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Adjacent Segment Motion After a Simulated Lumbar Fusion in Different Sagittal Alignments

A Biomechanical Analysis

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Study Design. An *in vitro* biomechanical study of adjacent segment motion (at L3–L4 and L5–S1) after a simulated lumbar interbody fusion of L4–L5 in different sagittal alignments was carried out.

Objectives. To test the hypothesis that an L4–L5 fixation in different sagittal alignments causes increased angular motion at the adjacent levels (L3–L4 and L5–S1) in comparison with the intact spine.

Summary of Background Data. Clinical experience has suggested that lumbar fusion in a nonanatomic sagittal alignment can increase degeneration of the adjacent levels. It has been hypothesized that this is the result of increased motion at these levels; however, to the authors' knowledge no mechanical studies have demonstrated this.

Methods. Eight fresh human cadaveric lumbar spines (L3–S1) were biomechanically tested. Total angular motion at L3–L4 and L5–S1 under flexion-extension load conditions (7-Nm flexion and 7-Nm extension) was measured. Each specimen was tested intact, and then again after each of three different sagittal fixation angles (at L4–L5): (1) *in situ* (21°lordosis), (2) hyperlordotic (31°lordosis), and (3) hypolordotic (7°lordosis). The simulated anterior/posterior fusion was performed at L4–L5 with pedicle screws posteriorly, vertebral body screws anteriorly, and an interbody dowel.

Results. The averaged values for flexion-extension motion at L3–L4 were as follows: intact specimen 2.0°, *in situ* fixation 4.0°, hyperlordotic fixation 1.7°, hypolordotic fixation 6.5°. The averaged values for flexion-extension motions at L5–S1 were as follows: intact specimen 2.3°, *in situ* fixation 2.6°, hyperlordotic fixation 3.6°, hypolordotic fixation 2.9°.

Conclusion. Hypolordotic alignment at L4–L5 caused the greatest amount of flexion-extension motion at L5–S1, and the difference was statistically significant in comparison with intact specimen but not *in situ* fixation or hypolordotic fixation. [Keys words: lumbar spine, lumbar interbody fusion, adjacent segment, biomechanics, sagittal alignment, lumbar lordosis, lumbar kyphosis, spinal instrumentation] **Spine 2003;28:1560–1566**

Degenerative changes at the levels adjacent to a lumbar spinal fusion have been well recognized,[1–7] although the reasons why they occur are not fully understood. Pathologic conditions that develop at adjacent levels after lumbar fusions range from degenerative arthritis of the facet joints, spinal canal stenosis, and severe disc degeneration to degenerative spondylolisthesis.[4] It is our experience that degenerative changes at the adjacent level above the fusion are more common than degenerative changes at the adjacent level below the fusion.

Biomechanical studies have shown that lumbar fusion produces increased motion and increased intradiscal pressure at the adjacent levels.[8–18] It has been suggested that this is caused by the patient, after surgery, attempting to obtain the same range of motion as before surgery. Several factors are thought to be involved in the increase in degeneration at adjacent levels: the stiffness of the fixation, the number of levels fused, the health of the adjacent level, and any postoperative lumbar sagittal malalignment. The importance of sagittal realignment and maintenance of lordosis during fixation have been documented in clinical studies,[19,20] and clinical experience suggests that lumbar fusion in a nonanatomic sagittal alignment can cause a deleterious effect at the adjacent level. Schlegel *et al*[7] investigated 58 patients and hypothesized that incorrect sagittal alignment of a lumbar fusion caused degeneration at the adjacent level by inducing too much motion at that level. However, they were unable to show statistical significance. There have been only limited experimental studies to determine the effect of lumbar sagittal malalignment on the biomechanics of the adjacent levels.[11,14,17] Umehara et al[17] used human cadaver spines and made a two-level instrumented posterior fixation with different sagittal alignments at L4–S1. They then measured the strain at the lamina of L3 under an extension moment. Their study showed that the strain at the L3 lamina increased when lordosis was decreased in the instrumented segments. Oda et al[14] investigated the effects of a kyphotic aligned fusion on the adjacent levels using an *in vivo* sheep

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Table 1.

