

Review

Biomechanics of the cervical spine 4: major injuries

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Abstract

This review presents considerations regarding major cervical spine injury, including some concepts that are presently undergoing evaluation and clarification. Correlation of certain biomechanical parameters and clinical factors associated with the causation and occurrence of traumatic cervical spine injuries assists in clarifying the pathogenesis and treatment of this diverse group of injuries. Instability of the cervical column based on clinical and mechanistic perspectives as well as the role of ligaments in determining instability is discussed. Patient variables such as pre-existing conditions (degenerative disease) and age that can influence the susceptibility or resistance to injury are reviewed. Radiological considerations of major injuries including dynamic films, CT and MRI are presented in the diagnosis and treatment of cervical trauma. Specific injury patterns of the cervical vertebral column are described including attention to the relative mechanisms of trauma. From a biomechanical perspective, quantification of injury tolerance is discussed in terms of external and human-related variables using laboratory-driven experimental models. This includes force vectors (type, magnitude, direction) responsible for injury causation, as well as potential influences of loading rate, gender, age, and type of injury. © 2002 Elsevier Science Ltd. All rights reserved.

1. Introduction

The categorization of injuries of the cervical spine into a “major” occurrence implies that the structural compromise of the cervical vertebral column has caused neurologic injury (spinal cord or nerve roots), or created an environment where instability offers a significant potential threat to spinal cord integrity. Numerous classifications of injury patterns have been presented but none has achieved widespread acceptance. A variety of factors contribute to this lack of unanimity of opinion including differences in interpretations of biomechanical studies, the frequent presence of varying mitigating or contributing processes, as well as potential clinical limitations in fully defining the injury patterns. The present discussion will, therefore, be mainly directed at determining the important clinical factors in defining the character of such major cervical vertebral injuries.

Numerous laboratory studies have assisted in clarifying the interrelationship between the various biomechanical parameters (rate of force application,

magnitude of force application, and vector) with the majority of investigations using “pure” moment force application (flexion, extension, rotation, axial, and lateral bending) to cause selective patterns of vertebral column injury. Other investigators have chosen to use complex loading such as combined flexion–compression, usually dynamically applied in order to more closely replicate the clinical situation. A more detailed analysis of the selective attributes of biomechanical methodology will be discussed in a separate part of this series and, although a few areas of overlap will occur, the present emphasis is directed to the clinical factors that influence cervical spine injury patterns.

Clarification of these potentially important clinical elements will both assist in designing preventive measures and treatment options. This latter consideration has special importance in helping the treating physician to avoid exacerbating causative injury forces by reapplication of the predominant injury vector with potential accentuation of instability and risk of neurologic compromise. The type and degree of cervical column injury may be influenced by clinical factors which are deduced from a variety of historic and radiologic evidence. The methodology of the clinical studies differs in comparison to laboratory investigations which are controlled and carefully observed, whereas, the clinical situation is

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uncontrolled and frequently unobserved. This paper provides insight into both clinical and laboratory investigations delineating the biomechanical aspects of major neck injuries.

2. Instability

The majority of acute injuries will have their basis in acute changes in column stiffness (fracture, dislocation, ligament compromise, etc.) with instability. Instability has been defined in anatomical, biomechanical, and clinical terms, but the variability of clinical presentations and the inability of sequential laboratory lesions to replicate clinical experiences indicates that instability should be defined in anatomical terms. The previous sections discussed reliance upon the integrity of the various spinal components for normal motion patterns of the column and the resultant interaction with the spinal cord. Mechanical compromise of the various spinal elements may result in deformation, either acute or chronic, of the cervical spine under physiologic loads. Larson has emphasized that the stable spinal column is symmetric in movement and configuration, whether normal or abnormal, and does not change with time. In considering spinal instability, it is necessary to generally review the column theory of vertebral stability [1,2]. The two- or three-column concepts of Holdsworth, White, Denis, and Louis are frequently used to conceptualize the mechanical integrity of the spinal column [3–6]. The definition of the extent of injury to soft tissue (ligaments, disc, etc.) and bony components of a column will assist the clinician in determining the risk to neural structures from alterations in curvature or alignment. Although inclusion of a middle column has a theoretical advantage in thoracic and lumbar injuries, these considerations are not as anatomically important in the cervical area and it is reasonable to use the two-column model in the cervical region (Fig. 1). As previously noted, laboratory studies of cervical instability determined through a sequential pattern of component section of a specific column are not usually representative of clinical injury which may include only selective elements of each column [7]. The degree and character of a column compromise, however, are important indicators alerting the clinician to the potential risk of instability. Additionally, this information, especially the potential role of ligament compromise, will assist in recognizing the possible development of delayed instability (subacute or chronic) which may be difficult to establish in the early post-trauma period. The degree of instability, irrespective of temporal occurrence, will be an important influence upon the threat or occurrence of neurologic injury and, therefore, clarification of the degree of component injury and a more precise measurement of displacement will assist in determining the threat to neurologic integrity.

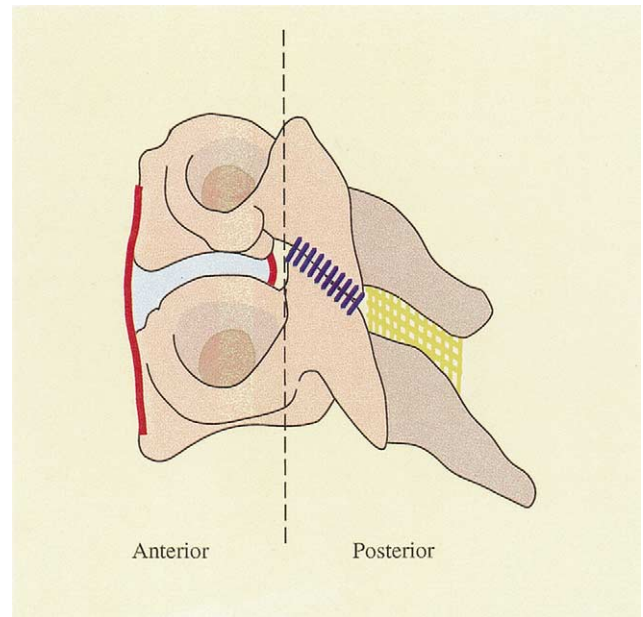


Fig. 1. The two-column model of the cervical region. Anterior column consists of vertebral bodies, intervertebral discs, and anterior and posterior longitudinal ligaments, whereas, the posterior column consists of zygapophysial joints, capsular ligaments, spinous processes, and lamina and interspinous ligaments.

3. Classification

A descriptive analysis of the local motions and forces acting upon the cervical spine has yielded a general consensus regarding the classification of these injuries and, in conjunction with the magnitude and vector of the force, the rate of force application is the third essential determinant of cervical spinal column injury [8–13]. The viscoelastic structures (ligaments and discs) are able to absorb greater levels of force at lower velocities of force application, but these structures will stiffen at higher rates with a decreasing capacity to absorb energy through deformation. Compromise of ligament integrity may result in mechanical instability with or without associated bone injury and the diagnostic difficulties of this occurrence will be discussed in the subsequent sections. The following classification for major injury patterns is proposed as a general guideline based upon load direction and load positioning with the understanding that other investigations may have certain inclusions or exclusions. Fig. 2 illustrates that a vector directed mainly in flexion will result in compression of the anterior components and distraction of the posterior elements, whereas, the reverse pattern of force application will occur with extension. The combining of axial compression or the less commonly occurring mode of distraction will potentially significantly magnify the forces acting upon the various segmental components and will be further detailed in evaluating injury patterns. As increased motions in the sagittal plane are applied,

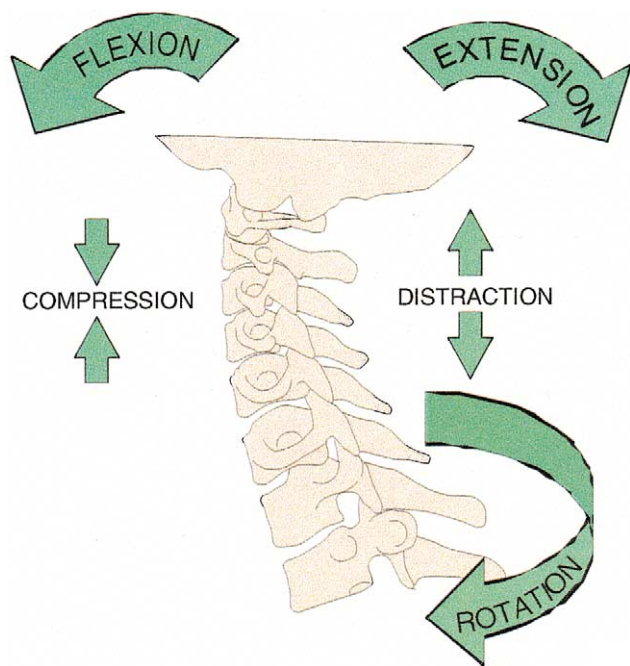


Fig. 2. Illustration of force vectors that may act upon the cervical spine.

shear forces (anterior–posterior translation) will induce additional changes, especially as a motion arc assumes increased magnitude (Fig. 2). The cervical spine is bounded by the head and thorax, and usually incurs injury from forces conducted in a cephalocaudal direction through the occipital condyles following head impact [14–18]. Inertial forces not associated with head or thorax impact are, therefore, unlikely to cause vertebral component compromise sufficient to elicit a major injury, especially fracture of bone elements [19–23]. Harris et al. [11] proposed that since the anterior and posterior columns are simultaneously affected in a reciprocal manner (hyperflexion causes compression in anterior column and distraction in posterior column, whereas hyperextension causes distraction in the anterior column and compression in the posterior column), it is not necessary to include the defining term of compression or distraction. This conceptualization, however, does not acknowledge the consistent experimental requirement for an external compression force to produce fracture patterns [22]. The terms compression and distraction, therefore, supply information regarding external load application in conjunction with load direction which is necessary to produce the component alterations in severe cervical spinal column injuries. The predominant vector, however, may assume a different direction at various levels. A cadaver study of high acceleration injuries produced a pattern of contiguous and non-contiguous injuries, but also illustrated that these associated alterations occasionally demonstrated structural changes at separate levels indicative of a different direction of

load application [24]. The alterations at the principal site, therefore, may change local motions inducing a different load vector in the distal load transmission.

To further define the mechanical determinants of cervical tolerance, consideration of certain patient variables that will influence the susceptibility or resistance to injury are necessary. Table 1 lists a number of these possible variables and is acknowledged not to be totally inclusive. These changes in the physiologic integrity of the spinal column or anatomical relationships may change the expectations regarding the mechanical determinants of cervical spinal column tolerance and require consideration in defining the risk of cervical column compromise.

4. Patient variables

Table 2 lists a number of possible variables that may influence the occurrence and extent of cervical column injury. Except for the agreement that cervical motion varies and decreases with age, presently, the clinical impact of age and gender in acute cervical injuries can only be hypothesized. A few laboratory studies offer some information regarding these factors. These are discussed in a later section entitled determination of quantified neck injury threshold.

