Low Back Pain:
Review of Diagnosis and Therapy

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Low back pain is a patient complaint frequently encountered in the emergency department setting. The disease entity is often a diagnostic challenge with a subtle presentation, but can be accompanied by significant neurovascular complications. Current topics of controversy include the utility of radiologic evaluation, pharmacologic and holistic treatment strategies, as well as guidelines for urgent referral of patients with lumbar pain. (Am J Emerg Med 1991;9:328-335. Copyright © 1991 by W.B. Saunders Company)

It has been suggested that 80% of the population will at some point suffer from low back pain, with as many as 50% progressing to a recurrent pain syndrome.[1] Quantitative estimates obtained from the National Center for Health Statistics data report that the 17 to 75 million patients affected are responsible for 32 million or 3% of all health care visits.[2,3] Analysis of disease classification finds that low back pain is the seventh most common presentation to internists, the second most likely cause of work absenteeism following the common cold, and the most common musculoskeletal presenting complaint.[2,4]

Expanding upon the occupational effects, low back pain is the major cause of disability in young patients and the third leading cause in patients over 45 years of age, after cardiac and arthritic conditions.[5,6] The financial expenditure is also significant with an estimated 20 billion dollars required for diagnosis, therapy, loss of productivity, and legal fees.[4] It has been suggested that the cost of diagnosis and therapy of low back pain is the least cost effective of any disease entity.[7]

Although most cases of low back pain are acute in onset and resolution, there is a finite progression to a refractory condition, the low back pain syndrome. Analysis of disease course shows that 74.2% of patients improve within 1 month, 87.3% within 3 months, and 92.6% within 6 months.[8] The remaining 7.4% progress to a chronic pain syndrome, responsible for the majority of health care and financial resources. Thus, efficient diagnosis and treatment of patients presenting in an ambulatory care setting is the goal of emergency department therapy.

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ANATOMY

Analysis of low back pain begins with an understanding of normal spine anatomy including bone, ligament, and neurovascular components. The spinal column consists of 33 vertebrae, with the lower back composed of 5 mobile lumbar segments and 9 fixed sacrococcygeal segments.[9] The normal curvature of the static spine finds the cervical and lumbar regions in a convex and the thoracic region in concave alignment.[11]

A functional classification of the lumbar vertebrae has been advanced by Cailliet.[10] The anterior weight-bearing segment consists of the vertebral body and intervertebral disc.[10] The disc is composed of the nucleus pulposus, a 90% water-based collagen and glycoprotein core, encased by the annular fibrosus.[6] Neural element protection is furnished by the posterior segment consisting of the vertebral arch, posterior spinal arch and transverse processes, and the superior and inferior facets.[6] These vertebrae are maintained in alignment by the anterior and posterior longitudinal ligaments with structural integrity provided by the former because of a 50% decrease in width of the posterior ligament at L1-5.[10] Spinal mobility is assisted by the paravertebral extensors, the strongest muscle unit present in the body.[12] The erector spinae or sacrospinalis group consists of a superficial layer consisting of the iliocostal, longissimus, spinalis, and a deep layer consisting of the semispinalis, multifidus, and rotatores muscles.[10] Lastly, low back pain and subsequent complications require focus on the nervous system. The nerve root of each spinal segment exits through the intravertebral foramen at the appropriate level. The posterior or dorsal root provides sensory innervation, and the anterior or ventral root is responsible for motor function.[6] Further subdivision establishes the posterior primary ramus innervating the facet joints, and the sinuvertebral nerve innervating the disc annulus, posterior longitudinal ligament, and periosteum. Thus, pain-sensitive structures implicated in low back pain include disc annulus, facet synovial lining, posterior longitudinal ligament, and paraspinous musculature.[10]

PATHOPHYSIOLOGY

Calliet has proposed a construct for understanding low back pain analyzing static and kinetic function.[10] Pathophysiologic components are structural abnormalities of the static

spine including postural changes such as kyphosis or scoliosis.[10] Functional abnormalities include accentuation of normal lumbar lordosis because of an increased lumbosacral angle, secondary to muscular weakness, responsible for 75% of low back pain.[10,11]

Further information concerning the mechanism of low back pain is obtained by examination of the kinetic spine. Analysis shows forward flexion is anatomically more difficult than extension, but crucial for most daily activity.[13] Normal range of motion is dependent on the extensibility of the longitudinal ligaments and elasticity of the articular capsule, intervertebral disc, and muscle groups.[10] This activity is quantified as a flexion range of 167° with hip flexor input of 122° for isolated spinal flexion and 45° in extension.[13] Thus, the anatomic basis for low back pain is the mobility of the lower lumbar segments, L5-S1 and L4-5 responsible for 60% to 75% and 20% to 25% of this motion, respectively.[10]