Number	Age	Sex	IS (intact)	Hyper	Hypo
1	59	M	−20	−27	−7
2	82	M	−21	−28	−7
3	81	M	−23	−28	−7
4	86	F	−23	−42	−6
5	74	F	−22	−29	−10
6	80	M	−23	−33	−13
7	84	M	−23	−34	−4
8	89	F	−16	−28	−3
Average	79.4		−21.3	−31.1	−7.2

model. Their study showed that a kyphotic aligned fusion may lead to degenerative changes of the facet joints at the cranial adjacent level. None of these studies, however, determined the effect of sagittal fusion alignment on the motions at adjacent levels.

Using human lumbar spines, we performed a biomechanical study to compare the motion at the adjacent disc levels after creating simulated lumbar fusions in different sagittal alignments (in the foregoing we use the term hypolordotic to mean kyphotic fixation in the lumbar spine).

■ Materials and Methods

Specimen Preparation. After multiple specimens were screened, eight fresh-frozen human lumbar motion segments (L3–S1) were obtained from five male and three female fresh cadavers. The average age at death was 79.4 years (range, 59–89 years) (Table1). None of the cadavers had a history of spinal disease. Each motion segment underwent radiography to ensure that no major structural abnormalities were present. In preparation for biomechanical testing, the specimens were thawed to room temperature and cleaned of all residual muscle, great care being taken to preserve the bone structure and all ligaments, joint capsules, and discs. The cranial (L3) and caudal (S1) vertebral bodies of the multisegmental specimens were then potted up to their midbodies in a 10-cm diameter polyvinylchloride (PVC) end cap by use of dental cement. Care was taken to ensure that the discs (L3–L4 and L5–S1) were clear of the cement and easily accessible.

The PVC end cap that contained S1 was clamped in a materials testing machine (MTS-868 Mini Bionix, MTS Systems, Eden Prairie, MN, USA) with the superior sacral endplate set approximately 45° to the horizontal.

Before each specimen was embedded in the cement, sagittal angle marking screws (2.5 mm in diameter, 60 mm long) were carefully inserted in the anterior aspect of each vertebral body so that each screw protruded by about 40 mm in the sagittal plane (Figure 1). These screws acted as sagittal angle markers during subsequent measurements of flexion-extension motions.

Biomechanical Loading Sequence. The specimen was then mechanically tested according to the following three-step loading sequence. The first step was a conditioning step, whereas steps 2 and 3 were carried out to obtain the relative motion at each disc level.

Step 1: Establishing the Center of Rotation and Conditioning. The center of rotation for flexion-extension was established in the intact motion segment at the beginning of the experiment. This was accomplished as follows: a pure compressive load of 50 N was applied through a ball race to the top of the surface of the PVC end cap, which contained the upper vertebra (L3). The ball race was positioned, the load was applied and then repositioned, and the load was applied until no angular rotation in the coronal or sagittal planes could be detected when the 50-N compressive load was applied. This position on the top surface of the end cap was designated the center of rotation and was clearly marked. This position remained as the reference position for the center of rotation for all the remaining steps carried out on that particular motion segment. Then, as conditioning, the motion segment was subjected to a period of cyclic compression (500 N ± 150 N, at 1 Hz, for 1000 cycles) applied through the center of rotation.

