

## Review Article

# Controversies surrounding posterior cervical laminoplasty for cervical spondylotic myelopathy with kyphotic deformity

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**Abstract:** Posterior cervical laminoplasty is a well-established surgical treatment for multilevel cervical spondylotic myelopathy (CSM). However, its application in patients with concomitant cervical kyphotic deformity remains a subject of significant debate and controversy. This review aims to critically examine and synthesize the current evidence surrounding key controversies in the use of laminoplasty for CSM with kyphosis. Specifically, it focuses on: (1) the ongoing debates regarding surgical indications and approach selection criteria, including the roles of cervical alignment (both global and segmental), spinal canal occupancy ratio, and the K-line; (2) the comparative safety profile of posterior versus anterior decompression; (3) the impact of the procedure on postoperative cervical sagittal balance and the risk factors for alignment loss; and (4) the relationship between preoperative cervical alignment and the development of postoperative axial symptoms. The evidence synthesized suggests that mild kyphosis may not preclude successful outcomes with laminoplasty, provided that meticulous patient selection is performed based on a comprehensive assessment of radiographic and clinical factors. Future studies should incorporate dynamic imaging, long-term follow-up, and refined biomechanical models to clarify decompression mechanisms and optimize surgical decision making for this complex patient population.

**Keywords:** Cervical laminoplasty, cervical spondylotic myelopathy, kyphotic deformity, surgical outcomes, controversies

## Introduction

Cervical spondylotic myelopathy (CSM) is a degenerative disorder characterized by intervertebral disc herniation, ossification of the posterior longitudinal ligament (OPLL), and spinal canal stenosis, ultimately leading to spinal cord compression or compromised vascular supply and consequent myelopathic dysfunction. Surgical intervention should be actively considered in patients exhibiting definite signs or symptoms of spinal cord compression. Current surgical strategies primarily include anterior, posterior, and combined anterior-posterior approaches. Anterior procedures achieve direct decompression by removing ventral compressive lesions, whereas posterior techniques indirectly alleviate anterior compression through enlargement of the spinal canal [1]. Owing to their relatively straightforward technique and

lower procedural risk, posterior approaches have gained widespread clinical acceptance [2]. This safety advantage is particularly evident when comparing approach-specific complications: meta-analyses have consistently shown that anterior cervical surgery carries a significantly higher risk of dysphagia - a complication largely avoided by the posterior approach; whereas laminoplasty is associated with a distinct, albeit different, set of risks such as C5 palsy [3]. Since Hirabayashi et al. first described posterior cervical laminoplasty in 1977 [1], this technique has become a mainstay in the surgical management of CSM, particularly for cases involving multilevel disease ( $\geq 3$  segments), continuous-type OPLL, or dorsal cord compression [4]. Nevertheless, laminoplasty remains associated with certain relative contraindications - most notably cervical kyphotic deformity,

a negative K-line, and cervical instability. Among these, the most contentious issue centers on the appropriateness of laminoplasty in patients with concomitant cervical kyphosis. Key unresolved questions include: (1) What is the critical threshold of cervical alignment beyond which laminoplasty yields suboptimal outcomes? (2) Which specific types or degrees of kyphotic deformity might still be amenable to this posterior approach?

This article therefore aims to critically review the ongoing controversies surrounding posterior cervical laminoplasty in patients with CSM and concomitant cervical kyphosis, with a particular focus on surgical indications, radiographic parameter evolution, and postoperative complications. Specifically, this review will address the criteria for selecting anterior versus posterior approaches, examine the evidence regarding their relative safety, and critically detail the specific drawbacks and outcome controversies associated with performing laminoplasty in the presence of cervical kyphosis.

### Pathophysiologic mechanisms of CSM

CSM is the most common cause of acquired spinal cord dysfunction in adults, and its pathophysiological mechanisms are complex and multifactorial. CSM is not solely a result of mechanical compression, but rather a progressive neurodegenerative process. This process involves a complex interplay of static and dynamic mechanical loads, microcirculatory deficits, secondary biochemical cascades, and chronic neuroinflammation [5, 6]. The underlying pathophysiologic basis of CSM is described in the following three core dimensions.

#### *Mechanical compression: the double whammy of structural stenosis and functional stress*

The initial cause of CSM usually stems from a decrease in the effective volume of the spinal canal due to cervical degeneration, a process that has a clear anatomical basis: static compression is mainly caused by structural changes such as loss of disc height, endplate sclerosis, formation of osteophytes in the hook and tuberosity, hypertrophy or ossification of the posterior longitudinal ligament (OPLL), and thickening of the ligamentum flavum folds, which result in a significant decrease in the anterior-posterior diameter and cross-sectional area of

the canal (especially at the C4-C6 segments); dynamic compression is manifested by a further reduction in the internal diameter of the canal during cervical motion (especially flexion-extension motion) [7]. These lesions cause a significant reduction in the anterior-posterior diameter and cross-sectional area of the spinal canal (especially in the C4-C6 segments), which directly and continuously compresses the cervical cord ventrally or dorsally [8]. For example, in cervical extension, the ligamentum flavum wrinkles into the spinal canal, and in forward flexion, the spinal cord is elongated and pressed against the anterior osteophyte or herniated intervertebral disc to form a “bowstring effect”, and in cervical kyphosis, the cervical spinal cord is forced to move anteriorly and tighten against anteriorly induced compressive objects, which amplifies the localized stress concentration and accelerates the damage to axon. This can interfere with normal nerve function [9].

