

# Enabling rapid decisions on ACC cover and entitlements

Consideration factors for: Lumbar disc pathology (lumbar disc injury; same-level fusion following lumbar disc injury; adjacent segment disease following fusion)

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This information has been developed by ACC's Clinical Services working together with the New Zealand Orthopaedic Spine Society in association with the New Zealand Orthopaedic Association (NZOA). It outlines factors ACC staff consider when making decisions on cover and entitlement requests. These factors are based on a review of published research evidence and expert opinion.

## Enabling rapid decisions for ACC clients

It's important that we make funding decisions for our clients as efficiently as possible, especially when, for some, getting surgery sooner is likely to lead to a better outcome.

ACC funding of entitlements is considered on a case-by-case basis. When we make a decision, it's based on information provided in the Assessment Report and Treatment Plan (ARTP), contemporaneous clinical information and imaging reports provided, along with information we already hold.

In all cases where ACC funding for cover and/or medical/surgical management is sought, the treating clinician should explain the causal link between the condition they are treating and the injury that ACC has covered.

## ACC assessment of cover and entitlement funding requests

ACC is required to ensure that its funding decisions comply with its legislation. The need to establish a causal link between a condition to be treated and an ACC-covered injury is critical to this assessment.

Applications for entitlements (e.g. surgery request) must be related to an accepted ACC claim for that body site. In the absence of such a covered claim ACC will not progress the application.

It should be noted that a temporal attribution of symptoms to an injury is not sufficient evidence of causation.

Where the conclusion using these consideration factors is that causation is unlikely to be established, the treating clinician should set these expectations with their patient and advise ACC.

## Consideration factors

ACC and the NZOA have developed General consideration factors for surgery funding requests. This document (ACC7637) can be found on the ACC website at [acc.co.nz](http://acc.co.nz). These factors apply across all surgery funding applications and are relevant here.

This document focuses specifically on:

- *Lumbar disc injury*
- *Same-level fusion following lumbar disc injury*
- *Adjacent segment disease following fusion*

# Lumbar disc pathology

In determining consideration factors for the causation of lumbar disc pathology, a standardised approach to lumbar disc nomenclature is required. The definitions put forward by the American Society of Spine Radiology and the American Society of Neuroradiology (Fardon et al, 2014) are the accepted definitions for lumbar disc terminology used throughout this document (Appendix A).

The literature describes a range of potential causes for disc pathology. Most commonly these are attributed to, or associated with, trauma, disc disease, degenerative/wear change, increasing age, and/or loading/activities causing repetitive microtrauma.

Radiculopathy results from nerve root compression, the cause of which is multifactorial.

Predisposition to nerve root compression may be altered by spinal canal morphology of multifactorial cause which includes:

- Disc degeneration
- Bony/spinal canal shape (developmental/constitutional)
- Facet joint hypertrophy
- Ligamentum flavum changes
- Osteophyte formation
- Spondylophyte formation
- Synovial (facet joint) cyst
- Annular fissure
- A combination of the above.

With increasing age, asymptomatic changes in disc structure and MRI appearances may occur including disc desiccation, annular fissure, spondylophyte formation, osteophyte formation and facet joint degeneration. These changes are within the normal spectrum (Brinjikji et al, 2015). It must be recognised that the presence of pathology does not necessarily assist in determining causation and that pathology (including nerve root compression) can be present without symptoms.

## Background prevalence

The background prevalence of asymptomatic disc degeneration (including disc desiccation, bulge, herniation, extrusion or sequestration) is an important consideration in determining the causation of a lumbar disc injury. Changes in the lumbar discs are a common finding in pain-free individuals as well as those with back pain. In a systematic review studying the prevalence of spine degeneration on imaging in asymptomatic individuals, the prevalence of disc degeneration increased from 30% of those 20 years of age to 84% of those 80 years of age. Disc protrusion prevalence increased from 29% of those 20 years of age to 43% of those 80 years of age. The prevalence of annular fissure increased from 19% of those 20 years of age to 29% of those 80 years of age (Table 1, Appendix B).

# 1. Lumbar disc injury

**Table 1:** Factors to consider in decisions on lumbar disc injury

**IMPORTANT:** The factors are not to be considered in isolation; rather the overall balance of factors that are more supportive or less supportive of a causal link must be considered.

