loading, and typically include parameters such as physical stress and the asymmetry of physical tasks.[9,10] The psychosocial risk factors pertain to psychogenic stress and are often related to job satisfaction, responsibility, and variety.[11,12] Personal risk factors have been acknowledged as physical, familial, anthropometric, gender, and personality traits.[13,14] The following risk factors have been associated with the development of spinal pain:

- Jobs that are stressful and that require heavy lifting and use of heavy equipment[15]
- Cigarette smoking[16,17]
- Psychiatric, emotional, and personality issues[11,12]
- Obesity[17]
- Spinal deformities and endplate injury[18]
- Genetic predisposition[19]
- Peripheral vascular disease[20]

ANATOMY

The human vertebral column consists of 7 cervical, 12 thoracic, 5 lumbar, 5 sacral, and 3 to 5 coccygeal vertebrae. Except for the sacral and coccygeal vertebrae, which are normally fused, two adjacent vertebral bodies and an intervening intervertebral disc comprise a vertebral motion segment. The linear array of adjacent spinal motion segments forms the continuum of the spinal column that houses dorsally the neural elements of spinal cord and nerve roots of the cauda equina. The latter are encompassed dorsally and laterally by the neural arch, which is comprised of spinous processes, spinal laminae and the ligamenta flava posteriorly, and pedicles and intervertebral foraminae laterally. In addition to the linkage of the vertebral bodies by intervertebral discs, the adjacent vertebral bodies are articulated dorsally by a pair of synovial joints, the zygapophysial or facet joints. Various components of the spinal column also enable attachment of the omnipotent trunk muscles and spinal ligaments. The most significant of the spinal ligaments include the anterior and posterior longitudinal ligaments and ligamentum flavum. The incredible forces applied to the spinal column are transmitted to the lower extremities by two large synovial-fibrous joints, the sacroiliac joints.

The vertebral bodies are largely composed of cancellous bone housed in a thin layer of cortical bone. The intervertebral discs (IVDs) are made of annulus fibrosus (AF), nucleus pulposus (NP), and vertebral endplates. The distinction between the AF and NP is most apparent at the lumbar levels and diminishes with advancing age. Both the NP and AF are populated by sparsely present cells immersed in abundant intercellular matrix. Cells populating the NP are found in clusters and are chondrocyte-like, whereas the cells found in AF have fibrocytic features.[21] The matrix composition of the two disc compartments is also significantly different. NP matrix is jelly-like, and is made

of high concentration of water and proteoglycans, whereas matrix constituting AF is high in collagen arranged in the form of interlacing lamellae. These collagenous lamellae are firmly attached to the adjacent vertebral bodies and are most dense anteriorly.[21] Although the cancellous vertebral bodies and the spinal canal contents are highly vascular, the IVDs are mostly avascular and the largest avascular structure in the body. The normal NP and inner third of the AF completely lack any vasculature; moreover, the avascular cartilaginous endplates act as a barrier separating the vertebral body vasculature from the IVD contents.[21]

Innervation of the IVDs and the neural canal contents (Fig. 43-1) is mainly by nerve plexuses along the anterior and posterior longitudinal ligaments.[22] The nerve plexus along the posterior longitudinal ligament receives its input mainly from the sinuvertebral nerve and the gray rami communicans, while the plexus along the anterior longitudinal ligament is contributed to mainly by the gray rami communicans.[22] The sinuvertebral nerve originates from the segmental spinal nerve as it exits the intervertebral foramen; it re-enters the vertebral canal and contributes mostly to the posterior longitudinal plexus. In addition to the segmental spinal nerve, the sinuvertebral nerve also receives contribution from the gray rami communicans.[22] The posterior longitudinal ligament plexus innervates the ventral half of the vertebral column, including the anterior dura and posterior intervertebral discs. The gray ramus communicans nerve emerges from the spinal segmental nerve; soon after, it enters the intervertebral foramina and runs anteriorly along the inferior third of the vertebral body. It connects to the sympathetic trunk before branching into lateral and anterior branches to innervate the lateral and anterior disc annulus of the disc levels above and below. The posterior primary ramus, soon after its division from the anterior primary ramus, branches into medial and lateral branches. The medial branch of the posterior primary ramus supplies most dorsal spinal column components, including facet joints, posterior neural arch components, and spinous processes. The AF of the IVD therefore has complex innervation from several sources and multiple spinal segments, including contributions from the sinuvertebral nerves, segmental spinal nerve, gray ramus communicans nerve, and the sympathetic trunk; thus, a normal IVD has rich autonomic connections. The latter may contribute to the hyperalgesia often exhibited by the chronically painful disc. Although almost all components of a spinal motion segment have been implicated in generating pain, the pain receptors—mostly mechanoreceptors—are found mainly in the spinal ligaments, paraspinal muscles, vertebral body periosteum, and the outer third of the AF and facet joints.[21,23]

PATHOPHYSIOLOGY

The forces applied to the spinal column are borne directly and efficiently by the vertebral bodies and the IVDs.[24] The flexibility and remarkable range of motion exhibited by an active spine depend almost entirely on the cumulative plasticity exhibited by the individual IVDs. The individual IVD, however, is only moderately plastic and the NP, like the vertebral body, is practically incompressible due to its high water content. The compressive forces applied to the IVD are borne by the NP and are distributed equally to the AF as a tensile force.[25]

NP incompressibility is maintained almost exclusively by the hydrostatic pressure generated by its proteoglycan

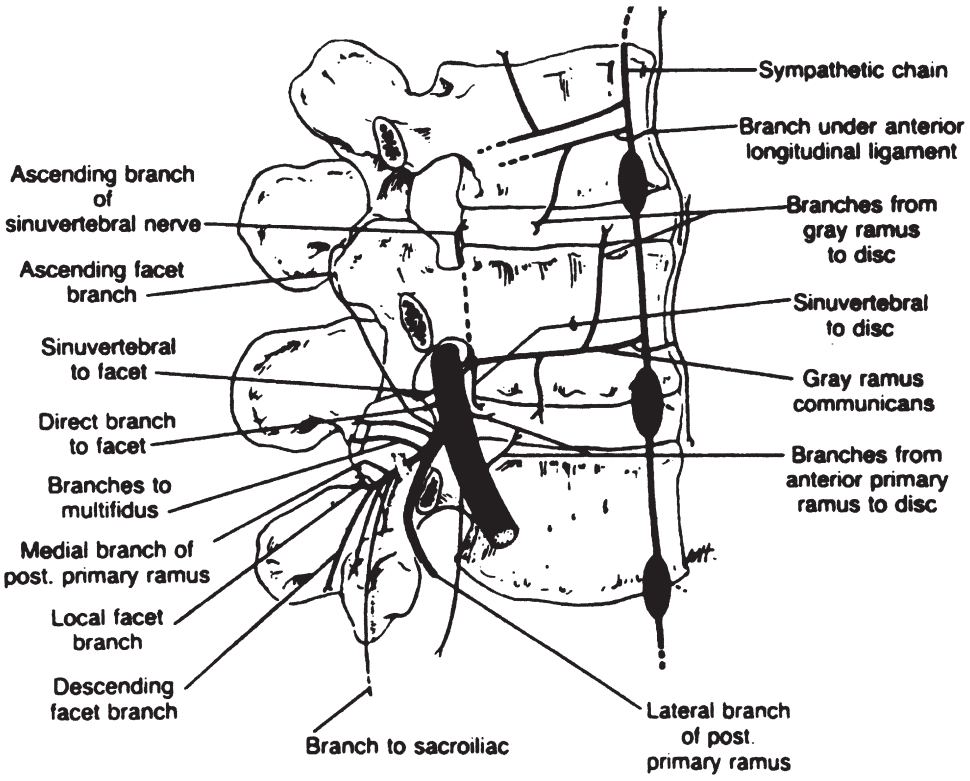

FIGURE 43-1 Segmental innervation of the lumbar spine. *(From Paris SV: Anatomy as related to function and pain. Symposium on Evaluation and Care of Lumbar Spine Problems.* Orthop Clin North Am *14:475–489, 1983.)*

