Eur Spine J (2001) 10 : 295–300
DOI 10.1007/s005860100278

ORIGINAL ARTICLE

Leonard Bastian
Uta Lange
Christian Knop
Guenter Tusch
Michael Blauth

Evaluation of the mobility of adjacent segments after posterior thoracolumbar fixation: a biomechanical study

Received: 19 August 1999
Revised: 14 February 2001
Accepted: 26 February 2001

This study was supported by Deutsche Forschungsgesellschaft (Bl 254/2–2). No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

L. Bastian (✉) · U. Lange · C. Knop
Department of Trauma Surgery,
Medizinische Hochschule,
30623 Hannover, Germany
e-mail: j.l.bastian@t-online.de,
Tel.: +49-511-5322050,
Fax: +49-511-532-5877,
URL: http://www.mhh-unfallchirurgie.de

G. Tusch
Department of Biometrics and Statistics,
Medizinische Hochschule,
Hannover, Germany

M. Blauth
Department of Trauma Surgery,
Leopold Franz University Innsbruck, Austria

Abstract An investigation was conducted into the effects of double-level T12–L2 posterior fixation on the mobility of neighboring unfused segments. The segmental mobility of adjacent segments above and below the fixation in ten cadaveric human thoracolumbar spine specimens was measured before and after fixation by biomechanical testing in flexion, extension, right lateral bending, and right rotation, and the data were compared. In flexion and extension, mobility of the segment above the double-level T12–L2 posterior fixation was significantly increased ($P<0.05$). In the adjacent segment below the fixation, there was no significant increased mobility after fixation for each moment applied. There is evidence that the adjacent segment above a double-level T12–L2 posterior fixation becomes more mobile, and this may lead to an accelerated degeneration in the facet joints due to increased stress at this point. This could be responsible for symptoms like low back pain after spinal surgery.

Keywords Mobility of adjacent segments · Accelerated degeneration · Posterior fixation · Posterior instrumentation

Introduction

Posterior transpedicular screw fixation is widely used for obtaining internal fixation of the thoracolumbar spine for management of the unstable spine caused by trauma, degenerative conditions and neoplasms. However, fusion generates a conflict between immediate benefit and later consequences [6]. Spinal fusion procedures, with or without instrumentation, have been reported to have various adverse effects, including pseudoarthrosis, spinal stenosis, spondylolysis and accelerated degeneration of the adjacent unfused segments [9]. Biomechanical and radiographic studies have shown increased forces [16], mobility [1, 14, 24], and intradiscal pressure [27] in adjacent segments after fusion. However, it is currently uncertain whether these increased mechanical demands lead to increased rates of pathology at the adjacent levels [5]. Lee [12] found that the most common pathologic condition responsible for new symptoms was symptomatic hypertropic facet joint arthritis. Increased motion at one or two motion segments above the fusion is supposed to be the reason for accelerated degeneration [21]. In a biomechanical study, Lee and Langrana [13] found that posterior fusion is the worst type of fusion in terms of producing the highest amount of stress in adjacent segments, especially in the facet joints.

An experimental study was conducted to investigate the reason for the hypertropic facet joint arthritis that developed after spinal fusions, especially after posterior pedicle screw fixation. In particular, the aim was to evaluate whether adjacent segments become hypermobile to compensate for the loss of spinal mobility caused by the fused segments.

