Annals of Biomedical Engineering, Vol. 39, No. 8, August 2011 *(© 2011)* pp. 2152–2162
DOI: 10.1007/s10439-011-0315-4

Cervical Spine Model to Predict Capsular Ligament Response in Rear Impact

JASON B. FICE, DUANE S. CRONIN, and MATTHEW B. PANZER

Department of Mechanical Engineering, University of Waterloo, 200 University Ave. West, Waterloo, ON N2L 3G1, Canada

(Received 26 December 2010; accepted 15 April 2011; published online 1 May 2011)

Associate Editor Stefan M Duma oversaw the review of this article.

Abstract—Predicting neck kinematics and tissue level response is essential to evaluate the potential for occupant injury in rear impact. A detailed 50th percentile male finite element model, previously validated for frontal impact, was validated for rear impact scenarios with material properties based on actual tissue properties from the literature. The model was validated for kinematic response using 4g volunteer and 7g cadaver rear impacts, and at the tissue level with 8g isolated full spine rear impact data. The model was then used to predict capsular ligament (CL) strain for increasing rear impact severity, since CL strain has been implicated as a source of prolonged pain resulting from whiplash injury. The model predicted the onset of CL injury for a 14g rear impact, in agreement with motor vehicle crash epidemiology. More extensive and severe injuries were predicted with increasing impact severity. The importance of muscle activation was demonstrated for a 7g rear impact where the CL strain was reduced from 28 to 13% with active muscles. These aspects have not previously been demonstrated experimentally, since injurious load levels cannot be applied to live human subjects. This study bridges the gap between low intensity volunteer impacts and high intensity cadaver impacts, and predicts tissue level response to assess the potential for occupant injury.

Keywords—Cervical spine injury, Rear impact, Whiplash, Finite element model.

INTRODUCTION

Whiplash or soft tissue cervical spine sprains/strains are the most common injury in motor vehicle collisions, with 28–53% of collision victims sustaining this type of injury.[12,44] The annual societal costs of whiplash in the United States are estimated to be between 4.5 and 8 billion dollars,[23,66] and whiplash can be a chronic injury with long-term symptoms, which can adversely affect the victim's quality of life. Radanov *et al.*[46] reported that 24% of whiplash victims had symptoms 1 year after an accident and 18% after 2 years, where it has been found that between 38 and 52% of whiplash cases occurred in rear impact scenarios.[2,12]

Although the specific mechanisms of whiplash injury are still under investigation, several theories link the facet joints, spinal ligaments, intervertebral discs, vertebral arteries, dorsal root ganglia, and neck muscles to possible sites of injury.[52] One of the most thoroughly investigated sites of injury in terms of clinical and biomechanical studies is the capsular ligament (CL) of the cervical spine facet joints. Clinical studies using double-blind anesthetic blocks reported 54–60% of whiplash patients had facet joint pain.[1,31] Similarly, *in vivo* animal models of the goat and rat have demonstrated that tensile force applied across the facet joint led to measurable changes in behavioral sensitivity or nerve discharge that has been linked to pain.[30,32] *In vitro* sub-catastrophic failure of the CL in a rat model was measured at a distraction magnitude of 0.57 mm, and the same distraction led to pain for up to 14 days *in vivo*.[29,30,45] The sub-catastrophic failure strain of isolated PMHS facet joints was reported to range from $35 \pm 21\%$[49] to $65 \pm 74\%$.[59] Low-speed rear impact studies using whole PMHS and cadaveric cervical spines reported peak CL strains from 29 to 40%, which is within the range of reported sub-catastrophic failure.[9,36,43,53] Post-impact tensile testing of the CLs exposed to rear impact showed a decrease in ligament stiffness compared to the untested control group, indicating the possibility that some damage had occurred in the ligaments during the impact tests.[20]

The finite element cervical spine model used in this study was previously validated at the segment level,

Address correspondence to Duane S. Cronin, Department of Mechanical Engineering, University of Waterloo, 200 University Ave. West, Waterloo, ON N2L 3G1, Canada. Electronic mail: dscronin@uwaterloo.ca

whole spine level in tension, and in frontal impact for head kinematics and local tissue response[41] (Appendix C—Electronic Supplementary Material). In the current study, the FE model was further validated for rear impacts based on global kinematics and local tissue deformation. Achieving this level of validation was noteworthy given the model geometry or material properties were not modified or calibrated to fit the experimental responses. The model was then exposed to rear impacts with increasing severity to determine a threshold for CL injury. These injuries were defined as predicted CL strains that exceeded published limits corresponding to a prolonged pain response. Finally, the influence of muscle activation on CL strain was investigated.

METHODS

Model Description

The finite element cervical spine model used in this study represents a 50th percentile male (Fig. 1); a full description of the model is presented in Appendix B—Electronic Supplementary Material. The model was developed with a focus on accurate geometric and material representation at the tissue level. The vertebrae geometries were in good agreement with published anthropometry and were modeled as rigid bodies for computational efficiency. The intervertebral discs were constructed with solid elements for the annulus fibrosus ground substance and nucleus pulposus, and layers of shell elements representing the fiber lamina (Fig. 1b). The facet joints were modeled with a superior and inferior layer of solid elements for the articular cartilage and a squeeze-film model to simulate the synovial fluid (Fig. 1c). Ligaments were represented using multiple 1D nonlinear rate-dependent tension-only spring elements. The model included both active and passive muscles that were modeled with 87 pairs of Hill-type 1D elements to represent 25 muscle pairs in the cervical spine. The material models for the components were based on studies in the literature (Table 1), and the properties were not modified to improve the fit to any data set. The model included 108,354 elements and was evaluated using the LS-Dyna explicit finite element software (LSTC, Livermore CA, version R3.2.1). As an example, the model took 46 h to solve a 280-ms duration impact, with a minimum time step of 1.1×10^{-4} ms, using four 2.1 GHz processors in a SMP configuration.

