

Spinal Implant Failure

A patient guide

In brief: Spinal implant failure is the mechanical breakdown of the metalwork used to hold the spine still after a fusion operation — the loosening, pulling out or breakage of pedicle screws, the fracture of the connecting rods, the migration or sinking of an interbody cage, or the loosening of the small set-screws and connectors that lock the construct together. The most important idea running through the whole subject is that this is a mechanical failure, intimately linked to but distinct from a failure of the bone to fuse. Spinal hardware is best understood as an internal splint engaged in a race against time: a race between the bone fusing solidly and the metal tiring out under repeated load. When a rod or screw breaks, it is usually not because the metal was faulty, but because that race was lost, and the broken metalwork is most often a signpost pointing to an underlying non-union beneath it.

What spinal implant failure is (and why it is mechanical, not biological)

When the spine is fused, the surgeon places metalwork — typically titanium or cobalt-chrome — to hold the vertebrae rigidly together while a bone fusion forms between them. The construct usually consists of pedicle screws anchored into the vertebrae, rods running between those screws, set-screws or caps that lock the rods into the screw heads, and often an interbody cage placed in the disc space at the front. This metalwork is sometimes described to patients as a permanent scaffold, but that description is misleading, and the misunderstanding sits at the heart of what implant failure actually is.

Spinal instrumentation is not a permanent load-bearing structure. It is a temporary internal splint. Its purpose is to hold the spine still long enough for living bone to bridge across the segment and fuse it solidly. Once that bony fusion has formed, the bone itself takes over the work of carrying load, and it shares the stress with the metal, protecting it. The metalwork then becomes largely redundant, quietly retired inside a spine that no longer needs it.

The difficulty arises if the bone does not fuse. In that situation the metal is left to carry the full cyclic load of every bend, twist and step, indefinitely and alone. No metal can do this forever. A material subjected to repeated stresses below its breaking point will eventually fail through a process called fatigue, which is the very same process that snaps a paperclip bent back and forth a few dozen times. This is the mechanical nature of implant failure, and it is what distinguishes it from a failure of biology.

It is worth stating plainly that implant failure and non-union are two different things that are nonetheless closely bound together. Non-union, also called pseudarthrosis, is a failure of the biology — the bone graft simply does not knit. Implant failure is the mechanical consequence that tends to follow. Because the two are so often found together, a broken rod or a broken screw is usually treated, in the literature, less as a problem in its own right and more as a clue that a non-union lies beneath it.

The race between fusion and fatigue

The most useful single way to think about spinal hardware is as a race, and the two competitors are fusion and fatigue. On one side, the bone is trying to fuse, a process that takes months. On the other side, the metal is accumulating microscopic fatigue damage with every load cycle, slowly working towards the point at which it will break. The outcome of the operation, in mechanical terms, depends on which competitor crosses the line first.

If the bone fuses before the metal fatigues, the construct is protected. The fused bone shoulders the load, the stress on the metalwork falls away, and the hardware survives intact and unremarkable for the rest of the person's life. This is the intended and by far the commonest course.

If, on the other hand, the metal fatigues before the bone fuses, the implant breaks. A rod fractures, or a screw snaps, or a screw works loose from its bony socket. Crucially, this breakage is rarely a sign that the metal was defective. In the great majority of cases it is a sign that the metal was asked to do the impossible — to carry load forever — because the fusion that should have relieved it never formed.

This is why the published literature is consistent on one point above all others: most rod and screw fractures are a marker of an underlying non-union rather than a primary fault of the implant. The discovery of broken hardware therefore tends to prompt a search for a non-union beneath it, rather than simply being recorded as a hardware problem. The metal lost the race, and the reason it lost is almost always that the bone failed to keep its side of the bargain.

How implant failure differs from its neighbours

Several distinct conditions are frequently confused with implant failure, and distinguishing them is central to understanding the subject. Each has a different mechanism, and conflating them obscures more than it clarifies.

****Pseudarthrosis, or non-union****, is the failure of the bone to fuse — a failure of biology. Implant failure is the mechanical sequel to it. The two are intimately linked, because broken metal is usually the visible signpost of a non-union hidden beneath, but they are not the same thing. One is bone not knitting; the other is metal breaking under the strain that the unknitted bone left it to bear.

****Screw malposition**** is a screw that was placed in the wrong position at the original operation, for example breaching the wall of the pedicle. This is a placement problem present from the very first day, not a process of loosening or fatigue that develops over months and years. The timing and the mechanism are entirely different.

****Adjacent segment disease**** is new wear and tear at the mobile level next to a fused segment, where the unfused neighbouring disc takes up extra strain over time. It is a disease of the bone and disc next door, not a failure of the metalwork, which in this situation typically remains perfectly intact.