Degenerative disease, especially significant radiologic evidence of spondylotic degeneration (SD) in the aging population, deserves emphasis. The influence of SD on the cervical column has the dual consideration of changes in the mechanical character and the morphology of the column [24,25]. As previously mentioned, laboratory studies on high acceleration injuries of the cervical spine demonstrated that the most severe levels of injury occurred adjacent to the regions of maximal degenerative change [24]. The increased local stiffness at the regions of most significant degeneration may result in increased motions and possible excessive deformations at neighboring, less involved levels. Recent clinical and radiographic studies of patients over 65 years with cervical spondylotic myelopathy (CSM) have shown the progression of myelopathy was frequently the result of increased motion at middle cervical levels (C3–C4 and C4–C5) in association with multilevel spinal stenosis most severely affecting lower cervical levels (C5–C6 and C6–C7) [26,27]. Therefore, such patients subjected to high acceleration forces, may incur increased local motions at these middle levels with resultant adverse stresses acting upon the spinal cord with potential traumatic myelopathy. These considerations, taken in conjunction with SD, may define a circumstance in which the spinal cord can be subjected to deleterious forces without incurring fracture or notable dislocation. Variation in injuries, therefore, can be anticipated such as marked increased shear stresses in the central cord

Table 1
Classification of injury (Mechanistic)

Axial compression
comminuted fracture of C1 (Jefferson)
vertical or oblique fractures (burst) of axis
comminuted fractures of vertebral bodies (burst)
Flexion(hyper)
instability (ligamentous)
Flexion–shear
odontoid fracture with posterior displacement
atlanto-axial instability from transverse atlantal ligament (TAL) compromise
Flexion–compression
vertebral body fractures (wedge, tear drop)
compromise of posterior ligamentous complex
Flexion–distraction
bilateral facet dislocation
Flexion–rotation
unilateral facet dislocation
Extension(hyper)
maintaining instability (ligamentous)
Extension–distraction
spondylolisthesis of C2
anterior C1 fracture
occipital–cervical (O–C) dislocation
Hangman’s fracture
Extension–compression
anterior longitudinal ligament and anular compromise
vertebral arch fracture (lamina, articular pillar, spinous process)
vertical vertebral body fracture
Extension–shear
odontoid fracture (anterior displacement)
posterior atlanto-axial dislocation (without fracture)

Rotation and lateral flexion injuries are not included because of the rare association with “major” injury situations.

during the pincer action compromise of the spinal cord incurred during a sudden forceful hyperextension. In this setting, a major spinal cord injury may occur in a patient with corresponding congenitally shallow canal or more commonly severe SD with resultant canal stenosis. The process of spinal cord injury associated with SD when the column undergoes rapid hyperextension (beyond physiologic limits) with resultant shortening of the cross-sectional area of the spinal canal and widening of the spinal cord permits a potential pincer-like compression of the spinal cord. The forces will act in a centripetal manner having the greatest potential effect on more centrally located spinal cord structures. The somatotopic structure of major motor tracts and the compromise of internuncial fibers will frequently cause a neurologic pattern of injury exhibiting a more profound motor impairment in the upper extremities, especially the hands, than in the lower extremities (central cord syndrome).

Osteopenia refers to decreased bone mineral content, and osteoporosis is defined as the parallel loss of both

bone mineral and matrix. The possible underlying cautions listed in Table 2 are conditions of physiologic bone loss that can render residual mineralized bone inadequate to withstand minor levels of trauma. This susceptibility to injury has been well recognized in the thoracic and lumbar regions, but such quantified and correlated information is not available for the cervical spine. In a like manner, certain endocrine-metabolic disturbances (hyperparathyroidism, Cushing’s disease, etc.) may alter the mineral content as well as varying loss of cortical and trabecular bone. The resultant risk of mechanical compromise at lower force levels may be encountered, but for the cervical region, the information is not presently adequate to clearly define the increased risk factors with these entities.

Certain of the arthritides, however, present conditions which have an acknowledged increased risk of injury in the cervical area. This consideration is especially valid for ankylosing spondylitis (AS). The spine appears stiff as a result of the multilevel ankylosis, but the strength in shear and flexion loading is decreased

Table 2
Variables potentially affecting injury tolerance

A	Age
B	Gender
C	Degenerative diseases
	1. Spondylosis degeneration
	2. Osteopenia
	(a) senile (>70 years)
	(b) postmenopausal (usually menopause before age 45)
	(c) hypogonadal (low gonadotrophins)
	(d) glucocorticoid induced
	(e) vitamin D deficiency
	(f) aluminum bone disease (dialysis, TPN)
D	Arthritis
	rheumatoid, ankylosing spondylitis (AS), diffuse idiopathic skeletal hyperostosis (DISH)
E	Endocrine
	hyperparathyroidism, hypoparathyroidism, acromegaly, Cushing's disease
F	Congenital (genetic)
	osteogenesis imperfecta, Down syndrome, sickle cell disease, lipid storage disorders, Klippel-Feil, atlanto-occipital fusion, agenesis of odontoid, os odontoidism, spondyloepiphyseal disease, achondrodysplasia

because of the brittleness of the spine. The ankylosed spine has a marked reduced capability to deform and absorb forces applied to the column, and, therefore, is increasingly susceptible to injury from low-force applications [28]. The patients incurring spinal column injury tend to be older men with advanced disease and resultant neurologic deficits, and mortality is high with some estimates approaching 35%. Loading factors will result in the majority of such injuries occurring in the thoracolumbar regions, but catastrophic cervical injuries are well known [29,30]. Inclusion of individuals with radiographic findings of diffuse idiopathic skeletal hyperostosis (DISH) as having an increased susceptibility to injury has been proposed, but presently no firm evidence confirms this impression.

A variety of relatively unusual congenital disorders, either through changes in anatomic configuration (Klippel-Feil, atlanto-occipital fusion, odontoid agenesis, etc.) or alterations of bony and ligamentous integrity (Down syndrome, osteogenesis imperfecta, etc.) of the cervical column, may reduce tolerance to applied loads. The clinical presentation will vary with realization that fused segments (Klippel-Feil, atlanto-occipital fusion) may result in altered caudal transmission of applied loads as well as potentiating ligamentous attenuation or tearing. The fulcrum/hinge concept is joined with potential decreased ligamentous integrity and increased potential for instability. The congenital changes in the odontoid (agenesis, os odontoidism) or ligamentous incompetence will also potentially alter the anatomic integrity of the column with increased susceptibility to injury.

5. Radiologic considerations in acute cervical injuries

The magnitude of the instability will have an important relationship to the deleterious threats to neural structures, and a precise measurement of displacement or angulation is desirable. As previously noted, radiologic definition of injury patterns through analysis of load direction and load application is the basis for the generally accepted mechanistic determination of injury (Table 1). Certain considerations and variations of the radiographic examination, however, need to be considered in order to clarify the value and limitations of these studies to derive the mechanism of injury. It is not feasible in this review to inclusively detail the multiple radiologic considerations of cervical spinal injury patterns, and a more comprehensive information is presented in texts dedicated to this subject [9,31].

The evaluator, however, must be cautioned that correlation of the appearance of static plane radiographs in the initial post-traumatic period to the deformations incurred by cervical column and related neural structures during the rapid and dynamic process of injury may be misleading or incomplete [14]. One cannot always predict that the initial post-traumatic radiographic appearance will replicate the changes that resulted in major injury, especially the relationship of transient biomechanical deformation of neural tissues to the structural changes. The initial injury occurred in a period of milliseconds but subsequent events including inertial movements and positional changes during patient removal and transport may induce curvature and alignment changes different from the immediate injury pattern.

6. Specific injury patterns

The distribution of cervical injuries by levels will always exceed 100% since contiguous or non-contiguous injuries are encountered [9,11]. The highest incidence of injury occurs at the upper and lower segments of the cervical column indicating a greater susceptibility of these regions to increasing force application. The application of force will differ between these regions with the upper segments incurring a pattern of injuries directly related to the direction of the skull contact forces at the skull occipital-atlantal junction, whereas, the pattern of injury to lower cervical spinal levels is primarily influenced by forces directly applied to the vertebrae or through a lever arm of several adjacent segments. Although the majority of details regarding the mechanisms of injury are fairly well defined, certain unusual or controversial issues require clarification.

Injury forces following head impact are usually conducted through the occipital condyles in a cephalocaudal direction (compressive) toward the vertebral axis. A

smaller percentage, however, will have cervical injury from forces directed away from the vertebral axis which are termed as distractive or disruptive (Fig. 2). Although a distractive mode of force application as a primary vector is unusual, the effects may be dramatic. The structural integrity of the occipital–cervical (O–C) junction is mainly dependent upon a strong musculoligamentous complex which may be compromised by a high-magnitude vertical distractive force causing attenuation or disruption of these structures. Neural injuries at the cervical medullary junction may potentially result not only in complete caudal motor-sensory loss, but also loss of voluntary respiration and, therefore, the clinical recognition of such occipital–cervical instability is compromised by the frequent immediate mortality [32–35]. In this regard, the majority of O–C injuries were discovered in postmortem examinations of pedestrians exposed to high-speed vehicle impacts, but improved resuscitation techniques at the crash scene and the occurrence of O–C instability as a singular injury has improved survivorship and increased presentation of these injuries to emergency facilities. The forceful employment of airbags in automobile collisions has recently been implicated in the production of such vertical distractive forces at the O–C junction or in the cervical column in children or adults of small stature [36]. The radiologic

identification of this entity, however, may be subtle or obscured by the poor technical quality of emergency radiographs. The disruption may be complete or incomplete and the anatomic details are more extensively outlined in texts dedicated to radiographic interpretation of vertebral injuries [9,11]. Powers, and later Lee, presented x-line methods on plain radiographic studies to determine O–C dislocation and anterior subluxation, and, although these methods improved awareness of O–C dislocation or subluxation, analyses presented certain technical difficulties which limited their diagnostic value. Harris et al. [11] proposed a measurement system based upon more reliably visible landmarks. The basis-axial interval (BAI) consists of the distance between a line from the posterior cortical margin of the axis body and the basion (top of clivus or anterior arch of foramen magnum), and the BDI interval is the distance from the tip of the dens to the basis. Neither of these distances should exceed 12 mm. Fig. 3 illustrates the radiographic examination of a 52-year-old female of short stature, who incurred such an airbag-induced injury. She presented to the emergency facility in an essentially “locked-in” syndrome with absence of neurologic function below the cervical medullary junction. Following emergency airway procedures, radiographs demonstrated O–C abnormalities indicative of O–C

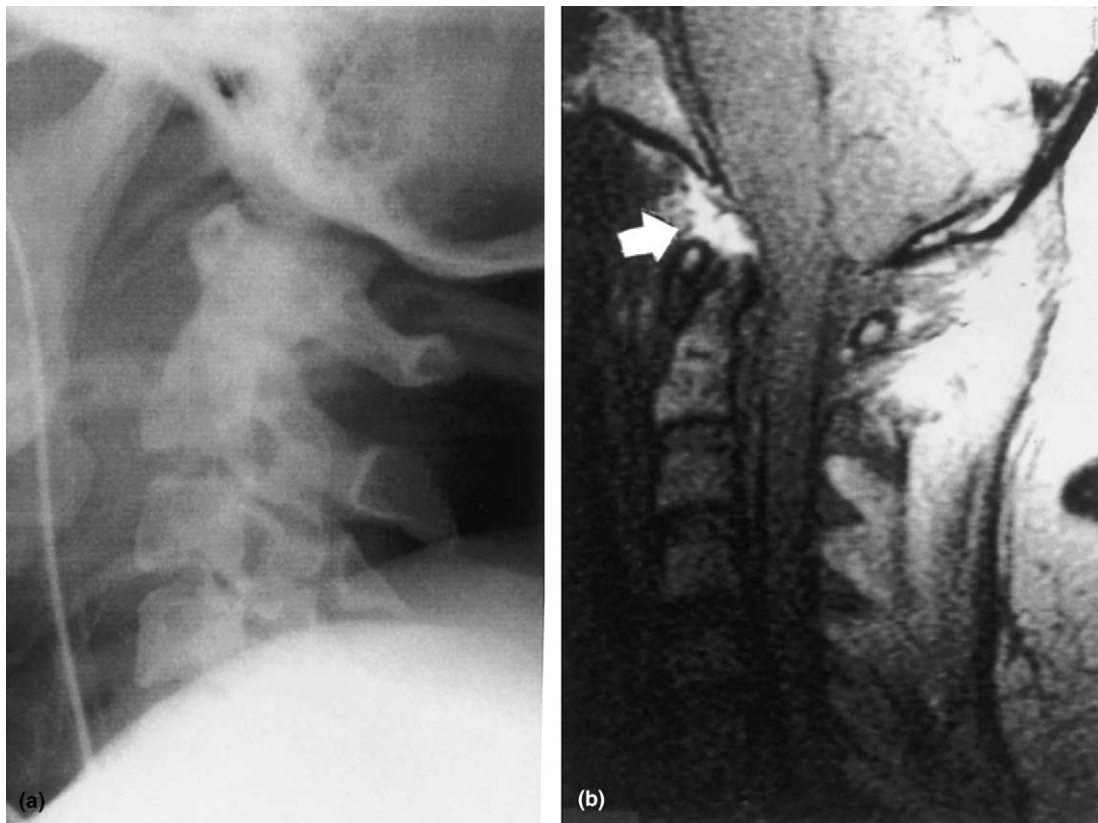


Fig. 3. (a) Lateral cervical radiography showing the occipital–atlantal subluxation; and (b) MR image showing the ligamentous injury of the anterior ligaments at the O–C junction (arrow).

dislocation and halo vest fixation was initially applied. Interestingly, tightening the cranial screws caused dramatic changes in extra-ocular eye movements indicative of brainstem compromise. These abnormalities were quickly reversed by relief of this trivial distractive force suggesting that even a minimal distractive force would induce brainstem compromise. The patient made a remarkable recovery to almost normal neurologic function following an occipital–cervical fixation and fusion procedure. This example emphasizes the need for recognition of these uncommon but potentially catastrophic injuries.