Muscle activation is critical for a cervical spine model to evaluate the response of a human subject.[4,40] Flexor and extensor muscles were found to activate at approximately the same time during an impact, and that EMG signals start at 60–79 ms after impact initiation.[35,48,50,54] The peak normalized EMG signal for the paraspinal muscles is 66–72% of the signal for the sternocleidomastoid, indicating that the flexor muscles in the neck activate with higher intensity than the extensor muscles during a rear impact.[3,50,51] In the current study, simulation of the living human response included the effect of the activated muscle response. Both the flexors and extensors were excited 74 ms after impact and remained active for 100 ms, while the activation level for the extensors was 70% of the flexor muscles (Appendix B—Electronic Supplementary

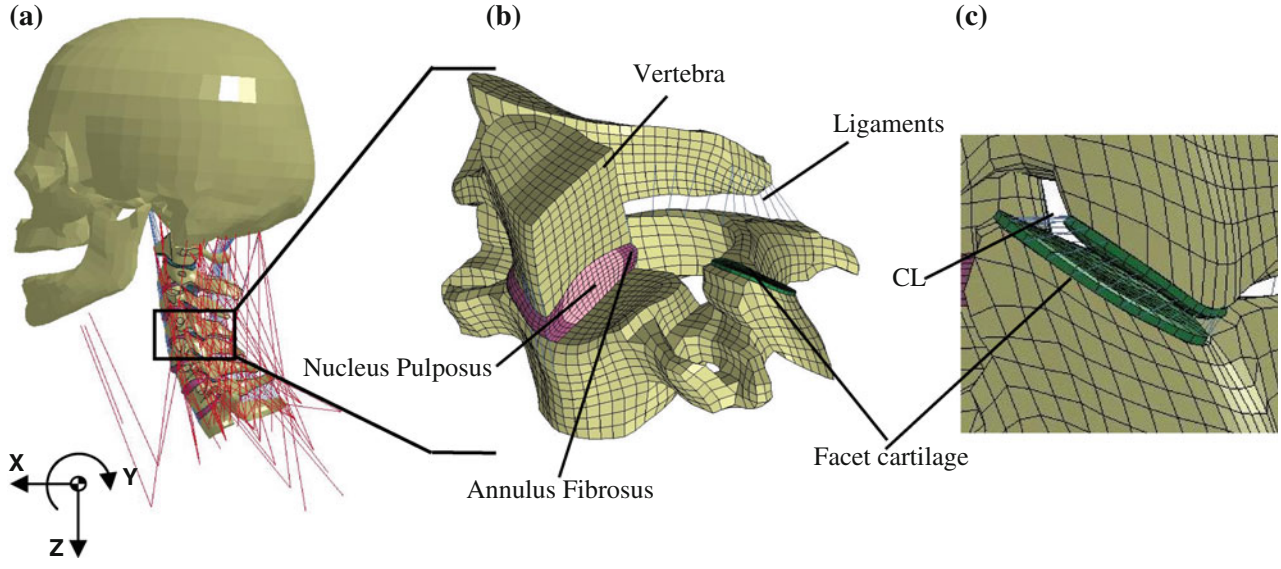

FIGURE 1. (a) Whole cervical spine model, (b) sectioned isometric view of the C4-C5 segment model, and (c) lateral close up of the C4-C5 facet joint.

TABLE 1. Material properties of the cervical spine model, including vertebral inertial properties.

Cervical tissue		Constitutive model	References
Skull		Rigid body Skull: $m = 4.376$ kg, $I_{yy} = 23{,}300$ kg mm^2	58
Vertebrae		Rigid body C1: $m = 22.6$ g, $I_{yy} = 3{,}759$ g mm^2 C2: $m = 25.4$ g, $I_{yy} = 5{,}372$ g mm^2 C3: $m = 16.2$ g, $I_{yy} = 2{,}166$ g mm^2 C4: $m = 17.0$ g, $I_{yy} = 2{,}183$ g mm^2 C5: $m = 18.8$ g, $I_{yy} = 2{,}559$ g mm^2 C6: $m = 19.1$ g, $I_{yy} = 3{,}260$ g mm^2 C7: $m = 18.5$ g, $I_{yy} = 3{,}461$ g mm^2	
Intervertebral disc	Ground substance	3D hyperelastic solid	41
	Annulus fibers	2D nonlinear elastic membrane	
	Nucleus pulposus	3D linear viscoelastic solid $K = 1.720$ GPa $G_1 = 0.5930$ kPa, $\beta_1 = 0.001477$ 1/s $G_2 = 0.6763$ kPa, $\beta_2 = 0.061524$ 1/s $G_3 = 0.9516$ kPa, $\beta_3 = 1.017893$ 1/s $G_4 = 2.0384$ kPa, $\beta_4 = 13.20041$ 1/s	18,63
Facet joint	Articular cartilage	3D linear viscoelastic solid $K = 2.0$ GPa $G_1 = 2.228$ MPa, $\beta_1 = 0.0248$ 1/s $G_2 = 0.5642$ MPa, $\beta_2 = 0.00545$ 1/s $G_{inf} = 0.210$ MPa	11
Ligaments		1D nonlinear, strain rate dependent beam (Appendix B—Electronic Supplementary Material)	6,64,65
Muscles		1D hill-type beam with activation (Appendix B—Electronic Supplementary Material)	25,57,60–62

Material). Due to the difficulty in defining a suitable muscle activation scheme to stabilize the model in a gravity field, the simulations were performed without gravity. Using a multi-body human cervical spine model, Van der Horst[56] showed that including gravity in a 15g frontal impact simulation resulted in a 5% maximum change of model kinematics.

Model Validation

The cervical spine model was previously validated at the segment level in flexion, extension, lateral bending, axial rotation, tension, compression, and anterior, posterior, and lateral shear.[41] The responses of model segments were generally within one standard deviation of the experimental responses. The full cervical spine model was previously validated in tension and was within one standard deviation of the experimental average (Appendix C, Fig. C1—Electronic Supplementary Material). The local tissue response of model in an 8g frontal impact was validated, and the strains were within one standard deviation of the experiments for 38 of 45 measures (Appendix C, Fig. C2—Electronic Supplementary Material). Finally, the global kinematics of the model head were compared to volunteer studies in 8g and 15g frontal impacts, and the response of the model was a good to excellent fit to the corridors (Appendix C, Fig. C3—Electronic Supplementary Material). The model validation was continued in this study for rear impacts in local tissue response and global kinematics. The data sets used for global kinematic validation were chosen based on the availability of data to describe the T1 *X*- and *Z*-acceleration and *Y*-rotation, so that the boundary conditions of the experimental tests could be fully implemented in the model.

The published results from a bench-top whole spine cadaver model with passive muscle force replication were used to validate the local tissue response.[21] The cadaver model consisted of a whole cervical spine from T1 to C0, with the T1 mounted to a pneumatic cylinder and a surrogate head mounted to the C0. The initial posture of the neck was maintained with a series of cables attached to springs, which were meant to model passive muscle resistance. The local tissue response in the cadaver model was measured by inferring tissue distractions based on the rigid body motion of the vertebra, which was tracked using opaque markers and high-speed cameras. These experiments were simulated by applying the experimental T1 acceleration trace in the anterior direction from an 8g rear impact onto the T1 of the model (Appendix A, Fig. A1—Electronic

Supplementary Material). The T1 in the model was constrained in all other directions, and the head was free. The mass and moment of inertia of the head were modified to 3.3 kg and 0.016 kg m^2, respectively, to agree with the experiments.[21] Muscle activation was not utilized to mimic the passive resistance modeled in the experiments. The ligament strains were calculated as tissue distraction divided by an anatomical neutral ligament lengths,[22,39] and the disc shear strains were calculated at the anterior, middle, and posterior aspects of the intervertebral discs as per the experiments.