content,[21] which is a function of intricate metabolic processes.[26] Being mostly avascular, IVD obtains metabolic requirements almost exclusively by diffusion from capillary plexuses in adjacent vertebral bodies and the outer AF. Discal catabolic activities are in addition facilitated by discal matrix metalloproteinases (MMPs).[27] A delicate balance therefore exists in the NP between the anabolic activities of the disc cells and the enzymatic catabolic activities. The IVD also lacks scavenger cells and the macromolecular end products of disc metabolism accumulate in the disc over time.[28] This arrangement is at best tenuous and the IVD cells function in a precarious anaerobic environment that can be adversely effected by a host of hereditary and environmental factors.[29] Dysfunction and decline in the viable NP cells,[26] enhanced MMP activity,[30] and increased disc cytokines and proinflammatory mediator concentration[31] can start a vicious cycle that can reduce NP proteoglycan and water content and consequent loss of disc hydrostatic pressure. The ensuing laxity of the NP exposes the AF to direct compressive forces.[25] In addition, the AF cells can undergo degenerative changes similar to those in the NP and result in loss of AF collagen. The cumulative effect of increased AF stress and collagen loss may lead to eventual AF failure with the consequent development of annular tears and fissures.[32]

Structural changes within the IVD alter its biomechanical properties and cause it to shrink and become less plastic. These changes in the IVD dynamics increases stress on adjacent vertebral motion segment and may propagate degenerative changes in several contiguous spinal structures. Some of these changes include sclerosis and hypertrophic new bone formation in adjacent vertebral bodies—Modic changes,[33] accelerated degenerative changes in the adjacent IVDs, hypertrophy and arthritis of the facet joints, sacroiliac joint dysfunction, and paraspinal myofascial syndrome.[34] Hypertrophic changes in the discs, facet joints and ligamenta flava may leads to narrowing of the spinal canal and the intervertebral foraminae. These stenotic changes may cause symptoms from compression of the spinal cord and the spinal nerve roots.[35] However despite the aforementioned, the spinal degenerative changes are commonly seen in asymptomatic individuals and their presence correlate poorly to patients' symptoms.[36,37]

TABLE 43–1 Etiology of Spinal Pain

Mechanical Spinal Pain
Herniated discs
Spondylosis or degenerative disc disease
Discogenic pain, internal disc disruption, or annular tears
Spondylolisthesis or displacement of one vertebral body over the other
Spondylolysis or defect in pars interarticularis without the vertebral slippage
Spinal instability or anomalous movement between the contiguous vertebral bodies
Foraminal stenosis or skeletal hypertrophy causing symptoms of nerve root compression
Spinal canal stenosis or neurogenic claudication or myelopathic symptoms and signs
Facet arthropathy
Musculoligamentous strains or sprains
Myofascial pain syndrome
Congenital spinal conditions such as kyphosis or scoliosis
Nonmechanical Spinal Pain
Primary and metastatic neoplasms of the spine or its neural contents
Infections, such as osteomyelitis of the vertebral bodies, septic discitis, paraspinal or epidural abscess
Noninfectious inflammatory spinal disorders such as ankylosing spondylitis, Reiter's syndrome, psoriatic spondylitis, and inflammatory bowel disease
Traumatic or pathologic fractures such as vertebral body compression fractures and dislocations
Metabolic disorders of the spine such as Paget's disease
Miscellaneous conditions such as Scheuermann's disease or osteochondrosis, and hemangiomas
Referred or Visceral Spinal Pain
Pelvic visceral disorders such as prostatitis, endometriosis, or pelvic inflammatory disease
Renal disease such as nephrolithiasis, pyelonephritis, or perinephric abscess
Vascular disease such as abdominal aortic aneurysm
Gastrointestinal disease such as pancreatitis, cholecystitis, or perforated bowel

ETIOLOGY

The differential diagnosis of SP has conventionally included specific and nonspecific causes (Table 43-1). Specific SP evidently originates from a definite pathophysiologic cause in contrast to nonspecific SP, which lacks a clear etiology. Although approximately 90% of all SP patients have conventionally been branded as having nonspecific SP,[38] this number is probably inexplicably high for diverse reasons. SP could originates not only from a variety of spinal column components such as IVDs, facet joints, paraspinal muscles, ligaments, and the various neural elements, but it can also initiate from adjacent spinal structures such as abdominal or pelvic viscera, sacroiliac and hip joints, and the adjoining neural plexuses. The pathologic conditions afflicting the spine could be widely diverse, ranging from an array of ubiquitously present benign degenerative conditions to rare, but often serious, neoplastic, vascular, infectious, traumatic, metabolic, or compressive lesions. The topographic localization of the spinal pain is often vague, as innervation of various spinal components is characteristically multisegmental, predominantly autonomic, and typically with extensive interneuronal convergence within the spinal cord.[39] The clinical presentation of the various SP syndromes is similar, and they are often present concomitantly, such as frequent simultaneous presence of degenerative disc disease, spinal stenosis, facet arthritis, and sacroiliac joint dysfunction. The range of spinal imaging techniques commonly employed for the diagnosis of SP may show similar abnormalities in symptomatic as well in asymptomatic individuals.[36,37]

In addition to the aforementioned types, SP has also been broadly divided into mechanical, nonmechanical, and visceral pain categories. Mechanical SP is ubiquitous and may be defined as pain emanating from the benign

degenerative conditions afflicting the various spinal structures, such as IVDs, facet joints, and the neural elements, or the immediately adjacent paraspinal structures, such as muscles, ligaments, periosteum and blood vessels. A range of terms has traditionally been used to describe mechanical SP such as lumbago, spondylosis, segmental or somatic dysfunction, ligamentous strain, subluxation, and facet joint, sacroiliac, or myofascial syndromes. The various conditions causing mechanical SP follow:

- Herniated discs
- Spondylosis or degenerative disc disease
- Discogenic pain, internal disc disruption or annular tears
- Spondylolisthesis or displacement of one vertebral body over the other
- Spondylolysis or defect in pars interarticularis without the vertebral slippage
- Spinal instability or anomalous movement between the contiguous vertebral bodies
- Foraminal stenosis or skeletal hypertrophy causing symptoms of nerve root compression
- Spinal canal stenosis or neurogenic claudication or myelopathic symptoms and signs
- Facet arthropathy
- Musculoligamentous strains or sprains
- Myofascial pain syndrome
- Congenital spinal conditions such as kyphosis or scoliosis

Nonmechanical SP is rare and typically has a more sinister etiology. It may result from widely diverse pathologic conditions, such as the following:

- Primary and metastatic neoplasms of the spine or its neural contents
- Infections, such as osteomyelitis of the vertebral bodies, septic discitis, paraspinal, or epidural abscess
- Noninfectious inflammatory spinal disorders such as ankylosing spondylitis, Reiter's syndrome, psoriatic spondylitis, inflammatory bowel disease
- Traumatic or pathologic fractures such as vertebral body compression fractures and dislocations
- Metabolic disorders of the spine such as Paget's disease
- Miscellaneous conditions such as Scheuermann's disease or osteochondrosis and hemangiomas

Visceral or referred SP is pain of extra spinal etiology that is referred to the low back, neck or dorsal spine. Referred SP is also less prevalent than mechanical SP, and can often be distinguished from the SP of other etiologies by the lack of spinal stiffness and the pain-free range of spinal movements. The etiology of visceral SP includes:

- Pelvic visceral disorders such as prostatitis, endometriosis, or pelvic inflammatory disease
- Renal disease such as nephrolithiasis, pyelonephritis, or perinephric abscess
- Vascular disease such as abdominal aortic aneurysm
- Gastrointestinal disease such as pancreatitis, cholecystitis, or perforated bowel

CLINICAL EVALUATION

Despite the diagnostic complexities and the daunting list of causal conditions, the majority of SP is caused by benign self-limiting conditions with symptoms characteristically resolving within 1 to 3 months.[40] A comprehensive history and physical examination are important determinants in the diagnosis of various SP syndromes.

