

Cervical Instability

A patient guide

In brief: Cervical instability of the subaxial spine (the segments from C3 to C7) describes a state in which one neck vertebra moves too much, or in an abnormal pattern, against its neighbour under ordinary physiological loads. That excess or disordered movement is what can threaten the spinal cord or nerve roots, or produce mechanical pain — and it is what distinguishes instability from a worn but steady neck, a trapped nerve, or ordinary neck pain.

What cervical instability is, and what it is not

The most enduring definition of clinical instability remains that of White and Panjabi: the loss of the ability of the spine, under physiological loads, to maintain its pattern of displacement so that there is no initial or additional neurological deficit, no major deformity, and no incapacitating pain. The operative idea is **abnormal motion under normal load**. A motion segment is unstable when it moves further, or in a different way, than a healthy segment would, and that abnormal movement is what can compromise the cord or roots or generate mechanical symptoms.

It is just as useful to be clear about what instability is not. It is not the same as a "worn" neck. A spine can be heavily degenerate, narrowed and even compressing the cord while remaining mechanically steady; conversely, a segment can be unstable while the nervous tissue is, for the moment, intact. Instability is a statement about **movement**, not about wear or compression alone.

It is also not simply a synonym for neck pain. Pain is a symptom; instability is a structural and kinematic diagnosis defined by measurable abnormal motion. Most neck pain is not instability. The point that tends to matter on a patient-facing page is a plain one: instability means too much, or abnormal, movement between the bones of the neck — something distinct from a worn but stable neck, from a trapped nerve, and from everyday neck pain.

How the cervical spine normally stays stable (anatomy & biomechanics)

The stability of any neck segment is the product of three cooperating subsystems, a framework set out by Panjabi. The **passive** subsystem is the bones, discs and ligaments. The **active** subsystem is the surrounding musculature. The **neural control** subsystem is the nervous system that senses position and times muscle activity. Stability is not a single structure but the coordinated output of all three.

Within the passive subsystem, the elements share the load in a patterned way. The anterior elements — the anterior longitudinal ligament, the anterior part of the anulus and the vertebral body — chiefly resist extension. The posterior ligamentous complex — the supraspinous and interspinous ligaments, the ligamentum flavum and the facet capsules — chiefly resists flexion. White and Panjabi's cadaveric sectioning studies showed that a subaxial segment behaves in a strikingly non-linear, almost all-or-nothing way: dividing ligaments one by one produces only small increments of extra motion until a critical amount of restraint is lost, at which point motion increases suddenly and completely.

Panjabi later refined this with the concept of the **neutral zone** — the low-stiffness region around the neutral resting posture where the passive restraints offer little resistance. Instability is better understood as an enlargement of this neutral zone than as a simple increase in total range of movement. The neutral zone widens with ligament or disc injury and with muscular insufficiency, and it narrows with the formation of bony spurs, with surgical fusion and with muscular reconditioning. This explains why some people show only modest movement on imaging yet still have symptoms that vary with position: the segment is loose around neutral even if its end-range travel is unremarkable.

What can make it unstable (degenerative, traumatic, post-surgical, inflammatory e.g. RA, congenital)

Several distinct processes can erode the restraints that keep a segment steady.

****Traumatic.**** Distractive-flexion injuries (subluxated, perched or jumped facets, and the so-called teardrop fracture), distractive-extension injuries and frankly translational injuries disrupt the disc, the ligaments and the facets. Bilateral facet injuries and frank translation are regarded as unstable almost by definition. These patterns are described mechanistically by the Allen–Ferguson system and graded by severity in the SLIC and AOSpine systems.

****Degenerative.**** Degenerative cervical spondylolisthesis — a slip of one vertebra forwards or backwards — and facet and disc degeneration can produce abnormal segmental motion. This is relevant because it can convert a stable narrowing into a dynamically compressive one in older people. Degeneration is, however, bidirectional for stability: advanced wear can also auto-stabilise a segment as bridging osteophytes stiffen it.

****Iatrogenic or post-surgical.**** The posterior tension band is a finite biomechanical resource. Removing spinous processes, the interspinous and supraspinous ligaments and the ligamentum flavum (as in a wide laminectomy), or violating the facets, transfers flexion loads forwards and can predispose to progressive kyphosis or a "swan-neck" deformity. Adjacent-segment degeneration and pseudarthrosis (failed fusion) are further post-surgical contributors.

