# *Overview of Back Pain and its Treatment*

Rasha Snan Jabri, M.D.
Mathew Hepler, M.D.
Honorio T. Benzon, M.D.

**Epidemiology**

Low back pain (LBP) is one of the most common complaints in our society today.At least 60-90% of U.S. adults will have LBP at some time during their lifetime and up to 50% have back pain within a given year[1,2,3,4,5,6,7] Acute low back pain is the fifth most common reason for all physician visits[8,9] Although symptoms are usually acute and self-limited, low back pain often recurs. Of those who develop acute LBP, 30% develop chronic LBP[10]

Low back pain has great financial and socioeconomic impact in industrial countries as a growing social economic problem. The cost for direct health care is more than $20 billion annually and as much as $50 billion per year when indirect costs are included.[11, 12] Low back pain is one of the most commonly cited problems for lost work time in industry. Back pain is the most frequently filed Workers' Compensation claim and is the most common reason for early Social Security disability in the U.S.A. for persons under age 45[13]. In 1990, direct medical costs for low back pain exceeded $24 billion. Total annual costs for back pain increase from $35 to $56 billion when disability costs are included[14,15]

**RISK FACTORS**:

Epidemiological studies have reported three general classifications of risk factors to be associated with LBP: biomechanical, psychosocial and personal. The biomechanical factors include weight lifting, lift rate, box position, reach distances, and task asymmetry. The amount of weight lifted, reach distances, task asymmetry and lift rate have all been found to significantly increase the three-dimensional spinal loads.[16,17] The psychosocial risk factors consist of mental concentration or demands, job responsibility, lack of variety, job satisfaction and mental stress.[18, 19,20,21,22] Studies have investigated the impact of psychosocial factors on spine loading[23]. Personal factors have also been identified as potential risk factors for LBP, such as physical strength, genetics, anthropometry, gender and personality[18,24,25,26] Furthermore, both psychosocial and biomechanical may contribute to spine loading as well as influence the loading response to the work factors[27,28].

Epidemiological studies have shown the following factors to be associated with the development of back pain:

- Jobs requiring heavy lifting[29,30,31]
- Use of jackhammers and machine tools,
- Operation of motor vehicles
- Cigarette smoking,[29, 30]
- Anxiety
- Depression
- Stressful occupations.
- Women with multiple pregnancies

- Scoliosis[31]
- Obesity[32,33]
- Genetics
- Personality[34,35]

**Definitions:**

Low back pain is defined as pain in the lumbosacral region localized between the costal margin and the inferior gluteal folds with or without sciatica. The Quebec Task Force on Spinal Disorders categorized patients based on the duration of symptoms[36].

Acute back pain: duration less than 2–4 weeks

Subacute back pain: up to 12 weeks,

Chronic: more than 12 weeks

Chronic pain can be classified as persistent or as multiple acute recurrences although few studies employ this distinction.

**Terminology in LBP:**

The North American Spine Society (NASS) recommended detailed definitions of lumbar disc pathology to standardize terminology among experts in the field.[37]

- Annular tears: loss of integrity of the annulus such as radial, transverse, and concentric separations.
- Bulging disk: a disc in which the contour of the outer annulus extends, or appears to extend, in the horizontal (axial) plane beyond the edges of the disc space, usually greater than 50% (180 degrees) of the circumference of the disc and usually less than 3 mm beyond the edges of the vertebral body apophysis.

Another (non-standard) definition of bulging disc is a disc in which the outer margin extends over a broad base beyond the edges of the disc space.

- Concentric tear**:** tear or fissure of the anulus characterized by separation, or break, of anular fibers, in a plane roughly parallel to the curve of the periphery of the disc, creating fluid-filled spaces between adjacent anular lamellae. See: radial tears, transverse tears.
- Contained herniation: displaced disc tissue that is wholly within an outer perimeter of uninterrupted outer annulus or capsule. Non-standard definition: a disc with its contents mostly, but not wholly, within annulus or capsule.
- Degenerated disk: changes in a disc characterized by dessication, fibrosis and cleft formation in the nucleus, fissuring and mucinous degeneration of the annulus, defects and sclerosis of the endplates, and/or osteophytes at the vertebral apophysis.
- Dessicated disc: disc with reduced water content, usually primarily of nuclear tissues.
- Displaced disc: a disc in which disc material is beyond the outer edges of the vertebral body ring apophysis (exclusive of osteophytes) of the craniad and caudad vertebrae, or as in case of intravertebral herniation, penetrated through the vertebral body endplate. The term includes, but is not limited to, disc herniation and disc migration.

- Extruded disc: a herniated disc in which, in at least one plane, any one distance between the edges of the disc material beyond the disc space is greater than the distance between the edges of the base in the same plane; or when no continuity exists between the disc material beyond the disc space and that within the disc space.
- Fissure of annulus: separations between anular fibers, avulsion of fibers from their vertebral body insertions, or breaks through fibers that extend radially, transversely, or concentrically, involving one or more layers of the anular lamellae. The terms fissure and tear are commonly used synonymously. Tear or fissure are both used to represent separations of anular fibers from causes other than sudden violent injury to a previously normal anulus, which can be appropriately termed "rupture of the anulus," which, in turn, contrasts to the colloquial, nonstandard, use of the term "ruptured disc," referring to herniation.
- Focal protrusion: protrusion of disc material so that the base of the displaced material is less than 25% (90 degrees) of the circumference of the disc. Focal protrusion refers only to herniated discs that are not extruded and do not have a base greater than 25% of the disc circumference. Protruded discs with a base greater than 25% are "broad-based protrusions."
- Free fragment: a fragment of disc that has separated from the disc of origin and has no continuous bridge of disc tissue with disc tissue within the disc of origin. Syn: sequestrated disc. Non-Standard definition: a fragment that is not contained within the outer perimeter of the annulus. Another non-standard definition: a

fragment that is not contained within anulus, posterior longitudinal ligament, or peridural membrane. Sequestrated disc and free fragment are virtually synonymous.

- Herniated disc: localized displacement of disc material beyond the normal margins of the intervertebral disc space. Non-standard definitions: a) any displacement of disc tissue beyond the disc space; b) any displacement of disc tissue beyond the disc space. Note: Localized disc herniation means less than 50% (180 degrees) of the circumference of the disc. Disc material may include nucleus, cartilage, fragmented apophyseal bone, or fragmented anular tissue. Herniated disc generally refers to displacement of disc tissues through a disruption in the anulus, the exception being intravertebral herniations (Schmorl's nodes) in which the displacement is through vertebral endplate.
- High intensity zone (HIZ): area of high signal intensity on T2-weighted magnetic resonance images of the disc, usually referring to the outer anulus. Note: High intensity zones within the posterior anular substance may reflect fissure or tear of the anulus, but do not imply knowledge of etiology, concordance with symptoms, or need for treatment.
- Internal disc disruption: disorganization of structures within the disc space.
- Intra-anular displacement: displacement of central, predominantly nuclear, tissue to a more peripheral site within the disc space, usually into a fissure in the anulus. Non-standard definition: intra-anular herniation, intradiscal herniation. Intra-anular displacement is distinguished from disc herniation, in that herniation of

disc refers to displacement of disc tissues beyond the disc space. Intra-anular displacement is a form of internal disruption.

- Intravertebral herniation: a disc in which a portion of the disc is displaced through the endplate into the centrum of the vertebral body. Syn: Schmorl's node.
- Normal disc: a fully and normally developed disc with no changes attributable to trauma, disease, degeneration, or aging. The bilocular appearance of the adult nucleus is considered a sign of normal maturation. Non-Standard definition: a disc that may contain one or more morphologic variants which would be considered normal given the clinical circumstances of the patient.
- Protruded disc: a herniated disc in which the greatest plane, in any direction, between the edges of the disc material beyond the disc space is less than the distance between the edges of the base, when measured in the same plane. Non-standard definition: a disc in which disc tissue beyond the disc space is contained within intact annulus. Non-Standard: any, or unspecified type of, disc herniation. The test of protrusion is that there must be a localized (less than 50% or 180° of the circumference of the disc) displacement of disc tissue so that the distance between the corresponding edges of the displaced portion must not be greater than the distance between the edges of the base. A disc that has broken through the outer anulus at the apex, but maintains a broad continuity at the base, is protruded and uncontained.
- Radial fissure or tear: disruption of anular fibers extending from the nucleus outward toward the periphery of the anulus, usually in the craniad-caudad

(vertical) plane, though, at times, with occasional horizontal (transverse) components. Occasionally a radial fissure extends in the transverse plane to include avulsion of the outer layers of anulus from the apophyseal ring.

- Ruptured annulus**:** disruption of the fibers of the anulus by sudden violent injury. Separation of fibers of the anulus from degeneration, repeated minor trauma, other nonviolent etiology, or when injury is simply a defining event in a degenerative process should be termed fissure or tear of the anulus. Rupture is appropriate when there is other evidence of sudden violent injury to a previously normal anulus. Ruptured anulus is not synonymous with ruptured disc, which is a colloquial equivalent of disc herniation.
- Ruptured disc: Non-standard: a herniated disc, a disc in which the anulus has lost its integrity. See herniated disc, ruptured anulus. Ruptured disc is used colloquially to encompass the same nonspecific meaning as the preferred term herniated disc.
- Sequestrated disc: an extruded disc in which a portion of the disc tissue is displaced beyond the outer anulus and maintains no connection by disc tissue with the disc of origin. An extruded disc may be subcategorized as “sequestrated” if no disc tissue bridges the displaced portion and the tissues of the disc of origin. If there is a fragment of disc tissue that is not continuous with parent nucleus, but still contained, even in part, by anular tissues the disc may be characterized as protruded or extruded, but not as sequestrated.

