Biomechanical Role of the Thoracolumbar Ligaments of the Posterior Ligamentous Complex: A Finite Element Study

Cong-Cong Wu, Hai-Ming Jin, Ying-Zhao Yan, Jian Chen, Ke Wang, Jian-Le Wang, Zeng-Jie Zhang, Ai-Min Wu, Xiang-Yang Wang

OBJECTIVES: To investigate the effect of sequential ligament failure on the range of motion (ROM) and location of the instantaneous axes of rotation (IAR) of the thoracolumbar spine (T12–L1) finite element (FE) model, and to verify the role of the supraspinous ligament (SSL) in maintaining the stability of the injured thoracolumbar spine.

METHODS: An FE model of the fractured thoracolumbar spine was developed and validated against published data. The posterior ligamentous complex (PLC) in the fractured T12–L1 segment was then reduced in a sequential manner from the facet capsular ligament (FCL), part of the interspinous ligament (ISL), SSL, and entire ISL, to the ligamentum flavum (LF). The ROM and IAR of the T12–L1 segment were measured at the fracture and at each reduced ligament step under 4 directions of flexion, extension, lateral bending, and rotation, and 4 bending motions of 1.5, 3.0, 4.5, 6.0 Nm.

RESULTS: The FE model showed a consistent increase in the ROM and location of the IAR as the ligaments were removed sequentially. Furthermore, failure of the SSL had the most significant influence on the change in the ROM and IAR in flexion. In extension, removal of the FCL caused the largest shift.

CONCLUSIONS: The SSL is a significant ligament that allows the PLC to maintain the stability of the thoracolumbar spine during injury.

INTRODUCTION

The thoracolumbar junction, and the T12–L1 segment in particular, are susceptible to traumatic fracture. Studies have shown complications after spinal fractures.1-3 Traumatic bone fracture of T12–L1 leads to spinal instability, which in turn decreases the patient’s quality of life. The motion of an intact spinal segment is well managed by the coordination of muscles, discs, facets, and ligaments. Any injury to these structures influences the function of the spine. A posterior element failure commonly occurs alongside an anterior structure injury; this is particularly the case with the posterior ligamentous complex (PLC).1,4

The PLC is composed of the interspinous ligament (ISL), supraspinous ligament (SSL), facet capsular ligament (FCL), and ligamentum flavum (LF). The Thoracolumbar Injury Classification and Severity (TLICS) scoring system suggested that PLC integrity was a key part of the stability assessment of the thoracolumbar spine.5,6 However, PLC integrity was assessed only as a whole in TLICS, which affects its application in a clinical setting. Although different methods have been used to describe the problem of the effect of failure of a particular PLC ligament on spinal stability, no consensus has been reached.

In some previous spinal kinematic studies, researchers analyzed changes in the range of motion (ROM) and locations of the instantaneous axes of rotation (IAR) of the spinal segments to assess the role of each ligament by sequentially cutting the ligaments.9,10 However, the method of sequentially reducing the ligaments was not realistic in these experiments. Some recent studies showed that the realistic sequential injury order of the PLC in a clinical setting is the FCL, part of ISL, SSL, entire ISL, and LF and that the SSL is the pivotal ligament that allows the PLC to maintain the stability of the
There has been no biomechanical finite element (FE) study of thoracolumbar spine fractures that follows the above realistic ligament failure order. The objective of the current study was to assess the ROM and IAR caused by various ligament failures and pure motion and to determine whether SSL rupture provides the key to PLC incompetence.

**MATERIALS AND METHODS**

Computed tomography scan data from a healthy 30-year-old man using a 512 × 512 pixel matrix at 1-mm intervals were used to construct a 3-dimensional FE model of the human T12–L1 segment. Informed consent was obtained from the volunteer. These data were then imported into Mimics version 19.0 (Materialise, Leuven, Belgium) to obtain a simplified skeletal model. After that, a 3-dimensional solid model was constructed using Geomagic version 2012 (Geomagic, North Carolina, USA). The intervertebral disc was modeled using SolidWorks version 2015 (SolidWorks, Massachusetts, USA). Finally, after FE modeling, the model was imported into ANSYS version 16.2 (Ansys, Canonsburg, Pennsylvania, USA) for FE analysis, in which ligaments were created and all structures were evaluated.

