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Lumbar Spine Degeneration and Flatback Deformity Alter Sitting-Standing Spinopelvic Mechanics—Implications for Total Hip Arthroplasty

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ABSTRACT

Background: Spinal degeneration and lumbar flatback deformity can decrease recruitment of protective posterior pelvic tilt when sitting, leading to anterior impingement and increased instability. We aim at analyzing regional and global spinal alignment between sitting and standing to better understand the implications of spinal degeneration and flatback deformity for hip arthroplasty.
Methods: Spinopelvic parameters of patients with full-body sitting-standing stereoradiographs were assessed: lumbar lordosis (LL), spinopelvic tilt (SPT), pelvic incidence minus LL (PI-LL), sagittal vertical axis (SVA), and T1 pelvic angle (TPA). Lumbar spines were classified as normal, degenerative (disc height loss >50%, facet arthropathy, or spondylolisthesis), or flatback (degenerative criteria and PI-LL >10°). Independent *t*-tests and analysis of variance were used to analyze alignment differences between groups.
Results: After propensity matching for age, sex, and hip osteoarthritis grade, 57 patients per group were included (62 ± 11 years, 58% female). Mean standing and sitting SPT, PI-LL, SVA, and TPA increased along the spectrum of disease severity. Increasing severity of disease was associated with decreasing standing and sitting LL. The flatback group demonstrated the greatest sitting SPT, PI-LL, SVA, and TPA. The amount of sitting-to-standing change in SPT, LL, PI-LL, SVA, and TPA decreased along the spectrum of disease severity.
Conclusion: Spinal degeneration and lumbar flatback deformity both significantly decrease lower lumbar spine mobility and posterior SPT from standing to sitting in a stepwise fashion. The demonstrated hypomobility in flatback patients likely serves as a pathomechanism for the previously observed increased risk of dislocation in total hip arthroplasty.

Patients with adult spinal deformity (ASD), characterized by progressive, degenerative changes in the spine leading to sagittal misalignment and postural imbalance, adopt (posterior) pelvic tilt as a compensatory mechanism to maintain upright posture [1]. Restoring sagittal plane alignment is of critical importance in the treatment of ASD. As a result, changes in spinal sagittal alignment, and spinal realignment surgery, cause changes in acetabular orientation; however, the effect of ASD on sitting alignment has remained unpublished. In ASD literature, a thorough understanding of the interrelationship of the hip and spine is crucial in the evaluation and operative planning for patients undergoing total hip arthroplasty (THA).

Lewinnek et al [2] attempted to account for pelvic orientation using the anterior pelvic plane for the assessment of optimal lateral inclination and anteversion of acetabular components. More recently, a number of studies have reported a wide variation in the anterior pelvic plane between sitting and standing positions and its inability to correlate with other spinopelvic parameters [3–5]. Furthermore, THA is performed in a patient supine or lateral

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position, which does not account for the patient's functional position in standing and sitting [6]. Spinopelvic alignment is most often measured in terms of PI-LL mismatch, which is the mathematical difference between pelvic incidence (PI) and standing lumbar lordosis (LL; Fig. 1) and can be used to quantify the severity of lumbar flatback deformity (normal, <10°; mild, 10°-20°; severe, >20°) [7]. In the ASD literature, it has been demonstrated greater PI-LL mismatch is associated with worse disability [8]. Although previous studies have evaluated lumbopelvic mechanics in weight-bearing positions, there are limited reports describing the regional and global sagittal spinal alignment in patients with concomitant hip osteoarthritis (OA) [9–22].

The aims of our study were 2-fold: (1) to describe the vertebral alignment of the thoracolumbar spine between standing and sitting and (2) to elucidate the effects of lumbar degeneration and flatback deformity on sitting-standing spinopelvic mechanics. We hypothesize that lumbar degeneration and flatback deformity alter regional and global spinal alignment in standing and sitting positions compared to patients with normal lumbar spines.

Methods

Data Collection

This is a single-center, retrospective, radiographic review of consecutive patients over age 18 undergoing primary THA. Appropriate institutional review board approval was obtained before study initiation. As part of our institution's standard preoperative assessment for THA, all patients had full-body, weight-bearing standing and sitting anteroposterior and lateral stereoradiographs. Inclusion criteria were all preoperative primary THA candidates who underwent stereoradiographic imaging and the ability to sit and stand for the duration of the radiographic study. Exclusion criteria were defined as poor visualization of the lumbar spine or femoral heads, transitional vertebrae, history of hip arthroplasty, hip ankylosis, and prior lumbar fusion. The severity of hip OA was graded using the Kellgren-Lawrence system, which measures joint space narrowing, osteophytes, and other evidence of OA on radiograph [23,24].