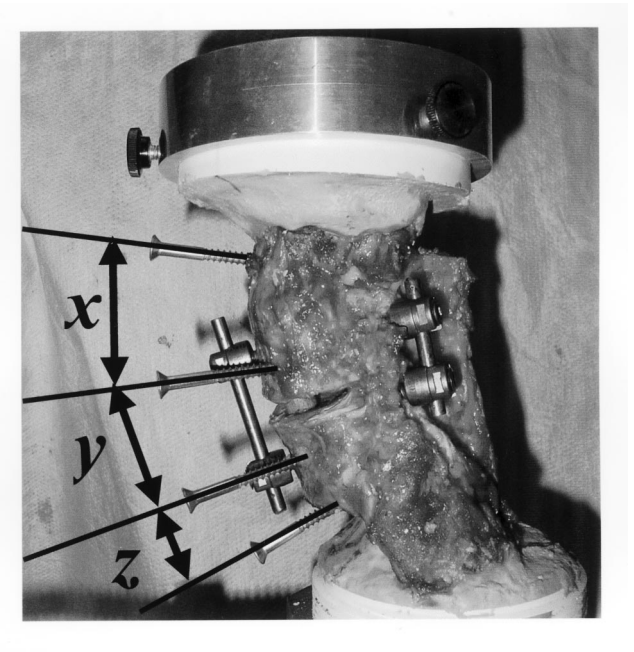

Figure 1. Specimen mounted on the testing machine. The anterior projecting screws acted as sagittal angle measurement markers during subsequent flexion-extension motions. A simulated anterior/posterior lumbar fixation was performed at L4–L5 with pedicle screws and rods posteriorly, vertebral body screws and a rod anteriorly, and a wood dowel as an interbody spacer. Angles between adjacent pairs of screws (*x, y, z*) were measured using a digital camera and a software package (Grab It version 1.51). The relative motion at each disc level in flexion (and extension) was calculated. Flexion-extension total motion was calculated by summing motion in flexion and extension.

Step 2: Flexion. By use of a fixed digital camera (DSC-S70, Sony, Tokyo, Japan), a lateral view image was obtained with the specimen in neutral position (before any load was applied). Then a 350-N load was applied 2 cm anterior to the center of rotation, to produce a flexion moment of 7 Nm (*i.e.*, 0.02 × 350 = 7 Nm) in flexion. The 350 N designation was chosen because it is about half bodyweight (a 70-kg person weighs approximately 700 N). With the specimens in the flexed position, a second lateral view image was obtained. This test was then repeated to obtain a second set of digital images: one set in the neutral position and one set in the flexed position (<7 Nm).

Step 3: Extension. A lateral view image was again obtained in the neutral position (before any load was applied). Then a

350-N load was applied 2 cm posterior to the center of rotation to produce an extension moment of 7 Nm, and a lateral view image was obtained. This test was then repeated to obtain a second set of images: one set in the neutral position, and one set in the extended position (<7 Nm).

Measurement of the Sagittal Angular Motion at the Disc Spaces. The images obtained in steps 2 and 3 were used to measure the relative sagittal angular motion at the disc spaces L3–L4, L4–L5, and L5–S1 when the specimen was subjected to 7 Nm in flexion and 7 Nm in extension. With the sagittal marking screws used as markers, the angles between adjacent pairs of screws were measured by use of a software package (Grab It version 1.51, Datatrend Software, Raleigh, NC, USA). The relative motion at each disc level in flexion (or extension) was calculated using the difference between the angle obtained in the neutral position with that obtained in the flexed (or extended) position. The average of the two tests carried out for step 2 (or step 3) was used as the result (the two results for the two tests were nearly always exactly the same). Flexion-extension total motion was calculated by summing motion in flexion and extension.

The accuracy of our method using the digital camera with the software package was checked against a digital goniometer (±0.1°; Smartlevel, Wedge Innovations, San Jose, CA), and observed to be within ± 0.2°.

Test Groups. Intact specimens were tested before simulated fixation. A simulated anterior/posterior lumbar fixation was performed at L4–L5 with pedicle screws and rods (Xia Spinal System, Stryker Spine, Allendale, NJ, USA) posteriorly, vertebral body screws and a rod (Xia Spinal System) anteriorly, and a wood dowel as an interbody spacer. Anterior and posterior longitudinal ligaments were cut at L4–L5, and the L4–L5 disc was resected totally before insertion of the wood dowel as the spacer. The sagittal fixation angle was varied at L4–L5 by changing the size of the dowel used as the spacers and adjusting the length of the rods (see below). Three different sagittal angles were tested. At each of these sagittal angles, steps 1, 2, and 3 of the biomechanical loading sequence were repeated, and lateral digital images were taken each time. From these the relative sagittal angular motion between L3–L4, L4–L5, and L5–S1 was measured each time (Table 1, Figure 2).