## Factors MORE SUPPORTIVE of a causal link

<b>Cover</b>	There is an ACC-covered lumbar spine injury and evidence of a lumbar spine injury documented in the contemporaneous clinical notes.
<b>Previous history</b>	No previous history of persisting low back symptoms or dysfunction and no clinical evidence suggesting pre-existing lumbar disc pathology.
<b>Demographic</b>	Younger age.
<b>Mechanism of injury</b>	History of a loading event involving axial compression combined with flexion, rotation and/or a sudden axial impact load to the lumbar spine.
<b>Current history</b>	Immediate low back pain and documented functional impairment/disability.  Continuity of back pain with development of radiculopathy.  No history mismatch between the history recorded in the ARTP and the contemporaneous medical records.

## Factors LESS SUPPORTIVE of a causal link

<b>Cover</b>	ACC cover has not been given for a lumbar spine injury and there is no evidence of a lumbar spine injury documented in the contemporaneous clinical notes.
<b>Previous history</b>	Documented clinical evidence of pre-existing lumbar spine symptoms or dysfunction in the lower back or pre-existing radiculopathy/sciatica.  Note: A history of prior low back problems or radiculopathy does not exclude a new accident causing a new lumbar disc herniation.
<b>Demographic</b>	Older age.
<b>Mechanism of injury</b>	Absence of event involving axial compression combined with flexion, rotation and/or a sudden axial impact load to the lumbar spine.
<b>Current history</b>	Does not present with immediate low back pain and documented functional impairment/disability.  Discontinuity of back pain with development of radiculopathy.  Able to continue participating in activities which load the lumbar spine in a way that would be expected to produce symptoms.  Unexplained mismatch between the history recorded in the ARTP and the contemporaneous medical records.

*Continued ...*

**Initial presentation** First documented clinical presentation to healthcare provider (<1 month).  
Clinical assessment findings consistent with lumbar disc pathology.

**General imaging features** Focal disc herniation causing compression of the nerve root and correlating with the clinical findings.

**Initial presentation** First documented clinical presentation. Delay in presentation (>1 month) without an adequate explanation for this delay.  
Clinical assessment findings inconsistent with lumbar disc pathology.  
Note: Delayed or unappreciated diagnosis of new leg pain from a lumbar disc injury can occur.

**General imaging features** Disc bulge without focal herniation.  
Multilevel disc disease and spondylosis.  
MRI and clinical evidence of spinal stenosis.  
Focal disc herniation causing compression of the nerve root and not correlating with clinical findings.  
Note: Latency of imaging record may be relevant (i.e. the time elapsed between the injury and imaging).

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## 2. Same-level fusion following lumbar disc injury

Where ACC has accepted that a client has sustained a traumatic lumbar disc injury (usually causing a radiculopathy), some of these clients may have persisting axial pain, and a subsequent fusion procedure may be required.

When determining ACC's liability to fund the treatment of lumbar disc pathology with a lumbar fusion, ACC Clinical Advisors must weigh the relative contributions from a covered physical injury to the disc against the presence of pre-existing lumbar disc pathology at that level. When considering the contribution of the covered physical injury, the lumbar disc injury consideration factors ([page 3](#)) apply.

**Table 2:** Factors to consider in decisions on same-level fusion following lumbar disc injury

**IMPORTANT:** The factors are not to be considered in isolation; rather the overall balance of factors that are more supportive or less supportive of a causal link must be considered.

### Factors MORE SUPPORTIVE of a causal link

**Mandatory:** The injury meets the criteria for sustaining lumbar disc injury as a result of the accident event. The fusion is required at the same level as the lumbar disc injury.

New single-level change that is seen on imaging subsequent to the event in a time frame consistent with causing that degenerative change.

Absence of a history of pre-existing symptoms and disability.

The absence of spondylolysis or spondylolisthesis.

### Factors LESS SUPPORTIVE of a causal link

Degenerative change at the time of the initial event and surgery at the level to be treated, e.g.:

- Disc degeneration
- Bony/spinal canal shape (developmental/constitutional)
- Facet joint hypertrophy
- Ligamentum flavum changes
- Osteophyte formation
- Spondylophyte formation
- Synovial (facet joint) cyst
- Annular fissure
- A combination of the above.

Presence of a history of pre-existing symptoms and disability.

The presence of spondylolysis or spondylolisthesis.

### 3. Adjacent segment disease following fusion

Adjacent segment disease (ASD) is a term describing symptomatic pathology at a level adjacent to a level that has undergone a spinal fusion. When a person has one or more levels of their spine fused surgically to treat disc pathology caused by a covered back injury, the adjacent disc level above or below the fused segment may be subject to extra load and stress because those levels are now the adjacent mobile segments.