The global kinematic response of the model was compared to 4g rear impact volunteer tests.[7] This group performed 28 rear impacts on 13 human volunteers at speeds between 5 and 7 kph with an average peak acceleration of 3.6g. The test involved the collision of a bullet sled with a stationary target sled and volunteer seated on a laboratory seat with a headrest. To model these rear impacts, the average T1 *X*-acceleration, *Z*-displacement, and *Y*-rotation were input into the cervical spine model T1 as prescribed motion constraints (Appendix A, Fig. A2—Electronic Supplementary Material). The model T1 was constrained in all other directions, and the head was unconstrained. Muscle activation was included for these validation cases to mimic the behavior of volunteers. The headrest used in the test was described as a stiff backing plate covered with 4 cm of foam, attached to a rigid frame with four coil springs of a specified preload and stiffness.[7] The mass of the headrest and dimensions were also specified. To model the headrest, the average sled *X*-acceleration was applied to the frame attachment points of the springs (Appendix A, Figs. A2, A3—Electronic Supplementary Material). The headrest material properties were nonlinear viscoelastic based on automotive seat cushion material, and the stiff wood backing was assumed to be pine with orthotropic elastic material properties.[5,15]

For higher severity rear impacts, it was necessary to validate against PMHS experiments. Deng[8] performed a series of 26 rear impacts on six whole body PMHS at delta velocities ranging from 5 to 15.5 kph, and accelerations from 5g to 9.9g. These experiments involved a PMHS seated in a custom seat, without a headrest, and being accelerated from rest using a pneumatic cylinder. Similar to the volunteer validation case, the experimental T1 *X*-acceleration, *Z*-acceleration, and *Y*-rotation from a 7g peak acceleration run were input into the model as prescribed motion (Appendix A, Fig. A4—Electronic Supplementary Material). However, muscle activation was not included in the validation of these tests to mimic the behavior of cadavers.

Following rear impact validation, the CL strains were computed for the 4g and 7g validation cases, but muscle activation was used for the 7g rear impact to model *in vivo* strains. The CL distractions were measured at the anterior and posterior aspects of the joint and then divided by anatomical ligament lengths reported in literature.[39] To define an impact threshold for CL injury, the model with active musculature was then exposed to a 10g rear impact, with T1 inputs from the PMHS test performed by Deng[8] (Appendix A, Fig. A5—Electronic Supplementary Material). This 10g rear impact was not suitable for validation because only three tests were performed at similar impact severities. The T1 *X*-acceleration from the 10g rear impact was then scaled to mimic impacts of 12g, 14g, and 16g severity. CL injury was defined as strain that entered the range of sub-catastrophic failure reported for human CL ligaments, 35–65% strain.[49,59] This injury criterion was chosen because sub-catastrophic ligament failure has been shown to relate to prolonged pain response in animal models.[29,30,45] The CL strains predicted by the model during rear impact were compared for passive and active musculature to determine the influence of no muscle activation in the cadaver studies. Finally, to determine the sensitivity of the CL strains predicted by the model to muscle activation timing, the model was run with muscle activation ±15 ms of the 74 ms currently implemented. The simulations performed are summarized with the relevant values and references in Table 2.

TABLE 2. Summary of model simulations.

Simulation	Impact severity	Muscle activation	Description	Reference
Model validation	4g	Yes	Volunteer global kinematic validation	7
	7g	No	Cadaver global kinematic validation	8
	8g	No	Cadaver bench-top local tissue validation	22,37,43
Injury threshold determination	10g	Yes	Increasing impact severities to determine threshold for CL injury	CL strain threshold[49]
	12g	Yes		
	14g	Yes		
	16g	Yes		
Muscle activation influence	4g	No	Model with and without muscle activation to determine its influence	–
	7g	Yes		
	10g	No		

RESULTS

The predicted ligament strains in the model with passive muscles during an 8g rear impact (Fig. 2) were compared to the bench-top cadaver results by Ivancic *et al.*[22] and Pearson *et al.*[43] for the ALL and CL, respectively. Four out the five ALL strains predicted by the model were within one standard deviation of the average experimental results (Fig. 3). The CL strains predicted by the model were within one standard deviation of the average results at all the spinal levels measured (Fig. 3). The peak shear strains measured in the model during an 8g rear impact were representative of the experimental results by Panjabi *et al.*[37] at every spinal level except for C5-C6 (Fig. 3).

When compared to volunteer data, the global kinematics of the model with muscle activation were a good fit to the volunteer displacement corridors (Fig. 4) presented by Hynd *et al.*[17] based on 4g rear impacts performed by Davidsson *et al.*[7] The head CG displacement relative to the sled in the posterior direction remained within the mean ± 1 SD response corridor for the duration of the impact. The head CG posterior translation relative to the T1 and the occipital condyle superior displacement both slightly deviated from the corridors beginning at ~140 ms. This timing coincides with the time that the head begins to over extend.

The response of the model without muscle activation for a 7g rear impact was compared to the experimental results from Deng[8] for rear impacts between 6g and 8g. The rotation of the head in the model initiated in the same manner and at the same time as the experiments, and the slope was representative, but the peak extension exceeded the experimental scatter by 6° (Fig. 5). The experimental head CG anterior acceleration displayed wide variation, but the response of the model remained within the experimental scatter (Fig. 5). In the inferior–superior direction, the response of the model displayed a similar shape and a moderate fit compared to the PMHS response, but the model was generally below the experimental curves and exhibited some higher frequency oscillations (Fig. 5).

Following model validation, the CL strains were predicted for increasing impact severities incorporating muscle activation and were compared to published sub-catastrophic failure values (Fig. 6). The CL strains measured increased with increasing impact severity. No injury was predicted by the model for impacts under 10g, and at 14g the C0-C1 CL strain exceeded the 35% threshold. At a 16g rear impact, the CL strain

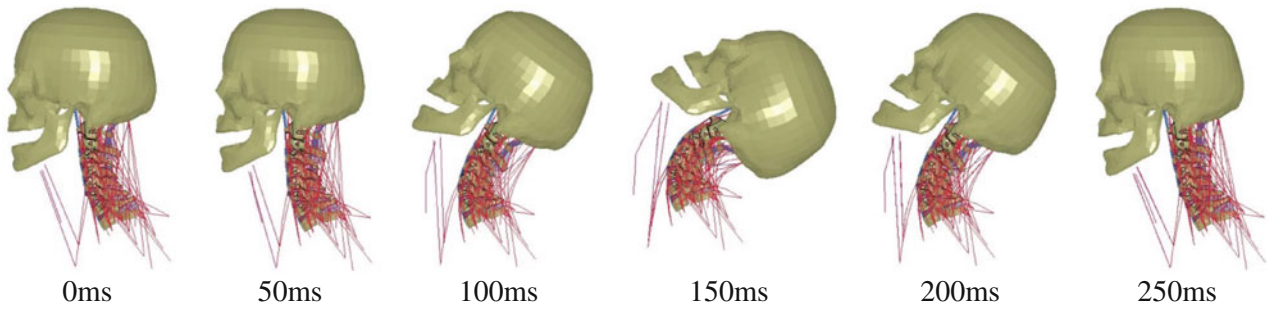

FIGURE 2. Time-lapse images of the model during an 8g rear impact.