****Infection**** deserves particular mention, because it can cause hardware to loosen. Bacteria and the inflammation they provoke resorb the bone immediately around the screws, so that a screw can work loose without any mechanical cause at all. For this reason, a loose screw that has no obvious mechanical explanation always raises the question of a low-grade infection.

****Cage subsidence**** is the sinking of an interbody cage into the soft bone of the vertebral endplate above or below it. Although it is often grouped under the heading of implant failure, it is mechanistically about the bone giving way rather than the device breaking, and it is therefore most strongly tied to bone quality.

Screw loosening, haloing and pull-out

Pedicle screws fail in a recognisable sequence. The earliest sign is loosening, in which a small amount of movement develops between the screw threads and the surrounding bone. This micromotion prevents new bone from forming against the screw; instead, the dead bone at the surface is resorbed and replaced by a thin layer of fibrous tissue. On imaging this shows up as a dark line, or halo, around the screw, and for this reason screw loosening is conventionally defined as a radiolucent zone of at least one millimetre in thickness around the screw, the so-called halo sign.

Once a screw has loosened, it loses its grip and can begin to toggle within the bone, a movement sometimes likened to a windscreen wiper, particularly at the screws at the top and bottom of a construct, which bear the highest leverage. With continued toggling, a screw may ultimately pull out of the bone altogether.

The dominant factor behind screw loosening and pull-out is the quality of the bone, and specifically osteoporosis. The strength with which a screw holds is directly related to the bone mineral density around it, so that screws in thinned, osteoporotic bone hold far less securely and loosen far more readily. Studies using opportunistic bone density measurement have found that patients with screw loosening have measurably lower bone density than

those without. This is why so much attention in the literature is paid to bone health, and why techniques such as cement augmentation, in which bone cement is injected to anchor the screw, have been developed to improve screw purchase in soft bone, reportedly increasing the force needed to pull a screw out very substantially.

Rod fracture

The rod is the long element that connects the screws and bears much of the bending load of the construct, and rod fracture is one of the most studied modes of implant failure. In large series of complex deformity operations, rods fracture in around one in fifteen patients overall, and the rate rises considerably after major corrective procedures such as a pedicle subtraction osteotomy, where reported rates range widely and reach much higher figures.

Rods do not break at random. They tend to fracture at two characteristic locations. The first is at or near the apex of a deformity correction or an osteotomy site, because this is where the bending stress on the rod is greatest. The second is at the lumbosacral junction, at the base of the spine, where the loads are highest and the demands on the metalwork most severe. Recognised risk factors for rod fracture include the presence of a non-union, a sharply contoured rod, the use of connectors or couplers along the rod, a construct that spans both the thoracolumbar and the lumbosacral junctions, smaller-diameter rods, and factors such as obesity and advanced age.

The relationship with non-union is again the central thread. An early rod fracture, occurring within the first year, tends to reflect a primary mechanical overload before fusion has had a chance to take, and may itself delay the union. A later rod fracture, occurring after the first year, is more often the result of metal fatigue accumulating in a construct where the fusion never solidified. Either way, a fractured rod points back towards the state of the underlying fusion.

Cage migration, subsidence and breakage

An interbody cage is a small spacer placed in the cleared disc space to restore height and to hold bone graft while the two vertebrae fuse across the front of the spine. Cages can fail in several ways. They can sink into the bone of the vertebra above or below, a process called subsidence, conventionally defined as the cage settling more than two millimetres into the endplate. They can slide out of position, called migration, and the most dangerous form of this is backward migration, or retropulsion, towards the spinal canal. Less commonly, a cage may itself break.

In series of interbody fusion in the lower back, migration without subsidence occurs in a small percentage of levels and migration with subsidence in a slightly larger percentage. The recognised risk factors echo those for screw failure and add some of their own. Osteoporosis greatly increases the risk, as does injury to the bony endplate during surgery, the use of a single cage rather than two, a larger cage, and a cage positioned too far back. As with subsidence generally, the common theme is that these are problems of the bone giving way around the device as much as failures of the device itself.

Set-screw and connector failure

A construct can also fail not at the bone and not in the middle of a rod, but at the joints between its own components. Each rod is locked into each screw head by a small set-screw or cap, and longer constructs may join rods together with connectors or couplers, sometimes called dominoes. If a set-screw loosens, the rod is no longer firmly gripped and can begin to slide or migrate within the screw head, with or without the rod itself breaking. In rare and extreme reports, a loosened rod has migrated a considerable distance.

These junction failures matter for two reasons. First, they represent a distinct failure mode that can occur even when the screws and rods themselves are sound. Second, the connectors used to join rods together are themselves a recognised risk factor for rod fracture, because they create points of concentrated stress. The literature also notes that, in some older stainless-steel systems, corrosion could develop at the junction between rod and screw where stagnant fluid was trapped, whereas titanium implants tend to show wear rather than corrosion.