Low back discomfort results from an increase in the lumbosacral angle as support is transferred from the posterior longitudinal ligament with minimal energy expenditure to the erector spinae muscles that can fatigue.[11,13] This causes secondary compensatory lordosis of the remaining L1-4 segments that bring about facet impingement.[10,13] This theory is supported by the finding that altered spinal curvature occurs in 15% of radiographic examinations, the most common diagnostic abnormality.[14] Finally, violation of the lumbar-pelvic rhythm, where reversal of lumbar lordosis is compensated for by the quadriceps, iliotibial band, and hamstring muscles, results in pelvic fatigue.[10] The normal ratio of lumbar to hip involvement of 3:2 reverses to a less efficient rate of 2:3, resulting in increased input of the fatiguable hip flexors.[13]

Thus, low back injury can be classified into three categories: an abnormal strain on a normal back, such as chronic postural changes due to obesity; normal strain on an abnormal back, such as facet disarticulation and scoliosis; and normal strain on an unprepared, normal back where there is inequality in anticipated and required muscle activity.[10-12] Another more concrete assessment suggests the importance of flexion, torsion, and compression mechanisms of disc injury.[6]

ETIOLOGY

The causes of low back pain are numerous and include congenital, metabolic, infectious, inflammatory, neoplastic, traumatic, degenerative, toxic, vascular, visceral, and psychosocial etiologies (Table 1).[9,11,12,15,16] Differential diagnosis should delineate referred leg pain versus nerve root irritation by its dermatomal distribution; mechanical, which is exacerbated by exertion, versus nonmechanical disease due to inflammatory effects of systemic illness; and orthopedic versus visceral disease.[17]

Classification of low back pain is also based on outcome and response to therapeutic intervention.[18] Most patients (91%) have acute disability that is self-limited with a good therapeutic response.[18] A second group of patients (7%) have potential disability if the condition is not recognized, and aggressive therapy may be warranted.[18] Finally, true spinal cord emergencies with life- or limb-threatening consequences requiring an urgent medical or surgical therapy occurs in only 2% of patients.[18] The diagnostic challenge is to identify and treat the latter two groups that account for only 9% of all patients presenting.

TABLE 1. Etiology of Low Back Pain

1 *Congenital*	6 *Trauma*
Kyphosis	Lumbar strain
Scoliosis	Fracture
Facet asymmetry	Facet subluxation
Transitional vertebrae lumbosacral unit	7 *Degenerative*
2 *Metabolic*	Disc herniation
Osteoporosis	Osteoarthritis
Osteopenia	Spinal stenosis
Hyperthyroidism	Spondylosis
Hyperparathyroidism	Spondylolithiasis
Cushing's syndrome	8 *Toxic*
3 *Infection*	Heavy metal
Osteomyelitis	9 *Vascular*
Discitis	Aortic aneurysm
Epidural abscess	Diabetes neuropathy
Paraspinous abscess	Aortic occlusion
Sacroilitis	10 *Visceral*
4 *Inflammatory*	Prostatitis
Ankylosing spondylitis	Pelvic inflammatory disease
Chronic inflammatory bowel disease	Ovarian cyst
	Endometriosis
Reiter's syndrome	Pregnancy
Psoriatic arthritis	Pyelonephritis
Rheumatoid arthritis	Nephrolithiasis
5 *Neoplasia*	Peripheral abscess
Multiple myeloma	Cholecystitis
Metastatic disease	Pancreatitis
Lymphoma	Peptic ulcer disease
Leukemia	11 *Psychosocial*
Primary osseous tumor	Hysteria
Spinal cord tumor	Malingering
	Litigation
	Somatization

LOW BACK PAIN SYNDROME

Disc herniation, the prototype low back pain syndrome, is the target of most diagnostic and therapeutic efforts. The incidence of disc herniation is in a range of 2% to 7.5% of patients presenting with lumbar complaints.[2,19] This disease entity was first described accurately by Walter Dandy and formerly thought to represent a spinal cord tumor, or osteochrondroma.[15] Disc herniation occurs when this syndesmosis, through a series of intrinsic or extrinsic mechanisms, undergoes extrusion of the nuclear material through the annular structure.[11] The posterior annulus is most susceptible to compromise and is also the area with the highest concentration of pain-sensitive structures.[6,11] The outcome of neural dysfunction depends on the degree of compression, with symptoms including sensory abnormality (pain, paresthesia, and motor involvement with increasing conduction blockade); continuance of compression; and the myelination of specific fibers with central parasympathetic nonmyelinated fibers affected initially, resulting in bowel or bladder incontinence.[11] This complication is known as the cauda

equina syndrome, in which a large central disc herniation results in incontinence and saddle paresthesia along with the loss of the anal reflex and sphincter tone.[20] Location of herniation can also be inferred from a prospective study of 113 patients with 100% sensitivity, where central lesions result in back pain and lateral lesions cause referred leg pain.[21]