#### *Microvascular dysfunction and ischemic injury: from inadequate perfusion to cell death*

Continuous or repeated mechanical compression will directly damage the microvascular network in the spinal cord, especially affecting the watershed zones in the anterior horn of the gray matter and the central region of the white matter and triggering hypoperfusion, in which the impaired arterial perfusion will lead to a decrease in the partial pressure of oxygen in the local tissues, a blockage of oxidative phosphorylation in the mitochondria, and a decrease in the generation of ATP, which will then trigger a failure of the sodium-potassium pump, an overload of calcium in the cells, and a large release of excitatory amino acids, such as glutamate [10]. The impaired venous return will cause venous hypertension and increased capillary permeability, followed by vasogenic edema, extravasation of erythrocytes, and even pitting hemorrhage; this “ischemia-reperfusion-like” injury, which does not have a typical reperfusion process but has similar pathological consequences, will trigger a burst of reactive oxygen species (ROS), lipid peroxidation, and DNA breakage [11]. This “ischemia-reperfusion-like” injury, which has no typical reperfusion process but has similar pathological consequences, triggers reactive oxygen species (ROS) bursts, lipid peroxidation, DNA rupture, and ultimately triggers apoptotic pathways such as caspase-3 activation [12, 13].

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Animal models have confirmed that even after decompression, some neurons fail to recover due to irreversible ischemic damage. This phenomenon may explain the limited neurological improvement observed in some patients post-operatively [9].

### *Secondary neuroinflammation and the glial response: central drivers of chronic progression*

Recent studies have emphasized that the deterioration of neurological function in CSM not only depends on the degree of initial compression, but also on the cascade of secondary damage induced by chronic compression, in which neuroinflammation plays a central role [11]. Under the effect of mechanical stimulation and ischemic signals such as ATP and HMGB1, microglia will change from a resting state (ramified) to an activated state (amoeboid) and secrete large quantities of pro-inflammatory factors such as TNF- $\alpha$ , IL-1 $\beta$ , IL-6, nitric oxide (NO), etc., and these inflammatory mediators can break the blood-spoke cord barrier (BSCB), leading to the exudation of plasma proteins, the infiltration of immune cells - inducing the reactive proliferation of astroglia, forming a glial scar and hindering axonal regeneration [10, 12]. The direct damage to oligodendrocytes leads to demyelination, and it also mediates excitotoxicity through NMDA receptor over-activation to exacerbate neuronal death [14]. In addition, the chronic inflammatory environment can activate the Wallerian degeneration pathway, leading to degeneration of distal axons; even if the compression is lifted, it is difficult to repair the axon rupture that has already occurred, which explains why patients with CSM often have an insidious onset of the disease, slow progression, and incomplete clinical recovery after surgery [15, 16].

### *Clinical manifestations and hazards of CSM with kyphotic deformity*

While the general pathophysiological mechanisms of CSM apply, the superimposition of a cervical kyphotic deformity significantly modifies the clinical presentation and amplifies the potential hazards for affected patients. Understanding this distinct clinical profile is crucial for appreciating the controversies surrounding its surgical management.

*Clinical manifestations:* Patients with CSM and concomitant kyphosis often present with a

constellation of symptoms that extend beyond classic myelopathy. In addition to the common findings of gait disturbance, hand clumsiness, and sensory deficits, they frequently present with pronounced axial symptoms early in the disease course. Chronic, debilitating neck pain and stiffness are more prevalent and severe, stemming from the abnormal biomechanical stresses on the posterior cervical musculature and facet joints as they contract to compensate for the forward head posture [17]. The kyphotic posture itself - characterized by a forward head position, loss of the natural cervical lordosis, and sometimes a visible "chin-on-chest" deformity in severe cases - constitutes a key clinical sign. Neurologically, the mechanical compression in kyphosis is often more dynamic and posture-dependent. Symptoms may markedly worsen with neck extension, as the spinal cord is pinched between the posterior vertebral elements and the anterior compressive pathology, a phenomenon less pronounced in lordotic spines [7].

### *Potential hazards and disease implications:*

The presence of kyphotic deformity significantly elevates the associated risks of the disease. Firstly, the kyphotic alignment subjects the spinal cord to constant tensile stress and direct anterior compression, potentially accelerating the progression of myelopathy compared to patients who maintain physiological lordosis [8]. Secondly, kyphosis exacerbates microcirculatory compromise at the concavity of the spinal cord curve. The irreversible damage caused by prolonged, focused stress may limit the potential for neural recovery even after decompression [9]. Furthermore, from a therapeutic perspective, kyphosis fundamentally challenges the principle of indirect decompression upon which posterior approaches like laminoplasty rely. This often necessitates the selection of more complex anterior or combined anteroposterior surgical approaches to achieve adequate neural decompression and sagittal realignment, which themselves carry higher inherent risks [2]. Moreover, cervical kyphosis does not exist in isolation; it can lead to compensatory thoracic hyperlordosis and disrupt overall spinal sagittal balance. This imbalance is a primary driver of persistent pain, functional disability, and reduced health-related quality of life, independent of the myelopathic symptoms themselves [18]. Finally, patients with kyphosis undergoing posterior surgery face a greater risk of complications

such as inadequate decompression, progression of kyphosis, and severe postoperative axial symptoms, making surgical outcomes less predictable and potentially less favorable [19].