- Spondylitis: inflammatory disease of the spine, other than degenerative disease. Spondylitis usually refers to noninfectious inflammatory spondyloarthropathies.
- Spondylosis: spondylosis deformans, for which spondylosis is a shortened form. Non-Standard definition: any degenerative changes of the spine that include osteophytic enlargement of apophyseal bone. Spondylosis deformans has specific characteristics that distinguish it from intervertebral osteochondrosis. Both processes include vertebral body osteophytes.
- Spondylosis deformans: degenerative process of the spine involving essentially the anulus fibrosus and characterized by anterior and lateral marginal osteophytes arising from the vertebral body apophyses, while the intervertebral disc height is normal or only slightly decreased.
- Transverse tear: tear or fissure of the anulus, running in the axial plane (horizontally), usually limited to rupture of the outer anular attachments to the ring apophysis. Transverse tears are usually small and are located at the junction of the anulus and ring apophysis. They may fill with gas and, thereby, become detectable on radiographs or CT. They may be early manifestations of spondylosis deformans.
- Vertebral body marrow changes (Modic's classification): reactive vertebral body modifications associated with disc inflammation and degenerative disc disease, as seen on MR images. Type 1 refers to decreased signal intensity on T1-weighted spin-echo images and increased signal intensity on T2-weighted images, indicating bone marrow edema associated with acute or subacute inflammatory

changes. Types 2 and 3 indicate chronic changes. Type 2 refers to increased signal intensity on T1-weighted images and isointense or increased signal intensity on T2-weighted images, indicating replacement of normal bone marrow by fat. Type 3 refers to decreased signal intensity on both T1 and T2-weighted images, indicating reactive osteosclerosis.

**Anatomy and Innervation of the lumbar spine**

The lumbar spine normally consists of 5 lumbar vertebrae and the sacrum. Two vertebrae and the intervertebral disc compose a motion segment. A motion segment with all its parts can be a pain generator. The intervertebral disc in adults is composed of the annulus fibrosus and the nucleus pulposus and the vertebral endplate. The annulus fibrosus consists of numerous concentric rings of fibrocartilaginous tissue. The rings are thicker anteriorly than posteriorly. The nucleus pulposus is a gelatinous loose material in the center of the disc. This material usually is under considerable pressure and is contained by the annulus. Because of the structural imbalance of the annulus, the nucleus is slightly posterior in the disc. The lumbar intervebral discs are supplied by a variety of nerves. The sinuvertebral nerves are responsible for the posterior innervations of the ventral compartment. The ramus communicans nerve is one of the important pathways for discogenic pain. The pain receptors are located in:

- Ligaments of the spine
- Paraspinal musculature

- Periosteum of vertebral bodies
- Outer third of the annulus fibrosus.
- Facet joints

The two main branches which provide sensory innervation to the various structures of the spine are[38]:

- Sinuvertebral or recurrent meningeal nerve,
- Medial branch of the posterior primary ramus.

The first nerve to emerge is the sinuvertebral nerve which emerges from the spinal nerve just outside the intervertebral foramen and then re-enters the vertebral canal to supply the ventral half of the vertebral column, including:

- Dura mater
- Posterior longitudinal ligament,
- Intervertebral discs
- Anterior longitudinal ligament[39]

The spinal nerve then branches into its anterior and posterior primary rami; the posterior ramus branches into the medial and lateral branches. The medial branch supplies the dorsal parts of the vertebral column including the following[40]:

- Facet joint
- Vertebral arch
- Spinous process

Note that the posterior aspect of the dura mater is not innervated.[41] The annulus fibrosus of the intervertebral disk has diverse innervations. The dorsal aspect of the

annulus fibrosus and the posterior longitudinal ligament are innervated are by the sinuvertebral nerve, the dorsal and lateral side is innervated by other branches of the anterior spinal nerve; the ventral and lateral side is innervated by branches of the ramus communicans nerve that connect the spinal nerve and the sympathetic trunk. The ramus communicans nerve branches from the spinal nerve just after it enters the intervertebral foramina. It runs anteriorly at the inferior third portion of the vertebral body, and connects to the sympathetic trunk before branching to the lateral and anterolateral aspects of the discs above and below. Therefore, each disc is innervated by 4 separate ramus communicans nerves; right and left, superior and inferior. Because of this pattern of innervation, the 2 ramus communicans nerves-superior and inferior-on either side should be denervated in case of unilateral discogenic pain. The ramus communicans nerve also innervates the vertebral body.

The mechanism for transmission of a noxious stimulus from a vertebral disc is not yet completely understood. However, one hypothesis suggests that the impulse is transmitted to the sympathetic trunk via the sinuvertebral nerve and the ramus communicans nerve. The gray ramus communicans nerve provides the greatest source of disc innervation.

**Etiology of back pain**

The differential diagnosis of low back pain is broad and variable and includes specific and nonspecific causes. Specific low back pain is defined as back pain caused by specific pathophysiological mechanism[42] such as HNP, infection, tumor, fracture, or

inflammation. Nonspecific back pain is defined as symptoms without a precise cause. Approximately 90% of all patients with low back pain patients have a nonspecific cause and a precise pathologic anatomical cause cannot be reliably identified[43]

In nonspecific, mechanical low back pain, the symptoms are thought to arise from local processes involving the spine and surrounding structures including the muscles, ligaments, facet joints, nerves, periosteum, blood vessels, and intervertebral discs. A wide range of terms are used for back pain due to mechanical causes including low back or lumbar pain/strain/sprain, lumbago, spondylosis, segmental or somatic dysfunction, ligamentous strain, subluxation, and facet joint, sacroiliac, or myofascial syndromes[44]

LBP arising from structures of the back can be distinguished from back pain referred from visceral diseases. In referred pain, there are no signs of stiffness, and movement of the back does not increase the pain.

**Mechanical structural back pain etiologies:**

- Spondylosis (degenerative disk disease)
- Spondylolisthesis: anterior displacement of one vertebra, typically L5, over the one beneath it.
- Spondylolysis: defect in the pars interarticularis without vertebral slippage.
- True disk herniation: presents with LBP with radiculopathy symptoms.
- Foraminal stenosis: bony material causing nerve oot compression and can not be distinguished from disk herniation symptoms
- Facet arthropathy
- Spinal stenosis:  nonspecific low back pain with typical neurogenic claudication.

- Fracture: traumatic or osteoporotic
- Musculoligamentous: lumbar strains or sprains can be considered due to a nonspecific idiopathic musculoligamentous etiology
- Discogenic pain: internal disc disruption and annular tear
- Congenital disease: severe kyphosis, severe scoliosis or flat spine syndrome

**Non-mechanical spinal etiologies**:

- Neoplastic and metastatic disease:
- Infection: osteomyelitis , septic discitis , paraspinal or epidural abscess
- Inflammatory arthritis: ankylosing spondylitis, Reiter's syndrome, psoriatic spondylitis or inflammatory bowel disease
- Paget's disease
- Scheuermann's disease (osteochondrosis)

**Referred pain from visceral disorders**:

- Pelvic organs: prostatitis , endometriosis or pelvic inflammatory disease
- Renal disease: nephrolithiasis, pyelonephritis or perinephric abscess
- Vascular disease: abdominal aortic aneurysm
- Gastrointestinal disease: pancreatitis, cholecystitis or perforated bowel

**Guidelines for managing acute back pain**:

Most acute LBP with or without sciatica or acute disk herniation is a self-limited process and will disappear within 1-3 months. A comprehensive history and physical examination are important determinants in the diagnosis of LBP syndrome. Because of the high prevalence of the problem, the variation in its management, and its generally

good prognosis, efforts to summarize evidence supporting common treatments for low back pain and to develop recommendations have been undertaken[45,46,47] In the United States, the Agency for Health Care Policy and Research (AHCPR) published a guideline on acute low back pain in 1994. A panel of experts reviewed the available literature using strict criteria to assess the quality of the evidence. The panel focused on recommendations for the initial and subsequent evaluation and treatment of individuals with low back and/or back-related leg symptoms of less than 3 months duration. A major finding of the guideline was that there was a paucity of reliable data on which to base treatment recommendations.

**History**

The history should include the patient's age, past medical and surgical history and any history of trauma. The presence of constitutional symptoms, night pain, bone pain or morning stiffness, claudication , numbness, tingling, weakness, radiculopathy, and bowel or bladder dysfunction should be noted. The onset of pain, its location, radiation, characteristics, and severity should be assessed. Aggravating and relieving factors should be noted. Previous therapy and its efficacy, and the functional impact of the pain on the patient's work and activities of daily living should be queried. The signs and symptoms of radiculopathy, facet syndrome, sacroiliac joint syndrome (see specific chapters on these syndromes) should be noted. Finally, as assessment of social and psychologic factors that may affect the patient's pain should be made.

**Physical examination**

A comprehensive general physical examination is recommended in patients with back pain. A detailed neurologic evaluation should be performed. These are outlined in the chapter on physical examination. The different tests for the different syndromes causing LBP, including nerve root irritation, facet syndrome, and sacroiliac joint syndrome, are discussed on the specific chapters on these syndromes.

**Red flags:**

Patients with low back pain should be screened for the possibility of potentially serious conditions including possible fracture, tumor, infection, or cauda equina syndrome[48]. Frequently, there are well described "red flags" which distinguish these serious conditions from the much more frequent "benign" causes (degenerative disc disease, disc herniation, spondylolisthesis) of low back pain. It's not uncommon, however, for a serious condition such as an infection or tumor to go undetected or mistaken for benign low back pain without a characteristic red flag. In general, patients with "benign low back pain" should have mechanical dysfunction with pain on sitting, bending, lifting or twisting and should improve with a short course of non-operative treatment. Those patients with atypical symptoms or who fail to improve should be evaluated with an MRI or other appropriate studies to confirm the benign diagnosis and rule out more serious conditions in the differential. Avoid the trap of making a diagnosis that cannot be confirmed (muscle sprain or myofascial pain) as this is the most common

reason appropriate workup is delayed and serious conditions identified late in their course.