HISTORY

A detailed history of SP patient should note the following (Table 43-2):

- Location and any radiation of pain, especially in the dermatomal distribution
- Characteristics of pain, such as burning, lancinating or aching quality
- Severity of pain; especially noted should be patient's ability to function and sleep at night
- Circumstances of onset of pain such as a history of trauma
- Factors aggravating and relieving the pain
- Patient's age
- Presence of any constitutional symptoms such as fever, malaise, or weight loss
- Special pain features such as night pains, bone pain, morning stiffness, and history of claudication
- Neurologic symptoms such as numbness, tingling, and weakness, along with any bowel or bladder dysfunction, and especially urinary retention and urinary or fecal incontinence
- History of any previous treatments and their efficacy
- Patient's detailed past medical and surgical history
- Assessment of social and psychological factors that may affect patient's pain
- Functional impact of the pain on the patient's work and activities of daily living

TABLE 43–2 Symptom Evaluation of Spinal Pain Patients

Location and any radiation of pain, especially in the dermatomal distribution
Characteristics of pain, such as burning, lancinating, or aching quality
Severity of pain, especially patient's ability to function and to sleep at night
Circumstances of onset of pain such as history of trauma
Factors aggravating and relieving the pain
Patient's age
Presence of any constitutional symptoms such as fever, malaise, or weight loss
Special pain features such as night pains, bone pain, morning stiffness, and history of claudication
Neurologic symptoms such as numbness, tingling, and weakness, along with any bowel or bladder dysfunction; especially urinary retention and urinary or fecal incontinence
History of any previous treatments and their efficacy
Patient's detailed past medical and surgical history
Assessment of social and psychological factors that may affect patient's pain
Functional impact of pain on patient's work and activities of daily living

PHYSICAL EXAMINATION

A comprehensive general physical and a detailed neurologic examination should be performed in all the patients with SP. Specific spinal examination should include:

- Assessment of gait.
- Range of spinal motion.
- Determination of local spinal and paraspinal tenderness.
- Specific tests for the clinical diagnosis of various SP syndromes, including those for nerve root irritation, facet syndrome, and sacroiliac joint dysfunction, are discussed in this book in the various chapters designated to these syndromes.

"RED FLAGS" IN PATIENT'S CLINICAL EVALUATION

Due to the high prevalence of SP, its frequent spontaneous resolution, the rarity of serious spinal disorders, and the frequent presence of abnormal findings in asymptomatic individuals, indiscriminate diagnostic testing for SP disorders would lead to inappropriate diagnosis and poor treatment results.[41] Therefore, in the United States the Agency for Health Care Policy and Research (AHCPR) developed guidelines to recognize clinical features that would signify the presence of conditions such as fractures, tumors, and infections that can pose significant threat to life or neurologic function—"the red flags" (Table 43-3).[41] Recognition of these notable clinical signs is essential as their existence would require further diagnostic testing to either rule out a serious condition or to confirm the presence of a benign diagnosis. However, it is probable that a serious spinal condition may go undetected despite a careful appraisal for these characteristic "red flags." In general, patients with benign mechanical SP should have pain mainly with spinal movements such as sitting, bending, lifting, or twisting, and the pain should improve over the course of few days to weeks. Diagnosis that cannot be confirmed, such as muscle sprain or ligamentous strain, should seldom be used in the presence of the "red flags" as this would further delay the appropriate workup; the latter is frequently the reason for serious spinal conditions being identified late in their course. The characteristic "red flags" follow.

TABLE 43–3 "Red Flags" in Patient's Clinical Evaluation

Age	<20 or >50 years of age
Duration of symptoms	Symptoms over 3 months indicate a less serious etiology
History of trauma	History of significant traumatic injury, or mild trauma in an elderly patient or in a patient with a serious medical condition
Presence of constitutional symptoms	Fever, chills, malaise, night sweats, unexplained weight loss, and so on
Presence of systemic illness	History of cancer, recent bacterial infections, intravenous drug abuse, immunosuppression, organ transplantation, and corticosteroid use
Unrelenting pain	Pain not relieved with rest, supine position, and analgesics
Presence of cauda equina syndrome	Caused by massive midline disc herniation or rarely by spinal metastases, hematoma, epidural abscess, traumatic compression, acute transverse myelitis, and abdominal aortic dissection. Symptoms include bilateral, but often unequal, lower extremity radicular pains and weakness, gait disturbances, abdominal discomfort and overflow incontinence. Physical examination exhibits neurologic dysfunction, saddle anesthesia, diminished anal sphincter tone, and urinary bladder retention. Diagnosis must be made by imaging the entire spine. Treatment is urgent decompressive surgery

Age: Patients less than 20 or over 50 years of age are suspect, as younger patients have a higher incidence of congenital and developmental anomalies, while older patients have a greater likelihood of neoplasms, pathologic fractures, serious infections, and life-threatening extraspinal pathologic conditions.

Duration of symptoms: Symptoms lasting over 3 months indicate a less serious etiology.

History of trauma: History of significant traumatic injury or mild trauma in an elderly patient or in a patient with a serious medical condition may indicate traumatic spinal injury.

Presence of constitutional symptoms: Examples such as a history of fever, chills, malaise, night sweats, and unexplained weight loss indicate a more sinister etiology of SP.

Presence of systemic illness: Patients with a history of cancer, recent bacterial infections, intravenous drug abuse, immunosuppression, organ transplantation, and corticosteroid use are at higher risk for pathologic fractures, epidural and vertebral body abscesses, and metastasis.

Unrelenting pain: Pain of a benign etiology is typically relieved with rest and the supine position, especially at night, while pain from a serious pathologic conditions is typically unrelenting, worse at night, and unresponsive to rest and analgesics.

Presence of cauda equina syndrome (CES): This syndrome is caused by acute compression of the spinal cord or the nerve roots of the cauda equina. CES is characteristically caused by a massive midline IVD herniation or a smaller disc herniation in a previous stenotic spine.[42,43] Rarely, CES may be caused by spinal metastases, hematoma, epidural abscess, traumatic compression, acute transverse myelitis, or abdominal aortic dissection.[44] Typical symptoms include bilateral, but often unequal, lower extremity radicular pains and weakness, gait disturbances, abdominal discomfort from urinary retention, and overflow incontinence. In addition to the positive findings on neurologic examination, the patient's physical examination typically exhibits saddle anesthesia—diminished sensation in the buttocks and perineum—diminished anal sphincter tone, and the evidence of urinary bladder retention. Due to the possibility of spinal cord compression at higher levels, CES must be diagnosed by imaging of the entire spine.[45] CES is one of the rare neurosurgical emergencies that requires urgent decompressive surgery in order to reduce permanent neurologic disability.[44]

DIAGNOSTIC TESTING

As the most commonly used tests for diagnosis of SP syndromes, especially the imaging studies, would reveal abnormal findings in asymptomatic individuals,[36,37,46,47] it is necessary that the imaging findings are corroborated with patient signs and symptoms. The diagnosis is not based solely on the test results. Additionally, as SP conditions are commonly self-limiting and benign, in the absence of "red flags" in the clinical history, diagnostic testing is not recommended for SP of less than 4 to 6 weeks.[41,48] Ordering tests selectively should then prevent inappropriate diagnosis and treatment and thus poor outcomes.[41] In addition to the diagnosis of specific SP syndromes, diagnostic tests are also used to determine the site of surgical or minimally invasive pain intervention. Following are the diagnostic modalities frequently used in the diagnosis of SP.

