

Osteoporosis and the Spine

A patient guide

In brief: Osteoporosis is a condition in which bone becomes thinner, more porous and mechanically weaker, so that it fractures under loads that healthy bone would withstand. Although it affects the whole skeleton, the spine is where it most often declares itself, because the vertebral bodies are made largely of the spongy, honeycomb-like bone that osteoporosis erodes first. The characteristic spinal event is the osteoporotic vertebral compression fracture, in which a vertebra crumples and loses height, frequently after a trivial strain or no obvious injury at all. These fractures are the commonest fragility fracture of all, and they tend to come not singly but in a cascade, each one increasing the load on its neighbours and the risk of the next. Over time, repeated fractures round the upper back into a forward-stooped curve, the kyphosis sometimes called a dowager's hump, and the person loses height. Beyond the fractures themselves, low bone density is the backdrop against which all spinal surgery is planned, because weak bone holds screws less securely, allows cages to sink and underlies several of the ways that spinal instrumentation can fail. The single most important idea is that osteoporosis is a disease of bone strength, and the spine is both its commonest victim and the place where that weakness most complicates treatment.

What osteoporosis is, and why the spine bears the brunt

Bone is not the inert scaffolding it is sometimes imagined to be. It is living tissue, constantly being broken down and rebuilt, and its strength depends on both how much bone is present and how well its internal architecture is arranged. Osteoporosis is the state in which this balance has tipped towards loss: the bone becomes thinner, its internal struts grow sparse and disconnected, and the whole structure loses the capacity to bear load. The result is bone that fractures under forces a healthy skeleton would shrug off, which is why these are called fragility fractures.

The spine is especially vulnerable, and the reason lies in its anatomy. The bulk of each vertebral body is made of trabecular bone, the spongy, lattice-like inner bone whose fine struts are turned over rapidly and lost early in osteoporosis. The dense outer shell, the cortical bone, is comparatively thin. When the internal lattice thins and its struts lose their connections, the vertebral body is left as a weakened box that can buckle under ordinary, everyday loads. This is why the spine is so often the first part of the skeleton to fracture in osteoporosis, and why a vertebral fracture is frequently the event that reveals the disease for the first time.

This framing matters for everything that follows. The same weakening of trabecular bone that lets a vertebra crumple also lets a surgical screw work loose, lets an interbody cage sink into the bone, and undermines the platforms on which spinal reconstruction depends. Osteoporosis, in other words, is not only a cause of fractures in its own right; it is the condition of the bone that shapes how the spine behaves under any load, surgical or natural.

Osteoporotic vertebral compression fractures: the commonest fragility fracture

The osteoporotic vertebral compression fracture is the signature spinal manifestation of the disease, and it is the most frequent of all fragility fractures. It occurs when a weakened vertebral body collapses, losing height at the front, at the back, or throughout. Because the front of the vertebra often gives way more than the back, the bone is left wedge-shaped, which tilts the spine forwards at that level.

What surprises many people is how little force is required. A vertebral fracture in osteoporotic bone may follow a minor stumble, a heavy lift, a vigorous cough or sneeze, or sometimes no identifiable event at all, the bone simply yielding under the load of daily life. This is the defining feature of a fragility fracture: the injury is out of all proportion to the force that caused it.

The fractures are also frequently silent. A substantial proportion of osteoporotic vertebral fractures are not recognised at the time, either because they cause no pain or because the pain is attributed to ordinary backache. Many are discovered only incidentally, when a chest X-ray or other scan taken for an unrelated reason shows that a vertebra has lost height. Others announce themselves with sudden, sharp back pain at the level of the fracture, often worse on movement and on standing, and easing with rest. The thoracolumbar junction, where the relatively stiff chest spine meets the more mobile lower back, is a particularly common site.

The fracture cascade and progressive kyphosis

One of the most important features of osteoporotic spinal fractures is that they tend not to occur in isolation. A single vertebral fracture is among the strongest predictors of further fractures, and the risk of a second fracture rises sharply, often within the first year after the first. This clustering is sometimes called the fracture cascade.

