Creating reproducible thoracolumbar burst fractures in human specimens: an in vitro experiment

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OBJECT The treatment of traumatic burst fractures unaccompanied by neurological impairment remains controversial and ranges from conservative management to 360° fusion. Because of the heterogeneity of fracture types, classification systems, and treatment options, comparative biomechanical studies might help to improve our knowledge. The aim of the current study was to create a standardized fracture model to investigate burst fractures in a multisegmental setting.

METHODS A total of 28 thoracolumbar fresh-frozen human cadaveric spines were used. The spines were dissected into segments (T11–L3). The T-11 and L-3 vertebral bodies were embedded in Technovit 3040 (cold-curing resin for surface testing and impressions). To simulate high energy, a metallic drop tower was designed. Stress risers were used to ensure comparable fractures. CT scans were acquired before and after fracture. All fractures were classified using the AO/OTA classification.

RESULTS The preparation and embedding of the spine segments worked well. No repositioning or second embedding of the specimen, even after fracture, was required. It was possible to create single burst fractures at the L-1 level in all 28 spine segments. Among the 28 fractures there were 16 incomplete burst fractures (Type A3.1), 8 burst-split fractures (Type A3.2), and 4 complete burst fractures (Type A3.3). The differences before and after fracture for stiffness and for anterior, posterior, and central heights were all significant (p < 0.05).

CONCLUSIONS The ability to create reproducible burst fractures of a single vertebral body in a thoracolumbar spine segment may serve as a basis for future biomechanical studies that will provide better understanding of mechanical properties or fixation techniques.

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KEY WORDS spine; fracture mechanism; burst fracture; fracture model; biomechanical; thoracolumbar; trauma

Burst fractures mostly occur in the thoracolumbar region and are one of the most common fracture types, especially in younger patients. Acute thoracolumbar burst fractures are classified according to the AO/OTA Classification as a subgroup of Type A fractures (A3). Burst fractures are defined by involvement of the posterior vertebral body wall. The height of the posterior vertebral wall is often reduced, and spinal canal compromise often accompanies this compression fracture. Burst fractures can be subdivided into incomplete burst fractures (Type A3.1), burst-split fractures (Type A3.2), and complete burst fractures (Type A3.3). Per definition for all Type A fractures, the posterior ligament complex (PLC) has to remain intact. Treatment options for burst fractures without neurological symptoms discussed in the literature are controversial. Recommendations range from operative to conservative treatment. However recent meta-analyses of the literature reveal no advantage for surgical treatment in comparison with conservative treatment. Various operative treatments are described in the literature: posterior stabilization with or without decompression, anterior fusion, 360° fusion, and percutaneous instrumentation as well as cement augmentation or a combination of techniques. Most recommendations and treatment options are based on biomechanical considerations, and clinical studies will continue to show advantages of different operative treatments. However, in vitro models are necessary to evaluate the stability of the reconstructed spine. The most common challenge in designing biomechanical studies is the creation of a repro-
ducible burst fracture, especially if the vertebral bodies are not dissected. For example, to evaluate posterior stabilization, it is necessary to use a specimen that has at least 2 connected motion segments and create a burst fracture in the middle vertebral body. Several biomechanical studies have already established methods for creating thoracolumbar burst fractures in vitro; however, the number of specimens in these studies is usually small, or animal vertebrae were used instead of human specimens.\(^7\) The aim of the current study was to develop and standardize a method for an in vitro experiment to create reproducible thoracolumbar burst fractures in human spinal segments.

**Methods**

**Specimens**

A total of 28 fresh-frozen human cadaveric thoracolumbar spines were used (T11–L3). Only male donors (mean age at the time of death 64.9 ± 6.5 years) were selected based on anonymized patient profiles to reduce the risk of osteoporosis or poorer bone quality. The donors had no history of tumor, osteoporosis, arthritis, or use of medications that could lead to secondary osteoporosis. Prior to fracture generation, CT scanning of all spines was performed to identify any pathologies, especially preexisting vertebral fractures or deformities. All 28 specimens could be used. The spines were dissected into segments (T11–L3). Soft tissue was removed except for the intervertebral discs and the anterior as well as the posterior ligament complex. The vertebral bodies of T-11 and L-3 were stably embedded in Technovit 3040 (cold-curing resin for surface testing and impressions). Specimens were stored at −20°C until testing. Just prior to the experiment, all specimens were thawed to room temperature.

**Fracture Generation**

To simulate a traumatic sudden impact we decided to use a high-speed trauma model, modified from the techniques described by Panjabi et al. and Kallemeier et al.\(^10,14\) To simulate high energy, a metallic drop tower with a height of 1.7 m was built, allowing a load of 7 kg to be dropped onto a horizontally aligned impounder. The design of the drop tower allows us to change the weight as well as the posterior ligament complex. The vertebral bodies of T-11 and L-3 were stably embedded in Technovit 3040 (cold-curing resin for surface testing and impressions). Specimens were stored at −20°C until testing. Just prior to the experiment, all specimens were thawed to room temperature.