Symptoms and signs include restricted flexion and a decrease in the intervertebral space respectively. Functional scoliosis, away from the lesion can also occur secondary to muscle spasm. Diagnosis is suggested by a positive straight-leg raise test, where pain occurs on 45° to 70° extension in the nerve root, dura mater, and enveloping sheath.[18,21] The pain induced is proportional to nerve root involvement, which occurs to a greater extent in the L4, L5 segment than in the L1, L2, and L3 segments.[10,21] Objective indices of herniation also include muscle weakness (12%), sensory abnormalities (10%), and abnormal deep tendon reflexes, specifically including the ankle (9.6%) and knee (7.5%).[14] These signs and symptoms are classified into specific nerve root syndromes. The L4 radiculopathy is associated with pain in the anteromedial thigh, quadriceps weakness, and an abnormal knee jerk reflex.[22] The L5 radiculopathy causes pain on the foot dorsum and great toe, weakness of foot extensor and ankle eversion, and an absent ankle jerk.[22] The S1 radiculopathy results in buttock, posterior calf, lateral foot, and great toe paresthesia; weakness in the gastrocnemius, toe flexors, gluteus maximus; and an abnormal ankle jerk.[22] These myotomal and dermatomal deficits are a theoretic construct and involvement may be irregular. Spondylolysis is a unilateral pars articularis defect without vertebral body displacement.[9] Pain occurs when the annulus fibers and longitudinal ligaments weaken, resulting in increased anteroposterior movement.[16] The disc may then progress to herniation with narrowing of the intervertebral space and a foreign body inflammatory response to form osteophytes.

Spondylolithesis is a bilateral pars articularis defect with anterior vertebral movement.[16] This defect occurs predominantly at the L5 level (70%) followed by L4 (25%) and L3 (4%).[10] A classification system based on displacement related to vertebral body width has been established.[9,10] Type I or isthmic (0% to 25%) is associated with a pars defect; Type II is congenital (25% to 50%) due to inadequate posterior elements; Type III is degenerative (50% to 75%) and secondary to posterior longitudinal ligament degeneration; Type IV or pedicle abnormality (75%) is due to an elongated neural arch; and Type V or destructive results from metastatic or infectious disease.[9,10] This radiographic finding, however, has a low specificity for disease with 50% occurring in normal patients.[23] Spinal stenosis, a congenital or acquired narrowing of the spinal canal, is found in 53% of patients undergoing discectomy.[7] This condition can involve the central canal, lateral recess, or intervertebral foramen.[24] The symptom complex is known as "pseudoclaudication" where vertebral arch narrowing causes effort-dependent ischemia of the neuronal vascular supply or vasovasorum simulating the symptoms of claudication or aortoiliac occlusions.[9,10]

Miscellaneous entities associated with lumbar pain include the pyriform syndrome or sciatica due to myofascial pain of the hip external rotators, and the sacralized transverse process, a congenital condition of an elongated L5 transverse process resulting in a pseudoarthrosis with the ipsilateral ileum.[9,10] The spondyloarthropathies such as rheumatoid arthritis, inflammatory bowel disease, and Reiter's syndrome are also implicated in low back pain.[18]

Sacroiliac pathology is manifested as a posttraumatic condition with restricted hip adduction and indicated by the Garnslen test with pain on hip hyperextension.[10] Ankylosing spondylitis is an inflammatory condition of the sacroiliac and lumbar region in young men aged 20 to 30 years.[25] Patients present with low back pain associated with decreased lumbar flexion and chest excursion.[25] Laboratory analysis finds a negative erythrocyte sedimentation rate and rheumatoid factor, and a significant HLA-B27 antigen titer with a sclerotic radiographic appearance of the sacroiliac joint.[9,25]

Infectious syndromes include arachnoiditis with inflammation of the neurovascular cord enveloping structures.[9,10] Discitis, manifested as disc space narrowing and endplate sclerosis due to staphylococcus aureus, can develop 2 to 8 weeks postoperatively.[15] This occurs in patients predisposed by intravenous drug abuse or a urinary tract infection and is indicated by an increased erythrocyte sedimentation rate.[15] Osteomyelitis affects the vertebrae in patients with pelvic inflammatory disease, urinary tract infection, or subacute bacterial endocarditis who undergo hematogenous or contiguous dissemination of disease.[10,26] The incidence is 1:100,000 in acute involvement in diabetics with *staph aureus,* intravenous drug abuse by *E. coli,* sickle cell patients by salmonella, and tuberculosis in chronic involvement.[15,27,28] Symptoms include chronic back pain lasting 8 to 12 weeks, percussion tenderness, and delayed radiographic findings of vertebral rarefication.[9,10] Malignancy can also present as back pain in rare cases with an incidence of 6:1,000.[26-28] Symptoms include failure to respond to therapy, nocturnal pain, and no alleviation of discomfort with position change or rest.[18] In fact, pain is the first symptom in metastatic epidural lesions followed by motor, sensory, or autonomic dysfunction worsened by movement or an increase in intrathoracic pressure.[26] Vertebral involvement is most commonly associated with metastasis from breast, lung, renal, ovarian, thyroid, uterine, prostate, and cardiac malignancies.[15] Lesions are predominantly osteoclastic, except for the latter two conditions associated with osteoblastic lesions.[15] Multiple myeloma is the most frequent primary malignant lesion, while osteoid osteoma is a benign lesion found in the young with nocturnal pain responding to aspirin.[15] Neural canal tumors are also implicated including meningioma, ependymoma, and neuroblastoma.[18]

