Seated Lateral X-ray Is a Better Stress Radiograph of the Lumbar Spine Compared to Standing Flexion

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Abstract

Study Design: Prospective lumbar radiograph analysis.

Objective: To compare changes in lumbar lordosis in standing flexion versus seated lateral radiographs.

Methods: Standing lateral, standing flexion, and seated lateral X-rays of the lumbar spine were obtained in patients presenting with low back pain. Trauma, tumor, and revision cases were excluded. Changes in global lumbar as well as segmental lordosis were measured in each position.

Results: Seventy adult patients were reviewed. Overall, the greatest changes in lordosis were seen at L4-S1 in both the seated and flexion X-rays (12.5° and 6.3°, respectively). Greater kyphosis was seen in seated versus flexion X-rays (21.6° vs 15.8°); changes in lordosis from L1-L3 were similar in both positions, with little change seen at these levels (approximately 5° to 7°). On subgroup analysis, these differences were magnified in analyzing only patients that moved at least 20° globally, and there were no significant differences between sitting and flexion in “stiff” patients that moved less than 20° globally.

Conclusion: Greater lumbar kyphosis was seen in the seated position compared to standing flexion, especially from L4-S1. Given these results we suggest the use of seated lateral X-rays to dynamically assess the lumbar spine. These findings may also guide future research into the mechanism and clinical relevance of a stiff versus mobile lumbar spine, as well as into the sensitivity of seated X-rays in detecting instability.

Keywords

lumbar, lordosis, low back pain, sagittal alignment, radiology, biomechanics

Introduction

Standing lateral radiographs in the flexed position are widely used as a dynamic stress view of the lumbar spine. However, this technique is limited by significant variation in patient effort/ability to flex forward, as well as by the fact that the pelvis anteverts during standing flexion, which may restrict full lumbar flexion—particularly at the most caudal segments (L4 to S1) where the majority of degenerative pathology occurs.1 Importantly, several studies have shown a significant decrease in global lumbar lordosis with sitting compared to standing radiographs.2-6 Biomechanically, it is intuitive that sitting may induce a greater flexion moment in the lumbar spine: when a person sits the pelvis retroverts, in turn the lumbar spine must go into kyphosis in order to maintain forward gaze.5,7,8

Recently, in a study of 60 patients, Hey et al showed greater lumbar kyphosis in sitting versus standing flexion lateral lumbar X-rays.9,10 A few additional studies have evaluated sitting versus standing radiographs as a means to determine the most

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appropriate alignment with deformity correction, although these studies have been aimed more at optimizing sagittal posture with deformity correction rather than as an assessment of angular range of motion.\(^2\)\(^,\)\(^11\) Apart from these studies there is a paucity of work that directly compares standing flexion versus sitting radiographs as a means for dynamic assessment of the lumbar spine. The purpose of the present study is to evaluate global and segmental kyphosis (changes in lordosis) in the lumbar spine in standing flexion versus seated lateral radiographs. We hypothesized that seated lateral radiographs will effect greater kyphosis and thus may be a simple, more effective method for dynamic assessment of the lumbar spine in clinical practice.

**Materials and Methods**

Consecutive adult patients presenting for lumbar complaints at an orthopedic surgical spine clinic at a single institution were evaluated. Standing lateral X-rays in the neutral, flexion, and extension positions as well as seated lateral X-rays were taken as part of the initial clinical evaluation in all patients, in that sequence. This series of radiographs is obtained for all patients presenting with lumbar complaints as part of the initial surgical evaluation. Patients were instructed to stand first in neutral position, then in maximum forward flexion, then maximum lumbar extension for the standing images. Patients were then instructed to sit upright in a hard-back chair for the seated lateral radiograph. Patients with prior fusion, tumor, trauma, and/or infection were excluded. Imaging software (InteleViewer, Montreal, Canada) was used to measure global lumbar as well as segmental lordosis in each position. The 2 fellowship-trained orthopedic spine surgeons at our institution performed the imaging analysis and measurements using the Cobb method: using the angle subtended between the superior endplate of the cranial level and the inferior endplate of the caudal level.\(^12\) Measurements of global lordosis (L1-S1), segmental lordosis (L1-2, L2-3, L3-4, L4-5, and L5-S1), and regional lordosis (L1-L3 and L4-S1) were recorded. Displacement/listhesis of vertebral segments was also noted between each posture and measurements of the amount of displacement were recorded. Pelvic tilt and sacral slope were also measured in each posture. Patients provided consent for their de-identified data to be analyzed and published.

