Cervical Spine Deformity: Indications, Considerations, and Surgical Outcomes

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Abstract

Cervical spinal deformity (CSD) in adult patients is a relatively uncommon yet debilitating condition with diverse etiologies and clinical manifestations. Similar to thoracolumbar deformity, CSD can be broadly divided into scoliosis and kyphosis. Severe forms of CSD can lead to pain; neurologic deterioration, including myelopathy; and cervical spine–specific symptoms such as difficulty with horizontal gaze, dysphagia, and dyspnea. Recently, an increased interest is shown in systematically studying CSD with introduction of classification schemes and treatment algorithms. Both major and minor complications after surgical intervention have been analyzed and juxtaposed to patient-reported outcomes. An ongoing effort exists to better understand the relationship between cervical and thoracolumbar spinal alignment, most importantly in the sagittal plane.

Cervical spinal deformity (CSD) remains a moving target in the current spine literature. It is a potentially debilitating condition with numerous etiologies, such as iatrogenic, inflammatory arthropathy, spondylosis, congenital, neuromuscular, traumatic, infectious, and neuromuscular processes. Severe cervical instability or sagittal malalignment can lead to pain; neurologic deterioration, including myelopathy; and cervical spine (CS)–specific symptoms such as difficulty with horizontal gaze, dysphagia, and dyspnea. Recent efforts have sought to classify CSD and formulate treatment algorithms. In this review, we discuss the anatomy, pathophysiology, and common etiologies of CSD. We then describe possible treatments, which are as myriad as the etiologies of CSD. Finally, we discuss the outcomes of CSD in the literature and its relationship to thoracolumbar deformity (TLD).

Etiology

Congenital

Atlanto-occipital fusion, os odontoideum, and basilar invagination are atlas and axis anomalies that can lead to CSD. Klippel-Feil syndrome, which causes most of the subaxial cervical congenital defects, involves fusion of cervical vertebrae (Figure 1). Achondroplasia, the common skeletal dysplasia, can lead to posterior vertebral scalloping, short pedicle canal stenosis, laminar thickening, and widening of intervertebral disks. Down syndrome is linked to ligamentous laxity that causes cervical instability seen at both the atlanto-axial joint and occiput-C1 level.

Traumatic

CSD can result from instability secondary to trauma. Upper CS injuries

Figure 1
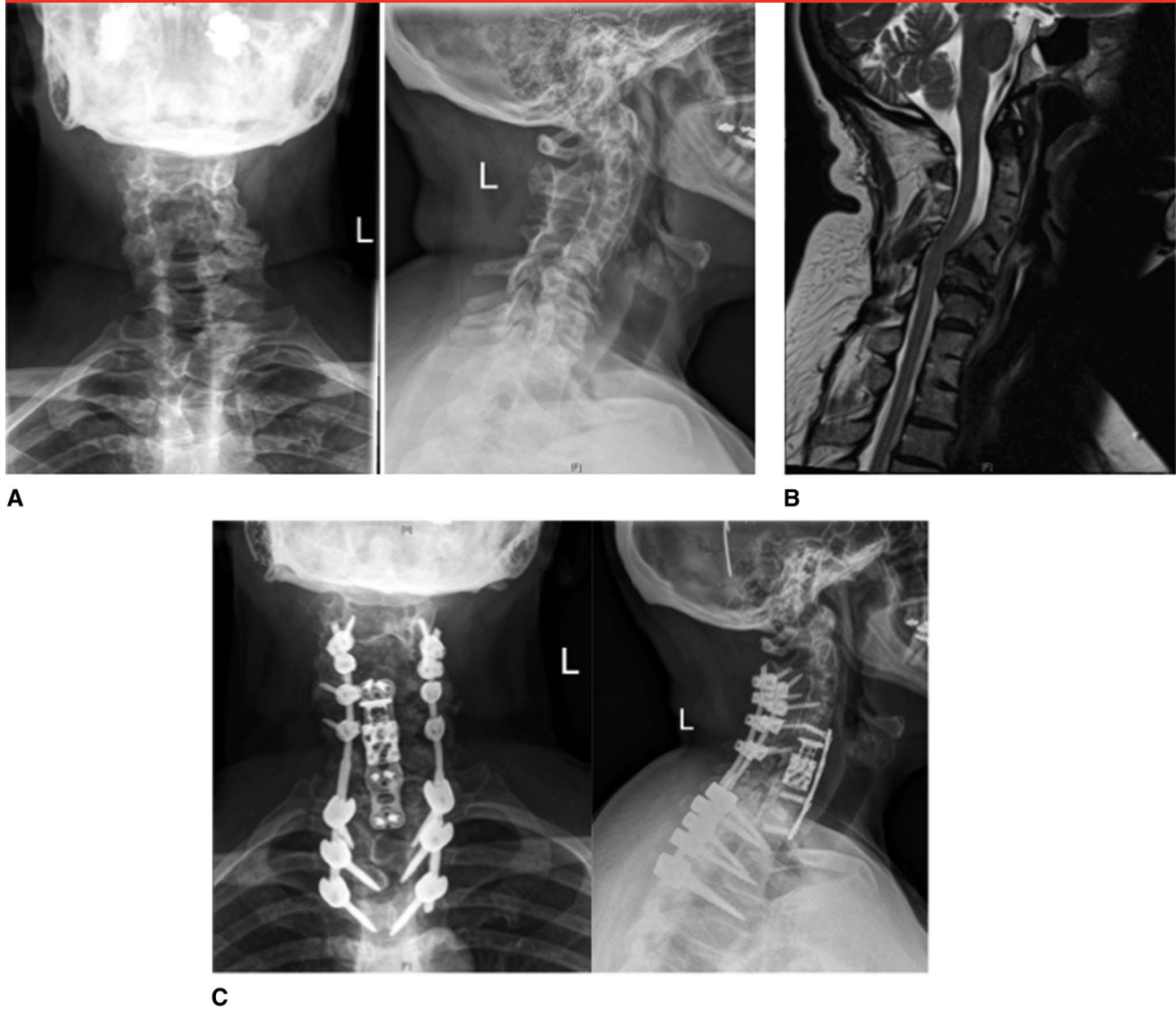
A, Preoperative AP and lateral radiographs of the cervical spine demonstrating kyphoscoliosis in an adult patient with Klippel-Feil syndrome. B, MRI of the cervical spine showing stenosis. C, Postoperative AP and lateral radiographs of the cervical spine after C2-T3 circumferential reconstruction.

