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CASE SERIES

Lordosis loss in degenerative spinal conditions

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Abstract

Purpose To establish whether common degenerative lumbar spine conditions have a predictable sagittal profile and associated range of lordosis.

Summary of background data The spinopelvic balance of a normal population and normal ranges are well described in the literature. There is also evidence that certain degenerative conditions can lead to a preponderance of loss of lordosis at specific spinal levels. There is limited literature on the range and magnitude of loss of lordosis for known degenerative lumbar spine pathologies.

Methods A retrospective analysis of *prospectively obtained radiographs* from a dual surgeon database was performed and imaging analysed for spinopelvic parameters. Degenerative conditions studied were; Lumbar degenerative spondylolisthesis (L3/4 and L4/5 analysed separately), L5/S1 degenerative disc disease, L5/S1 isthmic spondylolisthesis. Pelvic incidence, sacral slope, pelvic tilt, segmental and global lumbar lordosis, vertebral lordosis and lumbar vertical axis were measured.

Results The range of change in segmental lordosis was normally distributed for all studied degenerative spinal conditions except L5/S1 isthmic spondylolisthesis. L5/S1 degenerative disc disease affected younger adults (mean age 37), whilst degenerative spondylolisthesis at L3/4 and L4/5 affected older adults (mean ages 69.5 and 68.9 respectively). Removing an outlying high-grade L5/S1 isthmic spondylolisthesis made the data distribution approach a normal distribution.

Conclusion Most degenerative spinal pathologies cause a normally distributed spectrum of deformity which should be addressed and corrected with a tailored, individualised surgical plan for each patient. Universal treatment recommendations should be interpreted with caution.

Level of evidence 4.

Keywords Lumbar spine · Spinal alignment · Lordosis · Degeneration · Spondylolisthesis · Degenerative spinal conditions · Sagittal balance · Spinopelvic parameters

Introduction

With increased recognition that variability in pelvic shape as measured by pelvic incidence (PI) is associated with variation in sacral slope and the consequent lumbar lordosis (LL) required to maintain sagittal alignment [1, 2], there is increased recognition that spinal reconstruction with fusion needs to optimise sagittal realignment so as to optimise clinical outcomes and reduce adjacent segment degeneration [3], with its potential to cause late failure and the need for revision surgery.

Whilst LL is an acceptable global measure of lumbar sagittal alignment, it consists of five mobile articulations each of which contributes to the overall alignment. The individual contributions of each lumbar segment have been characterised [4–8]. Failure to recreate normal segmental alignment within a fusion requires compensation at adjacent segments with altered biomechanics that may lead to accelerated degeneration, but which may not be detected with an overall measure of multi-segmental lordosis—the LL [5].

Common pathologies that lead to low lumbar fusion procedures are associated with variable disc degeneration. This degeneration is associated with disc desiccation, loss of disc

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height, facet joint degeneration, annular bulging and loss of lordosis particularly in the lower lumbar spine where the motion segments are the major contributors to lordosis [9].

There is increasing evidence that different reconstruction options for lumbar fusion using a variety of interbody techniques result in variable lordosis gain [10–15]. It is becoming increasingly evident that if pathology occurring in the low lumbar spine requires treatment with fusion, treatment should attempt to optimise lordosis recreation to match the normal lordotic contribution of the treated segment within the overall lumbar lordosis [6].

If there is a goal to reconstruct the optimum lordosis at a pathological segment, there needs to be an understanding of what the normal lordosis at the operated segment should be, and the deformity created by the pathological process for which the surgery is indicated. The difference between the two will guide the magnitude of lordosis gain necessary in the reconstruction. To the best of our knowledge there is no definition of the deformity, the loss of normal sagittal alignment, associated with common pathologies of the low lumbar spine that may require intervention.

The purpose of this study is to document the sagittal deformity (loss of lordosis) associated with common lower lumbar disorders for which fusion surgery is indicated. We wished to specifically examine degenerative spondylolisthesis (DS) at both L3/4 and L4/5, degenerative disc disease (DDD) at L5/S1 (disc degeneration and loss of height without listhesis or due to degenerative anatomical change to the posterior elements), and isthmic spondylolisthesis (IS) at L5/S1, looking at the sagittal alignment of the relative motion segment in a population of patients coming forward for reconstructive surgery.

