Clinical and Biomechanics Analysis of Whiplash Injuries

Haibin Chen[1], Liying Zhang[1, 2], Zhengguo Wang[1], Xuemei Cheng[1,3], Yi Wang[1], Ling Zhang[1]

[1]*State Key Laboratory of Trauma, Burns, and Combined Injuries, Institute of Surgery Research, Daping Hospital, Third Military Medical University, Chongqing, China, 400042*

[2]*Bioengineering Center, Wayne State University, 818 W. Hancock, Detroit, MI 48201, USA*

[3]*College of Computer Science and Information Engineering, Chongqing Technology and Business University, Chongqing, China, 400067*

Abstract: An extensive series of biomechanics studies, including injury epidemiology, facet capsule ligament mechanics, injury mechanisms, was undertaken to help elucidate these whiplash injury mechanisms and gain a better understanding of cervical facet pain. These studies provide the following evidence: (1) Whiplash injuries are generally considered to be a soft tissue injury of the neck with symptoms such as neck pain and stiffness, shoulder weakness, dizziness, headache and memory loss. (2) Experimental findings have examined strains across the facet joint as a mechanism of whiplash injury, and suggested a capsular strain threshold or a vertebral distraction threshold for whiplash related injury, potentially producing neck pain. (3) Injuries to the facet capsule region of the neck are a major source of post-crash pain. (4) There are several hypotheses of how whiplash associated injury may occur and three of these are related strains within the facet capsule connected with events early in the impact. These results form the biomechanical basis for a hypothesis that the facet joint capsule is a source of neck pain and that the pain may arise from large strains in the joint capsule that cause pain receptors to fire.

Keywords: whiplash; pain; biomechanics; neck; facet joint; strain

1. Definitions of Whiplash Injury

1.1. Whiplash-Associated Disorder (WAD)

Harold Crowe first used the term whiplash in 1928. The term "whiplash-associated disorder" is used to describe the clinical manifestations of whiplash injury. The Québec Task Force on Whiplash-Associated Disorders1 describes these entities thus:

"*Whiplash is an acceleration-deceleration mechanism of energy transfer to the neck. It may result from rearend or side-impact motor vehicle collisions, but can also occur during diving or other mishaps. The impact may result in bony or soft-tissue injuries* (*whiplash injury*), *which may lead to a variety of clinical manifestations* (*Whiplash-Associated Disorders*)." (Spitzer et al., 1995)

1.2. Quebec Classification of WAD

Whiplash associated disorders (WAD) are generally considered to be a soft tissue injury of the neck with symptoms such as neck pain and stiffness, shoulder weakness, dizziness, headache and memory loss. The Québec Classification of Whiplash-Associated Disorders (Table 1) was devised by the Québec Task Force in 1995 to assist clinicians in making decisions about the treatment of whiplash injury and symptomatology. It was also proposed to allow research on WAD to be evaluated (Spitzer et al., 1995).

Table 1. The Québec classification of whiplash-associated disorders

Grade	Clinical presentation
0	No complaint about the neck, and no physical signs
I	Neck complaint of pain, stiffness or tenderness only, and No physical signs
II	Neck complaint, and musculoskeletal signs*
III	Neck complaint, and neurological signs**
IV	Neck complaint, and fracture dislocation

* Musculoskeletal signs include decreased range of motion and point tenderness; ** Neurological signs include decreased or absent deep tendon reflexes, weakness and sensory deficits

2. Clinical aspects of Whiplash Injury

In an extensive review of whiplash injury, Barnsley, Lord and Bogduk (1998) concluded[4][5] that the structures most likely to be injured in whiplash are the facet capsule, the intervertebral discs and the upper cervical ligaments.

Injuries to other structures may occur but the available evidence appears to suggest that these are less common. The most likely injuries to be associated with whiplash (Figure 1), were identified, and included the following:

--- *Facet capsule injury* - ligament tears, cartilage damage, contusion of the intraarticular meniscus hemarthrosis (joint haemorrhage) and possibly extending to microfractures;

--- *Disc injury* - AF ligament tears, cracks in the nu-

cleus pulposus and protrusions, and vertebral end plate avulsions;

--- *Major neck ligament injury* - tears to the ALL.