## Red flags for potentially serious conditions

| Possible fracture | Possible tumor or infection | Possible cauda equina syndrome |
|---|---|---|
| From medical history | | |
| Major trauma such as vehicle accident or fall from height | Age over 50 or under 20 | Saddle anesthesia |
| Minor trauma or even strenuous lifting (in older or potentially osteoporotic patient). | Constitutional symptoms, such as recent fever or chills or unexplained weight loss. | Recent onset of bladder dysfunction, such as urinary retention, increased frequency, or overflow Incontinence. |
| | Risk factors for spinal infection: recent bacterial infection (e.g. urinary tract infection); IV drug abuse; or immune suppression (from steroids, transplant, or HIV). | |
| | Pain that worsens when supine; Severe nighttime pain. | |
| **Substantial increase in pain or functional disability (fracture, tumor/infection,cauda equina**) | | |
| From physical examination | | |
| **Tenderness to palpation** | | |
| | | Unexpected laxity of the anal sphincter |
| | | Perianal/perineal sensory loss. |

| | |
|---|---|
| | Major motor weakness; quadriceps (knee extension weakness); ankle plantar flexors, evertors, and dorsiflexors (foot drop). |

**Imaging studies**

An acute episode of LBP does not warrant immediate imaging studies unless one or more of the following is present:

- Neurologic deficit
- History of trauma
- Pain does not subside spontaneously
- Pain is severe or unusual in character
- Systemic or other injury is suspected
- History of Cancer
- Corticosteroid use,
- Drug or alcohol abuse
- Temperature greater than 38°C (100.4°F)
- Unexplained weight loss

In the evaluation of patients with low back pain, it is essential to correlate all imaging findings with the patient's symptoms and signs on physical examination. Because most imaging studies reveal abnormal findings in asymptomatic patients, a diagnosis should not be based solely on diagnostic imaging without firm correlation to the patient's symptoms.

**Plain-film radiography:**

The simple x-ray films allows the evaluation of the bony anatomy, arthritic changes of the lumbar spine and the degenerative disk disease but does not show soft tissue anatomy which requires further testing for definite diagnosis. Plain x-ray films are rarely useful in the initial evaluation of patients with acute low back pain[49,50]. Studies have shown that plain x-ray films were normal or demonstrated changes of equivocal clinical significance in the majority (>75%) of patients with low back pain.

Traditionally, the plain radiograph has been the first imaging test performed in the evaluation of low back pain because it is relatively inexpensive, widely available, reliable, and easy to perform. The two major drawbacks of plain radiography are the difficulty in its interpretation and an unacceptably high rate of false-positive findings[51]. Plain radiographs are not required in the first month of symptoms unless the physical examination reveals specific signs of trauma or there is suspicion of tumor or infection[52]. It is important to obtain pictures that are free of motion or grid artifacts that display soft tissue and osseous structures of the entire lumbar spine.

Having a standard approach to evaluating radiographs can help prevent a missed diagnosis and it is crucial to develop and maintain a specific sequence of observation. The traditional sequence includes anteroposterior (AP) and lateral views of the lumbar spine, primarily to detect tumors or spinal misalignments such as scoliosis. In the AP view, the indicators of a normal spine 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 whole pelvis allowing for evaluation of the acetabulum and heads of the femur and for the detection of possible degenerative changes in the pelvis. The lateral view provides a good image of the vertebral bodies, facet joints, lordotic curves, disc space height, and intervertebral foramen. Decreased disc space height can be indicative of disc degeneration, infection, and postsurgical condition. Unfortunately, there is a poor correlation between decreased disc height and the etiology of low back pain. Anterior slippage (spondylolisthesis) of the fifth lumbar vertebra on the sacral base can be identified in lateral views.

Oblique views with the radiograph tube angled at 45 degrees improve visualization of the neural foramina and pars interarticularis and are used to confirm suspicions generated from the initial imaging assessment. Oblique views are used to show tumors, facet hypertrophy, and spondylosis or spondylolisthesis. Flexion-extension views are helpful in assessing ligamentous and bony injury in the axial plane. The use of these views should be limited to patients who do not have other radiographic abnormalities and patients who are neurologically intact, cooperative, and capable of describing pain or early onset of neurologic symptoms. Flexion-extension views can be used in trauma patients, especially those with muscle spasm, which may be the only sign of spinal instability. When examining the lumbar spine for possible fracture,

it is important to include the lower portion of the thoracic spine because of the high occurrence of injury between levels T12 and L2. This region is more prone to injury because of the change in orientation of the facet joints between the thoracic spine and the lumbar spine and because it lies directly beneath the more rigid thoracic spine, which is stabilized by the rib cage.

Degenerative changes are often evident on plain radiographs; caution must be used in making a diagnosis based on degenerative radiographic changes because of the high rate of asymptomatic degenerative changes. Radiographic evidence of degenerative change is most common in patients older than 40 years and is present in more than 70 percent of patients older than 70 years[51] Degenerative changes have been reported to be equally present in asymptomatic and symptomatic persons[51.] The incidence of intervertebral narrowing and irregular ossification of the vertebral end plates has also been shown to be associated with increased age[53]. Even though plain radiographs usually provide little definitive information, they should be included in the screening examination for patients with certain red flags.

**Bone Scintigraphy**

Bone scintigraphy is useful when clinical findings are suspicious of steomyelitis, bony neoplasm or occult fracture. Plain radiographs, CT scans and MRIs reveal morphologic changes in bone. Bone scintigraphy detects biochemical changes through images that are produced by scanning and mapping the presence of radiographic compounds (usually technetium Tc 99m phosphate or gallium67 citrate). The image

produced indicates bone turnover, a common occurrence in bone metastases, primary spine tumors, fracture, infarction, infection, and other metabolic bone diseases. Bone metastases normally appear as multiple foci of increased tracer uptake asymmetrically distributed. In extreme cases of bone metastases, diffusely increased uptake of tracer results in every bone being uniformly illustrated and can be falsely interpreted as negative. Aggressive tumors that do not invoke an osteoblastic response, such as myeloma, can also yield a negative examination. Primary spine tumors are usually benign. Osteoid osteoma, osteoblastoma, aneurysmal bone cyst, and osteochondroma produce an active bone scan. These tumors generally affect the posterior elements of the spine. Computerized tomography must be used to differentiate them and isolate their anatomic position.

Recent studies[54,55] evaluated the ability of bone scans, with the addition of single-photon emission computed tomography (SPECT), to distinguish benign lesions from malignant lesions. SPECT scan differs from bone scan because it provides a three-dimensional image that enables physicians to locate the lesion more precisely. Lesions that affect the pedicles are a strong indicator of malignancy, while lesions of the facets are likely to be benign. Lesions of the vertebral body or spinous process are just as likely to be benign as malignant and, therefore, offer little diagnostic evidence.

**Computed Tomography:**

CT is used to complement information obtained from other diagnostic imaging studies such as radiography, myelography, and MRI. The principal value of CT is its ability to demonstrate the osseous structures of the lumbar spine and their relationship to the neural canal in an axial plane. A CT scan is helpful in diagnosing tumors, fractures, and partial or complete dislocations. In showing the relative position of one bony structure to another, CT scans are also helpful in diagnosing spondylolisthesis. They are not as useful as MRI in visualizing conditions of soft tissue structure, such as disc infection. The data used to generate the axial images are obtained in contiguous, overlapping slices of the target area. The axial image data can be reformatted to construct views of the scanned area in any desired plane. Three-dimensional CT and CT–with myelogram are reserve for more complicated problems like failed back surgery syndrome.

The limitations of CT include less-detailed images and the possibility of obscuring nondisplaced fractures or simulating false ones. In addition, radiation exposure limits the amount of lumbar spine that can be scanned, and results are adversely affected by patient motion; spiral CT addresses these weaknesses because it is more accurate and faster, which decreases a patient's exposure to radiation exposure.

**Magnetic Resonance Imaging:**

MRI today has become the modality of choice in the evaluation of spinal degenerative disease. MR is superior even to CT with contrast in the distinction of bone, disc, ligaments, nerves, thecal sac, and spinal cord. On the T1WI (T1 weighted image),

the disc is a fairly homogenous structure and isointense compared to muscle. On long TR images (The TR is the time between consecutive 90 degree radiofrequency pulse**)**, the disc becomes brighter due to its water content. The CSF appears dark in the T1 weighted image and appears white on the T2 weighted image**.** The nucleus pulposus which is more hydrated than the annulus fibrosis becomes brighter than the annulus on the T2 weighted image. Therefore the disk appears black on T1 and white on T2.

MRI is the test of choice for the diagnostic imaging of neurologic structures related to low back pain. MRI can evaluate soft tissue and non-bony structurs pathology and disk herniation with greater accuracy than CT. For this reason, MRI remains the gold standard test in detecting early soft tissue pathologies like osteomyelitis, discitis, and epidural-type infections or hematomas. MRI is safe with no known biohazard effects. It can be problematic for patients with claustrophobia. The only contraindication to MRI is the presence of ferromagnetic implants, cardiac pacemakers or intracranial clips. Metal stabilization devices such as plates, rods, screws and loops, used in spinal operations impose local artifacts and usually render imaging of the spinal canal almost impossible with the MRI.

As with other imaging techniques, MRI can identify abnormalities in asymptomatic persons. In one study[56], MRIs of 67 asymptomatic persons 20 to 80 years of age were obtained. At least one herniated disc was identified in 20 percent of people younger than 60 years and in 36 percent of those older than 60 years. Another

study[57]discovered that 63 percent of asymptomatic persons had disc protrusion, and 13 percent had disc extrusion.

