

Pseudarthrosis (Failed Spinal Fusion)

A patient guide

In brief: Pseudarthrosis, meaning "false joint", is the failure of a spinal fusion to heal into a solid block of bone. When two or more vertebrae are fused, the aim is for the bone graft placed between them to grow across the gap and unite the segments permanently. When that bony bridge does not form, a persistent fibrous, mobile or partly mobile gap remains, and the fusion is said to have failed to unite, or to be a non-union. The single most important idea behind pseudarthrosis is that it is, at heart, a failure of bone healing. This sets it apart from the conditions it is most often confused with: breakage or loosening of the metalwork, wear at the level next to a fusion that did heal, and infection. These problems frequently travel together, but they are not the same thing, and telling them apart is the foundation of understanding the condition.

What pseudarthrosis is (and what makes it different)

A spinal fusion is, in essence, a controlled attempt to make the body weld two or more vertebrae into one. Bone graft is laid across the levels to be joined, and screws, rods, plates or cages are placed to hold everything still while that graft matures. Over the following months, in a successful fusion, the graft is gradually replaced by living, load-bearing bone that bridges the gap, and the operated segment becomes a single solid unit. Pseudarthrosis is what happens when this welding process does not complete: the bridge of bone fails to form, and a false joint, a zone of fibrous tissue or persistent micromotion, remains in its place.

The defining feature, and the one that distinguishes pseudarthrosis from its neighbours, is that it is a failure of **bone biology and bone healing**, not primarily a failure of metal. This distinction matters because the conditions it is most easily confused with are different in kind.

The first of these is **implant or hardware failure**, meaning breakage of a rod, fracture or loosening of a screw, or subsidence or migration of a cage. The metalwork is only ever intended to be a temporary internal splint, holding the spine still until the bone unites. It is not designed to bear load forever on its own. The crucial link, explored further below, is that hardware usually fails **because** the bone beneath it never fused, so that broken metal is often the visible signature of an underlying non-union rather than an independent problem.

The second is **adjacent segment disease**, which is degeneration and wear at the mobile level immediately next to a fusion that has, in fact, healed perfectly well. This is a problem of the altered mechanics imposed on neighbouring discs and joints, not a failure of the original fusion to unite. A fused level that has consolidated soundly can still be flanked by a neighbour that wears out over time, and that is a separate entity entirely.

The third is **infection**. A low-grade infection at the operated site can itself prevent bone from healing and can loosen metalwork, and it is therefore an important consideration whenever a fusion appears to be failing. But infection is a microbial problem with its own features and its own management, distinct from a simple mechanical or biological non-union.

Holding these four ideas apart, non-union, implant failure, adjacent segment disease and infection, is the single most useful framework for making sense of a fusion that is not behaving as hoped.

Why fusions fail to unite: biology versus mechanics

For a fusion to succeed, bone must heal across the gap, and bone healing has well-recognised requirements. It needs living cells capable of making bone, a scaffold for them to grow along, the biological signals that prompt bone formation, a good blood supply, and a mechanically stable environment in which to work. Pseudarthrosis arises when one or more of these requirements is not met, and the causes divide broadly into problems of

biology and problems of **mechanics**.

On the biological side, the most consistently reported and most important modifiable factor is **smoking**. Nicotine narrows small blood vessels and reduces the blood flow that delivers oxygen and nutrients to the healing graft, and it directly hampers the cells that build bone. The published evidence links smoking with markedly higher rates of non-union and worse outcomes after fusion, and it is the factor most often singled out in the literature. **Anti-inflammatory painkillers of the NSAID type**, taken in the early period after surgery, have been associated in several studies with higher rates of non-union and hardware failure, through their effect on the early chemical signals of healing, although the evidence here is not unanimous and appears to depend on dose and duration. **Diabetes**, particularly when blood sugar is poorly controlled, impairs the quality of new bone and the healing response. **Osteoporosis**, or thinned and weakened bone, both weakens the grip of the screws and reduces the quality of the bone available to fuse. Other recognised contributors include poor nutrition, obesity, long-term steroid use, inflammatory conditions such as rheumatoid disease, kidney disease, previous radiotherapy and older age.

On the mechanical side, the recurring theme is that bone will not bridge a gap that moves too much. **Multilevel fusions** carry a higher risk than single-level ones, simply because there are more surfaces that must all heal and a longer lever arm acting on the construct; multilevel fusion of the neck is a familiar example. **Long constructs**, fusions extending down to the sacrum and pelvis, and large deformity corrections all place high mechanical demands on the healing bone. **Inadequate fixation**, leaving too much motion at the graft, prevents union directly. The **choice and quantity of bone graft** also matters: graft taken from the patient's own body contains living bone-forming cells, whereas donor graft does not, and in some settings donor graft is associated with a higher non-union rate. Revision surgery in a previously operated, scarred fusion bed is biologically less favourable than surgery in untouched tissue.