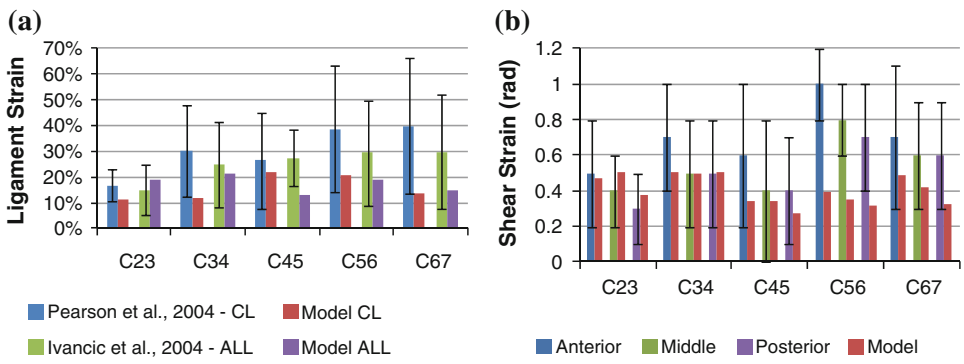

FIGURE 3. The peak ligament strains (a) and disc shear strains (b)[37] in the model during an 8g rear impact compared to experimental results (mean ± 1 SD). Anterior, middle, and posterior refer to the location were the shear strain measurement was taken.

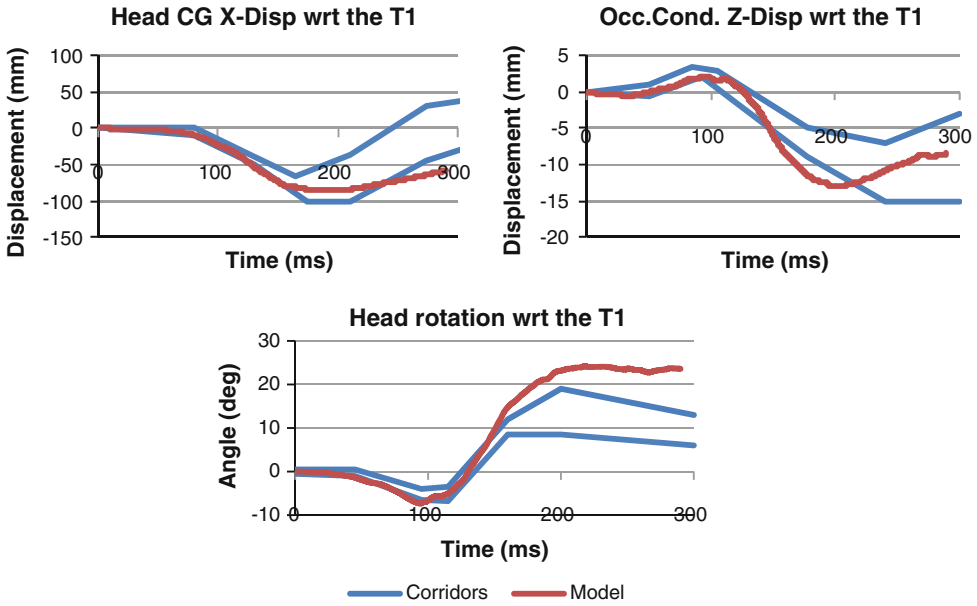

FIGURE 4. The head CG response of the model to a 4g rear impact. A negative X displacement, positive Z displacement, and positive angle imply relative posterior motion, superior motion, and extension, respectively.

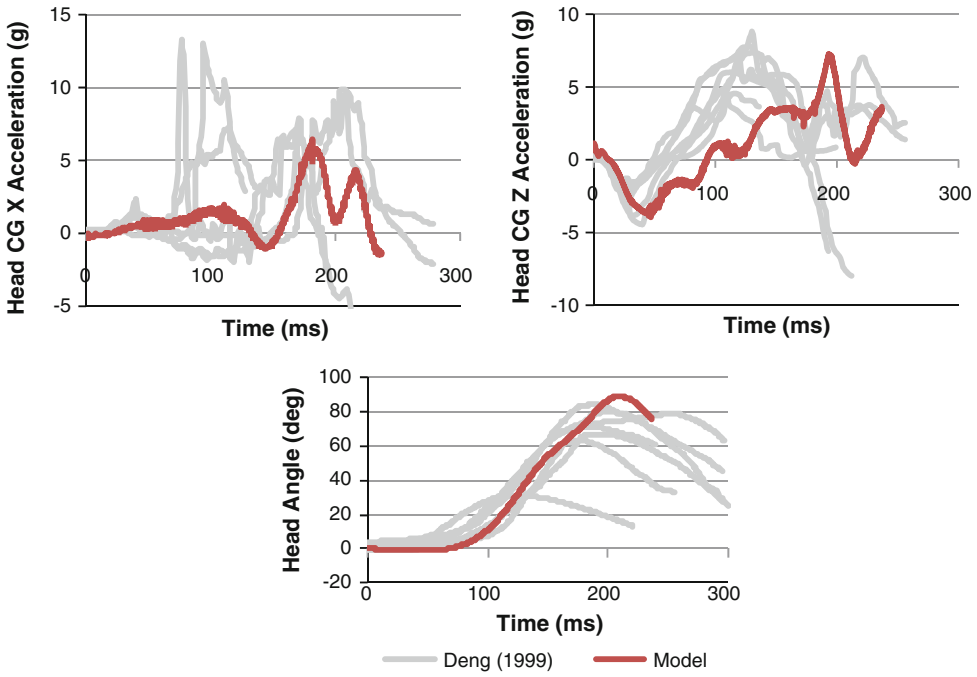

FIGURE 5. Global kinematic response of the model during a 7g rear impact. Muscle activation not utilized for direct comparison with cadavers.

entered the sub-traumatic failure region at the C0-C1, C1-C2, and C7-T1 joints.

