DISC DEGENERATION

TIMING of MRI findings

In MRI, the Thompson [Spine 1990;15:411-5.]

In conventional MRI, the first sign of degeneration is inhomogeneity of the intranuclear cleft followed by bulging of the disc. Late signs are decreased signal intensity and reduced disc height.

In a Finnish study of 151 men aged from 40 to 45 years in whom MRI was performed twice with a four-year interval, a clear sequence of these signs was shown.[Int Soc Study Lumbar Spine, Adelaide, 2000;23.]

Disc Degeneration Grading system Pfirrmann [Spine 2001:26: 1873]

<table>
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<tr>
<th>Grade</th>
<th>Description</th>
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<tbody>
<tr>
<td>I</td>
<td>Homogenous, Hyperintense Normal: Height.</td>
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<tr>
<td>II</td>
<td>Inhomogenous white signals with gray Normal disc height</td>
</tr>
<tr>
<td>III</td>
<td>Intermediate gray signal within the nucleus pulposus Disc Height Preserved</td>
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<tr>
<td>IV</td>
<td>Dark gray signals Mild loss of disc height</td>
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The pathogenesis of disk degeneration has been well-described. Changes that occur begin as **early as the second decade** and are very similar to those associated with disk aging [Semin Spine Surg 23:227-234].

The normal disk is avascular; obtains nutrition through passive and active transport of molecules through the disk substrate. As vascular penetration across the endplate diminishes, cell death in the disk occurs, resulting in reduced extracellular matrix production and a change in the relative composition of matrix proteoglycans. Disc degeneration is followed by secondary changes in the spine: including disc bulge or protrusion, endplate changes, Modic changes. These features are commonly present in the presence of or in the absence of symptoms. Eventually facet joint arthritis set in. Butler [Spine 1990;15:111-3.] performed CT and MRI of 330 discs; in 108 there was degeneration without changes in the facet joint, in 40 both the disc and the facet joints were degenerate, and in only one was there isolated **facet joint degeneration** without corresponding changes in the disc. **Instability:** It appears that instability can be assumed when dynamic AP translation is 5 mm or more.
CAUSATION

1. CONSTITUTIONAL

Age related cellular changes Degeneration in Children: [Spine (Phila Pa 1976), 2005 Apr 1;30(7):798-806]

Signs of disc degeneration were noted in approximately 1/3 of the subjects. Reduced signal intensity and irregular nucleus shape in the upper 3 lumbar discs were significantly associated with LBP within the last month, whereas reduced signal intensity and disc protrusion at L5-NS1 were associated with seeking care. Endplate changes in relation to the L3 discs were associated with LBP.

6jaer. SPINE Volume 30, Number 7, pp 798–806. Children

Signs of degenerative disc changes were present in approximately 1/3 of subjects at the age of 13 years and were associated with LBP to some degree. The loss of disc signal and disc height, as well as irregularity of the nucleus shape in the upper part of the lumbar spine were noted in 3% to 6% and strongly associated with LBP, particularly in boys.

Disc herniation and HIZ were most strongly associated with LBP in girls.

Spondylolisthesis with or without spondylolysis was significantly associated with LBP only in girls.


In asymptomatic volunteers of 60 20-year to 50-year-old asymptomatic patients, 67% had disc protrusions, 33% had high signal intensity zones, and 18% had disc extrusions based on MRI.

The finding that the risk of leaking annular tears in the lumbar discs increased from about 10% in -20%; 35% 20-40; 50% to 59 years of age implies that pain-related disc pathology exists in one-third of middle-age men.


MRI indicated degenerative changes in the lumbar spine in 79 subjects (84 %), with decreased disc signal intensity in 74.5 %, posterior disc protrusion in 78.7 %, anterior compression of the dura in 81.9 %, disc space narrowing in 21.3 %, and spinal canal
stenosis in 12.8%. These findings were more common in older subjects at caudal levels. MRI showed degenerative changes in both the lumbar and cervical spine in 78.7% of the volunteers.