Axial compression injuries of the upper cervical segments are unusual events, and normally, do not result in catastrophic neurologic compromise. Occipital condylar fractures may occur as the impact to the vertex of the skull is transmitted to the lateral masses of the atlas with centrifugal displacement of these elements potentially causing fractures of the lateral masses as well as the anterior and posterior arches of C1 (Jefferson fracture). A lateral displacement of greater than 7 mm indicates risk of tearing of the insertions of the transverse atlantal ligament (TAL) with resultant risk of atlanto-axial instability. The TAL is important in maintaining the mechanical integrity of the C1–C2 level with the principal function of restraining the odontoid process against the anterior arch of the atlas. A more common compromise of TAL occurs during forceful flexion in which the ligament tears near the midline before odontoid fracture occurs, and this injury usually presents in an older group of patients. The hypermobility of the C1–C2 articulation with posterior odontoid displacement in flexion is recognized when the space between the anterior margin of the odontoid and posterior aspect of the anterior arch of C1 (predental) exceeds 3 mm in the adult.

Fractures of the odontoid process are the most common injury at the atlanto-axial (C1–C2) level and the primary vector is usually flexion which may cause anterior fracture-dislocation (position of the proximal fragments), whereas, the less common extension vector will result in posterior displacement [9]. Odontoid fractures make up about one quarter of cervical spine fractures and have been classified by Anderson and D'Alonso as type I (fracture of tip of dens), type II (fracture of dens above the axis body), and type III (fracture of base of dens at the superior body of the axis) [37]. Injuries of the atlanto-axial articulation, except O–C disruption, share the low incidence of corresponding neurologic compromise as other trauma to the upper cervical levels (5–16%). The most important factor in the preservation of neural function is the relatively copious spinal canal dimensions in this region. Although the incidence of neurologic impairment is relatively low with upper cervical fractures, the potential for catastrophic neurologic compromise exists, especially with unstable

injuries to the atlanto-axial segment. Odontoid fractures may be difficult to document on initial plain radiographs, and the case of a 67-year-old female who incurred multiple trauma not associated with head or cervical injuries illustrates this consideration. Initial cervical spine radiographs were unremarkable for evidence of injury, but three months later, complaints of neck pain led to the discovery of a type III odontoid fracture with almost 100% anterior displacement (Fig. 4). She was neurologically intact, and this sequence of events emphasizes the importance of rate of force application in the causation of neurologic deficit. If the marked degree of displacement demonstrated in these radiographs had occurred as part of the initial injury, the potential for catastrophic neurologic injury would be almost inevitable. The process, however, probably represents an incremental displacement of the odontoid with the markedly diminished adverse forces being applied to the spinal cord, resulting in tolerable deformation of neural elements.

Traumatic spondylolisthesis of the axis is another of the upper cervical spinal fractures that is usually associated with a small risk of neurologic injury. This injury was originally described for cervical injuries resulting from judicial hanging (Hangman's fracture) and, although the general consensus of causation is by extension–distraction forces, certain fracture patterns with anterolisthesis of C2 on C3 have been suggested by a minority of investigators to be primarily a flexion injury [38]. If the fracture pattern extends to involve the superior articular facets and articulating pillars, the subluxation may be more extensive and increase the risk of neurologic impairment. Variations of this specific injury category again emphasize the need to define correctly the mechanism of injury in order to apply the appropriate therapy, as certain types of treatment may accentuate the deformity rather than offer reduction.

The fracture patterns to the middle and lower cervical spine share many similar characteristics, but the predilection for such injuries is greatest at lower levels. The relatively increased incidence of acute fracture-dislocation at lower levels is probably influenced by the approaching fixation point with the thoracic spine, as well as potentially experiencing an increased lever effect of the upper and middle cervical column. Flexion or extension modes usually modified by compression or distraction forces will result in varying patterns of vertebral column component injury. Irrespective of extension or flexion vectors, the production of fractures requires the application of a compressive force with anterior bony components (vertebral body) susceptible in flexion, whereas posterior bony elements (facets, articular pillar, and spinous processes) are involved in extension injuries [39]. The influence of eccentricity of the compressive force, therefore, will determine the susceptibility of posterior or anterior components to injury [18].

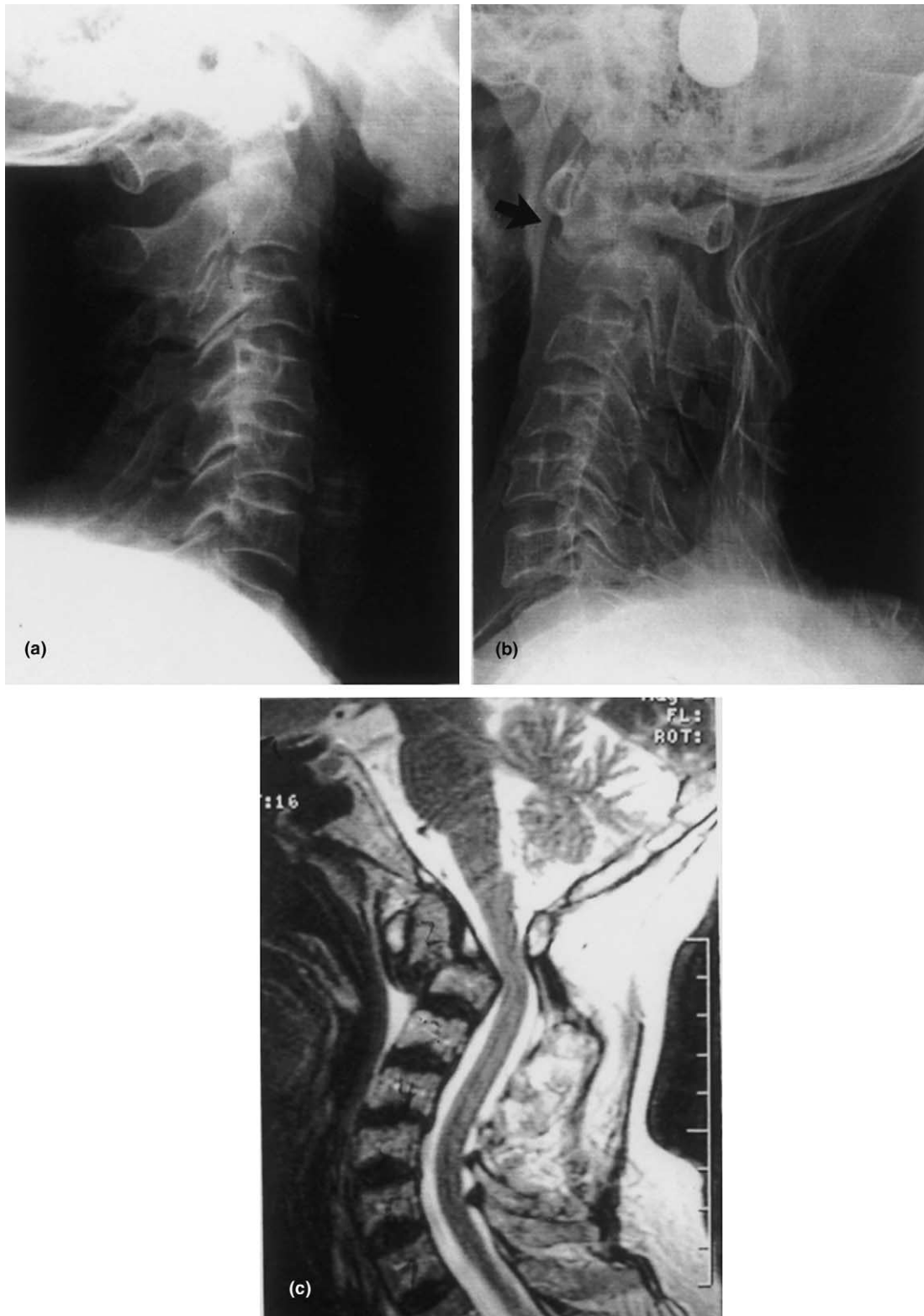


Fig. 4. (a) Initial lateral cervical spine showing normal alignment and soft tissue structures; (b) lateral cervical radiographs two months after initial trauma showing marked subluxation of C1 on C2 secondary to odontoid fracture (arrow); and (c) MR image demonstrating the significant canal compromise from the fracture dislocation of C1 on C2.

The forces acting upon the cervical space will be modified by the curvature of the column. The usual lordotic curvature of the column is identified as the

neutral position and, with small increments of flexion, the column assumes a straightened configuration. Pintar et al. [40,41] identified this alignment as the “stiffest

axis” and found that this configuration was necessary to produce a burst fracture. This fracture pattern was initially identified as a comminuted fracture of the vertebral body with variable retropulsion of the posterior aspect of the vertebral body into the canal. Computerized tomography (CT) has demonstrated that body fractures may also be associated with posterior element fractures indicating a wider dispersion of energy at the principal level of injury. Axial loads positioned along the axis of a straightened (neutral) spine is the position which sustains the highest loads to failure. The burst fracture is considered to result in potentially significant spinal canal compromise and instability, but the stenosis may be transient with partial recovery of the structural changes to the vertebral body following the initial event [14,16,42]. Therefore, injuries with relatively small canal compromise on post-injury studies may have been more severe during the acute injury event. The stiffest axis or straightened column presents a biomechanical situation where the column is capable of withstanding increased loads, but also having a greater risk of serious structural compromise as loads of greater magnitude are applied. The column, therefore, will demonstrate a greater tolerance to axial compressive loads, but exhibit increased risk of structural failure as load application approaches higher levels.

Additional anterior eccentricity of axial compressive load transfer with a larger ratio of bending moment to compressive force will result in a wedge-type fracture to the vertebral body. Experimentally, this fracture pattern exhibits minimal canal occlusion, and, therefore, has been acknowledged to represent a more benign clinical process than burst fractures [3,42]. Fig. 5, however, shows that a compression fracture can result in spinal cord injury. With further increases in anterior eccentricity, a marked increase in bending moment can be induced on the middle or lower column segments causing bilateral facet dislocations and spinal cord compromise (Fig. 6).

7. Determination of quantified neck injury tolerance

7.1. Normal cervical spine and spinal cord mechanics

The capacity of the various spinal components (vertebral bodies, intervertebral discs, particular pillars, facets, and ligaments) to tolerate a deforming force will determine the character of the structural compromise. The ligamentum nuchae represents the supraspinous ligament and offers a fairly strong attachment to the cervical spinous processes, but the other subaxial ligaments and intervertebral discs are relatively weak in comparison to other parts of the vertebral column. The orientation of the zygapophysial joints permits substantial motions in extension and flexion which are as-

sociated with a normal decreasing translational movement from C2 through C7, and the upper limits of normal for this latter motion have been suggested to be 3.5 mm [43]. Flexion and extension modes are descriptive of bending moments in the sagittal plane with hyperflexion or hyperextension suggesting the inclusion of ligamentous disruption or attenuation that allows excessive sagittal rotations. In the cervical spine, normal motion is usually coupled with motion about at least one other axis, and, since the zygapophysial joints have a medial orientation joining with the uncovertebral processes, rotation is coupled with lateral flexion. The consideration of rotation, therefore, as simply turning about a horizontal plane, is incomplete and necessitates consideration of lateral bending.

The corresponding effects of these dynamic changes of the spinal canal on spinal cord are integral to understanding the type and potential for neural injury in acute fracture-dislocations. Numerous investigations have demonstrated that the cervical spinal canal is lengthened in flexion and shortened in extension [44–47]. When the cervical spine is placed in flexion, the canal becomes longer at both the anterior and posterior walls with the posterior vertebral contour becoming convex. It has been estimated that the cervical canal lengthens 2.8 cm from full extension to full flexion, and in both flexion and extension the largest change in dimension takes place at the posterior wall. During maximal extension, the backward movement of the upper vertebra upon the inclined facet of the lower vertebra may result in laminar impingement and induce further shortening of the canal. Movement of the lamina and the possible infolding of the ligamentum flavum may contribute to a decrease in the cross-sectional diameter of the canal. The importance of pre-existing spinal canal dimensions has been proposed as an influence upon subsequent spinal cord compromise. Normal anterior–posterior canal dimensions are estimated to range from 16 to 18 mm at C3 through C7, and spinal cord diameter to range from 8.5 to 11.5 mm [6]. The capacity of the spinal canal decreases at lower cervical levels with the spinal cord occupying approximately 50% of the canal at the C1 level and 75% at the C6 level [46,48,49]. Wolfe et al. [50] suggested that a canal of 10 mm or less was a predisposing factor in developing subacute and chronic myelopathy and other investigators have noted that a smaller canal diameter may be a relative contributing factor in acute spinal cord injuries.

