Adjacent segmental degeneration following Wallis interspinous stabilization implantation

Biomechanical explanations and the value of magnetic resonance imaging

Zhiguo Zhou, MS\textsuperscript{a}, Wei Xiong, MD\textsuperscript{b}, Li Li, MS\textsuperscript{b,\#}, Feng Li, MD\textsuperscript{b,\#}

Abstract
Adjacent segmental degeneration (ASD) is a major issue after pedicular fixation. This study examined the degeneration of the adjacent levels due to the insertion of the Wallis interspinous stabilization system compared with discectomy, using magnetic resonance imaging (MRI).

Thirty-eight patients diagnosed with lumbar degeneration disorders at L4-L5 were reviewed: 19 patients underwent discectomy and Wallis system implantation (group A), and 19 patients underwent discectomy (group B). The Visual Analog Scale (VAS) and Oswestry Disability Index (ODI) were assessed preoperatively and postoperatively. ASD was evaluated by MRI.

There was no difference in the preoperative ODI scores between the 2 groups (non-normal distribution, median, 50 (40, 50) vs 50 (50, 50), \( P = .331 \)), but the postoperative ODI scores were different (non-normal distribution, median, 0 (0, 32) vs 20 (20, 30), \( P < .005 \)). Similar results were observed for VAS. In group A, ASD occurred in 4 patients (21.1\%) in the disc and 8 (42.1\%) in the facet joint at L3/4, and in 4 (21.1\%) in the disc and 5 (26.3\%) in the facet joint at L5/S1. In group B, ASD occurred in 3 patients (15.8\%) in the disc at L3/4, and in 4 (21.1\%) in the disc at L5/S1. In general, there was no difference between the 2 groups (\( P > .05 \), except at L3/4 (\( P = .015 \)). ASD of the facet joint in the cranial segment occurred after Wallis system implantation, suggesting that the Wallis system cannot prevent ASD of the facet joint, but could have some other benefits for the discs.

Abbreviations: ASD = adjacent segmental degeneration, BMI = body mass index, FOV = field of view, MRI = magnetic resonance imaging, ODI = Oswestry Disability Index, TE/TR = echo time and repetition time, VAS = visual analog scale.

Keywords: adjacent segmental degeneration, disc, facet joint, pedicular fixation, Wallis system implantation

1. Introduction
Acute or progressive disc lesions lead to instability of the spinal segments.\textsuperscript{[1,\#]} Currently, pedicular fixation (fusion) is the gold standard treatment in terms of increasing the biomechanical rigidity and clinical fusion rates because pedicle screws are the strongest component of spinal implants.\textsuperscript{[3]} Adjacent segment degeneration (ASD) is the development of a pathology at the mobile segment next to a lumbar or lumbosacral spinal fusion.\textsuperscript{[4]}

Several reports revealed that ASD could be accelerated due to the relative immobility of fused spinal segments transferring stress to adjacent segments after fusion.\textsuperscript{[5,7]} Symptoms and signs of ASD include pain, stenotic lesions, and instability, leading to additional surgeries such as extended fusion and neural decompression.\textsuperscript{[8]}

To reduce the incidence of fusion-related morbidity, non-fusion technologies have been developed, such as the Wallis interspinous stabilization system.\textsuperscript{[9]} Although the implant offers some advantages over fusion (e.g., motion of the involved levels and small operation wound), the efficacy of non-implants in the prevention of ASD is now well established.\textsuperscript{[1,\#]}

ASD was first described using x-ray indexes such as disc height and segmental range of motion,\textsuperscript{[10]} but a previous animal study suggested that the changes in x-ray indexes were less sensible than those extracted from magnetic resonance imaging (MRI),\textsuperscript{[11]} as supported by a study in humans.\textsuperscript{[12]}

Nevertheless, it is poorly known whether the use of the Wallis system could prevent ASD. Therefore, the aim of the present study was to compare the patients who underwent discectomy and Wallis system implantation with the patients who underwent discectomy only, based on MRI examinations.