**Statistical Methods**

Descriptive statistics were used for analysis of patient demographics. Changes in global and segmental lordosis from standing to sitting and standing to flexion were recorded; comparisons were made using the \(t\) test for independent means. A subgroup analysis was performed whereby patients were separated into groups based on the amount of angular motion detected from standing to sitting. Separate analyses were performed for patients that displayed at least 10\(^\circ\) of change ("mobile") from L4-S1 and those that did not ("stiff"). Additional comparisons were calculated for patients with at least 20\(^\circ\) of change globally (L1-S1) from standing to sitting. These cutoff values were chosen based on the mean changes seen globally (L1-S1 = 21\(^\circ\)) and at L4-S1 (12\(^\circ\)) for all patients in this study. Chi-squared analysis was performed to analyze differences in age, gender, diabetes, smoking status, opioid use, baseline Oswestry Disability Index (ODI), duration of symptoms, and psychiatric diagnoses in the mobile versus stiff patients. Data calculations were performed using SPSS statistical software (IBM, New York).

**Results**

Seventy adult patients were analyzed (30 male; Table 1). The greatest lumbar kyphosis compared to neutral standing were seen at L4-S1 in both the seated and standing flexion X-rays (12.5\(^\circ\) and 6.3\(^\circ\), respectively; Table 2). Greater kyphosis was seen in seated versus flexion X-rays (21.6\(^\circ\) vs 15.8\(^\circ\), \(P = .004\)), as well as in segmental lordosis from L4-S1 (12.5\(^\circ\) vs 6.3\(^\circ\), \(P < .001\)); changes in lordosis from L1-L3 were similar in both positions, with little change in both the standing and seated views seen at these levels (approximately 5\(^\circ\) to 7\(^\circ\), \(P = .3\)). As expected, large differences in pelvic tilt and sacral slope between standing and sitting were observed; average pelvic tilt was 20\(^\circ\) with standing, 42\(^\circ\) with sitting, and 8\(^\circ\) with standing flexion. Subgroup analysis of patients moving at least 20\(^\circ\) from L1-S1 showed even larger differences between standing flexion and sitting (Table 3), while no significant differences between sitting and standing flexion in “stiff” patients that moved less than 20\(^\circ\) globally were found (Table 4). Increased age was noted in patients with stiff versus flexible lumbar spines (59 years vs 48 years, \(P < .001\)). We did not identify a greater incidence or magnitude of spondylolisthesis in the seated versus flexion radiographs. Figure 1 is an illustrative example of these findings in a patient with dramatically different lumbar alignment between the flexed and seated postures.

**Discussion**

The results of this study confirm the hypothesis that greater kyphosis occurs in the lumbar spine in the seated position as compared with standing flexion. Greater kyphosis with sitting is most notable from L4 to S1, which is where the majority of symptomatic degenerative lumbar pathology occurs. Many patients are likely to spend a significant amount of time sitting at work and in daily life; thus, the seated lateral radiograph may be a better “everyday” dynamic view of the lumbar spine, especially for evaluating the most caudal segments. Given these results, we recommend the use of seated lateral

**Table 1. Demographics (n = 70).**

|          | L1-S1 movers | L1-S1 stiff | \(P\) value |
|----------|--------------|-------------|-------------|
| Male     | 16 (42%)     | 14 (44%)    | —           |
| Female   | 22 (58%)     | 18 (56%)    | .9          |
| Age      | 48.2         | 59          | .001        |

20\(^\circ\) of change globally (L1-S1) from standing to sitting. These cutoff values were chosen based on the mean changes seen globally (L1-S1 = 21\(^\circ\)) and at L4-S1 (12\(^\circ\)) for all patients in this study.

