Relation of the Development of Adjacent Segment Degeneration after Two Levels Posterolateral Fusion for Degenerative Lumbar Instability with Preoperative Facet Tropism and Sagittal Alignment at Full Fusion

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ABSTRACT

BACKGROUND: After lumbar spinal fusion, adjacent segment degeneration (ASD) is a concern to both patients and surgeons and is a potential cause of further spinal surgery. Although ASD may be considered as a part of the normal aging process and degenerative change, it could be influenced by changes in the stress acting on the adjacent segment after spinal fusion. There are confusing reports in literature on whether ASD development affects the patients’ outcome, in terms of changing his clinical status to the worse or not. No enough studies has correlated the development of ASD especially the symptomatic cases and postoperative sagittal alignment and the presence of preoperative facet tropism.

The authors hypothesized that mal-alignment of the sagittal balance after posterior spinal fusion at least increases (if not causes) the phenomenon of ASD development and progress. Also the authors hypothesized that facet tropism may play a role in the development and/or advancement of ASD.

PATIENTS AND METHODS: This prospective study was run in Ain Shams University hospitals and hospitals of ministry of health in Cairo from April, 2004 till January, 2008.

We had 53 patients (39 females and 14 males with ratio of 2.8:1). All were operated upon for degenerative indication and were selected according to strict inclusion criteria and all were fully fused by April of 2005. Range of follow up was 30-40 months with mean of 33 months but we considered the 30 months follow up visit as the final follow up.

Patients were categorized into group A with no facet tropism and B with facet tropism of the levels intended for fusion and their adjacent segments. Every group was subcategorized according to sagittal alignment at full fusion (measured using Cobb method) into group 1 with normal lordosis angle of 20-65 degrees and group 2 with hypolordotic alignment and group 3 with hyperlordotic alignment.

Patients were assessed clinically according to modified functional scale of Ghiselli et al and radiographically by AP and lateral plain films and dynamic laterals in the post fusion visits to assess ASD signs in the adjacent segments above and below fusion. The changes were graded according to University of California at Los Angeles Grading Scale for Intervertebral Space Degeneration. MRI was added for patients with symptomatic ASD and was done for all patients at the final follow up.

RESULTS: The results of this work prove that the incidence of asymptomatic ASD at 30 months follow up was only 5.2% and the symptomatic ASD was only 2.4% for the directly adjacent segment above fusion at 30 months follow up for group A1 patients (0.96 % per year) and this is far less than the recorded symptomatic ASD in most series that amounts to 3 % per year of follow up so it is 7.5% for that length of follow up.
INTRODUCTION

The prevalence of lumbar arthrodesis has continued to increase because of the emergence of newer techniques of spinal instrumentation and improved imaging modalities that allow for accurate recognition of spinal abnormalities. The levels involved in the arthrodesis typically are degenerative or unstable, and the ultimate goals are to provide relief of symptoms and to restore stability (12).

After lumbar spinal fusion, adjacent segment degeneration (ASD) is a concern to both patients and surgeons and is a potential cause of further spinal surgery. Although ASD may be considered as a part of the normal aging process and degenerative change, it could be influenced by changes in the stress acting on the adjacent segment after spinal fusion (5,13,20,27,30).

There are many reports that ASD is accelerated after lumbar spinal fusion(2,10,18,26,30). It is not clear whether ASD is a complication resulting from the increased activity and improved symptoms after lumbar fusion or part of the natural process of disc degeneration. Nevertheless, after lumbar fusion, an increase in stress, excess mobility, increased intra-disc pressure, and posterior displacement of the axis of motion have been observed in the adjacent segments (7,16,29,30). At the least, lumbar fusion is thought to accelerate ASD.

There are confusing reports in literature on whether ASD development affects the patients’ outcome, in terms of changing his clinical status to the worse or not.

