

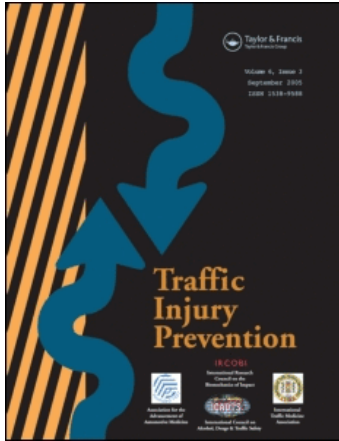
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## Traffic Injury Prevention

Publication details, including instructions for authors and subscription information:

<http://www.informaworld.com/smpp/title~content=t713456148>

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To cite this Article Yoganandan, Narayan , Pintar, Frank A. and Gennarelli, Thomas A.(2002) 'Biomechanical Mechanisms of Whiplash Injury', Traffic Injury Prevention, 3: 2, 98 – 104

To link to this Article: DOI: 10.1080/15389580211999

URL: <http://dx.doi.org/10.1080/15389580211999>

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# Biomechanical Mechanisms of Whiplash Injury

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*It is common knowledge that whiplash-associated disorders are soft-tissue-related. Rear-end crashes account for a majority of trauma. Specific objective diagnoses have eluded clinicians because of the soft tissue nature of the disorder. Sophisticated tools such as magnetic resonance images are inconclusive. The disorder is recognized in the Western world with significant societal impact and staggering economic costs. An increased level of awareness towards safety from the vehicle user coupled with the above factors and an interest by the automotive community to improve vehicle component designs have accelerated research in the area of whiplash-associated disorders from various perspectives. One of the main emphases has been to clearly delineate the mechanisms of the disorder. Consequently, the objective of this article is to describe the postulated mechanisms of injury and biomechanical studies attempting to prove the hypotheses. Results indicate that structures such as the facet joint are involved in chronic pain, and the kinematics of this joint are such that it undergoes characteristic motions during the early stages of rear impact acceleration. The presence of the transient nonphysiologic reverse curve, i.e., upper head-neck flexion is attributed to headaches, and the concomitant existence of lower cervical extension (particularly the inferior facet joints) during the early stages of rear impact acceleration are attributed to the mechanism of neck pain in whiplash. These studies have provided a fundamental basis for understanding the mechanism of the two most common complaints in whiplash, headache and neck pain.*

**Keywords** Biomechanics; Facet Joint; Headache; Kinematics; Neck Pain; Whiplash

From a biomechanical perspective, the mechanism of injury can be defined as the process in which injuries occur to the human tissues secondary to the application of external mechanical insult, e.g., inertial postero-anterior acceleration stemming from rear-end crashes in a motor vehicle environment. Although clinical literature is replete with reports of disorders due to rear-end crashes, often termed whiplash-associated disorders or simply whiplash, there is a lack of consensus in the biomechanical community with regard to the specific mechanism of injury. This is primarily because of the elusive nature of the disorder, i.e., lack of objective diagnosis in the patient population. It is common knowledge that whiplash is a soft tissue injury and belongs to the low end of the Abbreviated Injury Scale (AIS, 1990). Sophisticated imaging modalities, such as computed tomography and magnetic resonance scans, are not conclusive in identifying structural components involved in the disorder. More potentially confounding factors are the societal effects and the prevailing laws in the community, as whiplash

trauma bears a litigious face. From an epidemiological perspective, annual costs are estimated at \$10 billion in the United States, £2.5 billion in the United Kingdom, and 10 to 20 billion DM in Germany (Castro et al., 1997; Galasko, 1996; Kaufmann et al., 1995; Yoganandan & Pintar, 2000). These staggering costs and public awareness for safety, coupled with the interest by the vehicular community, appear to be responsible for the recent surge in biomechanical research to delineate the mechanisms of injury. Consequently, this article addresses issues regarding the mechanisms of injury and discusses developments in the area.

