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Postoperative Sagittal Balance Has Only a Limited Role in the Development of Adjacent Segment Disease After Lumbar Spine Fusion for Degenerative Lumbar Spine Disorders: A Subanalysis of the 10-year Follow-up Study

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Study Design. Retrospective additional analysis of a prospective follow-up study.

Objectives. We aimed to find out whether poor postoperative sagittal alignment increases revisions for adjacent segment disease (ASD) after lumbar spine fusion (LSF) performed for degenerative lumbar spine disease.

Summary of Background Data. Revisions for ASD accumulate over time after LSF for degenerative lumbar spine disease. The etiology of ASD is considered multifactorial. Yet, the role of postoperative sagittal balance in this process remains controversial.

Materials and Methods. A total of 215 consecutive patients who had undergone an elective LSF surgery for spinal stenosis with (80%) or without (20%) spondylolisthesis were analyzed. Spinal reoperations were collected from the hospital records. Preoperative and postoperative sagittal alignment were evaluated from standing

radiographs. The risk of revisions for ASD was evaluated by Cox proportional hazards regression models.

Results. We did not find the poor postoperative balance [pelvic incidence–lumbar lordosis (LL) > 9°] to significantly increase the risk of revisions for ASD. crude hazard ratio (HR)=1.5 [95% confidence interval (CI): 0.8–2.7], adjusted (by age, sex, pelvic incidence, fusion length, and the level of the caudal end of fusion): HR=1.7 (95% CI: 0.9–3.3). We found higher LL outside the fusion segment (LL–segmental lordosis) to decrease the risk of revisions for ASD: HR=0.9 (95% CI: 0.9–1.0).

Conclusion. Poor sagittal balance has only a limited role as a risk factor for the revisions for ASD among patients with degenerative spinal disease. However, the risk for ASD might be the greatest among patients with reduced spinal mobility.

Key words: lumbar spine fusion, degenerative spinal disease, sagittal balance, revisions, adjacent segment disease, adjacent segment pathology

Level of Evidence: 3

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Lumbar spine fusion (LSF) surgery is a common procedure in the treatment of several spinal pathologies. Degenerative lumbar spine disorders (DLSDs) are the most common reason for LSF, while isthmic spondylolisthesis (IS) covers up to 20% of the cases.^{1,2} LSF surgeries occasionally become complicated by the need for repeat surgeries.^{3,4} Adjacent segment disease (ASD) is a major reason for late reoperations after LSF.⁵ By definition, ASD is a degenerative condition that postoperatively develops to the disk level next to the fusion segment and causes symptoms via instability or neural compression.⁶ ASD is the most frequent among the patients with DLSD where reoperations accumulate by time, on contrast to the patients with IS, who infrequently acquire this complication.^{4,7,8}

Etiology of ASD is thought to be multifactorial. Yet, the detailed pathogenesis remains not thoroughly clarified. On the one hand, LSF surgery may contribute to the pathogenesis by altering the adjacent level biomechanics. On the other hand, the ongoing degenerative process outside the fusion itself seems to have a significant role, as well.⁹ Several potential risk factors are linked to the progression of ASD, but their significance varies in the literature.^{5,10} Sagittal alignment after LSF is generally considered relevant here, so that failure to restore normal lordosis or loss of lordosis in LSF increases the risk of ASD.^{5,11} If the postoperative balance can be linked to the occurrence of ASD, this would also support the role of surgery in the pathogenesis of ASD.

In a 10-year prospective follow-up study of elective LSF surgeries performed in a single university center, we found revisions for ASD to accumulate over time among patients with DLSD while they were sporadic with IS. Here, we performed additional analysis among the DLSD patients to find out whether poor postoperative sagittal alignment increases the revisions for ASD in a 10-year follow-up after LSF.

