

Burst Fracture

A patient guide

In brief: A thoracolumbar burst fracture is a high-energy injury in which a vertebra in the lower part of the spine is crushed from front to back, so that the back wall of the bone breaks and fragments can be driven into the spinal canal. It is more serious than a simple compression fracture because the middle of the vertebra fails rather than the front alone. The way the injury behaves, and the treatment it tends to attract, depend on whether the spine remains stable and whether the nerves are involved.

What a burst fracture is

A burst fracture is a break of a vertebral body caused by compressive load passing down the long axis of the spine. The vertebra does not simply wedge at the front, as it does in a straightforward compression injury; instead it is crushed through its full depth so that the back (posterior) wall of the bone gives way as well. Because the posterior wall forms the front boundary of the spinal canal, fragments of bone can be pushed backwards into the canal, a phenomenon called retropulsion. The word "burst" captures this outward spread of bone in several directions at once.

These injuries cluster at the thoracolumbar junction, the region between roughly the eleventh thoracic and second lumbar vertebrae (T11 to L2). This is a transition zone where the stiff, rib-supported thoracic spine, which is naturally curved into a gentle forward bend (kyphosis), meets the mobile lumbar spine, which curves the other way (lordosis). Mechanical stress concentrates at this changeover point, which is why a large proportion of vertebral fractures from falls and collisions occur there.

The clinical significance of a burst fracture lies in two questions that run through everything written below: is the spinal column still stable, and are the nerves affected? A vertebra that has burst but remains aligned, with the supporting ligaments intact and no pressure on the nervous tissue, behaves very differently from one in which the ligaments have torn, the spine has begun to angulate, or the spinal cord or nerve roots are being compressed.

The three-column idea and what "burst" means

Modern understanding of these injuries rests on the three-column concept described by Francis Denis in 1983, which built on the earlier two-column model of Holdsworth. In this scheme the spine is divided, from front to back, into three columns:

- The **anterior column** comprises the anterior longitudinal ligament, the front half of the vertebral body and the front part of the disc.
- The **middle column** comprises the back half of the vertebral body, the back part of the disc, and the posterior longitudinal ligament that runs along the rear surface of the vertebral bodies.
- The **posterior column** comprises the bony arch behind the canal (the pedicles, facet joints, laminae and spinous process) together with the posterior ligamentous complex: the supraspinous and interspinous ligaments, the ligamentum flavum and the facet joint capsules.

Within this framework a burst fracture is defined by failure of the **anterior and middle columns** under compression. The crucial element is the middle column: it is the involvement of the posterior vertebral wall, and the retropulsion of bone that can follow, that separates a burst fracture from lesser injuries. The middle column is sometimes described as the keystone of stability, because once it fails the spine loses an important part of its load-bearing capacity and its protection of the canal.

Whether the posterior column, and in particular the posterior ligamentous complex, has also been disrupted is the pivotal modifier. A burst fracture with an intact posterior ligamentous complex tends to behave in a stable fashion; one in which that complex is torn loses its tension band at the back and is far more likely to be unstable. This single distinction influences much of the assessment and classification described later on this page.

How it differs from a compression fracture and other injuries

A burst fracture sits within a family of thoracolumbar injuries that are best understood by which columns fail and how.

A **simple (wedge) compression fracture** involves the **anterior column only**. The front of the vertebra wedges down under flexion or axial load, but the middle column is intact, the posterior wall is preserved and there is no retropulsion. Because the canal is undisturbed, these injuries are generally stable and rarely involve the nerves. The defining contrast with a burst fracture is the integrity of that posterior wall: in a compression fracture it holds; in a burst fracture it breaks.

A **Chance, or flexion-distraction, injury** is fundamentally different in mechanism. Rather than being crushed, the spine is pulled apart at the back while hinging at the front, classically described in lap-belt restraint during a collision. The posterior and often the middle column fail in tension, producing a horizontal split through bone, ligament or both, with gapping of the posterior elements. The hallmark is distraction (pulling apart) rather than compression, and these injuries carry a notable association with intra-abdominal injury.