Moskowitz et al (22) followed 61 patients for up to 26 years and reported that there was no correlation between back pain and the number of segments fused. Gelalis and Kang (11) in 1998 found no correlation between the clinical outcome and spinal fusion, even with nonunion or instability.

Pallise et al in 2007(24) analyzed long term radiographic changes in all un-fused lumbar segments after instrumented posterolateral lumbar fusion by studying 212 un-fused segments in 62 patients of an average follow up of 7.5 years post fusion and found that no changes were observed at the segments located below the fusion and that all the un-fused segments above the fusion showed the same significant loss of disc height and that the loss of disc height did not depend on fusion parameters, and concluded that after posterior lumbar instrumented fusion, radiographic changes suggesting disc degeneration appear homogenously at several levels cephalad to fusion and seem to be determined by individual characteristics(24).

In group A2 with hypolordotic alignment there was 50% incidence of asymptomatic ASD in the directly adjacent segment above fusion and 50% incidence of symptomatic ASD in the directly adjacent segment above so actually all the segments directly above fusion got ASD in this group.

In group A3 all the levels directly below fusion showed ASD (66.7% Asymptomatic and 33.3 symptomatic) and all are in the directly adjacent segment below and that proves that hyperlordosis puts extra-demand on the adjacent segment below fusion.

In group B1 of facet tropism even with the preservation of physiological lordosis we had all levels directly above fusion levels showing ASD at the 30 months follow up (80% asymptomatic and 20% symptomatic) which is significantly different than the same alignment group with no facet tropism in our series(5.2% and 2.4% respectively) B2 and B3 groups patient population were insufficient for proper result analysis.

CONCLUSION: The results of this work prove that keeping lordotic alignment performing lumbar fusion for degenerative diseases within physiological ranges decreased the incidence of both symptomatic and non symptomatic ASD and that the disturbance of this alignment increased the incidence of symptomatic and non symptomatic ASD in the segments directly above fusion in hypolordotic alignment and in the segment directly below in hyperlordotic alignment and thus keeping physiologic lordosis plays detrimental role in decreasing the incidence of ASD and that this should be taken care of during surgery.

Also the authors concluded that patients with facet tropism are more likely to develop ASD than those with no tropism so those patients should be informed of this possibility and that they are more likely to need treatment that could be surgical for that condition in a while after fusion surgery. More patient population and longer follow up are needed to further solidify the concluded facts.

KEY WORDS: Adjacent segment, Facet tropism, Fusion, Sagittal alignment.
Axelsson et al in 2007 thus concluded that the significance of mechanical alterations in adjacent segment degeneration is uncertain and is possibly overestimated(3).

Conversely, Etebar and Cahill(9) reviewed 125 patients who underwent instrumented lumbar fusion with an average of 4.5 years of follow-up and found that 14% of the patients developed symptomatic disease related to ASD.

Jun Young Yang et al in 2008 in a retrospective study on 217 patients who underwent lumbar fusion and have more than 2 years of follow-up, found that the impact of ASD on clinical outcome after fusion showed a significant correlation, especially with the ASD after multiple-segments fusion(3).

Again the rate by which ASD develop after spinal fusion especially the symptomatic cases was never well studied until Ghiselli et al in 2004 did so and found that the rate of symptomatic degeneration at an adjacent segment warranting either decompression or arthrodesis was predicted to be 16.5% at five years and 36.1% at ten years and found that there appeared to be no correlation with the length of fusion or the preoperative arthritic degeneration of the adjacent segment(12).

In general, the causes, incidence, and risk factors of ASD remain topics of study. In addition, it is not clear whether there is a correlation between ASD and the clinical outcome.

Reviewing the literature the authors found no enough studies that correlated the development of ASD especially the symptomatic cases and postoperative sagittal alignment and the presence of preoperative facet tropism although we view them as possible contributing factors to the development of symptomatic ASD and may be responsible on the discrepancy of the results of studies on ASD in literature.

