

Adjacent Segment Disease

A patient guide

In brief: Adjacent segment disease describes the development of new symptoms at a mobile spinal level lying immediately next to a previous spinal fusion. A fusion deliberately stops movement across one or more vertebrae to relieve pain or stabilise the spine, but the levels above and below continue to move and bear load. Over time, some of these neighbouring levels show wear on imaging, and a smaller proportion go on to cause fresh pain, nerve symptoms or instability. The single most important idea in this whole subject is the distinction between mere wear seen on a scan, which is common and often silent, and genuine disease that produces symptoms, which is much less common. A great deal of confusion, and a great deal of unnecessary worry, comes from blurring these two very different things together.

What adjacent segment disease is (and the crucial distinction it rests on)

The spine is a column of vertebrae separated by discs, and it works as a chain of small joints that share movement and load between them. When a fusion is performed, two or more of these vertebrae are deliberately joined into a single solid block so that they no longer move relative to one another. This is done to settle pain coming from a worn or unstable level, to take pressure off nerves, or to correct a deformity. The fused segment stops moving; the segments immediately above and below it, by contrast, keep moving and keep carrying load, and it is these neighbouring, or adjacent, segments that the term refers to.

The entire field turns on a single distinction that is worth stating plainly at the outset, because it shapes everything that follows.

****Adjacent segment degeneration**** means changes seen on imaging at the level next to a fusion: narrowing of the disc, the formation of bony spurs, a small slip of one vertebra on another, thickening of the small joints, or changes in the signal of the disc on an MRI scan. These are structural, radiographic findings. They are extremely common as people age and as a fusion matures, and in a large proportion of cases they cause no symptoms whatsoever. A scan can look considerably worse than the person feels.

****Adjacent segment disease**** means something different and more specific: the appearance of **new symptoms**, such as recurrent back or neck pain, pain radiating into a limb, numbness or weakness, or the heavy-legged ache of spinal canal narrowing, that **correspond** to those changes at the adjacent level. Disease, in this technical sense, requires both the imaging change and a matching clinical problem.

The gap between the two is wide. Many segments degenerate on a scan and never trouble the person; only a minority become symptomatic. Because of this, modern reviewers increasingly use the umbrella term adjacent segment **pathology** to cover both, and then carefully specify whether they mean the radiographic kind or the clinical kind. Throughout what follows, this distinction is maintained, because reading a frightening scan report as though it were a diagnosis of disease is one of the commonest sources of avoidable alarm.

How it differs from other problems after a fusion

New symptoms appearing some time after a fusion can have several explanations, and adjacent segment disease is only one of them. Distinguishing it from its near neighbours is an important part of any assessment, because the explanations are managed quite differently.

****Recurrent or residual narrowing at the original level**** is the first to consider. This is a problem at the level that was actually operated on, rather than at a new neighbouring level. Scar tissue, incomplete decompression or a fresh disc protrusion at the index level can reproduce symptoms that resemble the original complaint. The distinguishing feature is that the imaging change sits at the operated level, not at the level next door.

****Pseudarthrosis, or non-union****, is failure of the intended fusion to knit solidly together. Instead of a single solid block, a small amount of persistent movement remains across the segment that was supposed to fuse. This can cause ongoing pain and, in time, loosening or breakage of the metalwork. Because its symptoms overlap with those of adjacent segment disease, careful imaging, often a CT scan and occasionally specialised nuclear-medicine scans, is used to tell them apart.

****Hardware-related discomfort**** from screws or rods, and, rarely, low-grade infection, are further possibilities that an assessment will weigh.

****Natural-history degeneration**** is the final and conceptually most important comparison, and it sits at the heart of the scientific debate described below. Degenerative change of the spine is a generalised, multilevel process driven by ageing, genetics and constitution. Some of what is seen at an adjacent level may simply be the continuation of a process that was always going to happen, fusion or no fusion. Separating the part of the change that the surgery may have accelerated from the part that would have occurred anyway is genuinely difficult, and is the subject of long-standing argument in the literature.

The genuine debate: stress transfer versus natural progression

One of the most interesting features of this topic is that its fundamental cause is not settled. There are two principal hypotheses, and the honest position, supported by most reviewers, is that both contribute and that no study has cleanly separated them.