Methods

Patient cohort

A surgical database was interrogated and data of patients who had undergone single-level fusion for L3/4 DS, L4/5 DS, L5/S1 DDD and L5/S1 IS were retrieved. Data retrieved included: patient demographics, surgical procedure and age.

Radiology

Erect lateral radiograph of the lumbar spine including the hip joints had been performed on all patients prospectively prior to surgery for the purposes of surgical planning.

The radiological measurements were; pelvic incidence (PI), sacral slope (SS), pelvic tilt (PT), lumbar lordosis (LL), segmental lordosis at each lumbar intervertebral disc (SL) (measured from caudal endplate of superior vertebra to cranial endplate of inferior vertebra, i.e. bottom of L4 to top of L5 for the L4/5 disc), vertebral lordosis at each vertebra (VL), and lumbar vertical axis (LVA—the distance from the posterior edge of the S1 superior endplate to the plumb line from the centre of the L1 vertebral body). Kyphosis was recorded as a negative value.

Exclusion criteria included; inadequate radiographs, abnormal vertebral differentiation, e.g. sacralisation of lumbar vertebra or lumbarisation of sacral vertebra and previous fusion or dual pathologies (e.g. L5/S1 DDD and L4/5 DS).

Measurements were performed on Inteleviewer Picture Archiving and Communication System (PACS) (Intelerad, 800 Boulevard De Maisonneuve East, Montreal, Canada). Measurement accuracy was ensured by a single observer using the software on the same PACS display system. Blinded repeat measurements were made for ten random cases and the accuracy was assessed. The sum of the vertebral lordosis and segmental lordoses was equivalent to the lumbar lordosis, and the difference between these components and the total was also assessed for accuracy.

Statistical analysis was performed with *t* test using GraphPad (GraphPad Software, 2365 Northside Dr. Suite 560, San Diego, CA 92108). A p value of < 0.05 was taken to be statistically significant.

Results

165 surgical cases were identified, and after exclusion of cases without adequate radiographs 89 patients remained: L3/4 DS—9; L4/5 DS—49; L5/S1 DDD—19; and L5/S1 IS—12 (see Fig. 1).

The mean and range of ages of the groups were L3/4DS—69.555.0–78.6); L4/5DS—68.9 (56.3–86.9); L5/S1DDD—37 (25.5–45.8); and L5/S1IS—55 (26.3–96.5). There was a statistically significant

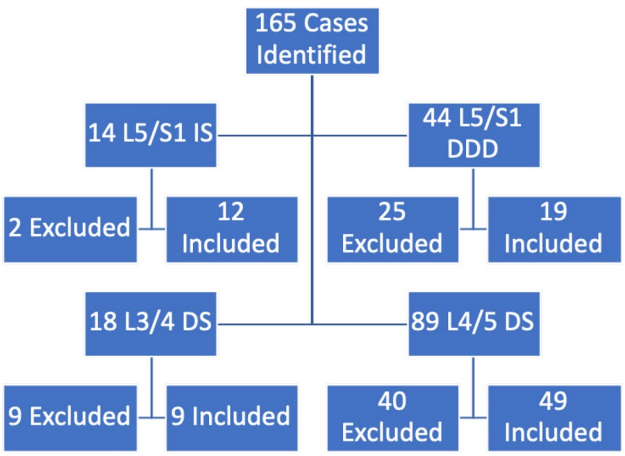

Fig. 1 Caseload and excluded cases. *IS* isthmic spondylolisthesis, *DDD* degenerative disc disease, *DS* degenerative spondylolisthesis

Table 1 Summary of findings for L3/4 degenerative spondylolisthesis

Mean age (years)	69.5 (56–78)
Male	5
Female	4
LL	51.8 (12–67)
PT	23.6 (14–34)
PI	59.7 (43–79)
SS	36.2 (17–45)
LVA (mm)	37.2 (1–90)
L3/4 SL	5.2 (0–11)
L4/5 SL	8.4 (3–13)
L5/S1 SL	6.0 (0–18)
PI–LL	7.9 (1–34)

All measurements are mean and are measured in degrees unless stated with ranges in brackets

Table 2 Summary of findings for L4/5 degenerative spondylolisthesis

Mean age (years)	68.9 (56–87)
Male	17
Female	32
LL	50.6 (15 to 77)
PT	25.2 (9 to 41)
PI	62.1 (37 to 83)
SS	36.9 (15 to 54)
LVA (mm)	28.9 (−27 to 106)
L3/4 SL	9.3 (1 to 19)
L4/5 SL	4.1 (− 12 to 16)
L5/S1 SL	7.7 (0 to 26)
PI–LL	11.5 (− 12 to 38)

All measurements are mean and are measured in degrees unless stated with ranges in brackets

difference between IS and DDD $p < 0.05$, IS and 3/4DS $p < 0.05$, IS and 4/5DS $p < 0.05$, L5/S1DDD and 3/4DS $p < 0.05$, 5/1DDD and 4/5DS $p < 0.05$, but not between 3/4DS and 4/5DS $p = 0.43$.