In general, whiplash injuries are considered to be a soft tissue injury of the neck. The clinical manifestations of whiplash injury include the collection of symptoms and signs that exist in a patient beyond a period in which recovery might normally be expected. These symptoms include headache, radicular deficit, cranial nerve/brainstem disturbance, cervical spine osteoarthritis, fatigue, anxiety, sleep disturbances, blurred vision, forgetfulness, illness/disability worry, and stress. The transition of a minority of cases of whiplash from an acute phase to a chronic phase is an important phenomenon that may depend on many factors, of which the initial injury is probably but one. However, the length of time since the crash that should be used to indicate chronic whiplash injury is inconsistently defined. The Quebec Task Force nominated 6 months post-crash as defining the transition from acute to chronic injury (Spitzer et al., 1995)[3] although one similar review used 8 weeks post crash.

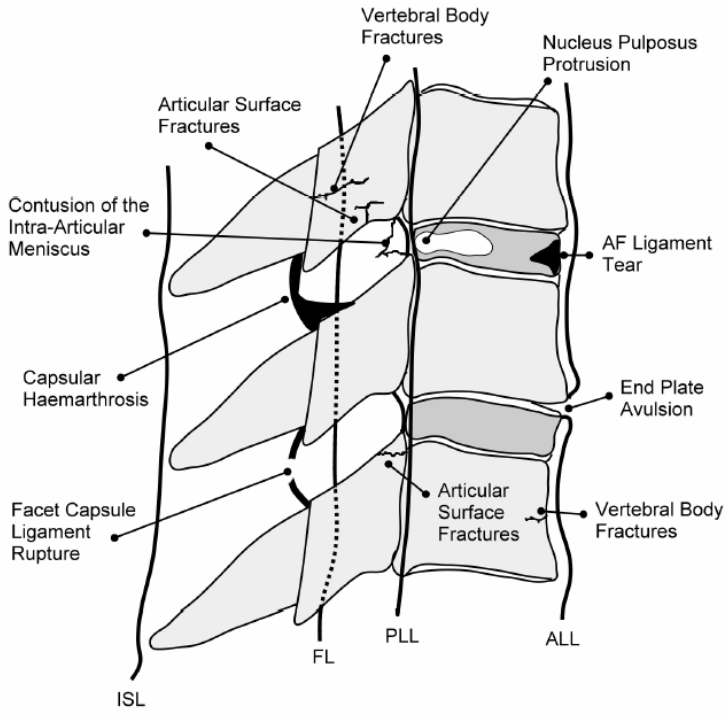

Figure 1. A lateral view of a section of the lower cervical spine showing possible whiplash associated injuries, adapted from Barnsley et al. (1995)

3. Cervical Facet Capsule Ligament Mechanics

Many studies have examined the cervical facet capsule specifically for its risk of mechanical injury. Kaneoka et al. (1999) demonstrated altered[6] facet joint motion during human volunteer studies of rear-impact collision with differential kinematics between upper and lower cervical spine regions. Panjabi et al. (1998) estimated linear capsular ligament strains using transducers inserted in the articular facets to quantify displacements across the C6/C7 joint[7]. For 6.5 g accelerations of cadaveric head-neck specimens, C6/C7 capsular strains reached a peak of 29.5 ± 25.7%. However, for these same specimens, the maximum C6/C7 capsule strain was 6.2 ± 5.6% for flexion-extension moments producing normal ranges of motion, suggesting capsular elongation in whiplash as a potential mechanism of injury. More recent work by that group (Pearson et al. 2004)[8] has further substantiated the C6/C7 joint as experiencing the greatest strains during simulated accelerations. For 8g accelerations, Pearson et al. (2004)[8] reported the maximum C6/C7 strain produced by facet joint sliding and separation was 39.9 ± 26.3%, consistent with earlier work of Panjabi et al. (1998)[7]. Yoganandan et al. (2002)[9] quantified relative facet motion (local sliding and compression) for human cadaveric head-neck whiplash simulations and demonstrated mean peak sliding motions in the anterior and posterior joint regions of 2.76 ± 0.78 mm and 1.94 ± 0.98 mm, respectively; mean peak compression motions in anterior and posterior regions of 2.02 ± 0.65 mm and 2.84 ± 0.47 mm, respectively. These studies provide evidence that whiplash kinematics alter strains across the bony surfaces of the facet joint and further hypothesize this as a mechanism contributing to painful capsule injury.