include occipitocervical dislocation, occipital condyle fractures, atlas fractures, atlanto-axial rotatory instability (AAI), atlanto-dens instability, and odontoid fractures. Subaxial injuries include traumatic spondylolisthesis of axis (Hangman's fracture), flexion injuries, vertical compression injuries, and subaxial extension injuries. Posttraumatic CSD develops in most patients because of the trauma itself, but interestingly, a minority of patients in whom a posttraumatic CSD develops do so because of nonunion, implant failure, Charcot joint, or technical error after surgery for the injury.[1] Injuries involving the posterior ligamentous structures, such as advanced-staged burst flexion-compression or flexion-distraction injuries, are prone to deformity overall (Figure 2), whereas lateral compression or burst injuries can result in posttraumatic coronal or scoliotic deformities.[1]

Spondylosis and Degenerative Disk Disease

Disk degeneration leads to increased mechanical stress at the cartilaginous end plates at the vertebral body (VB) lip. Generally, spondylosis begins with intervertebral disk desiccation, leading to bulging of the anulus fibrosus, loss of height anteriorly, and a positive feedback loop of increased anterior weight bearing leading to cervical kyphosis (CK)[2] (Figure 3).

Spondylosis is also associated with ossification of the posterior longitudinal ligament, which can contribute to ventral cord compression. This often leads to loss of lordosis in the subaxial spine and can negatively influence global sagittal alignment (GSA). Patients will often hyperextend through their high CS (occiput-C2) to compensate for the loss of subaxial segments. By contrast, pure scoliotic deformity is rarely from spondylosis.

Inflammatory

Rheumatoid arthritis (RA) is the most common inflammatory disorder that can affect the CS. The prevalence of CS involvement in RA ranges from 25% to 80%, depending on the algorithm used,[3] with seropositivity as a major risk factor. The three typical RA deformities in the CS are, in order of decreasing frequency, AAI or subluxation, superior migration of the odontoid process, and subaxial subluxation.

Seronegative spondyloarthropathies include ankylosing spondylitis (AS), Reiter's syndrome, psoriatic arthritis, and enteropathic arthritis. AS is the most common of the seronegative disorders and will affect the CS later in the disease course. Common manifestations in the CS are CK and AAI. Global spinal kyphosis progresses as a means to offload painful facet joints, and autofusion in this abnormal sagittal alignment leads to a fixed flexion deformity. Furthermore, susceptibility to spinal fractures leads to frequent and missed fractures that can worsen the existing deformity.

Iatrogenic

Iatrogenic (postoperative) remains the most common cause. Typically, postoperative CK is associated with a previous laminectomy or laminoplasty and demonstrates a loss of sagittal alignment, shifting the weight-bearing axis anteriorly (Figure 4). Surgery denervates the

Figure 2
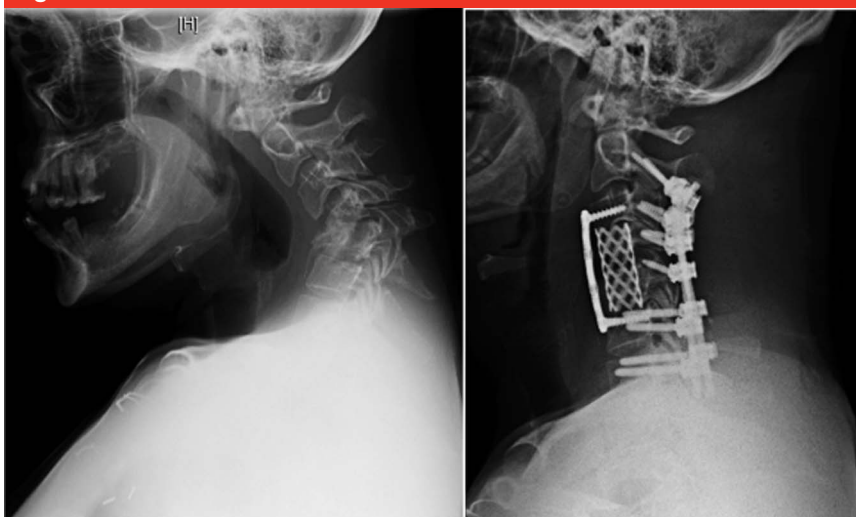
Chronic flexion-distraction injury leading to angular kyphotic deformity in the subaxial cervical spine. This patient successfully underwent C2-T1 circumferential spinal fusion with correction of deformity.

posterior cervical muscles, causing atrophy, and disruption of facet joints may lead to instability. Removal of the posterior tension band leads to worsening compressive forces on the anterior VB, thus exacerbating sagittal deformity and causing a kyphotic angulation.[4]

The incidence of iatrogenic CSD is difficult to measure because of heterogeneity of patient and case complexity. One case series demonstrated a 45% incidence in patients without any preoperative instability.[5] Contrarily, postoperative kyphosis developed in 10.6% of patients undergoing laminoplasty for cervical spondylosis, ossification of the posterior longitudinal ligament, and multilevel disk herniation.[6] A retrospective analysis investigating the incidence and outcomes of kyphotic deformity after laminectomy for cervical spondylotic myelopathy determined that kyphosis may develop in 21% of these patients.[7] In the pediatric population, weaker musculature, ligamentous elasticity, and greater horizontal facets have been theorized to account for the greater rates of postoperative kyphosis.[8]

Postoperative development of instability because of removal of static and dynamic stabilizing soft-tissue structures and facet violation is a well-known complication, and it was a major stimulus to the use of laminectomy with instrumented fusion and laminoplasty. The literature comparing outcomes of laminoplasty versus laminectomy are disparate, likely secondary to the heterogeneity between surgeon technique and patient as well as radiographic characteristics.[9,10] It can be agreed on, however, that the progression of loss of lordosis to kyphosis after laminoplasty appears to be dependent on the technique. Invariably, the preservation of muscle attachments has been shown to be essential for maintaining sagittal cervical alignment. The posterior cervical approach requires careful exposure from C3 to C7 while taking care to dissect in the avascular plane, or the raphe, between the left and the right paraspinal musculature. Additionally, during exposure of the lateral masses, facet capsule violation can lead to accelerated spondylosis, axial neck pain, and loss of lordosis.

