BIODYNAMICS OF CERVICAL SPINAL INJURY

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ABSTRACT

Using a new tool developed in our laboratory to quantify local pressure changes on an artificial spinal cord, quantitative results correlating fracture/soft tissue pathology with cord pressure changes, was accomplished. Human cadaveric head-neck complexes were dynamically loading by fixating the inferior of the preparation and removing the cadaver spinal cord The instrumented artificial spinal cord was inserted into the spinal canal. Under dynamic axial loading rates, compression injuries including burst and wedge fractures in the middle column were produced. Cord pressures at levels with minor injuries were 35 to 75 N/cm^2 and unaffected (non-failure) levels were generally low from 0 to 30 N/cm^2. Cord pressures at levels with significant fractures or dislocations were much higher from 50 to 200 N/cm^2.

SURVIVORS OF SPINAL CORD INJURY experience significant physical and psychological changes. The extensive long-term medical treatment and rehabilitation of spinal cord injury patients continues to generate large individual and societal costs. Spinal cord injuries are produced through a variety of scenarios including falls, diving, athletic related events, and motor vehicle crashes [18]. Although hypotheses are available for the mechanism of cervical spine fracture-dislocation, there is a lack of experimental evidence and correlation of fracture-dislocation with spinal cord damage.

The link between the biomechanics of spinal column injury (i.e., bony fracture/dislocation) and spinal cord injury is still in the initial stages. From clinical experience, it is known that survivors of spinal cord injury present in numerous scenarios. There may be significant bony injury without concomitant neurologic deficit. There may be significant neurologic loss without significant signs of bony instability. Although the spectrum is wide, a significant percentage of spinal cord injury survivors present with some type of compressive bony fracture corresponding to the nearest neurologic level of the spinal cord. For example, a patient with a burst fracture at the C5 body may likely have a C6 quadriplegia. There are others however, that may have significant bony injury without concomitant neurologic deficit.

Studies of spinal cord injury have been varied, but the greatest number of experimental studies on animals have incorporated the dorsal impact method first shown by Allen in 1911 [2]. This experimental technique induces injury by dropping a weight from a specified height onto the exposed cord of the animal. The potential energy of the impact can be correlated to the resulting neurologic deficit. The main purpose of the majority of these studies however, was to evaluate various treatment protocols [4-8,10-13, 15, 16, 19, 20, 26]. This method does produce a consistent cord injury but is not intended for the study of trauma due to fracture/dislocation. There are numerous other studies that have investigated the mechanisms of fracture/dislocation of the cervical spine [1, 3, 9, 14, 17, 21-25, 27-30, 32-34, 37-41]. These studies however, were not intended to examine potential for cord injury. The present investigation was designed to study both fracture/dislocation in the spinal column and the probability of spinal cord injury in one model. The purpose of the present study therefore, was to quantify spinal cord pressure changes during dynamic spinal column injury.

METHODS

An instrumented artificial spinal cord was developed [31]. It included seven 1 mm thick piezoelectric pressure sensors adjacent to a collagen encased gelatin substitute of the spinal cord. The normalized dynamic force-deflection characteristics of *in vivo* feline spinal cords were obtained using a weight drop technique. Compressive responses were physically modeled by varying the gelatin mixture until the appropriate characteristics were obtained. The physical model response therefore, is representative of all structures contained in the canal (i.e., cord, dura, CSF, fat, etc). The physical cord, with the transducer array are fit snug into the spinal canal.

A dorsal impact method to apply dynamic compression to the exposed cord of anesthetized feline preparations was used to obtain the normalized force-deformation response. The artificial cord properties were matched to this specification by adjusting the gelatin mixture appropriately. The transducer array was constructed from a thin-film piezo-electric material sensitive to pressure applied normal to the surface. Seven 1 cm^2 sensors were constructed and spaced approximately 15 mm apart in a water-tight seal. The transducer array and artificial cord were placed together such that the transducers contacted the anterior wall of the spinal canal of the cadaver specimen.