The spinal cord participates with the vertebral column in configurational changes relative to alterations in functional positioning, and the susceptibility to injury will vary with specific abnormalities of the vertebral column. The majority of information regarding the physical properties of the spinal cord and related nerve roots, dentate ligaments, and pia and dura mater has its basis in the studies of Breig and co-workers [44,45]. In

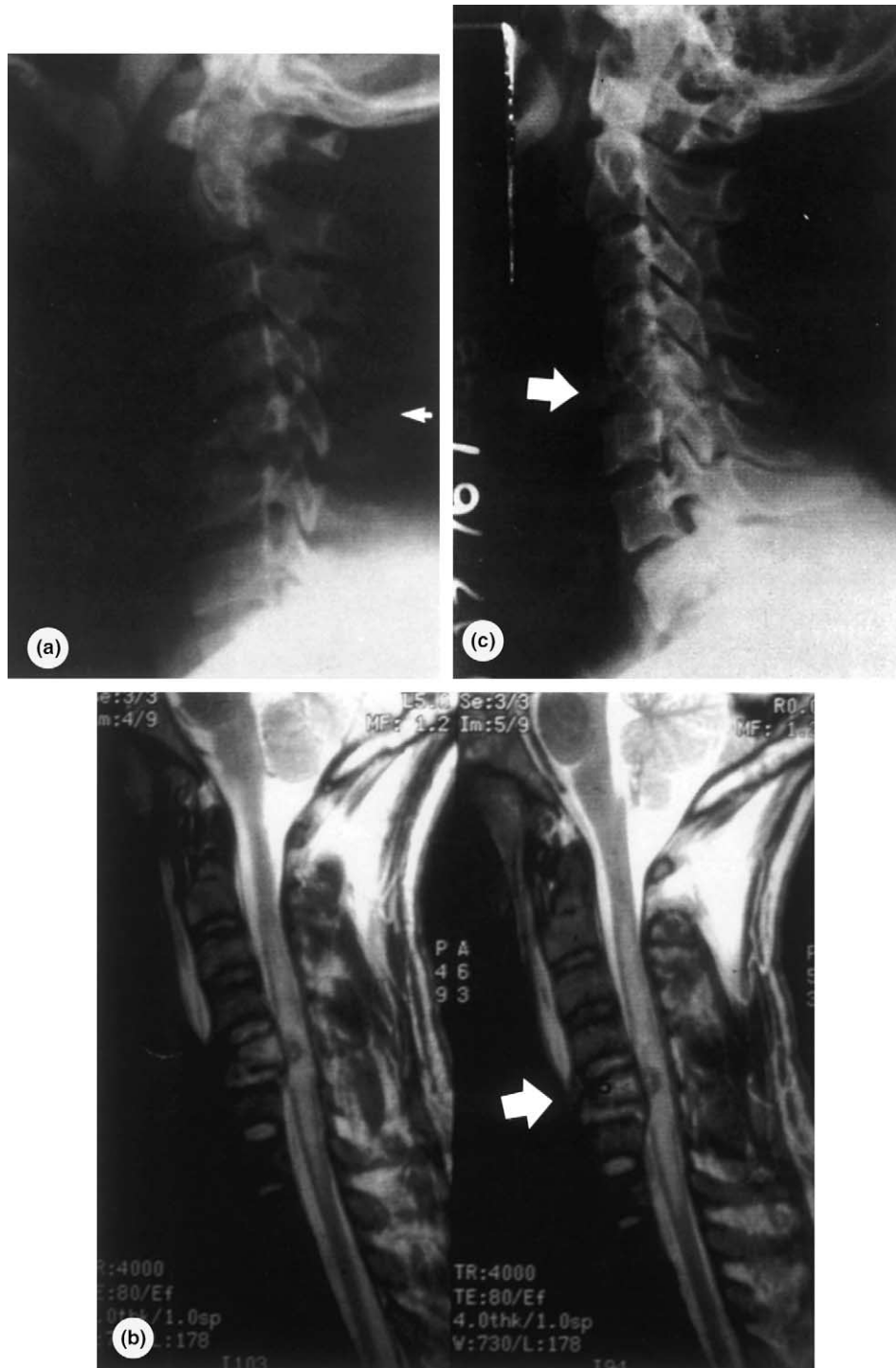


Fig. 5. (a) Lateral cervical radiograph of a compressive fracture of C5 with minimal retropulsion into the spinal canal. Arrow shows widening of the interspinous ligamentous complex (fanning); (b) sagittal MR showing the presence of small epidural hematoma (arrow) and mild canal compromise; and (c) lateral cervical radiograph showing burst (comminuted) fracture of C5 with retropulsion of fragments into the canal. The principal axial force does not compromise the posterior ligamentous complex.

considering the physical properties of the spinal cord, it is important to note Breig's clarification that the spinal cord is part of a continuous tissue tract originating in the mesencephalon and extending to the point where the nerve roots exit. This structure participates in physical

alterations as a whole, with the predominant effects occurring at the local level of distortion. This concept indicates, however, that abnormalities influencing the spinal cord may have compounding effects at distant sites along this brainstem-spinal cord tissue tract. In the

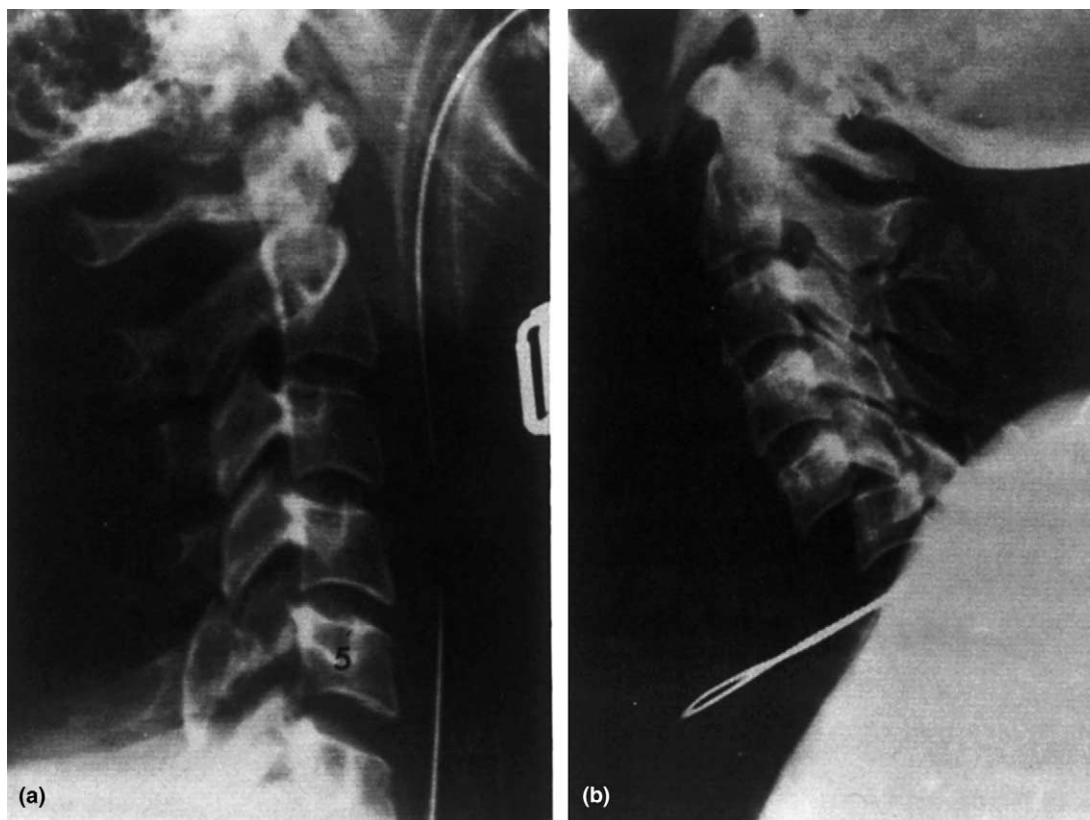


Fig. 6. (a) Lateral cervical radiograph showing unilateral locked facet (flexion rotation) with minimal subluxation; (b) lateral projection showing bilateral locked facets with almost 100% subluxation at C5 on C6 unassociated with evidence of fracture (flexion distraction).

cadaver preparation, distraction of the spinal cord demonstrates a load–displacement curve with two distinct phases. Large initial displacements are accomplished with small amounts of force, demonstrating the elastic qualities of the spinal cord, but these initial stages are followed by an abrupt stiffening in which additional small increments of stretch require large load levels [51]. These biomechanical findings agree with the physiologic evidence obtained in axial distraction studies in which initial load levels resulted in mild reversible distortion of axonal conduction until a critical level of stretch was accomplished. At this later stage, additional force resulted in marked deterioration of axonal conduction that was usually irreversible [52]. This situation mirrors a frequent clinical dilemma of distinguishing between physiologic (reversible) impairment of axonal conduction or anatomical (irreversible) damage to these neural elements in patients.

In addition to giving insight into the physical properties of the spinal cord in distraction, Breig [45] demonstrated that the spinal cord does not move in the longitudinal plane, but adapts itself to the length of the spinal canal by plastic deformation. In flexion, the cord elongates with the spinal canal and narrows in antero-posterior diameter. This induces increased axial tension in the axon cylinders of the white matter tracts and le-

sions of the vertebral canal that compromise the cross-sectional area, especially those processes anterior to the spinal cord, which cause further local and generalized increases in axial tension within the spinal cord (Fig. 7). Reid [53], in cadaver dissections, noted the anterior component of the force exerted on the cord and dura undergoing a 3-mm displacement, the neck in flexion was found to reach 0.21–0.27 MPa, whereas, with the neck in the neutral position, an anterior pressure of only 0.01 MPa was exerted on the cord and dura. In contrast to the elongation of the spinal cord during flexion, the spinal cord in extension shortens and increases in anteroposterior diameter with a relative relaxation of the axon cylinders. The corresponding decreased cross-sectional area of the spinal canal occurring from posterior bulging of the anulus, as well as the infolding of the ligamentum flavum and scaffolding of the lamina in extension, may result in a pincer-like action upon the cord (Fig. 8). In this setting, irreversible spinal cord damage becomes more likely when compression exceeds one-third of the normal cord diameter [44,45]. An important consideration in defining the risk for spinal cord compromise is consideration of both the degree and axis of applied forces. Tensile forces applied to the spinal cord in the neutral position will produce a relatively even load distribution across the structure, but if the

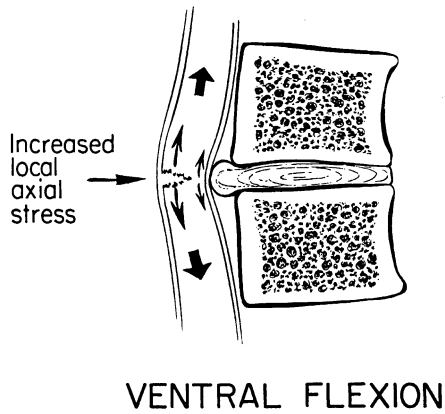
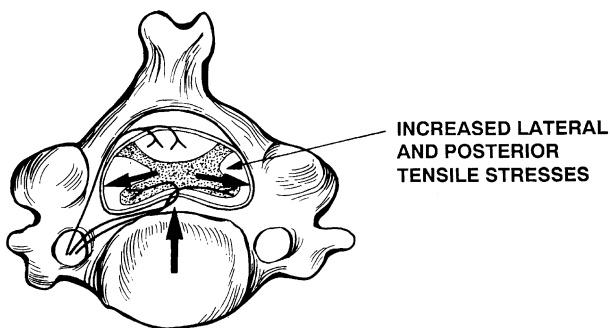
**FLEXION**

Fig. 7. Illustrates the tensile stresses acting upon the spinal cord from a ventral protrusion during flexion. The maximum local stresses are in the posterior aspect of the cord.

