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Basic Science

Dynamics of interpedicular widening in spinal burst fractures: an in vitro investigation

Nicola Brandolini, MSc*, Nikil Kapur, PhD, Richard M. Hall, PhD

School of Mechanical Engineering, University of Leeds, Woodhouse Lane, Leeds LS2 9JT, UK

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Abstract

BACKGROUND CONTEXT: Spinal burst fractures are a significant cause of spinal instability and neurologic impairment. Although evidence suggests that the neurologic trauma arises during the dynamic phase of fracture, the biomechanics underpinning the phenomenon has yet to be fully explained. Interpedicular widening (IPW) is a distinctive feature of the fracture but, despite the association with the occurrence of neurologic deficit, little is known about its biomechanics.

PURPOSE: To provide a comprehensive in vitro study on spinal burst fracture, with special attention on the dynamics of IPW.

STUDY DESIGN: Experimental measurements in combination with computed tomography scanning were used to quantitatively investigate the biomechanics of burst fracture in a cadaveric model.

METHODS: Twelve human three-adjacent-vertebra segments were tested to induce burst fracture. Impact was delivered through a drop-weight tower, whereas IPW was continuously recorded by two displacement transducers. Computed tomography scanning aided quantifying canal occlusion (CO) and evaluating sample anatomy and fracture appearance. Two levels of energy were delivered to two groups: high energy (HE) and low energy (LE).

RESULTS: No difference was found between HE and LE in terms of the residual IPW (ie, post-fracture), maximum IPW, or CO (median 20.2%). Whereas IPW was not found to be correlated with CO, a moderate correlation was found between the maximum and the residual IPW. At the fracture onset, IPW reached a maximum median value of 15.8% in approximately 20 to 25 milliseconds. After the transient phase, the pedicles were recoiled to a median residual IPW of 4.9%.

CONCLUSIONS: Our study provides for the first time insight on how IPW actually evolves during the fracture onset. In addition, our results may help shedding more light on the mechanical initiation of the fracture.

Keywords: In vitro biomechanics of the spine; Dynamics of spinal burst fracture; Dynamic interpedicular widening; Canal occlusion; High resolution peripheral quantitative computed tomography; Fracture of the pedicle; Laminar fracture

Introduction

Burst fractures account for about 30% of all spinal injuries [1] and are a cause of severe neurologic impairment and spinal instability [2]. Approximately 47% of cases present with a degree of neurologic deficit at the time of admission [3].

The onset of the fracture is usually traumatic and arises from a high-energy axial impact loading, commonly because of fall from heights and motor accidents [4]. The main features of the fracture are comminution of the end plates, loss of vertebral height, disruption of the posterior ligamentous complex, retropulsion of bony fragment into the spinal canal (fragment retropulsion), laminar fracture (LF), and widening of the interpedicular distance [1,3,5].

Canal occlusion (CO) caused by FR has been shown to be a significant risk factor of neurologic deficit [6]. However, CO alone appears not to fully explain the extent of the neurologic deficit [3,7]. Further additional insight

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* Corresponding author. Institute of Medical and Biological Engineering, School of Mechanical Engineering, University of Leeds, Woodhouse Lane, Leeds LS2 9JT, UK. Tel.: (44) 1133439408.

E-mail address: n.brandolini@leeds.ac.uk (N. Brandolini)

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into the generation of burst fractures can be accrued from the fact that neurologic deficit has been diagnosed in 68% of the patients with disruption of the posterior elements [8], while dural tears have been detected in 25% of low lumbar burst fractures [9], and their occurrence has been shown to be strongly associated with interpedicular widening (IPW) and LFs [10,11].

The clinical relevance of IPW has been also confirmed by Caffaro and Avanzi [12], where an approximately 25% widening has been found to be associated with a 51% probability of presenting neurologic deficit. Ultimately, assessment of posttraumatic IPW may provide a more time and cost-effective diagnostics because it can be better quantified on plain radiographs than spinal occlusion [13].