The honest summary is that pseudarthrosis is rarely a matter of biology *or* mechanics alone. A fusion fails to unite because the body's capacity to heal has been impaired, because the mechanical environment was too unstable, or, most often, because of some combination of the two.

How often it happens

The reported frequency of pseudarthrosis varies enormously according to the type of operation, the number of levels and the population studied, and the figures below are drawn from the published literature as general descriptions rather than predictions for any individual.

In surgery for adolescent idiopathic scoliosis, non-union is uncommon, on the order of one to two per cent. In neuromuscular scoliosis it is somewhat higher, around two per cent, and in adult spinal deformity, where the constructs are long and the demands great, rates of around six per cent and above are reported. In the lower back, posterolateral fusion has been reported across a wide band from roughly three to twenty-five per cent, and older series of anterior lumbar interbody fusion reported still wider ranges, with modern techniques generally toward the lower end. In the neck, fusion is achieved in the great majority of single-level operations, with success rates around ninety-six per cent, falling somewhat for two-level and three-or-more-level procedures, so that non-union becomes more likely as more levels are joined. Across all of these, the consistent message is that the more levels are fused and the greater the mechanical and biological challenge, the higher the chance that union fails.

Why a non-union on a scan is not always a problem

One of the most important and least intuitive facts about pseudarthrosis is that the appearance of a non-union on imaging does not, by itself, mean there is a clinical problem. The published literature repeatedly makes the point that **a radiographic non-union and a symptomatic non-union are not the same thing**.

A substantial proportion of pseudarthroses, by some accounts up to around half, are **completely without symptoms**, discovered only because a scan happens to show that the bone has not fully bridged, or because a

piece of metalwork is seen to have loosened or broken, with no accompanying pain or disability. Some non-unions identified at one year after surgery go on to **consolidate by themselves** over the following year. And in the early period after surgery, a thin line of lucency around a graft or cage is common and, in the majority of cases, eventually fills in with solid bone, so that early lucency is not the same as failure.

The practical consequence of this dissociation is that imaging findings are interpreted in the light of how the operated person actually is. A non-union that is stable and causing no trouble is regarded very differently from one that is associated with persistent or returning pain, progressive deformity or broken metalwork. This is why the literature distinguishes so carefully between the radiographic finding and the clinical syndrome, and it is the reason that management hinges far more on symptoms than on the scan appearance alone.

How it tends to present

When pseudarthrosis does cause symptoms, the commonest is **pain**, typically felt in the region of the operated spine, often described as a deep, mechanical, activity-related ache that may have eased for a time after surgery and then returned, or that never fully settled. Because the operated level retains an element of abnormal movement, the pain is frequently worse with loading, with twisting and bending, and toward the end of the day, and may be relieved by rest. In some cases the first sign is not pain at all but a change seen on imaging, such as a broken rod or a loosening screw found at a routine follow-up.

Where a non-union is associated with progressive collapse or angulation of the construct, there may be a gradual change in posture or alignment. Less commonly, if shifting bone or hardware begins to encroach on nearby nerves, there may be a return of leg or arm symptoms, although the great majority of non-unions do not involve the nerves. It bears repeating that a significant proportion of non-unions produce no symptoms whatsoever and are found incidentally. None of these descriptions corresponds to any particular person's situation, and the presence, absence or cause of symptoms in any individual is a matter for direct assessment.

The link between broken metalwork and non-union

The relationship between hardware failure and pseudarthrosis is close enough to deserve explaining in its own right, because it is one of the most useful diagnostic ideas in the whole subject.

The metalwork placed at a fusion, the screws, rods, plates and cages, is best thought of as an internal splint. Its job is to hold the spine still and share the load while the bone heals. In a fusion that unites successfully, the load is gradually transferred from the metal to the newly formed bone, and once solid union is achieved the metalwork is largely unloaded and protected. The metal has, in effect, done its job and is no longer carrying the body's weight on its own.

When a fusion fails to unite, this protective transfer never happens. The metalwork is left to bear repeated, cyclical loading at a level that keeps moving, day after day, step after step. No metal can withstand this indefinitely, and over time it fatigues and fails. The result is a **fractured rod, a broken or loosening screw, a halo of lucency around a screw, or a cage that has subsided or migrated**. A broken rod in particular is regarded as a reliable pointer to an underlying non-union, because the rod has effectively been carrying all the load at a level that never fused.

The key conceptual point is that, in this common scenario, the broken metal is the **consequence and the visible signature of the non-union, not the disease itself**. For this reason it is widely held that hardware breakage or loosening should be treated as evidence of pseudarthrosis until proven otherwise. The two conditions are intimately linked, but the bone, not the metal, is the heart of the matter.