The mechanics behind it are straightforward. When a vertebra collapses into a wedge, the spine above it tips forwards, and the centre of gravity of the upper body shifts ahead of the spine. This increases the bending load on the vertebrae below, which are themselves weakened by the same osteoporosis, making them more likely to fracture in turn. Each new fracture deepens the forward curve and adds to the load on the next vertebra down, so that fractures can accumulate in a self-perpetuating sequence.

The visible consequence is progressive kyphosis, the rounding of the upper back into a forward stoop. In its advanced form this is the curvature historically known as a dowager's hump. Alongside it comes measurable height loss, as each collapsed vertebra subtracts a little from the total length of the spine; a loss of several centimetres of height over time is a recognised marker of accumulated vertebral fractures. The forward curve also has knock-on effects: it can crowd the abdominal organs, reduce the space available to the lungs, alter balance and gait, and shift the head forward of the body in a way that strains the muscles of the neck and back. The progressive nature of this deformity, driven by the cascade of fractures, is what distinguishes the osteoporotic spine from a single isolated injury.

Pain, height loss and the wider toll

The pain of an acute vertebral fracture is often severe at first, concentrated at the level of the break and provoked by movement, by standing and by any jolt. In most cases this acute pain settles over a period of weeks to a few months as the fracture heals and the bone stabilises, much as a fracture elsewhere in the body would. For many, the episode resolves and the bone consolidates in its new, compressed shape.

For others, the consequences are more enduring. Chronic back pain can persist after the acute episode, arising not only from the healed fracture itself but from the altered mechanics of a spine that has lost height and tipped forwards. The muscles of the back work harder to hold the body upright against the forward curve, and this sustained effort is itself a source of fatigue and aching. Where multiple fractures have produced significant kyphosis, the deformity can interfere with breathing, with appetite and digestion through pressure on the abdomen, and with the simple ability to stand and walk comfortably.

There is also a wider toll that is easy to underestimate. Vertebral fractures are associated, across large population studies, with reduced quality of life, with loss of independence, and with a measurable increase in mortality, particularly where fractures are multiple. This is not because a spinal fracture is directly life-threatening in the way a hip fracture can be, but because it is a marker of frail, weakened bone and of the cascade of events, immobility, deconditioning and further fractures, that can follow. Understanding the vertebral fracture as a sentinel event, rather than an isolated nuisance, is central to the way it is regarded.

How bone density is measured: DEXA, T-scores and beyond

The standard measure of bone strength is bone mineral density, and the standard means of measuring it is dual-energy X-ray absorptiometry, the DEXA or DXA scan. This is a quick, low-dose X-ray examination, usually of the lumbar spine and the hip, that measures how much mineral is packed into a given area of bone. The result is

expressed as a T-score, which compares the measured density with that of a healthy young adult. By international convention, a T-score down to minus one is regarded as normal, a score between minus one and minus two and a half as low bone mass, or osteopenia, and a score of minus two and a half or below as osteoporosis. A person who has already sustained a fragility fracture is often considered to have established osteoporosis regardless of the precise number.

DEXA has well-known limitations in the spine. It measures density across an area rather than through a volume, and it is confounded by the very things common in older spines: the dense bone of arthritis, the calcification of the aorta in front of the spine, and previous fractures all artificially raise the apparent density and can mask underlying weakness. For this reason, attention has turned to other ways of judging bone quality, particularly in the context of surgery.

One increasingly used approach takes advantage of CT scans performed for other reasons. The density of the vertebral bone on a CT scan can be measured in units called Hounsfield units, and this opportunistic measurement correlates with bone strength and has been shown to predict surgical problems such as screw loosening and cage sinking. A related development is the vertebral bone quality score, derived from the signal of the vertebrae on a standard MRI scan, which similarly estimates bone quality without any additional test. These tools are not replacements for DEXA in diagnosing osteoporosis, but they give a more directly relevant picture of the bone a surgeon would actually be working with, and they are part of the reason bone quality is now assessed in several different ways rather than by a single number.