3. Takatola. Finish study. SPINE Volume 34, Number 16, pp 1716–1721
Disc Disease in asymptomatic 20-22 years old N =335
The prevalences of disc bulges [25%] and radial tear[ 9.1%].
HIZ lesions were more common among women than men (8.6% vs. 4.3%), whereas herniations were among men (5.6% vs. 2.5%).
Only 2 disc extrusions were observed, one in each gender.
All degenerative disc findings were more common at the L5–S1 level except HIZ lesions, which were most likely at L4–L5. The prevalence of the Modic changes was 1.4%, type I being more common than type II.
Conclusion. Almost half of young Finnish adult aged 21 years had at least one degenerated disc, and a quarter had a bulging disc.


| Table II. Incidence of MRI findings in individuals who had never had LBP according to Boden et al |
|-----------------|-----------------|-----------------|-----------------|-----------------|
| Age (yrs)       | Number          | Herniated disc (%) | Spinal stenosis (%) | Bulging disc (%) |
| 20 to 39        | 35              | 21               | 1                | 56              |
| 40 to 59        | 18              | 22               | 0                | 50              |
| 60 to 80        | 14              | 36               | 21               | 79              |

Forty percent of individuals under 30 years of age had lumbar intervertebral disc degeneration (LDD), the prevalence of LDD increasing progressively to over 90% by 50 to 55 years of age. There was a positive correlation between the DDD score and low back pain. L5-S1 and L4-L5 were the most commonly affected level.

6. Fraser. SPINE Volume 32, Number 25, pp 2797–2804
Tears in the L4–L5 disc show different patterns of incidence with aging, which can be explained by current biomechanical concepts. Tears may not only perturb disc function and cause segmental instability, but the frequency of neovascularization
accompanied by neoinnervation indicates that pain originating within the degenerate disc should not be dismissed as the frequent evidence of bleeding into the tear lumen indicates the susceptibility of the vessels to trauma.

While RL[radiolateral annular tear] may be unique by virtue of the fact that they are singularly capable of leading to advanced disc degeneration in experimental animals, discs do not require RL as an essential prerequisite to undergoing degeneration. They found RL to be present in 20% of the 10- to 30-year age group, and there was a linear increase thereafter so that they were found in nearly 90% of L4–L5 discs in the group aged 51 to 80 years. The overall incidence of 56%.

2. GENETIC

1,2: Genetic factors have been shown to play a significant role in the development of lumbar disc degeneration and the occurrence of herniation; however, the way in which these factors may weaken the disc structure, predisposing it to structural failure, remains unknown. [Spine 1995;20:2601–12; J Bone Joint Surg Am 1995;77:1662–70.]


We conclude that the combined genotype VEGF -2578CA+AA/-634CC is a possible risk factor for IVD degeneration and the VEGF -2578A/-1154A/-634C/936C haplotype may increase the risk for development of IVD degeneration
This study highlights importance of COL9A2 gene variation especially of homozygous variety in contrast to COL9A3 variation in causing disc disease in Indian population [single nucleotide polymorphism in collagen IX and intervertebral disc disease]

The Tt and the tt genotypes of Taq I polymorphism of the vitamin D receptor gene have been associated with IVD degeneration

3. MICRO AND MACRO TRAUMA
a. Mechanical Pressure:
Mean failure pressure of disc 14.1 +/- 3.9 Mpa With respect to the annular wall, the posterior region is most susceptible to failure in the presence of high nuclear pressure, even when loaded in the neutral position. Weak inter-lamellar cohesion of the outer posterior lamellae may explain why the majority of herniations remain contained as protrusions within the outer annular wall.
It is well documented that when loaded in pure compression, the elevated pressure generated within the nucleus of a motion segment causes vertebral end-plate failure before macroscopically visible annular damage. [Roaf R. J Bone Joint Surg 1960; 42:810–23, Yoganandan Microtrauma in the lumbar. Neurosurgery 1988;23:162–8]

Repeated application of loads amounting to between 50% and 80% of the ultimate tensile strength of the endplate can cause a fracture after as few as 100 cycles. There is a strong association between degeneration and defects in the endplate from Schmorl’s nodes, Scheuermann’s disease and fractures, with an increased incidence of disc prolapse, particularly at the lower lumbar levels. Damage to the endplate rapidly leads to depressurisation of the nucleus and a simultaneous increase in stress in the posterior annulus.
Destruction of cartilage from an endplate fracture would provoke an IL-1-mediated inflammatory response, inducing enzymes that destroy proteoglycans. Mechanical
factors can trigger biochemical reactions which, in turn, may promote the normal biological changes of ageing, which can also be accelerated by genetic factors.

c. Military service and back ache [Spine 36, Number 18, pp 1492]
Each service, when compared with the Marines as the referent category, had a significantly increased incidence rate Army: 2.19, Navy: 1.02, and Air Force: 1.54.