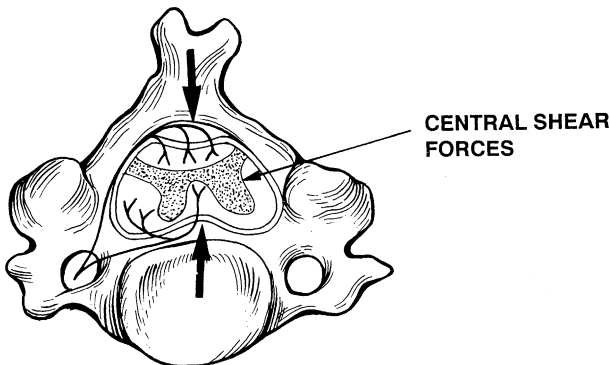
EXTENSION

Fig. 8. Axial section illustrating the centripetal transmission of forces upon the cord during extension. These forces will be magnified with pre-existing stenosis of the cervical canal.

cord undergoes bending, compressive force will increase on the concave side, causing increasing tensile (stretch) forces on the convex side (Fig. 7). Shear forces, in contrast to tensile forces, are maximal toward the center of the spinal cord and act in a perpendicular plane to the tensile forces. The interaction of these three different

forces being applied to varying areas of the spinal cord during flexion (bending) indicates the potential for a complex pattern of injury relative to the individual magnitude of each of the stresses and their possible interaction.

In extending these observations, as previously noted, laboratory studies provide the opportunity to control and observe the impact event that results in major cervical injury. Experimental models can delineate the mechanisms and quantify tolerances of the human neck to injury. Models have included segmented portions of the spine (single or multi-level), ligamentous columns, head–cervical columns, intact head–neck complexes, and whole-body human cadavers [12,13,15,40,54–76]. Although animal models can be used to create injuries, since precise scaling laws do not exist, it is difficult to extrapolate the quantified metrics to the human. Segmented portions of the spine represent a well-controlled and least complex model. With this model, the contribution from the adjacent levels cannot be directly incorporated in these functional spinal unit models. Ligamentous spinal columns automatically incorporate all vertebral levels. The column needs to be potted/fixated at one or both ends to apply loading. The artificial fixation condition at the proximal end is eliminated in the intact head–cervical column model. The applications of eccentric axial loading, necessary to create compression–flexion or compression–extension types of injuries, are difficult to accomplish with this model. Intact head–neck complex models with full inclusion of musculature and skin can be used to appropriately align the specimen and apply external loading to reproduce pure non-axial loading injuries. Although intact whole-body cadaver models are comprehensive, difficulties exist if detailed motion analysis of the various vertebrae is of interest. Thus, depending on the type of injury to be reproduced and nature of biomechanical variable under consideration, researchers have the option of using these models in injury biomechanics investigations. Factors that play a role in the biomechanics of major neck injury include both the external-and occupant-related variables. External variables include the force type, direction, and magnitude. Occupant-related parameters include age, gender, effects of neck musculature, and orientation or alignment of the head–neck at the time of impact.

7.2. External variables

7.2.1. Force type

Serious or major neck injuries belong to the Abbreviated Injury Scale (AIS ≥ 3) category and are associated with neurological deficit [77]. These injuries occur routinely with head impact (e.g., athletic events, motor vehicle crashes, falls, diving). Compressive loading applied to the head is transmitted to the cervical spine through the medium of occipital condyles. Because of

the three-dimensionality of the cervical spine, this load is internally transformed within the components of the neck into axial, lateral/anteroposterior shear and bending, and translational and rotational deformations [22]. Injury occurs when the forces/moments or deformations exceed the tolerance of an individual component(s). Because of the dynamic nature of the loading, tests using static methods are not fully applicable. This is due to the omission of the time factor in static load applications [78]. The biomechanical properties of the constituents of the cervical spine are time dependent [12]. They exhibit changes or sensitivity to loading rate. This viscoelastic property is not the same for the hard and soft tissue structures. Although bone, intervertebral disc, and ligament exhibit increasing stiffness with increasing loading rates, the rate of increase is higher in bone than in ligament [12,60]. Increasing loading rate also enhances the energy absorption characteristics. For example, with increasing loading rate from 1.0 to 250 cm/s the tensile failure load, stiffness, and energy of cervical spine anterior longitudinal ligament and ligamentum flavum increase by a factor of two to four [12]. Similar observations have been advanced by Pintar et al. [13] for compressive loading of the head–neck complex.

7.2.2. Force direction

It is important to specify the direction of the loading vector with respect to anatomy of the spine. For example, the applied compressive force on the head can be defined with respect to the position of the occipital condyles. Because of the curvature of the cervical spine and heterogeneity of the various components, pure compression, pure tension or pure bending moments do not occur. However, depending on the location of the load vector at the level or segment where injury occurs, one type of load (e.g. compression) may predominate. Axial compressive loading injuries include Jefferson, vertical, and burst fractures [79,80]. In contrast, compression–flexion bending moment-related injuries include wedge compression fractures with distraction of the posterior ligament complex. These injuries, by definition, have a combination of compression and flexion, i.e., from a mechanical perspective, a compressive load applied anterior to the center of the segment. This is termed as anterior eccentricity. If the eccentricity of the load is severe enough, i.e., with large eccentricities, ligaments disrupt leading to facet dislocations and vertebral body subluxations (without significant vertebral fractures) [66]. Application of compressive forces with a posterior eccentricity results in compression–extension-related trauma such as posterior element fractures with anterior longitudinal ligament ruptures. Likewise, tension forces applied due to airbag contact with chin induces tension–extension related injuries such as head–upper cervical zygapophysial joint and ligament dislocations [36]. While the above-described directions of the

load lie predominantly in one plane (sagittal), combined or out-of-plane loading is also possible. For example, unilateral facet dislocations have been attributed to a rotation component and lateral bending injuries in side impact crashes have been reported [81]. These types of out-of-plane injuries are relatively less common.

7.2.3. Magnitude

With regard to the magnitude of the force for inducing neck injury, human volunteer studies cannot be used. This is because difficulties exist to extrapolate human volunteer investigations from the non-injury domain to the injury domain. Human cadaver models can be subjected to injury-producing forces in a controlled environment, and the resulting trauma can be correlated with biomechanical variables such as force and/or deformation. Under compression, failures have been reported using various models with peak forces ranging from 300 N to 17 kN. For example, Culver et al. [76] subjected 11 cadavers to superior–inferior impact. Cervical spine injuries occurred at mean peak force of 7.58 ± 0.94 kN. Nusholtz et al. [62] impacted 12 intact cadavers using a pendulum at velocities ranging from 4.6 to 5.6 m/s. At a mean maximum impact force of 5.2 ± 3.1 kN, injuries to the anterior and posterior elements occurred at C2–T4 levels. In a later study, Nusholtz et al. [82] dropped eight cadavers on the head and produced upper and lower cervical spine injuries at forces ranging from 3.2 to 10.8 kN. A wider force range was reported by Alem et al. [54] wherein 14 cadavers were subjected to crown impacts; head impact forces ranged from 2.3 to 17 kN. However, the mean impact force at the head to cause neck injury occurred at a force level of 5.3 ± 2.4 kN. Yoganandan et al. [72] dropped 15 human cadavers at heights ranging from 0.9 to 1.5 m (impact velocities 4.2–5.5 m/s). Both head restrained and unrestrained cases were considered. Head impact forces for the restrained and unrestrained cases ranged from 9.8 to 14.7 kN and 3.0 to 7.1 kN. Forces measured in the cervical spine using a load cell inserted to the C5–C6 disc space ranged from 1.1 to 2.6 kN. Injuries included bilateral facet dislocations, odontoid and Jefferson fractures, and other spine trauma.

In the area of isolated ligamentous column tests, compression–flexion injuries occurred at axial loads ranging from 1.8 to 4.5 kN [83]. Maiman et al. [84] tested 13 specimens with varying initial positions (e.g., pre-flexed) at velocities ranging from 23 to 152 cm/s and reported peak loads ranging from 0.65 to 7.4 kN. McElhaney et al. [59] subjected isolated base of skull to lower cervical spine columns to compressive loading at 0.13–64 m/s. At peak forces ranging from 0.69 to 6.48 kN, burst, wedge, Jefferson, and posterior element fractures were produced. Nightingale et al. [61] dropped 22 inverted head–cervical spinal columns from a height of 0.53 m (velocity 3.2 m/s) and produced varying types

of cervical injuries which included facet dislocations, burst, and odontoid fractures. Injury occurred at a mean peak force of 2.04 ± 0.37 kN. In contrast, Pintar et al. [40] conducted studies using an upright intact head–neck model with pre-flexed spines in an attempt to replicate major injuries. The model simulated the effects of the anterior and posterior regions of the head–spine complex. Fig. 9 illustrates the test setup. With this initially pre-flexed (lordosis removed) configuration, typical cervical injuries which included wedge and burst fractures with and without posterior element distractions were produced at higher forces (mean: 3.3 kN). Fig. 10 illustrates the force–deflection corridors under compressive head impact. The cervical column compressive deformation to failure was determined to be 19 mm (sd: 3 mm).

With regard to tensile force, despite recent awareness in serious to fatal upper cervical injuries such as atlanto-occipital and atlanto-axial dislocations secondary to airbag deployment, data are very limited. Using dynamic loading as an external input for inducing neck injury, Yoganandan et al. [85] reported mean peak tensile failure loads of 3.4 ± 0.46 kN for intact human cadaver preparations. Distraction at failure, stiffness,

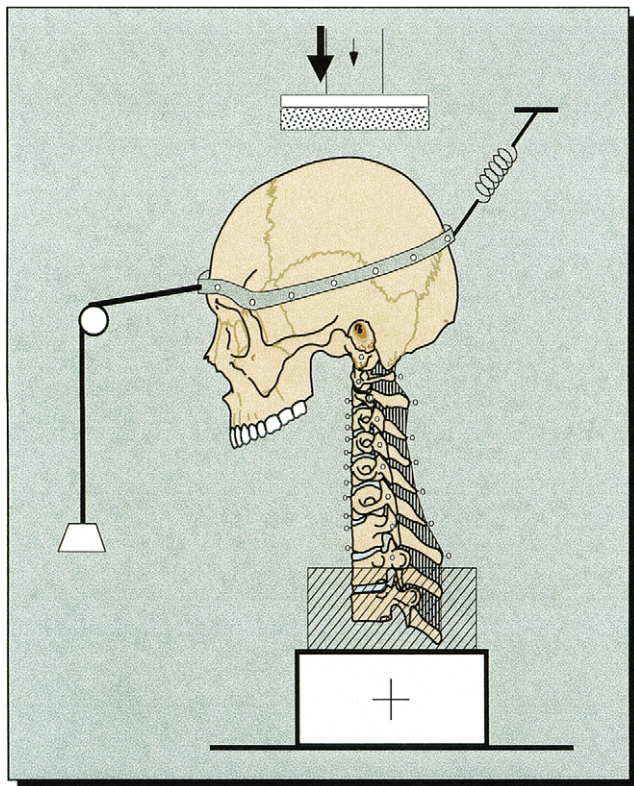


Fig. 9. Test setup for applying contact-induced impact injuries to the head–neck complex to reproduce typical compression-related injuries such as wedge and burst fractures with and without posterior element distractions resulting in neurological deficit. The effects of passive musculature were incorporated using preloaded spring and pulley weight system. Force–deformation corridors are shown in Fig. 13.