2. Methods
2.1. Study design and patients
Patients diagnosed with lumbar disc herniation at L4-L5 and operated (by the same surgeon) at the Department of Orthopedic
Surgery, Tongji Hospital affiliated to Tongji Medical University of HUST, in 2009 and 2010, were retrospectively reviewed after a 2-year follow-up. The project was approved by the institutional review boards and the ethics committee of Tongji Hospital affiliated to Tongji Medical University of HUST and followed the tenants of the Declaration of Helsinki. The need for informed consent was waived by the committee because of the retrospective nature of the study.

The inclusion criteria were: (1) history of lumbar disc herniation; (2) symptoms of sciatic and low back pain; and (3) failure of conservative treatment. The exclusion criteria were: (1) any other type of vertebral fracture; (2) patients without any indication for surgery or refused surgery; (3) adjacent segments with disc degeneration grade >5 and/or facet degeneration grade >2 according to MRI (Table 1[13] and Table 2[14]); (4) history of cardiovascular or cerebrovascular diseases, trauma, or cancer; (5) lost to follow-up; or (6) missing data.

During the study period, 100 patients were treated at our center, but after excluding patients lost to follow-up and those with missing data, and after matching the 2 groups for age, gender, and occupation, only 38 patients remained.

### 2.2. Surgery

The treatment approach was decided by the surgeon in consultation with patients. After oral and written explanations on the details of the surgery, all participants signed a written surgical informed consent. After discussion, the patients understood the nature of the study.

The treatment approach was decided by the surgeon in consultation with patients. After oral and written explanations on the details of the surgery, all participants signed a written surgical informed consent. After discussion, the patients understood the nature of the study.

The indications for Wallis system implantation were: (1) the sequence was stable and (2) no complications.

### 2.3. Data collection

Age, gender, body mass index (BMI), and duration of pain were collected preoperatively. The intensity of pain according to the visual analog scale (VAS) and Oswetry disability index (ODI) were collected preoperatively and postoperatively. The VAS ranged from 0 (no pain) to 10 (worst pain imaginable). The patients were asked to mark a point on the scale corresponding to their pain at that time. The ODI questionnaire contained 6 statements (denoted levels 0–5) in each of the 10 sections related to impairments such as pain and abilities such as personal care, lifting, walking, sitting, standing, sleeping, sex life, social life, and traveling. In each section, the patient chose the statement that best described his/her status. If the limitation fell between 2 levels, the higher point value was selected. The chosen statements received scores 0 to 5 corresponding to the level indicated. The total scores could range from 0 (the highest level of function) to 50 (the lowest level of function).

### 2.4. MRI

All patients had undergone magnetic resonance imaging (MRI) before and 6 months after operation. The lumbar spine MRI examination of each participant was done by the same clinical 1.5T system (Signa 1.5 T HD, GE Healthcare, Waukesha, WI) using a 4-channel Phased Array CTL Spine Coil. T1-weighted fast spin-echo sagittal images with effective echo time and repetition times (TE/TR) of 10/400 ms, T2-weighted fast spin-echo sagittal images with TE/TR of 102/3000 ms and T2-weighted fast spin-echo axial images with TE/TR of 120/3000 ms were included in

### Table 1

| Grade | Signal from nucleus and inner fibers of annulus | Distinction between inner and outer fibers of annulus at posterior aspect of the disc | Height of the disc |
|-------|-----------------------------------------------|---------------------------------------------------------------------------------|-------------------|
| 1     | Uniformly hyperintense, equal to CSF           | Distinct                                                                         | Normal            |
| 2     | Hyperintense (post-sacral fat and < CSF)± hypointense intranuclear cleft | Distinct                                                                         | Normal            |
| 3     | Hyperintense through < post-sacral fat         | Distinct                                                                         | Normal            |
| 4     | Mildly hyperintense (slightly > outer fibers of annulus) | Indistinct                                                                       | Normal            |
| 5     | Hypointense (=outer fibers of annulus)         | Indistinct                                                                       | <30% reduction in disc height |
| 6     | Hyperintense                                  | Indistinct                                                                       | 30–60% reduction in disc height   |
| 7     | Hypointense                                   | Indistinct                                                                       | >60% reduction in disc height   |
| 8     | Hypointense                                   | Indistinct                                                                       |                   |