MATERIALS AND METHODS

Patients

Between 2008 and 2012, all elective LSF patients in Tampere University Hospital were recruited into a prospective follow-up study. In Finland, a single public unit performs LSF surgeries and reoperations for a certain population. Hence, the study population represents a certain geographical catchment area. At the baseline, surgeons and study personnel filled in the demographic and surgical data, and the patients answered the following questionnaires: Oswestry Disability Index, Depression Scale, and a Visual Analog Scale for back and leg pain. All patients signed written consent, and the Tampere University Hospital Ethics Committee approved the study (R07108).

As ASD is mainly related to degenerative spinal disorders, we excluded patients with IS here. Our previous follow-up showed deformity patients to resemble DSLS patients demographically and in terms of revisions for ASD.⁴ However, given their condition which potentially requires more extensive surgery and individual judgement, we excluded patients with deformity here to facilitate answering to the present question. Hence, our exclusion criteria were: (1) fusion reaching the thoracic spine, (2) former spine surgery, (3) IS, (4) deformity, (5) fracture, or (6) tumor. Our whole study population suffered from degenerative lumbar spine pathology with related neural compression, that is, spinal stenosis with (80%) or without (20%) spondylolisthesis. Fusion was implemented to address the spondylolisthesis or to facilitate foraminal decompression. All surgeries were instrumented posterolateral fusions from midline incision with or without interbody fusion (transforaminal lumbar interbody fusion/posterior lumbar interbody fusion) combined with necessary decompression.

We investigated all spinal reoperations from the patient records. Death or reoperation for ASD ended the follow-up of a single patient—otherwise, the follow-up continued to June of 2020.

Spinopelvic Parameters

Lumbar lordosis (LL), pelvic incidence (PI), sacral slope, pelvic tilt, and segmental lordosis (SL) of the fusion segment were determined from sagittal standing lumbar spine radiographs before and 3 months after surgery. The preoperative standing radiograph was missing from 7 patients—they were excluded from the analysis. Figure 1 shows the definitions of these parameters. PI is regarded a constant value determined by individual pelvic anatomy. We determined LL as an angle between the upper endplates of L1 and S1 vertebrae. Schwab *et al*¹² postulated a formula $LL = PI \pm 9^\circ$ in the normal population. According to that, the patient can be considered hypolordotic in spine surgery settings with $PI - LL > 9^\circ$. The optimal target lordosis in LSF, however, decreases with the patient's age.^{13,14} A single threshold was chosen for statistical analysis. Further, analyses were performed separately to the patients under and over 65 years to avoid the potential effect of the difference between the age-appropriate threshold and the fixed cutoff of 9° . Sacral slope describes the pelvic alignment, and pelvic tilt indicates the amount of pelvic retroversion which is needed to maintain a standing posture. After LSF, $LL - SL$ represents the mobile segment of the lumbar spine.

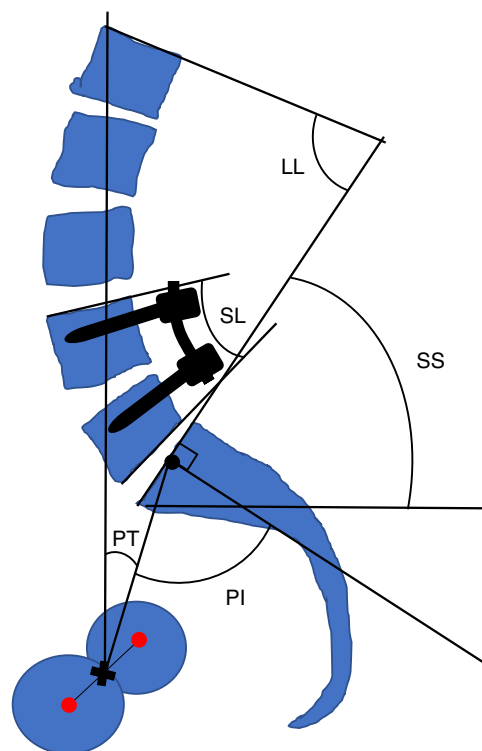


Figure 1. Lumbar spinopelvic parameters: lumbar lordosis (LL), pelvic incidence (PI), sacral slope (SS), pelvic tilt (PT), and segmental lordosis (SL) of the fusion segment. Values are presented in degrees. [full color online](#)