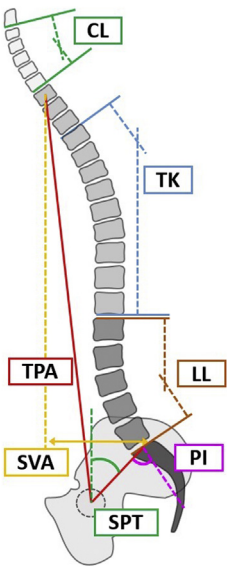


Fig. 1. Radiographic sagittal alignment parameters were measured in both sitting and standing positions for each patient. CL, cervical lordosis; LL, lumbar lordosis; PI, pelvic incidence; SPT, spinopelvic tilt; SVA, sagittal vertical axis; TK, T4-T12 thoracic kyphosis; TPA, T1 pelvic angle.

Image Acquisition

All patients underwent low-dose radiation, head-to-foot, biplanar stereoradiographic images (EOS imaging, Paris, France) [25,26]. The EOS system is a slot-scanning radiographic device consisting of 2 X-ray source-detector pairs, allowing simultaneous orthogonal image acquisition. The standardized protocol included a weight-bearing, free-standing position of comfort, and unsupported sitting position with femurs parallel to the floor, both with arms flexed at 45° and with fingers on clavicles [27]. Due to the field of view of the EOS, the lower extremity distal to the proximal femur was unable to be captured in sitting.

Radiographic Analysis

All radiographic measurements were performed using a dedicated surgical planning software (Surgimap; Nemaris, New York, NY) [28]. Radiographic evaluation of regional sagittal spinal parameters included LL (L1-S1 Cobb angle), thoracic kyphosis (TK; T4-T12 Cobb angle), and cervical lordosis (CL; C2-C7 Cobb angle). Spinopelvic parameters included PI (angle between the center of the femoral head axis, the midpoint of the sacral endplate, and a line perpendicular to the sacral endplate) and spinopelvic tilt (SPT; angle between the center of the femoral head axis, the midpoint of the sacral endplate, and the vertical; Fig. 1).

Spinopelvic alignment was assessed with radiographic analysis of the mismatch (mathematical difference) between PI and LL (PI minus LL or PI-LL), which is a measure of the severity of lumbar flatback deformity (normal, <10°; mild, 10°-20°; severe, >20°). From the ASD literature, it has been demonstrated that greater PI-LL mismatch is associated with worse disability [8]. Greater PI-LL mismatch has also been cited as a significant risk factor for THA dislocation [29].

Global sagittal spinal alignment was measured by the C7 sagittal vertical axis (SVA; sagittal offset in millimeters between a plumb-line dropped from the center of the C7 vertebral body and the posterosuperior aspect of S1) and T1 pelvic angle (TPA; angle between the center of the T1 vertebral body, the center of the femoral head axis, and the midpoint of the sacral endplate; Fig. 1). Larger values for SVA and TPA represent more forward sagittal alignment, which is associated with greater disability [8,30]. All measurements were performed in both the sitting and standing postures for each patient.

Patients were categorized based on radiographic assessment of lumbar spinal pathology into 3 groups: normal, degenerative, or lumbar flatback. Patients were placed in the degenerative group if there was radiographic evidence in at least 1 disc of loss of disc height >50%, facet arthrosis, or spondylolisthesis. Spondylolisthesis was defined as anterior migration of one vertebral body over the caudal body by >3 mm; facet arthrosis was defined as the presence of facet hypertrophy or osteophytes in anteroposterior or lateral radiographs. The lumbar flatback group included patients with radiographic evidence of lumbar spine degeneration, in addition to having PI-LL mismatch >10° [31].

Statistical Analysis

Statistical analysis was performed with SPSS v23.0 (IBM Corp, Armonk, NY). Propensity score matching by age, body mass index (BMI), and hip OA grade was performed to control for previously demonstrated confounding variables. Paired *t*-tests were used to assess changes in regional and global spinopelvic parameters from the standing position to the sitting position within groups. The assessment of differences between matched spinal pathology

Table 1
Patient Demographics for All Patients (Unmatched) and With Propensity Score Matching Controlling for Age, BMI, and Hip OA Grade (Matched).