Akamaru et al in 2003 in a bimechanical study of adjacent segment motion at L3-L4 and L5-S1 after simulated lumbar interbody fusion of L4-5 in different sagittal alignments found that hypolordotic alignment at L4-L5 caused the greatest amount of flexion-extension motion at L3-L4 level and that the differences were statistically significant in comparison with intact specimen, in situ fixation, and hyperlordotic fixation. Hyperlordotic alignment at L4-L5 caused the greatest amount of flexion-extension motion at L5-S1 level and the difference was statistically significant in comparison with intact specimen but not in situ fixation or hypolordotic fixation(1).

The results of this study were the basis by which we hypothesized that mal-alignment of the sagittal balance after posterior spinal fusion at least increases (If not causes) the phenomenon of ASD development and progress and that neglecting this point in most of the studies of ASD phenomenon was the cause of most of the discrepancy between the radiological findings and the clinical outcome for the patients of these series.

Park et al in 2007 evaluated the correlation between ASD and the pelvic parameters in patients with spondylotic spondylolisthesis and found that the development of ASD was closely related to post operative pelvic incidence and pelvic tilt angle and that the measurement of postoperative pelvic incidence angle can be used as a new indirect method to predict ASD(25).

Okuda et al in 2004 in a retrospective study of 87 patients who underwent posterior lumbar inter-body fusion at L4-5 level for degenerative spondylolisthesis found no correlation between radiological degeneration of cranial adjacent segment and clinical results and that the coexistence of horizontalization of the lamina and facet tropism at L3-4 level may be one of the risk factors for neurological deterioration resulting from accelerated L3-L4 degenerative changes after L4-L5 PLIF (23).

This study was one of the factors that made us hypothesize that facet tropism may play a role in the development and/or advancement of ASD in those patients and we designed our study as to try to verify the truth about this finding.

Facet tropism describes asymmetry of the facet joint angles of the lumbar or lumbosacral region, It has been suggested as being one of the possible causes of herniation of the lumbar disc(17).Facet tropism can be assessed by CT or MRI.

**Aim of the study**

To study the Relation between the development of adjacent segment degeneration [symptomatic and asymptomatic] after two levels postero-lateral fusion for degenerative lumbar instability and preoperative facet tropism and sagittal alignment at full fusion

**PATIENTS AND METHODS**

This prospective study was run in Ain Shams University hospitals and hospitals of ministry of health in Cairo from April, 2004 till January, 2008.
The study was designed to include all cases managed by the surgeon authors indicated for two levels lumbar fusion for degenerative indications according to strict inclusion criteria and not including any case that needs lumbo-sacral fusion so that we can assess the effect of fusion on at least one level above and below fusion level.

**Our indications for surgery were:**

1. Progressive neurogenic claudications, not relieved by NSAIDs or physical therapy with interference with activities of daily living, with radiological evidence of double level instability and at least single level compression of neurological structures.

2. Intractable back pain not relieved by NSAIDs or physical therapy with the same findings on MRI and plain x-rays as the previous indication.

3. Progressive neurological deficit with the same MRI and plain X-ray findings as the previous indication.

**Our exclusion criteria were:** Failed back surgery, compression or instability at the level of lumbosacral junction, the presence of spinal deformity above the levels intended for decompression and fixation and degenerated adjacent disc levels.

All patients had preoperative plain X-rays (A-P and lateral films on neutral position, and on flexion and extension), MRI.

We operated upon 66 cases with these criteria between April 2004, and November 2004.

**Surgical procedure**

All cases had two levels decompression, titanium pedicle screw fixation and grafting in order to achieve the goal of inter-transverse fusion.

We followed these cases till April 2005 and only the cases that went into complete fusion on follow up x-rays till this date were included in the study till the final follow up. Only 53 cases had complete fusion at that date and these cases were considered as our patient population during our study.

Of these 53 patients we had 39 females and 14 males with ratio of 2.8:1.

