



Cervical sagittal alignment after multilevel ACDF: correction goes along with loss of compensation

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Abstract

Background Cervical spondylosis can cause loss of lordosis or even kyphosis, altering sagittal alignment and triggering compensatory mechanisms to maintain horizontal gaze. Despite the widespread use of multilevel anterior cervical discectomy and fusion (ACDF), its impact on cervical sagittal alignment and compensatory mechanisms remains unclear. This study investigates degenerative changes in cervical sagittal alignment and compensatory mechanisms before and after multilevel ACDF.

Methods A retrospective analysis was conducted on 290 patients who underwent multilevel ACDF (2–3 levels) for cervical spondylosis. Preoperative and postoperative sagittal parameters including C0–C2 lordosis, C2–C7 lordosis, surgical (fused-level) lordosis, unfused-level lordosis, T1 slope (T1S), T1S minus cervical lordosis (T1S–CL), and cervical sagittal vertical axis (c–SVA) were measured using a previously described deep learning model. Demographic data were collected at the same time points. Changes from baseline to follow-up were calculated. Correlations between changes in surgical lordosis and sagittal parameters were assessed via linear regression, Pearson’s correlation, and multiple logistic regression analysis.

Results Mean preoperative C2–C7 lordosis was $11.4^\circ \pm 13.7^\circ$, increasing by 6.5° in two-level, and 8.6° in three-level ACDF patients. Statistically significant improvements were observed in C2–C7 lordosis, C0–C2 lordosis, and T1S–CL mismatch (all $p < 0.01$). Changes in surgical lordosis correlated negatively with changes in unfused segment lordosis ($R = -0.26$, $p < 0.01$). The strongest negative correlation was observed between changes in C2–C7 and changes in C0–C2 lordosis ($R = -0.60$, $p < 0.01$), highlighting subaxial realignment’s impact on upper cervical compensation. A moderate positive correlation emerged between changes in surgical lordosis and changes in T1S ($R = 0.29$, $p < 0.01$). Subgroup analyses by fusion area confirmed the consistency of these relationships across different ACDF configurations.

Conclusions Multilevel ACDF leads to a gain of surgical lordosis and reduces compensatory mechanisms. By recognizing and accounting for preoperative compensatory mechanisms, surgeons can optimize surgical planning to achieve a more stable and biomechanically favorable alignment. We propose a three-step algorithm to analyze cervical sagittal alignment and take into consideration the presence of compensatory mechanisms of the upper cervical, the unfused subaxial and when available the global spine.

Level of evidence IV.

Keywords multilevel ACDF · Compensatory mechanisms · Cervical lordosis · C0–C2 · Machine learning · Cervical sagittal alignment

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Introduction

The aging process is marked by degenerative changes such as reduced disc hydration and height, hypertrophy of the facet joints, osteophyte formation, and muscle atrophy, leading to a condition known as cervical spondylosis [1]. In symptomatic patients, these degenerative changes can disrupt the biomechanics of the cervical spine, alter its natural alignment, and potentially lead to segmental kyphosis [1], increasing the risk of cervical sagittal imbalance. This condition may lead to hyperextension of the upper cervical spine (C0–C2) and the adjacent non-pathological lower segments (C3–C7), accompanied by a decrease in T1 tilt. Except for the loss of native alignment in the degenerated segment(s), the other changes mentioned represent compensatory mechanisms to maintain an adequate horizontal gaze. Therefore, the severity of cervical spine degeneration is illustrated by the extent of structural damage but also by the magnitude of the body's attempt to compensate for these changes. Recognizing and accounting for these adaptive changes during the evaluation of patients with cervical spondylosis is essential to avoid underestimation of the severity of the condition and to guide effective surgical strategies.

Studies have shown that cervical sagittal imbalance, described as an increase in the cervical sagittal vertical axis (c-SVA) values, is associated with increased pain, disability and reduced quality of life [2]. This may result from the strain imposed on the cervical spine to maintain an adequate horizontal gaze.

This study aims to describe cervical sagittal alignment and compensatory mechanisms in patients with cervical spondylosis by highlighting the current evidence in literature and analyzing changes in cervical sagittal alignment from preoperative to follow-up stages in a large cohort of patients who underwent multilevel anterior cervical discectomy and fusion (ACDF) for cervical spondylosis. We also propose a three-step algorithm to analyze cervical sagittal alignment and take into consideration the presence of compensatory mechanisms of the upper cervical, the unfused subaxial and when available the global spine.

Cervical sagittal alignment

Measurement and physiological alignment

Recent studies on asymptomatic individuals have shown that the cervical spine can exhibit various alignments [3, 4], including neutral, kyphotic, lordotic, and sigmoid alignments. As such, a cervical kyphotic spine cannot be defined as deformed without the presence of symptoms and an inability to maintain a horizontal gaze.

In degenerative diseases, assessing global cervical alignment is crucial. One common approach is to measure the c-SVA, defined as the distance between a vertical line passing through the center of the vertebral body of C2 and the posterosuperior corner of C7 [5].

Another method for assessing cervical alignment involves measuring the center of gravity sagittal-vertical axis distance (COG-SVA) [6]. Full spine radiographs in a standing position are required to measure the distance between a vertical line passing through the COG (located at the anterior portion of the external auditory canal) and the posterosuperior corner of the sacrum. Physiologically, the distance between SVA and COG-SVA is below 3 cm. However, in severe degenerative disease with focal kyphotic deformities, this distance increases, reflecting a significantly imbalanced cervical spine.

Cervical lordosis (CL) can be measured using various methods. The lordosis of the upper cervical spine is assessed by the angle formed between McGregor's line (connecting the posterior margin of the hard palate to the posterior edge of the foramen magnum) and the inferior endplate of the C2 vertebral body [7]. The lordosis of the lower cervical spine is commonly measured using the Cobb angle between C2 and C7. In asymptomatic individuals, most of the cervical lordosis (75–80%) is located between C0 and C2, which also explains the high degree of flexion-extension mobility in this region [7].

The cervical spine rests on the proximal thoracic spine, where the upper endplate of T1 forms an angle with the horizontal line. This angle is known as the T1 slope (T1S) and is crucial in determining the amount of subaxial lordosis required to maintain the head's COG in a balanced position [8]. This parameter represents the trunk alignment and is influenced by global spinal alignment, with both physiological and pathological variations of the spine significantly impacting this parameter (Fig. 1).