Characteristic	Total	Normal	Degenerative	Flatback	*P* Value
Unmatched					
Cases	491	183	216	92	
Age, y (SD)	60.94 (±13.49)	52.29 (±13.54)	65.71 (±9.68)	66.33 (±12.79)	<.001
BMI, kg/m^2 (SD)	27.72 (±5.77)	26.91 (±5.11)	27.86 (±5.85)	29.01 (±6.59)	.018
Sex					.229
% Female	61.30%	64.70%	61.80%	53.80%	
% Male	38.70%	35.30%	38.20%	46.20%	
Hip OA grade (SD)	2.22 (±1.24)	1.79 (±1.19)	2.45 (±1.19)	2.49 (±1.21)	<.001
Matched					
Cases	171	57	57	57	
Age, y (SD)	61.76 (±11.42)	61.25 (±11.86)	62.55 (±9.96)	61.49 (±12.46)	.812
BMI, kg/m^2 (SD)	28.10 (±5.84)	28.41 (±5.61)	27.51 (±5.14)	28.38 (±6.71)	.646
Sex					.347
% Female	58.00%	58.90%	64.30%	50.90%	
% Male	42.00%	41.1%	35.7%	49.10%	
Hip OA grade (SD)	2.26 (±1.17)	2.28 (±1.00)	2.30 (±1.32)	2.21 (±1.19)	.915

BMI, body mass index; OA, osteoarthritis; SD, standard deviation.

groups was performed using one-way analysis of variance. Statistical significance was set at $P < .05$.

Results

Demographics

Overall, 491 patients met the inclusion criteria. Of these patients, 183 (37.27%) were placed in the normal group, 216 (43.99%) in the degenerative group, and 92 (18.74%) in the flatback group. Significant differences were noted in age, BMI, and hip OA grade between the groups. After propensity score matching for age, BMI, and hip OA grade, 171 patients were included (57 in each group). The overall mean age for the matched cohorts was 61.76 ± 11.42 years, mean BMI was 28.10 ± 5.84 kg/m^2, mean hip OA grade was 2.26 ± 1.17, and 58.00% of the patients were female. No significant differences in demographic characteristics were observed between matched cohorts (Table 1).

Sagittal Parameters

The 3 groups demonstrated significant differences in standing sagittal alignment. As the severity of disease increased across the groups from normal to degenerative to flatback, there was a corresponding increase in standing SPT (12.4° vs 12.9° vs 24.8°, $P = .048$), PI-LL (−6.7° vs −3.6° vs 16.1°, $P < .001$), SVA (9.9 mm vs 22.8 mm vs 38.9 mm, $P < .001$), and TPA (8.8° vs 10.6° vs 22.2°, $P < .001$). Across the same spectrum, there was also a decrease in standing LL (59.9° vs 54.8° vs 47.1°, $P < .001$).

Significant differences were also noted in the sitting position between the normal, degenerative, and flatback groups. Compared to the normal and degenerative groups, the flatback group demonstrated the greatest sitting SPT (26.3° vs 24.9° vs 32.8°, $P = .048$), PI-LL (15.7° vs 13.6° vs 29.3°, $P < .001$), SVA (57.3 mm vs 52.0 mm vs 69.8 mm, $P < .001$), and TPA (25.1° vs 23.3° vs 31.8°, $P < .001$). The flatback group also demonstrated the lowest sitting LL (38.0° vs 37.4° vs 33.8°, $P < .001$).

An analysis comparing the 3 groups demonstrated significant stepwise differences in lumbopelvic mechanics when transitioning from standing to sitting in normal, degenerative, and flatback groups, respectively (Table 2, Fig. 2). The lumbar flatback group was found to have least change in SPT (13.93° vs 11.98° vs 7.95°, $P = .029$) and LL (−21.91° vs −17.45° vs −13.23°, $P = .003$) when transitioning from standing to sitting. The flatback group also demonstrated the smallest change in TPA (16.35° vs 12.69° vs 9.64°, $P = .002$) and PI-LL (22.32° vs 17.28° vs 13.18°, $P = .001$). A significant difference in SVA change from standing to sitting was also noted between groups (48.99 mm vs 30.0 mm vs 32.1 mm, $P = .006$). The groups did not differ significantly for changes in PI, TK, or CL (Fig. 2).

