# VALIDATION OF THE NECK INJURY CRITERION (NIC) USING KINEMATIC AND CLINICAL RESULTS FROM HUMAN SUBJECTS IN REAR-END COLLISIONS

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## ABSTRACT

Neck Injury Criterion (NIC) values were calculated using human subject kinematic data and compared to their clinical results. Twenty-nine percent (29%) and 38% of the subjects exhibited whiplash-associated disorders (WAD) at rear-end speed changes of 4 and 8 km/h, respectively. None of the subjects' NIC values exceeded 15 $m^2/s^2$, which had been proposed as a tolerance level for AIS-1 cervical injury. NIC was not able to predict the presence of symptoms in our test population. This may be due to differences between our subjects' anatomical source of pain and the nature and type of injury predicted by NIC.

A NECK INJURY CRITERION (NIC) to mathematically model and predict neck injuries in low-speed rear-end automobile collisions has been proposed based on the relative acceleration and velocity between the top and the bottom of the cervical spine (Boström, et al., 1996). This criterion was based on a theory first presented by Aldman (1986) and a pig model developed by Svensson, et al. (1993), which produced histopathologic findings especially to the dorsal root ganglion in the lower cervical spine after rapid induced head motion in the sagittal plane (Örtengren, et al. 1996). Using Evans Blue dye conjugated to Albumin (EBA) Örtengren, et al. (1996) found leakage indicative of cell membrane dysfunction in the cervical spinal ganglia, which Svensson, et al. (1993) correlated with measured pressure gradients in the intervertebral canal of the cervical spine. Svensson (1993) proposed that the ganglia pathologies could have been caused by mechanical stresses and strains due to relative motion between vertebral segments but that a lack of observed trauma to the cervical vertebrae, discs, and ligaments supported Aldman's (1986) theory that the ganglia pathology was caused by hydro-dynamic effects from change in the inner volume of the spinal canal during the rapid extension/flexion motions of the neck. Svensson, et al. (1993) theorized that this pressure gradient injury

mechanism and resultant pathology could be responsible for AIS-1 symptoms experienced by occupants in rear-end collisions.

The work of Penning (1992) focused on rearward translation (retraction) of the head relative to the torso as a potential cervical injury mechanism and the recent work of Svensson, et al. (1993) and Boström, et al. (1996, 1997) addressed retraction as a potential injury mechanism during low-speed rear-end impacts. These authors postulated that as the head and neck passed through phases during the rearward motion the cervical spine changed shape between a straight and an S-shape, which probably caused the pressure transient along the spinal canal. They also suggested that the passage of maximal retraction was the critical phase in the head/neck kinematics. Consequently, Boström, et al. (1996) proposed a Neck Injury Criterion (NIC) with the equation

$$NIC = a_{rel} * L + v_{rel}^2,$$

where $a_{rel}$ and $v_{rel}$ were the relative horizontal acceleration and velocity between the bottom (T1) and top (C1) of the cervical spine (T1–C1). The term L represented the length of the cervical spine, which was set at 0.2 m for the pig model and assumed by these researchers to be a similar value in humans (Boström, et al. 1996). Svensson, et al. (1993) acknowledged that the pig model could not provide quantitative data regarding the injury threshold for human beings; however Boström, et al. (1996) proposed a preliminary estimate of human tolerance level of NIC < 15 $m^2/s^2$. The authors recommended that this proposed threshold be validated, falsified or modified using accident and volunteer test data.

Boström, et al. (1997) suggested that the NIC value should be calculated at the instant of maximal retraction but that due to insufficient human whiplash kinematic data that this point needed to be assumed. Therefore, it was proposed that the NIC value be calculated at 50mm of relative T1-C1 displacement, whereby NIC-50 equals NIC at the time when the double integral of $a_{rel}$ equals 50 mm. Boström, et al. (1997) used kinematic data from volunteer sled tests conducted at 8.5 and 10.5 km/h sled velocity with an average deceleration of 2.5 g to evaluate the NIC-50 (Eichberger, et al., 1996). These NIC-50 values were calculated from chest acceleration data rather than T1 acceleration data and estimated head center of gravity (CG) acceleration rather than C1 acceleration data. The averaged NIC-50 values reported from sixteen volunteer tests across five different car seats were all below the 15 $m^2/s^2$ threshold. Eichberger, et al. (1996) reported that three volunteers suffered from neck complaints on the day following the tests, which lasted approximately twenty-four hours and one volunteer complained of cervical distortion symptoms for about two weeks. No statistical analyses were reported by Boström, et al. (1997) regarding correlation between those volunteers with and without symptoms and NIC-50 values and it is not known if any of those individual NIC-50 values exceeded the 15 $m^2/s^2$ threshold.

