Craniocervical Junction Pathologies

Chiari Malformation, Rheumatoid Pannus, Atlantoaxial Instability & Surgical Decision-Making

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The craniocervical junction (CCJ) — the complex articulation between the skull base, the atlas (C1), and the axis (C2) — is one of the most anatomically intricate and surgically demanding regions of the spine. It houses the brainstem, upper cervical spinal cord, vertebral arteries, and the upper cranial nerves, all within a remarkably confined space. Pathology at this junction, whether from congenital anomaly, inflammatory disease, trauma, or tumor, can produce devastating neurological consequences if unrecognized or untreated.

This guide covers the most important CCJ conditions encountered in clinical practice: Chiari malformation, rheumatoid pannus and atlantoaxial instability, os odontoideum, and basilar invagination. For each, the key clinical and surgical question is: **Is decompression alone sufficient, or is decompression combined with stabilization and fusion required?** The answer depends on the specific pathology, the degree of instability, and the individual patient.

1. Anatomy of the Craniocervical Junction

Understanding the unique anatomy of the CCJ is essential to understanding why pathology here is so consequential:

The Occipital Condyles & Foramen Magnum

The skull base articulates with C1 via the paired occipital condyles, forming the occipito-atlantal joints. The foramen magnum is the large opening at the base of the skull through which the brainstem transitions to the spinal cord. It is also traversed by the vertebral arteries and lower cranial nerves (IX–XII). Any process that reduces the diameter of the foramen magnum compresses these critical structures.

C1 (Atlas)

A ring-shaped vertebra with no body and no intervertebral disc. It supports the skull and allows 50% of total cervical flexion-extension (nodding). The atlas has no spinous process and relies entirely on ligamentous attachments — particularly the transverse ligament — to maintain its relationship with C2.

C2 (Axis) & the Odontoid Process

The axis has a tooth-shaped bony projection (the odontoid process or dens) that projects upward through the C1 ring, restrained anteriorly by the anterior arch of C1 and posteriorly by the transverse ligament. Rotation of the head (50% of total cervical rotation) occurs at the C1-C2 articulation. Destruction of the odontoid or disruption of the transverse ligament produces atlantoaxial instability.

Key Stabilizing Ligaments

- **Transverse atlantal ligament:** The primary restraint preventing anterior displacement of C1 on C2. Its disruption — from rheumatoid disease, trauma, or Down syndrome — produces atlantoaxial instability and risks catastrophic cord compression
- **Alar ligaments:** Paired ligaments from the odontoid to the occipital condyles; limit rotation and lateral bending
- **Tectorial membrane:** The superior extension of the posterior longitudinal ligament; additional restraint against anterior translation
- **Posterior atlanto-occipital membrane:** Connects the posterior arch of C1 to the occipital bone; the posterior equivalent of the anterior atlanto-occipital membrane

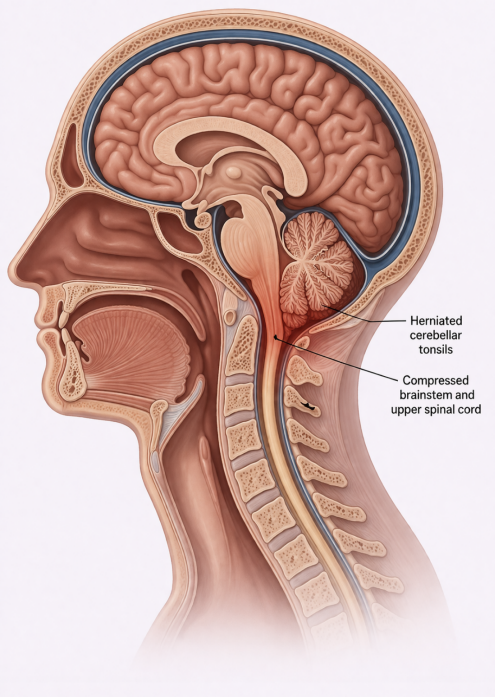
The Spinal Cord at the CCJ

At the level of C1-C2, the spinal cord is at its widest relative to the available canal space. A critical rule of thumb — **Steel's Rule of Thirds** — states that at the C1 level, one third of the ring is occupied by the odontoid, one third by the spinal cord, and one third by cerebrospinal fluid. This means there is some reserve space, but once CSF is effaced, the cord itself is under direct threat.