Weighing up the relative contributions from the natural history of pre-existing degeneration with the effects of the fusion surgery on the symptomatic adjacent segment is integral to the assessment of likely causation.

The presence of significant pre-existing degeneration in the adjacent segment may preclude access to ACC funding. The assessment of adjacent segment degeneration will include the disc, facet joints and other relevant anatomical structures.

Significant disc degeneration is categorised as the equivalent of grades 6-8 on the modified Pfirrmann grading system for lumbar intervertebral disc degeneration (Table 3, Appendix C).

Significant facet joint degeneration is categorised as the equivalent of grades 2-3 on the system developed and validated by Weishaupt et al (1999) (Table 4, Appendix C).

ASD arising from non-ACC-funded fusions would not be ACC's responsibility unless the disease met the criteria for a treatment injury, or there was clear evidence of a new injury caused by a new accident affecting that adjacent segment and where the new accident or event was the more likely cause of the new pathology.

**Table 3:** Factors to consider in decisions on adjacent segment disease following fusion

**IMPORTANT:** The factors are not to be considered in isolation; rather the overall balance of factors that are more supportive or less supportive of a causal link must be considered.

#### Factors MORE SUPPORTIVE of a causal link

**Mandatory:** Previous lumbar fusion to address ACC-covered injury.

Previous imaging (at the time of the index fusion or injury) doesn't show any evidence of significant existing degenerative change at the adjacent segment.

#### Factors LESS SUPPORTIVE of a causal link

Evidence of significant pre-existing degenerative change at the level in question (adjacent to the fusion segment) at the time of the initial injury and surgery, e.g.:

*Continued ...*

## Factors MORE SUPPORTIVE of a causal link

Note:

- Significant disc degeneration is categorised as the equivalent of grades 6-8 on the modified Pfirrmann grading system for lumbar intervertebral disc degeneration (Table 3, Appendix C).
- Significant facet joint degeneration is categorised as the equivalent of grades 2-3 on the system developed and validated by Weishaupt et al (1999) (Table 4, Appendix C).

Exclusion of ongoing symptoms linked to the original fusion (e.g. non-union).

New symptom complex of back and leg pain representing new spondylosis and/or new nerve root compression consistent with ASD.

## Factors LESS SUPPORTIVE of a causal link

- Disc degeneration
- Bony/spinal canal shape (developmental/constitutional)
- Facet joint hypertrophy
- Ligamentum flavum changes
- Osteophyte formation
- Spondylophyte formation
- Synovial (facet joint) cyst
- Annular fissure
- A combination of the above.

Note:

- Significant disc degeneration is categorised as the equivalent of grades 6-8 on the modified Pfirrmann grading system for lumbar intervertebral disc degeneration (Table 3, Appendix C).
- Significant facet joint degeneration is categorised as the equivalent of grades 2-3 on the system developed and validated by Weishaupt et al (1999) (Table 4, Appendix C).

No factors identified.

Absence of a period of time without new current symptoms attributable to ASD, i.e. there has been little or no change in symptoms following previous lumbar fusion.

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The clinical representatives involved in the document development:

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## References and Bibliography

1. Adams MA, Freeman BJ, Morrison HP, Nelson IW, Dolan P. Mechanical initiation of intervertebral disc degeneration. *Spine*. 2000 Jul 1;25(13):1625–36.
2. Adams MA, Hutton WC. Prolapsed intervertebral disc. A hyperflexion injury 1981 Volvo Award in Basic Science. *Spine*. 1982 May–Jun;7(3):184–91.
3. Adams MA, Roughley PJ. What is intervertebral disc degeneration, and what causes it? *Spine*. 2006 Aug 15;31(18):2151–61.
4. Albert HB, Manniche C. Modic changes following lumbar disc herniation. *European Spine Journal*. 2007 Jul;16(7):977–82.
5. Alkhatib B, Rosenzweig DH, Krock E, Roughley PJ, Beckman L, Steffen T, et al. Acute mechanical injury of the human intervertebral disc: link to degeneration and pain. *European Cells & Materials*. 2014 Sep;28:98–110.
6. Brinjikji W, Luetmer PH, Comstock B, Bresnahan BW, Chen LE, Deyo RA, et al. Systematic literature review of imaging features of spinal degeneration in asymptomatic populations. *American Journal of Neuroradiology*. 2015 Apr;36(4):811–6.
7. Brock M, Patt S, Mayer HM. The form and structure of the extruded disc. *Spine*. 1992 Dec;17(12):1457–61.