## HYPOTHESES

To analyze complaints following railway incidents, the terminology “railway spine” was used in 1886, and reports included headache and neck pain (Yoganandan et al., 1998c). To describe spinal motions in eight cases of neck trauma secondary to motor vehicle crashes, the term “whiplash” was coined in 1928. Although this was the first citation of the use of this phrase to our knowledge, early scientific papers about whiplash injuries and their mechanisms were not published until the 1950s (Braaf & Rosner, 1955; Gay & Abbott, 1953). More than 10,000 studies

Received 10 December 2001; accepted 7 March 2002.

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on whiplash injuries were published before 1995 (Spitzer et al., 1995). Many postulates exist on the mechanisms of injury. Almost all hypotheses on the mechanism of rear-impact-induced injury involve kinematics as a fundamental basis. Following are some of the theorized mechanisms.

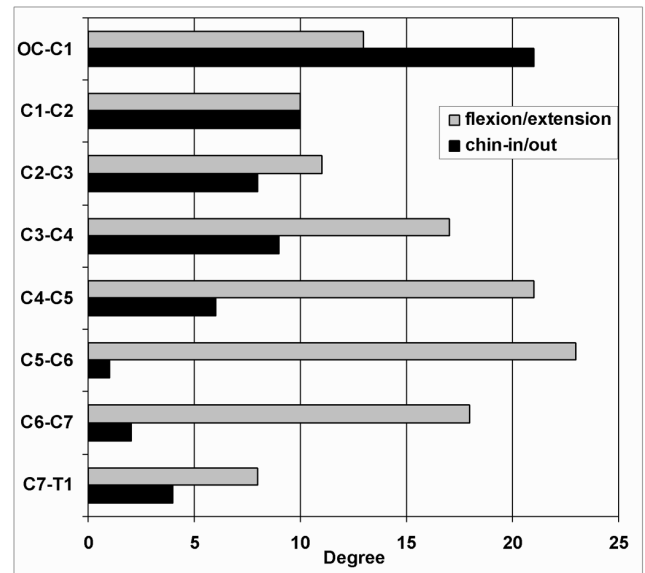
In 1953, one of the first proposals was made; the mechanism was thought to be flexion followed by extension based on clinical assessment including radiography and understanding of rear impact events (Gay & Abbott, 1953). However, in 1955 this was disproved by controlled laboratory tests that determined the opposite to be true, i.e., extension followed by flexion (Severy et al., 1955).

In 1964, based on disc-related injuries due to extension of the anterior cervical column from primate tests, the hyperextension mechanism of injury was proposed (Macnab, 1964). Clinicians have routinely used this mechanism to explain whiplash-associated disorders. Rear impact experiments with human volunteers have shown the absence of hyperextension of the head beyond physiological limits (Matsushita et al., 1994; McConnell et al., 1995). Therefore, the hyperextension mechanism refers to the motion of the neck. Although functional images provide information on the degree of physiologic extension of the human head-neck complex, precise limits of hyperextension are not clearly established in the dynamic domain. As explained later, recent biomechanical investigations, however, have not been able to fully confirm the hyperextension mechanism.

During the years 1967–1971, controlled rear impact experiments were conducted using physical models, volunteers, and embalmed cadavers; although this research was not primarily aimed to delineate the mechanisms of injury, extension limits were proposed (Mertz & Patrick, 1967; Mertz & Patrick, 1971). This implies extension or hyperextension is the plausible mechanism.

In 1975, the shear theory for whiplash was postulated based on radiographic studies (Aufdermaur, 1975). As described later, only recently has this mechanism received attention from biomechanics researchers. In fact, until the late 1980s a void seems to have existed in the proposals for additional mechanisms or studies to prove the proposed mechanisms of injury. Worldwide attention due to parameters such as public awareness, litigation, and societal costs appear to be the driving factors for the intensified research on this topic since the last decade.