Figure 3
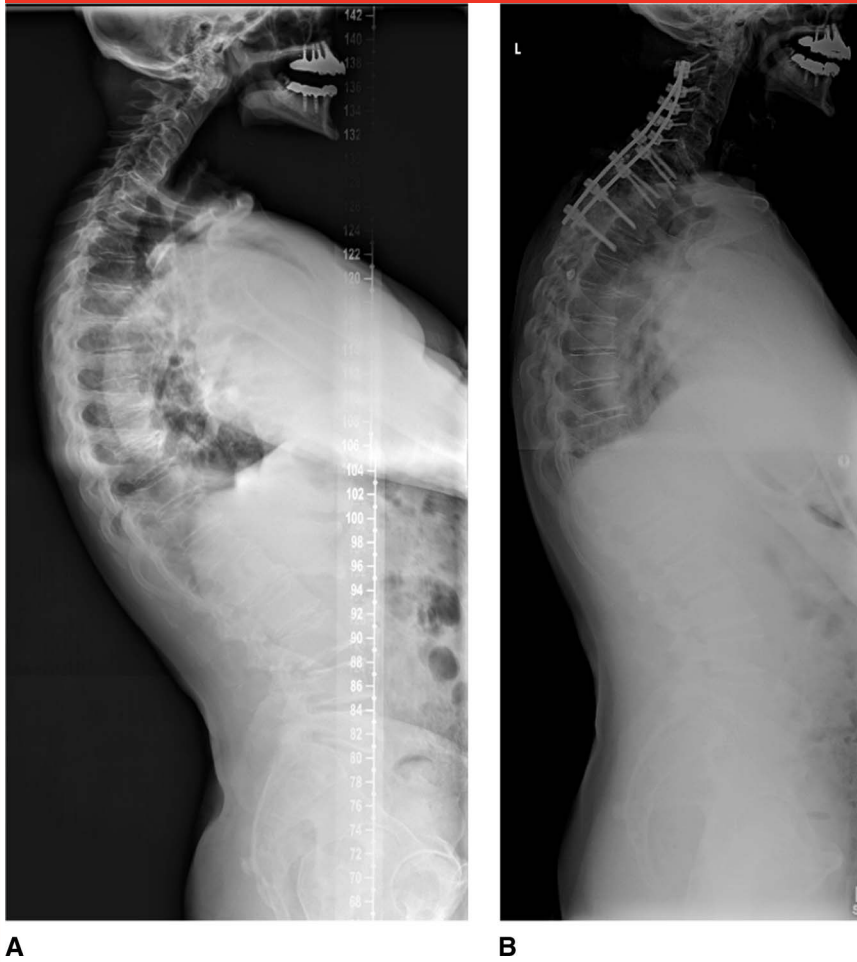
An adult patient with severe spondylotic kyphosis (**A**). He is hyperlordosing his lumbar spine to maintain horizontal gaze. C2-T4 posterior spinal fusion with C7 pedicle subtraction osteotomy to correct the deformity (**B**).

Infectious

Though a small contributor to the overall causes of CSD, spinal infections represent a growing area, given the use of illicit intravenous drugs in the younger population and genitourinary surgery and intravenous access devices in the elderly. Infections may involve any part of the spine including the VB, intervertebral disk, neural arch, or posterior elements, but most commonly it will affect the anterior and middle columns. Given its rich vascular supply, the VB are a common destination for dissemination of hematogenous osteomyelitis. Kyphosis is often a late finding but can present more frequently if *Mycobacterium tuberculosis* is the etiologic agent. In addition, early surgery for vertebral osteomyelitis has the benefit of improving stability and reducing kyphotic deformity relative to conservative management. Patients being treated with chemotherapy for spinal tuberculosis have, on average, an increase of 15° in deformity, and a kyphosis of >60° develops in 3% to 5% of patients.[11]

Neoplastic

Though tumors involving the spinal cord or surrounding structures in the CS are much more likely to cause deformity via the therapeutic approach used to manage them, certain entities exist which may engender malalignment and deformity even preoperatively. Kawabata et al[12] described their experience treating three patients with severe CK associated with neurofibromatosis (NF) and noted that a high risk of spinal cord injury, coexisting spinal cord and paraspinal tumors, difficulties in placing anchors in dystrophic vertebrae, and difficulty in obtaining solid fusion all potentially complicate the treatment of their patients' cervical disease.

Radiographic Definition

Lordosis/Kyphosis

It is important to consider the influence of CSD on global spinal alignment as compensatory changes occur to maintain horizontal gaze. Unlike the thoracolumbar spine (TLS), the CS can be divided into anterior (VB and disks) and posterior (facet joints) columns. The cervical load-bearing axis lies posterior to the VB of C2-C7, with posterior structures bearing about 64% of the axial load. This balance afforded by the stability of bony and ligamentous anatomy maintains the normal cervical curvature and maintains an upright head position[13]

PA and lateral radiographs should be used to assess coronal and sagittal alignment. Flexion-extension views allow for assessment of flexibility and stability. The most common method to evaluate cervical lordosis involves the use of Cobb angles. Similarly, the cervical curvature index (Ishihara) is an alternative method of assessing cervical spinal alignment numerically and is highly correlated with the C2-C7 Cobb angle.[14] Although the cervical Cobb angle is straightforward and has good interrater reliability, measurements such as the sagittal vertical axis (SVA) have gained traction in recent years (Figure 5).