The Vertebral Arteries

The vertebral arteries ascend through the transverse foramina of C6-C2, loop behind the lateral masses of C1, and enter the skull through the foramen magnum to form the basilar artery. Their proximity to C1 lateral mass screws and C2 pedicle screws makes their identification and protection essential in all CCJ surgery.

2. Chiari Malformation



Chiari I malformation: the cerebellar tonsils herniate through the foramen magnum, compressing the brainstem and upper cervical spinal cord and obstructing cerebrospinal fluid flow.

What Is Chiari Malformation?

Chiari malformation is a structural abnormality in which part of the cerebellum — specifically the **cerebellar tonsils** — herniates downward through the foramen magnum into the upper cervical spinal canal. Named after the Austrian pathologist Hans Chiari, it is classified into several types, of which **Chiari I** is by far the most common in adult practice.

Types of Chiari Malformation

- **Chiari I (most common):** Herniation of the cerebellar tonsils ≥5 mm below the foramen magnum. No herniation of the brainstem or fourth ventricle. Often asymptomatic; may be discovered incidentally on MRI. Symptoms arise from obstruction of CSF flow at the foramen magnum and direct brainstem/cord compression. Associated with syringomyelia in 30–70% of cases.
- **Chiari II:** More severe — the brainstem, fourth ventricle, and cerebellum all herniate through the foramen magnum. Nearly always associated with myelomeningocele (spina bifida). Presents in childhood.
- **Chiari III & IV:** Very rare, severe malformations associated with encephalocele and severe developmental anomalies.

Causes & Associated Conditions

Chiari I is thought to result from a congenitally small posterior fossa — the compartment of the skull housing the cerebellum. When the posterior fossa volume is insufficient, the cerebellum is crowded downward. Associated conditions include:

- **Syringomyelia:** A fluid-filled cavity (syrinx) within the spinal cord caused by CSF flow obstruction. If untreated, a syrinx can expand and damage the cord from within, causing progressive neurological deficits
- **Hydrocephalus** — increased intracranial pressure from impaired CSF circulation; treated with a shunt before or independently of Chiari decompression
- **Scoliosis** — adolescent scoliosis associated with a syrinx may partially resolve after successful Chiari decompression
- **Tethered spinal cord, basilar invagination, atlantoaxial instability** — may coexist and require concurrent treatment

Symptoms of Chiari I

Symptoms arise from two mechanisms: direct compression of the brainstem and cervical cord, and obstruction of CSF pulsatile flow at the foramen magnum. Classic features include:

- **Occipital and suboccipital headache** — the hallmark symptom. Pain at the back of the head that characteristically worsens with Valsalva maneuvers: coughing, sneezing, straining, or laughing ("cough headache"). This pattern is highly specific for Chiari.
- **Neck pain and stiffness** — often radiating to the shoulders or upper arms
- **Balance and gait disturbance** — from cerebellar compression; unsteady, wide-based gait
- **Upper extremity symptoms** — numbness, tingling, or weakness in the hands and arms from cord or syrinx involvement; a characteristic "cape-like" pattern of sensory loss across the shoulders (from syrinx involving the central cord)
- **Dysphagia and dysarthria** — difficulty swallowing or speaking from lower cranial nerve compression
- **Sleep apnea** — from brainstem compression affecting respiratory control centers
- **Tinnitus, vertigo, or nystagmus** — vestibular and cerebellar dysfunction
- **Syrinx-related deficits** — dissociated sensory loss (loss of pain and temperature with preserved touch), hand weakness and wasting, or spastic paraparesis as syrinx expands