RELEVANT ANATOMY
Sato. Spine 1999;24:2468–2474 Intradiscal pressure study
Using an advanced pressure sensor: in normal volunteers and in degenerated disc, the actual loading conditions in various body positions were calculated in relation to the angle between the two vertebrae of the studied motion segments. The intradiscal pressure was significantly reduced according to the degree of disc degeneration as estimated by magnetic resonance imaging. The spinal load calculated on the L4–L5 disc for healthy subjects with an average body weight of 73 kg and an average L4–L5 disc cross sectional area of 16 cm2. The spinal load increased in the following order of body positions:

<table>
<thead>
<tr>
<th>Body Position</th>
<th>Spinal Load</th>
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<tbody>
<tr>
<td>Prone</td>
<td>144 N</td>
</tr>
<tr>
<td>Lateral lying</td>
<td>240 N</td>
</tr>
<tr>
<td>Upright standing</td>
<td>800 N</td>
</tr>
<tr>
<td>Upright sitting</td>
<td>996 N</td>
</tr>
</tbody>
</table>

In the standing and sitting body positions, the spinal load increased not only with forward bending, but also with backward bending. The spinal load was highly dependent on the angulation in the motion segment.
The movements of the spine from a flexed to an extended position made the load of the spine change in a curvilinear fashion.

Conclusions. The spinal load was highly dependent on the angle of the motion segment in normal discs in vivo. The intradiscal pressure in degenerated discs was significantly reduced compared with that of normal discs.
PATHOLOGY

**Annular degeneration** [Spine Volume 29, Number 23, pp 2668–2676]

Appear earlier and are more clearly related to back pain than previously thought. Disc cellularity declines as age advances and is reduced in the vicinity of major tears. Extensive end-plate abnormalities reduce cellularity by impeding disc nutrition. a. SPINE Volume 33, Number 25, pp 2767–2773.

**Summary Interventional Neuroradiology 18: 227-241, 2012**

Significant changes occur in the disc including alteration of proteoglycan/water content (disc desiccation) and loss of anatomic integrity of the annulus fibrosus (concentric/lamellar separation, radial annular tears, anular fragmentation).

**Radial tears** that extend from the nucleus into the outer third of the annulus readily
reproduce a patient’s symptomatic low back pain on discography. Such tears, which are classified by Modified Dallas Discogram grade 3 or 4 manifestations of internal disc disruption, may account for 30% to 50% of chronic low back pain cases. [Br J Radiol 1992;65:361–9.]


**Protrusions:**

Focal Protrusion

Central, Paracentral, Foraminal and far lateral

Broad based: Thicker and Thinner

Bulge

Protrusion [A]

Extrusion [B]

Sequestrated [C]

Under most circumstances, acute LBP recovers spontaneously or with conservative medical management. In a study of work-related acute LBP, pain resolved in two
weeks in approximately 50% of patients with 85% experiencing functional recovery in four to six weeks and 95% developing significant improvement and return to work by three months.

In approximately 5% of patients LBP persists and becomes chronic. Acute-onset sciatica is less common but symptom resolution is often incomplete with persistent radiculopathy in approximately 50% of patients after four to six weeks. Even massive disc herniation can be treated non-operatively.

Disc protrusion was seen in the majority of these discs, generally related to out-pouching and stretch of the thin residual annular margin present. A variable degree of generalized disc bulging was present in these internally deranged discs, likely contributing to stretching and thinning of the annular margin. Given the focal location of the annular derangement in these discs, the source of discogenic pain most likely relates to the focal annular defect zone. Identification of the focal protrusion therefore, likely relates to the cause of discogenic LBP in these discs. The normal disc is innervated in up to the peripheral third annulus only.

1. In abnormal discs, nociceptors have been identified deep within the annulus.
2. Inflammatory byproducts leaking from the disc can promote the in-growth of local pain fibers and granulation tissue
3. Instability pain
4. Neurogenic
5. Facetal arthritis

CONCLUSION

In patients with chronic LBP, peripheral disc margin shape correlates with the features of internal annular derangement in significantly painful discs identified at provocation lumbar discography. Disc protrusion is seen in the presence of underlying radial defect (radial tear, annular gap) with thin overlying peripheral annular margin. Disc bulge suggests the presence of complex disc degeneration with annular fragmentation.

