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BIOMECHANICS OF LUMBAR SPINE AND LUMBAR DISC

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A better comprehension of the biomechanics of spine, provides a better understanding of the external factors causing spinal disorders. This situation, enables the best method to be applied to the patient both at the diagnosis and the treatment stage.

The spine; stands on four points (cervical, thoracic, lumbar and sacral) in the coronal plane and on one straight line in the vertical plane. In the sagittal plane, it contains regional variations while in the cervical and lumbar regions, it tends to have a kyphotic curve. In this chapter, the lumbar region and the biomechanical properties of the lumbar region will be emphasized.

The morphological structure of the lumbar spine can be evaluated in two groups as static and dynamic. *The static structure* consists of the vertebral body, the pedicle, the facet joints, the lamina and the transverse process.

Meanwhile, the dynamic structure consists of the intervertebral disc, the anterior longitudinal ligament (ALL), the posterior longitudinal ligament (PLL), the supraspinous ligament (SSL), the interspinous ligament (ISL), the ligamentum flavum (LF), the capsular ligament (CL) and the paravertebral muscles. In the sagittal plane, the lumbar vertebrae are aligned in a curve named "lordosis". In an upright person, the gravitational line of the force affecting the whole spine (drawn from C7 to S2, *the plumb line*), passes through the spinous processes in the coronal plane while passing through the pedicles of the lumbar vertebrae in the sagittal plane, for the lumbar region of the spine forms a lordosis angle of approximately 30°. In daily life when standing up, this

angle increases nearly 10-15 degrees while decreasing 20-35 degrees when we sit straight⁽¹⁾.

An increase in the lumbar lordosis has some disadvantages as; the deterioration of the supply of the posterior anulus, the narrowing of the spinal canal, the increment of the load transmitted through the facet joints and the posterior anulus. Lumbar lordosis, gives rise to relaxation of the muscles and some ligaments during motion and supports these structures to absorb energy. On the other hand, decrement of the lordosis; decreases the length of the motion arm of especially the posterior muscle group, to the lumbar spinal column. With the increment of the lumbar lordosis, a fall in the hydrostatic pressure within the disc is possible but this effect vanishes as the loading pressure increases along with the lordosis⁽²⁾.

Regarding the motion range of the lumbar area, the instant axis of rotation (IAR) is located on the anterior side of the disc space during the flexion movement and on the posterior side, at the facet joint level during the extension movement.

The lateral bending movement when it is to the right side, shifts the IAR to the left side in the coronal plane and when it is to the left side shifts the IAR to the right side in the coronal plane.

During the axial rotation (torsion) movements (right or left), IAR stays inside the disc space.

Thus the instant axis of rotation moves dynamically during the motion rather than being a static point. Especially in situations like trauma where the stabilization of the column deteriorates, the location of the IAR changes and in situations where

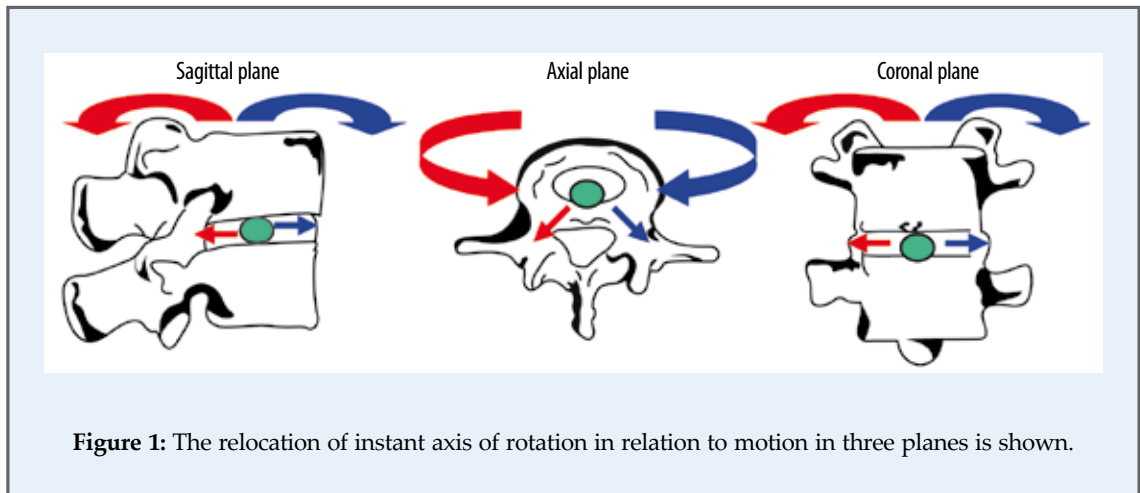


Figure 1: The relocation of instant axis of rotation in relation to motion in three planes is shown.

instability emerges, different surgical fixation techniques are applied to restore the IAR.

Combined movements are frequent in the lumbar area. The flexion-extension movement, while it is 12-14 degrees at the L1 level, rises up to 18 degrees at the L5 level.

The lateral bending is a bit more stable. At each segment it is approximately 7-9 degrees. The axial rotation is about 3 degrees at each segment (Table 1).

The contribution of the lumbar spinal column to the whole spinal column's motion is 95 degrees in flexion-extension while all spine's is 250 degrees; 40 degrees out of 150 in lateral bending and 18 degrees out of 100 in axial rotation⁽³⁾.

a) The Vertebral Body

When the geometrical properties of the vertebral bodies in three different planes are analysed, the

Segment	Flexion + Extension	Single-sided lateral bending	Single-sided axial rotation
L1-L2	12	6	2
L2-L3	14	6	2
L3-L4	15	8	2
L4-L6	16	6	2
L5-S1	17	3	1

Table 1: Lomber range of motion

following features are recognized⁽⁴⁾ (Figure 2): In the axial plane, except the L5 vertebra, they are kidney shaped. The fifth lumbar (L5) vertebral body has somewhat an oval shape. When the width of the vertebral body is examined in this plane, it is noticed that the anterior-posterior width is narrower than the left-right width. While the width of the lumbar vertebral body in the axial plane at the L1 level is 35-40 mm, it extends up to 50-55 mm at the L5 level. The axial anterior-posterior length is approximately 25-30 mm. The stated measurements display significance for assessing the length of the screws that may be used in the surgeries from anterior or posterior to this region. At the neighbouring edge of the vertebral body with the intervertebral disc, the *end plate* exists. The examination of the width and the depth of the end plate reveals that while the width increases approximately 14% from L1 to L5, the depth stays stable. The ratio of the width of the end plate (superior and inferior) to its depth, increases from L1 towards L5.

While the ratio increases 12% from L1 to L5 for the superior end plate, the increment reaches up to 21% for the inferior end plate at the L5 level. As for the measurement of the surface area of the end plates, from L1 to L5, the value increases 17% for the superior and 9% for the inferior end plates. The surface area of the inferior end plate is largest at the L3 level. In the coronary plane,

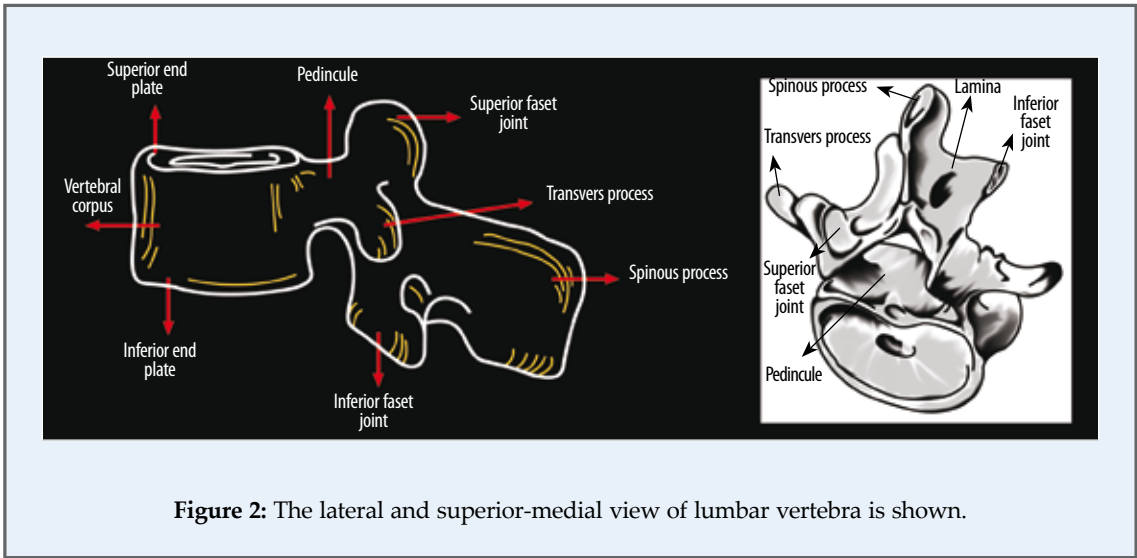


Figure 2: The lateral and superior-medial view of lumbar vertebra is shown.

we may notice that the lateral sides of the vertebral body are concave; and that in the sagittal plane, the height of the anterior side of the vertebral body is higher than the posterior side. This height inequality is approximately 20-30 mm and plays a part in the lumbar lordosis angle.