Similar to the lumbosacral spine, the mismatch between the cervical spine and lower spine can be assessed by measuring the T1 slope-cervical lordosis mismatch (T1S-CL), providing a measure of the neck response to trunk alignment.

Although previous studies [9, 10] have described models for predicting normative value for T1S-CL, these may not be universally applicable, particularly in cases of adult spinal deformity with altered global spine alignment. (Fig. 2)

Compensatory local and global mechanisms of the spine

The compensatory mechanisms are influenced by the extent of degenerative pathology, spinal stiffness, the patient's pain level, and the possible presence of cervical and global

Fig. 1 Variability of T1 slope (T1S) angular value: Evaluating the T1S requires analyzing the entire spine to identify potential compensatory mechanisms. In the figure on the left, the patient presents with lumbar hyperlordosis associated with a reduced sacral slope (SS) and increased pelvic tilt (PT). In the figure on the right, after normalizing the patient's SS and thereby removing the lumbosacral compensatory mechanism, the C7PL moves anteriorly and T1S value changes. C7PL=C7 plumb line. *This is a schematic representation intended to illustrate biomechanical principles; parameters such as C7 plumb line, sacral slope, and pelvic tilt were not quantitatively assessed in this study*

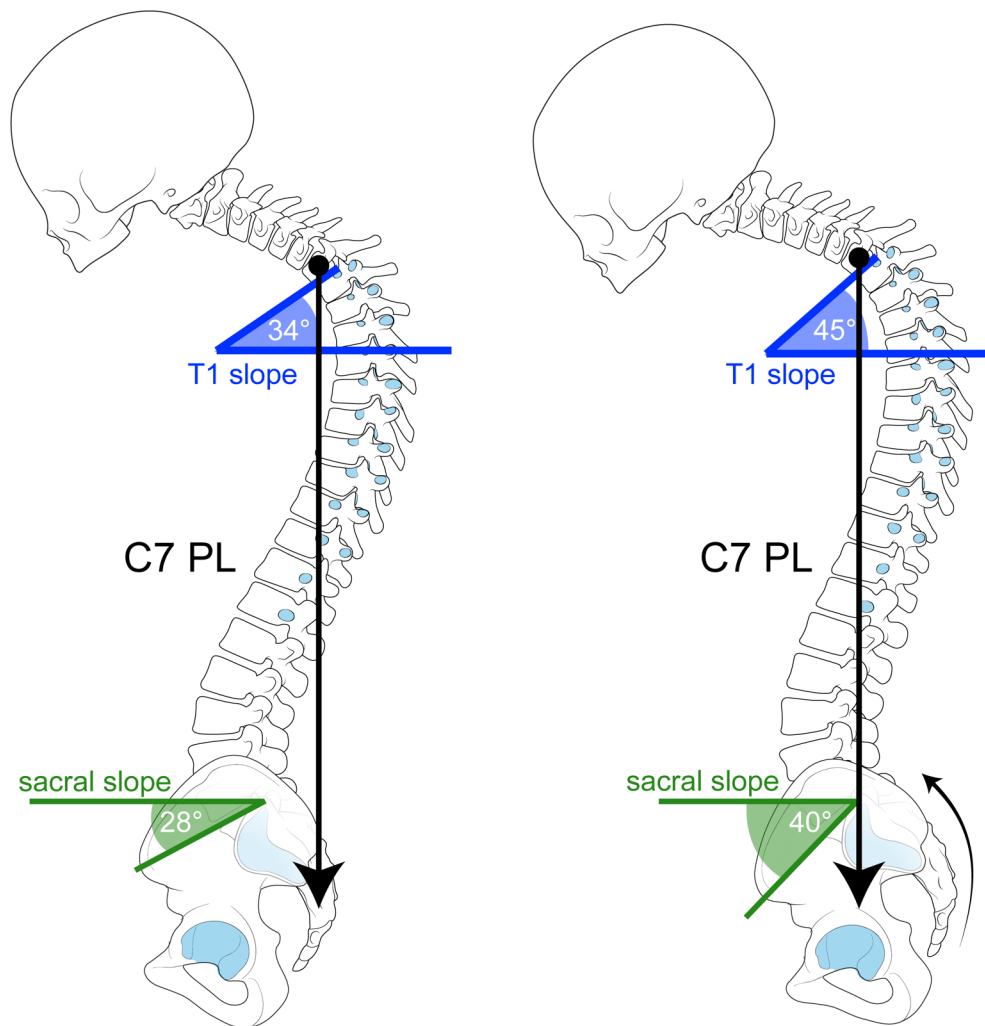
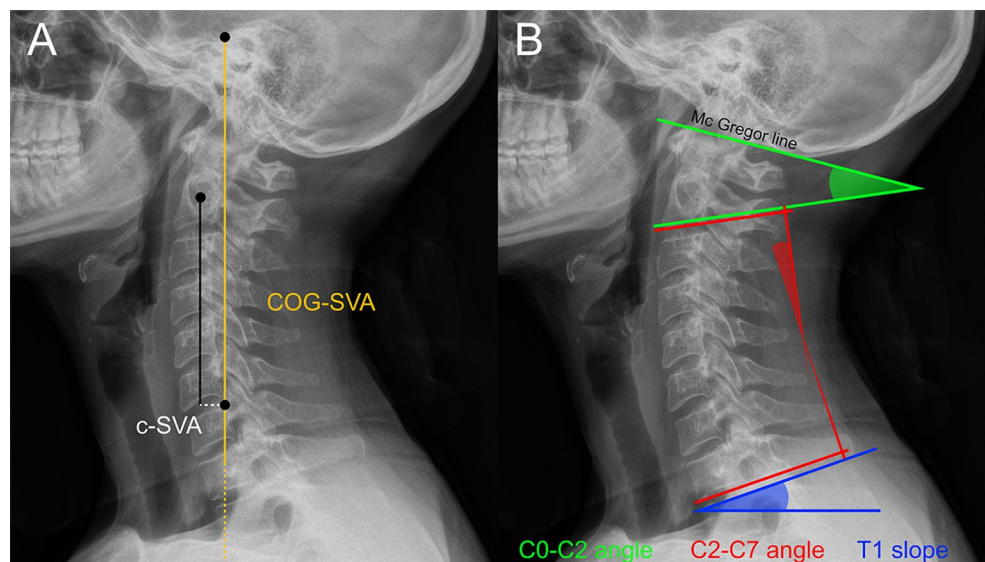


Fig. 2 Methods for assessing cervical spine alignment (A) and measuring C0-C2 lordosis, C2-C7 lordosis, and T1 Slope (B). c-SVA=cervical sagittal vertical axis; COG-SVA=center of gravity sagittal vertical axis



sagittal imbalance. As segmental degeneration progresses, the load axis shifts forward, accelerating disc degeneration, leading to segmental loss of lordosis or eventually increased

segmental kyphosis. To counteract these changes, the adjacent segments extend, helping to compensate for the anterior displacement of the load axis.

