

Review

Biomechanics of whiplash injury

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Despite a large number of rear-end collisions on the road and a high frequency of whiplash injuries reported, the mechanism of whiplash injuries is not completely understood. One of the reasons is that the injury is not necessarily accompanied by obvious tissue damage detectable by X-ray or MRI. An extensive series of biomechanics studies, including injury epidemiology, neck kinematics, facet capsule ligament mechanics, injury mechanisms and injury criteria, were undertaken to help elucidate these whiplash injury mechanisms and gain a better understanding of cervical facet pain. These studies provide the following evidences to help explain the mechanisms of the whiplash injury: (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, etc. (2) Based on kinematical studies on the cadaver and volunteers, there are three distinct periods that have the potential to cause injury to the neck. In the first stage, flexural deformation of the neck is observed along with a loss of cervical lordosis; in the second stage, the cervical spine assumes an S-shaped curve as the lower vertebrae begin to extend and gradually cause the upper vertebrae to extend; during the final stage, the entire neck is

extended due to the extension moments at both ends. (3) The *in vivo* environment afforded by rodent models of injury offers particular utility for linking mechanics, nociception and behavioral outcomes. 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. (4) Injuries to the facet capsule region of the neck are a major source of post-crash pain. There are several hypotheses on how whiplash-associated injury may occur and three of these injuries are related to strains within the facet capsule connected with events early in the impact. (5) There are several possible injury criteria to correlate with the duration of symptoms during reconstructions of actual crashes. 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 will cause pain receptors to fire.

Key words: Whiplash injuries; Pain; Biomechanics; Neck; Zygapophysel joint

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Definition

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 Disorders once described these entities as follows: Whiplash is an

acceleration-deceleration mechanism of energy transfer to the neck. It may result from rear end 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.¹

Québec classification of WAD WAD is 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 WAD (Table 1) was devised by Québec Task Force in 1995 to assist clinicians in making decisions about the treatment and symptomatology of whiplash injury. It was also pro-

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posed to allow research on WAD to be evaluated.¹

Epidemiology and clinical aspects of whiplash injury

Epidemiology of whiplash injury Motor vehicle accidents with a whiplash injury mechanism are one of the most common causes of neck injuries, with an incidence of perhaps 1 million per year in the US. It was estimated that the annual incidence was 3.8 per thousand populations in the US. In an extensive study, there was a steady increase in the number of cervical soft tissue injuries in patients in hospitals or emergency rooms after automobile accidents in the UK. In the 12 months before the introduction of compulsory seatbelts in the UK, the rate was 7.7% in 929 patients, which climbed to a rate of 42.5% in 2 661 patients by 1990. The rate continued to increase even after the introduction of mandatory seatbelts. Among insurance claims in the US, neck sprains are the most common injuries, making up 40% of claimants. The Québec Task Force reported that 61% of injury claims reimbursed by the Insurance Corporation of British Columbia in 1995 were from WAD at a cost of \$590 million.

Table 1. The Québec classification of WAD

| 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

The vast majority of reported WAD are AIS I (600 000 of 700 000 in 1992-1993), with an estimated annual cost of \$3.9 billion in the US. WAD, in addition, is not necessarily a self-limiting condition. A proportion of victims will be left with a significant disability that may interfere with jobs, everyday activities, and leisure-time pursuits. Most studies on the natural history of WAD have suggested a proportion between 6% and 18% with long-term disability. Thus, the societal cost of WAD is enormous.

Subjects typically complain of neck stiffness and pain in the neck muscles immediately after the impact.

A minority of patients have radiating pain. Symptoms can become worse as time goes on, and there is often an interval of little or no pain before the symptoms become worse.

Patients who note whiplash pain typically experience a low to moderate speed rear end vehicular impact. Rear impacts cause pain in the neck almost twice as frequently as frontal collisions. Of the drivers with WAD, 51.9% are injured in rear impacts, 27.2% in frontal and 16.4% in side impacts. Of passengers with WAD, 54.3% are injured in rear impacts, 21.3% in frontal and 12.2% in side impacts. The risk of initial AIS I neck injury for UK occupants is found to be 38% in rear impacts, while only 15% in frontal and 15% in side impacts; for Swedish occupants, 34% in rear, 16% in frontal and 11% in side impacts from Volvo's accident database. For AIS I neck injuries leading to disability, Krafft et al found that in Sweden 64% occurred in rear impact and 23% in frontal impact.²

There was a 34.7%-65.3% or 29%-71% ratio of male to female in whiplash patients. The majority of patients were 21-60 years old and had neck ache, headache and often limited neck motion. Two-thirds of subjects were pain-free within three months but others continued to have pain, including a group considered to have a late whiplash syndrome in which symptoms lasted more than six months.