**Initial Model**

The vertebrae were composed of a vertebral body and posterior elements, including pedicle, lamina, and transverse and spinous processes. The components of the intervertebral disc included the nucleus pulposus, cartilage endplate, and annulus fibrosus. The vertebral bodies were assumed to have a cancellous core covered by a cortical shell (with a 1-mm-thick wedge element). The FE model consisted of 41 springs, 69,106 nodes, and 216,588 solid elements. Spinal ligaments were simulated by body-to-body spring elements. The material properties used in the current study are provided in Table 1.

**Model with a Fracture and Ligament Failure**

To mimic a slight fracture of the L1 vertebra, a fracture model with a bone defect was constructed, as shown in Figure 1. In the fracture model, the superior half of the cortical bone of the vertebral body was removed, and the superior half of the cancellous bone was assigned 10% of the modulus of normal cancellous bone. As Figure 2 shows, ligament failures were simulated by the sequential reduction of the PLC in the following order: FCL, part of the SSL, ISL, entire SSL, and LF.

**Boundary and Loading Conditions**

For biomechanical tests, the inferior endplate and spinous process of the L1 vertebra were fixed in all degrees of freedom. Otherwise, the T12 vertebra was free to move in all directions. Loading and boundary conditions were replicated according to published experimental studies conducted using human cadaveric thoracolumbar spines and FE spine models. Pure moment loads were applied to the superior surface of the T12 vertebra in the 3 main planes, including flexion, extension, lateral bending, and axial rotation. For the sake of brevity, left lateral bending and anticlockwise rotation were ignored because of their symmetry, in line with other studies. For the validation analysis, a 7.5-Nm bending moment was applied to the intact spine model to match the published experimental studies for comparison. To validate the effect of each ligament on spine motion and the instantaneous center, bending moments of 1.5, 3.0, 4.5, and 6.0 Nm were applied to the injured spine model.

**Data Analysis**

The ROM and IAR of the motion segment were investigated for conditions leading to sequential ligament failure of the PLC under 4 motion directions (flexion, extension, right lateral bending, and anticlockwise axial rotation) and 4 levels of loading (1.5, 3.0, 4.5, and 6.0 Nm). The ROM during motion in all planes was determined as the degree of motion from the instantaneous center to the angle achieved at the particular bending motion. The IAR was calculated according to a published method, using the intersection of the instantaneous centers at the beginning and end of motion.

<table>
<thead>
<tr>
<th>Component Name</th>
<th>Young’s Modulus (MPa)</th>
<th>Poisson’s Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cortical bone</td>
<td>12000</td>
<td>0.3</td>
</tr>
<tr>
<td>Cancellous bone</td>
<td>100</td>
<td>0.2</td>
</tr>
<tr>
<td>Cartilage</td>
<td>32.9</td>
<td>0.3</td>
</tr>
<tr>
<td>Cartilage endplate</td>
<td>500</td>
<td>0.4</td>
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<tr>
<td>Nucleus pulposus</td>
<td>1</td>
<td>0.49</td>
</tr>
<tr>
<td>Annulus fibrosus</td>
<td>4.2</td>
<td>0.45</td>
</tr>
<tr>
<td>Posterior elements</td>
<td>3500</td>
<td>0.25</td>
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</table>

MPa, megapascal.
of the perpendicular bisectors of the displacement vectors of 2 peripheral nodes of the inferior endplate of the T12 vertebra.19-21 The center of the L1 superior endplate was chosen as the origin of the coordinates. Then, the change in the centrode, which the IAR followed in each direction during a full loading cycle, was evaluated.

RESULTS

Analysis of the ROM

ROM Analysis for the Intact Model. The predicted ROMs were 3.56°, 4.45°, 4.64°, and 1.85° for flexion, extension, lateral bending, and rotation under 7.5 Nm of pure bending motion. Figure 3 illustrates

Figure 2. Sequential reduction order of the posterior ligamentous complex in our study. (A) Cutting the facet capsular ligament. (B) Cutting part of the interspinous ligament (ISL). (C) Cutting the supraspinous ligament. (D) Cutting the entire ISL. (E) Cutting the ligamentum flavum.