The withstanding of the vertebra against the compressive and distractive forces is important. Panjabi⁽⁴⁾ has stated that the highest resistance against the static forces is present at the L4 vertebral body. Lumbar vertebral bodies' resistances against the compressive forces are between a range of 5500-8000 N⁽⁴⁾ (Figure 3). This resistance is achieved by the cortex which has a harder structure. Spongiform bone has a lower resistance against the loading forces. But the more notable thing is that the resistance alters depending on the location inside the vertebral body. The highest resistance is found in the spongiform bone located centrally⁽⁵⁾ (Figure 4). With the distractive forces applied, the weakest site is found at the vertebra-end plate junction. For example, in

Chance type injuries (flexion-distraction) the fracture line usually takes place here (Figure 5). Hanson presented in 1987 that the resistance of the vertebral body decreases under continual pressure. Approximately 5000 N pressure causes 50% fall in the vertebral body resistance.

b) The Pedicle

The significant anatomical properties of the lumbar area pedicles can be summarized as: The width of the cortex of the medial and inferior side of the pedicle is thicker than the cortex of the lateral and the

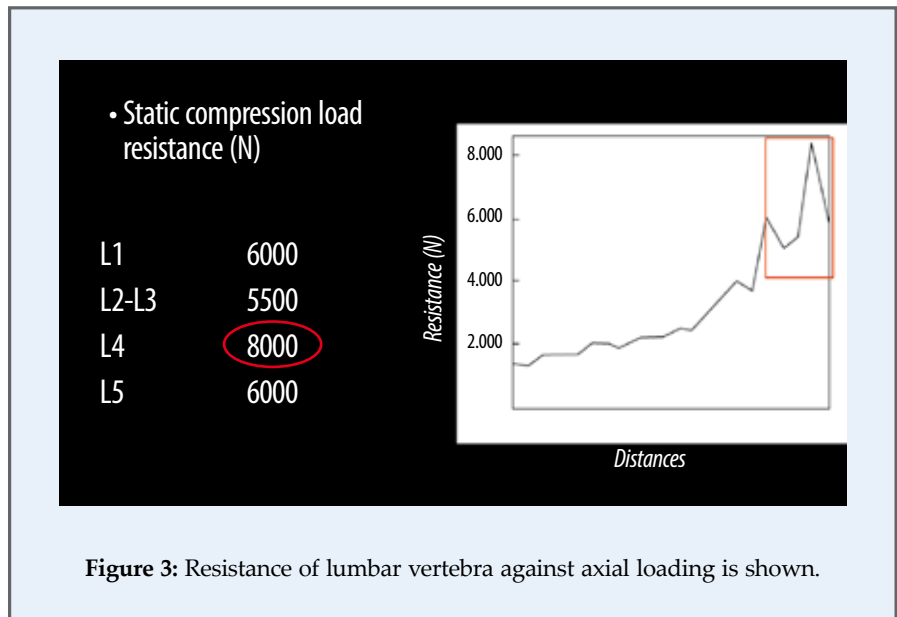


Figure 3: Resistance of lumbar vertebra against axial loading is shown.

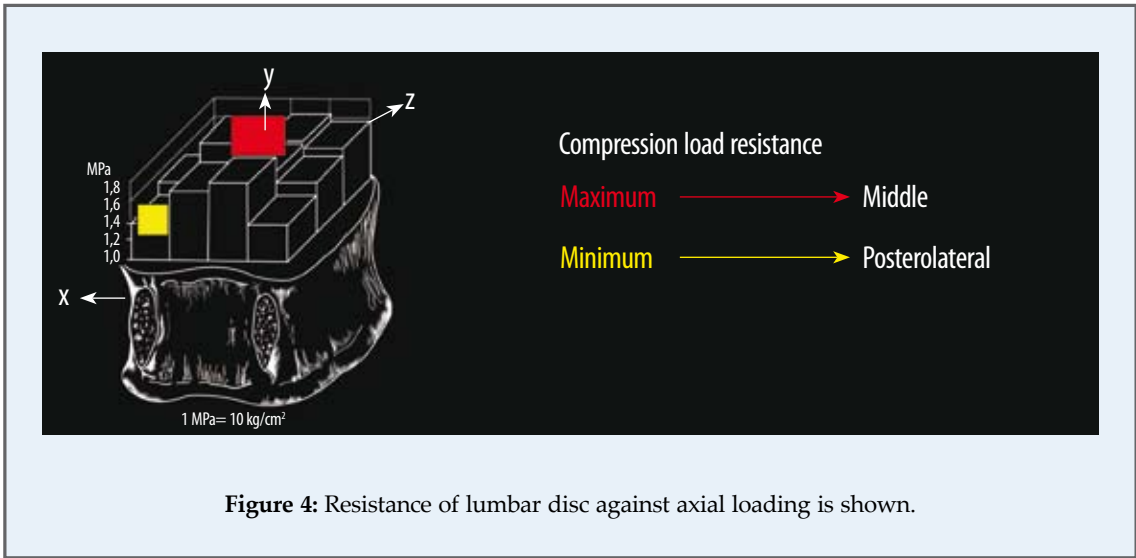


Figure 4: Resistance of lumbar disc against axial loading is shown.



Figure 5: Flexion-distraction injury at L3 level on T2 weighted sagittal MR images is shown.

superior side. The superior border of the pedicle is on the same level as the vertebral body. While the superior border is straight, the inferior border has a more concave shape.

The pedicle height is usually 15 mm in adults. The pedicle height is measured 15.9 mm at L1 level and 19.6 mm at L5 level. The width of the pedicle in the transverse plane ranges between 8.6 mm at L1 to 18.9 mm at L5 level⁽⁴⁾. The median orientation of the pedicles in the transverse plane increases 5 degrees per each level beyond T12 level.

The transverse plane orientation of the pedicle is about 15-30 degrees at the last lumbar vertebra, L5 level. At the pedicle screw applications, the medial angulation should be less at the upper lumbar levels compared to the lower levels. The applying of the screws to the vertebral body with an angle, makes it harder for the screw to be pulled out and the bone structure of the pedicle provides about the 60% of the stability of the screw⁽⁶⁾.

c) The Facet Joint

The facet joints are sagittally oriented in the lumbar area (Figure 6). This means that the facet joints allows the flexion-extension motion but restricts the axial rotation motion. The transverse plane view reveals that the superior facet joint has a concave and the inferior facet joint has a convex surface. The inferior facet joint of L5 at the lumbosacral joint is rather cor-

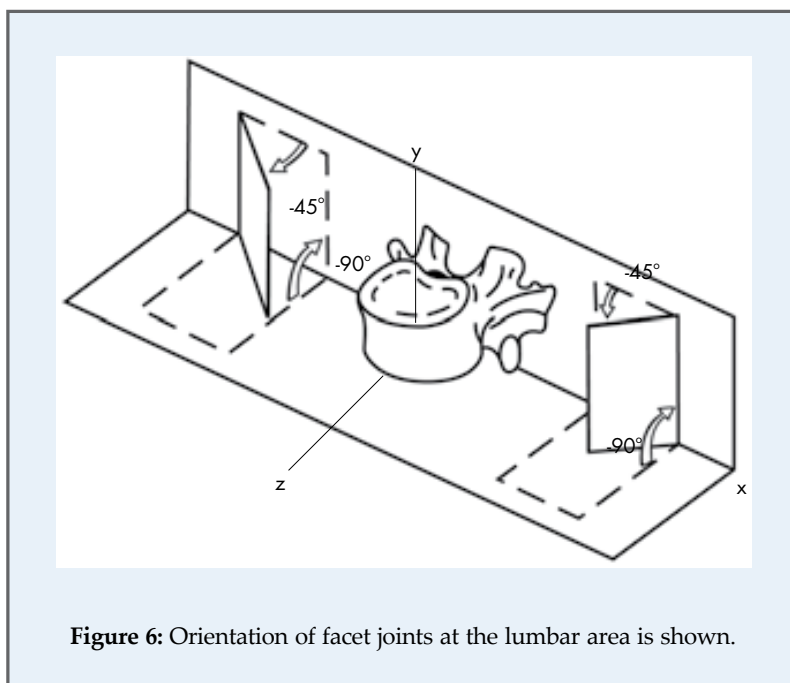


Figure 6: Orientation of facet joints at the lumbar area is shown.

of the anterior elements, with physiological loading. They have stated that the anterior translation increases 101.7% and posterior translation increases 117.1% after the resection of the facet joints. In various mechanical studies it's been presented that facet joints limit the rotation in extension by impact, similarly limit the movement in rotation by unilateral impact and exhibit limiting effect with the contributing regional ligaments in flexion (11-13). At the lumbar area, the width of the superior and the inferior facet is approximately 13 mm, the height is 15 mm. These values do not show significant differences between the left and the right

onally oriented. This structure displays a restrictive effect for flexion and extension.