Krafft et al² pointed out in a study of crash-recorder equipped cars, that 15 occupants sustained no injury when the peak acceleration was 6 g or less, 20 sustained short-term disability when the acceleration was 10 g or less and three occupants sustained long-term neck-related disability at peak accelerations of 13 g and 15 g. They also pointed out that cars manufactured in the 1980s resulted in a 0.15 ratio of long-term *versus* short-term disability, while those made in the 1990s had a ratio of 0.40. This better long-term outcome in older vehicles was hypothesized to be attributed to the less stiff seats of the earlier vehicles.³

Clinical aspects of whiplash injury According to an extensive review of whiplash injury, the structures most likely to be injured in whiplash were 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 are identified (Fig.1), and included the following aspects.(1) Facet capsule injury: ligament tears, cartilage damage, contusion of the intraarticular meniscus hemarthrosis (joint haemorrhage) and possibly extending to microfractures.(2) Disc injury: anulus fibrosus (AF) ligament tears, cracks in the nucleus pulposus and protrusions, and vertebral end plate avulsions.(3) Major neck ligament injury: tears to the anterior longitudinal ligament (ALL).

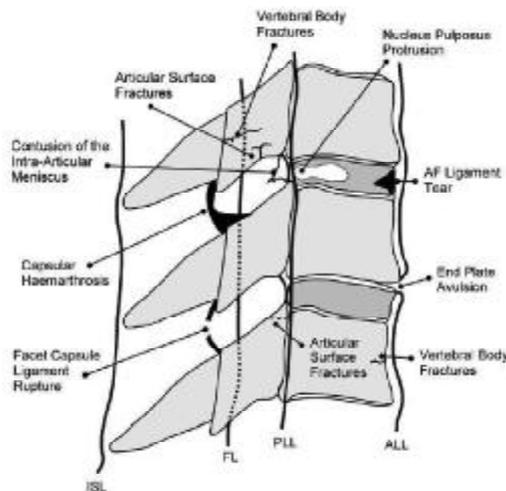


Fig. 1. A lateral view of a section of the lower cervical spine showing possible whiplash associated injuries.

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 be normally expected. These symptoms include headache, radicular deficit, cranial nerve/brain stem 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 Québec Task Force nominated 6 months post-crash as defining the transition from acute to chronic injury although one similar review used 8 weeks post-crash.

Neck kinematics

From a kinematical point of view, the head-neck complex has been shown in many studies to primarily sustain the inertially applied extension-flexion loading in a

sagittal mode.

Cadaveric kinematics Luan et al,⁴ based on their cadaveric kinematics study, illustrated that (Fig.2):

(1) In the first stage (0-100 ms after the onset of impact), flexural deformation of the neck is observed along with a loss of cervical lordosis. The initial lordotic neck at 20 ms becomes straight. After 50 ms, both upper and lower cervical spines are subjected to a flexion moment. The shear force is transmitted initially through the lower levels and eventually through the upper levels, but does not reach the superior end of the cervical spine. The axial force then changes from compressive to tensile at about the 60 ms mark.

(2) In the second stage (100-130 ms), the cervical spine assumes an S-shaped curve as the lower vertebrae begin to extend and gradually cause the upper vertebrae to extend. Eventually, the straightened neck once again becomes lordotic. An extension moment acts at the lower vertebrae, while a flexion moment acts at the upper levels. Shear forces are acting at all levels along with a tensile axial force.

(3) During the final stage (after 130 ms), the entire neck is in extension due to extension moments at both ends. Shear forces and tensile axial forces continue to act at all levels. The shear forces throughout the loading phase may subject the lower FJCs to excessive stretch while initial cervical spine compression may cause facet joint capsules (FJCs) to locally compress and slide along the joint. The posterior-most regions of the joint compress more than the anterior-most regions, exhibiting a "pinching" mechanism. Excessive joint compression/sliding may also induce pain if these joints contain pain-sensitive structures.