PLAIN RADIOGRAPHY

Plain radiography allows evaluation of the bony spinal anatomy. It can reliably diagnose pathologic spinal lesions such as fractures, deformities, transitional vertebra, and spondylolisthesis. Subtle spinal abnormalities seen on plain radiography, such as lumbar lordosis, disc space narrowing, arthritic changes, ossification of the vertebral end plates, and abnormal range of spinal movements or spinal instability, are frequently encountered in asymptomatic individuals.[49,50] Spinal radiography therefore exhibits a high rate of abnormal findings in asymptomatic individuals.[47,51] Major drawbacks of plain spinal radiography include its inability to visualize the soft tissue structures and their abnormalities, such as herniated disc, neural element compression, and soft tissue neoplasms. Spinal x-rays may therefore appear normal even in the presence of significant spinal soft tissue pathology. Spinal roentgenograms have traditionally been the earliest imaging test performed in the evaluation of patients with SP, chiefly because they are relatively inexpensive, widely available, and easy to perform. Therefore, although the routine use of spinal radiography has been discouraged,[47,52] in the presence of "red flags" in the clinical history, spinal roentgenograms are often the initial screening tests.

Traditional plain radiography sequences includes anteroposterior (AP), lateral, and oblique views. In the AP view indicators of normal spinal morphology include vertical alignment of the spinous processes, smooth undulating borders created by lateral masses, and uniformity among the disc spaces. Misalignment of the spinous processes suggests a rotational injury such as unilateral facet dislocation. The AP view of the lumbar spine should include the entire pelvis to allow the assessment of acetabulum and femoral heads and the lower portion of the thoracic spine due to the high occurrence of injury between T12 and L2 spinal levels. The lateral views provides a superior image of the vertebral bodies, facet joints, lordotic spinal curvature, disc space height, and spondylolisthesis. Decreased disc space height is a relatively non specific change and may indicate disc degeneration, disc space infection, and postsurgical changes. Oblique views, taken with the x-ray tube angled at (45 degrees), provide enhanced views of the neural foraminae and pars interarticularis. These views best demonstrate foraminal abnormalities and spondylosis. Flexion-extension views are typically used to demonstrate spinal instability as a cause of chronic pain. However, these views can also be used in trauma patients to assess ligamentous injury. When used to diagnosis ligamentous injury, the flexion-extension views should be used exclusively in patients with otherwise normal radiographs and who in addition are neurologically intact, are cooperative, and are able to recognize early onset of pain or neurologic symptoms with spinal movement.

BONE SCINTIGRAPHY

Bone scintigraphy creates images by scanning for the presence of radiographic compounds such as technetium-99m phosphate or gallium-67 citrate. Thus, whereas plain radiography and computerized and magnetic resonance scanning reveals simple morphologic changes, bone scintigraphy detects biochemical osseous processes and is valuable when clinical findings are suspicious of spinal osteomyelitis, neoplasms, or occult fracture. Primary spinal tumors, such as osteoid osteoma, osteoblastoma, aneurysmal bone cyst, and osteochondroma, are typically benign and show as active lesions on bone scintigraphy. Osseous spinal metastases typically appear as multiple foci of increased tracer uptake that are asymmetrically scattered. Occasionally, aggressive bony tumors, such as myeloma, may not invoke an osteoblastic response and may therefore yield a negative bone scan. Also, in occasional extreme cases of spinal metastases diffusely increased tracer uptake may result in a false-negative bone scan. Topographic location of the spinal osseous tumors is also pertinent. Lesions affecting the pedicles are typically malignant, while facet joint lesions are apt to be benign. The vertebral body and spinous process lesions are just as likely to be benign or malignant. Bone scintigraphy with the addition of single-photon emission computed tomography (SPECT) provides a three-dimensional spinal image and enhanced topographic tumor location. SPECT has been used to distinguish benign from malignant osseous neoplasms.[53,54]

COMPUTED TOMOGRAPHY

Computed tomography (CT) uses radiologic data to generate contiguous, overlapping axial images of the scanned area. The imaging data can also be reformatted to construct views in any desired plane. Spinal CT is most useful in evaluating osseous details of the spine in an axial plane particularly the facet joints and the lateral recesses. It is most valuable in diagnosing fractures, tumors involving the spine, and in showing the relative position of one osseous structure to another, such as partial or complete dislocations and spondylolisthesis. The resolution of the soft tissue structures on spinal CT is inferior to magnetic resonance imaging (MRI). Spinal CT cannot reliably distinguish between herniated IVD and epidural scar tissue and amongst various spinal canal lesions such as neoplasms of the spinal cord or the nerve roots. The routine use of spinal CT for the diagnosis of the soft tissue intraspinal canal lesions is therefore discouraged.[55] When combined with myelography (CT myelogram), the results are

comparable to spinal MRI and CT myelogram can be used as a substitute when MRI is contraindicated.[55] One significant limitation of spinal CT is motion artifact, the ensuing indistinct images and the chance of imprecise diagnosis of less distinguishing lesions, such as nondisplaced fractures. Radiation exposure is another significant hazard that limits the extent to which spinal CT can be employed. Spiral CT reduces exposure time, radiation hazard and motion artifact. Three-dimensional CT is a newer modality that provides higher resolution three dimensional images of the spine. This modality is currently being used only for complicated spinal problems such as failed back surgery syndrome.

MAGNETIC RESONANCE IMAGING

The powerful magnetic fields generated in the MRI scanner align the water molecules or protons, constituting the bulk of the body mass, in the direction of the magnetic fields applied. Brief bursts of radiofrequency (RF) waves are then applied and the resulting electromagnetic fields alter the proton alignment. Cessation of the RF field results is the protons decaying to their original state and releasing energy as photons, which are detected by the MRI scanner. The protons in the various tissues return to the equilibrium state at dissimilar rates and an image of various soft tissues is therefore created. By changing timing of the various scanner sequences, like the echo time (T_E) and the repetition time (T_R), the contrast between the various body tissues can be altered. T_2 weighted images use a spin echo (SE) sequence, with long T_E and long T_R intervals, and the water-containing tissues appear, whereas while fat-rich or water-deprived tissues appear dark. T_1 weighted images in contrast use a gradient echo (GRE) sequence, with short T_E and short T_R sequencing, and the tissue contrast on T_1 weighted images is the opposite of the T_2 weighted images. The cerebrospinal fluid appears dark on T_1 weighted images, and it appears white on T_2 weighted images. On T_1 weighted images a normal IVD appears dark and homogenous, whereas it appears brighter on T_2 weighted images—the NP with its greater water content appears brighter than the AF.

Although high quality osseous images can be achieved with spinal CT, MRI is currently considered the gold standard in spinal imaging. MRI provides sharper distinction between the various soft tissues, and the overall soft tissue resolution is superior. MRI offers excellent images of the spinal canal and its neural contents, the neural foraminae and the exiting nerve roots, and the disc spaces and its contents. MRI also allows evaluation of complete spine in various planes. A contrast enhanced MRI can be performed when greater distinction between various soft tissues is required, such as differentiation between scar tissue and recurrent IVD herniation in patients with a history of previous spine surgery. However, in contrast to spinal CT, which uses radiopaque contrast agents such as iodine or barium comprised of higher atomic weight elements than the surrounding tissues, MRI uses contrast agents such as gadolinium and manganese that enhance tissue resolution by their paramagnetic properties. MRI is considered relatively safe with no known biological effects. Limitations of MRI, however, include lengthy examination time, claustrophobia, and its effects on metallic objects. MRI is contraindicated in the presence of ferromagnetic implants, such as cardiac pacemakers, intracranial aneurysm clips, mechanical heart valves, and intraocular foreign bodies. Metallic stabilization devices used in spinal surgery cast artifacts and may render spinal imaging almost unattainable. Like other spinal imaging modalities, spinal MRI may frequently detect findings in asymptomatic individuals.[36,37]

ELECTRODIAGNOSTIC STUDIES

Electrodiagnostic studies encompass the following:

Electromyography (EMG): Study of spontaneous or evoked skeletal muscle electrical activity.
Nerve conduction studies (NCV): Study of conductive abilities of the motor and sensory nerves.
Evoked potentials: Study of brain electrical activity evoked from various nervous system locations, such as somatosensory-evoked potentials (SSEPs) and motor-evoked potentials (MEPs).