1. *In situ* fixation (range −16 to −23°; average −21.3°). The L4–L5 fixation was set at the same angle as the intact specimen.
2. Hyperlordotic fixation (range −27 to −42°; average −31.1°). This was obtained by using a large wood dowel interbody spacer and shortening the distance between the L4 and L5 posterior pedicle screws.
3. Hypolordotic fixation (range −3 to −13°; average −7.2°). This was obtained by using a small wood dowel interbody spacer and lengthening the distance between the L4 and L5 posterior pedicle screws.

The angles given in parentheses are the sagittal angles of the L4–L5 fixation. The sagittal angles were measured by the Cobb's method (angle between the top of L4 and the bottom of L5) using radiographs of each specimen. Negative values indicated lordosis.

Statistics. Means and standard deviations based on eight specimens are shown for each of the measured values: intact, *in situ*, hyperlordotic, hypolordotic. Because the same specimen was used for each of the measured values, paired *t* tests were used to identify statistically significant differences. Significance was stated at the 0.05 significance level.

Results

The average flexion-extension motions at L3–L4, L4–L5, and L5–S1 for the different sagittal fixation alignments at L4–L5 are shown in Table 2 and shown graphically in Figure 3. Note that the statistical analyses were carried out using paired differences, whereas the standard deviations shown in Table 2 are based on the averages. The standard deviations are shown only to give a perspective.

The average flexion-extension motions at L4–L5 were as follows: intact specimen 4.3°, *in situ* fixation 0.6°, hyperlordotic fixation 1.1°, hypolordotic fixation 1.0°. These latter three angles were significantly smaller than the angle measured for the intact specimen ($P < 0.01$). This significant decrease in motion at L4–L5 after fixation indicates that the instrumentation simulated a biologic fusion.

The averaged values for flexion-extension motions at L3–L4 were as follows: intact specimen 2.0°, *in situ* fixation 4.0°, hyperlordotic fixation 1.7°, hypolordotic fixation 6.5°. In comparison with the intact specimen, the percentage increases were as follows: *in situ* fixation +100% ($P < 0.05$), hyperlordotic fixation −15% ($P > 0.05$), hypolordotic fixation +225% ($P < 0.01$). The motion in hyperlordotic fixation was not significantly different in comparison with the intact specimen ($P > 0.05$). The value obtained for the motion in the hypolordotic fixation was significantly greater than the other three values ($P < 0.01$). In comparison with the other three, the percentage increases were as follows: intact specimen +225%, *in situ* fixation +63%, hyperlordotic fixation +300%.

The averaged values for flexion-extension motions at L5–S1 were as follows: intact specimen 2.3°, *in situ* fixation 2.6°, hyperlordotic fixation 3.6°, hypolordotic fixation 2.9°. In comparison with the intact specimen, the percentage increases were as follows: *in situ* fixation +13%, hyperlordotic fixation +57%, hypolordotic fixation +26%. The motion in hyperlordotic fixation was significantly greater than that for the intact specimen ($P < 0.05$) but not significantly different in comparison with the *in situ* fixation or the hypolordotic fixation ($P > 0.05$).

