Intradiscal Pressure Measurements Above an Instrumented Fusion
A Cadaveric Study

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Study Design. An in vitro study to determine the intradiscal pressure changes during flexion in levels above a simulated fusion was performed.

Objectives. To determine if intradiscal pressure increases more during flexion in discs above an instrumented spinal segment compared to an uninstrumented segment.

Summary of Background Data. The progressive degeneration of intervertebral discs adjacent to a fused or fixed segment is a phenomenon that is noted but poorly understood. Intuitively, the degeneration appears to be a function of altered biomechanics of the motion segments in the spine.

Methods. Two intervertebral disc levels were evaluated, L3–L4 and L4–L5 from each of six fresh frozen cadaver spines. Pressure measurements were taken with the spine uninstrumented, with bilateral pedicle screw instrumentation from L5 to S1, and with bilateral pedicle screw instrumentation from L4 to S1. Pressure measurements were accomplished with Millar Mikro-Tip pressure transducers. The transducers were placed within the nucleus pulposus of L3–L4 and L4–L5 intervertebral discs. Pressure data were recorded by computer data acquisition. The pressure data were compared by intervertebral level and by the effects of added instrumentation.

Results. In general, the addition of instrumentation significantly affected the intradiscal pressure in the levels above a simulated fusion. The intradiscal pressure increased as the amount of levels involved in the simulated fusion increased. The intradiscal pressure increased as flexion motion increased. A greater increase was seen at the L4–L5 level than the L3–L4 level. When L5–S1 fixation was added, the intradiscal pressure increased. When L4–S1 fixation was added, the intradiscal pressure further increased.

Conclusion. This study demonstrated increasing intradiscal pressure during flexion. [Key words: biomechanics, disc pressure, lumbar motion] Spine 1995;20: 526–531.

It has been reported that degenerative changes in the disc level adjacent to the fused segment develop in some patients. Lee et al. reported on 18 patients in whom adjacent disc symptoms developed after an average interval of 8.5 years. Lee hypothesized that this development may be due to alterations in the kinematics of the adjacent segment induced by the fusion. He felt that the use of internal fixation produces stress at the adjacent segment by increasing the fused segment stiffness and displacing its center of rotation. Hsu et al. reported on 30 patients who had a successful fusion that later led to breakdown of the adjacent discs. In patients with rigid fixation, the adjacent deterioration developed significantly faster, in an average of 5.3 years after surgery compared to 9.9 years among patients without instrumentation. Pintar et al. studied the kinematics of the lumbar spine for a variety of lumbar operative procedures. They found statistically significant increases in localized facet joint motion, which may suggest the potential for acceleration of degenerative changes.

Lee et al. and Yang et al. compared the stress changes at the adjacent spinal segment for posterior, anterior, and bilateral fusions. They found that a posterior fusion created the largest stress increase in the adjacent segment, especially in the facet joints. The anterior fusion created an intermediate stress increase whereas the bilateral fusion created the least adverse effects. Yoganandan et al. studied the kinematics of the lumbar spine after pedicle screw plate fixation. They found increased rigidity in the instrumented spinal segment, which can accelerate the healing process. Increases in motion at adjacent segments, however, demonstrate increased spine flexibility. Under in vivo conditions, this may lead to hypermobility and degeneration.

Data relating to in vivo disc pressures of a person in different body postures and performing certain tasks.
Table 1. Cadaver Morphologic and Bone Density Data

<table>
<thead>
<tr>
<th>Spine No.</th>
<th>Age</th>
<th>Cause of Death</th>
<th>Bone Density of L4 (g/cm²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>83</td>
<td>Racial failure</td>
<td>1.221</td>
</tr>
<tr>
<td>2</td>
<td>77</td>
<td>Acute myocardial infarction</td>
<td>0.683</td>
</tr>
<tr>
<td>3</td>
<td>61</td>
<td>Left pneumonitis</td>
<td>1.683</td>
</tr>
<tr>
<td>4</td>
<td>61</td>
<td>Gunshot to head</td>
<td>1.130</td>
</tr>
<tr>
<td>5</td>
<td>69</td>
<td>Arteriosclerotic cardiovascular disease</td>
<td>1.219</td>
</tr>
</tbody>
</table>

have been compiled\textsuperscript{1,2} most extensively by Nachemson.\textsuperscript{5} Although the portion of the body above the L3 disc constitutes 60% of the total body weight, the load on the L3 disc, in sitting and standing postures with 20° of flexion is 200%.\textsuperscript{3} This article also reports two cases with posterior fusions. The pressure within the fused segments was found to have an intradiscal pressure 30% less than persons without the fusion.