Vertebroplasty and kyphoplasty: the procedures and the controversy

When a painful vertebral fracture does not settle with time and supportive measures, two minimally invasive procedures exist that aim to stabilise the broken bone. In vertebroplasty, bone cement is injected through a needle directly into the fractured vertebral body, where it hardens and splints the bone from within. In balloon kyphoplasty, a small balloon is first inflated inside the vertebra to create a cavity and, in some cases, to partially restore lost height, before the cavity is filled with cement. Both are aimed primarily at relieving the pain of the fracture and allowing earlier movement.

These procedures are among the most debated interventions in spinal medicine, and the debate is instructive. Early observational studies and open trials reported dramatic and rapid pain relief, and on that basis the procedures became widely used. Then, in 2009, two influential trials published in the *New England Journal of Medicine* compared vertebroplasty against a sham procedure, in which patients underwent an identical-seeming intervention but no cement was actually injected. Both found no significant difference in pain relief between real and sham treatment, a result that called the whole practice into question and sharply reduced its use in many places.

The picture has not stayed that simple. Subsequent trials, including the VERTOS series, particularly those that treated fractures earlier and selected patients whose fractures were confirmed to be acute and painful on MRI, did report benefit over conservative care. The evidence is therefore genuinely mixed, and it is best understood as showing that the effect depends heavily on which fractures are treated, how acute they are, and what they are compared against. Major guideline bodies have reached differing conclusions, some recommending against routine use, others supporting it in carefully selected cases. The honest position is that vertebral augmentation is neither the cure-all of its early reputation nor the inert procedure suggested by the sham trials, but an intervention whose value is conditional and contested, and which is weighed case by case rather than applied as a rule.

Why low bone density complicates spinal instrumentation

Beyond fractures, osteoporosis casts a long shadow over any spinal operation that uses metalwork, because nearly every implant in spinal surgery relies on the strength of the bone it anchors into. Weak bone is, in the most basic sense, a poor foundation, and this single fact underlies a whole family of surgical problems.

The most direct is the behaviour of pedicle screws, the screws placed into the vertebrae to hold rods and reconstruct the spine. The grip of a screw depends on the density of the bone around its threads, and in osteoporotic bone that grip is markedly reduced. The consequence is a greater tendency for screws to loosen over time, or in the worst case to pull out altogether, which can destabilise the whole construct. Bone density is one of the strongest predictors of whether screws stay firmly seated.

A second problem is cage subsidence, the sinking of an interbody fusion cage into the soft bone of the vertebra above or below. The cage rests on the bony surface of the vertebra, the endplate, and where the bone beneath that endplate is weak, it yields and lets the cage settle downwards, undoing the height and alignment the operation achieved. Low bone density roughly doubles the risk of this happening.

A third, and particularly important, problem is proximal junctional failure. When several vertebrae are fused together, the junction between the rigid fused segment and the mobile spine above it is a point of concentrated stress. In osteoporotic bone, the vertebra at the top of a long construct can fracture, or the fixation there can fail, causing the spine to angle forwards sharply just above the metalwork, a problem known as proximal junctional kyphosis or, in its more severe form, proximal junctional failure. Low bone density is a recognised risk factor for this. Together, screw loosening, cage subsidence and junctional failure form a recurring theme: in weak bone, the implant is only ever as strong as the bone holding it, and that bone is the limiting factor.

Cement augmentation and surgical strategies in weak bone

Because weak bone is such a consistent obstacle, a range of techniques has developed to compensate for it, and these are an important part of how spinal surgery is adapted to the osteoporotic spine. They share a common aim: to spread load more widely, to anchor implants more securely, and to avoid concentrating stress on bone that cannot bear it.