Chi-squared analysis was performed to analyze differences in age, gender, diabetes, smoking status, opioid use, baseline Oswestry Disability Index (ODI), duration of symptoms, and psychiatric diagnoses in the mobile versus stiff patients. Data calculations were performed using SPSS statistical software (IBM, New York).
Table 2. All Patients (n = 70).

|                  | Change with sitting | %   | Change with flexion | %   | P value |
|------------------|---------------------|-----|---------------------|-----|---------|
| L1-S1 lordosis   | -21.60              | 36% | -15.8               | 26% | .004    |
| L1-L3 lordosis   | -5                  | 56% | -6.4                | 65% | .3      |
| L3-4 lordosis    | -5                  | 33% | -4.5                | 27% | .2      |
| L4-S1             | -12.5               | 33% | -6.3                | 16% | <.001   |
| L4-L5             | -6.7                | 34% | -2.8                | 8%  | <.001   |
| L5-S1             | -5.8                | 29% | -3.5                | 16% | .001    |
| Pelvic tilt       | 20                  | 126%| -12.7               | -66%| <.001   |
| Sacral slope      | -17.5               | 50% | -13.7               | 40% | <.001   |

Table 3. Patients That Move More Than 20° L1-S1 (n = 38).

|                  | Change with sitting | %   | Change with flexion | %   | P value |
|------------------|---------------------|-----|---------------------|-----|---------|
| L1-S1 lordosis   | -32                 | 53% | -20                 | 32% | <.001   |
| L1-L3 lordosis   | -7                  | 70% | -7.9                | 96% | .17     |
| L3-4 lordosis    | -7.4                | 50% | -5.2                | 33% | .02     |
| L4-S1             | -17.8               | 46% | -7.4                | 19% | <.001   |
| L4-L5             | -9.9                | 49% | -3.7                | 9%  | <.001   |
| L5-S1             | -7.9                | 39% | -4.1                | 19% | <.001   |
| Pelvic tilt       | 23.1                | 150%| -11.3               | -61%| <.001   |
| Sacral slope      | -20.9               | 61% | 13.6                | 39% | <.001   |

Table 4. Patients That Move Less Than 20° L1-S1 (n = 32).

|                  | Change with sitting | %   | Change with flexion | %   | P value |
|------------------|---------------------|-----|---------------------|-----|---------|
| L1-S1 lordosis   | -9.9                | 17% | -12.4               | 20% | 0.28    |
| L1-L3 lordosis   | -3                  | 28% | -4.9                | 42% | 0.29    |
| L3-4 Lordosis    | -2.5                | 17% | -4                  | 27% | 0.2     |
| L4-S1             | -6.5                | 18% | -5.1                | 12% | 0.06    |
| L4-L5             | -2.8                | 15% | -2                  | 6%  | 0.14    |
| L5-S1             | -3.6                | 19% | -3.1                | 14% | 0.19    |
| Pelvic tilt       | 17.2                | 104%| -14.4               | -69%| <0.001  |
| Sacral slope      | -13.6               | 37% | 14.4                | 41% | <0.001  |

Figure 1. Changes in lordosis from standing neutral, standing flexion, and sitting.
These differences in lumbar kyphosis between sitting and standing flexion are likely due to significant increase in pelvic retroversion with sitting; as our results confirm, the pelvis retroverts substantially with sitting as compared with standing flexion. The findings of this study may also suggest that sitting creates a more reproducible flexion moment on the lumbar spine as the pelvis retroverts in the seated position. We suspect that the seated radiograph may be a better means to detect cases of subtle spondylolisthesis and/or disc space wedging, although we were not able to confirm this with the small number of spondylolisthesis cases reviewed.

In 2017, Hey et al reported findings similar to those of the present study. In their work they were also able to show significantly greater kyphosis with sitting radiographs as compared with standing flexion. Indeed, they had originally planned to analyze 100 patients but terminated the study early due to a significant difference seen with just 60 patients. The results of our present study agree with those reported by Hey et al that sitting induces greater kyphosis in the lumbar spine, especially in the L4-S1 segments. Interestingly, we were able to show an even greater difference in the seated position on subgroup analysis of those with “flexible” lumbar spines as defined by at least 20° of angular change from L1-S1 with sitting. Our subanalysis shows that there is a subset of patients that do not have significant motion in the lumbar spine with either standing flexion or sitting. Increased age was the only significant factor associated with a “stiff” lumbar spine in this analysis. Somewhat surprisingly, we did not detect a significant difference in smoking, diabetes, baseline ODI, or duration of symptoms in those in the stiff versus flexible groups. These additional results thus cannot be fully explained based on the data available in this analysis, although the findings may have important clinical implications and certainly warrant future study.