### **Current treatment paradigms and the evolving role of laminoplasty in CSM**

The definitive management of symptomatic CSM is surgical, with strategies tailored to the primary compressive pathology and spinal alignment. Anterior approaches (e.g., ACDF, corpectomy) enable direct ventral decompression and are favored for 1-2 level disease, significant kyphosis requiring correction, or predominant anterior pathology. Posterior approaches, chiefly laminoplasty, provide indirect decompression via spinal canal expansion and are the established standard for multilevel ( $\geq 3$  segments) CSM or ossification of the posterior longitudinal ligament (OPLL) in patients with preserved cervical lordosis, due to its efficacy in neurological recovery and lower risk of major complications compared to extensive anterior reconstructions [1, 2, 4]. Specifically, systematic reviews have demonstrated a significantly higher incidence of dysphagia, hoarseness, and approach-related revision surgery following multilevel anterior cervical procedures compared to laminoplasty [20, 21]. This safety profile makes laminoplasty particularly advantageous for elderly patients or those with medical comorbidities.

### *Laminoplasty in CSM with kyphosis: a critical appraisal*

Extending laminoplasty to patients with concomitant cervical kyphosis remains contentious. Evidence and clinical experience delineate a nuanced risk-benefit profile.

Potential advantages in selected cases (e.g., mild, flexible global kyphosis) include avoidance of fusion-related complications and preservation of motion [22, 23].

However, significant disadvantages predominate, forming the core of the controversy: (1) The inefficacy of indirect decompression due to impaired spinal cord posterior drift, risking residual ventral compression and suboptimal neurological recovery, particularly with a high canal occupancy ratio [24, 25]; (2) Acceleration of sagittal deformity from iatrogenic detach-

ment of posterior stabilizers, leading to progressive kyphosis, imbalance, and pain [5, 6, 26]; (3) A markedly higher incidence of severe axial symptoms [27].

Author's perspective: In our practice, laminoplasty is considered only for a narrow subset of patients with mild kyphosis ( $< 15^\circ$ ), a positive K-line (especially in extension), and occupancy ratio  $\leq 60\%$ , after thorough counseling about tempered expectations. For moderate to severe kyphosis, anterior or combined procedures targeting direct decompression and sagittal correction are strongly preferred, such as laminoplasty, which in such contexts often yields unsatisfactory biomechanical and clinical outcomes.

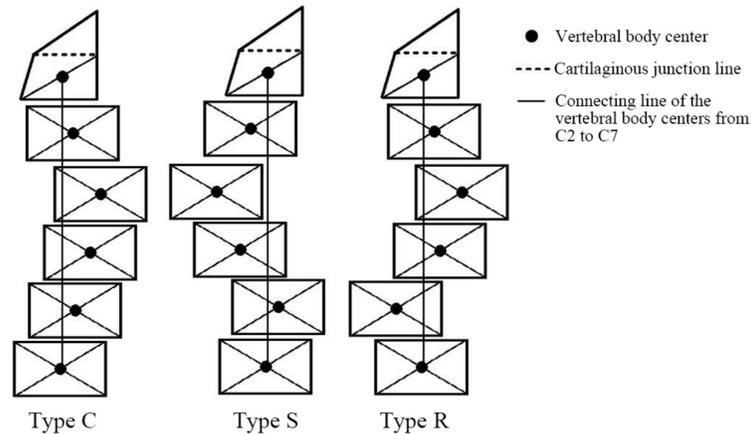
### **Controversies regarding the indications for laminoplasty**

It is generally accepted that the ideal candidates for posterior cervical laminoplasty are patients with multilevel cervical spondylotic myelopathy (CSM) or ossification of the posterior longitudinal ligament (OPLL) involving more than three vertebral segments, without cervical kyphotic deformity and with a negative K-line [28].

### *Cervical alignment*

A subset of researchers contends that impaired cervical alignment - particularly kyphotic deformity - constitutes a relative contraindication to laminoplasty. Sakaura et al. [29], in a retrospective analysis of 168 patients with CSM and 51 with OPLL followed for over two years, reported that patients with preoperative cervical kyphosis exhibited significantly lower postoperative Japanese Orthopaedic Association (JOA) score recovery rates ( $36.8 \pm 24.8\%$ ) compared to those with physiological lordosis ( $47.9 \pm 23.7\%$ ). Similarly, Sodeyama et al. [30], analyzing 65 CSM patients undergoing laminoplasty with a mean follow-up of 82.1 months, found that those achieving a posterior spinal cord shift exceeding 3 mm demonstrated significantly better JOA recovery rates ( $70.4 \pm 25.2\%$ ) than those with less than 3 mm of shift ( $48.7 \pm 23.5\%$ ). Furthermore, Baba et al. [31], in a study of 55 patients undergoing open-door laminoplasty with a mean follow-up of 2.4 years, observed a significant positive correlation between preoperative cervical lordosis and

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**Figure 1.** Modified Toyama classification [36].

postoperative spinal cord posterior shift ( $r = 0.276$ ,  $P = 0.048$ ), as well as between cord shift and neurological improvement ( $r = 0.303$ ,  $P = 0.033$ ). They concluded that only a physiologically lordotic cervical spine provides sufficient space for effective posterior cord migration, thereby facilitating decompression and functional recovery.