Ten human cadaveric head-neck complexes with an average age of 65 years were prepared for dynamic. The inferior of the preparation, T1-T2, was embedded in poly-methyl-methacrylate (PMMA) and mounted to the frame of the test apparatus. The cadaver spinal cord was removed and the instrumented artificial spinal cord was inserted into the spinal canal through an inferior access hole and craniotomy. Pre-radiography was taken to establish locations of each transducer with respect to anatomical landmarks (Figure 1). The head-neck preparation was pre-aligned by using a series of pulleys and dead-weights to position the spine such that the cervical lordosis was removed. Loading was accomplished through the piston of an electrohydraulic test apparatus (MTS Systems Corporation, Minneapolis, MN) to the top of the cranium. A 10 cm square aluminum plate with 15 mm of ensolite padding acted as the impacting surface. The specimens were tested at one of two dynamic loading rates, 300 or 600 cm/s. Instrumentation
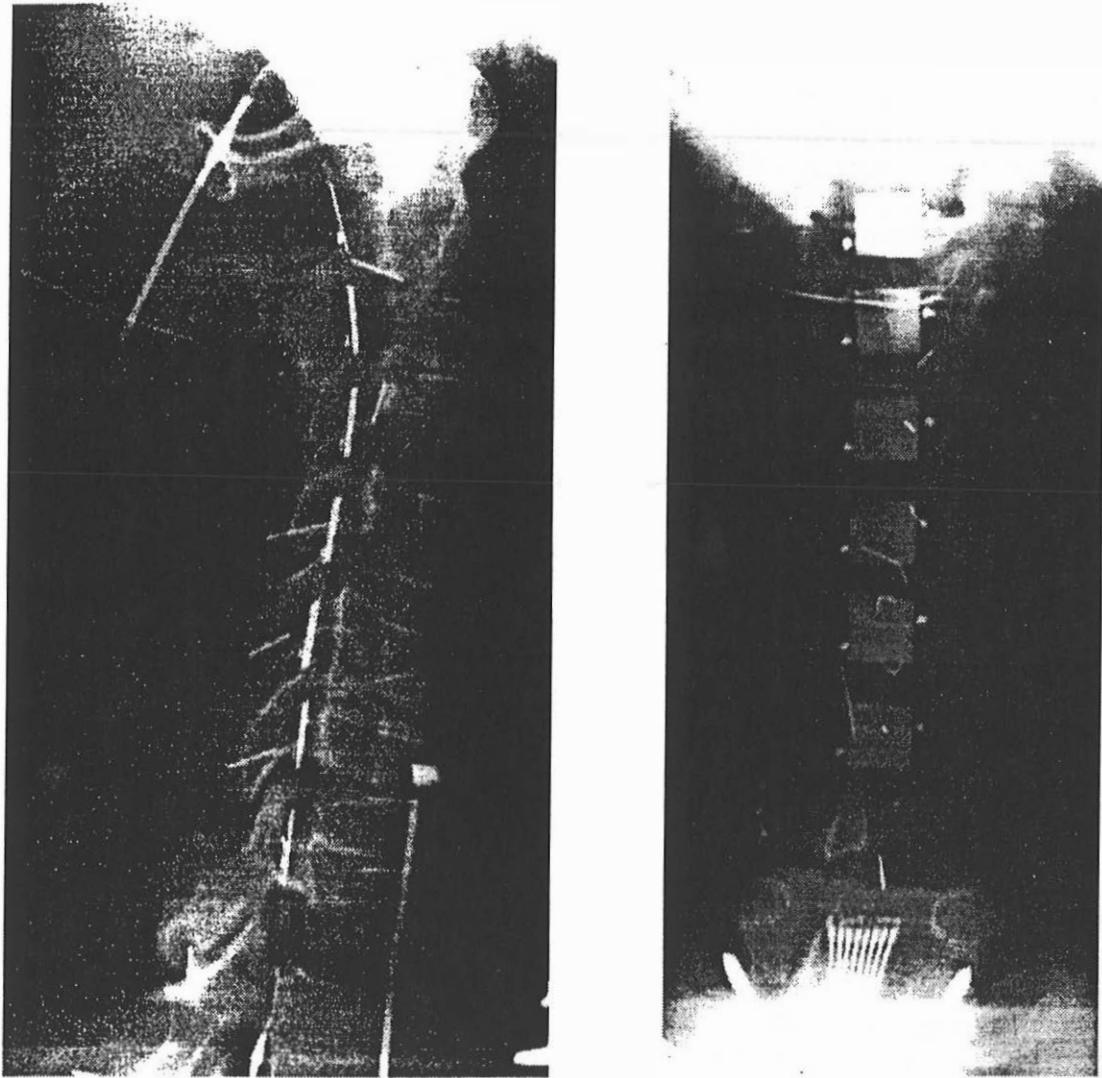
Figure 1:   Radiographic representations of a specimen with inserted instrumented artificial spinal cord. The left picture is the lateral view and the right picture is the anteroposterior view. The square shapes are the 1 cm^2 transducers.