Siegmund, et al. (1997) recently reported detailed head and neck kinematic data from human subjects exposed to a 4 and 8 km/h speed change. Brault, et al. (1998a) recently published the clinical results of these human subject tests including the presence, severity and duration of cervical symptoms. Symptoms consistent with whiplash-associated disorders (WAD) including cervical symptoms of AIS-1 level were recorded. The availability of

these combined kinematic and clinical data presents a unique opportunity to test the proposed Neck Injury Criterion. The primary purpose of this paper was to correlate NIC values calculated from human subject kinematic data to the subjects' presence of symptoms. The detailed human head and neck kinematic data were also used to investigate the differences between using data at the head CG and data at the atlanto-occipital (A/O) joint to represent C1 in the NIC equation and determination of retraction values.

## METHODS

The NIC validation tests were conducted using kinematic and clinical data acquired from human subjects during rear-end vehicle-to-vehicle impact tests. The impact tests were conducted as a part of a larger project investigating injury mechanisms in low-speed rear-end automobile impacts. Forty-two subjects (21 male, 21 female) between 20 and 40 years old (mean age 26.8 years) participated in the study. Human subject protection policies, test protocol, and human subject test procedures were reviewed and approved by the Western Institutional Review Board, an independent ethics review committee, and informed consent was obtained from each subject. Pre-test magnetic resonance images (MRI) were taken of each subject's head and cervical spine for the purposes of screening for pre-existing cervical pathology and obtaining head and neck geometry.

Each subject underwent a pre-test clinical examination in order to assess their musculoskeletal and neuromuscular systems and establish baseline values for several clinical measures. Pre-test measures included cervical range of motion, cervical muscle strength, C4-T1 dermatomal sensation and myotomal strength, upper extremity deep tendon reflexes, and evaluation for point tenderness. Please see Brault, et al. (1998a) for a more detailed description of the human subject clinical protocol.

Kinematic parameters measured and/or resolved included linear and angular acceleration, velocity, and displacements of the head CG and C7/T1 joint axis (Siegmund, et al., 1997). The head CG was assumed to be in the mid-sagittal plane and its superior-inferior and anterior-posterior position was estimated using regression equations published by Clauser, et al. (1969) for each subject. The C7/T1 joint axis was determined to be the center of rotation of the base of the neck and estimated to be in the mid-sagittal plane and at the midpoint between the C7 spinous process and the manubrium (Queisser, et al., 1994). The C7/T1 joint axis location was used to represent the T1 term in the NIC equation.

Head accelerations in six degrees of freedom were acquired via a nine accelerometer array (Kistler 8302B20S1; $\pm$20g) arranged in a 3-2-2-2 configuration, which was attached to the subject's head. Torso acceleration was measured via a tri-axial linear accelerometer (Summit 34103A; $\pm$7.5g) and a tri-axial angular rate sensor (ATA-Sensors DynaCube; $\pm$100rad/s). All data channels conformed to SAE J211 Channel Class 1000 and data were acquired at 10kHz.

Digital high-speed video captured sagittal plane motion using an OmniSpeed HS motion capture system (Speed Vision Technologies) and high-

speed camera (JC Labs 250). The high-speed video data were collected at 250 frames per second (fps) and at 1/1000 s shutter speed. Reflective targets were applied to the subject, seat, and vehicle. Head targets were affixed on the glabella, left temporomandibular joint (TMJ), left lateral aspect of the cranium, and left side of the head accelerometer unit. Torso targets were placed on the chest accelerometer unit and over the C7 spinous process. Target displacements were digitized (OmniSpeed AutoTracker Software) and used to resolve displacement data of the head CG, A/O joint, and C7/T1 joint axis.

Location and orientation of the head and torso instrumentation along with the reflective targets were measured relative to designated anatomical landmarks using a three-dimensional digitizer (FaroArm B08-02; $\pm$0.30mm). Head mounted accelerometer data were resolved to the estimated head CG and integrated to obtain velocity data, while torso mounted accelerometer data were resolved to the C7/T1 joint axis and integrated to velocity data as reported in Siegmund, et al. (1997).