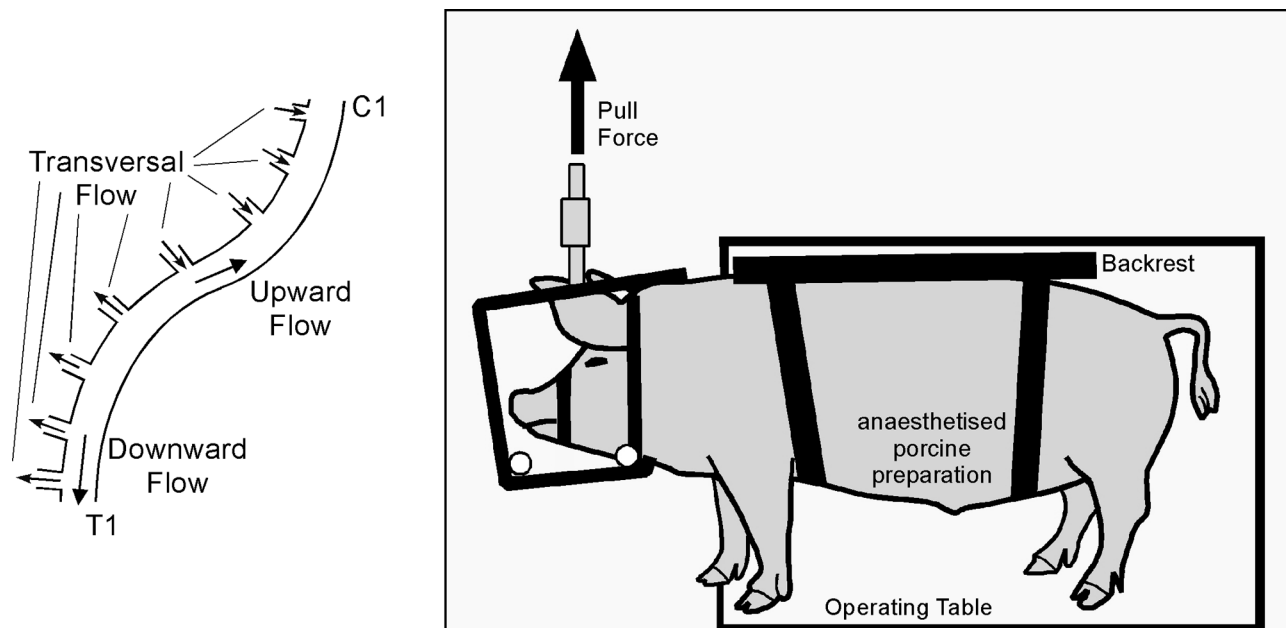
During the years 1992–1994, hypertranslation of the head was postulated as a plausible mechanism of whiplash injury (Penning, 1992; Penning, 1994). This mechanism was primarily based on a comparison of lateral x-rays of chin-in and chin-out subjects (representative of posterior and anterior head translations) with functional radiographs. Because rotations in the cranio-vertebral region, i.e., OC–C2, were greater with head translation than with head extension and flexion, whiplash trauma was attributed to the cephalad portion of the cervical spine (see Figure 1). This postulate, i.e., the injury concentrating in the upper cervical region, is yet to be supported by laboratory and clinical studies, which have consistently suggested the lower cervical spine to be a major source of trauma (Grauer et al., 1997).