Grades 1, 2, and 3 are based on the signal intensity of the nucleus and inner fibers of annulus. For Grade 4, the margins between the inner and other fibers of the annulus at the posterior margin of the disc are indistinct. For Grade 5, the disc is uniformly hypointense, but there is no loss of disc space height. For Grades 6, 7, and 8, there is progressive loss of disc space height. These could be broadly classified as mild, moderate, to severe loss of disc space height. Very occasionally, although obvious disc collapse is present, the hyperintense signal from the nucleus and inner fibers of the annulus is present. This is referred to by a double entry, for example, 4/7, with the former reporting the disc signal and the latter the degree of collapse.

### Table 2

| Grade | Criteria                                                                                                                                 |
|-------|------------------------------------------------------------------------------------------------------------------------------------------|
| 1     | Uniformly thick cartilage covers the articular surfaces completely. Articular processes have a thin layer of cortical bone. No osteophyte. |
| 2     | Cartilage covers the entire surface of the articular processes but with erosion of the irregular region evident. Cortical bone of the articular processes is focally thickened. Possible or small osteophyte. |
| 3     | Cartilage incompletely covers the articular surfaces, with regions of the underlying bone exposed to the joint. Thickened cortical bone covers less than half of the articular processes. Definite and moderate osteophyte. |
| 4     | Cartilage is absent except for traces on the articular surfaces; dense cortical bone covers greater than half the articular process. Large osteophyte. |
the examination. The field of view (FOV) was 360 mm and the matrix was 128 x 128, whereas 5-mm sections with a 1-mm section gap was used. There were 6 averages and the echo train length was 72 seconds.

The visual grading of intervertebral disc degeneration and the facet joint degeneration were based on the T2-weighted images and adjacent levels. Two operators (8 and 5 years of experience in MRI of the spine, respectively) graded the disc and facet joint in L3/4, L4/5, and L5/S1. The G value, defined as a measure of segment (disc and facet joint) degeneration, was obtained by adding the grades of intervertebral disc degeneration (Table 1) and facet joint degeneration (Table 2). The difference in the G-value after surgery was defined as \( \Delta G = G_{\text{postoperational}} - G_{\text{preoperational}} \) of intervertebral discs and facet joints of L3/4, L4/5, and L5/S1. Positive \( \Delta G_{\text{disc}} \) and \( \Delta G_{\text{facet}} \) values indicate that the grade of the intervertebral discs and facet joints worsened after surgery and the segment was marked as ASD. Negative \( \Delta G_{\text{disc}} \) and \( \Delta G_{\text{facet}} \) values indicate that the grade improved after surgery. The interobserver reliability of image grading was assessed using the kappa score. The final results were determined according to the results by 1 neuroradiologist.

2.5. Statistical analysis

Interobserver analyses of all MRI measurements showed fair to excellent agreement. Changes in scores from before to after surgery were calculated. Normally distributed data are presented as mean ± standard deviation and were analyzed using the Student t test. Non-normally distributed data are presented as median (range) and analyzed using the Mann–Whitney U test. SPSS 23.0 (IBM, Armonk, NY) was used for statistical analysis. Two-sided P-values < .05 were considered statistically significant.