Compensatory mechanisms can be classified as local or global. Local mechanisms are limited to the cervical spine itself, while global mechanisms involve the entire spine and typically occur in cases of severe cervical deformities.

Local compensatory mechanisms may occur either in the C0–C2 spine or in adjacent segments to the degenerated level(s).

In cases of degeneration in the lower cervical spine, both adjacent levels and the upper cervical spine hyperextend. According to Kim et al. [11], an ACDF procedure not only affects the lordosis at the surgical and adjacent levels but can also indirectly influence upper cervical lordosis (C0–C2). Specifically, the procedure led to an increase in lordosis at the surgical level, while reducing lordosis at the adjacent levels and in the upper cervical spine (Fig. 3).

In cases of C0–C2 degeneration and segmental kyphosis, the C2–C7 area extends as a compensatory mechanism, resulting from a downward sequential change [12].

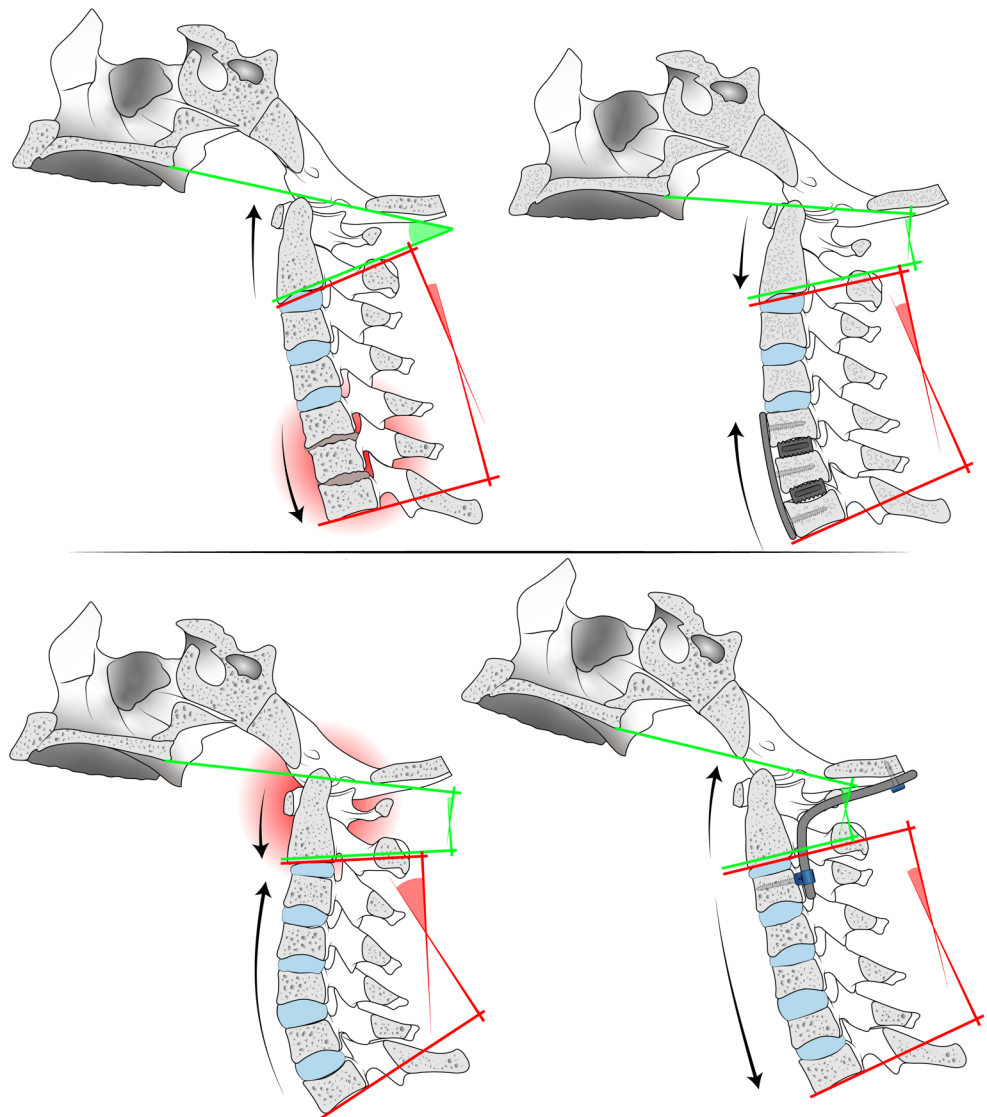
Global compensatory mechanisms, as described by Mizutani et al. [13], are rarely seen but can occur in patients with significant occipito-trunkal discordance, defined by a COG-SVA greater than 3 cm. These mechanisms typically arise when the cervical spine is fused and unable to compensate, prompting compensation in the lumbosacral spine (Fig. 4).

Materials and methods

Study sample

After Institutional Review Board approval (BASEC-ID 2023-01683), and in accordance with the Declaration of Helsinki for medical research involving human subjects, a retrospective analysis of prospectively collected data from a tertiary care spine center was carried out using the local

Fig. 3 Representation of compensatory mechanisms in subaxial cervical spondylosis (c3–c7, upper figures) and proximal cervical spondylosis (c0–c2, lower figures). When degenerative disease causes segmental kyphosis – defined as an abnormal forward curvature involving one or more motion segments and expressed as a positive Cobb angle between adjacent cervical vertebrae on lateral radiographs – non-pathological levels hyperextend to maintain horizontal gaze. Following surgical correction of the pathological levels (right-hand panels), the non-pathological segments reduce their hyperextension, thereby decreasing or eliminating the compensatory mechanism