**Figure 1** Range of motion in human volunteers under different initial conditions (adapted from Penning, 1992).

In 1994, using the hydrodynamic theory suggested in 1986, European researchers proposed another mechanism of rear-impact-induced whiplash injury (Aldman, 1986). Briefly, the hydrodynamic theory involves the concept of pressure alterations inside the spinal canal secondary to extension–flexion changes in the spinal curvature due to rear impact accelerations. The spinal canal lengthens and shortens in extension and flexion, and this change in length, which affects the pressure inside the canal (Figure 2), may traumatize the dorsal root ganglia leading to whiplash-associated disorders. Radiculopathy secondary to a pinched nerve or dorsal ganglia may lead to pain in specific areas (e.g., arm) of the human body depending on the level of the involved vertebral segment. It should be emphasized that radiculopathy is not the most common complaint in whiplash patients. Using the pressure concept as the basis, spinal canal pressures were recorded from experiments that involved pulling the head of two anesthetized pigs from the ventral to dorsal direction (Figure 2) and documenting dysfunctions in the cell membrane of the dorsal root ganglion (Ortengren et al., 1996). Although these data were obtained from an experimental animal model, soft tissue injuries resulting from pressure changes in the canal were not documented in studies by the same group of authors using the more realistic rear impact postero-anterior acceleration as the input and human cadaver as the experimental model (Darok et al., 2000).

Neck flexion and extension motions are often used to explain muscular trauma from rear impact accelerations. Injury to these structures occurs as secondary to eccentric contraction (Garrett et al., 1997). Anterior neck muscles undergo eccentric contraction during neck hyperextension. Although anterior muscles can be injured in this mode, they do not lead to chronic disorders. Another mechanism of whiplash injury, proposed using kinematic and anatomic concepts, attributes the disorder to eccentric



**Figure 2** Left: Schematic of the internal venous plexus demonstrating possible fluid flows from C1 to T1 levels. The curvature approximates the transient signature sustained by the human neck during the early stages of whiplash acceleration (redrawn from Bostrom et al., 1996). Right: Schematic of the test setup used by Ortengren et al., 1996 (redrawn) to simulate whiplash experiments on pigs.

contraction of posterior neck muscles during the rebound phase of neck motion (Tencer et al., 1999). During rebound, because of the flexion curvature of the neck, it is theoretically possible to alter muscle activity leading to injury in the extensors. Although this mechanism is biomechanically consistent with the finding that eccentric contractions can induce muscle trauma (Garrett et al., 1997), because of the low severity of rear impact accelerations in patients with whiplash-associated disorders, the following statement has been made to shed doubt on this mechanism. The hypothesis (of muscle injury in the rebound phase) “flies in the face of many more frontal impacts in which there is hyperflexion due to severe crashes, and there are not a large number of complaints of neck ache from these victims” (Deng et al., 2000). Additional studies are needed to fully investigate the validity of this mechanism.

In 1999, intervertebral rotations in the initial extension phase of the S-curve were used as a primary mechanism of injury to quantify whiplash trauma (Panjabi et al., 1999). These were based on tests carried out using isolated human cadaver ligamentous cervical spines attached to an artificial head and conducting repeated experiments with sled accelerations ranging from 2.5 g (change in velocity,  $\Delta V = 6$  km/h) to 10.5 g ( $\Delta V = 16$  km/h). Intervertebral rotations exceeded normal physiologic limits at input levels of 2.5 g at C2–3, C4–5, C6–7, and C7–T1; 4.5 g at C6–7, 6.5 g at C4–5, C6–7, and C7–T1; and 8.5 g at C6–7 and C7–T1 (Grauer et al., 1997). Elongations of the vertebral artery exceeded physiological limits by 1.0, 3.1, 8.9, and 9.0 mm at 2.5, 4.5, 6.5, and 8.5 g acceleration levels, respectively (Nibu et al., 1997). These findings should be interpreted with caution as repeated impacts do not occur in the real world, and the effects

on injury of using an artificial head in conjunction with a pure ligamentous column are not known. Along with this research, other human cadaver studies have reported that injuries occur to the lower cervical spine before the neck undergoes hyperextension, refuting the hyperextension mechanism of injury (Deng et al., 2000; Grauer et al., 1997).