GSA can be defined in multiple ways. Numerous studies have analyzed the health-related quality of life scores in relationship to adult TLD. These concepts have been echoed in the cervical deformity literature. Global alignment can be assessed by the C7 plumb line and the difference between the C2 and C7 SVA. The normal range of the C2–C7 SVA is reported to be 16.8 ± 11.2 mm.[15] Additionally, in a retrospective review of patients undergoing cervical laminoplasty, Oshima et al[16] found that postoperative functional outcome scores were markedly lower in patients with C2-C7 SVA of >+50 mm.

If full-standing spinal radiographs are unavailable, T1 sagittal angle or T1 slope is useful in predicting overall sagittal balance. The incidence of CK is likely to be twice as high if a patient has a higher T1 slope.[17] Although the high T1 slope demands a greater degree of cervical lordosis, patients may be unable to provide it, causing progressive kyphosis. Cervical lordosis is also strongly linked with T1 slope in maintaining horizontal gaze, and patients with higher T1 slope show more kyphotic changes after cervical laminoplasty at 2-year follow-up.[18] Lee et al[18] demonstrated that thoracic inlet alignment had notable correlation with craniocervical sagittal balance, similar to the effect that the pelvic incidence has on the lumbar spine. Thoracic inlet angle, along with neck tilt, affects the alignment of the CS in that the thoracic inlet angle must increase or decrease based on changes in T1 slope and cervical lordosis to maintain neck tilt at roughly 44° to minimize muscle energy expenditure.[19]

Measuring horizontal gaze is also a crucial clinical data point because it can occur with severe kyphosis and lead to a mechanical dysfunction of swallowing. Swallow dysfunction arises as a consequence of collapse of

Figure 4
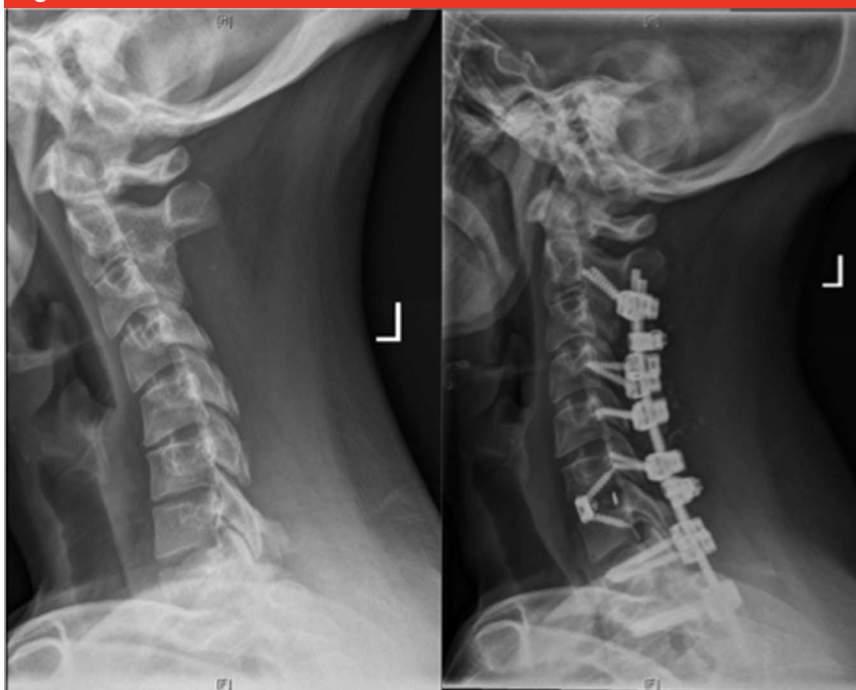
Post-laminectomy kyphosis. Patient required a long spinal fusion (C2-T1) to correct the deformity.

the pharyngeal space. The chin brow vertebral axis (CBVA) is used to assess horizontal gaze and is defined by the angle subtended between a line drawn from the patient's chin to brow and a vertical line. To obtain this distance, a photograph must be taken with the patient standing, with hips and knees extended and neck neutral or fixed. Lafage et al[20] reported on CBVA thresholds for disability and found that CBVA of <−4.8° or >+17.7° correlated with an Oswestry Disability Index of >40.

Ames Classification

Only one comprehensive CSD classification system exists, which was proposed by Ames et al;[21] it is an adaptation of the Scoliosis Research Society-Schwab classification for adult TLD. The system involves a deformity descriptor and five modifiers.

The classification system requires a full-length standing PA and lateral spine radiographs that include the

CS and femoral heads, standing PA and lateral CS radiographs, modified Japanese Orthopaedic Association (mJOA) scores, and a clinical photograph or radiograph that includes the skull for measurement of CBVA.

Scoliosis

Scoliosis, though infrequent, presents a distinct challenge for the spine surgeon. It is often found in association with congenital bony anomalies, Klippel-Feil syndrome, and NF type 1. Preoperative CT and MRI are both crucial, especially given the latter's ability to detect spinal dysraphism, which approaches a prevalence of 30% in patients with congenital spine deformity.[22] Surgical approach of this rare entity can be anterior, posterior, or combined (Figure 1). In a case series of 18 patients with isolated cervical scoliosis, the Cobb angle improved from 35.1° to 15.7°; however, a complication rate of 30.8% was related to surgery.[23]