PREDISPOSITION

Epidemiologic analysis of low back pain shows an incidence of 0.2% to 2.0% with a 2:1 male predominance for disc herniation.[27-29] Mechanical back pain, occurring in 99.8% of patients, is a disease of middle age with 35.9% in the 45-to-64-year age range.[2] Nonmechanical back pain, occurring in 0.2% of patients, has a bimodal peak at age extremes with 10% in the 0 to 20 age group, and 20% in those over 55 years of age presenting with vertebral involvement of systemic disease.[2] There is no occupational predisposition, in contrast to previous reports implicating heavy physical labor, in fact, white collar professionals account for 27.6% of chronic back pain patients.[30] Certain factors have been related including headache, living alone, previous hospitalization, multiple

physical complaints, and long distance travel.[31] Smoking also has a strong association because of decreased nutrient supply to the disc or increased intradiscal pressure with chronic cough.[31] It is conceivable that the unifying factor is depression, predisposing patients to both tobacco abuse and chronic pain syndromes. Pertinent historical factors include presence of "saddle anesthesia," urinary retention or incontinence, use of corticosteroids or anticoagulants, previous radiographic evaluation, poor general health, medications, rheumatoid arthritis or ankylosing spondylitis, and prior abdominal surgery.[32] Past medical history of patients with low back pain includes obesity (70%), psychiatric manifestations (33%), and hypertension (19%)[4]; however, the first episode of back pain has no associated cause in 79% of men and 89% of women.[33] The most significant historical point is the position of comfort or worsening of symptoms. Classically, disc herniation is associated with exacerbation when sitting (30%) or bending (65%) and relief while supine (53%) or standing (36%).[6,34] This finding is related to a progressive increase in intradiscal pressure estimated to be 30 kg supine, 70 kg standing, 100 kg sitting, and 120 kg bending forward.[10] The chief complaint is generally pain in men and weakness in women, with 36% to 51% presenting with radiating leg pain or sciatica.[34]

PHYSICAL EXAMINATION

A systematic physical examination is essential for accurate diagnosis of the etiology of low back pain. Deyo et al have suggested an examination based on patient positioning.[35] Initially the patient should be observed in the standing position noting pelvic symmetry with a 90° angle between the lumbar spine and pelvis.[32] Deviations from this posture in the sagittal plane include lordosis and kyphosis, with the latter correlated with organic back pain.[34,35] Deviation in the coronal plan occurs with scoliosis, which can be due to asymmetric paraspinous spasm in disc herniation.[36] Leg length discrepancy measured from the anterior superior iliac spine to medial malleolus of 2.5 cm is significant.[17]

Spinal mobility is best quantified by examination of lumbar flexion.[5,13] The Schober test measures the degree of distraction between two arbitrary points on the lumber spine, with a normal difference of 5 cm, and 3 cm or less being pathologic.[9] The Burns hysterical bench test requires the patient to kneel on a support 45 cm above the floor, and suggests that even with mechanical back pain may reach to within 15 cm of the surface.[37] This test has a 73% sensitivity and 71% specificity when associating failure of performance with probability of hysterical personality disorder as defined by the Minnesota Multiphasic Personality Inventory (MMPI).[37] Lumbar extension should be evaluated by a maneuver to arch the back. Extension results in pain in patients with degenerative disease or spinal stenosis, as opposed to those with disc herniation with flexion pain.[5] Lateral mobility is assessed where a normal range of motion allows the patient to touch the proximal fibular head with the hand by bending sideways at the hip.[5,35] Coordination of motion is assessed by lumbar pelvic dysfunction where the protective reflex spasm of paravertebral musculature in disc disease shifts the burden of movement to the hip flexors.[5,10] The ability to ambulate should be examined with a normal gait expected, even if disc herniation has occurred.[10] Abnormal gait is associated with mechanical causes other than disc herniation responsible for low back pain. Clarification is provided by reverse ambulation where a reproducible limp is a legitimate finding.[9,10] Accessory maneuvers such as the heel walk, where poor dorsiflexion correlates with L5 disc herniation, and the toe walk, where poor plantar flexion is implicated in S1 herniation, are compromised in 17% of patients.[6,38] The seated patient allows examination of the deep tendon reflexes involved in 8% of patients with disc herniation.[9,38] The knee reflex is controlled by the L4 nerve root; because herniation is rare at or above this level, an abnormal finding is unusual.[18,19] The ankle reflex of the S1 root is more common with a 90% specificity for the L5/S1 disc.[18,35] Muscle strength is assessed by the ability to draw the toes upward involving the extensor hallucus longus muscle innervated by L5. Finally, the "distraction straight leg raise" with extension of the knee to 90° is possible without difficulty in patients with functional pain, however, those with structural disease will exhibit the "tripod sign" arching backward with arm support to relieve the irritation.[20]