Discussion

The main results from these experiments were as follows:

1. The superior adjacent level (L3–L4) showed greater amounts of flexion-extension motion when the fixed segment (L4–L5) was aligned either *in situ* (the same alignment as when the specimen was nonfixed) or hypolordotic, but not hyperlordotic. The greatest amounts of flexion-extension motion at L3–L4 occurred with hypolordotic fixation alignment at L4–L5. In comparison with the other three, the hypolordotic fixation caused

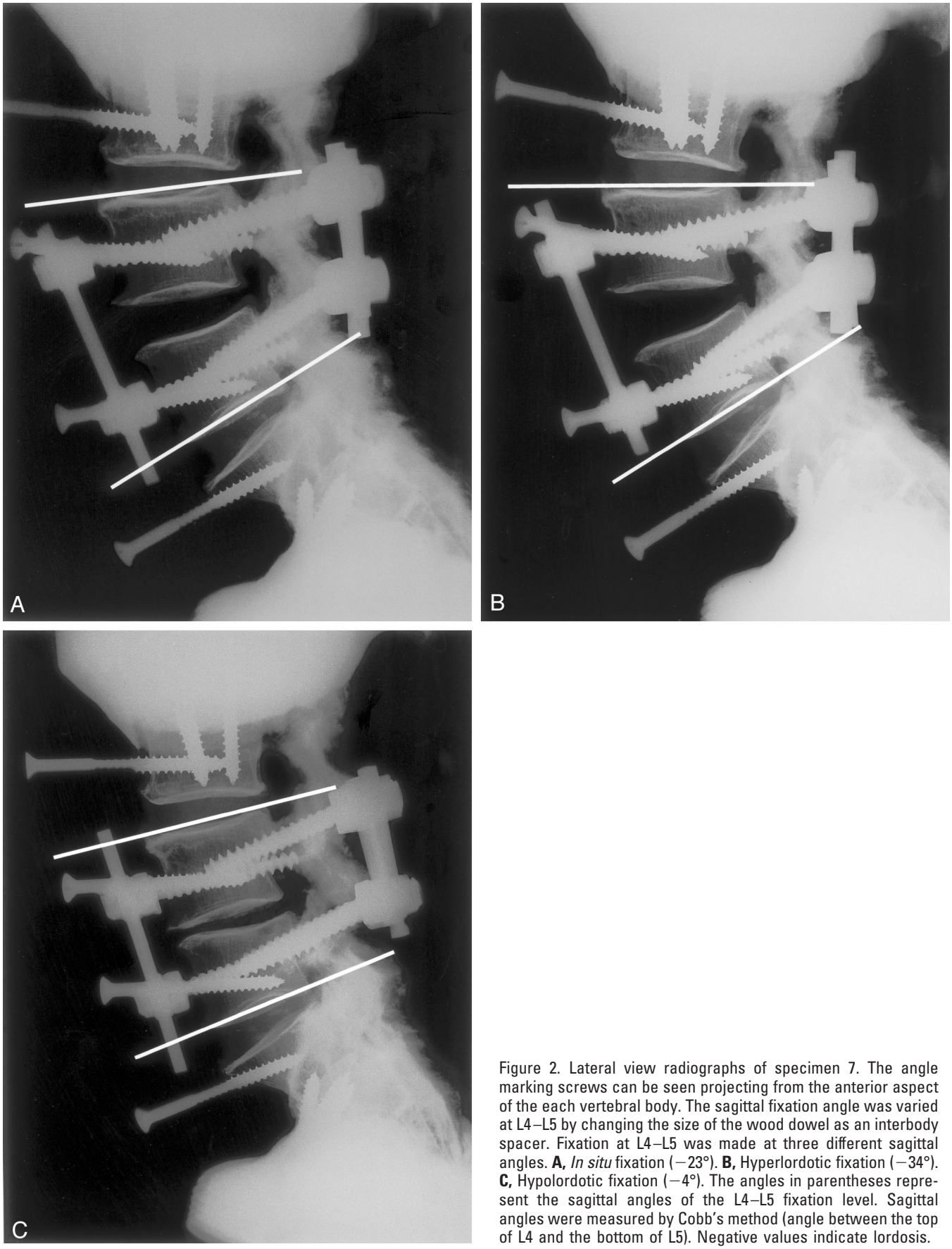

Figure 2. Lateral view radiographs of specimen 7. The angle marking screws can be seen projecting from the anterior aspect of the each vertebral body. The sagittal fixation angle was varied at L4–L5 by changing the size of the wood dowel as an interbody spacer. Fixation at L4–L5 was made at three different sagittal angles. **A,** *In situ* fixation (−23°). **B,** Hyperlordotic fixation (−34°). **C,** Hypolordotic fixation (−4°). The angles in parentheses represent the sagittal angles of the L4–L5 fixation level. Sagittal angles were measured by Cobb's method (angle between the top of L4 and the bottom of L5). Negative values indicate lordosis.

Table 2.

	L3–L4	L4–L5 (fixed level)	L5–S1
Intact specimen	2.0	4.3†#!	2.3
In situ fixation	4.0*#	0.6	2.6
Hyper-lordotic fixation	1.7	1.1	3.6*
Hypo-lordotic fixation	6.5*†#	1.0	2.9

the following percentage increases in motion: intact specimen +225%, *in situ* fixation +63%, hyperlordotic fixation +300%.