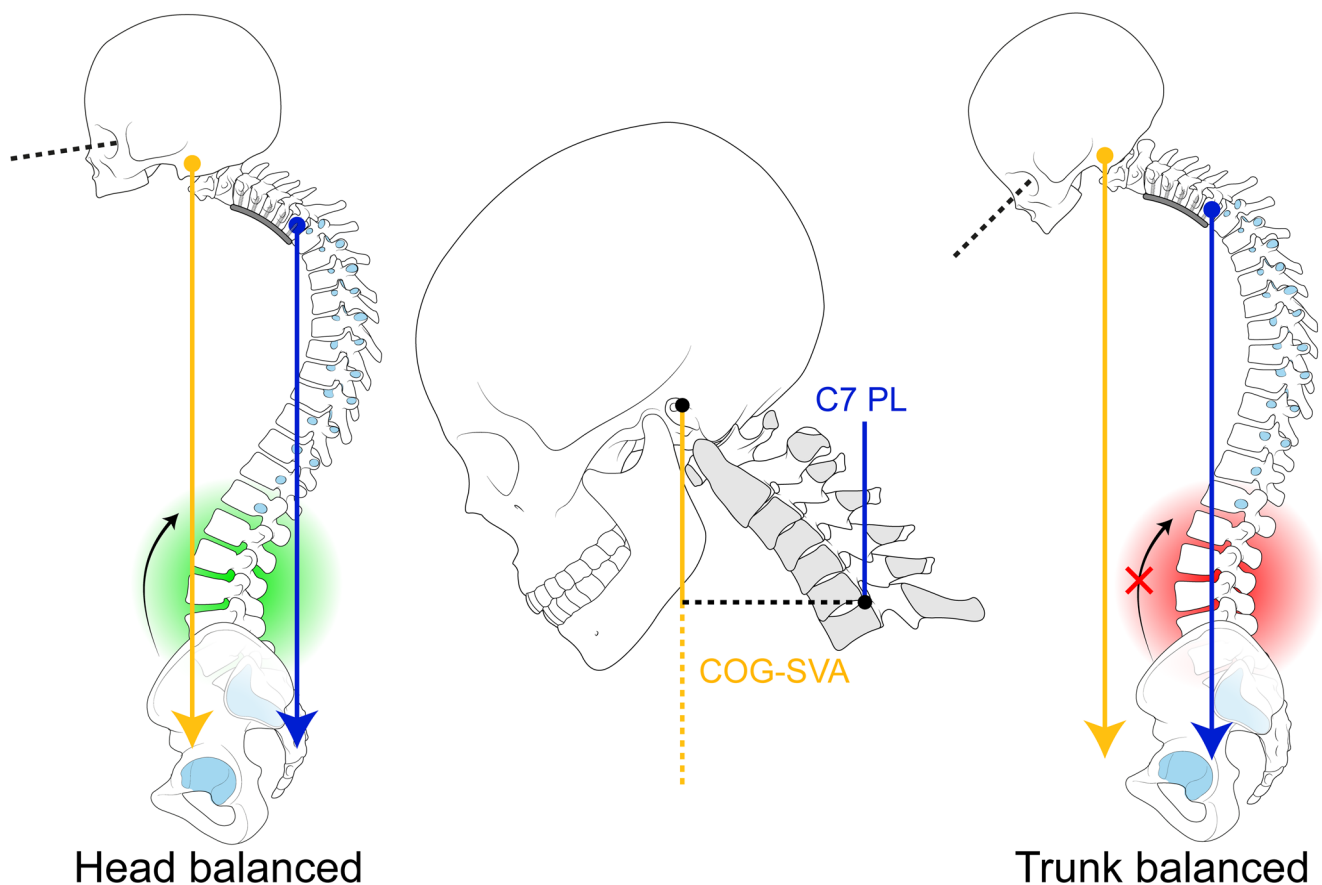


Fig. 4 Compensatory mechanisms in occipitotrunkal imbalance (central figure, with increased distance between C7PL and COG-SVA > 3 cm) and fused cervical spine. In the figure on the left, the patient achieves head balance (Head Balanced) through lumbosacral spine extension. In the figure on the right, the patient is unable to balance the head, achieving only trunk balance (Trunk Balanced), due to

lumbosacral extension impairment caused by degenerative disease in that region. *This is a schematic representation intended to illustrate biomechanical principles; Concepts such as head balanced and trunk balanced phenotypes and parameters such as C7PL and COG-SVA were not assessed or measured in this study*

spine registry together with the local radiographic database was performed. Patients treated with multi-level ACDF for degenerative disease were included. Subjects with non-available imaging or without a minimum 1-year follow-up were excluded.

A dataset of anonymized preoperative and postoperative lateral cervical x-ray images was acquired from the local radiographic database and processed using a novel automated image analysis algorithm [14] for the comprehensive measurement of sagittal cervical spine alignment parameters. This model employed a heatmap-based convolutional neural network to automatically detect 29 cervical spine landmarks from lateral x-rays, generating probability heatmaps that were converted into precise coordinates. Each x-ray was then reviewed by a spine surgeon, who confirmed, discarded, or recalibrated the landmarks based on their position and the image's adherence to inclusion criteria.

Images were excluded if they met at least one of the following conditions: inability to identify the McGregor line,

difficulty in recognizing the T1 upper endplate, absence of landmarks due to vertebral fusion, or abnormal landmark positioning caused by a vertebral fracture.

Data collection

Demographic data, age, gender, Charlson Comorbidity Index (CCI) score, American Society of Anaesthesiology (ASA), and Body Mass Index (BMI) were collected from the local spine registry. This Database was introduced in 2004 and collects surgical and patient-rated outcome.

Sagittal cervical spine alignment was evaluated by measuring C2-C7 lordosis, C0-C2 lordosis, T1S, surgical (fused-level) lordosis, unfused-level lordosis, and the c-SVA, both in the preoperative and follow-up periods (postoperative, 3 months, 6 months and 1-year).

Surgical lordosis was defined as the Cobb angle between the superior endplate of the most cranial and the inferior

endplate of the most caudal vertebra within the fused segment. Unfused lordosis represents the curvature of the cervical segments not included in the fusion (above and/or below the construct) and was calculated as the difference between the overall C2–C7 lordosis and the surgical lordosis. A segment was defined as kyphotic when the Cobb angle changed sign, indicating reversal of normal cervical curvature. Loss of lordosis was identified when the segmental angle fell below established normative values, typically ranging from -4° to -6° in asymptomatic adults [15]. Angles approaching 0° or turning positive were thus considered abnormal. This evaluation was supported by both quantitative measurements and qualitative radiographic assessment, including signs such as disc space narrowing or osteophyte formation.