A **fracture-dislocation** is the most severe pattern, with failure of **all three columns** accompanied by translation or rotation, so that one vertebra is displaced on the next. This shear-type injury is the most unstable of all and carries a very high rate of neurological damage. The distinguishing feature is displacement of one vertebral segment relative to another, which does not occur in an isolated burst fracture.

In short, the burst fracture is the compression injury that breaches the middle column and threatens the canal, distinct from the anterior-only compression fracture, from the tension-based Chance injury, and from the translational fracture-dislocation.

How it happens

The typical mechanism is axial compressive loading of a spine held in a neutral or slightly flexed position, transmitting force straight down the vertebral column until a vertebra fails through its depth. The energy involved is usually high. Falls from a height landing on the feet or buttocks, road traffic collisions and high-energy sport are the common causes in people with normal bone. Because the thoracolumbar junction is the mechanical changeover point of the spine, it absorbs a disproportionate share of this load and is the most frequent site.

The same fracture pattern can occur at much lower energy when the bone itself is weakened. In osteoporotic bone, or where the vertebra is affected by a tumour or other pathological process, a relatively minor load may be enough to produce a burst configuration. The pattern of the fracture is similar, but the context, the underlying bone quality and the wider clinical picture differ.

How it tends to present

The usual presentation is localised back pain at the level of the injury, with tenderness over the affected vertebra and spasm of the paraspinal muscles. Pain is typically worse on movement and on attempting to bear weight through the spine. A palpable gap between the spinous processes, or bruising over the back, can point towards disruption of the posterior column and its ligaments.

Neurological features vary widely and depend on the level of the injury and the degree to which the canal is compromised. At one extreme there is no neurological involvement at all. Beyond that, there may be radicular symptoms from a single compressed nerve root, or, where the cord, conus medullaris or cauda equina is affected, an incomplete or complete neurological deficit with weakness, sensory change, or disturbance of bladder and bowel function. Because these injuries are high-energy, other fractures elsewhere in the spine and injuries to the chest and abdomen are not uncommon and are routinely looked for.

How it is assessed (CT for bone/retropulsion, MRI for the posterior ligaments and cord)

Assessment draws on more than one imaging method, each answering a different question.

Plain radiographs give a first overview of alignment, the amount of vertebral height lost, widening between the pedicles and the degree of kyphotic angulation at the injured level. They are a starting point rather than the definitive study.

Computed tomography (CT) is the reference standard for bony detail. It shows the posterior wall fracture clearly, quantifies the degree of retropulsion and canal compromise, and demonstrates the comminution (the number and spread of fragments) and the displacement of those fragments. These bony features feed directly into several of the classification systems described below, and the cross-sectional area of the canal can be measured to express the degree of narrowing.

Magnetic resonance imaging (MRI) is the reference standard for the soft tissues. It assesses the spinal cord, conus and cauda equina, showing oedema, compression or haemorrhage within the nervous tissue, and it evaluates the integrity of the posterior ligamentous complex, where bright signal on fluid-sensitive sequences suggests that the ligaments have been disrupted. Because posterior ligamentous complex integrity is a core variable in the modern scoring systems, MRI often carries particular weight, although the literature acknowledges that the precision and reproducibility of MRI for ligamentous injury remain a subject of debate.

How stability is judged (Denis, TLICS, Load-Sharing, AO Spine)

Several classification systems have been developed to describe these injuries and to express how stable they are. Each emphasises different features.

The **Denis three-column classification** provides the conceptual foundation, defining the burst fracture as compressive failure of the anterior and middle columns and framing instability in terms of how many columns have failed. Denis also described descriptive subtypes of burst fracture according to which endplate is involved and whether there is rotation.

The **Magerl, or AO, comprehensive classification** of 1994 organises injuries by mechanism into type A (compression), type B (distraction) and type C (rotation or translation), with increasing severity across the groups. Burst fractures fall within the compression group as type A3 and A4 patterns.

The **Load-Sharing Classification** of McCormack, Karaikovic and Gaines scores three features of the fracture, the degree of comminution, the spread of the fragments and the amount of kyphosis to be corrected, on a scale from three to nine points. It was designed to predict whether a short-segment posterior fixation alone is likely to be sufficient, with higher scores suggesting that additional anterior support may be required.