**Electrodiagnostic studies:**

Electrodiagnostic studies have only a limited role in the evaluation of acute low back pain since it takes two to four weeks after the onset of symptom before any findings are present on EMG or nerve conduction studies. Electrodiagnostic studies may help if the clinical findings are suggestive of radiculopathy or peripheral neuropathy. These studies help in confirming the working diagnosis and identifying the presence or absence of previous injury. They are also useful in localizing a lesion, determining the extent of injury, predicting the course of recovery and determining whether structural abnormalities on radiographic studies are of functional significance[58]

**Psychosocial Evaluation**

Screening for non-physical factors is critical in the management of back pain. Psychological, occupational and socioeconomic factors can complicate both assessment and treatment. Studies have revealed that patients with lower job satisfaction are more likely to report back pain and to have a protracted recovery[59]. Patients with an affective disorder (e.g., depression) or a history of substance abuse are more likely to have difficulties with pain resolution. The physician should inquire if litigation is pending since this can often adversely affect the outcome of therapy.

**NONINVASIVE TREATMENTS**

In acute LBP, there is little or no evidence that most of the popular treatment and therapies alter the natural course of the disease. The conservative approach would be a

short period of rest, analgesics, retuning to function and NL activity as soon as possible and then an exercise program to minimize reoccurrence. In chronic LBP, the multidisciplinary biopsychosocial rehabilitation treatments with functional restoration have been shown to improve pain and function[60,61]

**REST:**

Evidences suggest that return to normal daily activity as soon as possible is a good approach to manage acute LBP. A randomized clinical trial found that patients with two days of bed rest had clinical outcomes similar to those in patients with seven days of bed rest.[62] Studies showed that a faster return of function and ordinary activity produced faster recovery. There was no evidence that early activity had any harmful effects or led to more recurrences. Bed rest for more than a week in patients with acute LBP is not advisable. The current recommendation is two to three days of bed rest in patients with acute radiculopathy.[63]

**Pharmacologic Therapy**

Recent evidence in the Cochrane Collaboration Back Review,[64,65]which included data from 51 trials, suggests that nonsteroidal anti-inflammatory drugs (NSAIDs) are moderately effective for the short-term symptomatic relief of patients with acute low back pain. There does not seem to be a specific type of nonsteroidal antiinflammatory drug that is clearly more effective than others. Evidence on the use of NSAIDs in chronic low back pain still is lacking.

If no medical contraindications are present, a two- to four-week course of an anti-inflammatory agent is suggested. Gastrointestinal prophylaxis might be necessary with the older types of NSAIDs for patients who are at risk for peptic ulcer disease. The newer NSAIDs with selective cyclo-oxygenase2 inhibition have fewer gastrointestinal side effects, but they still should be used with caution in patients who are at risk for peptic ulcer or kidney disease.

The short-term use of a narcotic may be considered for the relief of acute pain. The need for prolonged narcotic therapy should prompt a reevaluation of the etiology of a patient's back pain.

The use of muscle relaxants has been shown to have a significant effect in reducing back pain, muscle tension and increased mobility after one and two weeks[66]. All these medication can have significant adverse effects even after a short course and should be used cautiously.

**Intraspinal injections**

These modalities are discussed in several chapters on this book. These interventions are innovative and backed mostly by anecdotal reports; prospective randomized studies on the efficacy of some of these procedures are still lacking.

**Physical therapy:**

Although there have been randomized controlled trials and systematic reviews of the effectiveness of physical intervention therapies for the management of low back pain,

the role of these treatment remains unclear. There are data to suggest that general exercise programs may have beneficial effects on low back pain. Passive physical therapies such as heat, massage, electrical stimulation or ultrasound provide temporary comfort but no evidence of long term improvement[74] . In general, strengthening exercise programs that facilitate weight loss appear to be helpful in alleviating low back pain. Exercises that promote strengthening of the axial muscles that support the spine should be included in the physical therapy regimen. Aggressive exercise programs have been shown to reduce the need for surgical intervention.

There is limited evidence to show that specific back exercises produce clinical improvement in acute low back pain. More recently, a Cochrane review[67] identified 39 studies and concluded that the data did not support the efficacy of specific exercises in the treatment of acute low back pain. Waddell *et al*[68] **(Rasha: note reference cited)** cited evidence that general exercise programs can improve pain and functional levels in those with chronic low back pain. The general exercise program may be helpful for chronic low back pain patients to increase return to normal daily activities and work

Continuation of normal activities is recommended for acute low back pain. National guidelines in the USA[48] and UK[68,69] recommend a return to normal activity as soon as possible for patients with acute back pain and encourage the early access to physical therapy. Therapeutic exercises were found to be beneficial for chronic, subacute, and post-surgical low back pain.

In the review by Waddell et al,[70] they concluded that continuation of normal activities leads to less chronic disability and time off work than the traditional advice to rest and "let pain be your guide". Subsequent Cochrane reviews of the treatments for acute low back pain and sciatica concluded that the "advice to stay active" has little beneficial effect for patients[71] and that, compared to bed rest, advice to stay active alone will have limited beneficial effects[72] The treatment goals are to relieve pain, reduce muscle spasm, improve range of motion (ROM) and strength, correct postural problems, and ultimately improve functional status.

A number of rehabilitation interventions are used in the management of people with LBP. Among the current musculoskeletal interventions specific for LBP are body mechanics and ergonomics training, posture awareness training, strengthening exercises, stretching exercises, activities of daily living (ADL) training, organized functional training programs, therapeutic massage, joint mobilizations and manipulations, mechanical traction, biofeedback, electrical muscle stimulation, transcutaneous electrical nerve stimulation (TENS), thermal modalities, cryotherapy, deep thermal modalities, superficial thermal modalities, and work hardening[73].

The Philadelphia Panel efforts[74] to form evidence-based clinical practice guidelines (EBCPGs) for the management of LBP were developed based on a systematic grading of the evidence determined by an expert panel, and the evidence was derived from systematic reviews and meta-analyses using the Cochrane Collaboration methodology. The finalized guidelines were circulated for feedback from practitioners to verify their applicability and ease of use for practicing clinicians.

Exercises:

The Philadelphia Panel recommendations [74]are in agreement with those of the AHCPR guidelines that continuation of normal activities (such as walking) is more effective than bed rest for the management of acute LBP[75]. It showed that extension, flexion, or strengthening exercises are effective for subacute and chronic LBP and for postsurgical LBP. The results for acute LBP are in full agreement with the guidelines and other reviews [76] concerning moderate effectiveness of stretching or strengthening exercises, and highly effective for the patient "to stay active." [77] Certain authors recommend return to functional and work activities as soon as possible after lumbar injury to avoid the negative effects of immobilization and bed rest prescription[78]. Task-oriented activities are recognized in rehabilitation. Patients with LBP benefit from these activities as they improve ADL for chronic LBP[79].

There is evidence to support and recommend the use of continued normal activities for acute nonspecific LBP and therapeutic exercises for chronic, subacute, and postsurgical LBP. At the present time, there is insufficient evidence regarding the definite role of thermotherapy, therapeutic massage, EMG biofeedback, mechanical traction, therapeutic ultrasound, TENS, electrical stimulation, and combined rehabilitation interventions.

**Acupuncture[80,81]**

Two analyses of randomized controlled trials on the role of acupuncture (one in the framework of the Cochrane Collaboration Back Review) found that there was little or no evidence that acupuncture is effective in the management of back pain. Van Tulder's[80]

systematic review of 11 RCTs (n=542) assessed the effects of acupuncture for the treatment of non-specific low back pain. Some of the study populations contained people with acute or unspecified low back pain. Three RCTs compared acupuncture to no treatment and provided conflicting evidence. Two RCTs found that acupuncture was not more effective than trigger point injection or TENS. Eight RCTs compared acupuncture to a placebo or sham acupuncture. Of the two RCTs of higher methodological quality, one did not find any difference while the other study was positive for acupuncture although in this study the control group seemed to have more severe complaints at baseline. Five of the six remaining (lower quality) RCTs indicated that acupuncture was not more effective than placebo or sham acupuncture. In the last study the overall conclusion was 'unclear'. Van Tulder et al could not clearly conclude that acupuncture is effective in the management of back pain and con not recommend acupuncture as a regular treatment for patients with low back pain. There is clearly a need for more high-quality randomized controlled trials.

**Alternative therapies (spinal manipulation):**

The exact role of spinal manipulation is not clear. Spinal manipulation proved superior to other nonconventional therapies but was not found to be more effective than traditional back pain management [82]. For patients with acute lower back pain, spinal manipulation conferred statistically significant benefits in comparison with sham therapy. Similar results were noted among patients with chronic low back pain who received spinal manipulation when compared with sham manipulation. Assendelft et al,[82] on the other hand, concluded that there was no evidence for increased effectiveness of spinal

manipulative therapy compared with other advocated therapies for acute and chronic low back pain. Massage and spinal manipulation have relatively small clinical benefits for both acute and chronic back pain. However, they are cheaper than many conventional medical techniques and adverse side effects are rare.

Cherkin et al [83] analyzed original articles and systematic reviews of randomized controlled trials that evaluated acupuncture, massage therapy, and spinal manipulation for nonspecific back pain published since 1995. The authors concluded that "the effectiveness of acupuncture for back pain remains unclear, massage is effective for persistent back pain, spinal manipulation has small clinical benefits, similar to those of other commonly used therapies, for acute and chronic back pain. Assendelft and colleagues [82]**(ref?)** conducted a meta-analysis of 53 published articles, representing 39 studies, which compared spinal manipulation or mobilization with another treatment or control. A total of 5,486 patients were included, with individual study sample sizes varying from 19 to 666 (median, 92). Comparison therapies included sham therapies, conventional general practitioner care (which in most cases involved the prescription of analgesics), physical therapy and exercise, and treatments (eg, traction, bed rest, topical gel) for which there is a lack of evidence of benefits or evidence of harm. Assendelft *et al*., in a more recent systematic review[82], concluded that spinal manipulative therapy has no statistically or clinically significant advantage over general practice care, analgesics, physical therapy, exercise or back school for acute or chronic back pain.