Including muscle activation for the 7g rear impact validation case (Fig. 7) reduced the peak head extension by 17° and resulted in a broader lower peak response for the head anterior acceleration. The CL strains predicted by the model with passive muscles were generally higher than the same loading with active muscles, excluding the C4-C5 CL (Fig. 8). The peak CL strain increases were 4.4, 15.3, and 12.5% for the 4g, 7g, and 10g rear impacts, respectively. They occurred at the C1-C2, C0-C1, and C7-T1 joints, and the corresponding percent differences were 69, 55, and 37%, respectively. The activation scheme implemented

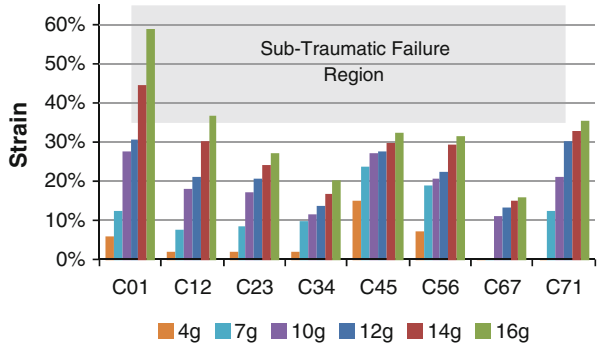

FIGURE 6. Capsular ligament strains predicted by the model during increasing rear impact severities.

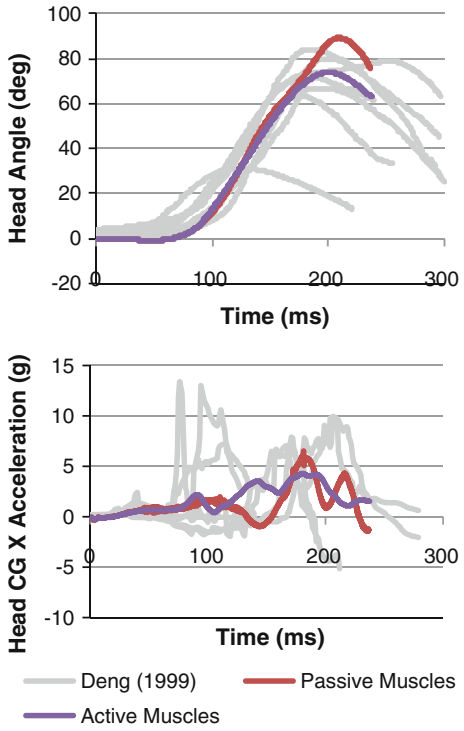

FIGURE 7. Comparing the global kinematic response of the head for active and passive muscles during a 7g rear impact.

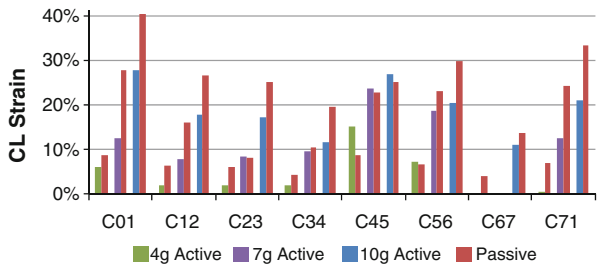

FIGURE 8. Comparing CL strains for active and passive muscles during 4g, 7g, and 10g rear impacts.

in the model activated the muscles 74 ms after the initiation of impact, and it was found that altering this value by ±15 ms during a 7g rear impact led to a maximum CL strain change of 3.3% between the 59 ms activation and the 89 ms activation at the C0-C1 spinal level. This indicates an activation time within ±15 ms does not greatly influence the CL strain predicted by the model.

DISCUSSION AND CONCLUSIONS

A detailed cervical spine model of a 50th percentile male was validated in rear impact, using both local tissue response and head kinematics. The model was previously validated at the segment level and for a variety of frontal impact crash scenarios. The focus of the model construction was accurate geometry and the best available tissue level material properties, and no aspect of the model was modified to improve the fit to a specific data set. The model was then used to investigate CL strain during increasing severity rear impacts. Finally, the influence of muscle activation on CL strain during rear impact was investigated.

The rear impact kinematic response of the model was generally a good fit to the volunteer data at 4g and the cadaver tests performed at 7g. For both impact severities, the head was predicted to over-rotate in extension. Two aspects of the model that could contribute to increased extension of the head are the distance between adjacent articular surfaces and the uncovertebral joints. When the articular surfaces of the facet joint come into contact, they provide resistance for further extension. Unfortunately, there was no data available for the distance between articular surfaces or detailed posture for these impact cases. The model geometry does not include uncovertebral joints, despite biomechanical evidence that has shown transecting these joints decreases motion segment stiffness in extension by up to 36%.[26] These joints have not been explicitly incorporated into detailed models likely due to the small size of the protuberance compared to the vertebral body, making it difficult to measure in detail with the imaging modalities used to create geometry for numerical models.

Oscillatory response was noted in the Z-acceleration and to a lesser extent the *X*-acceleration of the head during the 7g impact, compared to the experimental data. This difference was attributed to a lack of damping in the model, and the exclusion of some soft tissues in the neck such as the skin, fat, digestive tract, and airways. However, this is considered to be secondary to the overall response of the model. The kinematic validation of the model relied on a qualitative assessment of the response compared to the cadaver and volunteer data. Previously, a more quantitative measure was used that compared the size, phase shift, and shape cross-correlation of the model response to the average experimental results (CORA

software, Partnership for Dummy Technology and Biomechanics, Ingolstadt, Germany).[13,42] However, this method could not be used in the current study since a meaningful average experimental response and corridors could not be defined for the cadaver data. This was due to the large variation in response between the different tests, and some of the cadaver accelerometer traces were not provided for the entire impact duration. It should be noted that with a quantitative approach, there are still several qualitative decisions to be made to define the limits that constitute a good result for the different measures and the weighting of the measures into a final score.

For the local tissue validation in rear impact, the ALL and CL strains predicted by the model were typically within one standard deviation of the experimental data average response. The disc shear strains in the model during rear impact were accurate at all cervical levels except C5-C6, which was on average of 0.48 radians below the mean experimental results. There was no apparent reason that the shear strain at the C5-C6 level in the model should be lower, given that these strains were similar to the vertebral levels above and below this location. Also, there was no clear explanation why the cadaver model consistently predicted high shear strains at the C5-C6 level for each of the three positions measured. One possible explanation is that the initial posture of the cadaver model and the numerical model was different, causing the spinous process contact to occur at different times. Spinous process contact limits intervertebral extension and transfers load into adjacent vertebrae.

Injury to the facet joints is the most strongly supported mechanism or contributor to pain from whiplash injury, based on the wealth of biomechanical and clinical evidence in this area. Injury prediction in the model was based on a CL strain injury threshold of 35% for all spinal levels corresponding to sub-catastrophic failure. However, the actual 35% threshold value was determined from middle and lower cervical spine testing and does not include upper cervical spine CL results. It is known that the CL at C0-C1 and C1-C2 has different geometric and mechanical properties from the CL of the middle and lower cervical spine. For example, literature has reported the deformation at failure of the CL from C2 to T1 ranging from 6.8 to 10.2 mm and 9.3 to 11.4 mm for the C0-C1 and C1-C2 CL.[34,64] Unfortunately, data for the sub-catastrophic failure of the C0-C1 and C1-C2 CL were not available. Considering the higher failure deflection for these ligaments, it is likely the sub-catastrophic strain would be higher, and the 35% strain injury threshold is a conservative injury criterion at these joints.

