Understanding Causes of Low Back Pain

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WHEN TED Sorensen, the close confidante of John F. Kennedy, developed acute lower back pain during the 1956 political campaign, the future president recommended a series of steps to relieve the discomfort. Sorenson replied that he would follow the recommendations as soon as a "medical back expert" so advised, and Kennedy ruefully said, "Let me tell you, on the basis of 14 years of experience and three major back operations, there is no such thing!" Experienced surgeons who examine, treat, and operate on hundreds of patients with back pain will often echo the frustrations of the young president because low back pain is experienced by many people; yet, full knowledge of the spine is still wrapped in uncertainty, mystery, and enigma. However, there is a great fund of knowledge that we as orthopaedic surgeons and neurosurgeons have learned about this elusive, challenging problem.

The structure of the spine is composed of a strong ring of bone through which the spinal cord with its spinal nerves emanates. These bone blocks are connected by ligaments, which are in turn supported by thick muscles. To enhance further the stability of the back, as well as to promote motion, the back is composed of various curvatures with large muscle masses which contribute to our posture.¹

CAUSES OF LOW BACK PAIN

The chief cause of low back pain arises from the intervertebral disk, which actually serves as the shock absorber between the blocks of bone represented by the vertebrae. The disk may protrude or

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See Fig. 1.
bulge, it may extrude or slip out from its encircling thick ligament, or the disk may lose its water content and become a degenerative disk.

**DIFFERENTIAL DIAGNOSES**

**SACRO-ILIAC DISEASE**

The patient often complains of pain in the sacro-iliac area, and tenderness exists at that point. It is now generally accepted that the strong sacro-iliac ligaments allow for very little movement and that the pain here is often referred pain. At one time, a fusion of both sacro-iliac joints and the lumbo-sacral joint was thought to eliminate all motion at these affected joints and thereby eliminate the pain. Most experts now conclude that the sacro-iliac joint is not often involved in the mechanical low back problem and is not the source of the pain.

**LUMBAGO**

This term is frequently used to describe all manifestations of lumbar back pain. It is a convenient, wastebasket term but lacks scientific accuracy and cold, calculating precision.

**SCIATICA**

Sciatica is actually not a pathological, discrete entity but a rather physical symptom of an underlying process. One must determine the cause of this symptom. Is this a true sciatic radiation with involvement below the knee, or is it simply a diffuse spread of the lumbar pain into the buttocks region? Is the sciatica accompanied by true weakness of the toes or ankles? Are there any reflex changes?

**EPHYPYSISITIS OF THE SPINE**

This is a developmental defect that occurs during adolescence when the spine is growing. The growth process fails to complete itself and a fragment of bone does not unite to the main center of the vertebrae. This rarely causes back pain, but may cause posture changes characterized by the “adolescent round back” or the “dowager slouch.” The important legal point is to distinguish it from a compression fracture of the spine, which occurs as a result of a jack knife or flexion
fall from a height and compresses the vertebrae above against its neighboring vertebrae below.

THE SLIPPED INTERVERTEBRAL DISK

The protruded or the herniated disk causes nerve root pressure by pushing the nerve against the unyielding ring of bone surrounding it, forcing the typical sciatic pain into the buttocks, down the calf, and into the foot. The disk is allowed to extrude from its encircling thick ligament when the ligament becomes thin or breaks, permitting the disk substance to penetrate this protective barrier.\(^2\)

THE DEGENERATIVE INTERVERTEBRAL DISK

More and more evidence points to degenerative changes in the lumbar intervertebral disk as the source of most low back and sciatic pain. These disks have no blood supply after the age of twenty years. They are presumably nourished by the exchange of metabolites. The disks apparently have no nerve supply except to the anterior and posterior longitudinal ligaments and possibly the superficial layers of the annulus. Therefore, their capacity for repair and regeneration is very limited. Yet, these disks are subjected to severe strain and stress. It has been calculated that lifting a one hundred pound load places a stress of fifteen-hundred pounds on the lumbo-sacral disk space. How, therefore, does an insensitive disk lacking nerve endings become sensitive and cause low back pain? The process is as follows:

<table>
<thead>
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<th>Pathogenesis of Low Back Pain</th>
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<tr>
<td>Aging of Disk</td>
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<tr>
<td>Tears in Annulus (Ligament)</td>
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<tr>
<td>Inflammatory Tissue Invasion</td>
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<tr>
<td>Low Grade Inflammation within the Disk</td>
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<tr>
<td>PAIN</td>
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</tbody>
</table>

The presence of this new inflammatory tissue, particularly in the posterior annulus, changes an insentitive disk to an abnormal disk.