The facet joints play a load carrying role at the posterior column. As the superior facet joint bears the load from the upper vertebra, the inferior facet joint transmits the load to the vertebra below. In a normal intervertebral disc formation, as the vertebral body carries 80% of the axial plane compression load, the facet joints which are posterior elements, carry 20% of the load. However, in situations like the degeneration of disc material and the narrowing of the disc space, the load transmitted through the facet joints may rise up to 70%.

While the resistance of the lumbar vertebra to the compressive load is endowed by its cortical structure, this resistance at the facet joints is provided by their orientation, the capsular ligament and a little bit by the leaning of the inferior facet to the lamina below^(7,8). The biomechanical studies exposed the weight bearing capacity of the facet joint. Lamy et al.⁽⁹⁾ presented the weight bearing capacity at the lumbar area as 3000 N. Collapse with overloading happens at the level of the pedicle or the pars interarticularis. The resistance against the translational forces is much higher at the facet joint compared to anterior elements of the vertebra. Lu et al.⁽¹⁰⁾ have discovered a 12% rise in the anterior translation and an 18% rise in the posterior translation after the resection

sides. The ratio of the height of the facet joints to their width are almost the same. The thickness of the lumbar facet joint at L1 level is 10 mm and at L5 level it is about 20 mm. Facet joint height ranges between 12 and 20 mm.

Lumbar facet joints are arranged symmetrically on both sides, as left and right. Considering the superior and the inferior facets separately, the values of the angular orientation in the sagittal and transverse planes may be summarized as below: the superior facet joints at L1 level in the transverse plane make an angle of 82.9°; at L5 level 86°; in the sagittal plane at L1 level they make an angle of 139° and at L5 level 117°. The inferior facet joints at L1 level make an angle of 81.4°, at L5 level 71° in the transverse plane; at L1 level 152° and at L5 level 127° in the sagittal plane. In general, the angle of the facet joints in the sagittal plane decreases as the level descends from L1 to lower levels. Meanwhile, the angle of the facet joints does not display a significant change when the level descends from L1 to L5⁽¹⁴⁾.

d) The Lamina

The average thickness of the laminae is 4mm. It is well known that this thickness is 1-2 mm at the superior border and is 6-8 mm at the inferior border of the laminae. Since this thickness is lowest at

the L5 level, the translaminal screw application is harder at this level. The height of the laminae varies between 15-20 mm and the width grows towards the facet joint.

e) The spinous and the transverse processes

The length of the spinous process is 68 mm at L1 level, increasing up to 72 mm at L3 level then decreasing again back to 68 mm at L5 level. The spinous processes play an important role in the procedure of applying interspinous devices for supporting interspinous dynamic bands. Since there is not enough spinous process in the sacrum, these devices are not used between the L5-S1 vertebrae. The distance between the right and the left transverse processes is 71 mm at L1 level and 92 mm at L5 level. The transverse processes display a crucial part in the posterolateral fusion surgery as a suitable bed for fusion.

f) The Spinal Canal

When the anterior-posterior distance (depth) and the left-right distance (width) of the spinal canal, through which the spinal cord passes, is evaluated; we find that the width increases as it descends from L1 to L5. Considering the depth, it decreases from L1 to L3 and increases from L3 towards L5. The width of the spinal canal is 23.7 mm at L1 and 27 mm at L5 level. The depth of the spinal canal is 19 mm at L1, 17.5 mm at L3 and 19.7 mm at L5 level.

The surface area of the spinal canal decreases from L1 towards L2 level, remains the same from L2 to L4 level and increases at L5 level. The surface area is 320 mm² at L1 level, 280 mm² from L2 to L4 and 330 mm² at L5 level.

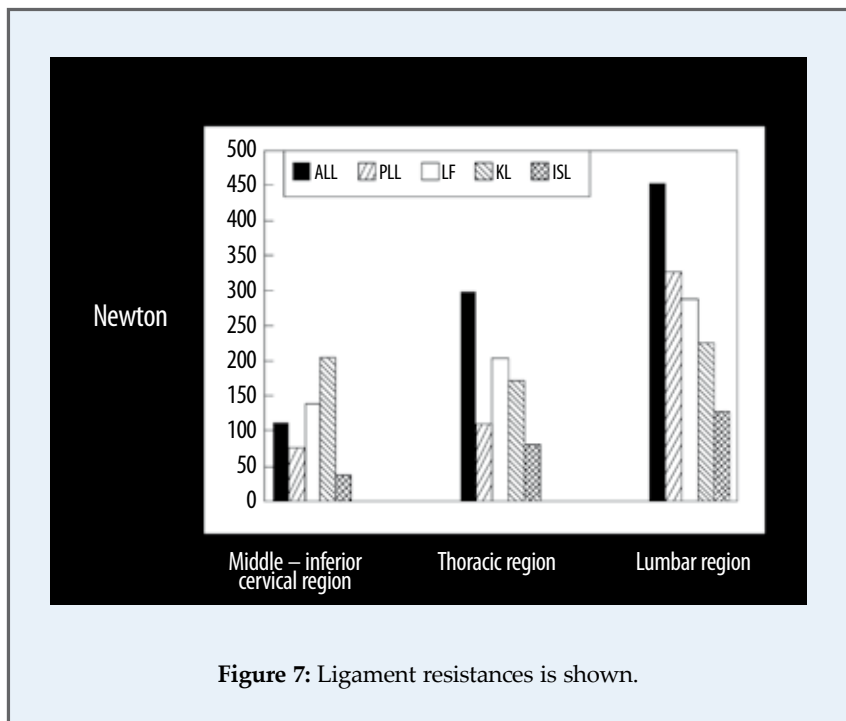
g) The Spinal Ligaments

The spinal ligaments can be listed from the strongest to the weakest among themselves as ALL > PLL > LF > KL > ISL (Figure 7).

Distraction loading in the axial plane is applied to test the resilience of the ligaments. Thus, application of the same load at the same time to all the ligaments is achieved. The ligaments that provide resistance against flexion are PLL, ISL, SSL and KL⁽¹⁶⁾. During flexion of the spinal column, the ligaments supply 70% and the disc material 30% of the resilience.

The extension and the axial rotation motions are limited especially by the disc and a bit by the facet joints⁽¹⁷⁾. The first ligament to be injured after an hyperflexion trauma is the ISL and then the KL. When lateral bending with flexion occurs as a complex motion, a unilateral KL injury happens. For a ligamentous injury to arise, minimally 60 Nm bending and 5°-20° flexion is necessary. An apparent injury comes about after a 120 Nm bending motion⁽¹⁸⁾. According to the morphological analysis of the ligaments at a functional spinal unit, a rise in the length of ALL, LF and ISL is noted from T12 to L5 level. KL length remains the same throughout all the levels. The supraspinous ligament (SSL) is longest at the L2-L3 level and keeps stable at other levels.

The length of the posterior longitudinal ligament (PLL) shortens distal to the L2-L3 level. The length of all the ligaments at the lumbar area are longest at the L2-L3 level. The reason for this is that the apex of



the lumbar lordosis is at the L2-L3 level. There is no statistically rational relation between the grading of the disc degeneration and the length of the anterior and the posterior longitudinal ligaments⁽¹⁹⁾.

Some other points that exhibit special significance about the ligaments of the lumbar area may be defined as: the supraspinous ligament (SSL) exists as a mesh structure at the L4-5 and L5-S1 levels, unlike other levels. Interspinous ligament (ISL) has a more regular fibrous structure at the L3-4 and L4-5 levels but sometimes may be seen as an empty or a fat filled fibrotic band. Because the apex of the lumbar lordosis is at the L2-3 level, the upper and lower endplates are positioned parallel to each other at this level. Thus the ALL and the PLL are in equal length at this level. Assessing the interrelation between the morphology and the function of the lumbar area ligaments some assumptions may be made as⁽⁶⁾: the surface area of the ligaments closer to the instant axis of lumbar rotation are smaller. On the other hand, the surface area of the ligaments far from the instant axis of rotation are wider. For instance, regarding the surface area of the ligaments at the apex of the lumbar lordosis, the L2-L3 level, the measurement of the anterior longitudinal ligament (ALL) is 54.5 mm², the PLL is 20.8 mm², the ligamentum flavum (LF) is 109.8 mm², interspinous ligament (ISL) is 43.5 mm² and the supraspinous ligament (SSL) is 82.1 mm². Especially in the flexion motion, the PLL is oriented for an extension motion. The functional meaning of this is that during the axial rotation motion, this ligament undergoes more alteration.