For the purposes of calculating NIC values, accelerations and velocities were also resolved to the A/O joint. The location of each subject's A/O joint relative to external anatomical landmarks was determined from sagittal plane MR images of the head and neck in neutral position. The left and right A/O joints were digitized separately, averaged, and projected onto the mid-sagittal plane. Head kinematics were then resolved to this point. The A/O joint was used to represent the C1 term in the NIC equation.

Each subject's cervical spine length (L) was also determined from sagittal plane MR images of the cervical spine in the neutral position. This length was taken as the linear distance between the midpoint of the A/O joint and midpoint of the C7/T1 joint. The measured length of each subject's cervical spine obtained from the MRI was used in their individual NIC calculations.

Subjects were seated in the front passenger seat of a 1990 Honda Accord LX four door sedan, which was struck from the rear by a 1983 Volvo 240DL station wagon. Vehicle speed change was measured using an MEA 5$^{th}$ wheel sampling at a rate of 128 Hz, while ribbon switches (Nortel TapeSwitch 121BP; 2N activation force) measured onset and duration of bumper contact. Data were synchronized to time zero at bumper contact. Each subject was exposed to two impact tests, one at a speed change of 3.95 $\pm$ 0.11 km/h and a second at 8.10 $\pm$ 0.11 km/h. The order of the two impact tests was randomized and separated by a minimum of seven symptom-free days. There were three subjects who completed the 4km/h speed change impact but did not complete the 8 km/h speed change, while some accelerometer data were lost to instrumentation problems. Thirty-nine (39) tests at 4 km/h and 36 tests at 8 km/h yielded complete kinematic and clinical data, resulting in 75 tests to evaluate the NIC.

Post-test clinical examinations were conducted within 30 minutes of the impact test. These examinations included repeating the pre-test clinical measures and administering the McGill Pain Questionnaire, which was designed to provide quantitative measures of clinical pain that can be treated statistically (Melzack, 1975). Another clinical examination was conducted 24 hours after the impact test. These data were treated statistically in order to

identify changes in the clinical measures attributable to the impact test (Brault, et al., 1998a).

NIC values were calculated and plotted across the time of impact. NIC values were calculated using two data sets: C7/T1 data relative to the A/O joint ($NIC_{A/O}$) and C7/T1 relative to the head CG ($NIC_{CG}$). NIC-50 values were also obtained at the point of 50 mm retraction of A/O joint translation relative to C7/T1 and 50 mm retraction of head CG translation relative to C7/T1 if retraction values reached the 50 mm level.

Logistic regression (modeled using a log linear model) was used to determine whether the calculated NIC values could be used to predict the presence of symptoms (Wilcox, 1996). The likelihood ratio chi squared ($\chi^2$) was used to test for significant effects of the various measurements. All data was pooled across speed change for the logistic regression analysis. Comparisons between the NIC values computed from the A/O joint data and the NIC values computed from the head CG data were performed using a Wilcoxon signed-rank test (Wilcox, 1996). A $p$ value of .05 level of significance was used for all statistical comparisons.

## RESULTS

Table 1 displays summary statistics regarding the count of subjects with and without symptoms at the two speed changes. Symptoms have been partitioned into two groups; GROUP I consists of those subjects who reported symptoms to any body region (cervical, thoracic, and/or headaches), while GROUP II consists of only those subjects who reported cervical and/or thoracic symptoms. For more detail regarding the clinical results please refer to Brault, et al. (1998a).

The mean length of the subjects' cervical spines was 0.119 $\pm$ 0.008 m with a range from 0.101 to 0.133 m. None of the subject's cervical spine length was equal to the 0.2 m assumed by Boström, et al. (1996). Figure 1 shows an exemplar plot of NIC and retraction values at an 8 km/h speed change. Time zero represents bumper contact. The NIC trace followed a pattern of two distinct peaks; an initial positive peak (labeled A) occurred at a mean time of 96 $\pm$ 12 ms and a second negative peak (labeled B) occurred at a mean time of 141 $\pm$ 27 ms. The second absolute peak value (NIC-B) was greater in magnitude than the first peak (NIC-A) and occurred just before maximum retraction (labeled C). Negative retraction values indicate rearward translation of the A/O joint relative to C7/T1. The mean time to maximum retraction was 153 $\pm$ 19 ms.

None of the subjects' peak NIC values at either speed change exceeded the proposed $15 m^2/s^2$ threshold. Table 2 shows the results of the logistic regression analyses for subjects with and without symptoms including the mean, minimum, maximum, and standard deviation for NIC peak values at points NIC-A, NIC-B, NIC-C, and NIC-50.