60–70% recover by 6 weeks,

90% by 12 weeks.
Recovery after 12 weeks is slow and uncertain.

**TREATMENT**

Provocation discography is perhaps one of the rare techniques, which is helpful in indicating the need for lumbar spinal fusion in patients with degenerative discs. It is, however, invasive, and a small amount of antibiotics should be added to the dye to protect against iatrogenic discitis. Provocation discography should be combined with psychometric testing. The painful disc should be fused and a 360° fusion is perhaps better than a posterior fusion in patients with positive provocation.

1. **Surgeon/patient factor:**

Volinn [*Spine* 1992;17:575-9] found a nearly 15-fold variation in spinal surgery, which was weakly related to explanatory variables. Those related to the surgeon included:

1) Variable diffusion of medical innovations (willingness to apply promising new *versus* well-established methods of treatment);
2) Preconceptualisation (knowledge learned during residency training, later acquired knowledge may be assimilated into a preconceived knowledge base);
3) Tolerance of uncertainty (surgeons are inclined towards action and resolving of uncertainty)
4) Reaction to patient expectations (pressures exerted by the patient vary by ethnic group, class

2. **Lumbar spinal fusion.** [VOL. 84-B, NO. 6, 883]

In a meta-analysis of 47 articles published on lumbar spinal fusion from 1966 to 1991 no randomised trials were found. On average, 68% of patients had a satisfactory outcome after fusion.

Reoperations had a worse outcome. There is some evidence that discogenic pain may persist after solid posterior fusion [*J Bone Joint Surg [Br]* 1986;68-B:142-3]

A bad outcome can be related to the wrong indication as well as the choice of the wrong technique. An average of 3.7% of patients had deep-vein thrombosis, 2.8% had neurological injury, and 8.7% developed chronic pain at the donor site. Pseudarthrosis averaged 14% overall.
Malteret have shown that lumbar fusion is associated with a higher rate of reoperation

3. Operative Vs Non-operative: In a study[Am J Ind Med 1996;29:584-9] of 507 patients, 133 were receiving workers' compensation. Of the latter, 65 had been treated surgically and 68 non-surgically. The patients treated surgically were more likely to have had preoperative imaging, worse leg pain and decreased physical function than the non-surgically-treated patients.


The valid argument: Many studies have compared a series of individuals with and without LBP and tried to demonstrate that a particular condition causes pain or is not likely to be painful. A condition occurring with the same frequency in patients with and without pain is likely not to be the cause of pain, and should therefore not be treated by fusion. Disability seems to play a role in the assessment by most surgeons, as preoperative disability in patients treated operatively is much greater than in those treated conservatively

Fritzell [Spine 26:2521-2532, 2001]

A randomized, controlled multicenter trial that compared lumbar fusion with nonoperative treatment. Two hundred ninety-four patients aged 25-65 with severe chronic low back pain were enrolled. Inclusion criteria included pain of at least 2-year duration and pain that was felt to be emanating from L4-L5 and/or L5-S1 with evidence of degenerative changes on imaging studies. Sixty-three percent of surgical patients rated themselves as “much better” or “better,” compared with 29% in the nonoperative group. The fusion rate was 83%. There was a 17% complication rate, and the authors recommended preoperative counseling before surgery.

A COCHRANE REVIEW115 came to the following conclusions:

1) There is no acceptable evidence (strength D) of the efficacy of any form of fusion
for degenerative lumbar spondylosis, back pain, or ‘instability’.

2) There is limited evidence (strength C) that adjunct fusion to supplement decompression for degenerative spondylolisthesis produces less progressive slip and better clinical outcomes than decompression alone.

3) There is limited evidence (strength C) that fusion alone may be as effective as combined decompression and fusion for patients with grade-I or grade-II isthmic spondylolisthesis and no significant neurology.

4) There is strong evidence that instrumented fusion may produce a higher rate of fusion (strength A), but does not improve clinical outcome (strength A).

Lumbar spinal fusion should therefore be undertaken with caution. It should be borne in mind that there is little evidence to suggest that it has a beneficial effect on patients. Psychosocial screening should be performed and if psychosocial distress is shown a thorough evaluation should follow. If both a psychosocial and organic disorder are found, both should be treated, with discussion between the psychiatrist and the surgeon as regards the sequence of treatment.