1) Intervertebral Disc Degeneration

As to the pathology science, the changes occurring through time are called as "aging". However, "degeneration" is defined as the disruption of the structural integrity or functional loss as a result of the retrogressive pathological changes along with the deterioration of the physical properties of the tissues and the cells. As the intervertebral disc ages, it degenerates. The physicians should be able to differentiate aging which is a morphological anatomical change, from degeneration which has the same changes plus clinical symptoms. It is possible to show the intervertebral disc degeneration (IVDD) radiologically among the adults with low

back pain⁽²⁰⁾, yet rational relation between the low back pain and the degenerative changes have not been established⁽²¹⁾. The common radiological signs may be demonstrated in a person without low back pain⁽²²⁾. There is no difference between the anatomical changes of degeneration during aging and the changes regarded as pathological^(23,24). Aging and degeneration are most probably the same pathological processes while some physical features may be causing the clinical symptoms. Lumbar intervertebral disc degeneration is a clinical entity to be investigated. Better evaluation of this issue is only possible with better understanding of the anatomical, morphological and biomechanical properties of the intervertebral disc.

2) Structural Components

The basic structure of the intervertebral disc consists of collagen, proteoglycan and water. As the collagen provides the intervertebral transmission, the lamellar configuration enables mobility. Proteoglycans, supply the hydration of the intervertebral disc material with their osmotic properties.

2a) Water

Normally, the intervertebral disc (IVD) contains high rate of water. Nucleus pulposus contains 85% and anulus fibrosus 78% of water. With aging and/or degeneration, the water rate in both of these tissues declines to 70%⁽²⁵⁻²⁷⁾. Especially the proteoglycan component and eventually the hydration decreases with the growing age^(5,28).

2b) Collagen

The intervertebral disc contains type I and type II collagen⁽²⁹⁾. The human anulus fibrosus comprises 60% type II and 40% type I collagen. The intermolecular space is greater in type II collagen. Therefore, it can attract more water. As a result, highly hydrated fibers of type II collagen can absorb more compressive force by better deforming. The rate of collagen in the outer anulus fibrosus is higher than it is in the inner anulus fibrosus or the nucleus pulposus, in both the elderly and the young people. With the growing age, type I collagen rate increases. IVD loses its capacity to withstand the compressive loading. Along with the degeneration, type III collagen

generation emerges within the IVD. Type III collagen is seen inside the annulus fibrosus.

2c) Proteoglycan

The proteoglycans within the intervertebral disc are composed of a basic protein and a glycosaminoglycan (chondroitin 4-sulphate and keratin sulphate) attached to it. The proteoglycans, form an aggregate by binding to hyaluronic acid using a binding protein. The proteoglycans located in the annulus fibrosus form more aggregates than the proteoglycans in the nucleus pulposus. However, there is a higher rate of proteoglycan in the nucleus pulposus compared to the annulus fibrosus^(30,31). As a consequence of aging and degeneration, the total proteoglycan component amount decreases^(27,28,32). The keratin sulphate / chondroitin 4-sulphate ratio increases^(26, 33, 34) and the capacity of the proteoglycans to form aggregates decreases⁽³³⁾. Eventually, the hydration of the IVD decreases.

3) The Anatomy and the Physiology of the Intervertebral Disc

The intervertebral disc (IVD) comprises 20% of the spine's height. The disc basically consists of an annulus fibrosus on the outside and a nucleus pulposus inside. Its borders in rostral and caudal are made by the cartilaginous end plates. This cartilaginous material braces the IVD and by its connection to the medullary bone, through its pores (*lamina cribrosa*) it supplies the disc. However, with aging the end plates start to calcify and the vascular structure vanishes. The IVD becomes almost avascular⁽¹⁰⁵⁾ and the anaerobic metabolism increases.

With ageing, the fall in the water content and the deterioration in vascularity causes a relative rise in the fibrocytes and chondrocytes, which are more resistant to an environment of low pH value. Up to 2 years of age, the nucleus pulposus is translucent⁽³⁵⁾. Beginning from the second decade on, as the fibrous material increases at the inner annulus and the nucleus, loss of height and proteoglycans ensues⁽³⁶⁾. With the third decade, nuclear fragmentation and fibrosis arises. At the fourth decade, progressive myomatous degeneration,

swelling and fissure formation develops^(37,38). Eventually, nucleus pulposus may become disorganised, dehydrated and fragmented with circumferential and radial tears. With plain radiographic examinations, the degenerative disc alterations may be evaluated in four stages. Stage 1; normal disc, Stage 2; minimal sclerosis with decline in the disc space or osteophyte formation, Stage 3; moderate sclerosis and Stage 4; severe sclerosis with decline in the disc space or osteophyte formation.

The annulus fibrosus consists of fibrous tissue (mainly collagen) lamellae. The fibrous fibers are arranged to form 30° angles at opposite directions (Figure 8). The inner fibers of the annulus fibrosus attach to the cartilaginous plate; the marginal fibers attach to the epiphysis ring of the vertebra corpus and the bony matter of the vertebra corpus. The fibrous fibers which attach to the bony matter (Sharpey's fibers) are stronger than the fibers which attach to the cartilaginous plate. The annulus fibrosus is stronger and firmer at the ventral and lateral sides than it is

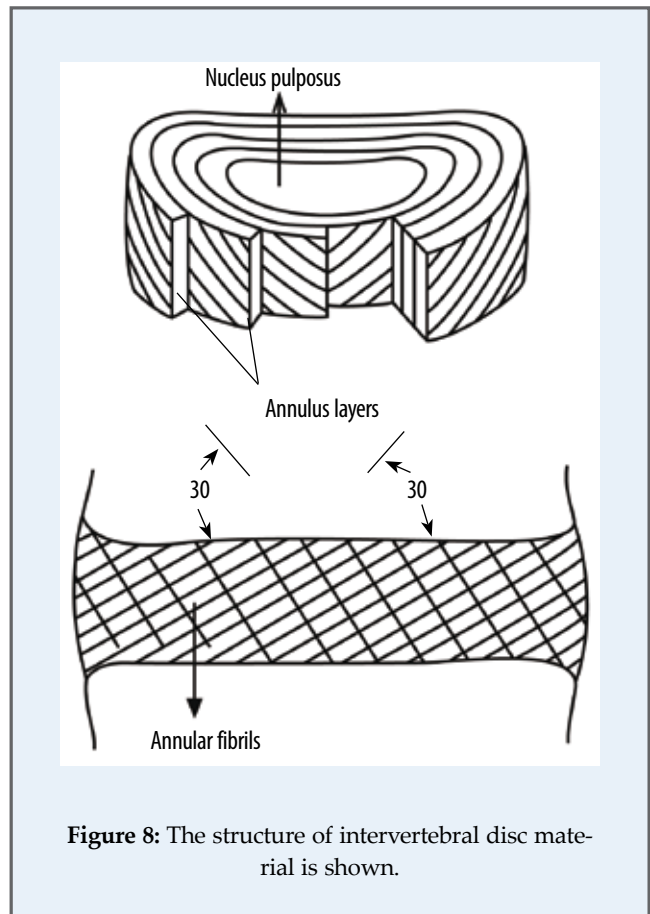


Figure 8: The structure of intervertebral disc material is shown.

at the dorsal side. Anulus fibrosus' being weak at the posterior side is an important factor in the progression of disc herniation.

Nucleus pulposus is a remnant of the notocord and is situated at the posterior side of the disc. The close reticular strips are composed of mucoid substance and the water content this substance decreases in time. The water inside the nucleus is not free. They reversibly bind to the macromolecules with their hygroscopic property. Depending on the pressure, a water movement is observed and the fluid moves in and out of the disc through a semi-permeable membrane. The other small molecular substances, e.g. waste materials and nutrients can move in and out of the disc by this way.

The exchange of the fluid amount inside the disc points that hydrostatic pressure influences the disc space physiology.

The balance of the outer and inner zone of an intervertebral disc is maintained by the formula below:

Extradiscal		Intradiscal		Intradiscal	
Hydrostatic pressure	+	Oncotic pressure	=	hydrostatic pressure	+ oncotic pressure

If any side of the equilibrium increases, the equilibrium is retained by fluid flow to the other side (Figure 9). For example, in case of an increase in axial loading, the intradiscal fluid escapes to the extradiscal space by the impact of hydrostatic pressure. This fluid interchange also enables the nutrient and the waste materials to pass through the membrane, beside its biomechanical effects.

3a) Disc Deformation

The extending of the anulus fibrosus outward, causes the periosteum of the adjacent vertebra to be ripped from the bone. In this case, subperiosteal bone formation (osteophyte) is noticed (Figure 10). While the intervertebral disc herniation

occurs towards the dorsal side, the osteophytic spurs usually arise at the ventral or lateral side.

3b) The Pathophysiology of Disc Degeneration

"Spondylosis" has been defined as the vertebral osteophytosis resulting from degenerative disc disease⁽³⁹⁾. According to the autopsy investigations, the intervertebral disc degeneration starts at the second decade in men and at the third decade in women. At the age of fifty, intervertebral disc degeneration is estimated at a ratio of 97%. L3-4 and L4-5 are identified as the most frequently degenerating discs. The degenerative changes of an intervertebral disc typically contains one or more or all of the developments below:

- Decline in the disc space height
- Irregularity of the disc end plate
- Sclerosis of the disc space
- Osteophyte formation

The basic deteriorations mentioned above causes the instant axis of rotation, which is normally on the intervertebral disc, move dorsally (Figure 11).