However, the real drawback in the diagnosis of any burst fracture caused impairment is that the actual injury originates during an extremely abrupt transient phase that cannot be quantified retrospectively. Hence, the need for more understanding on the dynamic biomechanics of burst fracture is paramount. Several in vitro studies have indeed shown that the maximum CO occurs during the onset of the fracture [14–18].

In addition, further biomechanical studies have suggested that the root of the pedicles is the site of initiation of burst fracture. Both in vitro [19] and numerical simulations [20] have detected significant strain concentration at the base of the pedicles. In Langrana et al. [21], the fracture initiation process has been demonstrated to be driven by the forces that originate at the pedicles when the superior facets wedge within their own adjacent joints. Unlike the dynamics of CO, which has been the subject of several biomechanical studies, IPW has not been investigated in a manner that would provide a greater understanding of the fracture process and aid its use in clinical diagnoses.

Therefore, the aim of this work was to investigate, for the first time, the dynamics underlying the behavior of the facet joints and pedicles during the generation of a spinal burst fracture. In addition, high-resolution peripheral quantitative computed tomography (HR-pQCT) was exploited to provide a comprehensive view of the phenomena, pre- and post-fracture.

Materials and methods

Specimen preparation

Four human spines were acquired after the ethics committee approval from the Leeds Tissue Bank (Leeds Teaching Hospitals Trust, Leeds, UK). Three, three-adjacent-vertebra segments (T9–T10–T11, T12–L1–L2, and L3–L4–L5) were harvested from each spine for a total of 12 specimens (Table 1). Care was taken to preserve the intervertebral discs, the principal ligamentous structures, and the integrity of the superior and inferior facet joints adjacent to the central vertebra. No alterations were performed to any of the vertebra to force the occurrence and appearance of a burst fracture.

The cranial and caudal vertebrae of each segment were partially embedded in polymethyl methacrylate (WHW Plastics, Hull, UK) to provide two flat parallel loading surfaces and consistently align the specimen within the testing rig. To this end, a stainless steel rod was firmly clamped against the posterior wall of the most distal vertebrae to firmly hold the sample in place while being embedded. The location of the rod within the canal was adjusted to make the superior and inferior rims of the central vertebral body as parallel to the ground as possible. The most anterior region of the central vertebra and its spinous process were used as references to define the sagittal plane of the segment that was aligned with the center lines of the potting molds.

Experimental protocol

A custom testing rig was designed to fit within a drop-weight tower (Fig. 1). Hence, to simulate an axial impact load, a weight was dropped down a guide rod

Table 1
Details of the donors together with the details of each specimen

Donor	Level	Age (y)	Height (m)	BW (kg)	Gender	BMD, mg HA/cm^3	PA (°)		CA_0 (mm^2)	l_0 (mm)
							Left	Right		
A	T9–T10–T11	44	1.60	55.0	F	148.3	4.8	−0.5	174	31.5
A	T12–L1–L2	44	1.60	55.0	F	138.6	22.8	5.2	350	44.3
A	L3–L4–L5	44	1.60	55.0	F	100.5	6.1	17.1	461	51.1
B	T9–T10–T11	46	1.70	89.5	M	150.6	5.6	8.9	201	29.6
B	T12–L1–L2	46	1.70	89.5	M	156.2	16.1	8.8	360	44.6
B	L3–L4–L5	46	1.70	89.5	M	143.5	14.2	21.1	305	49.8
C	T9–T10–T11	56	1.73	70.0	M	128.0	12.3	10.7	203	35.6
C	T12–L1–L2	56	1.73	70.0	M	98.5	21.2	16.2	273	46.7
C	L3–L4–L5	56	1.73	70.0	M	111.0	19.5	19.9	246	53.0
D	T9–T10–T11	38	1.75	85.6	M	191.9	14.0	9.0	214	32.6
D	T12–L1–L2	38	1.75	85.6	M	184.5	14.5	20.2	314	44.6
D	L3–L4–L5	38	1.75	85.6	M	161.7	37.4	35.8	258	55.7
Median		45	1.72	77.8	—	145.9	14.3	13.4	266	44.6

BMD, bone mineral density; BW, body weight; CA, canal area; F, female; HA, hydroxyapatite; l_0, initial interpedicular distance; M, male; PA, pedicle angle.