Diagnosis

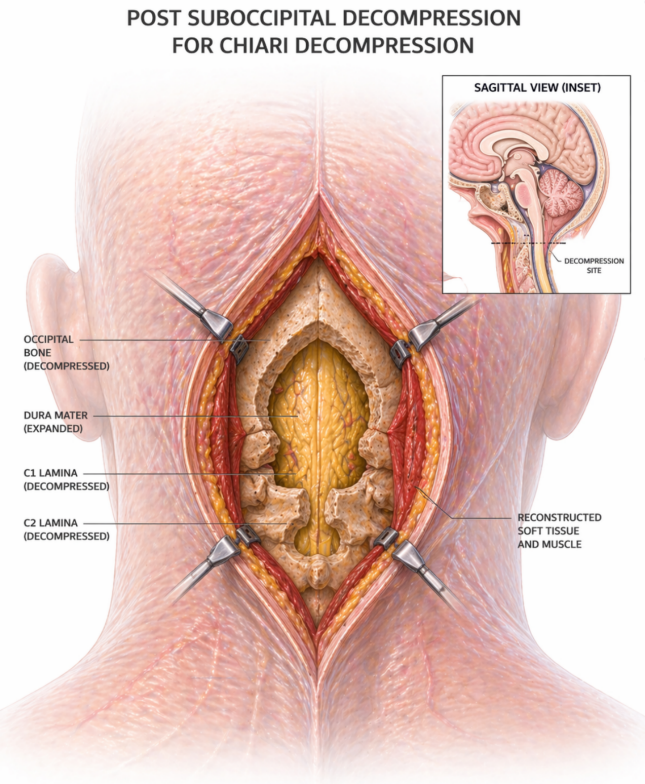
- **MRI brain and cervical spine** (with and without contrast) — the definitive study. Demonstrates the degree of tonsillar herniation, the size of the posterior fossa, brainstem and cord compression, syrinx presence and extent, and CSF flow at the foramen magnum
- **Phase-contrast MRI (CSF flow study)** — specialized MRI sequence that quantifies CSF pulsatile flow through the foramen magnum. Reduced or absent flow confirms symptomatic obstruction and is a strong indicator for surgical decompression
- **CT scan** — evaluates bony anatomy of the posterior fossa and foramen magnum; identifies associated bony anomalies such as basilar invagination or atlantoaxial instability
- **Full spine MRI** — screens for tethered cord, full extent of syrinx, and any thoracic or lumbar cord pathology

When Is Treatment Indicated?

Not all Chiari malformations require surgery. Asymptomatic Chiari I discovered incidentally may be managed with observation and serial MRI. Surgery is indicated when:

- Characteristic symptoms are present and attributable to the Chiari — particularly Valsalva-triggered headache
- A syrinx is present and symptomatic or enlarging on serial imaging
- Progressive neurological deficits are developing
- CSF flow is demonstrably obstructed on phase-contrast MRI

3. Surgical Treatment of Chiari — Suboccipital Decompression



Post suboccipital decompression: occipital bone craniectomy, C1 and C2 laminectomy, and dural expansion create a larger posterior fossa and restore CSF flow through the foramen magnum.

The Surgical Goal

The surgery for Chiari malformation is called a **posterior fossa decompression** (or suboccipital craniectomy). The goal is to enlarge the posterior fossa and foramen magnum — creating more room for the cerebellum and restoring normal pulsatile CSF flow around the brainstem. This relieves compression, resolves headaches, and in most cases leads to stabilization or resolution of syringomyelia.