Figure 5
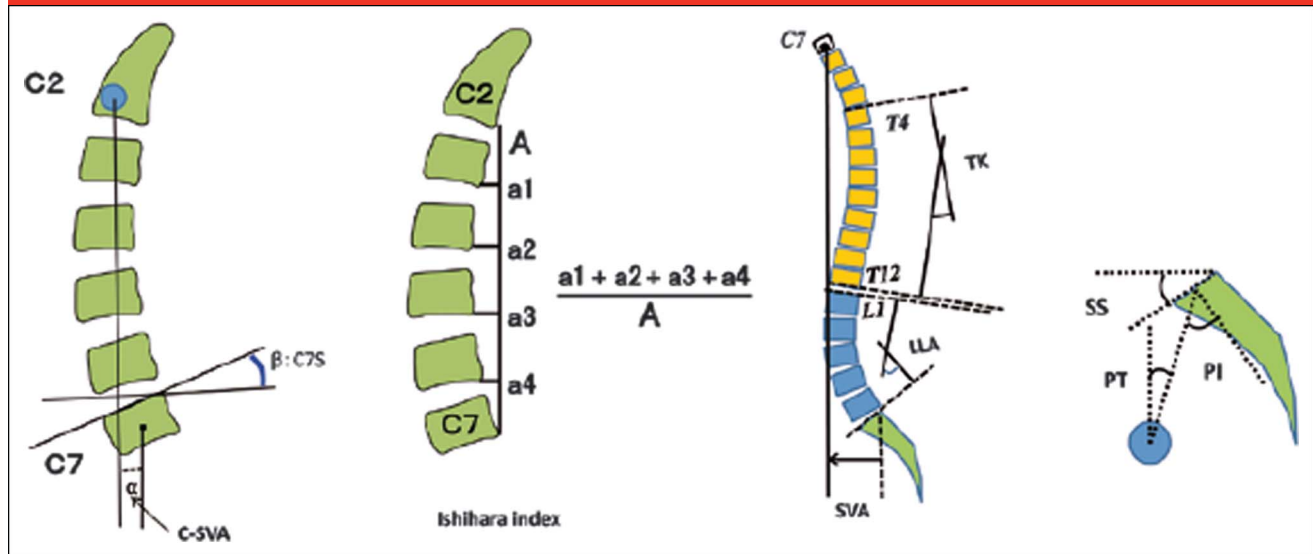
Cervical alignment, Ishihara index, and spinal sagittal alignment. C7S Beta = C7 tilt angle, c-SVA = cervical sagittal vertical axis, LS = lumbar lordosis, PI = pelvic incidence, PT = pelvic tilt, SS = sacral slope, SVA = sagittal vertical axis, TK = thoracic kyphosis. (Reproduced with permission from Endo K, Suzuki H, Sawaji Y, et al: Relationship among cervical, thoracic, and lumbopelvic sagittal alignment in healthy adults. *J Orthop Surg (Hong Kong)* 2016;24 [1]:92-96.)[26]

Cervical-Thoracic-Lumbar Relationship

Given the fact that CK may represent normal alignment in some patients, often a close relationship exists between cervical and thoracolumbar alignment.[24] Smith et al[25] found that 53% of adult patients with TLD had a concomitant cervical deformity. Furthermore, in patients with normal horizontal gaze, thoracolumbar alignment and thoracic kyphosis (TK) directly affect the cervical alignment.[24] Patients with poor sagittal alignment often develop painful compensatory alignment changes to maintain upright posture including knee flexion, pelvic retroversion, thoracic hypokyphosis, and cervical hyperlordosis. Diebo et al[24] found that patients with SVA of >50 mm require cervical lordosis to maintain gaze.

In the presence of spondylotic degenerative changes, loss of compensatory mechanisms for positive sagittal alignment may occur, result-ing in increased sagittal deformity.[26] Ames et al[27] analyzed spinal parameters in an asymptomatic population and found that pelvic incidence correlated with lumbar lordosis, lumbar lordosis correlated with TK, and TK correlated with cervical lordosis. The relationship between these parameters does not perfectly translate from one spinal segment to the next. For instance, the increase in cervical lordosis in response to TK may not be enough to maintain the head over the pelvis but does allow the patient to maintain horizontal gaze.

The relationship between cervical deformity and TLD has been analyzed before and after surgical treatment. In a study of 470 patients with TLD, a 53% prevalence of CSD and CSD was associated with C7-S1 SVA, pelvic tilt, and pelvic incidence-lumbar lordosis, suggesting that CSD should be investigated in patients presenting with other spine pathologies.[25]

After correction of the TLD, patients tend to be more misaligned 2 years from surgery with worse TK, T1 slope-cervical lordosis, cervical lordosis, cSVA, C2-T3 SVA, and global SVA compared with patients with TLD who were not operated on.[28] These patients additionally have worse Oswestry Disability Index and Scoliosis Research Society activity at 1 and 2 years. Even after controlling for magnitude of TLD, cervical alignment has a direct effect on health measures.[29]

Pathophysiology

Normal cervical lordosis is between 10° and 20° with an average of 14.4° (as measured by C2-C7 angle). Preservation of sagittal balance requires intact anterior and posterior structures. Anteriorly, the VB and intervertebral disks resist compression. Posteriorly, the facet joints, posterior CS musculature, and interspinous ligaments act as a tension band. When the integrity of the posterior or anterior structures is compromised and kyphosis is present, deformity is
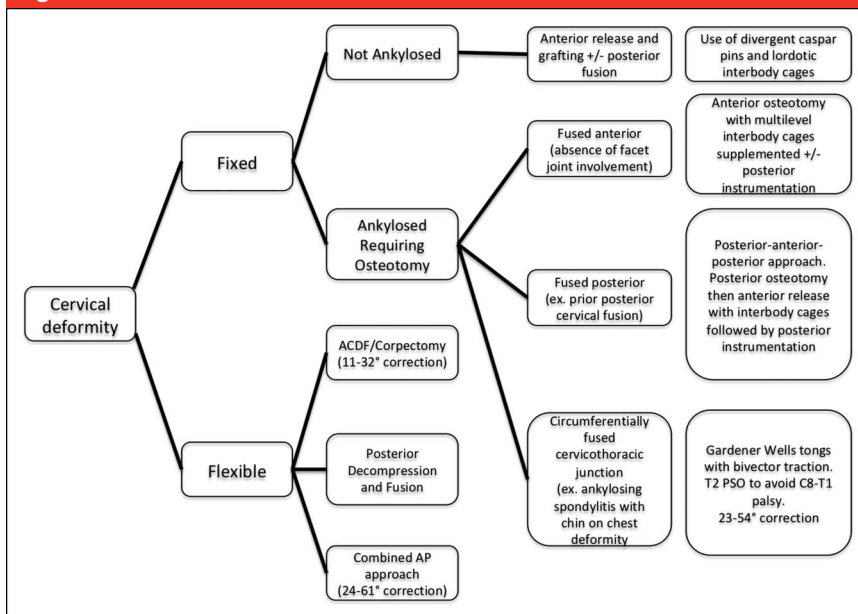
Figure 6

A patient whose head is completely suspended in air because of rigid cervical kyphosis (head suspension test).