Materials and methods

The experimental study was performed on ten fresh, human, cadaveric, thoracolumbar spines. The specimens between T10 and L4 from ten male cadavers (mean age: 45 years; range 26–63 years) were obtained at the time of postmortem autopsy. The specimens were stored in a freezer at –18°C until the day of testing, when each specimen was gradually thawed to room temperature. The specimens were cleaned and dissected from muscle and fat, taking care to preserve bone-ligament units intact. The test specimens included the spinal column between the vertebral bodies T10 and L4, which moves in all six directions. Throughout the entire testing procedure, the specimens were kept moist. Each specimen was then positioned in specially constructed cups and secured to the cups with rods, plates and specially constructed screws (Fig. 1). L3 and L4 were fixed in the bottom cup, T10 and T11 in the top, the L1/L2 discs were put in the horizontal plane. Then four Schanz screws (5 mm in diameter) were manually inserted into the pedicles of the T12 and L2 vertebrae for a later posterior fixation [Universal Spine System (USS), Fa. Synthes, Bochum, Germany], and four other screws were inserted into the bodies of T11, T12, L2, and L3 to hold the sensors (low-frequency magnet technology, resolution 0.1°) of the measurement system (3-Space-Tracker, Fa. Polhemus, Colchester, Vermont, USA; Fig. 2). After this, the specimen was positioned in an upright posture in the specially designed loading frame (Fig. 3). The L1/L2 discs were positioned in the horizontal plane. All measurements on each specimen were performed on a single day. First, the motion between L2 and L3 was measured without any instrumentation, followed once more after double-level T12–L2 posterior fixation. Because the pedicle screws had been inserted before positioning the specimen in the jig, the clamps and rods now could be applied easily. Neither taking out the specimen nor new calibration of the motion tracker was necessary. For measuring right and left lateral bending and torsion, the specimen would have had to be taken out of the jig and a new calibration had to be performed. To avoid having to repeat the calibration, right lateral bending and torsion was measured only. The rods and clamps of the posterior instrumentation were removed and measurements were repeated in the manner described above at the T11–T12 level. After all these procedures, a control measurement without instrumentation on six of the ten specimens was performed to demonstrate any increase in mobility independently of loss of ligament stiffness over the long testing time.

