Spinal Deformities
The Comprehensive Text

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Thieme
New York · Stuttgart

Thieme Medical Publishers, Inc.
333 Seventh Ave.
New York, NY 10001
Consulting Editor: Esther Gumpert
Assistant Editor: Owen Zurhellen
Director, Production and Manufacturing: Anne Vinnicombe
Production Editor: Becky Dille
Marketing Director: Phyllis Gold
Sales Manager: Ross Lumpkin
Chief Financial Officer: Peter van Woerden
President: Brian D. Scanlan
Compositor: Thomson Press (India) Limited
Printer: Maple-Vail Book Manufacturing Group

Library of Congress Cataloging-in-Publication Data

Spinal deformities : the comprehensive text / [edited by] Ronald L. DeWald
p.; cm.
Includes bibliographical references.
ISBN 1-58890-089-4 (alk. paper) – ISBN 3131305517 (alk. paper)
1. Spine–Abnormalities. 2. Spine–Diseases. I. Dewald, Ronald L., 1934-
[DNLM: 1. Spinal Diseases. 2. Spinal Injuries. 3. Spine–surgery. WE 725 S7548 2003]
RD768 .S645 2003
617.5'6–dc21 2002075002

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Printed in the United States of America

5 4 3 2 1

TMP ISBN 1-58890-089-4
GTV ISBN 3 13 130551 7

Biomechanics of the Intervertebral Disk

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CHAPTER 16

The intervertebral disk constitutes 20 to 33% of the entire height of the vertebral column. It forms the primary articulation between the vertebral bodies and is the major constraint to motion of the functional spinal unit (FSU). It is comprised of three distinct elements: the nucleus pulposus, the anulus fibrosus, and the cartilaginous end plates.

The nucleus pulposus consists of a random network of collagen and proteoglycans, which bind water. The water content ranges from 70 to 90%; it is highest at birth and decreasing with age. The lumbar nucleus occupies 30 to 50% of the total disk area.

The anulus fibrosus forms the outer boundary of the intervertebral disk. The structure is composed of fibrous tissue in concentric laminated bands. The fibers are arranged in a helicoid manner. Each layer is made of collagen fibers oriented vertically at the periphery but becoming progressively more oblique with each underlying layer. The fibers in adjacent sheets run at approximately 30 degrees to each other. The anulus fibers are attached to the cartilaginous end plates in the inner one, and in the more peripheral zone they attach directly into the vertebral bodies (Sharpey's fibers). Although the disk contains both types I and II collagen, there is a linear change in the proportion of type I in the outer anulus to type II in the inner sections. Type II fibers are more hydrophilic than type I and, therefore, contain about 25% more water than the latter.

The end plate is composed of hyaline cartilage that separates the other two components of the disk from the vertebral body. Distinctly different cell types have been demonstrated in the nucleus, transition zone, and anulus with respect to morphology, cytoskeletal arrangement, and biomechanical properties. Cells are of both mesenchymal and notochordal origins. These cells have been described as chondrocyte-like or fibrochondrocyte-like, and there are variations of each cell type in different zones of the disk. Studies have demonstrated that proteoglycan and collagen synthesis rates are the highest in the nucleus pulposus and the transitional zone between the nucleus and anulus.[1] As these are the regions of lowest cell density, this is evidence for the significant biosynthetic potential for these cells. Biomechanical factors may also influence metabolic activity of disk cells. Low magnitudes of hydrostatic pressure have been shown to increase proteoglycan or collagen synthesis, whereas higher pressures have been found to inhibit synthesis in all disk cells. Higher pressures have also been shown to induce decreases in proteoglycan content and aggrecan gene expression in the nucleus, as well as decreases in type II collagen gene expression and content in the nucleus and transitional zone. Similarly, high compressive loads in vitro have been shown to decrease production of proteoglycans and collagen in the entire disk, although high oscillating frequencies inhibit nuclear proteoglycan synthesis.

The human spine is a complex columnar structure. In performing biomechanical testing of the spine, applied loads or motions to the entire spine may lead to erroneous conclusions at the individual spinal component level. Biomechanical behavior of the spine is commonly tested over the smallest functional unit, the FSU, consisting of two adjacent vertebrae and the interconnecting soft tissue, devoid of musculature. The motion of an FSU is the right-handed, rectangular Cartesian coordinate system. This system consists of three straight intersecting axes that are mutually perpendicular (Fig. 16–1).

Compression testing is performed with a test specimen consisting of a lumbar disk with anterior and posterior longitudinal ligaments intact and a thin slice of bone on either side. The specimen is placed in a universal testing machine (MTS, Instron) that can apply large, controlled compressive loads. The load applied and the specimen deformation are measured continuously. Load-deformation curves can then be plotted to determine functional characteristics of the tested tissues.