Figure 7

Algorithmic selection of cervical deformity based on flexibility. (Reproduced with permission from Hann S, Chalouhi N, Madineni R, et al: An algorithmic strategy for selecting a surgical approach in cervical deformity correction. *Neurosurg Focus* 2014;36[5]:E5.)[31]

more than likely to progress. Ultimately, the spinal cord may become draped and tensioned over the posterior aspects of the VB, thereby compromising vascular supply. In this setting, myelopathic symptoms may develop in patients, which can lead to stepwise and potentially irreversible neurologic injury.

Clinical Presentation and Indications

Initial evaluation of the patient should be tailored based on the suspected etiology of their deformity. Patients with AS often exhibit chronic deformity that is gradually and progressively debilitating. Alternatively, in the setting of trauma, they can also exhibit acute deformity secondary to fracture, with sudden decompensation in posture and evidence of neurologic deficit. Patients with cervical deformity and evidence of ankylosis on imaging with sudden pain should be considered to have a fracture until proven otherwise.

Examination of patients with AS should comprise of the patient standing upright with the hips fully extended and in the seated and supine positions. Patients with cervical deformity often exhibit persistent cervical flexion despite lying flat. The rigidity of the deformity can be often assessed by

having the patient suspend his/her head in air when supine, the so-called head suspension test (Figure 6). Patients can also exhibit a chin-on-chest deformity that is characteristic of AS.

Iatrogenic deformities are unique in their presentation, and each requires their own corrective approach depending on the plane of deformity (sagittal, coronal, or both), the location of the fusion mass and instrumentation (anterior, posterior, or both), bone quality, soft-tissue quality, and the presence or absence of infection. In addition, patients with high to mid-cervical deformity may not present with an obvious abnormality on visual inspection (Figure 2). By contrast, those with cervicothoracic deformity often have a kyphotic appearance with difficulty raising the head and pain, similar to those suffering from flatback syndrome in the TLS (Figure 3). Revisions, especially in the setting of complex cervical deformity, require the utmost attention to detail to minimize complications.

The indications for corrective surgery in the setting of AS are intolerable deformity, neurologic deficit, airway compromise, esophageal dysmotility, and instability associated with fracture. The indications for surgery in the setting of iatrogenic deformity include intolerable posture, neurologic deficit, and intractable pain.

Preoperative Evaluation

Patients with cervical deformity whether AS or iatrogenic often have other medical comorbidities that increase the risk of morbidity and mortality. A thorough preoperative medical evaluation should be performed and the patient optimized before surgery. Nutrition and smoking status should be assessed in every case. Osteotomies can lead to substantial blood loss, and thus, preoperative measures should be taken to have blood prepared.

Radiographic evaluation should include long-standing radiographs to

Figure 8

Chin-on-chest kyphoscoliosis. This patient underwent C4-C7 anterior osteotomies (with standalone cages and one screw fixation) combined with C2-T3 posterior spinal fusion with multilevel Smith-Petersen osteotomies. C2 has two pars screws and one intralaminar screw as proximal anchors.

assess GSA and coronal alignment. A preoperative CT provides information on existing implants, fusion masses, and osseous landmarks for planned instrumentation. MRI should also be part of the workup in the setting of an abnormal preoperative neurologic examination, previous decompression surgery, or congenital anomalies.

Surgical management of cervical deformity requires meticulous preoperative planning to determine the approach and the degree of correction needed. Digital imaging software can allow surgeons to plan their osteotomies and provide insight into the postoperative alignment that can be attained.[30]

Surgical Treatment

One of the keys to surgical success is a thorough preoperative planning. Ideally, the CS should be made perpendicular to the clavicles in the coronal plane. Considerations in the sagittal plane should include flexibility and assessment of occiput-C2 motion. We prefer to align the posterior vertebral line of C2 as close to the anterior vertebral line of C7 as possible. This results in a balanced cervical posture, assuming the TLS is already well aligned. In the setting of a spine without mobile cervical segments, we attempt correction to a minimally flexed (15° to 20°) cervical alignment to allow the patient to be able to visualize the ground in front of him/her.

Approaches to deformity correction can be broadly categorized into anterior, posterior, and combined. The strategy for selecting a particular approach is often not straightforward. Hann et al[31] attempted to delineate an algorithm for surgical approach selection based on fixed versus passively correctable deformities. A detailed graphical summary of the article has been provided, delineating possible surgical approach and techniques for addressing various cervical deformities (Figure 7).

Anterior

Care should be taken to identify the vertebral arteries on preoperative MRI and to protect them during surgery. For maximal mobilization and induction of lordosis, we recommend wide exposure to the lateral margin of the uncinates bilaterally (Figure 8). If a previous fusion had been present, a high-speed burr can be used to take down the fusion at the original disk space to the level of the posterior longitudinal ligament. Concomitant coronal deformity can be corrected with asymmetric resection of the fusion mass. Prophylactic foraminotomies are performed to prevent root injury with spine extension. Diverging distraction pins can also be used so that distraction recreates lordosis. Two pins can be used on each VB to distribute forces if bone quality is poor. The head can be propped up with sheets initially and gently pushed down on the forehead with removal of the sheets one at the time on completion of the osteotomy to induce lordosis.[32]

Posterior

For posterior osteotomies, the patient is placed prone in a Jackson frame

with maximum reverse Trendelenburg. The foot of the Jackson frame is placed in the lowest rung of bottom bracket, and the top of the frame is placed in the lowest rung of the top bracket. We recommend that the head is placed in Gardner-Wells tongs with bivector traction. Typically, 15 pounds of weight is applied on the inline traction at the beginning of the procedure. This weight is then placed on the extension rope at the conclusion of the osteotomy to aid in correction.