****Inflammatory.**** Rheumatoid arthritis is the classic example, producing subaxial subluxation, the characteristic "staircase" spine and vertical settling, with myelopathy in a minority but potentially serious consequences. Synovitis erodes both ligamentous and bony restraints.

****Congenital or syndromic.**** Down syndrome (with ligamentous laxity and odontoid anomalies), os odontoideum, skeletal dysplasias and generalised connective-tissue laxity predispose a segment to give way at lower loads, although several of these primarily affect the upper cervical spine. For completeness, tumour and infection can also destroy stabilising bone and ligament, a situation captured for the spine generally by the Spinal Instability Neoplastic Score.

How it tends to present

Presentations fall broadly into mechanical and neurological groups. The mechanical picture is axial neck pain that is worse with movement and load and on sustained postures, sometimes with a sense that the neck is "giving way" or needs supporting, and at times an audible or palpable clunk. Some people describe relief when the neck is held in a collar, which can be a soft pointer rather than a defining feature.

The neurological group is the more concerning. When abnormal motion produces dynamic compromise of the cord, myelopathic features may appear: gait disturbance, clumsy hands, an electric-shock sensation down the spine on neck flexion (Lhermitte's phenomenon) and brisk reflexes with positive Hoffmann or Babinski signs. Dynamic narrowing of the nerve-root canals can instead produce radicular symptoms in an arm. A clinical signature that points towards dynamic instability rather than fixed compression is that symptoms are **positional** — provoked or relieved by flexion or extension. Features that the literature treats as red flags include a progressive deficit, deformity after trauma, deformity progression in rheumatoid disease and any neurological change that varies with neck position.

How it is assessed and measured (dynamic radiographs, White & Panjabi criteria, CT/MRI)

Plain dynamic radiography — flexion and extension lateral views — remains the first-line functional test, because it captures movement that static images cannot. The classical, still-cited thresholds derived by White and Panjabi are a sagittal translation greater than 3.5 mm between adjacent vertebrae, and/or segmental angulation greater than

11 degrees relative to an adjacent level. Translation is measured along the posterior margins of the vertebral bodies and angulation along the inferior endplates, on both flexion and extension films. The literature is candid about the caveats: muscle guarding after acute trauma limits sensitivity, so flexion–extension films are most useful for ruling out instability once a CT is clear and the guarding has settled, and there is recognised observer-to-observer variability in manual measurement.

The White–Panjabi instability checklist organises these observations into a point system, where a total of five or more indicates instability. It integrates the integrity of the anterior and posterior elements, the static radiographic thresholds above, a positive stretch test (an interspace separation greater than 1.7 mm or an angular change greater than 7.5 degrees with up to a third of body-weight traction), neurological status, abnormal disc narrowing and the anticipated future loads on the segment. It remains the reference framework for subaxial assessment.

Cross-sectional imaging adds different information. CT defines bony injury and facet morphology and is the workhorse of trauma assessment. MRI defines the soft-tissue, or discoligamentous, complex — the posterior ligaments, the disc and the longitudinal ligaments — and shows cord signal change; this matters because ligamentous disruption with normal-looking bones is precisely the "hidden" unstable injury, which is why MRI sits at the centre of the SLIC and AOSpine systems. Dynamic (flexion–extension) MRI and quantitative motion analysis are emerging adjuncts for detecting occult discoligamentous instability and dynamic cord compression. Trauma classification systems each capture a different facet of instability: Allen–Ferguson is mechanistic; SLIC sums morphology, the discoligamentous complex and neurology into a score; and the AOSpine subaxial system grades compression (A), tension-band (B) and translational (C) injuries with facet, neurological and case-specific modifiers, and was designed for reproducibility.

How it is distinguished from related conditions (atlantoaxial instability, cervical spondylotic myelopathy, neck pain, disc herniation)

What separates subaxial instability from its neighbours is worth setting out plainly, because the conditions are easily conflated.