**Radiological measurement of facet joint tropism and sagittal alignment**

We used the method described by Karacan et al(19) in measuring facet tropism. Axial scans were aligned parallel to the vertebral end plate using bone window in CT scan. By MRI facet joint angle was measured on the axial T1-or T2-weighted images. The right and left facet joint angles are measured; a line was drawn between the two margins of each of the superior articular facets. A mid sagittal line is defined as a line passing through the centre of the disc and the centre of the base of the spinous process. The angle between the facet line and the mid sagittal line is measured for each side of the spine and the facet joint tropism is the difference of the angles between the right and left facet (Figure 1).

We measured the extent of lumbar lordosis using Cobb method(4). The lordosis angle at the meeting points of two lines, one perpendicular to the line that passes through L1 superior endplate and the other one passes through superior end plate of S1 (Figure 2) the normal range of this angle ranges from 20-650(25,26).

We categorized these patients into two major subgroups according to the presence or absence of facet tropism on preoperative CT and/or MRI:

**Group A** : 46 patients. With no evident facet tropism.

**Group B** : 7 patients. With evident facet tropism.

Figure 1: The mid-sagittal line passed through the centre of the disc and the centre of the base of the spinous process. The angle between the facet line and the mid-sagittal line was measured for each side of the spine. The difference of the right and left facet angles (a-b) of each patient was then calculated(19).
each visit full clinical assessment was done and we evaluated the clinical condition for our patients according to a modified functional scale of Ghiselli et al. (12) (Table 1).

We considered degradation of patient’s clinical assessment by even one grade e.g from excellent to good grade during the follow ups associated by radiological signs of ASD to be symptomatic ASD and if the radiological signs are not accompanied by such a degradation we considered this as asymptomatic ASD keeping in mind that non of the patients had any ASD prior to surgery.

We performed plain x rays antero-posterior and lateral for each patient in all the visits and flexion extension films in all post fusion visits. In the lateral view projection the patient was standing, since when the patient is lying on his side the lumbar spine is much straighter especially in its upper three segments(4).

Lateral projections in flexion and extension to demonstrate mobility and stability were done in post fusion visits. The patient sits on a backless seat with either side against the vertical bucky , the patient first lean forwards flexing the lumbar region as far as possible, and grips the front of the seat to assist in maintaining the position. The patient then lean backward, extending the lumbar region as far as possible and grips the back of the seat. (28). In these films we looked for: Change in disc space height above and below the level of fusion. Disc height ratio is expressed as a percentage ratio of the height of the posterior border of the vertebra and inter-vertebral height (Figure 3)(21).

We also assessed the development of static or dynamic alignment change in the levels around fusion as  retrolisthesis or spondylolisthesis. The degree of vertebral slip was expressed as a percentage ratio of the height of the posterior border of the vertebra and inter-vertebral height (Figure 3)(21).

We also looked for osteophytes and end plate sclerosis to asses the presence and grade of ASD if any. The changes were graded according to University of California at Los Angeles Grading Scale for Intervertebral Space Degeneration (Table 2)(12).

The patients were classified according to their lordotic angle at full fusion into the following subgroups:

**Group A1**: with preserved or restored lumbar lordosis (angle range 20-65): 41 patients. These were classified according to the operated levels into 33 cases with L3-4 and L4-5 fusion, and 8 cases with L2-3, L3-4 fusion.

**Group A2**: with postoperative hypolordosis or kyphosis: two patients, both are L3-4 and L4-5 fusion.

**Group A3**: with postoperative hyperlordosis: three patients (Two L3-4 and L4-5 fusion and one L2-3 and L3-4 fusion)

**Group B1**: with preserved or restored lumbar lordosis: 5 patients

**Group B2**: with postoperative hypolordosis or kyphosis: one patient

**Group B3**: with postoperative hyperlordosis: one patient

All patients of this group were L3-4 and L4-5 fusion.