3c) Intradiscal Hydrostatic and Oncotic Pressure

A permanent rise in the intradiscal pressure causes a fall in the disc height. Finally, acceleration of the degeneration occurs with the distortion and distraction of anulus fibrosus. The fluid content and the

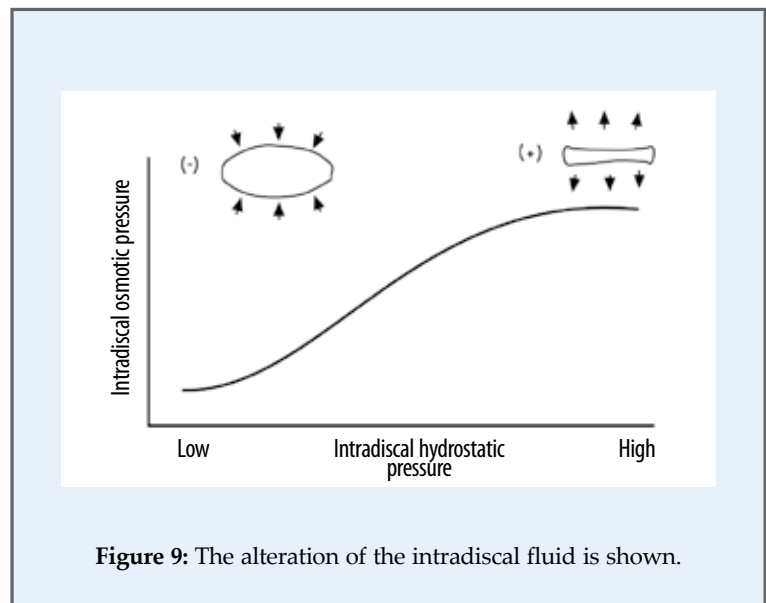


Figure 9: The alteration of the intradiscal fluid is shown.

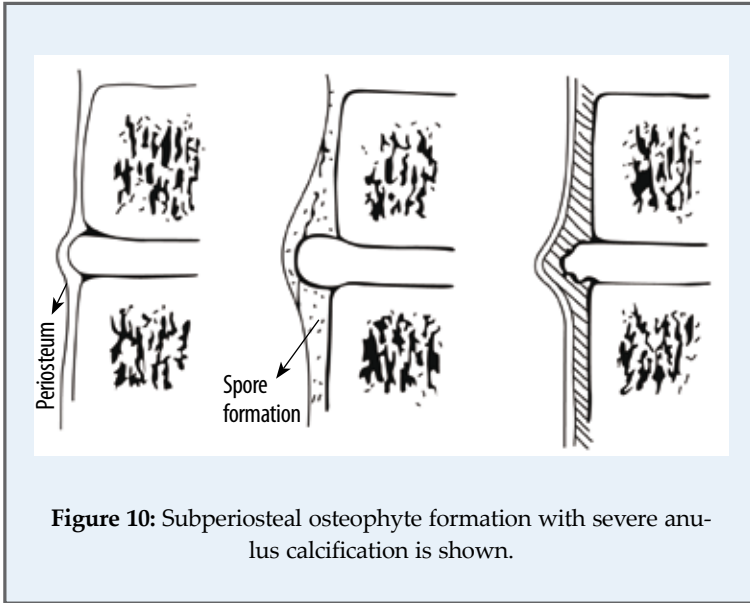


Figure 10: Subperiosteal osteophyte formation with severe annulus calcification is shown.

vascularisation of the intervertebral disc decreases in time. Other factors also play role in disc degeneration along with the factors mentioned above. Fissures arise in the cartilaginous end plate with the degeneration. Eventually, internal herniations (Schmorl's nodule) ensue. Gas is accumulated inside the intervertebral disc (vacuum phenomenon). Mucoïd degeneration and extended intervertebral disc degeneration culminate in spinal instability.

3d) Biomechanism of Intervertebral Disc

The intradiscal pressure is distributed symmetrically when axial loading of the intervertebral disc occurs. However, when the loading is unilateral, the intradiscal pressure is distributed asymmetrically. This situation gives rise to the shifting of nucleus pulposus from the high pressure site to the low pressure site. In contrast, the annulus fibrosus bulges out at the high pressure site (Figure 12).

3e) The Compression Features of the Intervertebral Disc

The hydration of the intervertebral disc (IVD) causes the disc act as a gelatinous substance in the early years of (first 30) life ^(40,41). In the compression tests, it is observed that the disc remains soft under low loads but stiffens under high loads, to increase the stability. For this reason, during the daily activities, the nucleus withstanding compressive loads shows little tendency to herniate by preserving its normal elasticity⁽⁴²⁾. A certain degree of

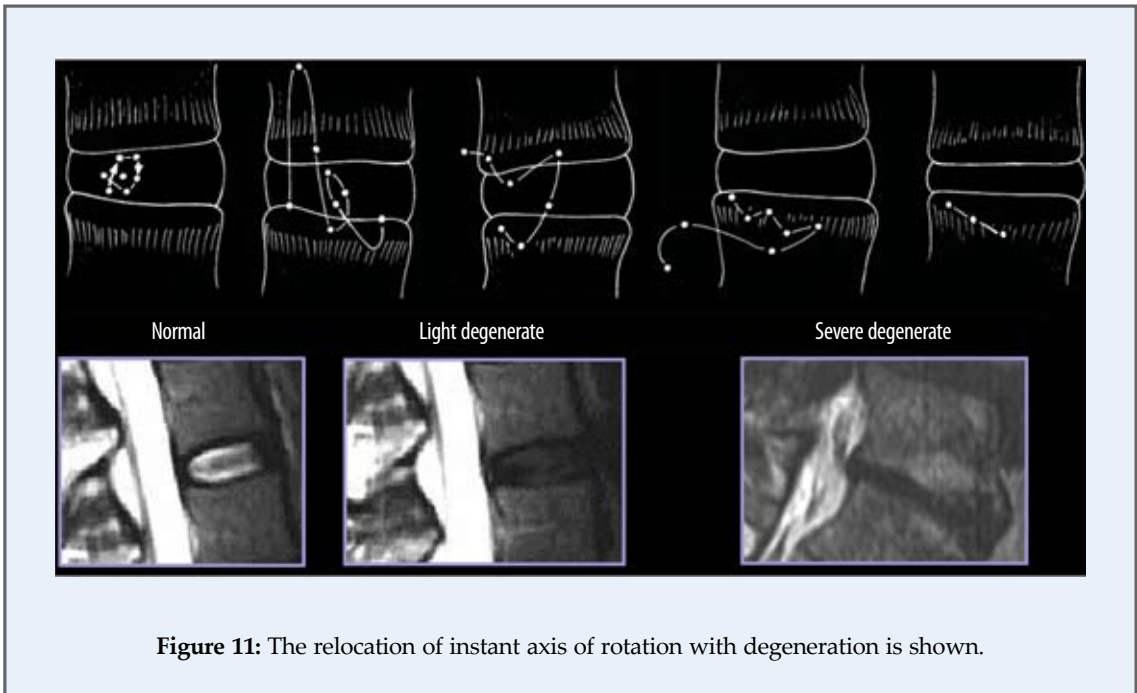
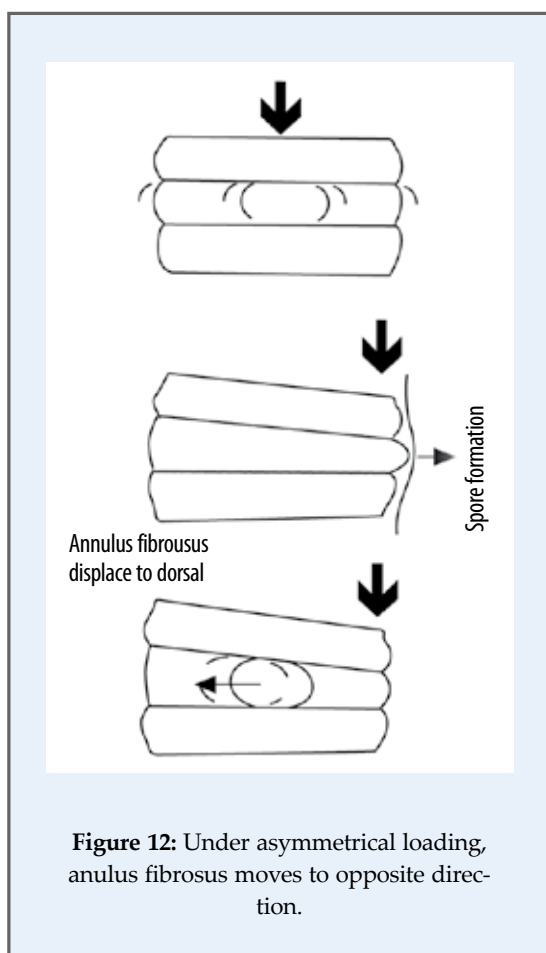


Figure 11: The relocation of instant axis of rotation with degeneration is shown.



pressure is generated inside the disc when there is loading to the IVD.