One-way analysis of variance was used to compare the mean change in SPT from sitting to standing position in patients with normal PI-LL (PI-LL, −10° to 10°), moderate lumbar flatback (PI-LL, 10° to 20°), and severe lumbar flatback (PI-LL, >20°). Changes in SPT from sitting to standing decreased based on the severity of flatback deformity (normal, 15.3°; moderate, 8.3°; and severe, 5.9°; $P < .010$).

Sitting and standing radiographs of a patient with lumbar flatback deformity and a patient without deformity are shown in Figure 3.

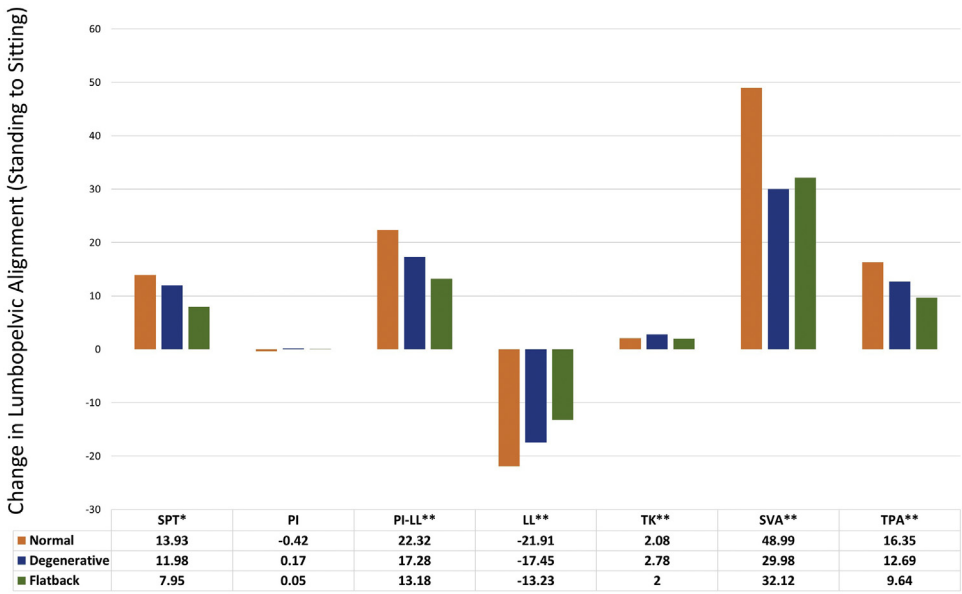
Discussion

Lumbar spinal degeneration is relatively common among patients undergoing THA. In a recently published study by Buckland

Table 2
Sagittal Parameters From Standing to Sitting in Normal, Degenerative, and Lumbar Flatback Deformity.

Spinopelvic Alignment Parameter	Normal (N = 57)			Degenerative (N = 57)			Lumbar Flatback (N = 57)		
	Standing	Sitting	*P* Value	Standing	Sitting	*P* Value	Standing	Sitting	*P* Value
SPT	12.40 ± 5.25	26.33 ± 10.97	<.0001	12.91 ± 7.81	24.89 ± 14.00	<.0001	24.81 ± 7.03	32.75 ± 9.96	<.0001
PI	53.22 ± 10.13	53.64 ± 11.65	.555	51.17 ± 11.33	51.00 ± 15.95	.914	63.13 ± 13.64	63.08 ± 12.82	.966
PI-LL	−6.65 ± 8.04	15.67 ± 15.47	<.0001	−3.64 ± 10.20	13.64 ± 16.73	<.0001	16.09 ± 5.27	29.26 ± 12.43	<.0001
LL	59.87 ± 9.21	37.97 ± 15.49	<.0001	54.81 ± 11.18	37.36 ± 15.91	<.0001	47.05 ± 14.84	33.82 ± 16.13	<.0001
TK (T4-T12)	−40.90 ± 11.22	−38.82 ± 11.59	.003	−40.43 ± 11.70	−37.65 ± 15.20	.028	−29.58 ± 10.99	−27.58 ± 11.26	.007
SVA (mm)	9.89 ± 26.90	57.30 ± 28.00	<.0001	22.76 ± 32.14	52.00 ± 29.99	<.0001	38.91 ± 40.23	69.76 ± 30.41	<.0001
TPA	8.79 ± 5.70	25.14 ± 10.20	<.0001	10.62 ± 7.13	23.31 ± 12.75	<.0001	22.15 ± 6.02	31.78 ± 9.88	<.0001