Conversely, other investigators argue that suboptimal cervical alignment should not be considered an absolute contraindication. Nagoshi et al. [32], in a retrospective review of 80 CSM patients who underwent laminoplasty, found no significant difference in neurological outcomes between patients with and without cervical kyphosis, suggesting that mild kyphotic deformity does not preclude successful laminoplasty. Zhu et al. [19] followed 81 CSM patients for a mean of 47 months and reported no statistically significant differences in JOA recovery rates among patients with kyphosis (59.81%), straightened alignment (69.25%), or normal lordosis (54.44%). Moreover, all three groups demonstrated comparable degrees of postoperative spinal cord posterior shift, leading the authors to conclude that patients with mild preoperative kyphosis remain suitable candidates for laminoplasty.

The authors of this review posit that the ongoing controversy regarding the relationship between cervical alignment and surgical outcomes may stem from several factors:

**Global versus segmental cervical curvature:** Miyamoto et al. [33] conducted a prospective study comparing postoperative recovery in two

groups of laminoplasty patients: one with segmental kyphosis (local Cobb angle  $> 5^\circ$ ) and another with severe global kyphosis but no significant segmental deformity. The segmental kyphosis group exhibited a markedly lower JOA recovery rate (32.6%) compared to the global kyphosis group (53.8%). Suda et al. [34], in a two-year follow-up of 114 laminoplasty patients, identified via multivariate regression analysis that both intramedullary MRI signal changes (OR = 4.10;  $P < 0.01$ ) and segmental

kyphosis (OR = 6.69;  $P < 0.01$ ) were the strongest predictors of poor surgical outcomes. They further proposed that a maximum segmental kyphotic angle of  $\leq 13^\circ$  is necessary to achieve favorable neurological recovery. In contrast, Zhang et al. [35], using multivariate regression, found that the most significant predictors of postoperative neurological recovery were intramedullary signal intensity (OR = 0.406;  $P = 0.000$ ), symptom duration (OR = 0.344;  $P = 0.005$ ), and severity of cord compression (OR = 0.244;  $P = 0.025$ ), whereas preoperative cervical alignment showed no statistically significant association ( $P = 0.548$ ). Collectively, these findings suggest that segmental kyphosis may exert a greater influence on postoperative neurological outcomes than global cervical alignment.

However, the Cobb angle has inherent limitations in characterizing segmental kyphotic deformity. To address this, Ruangchainikom et al. [36] proposed a modified TOYAMA classification, subdividing cervical kyphosis into three morphological types: global kyphosis (Type C), sigmoid (Type S), and reverse sigmoid (Type R) (**Figure 1**). Subsequently, Kimura et al. [37] applied this classification and found that S-type and R-type patients exhibited inferior postoperative neurological recovery compared to C-type patients. Nevertheless, Kim et al. [38] reanalyzed the same cohort using both the Cobb angle and the modified TOYAMA classification to define “lordotic” versus “non-lordotic” groups and found no significant difference in neurological improvement between the two classification systems, suggesting they may not be fundamentally distinct in predictive

value. Moreover, Uchida et al. [39], in a retrospective study of 43 CSM patients with cervical kyphosis exceeding 10°-including 11 S-type and 6 R-type cases - treated with either laminoplasty or anterior decompression and fusion, observed no significant difference in neurological recovery rates between morphological subtypes and other patients. Thus, the clinical utility of the modified TOYAMA classification in predicting laminoplasty outcomes warrants further investigation.

*Variability in postoperative evaluation timing:* Evidence suggests that 12 months postoperatively represents the typical timeframe for substantial neurological improvement, after which further recovery tends to plateau [40]. Consequently, JOA scores and neurological recovery rates assessed at 3 or 6 months may not fully capture the extent of functional recovery. To ensure a more accurate and comprehensive evaluation of surgical outcomes, a minimum follow-up duration of 12 months is recommended.

### *Spinal canal occupancy ratio*

An excessively high spinal canal occupancy ratio may compromise the efficacy of posterior cervical laminoplasty. Even with adequate posterior spinal cord drift or expansion following surgery, persistent anterior compression may remain unresolved, leading to suboptimal clinical outcomes - commonly defined as a postoperative Japanese Orthopaedic Association (JOA) score recovery rate of less than 50%. Consequently, the canal occupancy ratio is widely regarded as a critical determinant of surgical success in laminoplasty.

Sun et al. [41] retrospectively analyzed 83 CSM patients who underwent laminoplasty with a mean follow-up of 12 months and identified the canal occupancy ratio as a significant predictor of neurological recovery (OR = 0.940; P = 0.041). Zhang et al. [42] conducted a meta-analysis of 11 studies comparing anterior versus posterior approaches for OPLL and reported that when the occupancy ratio was below 60%, both surgical strategies yielded comparable outcomes; however, when it exceeded 60%, anterior decompression was associated with superior recovery rates. Furthermore, Hirai et al. [22], in a long-term study with a mean follow-up of 97 months, demonstrated that am-

ong patients with an occupancy ratio greater than 50%, those with residual anterior compressive lesions after laminoplasty exhibited significantly lower JOA recovery rates (34.0%) compared to those without residual anterior compression (56.6%), resulting in considerable disability and potentially impeding upper limb motor recovery.