Koes *et al*.[84]reviewed 38 trials and concluded that, although some results were encouraging, further trials were needed to establish the effectiveness of manipulation. In contrast, Shekelle *et al*.[85] did a meta-analysis combining data from nine trials and concluded that manipulation could increase the rate of recovery from acute uncomplicated low back pain, but that there were insufficient data to provide evidence for the effectiveness of manipulation in patients with chronic pain. The US Agency for Health Care Policy and Research (AHCPR)[48] reviewed four meta-analyses and 12 additional randomised trials and also concluded that manipulation could speed the recovery of patients with acute back pain and that the evidence to support the use of manipulation for radiculopathies or longer standing back pain was inconclusive. The systematic review by Assendelft *et al*.[86] was highly critical of the general standard of the other reviews. Nevertheless, some of the reviews reported some positive effects of manipulation.

**Biofeedback Treatments.**

The treatments involve external feedback to translate physiological activity of muscle response (often using electromyography) into visual or auditory signals that help the patient reduce muscle tension and pain. No studies have used these techniques in patients with acute symptoms, and there is limited evidence that biofeedback is ineffective for chronic low back pain[45,87]

**Patient Education**

It is critical that the patient understands the nature of his spine disorder and his role in avoiding re-injury. The appropriate postures for sitting, driving and lifting should be reviewed. Weight loss and healthy life-style should be emphasized.

**Surgical Treatment**

The surgical treatment of lumbar spinal disorders has made substantial advances in the last two decades. Rigid instrumentation systems, minimally invasive techniques, recombinant DNA, and joint replacement are just a few technologies which are rapidly changing what and how we treat spinal pathology. With these advances has come a corresponding increase in the rates of spine surgery; as high as 8.6/1000 Medicare enrollees in some regions. [88] Although many of these patients benefit immensely there is a definitive complication rate which must be carefully weighed against potential benefits when considering surgical intervention. Validated outcome measures and randomized trials must be applied to these new techniques to accurately assess both their effectiveness and inherent risks.

Low back pain most commonly results from degenerative changes which produce neural compression or mechanical dysfunction. Surgical treatment, therefore, typically requires some degree of neurologic decompression and or fusion respectively. More recently, disc replacement has demonstrated increasingly encouraging results and may, as it has in the peripheral skeleton, become a meaningful alternative to arthrodesis. This section will review some of the various surgical treatments for spinal disorders and is organized by the underlying treatment principle rather than specific diagnosis:

decompression, fusion, arthroplasty, and reconstruction. It's important to emphasize that each patient has a unique combination of pathology and expectations for treatment. Successful surgical management require a detailed clinical evaluation with confirmatory imaging studies to accurately identify the symptomatic pathology, a careful assessment of the risks and benefits associated with any procedure, and a strict adherence to orthopaedic principles while implementing treatment.

## Decompression

Back pain is the fifth most common complaint leading to physician visits and the majority of these relate to disc degeneration and herniation. The disc itself may produce significant back pain and even referred pain into the groin, hip, or leg. When degenerative changes encroach upon neurologic structures, they frequently produce back and leg pain from acute nerve compression in the younger patient or more insidious compression (neurogenic claudication) in the older patient population. The vast majority of these patients will improve with non-operative management including NSAID's, physical therapy, and injections.[89,90] For those who fail to improve with non-operative treatment surgical decompression remains an excellent option to definitively decompress neurologic structures and relieve pain. Patients with acute and dense motor deficits should be considered for early decompression as it remains the most effective means of relieving compression and optimizing recovery although some patients do improve with non-operative treatment.[91]

Since Mixter and Barr's classic report in 1934[92] discectomy has become the most commonly performed spinal surgery and remains the gold standard to which all other treatments must be compared. Less invasive microdiscectomy techniques were popularized in the late 1970's permitting faster recovery and return to work with improved patient outcomes. [93,94] More recently, endoscopic discectomy has been advocated as a safe and effective ambulatory procedure with superior results to other outpatient therapies (chemonucleolysis, percutaneous discectomy, and thermal coagulation). Indications include patients with primary leg pain, a positive straight leg raise, and imaging studies confirming compression at the symptomatic level. The principles of surgical treatment are decompression, mobilization of the affected nerve root, and removal of the herniated fragment. This typically includes release of the ligamentum flavum, partial laminotomy, medial facetectomy, and discectomy. Discectomy techniques differ but include at minimum removal of non-contained herniations and vertical annulotomy for removal of contained herniations. The endoscopic technique allows a limited exposure through an 18mm tubular retractor with results comparable to microdiscectomy (**Figure 1**). One study demonstrated complete relief of pain in 72% of patients and minimal discomfort requiring no further treatment in another 20% with a length of stay averaging 3.5 hours. [95] A separate lateral approach as described by Wiltse[96] maybe required to decompress the less common foraminal disc herniation.

Older patients with cumulative degenerative changes may ultimately develop symptomatic spinal stenosis (neurogenic claudication). Multiple lesions contribute to the stenosis including disc herniations/bulges, facet arthopathy, osteochondral spurs, ligament hypertrophy, and spondylolisthesis. Symptoms typically include low back and leg pain aggravated by standing and walking which must be differentiated from vascular claudication. Non-operative treatment includes physical therapy, NSAID's, and steroid injections. Selective nerve root blocks are helpful diagnostically as well as therapeutically as they identify symptomatic levels and may help predict response to surgical decompression (**Figure 2**). Patients who fail to improve with non-operative treatment are candidates for surgical decompression. Treatment often requires decompression of the central canal, lateral recess, and/or neural foramen. Determining which areas to decompress requires a careful correlation between patient symptoms and corresponding lesions on imaging studies. Studies demonstrate pain relief in 55-78% of patients compared to 28% of patients treated non-operatively.[97,98] A fusion procedure may be needed in addition to decompression when there is co-existing instability (spondylolisthesis is present or more than 50% of the facet joints are resected) or the patient has primarily back pain implicating degenerative joint pain opposed to neurogenic pain.

Lumbar Fusions

Fusion procedures have been used successfully for over one hundred years but have been much more frequently performed over the last10-15 years. The most common

indication is disabling mechanical low back pain secondary to an underlying disorder (spondylolysis, spondylolisthesis, degenerative arthritis, and scoliosis). Spine fusion is a salvage procedure in which painful degenerative joints are resected and dysfunctional motion segments stabilized. Results vary with specific pathology but many reports demonstrate good to excellent outcomes in as many as 94% of patients[99,100] (**Figure 3**). Treating degenerative disc disease with spine fusion is far more controversial with modest success rates. Most studies demonstrate clinical improvement in 65-75% of patients and return to work rates in 36%.[101] The actual fusion rates also vary and range from 80% in posterolateral fusions to 97% with circumferential (360°) fusions.[102] Although achieving fusion does not always correlate with clinical improvement, patients with non-unions are more likely to have a worse outcome. In addition, patients with degenerative disc disease tend to have greater clinical improvement when the pain generating disc is removed which can be can be accomplished with an anterior posterior spinal fusion and instrumentation (APSFI: **Figure 4**). More recently posterior approaches such as the transforaminal lumbar interbody fusion (TLIF) provide the advantages of a circumferential fusion through a lower risk posterior approach (**Figure 5**). Clinical studies demonstrate equal or superior results with lower complication rates.[103] Various devices can be placed in the interbody space including cylindrical cages, carbon fiber devices and bone. The highest fusion rates and clinical outcomes occur when following basic biomechanical principles (obtaining rigid fixation, loading bone under compression, and maintaining lumbar lordosis) and biologic principles with appropriate grafting material (autologous bone remains the gold standard) in a bed of vascularized tissue.

Most recently, recombinant human bone morphogenic protein has been shown to have similar clinical outcomes and equal or superior fusion rates in various studies[104]. This may be a useful alternative to autologous bone grafting but future studies are needed to assess effectiveness in larger populations including multilevel cases and patients with various other risk factors.

Disc replacement arthroplasty

Although spinal fusion has been beneficial in many patients, it remains a salvage procedure which reduces motion and increases stress and consequently degeneration at adjacent levels. Disc replacement has been advocated since the 1950's as it removes the painful and dysfunctional disc and restores physiologic motion. However, it wasn't until the early 1980's that a viable design began demonstrating encouraging results. Since then various implants have emerged including ProDisc (semiconstrained device manufactured by Spine Solutions), Maverick (nonconstrained device Medtronic Sofamor Danek), and Flexcore. The Link SB Charite III is the most commonly used prosthesis with as many as 5000 implanted worldwide. It is a nonconstrained design consisting of two cobalt-chrome endplates with a sliding polyethylene core (**Figure 6**). The implant is anchored to the vertebral bodies by teeth and a bony ingrowth on the endplate surface. Biomechanical studies demonstrate increased motion in flexion and extension, mobility in torsion, and relative immobility in lateral bending. Primary indication is disabling low back pain secondary to discogenic disc disease that has failed to improve with at least 6 months of adequate nonoperative treatment. The accurate diagnosis of discogenic back pain and

identification of the symptomatic level is best confirmed by MRI and concordant pain on discography. Exclusion criteria include nerve root compression and facet arthopathy. Clinical results are good in properly selected patients with as many as 79% of patients reporting substantial improvement and 87% returning to work.[105] The postoperative rehabilitation encourages early controlled, progressive spinal motion and rapid functional recovery compared to prolonged rehabilitation in fusion patients. It is hoped that long term studies will demonstrate continued clinical improvement and implant survivability with motion preservation and decreased adjacent degeneration. There are, however, no published prospective, randomized studies comparing disc replacement to fusion although several studies are ongoing in the United States.