Statistics

The descriptive statistics are presented as means with SD, as medians with interquartile range or as counts with percentages. Cox proportional hazards regression models were used to estimate the adjusted hazard ratios (HRs) and their 95% confidence intervals (CIs). Age, sex, fusion length, and the level of the caudal end of fusion were used as covariates in these models. The possible nonlinear relationship between LL and SL and the risk of revision for ASD was modeled using restricted cubic splines with 4 knots at the fifth, 35th, 65th, and 95th percentiles. Spline functions were estimated using multivariable Cox proportional hazard regression models, including age, sex, fusion length, and the level of the caudal end of fusion as a covariate. All analyses were performed using STATA software, version 16.1 (StataCorp LP, College Station, TX).

RESULTS

A total of 215 patients (mean age: 66 yr, SD: 10 yr) met the inclusion criteria. Most of them were women (76%) who most commonly underwent two-segment fusion in the lower lumbar spine (Table 1).

During the follow-up with a median of 9.2 years, 43 (20%) patients underwent a revision for ASD.

The spinopelvic parameters of the patients were equal preoperatively and postoperatively (Table 2). By mean, the difference PI–LL ranged in normal lordosis before and after surgery. However, 83 (39%) patients were hypolordotic after surgery according to the mismatch of PI–LL > 9°.

The postoperative imbalance (PI–LL > 9°) did not result in a significantly increased risk of revision for ASD according to the Cox multivariate model. The crude HR of 1.5 (95% CI: 0.8–2.7) and adjusted (by age, sex, PI, fusion length, and the level of the caudal end of fusion) HR of 1.7 (95% CI: 0.9–3.3) remained statistically insignificant. HR was the same, insignificant, if patients under and over 65 years were analyzed separately.

Postoperative segmental hypolordosis might lead to hyperlordosis outside the fusion segment (LL–SL) as a compensatory mechanism. Nevertheless, we found higher LL–SL to result in less revisions for ASD: HR = 0.9 (95% CI: 0.9–1.0). The effect of continuous difference LL–SL on revisions for ASD is shown in Figure 2 reinforced this finding.

DISCUSSION

Among patients who underwent LSF surgery for DLSD, we did not find postoperative hypolordosis (by PI–LL > 9°) to result in a significant increase of the risk for revision for ASD during a 10-year follow-up. However, mismatch of 9° does not always represent a clinical threshold for satisfactory and poor alignment. Older age groups reportedly tolerate lower lordosis and greater mismatch than younger patients.^{13,14} Nevertheless, one fixed cutoff was used to differentiate good and poor alignment in statistical analysis.

As previously indicated, revisions for ASD are infrequent after LSF for IS.⁴ Contrary to that, they accumulate almost linearly over time among patients that have undergone LSF for

TABLE 1. The Baseline Demographic Data, Self-reported (*) Symptoms and Comorbidities, and the Type of Primary Surgery

	N = 215
Women [n (%)]	164 (76)
Age [mean (SD)]	66 (10)
BMI [mean (SD)]	28.6 (4.4)
Smoking* [n (%)]	12 (6)
Education years [mean (SD)]	11.1 (3.9)
Physical activity* [mean (SD)] (h/wk)	4 (2, 9)
Duration of the spinal problem* [median (IQR)] (y)	9 (4, 20)
Back pain* VAS [mean (SD)]	61 (26)
Leg pain* VAS [mean (SD)]	68 (23)
ODI* [mean (SD)]	45 (15)
DEPS* [mean (SD)]	10.5 (6.1)
Comorbidity* [n (%)]	
Cardiovascular	118 (60)
Diabetes	24 (12)
Psychiatric disorder	5 (3)
Pulmonary	11 (6)
Neurological	5 (3)
Rheumatoid	14 (7)
Indication for surgery	
Spinal stenosis with spondylolisthesis [n (%)]	172 (80)
Spinal stenosis without spondylolisthesis [n (%)]	43 (20)
Fusion	
Level of the lower end [n (%)]	
L3 or L4	9 (4)
L5 or L6	114 (53)
S1	92 (43)
Length, levels [n (%)]	
1	59 (27)
2	84 (39)
3	54 (25)
4	17 (8)
5	1 (0)
Interbody cage (TLIF/PLIF) [n (%)]	23 (11)