Virgin[2] showed that compressive loads of sufficient magnitude to induce permanent deformations in intervertebral disks produced no herniations in the nucleus pulposus. This was true even when a longitudinal incision was made in the posterolateral part of the anulus fibrosus and compressive loading showed little change in elastic properties. Brown and colleagues[3] tested lumbar FSUs with the PLL removed in compression and found that the first component to fail in such a construct was the vertebra, with fracture of the end plates. No failure of the intervertebral disk was ever seen. They observed that there were no differences between the vertebrae with normal and degenerated disks. With central compressive loading, bulging was noted, but not in any particular direction. Clinically, posterolateral disk herniations, then, probably result from a loading mechanism other than purely compressive stress.

The intervertebral disk is rarely exposed to purely tensile loads, but under certain physiologic situations the disk anulus is exposed to tensile stresses. In flexion, the instantaneous axis of rotation passes through the middle of the disk. Thus, the posterior aspect of the disk is subjected to tensile stresses in flexion. In extension the opposite is true, the anterior disk experiences tension. In lateral bending, tensile stresses are produced in the convex side of the curve. Brown et al[3] divided disks into sections and tested each to tensile failure. Load-deformation curves showed variation between strength maps of the two disks, with anterior and posterior regions of the disk much stronger than the lateral region and the central region.

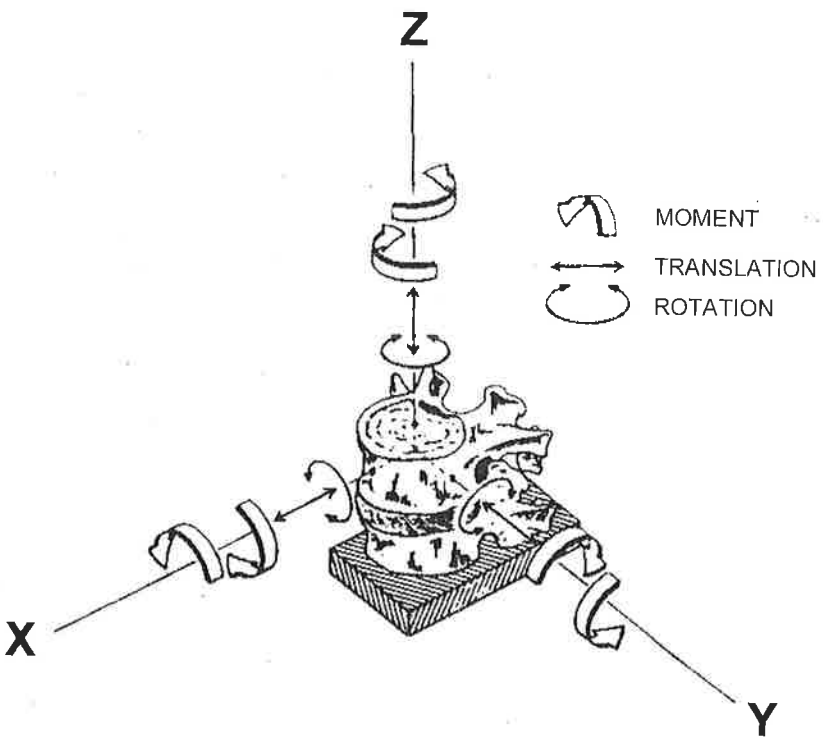

Figure 16–1 Standard coordinate system as described by the Scoliosis Research Society.

The results of these stiffness and strength tests indicate that the disk is an anisotropic structure, which means that its material properties are dependent of the direction of loading.

During normal physiologic motions, bulging of the disk occurs with bending motions. The disk bulges anteriorly during flexion, posteriorly during extension, and toward the concavity of the spinal curve during lateral bending. The disk curves inward on the opposite side (convexity).

Original work by Farfan[4] subjected a vertebra-disk-vertebra construct (including posterior elements) to torsional loading around a fixed axis. Torque was applied and the angle of deformation was recorded until failure. The load deformation curves were found to be sigmoid curves with an initial zone where small loads produced deformation, an intermediate zone where there was a linear relationship between applied torque and angular deformation, and a final zone where continued torque produced failure. Farfan and colleagues[5] also tested degenerated and nondegenerated spinal segments in a similar manner. They found that the average failure torque for the nondegenerated disks was 25% higher than that for the degenerated disks.

It has been shown that the anterior structures of the spine are responsible for rotational stability.[6,7] Haher et al[7] demonstrated that violation or destruction of the anterior column resulted in inability of the spine to resist rotation. These studies showed that 90% of torsional rigidity was lost with removal of the anterior two-thirds of the intervertebral disk.