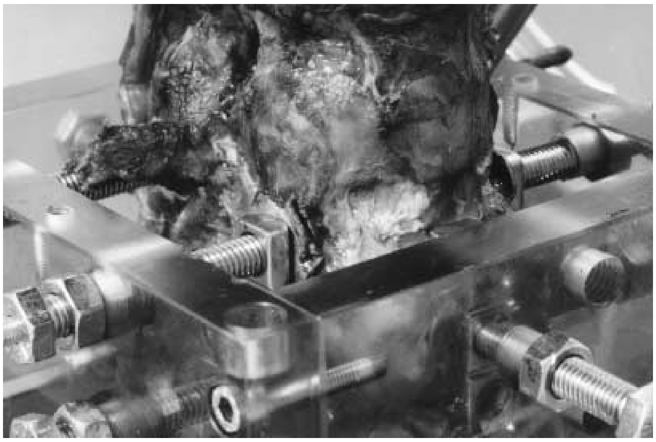

Fig. 1 Fixation of the specimen in the cup in detail

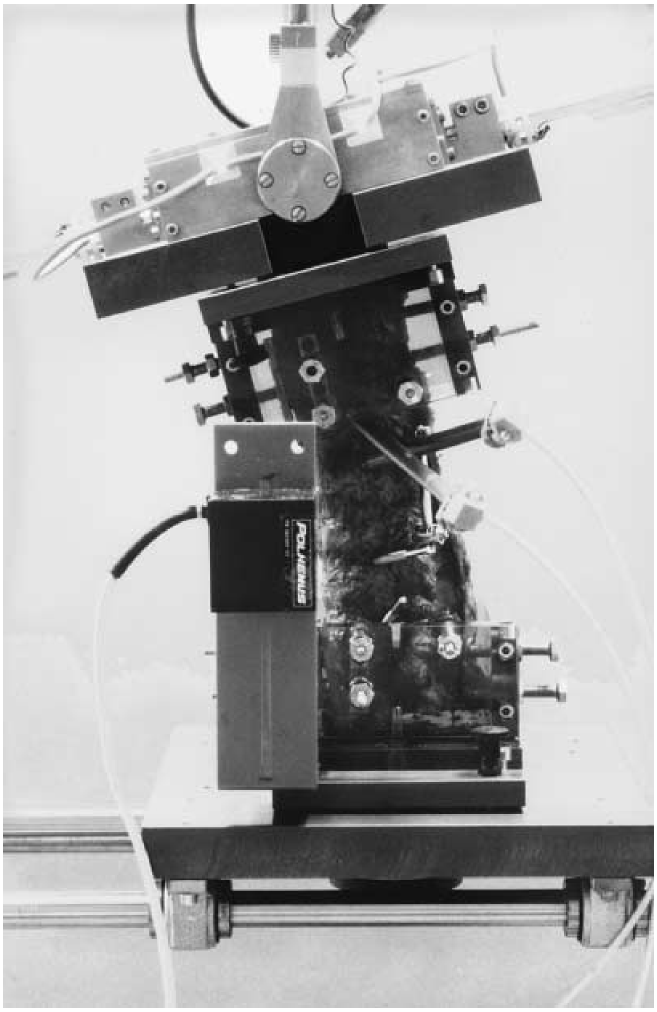

Fig. 2 Specimen in the loading jig in flexion (sensors of the motion tracker at T11/T12)

Bone mineral density

All specimens underwent bone mineral density (BMD) measurements before the experiment was started. Each specimen was placed in a 15-cm-deep bath of normal saline within a perspicuous container. The BMD (mgCa-HA/ml) of the T12–L2 vertebral bodies was measured by DE-QCT-BMD-evaluation (Somatom plus S and OsteoCT Software, Fa. Siemens, München-Erlangen, Germany).

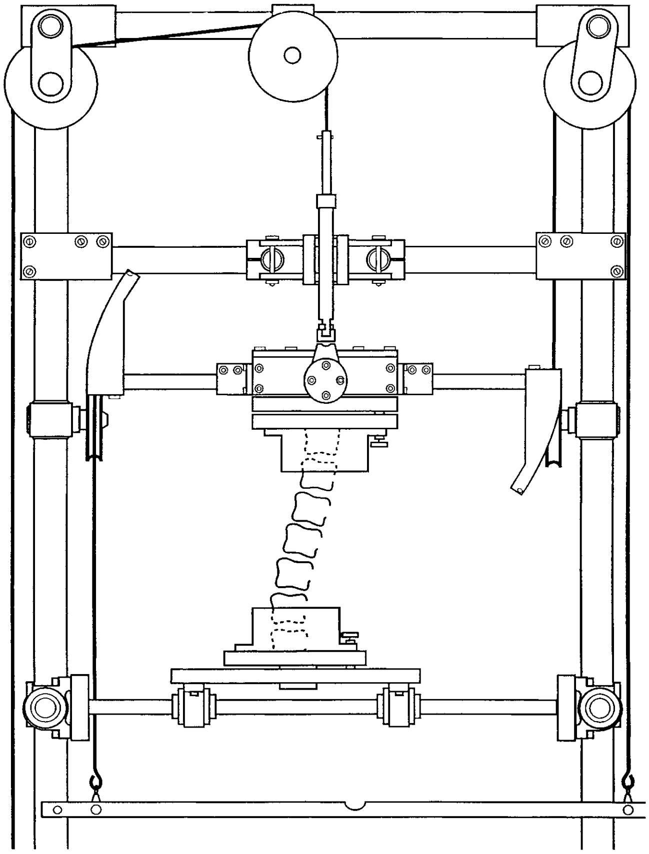

Fig. 3 A front view of the loading jig

Biomechanical testing

For a three-dimensional biomechanical study of spinal constructs (spinal specimen plus the instrumentation), a three-dimensional coordinate system was set up [18]. In this experiment, moments were applied only to the intact specimen and the construct, because such a load type produces uniform loading to each segment of a long specimen. The testing machine was specially designed (Fig. 3) to load the specimen in flexion, extension, right lateral bending and torsion, and to ensure free, three-dimensional movement of the whole specimen under the applied pure moments. An integrated stepper motor introduced the pure moments separately. The other five out of six degrees of freedom were free, enabling the specimen to move unconstrained. The maximum moment applied was 8 Nm. This was judged from preliminary experiments to be sufficient to produce physiologic motions, but to be small enough not to injure the spine specimen. Each moment was applied in three load-unload cycles, allowing 30 s of creep to occur at each loading step to precondition the specimen and minimize the viscoelastic effect of the specimen. The moments applied were: flexion, extension, right lateral bending, and right axial rotation. For each of the moments used, all six degrees of freedom, i.e., three translations along and three angulations about each of the three axes of the coordinate system, were measured by a motion tracker (3-Space-Tracker, Fa. Polhemus, Colchester, Vermont, USA) and range of motion (ROM), elastic zone (EZ), and neutral zone (NZ) were calculated [17]. Data were recorded and directly fed into a personal computer. Statistical analysis was performed using the paired *t*-test, exact Wilcoxon test and MANOVA ($P<0.05$) with computer software SPSS version 6.0.1 for Windows (SPSS Inc., Chicago, Illinois, USA).