In addition, the findings of this investigation may guide future research into the mechanisms and potential solutions for the problem of adjacent segment degeneration. Adjacent segment degeneration (ASD) has been demonstrated to occur more rapidly after lumbar fusion. The explanation for this phenomenon is certainly multifactorial and remains a topic of debate and continued investigation. Studies have shown that decreased segmental lordosis, higher body mass index, longer fusion constructs, and preexisting degeneration all may contribute to ASD. We have shown that most of the motion in the lumbar spine occurs from L4 to S1, and it of course follows that the majority of degenerative pathology occurs at these levels. Thus, we postulate that fusion of the L4-5 and/or L5-S1 levels will significantly alter the physiologic motion in the lumbar spine and contribute to further degeneration at more cranial levels. This is conjecture based on the present study, and will require future studies to analyze angular range of motion above fusion constructs.

Recently, segmental and global malalignment have been implicated as an independent risk factor for inducing ASD. In this study by Rothenfluh et al, 7-year reoperation for ASD was amazingly reported at nearly 80% in patients with decreased segmental lordosis after lumbar fusion. Even in those patients with appropriate lordosis after fusion (such that lumbar lordosis matched pelvic incidence), the reoperation rate at 7 years in this same series was roughly 25%. Thus, even if the segment is fused in the “proper” amount of lordosis (as measured by standing radiographs) there remains a high rate of ASD. The findings of our present study may give some indication as to why accelerated ASD may occur even when standing lordosis and pelvic incidence match. Adjacent levels to fusion are known to experience increased intra-disceal pressure, as cadaveric studies have demonstrated. We suspect the large angular changes shown in our study are likely to induce significant sheer stresses on the adjacent segment; further study is warranted to quantify and corroborate this hypothesis.

Our study is not without limitation. The clinical implications of the findings presented here are not fully established. Indeed, we suspect that there may be an increased ability to detect dynamic spondylolisthesis in the seated versus the standing posture; however, we were not able to show such differences with these results. Only 8 patients with radiographic spondylolisthesis were included in this analysis, and thus we were likely underpowered to detect a difference between standing and sitting. An important implication of our findings is that standing flexion radiographs could potentially miss dynamic listhesis or disc space wedging (indicative of facet incompetence) that occurs with sitting, thus leading the surgeon to decompress and not fuse that segment at surgery. The present findings cannot confirm such a proposition; however, future studies may consider analysis of a larger cohort of patients with known dynamic instability to assess the sensitivity and specificity of the seated radiograph as compared to standing flexion. In addition, patient effort is likely to have a significant influence on the posture of the lumbar spine and cannot be fully accounted for in this study. Indeed, even in the seated position it is possible for a patient to alter his or her lumbar lordosis significantly with either sitting fully upright or slumping forward. Thus, a limitation of the present study is that we were not able to objectively measure patient effort with forward flexion. Despite this limitation we suspect that patients may act as their own internal control, as similar amounts of pain-related guarding are likely to occur in both standing and sitting. In addition, it is possible that lack of effort with standing forward flexion limits the angular changes seen in this position, which itself is a potential argument for why the seated lateral is a better stress radiograph. The presence/degree of hip osteoarthritis and disc height was not studied; if those variables were controlled for, one might expect an even larger increase in lumbar kyphosis with seated flexion. Finally, we did not utilize the slumped-sitting posture in this study, which may have limited the differences seen between standing and sitting, and thus our findings may even underreport the true difference in angular motion between standing and sitting.
Conclusion
The findings of this study suggest that the seated lateral radiograph shows greater relative kyphosis as compared with standing flexion, particularly from L4-S1. Our analysis shows greater lumbar motion from standing to sitting, with greater kyphosis and pelvic tilt seen with sitting. These findings suggest that the seated lateral radiograph may be able to uncover more subtle dynamic listhesis, which could alter surgical decisions, although with the small number of spondylolisthesis patients in this analysis this is speculative. Given these results, we recommend the use of seated lateral radiographs for dynamic assessment of the lumbar spine in the clinical setting. These findings may also provide some insight into the potential mechanisms of accelerated adjacent segment degeneration after lumbar fusion; further study is warranted to corroborate the findings of this study and to investigate the impact of fusion on lumbar range of motion using seated rather than flexion radiographs. Finally, future work will be required to determine the pathophysiology and clinical significance of stiff versus mobile lumbar segments.