LL, lumbar lordosis; PI, pelvic incidence; PI-LL, mismatch between pelvic incidence and lumbar lordosis; SPT, spinopelvic tilt; SVA, sagittal vertical axis; TK, T4-T12 thoracic kyphosis; TPA, T1 pelvic angle.



	SPT*	PI	PI-LL**	LL**	TK**	SVA**	TPA**
Normal	13.93	-0.42	22.32	-21.91	2.08	48.99	16.35
Degenerative	11.98	0.17	17.28	-17.45	2.78	29.98	12.69
Flatback	7.95	0.05	13.18	-13.23	2	32.12	9.64

Fig. 2. Change in sagittal parameters during the transition from standing to sitting between normal, degenerative, and lumbar flatback groups. Change in SPT was significantly different with $P < .05$ (*) across groups, and change in PI-LL, LL, SVA, and TPA were significantly different with $P < .01$ (**) across groups. PI-LL, mismatch between pelvic incidence and lumbar lordosis; SVA, C7 sagittal vertical axis.

et al [32], 16% of THA candidates had a PI-LL mismatch greater than 10°. Although previous studies have demonstrated changes in lumbopelvic sagittal alignment from sitting to standing positions [9–13,19–22,33], an analysis with stratification by the severity of spinal pathology has not been previously reported.

In the sitting position, the innominate bone moves to the posterior point of the hip axis, the pelvis tilts posteriorly, psoas tension decreases, LL decreases, and hip extensors are under tension [33,34]. The present study demonstrates not only that there are significant changes in regional and global sagittal spinal alignment when transitioning from standing to sitting but also that the magnitude of change is significantly associated with the severity of degenerative lumbar spinal pathology.

The cascading sequence of sagittal compensatory mechanisms in ASD has been thoroughly described in previous spine surgery literature [19–22]. The initial compensation for loss of LL is to lordose flexible spinal segments and to increase SPT (increase posterior pelvic tilt) [35]. PI-LL mismatch serves as the primary instigator of sagittal spinal deformity, thus increasing SPT (posterior pelvic tilt) is the principal compensatory response [20,36]. As described by Diebo et al [20], the pelvis progressively tilts posteriorly as the first (and primary) compensatory mechanism for flatback deformity, as defined by PI-LL [37]. Our results support this finding with increasing standing SPT with increasing loss of LL. As the severity of spinal pathology increases across the 3 groups, the standing LL decreased from normal to degenerative to flatback. There was also a corresponding increase in compensatory posterior SPT from normal to degenerative to flatback. Patients with greater PI-LL mismatch therefore exhibited less change in pelvic tilt from standing to sitting as there is less available LL, and some of the available posterior tilt had already been recruited in standing.

The importance of the hip-spine relationship and the changes in spinopelvic mechanics in ASD have been illustrated by recent analyses of sagittal spinopelvic parameters and the impact on