The electrodiagnostic studies are useful in localizing the pathologic lesion, determining the extent of the neural injury, predicting the course of recovery, and in determining whether the radiologic abnormalities observed are the likely source of patient's symptoms.[56] These tests are especially useful when the clinical evaluation is inconclusive in distinguishing between radicular and peripheral neuropathic symptoms. EMG or NCVs however provide scant information on the symptom etiology, and the abnormal findings may take several weeks before they are first recognized. The use of SSEPs and MEPs is generally limited to identifying intraoperative nerve injury during spinal surgery. Compared to spinal imaging, the electrodiagnostic studies appear less sensitive; however, they have greater diagnostic specificity.[57]

PSYCHOSOCIAL TESTING

Screening for nonphysical factors is crucial in the management of SP patients. Psychological, occupational, and socioeconomic factors can complicate both the assessment and the treatment of SP patients. For example, patients with work dissatisfaction are at a greater risk for LBP with have poor outcomes.[58] Moreover, patients with affective disorder, such as depression, and those with a history of substance abuse are also more prone to chronic pain disorders. Pending litigation and disability issues also adversely affect SP treatment.

OTHER DIAGNOSTIC TESTS

A variety of other diagnostic and laboratory tests, such as complete blood count (CBC), urine analysis (UA), erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), rheumatoid factor (RH-factor), anti-nuclear antibodies (ANA) and HLA-B27 antigen, are useful when nondegenerative conditions, such as tumors, infections, and rheumatologic disorders, are considered a cause of SP.

MANAGEMENT OF SPINAL DISORDERS

NONINVASIVE TREATMENTS

Following are various noninvasive treatments employed in the treatment of SP (Table 43-4).

Rest

Strict bed rest has traditionally been the mainstay of acute SP treatment. The current evidence, however, suggests that for SP treatment prolonged bed rest is harmful,[59] and bed rest of more than a week is imprudent.[60] Furthermore, the continuation of daily activities and early return to work has been reported to shorten the chronic disability and duration of work absence.[61,62]

Pharmacologic Therapy

- *Nonsteroidal anti-inflammatory drugs (NSAIDs):* These drugs are often considered moderately effective for the short-term relief of acute LBP.[63] Nevertheless, the support for the use of NSAIDs in the treatment of chronic LBP is lacking. Additionally, information as to which specific NSAID is more effective for SP is also lacking.[64]
- *Narcotics:* The short-term use of narcotics may be contemplated for the relief of acute SP. Conversely, a need for prolonged narcotic therapy should prompt reevaluation of patient's motivations and the source of SP. Due to the chronicity of SP, these patients are at an increased risk of developing tolerance and addiction with prolonged narcotic use. These medications should therefore be limited to acute SP and exacerbations of chronic SP.[65]
- *Muscle relaxants:* The use of muscle relaxants has been shown to reduce pain, muscle tension, and immobility in patients with SP.[66]
- *Corticosteroids:* These are often prescribed orally, and often parenterally, in the treatment of acute IVD herniation; nevertheless, there is little evidence in the literature to support this practice.[67,68]
- *Calcitonin:* This drug has been shown to be beneficial for pain ensuing from spinal stenosis caused by Paget disease.[69]

Physical Therapy

Physical therapy and rehabilitation interventions used in the management of SP include the following[70]:

- Body mechanics, ergonomics, posture awareness, and activities of daily living (ADL) training
- Strengthening and stretching exercises
- Organized functional training programs
- Therapeutic massage
- Joint mobilizations and manipulations
- Mechanical traction
- Biofeedback
- Electrical muscle stimulation
- Transcutaneous electrical nerve stimulation (TENS)
- Application of superficial and deep thermal modalities
- Cryotherapy
- Work hardening

TABLE 43–4 Treatments for Spinal Pain

Noninvasive Treatments for Spinal Pain	
Rest	
Pharmacologic therapy	Nonsteroidal anti-inflammatory drugs Narcotics Muscle relaxants Corticosteroids Calcitonin
Physical therapy	Body mechanics, ergonomics, posture awareness, and activities of daily living (ADL) training Strengthening and stretching exercises Organized functional training programs Therapeutic massage Joint mobilizations and manipulations Mechanical traction Biofeedback Electrical muscle stimulation Transcutaneous electrical nerve stimulation (TENS) Application of superficial and deep thermal modalities Cryotherapy Work hardening
Acupuncture	
Spinal manipulation	
Minimally Invasive Treatments for Spinal Pain	
Injection therapy	Epidural steroid injections Facet joint injections Sacroiliac joint injection Trigger pain injections
Neuroablative procedures	Chemical neurolysis Cryoablation Radiofrequency ablation
Intradiscal procedure	Discography Percutaneous disc decompression Intradiscal electrothermal therapy Intradiscal bioculoplasty
Spinal Surgery	
Decompression surgery	Discectomy Microdiscectomy Endoscopic discectomy Decompression for fixed osseous stenosis
Fusion	Anterior fusion Posterior fusion Circumferential fusion Transforaminal lumbar interbody fusion
Disc arthroplasty	SB Charite III ProDisc Maverick Flexcore
Spinal reconstruction	Various techniques

The treatment goals of various physical therapy modalities include:

- Pain relief
- Reduction in muscle spasm
- Improved range of spinal motion (ROM)

- Improved strength
- Postural correction
- Improvement in functional status

Although the precise role of the various physical therapy modalities in the treatment of SP is not fully obvious, the evidence is suggestive of the beneficial effects of general exercise programs. Strengthening exercise programs that target the paraspinal musculature, and general exercise programs that promote weight loss are considered most beneficial in alleviating LBP, promoting return to work, resuming normal daily activities and reducing the need for surgical intervention.[71] There is inadequate evidence that specific back exercises and passive physical therapy techniques such as thermotherapy, therapeutic massage, biofeedback, mechanical traction, therapeutic ultrasound, and TENS produce valuable clinical improvement in patients with SP.[72]

Acupuncture

An analysis of 11 randomized controlled trials (RCTs) on the use of acupuncture in patients with nonspecific LBP led to the following conclusions: (1) overall methodologic quality of the RCTs was low; (2) none of the trials clearly evaluated acupuncture; (3) although moderate evidence existed of the efficacy of acupuncture, it was comparable to trigger-point injection and TENS; (4) evidence on the efficacy of acupuncture was lacking when compared to no treatment; and (5) there was limited evidence that acupuncture was as effective as placebo or sham treatment.[73] The authors of this review recommended against the routine use of acupuncture for the treatment of LBP.[73] Similar conclusions were presented in a comparable review.[74]

Spinal Manipulation

A number of RCTs and several meta-analyses of the use of spinal manipulations for the treatment of both acute and chronic LBP are available.[75–77] Overall, the results of these studies demonstrate that although there may be some advantage of manipulative therapy in the treatment of acute LBP, no statistical or clinical advantage of spinal manipulations over the conventional therapy for the treatment of chronic LBP exists.