The supine patient should undergo visual inspection for signs of muscle atrophy such as a decrease in calf circumference.[6] Palpation may show a pulsatile abdominal mass consistent with aneurysm or facilitate identification of dermatomal sensory defects. Manual testing includes the "Bowstring" test where a flexed knee and partially extended lower leg that undergo popliteal compression will reproduce sciatic pain with radiation if root irritation is present.[19] The "Patrick" test elicits pain of sacroiliac origin by external rotation of the hips.[16] This is designated the FABRE (flexion, abduction, external rotation) maneuver indicative of sacroiliac pathology.[20] The testing standard for disc herniation is the straight-leg raise or Lasegue's sign indicated by sciatic pain radiation with extension found in 36% of cases.[10,38] The distribution of pain allows accurate localization of the herniated structure in 88.5% of patients.[21] This test offers a sensitivity of 98.2% for cases resulting in surgery, and specificity in 89% with disc herniation found intraoperatively.[21] The degree of herniation is also suggested by a "crossed straight-leg raise sign" sensed in the opposite extremity implying a large central herniation.[10,18] Confirmation of an equivocal straight-leg raise test is offered by the "nuchal flexion" and "ankle dorsiflexion" maneuvers, positive in 70.8% of patients with disc herniation.[19,21]

Finally, lateral positioning of the patient allows evaluation of gluteus medius adduction dependent on L5, the trochanteric bursa, and a rectal examination.[9] The issue of validity of physical examination has been addressed by Waddell who suggested that if 3 of 5 "symptom amplifiers" were present there was a high likelihood of malingering or hysteria.[39] These correlates included the "spinal loading" or "acetabular rotation test," which are painless maneuvers even with disc herniation; nonorganic tenderness where light palpation of paraspinous region is painful; a negative distraction straight-leg raise test; nondermatomal sensory findings; and overreaction to history and physical examination.[39]

DIAGNOSIS

The diagnostic evaluation of low back pain shows that 57% of patients have idiopathic disease, followed by muscle sprain or strain in 32.7%, degenerative joint disease in 19%, miscellaneous causes in 13%, and disc disease in only 11%.[2,38] Diagnosis is suggested by a careful history and

physical examination in 63% of patients; the most commonly used modality is radiographic evaluation in 17.6% of patients.[2]

There has been a recent trend toward limiting radiographic evaluation of the lumbar spine in light of minimal clinical information that is gained. The most commonly identified condition is degenerative joint disease in 70% of patients, while another prospective evaluation found 46% were normal and 33% demonstrated abnormalities of questionable significance.[38,40] Deyo has suggested a series of 11 high yield criteria for lumbar radiography that results in a 3-fold increase in clinically-significant findings.[27] These criteria include age over 50, significant trauma, neuromotor deficit, weight loss of 10 pounds, ankylosing spondylitis, drug or alcohol abuse, malignancy, corticosteroids, fever of 100°, revisit without improvement or financial compensation.[27]

If radiographic evaluation is warranted, then selective interpretation criteria should be stressed. Radiographic correlates of low back pain include vertebral wedging, disc space narrowing, and the presence of osteophytes.[22,41] However, these findings correlated to only a small degree in patients with low back pain compared with normals.[22,42] Radiographic evaluation should include oblique views suggested by a study that showed a 12% incidence of undiagnosed lesions, if a standard three-view series was used.[42] The issue of repeat radiograph on revisit is addressed by a study that found 64% of patients were unchanged and 36% demonstrated resolving lesions.[40] Thus, interval radiographic studies are clearly not warranted without reinjury.