Figure 3. Comparison of the predicted ranges of motion with published experimental results under 7.5 Nm of pure bending motion.
the comparison of the predicted ROMs with published experimental results.17,18 The current FE model successfully fell within the range of the aforementioned experimental data for the T12–L1 segment.

ROM Analysis for Models with Fracture. As shown in Table 2, the fracture of the L1 model caused a slight increase in the ROM under 6.0 Nm of pure bending motion, with 1.04° in flexion, 1.54° in extension, 0.58° in lateral bending, and 0.79° in axial rotation.

ROM Analysis for Models with Ligament Failure. In terms of flexion, failure of the FCL did have a slight impact on the ROM, and removing part of the ISL was also observed to cause a slight increase in the ROM. However, the SSL had the most significant influence on the ROM in flexion. Furthermore, failure of the entire ISL and LF in the process caused a slight increase in the ROM. In terms of extension, failure of the FCL had the most significant impact on the ROM. Otherwise, all other ligaments made only a slight contribution to the increase in the ROM. For lateral bending and axial rotation, failure of all ligaments had only a slight impact on the increase in the ROM.

Analysis of the IAR

IAR Analysis for the Intact Model. In all directions of motion, the IAR was observed to be closer to the superior endplate of the L1 vertebral body. In sagittal motion, the IAR was observed to be closer to the superior endplate of the L1 vertebral body, and stayed within the disc space. During sagittal motion, the length of the centrode along with the total translation of the IAR increased markedly compared with those during axial and lateral motion. The length of the centrode and the total displacement of the IAR were similar in both flexion and extension. In lateral bending and axial rotation, the IAR was also located within the disc space, close to the center.

IAR Analysis for the Model with a Fracture. In flexion, the IAR shifted slightly anteriorly and superiorly as the ligaments were removed. By contrast, the IAR and centrode shifted slightly posteriorly and superiorly in extension, and anterolaterally in lateral bending and axial rotation.

The IAR Versus Fracture in the Model with Ligament Failure. As shown in Table 3 and Figures 4–7, in flexion, the IAR was located in the middle third of the intervertebral disc, close to the L1 superior

<table>
<thead>
<tr>
<th>Directions of Motion</th>
<th>Intact (°)</th>
<th>Fracture (°)</th>
<th>Without FCL (°)</th>
<th>Without Part of ISL (°)</th>
<th>Without SSL (°)</th>
<th>Without Entire ISL (°)</th>
<th>Without LF (°)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flexion</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1.5 Nm</td>
<td>1.04</td>
<td>1.38</td>
<td>1.45</td>
<td>1.53</td>
<td>1.74</td>
<td>1.77</td>
<td>1.80</td>
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<tr>
<td>3.0 Nm</td>
<td>1.95</td>
<td>2.39</td>
<td>2.56</td>
<td>2.75</td>
<td>3.25</td>
<td>3.31</td>
<td>3.35</td>
</tr>
<tr>
<td>4.5 Nm</td>
<td>2.47</td>
<td>3.42</td>
<td>3.44</td>
<td>3.60</td>
<td>4.68</td>
<td>4.73</td>
<td>4.74</td>
</tr>
<tr>
<td>6.0 Nm</td>
<td>3.12</td>
<td>4.16</td>
<td>4.18</td>
<td>4.56</td>
<td>5.85</td>
<td>5.96</td>
<td>6.00</td>
</tr>
<tr>
<td>Extension</td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>1.5 Nm</td>
<td>1.30</td>
<td>1.79</td>
<td>1.92</td>
<td>1.95</td>
<td>1.96</td>
<td>1.97</td>
<td>2.04</td>
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<td>3.0 Nm</td>
<td>2.29</td>
<td>3.25</td>
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<td>3.62</td>
<td>3.64</td>
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<td>4.5 Nm</td>
<td>3.18</td>
<td>4.46</td>
<td>5.20</td>
<td>5.23</td>
<td>5.25</td>
<td>5.26</td>
<td>5.31</td>
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<tr>
<td>6.0 Nm</td>
<td>3.77</td>
<td>5.31</td>
<td>6.78</td>
<td>6.82</td>
<td>6.83</td>
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<td>Axial rotation</td>
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<tr>
<td>1.5 Nm</td>
<td>0.62</td>
<td>0.76</td>
<td>0.80</td>
<td>0.81</td>
<td>0.82</td>
<td>0.83</td>
<td>0.86</td>
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<tr>
<td>3.0 Nm</td>
<td>0.96</td>
<td>1.33</td>
<td>1.34</td>
<td>1.35</td>
<td>1.36</td>
<td>1.38</td>
<td>1.39</td>
</tr>
<tr>
<td>4.5 Nm</td>
<td>1.13</td>
<td>1.78</td>
<td>1.80</td>
<td>1.81</td>
<td>1.82</td>
<td>1.83</td>
<td>1.84</td>
</tr>
<tr>
<td>6.0 Nm</td>
<td>1.65</td>
<td>2.21</td>
<td>2.23</td>
<td>2.25</td>
<td>2.26</td>
<td>2.27</td>
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<tr>
<td>1.5 Nm</td>
<td>1.36</td>
<td>1.37</td>
<td>1.43</td>
<td>1.44</td>
<td>1.47</td>
<td>1.51</td>
<td>1.52</td>
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<tr>
<td>3.0 Nm</td>
<td>2.19</td>
<td>2.67</td>
<td>2.69</td>
<td>2.76</td>
<td>2.82</td>
<td>2.84</td>
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<tr>
<td>4.5 Nm</td>
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<td>3.98</td>
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<tr>
<td>6.0 Nm</td>
<td>3.98</td>
<td>4.77</td>
<td>5.03</td>
<td>5.12</td>
<td>5.18</td>
<td>5.20</td>
<td>5.31</td>
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</tbody>
</table>