Biofeedback Treatments

Biofeedback entails external feedback, which translates physiologic muscle activity (often using EMG) into visual or auditory signals that help the patient reduce muscle tension and pain. There is limited evidence that biofeedback techniques are ineffective for the treatment of chronic LBP, and studies of the use of these techniques in the treatment of acute LBP are lacking.[78]

MINIMALLY INVASIVE TREATMENTS

The minimally invasive treatments for SP (Table 43-4) such as a range of spinal injections (Fig. 43-2), neuroablation techniques, and percutaneous disc procedures are discussed in detail in the sections of this book pertained to the specific pain syndromes.

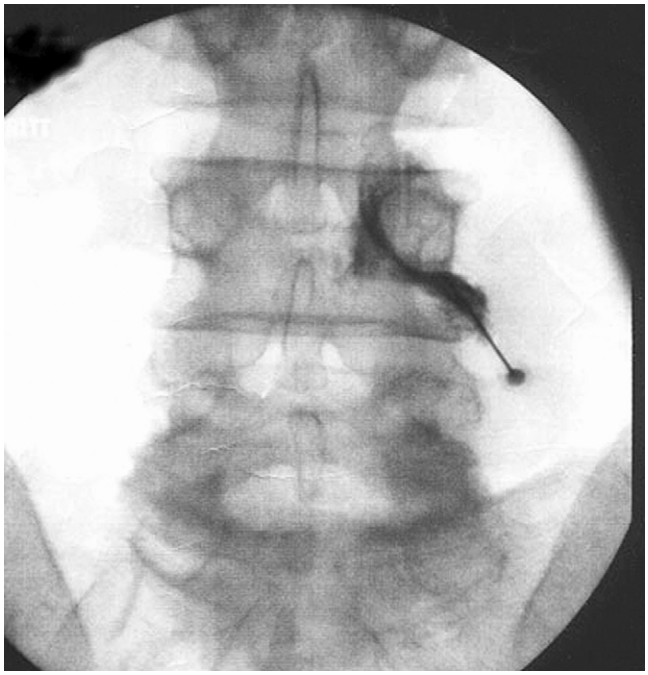

FIGURE 43-2 Fluoroscopic image of right-sided L4–L5 transforaminal steroid injection. Dye injection prior to steroid demonstrating proper position and backflow along L4 nerve root sheath.

SURGICAL TREATMENT

Although a detailed discussion of the various surgical treatments available for SP patients is beyond the scope of this book, an overview is pursued in this section. The majority of surgical treatments intending to relieve SP (Table 43-4) typically incorporate an element of neurologic decompression and/or fusion; more recently, though, disc replacement surgery has been regularly employed.

Spinal Decompression

Disc decompression surgery is typically reserved for patients with a herniated IVD, with distinctive symptoms of persistent radicular pain, positive straight leg raise test, and the imaging studies confirming the presence of herniated IVD. The target for surgical decompression is determined by careful correlation of patient's symptoms and the lesions on the imaging studies. Classical discectomy has been in vogue since Mixter and Barr's classic report in 1934.[79] Classical discectomy has since remained the most commonly performed spinal-surgery procedure to which all other disc surgeries are commonly compared. Popularized in the late 1970s, the microdiscectomy procedure is less invasive and aims to permit faster recovery and early return to work.[80,81] Although the various discectomy techniques differ, the procedure in essence involves laminectomy or laminotomy, release of ligamentum flavum, removal of the herniated disc fragments, and a vertical annulotomy for the removal of nonherniated disc material. More recently, minimal invasive endoscopic discectomy has been performed, which involves limited exposure through an 18-mm tubular retractor (Figure 43-3).

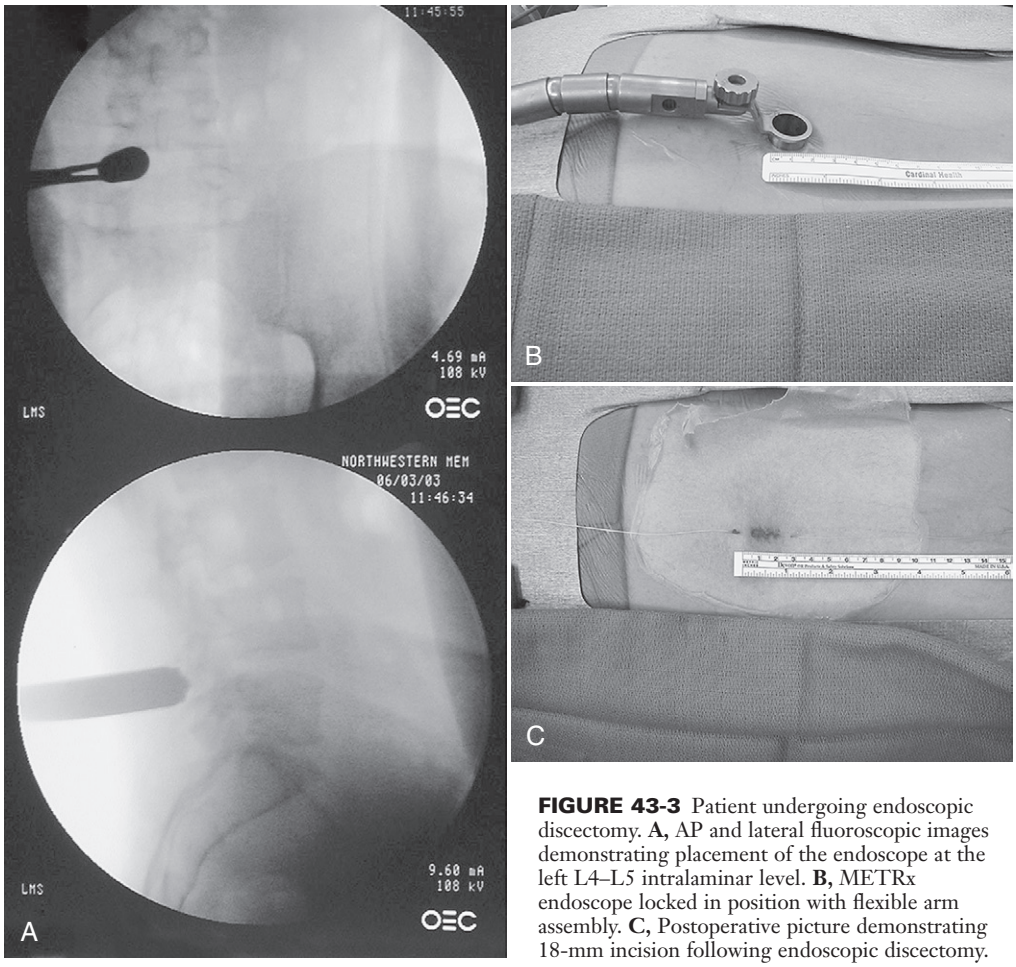

FIGURE 43-3 Patient undergoing endoscopic discectomy. **A,** AP and lateral fluoroscopic images demonstrating placement of the endoscope at the left L4–L5 intralaminar level. **B,** METRx endoscope locked in position with flexible arm assembly. **C,** Postoperative picture demonstrating 18-mm incision following endoscopic discectomy.

In older patients the nerve root compressive symptoms are often the result of local degenerative changes involving the disc, facet joints, and the local ligaments. Such changes may include disc bulges and herniations, facet and ligamentous hypertrophy, osteochondral spurs, and spondylolisthesis. Surgical decompression for these patients characteristically involves osseous decompression of the neural foraminae, central canal, and the lateral recess. In many such cases, especially in the presence of spondylolisthesis and when greater than 50% of the facet joints are resected, a fusion procedure may needed in addition to avoid the ensuing spinal instability.