Although the proposed NIC threshold value was never reached, logistic regression analyses on NIC-50 using head CG translation predicted GROUP I symptoms (cervical, thoracic, and/or headaches). The presence of any symptoms representing by the Group I count was then modified to GROUP II symptoms, which eliminated headache symptoms and consisted only of cervical

and/or thoracic symptoms. This was done because of a potential confounding effect of the tightly head mounted tri-axial unit, which the researchers felt might have been a source of some of the subjects' headache complaints. With this potential confounding effect removed, NIC-50 did not predict the presence of symptoms in GROUP II subjects (cervical and/or thoracic complaints).

Tables 3a and 3b show summary statistics regarding the maximum retraction for female and male subjects at the two speed changes. There were significant differences noted in maximum retraction between gender and speed change. Table 4a and 4b show a count of those subjects whose maximum retraction was greater than or less than 50 mm and the presence of symptoms. When using head CG translation 44 of the 81 impact tests (54%) resulted in maximum retraction beyond 50 mm, which accounted for only 13 of the 27 subjects with any symptoms (48%) and only 8 of the 19 subjects with cervical or thoracic symptoms (42%). When using A/O translation only 13 of the 81 impact tests (16%) resulted in maximum retraction beyond 50 mm, which accounted for 6 of the 27 subjects with any symptoms (22%) and 6 of the 19 subjects with cervical or thoracic symptoms (32%).

Figure 2 displays an exemplar plot comparing NIC calculated using A/O joint data ($NIC_{A/O}$) and NIC calculated using head CG data ($NIC_{CG}$). The $NIC_{CG}$ peak values were greater than $NIC_{A/O}$ peak values at NIC-A in all seventy-five trials (100%) with a mean difference of 25%. The differences between $NIC_{A/O}$ peak values and $NIC_{CG}$ peak values at NIC-A were found to be significant ($p < .05$). $NIC_{CG}$ peak values were greater than $NIC_{A/O}$ peak values at NIC-B in forty-nine of the seventy-five trials (65%) with a mean difference of 8%.

## DISCUSSION

None of the test subjects' NIC values exceeded the previously proposed 15 $m^2/s^2$ threshold, yet overall 33% of the tests resulted in symptoms. Of the 42 subjects tested 22 (52%) reported symptoms at either the 4 or 8 km/h speed change and 3 subjects who reported symptoms at 4 km/h elected not to submit to the 8 km/h test. One reason that the NIC may not have predicted the occurrence of WAD symptoms in our test subjects is because NIC is based on a pressure gradient injury mechanism model that predicts dorsal root ganglion pathology, while the precise source of our subjects' symptoms was not known. It was not possible to verify by histopathological examination whether or not dorsal root ganglia injury occurred to our subjects. Furthermore, no significant differences were noted in post-impact clinical examinations for reflex, sensory, or upper extremity muscle strength, which suggested that our subjects' symptoms were not nerve based. Our subjects' measured deficits in cervical range of motion and tenderness upon palpation of cervical and upper thoracic muscles may suggest musculotendinous trauma. We are continuing to investigate the role of cervical muscle response in combination with the head/neck kinematics as a potential muscle-based injury mechanism (Brault, et al., 1998b).

Another reason that NIC did not predict our subjects' symptoms is that the severity and duration of our subjects' symptoms may not correlate with the intended use of NIC. Symptom mean Present Pain Intensity (PPI) scores were

1.3 ± 1.2 and 0.8 ± 0.5 (scale 1 to 5) and symptom median duration were 5.8 (range 1 to 48) and 24 (range 0.3 to 111) hours for the 4 km/h and 8 km/h speed changes, respectively (Brault, et al., 1998a). The concern of the researchers developing the NIC may have been symptoms and disorders more severe and/or of longer duration. This may explain why Boström, et al. (1997) interpreted that the volunteers in Eichberger, et al. (1996) underwent "non-injurious motions" even though several volunteers reported symptoms and thus concluded that the NIC was a useful predictor of real-life neck injuries because average NIC values were below their proposed 15 $m^2/s^2$ threshold. Therefore, the nature and severity of our subjects' reported symptoms may be below NIC's intended level of application.