Spinal Reconstruction

Spinal reconstruction is necessary when a disease process destroys the structural integrity of the spine or produces a deformity, which alters normal spinal balance and biomechanics. The most common conditions requiring spinal reconstruction include trauma, infection, tumor, scoliosis, kyphosis, and increasingly iatrogenic causes from failed spinal surgery. The principles of reconstruction include resection and soft tissue release to allow realignment, anterior column support with structural grafting, rigid fixation, and biologic fusion. There are various surgical techniques employed to effect reconstruction some of which are described below.

Reconstruction frequently requires resection of diseased tissue and release of soft tissues in malaligned segments of the spine. Anteriorly, this is accomplished with

vertebral body resection (corpectomy) and discectomy (**Figure 7**). Once a corpectomy is performed the anterior column must be reconstructed with structural support. This can be accomplished with implants such as mesh cages or structural allograft or autograft. It's essential the spine is properly realigned after release to restore physiologic lumbar lordosis and thoracic kyphosis and the appropriate graft or implant length selected to maintain this sagital balance. Most structural grafts will require some form of internal fixation to maintain stability until fusion is successfully achieved. In severe cases of spinal deformity, such as scoliosis exceeding 90°, the rib cage itself may become ankylosed and also require release in the form of rib head resections to effect realignment (**Figure 8**).[106] Such reconstruction will similarly require posterior releases. These may include chevron osteotomies which can correct sagital and coronal malalignment[107], rib resection or osteotomy, and pedicle subtraction osteotomy[108] (**Figure 9**).

Once a spinal segment is properly realigned it must be rigidly fixed to maintain alignment and effect successful fusion. Modern instrumentation systems include hooks, sublaminar cables, and most frequently pedicle screws connected by rods. These "segmental" instrumentation systems allow much greater correction than earlier systems and have substantially improved the treatment of spinal deformity over the last 20 years. Nonetheless, they are subject to fatigue failure and will fracture if the spine does not go on to a solid union.

Spinal fusion remains a primary goal of most reconstruction procedures for long term stability and function. Typically, this requires resection of articulations (disc space

and facet joints), decortication of the fusion area, rigid stabilization, and an adequate volume of bone graft. The biology of lumbar fusion and bone grafts has been well characterized over the last decade and requires three key elements: precursor cells capable of transformation into bone forming osteoblasts, osteoconductive materials (which serve as scaffolds for formation of new bone), and osteoinductive growth factors which promote differentiation of progenitor cells into osteoblasts.[109] Autologous bone graft contains all three materials and remains the gold standard which all other products must be compared. Limitations in the amount of graft available and morbidity associated with harvesting have led to use of various other products including bone graft extenders (demineralized bone matrix, calcium carbonate, hydroxyapatite-tricalcium phosphate). bone graft substitutes, and more recently osteoinductive substitutes such as BMP. Although preliminary clinical studies have demonstrated promising results these products must be validated by prospective, randomized trials and they do not replace the need for following well established biomechanical and biological principles.

There have been tremendous advances in both the understanding and treatment of lumbar spinal disorders over the last two decades.[110] These advances have dramatically increased our ability to manage various spinal disorders with a corresponding increase in rates of surgery and devices used. Although many patients obtain substantial benefit there are inherent and quantifiable risks which must be carefully assessed before considering surgical treatment. The injudicious use of surgery and spinal devises exposes patients to unnecessary risks and society to excessive costs. As a result, there has already been a call for restraint in the performance of such procedures.[111]

Disorders of the lumbar spine are extremely common and increasing with the age and activity of the population. Fortunately, the vast majority of these patients improve with appropriately guided low risk non-operative care. For the small group of patients who fail to improve there are now a wide array of surgical options available. By thoroughly evaluating each patient's unique condition, carefully balancing the risks and benefits of various interventions, and employing well established treatment principles we ensure the best chance for a satisfactory outcome.

Illustration Legends

**Figure 1.** Patient undergoing endoscopic discectomy. AP and lateral fluoroscopic images demonstrating placement of the endoscope at the left L4-5 intralaminar level (A). METRx endoscope locked in position with flexible arm assembly (B). Postoperative picture demonstrating 18 mm incision following endoscopic discectomy.

**Figure 2.** Fluoroscopic image of right sided L4-5 transforaminal steroid injection. Dye injection prior to steroid demonstrating proper position and backflow along L4 nerve root sheath.

**Figure 3.** AP (A) and lateral (B) lumbar spine radiographs demonstrating grade 1 spondylolisthesis in 47 yo woman with disabling back and leg pain refractory to non-operative treatment. Postoperative radiographs demonstrating stable fusion 1 year following posterior decompression and fusion with supplemental instrumentation (C+D). Note the robust fusion mass bridging transverse processes laterally. The patient is pain free and has returned to full level of activity including triatholons and skiing.

**Figure 4.** 64 yo woman with degenerative scoliosis and disabling low back and radicular leg pain. AP radiograph demonstrates severe lateral listhesis at L2-3 and L3-4 resulting in symptomatic compressive neuropathy (A) and lateral radiograph demonstrating severe disc degeneration and consequent loss of lumbar lordosis (B). She was treated with anterior-posterior fusion, instrumentation and decompression. AP radiograph demonstrates correction of lateral listhesis and tilt (C) and lateral film shows excellent restoration of lumbar lordosis with structural interbody allograft.

**Figure 5.** 51 yo male lawyer with recurrent L4-5 disc herniation with disabling back and leg pain treated with revision discectomy and transforaminal lumbar interbody fusion (TLIF) L4-5. Circumferential fusion avoids exposure and fusion of transverse processes and resulting denervation of paraspinal muscles. (A). The lateral radiograph demonstrates excellent interbody support and trabeculating bone (B). Diagram illustrating technique of inserting structural allograft through transformainal approach (C).

**Figure 6.** The Link SB Charite III artificial disc (A) and lateral radiograph of a patient treated for degenerative disc disease with the Link SB Charite III at L4-5.

**Figure 7.** 43 yo woman with blastomycosis involving T9 and T10 with progressive collapse (A) and lower extremity weakness secondary to neurologic compromise (B). Reconstruction involved T9 and T10 vertebrectomies and anterior column support with fibular allograft and vascularized rib autograft followed by posterior fusion and instrumentation (C+D). The patient had resolution of infection with full functional and motor recovery.

**Figure 8.** 22 yo male with progressive idiopathic scoliosis, stiff right thoracic curve measuring 97 degrees, decompensation, and FVC 37% (A). Lateral radiograph demonstrates thoracic lordosis and positive sagittal balance measuring 5 cm (B). The patient was treated with T9 vertebrectomy, internal thoracoplasties, and posterior osteotomies to safely release the stiff deformity and stabilization with fusion and instrumentation from T2-L3. Two year follow up demonstrates excellent correction of scoliosis (C) and restoration of balance in both coronal and sagittal planes. Spondylolisthesis remains asymptomatic without progression (D).

**Figure 9.** 42 yo male with ankylosing spondylitis and progressive kyphotic deformity (A). Lateral radiographs demonstrated kyphosis involving primarily the lumbar spine (B). AP and lateral radiographs following a pedicle subtraction osteotomy of L3 (C+D). Note the substantial improvement in forward gaze and neutralization of C-7 plumbline (E).

**References:**


1. National Institute for Occupational Safety and Health, Musculoskeletal disorders and workplace factors: a critical review of epidemiological evidence for work-related musculoskeletal disorders of the neck, upper extremity, and low back. , National Institute for Occupational Safety and Health, US Department of Health and Human Services, Rockville, MD (1997) NIOSH technical report 97-141 .

2. Andersson GBJ, Frymoyer JW, eds. The Epidemiology of Spinal Disorders.. 2nd ed. New York, NY: Raven Press, 1997:93-141

3. G.B. Anderson, Epidemiological features of chronic low back pain. Lancet 354 (1999), pp. 581–585

4. H.B. Bressler, W.J. Keyes, P.A. Rochon and E. Badley, The prevalence of low back pain in the elderly: a systematic review of the literature. Spine 24 (1999), pp. 1813–1819.

5. A.L. Nachemson, Newest knowledge of low back pain: a critical look. Clin Orthop 279 (1992), pp. 8–20

6 . Andersson GBJ, Frymoyer JW, eds. The Epidemiology of Spinal Disorders.. 2nd ed. New York, NY: Raven Press, 1997:93-141.

[7] . Skovron ML. Epidemiology of low back pain. Baillieres Clin Rheumatol. 1992;6:559-573.

[8]. McCaig LF. National Hospital Ambulatory Medical Care Survey: 1992 emergency department summary. Advance data from vital and health statistics; no 245. Hyattsville, MD: National Center for Health Statistics, 1994

[9]. Hart LG, Deyo RA, Cherkin DC. Physician office visits for low back pain. Frequency, clinical evaluation, and treatment patterns from a U.S. national survey. Spine 1995;20:11-9

[10]. Bowman JM. The meaning of chronic low back pain. AAOHN J. 1991;39:381-438.

[11]. Deyo RA, Cherkin D, Conrad D, Volinn E. Cost, controversy, crisis: low back pain and the health of the public. Annu Rev Public Health 1991;12:141-56.

[12]. Frymoyer JW. Can low back pain disability be prevented? Bailliere's Clin Rheumatol 1992;6:595-606.

[13] . Bowman JM. The meaning of chronic low back pain. AAOHN J. 1991;39:381-438.

[14]. Van Tulder MW, Koes BW, Assendelft WJ, et al. The Effectiveness of Conservative Treatment of Acute and Chronic Low Back Pain.. Amsterdam, the Netherlands: EMGO Institute, 1999

[15] . Deyo RA, Tsui-Wu YJ. Functional disability due to low-back pain: a population-based study indicating the importance of socioeconomic factors. Arthritis Rheum. 1987;30:1247-1253.