A limitation of this study is the method of strain calculation for the ligaments, where strains were calculated as the joint distraction divided by the neutral ligament length as defined by in the related literature. The researchers that defined a CL injury threshold of 35% used a high-speed camera to measure the distortion of a grid of markers and calculated principal strain.[49] These localized strains could be predicted by replacing the 1D elements currently used to model the ligaments with shell elements, but most of the available ligament characterization studies report their results in force vs. displacement which is only compatible with strain based on distraction and original length, as predicted by the 1D elements. It is expected that using distraction over neutral length will result in lower strain values compared to local principal strain which could be higher.

Using the 35% strain injury threshold criterion, the model predicted CL injury at the C0-C1 level during a 14g rear impact, in agreement with available epidemiological data. In a study of 28 instrumented real-life accidents with 38 occupants, 20 occupants had short-term consequences at less than 10g, and two had long-term consequences at 13g and 15g.[27] In another study of 66 real-life accidents, 13 of the 15 people that sustained neck injuries for longer than a month experienced a rear impact of greater than 9g.[28] The numerical model did not predict any injuries during a 7g rear impact (delta V of 7.5 mph), which is in agreement with volunteer impact tests that have been performed up to 6.8 mph delta V without mild symptoms (defined as lasting longer than 4 days).[33] The location of predicted CL injury (C0-C1) was in the upper cervical spine, while clinical findings show injury in the C2-C3 or C5-C6 spinal levels. The difference may be due to the initial posture of the model and the assumed sub-catastrophic failure threshold as noted above.

Sundararajan *et al.*[53] exposed four female cadavers to 12 rear impacts (10.7g–11.6g severity) and calculated CL distraction using cineradiography to capture the motion of implanted spheres. The distractions measured by this group were converted to strain by dividing by the neutral ligament lengths used in this work and then compared to CL strain measured in the model during a 12g rear impact (Table 3). The strains

TABLE 3. Comparison of CL strains measured in the model and in a full cadaver sled test.[53]

Spinal level	Model at 12g (%)	Full body cadaver at ~11g Mean ± SD
C1-C2	19.5	31.9 ± 13.7
C2-C3	19	31.7 ± 27.9
C3-C4	12.9	26.3 ± 9.7
C4-C5	27.8	31.4 ± 11.8
C5-C6	22.7	21.5 ± 6.1
C6-C7	13.1	27.9 ± 7.2

predicted by the model agreed well with the cadaver study at the C4-C5 and C5-C6 spinal levels, but the model under predicted CL strain at the other spinal levels, with the difference attributed to the musculature It should be noted that both the cadaver study and the model showed the highest CL strain in the upper cervical spine, C1-C2 for the experiments and C0-C1 for the model.

This is the first study to show the influence of muscle activation on ligament strains in rear impact. Muscle activation was shown to reduce peak strains of the CL in rear impact, with the exception of the C4-C5 joint. Muscle activation should be expected to reduce ligament strains because the muscles stiffen the response of the neck and reduce joint distractions. It is also possible that, due to the increased strength of the extensor muscles in comparison to the flexor muscles, muscle activation could cause increased CL strain, as seen at the C4-C5 joint. Including muscle activation led to peak CL strain reductions of 4, 15, and 12% for rear impacts of 4g, 7g, and 10g, respectively. The significance of these results is that the strains predicted by bench-top cadaver sled tests may be high because they lack a representation of active musculature. Authors of these cadaver cervical spine models have published CL strains exceeding the 35% threshold for sub-traumatic failure at 5g and 10.5g.[36,43] These impact severities were low considering the 9g to 15g impact severities for injury predicted in this work and published by Krafft *et al*. in real-life crashes.

The CL strain response of the model was shown to be relatively insensitive to muscle activation timing of 59–89 ms for a 7g rear impact. This range of muscle activation timing covers the range of 60–79 ms from EMG measurements of volunteers in rear impact.[35,48,50,54]

Although this cervical spine model has been developed with the goal of predicted neck injury in automotive impacts, there are other potential applications including sport and military, in which the model could be used to understand injury mechanisms and design preventative measures. The model should be versatile because it has been validated across a range of loadings from quasi-static physiologic loading up to automotive impacts in various directions. In the case of chronic or repetitive loading, there is a great deal of development that would be required before suitable injury tolerances could be used with the model.

This study demonstrated that a cervical spine model can be developed at the tissue level and provide accurate biofidelic kinematic and local tissue response, leading to injury prediction in automotive crash scenarios. Importantly, this model bridges low severity impact volunteer data including active musculature, and the available higher severity PMHS impact data with no muscle activity available in the literature. Future studies will include evaluation of the model in out of position (head turned) rear impacts, which have been shown to increase the risk of CL injuries.[59]

ELECTRONIC SUPPLEMENTARY MATERIAL

The online version of this article (doi:10.1007/s10439-011-0315-4) contains supplementary material, which is available to authorized users.

ACKNOWLEDGMENTS

The authors gratefully acknowledge support from the Global Human Body Models Consortium.

REFERENCES

[1]Barnsley, L., S. M. Lord, B. J. Wallis, and N. Bogduk. The prevalence of chronic cervical zygapophysial joint pain after whiplash. *Spine* 20(1):20–26, 1995.
[2]Berglund, A., L. Alfredsson, I. Jensen, L. Bodin, and A. Nygren. Occupant- and crash-related factors associated with the risk of whiplash injury. *Ann. Epidemiol.* 13(1):66–72, 2003.
[3]Brault, J. R., G. P. Siegmund, and J. B. Wheeler. Cervical muscle response during whiplash: evidence of a lengthening muscle contraction. *Clin. Biomech.* 15(6):426–435, 2000.
[4]Brolin, K., P. Halldin, and I. Leijonhufvud. The effect of muscle activation on neck response. *Traffic Inj. Prev.* 6(1):67–76, 2005.
[5]Cambell, B., and D. S. Cronin. High rate characterization of automotive seat foams. In: Proceedings of SEM, 2007.
[6]Chazal, J., A. Tanguy, M. Bourges, G. Gaurel, G. Escande, M. Guillot, *et al*. Biomechanical properties of spinal ligaments and a histological study of the supraspinal ligament in traction. *J. Biomech.* 18(3):167–176, 1985.
[7]Davidsson, J., C. Deutscher, W. Hell, A. Linder, P. Lovsund, M. Svensson, *et al*. Human volunteer kinematics in rear-end sled collisions. In: Proceedings of IRCOBI, Goteborg, 1998, pp. 289–301.
[8]Deng, B. Kinematics of human cadaver cervical spine during low speed rear end impacts. PhD Thesis, Wayne State University, 1999.
[9]Deng, B., P. C. Begeman, K. H. Yang, S. Tashman, and A. I. King. Kinematics of human cadaver cervical spine during low speed rear-end impacts. *Stapp Car Crash J.* 44:171–188, 2000.
[10]Dibb, A. T., R. W. Nightingale, J. F. Luck, V. C. Chancey, L. E. Fronheiser, B. S. Myers, *et al*. Tension and combined tension-extension structural response and tolerance properties of the human male ligamentous cervical spine. *J. Biomech. Eng.* 131(8):081008, 2009.
[11]DiSilvestro, M. R., and J. K. Suh. A cross-validation of the biphasic poroviscoelastic model of articular cartilage in unconfined compression, indentation, and confined compression. *J. Biomech.* 34(4):519–525, 2001.
[12]Galasko, C. S., P. M. Murray, M. Pitcher, H. Chambers, S. Mansfield, M. Madden, *et al*. Neck sprains after road