The noted tropism was at L2-L3 level in all the cases

The mean follow up period was 33 months (Range of follow up 30-40 months), and the 30 months follow up visit was considered to be the final follow up.

The follow up schedule was every 3 months for the first 9 months and then every 6 months for the next 21 months. In

![Figure 2: Lumbar lordosis angle (L1S1) based on the Cobb method](14,15).
In the MRI we compared the disc condition above and below fusion levels with the preoperative MRI assessment of disc degeneration and its sequelae, facet joint arthritis, spinal canal stenosis and malalignment and instability. MRI studies were performed by a 1.5 tesla MRI machine. A coronal localizer was performed for the lumbar region then the needed sections were installed. The image sequences obtained were as follows, sagittal T1 and T2 weighted images, axial T1&T2 weighted images. Post contrast gadolinium injection in some situations.

Midsagittal T2-weighted images were used to evaluate the degree of disc degeneration according to T2 signal intensity on a 5 point grading scale(8), namely: 0, very intense: 1, intense, 2, moderate, 3 slight, 4, none.

### RESULTS

Our results are summarized in the following tables(3-8.)

We have to note to the fact that none of our patients with symptomatic ASD needed re-operation till 30 months final follow up. Medical treatment and physical therapy was the mode of their treatment and none showed an indication for decompression and/or fusion for adjacent levels according to our indications in the inclusion criteria. Figure 4 and 5 demonstrate two illustrative cases.

### DISCUSSION

The results of this work as shown in (Table 3) proves that the incidence of asymptomatic ASD at 30 months follow up was only 5.2% and the symptomatic ASD was only 2.4% for

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**Table 1: Criteria for the Assessment of Clinical Outcome(1).**

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Pain</th>
<th>Medication</th>
<th>Activity</th>
<th>Work status</th>
</tr>
</thead>
<tbody>
<tr>
<td>Excellent</td>
<td>None except for occasional back pain</td>
<td>None</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Good</td>
<td>Markedly improved, occasional pain</td>
<td>Occasional use of pain medication</td>
<td>Minimal functional limitations</td>
<td>Return to work, although not at the same job activity</td>
</tr>
<tr>
<td>Fair</td>
<td>Some improvement</td>
<td>Frequent use of pain medication</td>
<td>Restricted</td>
<td>Limited</td>
</tr>
<tr>
<td>Poor</td>
<td>No change in symptoms or a worsening of the patient’s condition</td>
<td>Oral use of narcotics</td>
<td>Incapacitated</td>
<td>Disabled</td>
</tr>
</tbody>
</table>

**Table 2: University of California at Los Angeles Grading Scale for Intervertebral Space Degeneration(1).**

<table>
<thead>
<tr>
<th>Grade</th>
<th>Disc-Space Narrowing</th>
<th>Osteophytes</th>
<th>End Plate Sclerosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>II</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>III</td>
<td>±</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>IV</td>
<td>± ±</td>
<td>±</td>
<td>+</td>
</tr>
</tbody>
</table>

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**Figure 3:** Measurement of disc height ratio, Disc height ratio=b/a. a=the height of the posterior border of the vertebra, b=intervertebral height (21)
the directly adjacent segments at 30 months follow up for group A1 patients (0.96 % per year) and this is far less than the recorded symptomatic ASD in most series that amounts to 3% per year of follow up so it is 7.5% for that length of follow up in most series(12).

That proves that keeping anatomical alignment (Lordosis at the range of 20-65 degrees) decreased the incidence of ASD both symptomatic and asymptomatic significantly. Also this alignment kept the next adjacent segments free of disease.

No ASD is recorded below the fusion level in this group.