FCL, facet capsular ligament; ISL, interspinous ligament; SSL, supraspinous ligament; LF, ligamentum flavum.
endplate in the ligament failure model. With the increase in bending motion, it shifted slightly anteriorly and superiorly after the subsequent removal of the FCL and part of the ISL. After removal of the SSL, the IAR shifted significantly, nearly 0.39 mm. Furthermore, the subsequent failure of the entire ISL and LF did not have much influence on the IAR; it shifted 1.00 mm anteriorly and superiorly when all the ligaments were reduced. In extension, the IAR was also initially located in the middle third of the intervertebral disc, and shifted 0.65 mm posteriorly and superiorly when all the ligaments were reduced. With removal of the FCL, the IAR showed the most significant shift. Otherwise, none of the other ligament failures of the process had a great influence on the IAR. In lateral bending and axial rotation, the IAR was also initially observed near the center of the disc. However, the IAR changed more after failure of the ISL and SSL compared with other ligaments in lateral bending. The IAR moved into the superior and right lateral directions by 1.11 mm with the simulation of full ligament failure in lateral bending. On the other hand, with the simulation of ligament failure, the IAR moved anterolaterally by 0.69 mm under clockwise rotation. There was no remarkable shift under all conditions of ligament failure in the axial rotation motion; that is, the centrode remained close to a near-linear shape.

Table 3. Mean Position of IAR in Different Levels of Ligament Failure and Loading Conditions

<table>
<thead>
<tr>
<th>Directions of Motion</th>
<th>Flexion</th>
<th>Extension</th>
<th>Axial Rotation</th>
<th>Lateral Bending</th>
</tr>
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<tr>
<td>Intact</td>
<td>4.01, 1.06</td>
<td>2.51, 1.59</td>
<td>2.67, 0.08</td>
<td>1.57, 0.59</td>
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<tr>
<td>Fracture</td>
<td>3.98, 1.06</td>
<td>2.54, 1.67</td>
<td>2.73, 0.09</td>
<td>1.53, 0.63</td>
</tr>
<tr>
<td>Without FCL</td>
<td>3.64, 1.13</td>
<td>2.64, 1.91</td>
<td>2.85, 0.18</td>
<td>1.47, 0.79</td>
</tr>
<tr>
<td>Without part of ISL</td>
<td>3.68, 1.29</td>
<td>2.67, 1.97</td>
<td>2.96, 0.22</td>
<td>1.36, 0.81</td>
</tr>
<tr>
<td>Without SSL</td>
<td>3.39, 1.54</td>
<td>2.71, 1.98</td>
<td>3.04, 0.27</td>
<td>1.16, 1.04</td>
</tr>
<tr>
<td>Without entire ISL</td>
<td>3.31, 1.65</td>
<td>2.73, 2.14</td>
<td>3.14, 0.31</td>
<td>0.93, 1.25</td>
</tr>
<tr>
<td>Without LF</td>
<td>3.25, 1.75</td>
<td>2.77, 2.28</td>
<td>3.33, 0.44</td>
<td>0.78, 1.45</td>
</tr>
</tbody>
</table>