Adverse effects of excess radiation from unnecessary radiographic evaluation have been investigated by Liang.[28] He estimates the incidence of occult pathology at 0.2%, arguing the radiation risk of .03 Gy as the primary source of gonadal irradiation associated with 400:1,000,000 fetal malformations, and the cost calculated at $2,072 for diagnosis does not justify their use.[28] Most recently, Deyo's original high-yield criteria were examined in a retrospective study that suggested an actual increase in use from 21.1% to 46.1% compared with routine practice.[19] Suggested revised criteria include old age, duration of 60 days, asymmetric reflexes, and vertebral point tenderness.[19]

Other diagnostic modalities including discography, where radiopaque dye is injected into the disc structure, is cited for historical interest.[10] Traditionally, the gold standard for diagnosis has been myelography with a 92% sensitivity and 87% specificity.[36] Advantages include definition of surgical anatomy; however, the cost, about $2,000 for admission and the procedure, must be a consideration.[16,30]

The current diagnostic standard is high resolution computerized tomography (CT). This noninvasive modality associated with a minimal .00005 Gy radiation exposure offers diagnostic accuracy with a sensitivity of 95% and specificity of 66% to 88%, and is readily available from the emergency department.[43,44] CT allows versatility by delineating disc herniation, spinal stenosis, facet disease, spondylolysis, arachnoiditis, and sacroilitis subsequently decreasing the onset of "failed back syndrome" occurring in 25% to 30% of patients postoperatively.[23,44] Also, the financial advantage of slightly lessened procedural costs without requiring hospital admission suggests the superiority of CT scan over myelography.[16,44] Finally, comparison studies have found CT scan as effective as myelography in multiple clinical trials.[23,43-45] Radionuclide scanning is useful in the diagnosis of inflammatory conditions, such as malignancy or infection, with a 0.4% false negative rate compared with a 9.1% rate for standard radiography.[4,46] The most recent radiographic modality, magnetic resonance imaging (MRI) is superior to CT for anatomic detail, but cost considerations and availability limit its use for acute low back pain.[43] However, MRI has been used in the emergent setting with sensitivity of 88.8% for both intramedullary and extramedullary spinal lesions.[47]

Corroborative, although not conclusive, diagnostic information is provided by laboratory analysis. Leukocytosis, or a white blood cell count of 12,000/mm, is present in 4% to 65% of patients with neoplastic or infectious lumbar disease.[36] However, only 16% of patients with low back pain have leukocytosis.[4] The erythrocyte sedimentation rate is elevated to 50 mm, in 18% to 87% of patients with occult inflammatory lesions, approximating the sensitivity of myelography.[4] Urinalysis utilized in 26% of patients may demonstrate abnormalities indicative of a urinary tract infection.[4] Examination for histocompatibility antigens, specifically HLA-B27 associated with ankylosing spondylitis, similarly cannot be advocated as an efficient screening test, because of an 8% false positive rate and an incidence of 5% to 13% equal to that present in a normal population.[4,25]

THERAPEUTIC INTERVENTIONS

Most patients with low back pain are evaluated and treated by primary care physicians (61%) as opposed to specialists (28%).[40] Thus, it is apparent that most practitioners should have a clear therapeutic plan formulated. Goals of therapy include the alleviation of pain, restoration of mobility, minimization of residual impairment, prevention of recurrence, and intervention in the chronic pain syndrome.[10] Patients' understanding is the key to an effective therapeutic approach. A retrospective analysis of low back pain patients found the most common complaint from disgruntled patients was inadequate physician explanation.[48] Patient expectations can be affected by both diagnosis and testing strategy. Those given a concrete diagnosis such as degenerative joint disease were satisfied to a greater extent (93%) than those with a nonspecific diagnosis such as muscle strain (63%).[38] It has been suggested previously that radiographic evaluation is essential to patient satisfaction.[49] A study comparing immediate radiograph versus delayed filming with a modified educational program found equivalent results in patients with minor back pain.[49]

Therapy can be delineated into treatment directed at the acute condition from 0 to 3 months, and chronic low back pain present for 3 months or greater.[10] Most patients are in the former group, 52% to 90% with onset less than 1 week; 16.6% from 1 to 3 weeks; and 12.7% where pain has been present for 1 to 3 months.[2,33]

Conservative therapy of acute low back pain present in 78% of patients begins with bed rest.[33] Bed rest is utilized in 33% of low back pain cases and has proven superior to pharmacologic therapy, demonstrating a return to activity in 48% of patients.[4] Traditionally, the length of therapy is between 7 and 14 days for radicular symptoms and 5 weeks for disc herniation.[10] Shorter duration of bed rest appears to be beneficial as suggested by an evaluation of 2 versus 7 days of

immobilization, demonstrated by 45% fewer work days missed and improved compliance.[50]

Local therapy is utilized in 43% of cases, specifically cold application in the first 24 hours and heat application in the second 24 hours.[4] Cooling therapy provides sensory input competing with slower pain fibers, resulting in a prolonged refractory period to pain stimulus.[10] Warming treatment can prevent secondary cold-induced muscle spasm. Systemic therapy such as whirlpool or diathermy provides more effective relief than topical application that does not penetrate below the skin surface.[5]

Pharmacologic therapy is the mainstay of low back pain treatment, utilized in 50% to 62% of cases.[2,29] Traditional therapy includes aspirin, which has proved superior to Tylenol (McNeil PPC, Ft Washington, PA) or Butazoladin (phenylbutazone; Gergy, Summit, NJ) at 10% to 20% of the cost of nonsteroidal antiinflammatory agents (NSAIA).[51] Disadvantages include the gastric irritant side effects.