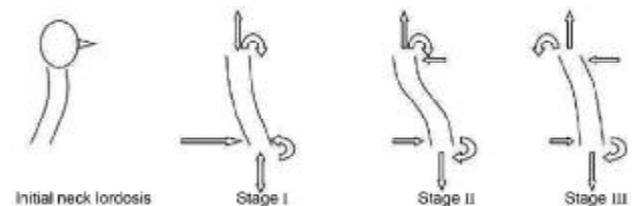


Fig. 2. Models of neck deformation, and force and moment diagrams at three stages of a rear-end impact.

Volunteer kinematics Kaneoka et al⁵ tested 10 volunteer subjects seated on a sled to simulate car rear impact acceleration (Fig.3). An impact speed of 8 km/h was used to study the head-neck-torso kinematics and cervical spine responses. The acceleration pulse generated by the sled in the 8 km/h impact speed is

shown in Fig.4. A headrest was not used in the experiment. The activity of the sternocleidomastoid muscle and the paravertebral muscles were measured by surface electromyography (EMG). The neck axial and shear forces, and the flexion/extension bending moments at the occipital condyle, were calculated by treating the head as a free body. The results for one of the volunteers are plotted in Fig. 5.

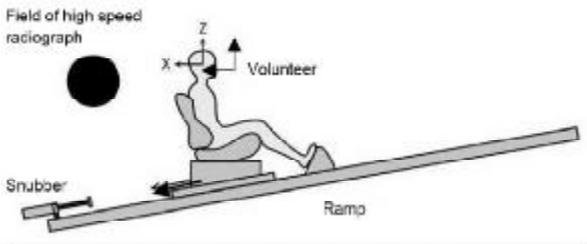


Fig. 3. Volunteer seated on a sled inclined at 10°, simulating a car rear impact at 8 km/h.

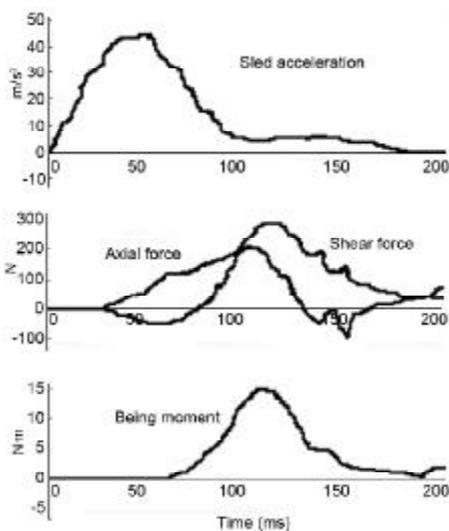


Fig. 4. The acceleration pulse generated by the sled for the 8 km/h impact speed is shown along with the neck axial and shear forces and flexion/extension bending moment at the occipital condyle for one volunteer.

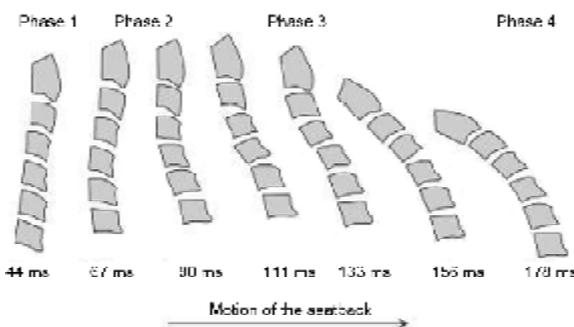


Fig. 5. The alignment of the C₂ to C₇ vertebrae of a volunteer during a rear impact obtained by high-speed radiography for the 4 phases described by Kaneoka and Ono. The alignment at 111ms also includes the facet capsule and spinous processes to illustrate the possibility of impingement of the facet surfaces.

This study is of particular importance because the cervical motion is recorded by cineradiography (90 frames per second X-ray) and analyzed to quantify the rotation and translation of individual cervical vertebrae resulting from the impact. This method allows the motion patterns of the cervical vertebrae in the crash motion and in normal motion to be compared.