The fluid distributes this pressure equally. But at the annulus ring there is a complex tension (Figure 13). The IVD dehydrates with degeneration, cannot generate enough hydrostatic pressure, thus the load distributing mechanism deteriorates.

Eventually, the centre of the end plate faces less pressure and the load spreads rather to the periphery (Figure 14). Less peripheral tension, more axial loading occurs at the outer annulus layers of the degenerated disc and too much loading is caused at the fibers.

The altered load distributing mechanism due to degeneration may result in the creation of a Schmorl's node. According to a load bearing test exerted on the functional spinal unit, it is identified that the vertebral end plates are damaged before the disc material.

Even though it is theoretically possible to cause a disc herniation, Virgin⁽⁴¹⁾ stated in his study that the degenerated disc may experience permanent damage with over loading under compressive forces but does not undergo herniation. Therefore, it is accepted that the progress of lumbar disc herniation is not just a consequence of structural changes of IVD but may result from certain types of loadings.

Adams and Hutton⁽⁴²⁾ reported after a study under laboratory circumstances that for an intervertebral disc to develop herniation, the disc had to be degenerated and a special force to be exerted:

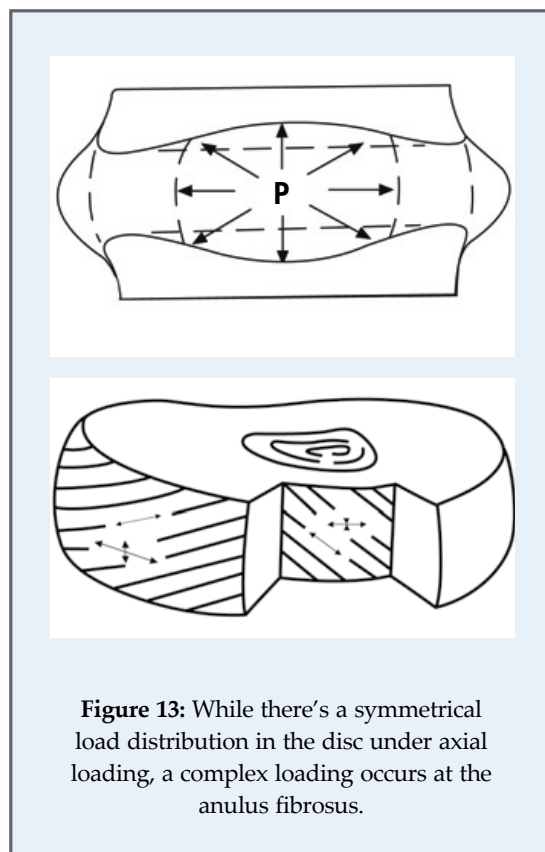
Flexion (shift of nucleus pulposus backwards)

Lateral bending

Axial loading to increase the intervertebral disc pressure (Figure 15)

3f) The Distraction Feature of the Intervertebral Disc

Although pure distractive forces are not applied clinically, in laboratory studies it has been reported that IVD substance is less resistant against the distractive



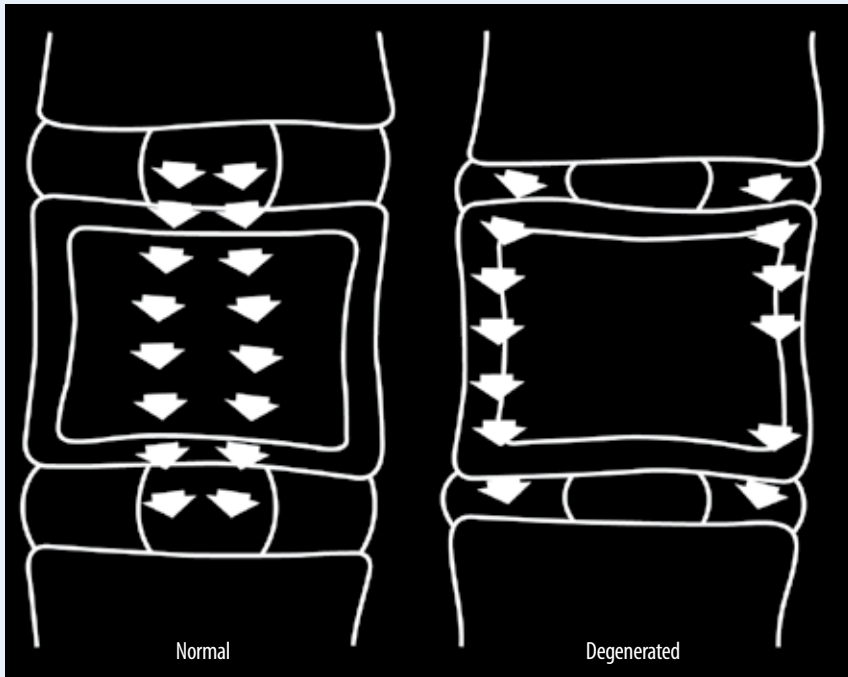


Figure 14: The downward transferring of load takes place more peripherally as the disc material degenerates.

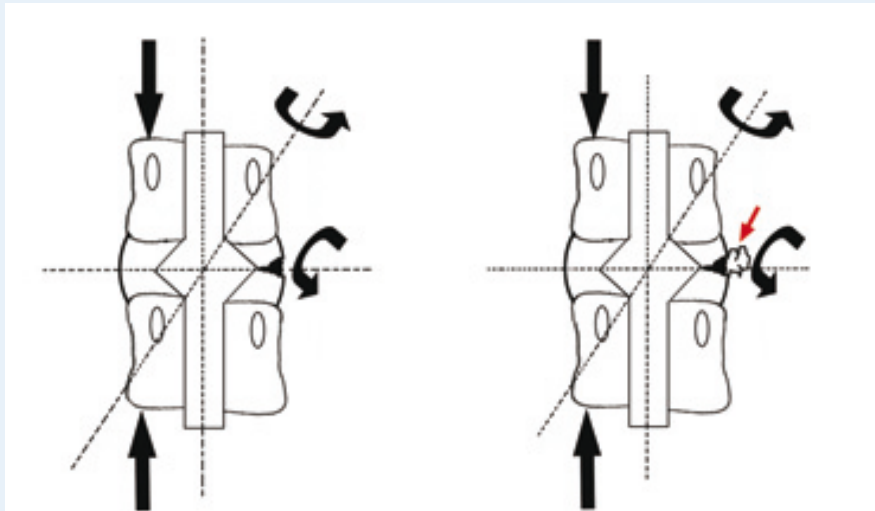


Figure 15: The intervertebral disc herniation generation mechanism under experimental conditions: Asymmetrical axial loading, flexion and rotation motions are shown.

forces than the compressive forces⁽⁴⁴⁾. An experiment, testing the axial tensile loading in a vertebra-disc-vertebra model, revealed that the annulus is strongest at the ventral and dorsal sides and weakest at lateral sides and the middle. In this formation, nucleus is the least durable part against tensile loading⁽⁴³⁾ (Figure 16). This situation is related to the supportive effect of the intradiscal fluid.

3g) The Bending Feature of the Intervertebral Disc

In any one of the flexion, extension or lateral bending motions of the spine, normally one half of the IVD sustains compressive forces (the concave side) and the other half sustains distractive forces (the convex side) at the same time. In regard to this, bending forces are combinations of distractive and compressive forces.

3h) The Torsional Motion of the Intervertebral Disc

When the intervertebral disc (IVD) undergoes a torsion motion, it sustains shear forces both at the axial and the horizontal plane (Figure 17). Farfan⁽⁴⁵⁾, in his study proclaimed that the normal disc material

could resist 25% more to the torsion forces than the degenerated disc material could.

3i) The Shear Features of the Intervertebral Disc

The shear forces affect the IVD in the horizontal plane. Experimental studies revealed that for a disc to deform by just shear forces, it is possible only by a force at approximately 260 N/mm high⁽⁴⁶⁾. For this reason, when a tear is identified in an IVD clinically, it can be supposed that probably another (maybe more) force (bending, torsion or distraction) may be present accompanying to the shear force.

The resisting powers of the intervertebral disc materials are shown in table 2.

	Maximum load	Resistance
Compression	4500 N	2.5 MN/m
Distraction	1800 N	1.0 MN/m
Shear	150 N	0.26 MN/m
Torsion	31 N	2.0 Nm/degree

Table 2: Intervertebral disc endurance.