Nevertheless, some investigators argue that, with appropriate patient selection, even those with high canal occupancy ratios can achieve favorable outcomes (JOA recovery rate > 50%) following laminoplasty. Chen et al. [23] retrospectively evaluated 55 patients with occupancy ratios exceeding 50% and a mean follow-up of 37 months, reporting that 63.6% (35 patients) achieved satisfactory neurological recovery. Notably, these patients demonstrated significantly greater posterior spinal cord shift. Their findings further indicated that the degree of cord posterior migration - and thus the efficacy of indirect decompression - was closely associated with preoperative cervical lordosis: patients with sufficient lordosis enabling adequate dorsal cord displacement tended to achieve better outcomes. However, preoperative cervical alignment alone is insufficient to reliably predict the extent of cord shift. Jokich et al. [43] observed that in cases of severe anterior compression, posterior cord migration after isolated posterior decompression is often limited, as the mobility of the spinal cord is constrained by the dentate ligaments and nerve roots.

In summary, the authors contend that the spinal canal occupancy ratio is indeed a pivotal factor influencing the outcomes of laminoplasty. An excessively high occupancy ratio (> 50-60%) may adversely affect postoperative neurological recovery. Nonetheless, for selected patients with high occupancy ratios, laminoplasty remains a viable surgical option when preoperative cervical alignment and other relevant factors are comprehensively evaluated and deemed favorable.

### *The K-line*

The majority of researchers consider a negative K-line to be a relative contraindication for posterior cervical laminoplasty [24, 44-46]. Fujiyoshi et al. [47] stratified 27 patients with ossification of the posterior longitudinal ligament

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(OPLL) who underwent laminoplasty into K-line-positive and K-line-negative groups. Their results showed that the K-line-positive group achieved a significantly higher postoperative Japanese Orthopaedic Association (JOA) score recovery rate (66.1%) compared to the K-line-negative group (13.9%). This finding suggests that patients with a positive K-line possess sufficient space for posterior spinal cord drift, which facilitates neurological recovery. In contrast, K-line-negative patients lack adequate room for cord posterior migration, resulting in inferior surgical outcomes. Consequently, the K-line is widely regarded as a valuable predictor of laminoplasty efficacy.

Keda et al. [27], in a study with a mean follow-up of 74.2 months involving 68 laminoplasty patients, reported that the K-line-positive group exhibited a significantly better JOA recovery rate ( $50.6 \pm 25.8\%$ ) than the K-line-negative group ( $19.4 \pm 25.2\%$ ). Moreover, they subclassified K-line-negative cases based on the nature of the compressive lesion-disc-type versus ossified-type and found that ossified-type compression was associated with worse prognosis, implying that bony compression is less amenable to indirect decompression via posterior drift.

However, conflicting perspectives exist in the literature [48]. Li et al. [49], in a multicenter study, found no statistically significant difference in JOA recovery rates between K-line-positive and K-line-negative groups. Similarly, Taniyama et al. [50] followed 46 CSM patients for two years and observed that 21.7% (10 patients) of those with a positive K-line still failed to achieve satisfactory outcomes after laminoplasty. These findings highlight the limitations of the K-line as a sole prognostic indicator.

The principal limitations of the K-line include the following: Insufficient imaging precision. On plain radiographs, it is challenging to accurately delineate the extent of anterior spinal cord compression or precisely locate the midpoint of the C7 spinal canal - particularly in obese or short-statured individuals - leading to inevitable inter-observer variability. To address this, Boudreau et al. [51] proposed measuring a modified K-line on MRI to mitigate bias arising from radiographic limitations. However, since CT and MRI are typically acquired in the supine

position rather than the upright stance, and cervical alignment is highly posture-dependent, the K-line measured on supine MRI may not reflect true spinal biomechanics in the functional (standing) position. Thus, further advancements in imaging technology - particularly weight-bearing MRI or upright CT - are needed to enhance the accuracy of K-line assessment.

**Influence of cervical positioning.** The K-line is highly sensitive to cervical posture during radiographic acquisition. Several studies have questioned whether neutral-position radiographs alone can reliably predict surgical outcomes [49]. Takeuchi et al. [52] suggested that evaluating the K-line on flexion-view radiographs may offer a more rational basis for surgical decision making, as anterior cord compression often worsens during cervical flexion. Conversely, Tsujimoto et al. [53] demonstrated that patients whose K-line was positive in extension but negative in neutral alignment could still achieve favorable postoperative outcomes, underscoring the dynamic nature of cervical alignment and its impact on decompression efficacy.

**Postoperative changes in cervical curvature.** Some patients experience loss of cervical lordosis or even develop postoperative kyphosis following laminoplasty. Such alignment alterations complicate the prediction of whether a preoperatively positive K-line might become negative postoperatively, thereby undermining the reliability of preoperative K-line status in forecasting the adequacy of spinal cord decompression.

Therefore, the prognostic value of the K-line in laminoplasty continues to be debated. Future research should aim to establish more precise, dynamic, and standardized criteria - potentially integrating multimodal imaging and functional posture assessment to enhance its clinical utility and predictive accuracy.

### *Anterior vs. posterior surgical approach: decision criteria and safety considerations*

The selection between anterior and posterior surgical approaches for CSM is guided by a synthesis of patient specific anatomical and pathological factors, and is central to the controversies surrounding laminoplasty in kyphosis. The general criteria favoring an anterior approach (e.g., anterior cervical discectomy and fusion,

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corpectomy) include: (1) pathology primarily localized to one or two vertebral segments; (2) presence of significant cervical kyphosis requiring direct correction to restore sagittal alignment; (3) compression predominantly from ventral structures (e.g., large central disc herniations, focal ossification of the posterior longitudinal ligament (OPLL)); and (4) the presence of a “negative” K-line, which indicates insufficient space posterior to the compressive lesion for adequate spinal cord drift after posterior decompression [47, 53]. Conversely, the posterior approach, particularly laminoplasty, is classically indicated for: (1) multilevel CSM or OPLL involving three or more segments; (2) pathology primarily causing dorsal compression (e.g., hypertrophied ligamentum flavum); (3) a physiologically lordotic or straightened cervical alignment; and (4) a “positive” K-line, suggesting potential for effective posterior cord migration [28, 44, 47].