Kaneoka and Ono divided the motion and head-neck-torso responses of the test subjects into the following four phases (Fig. 5).

(1) Phase 1: Sled motion (0-40 ms). a. The seat begins to press the back of the volunteer; b. The spine begins to straighten; c. Cervical motion has not occurred; d. No muscular response in the neck.

(2) Phase 2: Neck axial force (40-100 ms). a. The torso moves forward—pushed by the seat back; b. The torso moves upward—parallel to the seat inclination, causing axial compression of the cervical spine due to the inertia of the head, which reaches a maximum; c. The head remains stationary due to inertia, with a slight initial flexion; d. C₆ rotates earlier into extension than the upper vertebral segments (C₃, C₄ and C₅); e. The vertebrae of the neck assumes an “S” shape with the upper region in flexion and the lower region in extension; f. No muscular response in the neck.

(3) Phase 3: Axial and shear force (100-160 ms). a. The sled slows the torso rebounds and moves forward with some backward rotation; b. The axial force on the neck decreases while the shear force on the neck reaches a peak at about 120 ms; c. The head begins to rotate into extension; d. The cervical spine moves into alignment in extension; e. The EMG of the sternocleidomastoid discharges from about 115 ms.

(4) Phase 4: Full extension (150-220 ms). a. The torso moves forward and downward; b. The head and neck rotation reaches full extension; c. Shear and axial forces in the neck decrease; d. The muscular discharge finishes by around 220 ms.

The exact timing of the events in a volunteer test is quite variable and depends on the shape and magnitude of the acceleration pulse, the stiffness of the seat back, the angle of the seat back, the posture and anthropometry of the subject, and whether a head restraint is present. The S-shaped response in Phase 2 of the neck in a rear impact has been verified by other studies using cadaver's head and neck, the whole cadavers and volunteers.

If the seat used in the test is fitted with a head restraint, during Phase 3 the head will make contact and start to receive additional support. The maximum retraction of the head is most likely to occur before contacting with the head restraint. The effectiveness of this extra head support depends on the geometry and stiffness of the head restraint and its mounting on the seat back. A head restraint located at an appropriate proximity to the head, in terms of offset and height, and with ample crash stiffness, has the potential to reduce the neck loads in Phases 3 and 4.

In Phase 4, the motion halts when a restrained subject moves forward into the shoulder portion of the seatbelt. Seatbelts also reduce the upward motion of the torso in Phase 2. Phase 4 may possibly accounts for the increase in whiplash injury noted with the use of seatbelt in field accident studies. Based on these phases of motion, there are three distinct periods that have the potential to cause injury to the neck: (1) Early in the impact event during the head retraction period and leading to the "S" shape of the neck (Phase 2); (2) Due to the impact with the head restraint, if it is poorly positioned with respect to the head and neck at the time of contact (Phase 3); (3) Due to hyperextension for a severe impact with a poorly fitted head restraint or without one (Phase 4); and (4) During the rebound into the seatbelt (Phase 4).

Cervical facet capsule ligament mechanics

Many studies have examined the cervical facet capsule specifically for its risk of mechanical injury. Kaneoka et al⁶ demonstrated the altered facet joint motion during the human volunteer studies of rear-impact collision with differential kinematics between upper and lower cervical spine regions. Panjabi et al⁷ estimated the linear capsular ligament strains using transducers inserted in the articular facets to quantify displacements across the C₆/C₇ joint. 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 group⁸ has further substantiated the C₆/C₇ joint as experiencing the greatest strains during simulated accelerations. For 8 g accelerations, Pearson et al⁸ 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.⁷ Yoganandan et al⁹ 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 mm±0.78 mm and 1.94 mm±0.98 mm, respectively; mean peak compression motions in anterior and posterior regions of 2.02 mm±0.65 mm and 2.84 mm±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.¹⁰ For vertebral bending motions matching human volunteer whiplash kinematics, full-field capsular strains have been quantified for cervical motion segments. For the joint kinematics, maximum principal strains are found to be directed across the joint, in a direction perpendicular to the joint articulation.¹¹ While not sustaining any gross capsule injury during this vertebral kinematics, maximum principal strains reach as high as 23.0%±4.4%. These strains are not significantly different from those capsular strains (64.6%±73.8%) produced at the first ("subcatastrophic") failure during the 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 lead 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 al¹² 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 the full spine and the motion segment studies suggests a capsular strain threshold for whiplash-related injury, potentially producing neck pain. While these studies provide mechanical bases for whiplash pain and a potentially painful facet capsule subcatastrophic injury, they do not provide physiologic context for those subcatastrophic injuries.