In group A2 (Table 4) with hypolordotic alignment there was 50% incidence of asymptomatic ASD in the directly adjacent segment above fusion and 50% incidence of symptomatic ASD in the directly adjacent segment above so actually all the segments directly above fusion got ASD in this group. This shows a significant difference to ASD rates in most series in literature (7.5% symptomatic ASD for this length of follow up) and this shows that the hypolordotic posture has put severe working load on the segment above and this is in line with the study of Akamaru et al in 2003(19) that showed that hypolordotic posture caused the greatest amount of flexion and extension motion at the immediate adjacent segment above the fused segment shown on a biomechanical cadaveric model. This was also shown to be true in the work of Chen et al in 2004(6) by biomechanically testing posteriorly instrumented lumbar specimens.

Again no ASD was found in the segments below fusion in this group.

In group A3 (Table 5) all the levels directly below fusion showed ASD (66.7% Asymptomatic and 33.3 symptomatic) and all are in the directly adjacent segment below and that proves that hyperlordosis puts extra-demand on the adjacent segment below fusion in consistency with the study of Akamaru et al in 2003(1) that showed that hyperlordotic alignment caused the greatest amount of motion at the directly adjacent segment below fusion in a cadaveric lumbar spine fusion model. Non of the previous studies to our knowledge has studied the effect of hyperlordosis on the development and progress of ASD in the directly adjacent distal segment to the fused levels. No ASD was found in segments above fusion level in this group.
Table 3: A1 group Relative Risk of New Disease Developing at an Adjacent Vertebral Level.

<table>
<thead>
<tr>
<th>Vertebral Level</th>
<th>Directly Adjacent Above (D AD. AB)</th>
<th>Directly adjacent Below (D AD. BL)</th>
<th>Next Adjacent Above (N Ad. Ab.)</th>
<th>Next Adjacent below (N Ad. BL)</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total no. of discs at risk</td>
<td>41</td>
<td>41</td>
<td>41</td>
<td>8</td>
<td>131</td>
</tr>
<tr>
<td>No. of discs with new asymptomatic degeneration</td>
<td>2 (All Grade II)</td>
<td>0</td>
<td>2 (All Grade II)</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>Percentage (%)</td>
<td>4.9</td>
<td></td>
<td>4.9</td>
<td></td>
<td>5.24</td>
</tr>
<tr>
<td>No. of discs with new (Symptomatic) disease</td>
<td>1 (Grade III)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Percentage (%)</td>
<td>2.4</td>
<td></td>
<td></td>
<td></td>
<td>1.31</td>
</tr>
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</table>

Table 4: A2 group Relative Risk of New Disease Developing at an Adjacent Vertebral Level.

<table>
<thead>
<tr>
<th>Vertebral Level</th>
<th>Directly Adjacent Above (D AD. AB)</th>
<th>Directly adjacent Below (D AD. BL)</th>
<th>Next Adjacent Above (N Ad. Ab.)</th>
<th>Next Adjacent below (N Ad. BL)</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total no. of discs at risk</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>0</td>
<td>6</td>
</tr>
<tr>
<td>No. of discs with new asymptomatic degeneration</td>
<td>1 (All Grade II)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Percentage (%)</td>
<td>50</td>
<td></td>
<td></td>
<td></td>
<td>16.6</td>
</tr>
<tr>
<td>No. of discs with new (Symptomatic) disease</td>
<td>1 (Grade III)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Percentage (%)</td>
<td>50</td>
<td></td>
<td></td>
<td></td>
<td>16.6</td>
</tr>
</tbody>
</table>

Table 5: A3 group Relative Risk of New Disease Developing at an Adjacent Vertebral Level.