**Fracture Classification**

All fractured spine segments were classified by 2 experienced spine surgeons (L.O. and A.K.) using CT scans. The AO/OTA Classification was used to classify the fracture types.\(^12\)

**Stiffness Measuring**

To measure the instability of the created thoracolumbar burst fractures, stiffness was measured with a servo-hydraulic test bench (Bose Electroforce LM2 Test Bench). For testing deformation, the specimens were exposed to an axial load of 600 N (50 cycles, 1 Hz) before and after fracturing.

**Measurements of Vertebral Height**

Before and after creating the burst fractures, measurements of the vertebral body height of L-1 were performed by analyzing the CT scans. Vertebral heights were measured at the anterior and posterior vertebral walls as well as in the center of the vertebral bodies in the midsagittal plane of the vertebral body.

**Cobb Angle and Spinal Canal Compression**

The Cobb angle was measured in the midsagittal plane on CT scans, and spinal canal compromise was measured by analyzing the CT scans in the axial plane. Spinal canal compromise was measured as a percentage of the difference between the surface area of the spinal canal before and after fracture. Surface area was measured at the point of maximum compromise.

**Statistical Analysis**

For all parameters determined, the results are expressed as means and ranges ± SD. The test of significance
between results from study pairs was conducted by using the Student t-test and the Mann-Whitney test with significance p < 0.05.

**Results**

**Fracture Types**

The preparation and embedding of the spine segments worked well. No repositioning or renewal of the embedding was required, even after creation of the fracture. It was possible to create single burst fractures at the L-1 level in all 28 spine segments. A single drop of the weight was sufficient to create the fracture in all cases, and repetition was not necessary in any case. All fractures were classified according to the AO/OTA Classification. Basic characteristics of burst fractures include involvement of the posterior vertebral wall, reduction of the posterior height of the vertebral body, and some degree of narrowing of the spinal canal (Fig. 4).

Among the 28 fractures there were 16 incomplete burst fractures (A3.1), which were characterized by superior endplate destruction, depression in the anterior column, and shattering of the middle column into 3–5 fragments, as well as the typical fragment retropulsed into the spinal canal. There were also 8 burst-split fractures (A3.2) and 4 complete burst fractures (A3.3) (Table 1).

**Stiffness**

The average stiffness before fracture was 728.5 N/mm (± 144.9). The average stiffness after fracture was 386.4 N/mm (± 84.6) (Table 1), and this difference was significant on the Student t-test (p < 0.05).
After fracture the anterior, central, and posterior height of all L-1 vertebral bodies was significantly reduced. The average anterior height after fracture was 88.3% of initial prefraction height (range 68.2–97.7 [± 7.6]), the average central height after fracture was 72.1% of initial prefraction height (range 50.0–94.4 [± 10.9]), and the average posterior height after fracture was 86.9% of initial prefraction height (range 68.1–99.7 [± 6.1]). All postfraction parameters were significant in comparison with prefraction values for vertebral body height when assessed by the Mann-Whitney test (p < 0.05) (Table 1).

Measurements of the Cobb Angle and Spinal Canal Compression

The average Cobb angle after fracture was 6.3° (range −3.3° to 17.4° [± 5.2°]). The average spinal canal surface area of the unfractured specimens was 313.2 mm² (± 39.9 mm²) and of the fractured vertebrae 252.3 mm² (± 43.5 mm²). The average spinal canal compression was 19.5% (± 8.7%). These differences were significant as assessed by the Student t-test (p < 0.05) (Table 1).

**Discussion**

Acute thoracolumbar burst fractures seem to be one of the most common thoracolumbar lesions, especially in young patients.2–4,13,15 Acute traumatic burst fractures are classified according to the AO/OTA Classification12 as a subgroup of Type A fractures (A3).7 They present with involvement of the posterior wall of the vertebral body and often with spinal canal compromise.11 Type A3 burst fractures are subdivided into incomplete burst fractures (A3.1), burst-split fractures (A3.2), and complete burst fractures (A3.3). An intact posterior ligament complex7

**Table 1. Measurements and values for each fractured vertebral body specimen**

<table>
<thead>
<tr>
<th>Specimen No.</th>
<th>AO/OTA Fx Type</th>
<th>Post-Fx Vertebral Body Height (% of initial value)</th>
<th>Stiffness (N/mm) Pre-Fx</th>
<th>Post-Fx</th>
<th>Post-Fx Cobb Angle (°)</th>
<th>Area of Spinal Canal (mm²)</th>
<th>Spinal Canal Compromise (%)</th>
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Ant = anterior; Fx = fracture; pst = posterior.
(PLC) is characteristic of all Type A fractures. The treatment of burst fractures without neurological deficit remains controversial. Recommendations range from operative to conservative treatment. However, previous reviews have shown no superior results for operative treatment in comparison with conservative treatment at an average follow-up of 4 years.