The shear and compression hypothesis attributes neck pain to the stretch of the facet capsule resulting from these two biomechanical variables. In a rear impact, shear forces develop in the cervical spine because the forward motion of the torso occurs before the head motion. The shearing action imparts a relative motion between adjacent vertebrae, and this motion is highest in the lower cervical level because of the facet orientation (Deng et al., 2000). In addition, compressive forces are generated early in the acceleration phase due to uncoiling or straightening of the thoracic spine, a finding reported in rear impact simulations using human volunteers (Matsushita et al., 1994; McConnell et al., 1993; Ono et al., 1999). In rear impacts, anterior shearing of the lower vertebra occurs, and its facet joint offers little or no protection. This is in contrast to frontal impacts wherein the anterior shearing of the upper vertebra occurs, and its contact with the facet joints provides protection because of the anatomical orientation of these structures. Axial compressive force does not exist in the frontal mode.

In 2000, facet capsule stretches from sled experiments were measured (Deng et al., 2000). Repeated accelerations were applied to whole-body cadavers, and high-speed cine-radiography techniques were used to track cervical intervertebral motions. Six human whole-body cadavers were subjected to 26 tests at velocities ranging from 4.8 to 16 km/h (sled acceleration

levels 5 to 9 g) with 0° and 20° combinations of seatback angles, with and without headrests. Injuries to the disc, facet joints, and ligaments of the cervical structures were discovered at autopsy in four out of the six specimens. Repeated acceleration loading is atypical in real world rear-impact-induced whiplash trauma. Nonetheless, these studies clearly showed the role of kinematics in rear impact analysis. Furthermore, peak capsular strains occurred before maximum head rotation and before head contact with the head restraint. This finding may have important implications with regard to the effects of muscles on the injury. A study examining electromyography signals from neck musculature during human volunteer rear impact auto collisions reported that initial muscle activation does not occur until 100 msec after event start, and full muscle contraction does not occur until 150–170 msec after event onset when the head–neck is already in extension (Szabo & Welcher, 1996). Similar findings have been confirmed by other human volunteer studies. Furthermore, animal experiments have demonstrated that the time to develop muscle force is approximately 200 ms (Tennyson et al., 1977). Therefore, the hypothesis from this literature is that injury occurs before this time and, therefore, active muscle contraction is not a primary determinant.

An evaluation of previous research aiming at the identification of soft-tissue-related trauma secondary to a single application of rear impact acceleration shows inconsistent data (Table I). In fact, a systematic documentation of the injury and the associated mechanism for injury production has not been done. For example, studies reported disc injuries with tears of the anterior longitudinal ligament, vertebral fractures, and some capsular tears (Clemens & Burow, 1972). Severe compression fractures of cervical vertebra have also been reported (Hu et al., 1977). Because the input pulse used in these studies is severe [25 g (Clemens & Burow, 1972) and 18 g (Hu et al., 1977)], it is not representative of real world whiplash (King, 2000). The common inconsistency among most of the previous studies is repeated loading to the same embalmed cadaver (Mertz & Patrick, 1967; Mertz & Patrick, 1971); five sled tests on one embalmed

cadaver (Prasad et al., 1975); 28 sled tests on five cadavers (Eichberger et al., 1998); 19 tests on three cadavers (Bertholon et al., 2000); 26 tests on six cadavers (Deng et al., 2000); and 49 tests on six cadavers (Geigl et al., 1995). As remarked earlier, repeated loading is atypical of real world, rear crash-induced whiplash injury. Another comment is that the injury documentation generally involved male (Kallieris et al., 1996; Mertz, 1967; Mertz & Patrick, 1967) or unknown gender and anthropometry (Eichberger et al., 1998). Furthermore, injury determinations were inconsistent (x-ray, autopsy) and nonspecific (Table I). It is difficult to detect hemorrhage and other types of soft tissue injuries from autopsy (Eichberger et al., 2000). These studies highlight the need for production and detailed documentation of soft-tissue-related neck injuries due to a single event whiplash acceleration input and the associated mechanism of injury.

