Disc herniation may cause mechanical compression or inflammation. The normal intervertebral disc is avascular and aneural, except for the outer third of the annulus fibrosus. Maintaining a dynamic equilibrium between the synthesis and degradation of the extra cellular matrix is pivotal in intervertebral disc homeostasis, but degeneration of the extra cellular matrix occurs in patients with low back pain. Ingrowth of nociceptive neural fiber into deeper parts of the degenerated intervertebral disc is considered as one of the most widely accepted pathophysiological mechanisms related to chronic discogenic pain (426,427). Disc inflammation may promote axonal growth of afferent fibers innervating the disc by secreting pro-inflammatory mediators, such as tumor necrosis factor (TNF) and interleukin 6 (IL-6) as disc degeneration proceeds (428-432). In addition, nerve growth factor (NGF) is known to be a trophic growth factor for sympathetic and sensory nerve cells and to stimulate their differentiation, growth, maintenance, and survival (433). Further, it also has been shown that NGF also shows a hyperalgesic property by sensitizing and sprouting sensory nerve fibers in painful pathologic conditions (434,435). It has also been proposed that actions of NGF in painful intervertebral disc not only sensitize the sensory neuron, but also stimulate the peripheral nociceptive sensory neurons to grow into the intervertebral disc tissue where the extracellular matrix is degenerated in most cases (420). Inflammatory involvement has been extensively described (127,427-437). Consequently, the proposed etiologies and radiculitis may be summarized to include neural compression with dysfunction, vascular compromise, inflammation, and biochemical influences. Risbud and Shapiro (438) described the pathophysiology of disc degeneration and pain showing that in the inflammatory milieu neurogenic factors, in particular NGF and brain-derived neurotrophic factor (BDNF) generated by the disc and immune cells along with a multitude of other factors including TNF and IL-6. Phospholipase A-2 induces an expression of pain associated with cation channels in the dorsal root ganglia (DRG). Risbud and Shapiro (438) also showed that disc degeneration is characterized by 3 distinct, but overlapping phases in which cytokines play a central role with an initiating event resulting in phenotypic changes in production of cytokines and chemokines by both the nucleus pulposus and annulus fibrosus cells in the first phase, followed by further application of the inflammatory response by infiltrating immunocytes, as well as neovascularization and nerve ingrowth into the structurally deficient disc tissues in the second phase. In the final phase, nerve endings are sensitized and the modulation of DRG pain channel activities are altered by inflammatory mediators and neurotrophins resulting in pain. Further, Olmarker et al (439,440) also showed that spinal nerve roots, when compressed, exhibited interneural edema, deprived nutritional supply (439) and loss of amplitude of nerve conduction (440). In fact, these experimental findings were confirmed by Kuslich et al (130), who reported that noncompressed nerve roots did not reproduce the patients' pain when stimulated intraoperatively in awake surgical patients. Consequently, epidural procedures are suited in managing chronic lumbar disc herniation, with a natural history of radicular pain showing a favorable prognosis (55-65,70,71,75,76,436). Disc regression or resorption have been shown to be most favorable in disc sequestration (96%), disc extrusion (70%), and disc protrusion (40%); however, it was only 13% in disc bulging (413). Saal et al (420) reported that 90% of the patients showed good to excellent outcomes and 92% had achieved return to work status with nonoperative treatment of disc extrusions.

5.1.2 Lumbar Spinal Stenosis

Spinal stenosis is the result of abnormal narrowing of the spinal canal, lateral recess or the intervertebral foramina, resulting in pressure on the spinal cord or nerve roots (441,442). The two lower motion segments (L3-L4 and L4-L5) are the most commonly affected related to being more vulnerable to rotatory strains (443). Spinal stenosis may develop by a combination of chronic mechanical compression and cord instability. Along with ischemia, these factors may lead to neurologic symptoms or claudication (444-451). Some common causes of spinal stenosis include disc-related degeneration, osteophyte formation, herniated discs, arthritis, ligament or facet joint hypertrophy, epidural fat deposition, spinal tumors, and traumas (447-451). Adult degenerative scoliosis and degenerative spondylolisthesis may lead to instability and facet hypertrophy, resulting in stenosis (441-444).

Lumbar spinal stenosis is a common source of back and leg pain, and often involves weakness and numbness (441-443). More severe cases may present with neurological symptoms such as radiculopathy, myelopathy or cauda equina syndrome. Permanent numbness or paralysis can result from a long-term compression of spinal nerves or of the spinal cord. Lumbar spinal stenosis is the most common cause of spinal surgery in patients over 65 years old (441-444).