While experimental findings have examined strains across the facet joint as a mechanism of whiplash injury, more recent work has focused specifically on closer examination of the cervical facet capsule strain field (Lu et al, 2005)[10]. For vertebral bending motions matching human volunteer whiplash kinematics, full-field capsular strains have been quantified for cervical motion segments. For these joint kinematics, maximum principal strains were found to be directed across the joint (Winkelstein et al., 2000)[11], in a direction perpendicular to the joint articulation. While not sustaining any gross capsule injury during these vertebral kinematics, maximum principal strains reached as high as 23.0 ± 4.4%. These strains were not significantly different from those capsular strains (64.6 ± 73.8%) produced at the first ("subcatastrophic") failure during tensile testing of the isolated capsule. Despite the 2.5-fold difference in strains reported for those conditions, the lack of statistical difference due to high variation in subcatastrophic strains led the authors to suggest that whiplash-like bending of the facet joint can produce maximum capsular strains that are similar to those produced during pure tension. Likewise, Siegmund et al. (2001)[12] also documented the likelihood of subcatastrophic failures in combined shear loading during whiplash kinematics, with the capsule sustaining strains of 35.0 ± 21.0%. The broad collection of full spine and motion segment studies suggests a capsular strain threshold for whiplash related injury, potentially producing neck pain. While these studies provided mechanical bases for whiplash pain and a potentially painful facet capsule subcatastrophic injury, they did not provide physiologic context for those subcatastrophic injuries.

Considering all data from biomechanical testing using human volunteers, head-neck preparations and motion segments, it is possible that a critical distraction of the facet joint may be required for its painful capsular injury. It is hypothesized that such a distraction threshold may initiate nociception and/or pain symptoms. As such, this study examines a range of vertebral distractions, which is inclusive of those distractions producing subcatastrophic C6/C7 capsular strains, as noted in human cadaveric whiplash studies (Winkelstein et al, 2000; Siegmund et al, 2001)[11,12]. Using human capsule dimensions and displacement responses under tensile loading (Winkelstein et al, 2000)[11], geometric scaling between the human and rat species defined vertebral distraction ranges for the present study. Accordingly, vertebral distractions in the rat (0.9 mm = *SV*), scaled to be equivalent to joint distractions for human subcatastrophic failures, are examined for their potential to induce pain symptoms. Moreover, to evaluate whether joint distraction below these levels initiates any nociceptive or symptom outcomes, vertebral distractions sufficiently below (<10%) the *SV* magnitude are also examined (0.1 mm = *PV*). This study examines these two categories of vertebral distraction *in vivo*, in the context of pain behavioral outcomes and one indicator of nociception for insight into facet-mediated neck pain.

4. Injury Mechanisms

4.1. Hyperextension of the Neck

Formerly, hyperextension of the neck was thought to be a cause of injury. These early studies[1] included primate studies (MacNab, 1965), volunteer and cadaver studies (Mertz & Patrick, 1967) and field accident studies (States et al, 1972). However, it was inadequate to explain the continuous occurrence of whiplash injuries even after most vehicles had been equipped with head restraints as a result of motor vehicle safety regulation in the 1980s. In addition, the increasing levels of whiplash associated injury in the last decade combined with the results of the volunteer testing, which suggests possible injury in the early phase of motion, are indications that simple hyperextension of the neck is not the problem

4.2. Muscle Strains

The motion of the head leading to extension of the neck stretches the anterior muscles such as the sternocleidomastoid muscles. One hypothesis[13] is that these muscles are at risk of injury from attempting eccentric contraction (Phase: 100-160ms) of whiplash motion. Eccentric contraction occurs when a muscle contracts as it is stretched. Studies have shown that muscle failure occurs at forces much larger than maximal isometric force and stretch is necessary to create injury (Garrett et al, 1997)[13]. The contraction is due to the stimulation of muscle spindles in the flexor muscles that are being stretched as the neck and head move into extension (Phase: 40-100ms). At this stage, the large extensor muscles in the back of the neck are moving into compression and are hence unlikely to contract at the time of impact.