Considering all data from biomechanical tests us-

ing 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 are inclusive of those distractions producing subcatastrophic C_6/C_7 capsular strains, as it noted in human cadaveric whiplash studies. Using human capsule dimensions and displacement responses under tensile loading, geometric scaling between human and rat species defined vertebral distraction ranges for the present study. Accordingly, vertebral distractions in the rat ($0.9\text{ mm}=\text{SV}$), scaled to be equivalent to the 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 symptomatic outcomes, vertebral distractions sufficiently below ($<10\%$) the SV magnitude are also examined ($0.1\text{ mm}=\text{PV}$). 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.

Injury mechanisms

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.⁷

Hyperextension of the neck Formerly, hyperextension of the neck was thought to be a cause of injury. These early studies included primate studies, volunteer and cadaver studies and field accident studies. 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 tests, which suggests possible injury in the early phase of motion, are indications that simple hyperextension of the neck is not the problem.

Muscle strains The motion of the head leading to extension of the neck stretches the anterior muscles such as the sternocleidomastoid muscles. One hypothesis is that these muscles are at risk of injury from

attempting eccentric contraction during Phase 3 (Fig. 5) 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. 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 2 (Fig. 5). 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 in Phase 4 (Fig. 5). Hell et al¹³ 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 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 indicate that muscles are also focused on as a candidate of soft tissue injuries in rear impacts. However, the assumption is not consistent with the fact that most patients have pain in the posterior region of the neck, whereas the anterior muscles would be stretched first in rear impacts.

Spinal column pressure pulses An animal study to investigate whether whiplash injury was produced by pressure pulses generated in the spinal column was conducted. The necks of pigs were exposed to rapid flexion-extension motion in simulated rear impacts. Pressure pulses of up to 150 mm Hg 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 al¹⁴ 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 mm Hg at approximately 100 ms in the cadaver tests. This confirms that the pressure pulse amplitudes and times obtained in the animal experiments by Svensson et al are 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.

Facet impingement Based on the neck radiographs from the volunteer tests, the researchers found that the lower motion segments had the larger relative rotation angle.⁶ The rotation between the fifth and sixth vertebral segments is the largest and earliest (Fig. 6).

To quantify this motion, the position of the instantaneous axis of rotation (IAR) was analyzed for the C₅/C₆ motion segment. Volunteer neck measurements provided the expected positions of the IAR within the C₆ vertebral body, in normal cervical extension (Fig. 6).

When the S-shape of the neck occurs in the whiplash motion, the IAR moves upward to a position within the C₅ vertebral body (Fig. 6). 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 is only observed in the C₅/C₆ motion segment. It is hypothesized that, as a result of the motion, the articular facet surfaces would collide, resulting in mechani-

cal impingement on the synovial fold or meniscoid in the facet capsule. Further, it is hypothesized that if this torque is large enough, there is the possibility of tearing the anterior longitudinal ligament or separating the annulus fibrosus from the end plate of the associated vertebrae (a rim lesion).

Subsequent tests of cadaver head and necks by both Yoganandan et al and Pearson et al have supported the impingement motion of the facet capsule.

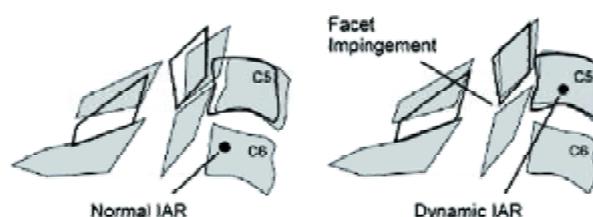


Fig. 6. With normal cervical extension motion, the IAR is positioned in the C₆ vertebral body. When the S-shape is reached in the whiplash motion, the IAR moves upward to a position within the C₅ vertebral body.