### Disclaimer

All information in this publication was correct at the time of printing. This information is intended to serve only as a general guide to arrangements under the Accident Compensation Act 2001 and regulations. For any legal or financial purposes this Act takes precedence over the contents of this guide.



8. Fardon D, Williams A, Dohring E, Murtagh F, Rothman S, Sze G. Lumbar disc nomenclature: version 2.0: Recommendations of the combined task forces of the North American Spine Society, the American Society of Spine Radiology and the American Society of Neuroradiology. *The Spine Journal*. 2014 Nov 1;14(11):2525–45.
9. Griffith et al (2007) Modified Pfirrmann Grading System for Lumbar Intervertebral Disc Degeneration. *Spine*, 32(24), 708–712
10. Kanna RM, Shetty AP, Rajasekaran S. Patterns of lumbar disc degeneration are different in degenerative disc disease and disc prolapse magnetic resonance imaging analysis of 224 patients. *The Spine Journal*. 2014 Feb;14(2):300–7.
11. Lama P, Le Maitre CL, Dolan P, Tarlton JF, Harding IJ, Adams MA. Do intervertebral discs degenerate before they herniate, or after? *The Bone & Joint Journal*. 2013 Aug;95-B(8):1127–33.
12. Lama P, Zehra U, Balkovec C, Claireaux HA, Flower L, Harding IJ, et al. Significance of cartilage endplate within herniated disc tissue. *European Spine Journal*. 2014 Sep;23(9):1869–77.
13. Mannion AF, Adams MA, Dolan P. Sudden and unexpected loading generates high forces on the lumbar spine. *Spine*. 2000 Apr;25(7):842–52.
14. Osti OL, Vernon-Roberts B, Fraser RD. 1990 Volvo Award in experimental studies: Anulus tears and intervertebral disc degeneration: An experimental study using an animal model. *Spine*. 1990 Aug;15(8):762–7.
15. Ramani PS. Variations in size of the bony lumbar canal in patients with prolapse of lumbar intervertebral discs. *Clinical Radiology*. 1976 Jan;27(3):301–7.
16. Schmidt H, Kettler A, Heuer F, Simon U, Claes L, Wilke HJ. Intradiscal pressure, shear strain, and fiber strain in the intervertebral disc under combined loading. *Spine*. 2007 Apr 1;32(7):748–55.
17. Smartt P. Causation reviews: Spinal injuries and diseases. Health Services Assessment Collaboration. 2012. Retrieved from <https://www.acc.co.nz/assets/provider/886cobb5d6/Causation-reviews-spinal-injuries-and-diseases.pdf>
18. Tsuchiya K, Doi T, Oda Y, Iwamoto Y, Nakashima Y. Effect of cartilaginous endplates on extruded disc resorption in lumbar disc herniation. *PLoS One*. 2018 Apr;13(4).
19. Wade KR, Robertson PA, Thambyah A, Broom ND. How healthy discs herniate: A biomechanical and microstructural study investigating the combined effects of compression rate and flexion. *Spine*. 2014 Jun 1;39(13):1018–28.
20. Wade KR, Robertson PA, Thambyah A, Broom ND. “Surprise” loading in flexion increases the risk of disc herniation due to annulus-endplate junction failure. *Spine*. 2015 Jun 15;40(12):891–901.
21. Weishaupt, W, Zanetti, M, Boos N, Hodler J (1999) MR imaging and CT in osteoarthritis of the lumbar facet joints. *Skeletal Radiol*, 28:215–219
22. Willburger RE, Ehiosun UK, Kuhnen C, Krämer J, Schmid G. Clinical symptoms in lumbar disc herniations and their correlation to the histological composition of the extruded disc material. *Spine*. 2004 Aug 1;29(15):1655–61.