Blood pressure should be closely monitored by an arterial line or a well-placed blood pressure cuff. We prefer to keep the blood pressure relatively high (around 85 mm Hg) in the setting of myelopathy to ensure adequate spinal cord perfusion. Foley catheter placement is inserted to assess fluid balance. A warming blanket can prevent hypothermia and thereby coagulopathy. Hemostatic techniques (eg, hemostatic agents, intraoperative blood salvage) can be used to minimize blood loss.

A posterior midline incision is made. The raphe of the paraspinal muscles is identified and dissected to minimize blood loss during exposure. Previous posterior fusions with a mobile anterior column are amenable to correction with multiple Smith-Petersen osteotomies. We advise prophylactic foraminotomies to prevent nerve root entrapment with correction of the deformity. A tension band construct can be used by connecting available spinous processes with a cable. This is done to help maintain the extension of the spine and hold correction until rods can be placed and tightened into position.

The Simmons osteotomy was classically described as an opening wedge osteotomy of the lower CS that compromises the anterior column opening by hinging on the posterior column. This was inherently unstable, and subsequently, pedicle sub-

Figure 9
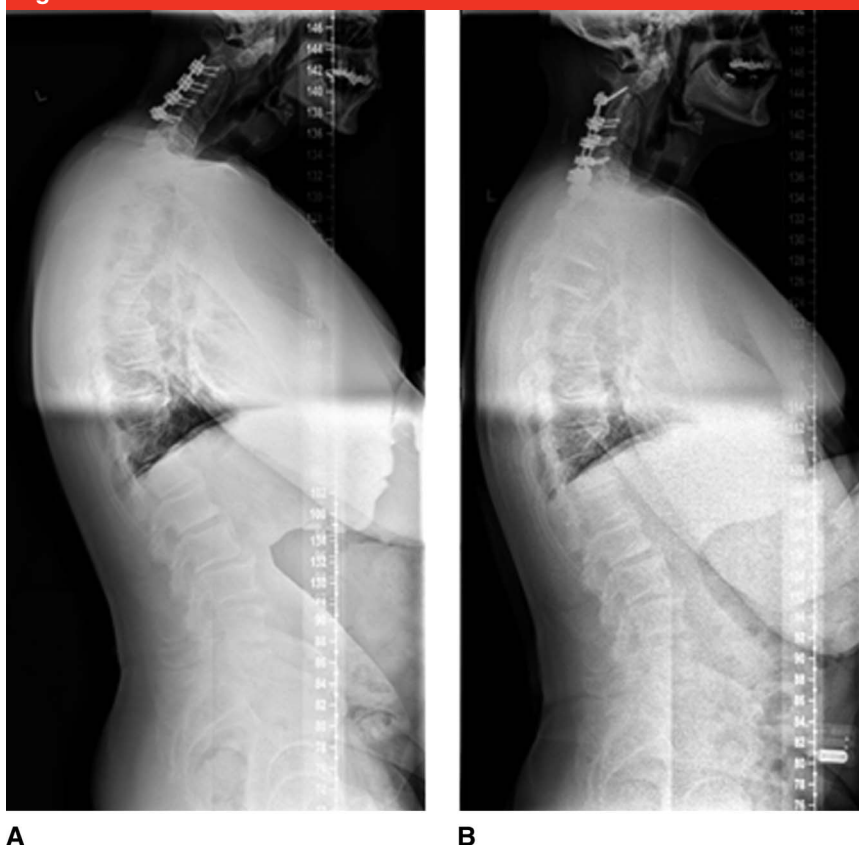
Rigid cervical kyphosis after laminectomy and fusion (**A**). The patient underwent C2-T4 revision fusion with T2 pedicle subtraction osteotomy to correct the deformity while avoiding C8 or T1 radiculopathy (**B**).

traction osteotomy (PSO) at C7 was developed as a means of shortening the posterior column while leaving the anterior column intact (Figure 3). We recommend that all available points of fixation be used. In cases where the occipital-cervical junction is mobile, we leave this joint alone. In cases where the occipital-cervical junction is autofused, good boney purchase can be made in the occipital protuberance. It is the author's preference to use C2 pedicle screws over laminar screws because they allow for collinear rod attachment. Pedicle or pars screws and laminar screws can be used together, that is, three or four points of fixation at C2, in case of poor bone quality resulting in poor fixation (Figure 8). The fusion should be extended down into the thoracic spine inferior to the level of the osteotomy. We recommend that if any question of bony purchase comes up, then the surgeon supplements the posterior fixation with anterior plate fixation at the level of the osteotomy. The technical details of a cervical PSO have been described in the literature.[33] Recently, some surgeons have found C8 or T1 nerve root palsy with profound intrinsic hand weakness after C7 or T1 PSO and recommend that the three-column osteotomy be performed at T2 or below (Figure 9).