The ****stress-transfer hypothesis**** is the mechanical argument. A fusion abolishes movement across the segment it joins. The movement and load that the fused segment can no longer accommodate must go somewhere, and the neighbouring mobile segments take up the slack. Laboratory studies on cadaver spines and computer models have shown that, after a fusion, the pressure inside the adjacent disc rises, the small joints are loaded more heavily, and the demand for movement at those levels increases. On this view, the fusion itself accelerates the wear of its neighbours, and the longer and more rigid the construct, the greater the effect should be.

The ****natural-progression hypothesis**** is the biological argument. Degenerative disease of the spine is rarely confined to one level; it is a diffuse process, and a person who needed a fusion at one level very often has discs elsewhere that are already predisposed to wear. On this view, the adjacent segment was going to degenerate regardless, and the fusion simply identifies a person whose spine is prone to degeneration. This argument is supported by the observation that people with degenerative disease who never have surgery show similar progression at neighbouring levels on serial scans, and by the inconsistent relationship in some studies between the length of a fusion and the rate of symptomatic disease.

A ****third strand**** concerns the surgery itself. Damage done to the adjacent level during the original operation, such as injury to the small joint capsule just above a fusion, disruption of the ligaments and muscles, or fusing the spine in a poorly balanced position, may set up the neighbouring level to fail. This part is at least partly avoidable, which is why careful technique and attention to spinal balance are emphasised.

The prevailing conclusion is that the truth lies somewhere in the middle: mechanical stress transfer, a pre-existing tendency to degenerate, and the effects of the surgery all play a part, in proportions that vary from person to person and that cannot be precisely disentangled. This uncertainty is not a weakness in the evidence so much as an honest reflection of how complex the problem is.

How often it happens

The figures most often quoted come from two landmark studies, one in the neck and one in the lower back, and they illustrate both the scale of the problem and the importance of the degeneration-versus-disease distinction.

In the neck, the seminal study by Hilibrand and colleagues, published in 1999, followed patients after anterior cervical discectomy and fusion. It found that new symptomatic disease at an adjacent level appeared at a rate of

roughly **three per cent per year**, and projected that around **a quarter of patients would develop symptomatic adjacent segment disease within ten years**, with about two-thirds of those eventually coming to further surgery. Later systematic reviews placed the annual rate broadly between one and a half and four per cent.

In the lower back, the landmark survivorship study by Ghiselli and colleagues, published in 2004, followed patients after lumbar fusion and estimated that the proportion needing further surgery for symptomatic degeneration at an adjacent level was about **sixteen per cent at five years and thirty-six per cent at ten years**. The level just above the fusion was affected more often than the level below.

Across the wider literature, pooled estimates suggest that radiographic degeneration at an adjacent level is common, on the order of a quarter to a third of patients or more, while symptomatic disease is considerably less frequent, in the region of one in ten, and reoperation for it less frequent still, in the region of one in twenty. The consistent message is that the scan changes outnumber the symptoms by a wide margin, and that symptomatic disease accumulates slowly over years rather than appearing as a single early event.

Risk factors

The literature identifies a fairly consistent set of factors associated with a higher likelihood of symptomatic adjacent segment disease, although none of these predicts the outcome for any individual.

Spinal balance, or sagittal alignment, is among the most important and is partly within a surgeon's control. The spine has natural front-to-back curves that keep the head balanced economically over the pelvis. When a fusion leaves the spine out of balance, for example with a mismatch between the curve of the lower back and the shape of the pelvis, or with the trunk tipped forwards, the neighbouring segments are loaded abnormally and the risk of disease rises. Restoring good balance at the original operation is therefore regarded as one of the most effective ways of protecting the adjacent levels.

The number of levels fused matters, with longer constructs, and in particular fusions of three or more levels, carrying a greater reported risk than single-level fusions.

Age and pre-existing wear are strongly linked. Older patients, and those whose neighbouring level already shows degeneration, narrowing or loss of disc height on imaging before surgery, are more likely to develop symptomatic disease. Pre-existing degeneration at the level destined to become the adjacent segment is one of the most powerful predictors in several series.

Injury to the adjacent level during surgery, such as violation of the small joint just above the fusion, and other factors including fusion extending down to the sacrum, osteoporosis, raised body weight and, in some reports, female sex, are also described.

The proximal-junctional overlap

In long fusions performed to correct spinal deformity, exactly the same idea appears under a different name at the top end of the construct, where it is called proximal junctional kyphosis and, in its more severe form, proximal junctional failure.

Proximal junctional kyphosis is a radiographic measurement: an increase in the forward angle at the junction just above the top of a long fusion beyond a defined threshold. Like adjacent segment degeneration, it is frequently a scan finding without symptoms.