Another possible explanation for the failure of NIC to predict our subjects' symptoms is that our speed changes were too low. Svensson, et al. (1993) and Boström, et al. (1996, 1997) have considered low-velocity impacts as below 20 km/h speed change. Our tests were conducted at 4 km/h and 8 km/h speed change. Perhaps our minimal and short duration symptoms represent different injuries and/or precursors to more severe injuries that may result at higher speed changes. Consequently, NIC and its proposed threshold of 15 $m^2/s^2$ cannot be used to predict the onset of minor whiplash symptoms as a result of low-speed rear-end impacts at 4 and 8 km/h speed change. It will be difficult, if not impossible, to use human subjects to bridge the gap between our reported onset of symptoms at 4 and 8 km/h speed change and higher impact speeds. Ethical standards and adherence to human subject protection policies restrict the use of subjects at higher impact speeds.

Oddly, the only NIC parameter which correlated with the presence of symptoms was NIC-50 when using head CG retraction values. At this point, NIC values were decreasing from their initial peak (NIC-A) and in almost all subjects were negative. This negative NIC value corresponds to a forward acceleration of the A/O joint relative to C7/T1, although retraction was still increasing. This behavior was likely the result of relative acceleration's ($a_{rel}$) dominant role in the NIC calculation. It is unknown if the NIC is intended to be used in this region.

When using head CG translation only 54% of our test subjects' maximum retraction reached 50 mm. The remaining 46% of the subjects' maximum retraction never reached 50 mm including 52% of those subjects with Group I symptoms and 58% of those subjects with Group II symptoms. When using A/O translation only 16% of our test subjects' maximum retraction reached 50 mm. The remaining 84% of the subjects' maximum retraction never reached 50 mm including 78% of those subjects with Group I symptoms and 68% of those subjects with Group II symptoms. The decrease of the number of subjects that reached 50 mm retraction from 44 to 13 subjects highlights the influence of using head CG data versus A/O data. As previously noted, it is believed the NIC was intended to use C1 (represented by the A/O joint data) relative to T1 (represented by the C7/T1 joint axis data) to calculate retraction values and not head CG data. Additional work is being conducted to determine true retraction values by subtracting out rotational effects, which will likely reduce the values presented here.

The 50 mm requirement to calculate NIC-50 excluded a significant portion of our test subjects and may potentially miss a significant portion of the

general population whose maximum retraction is less than 50 mm yet might still sustain injury. This may be especially true if recommendations to decrease backset (horizontal distance between the back of the head and front surface of the head restraint) and limit retraction are embraced by automotive safety design engineers. Olsson, et al. (1990) observed an increased injury risk when the backset was greater than 10 cm and thus current seat back and head restraint design developments are focusing on minimizing the backset distance both before and during the impact. Wiklund, et al. (1998) showed that an active head restraint design can be successful in minimizing the retraction motion during a rear impact but it has yet to be determined if this would effectively eliminate the injury mechanism observed in our study. Our subjects all had initial backsets of less than 8 cm with a mean of less than 5 cm. Yet our published clinical results suggest that an injury mechanism does exist under these conditions (Brault, et al., 1998a). Based upon the data from our human subject tests, using NIC-50 to predict WAD to subjects' with adequate head restraint protection at rear-end speed changes of 4 and 8 km/h was not practical.

NIC was calculated using both head CG and A/O data sets in order to test for significance. $NIC_{CG}$ and $NIC_{A/O}$ peak values were significantly different at NIC-A with the mean $NIC_{CG}$ peak value 25% greater in magnitude. Considering the NIC mathematical model was based on kinematics at the top and bottom of the cervical spine and not at head CG, these differences should be kept in mind by researchers when making assumptions regarding the kinematic variable input to the NIC equation as well as the retraction measurement.

## CONCLUSIONS

Computation of NIC values using human subject kinematic data at 4 km/h and 8 km/h rear-end speed changes did not result in NIC values in excess of the previously proposed 15 $m^2/s^2$ threshold despite symptoms reported in 33% of the tests and in 52% of the subjects at either the 4 or 8 km/h speed change. This discrepancy may be due to differences between the mechanism, nature, and severity of the reported symptoms and those modeled and predicted by the NIC. The symptoms reported were minimal, short duration, and may have been musculotendinous in nature, while the NIC was modeled on a pressure gradient injury mechanism theory, which may predict dorsal root ganglion injuries and may better predict more severe and/or longer symptoms. NIC-50 was not a practical parameter for predicting the presence of WAD symptoms during rear-end speed changes of 4 and 8 km/h considering a significant portion of our test subjects did not exceed 50 mm retraction during these impacts with properly positioned head restraints. Future developments in automotive safety including improved seat back and head restraint designs aimed at limiting retraction may also limit the application of the NIC-50. Finally, calculation of NIC and retraction values were influenced by the assumption of data taken at the head CG or A/O joint. Future researchers should consider this when applying kinematic data to the calculation of NIC and retraction values.