Complications

Complications resulting from cervical deformity surgery are numerous

Table 1

Patient Outcomes Table Exploring Correction and HRQOL Measures

Study	Surgical Correction Approach	Preoperative Kyphosis	Postoperative Kyphosis	Degree of Correction	HRQOL Measures
Kim et al[36]	Group 1 (17): Anterior cervical osteotomy w/ w/out posterior instrumentation	Lordosis ($P = 0.10$)	Lordosis ($P = 0.92$)	Group 1: 23.1° angular correction, 1.4 cm translational correction	NDI
	Group 2 (21): Anterior osteotomy and SPOs w/posterior instrumentation	Group 1: 18.8°	Group 1: −5.0°	Group 2: 32.4° angular correction, 3.7 cm translational correction ($P = 0.15$ and 0.03, respectively)	Group 1: 26.3 → 25.5
		Group 2: 29.3°	Group 2: −4.7°		Group 2: 20.8 → 19.7 ($P = 0.56$, $P = 0.78$, respectively)
Du et al[37]	43 pts CDM associated with kyphosis-enlarged laminectomy w/lateral mass screw fixation	8.4% (Ishihara index) ($P < 0.001$)	19.3% (Ishihara index) ($P < 0.001$)	—	VAS: 37.4 ± 12.1 → 10.6 ± 5.3 ($P < 0.001$)
				—	JOA: 6.2 ± 1.9 → 14.9 ± 1.4 ($P < 0.001$)
Yeh et al[38]	20 pts anterior fusion, EOLP, lateral mass or pedicle screw instrumented fusion	−5.0° (cervical curvature) ($P < 0.001$)	9.3° (cervical curvature) ($P < 0.001$)	—	NDI: 39.9 ± 6.1 → 22.4 ± 3.8 ($P < 0.001$)
				—	Nurick scale: 2.6 ± 0.7 → 0.4 ± 0.9 ($P = 0.001$)
				—	VAS: 6.2 ± 0.8 → 2.0 ± 1.3 ($P = 0.118$)
				—	JOA: 10.1 ± 1.6 → 15.7 ± 1.8 ($P < 0.001$)
Grosso et al[35]	34/13/53 pts dorsal/ ventral/combined	Focal 23.9° (SD 14.5°)	Focal 2.8° (SD 7.4°)	Focal 1.5° (SD 7.8°)	mJOA change
		Global 17.2° (SD 14.0°)	Global −4.2° (SD 11.2°)	Global 1.3° (SD 5.6°)	Dorsal: 1.42
					Ventral: 2.37 Combined: 1.05 ($P = 0.097$)
Yeh et al[39]	109 pts EOLP and adjunct short-segment ACDF	7.7° (CC)	16.1° (CC)	—	VAS: 5.4 → 3.9
				—	Nurick scale: 2.7 → 0.3
				—	JOA: 10.9 → 15.8
Lau et al[40]	100 pts sequential interbody dilation	5.7° ± 7.3° ($P < 0.001$)	6.7° ± 7.3° ($P = 0.001$)	12.4° ± 8.0° ($P < 0.001$)	Nurick grade: 1.4 ± 1.5 → 0.9 ± 1.5 ($P = 0.037$) Neck VAS: 6.2 ± 2.6 → 4.4 ± 2.0 ($P = 0.02$)

(continued)

ACDF = anterior cervical, diskectomy and fusion, ATO = anterior osteotomy, CC = cervical curvature, CDM = cervical degenerative myelopathy, EOLP = expansive open door laminoplasty, HRQOL = health-related quality of life, JOA = Japanese Orthopaedic Association, mJOA = modified Japanese Orthopaedic Association, NA = not applicable, NDI = Neck Disability Index, PSO = pedicle subtraction osteotomy, SPO = Smith-Petersen osteotomy, VAS = visual analog scale

Table 1 (*continued*)

Patient Outcomes Table Exploring Correction and HRQOL Measures

Study	Surgical Correction Approach	Preoperative Kyphosis	Postoperative Kyphosis	Degree of Correction		HRQOL Measures
Kim et al[41]	Group 1 (31): Anterior osteotomy w/w/out posterior instrumentation	Lordosis (P = 0.73)	Lordosis (P = 0.03)	Angular (P = 0.03)	Translational (P = 0.56)	NDI: (P = 0.99)
	Group 2 (4): PSO	Group 1: 24.8°	Group 1: −5.0°	Group 1: 27.7°	Group 1: 1.8 cm	Group 1: 26.0 → 21.5
		Group 2: 4.3°	Group 2: −16.2°	Group 2: 48.8°	Group 2: 2.8 cm	Group 2: 25.2 → 20.5
Kim et al[42]	4 groups (61 total): SPO PSO ATO ATO + SPO	Lordosis 13.8° 20.3° 13.9° 30.9°	Lordosis −5.6° −16.2° −4.9° −4.8°	Angular 19.4° 44.8° 22.4° 32.5	Translational 3.5 cm 2.8 cm 1.3 cm 3.6 cm	NA
Mahesh et al[43]	9 pts single-stage posterior decompression and deformity correction for multilevel cervical myelopathy w/ kyphosis	6.33° (C2-C7 Cobb angle)	2° (C2-C7 Cobb angle)	—		mJOA: 7.88 → 15
		−15.8% (Ishihara Index)	−3.6% (Ishihara Index)	—		

ACDF = anterior cervical, diskectomy and fusion, ATO = anterior osteotomy, CC = cervical curvature, CDM = cervical degenerative myelopathy, EOLP = expansive open door laminoplasty, HRQOL = health-related quality of life, JOA = Japanese Orthopaedic Association, mJOA = modified Japanese Orthopaedic Association, NA = not applicable, NDI = Neck Disability Index, PSO = pedicle subtraction osteotomy, SPO = Smith-Petersen osteotomy, VAS = visual analog scale

and include implant displacement, graft dislodgement, pseudarthrosis, dysphagia, hoarseness, wound infection, dural tear, pneumonia, neurologic deficits, airway issues, and vertebral artery injury.[13] Complications can be divided into categories based on the operative technique. Anterior approaches are more likely to cause vocal cord palsy, tracheal/esophageal injury, graft failure, and infection or hematoma. The posterior approach is associated with higher rates of spinal cord or nerve root injury, hardware failure or fracture, nonunion, vertebral artery injury, and infection or hematoma. Finally, osteotomies are likely to cause infectious, respiratory, and cardiovascular morbidities.

A review of a multicenter database for adult patients undergoing cervical deformity surgery found 52 early complications (≤30 days postop) of 78 patients, with 28.2% of patients having ≥1 minor complication and 24.4% of patients having ≥1 major complication.[34] Of the three approaches (anterior, posterior, and combined), some have found the combined approach to be most fraught with complications at a rate of 40% versus 30% and 27% for anterior and dorsal, respectively.[35]

Patient Outcomes

Patients undergoing surgery for CSD can be analyzed postoperatively by examining the effects on the magnitude of deformity and the function and quality of life. Table 1 demonstrates a series of studies that explored the effects of surgical correction on degree of deformity and health-related quality of life measures.

Summary

CSD is an uncommon but debilitating condition with myriad etiologies but with iatrogenic-induced deformity being the most common cause. Depending on the severity of the deformity, the flexibility, and the surgical approach, the complication rate can be high. The relationship of CSD to TLD has been studied; sagittal imbalance in the lumbar spine can lead to painful compensatory changes in the CS. At this time, the subject of CSD represents a moving target as our understanding of cervical and thoracolumbar alignment evolves.

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