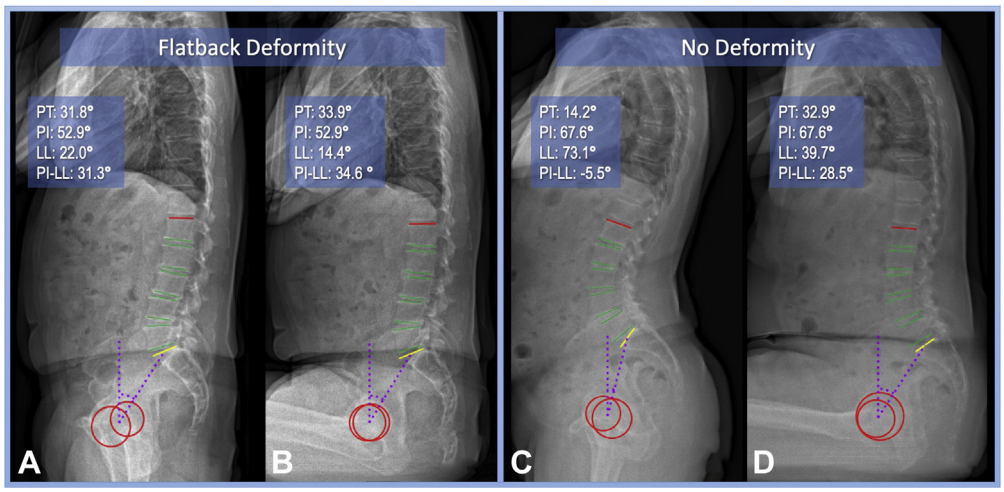


Fig. 3. Sitting and standing radiographs in a patient with flatback deformity and a patient without flatback deformity including measurements of PT, PI, LL, and PI-LL. (A and B) Sitting and standing lateral radiographs of a patient with flatback deformity. (C and D) Sitting and standing lateral radiographs of a patient with no deformity. Patients with flatback deformity were found to have a smaller change in spinopelvic parameters when going from sitting to standing.

acetabular positioning in THA. Several studies have demonstrated that every 1° of increased posterior pelvic tilt causes approximately 0.7° of acetabular anteversion [6,38,39]. This has significant implications in acetabular cup positioning in patients with concurrent spinal deformity or who are undergoing surgical correction of spinal deformity, as subsequent changes in component position due to changes in pelvic tilt, which may increase the risk of implant instability [6,40–42]. Furthermore, patients who require both spinal realignment and THA may elect to have spinal realignment first if significant pelvic tilt change is anticipated [6]. DelSole et al [29] demonstrated a dislocation rate of 8.0% in primary THA despite acetabular placement within Lewinnek's "safe zone," suggesting that the traditional safe zone may not be applicable in the spinal deformity patient population. The patients in this study found to be at the highest risk of dislocation were those with increased degrees of standing SPT and larger PI-LL mismatch (ie, patients with more severe sagittal spinal deformity). As suggested by the authors, the high rate of dislocation likely results from changes in lumbopelvic mechanics between standing and sitting, with less protective change in pelvic tilt and acetabular anteversion [29].

The observed direct relationship between SPT and acetabular anteversion has led to the suggestion that the placement of the acetabular component be adjusted based on the patient's individual degree of pelvic tilt [11,43,44]. Logically, patients with more posterior pelvic tilt may require less acetabular anteversion to optimize standing acetabular component position; however, due to limited pelvic tilt change between standing and sitting, this may make anterior femoroacetabular impingement more likely in a seated posture. For this reason, ASD patients would likely benefit from preoperative standing and sitting radiographs to better plan for surgery. Furthermore, it is our opinion that THA candidates with concomitant spinal pathology would benefit from evaluation by a spine surgeon before undergoing THA in order to ensure optimal positioning of the acetabular component [6].

Our study was not without limitations. Although previous research has demonstrated significant associations between spinopelvic alignment and THA stability, the present study was a radiographic analysis of preoperative spinal alignment among THA candidates. As a result, we were unable to evaluate the relationship between sit-stand mechanics and spinal deformity with respect to post-THA outcomes and dislocation. It is also important to note that the degenerative group in this study encompassed a spectrum of disease severity. Although we observed distinct differences between the degenerative group and the normal and flatback groups, further stratification may provide a more accurate analysis of sit-stand mechanics. Additionally, magnetic resonance imaging is more sensitive to diagnose facet arthrosis, which may reduce the differences observed between normal and degenerative groups if some patients were inadvertently placed into the normal group. Another limitation of this study is that the average PI-LL mismatch in the flatback deformity group was 16°, which is traditionally considered to be "mild" deformity (PI-LL = 10°-20°). While the differences in spinopelvic mobility are likely greater in patients with moderate-severe flatback (PI-LL > 20°), our patient cohort unfortunately did not contain sufficient numbers in the "severe" flatback group to make definitive conclusions. However, we are starting to collaborate more with our spine colleagues to study patients with severe spinal deformity to address this limitation.

Conclusion

In summary, lumbar spinal degeneration occurs on a spectrum and results in lumbar flatback deformity. Among preoperative THA candidates, greater lumbar spinal degeneration is associated with a progressive reduction in spinopelvic mobility and loss of protective posterior pelvic tilt during postural changes. The demonstrated spinopelvic hypomobility in degenerative spinal pathology may help explain the higher rate of THA dislocations observed in this patient population.

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