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 cadaver tests simulating whiplash. As the torso pulls the head forward, a shear force is generated at each level of the cervical spine. This shear force is 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 inflammation and pain.

Deng et al¹⁵ carried out 26 low-speed rear-end impacts on 6 human cadavers in a rigid seat. The study shows 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 (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 is concluded that if stretching of the facet capsular ligaments is 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 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.

Axial compression A hypothesis to explain the rear-end neck injury mechanism states that axial compression can cause loosening of ligaments and make it easier for the facet joint capsule and other soft tissues to be injured.

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 moved 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 rear-end impact for 20 mm displacement at a quasi-static speed of 0.04 m/s. In the next four tests, an axial compression of 44.45 N, 88.90 N, 133.35 N and 177.8 N of dead weight were applied through a cable-pulley system. The same procedure as in the first test was then repeated. The data indicate that at C₅-C₆ level a shear of 22.5 N without any pre-compression produces a 2.5 mm deflection and a shear of approximately 10 N with the axial pre-compression of 177.8 N produced a 3.5 mm deflection. Further analysis shows that shear stiffness values are reduced significantly with increased axial compressions.

Injury criteria

There are several possible injury criteria to correlate with the duration of the symptoms in reconstructions of actual crashes.

Neck injury criterion (NIC) A mathematical model of the transient pressure pulses, measured by Svensson et al in the spinal canal of pigs, was developed to establish the NIC criterion. These pulses were due to volume changes resulting from forcing the head

and torso to translate horizontally relative to each other. Bostrom et al hypothesized that a neck injury would occur during the initial head/thorax motion when the spine takes the 'S' shape as the thorax is pushed forward. Anatomically, this is a retraction motion of the neck and it occurs in the first 100 ms of the rear impact before the head begins to rotate. Injury is thought likely to occur if:

$$NIC = a_{rel} \times 0.2 + v_{rel}^2 < 15 \text{ m}^2/\text{s}^2$$

where a_{rel} and v_{rel} are the relative acceleration and velocity between the head (C₁) and the upper torso (T₁). The criterion for the threshold of human tolerance of 15 m²/s² is estimated to be appropriate.

NIC has been validated against volunteer tests, cadaver tests and dummy tests. The test confirmed aspects of the use of NIC. For the volunteers, the peak NIC correlated well with the maximum retraction of the head and no complaints of pain were made below a NIC of 8, while some complaints of pain were made at NIC values of about 10. For the cadavers, a ligament rupture occurred at an NIC of 18.6 and NIC also correlated with the magnitude of the peak pressure readings in the spinal canal.

A group of 79 rear-impact crashes with known injury outcomes and a crash-pulse recorder fitted to the vehicle were used to validate the maximum NIC (or NIC_{max}) as a criterion for injury. The crashes were all reconstructed in a mathematical (MADYMO) model of the BioRID II dummy and seat. The model was validated with sled testing. The study found that an NIC_{max} threshold of 15.3 m²/s², where the proportion of occupants with lasting symptoms is 12/13 (sensitivity=0.92), showed relatively high positive predictive values (33%±15%) and very high negative predictive values (99%±2%) for neck injury with long lasting symptoms (greater than 1 month).

NIC_{max} has been shown to be sensitive to the major risk factors of a rear impact such as crash pulse, seat deflection characteristics and head-to-head restraint distance.

N_{ij} criterion The neck injury criterion, N_{ij}, has been proposed to assess AIS 2+ neck injuries (not normally classified as "whiplash injuries") in frontal impacts including those with airbag deployment. This criterion

could potentially be of interest if a high speed rear impact test is to be included. AIS 2+ neck injuries are however rare in rear impacts. N_{ij} is based on dimensional analysis of the load to the neck. It combines the effects of force and moment measured at the occipital condyles and is based on both the tolerance levels for axial compression and bending moment. The N_{ij} criterion is calculated by:

$$N_{ij} = \frac{F_z}{F_{int}} + \frac{M_y}{M_{int}}$$

where F_z represents the axial force and M_y represents the flexion/extension bending moment. The index "int" gives a critical intercept value for the load and the moment, respectively. The intercept values for the 50th percentile Hybrid III male are proposed to be F_{int} (tension) = F_{int} (compression) = 4500 N, M_{int} (flexion) = 310 Nm and F_{int} (extension) = 125 N·m. The threshold for injury levels based on N_{ij} is 1. Since the intercept values for the forces are based on the corresponding values for the Hybrid III and do not represent human physiological values, they must be redefined if a dummy other than the Hybrid III is used. The N_{ij} may be of interest in high severity seat back integrity tests. There is however currently no validated dummy available and the frequency of AIS 2+ injuries in rear impacts is relatively small (>10% of the neck injuries in rear impacts according to GIDAS and CCIS databases).