[16]. P. Dolan, I. Kingma, J. van Dieen et al., Dynamic forces acting on the lumbar spine during manual handling—can they be estimated using electromyographic techniques alone?. Spine 24 (1999), pp. 698–703

[17]. F.A. Fathallah, W.S. Marras and M. Paranianpour, An assessment of complex spinal loads during dynamic lifting tasks. Spine 23 (1998), pp. 706–716.

[18]. National Institute for Occupational Safety and Health, Musculoskeletal disorders and workplace factors: a critical review of epidemiological evidence for work-related musculoskeletal disorders of the neck, upper extremity, and low back. , National Institute for Occupational Safety and Health, US Department of Health and Human Services, Rockville, MD (1997) NIOSH technical report 97-141

[19]. P.M. Bongers, C.R. Dewinter, M.A.J. Kompier and V.H. Hildebrandt, Psychosocial factors at work and musculoskeletal disease. Scand J Work Environ Health 19 (1993), pp. 297–312.

[20]. A. Burdorf and G. Sorock, Positive and negative evidence of risk factors for back disorders. Scand J Work Environ Health 23 (1997), pp. 243–256

[21]. K.G. Davis and C.A. Heaney, The relationship between psychosocial work characteristics and low back pain: underlying methodological issues. Clin Biomech 15 (2000), pp. 389–406

[22]. W.E. Hoogendoorn, M.N.M. van Poppel, P.M. Bongers, B.W. Koes and L.M. Bouter, Systematic review of psychosocial factors at work and private life as risk factors for back pain. Spine 25 (2000), pp. 2114–2125

[23]. W.S. Marras, K.G. Davis, C.A. Heaney, A.B. Maronitis and W.G. Allread, The influence of psychosocial stress, gender, and personality on mechanical loading of the lumbar spine. Spine 25 (2000), pp. 3045–3054

[24]. J.D. Cassidy, L.J. Carroll and P. Cote, The Saskatchewan health and back pain survey—the prevalence of low back pain and related disability in Saskatchewan adults. Spine 23 (1998), pp. 1860–1866

[25] M.C. Battie, T. Videman, L.E. Gibbons, L.D. Fisher, H. Manninen and K. Gill, Determinants of lumbar disc degeneration—a study relating lifetime exposures and magnetic resonance imaging findings in identical twins. Spine 20 (1995), pp. 2601–2612

[26]. S.J. Bigos, D.M. Spengler, N.A. Martin, J. Zeh, L. Fisher and A. Nachemson, Back injuries in industry: a retrospective study: III. Employee-related factors. Spine 11 (1986), pp. 252–256

[27]. W.S. Marras, K.G. Davis, C.A. Heaney, A.B. Maronitis and W.G. Allread, The influence of psychosocial stress, gender, and personality on mechanical loading of the lumbar spine. Spine 25 (2000), pp. 3045–3054

[28]. W.S. Marras, K.G. Davis and M. Jorgensen, Spine loading as a function of gender. Spine 27 (2002), pp. 2514–2520.

[29]. S.M. McGill, The biomechanics of low back injury: implications on current practice in industry and the clinic. J Biomech 30 (1997), pp. 465–475

[30]. Boshuizen HC, Verbeek JH, Broersen JP, et al. Do smokers get more back pain? Spine 1993;18:35-40

[31]. Jackson RP, Simmons EH, Stripinis D: Incidence and severity of back pain in adult idiopathic scoliosis, Spine 8:749, 1983.

[32]. Deyo RA, Bass JE. Lifestyle and low-back pain; the influence of smoking and obesity. Spine 1989;14: 501-506

[33]. Leino PI. Does leisure time physical activity prevent low back disorders? A prospective study of metal industry employees. Spine 1993;18:863-871

[34]. Deyo RA, Loeser JD, Bigos SJ. Herniated lumbar intervertebral disk. Ann Intern Med 1990;112:598-603.

[35]. Frymoyer JW. Can low back pain disability be prevented? Bailliere's Clin Rheumatol 1992;6:595-606

[36]. Spitzer WO. Scientific approach to the assessment and managemen of activity-related spinal disorders: a monograph for clinicians. Report of the Quebec Task Force on Spinal Disorders. Spine 1987;12(Suppl 7):1–59.

[37]. Fardon, David F. and; Milette, Pierre C.,M.D., Nomenclature and Classification of Lumbar Disc Pathology: Recommendations of the Combined Task Forces of the North American Spine Society, American Society of Spine Radiology, and American Society of Neuroradiology, Spine,Volume 26(5) 1 March 2001

[38]. Gordon, S.J., Yang, K.H., Mayer, P.J., Mace, A.H., Jr., Kish, V.L., and Radin, E.L., (1991). Mechanism of disc rupture. A preliminary report. Spine 16**,** 450-456.

[39]. Williams, P.L., and Warwick, R. (1980). "Gray's Anatomy." Churchill Livingstone, Edinburgh.

[40]. Bogduk, N., and Long, D.M., (1979). The anatomy of the so-called "articular nerves" and their relationship to facet denervation in the treatment of low-back pain. J. Neurosurg. 51**,** 172

[41]. Grieve, G.P. (1981). "Common vertebral joint problems." Churchill Livingstone, Edinburgh New York page 10

[42]. Van tudler 1997 low back pain , best practice and research clinical rheumatology, vol. 16, No 5, pp 761-775,2002)

[43]. White AA, Gordon SL. Synopsis: workshop on idiopathic low-back pain. Spine 1982;7:141–149.

[44]. Steven j. Atlas, MD, MPH,1 and Rachel A. Nardin, MD, Evaluation and Treatment ofLow Back Pain: an evidence-based approach to clinical care muscle nerve 27: 265–284, 2003

[45]. Bigos SJ, Bowyer OR, Braen GR, Brown K, Deyo R, Haldeman S, Hart JL, Johnson EW, Keller R, Kido D, Liang MH, Nelson RM, Nordin M, Owen BD, Pope MH, Schwartz RK, Stewart DH, Susman J, Triano JJ, Tripp LC, Turk DC, Watts C, Weinstein JN. Acute low back problems in adults. Clinical practice guideline no. 14. Rockville, MD: Department of Health and Human Services; 1994 (AHCPR publication no. 95-0642.

[46]. Burton AK, Waddell G. Clinical guidelines in the management of low back pain. Baillieres Clin Rheumatol 1998;12: 17–35.280 Low Back Pain MUSCLE & NERVE March 2003

[47]. Koes BW, van Tulder MW, Ostelo R, Burton AK, Waddell G.Clinical guidelines for the management of low back pain in primary care. Spine 2001;26:1504 –1514.

[48]. US Dept. of Health and Human Services Public Health Service Agency for Health Care and Policy Research (AHCPR). Acute Low Back Problems in Adults: Assessment and Treatment. AHCPR Publication No. 95-0643, December 1994.

[49]. Scavone JG, Latshaw RF, Rohrer GV. Use of lumbar spine films. Statistical evaluation at a university teaching hospital. JAMA 1981;246:1105-8.

[50]. Scavone JG, Latshaw RF, Weidner WA. Anteroposterior and lateral radiographs: an adequate lumbar spine examination. AJR Am J Roentgenol 1981; 136:715-7

[51]. Bell GR, Ross JS. Diagnosis of nerve root compression. Myelography, computed tomography, and MRI. Orthop Clin North Am 1992;23:405-19.

[52]. Bigos SJ. Acute low back problems in adults. Rockville, Md.: U.S. Department of Health and Human Services, Public Health Service, Agency for Health Care Policy and Research, 1994; AHCPR publication no. 95-0642

[53]. Inaoka M, Yamazaki Y, Hosono N, Tada K, Yonenobu K. Radiographic analysis of lumbar spine for low-back pain in the general population. Arch Orthop Trauma Surg 2000;120:380-5

[54]. Reinartz P, Schaffeldt J, Sabri O, Zimny M, Nowak B, Ostwald E, et al. Benign versus malignant osseous lesions in the lumbar vertebrae: differentiation by means of bone SPET. Eur J Nucl Med 2000;27:721-6

[55]. Savelli G, Chiti A, Grasselli G, Maccauro M, Rodari M, Bombardieri E. The role of bone SPET study in diagnosis of single vertebral metastases. Anticancer Res 2000;20:1115-20.

[56]. Boden SD, Davis DO, Dina TS, Patronas NJ, Wiesel SW. Abnormal magnetic-resonance scans of the lumbar spine in asymptomatic subjects. Aprospective investigation. J Bone Joint Surg Am 1990;72:403-8.

[57]. Boos N, Reider R, Schade V, Spratt KF, Semmer N, Aebi M. The diagnostic accuracy of magnetic resonance imaging, work perception, and psychosocial factors in identifying symptomatic disc herniations. Spine 1995;20:2613-25.

[58]. Wilbourn AJ, Aminoff MJ. AAEM minimonograph 32: the electrodiagnostic examination in patients with radiculopathies. American Association of Electrodiagnostic Medicine. Muscle Nerve 1998;21: 1612-31.

[59]. Bigos SJ, Battie MC, Spengler DM, Fisher LD, Fordyce WE, Hansson TH, et al. A prospective study of work perceptions and psychosocial factors affecting the report of back injury. Spine 1991;16:688.

[60]. H. Flor, T. Fydrich and D.C. Turk, Efficacy of multidisciplinary pain treatment centers: a meta-analytic review. Pain 49 (1992), pp. 221–230

[61]. J. Guzman, R. Esmail, K. Karjalainen, A. Malmivaara, E. Irvin and C. Bombardier, Multidisciplinary rehabilitation for chronic low back pain: systematic review. BMJ 322 (2001), pp. 1511–1516.