3. Results

Table 3 presents the characteristics of the patients. There were no differences in age, gender, BMI, and pain duration between the 2 groups (all \( P > .05 \)). The median preoperative ODI scores in groups A and B were 50 (40, 50) and 50 (50, 50) respectively (non-normal distribution; \( P = .331 \)). The postoperative ODI scores were 0 (0, 32) and 20 (20, 30), respectively (non-normal distribution; \( P < .005 \)). The median preoperative VAS scores in group A were 9 (9, 10) and 10 (9, 10) (non-normal distribution; \( P = .079 \)). The postoperative VAS scores were 0 (0, 6) and 2 (2, 4) (non-normal distribution; \( P = .067 \)).

3.1. Occurrence of ASD

For all patients (n = 38), ASD occurred in 7 patients (18.4%) in the disc and 8 (21.1%) in the facet joint at L3/4, and in 8 (21.1%) in the disc and 5 (13.2%) in the facet joint at L5/S1. For group A, ASD occurred in 4 patients (21.1%) in the disc and 8 (42.1%) in the facet joint at L3/4, and in 4 (21.1%) in the disc and 5 (26.3%) in the facet joint at L5/S1. For group B, ASD occurred in 3 patients (15.8%) in the disc at L3/4 and in 4 (21.1%) in the disc at L5/S1 (Table 4).

3.2. Changes in G value during follow-up

The comparison of the \( G_{\text{preoperational}} \), \( G_{\text{postoperational}} \), and \( \Delta G \) value of the discs and facets in the 2 groups are summarized in Table 5 and Fig. 1. There was no difference between the 2 groups for \( \Delta G_{\text{disc}} \) (\( P > .05 \)), but there was a difference for \( \Delta G_{\text{facet}} \) at L3/4 (\( P = .015 \)) but not at L5/S1 (\( P = .217 \)). In Fig. 2, the \( \Delta G_{\text{disc}} \) of the 2 groups were negative, and the changes in MRI were obvious. Detailed MRI examination of a patient from group B at the facet joints of L3/4, L4/5, and L5/S1 is shown in Fig. 3. Preoperatively, cartilage covers the surfaces of the articular processes with some erosion; the cortical bone of the articular processes is focally thickened with small/moderate osteophyte. After operation, regions of the underlying bone are exposed to the joint, with moderate/large osteophyte.

4. Discussion

ASD after lumbar spinal fusion is a potential cause of further spinal surgery, which is disquieting to both patients and surgeons. The Wallis system can be used to stabilize the spine, but its effect on ASD is unknown. Therefore, the aim of the present study was to examine the degeneration of the adjacent levels due to the insertion of the Wallis interspinous stabilization system compared with discectomy, and using MRI. The results showed that in group A, ASD occurred in 4 patients (21.1%) in the disc and 8 (42.1%) in the facet joint at L3/4, and in 4 (21.1%) in the disc and

### Table 3
Characteristics of the patients.

| Data                        | A     | B     | P     |
|-----------------------------|-------|-------|-------|
| N                           | 19    | 19    | –     |
| Gender                      | Male  | 11    | 10    | 1.00  |
| Female                      | 8     | 9     |       |
| Age, years                  | 47.5±13.7 | 47.3±13.2 | .96  |
| BMI, kg/m²                  | 22.6±1.9 | 22.5±1.8 | .87  |
| Duration of pain            | 56 m, 2 weeks-17 years | 37 m, 2 weeks-10 years |       |
| Preoperative ODI*           | 50 (40, 50) | 50 (50, 50) | .331 |
| Postoperative ODI*          | 0 (0, 32) | 20 (20, 30) | < .005 |
| Preoperative VAS*           | 9 (9, 10) | 10 (9, 10) | .08  |
| Postoperative VAS*          | 0 (0, 6) | 2 (2, 4) | .07  |

BMI = body mass index, ODI = Oswestry disability index, VAS = visual analog scale.