When broken hardware is silent, and when it speaks

One of the most important and most reassuring points about implant failure is that broken hardware is not automatically a problem. A great deal depends on whether the spine beneath the metalwork has fused.

If the spine has fused solidly, the bone is carrying the load and the metalwork has become redundant. In that setting, a rod or screw that breaks may cause no symptoms at all and may be discovered only by chance on a scan taken for another reason. Series of patients with rod fractures at the base of the spine have described cases that were entirely without symptoms, all with a fusion confirmed on a CT scan. The broken metal, in these cases, is simply a passenger in a spine that no longer needs it.

The picture is different when the hardware fails and the spine has not fused. Here the broken metal usually announces itself, with a return of pain at the operated level, sometimes a sensation of movement or a click, and in some cases a gradual loss of the correction that the operation achieved. In one notable series, only the symptomatic rod fractures came to surgery, and every one of those was found to have a non-union at the time of revision, while the symptomless ones included cases with a solid fusion. This is the practical expression of the central idea: a broken implant that hurts is usually telling of a non-union beneath it, whereas a broken implant in a fused spine is often saying nothing at all. None of this describes any particular person's situation, which is always a matter for direct assessment.

How implant failure is assessed

The assessment of suspected implant failure rests on imaging, and on a particular question that imaging is asked to answer: not merely whether the metal has broken, but whether the bone beneath it has fused.

****Plain X-rays**** are the first-line investigation. They readily show a frankly broken rod or screw, a migrated cage, or the halo of a loosened screw, and films taken bending forwards and backwards can reveal abnormal movement at a level that should be fused. Their limitation is that they are relatively insensitive to early loosening and cannot reliably confirm a fusion.

****Computed tomography, the CT scan****, is the principal advanced investigation. It defines the fracture line through the metal, demonstrates the lucent halo around a loosened screw, shows the exact position of a cage, and, most importantly, is the best test for determining whether a solid bony fusion has actually formed across the segment. Because the management of broken hardware turns so heavily on the state of the fusion, this is the question that CT is most often called upon to settle. Peri-screw lucency on CT is regarded as the single best indicator of screw loosening.

****Magnetic resonance imaging**** is more limited in this setting because the metal distorts the image, but with modern techniques it is used to assess the surrounding soft tissues, to look for nerve compression, and, with contrast, to investigate the possibility of infection as a cause of loosening. Where a low-grade infection is suspected, blood tests for inflammation and, occasionally, sampling of the area may be added.

What the evidence shows about management — impersonal

It should be stated at the outset that the evidence base for managing implant failure is built on retrospective series, biomechanical studies and registry data rather than on large randomised trials. The descriptions that follow report what the literature documents, and are not guidance for any individual, whose circumstances are a matter for assessment by the treating team.

****Observation**** is the approach most often described for the broken implant that is causing no symptoms in a spine that has fused solidly. In this situation the fusion is doing the work, the metalwork is redundant, and the literature documents that such broken hardware is frequently simply monitored rather than removed. The rate at which hardware failure leads to further surgery is, across series, low.

****Revision surgery**** is described where the failure is symptomatic, where a non-union is confirmed or strongly suspected, where the deformity is progressing, or where there is instability or pressure on the nerves. The

techniques reported include replacing the broken rod, often with a stronger or larger-diameter one, adding extra rods to share the load, providing additional support at the front of the spine through an interbody fusion, revising and re-grafting the fusion bed itself, and, in osteoporotic bone, augmenting or upsizing the screws and using cement to improve their hold. Running through all of these is the principle that revision for a broken implant is, in essence, revision for the non-union beneath it.

Alongside surgery, the literature consistently emphasises the importance of addressing the factors that allowed the failure in the first place, in particular the assessment and treatment of osteoporosis and the cessation of smoking, both of which influence whether a fusion will succeed. In summary, the evidence points towards observation for asymptomatic failure in a fused spine, and revision directed at the underlying non-union for symptomatic failure, all within a framework of individualised, shared decision-making.

Outlook

The outlook after implant failure depends almost entirely on the state of the fusion beneath the broken metal. Where the spine has fused and the failure is asymptomatic, the outlook is generally good; the broken hardware is a redundant passenger, the fusion is secure, and intervention is frequently unnecessary. Where the failure reflects a non-union, the outlook is bound up with that non-union, and recovery tends to follow successful revision and the eventual achievement of a solid fusion.

Across the field, the consistent themes are that broken hardware should be interpreted in the light of the underlying fusion rather than in isolation, that osteoporosis and smoking are recurring contributors that reward attention, and that the great majority of hardware failures, particularly the asymptomatic ones, never require further surgery. The variation between individuals is wide, and any individual outlook is a matter for assessment by the treating team rather than something that can be read from a category alone.