Results

Bone mineral density

Average BMD values and standard deviation of the T12–L2 vertebrae are shown in Table 1. The BMD of all ten specimens used for this study fit within the normal range according to Siemens-Somatom Database and Kalender et al. [10].

Motion segment T11/T12

In flexion the specimens after double-level T12–L2 posterior fixation showed an increased mobility in the adjacent segment (T11/T12) for ROM above the fixation ($P<0.05$) (Fig. 4). In extension there was a significant difference in ROM, as well as for the EZ ($P<0.05$) (Fig. 5). Lateral bending and rotation did not show a greater mobility after posterior instrumentation (Fig. 6, Fig. 7).

Table 1 Specimen age and bone mineral density (BMD)

Specimen no.	Age (years)	Gender	BMD (g/cm^2) Mean ±SD
1	47	m	1.433 ± 0.22
2	55	m	1.213 ± 0.18
3	49	f	1.216 ± 0.46
4	26	f	1.343 ± 0.36
5	31	m	1.329 ± 0.62
6	55	m	1.314 ± 0.66
7	33	f	1.612 ± 0.54
8	33	m	1.585 ± 0.30
9	57	m	1.189 ± 0.21
10	63	f	1.016 ± 0.12

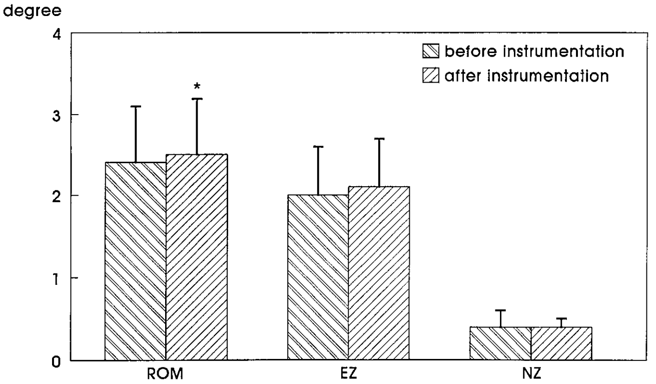

Fig. 4 Comparison of range of motion (ROM), elastic zone (EZ), and neutral zone (NZ) at T11/T12 before and after double-level fusion T11–L2 in flexion (mean values and standard deviation, $*P<0.05$)

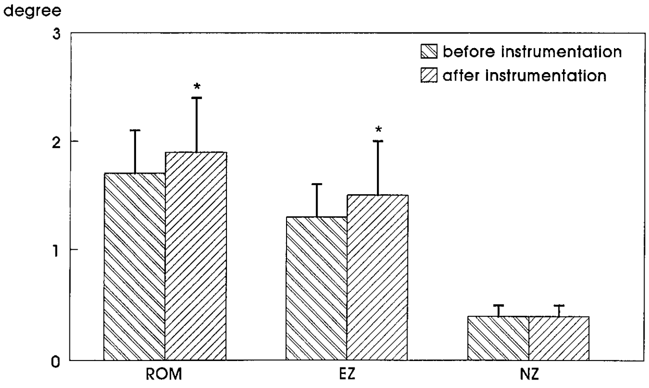

Fig. 5 Comparison of ROM, EZ, and NZ at T11/T12 before and after double-level fusion T11–L2 in extension (mean values and standard deviation, $*P<0.05$)