The mean PIs of the groups were L3/4DS—59.7 deg; L4/5DS—62.1 deg; L5/S1DDD—49 deg; and L5/S1IS—66.2 deg. The PI was significantly lower for L5/S1DDD compared with the other three diagnoses (vs L3/4DS $p < 0.05$, vs L4/5DS $p < 0.05$, vs L5/S1 IS $p < 0.05$), but there was no difference of the PI between L3/4DS, L4/5DS and L5/S1IS (L3/4DS vs L4/5 DS p = 0.27, L3/4DS vs L5/S1IS $p = 0.15$, L4/5DS vs L5/S1IS $p = 0.14$).

The mean and range of segmental lordosis at each operated level prior to surgery were L3/4DS 5.2 deg (0–11); L4/5DS 4.1 deg (− 12 to 16); L5/S1DDD 7.9 deg (0–17); and L5/S1IS − 1.8 deg (− 40 to 14).

The mean and range of LL-PI for each condition was L3/4DS—7.9 deg (− 34 to − 1); L4/5DS—11.1 deg (− 38 to 8); L5/S1DDD— − 4.8 deg (− 4 to 22); and L5/S1IS—6.8 deg (− 25 to 12).

Mean PT was > 20 deg in L3/4DS, L4/5 DS, and L5/S1IS, but not in L5/S1DDD. This information is demonstrated in Tables 1, 2, 3, and 4.

The range of SL for the operated level in each condition is shown in histogram form in Figs. 2, 3, 4, and 5. Included is a reference line for the normal segmental lordosis for that anatomical level as derived from literature norms.

L4/5 degenerative spondylolisthesis

The Shapiro–Wilk test for normality of L4/5 DS was $p = 0.12$, revealing the data to be normally distributed.

$W = 0.96$ (0.95–1.00).

Table 3 Summary of findings for L5/S1 degenerative disc disease

Mean age (years)	37 (25–26)
Male	13
Female	6
LL	53.8 (41–68)
PT	12.7 (2–25)
PI	49 (32–66)
SS	36.3 (21–53)
LVA (mm)	5.2 (− 33 to 4)
L3/4 SL	8.6 (2–13)
L4/5 SL	12.7 (9–18)
L5/S1 SL	7.9 (0–17)
PI–LL	− 4.8 (− 22 to 6)

All measurements are mean and are measured in degrees unless stated with ranges in brackets

Table 4 Summary of findings for L5/S1 isthmic spondylolisthesis

Mean age	55.2 (26–96)
Male	6
Female	6
LL	59.4 (37–78)
PT	21.2 (10–37)
PI	66.2 (47–94)
SS	44.8 (32–59)
LVA (mm)	21.2 (− 8 to 48)
L3/4 SL	9.3 (3–12)
L4/5 SL	12.3 (2–18)
L5/S1 SL	− 1.8 (− 40 to 14)
PI-–LL	6.8 (− 12 to 25)

All measurements are mean and are measured in degrees unless stated with ranges in brackets

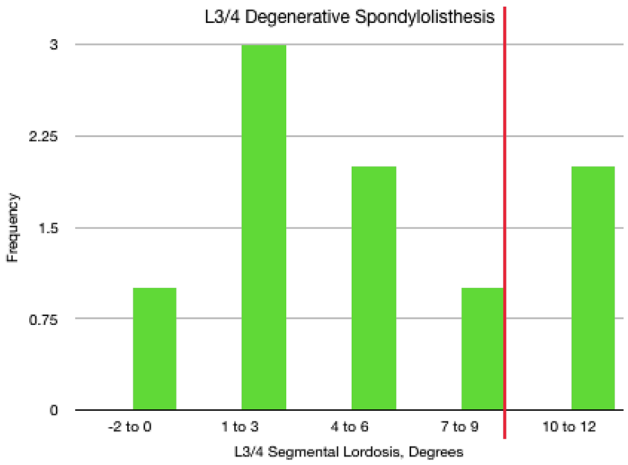

Fig. 2 Histogram of lordosis ranges for L3/4 degenerative spondylolisthesis. Expected segmental lordosis (SL) is represented by the red line (9°)

L3/4 degenerative spondylolisthesis

The Shapiro–Wilk test for normality of L3/4 DS was $p = 0.27$, revealing the data to be normally distributed.