****Atlantoaxial and craniocervical instability**** is a problem of a **different region** — the C1–C2 joint and the junction with the skull — with its own kinematics. The C1–C2 joint contributes roughly half of the neck's axial rotation and has no intervertebral disc. It is measured by the atlanto-dental interval rather than by subaxial translation and angulation, and its common drivers (rheumatoid pannus, os odontoideum, the ligamentous laxity of Down syndrome, transverse-ligament rupture) differ from the degenerative and traumatic causes typical lower down. Subaxial instability is the C3–C7 problem, quantified by the White–Panjabi sagittal-plane criteria.

****Cervical spondylotic myelopathy**** (also called degenerative cervical myelopathy) is defined by cord compression and dysfunction, which may be entirely static. A stenotic, myelopathic spine can be perfectly stable, and an unstable segment can be neurologically intact. Instability concerns motion; myelopathy concerns compression. The two overlap precisely where abnormal motion becomes the mechanism of dynamic cord injury, and that intersection is the surgically important one.

****Simple mechanical neck pain**** is pain without demonstrable abnormal segmental motion or neural compromise — a symptom rather than a structural diagnosis. ****Cervical disc herniation and radiculopathy**** is a focal compressive lesion, a fragment of disc pressing on a root or the cord, on a segment that is typically stable; the problem there is the fragment, not excessive movement.

What the evidence shows about management (conservative vs stabilisation/fusion) — impersonal

The literature divides management along conservative and operative lines, and describes — rather than prescribes — when each tends to be considered.

On the conservative side, stable injuries and many degenerative or mechanical presentations without neural compromise are managed without surgery, using analgesia, activity modification, physiotherapy directed at the active and neural stabilising subsystems, and an orthosis for selected ligamentous-equivocal injuries. The neutral-zone model provides the rationale for muscular reconditioning, since strengthening the active subsystem can compensate for a loosened passive one. In cervical spondylotic myelopathy with mild symptoms, conservative care stabilises or improves the majority; the evidence is, however, of moderate strength that between roughly 20 and 60 per cent of such patients deteriorate over time without surgery, which is why surveillance is emphasised wherever a dynamic or myelopathic element exists.

On the operative side, stabilisation with or without decompression and fusion is described in the literature as being considered for demonstrable instability accompanied by neurological compromise, a progressive deficit, frank translational or bilateral facet injuries, deformity or deformity progression (including rheumatoid subaxial subluxation with myelopathy), and intractable pain clearly correlated with the abnormal movement. The SLIC and AOSpine scores operationalise the surgical threshold for trauma. The stated goals are realignment, neural decompression where compression coexists, and rigid stabilisation or fusion to abolish the pathological motion.

A distinct theme concerns avoiding instability created by surgery itself. Because multilevel laminectomy alone carries a historical risk of post-laminectomy kyphosis or swan-neck deformity reported at around 20 to 34 per cent, the literature favours laminoplasty (which preserves motion and carries a lower deformity rate) or laminectomy combined with instrumented fusion (which gives immediate rigid stability and allows deformity correction) over stand-alone laminectomy. The choice is described as being driven by the pre-existing alignment and stability — fusion where lordotic reserve is deficient or instability is already present, laminoplasty where lordosis and stability are preserved. Functional outcomes between laminoplasty and laminectomy-with-fusion are broadly comparable across comparative series, with technique-specific trade-offs between kyphosis risk on one hand and adjacent-segment disease and fusion-related complications on the other.

Outlook

The outlook is best understood through the lens of what is driving the instability and whether the nervous tissue is involved. A great many mechanical and degenerative presentations without neural compromise are stable enough to be managed without surgery, and the active subsystem — the muscles — can be reconditioned to compensate for a loosened passive one. Where there is a dynamic or myelopathic element, the literature is clear that a meaningful proportion of people drift towards deterioration over time, which is why the emphasis falls on recognition and surveillance rather than reassurance alone. Where surgery is undertaken for clear instability with neural compromise or deformity, its aims of realignment, decompression and rigid stabilisation are well defined, and comparative outcomes between the principal techniques are broadly similar. The recurring message of the evidence is that the prognosis tracks the mechanism: too much or abnormal movement that can pinch the cord or nerves behaves differently from a worn but steady spine, and the two should not be read as the same thing.