Regarding procedural safety, posterior decompressive procedures like laminoplasty are widely considered to have a lower risk profile for certain major complications compared to multilevel anterior cervical surgeries. Anterior approaches carry inherent risks related to the anterior cervical visceral and vascular anatomy, including dysphagia, hoarseness due to recurrent laryngeal nerve injury, esophageal injury, and vertebral artery injury [2]. Laminoplasty largely avoids these specific risks. Its safety advantage is particularly relevant in elderly patients or those with medical comorbidities, as it typically involves less extensive visceral dissection, may have shorter operative times, and avoids the potential for graft-related complications (e.g., subsidence, dislodgement) or pseudarthrosis associated with anterior fusion [4, 28]. However, it is crucial to emphasize that laminoplasty is associated with its own unique set of complications, most notably postoperative axial neck pain and the potential for loss of cervical lordosis.

### *Specific drawbacks of laminoplasty in CSM with cervical kyphosis*

Performing laminoplasty in the context of cervical kyphosis introduces several specific drawbacks and potential compromises to the surgical outcome, which underpin the ongoing controversy and the need for stringent patient selection:

*Compromised mechanism of indirect decompression:* The efficacy of laminoplasty hinges on posterior migration of the spinal cord away from anterior compressive elements. In a kyphotic spine, the cord is draped over and tensioned against the anterior vertebral bodies. This anatomical configuration severely limits the potential for posterior drift, thereby negating the efficacy of the classical “bowstring” mechanism [34]. Even alternative mechanisms like dural sac expansion may be insufficient to overcome this anterior tethering effect [43, 54].

*Risk of persistent anterior compression:* After posterior canal expansion, the spinal cord may remain persistently apposed to anterior osteophytes or OPLL masses due to the kyphotic alignment. This is critically important in patients with a high spinal canal occupancy ratio (> 50-60%), where the space for decompression is already marginal. As Hirai et al. demonstrated, residual anterior compression after laminoplasty directly inhibits neurological recovery, particularly in the upper extremities [22]. Thus, for kyphotic patients with predominant ventral pathology, laminoplasty may fail to address the primary compressive lesion.

*Accelerated postoperative sagittal imbalance:* The cervical extensor musculature and posterior ligamentous complex, which are partially detached or injured during laminoplasty, are vital for maintaining sagittal balance. The procedure can weaken these posterior tension bands, thereby accelerating the loss of lordosis or worsening pre-existing kyphosis [55]. This risk is amplified in patients with unfavorable baseline sagittal parameters, such as a high T1 slope (> 29°) or a large C2-C7 SVA, predisposing them to progressive kyphosis, sagittal imbalance, chronic neck pain, and functional decline [17, 18].

*Higher incidence of debilitating axial symptoms:* As highlighted by Iseda et al., patients with preoperative kyphosis who undergo laminoplasty have a markedly higher incidence of postoperative axial symptoms (chronic neck pain, stiffness, shoulder discomfort) compared to those with lordotic alignment [56]. This is attributed to the combined biomechanical insult of pre-existing abnormal spinal alignment and the surgical disruption of posterior musculoligamentous structures, leading to pain from

instability, muscular fatigue, and altered load distribution.

We posit that the binary classification of cervical alignment as simply “lordotic” or “kyphotic” is insufficient for surgical decision-making. The key is to distinguish between compensatory global curvature and rigid segmental kyphosis. For the former, laminoplasty may be effective if other factors (e.g., K-line, occupancy ratio) are favorable. For the latter, especially with an angle  $> 13^\circ$ , it often represents a biomechanical failure point where indirect decompression is fundamentally compromised, and anterior or combined procedures should be strongly considered.

### **Controversies regarding the mechanism of spinal cord decompression in laminoplasty**

The classical understanding of the decompression mechanism in posterior cervical laminoplasty encompasses two interrelated components: direct and indirect decompression. Direct decompression is achieved through the surgical removal of posterior compressive structures—such as hypertrophied ligamentum flavum, bony stenosis, or other anatomical abnormalities—thereby providing immediate mechanical relief to the spinal cord. Indirect decompression, commonly described by the “bowstring mechanism”, relies on the preservation of physiological cervical lordosis, which serves as the “bow”, while the spinal cord functions as the “string”. Following laminar opening, the spinal cord migrates posteriorly, effectively distancing itself from anterior compressive pathology and thereby achieving decompression without direct manipulation of the anterior spinal column [25]. Central to this model is the assumption that an intact cervical lordotic curvature is essential for effective posterior cord drift and optimal decompression.

Suda et al. [34] emphasized that cervical lordosis is a prerequisite for successful indirect decompression via the bowstring mechanism; in the absence of lordosis—or worse, in the presence of kyphotic deformity—this mechanism is deemed ineffective. Consequently, cervical kyphosis has long been regarded as a contraindication to laminoplasty.