The **Thoracolumbar Injury Classification and Severity Score (TLICS)**, described by Vaccaro and colleagues in 2005, combines three elements: the morphology of the fracture, the integrity of the posterior ligamentous complex and the neurological status of the patient. The points are summed, with lower totals pointing towards non-operative management and higher totals towards surgery, and an intermediate band regarded as equivocal. Its explicit inclusion of the ligaments and the nerves marked a shift away from purely bony description.

The **AO Spine Thoracolumbar Classification** of 2013 modernised the A, B and C scheme with subtypes (the A3 and A4 categories being burst patterns), a neurological modifier and case-specific modifiers. A related surgical algorithm, sometimes expressed as the TL AOSIS score, converts the classification into a points-based treatment recommendation. Across all these systems the recurring themes are the same: the state of the posterior ligamentous complex, the neurological picture and the degree of deformity.

What the evidence shows about management (non-operative vs surgery) — impersonal

The central question in the literature is whether a burst fracture that is stable and not associated with any neurological deficit should be treated with surgery or without it.

A burst fracture is broadly regarded as **stable** when the person is neurologically intact, the posterior ligamentous complex is intact, the kyphosis is modest and the comminution limited. It is regarded as **unstable** where there is a significant or progressive neurological deficit, disruption of the posterior ligamentous complex, marked or progressive kyphotic deformity, extensive comminution, or any translational element of the kind seen in a fracture-dislocation.

For the **neurologically intact, stable burst fracture**, the best randomised evidence does not favour surgery. The trial by Wood and colleagues, first reported in 2003 and followed up at sixteen to twenty-two years in 2015, found no major advantage for operative over non-operative treatment; at long-term follow-up the non-operatively treated group reported less pain and better function, while surgery carried higher complication rates and greater cost. A Cochrane systematic review concluded that the evidence is insufficient to recommend surgery and noted the higher complication burden associated with operative treatment. A meta-analysis by Gnanenthiran and colleagues likewise found no difference in pain, disability or return to work, again with more complications after surgery. A multicentre randomised trial by Siebenga and colleagues is the notable dissenting study, reporting better short-term outcomes after surgery.

The role of bracing has also been examined. Randomised data from Bailey and colleagues found that managing an AO type A3 burst fracture without neurological deficit using no orthosis was equivalent to using a thoracolumbosacral orthosis, which has called the routine use of bracing into question.

The relationship between canal narrowing and outcome is less direct than might be expected. The degree of canal compromise measured on CT correlates only weakly with the presence of neurological deficit and does not reliably predict neurological recovery. Furthermore, retropulsed fragments tend to resorb spontaneously and the canal remodels over the following one to two years when the fracture is managed without surgery, so canal narrowing on its own is not regarded as an indication for operating in a person who is neurologically intact.

Where surgery is indicated, the literature addresses technique rather than the decision to operate. The Load-Sharing score helps guide the choice between anterior and posterior approaches, and a randomised study by Dai and colleagues found that adding a fusion conferred no advantage over fixation without fusion in short-segment posterior surgery.

Taken together, the current evidence supports non-operative management of the stable, neurologically intact burst fracture, with surgery reserved for instability, in the form of posterior ligamentous complex disruption, fracture-dislocation or progressive deformity, or for a significant or progressive neurological deficit. Such decisions are individualised and made by the treating spinal team in the light of the whole clinical picture.

Outlook

The outlook for a burst fracture depends heavily on which of the patterns described above is present. A stable, neurologically intact burst fracture managed without surgery generally has a favourable long-term course, with the bone healing, the retropulsed fragments resorbing and the canal remodelling over time; the long-term randomised data describe good function and modest pain in this group. Where the spine is unstable or the nerves are involved, the picture is more complex and the recovery less predictable, shaped by the severity of any neurological injury and by the demands of surgery and rehabilitation. Across the spectrum, the consistent message of the evidence is that the burst fracture is a heterogeneous injury whose prognosis is set chiefly by stability and by neurological status rather than by the fracture label alone.