Once the dataset was completed, the difference (delta, Δ) between the preoperative and follow-up values (3 months if available, otherwise 6 months or 1-year) for surgical lordosis, C2–C7 and C0–C2 lordosis, T1S, T1S - CL and unfused-levels lordosis were calculated.

The primary outcomes were to calculate the variations in these parameters and investigate the relation between Δ surgical lordosis and Δ C2–C7 lordosis, Δ C0–C2 lordosis, Δ T1S, Δ unfused-levels' lordosis, Δ T1S - CL and the relation between Δ C2–C7 lordosis and Δ C0–C2 lordosis.

Statistical analysis

After collection of x-rays parameters and outcomes the entire cohort was subdivided. First, we evaluated the results for the entire cohort, assessing for differences among groups via Student T-Test. Following sub analysis for patients with two- and three-level ACDF were performed with the same analysis.

Subsequently, to verify the primary outcomes univariate and multivariate analyses were performed in the entire cohort and both subgroups. For each radiological parameter a linear regression analysis was performed calculating p-values, R^2 values and Pearson's correlation R values. The differences between the two subgroups were assessed using a hypothesis test based on the Student's T test.

A multivariate analysis for prespecified patient-specific variables, such as ASA score, or CCI score, or age and gender was performed.

All statistical analyses were conducted with Jamovi (The Jamovi Project, Sydney, Australia) software. P value < 0.05 was considered significant.

Results

Demographic data

A total of 289 patients (149 females, 51.3%) met the inclusion criteria: 245 with to 2-level ACDF, 44 with 3-level ACDF. Mean age at surgery was 59.5 ± 11.5 (range 26 to 86) with a preponderance of patients below 65 years (68.8%).

With a mean BMI of 25.7 ± 4.5 , 29.2% had an ASA above 2 and 6.7% had a CCI above 4 (Table 1).

Radiological parameters and correlations

The mean preoperative C2–C7 lordosis in the overall cohort was $11.4^{\circ} \pm 13.7^{\circ}$, with a mean C0–C2 lordosis of $20.5^{\circ} \pm 13.1^{\circ}$, a T1 slope (T1S) of $32.3^{\circ} \pm 9.8^{\circ}$, a T1S–CL mismatch of $20.9^{\circ} \pm 11.5^{\circ}$, and a cSVA of 29.3 ± 15.6 mm.

After surgery, postoperative radiographic values in the overall cohort showed a C2–C7 lordosis of $19.4^{\circ} \pm 15.3^{\circ}$, a C0–C2 lordosis of $13.9^{\circ} \pm 12.5^{\circ}$, a T1 slope of $33.5^{\circ} \pm 8.9^{\circ}$, a T1S–CL mismatch of $14.1^{\circ} \pm 14.3^{\circ}$, and a cSVA of 31.3 ± 15.2 mm. The average postoperative surgical lordosis was $9.4^{\circ} \pm 9.4^{\circ}$, while unfused lordosis measured $11.9^{\circ} \pm 14.4^{\circ}$ (Table 2). Across the entire cohort, mean radiographic changes between pre- and postoperative assessments included: Δ C2–C7 lordosis of $8.0^{\circ} \pm 14.9^{\circ}$, Δ C0–C2 lordosis of $-6.5^{\circ} \pm 13.6^{\circ}$, Δ T1 slope of $1.1^{\circ} \pm 9.1^{\circ}$, Δ unfused lordosis of $1.2^{\circ} \pm 12.7^{\circ}$, and Δ T1S–CL mismatch of $-6.8^{\circ} \pm 15.1^{\circ}$.

When comparing pre- and postoperative values within the cohort, statistically significant changes were observed in several alignment parameters, including C2–C7 lordosis, C0–C2 lordosis, T1S–CL mismatch, surgical lordosis, and unfused lordosis (Table 2). Upon stratifying the cohort by fusion length (2-level vs. 3-level ACDF), the average Δ surgical lordosis was $6.5^{\circ} \pm 11.8^{\circ}$ for 2-level ACDF patients and $8.6^{\circ} \pm 10.6^{\circ}$ for 3-level ACDF patients (Table 2). However, no significant differences were found between groups for most preoperative, postoperative and variations parameters—except for postoperative surgical lordosis, which was significantly greater in the 3-level group ($p=0.02$).

In the total cohort, a moderate positive correlation was found between Δ surgical lordosis and Δ C2–C7 lordosis ($R=0.57$), showing that increases in surgical lordosis are

Table 1 Baseline characteristics of patients included in the study

	(Avg $\pm$ SD)
Age	59.5 ± 11.5
♂/♀	149/141
CCI	3.1 ± 1.5
ASA	1.28 ± 3.2
BMI	25.7 ± 4.5

Data are presented as mean value and standard deviation (Avg $\pm$ SD), when available. CCI=Charlson comorbidity Index; ASA=American society of Anesthesiology; AVG=Average value

Table 2 Preoperative, postoperative and variations radiological parameters of patients included in the study, stratified per fusion area

	Total cohort (Avg±SD)	ACDF 2 levels (Avg±SD)	ACDF 3 levels (Avg±SD)	<i>P</i> -value (2 vs. 3 lev.)	<i>P</i> -value (pre vs. post total cohort)
<i>Preoperative</i>					
<i>N</i>	<i>N</i> =289	<i>N</i> =245	<i>N</i> =44		
C2-C7 lordosis	11.4° ± 13.7°	11.62° ± 13.4°	10.1° ± 15.2°	0.54	
C0-C2 lordosis	20.5° ± 13.1°	20.4° ± 13.3°	20.5° ± 11.8°	0.98	
T1S	32.3° ± 9.8°	32.5° ± 9.4°	31.1° ± 11.6°	0.99	
T1S – CL	20.9° ± 11.5°	20.9° ± 11.4°	20.9° ± 11.9°	0.31	
c-SVA	29.3 ± 15.6 mm	29.3 ± 15.6 mm	30.2 ± 16.8 mm	0.71	
Surgical lordosis	2.3° ± 10.4°	2.3° ± 10.4°	4.1° ± 12.9°	0.39	
Unfused lordosis	9.3° ± 12.0°	9.3° ± 12.0°	6.0° ± 9.7°	0.05	
<i>Postoperative</i>					
C2-C7 lordosis	19.4° ± 15.3°	19.5° ± 15.0°	19.0° ± 15.6°	0.84	<0.0001
C0-C2 lordosis	13.9° ± 12.5°	13.9° ± 12.6°	13.9° ± 11.5°	≈ 1	<0.0001
T1S	33.5° ± 8.9°	33.3° ± 8.8°	34.7° ± 10.3°	0.40	0.06
T1S – CL	14.1° ± 14.3°	13.8° ± 14.5°	15.6° ± 12.1°	0.38	<0.0001
c-SVA	31.3 ± 15.2 mm	30.6 ± 14.7 mm	35.5 ± 16.7 mm	0.07	0.08
Surgical lordosis	9.4° ± 9.4°	8.8° ± 9.1°	12.7° ± 9.7°	0.02	<0.0001
Unfused lordosis	11.9 ± 14.4°	12.1° ± 14.7°	11.2° ± 12.3°	0.66	0.019
<i>Variations</i>					
Δ C2-C7 lordosis	8.0° ± 14.9°	7.9° ± 15.2°	8.9° ± 13.6°	0.65	
Δ C0-C2 lordosis	−6.5° ± 13.6°	−6.5° ± 14.1°	−6.6° ± 10.8°	0.97	
Δ T1 slope	1.1° ± 9.1°	0.7° ± 9.1°	3.6° ± 9.0°	0.054	
Δ Surgical lordosis	6.8° ± 11.6°	6.5° ± 11.8°	8.6° ± 10.6°	0.23	
Δ Unfused lordosis	1.2° ± 12.7°	1.4° ± 12.7°	0.3° ± 12.6°	0.60	
Δ T1S-CL	−6.8° ± 15.1°	−7.1° ± 15.5°	−5.3° ± 12.3°	0.38	