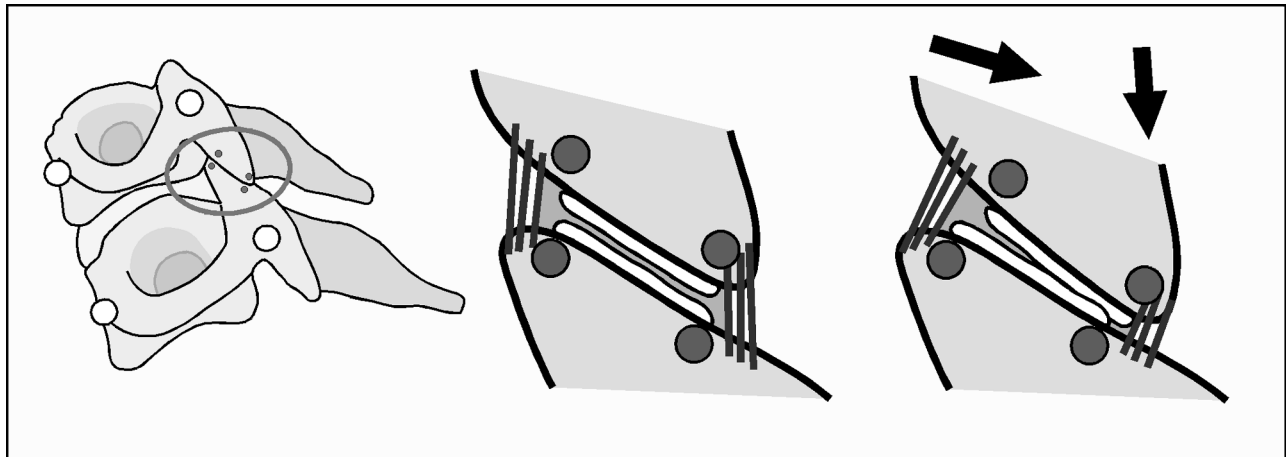
During the years 1996–2001, researchers from the Medical College of Wisconsin proposed kinematics-based hypotheses for the mechanisms of injury applicable to the two most common complaints reported by patients sustaining whiplash-associated disorders (Yoganandan et al., 1996). Mechanisms of headache and neck pain were explained using head–neck motions determined from in vitro experiments simulating rear impact accelerations (Yoganandan et al., 1999; Yoganandan et al., 1998a). Based on the transient, nonphysiological reverse (S-shape) curve that occurs during the initial stages of acceleration, these studies indicated local flexion of the head–upper cervical spine. Posterior stretch in the suboccipital region secondary to this local flexion may alter the mechanics and electrical activity of suboccipital muscles, leading to headaches. Facet joint compromise at OC–C2 levels, in the form of distraction of the capsule, may also lead to headache (Cusick et al., 2001).

Thus, the transient flexion at the upper cervical spinal region explained the mechanism of one type of whiplash injury. The same group of researchers proposed a hypothesis for the mechanism of neck pain (Cusick et al., 2001; Yoganandan et al., 1999; Yoganandan et al., 1998a). The pinching and resultant stretch of the facet joints of the lower cervical spine secondary to local

**Table I** Injury data from intact cadaver tests

First author (reference)	# cadavers (# of tests)	Injury	Injured component
Mertz (Mertz & Patrick, 1967)	2 (4)	X-rays: One embalmed male cadaver “minor ligamentous damage occurred between the third and fourth cervical vertebrae”	NS
Prasad (Prasad et al., 1975)	1 (5)	NR	NS
Kallieris (Kallieris et al., 1996)	2 (2)	Autopsy: One of two male unembalmed cadavers “the sclerotic front longitudinal ligament was fractured at the lower edge of C3”	NS
Geigl (Geigl et al., 1995)	6 (49)	NR	NS
Eichberger (Eichberger et al., 1998)	5 (28)	Autopsy: One of five cadavers “minor ligament damage to the cervical spine to a cadaver”	NS
Bertholon (Bertholon et al., 2000)	3 (19)	NR	NS
Deng (Deng et al., 2000)	6 (26)	Autopsy: Disc and anterior ligament injuries of the lower spine in four cadavers and tear of C7–T1 capsule in one unembalmed cadaver.	Disc in 4 Facet in 1
Yoganandan (Yoganandan et al., 2000)	5 (5)	X-ray, computed tomography and cryomicrotomy: Soft tissue-related injuries in four out of five specimens with injuries concentrated in the lower cervical regions, specifically at the C5–6 level	Disc annulus, facet joint, ligaments