Declaration of Conflicting Interests
The author(s) declared the following potential conflicts of interest with respect to the research, authorship, and/or publication of this article: Dr Sielatycki, Dr Devin, and Dr Hodges have stock ownership with respect to the research, authorship, and/or publication of this article. The author(s) received no financial support for the research, authorship, and/or publication of this article.

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References
1. Dvorak J, Panjabi MM, Chang DG, Theiler R, Grob D. Functional radiographic diagnosis of the lumbar spine. Flexion-extension and lateral bending. Spine (Phila Pa 1976). 1991;16:562-571.
2. Hey HWD, Teo AQA, Tan KA, et al. How the spine differs in standing and in sitting-important considerations for correction of spinal deformity. Spine J. 2017;17:799-806.
3. De Carvalho DE, Soave D, Ross K, Callaghan JP. Lumbar spine and pelvic posture between standing and sitting: a radiologic investigation including reliability and repeatability of the lumbar lordosis measure. J Manipulative Physiol Ther. 2010;33:48-55.
4. O’Sullivan K, O’Dea P, Dankaerts W, O’Sullivan P, Clifford A, O’Sullivan L. Neutral lumbar spine sitting posture in pain-free subjects. Man Ther. 2010;15:557-561.
5. Sparrey CJ, Bailey JF, Saffae M, et al. Etiology of lumbar lordosis and its pathophysiology: a review of the evolution of lumbar lordosis, and the mechanics and biology of lumbar degeneration. Neurosurg Focus. 2014;36:E1.
6. Endo K, Suzuki H, Nishimura H, Tanaka H, Shishido T, Yamamoto K. Sagittal lumbar and pelvic alignment in the standing and sitting positions. J Orthop Sci. 2012;17:682-686.
7. Been E, Kalichman L. Lumbar lordosis. Spine J. 2014;14:87-97.
8. Bailey JF, Sparrey CJ, Been E, Kramer PA. Morphological and postural sexual dimorphism of the lumbar spine facilitates greater lordosis in females. J Anat. 2016;229:82-91.
9. Hey HW, Lau ET, Lim JL, et al. Slump sitting X-ray of the lumbar spine is superior to the conventional flexion view in assessing lumbar spine instability. Spine J. 2017;17:360-368.
10. Dennis Hey HW, Choong DAW, Lin AZ, et al. Patient and radiographer assessment of slump sitting flexion compared to conventional standing forward bending flexion. J Spine Surg. 2018;4:750-756.
11. Hey HW, Wong CG, Lau ET, et al. Differences in erect sitting and natural sitting spinal alignment-insights into a new paradigm and implications in deformity correction. Spine J. 2017;17:183-189.
12. Gstoettner M, Sekyra K, Walochnik N, Winter P, Wachter R, Bach CM. Inter- and intraobserver reliability assessment of the Cobb angle: manual versus digital measurement tools. Eur Spine J. 2007;16:1587-1592.
13. Been E, Gomez-Olivencia A, Shefi S, Soudack M, Bastir M, Barash A. Evolution of spinopelvic alignment in hominins. Anat Rec (Hoboken). 2017;300:900-911.
14. Hilibrand AS, Robbins M. Adjacent segment degeneration and adjacent segment disease: the consequences of spinal fusion? Spine J. 2004;4(6 suppl):190S-194S.
15. Ekman P, Moller H, Shalabi A, Yu YX, Hedlund R. A prospective randomised study on the long-term effect of lumbar fusion on adjacent disc degeneration. Eur Spine J. 2009;18:1175-1186.
16. Bagheri SR, Alimohammadi E, Zamani Froushani A, Abdì A. Adjacent segment disease after posterior lumbar instrumentation surgery for degenerative disease: incidence and risk factors. J Orthop Surg (Hong Kong). 2019;27:2309499019842378.
17. Rothenfluh DA, Mueller DA, Rothenfluh E, Min K. Pelvic incidence-lumbar lordosis mismatch predisposes to adjacent segment disease after lumbar spinal fusion. Eur Spine J. 2015;24:1251-1258.
18. Weinhoffier SL, Guyer RD, Herbert M, Griffith SL. Intradiscal pressure measurements above an instrumented fusion. A cadaveric study. Spine (Phila Pa 1976). 1995;20:526-531.