2. The flexion-extension motion at the inferior adjacent level (L5–S1) was not significantly different for any of the fixation alignments at L4–L5.

3. When the fixed segment (L4–L5) was aligned in a hyperlordotic fashion, the level above (L3–L4) the fixed segment (L4–L5) did not show significantly greater amounts of flexion-extension motion, in comparison with the intact specimen. However, the level below (L5–S1) the fixed segment (L4–L5) did show greater amounts of flexion-extension motion in comparison with the intact specimen but not with the other two fixation alignments.

It should be noted that the percentage differences given above and in the results are quite large, whereas the actual values in degrees are quite small.

In the study presented here, the average segmental lordosis of the intact spine at L4–L5 was observed to range from −16° to −23° (mean −21.3°) (Table 1). Average radiographic assessed values of the lumbar sagittal profile in healthy individuals have significant variance.[1,19,21] Gelb et al[22] measured 100 asymptomatic volunteers and observed that the L4–L5 lordosis angle ranged from 0° to −44° (mean −24°). Jackson et al[19] measured 50 volunteers and observed that the L4–L5 lordosis angle ranged from −10° to −29° (mean −17°). Thus, our measured values fall in line with those from previous studies.

The hypolordotic alignment at L4–L5 caused the greatest amounts of motion at L3–L4, and hyperlordotic alignment caused the least amounts of motion to be seen at L3–L4. Although the differences in L3–L4 motion between hypolordotic and the other alignments were sig-