included accelerometer and load cell on the piston, a triaxial accelerometer on the cranium, a uniaxial accelerometer on the mastoid process of the skull, a uniaxial accelerometer glued to C4, and a six-axis load cell mounted in line with the base of the preparation. All transducer data were recorded with a digital data acquisition system sampling at 8 kHz according to SAE J211b specifications. Each level of the cervical column had retroreflective markers fixed to the vertebral body, lateral mass, and spinous process. High-speed photography documented the motion of the markers during the test.

RESULTS

Each of the columns failed in one of three predominant injury mechanisms: direct compression, flexion-compression, extension-compression. Burst and wedge compression fractures of the mid column (C4, C5, C6) occurred for direct compression mechanism and flexion-compression mechanism. Anterior ligament and disc injuries occurred for specimens failing in extension-compression. On a general basis, the more severe the spinal column injury, the higher the local pressure change observed from the instrumented artificial spinal cord. Cord pressures at levels with minor injuries were 35 to 75 N/cm^2 (Figure 2) and unaffected (non-failure) levels were generally low from 0 to 30 N/cm^2. Cord pressures at levels with significant fractures or dislocations were much higher from 50 to 200 N/cm^2 (Figure 3). On one occasion however, a direct compression fracture of the C5 vertebral body produced very low pressure changes. Specimens loaded at the higher rate of 600 cm/sec demonstrated greater impact forces from 4.6 kN to 9.0 kN whereas, specimens tested at 300 cm/sec produced impact forces from 2.1 kN to 4.3 kN. The maximum compressive force recorded from the inferior load cell however, was more uniform between the two loading rates and resulted in a mean of 2.8 kN. Failure of the column was defined as the first significant drop in the vertical force from the inferior load cell with continuing compressive displacement. Compressive displacements to failure ranged from 14 to 27 mm.