**Future**

The ideal study to evaluate the efficacy of lumbar fusion for discogenic pain has not yet been performed. All the studies to date have flaws that limit their validity. Ideally one would start with patients with chronic low back pain that was believed to be discogenic on the basis of history, physical examination, imaging studies, and diskography. Patients should have no clear contraindications for fusion (eg, osteoporosis) or problems that might impact outcome determinations (eg, psychiatric disorders, workers compensation).

The patients then would be sent for a comprehensive physical therapy program, including behavioral and cognitive components, that has been shown to be effective. Only patients who have failed 6 months of conservative management would then be eligible for randomization and would be allocated to continuing nonoperative management or surgery. The surgical procedure would consist of circumferential fusion with interbody and posterolateral fusion to completely fuse the motion segment, including the painful disk.
Adjacent disc Disease [Spine J. 2011 Jan;11(1):21-3]
Incidences and prevalence of surgery at segments adjacent to a previous posterior lumbar arthrodesis.
An overall annual incidence and predicted 10-year prevalence of further surgery for ASD after lumbar arthrodesis were 2.5% and 22.2%. These rates varied widely depending on the identified risk factors:
1. Level of fusions: More in 2 level than single level
2. Age of the patient: Older more chances.

Spontaneous Vs Injury
MRI: Common degenerative findings are often interpreted as recent developments and the probable anatomic cause of the new symptoms. Findings on MR imaging within 12 weeks of serious LBP inception are highly unlikely to represent any new structural change. Primary radicular syndromes may have new root compression findings associated with root irritation. Support an alternative hypothesis:
Degeneration begin very early in life- the basis of nutritional, developmental, and genetic factors. Later minor traumatic or repetitive occupational events play a minor role, if any, in eventual structural changes and serious disability.

However, the fact that many patients report no injury before the onset of sciatica suggests that some discs are more susceptible to herniation than others, probably on account of the weakening effects of middle age [Spine (Phila Pa 1976) 2006;31:2151–2161] or genetic factors [Spine (Phila Pa 1976) 2008;33:2801–2808]

Predictors of outcome
1. Duration of symptoms [>6 m of chronic back pain]
In chronic LBP, and especially in patients who have been on sick leave for more than a month, resumption of work can be predicted almost exclusively by psychosocial factors, disability, and comorbidity. [Hiebert R, Pietrek M, Crane M, Nordin M; Int Soc Study Lumbar Spine, Adelaide, 2000:6]
2. **Psychosocial factors:** Hasenbring [*Spine* 1994;19:2759-65] found that persistent pain six months after discectomy was best predicted by a combination of somatic (the degree of disc displacement), psychological (depression, pain-coping strategies, endurance strategies, non-verbal pain behavior. Look for Waddell signs, inappropriate non-organic signs, signs when patient is distracted [SPINE Volume 19, Number 24, pp 2759]

3. **Compensation, Litigation: Poor prognosis**

   Compensation has a negative influence on the length of disability. **Adelaide study**

   - The average time off work: 12 months for men with compensation Vs 1 month with no compensation, for women, 15 M Vs 0.5 Months not on compensation.
   - Filing a compensation claim for costs, retaining a lawyer, or higher pain intensities were limited predictors of longer claims (level 3). As the ratio of compensation to preinjury wage increases, there is moderate evidence (level 2) that the duration of the claim increases and that disability is more likely. Compensation status, particularly combined with higher pain intensities, is associated with poorer prognosis after rehabilitation treatment programs [*Clin J Pain*, 2001 Dec;17(4 Suppl):S46-64.]

4. **Occupation**

   Several studies showed that LBP is strongly associated with occupation [*Spine* 2000;25:487-92]. Machine driving with whole body vibration and prolonged sitting were notable factors.

5. **Smoking** [*Spine*:1998;23,2207 Leboef]
Consistent evidence in favor of a causal link between smoking and low back pain.

6. **Job**

- Unavailability of light duties on return to work
- Availability of compensation with no end point [80% of the income]
- Belief that working causes increase symptoms
- Belief that need for 100% fit to return to work
- Lack of job satisfaction

**REFERENCES**

- *Lumbar spinal fusion. [VOL. 84-B, NO. 6, 883]*