IAR, instantaneous axes of rotation; FCL, facet capsular ligament; ISL, interspinous ligament; SSL, supraspinous ligament; LF, ligamentum flavum.

Figure 4. Change of the mean position of the instantaneous axes of rotation (IAR) with fracture and subsequent transection of ligaments in flexion. – Values for anterior and inferior directions. Values for the coordinate system are in millimeters. FCL, facet capsular ligament; ISL, interspinous ligament; SSL, supraspinous ligament; LF, ligamentum flavum.
Figure 5. Change of the mean position of the instantaneous axes of rotation (IAR) with fracture and subsequent transection of ligaments in extension. + Values for posterior and superior directions. – Values for anterior and inferior directions. Values for the coordinate system are in millimeters. FCL, facet capsular ligament; ISL, interspinous ligament; SSL, supraspinous ligament; LF, ligamentum flavum.

Figure 6. Change of the mean position of the instantaneous axes of rotation (IAR) with fracture and subsequent transection of ligaments in lateral bending. + Values for superior and left lateral directions. – Values for inferior and right lateral directions. Values for the coordinate system are in millimeters. FCL, facet capsular ligament; ISL, interspinous ligament; SSL, supraspinous ligament; LF, ligamentum flavum.
DISCUSSION

In this study, the 2 important physical parameters of the ROM and IAR were considered in an FE model of the thoracicolumbar spine, which contained various conditions of ligament failure by means of a stepwise reduction of the ligaments. In TLICS, the integrity of the PLC was described as a key point for stratifying patients into surgical and nonsurgical treatment groups. Previous biomechanical studies also showed the significance of PLC integrity in spine stability and attempted to reveal the function of each ligament of the PLC. Heuer et al.10 analyzed the ROM of 8 spinal segments (L4–5) by sequentially cutting ligaments in cadaveric specimens. Alapan et al. 11 used an FE model of the L4–5 segment to analyze the instantaneous center of rotation behaviour of the lumbar spine with ligament failure. However, those 2 in vitro studies did not follow a realistic ligament failure order. Thus, their results might not indicate the real mechanism of the ligaments in maintaining the stability of the spinal segments. Pizones et al.13,14 studied the sequential pattern of PLC rupture caused by deforming traumatic forces by analyzing the magnetic resonance images of 74 traumatic fractures. He concluded that with the increase in force, the real injury order of the PLC was the FCL, part of the ISL, SSL, entire ISL, and LF, and that the SSL was the key ligament in the function of the PLC. Li et al. 15 investigated the stability of the thoracicolumbar spine by stepwise resection of the posterior ligamentous complex in 12 fresh human spinal specimens based on the research of Pizones et al. 13,14 and Li et al.15 also concluded that the stability of the segment decreased significantly with rupture of SSL. In our study, the ligament failure order was also based on the research of Pizones et al.13,14

Clinically, deforming traumatic forces always cause simultaneous ligament failure and vertical fracture. These forces limit the motion of the spine, especially in flexion—the most important function. Few previous studies considered the influence of a vertebra fracture while analyzing ligament failure. In this study, vertical fracture models were made in FE before ligament failure analysis. The superior half of the cortical bone of the vertebral body was removed, and the superior half of the cancellous bone was assigned 10% of the modulus of normal cancellous bone. Several fracture models have been published previously.22-26 Compared with these mimic fracture models, the model established in our study was more realistic. Although no consensus has been reached until now regarding the bending motion, we calculated the ROM and IAR of an intact spinal segment under a 7.5-Nm bending motion to allow for a comparison with previous studies. Moreover, 4 motion directions (flexion, extension, right lateral bending, and clockwise axial rotation) and 4 levels of loading (1.5, 3.0, 4.5, and 6.0 Nm) were applied to the fracture and ligament failure in our study; these also matched some previously published experimental studies.

