Lumbar instability may be defined as, extending beyond normal physiological movement limits, along with the disordered cooperative act of the disc, ligament and joints of the low back region and manifests itself with chronic low back pain.

Following the impairment of the systems working together and simultaneously, abnormal loading rather than over mobility is considered to be the real reason of low back pain.

Many patients complain from the posture of their body or the pain precipitated while doing their daily activities, as the main symptom (1).

The pain at the symptomatic mobile segment may be originating from several parts. Vertebral end plates, annulus of the disc, vertebral periosteum, facet joints or the surrounding protective soft tissue structures may be causing this pain (2). With the aging of the lumbar spine, these structures undergo degenerative changes such as the dehydration, shrinking of the disc space and the impairment of the facet joints. The addition of the rise in rigidity to these changes, augments the reduction in the sagittal balance and the disorder in the coronal-sagittal planes (3, 4). The common sense is that the degenerative changes of the disc increases the mobility of the spine except for the early stages (4). The deteriorations in the disc space, leads to abnormal conduction of the load to the end plates. Normal disc, is an homogenous jelly like structure composed of collagen and proteoglycans and distributes the applied load to the annulus and the cartilaginous end plates (5, 6). The significant detail at this point is that this feature enables the load to be distributed evenly in position changes.

This way, bending forward, backward and side movements becomes possible. Degeneration of a disc, impairs the general properties of the disc (6, 7).

The teared and sheared disc turns into a non-homogenous substance composed of collagen, liquid and gas (8). This situation renders the load conducted to the end plates uneven. The emerging of the pain while standing upright, arises from the failure of the degenerated disc to distribute the load properly. Meanwhile, the decline in the disc space increases the pressure inside the annulus and causes tears at the annular structures (9, 10).

Theories of Pain Genesis

As the disc undergoes degeneration, the harmony of the mobile segment fails. At this point, existence of two theories explaining the genesis of pain are noticed.

The loading theory; according to this concept, the load conducted to the impaired motion segment is no more distributed equally to the inner surface of the disc by the nucleus and the load transmission starts shifting away from the centre to pass through the annulus.

Instant axis of rotation, shifts from its normal position to upward or backward depending on the severity of the degeneration. At the onset of the degeneration, because of the progressively deteriorating and malfunctioning nucleus which is trying to function normally under the daily loadings and because of the developing tears now even in physiological movements in the annulus themselves or the succeeding fibrosis tissue, the annulus becomes more susceptible to pain.

The pain receiving mechanoreceptors located in the annulus and in the consequently growing fibrous tissue, act almost like pain generating stations by becoming relentlessly irritated (Figure 1).
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Sabri AYDIN M.D., Ali Fahir OZER M.D.

The intradiscal toxic liquid material of low pH value extruding through the torn layers of annulus, causes chemical irritation with pain by deteriorating the pH level of the tissues surrounding the nerve roots (Figure 2) (8,12-16). According to the other theory attempting to explain the pain, inside the highly degenerated disc the nucleus deforms and at the advancing stages, forms bead like solid structures composed of conglomerated collagen and fibrous tissue. Especially while upright, depending on the degeneration’s severity, when even just the body weight is applied onto the lumbar spine, these beads naturally exerts a force high above the normal limits, from their positions to the cartilaginous end plates which are rich in pain transmitting nerve endings (Figure 3). These points of pressure provoke too much pain. This way of pain generation is considered to be similar to the pain originated by the “stone in the shoe.” (6,11,15,16)

According to this theory; when trying to accomplish a fusion between the vertebrae but confined in a small area, not across the whole vertebral surface, even when a fusion is achieved, constant pressure applied to the cartilaginous end plates in a restricted area is considered to be the reason of the pain (16).

There are reasonable oppositions to both of these theories. For instance; everyday at the outpatient clinic we see many patients, with degenerative spondylolisthesis which could be regarded as an advanced stage of degeneration, not complaining of pain so much. Again, we notice that several patients highly presumed to suffer pain with severe degeneration and radiological findings, are not even aware of the changes in their spines.

However, it is a truth that these two theories undoubtedly take part at the genesis of low back pain syndrome.
References