A second hypothesis is that the extensor muscles are injured during rebound of the head and neck as they undergo eccentric contraction during the rebound phase of the impact (Phase: 150-200ms). Hell et al (2002)[14] regarded the rebound into the belt system as a possible additional injury source, because the measured head velocities in this phase have been shown to reach higher values than previously expected. This mechanism is consistent with the findings of Garrett et al (1997)[13] but fails to explain the significant number of belted occupants in severe frontal impacts who do not have neck pain following a crash. Further, the muscle strain mechanism may explain short-term muscle stiffness following the impact, but such injuries typically last only a few days.

These two hypotheses indicated that muscles were also focused on as a candidate of soft tissue injuries in rear impacts. However, the assumption was not consistent with the fact that most patients had pain in the posterior region of the neck, whereas the anterior muscles would

be stretched first in rear impacts.

4.3. Spinal Column Pressure Pulses

Svensson et al (1993)[15] conducted an animal study to investigate whether whiplash injury was produced by pressure pulses generated in the spinal column. The necks of pigs were exposed to rapid flexion-extension motion in simulated rear impacts. Pressure pulses of up to 150 mmHg were found in the lower cervical spinal canal during neck motion and were greater in magnitude across the vertebral foramen than along the canal. Microscopic analysis of the nerve cells in the spinal dorsal root ganglia (DRG) revealed a leakage of dye from the CFS across the cell membranes, indicating membrane damage.

Eichberger et al (2000)[16] conducted a total of 21 tests including pressure measurements with 5 cadavers. Sled experiments were performed using a test set-up similar to real rear-end collisions. Impact velocities of approximately 9 km/h and 15 km/h were chosen. The subjects were fitted with 2 triaxial accelerometers on the head and chest, one biaxial accelerometer at the height of T1, and one angular accelerometer at the head. Pressure measurements in the cerebrospinal fluid (CSF) were performed using 2 catheter-tip pressure transducers, placed subdurally in the spinal canal. The upper transducer was placed at the C1/C2 level and the lower transducer at C6/C7. The researchers found pressure peaks reaching 220 mmHg at approximately 100 ms in the cadavers tested. This confirmed the pressure pulse amplitudes and times obtained in the animal experiments by Svensson et al (1993)[15] were also possible in humans. Injuries to the nerve tissue in the neck resulting from these pressure effects could not be observed due to limitations with the use of cadavers.

There is a need to note that their theory does not explain the fact that many patients indicate the location of pain at the inferior region of the neck, while the pressure gradient can be raised anywhere in the spinal column.

4.4. Facet Impingement

Based on the neck radiographs from the volunteer tests, the researchers[6, 17] (Kaneoka et al, 1999; Ono et al, 1997) found that the lower motion segments had the larger the relative rotation angle. The rotation between the fifth and sixth vertebral segments is the largest and earliest (Figure 2).

To quantify this motion, the position of the instantaneous axis of rotation (IAR) was analyzed for the C5/C6 motion segment (Ono et al, 1997)[17]. Volunteer neck measurements provided the expected positions of the IAR within the C6 vertebral body, in normal cervical extension (Figure 2).

When the S-shape of the neck occurs in the whiplash motion, the IAR moves upward to a position within the C5 vertebral body (Figure 2). This upward motion of the IAR indicates that the C5 motion at this point is largely one of rotation rather than shear.

This upward shift of the IAR during the crash motion was only observed in the C5/C6 motion segment. It was hypothesised that, as a result of the motion, the articular facet surfaces would collide, resulting in mechanical impingement on the synovial fold or meniscoid in the facet capsule. Further, it was hypothesized that if this torque is large enough, there was the possibility of the anterior longitudinal ligament or separating of the annulus fibrosus from the end plate of the associated vertebrae (a rim lesion).

Subsequent testing of cadaver head and necks by both Yoganandan et al [9] and Pearson et al (2004)[8] has supported the impingement motion of the facet capsule.