To our knowledge, no in vitro studies of disc pressure changes during flexion in levels above a simulated fusion have appeared in the literature. The purpose of this biomechanical study was to determine if intradiscal pressure increases more during flexion in discs above an instrumented spinal segment compared to an uninstrumented one.

- **Materials and Methods**

Because solid pedicle screw purchase, and therefore rigidity, is dependent on bone quality, the bone mineral density of live fresh, frozen cadaver spines were determined with dual-energy x-ray absorptiometry (model DPX-L, Lunar Corporation, Madison, WI; Table 1). Two intervertebral disc levels, L3–L4 and L4–L5, were evaluated from each of the spines. For all comparisons of disc pressure, each spine served as its own control. Pedicle screws (Wiltse-type, Advanced Spine Fixation Systems, Inc., Cypress, CA) were inserted bilaterally into the pedicles of L4, L5, and S1. The spines were potted in methyl methacrylate at T10 and the sacrum for attachment to an Instron materials testing machine (model 8511, Braintree, MA). A caliper and gross visualization were used to determine the position of the center of each disc. A small hole through the center of the anterior surface of the center position of the disc was made with a small Steinman pin so that the hole would be slightly smaller than the transducer catheter. Two Millar SPR-524 Mikro-Tip pressure transducers (Millar Instruments, Inc., Houston, TX) were used to measure intradiscal pressure (Figure 1). The transducer sensor has an outside diameter of 1.165 mm and the transducer catheter has an outside diameter of 0.84 mm. The transducer voltage output is linearly related to pressure for values up to 300 mm Hg as specified by the manufacturer. Because pressures higher than 300 mm Hg were anticipated, each transducer was calibrated to 2000 mm Hg to ensure nonlinear repeatability and to determine the relationship between the pressure and the voltage output of the transducer. A transducer was placed in each hole of each nucleus pulposus and attached with sutures to maintain stationary positions between tests (Figure 2).

Two markers were placed on the sacrum and in the body of L2. Photographs were taken at a rate of 1, 2, 3, 4, and 5 cm. From the photographs, the relationship of the actuator displacement to flexion angle was determined by measuring the angles between the markers. An overall average from L2 to S1 was determined.

Spinal flexion was controlled by the Instron 8511 with an actuator displacement rate of 2 mm/sec. A custom jig was used to convert this actuator vertical translation into a combination of flexion and translation; thereby mimicking the normal motion of the spine. Figures 3A and 3B illustrate the two extremes.
of motion with this experimental setup. Each spine was flexed from 0° to 20–40° depending on the size of the spine. Pressure measurements were taken with the spine uninstrumented, with bilateral pedicle screw-rod instrumentation from L5 to S1, and with bilateral pedicle screw-rod instrumentation from L4 to S1. Each test condition was replicated three times to ensure repeatability. The entire test sequence was conducted without removing the spine on transducers from the testing setup.

Pressure data were recorded simultaneously from each disc while the spine was being flexed to 20–40° (equivalent to 5 cm actuator displacement). The spines were kept moist with a saline spray and moist towels during the testing.

Statistical comparison between the uninstrumented condition and each instrumented condition was made with a paired t test at five intervals along the pressure profiles. Significant differences were accepted at the P < 0.05 level.

Results

Intradiscal pressure data was checked for repeatability among the three replicated motions at each condition. Correlation analysis found the data to be repeatable (r > 0.80) and was subsequently averaged. The pressure data were corrected based on the high pressure nonlinear relationship observed in the transducer calibration procedure. The actuator data were transposed into angular displacement based on the angular motion during photographic calibration. Finally, data from the five spines were averaged to give an average disc pressure at each flexion angle for each test condition (no instrumentation, posterior instrumentation from L5 to S1, and posterior instrumentation from L4 to S1).

In the uninstrumented spine the change in pressure in the L4–L5 disc was found to be greater than the change in pressure in the L3–L4 disc; however, this difference was not significant (Figure 4 and Table 2).