### Table 4
Occurrence of ASD in the 2 groups.

|        | A     | B     | P     |
|--------|-------|-------|-------|
| Disc L3/4 | 4     | 3     | .484  |
| Disc L5/S1| 4     | 4     | .869  |
| Facet joint L3/4 | 8     | 0     | .015  |
| Facet joint L5/S1| 5     | 0     | .217  |

### Table 5
Comparison of the \( G_{\text{preoperational}}, G_{\text{postoperational}}, \) and \( \Delta G \) value of the discs and facets in the 2 groups.

| Level (P-values) | L3/4 | L5/S1 |
|------------------|------|-------|
| \( G_{\text{pre-disc}} \) | .137 | .079  |
| \( G_{\text{post-disc}} \) | .530 | .238  |
| \( \Delta G_{\text{disc}} \) | .484 | .869  |
| \( G_{\text{pre-facet}} \) | .693 | .289  |
| \( G_{\text{post-facet}} \) | .034* | .050  |
| \( \Delta G_{\text{facet}} \) | .015* | .217  |

* \( P < .05 \) was considered to be statistically significant.

The \( G \) value is obtained by adding the disc degeneration grade (Table 1) to the facet degeneration grade (Table 2), as assessed by 2 radiologists.

---

**Note:**

- **Table 3** presents the characteristics of the patients. There were no differences in age, gender, BMI, and pain duration between the 2 groups (all \( P > .05 \)). The median preoperative ODI scores in groups A and B were 50 (40, 50) and 50 (50, 50), respectively (non-normal distribution; \( P = .331 \)). The postoperative ODI scores were 0 (0, 32) and 20 (20, 30), respectively (non-normal distribution; \( P < .005 \)). The median preoperative VAS scores in group A were 9 (9, 10) and 10 (9, 10) (non-normal distribution; \( P = .079 \)). The postoperative VAS scores were 0 (0, 6) and 2 (2, 4) (non-normal distribution; \( P = .067 \)).

- **Table 4** shows the occurrence of ASD in the 2 groups. There were no differences in ASD between the 2 groups (all \( P > .05 \)). For group A, ASD occurred in 4 patients (21.1%) in the disc and 8 (42.1%) in the facet joint at L3/4, and in 4 (21.1%) in the disc and 5 (26.3%) in the facet joint at L5/S1. For group B, ASD occurred in 3 patients (15.8%) in the disc at L3/4 and in 4 (21.1%) in the disc at L5/S1.

- **Table 5** compares the changes in \( G \) value during follow-up. The comparison of the \( G_{\text{preoperational}}, G_{\text{postoperational}}, \) and \( \Delta G \) value of the discs and facets in the 2 groups are summarized in Table 5 and Fig. 1. There was no difference between the 2 groups for \( \Delta G_{\text{disc}} \) (\( P > .05 \)), but there was a difference for \( \Delta G_{\text{facet}} \) at L3/4 (\( P = .015 \)) but not at L5/S1 (\( P = .217 \)). In Fig. 2, the \( \Delta G_{\text{disc}} \) of the 2 groups were negative, and the changes in MRI were obvious.

- **Discussion:** ASD after lumbar spinal fusion is a potential cause of further spinal surgery, which is disquieting to both patients and surgeons. The Wallis system can be used to stabilize the spine, but its effect on ASD is unknown. Therefore, the aim of the present study was to examine the degeneration of the adjacent levels due to the insertion of the Wallis interspinous stabilization system compared with discectomy, and using MRI. The results showed that in group A, ASD occurred in 4 patients (21.1%) in the disc and 8 (42.1%) in the facet joint at L3/4, and in 4 (21.1%) in the disc and
In the cranial segment, ASD occurred in 3 patients (15.8%) in the disc at L3/4, and in 4 (21.1%) in the disc at L5/S1. In general, there was no difference between the 2 groups ($P > .05$), except at L3/4 ($P = .015$). Therefore, ASD of the facet joint in the cranial segment occurred after Wallis system implantation, suggesting that the Wallis system cannot prevent ASD of the facet joint, but could have some other benefits for the discs, highlighted by the significantly lower ODI scores in group A compared to group B.