BMI indicates body mass index; DEPS, Depression Scale; IQR, interquartile range; ODI, Oswestry Disability Index; PLIF, posterior lumbar interbody fusion; TLIF, transforaminal lumbar interbody fusion; VAS, Visual Analog Scale.

TABLE 2. The Spinopelvic Parameters (°) Before and After Lumbar Spine Fusion Surgery

	Mean (SD)	
	Preoperative	Postoperative
LL	50 (13)	49 (12)
PI	56 (10)	—
PI–LL	6.7 (11.1)	6.7 (11.1)
PT	20 (8)	21 (7)
SS	37 (9)	36 (8)
SL	29 (14)	27 (12)
LL–SL	21 (14)	22 (13)

LL indicates lumbar lordosis; PI, pelvic incidence; PT, pelvic tilt; SL, segmental lordosis; SS, sacral slope.

DLSA. This phenomenon highlights the role of the ongoing degenerative process in the spine in the development of ASD.

Generally, the effect of postoperative sagittal alignment on clinical outcome is established, but its role in the prevention of ASD is more unclear.^{15,16} In the literature, the case-control study of Djurasovic and colleagues is often referred to as a proof of an association between postoperative hypolordosis and the increased revisions for ASD.^{5,11,17} In that study, the mean interval between the

initial surgery and the revision was 58 months, while the mean follow-up period for controls was only 55 months, which we consider relatively short. As revisions accumulate over time, and secondly, patients may die during the follow-up, we consider the Kaplan-Meier method an appropriate way to assess this phenomenon.

Kim *et al*¹⁸ retrospectively analyzed 69 patients who underwent L4–L5 fusion for IS or degenerative spondylolisthesis. They concluded that maintaining a segmental lordosis of 20° or more was important in the prevention of ASD. Bae *et al*,¹⁹ in their retrospective analysis, suggested that restoration of segmental lordosis is important in the prevention of ASD. Nevertheless, they found only a statistically insignificant difference of 3° between ASD and non-ASD groups. In a prospective 5-year follow-up after LSF, Anandjiwala *et al*²⁰ found preexisting adjacent segment degeneration, not postoperative balance, to be a risk factor for radiological ASD. Furthermore, they found no correlation between the clinical outcome and radiological ASD. In a retrospective 10-year follow-up of posterior lumbar interbody fusion surgeries, Nakashima *et al*²¹ found high PI, not LL, a significant risk factor for early-onset ASD. In a retrospective analysis of Alentado *et al*,¹⁰ SL and LL were not significant risk factors for ASD.

Despite a relatively large study population and a long follow-up, we did not find a statistically significant effect of poor postoperative balance on the rate of revisions for ASD. Hence, we postulate that alignment plays a less significant role in the multifactorial pathogenesis of ASD than commonly proposed. We consider the ongoing degenerative spinal disease the most important single factor in this entity.

Poor segmental alignment requires compensatory mechanisms from the patient to maintain global balance. Hyperlordosis in the mobile segment of the lumbar spine, usually above the fused segment, is one of the compensatory mechanisms after LSF.²² Thus, we expected higher LL–SL to relate to increased revisions for ASD caused by the increased stress at the adjacent segments. However, the connection was the opposite. This may indicate that the patients with mobile spine present more capacity to compensate and thus less stress to the adjacent segments. Moreover, Figure 2 indicated a strong effect from the change in LL–SL on the revisions for ASD. Our data provide no definitive answer whether this, in fact, more reflects the individual alignment or mobility in the mobile segment. It is also possible that some of the patients had an unfulfilled need for compensation before and after surgery due to a stiff spine. Earlier, diffuse idiopathic skeletal hyperostosis, a condition resulting in severely restricted spinal mobility, is reported as a significant risk factor for ASD after short segment LSF.²³ We assume that the benefit of reasonable segmental lordosis in the prevention of ASD might be the most important with reduced spinal mobility.