Narcotic analgesics have been used routinely in the treatment of moderate to severe low back pain. Advantages include a variety of administration routes, an anxiolytic or soporific effect and well defined efficacy in structural back dysfunction. The potential for misuse and abuse of narcotics is well described and should be limited to the therapy of acute pain only. Interestingly, a comparison of Tylenol III and codeine compared with difusinal (Dolobid, Merck, Sharp, and Dohme, West Point, PA) in equipotent doses found similar efficacy but better tolerance of difusinal side effects.[3,52]

NSAIA are utilized for minor or moderate pain, demonstrating minimal potential for abuse. Specific agents such as piroxicam (Feldene, Pfizer, New York, NY) has proven efficacious by oral and intramuscular routes compared with indomethacin (Indocin, Lederle, Wayne, NJ) in clinical trials.[53,54] Also, difusinal has proven superior to naproxen (Naprosyn, Syntex, Humacao, PA) in clinical trials demonstrating an 81% and a 41% improvement in symptoms, respectively.[55] A new, injectable nonsteroidal agent ketorolac (Toradal, Syntex) is amenable to emergency department use for low back pain.

Skeletal muscle relaxants are suggested for use when paraspinous spasm accompanies lumbar pain and are utilized in 56% of cases.[4] Disadvantages include behavioral alterations including sedation, paradoxical agitation and some abuse potential in light of these side effects.[22,56] A multidrug comparison trial found that all agents were more efficacious than placebo, except for diazepam (Valium, Roche, Nutley, NJ) that ironically was found least effective in acute musculoskeletal strain.[56] They also concluded that combination agents such as carisoprodol (Soma, Wallace, Cranbury, NJ) and chlorzoxazone (Parafon Forte, McNeil) were superior to single mechanism medication.[56] This may establish support for dual therapy with nonsteroidal agents and muscle relaxants for ambulatory low back pain. Cyclobenzaprine (Flexeril, Merck, Sharpe, and Dohme) has also been proven to be a more effective oral agent compared with diazepam by electromyelography.[51] Orphenadrine citrate (Norflex, Riker, St Paul, MN) has also proven efficacious administered by intramuscular and by oral routes.[58,59]

Corticosteroids have been traditionally utilized for intradural compression syndrome due to malignancy, diagnosed by myelography. The suggested regimen includes dexamethasone in high dose (100 mg) for 80% to 100% compression and low dose (60 mg) for 80% compression.[26] Injectable corticosteroids have been utilized for iliolumbar ligament infiltration with subjective improvement, but no objective change in flexion range.[60] However, there is little indication for steroid use in cases of radicular pain caused by nerve root and dorsal sheath inflammation based on a clinical trial that demonstrated minimal to no improvement.[61]

Adjunct therapeutic modalities may be indicated by individual circumstances. The lumbar brace, utilized in 1% of patients, provides abdominal support, moving the sacrum posteriorly and limiting thoracoabdominal motion.[4,5] The lumbar brace has no proven efficacy, except for spinal stenosis or spondylolithesis, but the abdominal binder may be effective for postural lumbar pain caused by obesity.[5,62]

Exercise programs are suggested and utilized in 7% of patients.[4] A study comparing the effects of physical therapy and exercise with bed rest found no advantage in 27% of patients and suggested early mobilization rather than a recuperation period.[63] However, a multicenter trial involving 12,000 problematic patients with chronic lumbar pain demonstrated improvement in 80.7% of those enrolled in a 6-week exercise program.[30] This study was without a control group, and improvement may have occurred with time; however, this is unlikely considering a mean pain duration of 8.1 years.

Manipulation or rotational manual alignment coupled with acute and passive range of motion exercise is suggested to facilitate facet integrity and to reduce spasm.[10] However, a large single blinded clinical trial found no apparent efficacy compared with controls.[64] Similarly, traction where a pelvic or shoulder harness is fitted with 25% to 35% of body weight has not proven to be effective.[5]