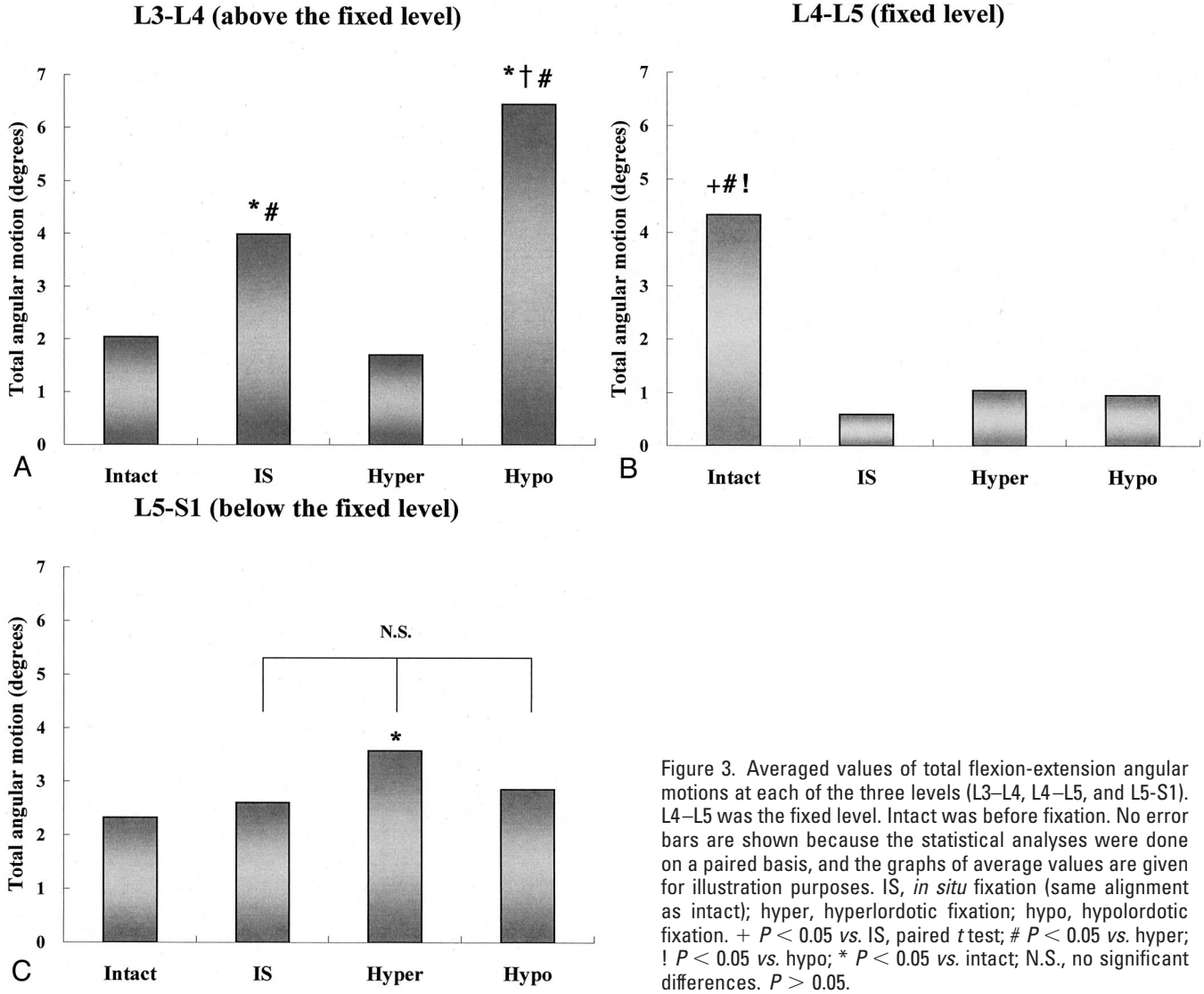

Figure 3. Averaged values of total flexion-extension angular motions at each of the three levels (L3–L4, L4–L5, and L5-S1). L4–L5 was the fixed level. Intact was before fixation. No error bars are shown because the statistical analyses were done on a paired basis, and the graphs of average values are given for illustration purposes. IS, *in situ* fixation (same alignment as intact); hyper, hyperlordotic fixation; hypo, hypolordotic fixation. + $P < 0.05$ *vs.* IS, paired *t* test; # $P < 0.05$ *vs.* hyper; ! $P < 0.05$ *vs.* hypo; * $P < 0.05$ *vs.* intact; N.S., no significant differences. $P > 0.05$.

nificant, they were only a few degrees and would be difficult to detect in the clinical situation. However, the percentage differences in motion were quite large. Thus, these findings suggest that a hypolordotic fixation at L4–L5 causes a large increase in relative motion at L3–L4, and this could, over time, lead to a higher rate of degenerative changes at the superior adjacent level.