It has been shown that although torsional testing subjects spinal specimens to shear stresses, these stresses are not uniformly distributed, being larger at the periphery and lower in the center. Because torsional experiments do not provide a good model for direct shear of the disk, experiments were carried out to study disks loaded in direct shear. The shear stiffness in the horizontal plane was found to be about 260 N/mm.[8] This high value shows that a large force is required to cause an abnormal horizontal displacement of a normal vertebral disk unit. This means that it is relatively rare for the anulus to fail clinically because of pure shear loading.

Range of motion usually is measured on the basis of a brief force application. However, if a force is applied to a collagenous viscoelastic structure for a prolonged period, further movement can be detected. This movement, creep, is small in amplitude and occurs slowly. Markolf and Morris[9] demonstrated that higher loads produced greater deformation and faster rates of creep. Kazarian performed compression creep tests on normal and degenerated disks and noted that nondegenerated disks creep slowly and reach their final deformation value after considerable time when compared to degenerated disks.[10] Thus, the process of degeneration makes the disks less viscoelastic and implies that as a disk degenerates it loses the capability to attenuate shocks and to distribute the load uniformly over the entire end plate.

Time-dependent (viscoelastic) intervertebral changes have been demonstrated. It is currently understood that an increase in height occurs if there is a reduction in the overall disk compressive pressure; the reduced intradiskal hydrostatic pressure allows for water to flow into the intervertebral disk. The reverse is true if intradiskal pressure is increased. Reported diurnal changes in the overall height of individuals ranged

from 6.3 to 19.3 mm.[11] The average person is 1% shorter in the evening than in the morning; children are 2% shorter in the evening; the elderly are 0.5% shorter in the evening. Fifty percent of the total length change occurs during the first 2 hours in the upright position. Thus, for most, the first 2 hours out of bed in the morning are critical for disk metabolism.

The nucleus pulposus acts to hydrostatically pressurize the disk. Pressure is present even at rest. The disk pressure is about 1.5 times the compressive load divided by the cross-sectional area. The actual loads to which a disk is subjected can be determined by using an in vivo measurement of disk pressure. Lumbar intradiskal pressure is lowest in the recumbent posture, higher in a standing posture, and highest in unsupported sitting. The pressure within the disk is directly related to the axial compression applied to the disk. Approximately 60% of the body mass is located above the L3–L4 level. In vivo, however, the load acting on the disk between L3 and L4 is related to both the body weight above the disk and the additional forces produced by the stabilizing muscles. In a sitting and standing position with 20 degrees of flexion, the load at the disk at the L3–L4 level is 200% body weight. It increases to 300% with the addition of 20 kg of weight, held in the hands. This data has been reproduced several times since Nachemson's[12] original studies. Disk pressure has been shown to be uniform throughout the nucleus and inner anulus. Small peaks of vertical compressive stress exist in the outer anulus. Damage to the vertebral body, particularly in the vicinity of the end plate, causes decompression of the adjacent nucleus pulposus, which produces a high compressive stress in the posterior anulus. Recent work by Wilke et al demonstrated that after 7 hours in a lying position, intradiskal pressure increased to 240% of the pressure before lying down.[13] Presumably, intradiskal pressure increased because of rehydration of the disk. In vivo tests have shown decreased pressures in painful disks.

Asymmetric and cyclic loading in combined lateral bend, compression, and flexion are risk factors for disk herniation. The lumbar motion segment can resist a combination of bending moment and shear force of 156 Nm and 620 N, respectively, before complete disruption occurs. This is much less than the failure load in compression.

Sudden disk prolapse has been produced in vitro by application of specific loading patterns.[14] A two-vertebrae laminectomized specimen was placed in a laterally bent and hyperflexed position, placing the posterolateral disk in tension. Application of a sudden compressive force resulted in disk prolapse. Disks in this experiment were found to be from lower lumbar levels (L4–L5 or L5–S1), in the 40 to 49 year age group, with disk degeneration evident. This implicates both the loading conditions and a predisposition of certain disks to sudden prolapse. Similar in vitro loading of FSUs in flexion, lateral bending, and varying compression with a cyclic frequency was tested in an attempt to simulate gradual disk prolapse.[15] A very small number of specimens were found to be prolapsed by this method, suggesting that mechanisms in addition to flexion, lateral bending, and compression loading are responsible for gradual intervertebral disk prolapse.