DISCUSSION

The human cervical spine is a mechanical structure which transmits the load from the cranium to the other regions of the human body. The principal functional purpose of the cervical spinal column is to maintain the load-carrying capacity as well as preserve the structural integrity of the cervical spinal cord. The articulated inhomogeneous cervical spine structure consists of seven vertebrae, connected by intervertebral discs from C2 to C7 and the apophyseal joints, interlaced with the various soft tissues including ligaments and muscles. The mechanism of compressive load transfer principally takes place through the occipital condyles of the cranium to the vertebrae, intervertebral discs, and the ligaments. Because of the variations in the mechanical characteristics, the nature and magnitude of the deformations of the tissues that have the potential to induce spinal cord injury are not identical under a given loading situation [37-39]. In other words, the intrinsic load-carrying capacities of these components differ. It is important to understand the biomechanical characteristics of the spinal column components to determine the mechanism of related injury to the cord.
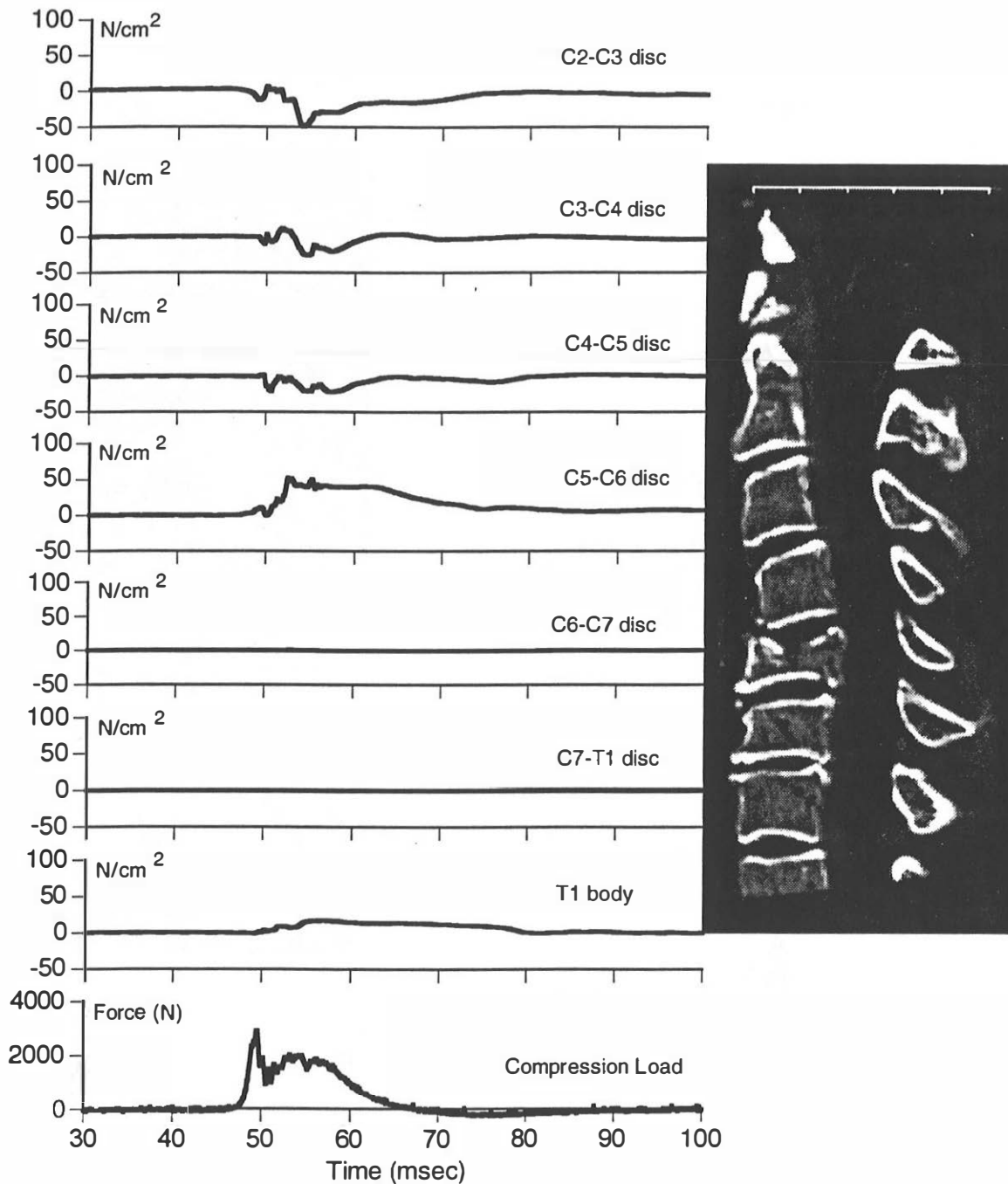