The reason for these differences in motion at L3–L4 could be in the load sharing arrangement with the posterior column at L3–L4 when the overall sagittal alignment is altered. When L4–L5 was fixed in hypolordotic alignment, we observed that the upper surface of the L3–L4 segment tilted forward, as in flexion (in relation to L4–L5); this could have relieved the posterior column and allowed the facet joints to open slightly. Thus, although the L3–L4 segment saw the same magnitude of compression and moment in flexion and in extension, the orientation of the L3–L4 joint might have caused it to become lax and resist differently. This is indicated by the fact that the different alignments affected the level above (*i.e.*, L3–L4) rather than the level below (*i.e.*, L5–S1) because the level below never changed alignment with changes at L4–L5. This interpretation is somewhat validated by Umehara et al,[17] who made a two level instrumented posterior fixation at L4–S1 in cadaver spines and measured the strain at the lamina of L3 under an extension moment of 5 Nm. The fixation alignment at L4–S1 was changed as a baseline lordosis, loss of lordosis of 4°, and loss of lordosis of 8°. In that study, the strain in the L3 lamina increased when lordosis was decreased (*i.e.*, hypolordosis).

In the clinical situation, when an L4–L5 fusion is set in a hypolordotic sagittal alignment, the levels above and below the fusion level may both be required to compensate for the changes in spinal alignment in order to maintain normal posture and center of gravity (for example, hypokyphosis in thoracic spine, hyperlordosis in upper lumbar spine, and a decrease in sacral inclination). This means that the changes in motion measured at L3–L4 in our experiment would be compensated to some extent *in vivo*, at L5–S1, and may lead to changes at both adjacent levels in the clinical situation.

The importance of realigning the sagittal lumbar profile during spinal reconstruction from a clinical viewpoint has been documented.[19,20] Loss of segmental lumbar lordosis (or hypolordosis) is associated with an increased incidence of low back pain from iatrogenic flat back syndrome and with an increased rate of the degenerative changes seen at the adjacent levels. This study showed that flexion-extension motion at the segment above the fixation was increased when the fixed level was aligned in a hypolordotic fashion; this suggests that it may be beneficial to fuse the lumbar spine in an *in situ* or hyperlordotic alignment.

Slippage occurs most often at L4–L5 in degenerative spondylolisthesis, and patients with L4–L5 spondylolisthesis may have associated L3–L4 or L5–S1 disc degeneration. The need to extend the fusion segment to include the mild or moderately degenerated adjacent discs in such patients is controversial.[2,8,6,15] The results presented here suggest that the surgeon may wish to avoid fusing the L4–L5 joint in a hypolordotic alignment, to avoid the potential of cephalad adjacent segment breakdown.

It should be added that because of the vagaries of postmortem collection, we were not able to acquire specimens from young cadavers, and one disadvantage of this experiment is that our specimens were from cadavers with an average age of 79.4 years. Clearly, this group of cadavers does not represent patients in the general population who undergo surgery and fusion. Furthermore, our results could have been influenced by the removal of the residual muscles and to some extent by the fact that we used only L3 to S1. In life, the paraspinal muscles would act as stabilizers, so that excessive motion at one level would be somewhat inhibited by the muscle attachments at those levels and the levels above L3.

■ Conclusion

Hypolordotic alignment at L4–L5 caused the greatest amount of flexion-extension motion at L3–L4, and the differences were statistically significant in comparison with the intact specimen, *in situ* fixation, and hyperlordotic fixation. Hyperlordotic alignment at L4–L5 caused the greatest amount of flexion-extension motion at L5–S1, and the difference was statistically significant in comparison with the intact specimen but not with *in situ* fixation or hypolordotic fixation.

■ Key Points

- An *in vitro* biomechanical study of L4–L5 lumbar fixation in three different sagittal alignments was carried out and the total flexion-extension motions at the adjacent levels (L3–L4 and L5–S1) were measured. The three different fixation alignments were *in situ* fixation, hyperlordotic fixation, and hypolordotic fixation.
- Hypolordotic alignment at L4–L5 caused the greatest amount of flexion-extension motion at L3–L4, and the differences were statistically significant in comparison with intact, *in situ* fixation, and hyperlordotic fixation.
- Hyperlordotic alignment at L4–L5 caused the greatest amount of flexion-extension motion at L5–S1, and the difference was statistically significant in comparison with intact, but not with *in situ* fixation or hypolordotic fixation.

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