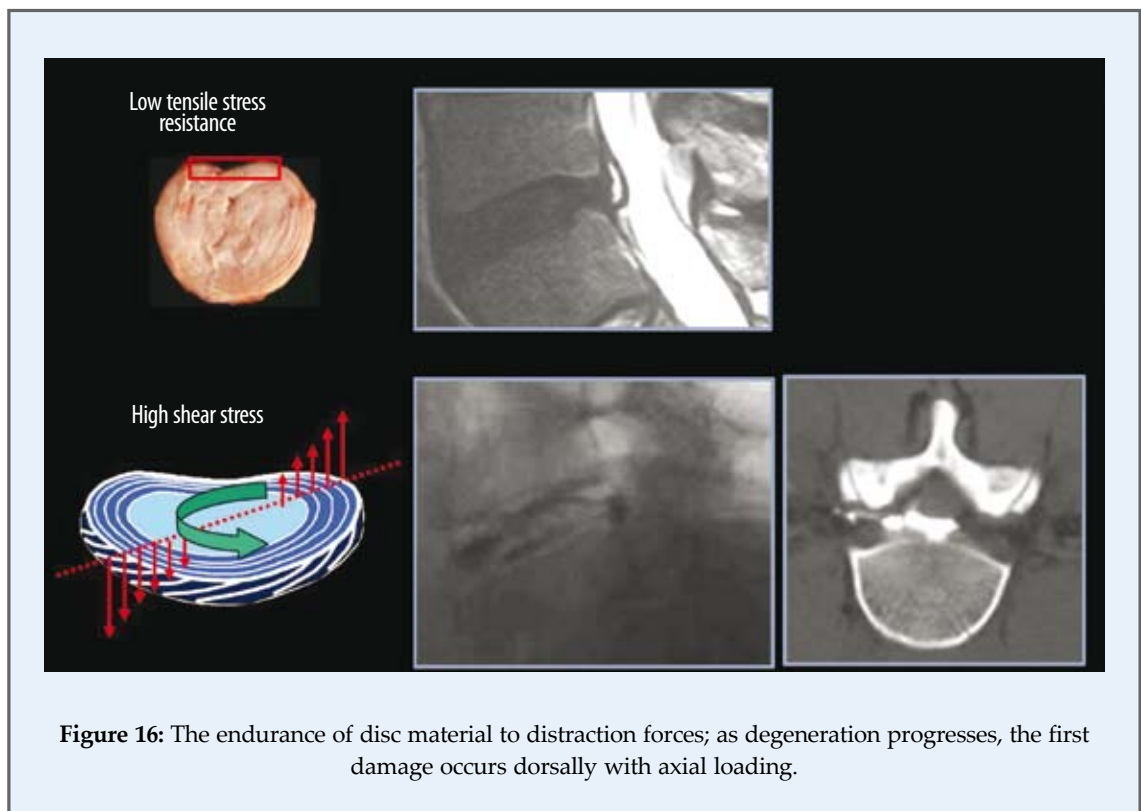


Figure 16: The endurance of disc material to distraction forces; as degeneration progresses, the first damage occurs dorsally with axial loading.

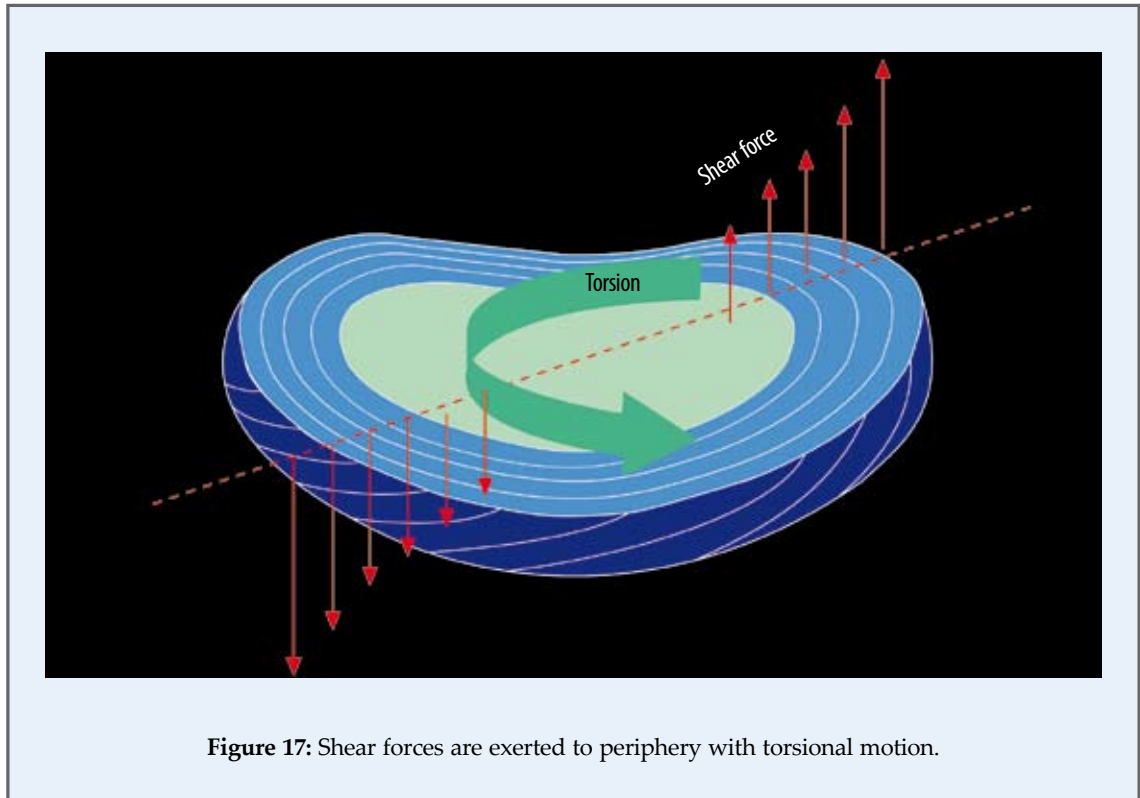


Figure 17: Shear forces are exerted to periphery with torsional motion.

3j) The Resting And Loading Properties of the Intervertebral Disc

Kazarian⁽⁵⁾ accomplished an experimental study about the loaded and resting IVD. He used 4 kinds of IVDs in his study, and staged them from 0 to 3 (0, normal disc; 3, severe degenerated disc) to which he applied sudden, constant force. As a conclusion, he showed that the time spent until the discs develop their final deformed state, is proportional with the disc degeneration. He detected that this period is longest for the non-degenerated disk and shortest for the grade 3, most severely degenerated disc. Thereby, he stated that degeneration decreases the viscoelasticity of the disc and as the degeneration increases, the ability of the disc to withstand shock forces and to distribute these loads to the entire end plate decreases.

3k) The Fatigue Tolerance Feature of the Intervertebral Disc

A short period of over loading may cause an irreversible damage to the IVD material. On the other

hand, the injury mechanism from a long lasting low pressure loading is different and relates to the fatigue of the IVD. After an injury, the regeneration and repair potential of the disc material is low. For this reason, in the fatigue tolerance tests done by cyclic loadings, after 1000 cycles, total injury ensues⁽⁴³⁾. With this type of loading, little injury is done at the beginning but as the loading continues the injury grows and IVD failure develops.

3m) Pressure Inside the Intervertebral Disc

In the early in vivo experiments, pressure transducers were placed inside the disc substance to investigate the pressure. It's been discovered that, when a 20 kg load is carried and bended forward 20°, the pressure at the L3-L4 disc level rises 300% of the normal value⁽⁴⁷⁾. The intradiscal pressure may change according to different body postures. It is 154 kPa while lying, 550 kPa while standing up and 700 kPa while sitting. Besides, it is noted that disc degeneration increases the intradiscal pressure⁽⁴⁸⁾.

4) The Clinical Presentation of Lumbar Disc Degeneration

Intervertebral disc degeneration (IVDD) is defined as the normal anatomical and morphological changes causing clinical complaints. Various clinical presentations are related with lumbar IVDD. The most common among these are disc herniation and spinal instability.

4a) Disc Herniation

The formation of annulus fibrosus tears in IVDD with an increasing rate, the loading of IVD mostly in the age group of 35-55, explains the high rate of lumbar disc herniation in this age group. As mentioned above, although the progression of degeneration does not directly cause the development of disc herniation, it is an important predisposing factor.

4b) Instability

Intervertebral disc provides most of the stiffness of the motion segment because the ligaments and facet joints display low resistance to compression. Panjabi⁽⁴⁸⁾, investigated the relation between degeneration and spinal instability in vitro and in his study demonstrated that degeneration could give rise to spinal instability. *Dysfunctional segmental motion*, is defined as the instability state following the degeneration of IVD and/or vertebral corpus or a tumour. Diagnosis is made by the anamnesis of the typical pain the patient describes. Pain aggravated by activity and eased with resting is characteristic. Neuroradiologically, identification of the mobile segment on plain x-rays and degenerated (black) disc on MRI is essential for the diagnosis. Discography is an important diagnostic tool to display IVDD and also to identify the IVD causing pain with provocative test.