N_{km} criterion The N_{km} criterion was proposed to assess neck injuries in rear impacts. It is a combination of moments and shear forces. The N_{km} criterion is calculated as

$$N_{km} = \frac{F_x}{F_{int}} + \frac{M_y}{M_{int}}$$

where F_x represents the shear force and M_y the flexion/extension bending moment. The index "int" gives a critical intercept value for the load and the moment. The intercept values for the 50th percentile Hybrid III male where F_{int} (anterior) = F_{int} (posterior) = 845 N, M_{int} (flexion) = 88.1 N·m, and M_{int} (extension) = 47.5 N·m. The threshold for injury levels based on N_{km} is 1. Schmitt et al have shown that N_{km} varies depending on the dummy used in the test.

The lower neck load-index (LNL) The lower neck moment is sensitive to seat design parameters.¹⁶ Lower neck loads are also consistent with the facet-

based injury mechanism supported by the works of Yoganandan et al,¹⁷ presented LNL. It incorporates a combination of neck loads at T_1 level. Indications of LNL correlation to injury risk are reported but the need for a more extensive evaluation of the LNL is also emphasized.

The IV-NIC criterion developed by Panjabi et al is based on the hypothesis that a neck injury occurs when an intervertebral extension-flexion angle exceeds its physiological limits. It is defined as the ratio of the intervertebral motion Θ_{trauma} under traumatic loading and the physiological range of motion $\Theta_{physiological}$. The IV-NIC is calculated by:

$$IV-NIC_i = \frac{\Theta_{trauma, i}}{\Theta_{physiological, i}}$$

This criterion still lacks a threshold. Using the IV-NIC requires a dummy neck capable of simulating intervertebral motion. At present, only the neck of the BioRID has this capacity in the sagittal plane. The biofidelity of the angular motion of the individual BioRID spinal units has however not been evaluated.

NDC criterion The NDC, proposed by Viano and Davidsson, is based on the angular and linear displacement response of the head relative to T_1 from volunteer tests.¹⁸ The criteria are given as corridors of the z versus angular and x versus angular displacement of the occipital condyle of the head relative to the T_1 . Working performance guidelines for NDC in the Hybrid III and the BioRID P3 for low speed rear impacts are proposed in four different categories: Excellent, Good, Acceptable and Poor.

For the Hybrid III, the requirements for Excellent are: a. The head relative to T_1 angle should be <20 degrees; b. The x displacement of the head relative to the T_1 <30 mm; c. The z displacement of the head relative to the T_1 <15 mm. The requirements for Good are: a. The head relative to T_1 angle should be <35 degrees; b. The x displacement of the head relative to the T_1 <50 mm; c. The z displacement of the head relative to the T_1 <25 mm. The requirements for Acceptable are: a. The head relative to T_1 angle should be <50 degrees; b. The x displacement of the head relative to the T_1 <70 mm; c. The z displacement of the head relative to the T_1 <35 mm. The requirements for Poor are: a. The head relative to T_1 angle is >50 degrees; b. The x displacement of

the head relative to the T_1 >70 mm; c. The z displacement of the head relative to the T_1 >-35 mm.

In addition, a response outside the corridor places the response in the category "Poor". For the BioRID, the guidelines are 5 degrees higher for the head relative to T_1 angle, 5 mm more for the x displacement of the head relative to the T_1 and the same for the Hybrid III for the z displacement of the head relative to the T_1 . The correlation between these three injury criteria and the risk of long term soft tissue neck injury has not yet been established.

The N_{ij} , N_{km} , NIC, NDC and lower neck moment can be applied to current rear impact dummies. Reference values have to be adapted to the chosen dummy. The validity of all these criteria, in predicting the injury risk, needs to be established.

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