traffic accidents: a modern epidemic. *Injury* 24(3):155–157, 1993.
[13]Gehre, C., H. Gades, and P. Wernicke. Objective rating of signals using test and simulation responses. In: Enhanced Safety of Vehicles, Stuttgart, Germany, 2009.
[14]Gilad, I., and M. Nissan. A study of vertebra and disc geometric relations of the human cervical and lumbar spine. *Spine* 11(2):154–157, 1986.
[15]Green, D. W., J. E. Winandy, and D. E. Kretschmann. Mechanical Properties of Wood. Madison, WI: U.S. Department of Agriculture, 1999.
[16]Happee, R. Inverse dynamic optimization including muscular dynamics, a new simulation method applied to goal directed movements. *J. Biomech.* 27(7):953–960, 1994.
[17]Hynd, D., M. Svensson, X. Trosseille, M. van Ratingen, and J. Davidsson. Dummy requirements and injury criteria for a low-speed rear impact whiplash dummy. European Enhanced Vehicle-Safety Committee, EEVC WG12 #505A, 2007.
[18]Iatridis, J. C., L. A. Setton, M. Weidenbaum, and V. C. Mow. The viscoelastic behavior of the non-degenerate human lumbar nucleus pulposus in shear. *J. Biomech.* 30(10):1005–1013, 1997.
[19]Ito, S., P. C. Ivancic, A. M. Pearson, Y. Tominaga, S. E. Gimenez, W. Rubin, *et al*. Cervical intervertebral disc injury during simulated frontal impact. *Eur. Spine J.* 14(4):356–365, 2005.
[20]Ivancic, P. C., S. Ito, Y. Tominaga, W. Rubin, M. P. Coe, A. B. Ndu, *et al*. Whiplash causes increased laxity of cervical capsular ligament. *Clin. Biomech.* 23(2):159–165, 2008.
[21]Ivancic, P. C., M. M. Panjabi, S. Ito, P. A. Cripton, and J. L. Wang. Biofidelic whole cervical spine model with muscle force replication for whiplash simulation. *Eur. Spine J.* 14(4):346–355, 2005.
[22]Ivancic, P. C., A. M. Pearson, M. M. Panjabi, and S. Ito. Injury of the anterior longitudinal ligament during whiplash simulation. *Eur. Spine J.* 13(1):61–68, 2004.
[23]Kleinberger, M. Importance of head restraint position on whiplash injury. In: Frontiers in Whiplash Trauma: Clinical & Biomechanical, edited by N. Yoganandan, and F. A. Pintar. Amsterdam: ISO Press, 2000, pp. 477–490.
[24]Klinich, K. D., S. M. Ebert, C. A. Van Ee, C. A. Flannagan, M. Prasad, M. P. Reed, *et al*. Cervical spine geometry in the automotive seated posture: variations with age, stature, and gender. *Stapp Car Crash J.* 48:301, 2004.
[25]Knaub, K. E., and B. S. Myers. Project F.2(c): Cervical Spine Muscles. Durham, NC: Duke University, 1998.
[26]Kotani, Y., P. S. McNulty, K. Abumi, B. W. Cunningham, K. Kaneda, P. C. McAfee, *et al*. The role of anteromedial foraminotomy and the uncovertebral joints in the stability of the cervical spine. A biomechanical study. *Spine* 23(14):1559–1565, 1998.
[27]Krafft, M., A. Kullgren, C. Tingvallb, O. Boströmc, and R. Fredrikssonc. How crash severity in rear impacts influences short- and long-term consequences to the neck. *Accid. Anal. Prev.* 32(2):187–195, 2000.
[28]Krafft, M., A. Kullgren, A. Ydenius, and C. Tingvall. Influence of crash pulse characteristics on whiplash associated disorders in rear impacts—crash recording in real life crashes. *Traffic Inj. Prev.* 3(2):141–149, 2002.
[29]Lee, K. E., A. N. Franklin, M. B. Davis, and B. A. Winkelstein. Tensile cervical facet capsule ligament mechanics: failure and subfailure responses in the rat. *J. Biomech.* 39(7):1256–1264, 2006.
[30]Lee, K. E., J. H. Thinnes, D. S. Gokhin, and B. A. Winkelstein. A novel rodent neck pain model of facet-mediated behavioral hypersensitivity: implications for persistent pain and whiplash injury. *J. Neurosci. Methods* 137(2):151–159, 2004.
[31]Lord, S. M., L. Barnsley, B. J. Wallis, and N. Bogduk. Chronic cervical zygapophysial joint pain after whiplash. A placebo-controlled prevalence study. *Spine* 21(15):1737–1745, 1996.
[32]Lu, Y., C. Chen, S. Kallakuri, A. Patwardhan, and J. M. Cavanaugh. Neurophysiological and biomechanical characterization of goat cervical facet joint capsules. *J. Orthop. Res.* 23(4):779–787, 2005.
[33]McConnell, W. E., R. P. Howard, J. V. Poppel, R. Krause, H. M. Guzman, J. B. Bomar, *et al*. Human head and neck kinematics after low velocity rear-end impacts understanding whiplash. *Stapp Car Crash J.* 39:215–238, 1995.
[34]Myklebust, J. B., F. Pintar, N. Yoganandan, J. F. Cusick, D. Maiman, T. J. Myers, *et al*. Tensile strength of spinal ligaments. *Spine* 13(5):526–531, 1988.
[35]Ono, K., K. Kaneoka, A. Wittek, and J. Kajzer. Cervical injury mechanism based on the analysis of human cervical vertebral motion and head-neck-torso kinematics during low-speed rear impacts. *Stapp Car Crash J.* 41:339–356, 1997.
[36]Panjabi, M. M., J. Cholewicki, K. Nibu, J. Grauer, and M. Vahldiek. Capsular ligament stretches during in vitro whiplash simulations. *J. Spinal Disord.* 11(3):227–232, 1998.
[37]Panjabi, M. M., S. Ito, A. M. Pearson, and P. C. Ivancic. Injury mechanisms of the cervical intervertebral disc during simulated whiplash. *Spine* 29(11):1217–1225, 2004.
[38]Panjabi, M. M., T. Oxland, K. Takata, V. Goel, J. Duranceau, M. Krag, *et al*. Articular facets of the human spine. Quantitative three-dimensional anatomy. *Spine* 18(10):1298–1310, 1993.
[39]Panjabi, M. M., A. M. Pearson, S. Ito, P. C. Ivancic, S. E. Gimenez, Y. Tominaga, *et al*. Cervical spine ligament injury during simulated frontal impact. *Spine* 29(21):2395–2403, 2004.
[40]Panzer, M. B. Numerical modelling of the human cervical spine in frontal impact. M.A.Sc Thesis, University of Waterloo, 2006.
[41]Panzer, M. B., and D. S. Cronin. C4-C5 segment finite element model development, validation, and load-sharing investigation. *J. Biomech.* 42(4):480–490, 2009.
[42]Panzer, M. B., J. B. Fice, and D. S. Cronin. Cervical spine response in frontal crash. Medical Engineering and Physics, 2010 (under review #MEP-D-10-00414).
[43]Pearson, A. M., P. C. Ivancic, S. Ito, and M. M. Panjabi. Facet joint kinematics and injury mechanisms during simulated whiplash. *Spine* 29(4):390–397, 2004.
[44]Quinlan, K. P., J. L. Annest, B. Myers, G. Ryan, and H. Hill. Neck strains and sprains among motor vehicle occupants—United States, 2000. *Accid. Anal. Prev.* 36(1):21–27, 2004.
[45]Quinn, K. P., and B. A. Winkelstein. Cervical facet capsular ligament yield defines the threshold for injury and persistent joint-mediated neck pain. *J. Biomech.* 40(10):2299–2306, 2007.
[46]Radanov, B. P., M. Sturengger, and G. Di Stefano. Long-term outcome after whiplash injury: a 2-year follow-up considering features of injury mechanism and somatic, radiologic, and psychosocial findings. *Medicine* 74(5):281–297, 1995.