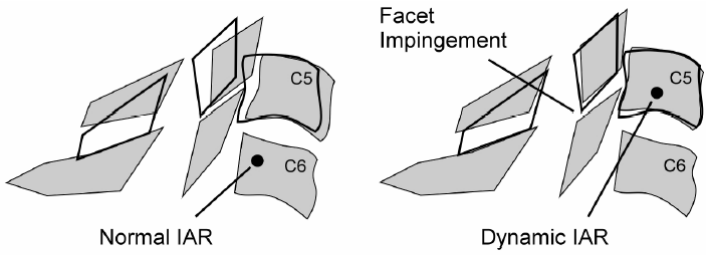

Figure 2. With normal cervical extension motion the IAR is positioned in the C6 vertebral body. When the S-shape is reached in the whiplash motion, the IAR moves upward to a position within the C5 vertebral body, from Ono et al. (1997)

4.5. Shear

A rear impact causes the seatback to push the torso forward, while the head remains stationary. The effect of the seatback pushing on the cervical spine is to straighten the thoracic spine. The inertia of the head converts this vertical motion of the spine into a compression loading to the cervical spine. This compression has been observed in volunteer and cadaveric tests simulating whiplash. As the torso pulls the head forward, a shear force is generated at each level of the cervical spine. Yang and Begeman (1996)[18] suggested that this shear force was a candidate to cause soft tissue injury to the intervertebral joints of the cervical spine. Under compression, the cervical vertebrae slide relative to each other and the facet capsules are stretched and possibly torn, resulting in in-

flammation and pain.

Deng et al (2000)[19] carried out 26 low-speed rear-end impacts on six human cadavers in a rigid seat. The study showed that the upper cervical vertebrae go into relative flexion with respect to the lower cervical vertebrae during whiplash motion, while the entire neck is in extension (the S-shape). In addition, the upper neck is under flexion when the head contacts the head-rest, while the facets reach peak strain prior to head contact with the head-rest. It was concluded that if stretching of the facet capsular ligaments were the reason for the high incidence of neck pain, the upper cervical spine would sustain a flexion injury while injury to the lower cervical spine would be due to a combination of shear and compression.

Deng et al (2000)[19] also reported that a 20-degree seatback as compared to a 0-degree seatback resulted in less cervical lordotic curvature, more upward ramping motion of the thoracic spine, and greater relative rotation of each cervical motion segment.

4.6. Axial Compression

Yang et al (1996)[18] proposed a hypothesis to explain the rear-end neck injury mechanism stating that axial compression can cause loosening of ligaments and make it easier for the facet joint capsule and other soft tissues to be injured.

In Yang's studies, cervical spine specimens from C1-T1 were tested. The C1 vertebra was fixed to an aluminum plate with screws. The other end (T1) was potted in epoxy and attached to a six-axis load cell. The entire assembly was placed in a jig on an INSTRON testing machine. This jig limits the C1 vertebra from moving to simulate the inertial effect of the head. The T1 vertebra was attached to the actuator of the INSTRON testing machine. During the test, the actuator moves upward to simulate the seat back pushing from behind. Five tests were done for each specimen. In the first test, the T1 was moved anteriorly to simulate a rearend impact for 20mm displacement at a quasi-static speed of 0.04 m/s. In the next four tests, an axial compression of 10, 20, 30 and 40 lbs of dead weight were applied through a cable-pulley system. The same procedure as in the first test was then repeated.

Yang's data indicated that at C5-C6 level a shear of 22.5 N without any pre-compression produced a 2.5-mm deflection and a shear of approximately 10 N with the axial pre-compression of 40 lbs produced a 3.5-mm deflection. Further analysis showed that shear stiffness values were reduced significantly with increased axial compressions and validated Yang's hypothesis.

5. Discussions

Whiplash injury is not necessarily accompanied by obvious tissue damage detectable by X-ray or MRI. Many different injury mechanisms of the cervical spine have been identified thus far, but the extent to which a single mechanism of injury is responsible remains uncertain. The future research should provide more clinical evidence for injury mechanisms of whiplash injury.

Acknowledgements

This work was supported by grants from the National Natural Science Foundation of China (No. 30928005), the Chongqing Natural Science Foundation (No. CSTC2009BB5013), and the Third Military Medical University Research Foundation (No. 2009XHG16).