$W = 0.90$ (0.83–1.00).

L5/S1 degenerative disc disease

The Shapiro–Wilk test for normality of L5/S1 DDD was $p = 0.13$, revealing the data to be normally distributed.

$W = 0.92$ (0.90–1.00).

L5/S1 isthmic spondylolisthesis

The Shapiro–Wilk test for normality of L5/S1 IS was $p = 0.005$, revealing the data to NOT be normally distributed.

$W = 0.78$ (0.86–1.00).

Removing the single outlier for significant kyphosis (Grade III isthmic spondylolisthesis with a segmental lordosis at L5/S1 of − 40°) does not alter this outcome although the W value does approach a normal value. $p = 0.03$, $W = 0.84$ (0.85–1.00).

There was no statistically significant difference between the SL at L4/5 between the L4/5 DS and L3/4 DS patients $p = 0.0546$.

The difference in SL at L5/S1 was significant between DDD and L5/S1 IS, $p = 0.0084$.

Measurement accuracy was assessed. A single observer performed the measurements on a single PACS compliant Computer Display. Measurements were repeated for ten cases at a separate sitting to confirm reliability. All repeat measurements varied by less than 1%. Summation of all of the VL and SLs provided a combined total equal to LL. This allowed accuracy assessment within each case as LL was also measured separately. All measurements of sum VL and SL were within 1 degree of the LL for each case.

Discussion

This study has demonstrated that loss of normal sagittal alignment at the abnormal level can be very significant, but is highly variable in patients coming forward for single-level fusion surgery. The normative values for the lumbar disc

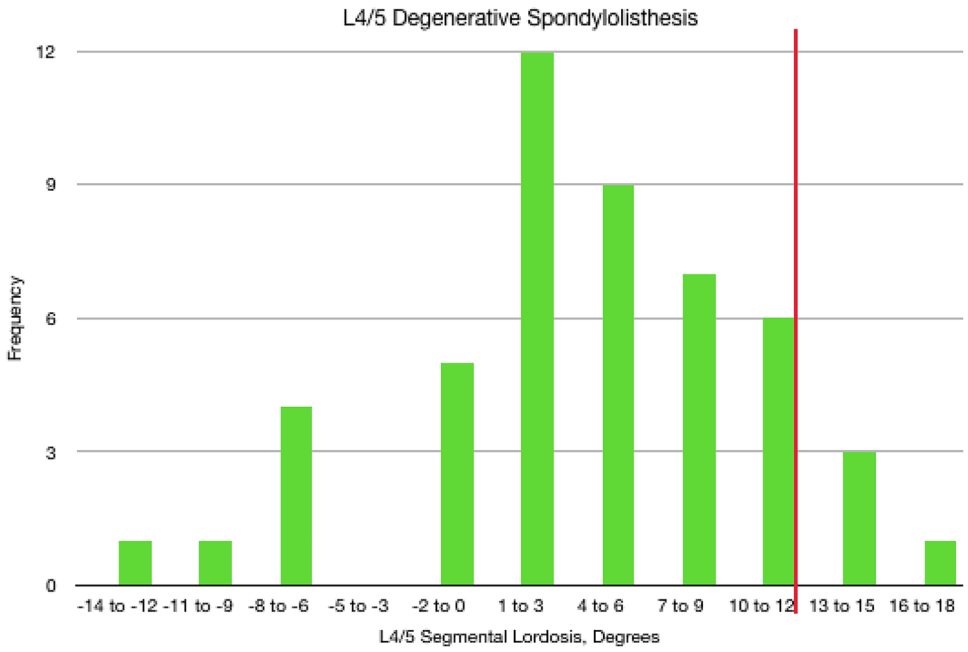

Fig. 3 Histogram of lordosis ranges for L4/5 degenerative spondylolisthesis. Expected segmental lordosis (SL) is represented by the red line (12°)