NR: Not reported; NS: Not specific.



**Figure 3** Schematic representation of local lower cervical facet joint kinematics occurring during the early stages of whiplash acceleration. Left: Lateral view of the lower cervical functional unit (entire cervical spinal column not shown) indicating the four targets surrounding the facet joint. Middle: Facet joint in the normal physiologic position. Right: Pinching action on the posterior region of the facet joint with a tensing action on the anterior region combined with the anteroposterior slide, contributing to resultant joint stretch. The facet joint cartilage does not cover the entire surface of the articular process. The implications of these motions for chronic neck pain are discussed in the text (adapted from Cusick et al., 2001).

extension may alter the integrity of the facet joints, leading to neck pain (Figure 3). Intact head–neck complex tests were used to determine the following kinematics to support the mechanism: superior–inferior (SI) compression of the dorsal region, SI stretch of the ventral region, and a simultaneous sliding of the facet joint resulting in a stretch of the joint during the early phase of the whiplash acceleration pulse. The lower cervical spine was in extension during this period. The pinching action suggests the potential of the dorsal regions of the facet joint to come in contact with the subchondral bone via cartilage compression (Cusick et al., 1999; Yoganandan et al., 1998b). Human volunteer tests supporting the mechanism have referred to this as “facet impingement” or “collision” (Kaneoka et al., 1999; Ono et al., 1999). Mere compression of cartilage is inadequate to induce pain because it is deprived of nerve endings. However, cartilage degradation in the form of surface microfissures accentuates osteoarthritis, leading to long-term changes in the mechanics of the innervated subchondral bone. It should be noted that the “pinching” action does not refer only to the SI/axial component of the facet joint motion. The resultant motion of the ventral and dorsal regions of the joint (axial and sliding motions) describes the stretch of the joint. As delineated above, intact head–neck complex tests showed that the joint stretches nonuniformly across its articulation because the individual (axial) motion components are not identical at the dorsal and ventral ends of the joint, although the shear/sliding is uniform (Stemper et al., 2001). Any combination of these motions (stretch) may compromise the integrity of the joint, fire nerve endings, and elicit pain.

The same group of researchers conducted sled tests using intact whole-body human surrogates. The surrogates were restrained, and a single application of rear impact pulse ( $\Delta V = 15$  or  $25$  km/h) was used to determine the biomechanics of rear impact acceleration and to document soft tissue injuries. The interaction of the human torso in the form of uncoiling of the thoracic

spine introducing a compressive force on the neck was automatically incorporated into this experimental model. Tests with specimens with 5th percentile female and 95th percentile male anthropometry and at velocities cited above resulted in peak axial compressive forces ranging from 100 to 254 N (Yoganandan et al., 2000). These data were remarkably consistent with the maximum compressive loads (ranging from 33 to 258 N) calculated from other intact whole-body cadaver sled tests conducted at 5 to 16 km/h velocities (Deng et al., 2000). The finding of similar levels of peak compressive force regardless of specimen anthropometry, test speed, and other varying experimental parameters appears to indicate a secondary role for the compressive component (attributed to be stemming from the straightening of the thoracic spinal column) on the biomechanical mechanisms of injury. However, because of the small sample size, additional studies are needed to clearly quantify the effect of the compressive force reflecting the role of the uncoiling of the dorsal spinal column on injury.