Local injection of involved structures with narcotics, anesthetics, or corticosteroids has been utilized in both the acute and chronic condition. The concept of the myofascial "trigger point" has been implicated in 30% of patients with pain syndromes, although only 10% involve the lumbar region.[65] This regional pain syndrome involves a cutaneous trigger point for a band of skeletal muscle localized as discrete tenderness with radiation.[65] The multifidus triangle lateral to L5 has the highest concentration of lumbar pain producing structures amenable to superficial injection.[10] Intraarticular facet injection of steroids and a local anesthetic demonstrated improvement in 63.1% of patients with lumbar facet syndrome for a 6-month period.[66] The extradural injection provides a nerve root block of the posterior primary division with proven efficacy in a double blinded study of 36 patients.[61] The epidural block anesthetizes the recurrent sinuvertebral nerve at the intertransversus ligament.[67] This method utilizing morphine and methylprednisolone provided pain relief in a study of 20 patients, except for those undergoing litigation, isolated as a negative correlate.[67] Finally, the intradural injection affecting the dura and nerve roots, analogous to a lumbar puncture, has been utilized.[67] Most injection techniques are best reserved for the specialist, although facet and myocutaneous injection have been successful in the emergency department setting.

The most significant dilemma is to identify and refer lumbar conditions associated with morbidity and neurovascular

compromise. Disc herniation without sensory motor deficit can be managed as an outpatient with urgent referral over 24 to 48 hours. Admission and emergent referral is warranted for the cauda equina syndrome, disc herniation with sensory-motor compromise. Progressive neurologic deficit, and multiple root involvement.[19] These conditions usually require operative intervention and become irreversible if untreated for 24 hours.[11]

Surgical intervention for disc herniation has long proven efficacy.[6] A study of laminectomy for low back pain of less than 1 year's duration demonstrated excellent recovery in 53% and good recovery in 23%, leaving 24% of cases refractory to surgical therapy.[68] Standard unilateral or bilateral discectomy is performed 150,000 times annually with a 95% success rate.[12] Alternative therapy includes microdiscectomy or removal of the extruded disc only.[6] Chemonucleosus or injection of chymopapain, a proteolytic enzyme has a 70% to 90% success rate.[6,12]

Finally, chronic back pain occurring for a duration of 3 months or greater requires a distinct therapeutic approach. Chronic back pain is defined by an incidence of 15.5% with a 44.4% to 60% rate of recurrence and refractory to 2- to 4-months of multidisciplinary therapy.[2,5,69]

Pain fibers implicated include the myelinated afferent α (12 to 21 microns), β (8 to 12 microns), and γ (5 to 6 microns), myelinated preganglionic and unmyelinated somatic fibers.[10,60] These fibers transmit via localizing spinothalmocortical and nonlocalizing spinoreticulothalmic pathways.[10,69] Transmission of pain sensation involves the "specificity theory" or unique nerve terminals for unpleasant stimuli or the "pattern theory" where a processing malfunction creates unpleasant sensations from a normal stimulus.[10,69] The Wahl and Melzack gate control theory states that the fast α fibers stimulate the substantia gelatinosa inhibitory center to decrease the pain stimulus causing the slow γ fibers to encounter a refractory period for pain generation.[10,70] Thus, the therapeutic approach attempts to facilitate α and decrease γ transmission.

Therapy of chronic low back pain should prevent drug dependency utilizing low-dose narcotic or nonnarcotic pain relief for a limited duration, minimal as-required (prn) medications, and positive feedback for desired behavior. Behavior modification includes stress management techniques and lifestyle changes; biofeedback demonstrates a 10% to 20% response rate.[5] Depression has been implicated by the "biochemical hypothesis" suggesting that low serotonin stores are responsible for both depression and lumbar pain.[71] A double blinded study of desipramine and doxepin showed pain relief in 60% of patients.[71] Anxiety is also implicated as 40% of back pain patients state concerns over their disability, financial difficulties, or fear of malignancy.[72] The efficacy of muscle relaxants may be due to a central anxiolytic effect, as well as musculoskeletal activity. Lastly, the transcutaneous electrical nerve stimulation (TENS) unit modifies the amplitude frequency and pulse width of the fast α stimulus to decrease pain perception according to the Wahl and Melzack theory in chronic pain suffers.[5,10,69]

CONCLUSION

The presentation of low back pain requires diagnostic acumen based predominantly on a comprehensive history and physical examination. Appropriate diagnostic modalities include plain lumbar radiography followed by high-resolution computerized tomography where warranted. Specific disease consideration may necessitate myelography or nuclear scanning. Hematologic analysis for leukocytosis, an elevated erythrocyte sedimentation rate, or HLA-B27, are probably not warranted during a screening examination.

Therapy for low back pain syndrome should delineate the acute and chronic condition. Acute modalities include narcotic or nonnarcotic pain relief, antiinflammatory agents and muscle relaxants where appropriate, along with rest and early mobilization. Chronic back pain requires alternative techniques including exercise, stress and lifestyle modification usually in the multidisciplinary environment. Thus, the morbidity of this commonly encountered disease entity can be minimized by a well organized effective diagnostic and therapeutic strategy.

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