References

[1] Chen H B, Yang K H, Wang Z G. Biomechanics of whiplash injury. Chin J Traumatol, 2009; 12(5): 305-314.

[2] Yang J K, Xiao Z, Wan X M. A study on neck injury in vehicle rear impacts using human neck FE model. Automotive Engineering; 2007, 29(6): 1-6.

[3] Spitzer W O, Skovron M L, Salmi L R, et al. Scientific monograph of the Quebec Task-Force on Whiplash-Associated Disorders - Redefining whiplash and its management. Spine, 1995; 20(8): S1-S73.

[4] Barnsley L, Lord S, Bogduk N. Whiplash injury. Pain 1994; 58: 283-307.

[5] Barnsley L, Lord S and Bogduk N. The pathophysiology of whiplash. in GM Malanga (ed.), SPINE State of the Art Reviews – Cervical Flexion-Extension/Whiplash Injuries, Hanley and Belfus Inc, Philadelphia, United States, 1998; 12(2): 42-77.

[6] Kaneoka K, Ono K, Inami S, et al. Motion analysis of cervical vertebrae during whiplash loading. Spine, 1999; 24(8): 763-770.

[7] Panjabi M M, Cholewicki J, Nibu K, et al. Capsular ligament stretches during in vitro whiplash simulations. Journal of Spinal Disorders, 1998; 11: 227-232.

[8] Pearson A M, Ivancic P C, Ito S, et al. Facet joint kinematics and injury mechanisms during simulated whiplash. Spine, 2004; 29(4): 390-397.

[9] Yoganandan N, Pintar F A, Cusick J F. Biomechanical analyses of whiplash injuries using an experimental mode. Accident Analysis and Prevention, 2002; 34: 663-671.

[10] Lu Y, Chen C, Kallakuri S, et al. Neural response of cervical facet joint capsule to stretch: a study of whiplash pain mechanism. Stapp Car Crash Journal, 2005; 49: 49-65.

[11] Winkelstein B A, Nightingale R W, Richardson W J, et al. The cervical facet capsule and its role in whiplash injury. Spine, 2000; 25(10): 1238-1246.

[12] Siegmund G P, Myers B S, Davis M B, et al. Mechanical evidence of cervical facet capsule injury during whiplash. Spine, 2001; 26(19): 2095-2101.

[13] Garrett W E, Seaber A V, Nest T M, et al. Muscle strain injury: basic science and clinical application. Kappa Delta Papers in the Transactions of the 43rd Annual Meeting of Orthopaedic Research Society, San Francisco. Orthopaedic Research Society, Palant, United States, 1997.

[14] Hell W, Schick S, Langwieder K, et al. Biomechanics of cervical spine injuries in rear end car impacts: influence of car seats and possible evaluation criteria. Journal of Traffic Injury Prevention, 2002; 3(2): 127-140.

[15] Svensson M Y, Aldman B, Hansson H A, et al. Pressure effects in the spinal canal during whiplash extension motion: a possible cause of injury to the cervical spinal ganglia. Proceedings of the International IRCOBI Conference on the Biomechanics of Impact, Eindhoven, Netherlands, International Research Council on the Biomechanics of Impact, Bron, France, 1993: 189-200.

[16] Eichberger A, Darok M, Steffan H, et al. Pressure measurements in the spinal canal of post-mortem human subjects during rear-end impact and correlation of results to the neck injury cri-

terion. Accident Analysis and Prevention, 2000; 32(2): 251-260.
[17] Ono K, Kaneoka K, Wittek A, et al. Cervical injury mechanism based on the analysis of human cervical vertebral motion and head-neck-torso kinematics during low speed rear impacts. 41st Stapp Car Crash Conference, Orlando, United States. SAE Paper 973340, Society of Automotive Engineers, Warrendale, United States, 1997: 339-56.
[18] Yang K H, Begeman P C. A proposed role for facet joints in neck pain in low to moderate speed rear end impacts, Part I: Biomechanics. 6th Injury Prevention through Biomechanics Symposium, 1996: 59-63.
[19] Deng B, Begeman P C, Yang K H, et al. Kinematics of human cadaver cervical spine during low speed rear-end impacts. Stapp Car Crash Journal, 2000; 44: 171-188.