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REFERENCES

Aldman B. An Analytical Approach to the Impact Biomechanics of Head and Neck. In: 30th Annual Proceedings Association for the Advancement of Automotive Medicine, 1986, 439-454.

Boström O, Svensson MY, Aldman B, Hansson HA, Håland Y, Lövsund P, Seeman T, Suneson A, Säljö A, Örtengren. A New Neck Injury Criterion Candidate – Based on Injury Findings in the Cervical Spinal Ganglia after Experimental Neck Extension Trauma. In: Proceedings of the International Conference on the Biomechanics of Impact, Dublin, Ireland, 1996.

Boström O, Krafft M, Aldman B, Eichberger A, Fredriksson R, Håland Y, Lövsund P, Steffan H, Svensson M, Tingvall C. Prediction of Neck Injuries in Rear Impacts Based on Accident Data and Simulations. In: Proceedings of the International Conference on the Biomechanics of Impact, Germany, 1997.

Brault JR, Wheeler JB, Siegmund GP, Brault EJ. Clinical Response of Human Subjects to Rear-End Automobile Collisions. Archives of Physical Medicine and Rehabilitation, 79, 1998a, 72-80.

Brault JR, Smith TA, Wheeler JB, Siegmund GP. Cervical Muscle Response to Rear-End Automobile Collisions: Implications for Injury. In: Proceedings of the North American Congress on Biomechanics, Waterloo, Ontario, 1998b.

Clauser CE, McConvilee JT, Young JW. Weight, Volume, and Center of Mass of Segments of the Human Body (AMRL-TR-69-70). Wright Patterson Air Force Base, Aerospace Medical Research Laboratory, Yellow Springs, Ohio, August 1969.

Eichberger A, Geigl BC, Moser A, Fachbach B, Steffan H, Hell W, Langwieder K. Comparison of Different Car Seats Regarding Head-Neck Kinematics of Volunteers During Rear End Impact. In: Proceedings of the International Conference on the Biomechanics of Impact, Dublin, Ireland, 1996, 153-164.

Melzack R. The McGill Pain Questionnaire: Major Properties and Scoring Methods. Pain, 1975, 1:277-299.

Olsson I, Bunkertop O, Carlsson G, Gustafsson C, Planath I, Norin H, Ysander L. An In-depth Study of Neck Injuries in Rear End Collisions. In: Proceedings of the International Conference on the Biomechanics of Impact, Bron-Lyon, France, 1990, 269-282.

Penning L. Acceleration Injury of the Cervical Spine by Hypertranslation of the Head, Part I, Effect of Normal Translation of the Head on Cervical Spine Motion: A Radiology Study. European Spine Journal, 1, 1992, 7-12.

Queisser F, Bluthner R, Seidel H. Control of Positioning the Cervical Spine and its Application to Measuring Extensor Strength. Clinical Biomechanics, 1994, 9:157-161.

Siegmund GP, King DJ, Lawrence JM, Wheeler JB, Brault JR, Smith TA. Head/Neck Kinematic Response of Human Subjects in Low-Speed Rear-End Collisions. SAE Paper No. 973341, In: Proceedings of the 41st Stapp Car Crash Conference, Lake Buena Vista, Florida, 1997, 357-385.

Svensson MY, Aldman B, Hansson HA, Lövsund P, Seeman T, Suneson A, Örtengren T. Pressure Effects in the Spinal Canal during Whiplash Extension Motion: A Possible Cause of Injury to the Cervical Spinal Ganglia. In: Proceedings of the International Conference on the Biomechanics of Impact, Eindhoven, The Netherlands, 1993, 189-200.

Wiklund K, Larsson H. Saab Active Head Restraint (SAHR) – Seat Design to Reduce the Risk of Neck Injuries in Rear Impacts. SAE Technical Paper Series No. 980297, International Congress and Exposition, Detroit, Michigan, 1998.

Wilcox RM. Statistics For The Social Sciences. Academic Press Inc., San Diego, California, 1996.