The Procedure — Step by Step

- **Positioning:** Patient is placed prone (face-down) with the head secured in a rigid Mayfield skull clamp and flexed to open the posterior fossa.
- **Suboccipital craniectomy:** A small amount of bone is removed from the posterior skull base (occipital bone) to widen the foramen magnum — typically a 3–4 cm craniectomy. This is the bony decompression.
- **C1 laminectomy:** The posterior arch of C1 is removed to further open the neural canal at the top of the cervical spine and relieve pressure on the upper cord.
- **C2 laminectomy (if needed):** Additional decompression may extend to C2 if the tonsils herniate this far or if additional exposure is required.
- **Dural opening and duraplasty:** The dura (the tough outer membrane covering the brain and cord) is opened in the midline and a patch (duraplasty) — made from pericranium, synthetic dura, or cadaveric tissue — is sewn in to permanently enlarge the dural sac and maximize CSF flow restoration. This is the most important step for long-term symptom relief.
- **Tonsillar manipulation:** In some cases, the herniated tonsils are gently cauterized (bipolar coagulation) to shrink them and further open the foramen magnum — though this is not universally performed.

Chiari — Decompression Alone vs. Decompression + Fusion

✓ Decompression Alone Is Sufficient When:

- No atlantoaxial instability on flexion-extension X-rays
- No basilar invagination or cranial settling
- No os odontoideum or odontoid fracture
- Chiari symptoms without pre-existing instability at C0-C2
- The vast majority of Chiari I patients fall into this category

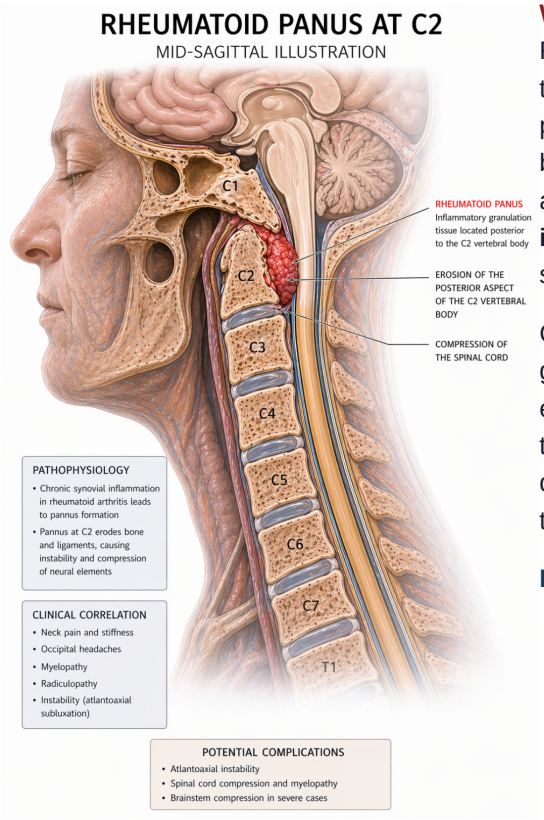
Procedure: Suboccipital craniectomy ± C1 laminectomy + duraplasty alone. No instrumentation placed. Excellent outcomes — 85–90% of patients improve or stabilize.

⊕ Decompression + Fusion Is Required When:

- Coexisting atlantoaxial instability (ADI >3 mm on X-ray)
- Basilar invagination — odontoid migrating above Chamberlain's line into the posterior fossa
- Os odontoideum with instability
- Ehlers-Danlos syndrome or connective tissue hyperlaxity
- Revision surgery after prior decompression that has destabilized C1

Procedure: Decompression + posterior C1-C2 or occipito-cervical fusion. Fusion eliminates residual instability and prevents progressive neurological deterioration.

4. Rheumatoid Pannus & Atlantoaxial Instability



Rheumatoid pannus at C2: inflammatory granulation tissue erodes the posterior C2 vertebral body and transverse ligament, producing cord compression, atlantoaxial instability, and risk of brainstem compression.

What Is Rheumatoid Pannus?