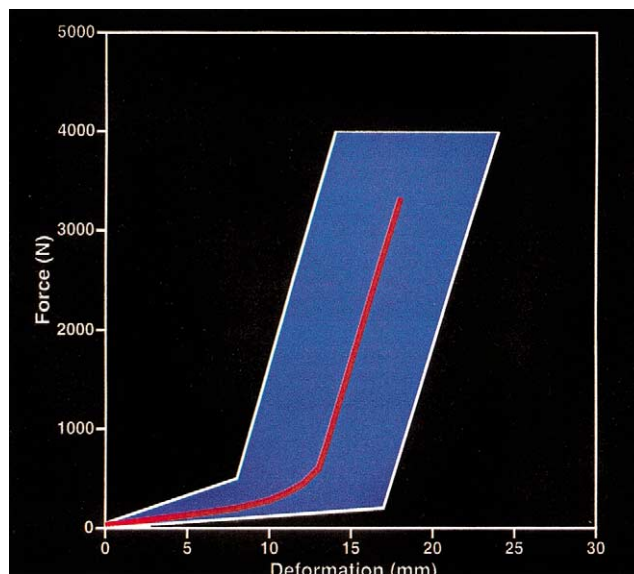


Fig. 10. Force–deformation corridors of the human neck under dynamic impact loading to the head. The dark middle line indicates the mean response curve derived from the force (3.3 kN) and deformation at failure (19 mm). These data quantify compression tolerance of the human cervical spine.

and energies were 21.3 ± 1.9 mm, 164.8 ± 34.2 N/mm, and 35.1 ± 4.0 Nm, respectively. In order to determine the dynamic tension biomechanics applicable to the pediatric and small female (populations at risk for airbag-related injury), experiments are underway [36]. Models include intact occiput to T1 column with and without the skin and musculature, and segmented single and multilevel spinal units from the occiput and intact human cadaver preparations. In order to reproduce upper cervical injuries typically seen in vehicular crashes, it is necessary to apply a high rate of onset dynamic loading to the intact human cadaver or intact head–neck complex with intact skin and musculature. In addition, the Caprine was shown to be a valid model to study age-specific pediatric injuries because of its close relationship with the growth and development of the human neck structure (e.g., ossification and chondrification processes similar to the human with a reduced age pattern).

7.3. Occupant-related variables

7.3.1. Age

Studies by Cusick et al. [57] have shown that, in general, segment(s) superior to the level(s) of spondylosis are vulnerable to injury due to impact loading to the head. Spondylotic degeneration decreases cervical mobility and this stiffening effect at the degenerated level of the column acts as a hinge/fulcrum to induce trauma to the rostral spine. Degeneration decreases bone mineral content in the load-bearing regions of the skeleton and contributes to reduced vertebral strength. Pathologic fractures due to osteoporosis have been reported

frequently in the thoracolumbar spine [86]. However, such quantified and correlated information is not available for the cervical spine [81]. Despite this paucity of data relating age or bone-related changes to fracture production or tolerance, a general consensus exists with regard to lower thresholds for female and aged populations. Similarly, pediatric bone growth and development due to primary and secondary ossification processes also influence the strength of the cervical spine. The increased flexibility of the pediatric neck structures (distraction limit of up to 5.0 cm compared to approximately 2.0 cm in the adult) may be beneficial to sustain larger deformations. However, spinal cord injuries without radiological abnormalities are a severe form of neck trauma to the pediatric population. Another factor is the increased mass of the pediatric head in relation to the neck mass [87,88]. This is particularly important during high rate of onset load applications such as airbag deployments, creating a tension–extension moment-type injury at the upper cervical levels. Quantified data are very limited with regard to pediatric tissues.

7.3.2. *Musculature*

Muscles play a role in stabilizing the cervical spine particularly with regard to initial alignment. In addition, depending on the rate of application of the external force vector, muscle activation may influence the mechanism of load transfer in the neck structures and hence, injury. Impact load on the head resulting in major neck injuries such as wedge and burst fractures of the mid-lower cervical spine occur approximately within the first 15 ms after the initiation of the dynamic load [22,61,63,70,89,90]. This time is significantly shorter than the time required for muscle activation which is reported to be approximately 60 ms in human volunteer studies [91]. Consequently, the role of active muscle response may be minimal in these major cervical injuries induced by compressive forces. However, for other trauma such as bilateral facet dislocation, it has been hypothesized that the injury begins with a tear of the posterior ligaments [66]. As hyperflexion disrupts the joint, the reactionary response of the cervical musculature initiates to stabilize the spine resulting in a pulling-down action on the upper regions of the spine. The downward contraction of the muscles slides the upper portion of the dislocated spine forward and down causing the superior facet to lock in front of the inferior facet, resulting in neurological trauma to the cervical spine. Researchers have attributed the neck musculature to influence the local bending modes during injury [92].

7.3.3. *Spinal alignment*

The human neck has a resting lordotic curvature. The orientation or alignment of the neck structure with respect to the head and/or the torso at the time of impact can affect the resulting injury and injury mechanisms.

Culver et al. [76] and Nusholtz et al. [82] suggested a relationship between initial neck position and injury although no specific quantification was made of spinal alignment. Alem et al. [93] subjected 19 human cadavers with pre-flexed (lordosis removed) and natural lordotic (without pre-flexion) configuration. Pre-flexed spines had higher impact forces (4.69 ± 0.5 kN) than lordotic spines (4.45 ± 1.0 kN) for cervical injury. Using pre-flexed head–neck complexes, Pintar et al. [40] produced clinically relevant neck trauma typically concentrated at one motion segment level. Peak forces were higher in these pre-flexed spines (mean force 3.3 kN) than the peak forces reported by Nightingale et al. [61] who dropped head–ligamentous–column torsos without pre-flexion (mean force 2.0 kN). Because of the presence of curvature, in addition to the axial force due to impact, bending moments contribute to the intrinsic biomechanics and load transfer within the cervical column. Thus, the resistance of the spine to axial loads decreases in the presence of bending moment. Increased forces due to initial pre-flexion have been shown to produce lower cervical injuries such as burst and wedge fractures than neutrally aligned (lordotic curvature maintained) columns [40,63,70,94]. Maiman et al. [84] evaluated the effects of pre-flexion and pre-extension on human injury using base of skull to T1 and intact human cadaver models. In this study which used 13 specimens, the mean peak loads to failure for pre-flexed and pre-extended spines were 1.82 and 1.09 kN.

Yoganandan et al. [72] in their 15 intact cadaver drops, accounted for the alignment by restraints and produced more cervical injuries with restraint conditions. McElhaney et al. [59] reported that moving the load vector from anterior to posterior direction altered the mechanism of injury from compression–flexion to compression–extension, i.e., from vertebral body fractures to posterior element fractures. However, in these studies, quantification of the alignment and its effect on the biomechanics were not made. Yoganandan et al. [18] statistically correlated alignment with neck injury. Results indicated that initial spinal alignment affects the type of injury and injury mechanism, abbreviated injury classification of trauma, and differentiation between bony and ligamentous trauma ($p < 0.05$ in all cases). However, spinal alignment was not a good predictor to statistically differentiate between stable and unstable injuries. These data suggest that while alignment may control the injury mechanism, the decision for operative treatment may be influenced by factors such as the potential for further neurologic deterioration secondary to initial anatomic disruption.

7.4. *Neck injury threshold*

Yoganandan and Pintar [69] conducted a series of impact tests using human cadavers. The force–

deformation corridors for axial compression loading injuries were discussed earlier (Figs. 9 and 10). The probability of injury secondary to hyperflexion injury mechanism was determined using a similar model. Preflexed intact human cadaver head–neck complexes were subjected to dynamic loading using an electrohydraulic piston at varying eccentricities measured as the position of the occipital condyles with respect to the first thoracic vertebra [66]. The test setup is shown in Fig. 11. The injury probability functions expressed as a function of axial neck force and bending moment at the level of spinal injury are included in Figs. 12 and 13. In a later study, Pintar et al. [13] examined the effects of age, gender, and loading rate on compression-related injury. The age of the cadavers ranged from 29 to 95 years and loading rate from 0.25 to 800 cm/s. Statistical modeling included multiple linear regression and proportional hazards analyses. Age was significantly related to loading rate ($p < 0.05$) and an interactive effect was demonstrated between the two parameters. Although gender was a significant parameter, higher variations were found with females sustaining a lower force at a given probability than males. While both genders exhibited

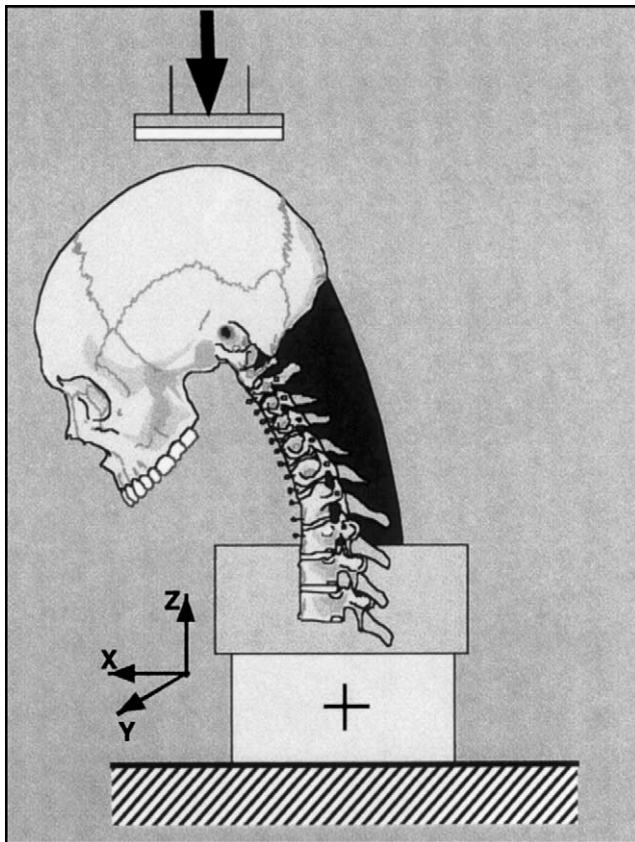


Fig. 11. Schematic of test setup for inducing flexion-related injuries to the head–neck complex. A six-axis load cell was placed at the inferior end to record and translate the bending moment at the site of spinal injury due to impact loading to the head.

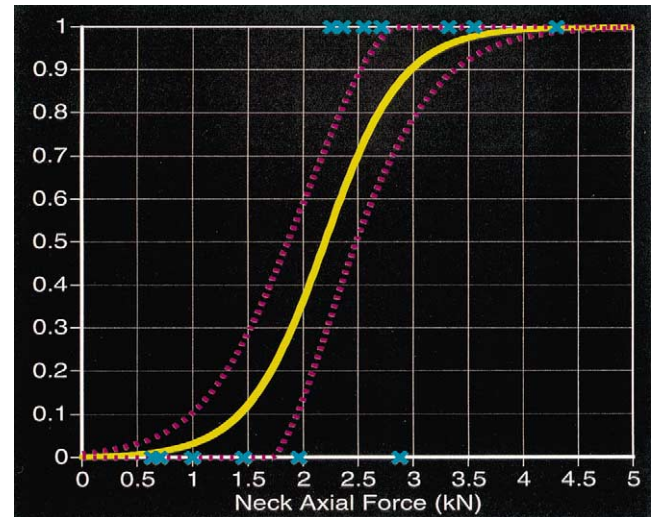


Fig. 12. Probability of cervical spine failure as a function of peak neck axial force under hyperflexion injury mechanism.

viscoelastic effects (Figs. 14 and 15), males consistently exhibited higher failure force (600 N) than females at all age groups and loading rates (Fig. 16). The probability distribution is shown in Fig. 17. These age, gender, and loading rate dependent injury probabilities represent a comprehensive data for assessing compression-related injury in impact environment [13].

8. Conclusions

Correlation of certain biomechanical parameters and clinical factors associated with the causation and occurrence of traumatic cervical spine injuries assists in clarifying the pathogenesis and treatment of this diverse group of injuries. The differing methodology of these two forms of investigation offers different potentially supportive information in clarifying the relative importance of certain histories, radiological and conceptual factors. In this regard the principal characteristics of the cervical vertebral column and the associated spinal cord, especially relative to dynamic positional alteration, will be important influencing factors on the type and magnitude of potential neurological compromise; another important parameter to establish is the presence of vertebral column instability and the descriptive analysis of the local forces responsible for the change in the mechanical integrity of the spinal column. These mechanical determinants of cervical spinal column may be influenced by certain patient variables which may alter the physiologic integrity or anatomic relationships of vertebral column compromise rendering the column more susceptible to injury. The localization, magnitude of development and rate of instability are important in determining the risk of neurological damage and therefore, the determination of angulation or displacement by

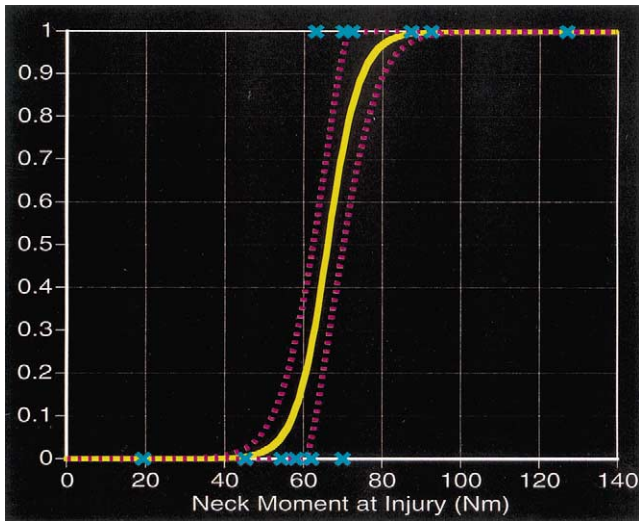


Fig. 13. Probability of cervical spine failure as a function of peak neck moment at the injury level under hyperflexion injury mechanism.