Soft tissue injuries in these studies were identified using radiography, close-up computed tomography, and sequential cryomicrotomy (Yoganandan et al., 2001a). Injuries were mainly concentrated to the facet and disc joints at the lower cervical spine. More specifically, in the lower cervical spine the C5–C6 level was identified as the region most susceptible to trauma. These findings were correlated with the microlevel kinematics of the facet joints in the head–neck complex model (described in the earlier paragraph) wherein significantly different motions were derived for the C5–C6 joint compared to its cephalad caudal counterparts (Yoganandan et al., 2001b). These results parallel clinical literature wherein the C5–C6 joint is cited as the anatomic source for neck pain in whiplash patients (Yoganandan & Pintar, 2000). Controlled biomechanical investigations from this group of researchers have provided explanations for the two most common complaints in whiplash, i.e., two plausible mechanisms of injury for headache and neck pain, and documented

the occurrence of soft tissue injuries due to a single application of rear impact acceleration. It should be emphasized that because soft tissue injuries were produced and documented secondary to a single event, the proposed mechanisms have biomechanical support. Further analyses of these data with a focus on developing injury criteria should be of value as clinically applicable soft tissue trauma has been identified.

### SUMMARY

The postulated mechanisms of injury and biomechanical studies attempting to prove the hypotheses are described. Although knowledge has been gained from recent studies, consensus does not fully exist on the precise cause of injury production as the trauma is confined to soft tissues and still eludes objective clinical diagnoses. However, structures such as the facet joint have been clearly shown in clinical and experimental biomechanical studies to be involved in chronic pain, and the kinematics of this joint are such that it undergoes characteristic motions during the early stages of rear impact acceleration. The presence of the transient nonphysiologic reverse curve, i.e., upper head-neck flexion is attributed to the headaches, and the concomitant existence of the lower cervical spine extension during the early stages of rear impact acceleration are attributed to the mechanism of neck pain in whiplash. The foregoing discussions have enriched our knowledge on the mechanism and production of whiplash injuries.

The principal components involved in defining the mechanisms of injury include the identification of tissues (e.g., capsular ligament) sustaining the stresses/strains that result in damage, determination of local mechanical variables (e.g., facet joint strain distributions), documentation of the temporal sequence in the attainment of postures (e.g., changes in the local kinematics of the various cervical segments), derivation of injury metrics (e.g., extension moment at the occipital condyles), and establishment of injury criteria. As discussed before, many mechanisms have been proposed and in some cases injury criteria have been derived based on the postulates. Because the actual injury/damage sustained by the structural components was not identified, the validity of the mechanism and/or the applicability of the injury criteria is questionable. This inconclusive output is chiefly responsible for the confusion that exists in the biomechanical assessment of whiplash-associated disorders. In fact, dummies have been designed and validated based on other dummies for rear impact-induced injury without actually possessing injury-related biomechanical data (though a discussion on this topic is beyond the scope of this paper). Identification of the structural components that may sustain injury is a critical step in understanding the disorder. In order to simulate the real world whiplash scenario, it is imperative to subject experimental models to a single event rear impact acceleration, a condition often not reproduced in literature. Because biological tissues including the human vertebral column are sensitive to loading history, repeated acceleration input alters the load carrying capacity and, hence, injury to its structures.

The two critical factors in the determination of whiplash injury mechanisms are, therefore, the application of single event acceleration and documentation of injury to the soft tissues structures secondary to the load. It is worth noting that these two factors have received due attention in contact-induced injuries (e.g., compression-related burst and wedge fractures of the cervical spine) and have assisted in the determination of mechanisms of injury and derivation of human tolerance limits (Pintar et al., 1998). Such attention is clearly needed in whiplash injury research.

### ACKNOWLEDGMENTS

This research was supported in part by PHS CDC 515433 and VA Medical Research.

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