Rheumatoid arthritis (RA) is a systemic autoimmune disease that targets synovial joints throughout the body. The cervical spine — particularly the C1-C2 articulation — is uniquely vulnerable because of the rich synovial lining of the atlantoaxial and atlanto-occipital joints. In established RA, **cervical spine involvement occurs in up to 80% of patients**, with clinically significant instability developing in up to 40%.

Chronic synovial inflammation generates an invasive mass of granulation tissue called **pannus**. At the C1-C2 level, pannus: erodes the odontoid process and surrounding bone; destroys the transverse ligament (the primary stabilizer of C1 on C2); and directly compresses the spinal cord and brainstem from behind the eroded C2 vertebral body.

Patterns of Rheumatoid CCJ Involvement

- **Atlantoaxial subluxation (AAS):** Forward slippage of C1 on C2 from transverse ligament destruction — the most common pattern. An atlanto-dens interval (ADI) >3 mm in adults is abnormal; >10 mm indicates severe ligament disruption.
- **Vertical translocation (cranial settling / basilar invagination):** Destruction of the C1 lateral masses allows the skull to sink downward onto C2, driving the odontoid upward into the foramen magnum toward the brainstem — a life-threatening complication.
- **Subaxial subluxation:** RA also affects C2-C7, producing multilevel instability in a "staircase" or "stepladder" pattern.
- **Pannus at C2:** The mass of inflammatory tissue compresses the cord and nerve roots directly, independent of bony instability.

Symptoms

- Neck pain and occipital headache — often the earliest symptom
- Myelopathy — hand clumsiness, grip weakness, spastic gait, hyperreflexia, Babinski sign; can be rapidly progressive
- Radiculopathy — arm and hand symptoms from nerve root compression
- Brainstem symptoms — dysphagia, dysarthria, vertigo in severe cases
- Sudden death from cord transection with minor trauma in severely unstable cases — a catastrophic but recognized risk

Diagnosis

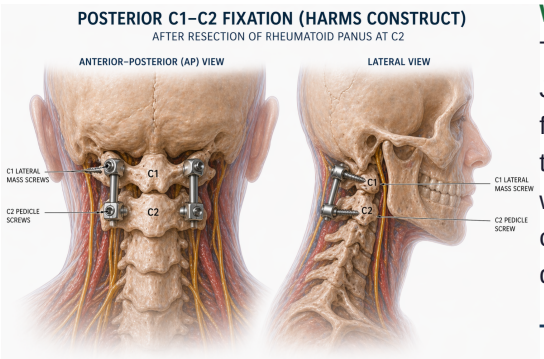
- **Standing and flexion-extension cervical X-rays:** Measure the atlanto-dens interval (ADI) and space available for the cord (SAC). Dynamic views reveal the degree of instability. The McRae and Chamberlain lines assess cranial settling.
- **MRI:** Demonstrates the pannus mass, degree of cord compression, presence of myelomalacia (cord signal change = injury), and the condition of the transverse ligament. Myelomalacia on MRI indicates established cord injury and mandates urgent surgical planning.
- **CT scan:** Defines bony erosion of the odontoid and C1-C2 architecture. Essential for pre-surgical planning — confirms pedicle and lateral mass anatomy for screw placement.
- **Neurological examination:** Myelopathy scales (mJOA, Nurick) document severity and guide urgency of intervention.

Surgical Treatment

Rheumatoid CCJ instability almost always requires surgical stabilization. Unlike Chiari malformation, where decompression alone is usually sufficient, RA-related CCJ disease nearly universally requires **fusion** because the underlying ligamentous destruction cannot heal with decompression alone:

- **Reduction of subluxation:** Pre-operative halo traction or intraoperative positioning is used to reduce the C1-C2 subluxation before fixation, restoring normal alignment
- **Pannus resection:** The inflammatory mass is surgically removed from the posterior aspect of C2 to directly decompress the spinal cord — as demonstrated in the Harms construct illustrations
- **C1-C2 posterior fusion (Harms technique):** The definitive stabilization — C1 lateral mass screws and C2 pedicle screws connected by rods eliminate motion at the destroyed atlantoaxial joint. Bone graft is placed to achieve solid fusion.
- **Occipito-cervical fusion (C0-C2 or longer):** When cranial settling is present or the C1 lateral masses are too eroded for screw fixation, the construct is extended to include the occiput — a plate is fixed to the occipital bone and rods are extended to C2 or lower
- **Important note on pannus:** In the majority of cases, the rheumatoid pannus regresses spontaneously after solid C1-C2 fusion eliminates the motion that drives ongoing inflammation — without the need for direct anterior surgical resection

5. C1-C2 Fusion — The Harms Construct



Posterior C1-C2 fixation (Harms construct) after rheumatoid pannus resection: C1 lateral mass screws and C2 pedicle screws connected by titanium rods — lateral and AP views. The construct eliminates atlantoaxial motion and provides the stability required for solid bony fusion.

What Is the Harms Construct?

The Harms technique — named after German spine surgeon Jürgen Harms — is the gold-standard method for posterior C1-C2 fixation and fusion. It replaced the older Gallie and Brooks wiring techniques because it provides three-dimensional rigid fixation without the need to pass wires beneath the C1 arch (which risks cord injury), and allows complete C1 laminectomy for cord decompression.

The Technique

- **C1 lateral mass screws:** Screws are placed through the lateral mass of C1 (the bony mass lateral to the C1 ring that supports the weight of the skull). Entry point and trajectory are precisely planned to avoid the vertebral artery above and the C2 nerve root below. These provide powerful purchase in the dense bone of the C1 ring.
- **C2 pedicle screws:** Screws are placed into the pedicle of C2 — a dense bony bridge connecting the posterior elements to the vertebral body. C2 pedicle screws provide the strongest fixation point in the upper cervical spine, but must be placed with great precision to avoid the adjacent vertebral artery.
- **Titanium connecting rods:** Rods connect the C1 and C2 screws bilaterally, creating a rigid four-point fixation frame that eliminates all C1-C2 motion in flexion-extension, rotation, and lateral bending.
- **Bone graft:** Autologous bone graft (from the iliac crest or local decorticated bone) is placed between the C1 and C2 posterior elements to induce solid bony fusion — the biological endpoint that makes the fixation permanent.

When the Construct Is Extended to the Occiput

In cases of cranial settling, severe C1 lateral mass erosion, or occipito-atlantal instability, the construct is extended proximally using an occipital plate fixed to the skull base — creating an **occipito-cervical fusion (C0-C2)**. This sacrifices occipital flexion-extension (nodding) but provides definitive stability when C1 cannot be used as a fixation point.

6. Other Craniocervical Junction Pathologies

Os Odontoideum

Os odontoideum is an anomalous ossicle — a rounded, smooth bony fragment separated from the body of C2 where the odontoid process should be. It may be congenital (failure of odontoid fusion) or acquired (from an unrecognized childhood odontoid fracture that remodeled). The critical issue is that the os does not provide the normal bony restraint against C1-C2 subluxation — making atlantoaxial instability common.

- Diagnosed on CT (clearly shows the separated ossicle) and MRI (shows cord compression and ligamentous injury)
- Symptomatic os odontoideum with instability requires posterior C1-C2 fusion — decompression alone is never adequate when significant subluxation is present
- Asymptomatic os odontoideum with minimal instability may be observed, but contact sports and high-risk activities are restricted

Basilar Invagination

Basilar invagination describes upward migration of the cervical spine into the skull base — most commonly the odontoid process rising above Chamberlain's line (a reference line drawn from the hard palate to the posterior lip of the foramen magnum). This drives the odontoid directly toward the brainstem and can produce compression even without frank C1-C2 subluxation.