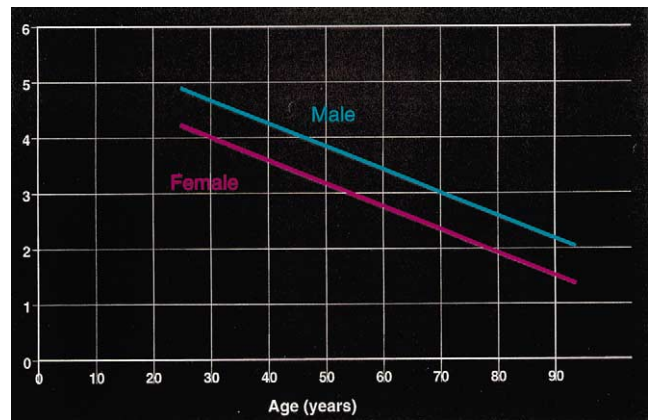


Fig. 16. Comparison of the tolerance characteristics of the human cervical spine (Force kN) under axial impact loading for male and female specimens at a loading rate of 4 m/s.

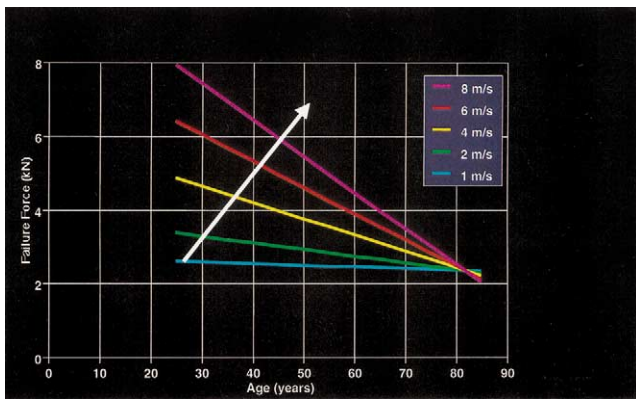


Fig. 14. Multiple linear regression model plots for human cervical spine impact tolerance as a function of loading rate and age. Results are applicable to male specimens.

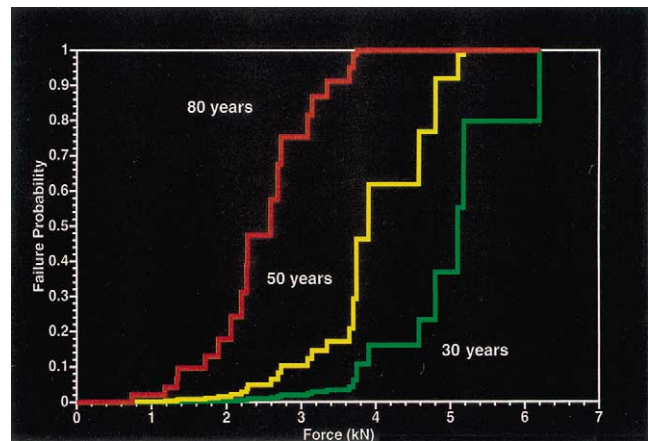


Fig. 17. Probability of failure of human cervical spine as a function of age.

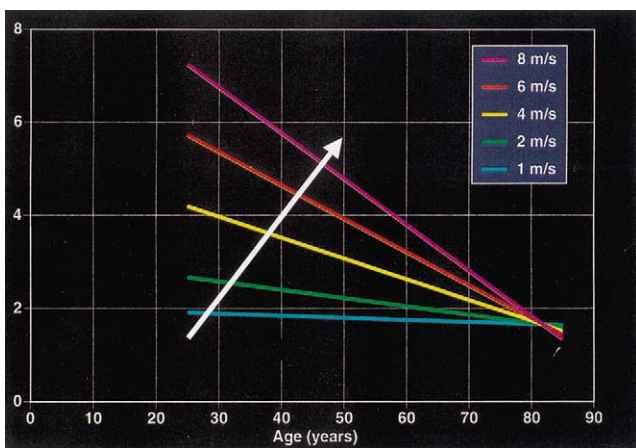


Fig. 15. Multiple linear regression model plots for human cervical spine impact tolerance as a function of loading rate and age. Results are applicable to female specimens.

radiographic studies has an important role. The definition of injury patterns through the analysis of load direction is a basis for the mechanistic determination of injury, but variations in radiologic studies need to be considered to clarify the value of specific radiological ramifications. Plain radiography is frequently able to establish the presence of cervical column compromise as well as defining the probable mechanisms of injury, but occasionally adjunctive radiological studies (CT, MRI, tomography) may be required. In the armamentarium of radiological examinations, the diagnostic role of dynamic radiography deserves attention, especially relative to delayed instability. The highest incidence of cervical column injuries occurs at the upper and lower segments. Regional differences exist not only to relative susceptibility to column injury but also for potential neurologic compromise, and the recognition of treatment of many common injury mechanisms as well as certain unusual injuries, including occipital–cervical dislocations are emphasized in this review. The influence of cervical alignment and curvature in association with the direc-

tion of force application on the potential vertebral component compromise is an important consideration in clarifying the causative forces of specific fracture patterns. Full consideration of the radiologic and historic factors in conjunction with an understanding of the biomechanical principles of the cervical spinal column and spinal cord will allow the clinician to enhance diagnostic accuracy and improve the management of such injuries.

Acknowledgements

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References

- [1] Larson SJ. Vertebral injury and instability. In: Holtzman RNN, editor. *Spinal instability*. New York: Springer; 1993. p. 101–37.
- [2] Larson SJ, Maiman DJ. In: *Surgery of the lumbar spine*. New York, NY: Thieme; 1999. p. 334.
- [3] White III A, Panjabi M. *Clinical biomechanics of spine*. 2nd ed. Philadelphia, PA: JB Lippincott; 1990. 722pp.
- [4] Holdsworth H. Fractures, dislocations and fractures-dislocations of the spine. *J Bone Joint Surg Br* 1963;45:6–20.
- [5] Denis F. Three-column spine and its significance in classification of acute thoracolumbar spinal injuries. *Spine* 1983;8:817–31.
- [6] Louis R. Spinal stability as defined by three-column spine concept. *Anat Clin* 1985;7:33–42.
- [7] White III A, Johnson R, Panjabi M, et al. Biomedical analysis of clinical stability in cervical spine. *Clin Orthop* 1975;109(III):85–95.
- [8] Allen Jr. B, Ferguson R, Lehmann T, O'Brien R. Mechanistic classification of closed indirect fractures and dislocations of the lower cervical spine. *Spine* 1982;7:1–27.
- [9] Gehweiler Jr. J, Osborne Jr. R, Becker R. *Radiology of vertebral trauma*. Philadelphia, PA: Saunders; 1980.
- [10] Whitely J, Forsyth H. Classification of cervical spine injuries. *Am J Roentgenol* 1960;83:633–44.
- [11] Harris Jr. J, Edeiken-Monroe B, Kopaniky D. Practical classification of acute cervical spine injuries. *Orthop Clin North Am* 1986;17:15–30.
- [12] Yoganandan N, Pintar FA, Butler J, Reinartz J, Sances Jr. A, Larson SJ. Dynamic response of human cervical spine ligaments. *Spine* 1989;14:1102–10.
- [13] Pintar FA, Yoganandan N, Voo L. Effect of age and loading rate on human cervical spine injury threshold. *Spine* 1998;23:1957–62.
- [14] Yoganandan N, Pintar FA, Sances Jr. A, Maiman DJ. Strength and motion analysis of the human head–neck complex. *J Spinal Disord* 1991;4:73–85.
- [15] Yoganandan N, Pintar F, Cusick JF. Biomechanics of compression–extension injuries to the cervical spine. In: *Proceedings of the 41st Association for the Advancement of Automotive Medicine*, 1997; Orlando, FL. p. 331–44.
- [16] Yoganandan N, Pintar FA, Arnold P, Reinartz J, Cusick JF, Maiman DJ, et al. Continuous motion analysis of the head–neck complex under impact. *J Spinal Disord* 1994;7:420–8.
- [17] Yoganandan N, Halliday A, Dickman C, Benzel E. *Practical anatomy and fundamental biomechanics*. In: Benzel E, editor. *Spine surgery: techniques, complication avoidance, and management*. New York, NY: Churchill Livingstone; 1999. p. 93–118.
- [18] Yoganandan N, Pintar FA, Gennarelli T, Eppinger RH, Voo LM. Geometrical effects on the mechanism of cervical spine injury due to head impact. In: *Proceedings of the IRCOBI*, 1999; Barcelona, Spain. p. 261–70.
- [19] Yoganandan N, Pintar FA, Kleinberger M. Cervical spine vertebral and facet joint kinematics under whiplash. *J Biomech* 1998;120:305–7.
- [20] Yoganandan N, Pintar FA, Cusick JF, Kleinberger M. Head–neck biomechanics in simulated rear impact. In: *Proceedings of the 42nd Association for the Advancement of Automotive Medicine*, 1998; Charlottesville, VA. p. 209–31.
- [21] Yoganandan N, Pintar F, Kleinberger M. Whiplash injury – biomechanical experimentation. *Spine* 1999;24:83–5.
- [22] Yoganandan N, Pintar FA, Larson SJ, Sances Jr. A, editors. *Frontiers in head and neck trauma: clinical and biomechanical*. The Netherlands: IOS Press; 1998.
- [23] Yoganandan N, Pintar FA, editors. *Frontiers in whiplash trauma: clinical and biomechanical*. The Netherlands: IOS Press; 2000.
- [24] Cusick J, Yoganandan N, Pintar F, Gardon M. Cervical spine injuries from high-velocity forces: pathoanatomic and radiologic study. *J Spinal Disord* 1996;9:1–7.
- [25] Cusick J. Pathophysiology and treatment of cervical spondylotic myelopathy. In: Black P, editor. *Clinical neurosurgery*. Baltimore, MD: Williams & Wilkins; 1991. p. 661–81.
- [26] Hayashi H, Okada K, Hashimoto J, et al. Cervical spondylotic myelopathy in the aged patient: radiographic evaluation of aging changes in cervical spine and etiologic factors of myelopathy. *Spine* 1988;13:618–25.
- [27] Tani T, Yamamoto H, Kimura J. Cervical spondylotic myelopathy in elderly people: high incidence of conduction block at C3–4 or C4–5. *J Neurol Neurosurg Psychiatry* 1999;66:456–64.
- [28] Murray G, Persellin R. Cervical fracture complicating ankylosing spondylitis. *Am J Med* 1981;4:1033–41.
- [29] Karasick D, Schweitzer M, Abidi N, et al. Fractures of vertebrae with spinal cord injuries in patients with ankylosing spondylitis: imaging findings. *Am J Roentgenol* 19xx;165:1205–8.
- [30] Thorngren K, Liedberg E, Aspelin P. Fractures of thoracic and lumbar spine in ankylosing spondylitis. *Arch Orthop Trauma Surg* 1981;98:101–7.
- [31] Harris Jr. J, Mirvis S. *Radiology of acute cervical spine trauma*. 3rd ed. Baltimore, MD: Williams & Wilkins; 1996. p. 512.
- [32] Bucholz R, Burkhead W. Pathologic anatomy of fatal atlanto-occipital dislocations. *J Bone Joint Surg Am A* 1979;61:248–50.
- [33] Davis D, Bohlman H, Walker A, Fisher R, Robinson R. Pathological findings in fatal craniospinal injuries. *J Neurosurg* 1971;34:603–13.
- [34] Jonsson Jr. H, Bring G, Rauschnig W, Sajlstedt B. Hidden cervical spine injuries in traffic accident victims with skull fractures. *J Spinal Disord* 1991;4:251–63.
- [35] Yoganandan N, Haffner M, Maiman DJ, Nichols H, Pintar FA, Jentzen J, et al. Epidemiology and injury biomechanics of motor vehicle related trauma to the human spine. *SAE Trans* 1990;98:1790–807.
- [36] Pintar FA, Yoganandan N, Gennarelli TA. Head–neck tension biomechanical models for pediatric and small female populations. In: *Proceedings of the 43rd AAAM Conference*, 1999; Barcelona, Spain. p. 357–66.
- [37] Anderson L, D'Alonzo R. Fractures of odontoid process of the axis. *J Bone Joint Surg Am A* 1974;56:1663–74.
- [38] Levine A, Edwards C. Treatment of injuries in C1–C2 complex. *Orthop Clin North Am* 1986;17:31–44.