[62]. Hilde G, Hagen K, Jamtvedt G, et al. Stay active for acute, subacute and chronic low back pain (Protocol for a Cochrane Review). The Cochrane Library. Oxford:Update , 2000.

[63]. Deyo RA, Diehl AK, Rosenthal M. How many days of bed rest for acute low back pain? A randomized clinical trial. N Engl J Med 1986;315:1064-70

[64]. Van Tulder, M.W., Scholten, R.J., Koes, B.W., and Deyo, R.A., (2000). Nonsteroidal anti-inflammatory drugs for low back pain: a systematic review within the framework of the Cochrane Collaboration Back Review Group. Spine 25,2501-2513. Porter RW, Ralston SH. Pharmacological management of back pain syndromes. Drugs 1994;48:189-98.

[65]. Moskowitz RW. The appropriate use of NSAIDs in arthritic conditions. Am J Orthop 1996;25(9 Suppl):4-6.

[66]. Derebery V, Tullis W. Delayed recovery in the patient with a work compensable Waddell G, McIntosh A, Hutchinson A, et al. Low back pain evidence review. London: Royal College of General Practitioners, 1999

[67]. Tulder M, van, Koes B, Assendelft W, et al.Chronic low back pain: exercise therapy, multidisciplinary programms, NSAID's back schools and behavioural therapyeffective; traction not effective; results of systematic reviews. Ned Tijdschr Geneeskd 2000;144:1489-94.

[68]. Waddell G, Feder G, McIntosh G, Lewis M, Hutchinson A.: Low Back Pain Evidence Review.London, Royal College of General Practitioners 1996

[69]. Waddell G, Feder G, McIntosh G, Lewis M, Hutchinson A.: Low Back Pain Evidence Review.London, Royal College of General Practitioners 1999.

[70]. Waddell G, Feder G, Lewis M: Systematic reviews of bed rest and advice to stay active for acute low back pain.Br J Gen Pract 1997, 47:647-652.

[71]. Hagen KB, Hilde G, Jamtvedt G, Winnem MF: The cochrane review of advice to stay active as a single treatment for low back pain and sciatica.. Spine 2002, 27:1736-1741

[72]. Hagen KB, Hilde G, Jamtvedt G, Winnem MF: The Cochrane review of bed rest for acute low back pain and sciatica.
Spine 2000, 25:2932-293

[73]. Nelson BW, Carpenter DM, Dreisinger TE, Mitchell M, Kelly CE, Wegner JA. Can spinal surgery be prevented by aggressive strengthening exercises? A prospective study of cervical and lumbar patients. Arch Phys Med Rehabil 1999;80:20-5.

[74]. Philadelphia Panel Evidence-Based Clinical Practice Guidelines on Selected Rehabilitation Interventions for Low Back Pain; Physical Therapy Volume 81 Number 10 · October 2001

[75]. Malmivaara A, Hakkinen U, Aro T, et al. The treatment of acute low back pain: bed rest, exercises, or ordinary activity? N Engl J Med. 1995;332:351-355

[76]. Van Tulder MW, Koes BW, Assendelft WJ, et al. The Effectiveness of Conservative Treatment of Acute and Chronic Low Back Pain.. Amsterdam, the Netherlands: EMGO Institute, 1999

[77]. Riihimäki H. Hands up or back to work: Future challenges in epidemiologic research on musculoskeletal diseases. Scand J Work Environ Health. 1995;21:401-403

[78]. Nordin M, Campello M. Physical therapy exercises and the modalities: when, what and why? Neurol Clin. 1999;17:75-89

[79]. Van Tulder MW, Koes BW, Assendelft WJ, et al. The Effectiveness of Conservative Treatment of Acute and Chronic Low Back Pain. Amsterdam, the Netherlands: EMGO Institute, 1999

**[80]**. Tulder M, van, Cherkin D, Berman B, et al. Acupuncture for low back pain (Cochrane Review). The Cochrane Library. Oxford:2002

**[81]**. Tulder MW van, Cherkin DC, Berman B, Lao L, Koes B. The effectiveness of acupuncture in the management of acute and chronic low back pain. A systematic review within the framework of the Cochrane Collaboration Back Review Group. Spine 1999; 24(11): 1113–1123.

[82]. Assendelft WJJ, Morton SC, Yu El, et al. Spinal manipulative therapy for low back pain: a meta-analysis of effectiveness relative to other therapies. Ann Intern Med. 2003;138:871-881

[83]. Cherkin DC, Sherman KJ, Deyo RA, Shekelle PG. A review of the evidence for the effectiveness, safety, and cost of acupuncture, massage therapy, and spinal manipulation for back pain. Ann Intern Med. 2003;138:898-906

[84]. Koes BW, Assendelft WJ, van der Heijden GJ, Bouter LM, Knipschild PG: Spinal manipulation and mobilisation for back and neck pain: a blinded review. BMJ 1991, 303:1298-1303

[85]. Shekelle PG, Adams AH, Chassin MR, Hurwitz EL, Brook RH: Spinal manipulation for low-back pain. Ann Intern Med 1992, 117:590-598

[86]. Assendelft WJ, Koes BW, Knipschild PG, Bouter LM: The relationship between methodological quality and conclusions in reviews of spinal manipulation. JAMA 1995, 274:1942-1948.

[87]. van Tulder MW, Koes BW, Bouter LM. Conservative treatment of acute and chronic nonspecific low back pain. A systematic review of randomized controlled trials of the most common interventions. Spine 1997;22:2128 –2156.

[88]. Lurie JD, Birkmeyer NJ, Weinstein JN. Rates of advanced spinal imaging and spine surgery. Spine 2003; 28(6): 616-620.

[89] Riew KD, Yin Y, Gilula L et al. The effect of nerve-root injections on the need for operative treatment of lumbar radicular pain: a prospective, randomized, controlled, double-blinded study. JBJS 2000; 82-A(11): 1589-1593.

90. Simotas AC, Dorey FJ, Hansraj KK et al. Nonoperative treatment for lumbar spinal stenosis. Spine 2000; 25:197-209.

[91] Dubourg G, Rozenberg S, Fautrel B, et al. A pilot study on the recovery from paresis after lumbar disc herniation. Spine 2000; 27:1426-1432.

[92] MixterWJ, Barr JS. Rupture of the intervertebral disc with involvement of the spinal canal. N Engl J Med 1934; 211:210-215.

[93]. Caspar W. A new surgical procedure for lumbar disc herniation causing less tissue damage through microsurgical approach. Adv Neurosurg 1977; 4:74-79.

[94]. Williams RW. Microlumbar discectomy: A conservative surgical approach to the virgin herniated lumbar disc. Spine 1978; 3:175-182.

[95]. Hilton DL Microdiscectomy with minimally invasive tubular retractor. Outpatient Spinal Surgery 2002 171-195.

[96]. Wiltse LL, Bateman JG, Hutchinson RH, et al. The paraspinal sacro-spinalis approach to the lumbar spine. Clin Orthop 35:80, 1964.

[97]. Atlas SJ, Deyo RA, Keller RB, et al. The Maine lumbar spine study, part III. Spine 1996; 21:1787-1794.

[98]. Verbiest H. Results of surgical treatment of idiopathic developmental stenosis of the lumbar vertebral canal. A review of twenty- seven years of experience. JBJS [Br] 1977;59-B:181-8.

[99]. L'Heureux EA, Perra JH, Pinto MR, et al. Functional outcome analysis including preoperative and postoperative SF-36 for surgically treated adult isthmic spondylolisthesis. Spine 2003;28:1269-1274.

[100]. Shapiro GS, Gaku T, Ohenaba B-A. Results of surgical treatment of adult idiopathic scoliosis with low back pain and spinal stenosis: a study of long term clinical radiographic outcomes. Spine 2003; 28:358-363.

[101]. Fritzell P, Hagg O, Wessberg P, et al. Lumbar fusion versus nonsurgical treatment for chronic low back pain. Spine 2001 ;26:2521-2534.

[102] Gertzbein SD, Betz R, Clements D, et al. Semirigid instrumentation in the management of lumbar spinal conditions combined with circumferential fusion. Spine 1996; 21:1918-1925

[103]. Hee HT, Castro FP, Majd ME, et al. Anterior/posterior fusion versus transforaminal lumbar interbody fusion: analysis of complications and predictive factors. Journal of Spinal Disorders 2001 ; 14:533-540.

[104]. Boden SD, Kang J, Sandhu H, et al. Use of recombinant human bone morphogenetic protein-2 to achieve posterolateral lumbar spine fusion in humans: a prspective, randomized clinical pilot trial: 2002 Volvo award in clinical studies. Spine 2002 ; 27:2662-2673.

[105]. Lemaire JP, Skalli W, Lavaste F, et al. Intervertebral disc prosthesis: results and prospects for the year 2000. Clin Orthop Rel Res. 1997 ;337:64-76.

[106]. Bradford DS, Tribus CB. Vertebral column resection for the treatment of rigid coronal decompensation. Spine 1997;22:1590-1599.

[107]. Voos K, Boachie-Adjei O, Rawlins BA. Multiple vertebral osteotomies in the treatment of rigid adult spinal deformities. Spine 2001;26:526-533.

[108]. Thiranont N, Netrawichien P. Transpedicular decancellation closed wedge vertebral osteotomy for treatment of fixed flexion deformity of spine in ankylosing spondylitis. Spine 1993;18:2517-2522.

[109] Boden, SD Overview of the biology of lumbar spine fusion and principles for selecting a bone graft substitute. Spine 2002; 27(16S):S26-S31.

[110] . Lipson SJ. Spinal-fusion surgery-Advances and concerns. N Engl J Med 2004;350:643-645.

[111] . Deyo RA, Nachemson A, Mirza SK. Spinal-fusion surgery-The case for restraint. N Engl J Med 2004;350:722-726