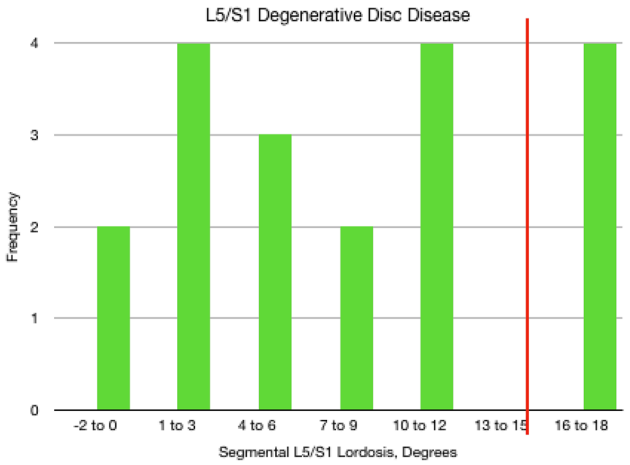

Fig. 4 Histogram of lordosis ranges for L5/S1 degenerative disc disease. Expected segmental lordosis (SL) is represented by the red line (15°)

contribution to lordosis are well established [4]. The maximum contribution to lordosis from the discs comes from L5/S1 and L4/5 levels where the mean lordotic angle of the disc is 15° and 12°, respectively [4]. Commonly treated pathologies at these levels, where disc height has been dramatically reduced as is often seen, will require operative procedures capable of dramatically improving lordosis of the segment if alignment is to be optimised and biomechanical forces at adjacent segments minimised.

The clinical outcomes of patients treated are not included within this paper as this was not the purpose of the paper. It is accepted that restoration of the spinopelvic balance decreases the risks of adjacent segment disease and clinical outcomes as demonstrated by Radovanovic amongst others [45].

From the study population three relatively common pathologies for which fusion is a common component of surgical treatment were selected, with degenerative spondylolisthesis being subdivided into cases at L3/4 and L4/5. This separation of the DS group was performed because of the recognised difference in lordosis in disc at the differing levels in the normal lumbar spine. The differences in the ages at the time of presentation for surgery represented the well-recognised differences in the pathologies. DS is symptomatic later in life, whereas IS is symptomatic across adult life [16, 17]. DDD included painful motion segments, and back dominant pain after disc prolapse treated with or without surgery, and this occurs in a younger demographic [18]. This undoubtedly influences the ability of the other motion segments of the spine to 'compensate' for deformity as demonstrated by the reduction in lordosis across uninstrumented motion segments [43] whilst the more aged spine has less flexibility and therefore less ability to compensate for the acquired deformity for example in DS patients.

The difference in the mean PI values is also consistent with the hypothesised influence of the PI and SS on lumbar spine disorders: IS and DS result in translation of the cephalad vertebra due to increased shear forces upon the posterior elements related to increased vertebral slope as seen in the lower lumbar spine where there is greater SS [19], whilst DDD is associated with more horizontal disc alignment due to a lower PI and SS, and this may result in greater loads through the anterior column of the spine, where the disc may be more susceptible to failure under axial loading [20].

The published literature suggests a wide range of lordotic gain associated with various surgical techniques used to perform fusion [12, 21–36]. Fusion in situ, with or without rigid instrumentation, is unlikely to significantly increase lordosis. At the other extreme experience suggests that the maximum gain of lordosis occurs where operative intervention involves wide posterior column osteotomy and facetectomy, and this can be combined with excision of the anterior longitudinal ligament with ALIF to maximise lordosis gain [37]. In