The change in pressure in the L3–L4 disc with no instrumentation compared to one-level posterior instrumentation (L5–S1) and two-level posterior instrumentation (L4–S1) is illustrated in Figure 5 and Table 3. For

<table>
<thead>
<tr>
<th>Flexion Angle</th>
<th>L3–L4</th>
<th>L4–L5</th>
</tr>
</thead>
<tbody>
<tr>
<td>5°</td>
<td>24.7 ± 5.3</td>
<td>80.1 ± 46.1</td>
</tr>
<tr>
<td>10°</td>
<td>72.8 ± 19.3</td>
<td>149.3 ± 82.1</td>
</tr>
<tr>
<td>15°</td>
<td>175.5 ± 53.1</td>
<td>275.7 ± 106.4</td>
</tr>
<tr>
<td>20°</td>
<td>392.3 ± 66.4</td>
<td>462.5 ± 122.9</td>
</tr>
</tbody>
</table>
instance, at 20°, the L3–L4 pressure without instrumentation was 392.2 mm Hg, and with a one-level simulated fusion the pressure was 536.47 mm Hg. This represents a 37% increase in intradiscal pressure two disc spaces above the fixed segment. With a two-level simulated fusion, the pressure was 714.14 mm Hg at 20°. This is a 33% increase compared to the one-level simulated fusion condition and an 82% increase one disc space above the fixed segment compared to the noninstrumented condition. Significantly increased pressure was noted at the end of the flexion range in a one-level posterior instrumentation compared to no instrumentation (P < 0.05). Significantly increased pressure was noted along the entire flexion range in a two-level posterior instrumentation compared to the noninstrumented condition (P < 0.05).

The change in pressure in the L4–L5 disc with no instrumentation compared to one-level posterior instrumentation (L5–S1) and two-level posterior instrumentation (L4–S1) is illustrated in Figure 6 and Table 4. For instance, at 20°, L4–L5 pressure was 462.46 mm Hg and with a one-level simulated fusion the pressure was 600.19 mm Hg. This represents a 30% increase in the intradiscal pressure one disc space above the fixed segment. The data were found to be significant at the end of the measured flexion range, that is, 20°, in the one-level posterior instrumentation compared to the no instrumentation condition (P = 0.02). The two-level simulated fusion resulted in an L4–S pressure of 615.15 mm Hg at 20° of flexion. There was no significant difference between the two-level simulated fusion condition at L4–L5 and the uninstrumented condition.

**Discussion**

The progressive degeneration of intervertebral discs adjacent to a fused or fixed segment is a phenomenon that has been noted but poorly understood. Intuitively, the degeneration appears to be a function of altered biomechanics of the motion segments in the spine. One of the results of altered spinal biomechanics is speculated to be changes in the pressures seen within the disc. We tested this hypothesis in a cadaveric model using pedicle screw fixation.

**Table 3. Intradiscal Pressure (mm Hg) at L3–L4 (mean ± standard error of the mean; n = 5)**

<table>
<thead>
<tr>
<th>Flexion Angle</th>
<th>No Instrumentation</th>
<th>One-Level Fixation (L5–S1)</th>
<th>Two-Level Fixation (L4–S1)</th>
</tr>
</thead>
<tbody>
<tr>
<td>5°</td>
<td>24.64 ± 5.33</td>
<td>34.22 ± 24.72</td>
<td>50.99 ± 14.48</td>
</tr>
<tr>
<td>10°</td>
<td>72.78 ± 19.87</td>
<td>114.58 ± 41.07</td>
<td>187.40 ± 66.86</td>
</tr>
<tr>
<td>15°</td>
<td>175.35 ± 53.09</td>
<td>266.60 ± 71.21</td>
<td>426.09 ± 153.24</td>
</tr>
<tr>
<td>20°</td>
<td>392.26 ± 96.39</td>
<td>536.47 ± 110.33*</td>
<td>714.14 ± 213.99</td>
</tr>
</tbody>
</table>

* Significantly greater than control; P < 0.05 (paired t test).

**Table 4. Intradiscal Pressure (mm Hg) at L4–L5 (mean ± standard error of the mean; n = 5)**

<table>
<thead>
<tr>
<th>Flexion Angle</th>
<th>No Instrumentation</th>
<th>One-Level Fixation (L5–S1)</th>
<th>Two-Level Fixation (L4–S1)</th>
</tr>
</thead>
<tbody>
<tr>
<td>5°</td>
<td>80.06 ± 45.16</td>
<td>77.69 ± 52.13</td>
<td>81.58 ± 19.89</td>
</tr>
<tr>
<td>10°</td>
<td>149.87 ± 42.10</td>
<td>154.25 ± 50.70</td>
<td>152.75 ± 39.58</td>
</tr>
<tr>
<td>15°</td>
<td>275.68 ± 106.40</td>
<td>311.92 ± 101.26</td>
<td>302.36 ± 73.67</td>
</tr>
<tr>
<td>20°</td>
<td>462.46 ± 122.91</td>
<td>600.19 ± 86.85*</td>
<td>615.15 ± 143.27</td>
</tr>
</tbody>
</table>