Data are presented as mean value and standard deviation (Avg±SD), when available. Δ=difference among preoperative and postoperative values, T1S=T1 slope, T1S-CL=T1 slope – cervical lordosis mismatch, surgical lordosis=lordosis present in fusion area, unfused lordosis=lordosis present in unfused segments, AVG=Average value. Numbers in bold show statistical significance ($p<0.05$)

associated with increases in C2-C7 lordosis, with the model being highly significant ($F=135.77$, $p<0.0001$). A negative correlation was found between Δ surgical lordosis and Δ unfused lordosis ($R = -0.26$), with a highly significant regression model ($F=26.28$, $p<0.0001$), suggesting that increased surgical lordosis is associated with a reduction in compensatory lordosis at unfused levels. Likewise, the correlation between Δ surgical lordosis and Δ C0-C2 lordosis was negative ($R = -0.11$), indicating that increased surgical lordosis tends to correspond with decreases in compensatory C0-C2 lordosis; however, this relationship missed statistical significance ($F=3.55$, $p=0.061$). The strongest correlation was found between Δ C2-C7 lordosis and Δ C0-C2 lordosis, which exhibited a moderate-to-strong negative correlation ($R = -0.60$) with a highly significant regression model ($F=168.45$, $p<0.0001$) (Fig. 5).

A weak-to-moderate positive correlation was observed between Δ surgical lordosis and Δ T1 slope ($R=0.29$), indicating that increases in surgical lordosis are linked to increases in T1S, with a highly significant regression model ($F=26.28$, $p<0.0001$). Lastly, a moderate negative correlation was observed between Δ surgical lordosis and Δ T1S – CL ($R = -0.40$), suggesting that increases in surgical lordosis are associated with decreases in T1S – CL, with a

highly significant regression model ($F=51.99$, $p<0.0001$) (Fig. 5).

To assess whether the correlation patterns observed in the total cohort were influenced by fusion level, subgroup analyses were performed for common ACDF configurations (C3–C5, C3–C6, C4–C6, C4–C7, and C5–C7). The results demonstrated consistent trends across all subgroups, mirroring those of the overall cohort—particularly the positive correlation between Δ surgical lordosis and Δ C2–C7 lordosis, and the negative correlation with Δ C0–C2 lordosis. The strongest association between surgical and subaxial correction was found in the C5–C7 group ($R=0.75$, $p<0.001$), while the most pronounced inverse relationship with cranio-cervical compensation emerged in the C4–C6 group ($R = -0.78$, $p<0.001$) (Table 3).

Multivariate analysis of the entire cohort revealed non-statistically significant demographic correlations, and radiological parameters remained significant. A secondary analysis was conducted specifically on patients who underwent three-level ACDF to compare their results with those of the entire cohort. Even within this subgroup, non-statistically significant correlations were identified, while radiological parameters remained significant.