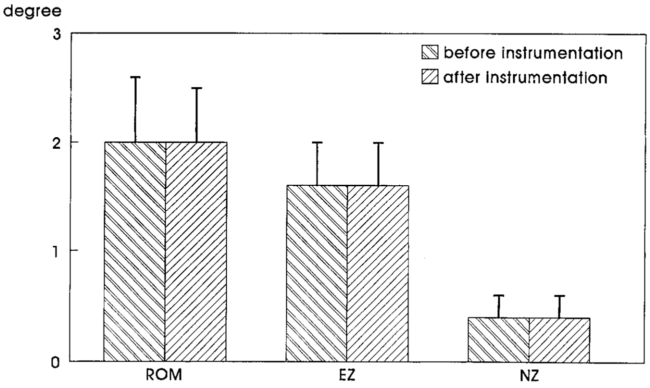

Fig. 6 Comparison of ROM, EZ, and NZ at T11/T12 before and after double-level fusion T11–L2 in lateral bending (mean values and standard deviation, $*P<0.05$)

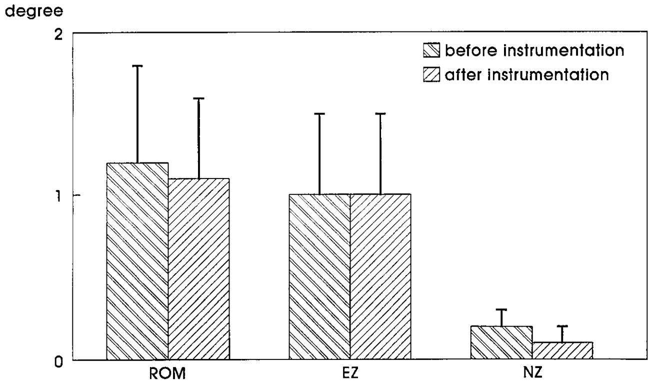

Fig. 7 Comparison of ROM, EZ, and NZ at T11/T12 before and after double-level fusion T11–L2 in axial rotation (mean values and standard deviation, $*P<0.05$)

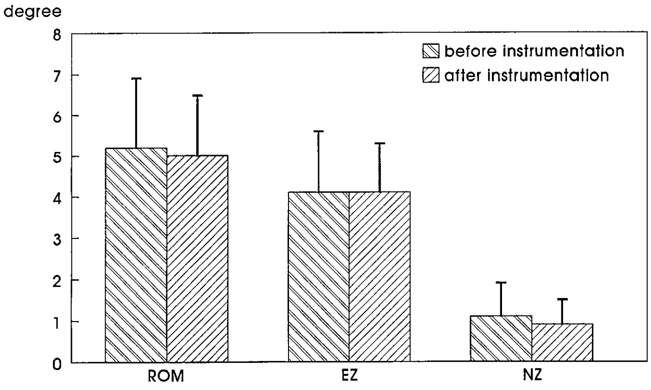

Fig. 8 Comparison of ROM, EZ, and NZ at L2/L3 before and after double-level fusion T11–L2 in flexion (mean values and standard deviation, $*P<0.05$)

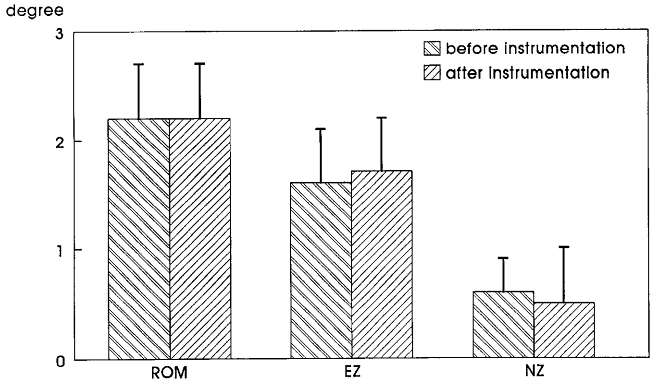

Fig. 9 Comparison of ROM, EZ, and NZ at L2/L3 before and after double-level fusion T11–L2 in extension (mean values and standard deviation, $*P<0.05$)