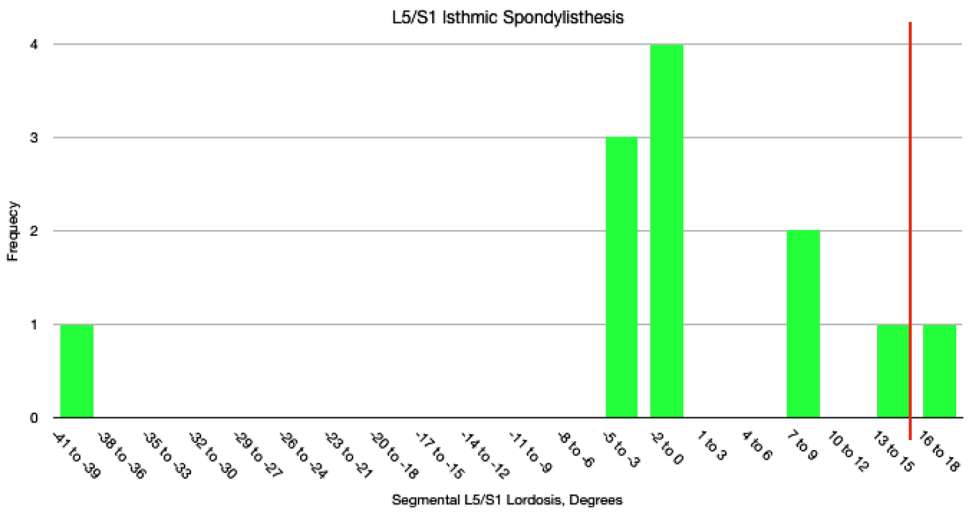

Fig. 5 Histogram of lordosis ranges for L5/S1 isthmic spondylolisthesis. Expected segmental lordosis (SL) is represented by the red line (15°)

general, minimal bone resection as seen in minimally invasive procedures reduces lordosis gain [38–40] with conflicting evidence as to which technique produces the most lordosis, including minimally invasive surgery and expandable cages [38, 41, 42]. It is clear that the wide range of operative options resulting in variable increases in SL [12, 21] could ideally be matched with the wide range of reductions in SL demonstrated in the study across each of the pathologies. It is highly likely that a single technique will not be suitable for the range of SL seen across any single pathology coming to fusion surgery. Schwab's classification of osteotomies [37] provides a useful escalation of posterior-based osteotomies, whilst transforaminal, anterior only, posterior only or combined anterior–posterior techniques provide increasing lordotic gains whilst respecting the surgeon's preferred, individual technique.

This study again questions the relevance of using the global measure of lordosis (LL) when looking at pathologies with potential for focused alignment abnormalities within the multi-articulated lumbar spine. The measure of mismatch between PI and LL, PI–LL < 10°, was not exceeded in L3/4DS, L5/S1IS or L5/S1DDD, and only marginally exceeded for L4/5 DS. This is not surprising. In younger spines, compensation occurs at levels adjacent to the pathology with the frequent appearance of compensatory hyperlordosis above a hypolordotic segment [43] and the global LL remains normal. The modest mismatch between PI and LL seen in the L4/5 DS group likely represents increasing stiffness with increasing spondylosis and lesser ability to compensate for regional abnormalities in this older population [44]. What is also evident from the data is the wide spectrum of all measurements of alignment, including PT and sagittal balance (as measured by LVA), which must be considered on an individual basis when addressing these segmental deformity corrections and the array of surgical treatment options and their respective lordosis gains should be considered when assessing individual patients and their segmental deformities.

Whilst it is tempting to recommend an optimum case by case increase in SL based on the SL associated with the pathology before surgery, it must be recognised that the normative anatomical data is based on population means. Given that the 'shearing' pathologies of IS and DS are associated with a higher PI and SS, it may well be that the disc contribution to the greater LL needed to match the PI in these disorders is greater than the published means. Whilst there is some evidence that more lordotic lumbar spines are associated with greater lordotic contributions from the upper lumbar spine [6], it seems likely that such spines also have a greater contribution to the LL by the SL in the lower discs at L4/5 and L5/S1. To the best of our knowledge there is no normative data for the disc contribution to the LL for the extremes of PI, yet it is the extremes of PI (low for DDD and high for DS and IS) which are commonly associated with these common pathologies requiring intervention.

This paper is the first that attempts to characterise the focal sagittal deformities associated with common lumbar pathologies requiring fusion as part of the treatment. It clearly demonstrates that the wide range of deformities means that a 'typical' loss of SL cannot be described for a specific pathology, and therefore attempts to both characterise the deformity and prescribe universal treatment recommendations are flawed. Individualised planning is required dependent upon the individual SL if reconstruction aims to optimise lordosis at the fused segment.

Author contributions WKMK, AD, AF, PAR: made substantial contributions to the conception or design of the work; or the acquisition, analysis, or interpretation of data; or the creation of new software used in the work; drafted the work or revised it critically for important intellectual content; approved the version to be published; and agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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Declarations

Conflict of interest There are no conflict of interest disclosures relevant to this work.

Ethics committee The work was reviewed by the Health and Disabilities Ethics Committees (HDEC) and granted exemption.

Copyright There is no copyrighted material or patient identifiable data/consent forms within this work.

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