Spinal Fusion

Although spinal fusion surgery has been performed for the past 100 years, it has been particularly popular in recent years. Common indications, for spinal fusion surgery include ensuing instability after decompressive spinal surgery and diverse causes of mechanical SP such as spondylolysis, spondylolisthesis, degenerative arthritis, spinal instability, discogenic pain and scoliosis. During the spinal fusion surgery the dysfunctional spinal motion segments—including the incriminating disc and the painful degenerative joints—may be resected and the spine is characteristically rigidly stabilized by using various mechanical fusion devices such as pedicular screws, interpedicular fixation plates, and intervertebral spacers such as cylindrical cages (Figure 43-4). Mechanical spinal instrumentation, however, is subject to fatigue failure and eventual fracture unless osseous spinal fusion is attained by osteogenesis, classically by the use of bone graft in the vascularized tissue bed. The key elements required for spinal osteogenesis include precursor cells capable of transformation into bone-forming osteoblasts, osteoconductive materials that would serve as scaffolds for the formation of new bone, and osteoinductive growth factors that will promote differentiation of progenitor cells into osteoblasts.[82] Autologous bone graft remains the gold standard osteogenetic material because it contains all three essential elements. Limitations of autologous bone graft, however, include the amount of available graft material and the morbidity associated with harvesting autologous bone graft. These limitations have led to the use of other osteogenetic materials including bone graft extenders—demineralized bone matrix, calcium carbonate, hydroxyapatite-tricalcium phosphate, bone graft substitutes, and, more recently, osteoinductive substitutes, such as recombinant human bone morphogenic protein (BMP).[83] Spinal fusion can be performed by either posterior, posterolateral, anterior, or combined circumferential (360°) approach. More recently, a transforaminal lumbar interbody fusion (TLIF) technique is used, which provides the

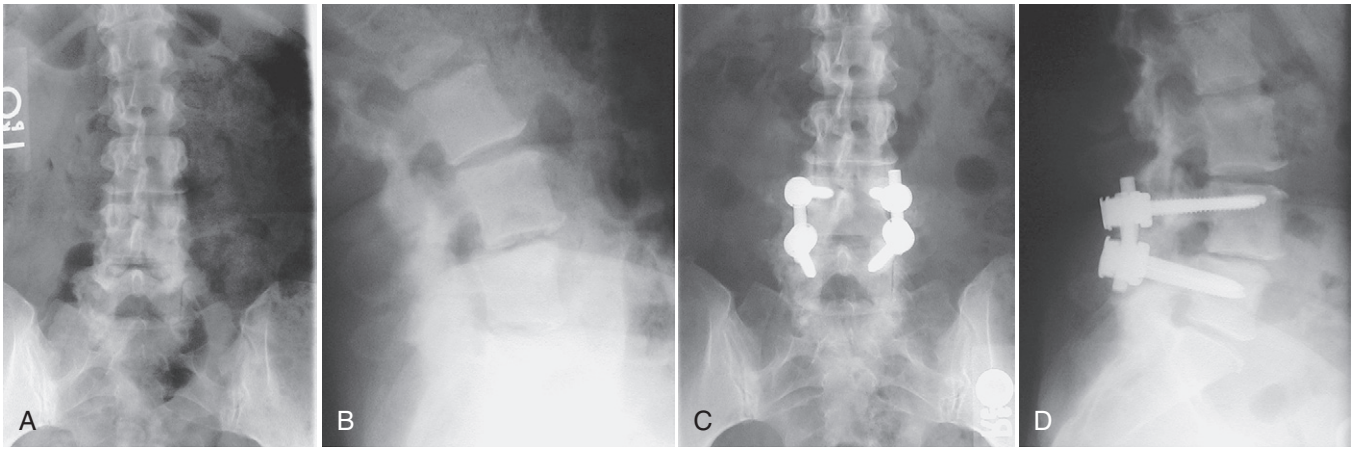

FIGURE 43-4 **A,** AP and, **B,** lateral lumbar spine radiographs demonstrating Grade 1 spondylolisthesis in a 47-year-old woman with disabling back and leg pain refractory to nonoperative treatment. **C, D,** Postoperative radiographs demonstrating stable fusion 1 year following posterior decompression and fusion with supplemental instrumentation. Note the robust fusion mass bridging transverse processes laterally. The patient is pain free and has returned to full level of activity including triathlons and skiing.

advantages of a circumferential fusion through a lower-risk posterior approach.[84] The actual fusion rates after fusion surgery vary from 80% for posterolateral fusions to 97% for circumferential fusions.[85] The results of the spinal fusion surgery vary vastly depending on the condition for which the surgery is performed. When performed for spinal deformities and spondylolisthesis, the results reported are generally favorable.[86,87] However, treatment of degenerative disc disease and discogenic pain with spinal fusion has remained controversial and the reported success is modest.[88] Patients with discogenic pain tend to have greater clinical benefits when the incriminating disc is removed.

Disc Arthroplasty

Despite its popularity, spinal fusion surgery remains a salvage procedure, as it reduces spinal mobility and increases stress and consequently degeneration at adjacent spinal levels. The concept of disc arthroplasty was envisaged to avert these shortcomings of spinal fusion surgery. During disc arthroplasty the offending disc is surgically removed and replaced by an artificial disc. When compared to spinal fusion, the major benefit of disc replacement surgery include preservation of spinal range of motion and decreased adjacent spinal segment degeneration. The primary indication for disc arthroplasty is recalcitrant disabling LBP secondary to discogenic disc disease, which is confirmed by MRI and discography. The exclusion criteria include evidence of nerve root compression, facet, and sacroiliac joint arthropathy. In contrast to the prolonged rehabilitation that typically follows spinal fusion surgery early and progressive spinal motion and functional recovery is characteristically encouraged after disc arthroplasty.

Although disc replacement surgery has been advocated since the 1950s, it was not until the early 1980s that a viable design with encouraging results was introduced. Several types of artificial discs are currently marketed; these include SB Charite III, ProDisc, Maverick, and Flexcore. The Link SB Charite III is currently the most commonly used prosthesis. It consists of two cobalt-chrome endplates, with a sliding polyethylene core. The endplates are anchored to the vertebral bodies by teeth and later by the bony in-growth. The biomechanical studies of artificial disc replacement demonstrate increased range of motion in flexion, extension, and torsion, whereas relative immobility was seen in lateral bending. Although the initial clinical results of artificial disc replacement are encouraging, this technique remains largely untried.[89]

Spinal Reconstruction

Spinal reconstruction is contemplated when the disease progression either destroys the structural integrity of the spine or produces deformity that alters its normal biomechanics. Conditions requiring spinal reconstruction surgery may include traumatic spine injuries, spinal infections and tumors, and spinal deformities such as scoliosis and kyphosis. More recently adverse consequences of failed prior spinal surgery are a major cause of spinal reconstruction surgery. The principles of spinal reconstruction surgery include resection of diseased tissues, soft tissue, and bony release to allow spinal realignment and rigid fixation to maintain spinal stability until the biologic fusion is achieved. Proper spinal realignment must restore the physiologic lumbar lordosis and thoracic kyphosis. An appropriate graft or implant length must be selected to maintain this sagittal balance. Spinal reconstruction typically involves anterior release in the form of vertebral body (corpectomy) and disc (discectomy) resection and posterior release that incorporates chevron osteotomies.[90] In severe cases of spinal scoliosis the rib cage itself may become ankylosed and may require release in the form of rib head resections, rib osteotomy, and pedicle subtraction osteotomy.[91,92] Once the spinal segment is appropriately realigned it must be rigidly fixed to maintain the alignment, until the successful osseous fusion is achieved (Fig. 43-5). The various currently used instrumentation systems include pedicle screws connected by rods, hooks, and sublaminar cables.