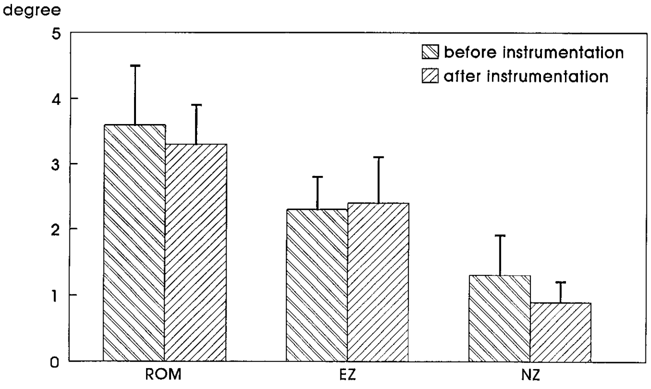

Fig. 10 Comparison of ROM, EZ, and NZ at L2/L3 before and after double-level fusion T11–L2 in lateral bending (mean values and standard deviation, $*P<0.05$)

Motion segment L2/L3

In the adjacent segment below the double-level T12–L2 posterior fixation there was no significant difference in segmental mobility for each moment applied (i.e., flexion, extension, right lateral bending, right rotation), either for ROM and EZ, or for NZ (Fig. 8, Fig. 9, Fig. 10, Fig. 11).

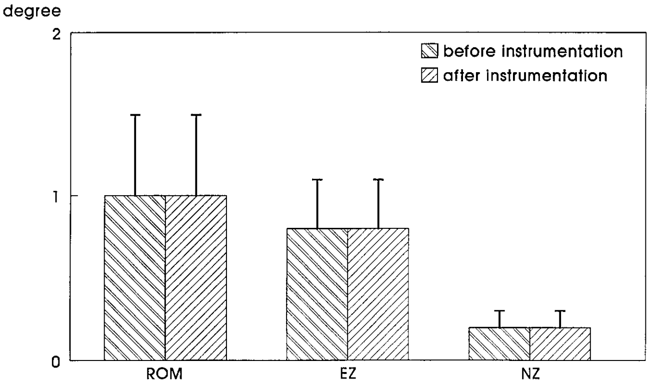

Fig. 11 Comparison of ROM, EZ, and NZ at L2/L3 before and after double-level fusion T11–L2 in axial rotation (mean values and standard deviation, *$P<0.05$)

Control (motion segment T11/T12)

After all these procedures, a control measurement was performed without any instrumentation on six of the ten specimens, and the results demonstrate that, in the segment T11/T12, there was no significant change in mobility due to loss of ligament stiffness for any of the moments applied, i.e., flexion, extension, right lateral bending, and right rotation, despite the long testing time.

Discussion

When motion at a highly flexible segment is eliminated through fixation, the motion and accompanying forces are thought to be transferred to the adjacent level. This can result in both hypermobility and abnormal patterns of motion. If the adjacent level is originally stiffer, this could increase the risk of accelerated degeneration [5, 13]. A previous biomechanical study has demonstrated that stress on the facet joints is increased by posterior fusion in particular, but also by anterior and bilateral fusion [13]. This corresponds with clinical findings that patients develop new symptoms from the segment adjacent to a fusion after an average symptom-free interval of 8.5 years [13] or an adjacent segment breakdown after an average symptom-free interval of 13.1 years [24]. The most common pathologic condition responsible for these new symptoms was symptomatic hypertropic facet joint arthritis [4, 7, 12], but spondylolisthesis and spinal canal stenosis also figured in some cases [7]. Baker et al. [2] reported changes in the cartilage of the posterior intervertebral joints after anterior fusion due to tuberculosis. They noted that it seems likely that the changes were initiated by two factors: first, prolonged immobilisation with consequent interference with the nutrition of articular cartilage; second, abnormal strains that produced changes in cartilage both from defective nutrition and from alterations of pressure of one cartilage surface on another. This accords with the findings of Salter and Field, that cell necrosis and subchondral bone thickening occurred at the point of abnormally high pressure on the joint surface [23].