Biomechanical changes of ASD consist of increased intradisc pressure, increased facet load, and increased mobility after fusion. X-ray indexes such as disc height and segmental range of motion can describe ASD to some degree, but MRI indexes provide more reliable data. However, fusion surgery may cause artifacts with imaging. From the results of the present study, it seems that ASD occurs above the operated segment after implantation of the Wallis system, especially at the facet joint. Based on several studies, after spinal fusion, increased stress on the adjacent facet joints and a change in the load of the adjacent disc have been proved. In the studies of spinal fusion, several authors support the point of view that the load is shifted to the free and mobile cranial lumbar segments for compensation. Therefore, ASD always occurred in the facet joints above the reconstructed segment. Akamaru et al demonstrated that the highest increase in motion is the cranial segment (L3/4) to L4/5 after its hypolordotic floating fusion. In addition, the change in joint orientation is a major risk factor in the degenerative process of that segment. The Wallis implants can restrict the motion...
of the lumbar spine. The Wallis implant consists of an interspinous spacer that limits the extension and 2 bands that secure the implant in the interspinous space and limit flexion.[9,10] Therefore, the motion and the load is shifted from L4/5 to the adjacent segments (L3/4 and L5/S1) after Wallis system implantation at L4/5, especially at the cranial segment (L3/4). The reason for ASD at the L5/S1 facet in this study could be due to damage to the posterior structure resulting from the implantation, but this requires further investigation.

In some studies, the intradisc pressure was strongly reduced in extension after the implantation of the Wallis system,[19] but without difference in all other loading directions (flexion, lateral bending, and axial rotation), which has been observed in the present study. Nevertheless, the use of an interspinous implant could cause adjacent level facet pain or accelerated facet joint degeneration.[19] At the implanted level, the mean peak pressure, average pressure, contact area, and force were significantly reduced, but there were no significant changes at the level above the implant. The implant appears to redirect a large portion of the load away from the intervertebral disc and to transfer that load to the spinous processes. In a study by Adams et al.[19] there was a paradoxical decrease in posterior annular pressure during hyperextension at the tested level. They attributed this observation to the facet joints acting as a fulcrum and redirecting most of the force from the respective disc. When using the Wallis system, the lumbar spine is kept slightly flexed, meaning that the anterior part of the intervertebral disc is compressed, keeping the articular facets separated during movement of the lumbar spine.[21] As superior-segment facet contact has been presumed to play a role in the onset of ASD, it is unclear why the Wallis system does not prevent ASD. Nevertheless, additional mechanical studies are necessary to characterize the spinal changes leading to ASD. Unfortunately, there is currently no relevant literature about the prevention of ASD and the present study does not allow drawing conclusions about ASD prevention. Additional studies are also necessary to address these issues.

The present study is not without limitations. The sample size was small, from a single center, and was operated by a single surgeon. The ODI scores were self-assessed and could be more severe than in reality. No patient with pedicular fixation (fusion) could be included as controls because the fixation affected MRI quality. Finally, the follow-up was short and was based on retrospective data.

In conclusion, ASD of the facet joint in the cranial segment occurred after Wallis system implantation, suggesting that the Wallis system cannot prevent ASD of the facet joint, but could have some benefits for the discs.

**Acknowledgments**

The authors thank Dr. Yuwei Liu for his help in data analysis, as well as Dr. Lingyun Zhao and Dr. Liang Qi for their help in dealing with the MRI and picture reporting in this study.

![Figure 3](image-url)
References

[1] Ebara S, Harada T, Hosono N, et al. Intraoperative measurement of lumbar spinal instability. Spine (Phila Pa 1976) 1992;17:544–50.

[2] Mimura M, Panjabi MM, Oxland TR, et al. Disc degeneration affects the multidirectional flexibility of the lumbar spine. Spine (Phila Pa 1976) 1994;19:1371–80.

[3] Erbulut DU, Kiapour A, Oktenoglu T, et al. A computational biomechanical investigation of posterior dynamic instrumentation: combination of dynamic rod and hinged (dynamic) screw. J Biomech Eng 2014;136:031007.