**Table 1: Summary Statistics – Presence Of Symptoms And Speed Change**

| | 4 km/h Speed Change<br>Count (Percent) | 8 km/h Speed Change<br>Count (Percent) | Total<br>Count (Percent) |
|---|---|---|---|
| GROUP I: | | | |
| With Symptoms: Cervical, Thoracic, and/or Headaches | 12 (15) | 15 (19) | 27 (33) |
| Without Symptoms | 30 (37) | 24 (30) | 54 (67) |
| Total | 42 (52) | 39 (48) | 81 (100) |
| | | | |
| GROUP II: | | | |
| With Symptoms: Cervical and/or Thoracic | 7 (9) | 12 (15) | 19 (23) |
| Without Symptoms: Cervical or Thoracic | 35 (43) | 27 (33) | 62 (77) |
| Total | 42 (52) | 39 (48) | 81 (100) |

**Figure 1: NIC and Retraction**
**Exemplar Data For 8 km/h Speed Change**

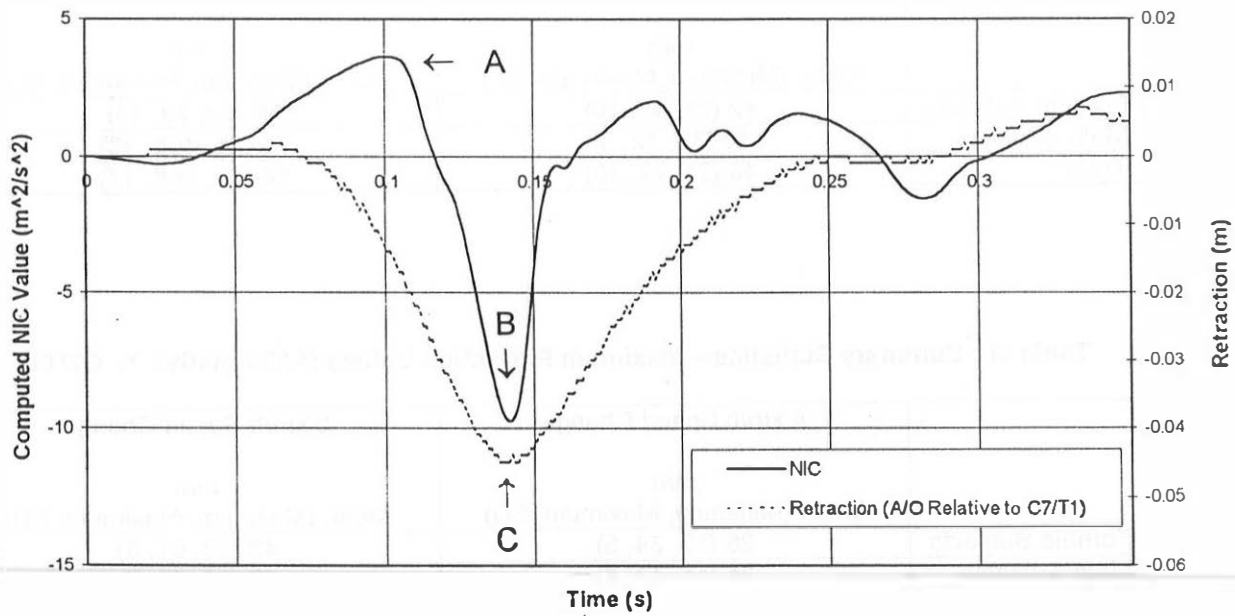

**Table 2: Logistic Regression Of NIC Values Used To Predict For The Presence Of Symptoms Following A 4 km/h Or 8 km/h Collision**

| GROUP I: | | | |
|---|---|---|---|
| | Subjects Without Symptoms<br><br>$m^2/s^2$<br>Mean (Minimum, Maximum, SD) | Subjects With Symptoms: Cervical, Thoracic, and/or Headaches<br><br>$m^2/s^2$<br>Mean (Minimum, Maximum, SD) | *p* value * |
| NIC – A | 2.15 (0.86, 4.67, 0.94) | 2.46 (1.28, 3.97, 0.81) | .171 |
| NIC – B | -4.59 (-1.65, -11.92, 2.57) | -5.09 (-2.17, -8.61, 2.09) | .405 |
| NIC – C | -1.97 (-6.75, 7.19, 1.96) | -1.79 (-5.76, 4.02, 1.79) | .707 |
| NIC – 50 | -2.96 (-7.08, 1.02, 1.68) | -1.61 (-3.77, 0.71, 1.61) | .027† |
| | | | |
| GROUP II: | | | |
| | Subjects Without Symptoms: Cervical or Thoracic<br><br>$m^2/s^2$<br>Mean (Minimum, Maximum, SD) | Subjects With Symptoms: Cervical and/or Thoracic<br><br>$m^2/s^2$<br>Mean (Minimum, Maximum, SD) | *p* value * |
| NIC – A | 2.13 (0.86, 4.67, 0.92) | 2.61 (1.28, 3.97, 0.79) | .059 |
| NIC – B | -4.56 (-1.65, -11.92, 2.56) | -5.37 (-2.79, -8.61, 1.86) | .219 |
| NIC – C | -2.00 (-6.75, 7.19, 1.90) | -1.64 (-5.76, 4.02, 2.43) | .509 |
| NIC – 50 | -2.81 (-7.08, 1.02, 1.66) | -1.44 (-3.77, 0.71, 1.84) | .052 |