During the data collecting period, use of interbody cage was less common than nowadays. The main indication for interbody cage then was foraminal decompression or strengthening the fusion to prevent instrumentation failures.

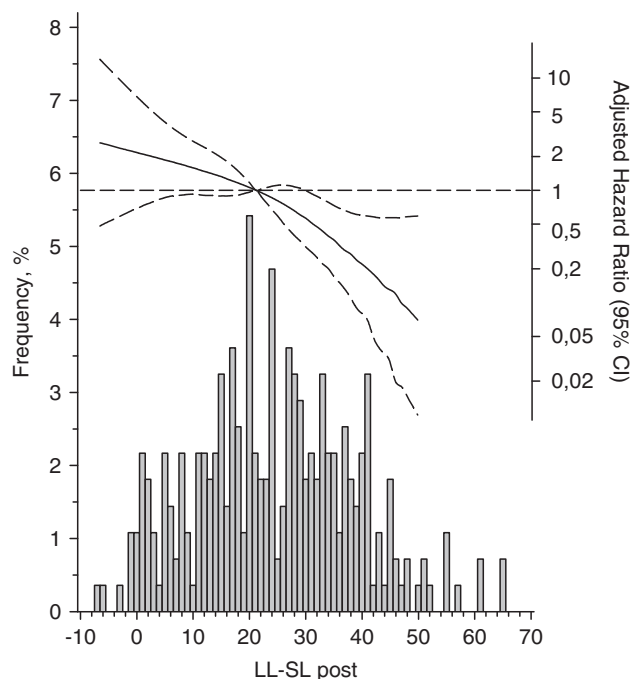


Figure 2. Higher lordosis in the mobile segment of the lumbar spine (LL–SL) after lumbar spine fusion was linked to decreased revisions for adjacent segment disease. Reference level (hazard ratio = 1) of LL–SL was here set to 21°. CI indicates confidence interval; LL, lumbar lordosis; SL, segmental lordosis.

The use of transforaminal lumbar interbody fusion in the correction of sagittal alignment has increased thereafter. Therefore, we did not assess the role of the interbody cage in the prevention of ASD here.

Although the connection between postoperative sagittal alignment and the occurrence of ASD seems less straightforward as occasionally proposed, the pursuit of normal alignment is important, especially for the clinical outcome. In this study, we have not investigated how postoperative sagittal balance affects the functionality or the health-related quality of life. Moreover, ending up in kyphosis during LSF surgery usually hampers future revision surgeries, where restoring normal balance may require considerably heavier surgery. All this might have the greatest impact with limited spinal mobility.

This study does not prove that sagittal alignment has no effect on the development of ASD. However, our results reinforce the perception from the literature that sagittal alignment has only a limited effect on the progression of ASD.

CONCLUSION

Poor sagittal alignment (mismatch PI-LL >9°) did not significantly increase revisions for ASD in a 10-year follow-up of the patients who underwent LSF for DLSD. Achieving appropriate segmental lordosis in LSF might be the most important in patients with reduced spinal mobility.

➤ Key Points

- ❑ We performed a retrospective additional analysis to evaluate the effect of sagittal alignment on the risk of revisions for adjacent segment disease after LSFs.
- ❑ The study population had been prospectively followed up for 10 years after having undergone LSF for a degenerative spinal disorder (stenosis with or without spondylolisthesis).
- ❑ We did not find poor postoperative balance to significantly increase the risk of revisions for ASD.

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