<table>
<thead>
<tr>
<th>Vertebral Level</th>
<th>Directly Adjacent Above (D AD. AB)</th>
<th>Directly adjacent Below (D AD. BL)</th>
<th>Next Adjacent Above (N Ad. Ab.)</th>
<th>Next Adjacent below (N Ad. BL)</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total no. of discs at risk</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>1</td>
<td>10</td>
</tr>
<tr>
<td>No. of discs with new asymptomatic degeneration</td>
<td>0</td>
<td>2 (All Grade II)</td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Percentage (%)</td>
<td>66.7</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of discs with new (Symptomatic) disease</td>
<td>0</td>
<td>1 (Grade III)</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Percentage (%)</td>
<td>33.3</td>
<td></td>
<td></td>
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In group B1 (Table 6) of facet tropism even with the preservation of physiological lordosis we had all levels directly above fusion levels showing ASD at the 30 months follow up (80% asymptomatic and 20% symptomatic) which is significantly different than the same alignment group with no facet tropism in our series (5.2% and 2.4% respectively) and this fact in addition to the fact that also the next adjacent segments above had 60% incidence of ASD although non symptomatic, showed that facet tropism is a factor in development of ASD rather than the fusion process itself and this proves our hypothesis.

No ASD was found in segments below the level of fusion in this group.

The fact that we had only one patient in B2 (Table 7) group made it difficult to get any correlations in this group but it shows that the patient had ASD in adjacent segment directly above to the hypolordotic segment fused but having only one patient made it impossible to figure out the role of hypolordosis and facet tropism separately as a cause of ASD.

The fact that we had only one patient in B3 group (Table 8) made it difficult to get any correlations in this group but it shows that the patient had ASD in adjacent segment directly below the hyperlordotic segment fused but having only one patient made it impossible to figure out the role of hyperlordosis and facet tropism separately as a cause of ASD.

So in conclusion the authors found that the results of this work prove that keeping lordotic alignment performing lumbar fusion for degenerative diseases within physiological

### Table 6: B1 group Relative Risk of New Disease Developing at an Adjacent Vertebral Level.

<table>
<thead>
<tr>
<th>Vertebral Level</th>
<th>Directly Adjacent Above (D AD. AB)</th>
<th>Directly adjacent below (D AD. BL)</th>
<th>Next Adjacent Above (N AD. Ab.)</th>
<th>Next Adjacent below (N AD. BL)</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total no. of discs at risk</td>
<td>5</td>
<td>5</td>
<td>5</td>
<td>0</td>
<td>15</td>
</tr>
<tr>
<td>No. of discs with new asymptomatic degeneration</td>
<td>4 (All Grade II)</td>
<td>0</td>
<td>3 (All Grade II)</td>
<td>0</td>
<td>7</td>
</tr>
<tr>
<td>Percentage (%)</td>
<td>80</td>
<td>60</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of discs with new (Symptomatic) disease</td>
<td>1 (Grade III)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Percentage (%)</td>
<td>20</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
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</table>

### Table 7: B2 group Relative Risk of New Disease Developing at an Adjacent Vertebral Level.

<table>
<thead>
<tr>
<th>Vertebral Level</th>
<th>Directly Adjacent Above (D AD. AB)</th>
<th>Directly adjacent below (D AD. BL)</th>
<th>Next Adjacent Above (N AD. Ab.)</th>
<th>Next Adjacent below (N AD. BL)</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total no. of discs at risk</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>No. of discs with new asymptomatic degeneration</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Percentage (%)</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>No. of discs with new (Symptomatic) disease</td>
<td>1 (Grade III)</td>
<td>0</td>
<td>0</td>
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<td>1</td>
</tr>
<tr>
<td>Percentage (%)</td>
<td>100</td>
<td></td>
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ranges decreased the incidence of both symptomatic and non symptomatic ASD and that the disturbance of this alignment increased the incidence of symptomatic and non symptomatic ASD in the segments directly above fusion in hypolordotic alignment and in the segment directly below in hyperlordotic alignment and thus keeping physiologic lordosis plays detrimental role in decreasing the incidence of ASD and that this should be taken care of during surgery. Also the authors concluded that patients with facet tropism are more likely to develop ASD than those with no tropism so those patients should be informed of this possibility and that they are more likely to need treatment that could be surgical for that condition in a while after fusion surgery. We do not know if motion preservation strategies would save those patients from this phenomenon or not and this is to be investigated.

More patient population and longer follow up are needed to further prove our results.

REFERENCES


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