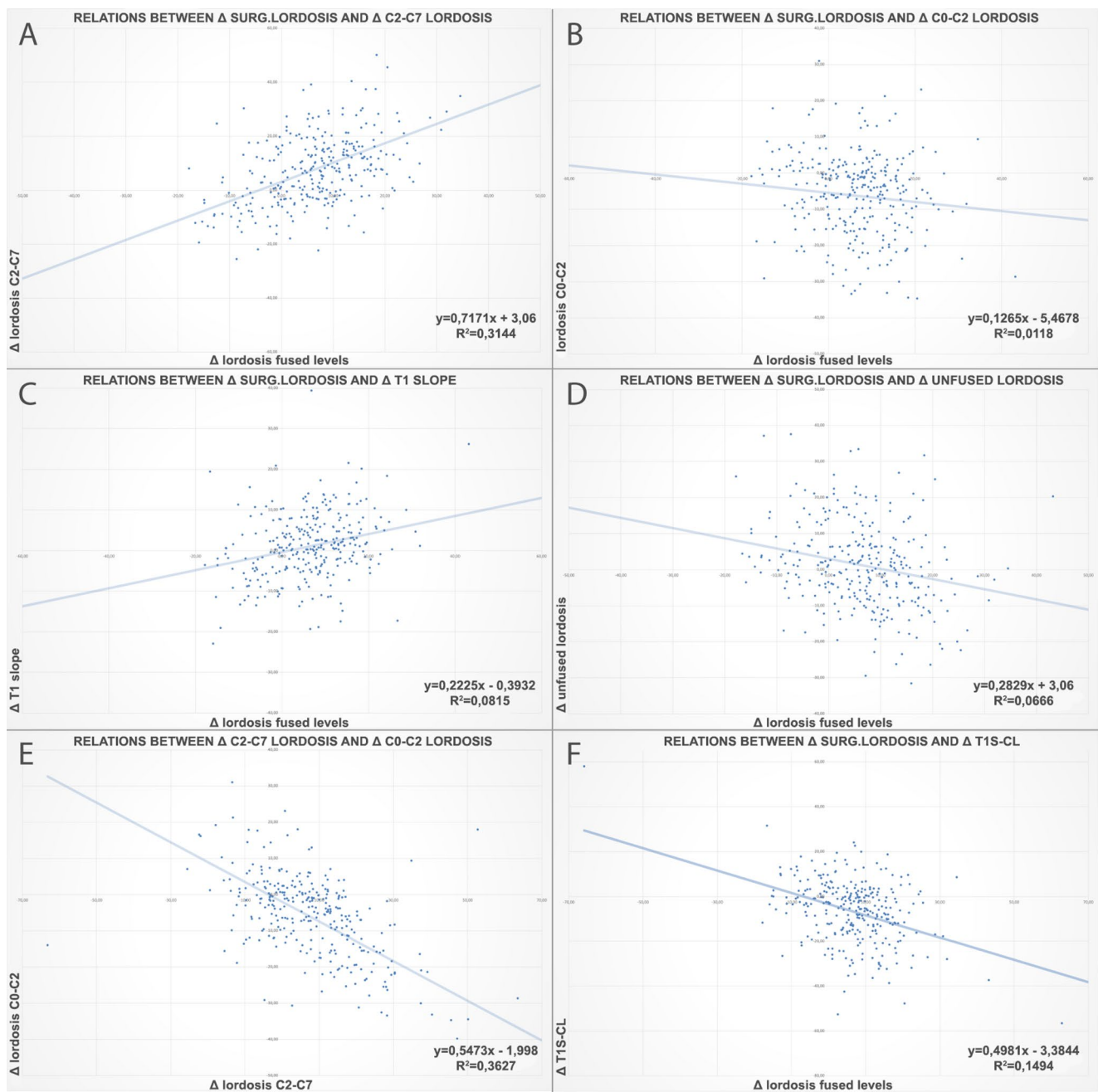


Fig. 5 Graphs of correlations obtained in the study population: Correlations between Δ surgical lordosis and: **(a)** Δ C2–C7 lordosis ($R=0.57$; $R^2=0.31$; $F=135.77$; $p<0.0001$), **(b)** Δ C0–C2 lordosis ($R=-0.11$; $R^2=0.02$; $F=3.55$; $p=0.061$), **(c)** Δ T1S ($R=0.29$; $R^2=0.08$; $F=26.28$; $p<0.0001$), **(d)** Δ lordosis of unfused segments ($R=-0.26$;

$R^2=0.06$; $F=26.28$; $p<0.0001$), **(f)** and Δ T1S–CL ($R=-0.40$; $R^2=0.14$; $F=51.99$, $p<0.0001$), and **(e)** the correlation analysis between Δ C2–C7 lordosis and Δ C0–C2 lordosis ($R=-0.6$; $R^2=0.36$; $F=168.45$; $p<0.0001$)

Table 3 Correlation analysis between changes in surgical lordosis (Δ Surg. lord.) and other radiographic alignment parameters, stratified by fusion area

	C3-C5 (<i>R</i> ; <i>R</i> ² ; <i>F</i> ; <i>p</i>) <i>N</i> =32	C3-C6 (<i>R</i> ; <i>R</i> ² ; <i>F</i> ; <i>p</i>) <i>N</i> =23	C4-C6 (<i>R</i> ; <i>R</i> ² ; <i>F</i> ; <i>p</i>) <i>N</i> =89	C4-C7 (<i>R</i> ; <i>R</i> ² ; <i>F</i> ; <i>p</i>) <i>N</i> =26	C5-C7 (<i>R</i> ; <i>R</i> ² ; <i>F</i> ; <i>p</i>) <i>N</i> =119
Δ Surg. lord. $\leftrightarrow \Delta$ C2-C7	0,38; 0,15; 5,19; 0,03	0,10; 0,01; 0,18; 0,68	0,34; 0,12; 11,34; < 0,0001	0,33; 0,11; 2,86; 0,12	0,75; 0,56; 151,85; < 0,0001
Δ Surg. lord. $\leftrightarrow \Delta$ C0-C2	-0,09; 0,01; 0,23; 0,63	0,02; 0,00; 0,01; 0,94	-0,19; 0,04; 3,19; 0,08	0,12; 0,01; 0,31; 0,58	-0,05; 0,00; 0,35; 0,56
Δ Surg. lord. $\leftrightarrow \Delta$ T1S	0,13; 0,02; 0,51; 0,48	-0,17; 0,03; 0,49; 0,50	0,22; 0,05; 4,64; 0,03	0,15; 0,02; 0,52; 0,48	0,34; 0,12; 15,42; < 0,0001
Δ Surg. lord. $\leftrightarrow \Delta$ Unfu. lord.	-0,33; 0,11; 3,65; 0,07	-0,70; 0,49; 15,59; < 0,0001	-0,35; 0,12; 12,06; < 0,0001	-0,41; 0,17; 4,5; 0,05	-0,14; 0,019; 2,21; 0,140
Δ C2-C7 $\leftrightarrow \Delta$ C0-C2	-0,65; 0,42; 21,93; < 0,0001	-0,30; 0,09; 1,58; 0,23,	-0,78; 0,61; 136,85; < 0,0001	-0,47; 0,22; 6,23; 0,02	-0,46; 0,21; 31,39; < 0,0001
Δ Surg. lord. $\leftrightarrow \Delta$ T1S-CL	-0,27; 0,07; 2,38; 0,13	-0,21; 0,04; 0,71; 0,41	-0,18; 0,03; 3,05; 0,08	-0,21; 0,04; 0,99; 0,33	-0,58; 0,34; 60,62; < 0,0001

Data are presented as pearson's correlation coefficient (*R*), coefficient of determination (*R*²), *F* statistic, and *p*-value (*R*; *R*²; *F*; *p*). Δ =difference between postoperative and preoperative values; C2–C7=subaxial cervical lordosis; C0–C2=upper cervical lordosis; T1S=T1 slope; T1S–CL=T1 slope – cervical lordosis mismatch; Unfus. lord. = unfused lordosis. Numbers in bold indicate statistical significance (*p*<0.05)

Discussion

This study analysed cervical sagittal alignment in 290 patients undergoing multilevel ACDF for degenerative disease, via a novel automated image analysis algorithm. We hypothesized that surgical correction of cervical segmental alignment leads to measurable changes in cervical sagittal parameters, particularly in adjacent and unfused segments, reflecting preoperative compensatory mechanisms developed in response to degenerative segmental misalignment.

Before surgery, compensatory mechanisms can be observed, particularly at the upper cervical spine (C0–C2 lordosis) and at the unfused segments. After multilevel ACDF, these compensatory mechanisms were reduced, confirming our hypothesis.