[47]Robbins, D. H. Anthropometric Specifications for Mid-Sized Male Dummy, Vol. 2. Ann Arbor, MI: NHTSA, University of Michigan Transportation Research Institute, 1983.
[48]Roberts, A. K., D. Hynd, P. R. Dixon, O. Murphy, M. Magnusson, M. H. Pope, *et al*. Kinematics of the human spine in rear impact and the biofidelity of current dummies. In: IMechE Conference Proceedings, 2002, pp. 227–246.
[49]Siegmund, G. P., B. S. Myers, M. B. Davis, H. F. Bohnet, and B. A. Winkelstein. Mechanical evidence of cervical facet capsule injury during whiplash: a cadaveric study using combined shear, compression, and extension loading. *Spine* 26(19):2095–2101, 2001.
[50]Siegmund, G. P., D. J. Sanderson, B. S. Myers, and J. T. Inglis. Rapid neck muscle adaptation alters the head kinematics of aware and unaware subjects undergoing multiple whiplash-like perturbations. *J. Biomech*. 36(4): 473–482, 2003.
[51]Siegmund, G. P., D. J. Sanderson, B. S. Myers, and J. T. Inglis. Awareness affects the response of human subjects exposed to a single whiplash-like perturbation. *Spine* 28(7): 671–679, 2003.
[52]Siegmund, G. P., B. A. Winkelstein, P. C. Ivancic, M. Y. Svensson, and A. Vasavada. The anatomy and biomechanics of acute and chronic whiplash injury. *Traffic Inj. Prev*. 10(2):101–112, 2009.
[53]Sundararajan, S., P. Prasad, C. K. Demetropoulos, S. Tashman, P. C. Begeman, K. H. Yang, *et al*. Effect of head-neck position on cervical facet stretch of post mortem human subjects during low speed rear end impacts. *Stapp Car Crash J*. 48:331–372, 2004.
[54]Szabo, T. J., and J. B. Welcher. Subject kinematics and electromyographic activity during low speed rear impacts. *Stapp Car Crash J*. 40:295–315, 1996.
[55]Thunnissen, J. G., J. S. Wismans, C. L. Ewing, and D. J. Thomas. Human volunteer head-neck response in frontal flexion: a new analysis. *Stapp Car Crash J*. 39:439–460, 1995.
[56]Van der Horst, M. J. Human head neck response in frontal, lateral and rear end impact. PhD Thesis, Eindhoven University of Technology, 2002.
[57]Van Ee, C. A., A. L. Chasse, and B. S. Myers. Quantifying skeletal muscle properties in cadaveric test specimens: effects of mechanical loading, postmortem time, and freezer storage. *J. Biomech. Eng*. 122(1):9–14, 2000.
[58]Walker, L., E. Harris, and U. Pontius. Mass, volume, center of mass, and mass moment of inertia of head and head and neck of human body. *Stapp Car Crash J*. 17:525–537, 1973.
[59]Winkelstein, B. A., R. W. Nightingale, W. J. Richardson, and B. S. Myers. The cervical facet capsule and its role in whiplash injury: a biomechanical investigation. *Spine* 25(10): 1238–1246, 2000.
[60]Winters, J. How detailed should muscle models be to understand multi-joint movement coordination? *Hum. Mov. Sci*. 14(4–5):401–442, 1995.
[61]Winters, J. M., and L. Stark. Estimated mechanical properties of synergistic muscles involved in movements of a variety of human joints. *J. Biomech*. 21(12):1027–1041, 1988.
[62]Winters, J., and S. L. Woo. Multiple Muscle Systems: Biomechanics and Movement Organization. New York: Springer, 1990.
[63]Yang, K. H., and V. L. Kish. Compressibility measurement of human intervertebral nucleus pulposus. *J. Biomech*. 21(10):1–3, 1988.
[64]Yoganandan, N., S. Kumaresan, and F. A. Pintar. Biomechanics of the cervical spine Part 2. Cervical spine soft tissue responses and biomechanical modeling. *Clin. Biomech*. 16(1):1–27, 2001.
[65]Yoganandan, N., F. Pintar, J. Butler, J. Reinartz, A. Sances, S. J. Larson, *et al*. Dynamic response of human cervical spine ligaments. *Spine* 14(10):1102–1110, 1989.
[66]Zuby, D. S., and A. K. Lund. Preventing minor neck injuries in rear crashes—forty years of progress. *J. Occup. Environ. Med*. 52(4):428–433, 2010.