How it is assessed, and the limits of plain X-rays

The assessment of a possible non-union is shaped by the well-recognised shortcomings of the ordinary X-ray. Plain radiographs are **insensitive** for detecting whether a fusion has truly united. Metalwork casts shadows

that obscure the very graft one is trying to see, and a film that looks acceptable does not confirm that solid bone has bridged the gap. Plain films can suggest a problem, by showing broken hardware or a lack of bridging bone, but a normal-looking film cannot be relied upon to prove that a fusion has healed.

Dynamic, or flexion-extension, X-rays are taken with the spine bent forwards and backwards to look for movement at the operated level. Their logic is that a level that has truly fused should not move. In practice their value is limited: an absence of motion is reassuring and suggests union, but the presence of some motion does not reliably prove a non-union, the amount of movement that distinguishes a healed from an unhealed fusion is not firmly agreed, and the metalwork itself tends to mask whatever movement is present.

The **reference standard** among the scans is the **fine-cut CT**, a computed tomography scan acquired in thin slices and reconstructed in several planes. This is the investigation that comes closest to the truth, because it can show whether trabecular bone has genuinely bridged across the graft or interbody space, and it reveals lucency or haloing around screws, broken hardware, graft resorption and subsidence. Among the available imaging tests, fine-cut CT has the best diagnostic accuracy for non-union and is the test most relied upon to settle the question. Grading systems exist to describe what the CT shows, ranging from clear non-union, through an indeterminate "locked" appearance, to solid fusion with bridging bone across at least half of the fusion area.

The **absolute gold standard** remains direct surgical exploration of the fusion, but this is invasive and is not used as a routine test. Other investigations, such as certain nuclear medicine scans, can show the metabolic activity of bone but are used selectively and can themselves be misleading. Where infection is a possibility, blood tests and other investigations are used to address that separate question.

What the evidence shows about management

It is important to state at the outset that the evidence base for managing pseudarthrosis is built largely on retrospective series, registry data, biomechanical experiments and reliability studies, rather than on large, high-quality randomised trials. The recommendations that exist are therefore consensus-based and series-driven, and decisions in practice are individualised and shared between the treating team and the person concerned. What follows is a description of what the literature reports, not guidance for any individual.

The first and most fundamental division in management mirrors the division described above between the radiographic finding and the clinical syndrome. A **stable, asymptomatic non-union** is, in the great majority of cases, **observed** rather than operated upon. It is monitored with imaging over time, some such non-unions go on to consolidate of their own accord, and the literature describes attention to the modifiable factors that influence bone healing, including stopping smoking, control of diabetes, attention to bone health and nutrition, as part of optimising the situation. Surgery is not routinely undertaken simply because a scan shows that a fusion has not fully united, if the person is well and the construct is stable.

Surgery, in the form of revision fusion, is reserved in the literature for the symptomatic non-union, that is, one causing persistent or recurrent pain, progressive deformity, or hardware failure that is itself causing problems. The governing principle of revision is to improve **both the biology and the mechanics** that allowed the first fusion to fail. On the mechanical side, this typically means removing or revising the failed metalwork and applying more rigid, and often longer or circumferential, fixation, meaning fixation from both the front and the back of the spine, which is reported to achieve higher union rates than a single-sided approach. On the biological side, it means cleaning out the false joint, freshening the bone surfaces and adding new bone graft, often taken from the patient's own body, sometimes supplemented by biological bone-stimulating agents such as bone morphogenetic protein in selected, higher-risk cases. These agents are effective at promoting union but carry their own recognised complications and are used selectively, with particular caution noted in the literature regarding their use at the front of the neck. Throughout, the importance of optimising the host, and of excluding a low-grade infection as the underlying cause, is consistently emphasised.

In summary, the literature points toward observation for the stable, asymptomatic non-union, and revision surgery that improves both biology and fixation for the symptomatic one, all within a framework of individualised, shared decision-making rather than fixed rules.

Outlook

The outlook after pseudarthrosis varies as widely as its causes. For the large group of people whose non-union is stable and causes no symptoms, the outlook is generally good, and the situation is often one of monitoring rather than treatment, with some non-unions consolidating over time. The principal significance in this group is that the finding is noted and followed, and that the factors influencing bone health are addressed.

For those with a symptomatic non-union who undergo revision surgery, the published outcomes are broadly encouraging, particularly where the revision improves both the biology and the mechanics. Reported union rates are high after well-planned revision, especially with circumferential fixation and fresh bone graft, although revision surgery is inherently more demanding than the original operation, and a small proportion of revisions themselves fail to unite and require further attention. The literature consistently notes that addressing the underlying contributors, above all stopping smoking, gives the best chance of a sound result.

Across the whole field, the recurring themes are that the imaging finding must be read alongside the clinical picture, that broken metalwork is usually a clue to an underlying non-union rather than a problem in its own right, and that recovery is best understood as an individual matter shaped by the particular fusion, the particular biology and the particular mechanics involved, rather than something that can be read from a label alone.