\(^2\) See Fig. 2.
Cracks occur in the cartilage plate. The disk then loses its elasticity and the ground is laid for the *acute low back syndrome* and *degenerative disk disease*.

**DEGENERATIVE DISK AND CAUSE OF PAIN**

The degenerative disk can result in pain through three different avenues:

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<td>I</td>
<td>II</td>
<td>III</td>
</tr>
<tr>
<td>Unstable Spine</td>
<td>Ligamentous strain and Muscle spasm</td>
<td>“Pinched” nerve</td>
</tr>
</tbody>
</table>

Disk degeneration results in mechanical changes in the low back. The spinal disk serves as a shock absorber and checks movement between adjacent spinal segments. After the annulus has lost its elasticity, excessive motion and abnormal posture is permitted, and the so-called “unstable low back” occurs. Obesity, pregnancy with resultant relaxed abdominal muscles, frequent lifting of objects above waist level, prolonged stooping, sleeping face down, all contribute to hyperextension of the lumbar spine. This in turn allows for the *posterior joint subluxation* due to the loss of the elasticity of the spinal disk. These changes frequently are accompanied by articular degenerative changes in the facet joints permitting telescoping of vertebrae and forward displacement (pseudospondylolisthesis) or retro-displacement. This abnormal motion is best seen in the lateral roentgen view of the lumbar spine taken in maximum flexion and maximum extension with the patient standing.

With resultant instability of the lumbar spine, the spinal nerves may be stretched causing inflammation and adhesions to surrounding bone structures and soft tissues. This may result in incarceration of the nerve root between the superior facet of the vertebrae below and the surface of the lamina above.

New and fertile areas lie ahead in the research fields in investigation of the collagen content and changes associated with the aging process. The hygroscopic properties of the disk substance have been

3. *See Fig. 3.*
4. *See Fig. 4.*
suggested as accompanying injury, possibly as the result of depolymerisation of the mucopolysaccharide, as suggested by Mitchell et al.

Thus, it becomes evident that much of the disorder of the low back pain is preceded by *degeneration of the spinal disk*. This allows for increased mobility of the facet joints of the vertebral column. Three causes of pain may then be manifested: (1) The sacrospinalis muscle (extensor muscle of the spine) goes into *spasm and ligamentous stress* occurs to prevent the abnormal motion; (2) *arthritic changes* develop due to the stress and strain at the facet joints; and (3) *neural irritation* due to stretching or incarceration of the nerve root ensues. This pain may be directed along any or all of these avenues.

**TREATMENT**

It becomes evident that a focal point of therapy is to limit the *abnormal back excursions*. Avoid lifting heavy objects above the waist; prevent postural strain of prolonged stooping, and eschew an obese, pendulous abdomen. A corset or back support aids in limiting this excess motion. Rest in the acute stage is mandatory. As the patient ages beyond fifty years, the anatomical motion of the lumbar spine is gradually lost and back pain tends to diminish in frequency. This is one of the few compensations of middle age.

Back and hip exercises to keep strong muscle tone and stretch out hamstring contracture are beneficial. It has been suggested that when osteopathic manipulations are beneficial, it is because of this factor as well as a reduction of the facet joints which may have been locked in an abnormal position causing pain. A strong mattress and avoidance of sleeping on the abdomen are indicated to prevent abnormal pressure on the posterior vertebral elements.

Rest, exercises, and guarded activity will usually overcome the acute low back pain. However, surgery may have to be considered for repeated attacks causing a man to lose employment at frequent intervals. Spinal fusion, laminectomy, and nerve root decompression are procedures that may be required if conservative measures are ineffective.

**LEGAL IMPLICATION**

Man has paid the price with low back pain for standing on his own
two feet when he decided to turn away from quadriped existence. Any legal inquiry into this area must take into consideration the phylogenetic evolution as well as the biomechanical, anatomical, and physiological aspects of the spine. With this knowledge, the lawyer will be able to understand, protect, and defend the rights of his client who has incurred injury to his lumbar spine.
Figure 1. Influence of spinal muscles on postural curves.
Figure 2. "Slipped disk" showing compression of the nerve roof after the disk has extruded. (after Abbott).

Figure 3. Intervertebral disk compression forces. (after Cailliet).
Figure 4. Degenerative disk disease with loss of disk space.