Figure 2: Pressure trace recordings from the instrumented artificial spinal cord measured at the various spinal locations. The bottom trace is the vertical compression force from the inferior six-axis load cell. On the right is a sagittal plane CT image of the specimen post-test. Note the compression fracture of the C5 vertebral body, but with minimal residual encroachment into the spinal canal. Compared to the specimen in Figure 3, there would be a lower probability of spinal cord injury in this preparation.
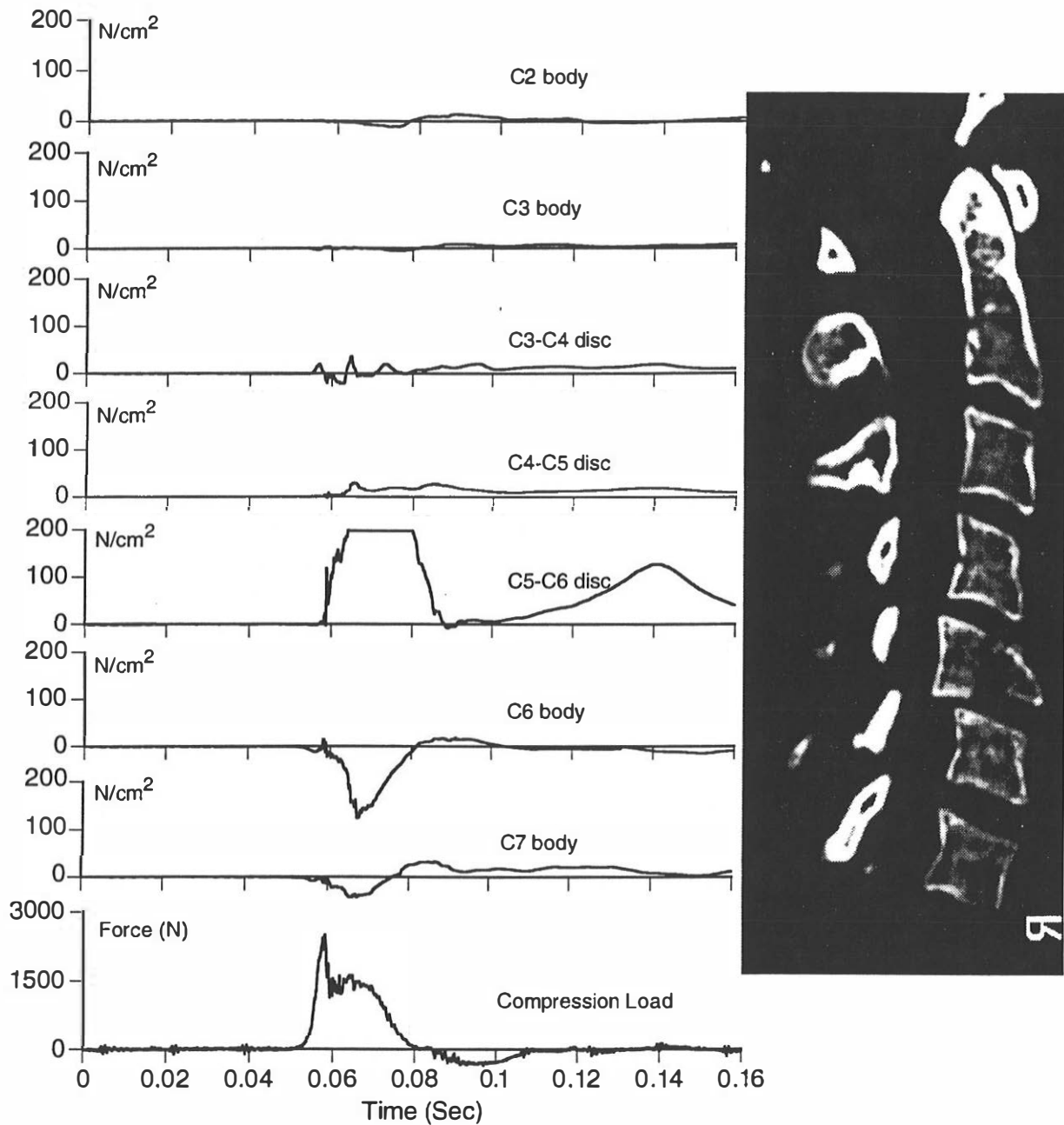
Figure 3: Pressure trace recordings from the instrumented artificial spinal cord measured at the various spinal locations. The bottom trace is the vertical force from the distal six-axis load cell. On the right is a post - test CT taken in the sagittal plane. Note the burst fracture at C5 and the resulting pressure traces that are extremely high in the area of the fracture site. At the C4-C5 disc level the amplifier setting for the transducer was not set high enough and clipping occurred. The instrumented artificial spinal cord quantifies what the cord experiences during impact. There is a high probability that spinal cord injury would have occurred under this mechanism.