The best known is cement augmentation of pedicle screws. Specially designed fenestrated screws have small openings through which bone cement can be injected into the vertebral body after the screw is placed. The cement hardens around the threads and locks the screw into a far larger volume of bone than the threads alone would engage, substantially increasing the force needed to loosen or pull it out. Biomechanical and clinical studies consistently show improved fixation and reduced loosening with this technique, at the cost of a small risk of cement leaking beyond the vertebra, which is the main reason it is used selectively.

Other strategies address the same problem in different ways. Constructs in weak bone are often longer, engaging more vertebrae so that the load is shared across many anchor points rather than a few. More screws may be placed, and the bone at the very top of a construct may be protected with techniques designed to soften the transition between the fixed and mobile spine, in an effort to reduce junctional failure. Cages with a larger footprint, designed to rest on the strong outer rim of the endplate, are favoured to resist subsidence. The unifying idea behind all of these is that operating on osteoporotic bone is less about the implants themselves than about respecting the weakness of the foundation, and engineering the construct so that no single point of bone is asked to bear more than it can.

The role of medical therapy around spinal surgery

Running alongside the surgical considerations is the medical treatment of osteoporosis itself, and its relationship to spinal surgery has become an area of considerable interest. The drugs used to treat osteoporosis fall broadly into two groups, and the distinction between them matters for the spine.

The first group are the antiresorptive agents, which work by slowing the cells that break bone down. These include the bisphosphonates, such as alendronate and zoledronic acid, which are the long-established mainstay of osteoporosis treatment and reduce the risk of vertebral fractures substantially. Denosumab, given by injection, is a more recent antiresorptive that is highly effective at building density, with the important caveat that its effect is rapidly lost if it is stopped, and that stopping it abruptly has been associated with a rebound of multiple vertebral

fractures. These agents reduce fracture risk and are widely used in people with osteoporotic spines.

The second group are the anabolic, or bone-building, agents, which stimulate the formation of new bone rather than merely slowing its loss. Teriparatide and the related abaloparatide are the principal examples, and romosozumab, which both builds bone and slows resorption, is a newer addition. Interest in these drugs around spinal surgery comes from a body of work, much of it originating from comparative studies of teriparatide against bisphosphonates, suggesting that bone-building agents given before and after fusion surgery may improve the rate at which the bones fuse, reduce screw loosening and lower the rate of mechanical complications in people with poor bone. The evidence is still developing, drawn largely from smaller comparative series rather than large trials, but the direction of travel is consistent enough that the perioperative use of anabolic agents has become a recognised strategy in the osteoporotic spine. The overarching theme is that, increasingly, the bone is treated as well as the spine, with medical and surgical management regarded as two halves of the same problem rather than separate concerns.

Outlook

The outlook in osteoporosis of the spine is best understood as two related stories. The first concerns the fractures themselves. A single acute vertebral fracture frequently settles over weeks to months as the bone heals, and many people recover well from an isolated event. The concern is the cascade: the way one fracture raises the risk of the next, and the way accumulated fractures produce progressive kyphosis, height loss and chronic discomfort. The trajectory of the osteoporotic spine depends heavily on whether this cascade is interrupted, which is why a vertebral fracture is regarded as a sentinel event rather than a closed episode.

The second story concerns the bone as the foundation for any treatment. Where surgery is contemplated, the quality of the bone is the backdrop against which everything is judged, because weak bone holds implants less securely and is prone to the loosening, subsidence and junctional failure described above. This is why so much attention now falls on measuring bone quality accurately, on adapting surgical technique to weak bone, and on treating the osteoporosis medically around the time of any operation.

Across the whole field, the consistent themes are that osteoporosis is a disease of bone strength rather than of any one symptom, that the spine is its commonest and most revealing site, that the vertebral fracture is both a problem in itself and a warning of more to come, and that any individual outlook depends on the density and quality of the bone, the number and pattern of fractures, and the wider health of the person, rather than on any single measurement. These are descriptions of how the condition behaves across groups of people, and the situation of any individual is a matter for assessment by the treating team.