The ROM was proved to be an important parameter in previous studies analyzing spinal stability. From the raw data, the angular ROM in all planes during motion was defined as the degree of motion from the location of the instantaneous center of rotation to the angle achieved at the particular end of a bending motion. Our study showed that the ROM increased in an unabridged spinal segment under all bending motions, which agreed with some previous studies. Moreover, the ROM increased more after sequential resection of the ligaments in all motions compared...
with the intact spinal model. In flexion, failure of the SSL had the most significant effect on the ROM. Otherwise, failure of any of the other ligaments did not cause such a noteworthy increase in the ROM. In extension, the ROM changed most when the FCL was removed. Except for the FCL, other ligament failures caused only a slight shift of the ROM. In lateral bending and axial rotation, no significant shift of the ROM was observed under any condition of ligament failure. Beyond that, ROMs measured in the sagittal and coronal planes were much larger than in lateral bending; that is, ligament failure of the PLC in a fracture vertebral model influenced spinal stability more in the sagittal and coronal planes.

The location of the IAR was widely used to investigate spinal stability in previous studies and was shown to have diagnostic value in defining the source of segmental instability. The IAR measurement method in this study matched that of previous studies. The normal location of the IAR has been studied in cadavers and in normal volunteers, and an abnormal location of the IAR has also been shown to correlate with spinal instability in previous studies. In the current study, we investigated the IAR in FE under the conditions of intact, fracture, and sequential ligament failure in different bending motions. In every ligament failure condition, 4 series of mean instantaneous axes were measured, linked to 4 bending motions, which constituted the centrode (i.e., the path that the IAR followed during the increase in pure motion). The centrode shift was observed in the intact spinal segment under all bending motions, which was in agreement with some previous studies. In flexion, removal of all the ligaments carried the IAR anteriorly and superiorly, toward the center of the superior endplate of the L1 vertebral body. Compared with other ligaments, the shift of the IAR was much more significant after the SSL was resected in flexion. With proceeding extension movements, the IAR was carried posteriorly and superiorly during sequential ligament failure. The most remarkable change happened when the FCL was removed. In lateral bending, the IAR changed more after ISL and SSL failure compared with the other ligaments. In the axial rotation, the centrode changed smoothly into a near-linear shape; that is, no ligament failure caused a noteworthy shift during the process.

The ROM and IAR have been used to analyze spinal stability in many different surgeries and clinical implantations, such as lumbar total disc arthroplasty,27 the pedicle-based dynamic stabilization system,28 lumbar interspinous spacer,29 and dynamic pedicle screw systems.30 However, few studies have taken realistic ligament failures and vertebral fractures into consideration when analyzing the alteration of implants or the spine. Kinematic studies should produce a better understanding of what the workers investigated or are investigating by using a realistic injury spine model. Furthermore, our investigation provides biomechanical evidence for clinicians that repairing the PLC is a crucial step in surgery. For the patient to recover and maintain spinal stability, surgeons should repair all broken ligaments if possible, or at least fix the SSL and maintain facet integrity.

Injury of the PLC is a dynamic process. Thus, our study can only prove that the SSL plays the most significant role during this process. When this dynamic process is not considered, our conclusion may not be applicable.

CONCLUSION

The location of the ROM and IAR was changed in all planes of motion in intact, fracture, and ligament failure models. Particular ligament failure caused a much more significant shift of the ROM and IAR. Furthermore, after removal of the SSL, the ROM, and IAR, of the T12–L1 segment increased sharply under flexion motion. In extension, excision of the FCL caused the most remarkable increase. The changes in the ROM and IAR reveal the mechanism of unstable motion of a fractured spinal segment under the condition of sequential ligament removal. Moreover, the SSL, as investigated, is the most significant ligament for the PLC to maintain the stability of the thoracolumbar spine.

REFERENCES