References

- 1- Dolan P, Adams MA, Hutton WC: Commonly adopted postures and their effect on the lumbar spine. *Spine (Phila Pa 1976)* 13(2):197-201, 1988.
- 2- Adams MA, McNally DM, Chinn H, Dolan P: Posture and the compressive strength of the lumbar spine. *International Society of Biomechanics Award Paper. Clin Biomech* 9:5-14, 1994.
- 3- Yoganandan N, Ray G, Pintar FA, Myklebust JB, Sances A Jr: Stiffness and strain energy criteria to evaluate the threshold of injury to an intervertebral joint. *J Biomech* 22(2):135-42, 1989.
- 4- Panjabi MM, Goel V, Oxland T, Takata K, Durancieu J, Krag M, Price M: Human lumbar vertebrae: Quantitative three-dimensional anatomy. *Spine (Phila Pa 1976)* 17(3):299-306, 1992.
- 5- Kazarian, LE: Creep characteristics of the human spinal column. *Orthop. Clin North Am* 6:3, 1975.
- 6- Hirano T, Hasegawa K, Takahashi HE, Uchiyama S, Hara T, Washio T, Sugiura T, Yokaichiya M, Ikeda M: Structural characteristics of the pedicle and its role in screw stability. *Spine (Phila Pa 1976)* 1;22(21):2504-2509, 1997.
- 7- Adams MA, Hutton WC: The effect of posture on the role of the apophysial joints in resisting intervertebral compressive forces. *J Bone Joint Surg Br* 62(3):358-362, 1980.
- 8- Dunlop RB, Adams MA, Hutton WC: Disc space narrowing and the lumbar facet joints. *J Bone Joint Surg Br.* 66(5):706-710, 1984.
- 9- Lamy C, Bazergui A, Kraus H, Farfan HF: The strength of the neural arch and the etiology of spondylolysis. *Orthop Clin North Am* 6:215-231, 1975.
- 10- Lu WW, Luk KD, Holmes AD, Cheung KM, Leong JC: Pure shear properties of lumbar spinal joints and the effect of tissue sectioning on load sharing. *Spine* 30:E204-E209, 2005.
- 11- Weinstein JN, Kim YE: Load sharing among spinal elements of a motion segment in extension and lateral bending. *J Biomech Eng* 109:291-297, 1987.
- 12- Little JS, Khalsa PS: Material properties of the human lumbar facet joint capsule. *J Biomech Eng* 127:15-24, 2005.
- 13- Yang KH, King AI: Mechanism of facet load transmission as a hypothesis for low-back pain. *Spine* 9:557-565, 1984.
- 14- Panjabi MM, Oxland T, Takata K, Goel V, Durancieu J, Krag M: Articular facets of the human spine: Quantitative three-dimensional anatomy. *Spine (Phila Pa 1976)* 18(10):1298-1310, 1993.
- 15- Xu R, Burgar A, Ebraheim NA, Yeasting RA: The quantitative anatomy of the laminae of the spine. *Spine (Phila Pa 1976)* 15;24(2):107-113, 1999.

- 16- Myklebust JB, Pintar F, Yoganandan N, Cusick JF, Maiman D, Myers TJ, Sances A Jr: Tensile strength of spinal ligaments. *Spine (Phila Pa 1976)*. 13(5):526-531, 1988.
- 17- Adams MA, Hutton WC, Stott Jr: The resistance to flexion of the lumbar intervertebral joint. *Spine (Phila Pa 1976)* 5(3):245-253, 1980.
- 18- Neumann P, Osvalder AL, Nordwall A, Lövsund P, Hansson T: The mechanism of initial flexion-distraction injury in the lumbar spine. *Spine (Phila Pa 1976)* 17(9):1083-1090, 1992.
- 19- Panjabi MM, Greenstein G, Duranceau J, Nolte LP: Three-dimensional quantitative morphology of lumbar spinal ligaments. *J Spinal Disord* 4(1):54-62, 1991.
- 20- Coventry MB, Ghormley RK, Kernohan JW: The intervertebral disc: Its microscopic anatomy and pathology. Part III: Pathologic changes in the intervertebral disc. *J Bone Joint Surg*. 27A:460-474, 1945.
- 21- Eckert C, Decker A: Pathological studies of intervertebral discs. *J. Bone Joint Surg*. 29A:447-454, 1947.
- 22- Eyring EJ: The biochemistry and physiology of intervertebral disc. *Clin. Orthop* 67:16-28, 1969.
- 23- Adams M, Deyl Z: Degenerated annulus fibrosus of the intervertebral disc contains collagen type III. *Ann Rheum Dis* 43:258-263, 1984.
- 24- Eyre DR, Muir H: Collagen polymorphism: Two molecular species in pig intervertebral disc. *FEBS Lett* 42:192-196, 1974.
- 25- Harris RI, Macnab I: Structural changes in the lumbar intervertebral discs: Their relationship to low back pain and sciatica. *J. Bone Joint Surg* 36B:304-322, 1954.
- 26- Hirsch C, Paulson S, Sylven B, Snellman O: Biophysical and physiological investigations on cartilage and other mesenchymal tissues. *Acta Orthop Scand* 22:175-181, 1952.
- 27- Hult L: Cervical, dorsal and lumbar spine syndromes. *Acta Orthop. Scand (Suppl 17)*:65-73, 1954.
- 28- Kellgren, JH, Lawrence JS: Osteoarthritis and disc degeneration in an urban population. *Ann Rheum Dis* 17:388-397, 1958.
- 28- Lewin T: Osteoarthritis in lumbar synovial joints. *Acta Orthop Scand (Suppl 73)*:1-112, 1964.
- 30- Lumsden, RM, Morris JM: An in vivo study of axial rotation and immobilization at the lumbo-sacral joint. *J. Bone Joint Surg* 50A:1591, 1968.
- 31- Lyons G, Einsenstein SM, Sweet MBE: Biochemical changes in intervertebral disc degeneration. *Biochim Biophys Acta* 673:443-453, 1981.
- 32- Magora A, Schwartz A: Relation between the low back pain syndrome and X-ray findings: I. Degenerative osteoarthritis. *Scand J Rehabil Med* 8:115-125, 1976.
- 33- Markolf KL: Stiffness and damping characteristics of the thoracic-lumbar-spine: In Proceedings of workshop on bioengineering approaches to the problems of the spine. NIH, 1970.
- 34- McDevitt CA: Proteoglycans of the intervertebral disc. In Ghosh P (ed): *The biology of the intervertebral disc*. Vol 1, CRC Press, Boca Raton FL, 1988, pp 151-170.
- 35- Miller JAA, Schmartz C, Schultz AB: Lumbar disc degeneration: Correlation with age, sex, and spine level in 600 autopsy specimens. *Spine* 13:173-178, 1988.
- 36- Mitchell PEG, Hendry NGC, Billewicz WT: The chemical background of intervertebral disc prolapse. *J. Bone Joint Surg* 43B:141-151, 1961.
- 37- Nachemson A: Lumbar intradiscal pressure. *Acta Orthop. Scand (Suppl 43)*:43-44, 1960.
- 38- Naylor A, Happy F, MacRae T: Changes in the lumbar intervertebral disc with age: A biophysical study. *J. Am Geriatr Soc* 3:964, 1955.
- 39- Resnick D, Niwayama G: Intravertebral disc herniations: Cartilaginous (Scmorl's) nodes. *Radiology* 126:57-65, 1978.
- 40- Van den Hoof A: Histological age changes in the annulus fibrosus of the human intervertebral disc. *Gerontologia* 9:136-149, 1964.
- 41- Virgin W: Experimental investigations into physical properties of intervertebral disc. *J Bone Joint Surg* 33B:607, 1951.
- 42- Yu S, Haughton VM, Sether LA, et al: Criteria for classifying normal and degenerated lumbar intervertebral discs. *Radiology* 170:523-526, 1989.
- 43- Brown T, Hanson R, Yorra A: Some mechanical tests on the lumbo-sacral spine with particular reference to the intervertebral discs. *J Bone Joint Surg Am* 39:1135, 1957.
- 44- Yasuma T, Koh S, Okamura T, Yamauchi Y: Histological changes in aging lumbar intervertebral discs: Their role in protrusions and prolapses. *J Bone Joint Surg* 72A:220-229, 1990.
- 45- Farfan HF, Cossette JW, Robertson GH, Wells RV, Kraus H: The effects of torsion on the lumbar intervertebral joints: The role of torsion in the production of disc degeneration. *J. Bone Joint Surg* 52A:468, 1970.
- 46- Wolfe HJ, Putschar GJ, Vickery AL: Role of the notochord in human intervertebral disc: I. Fetus and infant. *Clin Orthop* 39:205-212, 1965.
- 47- Nachemson A: The load on lumbar discs in different positions of the body. *Clin Orthop* 45:107, 1966.
- 48- Panjabi MM, Brown M, Lindahl S, et al: Intrinsic disc pressure as a measure of integrity of the lumbar spine. *Spine* 13(8):913, 1988.