In the current study, flexion, extension, lateral bending and rotation of the lumbar spine were simulated to investigate the behavior of adjacent segment motion before and after posterior fixation. It was found that there was a redistribution of segmental mobility along the lumbar spine after double-level T12–L2 instrumentation, with a significant increase in the adjacent unfused segment above the posterior fixation (T11/T12) for flexion and extension compared to the unfused spine. These findings accord well with those reported by Chow et al. [3] in a study on human cadaveric spine specimens, as well as with the findings reported by Shono et al. [26], who used calf lumbosacral spines in a material testing machine; Nagata et al. [16], in a study on canine spine specimens; Quinnel and Stockdale [22], after a single lumbar floating fusion; and also Lee and Langrana [13], after posterior instrumentation. The loss of segmental mobility at the fused segments tended to be compensated for by the unfused segments above the fusion [21, 26], although Luk et al. [15] reported that 5/7 years after single-level L4–L5 or double-level L4–S1 fusion, the lumbar spine becomes significantly less mobile than that of control subjects, and that the unfused segments are not required to compensate for this by becoming hypermobile. However, these findings without instrumentation may not be applicable to short fusion with instrumentation. Some authors reported significantly earlier degenerative changes after lumbar fusion using instrumentation [24], and other investigators showed that the development of these degenerative changes depends on the extent of the fusion [4, 8, 25].

We also investigated the motion of the unfused segment below the fusion, but could not find an increase in segmental mobility at this level (L2/L3). This is in correspondence with the clinical investigations by Lehmann et al. [14] in a long-term follow-up after lower lumbar fusions. They found that accelerated degeneration occurs as spinal stenosis in 42% of patients; in 30% it occurs within the segment above the fusion, and in 12% in the second segment above the fusion, 15% occur on multiple levels and 15% occur under the fusion mass as well. Stenosis never occurred under the fusion mass without occurring above the fusion level. For rotation and lateral bending, the posterior fixation produced no significant changes in the mobility of the adjacent segments above or below. However, it is flexion and extension that seem to be the most frequent movements in our daily activities.

To demonstrate further that the increase of mobility in the adjacent segment above the instrumentation is independent of losing ligament stiffness, the segmental motion of six of the ten specimens was measured after the whole testing procedure, again without any instrumentation. Our results showed no significant difference in mobility, particularly no increase of mobility at the T11/T12 level.

In our study, we did not use preloads and simulated muscle activity as reported by other groups [19, 20, 28], because of conflicting results. Wilke et al. [28] simulated the combination of five muscles, attached only to L4, with the muscle forces kept constant. However, the combination of muscles best simulating in vivo motions are not known. In reality, it is clear that a more complex muscular apparatus exists.

Conclusions

Living tissue responds to chronic changes in stresses and strains [11]. The hypermobility in the adjacent segment above the posterior fixation seems to accelerate degeneration in the facet joints, which is responsible for clinical symptoms like low back pain after spinal surgery. In order to avoid degenerative changes in the adjacent segments after spinal fusion with an instrumentation, the fusion should be as short as possible and the removal of the implant as early as justifiable.