## APPENDIX A – Lumbar disc nomenclature – relevant terminology (Fardon et al, 2014)

- **Annular fissure** is to be used in the place of annular tear. Annular fissures are separations between the annular fibres or separations of annular fibres from their attachment to the vertebral bone. Use of the term ‘tear’ can be misunderstood because the analogy to other tears has a connotation of injury, which is inappropriate in this context. The term ‘fissure’ is the correct term. Use of the term ‘tear’ should be discouraged and, when it appears, should be recognised that it is usually meant to be synonymous with ‘fissure’ and not reflective of the result of injury. The term ‘tear’ is regarded as nonstandard usage.
- **Normal** defines discs that are morphologically normal, without the consideration of the clinical context and not inclusive of degenerative, developmental or adaptive changes that could, in some contexts (e.g. normal ageing, scoliosis, spondylolisthesis), be considered clinically normal.
- **Congenital/developmental variation** category includes discs that are congenitally abnormal or that have undergone changes in their morphology as an adaptation of abnormal growth of the spine, such as from scoliosis or spondylolisthesis.
- **Disc degeneration** – Degenerative changes in the discs are included in a broad category that includes the subcategories annular fissure, degeneration, and herniation. Degeneration may include any or all of the following: desiccation, fibrosis, narrowing of the disc space, diffuse bulging of the annulus beyond the disc space, fissuring (i.e. annular fissures), mucinous degeneration of the annulus, intradiscal gas, osteophytes of the vertebral apophyses, defects, inflammatory changes, and sclerosis of the endplates.
- **Desiccated disc** – Disc with reduced water content, usually primarily of nuclear tissues. Imaging manifestations of reduced water content of the disc, such as decreased (dark) signal intensity on T2-weighted images, or of apparent reduced water content, as from alterations in the concentration of hydrophilic glycosaminoglycans.
- **Disc space** – Space limited, cranial and caudal, by the endplates of the vertebrae and peripherally by the edges of the vertebral body ring apophyses, exclusive of osteophytes. Synonym: **intervertebral disc space**.
- **Herniation** is broadly defined as a localised or focal displacement of disc material beyond the limits of the intervertebral disc space. The disc material may be nucleus, cartilage, fragmented apophyseal bone, annular tissue, or any combination thereof. The disc space is defined cranial and caudal by the vertebral body endplates and, peripherally, by the outer edges of the vertebral ring apophyses, exclusive of osteophytes. The term ‘localised’ or ‘focal’ refers to the extension of the disc material less than 25% (90°) of the periphery of the disc as viewed in the axial plane. The presence of disc tissue extending beyond the edges of the ring apophyses, throughout the circumference of the disc, is called ‘**bulging**’ and is not considered a form of herniation. Asymmetric bulging of disc tissue greater than 25% of the disc circumference, often seen as an adaptation to adjacent deformity, is also not a form of herniation. In evaluating the shape of the disc for a herniation in an axial plane, the shape of the two adjacent vertebrae must be considered. Herniated disc may be classified as **protrusion** or **extrusion**, based on the shape of the displaced material.

- **Protrusion** is present if the greatest distance between the edges of the disc material presenting outside the disc space is less than the distance between the edges of the base of that disc material extending outside of the disc space. The base is defined as the width of the disc material at the outer margin of the disc space origin, where disc material displaced beyond the disc space is continuous with the disc material within the disc space.
- **Extrusion** is present when, in at least one plane, any one distance between the edges of the disc material beyond the disc space is greater than the distance between the edges of the base of the disc material beyond the disc space or when no continuity exists between the disc material beyond the disc space and that within the disc space. The latter form of extrusion is best further specified or subclassified as **sequestration** if the displaced disc has lost continuity completely with the parent disc. The term “**migration**” may be used to signify displacement of disc material away from the site of extrusion.
- **Modic classification (types I, II, and III)** – A classification of degenerative changes involving the vertebral endplates and adjacent vertebral bodies associated with disc inflammation and degenerative disc disease, as seen on MRIs. **Type I** refers to decreased signal intensity on T<sub>1</sub>-weighted spin-echo images and increased signal intensity on T<sub>2</sub>-weighted images, representing penetration of the endplate by fibrovascular tissue, inflammatory changes, and perhaps oedema. Type I changes may be chronic or acute. **Type II** refers to increased signal intensity on T<sub>1</sub>-weighted images and isointense or increased signal intensity on T<sub>2</sub>-weighted images, indicating replacement of normal bone marrow by fat. **Type III** refers to decreased signal intensity on both T<sub>1</sub>- and T<sub>2</sub>-weighted images, indicating reactive osteosclerosis.
- **Ruptured disc** – A herniated disc. The term ‘ruptured disc’ is an improper synonym for herniated disc, not to be confused with violent disruption of the annulus related to injury.