However, recent investigations have challenged the universality of the bowstring principle. Mul-

multiple studies have demonstrated that the degree of postoperative spinal cord posterior shift does not consistently correlate with preoperative cervical curvature. Notably, even in patients with mild kyphotic alignment, significant cord posterior migration has been observed postoperatively [19, 32], thereby questioning the exclusivity of the bowstring model.

An alternative hypothesis: the “tethering mechanism”—has been proposed to explain this phenomenon. One study [54] suggested that the extent of spinal cord posterior shift is closely associated with the posterior displacement of the dural sac at the same level. According to this model, postoperative cord migration results primarily from posterior expansion of the dural sac, which, via the tethering effect of the dentate ligaments, pulls the spinal cord dorsally.

Further evidence undermining the classical bowstring theory comes from Sodeyama et al. [30], who found that expanding the laminoplasty decompression span increased posterior cord shift only at adjacent levels, with no significant enhancement in distal segments, contrary to what would be expected if a larger “bow” uniformly amplified cord migration along its entire length. Additionally, Tsuji et al. [57] reported that even in patients undergoing segmental (limited-level) laminoplasty, the spinal cord at the decompressed levels still exhibited substantial posterior shift, further indicating that global cervical curvature may not be the dominant determinant of local cord movement.

In summary, the authors propose that the indirect decompression achieved by laminoplasty likely results from a combined effect of the bowstring mechanism and the tethering mechanism, rather than either principle acting in isolation. Nevertheless, the interplay between these two mechanisms and their relative contributions under varying anatomical and pathological conditions remains incompletely understood and warrants further biomechanical and clinical investigation to elucidate the true pathophysiology of spinal cord decompression in laminoplasty.

We favor a unified “dural sac expansion” model over the classical “bowstring” theory as the primary driver of cord shift. In this model, surgical enlargement of the canal allows the dura to

expand posteriorly, which then translocates the cord via its dural attachments. This explains why limited lordosis does not preclude some posterior migration. However, the efficacy of this migration in achieving clinical decompression remains critically dependent on the absence of rigid anterior tethering, a condition often violated in kyphosis.

### Controversies regarding postoperative cervical alignment and sagittal balance

Postoperative cervical kyphosis may impede posterior spinal cord migration, leading to inadequate decompression [22]. Moreover, cervical kyphotic deformity is associated with an increased risk of sagittal imbalance, chronic neck pain, and recurrence of myelopathy [55]. Maintaining sagittal alignment of the cervical spine is crucial for overall spinal balance. Among the various radiographic parameters, the T1 slope (T1S) has emerged as a key predictor of post-laminoplasty cervical alignment loss [18].

Kim et al. [58] retrospectively analyzed 64 patients who underwent laminoplasty with a 2-year follow-up and stratified them into high- and low-T1S groups based on the preoperative mean T1S. They found that patients with higher preoperative T1S were significantly more prone to postoperative loss of cervical lordosis (74.3% vs. 48.3%) and exhibited greater degrees of alignment deterioration ( $6.2 \pm 10.1^\circ$  vs.  $1.6 \pm 9.3^\circ$ ). Similarly, Lee et al. [59], in a regression analysis, identified a T1S  $> 29^\circ$  as a significant risk factor for postoperative cervical kyphosis following laminoplasty.

In addition, Zhang et al. [17], in a prospective study, demonstrated a significant correlation between the C2-C7 sagittal vertical axis (SVA) and postoperative loss of cervical lordosis: higher preoperative C2-C7 SVA was associated with more pronounced alignment deterioration ( $r = 0.393$ ,  $P < 0.001$ ).

However, Lin et al. [60] reported a more nuanced relationship. Their study showed that regardless of preoperative C2-C7 SVA values, patients with a low T1S ( $\leq 20^\circ$ ) experienced minimal postoperative loss of lordosis, whereas those with a high T1S ( $> 20^\circ$ ) exhibited significantly greater alignment deterioration. Notably, they observed an unexpected phenome-

non: in patients with low T1S but elevated C2-C7 SVA, cervical lordosis actually increased after laminoplasty. This suggests that individuals with a lower T1S may possess a greater compensatory capacity to generate lordosis, thereby counteracting the inherent kyphotic tendency induced by posterior surgery. Consequently, C2-C7 SVA may be better interpreted not as a direct cause of alignment loss, but rather as a reflective parameter of the underlying cervical sagittal status.

Furthermore, some investigators [61] have integrated the K-line with sagittal parameters and introduced the K-line inclination angle, defined as the angle between the K-line and the vertical reference line. Rao et al. [62], in a retrospective analysis of 45 OPLL patients undergoing laminoplasty (mean follow-up: 28.2 months), divided patients into high- and low-K-line inclination groups and found that those with a greater preoperative K-line inclination were more likely to develop postoperative cervical kyphosis and axial symptoms. Compared with T1S, which can be difficult to measure accurately due to poor visualization of the thoracic spine on standard radiographs and C2-C7 SVA, which is susceptible to magnification artifacts as a linear measurement, the K-line inclination angle offers a simpler and more intuitive assessment. Rao et al. [45] therefore proposed that the K-line inclination angle holds significant value in predicting postoperative sagittal balance.

Supporting this, Lee et al. [63] followed 108 CSM patients for 2 years after laminoplasty and reported that a larger K-line inclination angle significantly increased the risk of postoperative sagittal imbalance (defined as C2-C7 SVA  $> 40$  mm), with a critical threshold of  $12.5^\circ$ .