We have three relevant findings: First, the significant negative correlation between the amount of surgical correction and the decrease in the unfused segment lordosis. This suggests that, once the segmental sagittal misalignment is corrected, adjacent segments no longer need to hyperextend to compensate, leading to a more balanced distribution of alignment across the unfused cervical spine and suggesting a normalization of biomechanical stress across different levels. Second, a similar trend can be observed in the correlation between amount of surgical correction and the decrease in C0–C2 lordosis, just missing statistical significance. Further supporting this, the strongest negative correlation was found between the total increase in C2–C7 lordosis and the decrease in C0–C2 lordosis. Changes at fused levels may have influence on upper cervical alignment, but restoring global subaxial cervical lordosis has a greater impact on reducing compensatory hyperlordosis in the upper cervical spine. This was further supported by

subgroup analyses, which confirmed that the biomechanical relationships observed in the total cohort were consistently maintained across different ACDF fusion areas. Notably, the strength of these associations varied by fusion level, suggesting that the site of surgical correction may influence how sagittal alignment is transmitted. In the C3–C5 group, a strong inverse correlation between Δ C2–C7 and Δ C0–C2 lordosis (*R* = - 0.65) suggests a more direct mechanical effect on the cranio-cervical junction. Conversely, in the C5–C7 group, a strong association between Δ surgical lordosis and Δ T1S–CL (*R* = - 0.58), coupled with a weak correlation to Δ C0–C2 (*R* = - 0.05), indicates that caudal fusions primarily act through restoration of global sagittal balance. Third, the significant positive correlation between the amount of surgical correction and the increase in T1S, suggesting that restoring segmental lordosis can reverse the compensatory reduction in T1S. Considering that T1S is strongly influenced by the alignment of the thoracic and lumbosacral spine [16] emphasizes the need for full-spine evaluation when planning multilevel ACDF¹⁷.

Although cervical spine alignment is an increasingly debated topic, few studies have investigated the effects of restoring segmental lordosis in ACDF procedures involving more than two levels on overall cervical alignment and compensatory mechanisms.

Zhang et al. [17] demonstrated that after ACDF, the C2–C7 lordosis and T1S increased, confirming that ACDF can contribute to restoring cervical sagittal alignment. Similarly, a recent case series study [11] of 117 patients undergoing two-level ACDF reported a surgical segmental correction (5.8°) consistent with our findings (6.5°), along with a reduction in unfused segment lordosis of approximately 3°, confirming the resolution of compensatory mechanisms. Furthermore,

the restoration of surgical lordosis led to an improvement in C2-C7 lordosis, with values comparable to our cohort.

As shown in previous studies [11, 12, 18–22], when segmental degeneration with loss of lordosis develops, increased biomechanical forces on the anterior vertebral bodies, accelerating disc degeneration and worsening compensatory hyperlordosis at non-degenerated levels [23, 24]. To maintain horizontal gaze, compensatory mechanisms trigger a cascade of alignment adjustments in the upper cervical and non-degenerated levels, reinforcing the spine's role as a dynamic unit. If left unaddressed, this cycle of progressive misalignment can generate additional mechanical stress, predisposing patients to long-term functional deterioration.

Thus, restoring both segmental and global cervical lordosis should be a priority in ACDF planning, as it contributes to reducing compensatory mechanisms and optimizing overall sagittal alignment.

We propose a three-step algorithm for analyzing cervical sagittal alignment: first, a preoperative assessment of cervical sagittal parameters; second, the identification of loss of lordosis or presence of kyphosis at the pathological level; and third, the evaluation of compensatory mechanisms in the upper cervical spine, the unfused subaxial segments, and, when available, the global spine.

This study does not come without limitations. First, its retrospective design, utilizing data from a single institution. To the best of the authors' knowledge, no larger studies currently exist; however, a multicenter database approach would allow the inclusion of a broader patient population. Nonetheless, the literature on this topic is consistent with our findings.

Another limitation of this study is the lack of full-spine x-ray, which prevented us from assessing global sagittal alignment in a comprehensive way. We were unable to measure key parameters such as PI, sacral slope (SS), and overall thoracic, lumbosacral, and global alignment. Had such imaging been available, the analysis would have undoubtedly benefited from a more complete biomechanical context. Nonetheless, the focus of our study was intentionally limited to the cervical spine. We considered cervical regional imaging sufficient to address the aims of this work, which were to describe compensatory patterns and alignment changes at the cervical level.

Moreover, we did not evaluate the influence of resolution of compensatory mechanisms on patient-reported outcomes. We believe that spinal cord decompression holds a greater importance than the mere resolution of radiological alignment parameters, as myelopathy improvement in ACDF patient is the key driver for optimal outcomes. However, as demonstrated by Passias et al. [20], while no direct relationship was found between cervical-specific sagittal outcomes

and patient-reported outcomes, C2-S1 SVA and C7-S1 SVA showed a significant correlation with overall 1-year outcomes. This underscores sagittal realignment importance, particularly in patients with axial pain.

Despite these limitations, to the best of authors' knowledge, this is the first retrospective high-volume series to specifically evaluate not only the impact of multilevel ACDF on cervical alignment but also on its compensatory mechanism, both in upper cervical spine and unfused segments. Moreover, the use of an automated image analysis algorithm, followed by revision by a spine surgeon, not only enhanced the precision of parameter measurements but also allowed us to perform these assessments in a straightforward and efficient manner. This approach was particularly valuable in cases where severe degeneration or surgical implants could compromise the confidence of spine surgeons or radiologists in obtaining accurate measurements.

As the population ages [25] and the number of procedures for cervical spondylosis increase, these findings are particularly relevant to avoid subsequent degeneration in unfused levels. That is why, spine surgeons should adopt a full cervical sagittal alignment assessment in evaluating patients for ACDF surgery, as individual variability necessitates tailored management.

Conclusions

This study highlights the necessity of considering cervical spine surgery as a global intervention rather than an isolated segmental correction. Restoring both segmental and global cervical lordosis should be a priority in ACDF planning, as it contributes to reducing compensatory mechanisms at the upper cervical spine, the unfused subaxial spine and the overall sagittal alignment. By recognizing and accounting for preoperative compensatory mechanisms, surgeons can optimize surgical planning to achieve a more stable and biomechanically favourable alignment.

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Declarations

Competing interests The authors declare no competing interests.

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