* Likelihood ratio $\chi^2$ from logistic regression.
† $p < .05$.

**Table 3a: Summary Statistics – Maximum Retraction Values (Head CG Relative To C7/T1)**

| | 4 km/h Speed Change<br><br>mm<br>Mean (Minimum, Maximum, SD) | 8 km/h Speed Change<br><br>mm<br>Mean (Minimum, Maximum, SD) |
|---|---|---|
| Female Subjects | 42 (21, 57, 10) | 56 (32, 79, 13) |
| Male Subjects | 50 (33, 72, 9) | 70 (25, 108, 18) |
| Total | 46 (21, 72, 10) | 64 (25, 108, 17) |

**Table 3b: Summary Statistics – Maximum Retraction Values (A/O Relative To C7/T1)**

| | 4 km/h Speed Change<br><br>mm<br>Mean (Minimum, Maximum, SD) | 8 km/h Speed Change<br><br>mm<br>Mean (Minimum, Maximum, SD) |
|---|---|---|
| Female Subjects | 26 (19, 34, 5) | 43 (33, 61, 8) |
| Male Subjects | 31 (11, 43, 7) | 47 (35, 57, 7) |
| Total | 29 (11, 43, 6) | 45 (33, 61, 8) |

**Table 4a: Summary Statistics – Maximum Head CG Retraction Values And Presence Of Symptoms**

| GROUP I: | | | |
|---|---|---|---|
| | Subjects Without Symptoms<br><br>Count (Percent) | Subjects With Symptoms: Cervical, Thoracic, and/or Headaches<br><br>Count (Percent) | Total<br><br>Count (Percent) |
| Retraction > 50 mm | 31 (38) | 13 (16) | 44 (54) |
| Retraction < 50 mm | 23 (28) | 14 (17) | 37 (46) |
| Total | 54 (67) | 27 (33) | 81 (100) |
| | | | |
| GROUP II: | | | |
| | Subjects Without Symptoms: Cervical or Thoracic<br><br>Count (Percent) | Subjects With Symptoms: Cervical and/or Thoracic<br><br>Count (Percent) | Total<br><br>Count (Percent) |
| Retraction > 50 mm | 36 (44) | 8 (10) | 44 (54) |
| Retraction < 50 mm | 26 (32) | 11 (14) | 37 (46) |
| Total | 62 (77) | 19 (23) | 81 (100) |

**Table 4b: Summary Statistics – Maximum A/O Retraction Values And Presence Of Symptoms**

| GROUP I: | | | |
|---|---|---|---|
| | Subjects Without Symptoms<br><br>Count (Percent) | Subjects With Symptoms: Cervical, Thoracic, and/or Headaches<br><br>Count (Percent) | Total<br><br>Count (Percent) |
| Retraction > 50 mm | 7 (9) | 6 (7) | 13 (16) |
| Retraction < 50 mm | 47 (58) | 21 (26) | 68 (84) |
| Total | 54 (67) | 27 (33) | 81 (100) |
| | | | |
| GROUP II: | | | |
| | Subjects Without Symptoms: Cervical or Thoracic<br><br>Count (Percent) | Subjects With Symptoms: Cervical and/or Thoracic<br><br>Count (Percent) | Total<br><br>Count (Percent) |
| Retraction > 50 mm | 7 (9) | 6 (7) | 13 (16) |
| Retraction < 50 mm | 55 (68) | 13 (16) | 68 (84) |
| Total | 62 (77) | 19 (23) | 81 (100) |

**Figure 2: Comparison Of NIC Using A/O Joint Versus Head CG**