Mechanical Properties of the Disks in Vivo and in Vitro

Some caution should be exercised in evaluating data from biomechanical testing of spinal specimens, especially the intervertebral disks. As was stated previously, considerable variation exists in the biochemical makeup of intervertebral disks. The inner anulus is composed of a higher percentage of type II collagen, which is much more hydrophilic, leading to a higher water content. Further, with age, water content of the intervertebral disk decreases. Objectively, disk degeneration is, in part, a measure of the amount of water content in an intervertebral disk. Viscoelastic principles suggest that loading history plays an important role in fully describing the biomechanical properties of the disk.

Postmortem changes in the water content of intervertebral disks may have an important effect on the mechanical properties of the spine. Prolonged unloading after death causes the hydrophilic disks to swell with tissue fluid, and the resulting increase in disk height profoundly affects spinal mechanics. The apophyseal joints resist less of the axial compressive force on the spine and the disk resists bending more strongly. Loading history has become so important that it has become standard practice to expose disk specimens to sustained compressive creep loading prior to testing to bring the disk into what is felt to be a physiologic range of hydration. One protocol exposes all specimens to a preliminary creep load of 300 N for a minimum of 15 minutes; to simulate disk hydration late in the day, 1 to 2 kN is applied to specimens for 2 to 6 hours.[16]

Spinal tissues become stiffer the more they are deformed, and the distribution of applied loads will change with increasing load. Thus, it is not possible to infer mechanical behavior at high load levels based on experiments performed at low load levels. Also, spinal tissues become stiffer at faster loading rates, and similarly, mechanical behavior at fast loading rates cannot be predicted from experiments using slow or static loading techniques.

Biochemistry/Anatomical Etiologic Considerations of Disk as Pain Generator

Significant debate exists as to if and how the intervertebral disk might produce back pain. The clinical presentation of acute lumbar radiculopathy is most often attributed to a compressed lumbar nerve root by a herniated intervertebral disk. Some patients with large herniations have no radicular symptoms, and, in contrast, some patients with no evidence of disk herniation have severe radicular symptoms. Direct mechanical compression of a nerve root plays a role, but this may only partly explain the pathophysiology of radicular pain. Recent work has implicated local innervation patterns and local release of biochemical mediators of inflammation as possible important factors in the production of radiculopathy.[17]

It is currently accepted that fine nerve endings reside on the outer anulus and that these nerve endings contribute to back pain in a variety of clinical conditions. The intervertebral disk receives its innervation from the sinuvertebral nerve, which supplies the posterior longitudinal ligament, ventral dura, and posterior and posterolateral anulus, and from the gray ramus communicans, which innervates the lateral and anterior aspects of the disk. Considerable overlap exists in the ascending and descending branches of these nerves, with branches from a single trunk supplying one or two levels higher or lower. When disk degeneration occurs at one level, pain may be referred to adjacent levels by this overlap, making clear localization of the painful level difficult. Further, the PLL is the most richly innervated of all of the spinal ligaments. The PLL and posterior anulus are tightly connected, causing localized back pain from not only disk herniation itself but from distortion of the PLL.

The close anatomical proximity of the dorsal root ganglion (DRG) to the intervertebral disk is another indirect mechanism of intradiskal pathology to produce pain. The DRG is very sensitive to irritation, and elaboration of irritating neuropeptides and prostaglandins may result in sensitization or stimulation of DRG neurons, producing radicular pain and sciatica.

Production of inflammatory mediators by degenerative or herniated disks is postulated to be an inciting factor in the pain cycle. Several studies have shown increased production of inflammatory mediators nitric oxide, phospholipase A2, prostaglandin E2, metalloproteinases, and various cytokines in herniated lumbar disks.[17] Autologous nucleus pulposus can induce pronounced histologic and functional changes in spinal nerves via these mediators when applied epidurally. Phospholipase A2 is the enzyme responsible for the liberation of arachidonic acid from cell membranes at the site of inflammation and is considered to be the limiting agent in the production of prostaglandins and leukotrienes, which are powerful mediators of inflammation. The liberation of prostaglandins and leukotrienes has been shown to cause sensitization of nociceptors; it also enhances pain generation, suggesting a possible mechanism for the sciatica in lumbar disk herniation. This mechanism also might explain the positive effect of epidural and systemic steroids in the management of pain after surgery for lumbar disk herniation. Corticosteroids inhibit the activation of phospholipase A2; thus, the production of prostaglandins and leukotrienes is blocked.

Additionally, progressive deterioration of the disk results in increasing mechanical instability, which in turn leads to more pain whenever the patient tries to become active. Decreased annular strength may result in bulging of the anulus under axial loading, which puts the outer annular fibers under increased tension and subjects nerve fibers in these layers to abnormal stretch and deformation.

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