Proximal junctional failure is the more serious, symptomatic counterpart: an actual mechanical breakdown at that junction, which may involve a fracture, the metalwork pulling out, or a slip of one vertebra on another, often with pain and sometimes with nerve symptoms, and frequently requiring revision surgery.

The parallel with adjacent segment degeneration and disease is exact: a common radiographic change on one hand, and a less common symptomatic failure on the other. The risk factors overlap too, including poor bone quality, raised body weight, advanced age, and a spine corrected into poor balance. Conceptually, proximal junctional

problems are best understood as adjacent segment pathology occurring at the top junction of a long deformity fusion, governed by the same principles of stress transfer and spinal balance.

What the evidence shows about motion preservation

If the stress-transfer hypothesis were the whole story, then replacing a worn disc with a moving artificial disc, rather than fusing it, ought to spare the neighbouring levels. This is the rationale behind disc replacement, also called arthroplasty, and the evidence for it is genuinely mixed, which is part of what keeps the underlying debate alive.

In the **neck**, several randomised trials and their pooled analyses have reported that cervical disc replacement is associated with fewer further operations and a lower rate of surgery at adjacent levels than fusion, together with preserved movement at the treated level. Against this stands an important counterpoint: a carefully conducted randomised trial known as the NECK trial reported that implanting an artificial cervical disc did **not** prevent adjacent segment disease compared with fusion. Some studies have also found no difference in the true rate of degeneration at the adjacent level between the two operations. The picture, in other words, is not one-sided.

In the **lower back**, analyses from regulated trials of lumbar disc replacement have suggested a protective effect on radiographic degeneration at the adjacent level, with less neighbouring wear where more movement is preserved at the treated level.

Taken together, the evidence indicates that preserving motion *may* reduce reoperation and radiographic change in carefully selected patients, but that the highest-quality trial evidence shows it does **not** reliably abolish clinical adjacent segment disease. That finding fits the view that degeneration is substantially driven by a person's underlying biology, not by the mechanics of the fusion alone. This is a description of what the trials report, not a recommendation for or against any particular operation.

What the evidence shows about management — impersonal

It is important to be clear that the evidence base for managing adjacent segment disease rests largely on retrospective series, cohort studies, biomechanical work and consensus, rather than on large randomised trials addressing this specific problem. Decisions in practice are therefore individualised, and what follows describes what the literature reports rather than guidance for any person.

Conservative, non-surgical care is the starting point for most people. The reported approach centres on pain relief, modification of activity, physiotherapy and core-strengthening rehabilitation, attention to body weight and bone health, and, in selected cases, targeted injections around the affected level, which can both ease symptoms and help to confirm where they are coming from. A substantial proportion of people settle with these measures, and the literature reserves surgery for those whose symptoms persist despite conservative care or whose nerve symptoms are progressing.

Surgical treatment, when it is considered, takes one of a few forms. Where the trouble is mainly compression of a nerve at a stable adjacent level, **decompression alone**, sometimes performed through minimally invasive or keyhole techniques, is described as offering good results with less disturbance than a larger operation. Where the adjacent level is unstable or malaligned, the traditional approach is to **extend the fusion** to include the affected level, often through the back. Approaches from the front or the side, which can add a level while sparing the muscles and ligaments at the back, are increasingly described as a way of reducing the burden of revision surgery. Where poor spinal balance has driven the problem, **restoring alignment** is emphasised.

A recognised limitation of extending a fusion is that it solves the problem at one level while creating a *new* adjacent level, which can in turn become symptomatic in time. This is one reason decisions are made carefully and individually, weighing the likely benefit against the prospect of simply moving the problem along the spine.

Outlook

The outlook in adjacent segment disease is best understood through the same distinction that runs through the whole subject. Radiographic degeneration at a level next to a fusion is common, accumulates with time, and in the great majority of cases causes no symptoms; on its own it is a finding rather than a disease. Symptomatic disease is much less frequent, and when it does occur it tends to build gradually over years rather than appearing suddenly.

For those who develop symptoms, conservative measures help many, and surgery, when needed, generally improves the symptoms, although it introduces a fresh neighbouring level and so the problem can recur further along the spine. The most consistent themes across the literature are that good spinal balance at the original operation is the most actionable way of reducing the risk, that the changes seen on a scan should not be mistaken for a diagnosis of disease, and that any individual outlook depends on the particular spine, the particular fusion and the particular pattern of symptoms, and is a matter for assessment by the treating team rather than something that can be read from a category alone.