References

1. Axelsson P, Johnsson R, Stromquist B (1997) The spondylolytic vertebra and its adjacent segment. Mobility measured before and after posterolateral fusion. Spine 22:414–417
2. Baker WC, Thomas TG, Kirkaldy-Willis WH (1969) Changes in the cartilage of the posterior intervertebral joints after anterior fusion. J Bone Joint Surg Br 51:736–746
3. Chow GH, Nelson BJ, Ebhard JS, Rugman JL, Rown CW, Donaldson DH (1996) Functional outcome of thoracolumbar burst fractures managed with hyperextension casting or bracing and early mobilization. Spine 21:2170–2175
4. Cochran T, Irstam L, Nachemson A (1983) Long-term anatomic and functional changes in patients with adolescent idiopathic scoliosis treated by Harrington rod fusion. Spine 8:576–584
5. Eck JC, Humphreys SC, Hodges SD (1999) Adjacent-segment degeneration after lumbar fusion: a review of clinical, biomechanical, and radiologic studies. Am J Orthop 28:336–340
6. Ehni G (1981) The role of spine fusion. Question 9. Spine 6:308–310
7. Etebar S, Cahill DW (1999) Risk factors for adjacent-segment failure following lumbar fixation with rigid instrumentation for degenerative instability. J Neurosurg 90:163–169
8. Hayes MA, Tompkins SF, Herndon WA, Gruel CR, Kopta JA, Howard TC (1988) Clinical and radiological evaluation of lumbosacral motion below fusion levels in idiopathic scoliosis. Spine 13:1161–1167
9. Hunter LY, Braunstein EM, Bailey RW (1980) Radiographic changes following anterior cervical fusion. Spine 5:399–401
10. Kalender WA, Felsenberg D, Louis O, Lopez P, Klotz E, Osteaux M, Fraga J (1989) Reference values for trabecular and cortical vertebral bone density in single and dual-energy quantitative computed tomography. Eur J Radiol 9: 75–80
11. Kim YE, Goel VK, Weinstein JN, Lim TH (1991) Effect of disc degeneration at one level on the adjacent level in axial mode. Spine 16:331–335
12. Lee CK (1988) Accelerated degeneration of the segment adjacent to a lumbar fusion. Spine 13:375–377
13. Lee CK, Langrana NA (1984) Lumbosacral spinal fusion. A biomechanical study. Spine 9:574–581
14. Lehmann TR, Spratt KF, Tozzi JE, Weinstein JN, Reinarz SJ, El-Khoury GY, Colby H (1987) Long-term follow-up of lower lumbar fusion patients. Spine 12:97–104
15. Luk KDK, Chow DHK, Evans JH, Leong JCY (1995) Lumbar spinal mobility after short anterior interbody fusion. Spine 20:813–818
16. Nagata H, Schendel MJ, Transfeldt EE, Lewis JL (1993) The effects of immobilization of long segments of the spine on the adjacent and distal facet force and lumbosacral motion. Spine 18: 2471–2479
17. Panjabi MM (1988) Biomechanical evaluation of spinal fixation devices. I. A conceptual framework. Spine 13: 1129–1134
18. Panjabi MM, Abumi K, Duranceau J, Crisco JJ (1988) Biomechanical evaluation of spinal fixation devices. II. Stability provided by eight internal fixation devices. Spine 13:1135–1140
19. Panjabi MM, Abumi K, Duranceau J, Oxland T (1989) Spinal stability and intersegmental muscle forces – a biomechanical model. Spine 14:194–200
20. Patwardhan AG, Havey RM, Meade KP, Lee B, Dunlap B (1999) A follower load increases the load-carrying capacity of the lumbar spine in compression. Spine 24:1003–1009
21. Pearcy MJ, Burrough S (1982) Assessment of bony union after interbody fusion of the lumbar spine using a biplanar radiographic technique. J Bone Joint Surg Br 64:228–232
22. Quinnell RC, Stockdale HR (1981) Some experimental observations of the influence of a single lumbar floating fusion on the remaining lumbar spine. Spine 6:263–267
23. Salter RB, Field P (1960) The effects of continuous compression on living articular cartilage. J Bone Joint Surg Am 42:31–49
24. Schlegel JD, Smith JA, Schleusener RL (1996) Lumbar motion segment pathology adjacent to thoracolumbar, lumbar, and lumbosacral fusions. Spine 21:970–981
25. Schulitz KP, Wiesner L, Wittenberg RH, Hille E (1996) Das Bewegungssegment oberhalb der Fusion. Z Orthop 134:171–176
26. Shono Y, Kaneda K, Abumi K, McAfee PC, Cunningham BW (1998) Stability of posterior spinal instrumentation and its effects on adjacent motion segments in the lumbosacral spine. Spine 23:1550–1558
27. Weinhoffer SL, Guyer RD, Herbert M, Griffiths SL (1995) Intradiscal pressure measurements above an instrumented fusion. A cadaveric study. Spine 20: 526–531
28. Wilke HJ, Wolf S, Claes LE, Arand M, Wiesend A (1995) Stability increase of the lumbar spine with different muscle groups. A biomechanical in vitro study. Spine 20:192–198