Various operative treatments are described in the literature: posterior stabilization with or without decompression, anterior fusion, 360° fusion, and percutaneous instrumentation, as well as cement augmentation or a combination of techniques. In the US conservative treatment is often recommended if burst fractures are considered stable and if the PLC is intact. Treatment is guided by the Thoracolumbar Injury Classification and Severity (TLICS) score. In European countries burst fractures defined by involvement of the posterior wall of the vertebral body are surgically treated in most cases. These considerable differences in treatment underline the importance of understanding these fractures better.

Most recommendations for treatment are based on biomechanical considerations. Therefore, in vitro models in addition to clinical studies are necessary to evaluate the stability of the reconstructed spine. The creation of reproducible burst fractures seems to be the most common challenge for biomechanical studies, especially if the vertebral bodies were not dissected. For example, to evaluate posterior stabilization, it is necessary to have at least 2 connected motion segments and to create a burst fracture in the middle vertebral body without lesions in the upper and lower bodies. Several biomechanical studies have already established methods for creating thoracolumbar burst fracture in vitro; however, the numbers of specimens were small or animal specimens were used. Panjabi et al. described a high-speed trauma model in 1995. They used 16 human specimens, but did not indicate the level at which the fracture was created. Kallemeier et al. used a model similar to that of Panjabi et al. for producing thoracolumbar burst fractures. They were able to produce burst fractures in every segment, but only 9 specimens were investigated. Jones et al. created burst fractures of L-1 in 5 cadaveric specimens, also using a free-fall protocol. Hartensuer et al. described a protocol using an Instron Testing System with an axial load of 300 mm/second in 10 calf and 7 human specimens. In our study we decided to use a high-speed trauma model to simulate a traumatic sudden impact. Therefore, a modified system and protocol based on the ones used by Panjabi et al. and Kallemeier et al. was used. To ensure a burst fracture of L-1 only, stress risers were generated by cutting the superior end plate and lamina of L-1 prior to impaction, as described by Kallemeier et al. and Hartensuer et al. We were able to generate typical burst fractures in all 28 specimens (Fig. 2). Fractures appeared in the targeted L-1 without any lesions of the upper and lower vertebral bodies. We were mainly able to achieve incomplete burst fractures (Type A3.1), followed by burst-split fractures (Type A3.2), and complete burst fractures (Type A3.3). The latter group included only 4 cases. It is difficult to achieve consistent results when biological materials having different properties are used. A sudden impact is necessary for the creation of “traumatic” fractures. To compare different treatment methods, it is important to create a fractured vertebral body between unfractured vertebral bodies. We managed to create fractures at L-1 in every case. The distribution of fractures in our experimental series is comparable to the categories in the clinical series of burst fractures published by Altay et al. The distribution in their series was 61.9% for Type A3.1 fractures; 12.7% for Type A3.2 fractures; and 25.4% for Type A3.3 fractures. Similar percentages to these clinical values were obtained using our model.

In all fractures we were able to generate a significant loss of vertebral body height comparable to the results of Kallemeier et al. and Jones et al. Cobb angles as well as spinal canal compression were comparable to the results of these two studies as well.

Our study has some limitations. To create a single L-1 fracture, standard bone defects were necessary to create pre-stressing. Jones et al. criticized this type of model in their discussion. Their recommendation is a complete potting of the upper and lower vertebral bodies with rigid foam to get a single burst fracture. However, if further biomechanical investigation follows burst fracture generation, access to adjacent vertebral bodies is necessary, especially for testing posterior stabilization.

Despite these limitations this study presents, to the best of our knowledge, the largest number of cases of in vitro burst fracture generation in human fresh-frozen specimens. We created reproducible burst fractures of a single vertebral body in thoracolumbar spines. We were able to create burst fractures in all 28 specimens at a defined level (L-1) leaving the adjacent levels above and below intact, which makes the model very cost-effective. Our results may help substantiate those of previous studies and serve as a basis for further biomechanical studies. There are different ways in which this model can help us understand burst fractures better. Biomechanical studies on concomitant injuries (e.g., injuries to the PLC) would be possible. Most importantly, this model has the potential to evaluate different treatment strategies (e.g., anterior or posterior approaches and augmentation techniques) in a multilevel setting.

Conclusions

To our knowledge, this study is the first investigation to create a large number of burst fractures (AO/OTA Type A3) in multisegmental spine samples. The ability to create reproducible burst fractures of a single vertebral body in a thoracolumbar spine segment may serve as a basis for future biomechanical studies to develop and test treatment strategies for thoracolumbar burst fractures.

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Disclosures
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Author Contributions
Conception and design: Oberkircher, Krüger. Acquisition of data: Oberkircher, Schmuck, Bergmann. Analysis and interpretation of data: Oberkircher, Schmuck, Lechler, Krüger. Drafting the article: Oberkircher, Schmuck, Krüger. Critically revising the article: all authors. Approved the final version of the manuscript on behalf of all authors: Oberkircher. Statistical analysis: Oberkircher, Schmuck, Krüger. Administrative/technical/material support: Bergmann, Ruchholtz. Study supervision: Oberkircher, Ruchholtz, Krüger.

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