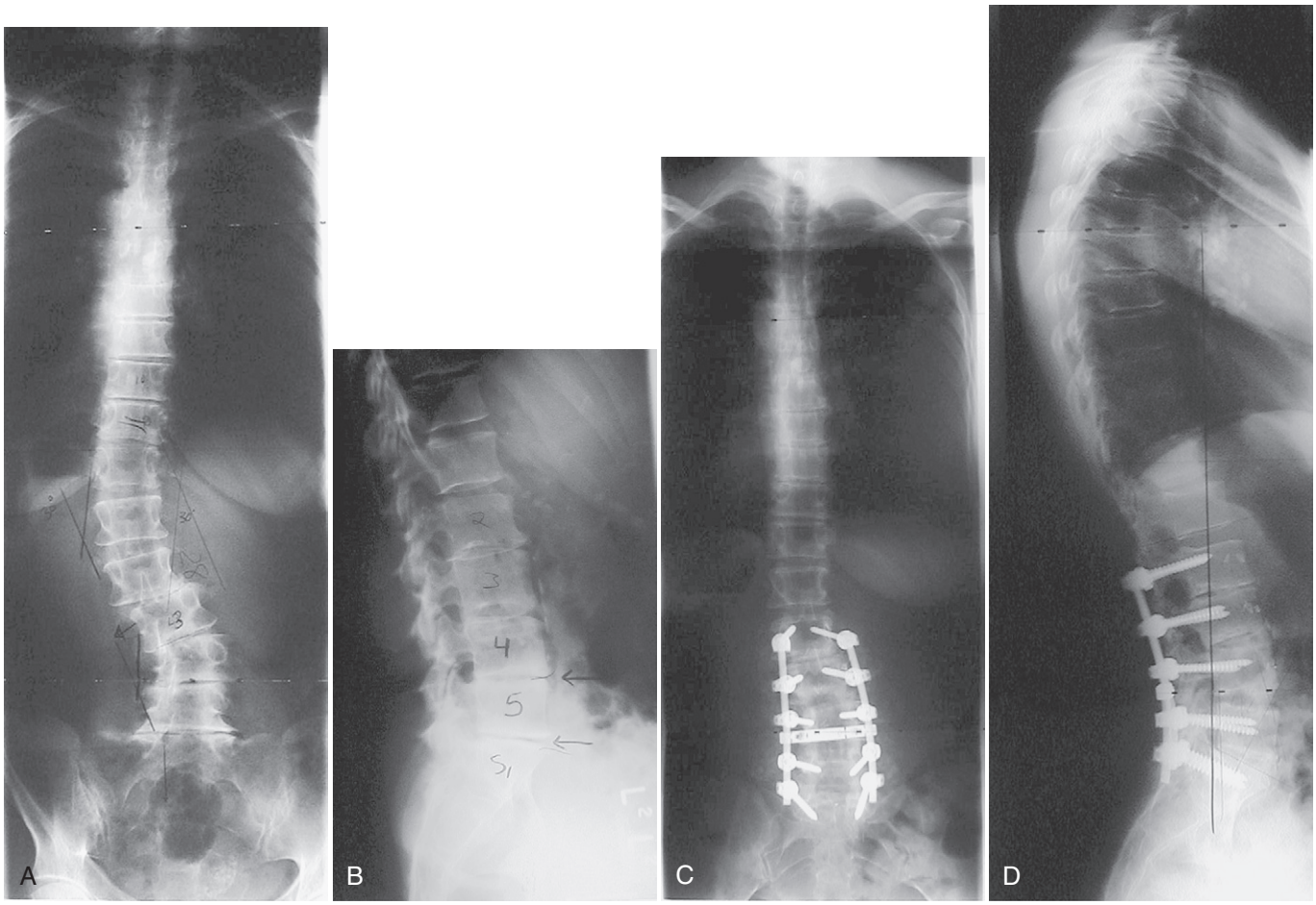

FIGURE 43-5 A 64-year-old woman with degenerative scoliosis and disabling low back and radicular leg pain. **A,** AP radiograph demonstrates severe lateral listhesis at L2–L3 and L3–L4 resulting in symptomatic compressive neuropathy. **B,** Lateral radiograph demonstrating severe disc degeneration and consequent loss of lumbar lordosis. She was treated with anterior–posterior fusion, instrumentation, and decompression. **C,** AP radiograph demonstrates correction of lateral listhesis and tilt. **D,** Lateral film shows excellent restoration of lumbar lordosis with structural interbody allograft.

CONCLUSION

Patients with SP frequently have pain emanating from multiple co-existing pain generators; the diagnosis of a specific pain syndrome consequently is often uncertain. In addition, with their propensity to show positive results in asymptomatic individuals and relatively unimposing findings in many patients with SP, the available diagnostic tests are frequently unable to precisely diagnose the basis of patient's pain and therefore the target for various treatments. This situation is further aggravated by the fact that the available treatments for SP are not universally effective. It is therefore not surprising that due to the frequent lack of adequate explanation for their symptoms and poor treatment results, chronic SP patients are often resentful towards the care they have received and may develop in addition a variety of behavioral, substance abuse, disability, and other psychosocial issues. To avoid these predicaments, it is vital that patients with chronic SP clearly understand the nature of their spinal disorder, develop reasonable expectations from their treatments and care providers, and have realistic outlook for their quality of life.

Because acute SP frequently has a favorable natural history, in the absence of progressive neurologic deficits or "red flags," expectant and symptomatic treatment may be sufficient. This may include a short period of rest and analgesics and early return to function and normal activities. On the contrary, due to its persistent and recurrent nature and with often accompanying psychosocial issues, chronic SP may best be treated by adopting a multidisciplinary approach with addition of psychosocial, rehabilitative, and functional restoration. An exercise program may be introduced in all patients to minimize the risk of recurrent SP. Furthermore, SP patients should be educated in their role for preventing spinal injury and in reducing their ongoing pain. Some of these general preventative instructions may include measures as simple as assuming appropriate posture for sitting, driving, and lifting; attempts to lose weight; smoking cessation; and adopting a healthy lifestyle.

Because the invasive spinal procedures are often associated with significant risks and can be exorbitantly expensive, the potential risks of these procedures must be carefully weighed against any potential benefits. Due to the self-limiting nature of many painful spinal conditions, the invasive spinal procedures are best suited for the small group of patients who have failed to improve with more conservative treatments. Despite an ever-growing array of invasive

treatment options available, each patient must be uniquely and thoroughly evaluated before a specific treatment option is recommended. Flagrant and improper use of the various invasive spinal procedures exposes SP patients to unnecessary risks at an extreme cost to the individual patient and the society as a whole.[93]

KEY POINTS

- Spinal pain is prevalent in general population, and it is considered the most common reason for lost work time, workers' compensation claims, and early social security disability.
- Acute spinal pain is typically self-limiting, whereas chronic spinal pain could often be persistent, recurring, and frequently associated with psychosocial, behavioral, and substance abuse– and disability-related issues.
- Intervertebral disc cells function in a precarious anaerobic environment that can be adversely affected by a host of hereditary and environmental factors.
- The structural changes within the disc could change the disc dynamics that may lead to degenerative changes within the spinal motion segment and the contiguous spinal structures.
- The origin of spinal pain is versatile and it can originate from a range of spinal column components and from structures adjacent to the spine.
- The innervation of the various spinal components is complex and the topographic localization of the spinal pain is often vague.
- The ubiquitous nonspecific or mechanical spinal pain caused by benign degenerative conditions must be differentiated from a wide range of other uncommon but often perilous pathologic conditions.
- The diagnosis of the various spinal pain syndromes is often made arduous by the lack of specific diagnostic tests.
- Due to its high prevalence and frequent spontaneous resolution, spinal pain should be further appraised only when red flags are present in the clinical evaluation of a spinal pain patient.
- In the absence of progressive neurologic deficits or the red flags, acute spinal pain should be treated symptomatically, whereas chronic spinal pain is best treated by implementing a multidisciplinary approach.
- Invasive spinal procedures are best suited for a small group of patients with unrelenting chronic spinal pain who have failed to improve with conservative treatments.

REFERENCES

Access the reference list online at http://www.expertconsult.com