* Significantly greater than control; P < 0.05 (paired t test).
The dynamic nature of the pressure increase was demonstrated in spines without hardware instrumentation (i.e., control conditions). The intradiscal pressure increased slowly and continued to rise along the entire range of motion. The L4–L5 disc space showed a higher pressure than that noted in the L3–L4 disc space but given the low specimen numbers, our study could not show significant differences. This result was expected because others have shown increasing intradiscal pressures at lower levels of the spine. We believe this is the first demonstration of the dynamics of the pressure rise during a flexion movement. These intradiscal pressure measurements were subsequently used to determine the effect of spinal fixation on the pressures within the intervertebral disc.

The results of this study indicate that during flexion, intradiscal pressure increased in discs above an instrumented spine segment. The pressure increase is likely caused by the force transmission from the level(s) involved in the fusion because their ability to absorb force is diminished as a result of the loss of motion. The results also indicate that two-level instrumentation affects the adjacent disc pressure more than one-level instrumentation and occurs earlier in the range of motion. This may be caused by the additional segment involved in the rigid fusion, thus increasing the flexibility of the spine above the fusion to a greater degree. This force transmission may also account for the many reports of accelerated degeneration in adjacent discs.

It was interesting to note that the pressure measured in the L4–L5 disc during a two-level fixation (i.e., the pressure measurement taken at the level that was included in the fixed segment length) was essentially the same as that measured in the control situation. Recent reports indicate that internal spinal fixation facilitates fusion mass healing by providing a stable environment free from gross motion. But the pressure measurements made in this study indicate that micromotion in the anterior column, and therefore the discs, may still be sufficient to produce potentially important dynamics. If the disc represents a pain source, then the intradiscal pressure even during posterior fixation may be clinically important. Removal of the disc in an anterior fusion procedure or in conjunction with posterior fusion/fxation may be more appropriate in some cases.

This study created motion from L2 to the sacrum of only 20°. This motion is approximately one third of the expected range for these levels. Therefore, the increase in intradiscal pressure occurred even with a small range of motion. The normal lumbar range of motion is approximately 60°. This larger motion angle may possibly compound this effect because there did not appear to be a plateau in the pressure increase up to the angles measured.

While traditional rehabilitation efforts in the past were generally directed at getting the patient "back to normal," these results would reaffirm that to better rehabilitate a spinal fusion patient, flexion motion should be limited, stabilization exercises should be increased, and the patient should not be expected to return to normal range of motion. Compelling patients with instrumented spinal segments to move through a normal range of motion may place undue stress at the adjacent disc level. This motion, repeated over time, may lead to the breakdown of the adjacent disc. The target range of motion should perhaps be reduced for fusion patients, especially those with multilevel fusion. The changes in rehabilitation may help increase the longevity of the disc adjacent to the fusion.

Further clinical importance of this research is in the area of presurgical planning. Because intradiscal pressure may play a role in disc breakdown; any disc that is already slightly degenerated or disrupted may be at an even greater risk after successful fusion. Therefore, it is imperative that surgical planning for spinal fusion take this into consideration. The normalcy of the adjacent discs is important in planning how many levels of the spine to fuse. It may be necessary to include additional motion segments in the length of the fusion if the disc levels are present that show early signs of degeneration as evidence by positive discography.

The results of this study are based on in vitro cadaver experiments. Therefore in the in vivo situation, the intradiscal pressures may be different from those illustrated here, although the relative changes will be the same. However, the results of this study can help us begin to understand the changes in intradiscal pressure with a simulated fusion. The effects in patients may be greater than demonstrated in this study. In patients, where solid bony fusion occurs, the pressure within the adjacent disc may be increased even more because the fused segments are providing less motion than in the current study (the study only restricted posterior motion). The effects may be magnified even more in a combined anteroposterior fusion, which would more rigidly stabilize the fused segments than just the instrumentation used in this cadaveric study.

In summary, this study has demonstrated increasing intradiscal pressures during flexion. We speculate that this has importance in the subsequent accelerated breakdown of the discs adjacent to fused segments that has been observed clinically. It may be inappropriate to encourage spinal fusion patients to regain a normal range of motion and therefore postoperative rehabilitation may need to be modified accordingly. Finally, for spinal fusions, it may be important to determine the normalcy of discs adjacent to a proposed fusion because failure to do so may result in accelerated disc degeneration requiring further surgery.

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References


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