## APPENDIX B – Background prevalence of spine degeneration (Brinjikji et al, 2015)

**Table 1:** Estimated number of patients by age used to inform prevalence of degenerative spine imaging findings in asymptomatic patients<sup>1</sup>

Imaging Finding	Age (yr)						
	20	30	40	50	60	70	80
Disk degeneration	273 (9)	604 (16)	415 (12)	311 (10)	80 (4)	20 (2)	19 (2)
Disk signal loss	46 (2)	142 (5)	352 (4)	73 (2)	35 (1)	15 (1)	14 (1)
Disk height loss	15 (1)	163 (5)	186 (5)	208 (5)	35 (1)	15 (1)	14 (1)
Disk bulge	55 (4)	101 (7)	151 (8)	123 (7)	66 (5)	24 (3)	22 (3)
Disk protrusion	87 (5)	468 (14)	490 (14)	363 (12)	86 (5)	19 (2)	17 (2)
Annular fissure	167 (5)	350 (5)	426 (7)	53 (3)	35 (3)	15 (1)	14 (1)
Facet degeneration	0 (0)	0 (0)	596 (3)	53 (3)	35 (3)	15 (1)	14 (1)
Spondylolisthesis	0 (0)	0 (0)	31 (1)	53 (1)	35 (1)	15 (1)	14 (1)

**Table 2:** Age-specific Prevalence estimates of degenerative spine imaging findings in asymptomatic patients<sup>2</sup>

Imaging Finding	Age (yr)						
	20	30	40	50	60	70	80
Disk degeneration	37%	52%	68%	80%	88%	93%	96%
Disk signal loss	17%	33%	54%	73%	86%	94%	97%
Disk height loss	24%	34%	45%	56%	67%	76%	84%
Disk bulge	30%	40%	50%	60%	69%	77%	84%
Disk protrusion	29%	31%	33%	36%	38%	40%	43%
Annular fissure	19%	20%	22%	23%	25%	27%	29%
Facet degeneration	4%	9%	18%	32%	50%	69%	83%
Spondylolisthesis	3%	5%	8%	14%	23%	35%	50%

<sup>1</sup> The number of studies are in parentheses.

<sup>2</sup> Prevalence rates estimated with a generalized linear mixed-effects model for the age-specific prevalence estimate (binomial outcome) clustering on study and adjusting for the midpoint of each reported age interval of the study.

## APPENDIX C

**Table 3:** Modified Pfirrmann grading system for lumbar intervertebral disc degeneration (Griffith et al, 2007)

Grade	Signal from nucleus and inner fibers of anulus	Distinction between inner and outer fibers of anulus at posterior aspect of disc	Height of disc
1	Uniformly hyperintense, equal to CSF	Distinct	Normal
2	Hyperintense (>presacral fat and <CSF) +/- hypointense intranuclear cleft	Distinct	Normal
3	Hyperintense though <presacral fat	Distinct	Normal
4	Mildly hyperintense (slightly >outer fibers of anulus)	Indistinct	Normal
5	Hypointense (=outer fibers of anulus)	Indistinct	Normal
6	Hypointense	Indistinct	<30% reduction in disc height
7	Hypointense	Indistinct	30%-60% reduction in disc height
8	Hypointense	Indistinct	>60% reduction in disc height

Grades 1, 2 and 3 are based on the signal intensity of the nucleus and inner fibers of anulus. For Grade 4, the margins between the inner and the other fibers of the anulus at the posterior margin of the disc are indistinct. For Grade 5, the disc is uniformly hyperintense, although there is a loss of disc space height. For Grades 6, 7 and 8, there is progressive loss of disc space height. These could be broadly classified as mild, moderate, to severe loss of disc space height. Very occasionally, although obvious disc collapse is present, hyperintense signal from the nucleus and inner fibers of the anulus is preserved. This is referred to by a double entry, e.g. 4/7, with the former reporting the disc signal and the latter the degree of collapse.

**Table 4:** Grading scale developed and validated by Weishaupt (1999) for zygapophyseal (facet) joint degeneration

Grade	Weishaupt et al (1999) validated on CT and MRI
0	Normal facet joint space (2±4 mm width)
1	Narrowing of the facet joint space (< 2 mm) and/or small osteophytes and/or mild hypertrophy of the articular process
2	Narrowing of the facet joint space and/or moderate osteophytes and/or moderate hypertrophy of the articular process and/or mild subarticular bone erosions
3	Narrowing of the facet joint space and/or large osteophytes and/or severe hypertrophy of the articular process and/or severe subarticular bone erosions and/or subchondral cysts