Of necessity, the human cervical spinal column has to resist physiologic as well as traumatic forces. Under these conditions, it is important to maintain the normal functional relationships between the bony elements of the spine and the cervical spinal cord. Clinical studies indicate that, under traumatic situations such as surviving victims of motor vehicle crashes, cervical injuries such as wedge or burst compression fractures are often associated with neurologic deficit [36]. These injuries have significant societal costs. Epidemiological studies indicate that cervical injuries unaccompanied by significant bony trauma may also result in cord injury which can render the patient a complete or incomplete quadriplegic. Recent epidemiological studies using computerized National Accident Sampling System Data (NASS) files, clinical data from an urban population for the years 1979 through 1986, and the fatalities from a similar community, have indicated the following mechanisms regarding cervical spine injury: transmission of the dynamic force from the head to the neck results in cervical trauma; commonly seen cervical injuries in surviving victims of motor vehicle crashes can be classified into compression/axial loading, compression-flexion, compression-extension, and locked facet mechanisms; the fatal victims in motor vehicle crashes sustain upper cervical spine injuries routinely at the occiput-C1-C2 level (such as atlanto-occipital dislocation); in contrast, the surviving victims sustain injuries to the mid-lower cervical spine regions [35, 36]. Because of the involvement of the cervical spinal cord in producing the neurologic deficit, it is important to understand the biomechanics of the cervical spine from both the vertebral column (bony architecture) as well as from the spinal cord perspective [7, 31, 35].

Using a new tool developed in our laboratory to quantify local pressure changes on an artificial spinal cord, quantitative results correlating fracture/soft tissue pathology with cord pressure changes, were accomplished. Commonly seen clinical injuries were produced under compression-flexion, compression-extension, and direct axial compression loading mechanisms. Severe wedge and burst fractures with bony fragments in the spinal canal produced extremely high local cord pressure changes at the site of bony injury. There were sometimes even negative cord pressures recorded adjacent to high positive pressures (Figure 3). This is due primarily to the construction of the sensor array which is pressed posteriorly by the incoming bone fragment on one sensor level and the adjacent sensor actually experiences a negative pressure wave as it is drawn away from the anterior wall. Mild compression fractures and extension-related trauma without significant subluxation produced consistently low cord pressure changes.

One preparation that failed under direct compression produced a significant C5 compression fracture, but resulted in very low cord pressure changes. This event may explain the more rare clinical case where significant bony fracture does not produce neurologic deficit. Examination of the kinematics of the spine during impact revealed the greatest difference between this test and a similar one that produced high cord pressures, was the resulting flexion of the column after initial compression fracture of the vertebrae. This local flexion of the column appeared to have enhanced the protrusion of the bone fragments into the spinal canal, causing the high local pressure changes. Additional studies with this experimental method will lead to an understanding of the injury tolerance of the human spinal cord correlated to the injuries produced in the spinal column.

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