- May be congenital (platybasia, assimilation of atlas) or acquired (from RA, Paget's disease, osteogenesis imperfecta, or Chiari-related cranial settling)
- Treatment depends on reducibility: reducible basilar invagination is treated with posterior occipito-cervical fusion after traction reduction; irreducible cases may require anterior transoral or endonasal odontoidectomy followed by posterior fusion

Traumatic Atlantoaxial Instability

High-energy cervical trauma can produce atlantoaxial instability through fractures or ligament ruptures at the C1-C2 level:

- **Jefferson fracture (C1 burst fracture):** Axial load shatters the ring of C1. If the transverse ligament is intact, the fracture is stable and treated with a halo brace. If the transverse ligament ruptures (rule of Spence: >8.1 mm total overhang of C1 on C2), the injury is unstable and requires C1-C2 fusion.
- **Odontoid fractures:** Type II (through the odontoid base) are most common and may be treated with anterior odontoid screw fixation or posterior C1-C2 fusion depending on age, displacement, and angulation.
- **Transverse ligament rupture:** Isolated ligament disruption from trauma cannot heal and requires C1-C2 fusion. Diagnosed on MRI (ligament signal change) and flexion-extension CT.

Down Syndrome & Ligamentous Laxity

Trisomy 21 is associated with laxity of the transverse atlantal ligament, producing atlantoaxial instability in up to 15% of affected individuals. Most are asymptomatic and managed with activity restriction (no contact sports, gymnastics, or diving). Symptomatic instability with cord compression requires posterior C1-C2 or occipito-cervical fusion.

Tumors & Infection at the CCJ

Primary or metastatic tumors involving the C1-C2 complex, as well as discitis-osteomyelitis, can destroy bony and ligamentous stability. Treatment combines tumor or infection management with posterior stabilization to prevent cord injury from progressive collapse. Anterior approaches (transoral, retropharyngeal) may be required to resect anterior pathology before posterior fusion.

7. The Key Surgical Decision: Decompression Alone vs. Fusion

The most important surgical question in CCJ pathology is whether neural decompression alone is adequate, or whether the decompression must be combined with rigid stabilization and fusion. The answer is determined by a careful assessment of **stability** — the ability of the CCJ to maintain normal alignment under physiological loads without risking neurological injury:

Chiari I	✓ Standard treatment for most patients	Required if coexisting instability, basilar invagination, or os odontoideum	Presence or absence of atlantoaxial instability
Rheumatoid Pannus	✗ Rarely sufficient — ligament is destroyed	✓ Almost always required (C1-C2 or C0-C2 fusion)	Degree of ligamentous destruction and cranial settling
Os Odontoideum	✗ Insufficient when instability present	✓ Required for symptomatic instability	ADI on flexion-extension X-rays; cord compression on MRI
Basilar Invagination	✗ Incomplete without addressing instability	✓ Occipito-cervical fusion after reduction or odontoidectomy	Reducibility; degree of brainstem compression
Jefferson Fracture (stable)	✓ Halo brace immobilization	Required if transverse ligament ruptures (>8.1 mm Spence)	Transverse ligament integrity (MRI + Spence rule)
Down Syndrome Instability	Activity restriction for asymptomatic cases	✓ C1-C2 fusion when symptomatic or cord compressed	Neurological symptoms; cord compression on MRI

8. Risks & Complications of CCJ Surgery

Craniocervical junction surgery is performed in one of the most demanding anatomical locations in the body. All procedures in this region carry risks that must be clearly understood:

- **Spinal cord and brainstem injury:** The most serious risk. Intraoperative neuromonitoring (MEPs, SSEPs, cranial nerve EMG) is used throughout all CCJ procedures. Even with monitoring, positioning, manipulation, or instrumentation can cause neurological deterioration — including paralysis or, in rare cases, death.
- **Vertebral artery injury:** The vertebral arteries are immediately adjacent to C1 lateral mass and C2 pedicle screw trajectories. Injury can cause catastrophic hemorrhage or posterior fossa stroke. Pre-operative CT angiography defines arterial anatomy in complex cases.
- **Dural tear and CSF leak:** Particularly relevant in Chiari surgery, where the dura is intentionally opened and repaired; CSF leakage from the repair may require prolonged bed rest or additional surgery.
- **C2 nerve root injury:** Sacrifice or injury of the C2 nerve root during C1 lateral mass screw placement causes occipital numbness — usually well-tolerated but occasionally bothersome.
- **Pseudoarthrosis / fusion failure:** C1-C2 fusion failure is more common in rheumatoid patients due to poor bone quality, ongoing immunosuppression, and steroid use. External bone stimulator use and optimization of disease-modifying therapy are recommended.
- **Dysphagia and airway compromise:** Post-operative swelling at the CCJ can affect swallowing and airway patency; patients are monitored closely and may require temporary nasogastric feeding.
- **Aseptic meningitis:** An inflammatory reaction to dural patch material used in Chiari duraplasty, causing fever, headache, and neck stiffness in the early post-operative period. Usually self-limiting.
- **Infection:** Posterior wound infections are more common in rheumatoid patients on immunosuppressive medications. Methotrexate and biologics are typically held peri-operatively per rheumatology guidance to reduce infection risk.
- **Failure of symptom resolution:** Particularly in Chiari surgery, some symptoms — especially chronic pain and non-Valsalva headaches — may persist after technically successful decompression.

9. Recovery After Craniocervical Junction Surgery

Hospital Stay 2–5 days	Neurological monitoring in the ICU or step-down unit overnight. Rigid cervical collar applied before discharge. Swallowing function assessed post-operatively. Headache is common after Chiari decompression and typically improves over weeks. Head-of-bed positioning instructions given.
Weeks 1–4 Home Recovery	Wear rigid collar as instructed. Limit head and neck movement. No driving, lifting, or strenuous activity. Wound check at 10–14 days. Chiari patients often notice headache and Valsalva symptoms improve within the first 2–4 weeks. Neurological recovery from myelopathy may take months.
Weeks 4–12 Rehabilitation	Collar weaned per Dr. Caridi's assessment. Outpatient physical therapy for cervical strengthening and balance rehabilitation. X-rays at 6 weeks assess hardware position and early fusion. Syrinx resolution is monitored with MRI at 3–6 months.
Months 3–6 Fusion & MRI Assessment	CT scan at 3–6 months assesses C1-C2 fusion progress. MRI at 3–6 months evaluates syrinx response (Chiari) and pannus regression (RA). Most patients experience continued neurological improvement during this period. Activity expanded progressively.
Months 6–18 Full Recovery	Fusion confirmed at 12 months. Neurological recovery from myelopathy continues for up to 18–24 months. Outcomes for Chiari decompression are excellent — 85–95% improvement in Valsalva headache. RA patients with myelopathy show meaningful improvement when surgery is performed before myelomalacia becomes irreversible.

10. When to Seek Evaluation

Seek Urgent Evaluation With Dr. Caridi If You Experience Any of the Following:

• Headache at the back of the head that worsens with coughing, sneezing, or straining — the hallmark symptom of Chiari malformation

• Progressive hand clumsiness, grip weakness, or difficulty walking — signs of cervical myelopathy requiring urgent evaluation

• New or worsening arm numbness, tingling, or weakness

• Neck pain and occipital headache in a patient with known rheumatoid arthritis — cervical involvement must be ruled out

• Difficulty swallowing, changes in speech, or episodes of dizziness or vertigo — possible brainstem or lower cranial nerve compression

• Any sudden neurological deterioration — loss of arm or leg strength, bladder or bowel dysfunction — seek emergency evaluation immediately

• Known Chiari malformation with worsening or new symptoms, or an enlarging syrinx on surveillance MRI

• Prior C1-C2 or occipito-cervical fusion with new neck pain or neurological symptoms — possible hardware failure or adjacent pathology