[4] Park P, Garton HJ, Gala VC, et al. Adjacent segment disease after lumbar or lumbosacral fusion: review of the literature. Spine (Phila Pa 1976) 2004;29:1938–44.

[5] Epstein NE. Older literature review of increased risk of adjacent segment degeneration with instrumented lumbar fusions. Surg Neurol Int 2016;7: S70–6.

[6] Lee CS, Hwang CJ, Lee SW, et al. Risk factors for adjacent segment disease after lumbar fusion. Eur Spine J 2009;18:1637–43.

[7] Lee JC, Choo SW. Adjacent segment pathology after lumbar spinal fusion. Asian Spine J 2015;9:807–17.

[8] Hwang DH, Cho YJ, Cho SM, et al. Adjacent segment degeneration after lumbar dynamic stabilization using pedicle screws and a nitinol spring rod system with 2-year minimum follow-up. J Spinal Disord Tech 2012;25:409–14.

[9] Sandu N, Schaller B, Arasho B, et al. Wallis interspinous implantation to treat degenerative spinal disease: description of the method and case series. Expert Rev Neurother 2011;11:799–807.

[10] Korovessis P, Re帕ntis T, Zacharatos S, et al. Does Wallis implant reduce adjacent segment degeneration above lumbosacral instrumented fusion? Eur Spine J 2009;18:830–40.

[11] Sobajima S, Kompel JF, Kim JS, et al. A slowly progressive and reproducible animal model of intervertebral disc degeneration characterized by MRI, X-ray, and histology. Spine (Phila Pa 1976) 2005;30:15–24.

[12] Siepe CJ, Zelenkov P, Sauri-Barraza JC, et al. The fate of facet joint and adjacent level disc degeneration following total lumbar disc replacement: a prospective clinical, X-ray, and magnetic resonance imaging investigation. Spine (Phila Pa 1976) 2010;35:1991–2003.

[13] Griffith JF, Wang YX, Antonio GE, et al. Modified Pfirrmann grading system for lumbar intervertebral disc degeneration. Spine (Phila Pa 1976) 2007;32:E708–12.

[14] Grogan J, Nowicki BH, Schmid TA, et al. Lumbar facet joint tropism does not accelerate degeneration of the facet joints. AJNR Am J Neuroradiol 1997;18:1325–9.

[15] Min JH, Jang JS, Jung B, et al. The clinical characteristics and risk factors for the adjacent segment degeneration in instrumented lumbar fusion. J Spinal Disord Tech 2008;21:305–9.

[16] Yang JY, Lee JK, Song HS. The impact of adjacent segment degeneration on the clinical outcome after lumbar spinal fusion. Spine (Phila Pa 1976) 2008;33:503–7.

[17] Disch AC, Schmoelz W, Matziolis G, et al. Higher risk of adjacent segment degeneration after floating fusions: long-term outcome after low lumbar spine fusions. J Spinal Disord Tech 2008;21:79–85.

[18] Akamaru T, Kawahara N, Tim Yoon S, et al. Adjacent segment motion after a simulated lumbar fusion in different sagittal alignments: a biomechanical analysis. Spine (Phila Pa 1976) 2007;32:1360–6.

[19] Parchi PD, Evangelisti G, Verrucchio A, et al. Biomechanics of interspinous devices. Biomed Res Int 2014;2014:839325.

[20] Adams MA, May S, Freeman BJ, et al. Effects of backward bending on lumbar intervertebral discs. Relevance to physical therapy treatments for low back pain. Spine (Phila Pa 1976) 2000;25:431–7, discussion 438.

[21] Lai PL, Chen LH, Niu CC, et al. Relation between laminectomy and development of adjacent segment instability after lumbar fusion with pedicle fixation. Spine (Phila Pa 1976) 2004;29:2527–32. discussion 2532.