- [39] Pintar F, Yoganandan N, Voo L, Cusick J, Maiman D, Sances Jr. A. Dynamic characteristics of human cervical spine. *SAE Trans* 1995;104:3087–94.
- [40] Pintar FA, Yoganandan N, Voo LM, Cusick JF, Maiman DJ, Sances Jr. A. Dynamic characteristics of the human cervical spine. *SAE Trans* 1995;104:3087–94.
- [41] Pintar FA, Yoganandan N, Schlick MB. Biodynamics of cervical spinal injury. In: *Proceedings of the International IRCOBI Conference on Biomechanics of Impacts*, 1995; Brunnen, Switzerland. p. 285–94.
- [42] Ching R, Tenser A, Anderson P, Daly C. Comparison of residual stability in thoracolumbar spine fractures using neutral zone measurements. *J Orthop Res* 1995;13:533–41.
- [43] Fielding J. Normal and selected abnormal motion of cervical spine from second cervical vertebra to seventh cervical vertebra on cinerentgenography. *J Bone Joint Surg Am A* 1964;46:1779–81.
- [44] Breig A. *Biomechanics of central nervous system*. Chicago, IL: Year Book Medical; 1960.
- [45] Breig A, Turnbull I, Hassler O. Effects of mechanical stress on spinal cord in cervical spondylosis: study of fresh cadaver material. *J Neurosurg* 1966;25:45–56.
- [46] Ehni G. *Cervical arthrosis: diseases of cervical motion segments*. Chicago, IL: Year Book Medical; 1984.
- [47] Penning L. *Functional pathology of the cervical spine* 1968.
- [48] Edwards W, LaRocca H. Development segmented sagittal diameter of cervical spinal cord in patients with cervical spondylosis. *Spine* 1983;8:20–7.
- [49] Hashimoto I, Tak Y. True sagittal diameter of cervical spinal canal and its diagnostic significance in cervical myelopathy. *J Neurosurg* 1977;47:912–6.
- [50] Wolf B, Khilnani M, Malis L. Sagittal diameter of bony cervical spinal canal and its significance in cervical spondylosis. *J Mt Sinai Hospital* 1956;23:283–92.
- [51] Brain R. Cervical spondylosis. *Ann Intern Med* 1954;41:115–25.
- [52] Cusick J, Myklebust J, Zyvoploski M, Sances Jr. A, Hauterman C, Larson S. Effects of vertebral column distraction in the monkey. *J Neurosurg* 1982;57:651–9.
- [53] Reid J. Effects of flexion–extension movements of head and spine upon spinal cord and nerve roots. *J Neurol Neurosurg Psychiatry* 1960;23:214–21.
- [54] Alem N, Nusholtz G, Melvin J. Superior–inferior head impact tolerance levels. Ann Arbor, MI: University of Michigan; 1982. 271pp.
- [55] Clark CR, Ducker TB, Dvorak J, Garffin SR, Herkowitz HN, Levine AM, et al. In: *The cervical spine*. 3rd ed. Philadelphia, PA: Lippincott–Raven; 1998, 1003 pp.
- [56] Cusick JF, Pintar FA, Yoganandan N, Baisden J. Wire fixation techniques of the cervical facets. *Spine* 1997;22:970–5.
- [57] Cusick JF, Yoganandan N, Pintar FA, Gardon M. Cervical spine injuries from high velocity forces: a pathoanatomical and radiological study. *J Spinal Disord* 1996;9:1–7.
- [58] Huelke DF, Nusholtz GS. Cervical spine biomechanics: a review of the literature. *J Orthop Res* 1986;4:232–45.
- [59] McElhaney JH, Paver JG, McCrackin HJ, Maxwell GM. Cervical spine compression responses. In: *Proceedings of the 27th Stapp Car Crash Conference*, 1983; San Diego, CA. p. 163–77.
- [60] McElhaney JH, Roberts VL, Hilyard JF, editors. *Handbook of human tolerance*. Tokyo, Japan: Japan Automobile Research Institute, Inc; 1976.
- [61] Nightingale RW, McElhaney JH, Camacho DL, Kleinberger M, Winkelstein BA, Myers BS. The dynamic responses of the cervical spine: buckling, end conditions, and tolerance in compressive impacts. In: *Proceedings of the 41st Stapp Car Crash Conference*, 1997; Orlando, FL. p. 451–71.
- [62] Nusholtz GS, Melvin JW, Huelke DE, Alem NM, Blank JG. Response of cervical spine to superior inferior head impact. In: *Proceedings of the 25th Stapp Car Crash Conference*, 1981; San Francisco, CA. p. 197–237.
- [63] Pintar FA, Sances A Jr., Yoganandan N, Reinartz JM, Maiman DJ, Suh JK. Biodynamics of the total human cadaver cervical spine. In: *Proceedings of the 34th Stapp Car Crash Conference*, 1990; Orlando, FL. p. 55–72.
- [64] Pintar FA, Yoganandan N, Sances Jr. A, Reinartz J, Harris G, Larson SJ. Kinematic and anatomical analysis of the human cervical spinal column under axial loading. *SAE Trans* 1990;98:1766–89.
- [65] Maiman DJ, Yoganandan N. Biomechanics of cervical spine trauma. In: Black P, editor. *Clinical neurosurgery*, vol. 37. Baltimore, MD: Williams & Wilkins; 1991. p. 543–70.
- [66] Pintar F, Voo L, Yoganandan N. The mechanisms of hyperflexion cervical spine injury. In: *Proceedings of the 16th International Research Council on the Biomechanics of Impact Conference*, 1998; Goteborg, Sweden. p. 349–63.
- [67] Pintar FA, Schlick MB, Yoganandan N, Maiman DJ. Instrumented artificial spinal cord for human cervical pressure measurement. *Bio-Med Mat Eng* 1996;6:219–29.
- [68] Sances Jr. A, Myklebust JB, Maiman DJ, Larson SJ, Cusick JF, Jodat R. The biomechanics of spinal injuries. *CRC Crit Rev Bioeng* 1984;11:1–76.
- [69] Yoganandan N, Pintar FA. Biomechanics of the cranio-cervical region. In: Boeker D, editor. *Cranio-cervical junction – anatomy, physiology therapy*. GMBH: Biermann Verlag [in press].
- [70] Yoganandan N, Sances Jr. A, Pintar FA, Maiman DJ, Reinartz J, Cusick JF, et al. Injury biomechanics of the human cervical column. *Spine* 1990;15:1031–9.
- [71] Yoganandan N, Maiman DJ, Pintar FA, Sances Jr. A, Chintapalli K, Unger GF. Cervical spine injuries from motor vehicle accidents. *J Clin Eng* 1990;15:505–13.
- [72] Yoganandan N, Sances Jr. A, Maiman DJ, Myklebust JB, Pech P, Larson SJ. Experimental spinal injuries with vertical impact. *Spine* 1986;11:855–60.
- [73] Yoganandan N, Sances Jr. A, Pintar FA. Biomechanical evaluation of the axial compressive responses of the human cadaveric and manikin necks. *J Biomech Eng* 1989;111:250–5.
- [74] Yoganandan N, Pintar FA, Wilson CR, Sances Jr. A. In vitro biomechanical study of female geriatric cervical vertebral bodies. *J Biomed Eng* 1990;12:97–101.
- [75] Yoganandan N, Pintar FA, Kleinberger M. Cervical vertebral and facet joint kinematics under whiplash. *J Biomech Eng* 1998;120:305–8.
- [76] Culver R, Bender M, Melvin J. Mechanisms, tolerances, and responses obtained under dynamic superior–inferior head impact. Ann Arbor, MI: University of Michigan; 1978. 103pp.
- [77] AIS. *The Abbreviated Injury Scale*. Arlington Heights, IL: American Association for Automotive Medicine; 1990. p. 74.
- [78] Yoganandan N, Myklebust JB, Ray G, Sances Jr. A. Mathematical and finite element analysis of spinal injuries. *CRC Rev Biomed Eng* 1987;15:29–93.
- [79] Allen BL, Ferguson RL, Lehmann TR, O'Brien RP. A mechanistic classification of closed indirect fractures and dislocations of the lower cervical spine. *Spine* 1982;7:1–27.
- [80] Sherk HH, Dunn EJ, Eismont FJ, Fielding JW, Long DW, Ono K, et al. In: *The cervical spine*. 2nd ed. Philadelphia, PA: JB Lippincott, Co; 1989. p. 881.
- [81] Winkelstein B, Myers B. Determinants of catastrophic neck injury. In: Yoganandan N, Pintar FA, Larson S, Sances AJ, editors. *Frontiers in head and neck trauma: clinical and biomechanical*. The Netherlands: IOS Press; 1998. p. 266–95.
- [82] Nusholtz GS, Huelke DE, Luz P, Alem NM, Montavo F. Cervical spine injury mechanisms. In: *Proceedings of the*

- 27th Stapp Car Crash Conference, 1983; San Diego, CA. p. 179–98.
- [83] Sances Jr. A, Myklebust JB, Cusick J, Weber R, Houterman C, Larson S, et al. Experimental studies of brain and neck injury. *SAE Trans* 1981;90:3378–400.
- [84] Maiman DJ, Sances Jr. A, Myklebust JB, Larson SJ, Houterman C, Chilbert M, et al. Compression injuries of the cervical spine: a biomechanical analysis. *Neurosurgery* 1983;13:254–60.
- [85] Yoganandan N, Pintar FA, Maiman DJ, Cusick JF, Sances Jr. A, Walsh PR. Human head–neck biomechanics under axial tension. *Med Eng Phys* 1996;18:289–94.
- [86] Rothman RH, Simeone FA.. 3rd ed. *The spine*, vol. 1. Philadelphia, PA: Saunders; 1992. p. 969.
- [87] Yoganandan N, Kumaresan S, Pintar F, Gennarelli T. Pediatric biomechanics. In: Nahum A, Melvin J, editors. *Accidental injury: biomechanics and prevention*. New York: Springer. in press.
- [88] Yoganandan N, Pintar FA, Cusick JF. Biomechanical analyses of whiplash injuries using experimental model. *Accid Anal Prev* [in press].
- [89] Nightingale RW, McElhaney JH, Richardson WJ, Myers BS. Dynamic responses of the head and cervical spine to axial impact loading. *J Biomech* 1996;29:307–18.
- [90] Yoganandan N, Pintar FA, Sances Jr. A, Reinartz J, Larson SJ. Strength and kinematic response of dynamic cervical spine injuries. *Spine* 1991;16:511–7.
- [91] Foust DR, Chaffin DB, Snyder RG, Baum JK. Cervical range of motion and dynamic response and strength of cervical muscles. In: *Proceedings of the 17th Stapp Car Crash Conference*, 1973; Oklahoma City, OK. p. 285–308.
- [92] McElhaney JH, Myers BS. Biomechanical aspects of cervical trauma. In: Nahum AM, Melvin JW, editors. *Accidental injury: biomechanics and prevention*. New York: Springer; 1993. p. 311–61.
- [93] Alem NM, Nusholtz GS, Melvin JW. Head and neck response to axial impacts. In: *Proceedings of the 28th Stapp Car Crash Conference*, 1984; Warrendale, PA. p. 275–88.
- [94] Bates-Carter J, Ching R, Tencer AF. Transient changes in canal geometry during axial burst fracture of the cervical spine. In: *Proceedings of the Injury Prevention Through Biomechanics*, 1995; Detroit, MI. p. 171–77.