Nevertheless, conflicting evidence exists. Another study [64] found that the K-line inclination angle failed to predict postoperative changes in cervical curvature. Thus, the prognostic utility of the K-line inclination angle remains inconclusive and warrants validation through larger, multicenter cohorts with long-term follow-up.

In summary, while T1S appears to be a robust predictor of post-laminoplasty cervical alignment loss, the interplay among T1S, C2-C7 SVA, and dynamic parameters such as the K-line inclination angle underscores the com-

plexity of sagittal balance. Future research should focus on integrating these parameters into a comprehensive, individualized preoperative risk assessment model to optimize surgical planning and improve long-term outcomes.

We consider the T1 slope (T1S) to be the most pivotal preoperative predictor of post-laminoplasty alignment loss. A high T1S ( $> 25\text{-}30^\circ$ ) indicates a high gravitational moment arm that the compromised posterior musculature after surgery cannot counteract, predisposing one to kyphosis. Therefore, in patients with high T1S, even mild preoperative kyphosis should be viewed with extreme caution, and strategies to preserve or augment posterior tension band integrity should be prioritized.

### **Relationship between preoperative cervical alignment and postoperative axial symptoms**

Axial symptoms characterized by chronic neck pain, stiffness, and referred shoulder discomfort are among the most common complications following cervical laminoplasty. Iseda et al. [56], in a 7-year follow-up study of 106 patients who underwent muscle- and ligament-preserving laminoplasty, found that although neurological recovery was comparable between patients with global cervical kyphosis accompanied by pronounced segmental kyphosis and those with overall lordotic alignment without significant local kyphosis, the incidence of axial symptoms was markedly higher in the kyphotic group (21.9% vs. 1.4% in the lordotic group). Notably, among patients with preoperative kyphotic deformity, those who experienced postoperative loss of cervical alignment exhibited an axial symptom rate as high as 46%, whereas those who maintained favorable postoperative curvature had a rate of only 5%. These findings underscore the critical importance of preserving cervical sagittal alignment in mitigating postoperative axial symptoms.

However, conflicting evidence exists. Gong et al. [65], in a study of 79 CSM patients with a mean follow-up of 36 months, reported that preoperative cervical curvature was not an independent risk factor for axial symptoms ( $P = 0.221$ ). From the authors' perspective, it is not preoperative kyphosis per se, but rather the combination of preexisting kyphotic deformity and postoperative loss of cervical alignment that may jointly contribute to cervical instability

and abnormal stress distribution across the posterior elements, thereby triggering axial symptoms. In isolation, preoperative kyphosis may not directly cause axial pain.

Although the precise pathophysiology of axial symptoms remains incompletely understood, the prevailing consensus attributes their development to a multifactorial etiology. Proposed mechanisms include injury to the facet joint capsules, excessive laminar opening angle, postoperative cervical instability, reduced cervical range of motion, and damage or atrophy of the posterior cervical extensor musculature [26]. Additionally, prolonged use of cervical collars postoperatively has also been implicated as a potential contributing factor, possibly due to disuse-induced muscle deconditioning.

In summary, while preoperative cervical kyphosis alone may not be a definitive predictor of axial symptoms, the dynamic interplay between preoperative alignment and postoperative sagittal balance appears pivotal. Preservation of cervical lordosis and minimization of iatrogenic biomechanical disruption during surgery are essential strategies to reduce the risk of this debilitating complication.

### **Summary and future perspectives**

In summary, considerable controversy persists regarding the efficacy and complication profile of laminoplasty in patients with cervical spondylotic myelopathy (CSM) accompanied by cervical kyphosis. Based on the synthesis of existing evidence and our clinical experience, we contend that laminoplasty retains a limited but definite role in this population. Its successful application is not governed by a single parameter but by the convergence of multiple favorable factors: mild, flexible kyphosis; a positive dynamic K-line; a canal occupancy ratio  $< 60\%$ ; and the absence of a high T1 slope. For patients with mild kyphotic deformity meeting these criteria, laminoplasty may remain a viable surgical option - provided a comprehensive preoperative assessment is performed, incorporating factors such as spinal cord signal intensity on MRI, the magnitude and pattern of local kyphosis, and the nature of anterior compression. When these criteria are not met, laminoplasty likely represents a suboptimal biomechanical solution, and alternative anterior or combined approaches should be employed.

Moreover, postoperative changes in cervical sagittal alignment significantly influence surgical outcomes. Consequently, risk factors associated with loss of cervical lordosis - including T1 slope, C2-C7 SVA, and K-line parameters warrant thorough evaluation and ongoing investigation. The long-term trajectory of cervical curvature and the development of complications such as axial symptoms have profound implications for both functional recovery and health-related quality of life. Therefore, future studies should prioritize long-term follow-up (> 5 years) to better understand the durability of outcomes and the natural history of postoperative alignment changes.

Regarding the underlying mechanism of spinal cord decompression in laminoplasty, the relative contributions and interplay between the bowstring principle and the tethering (or dural expansion) mechanism remain incompletely elucidated and require further biomechanical and clinical validation.

With ongoing advances in imaging technology, biomechanical modeling, and evidence-based research, it is reasonable to anticipate that these current controversies will be progressively resolved. Such progress will ultimately enable more precise patient selection, refined surgical decision-making, and most importantly more effective and scientifically grounded clinical management of CSM.

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### Disclosure of conflict of interest

None.

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