FOLLOWER LOAD INFLUENCES THE KINEMATICS AND KINETICS OF CERVICAL SPINE BUCKLINGDURING SIMULATED HEAD FIRST IMPACT: AN EX VIVO STUDY

Christopher R. Dennison[1,2,4], Amy Saari[1,2,4], Qingan Zhu[1,2,4] , Timothy S. Nelson[1,2,4], , Philip Morley[1,2,4], Eyal Itshayek[1,3,4,5], Thomas R. Oxland[1,2,3,4] and Peter A. Cripton[1,2,3,4]
[1]Orthopaedic and Injury Biomechanics Group; [2]Department of Mechanical Engineering [3]Department of Orthopaedics; [4]International Collaboration on Repair Discoveries University of British Columbia Vancouver, Canada; [5]Department of Neurosurgery. Hadassah - Hebrew University Hospital. Jerusalem. Israel

ABSTRACT

Current understanding of the biomechanics of cervical spine injuries in head first impact is based on decades of epidemiology, mathematical models and on ex vivo experimental studies. Recent mathematical modeling suggests that muscle activation and muscle forces are relevant to injury risk in head first impact. It is also known that muscle forces are central to the overall stability of the cervical spine. Despite this knowledge, the vast majority of ex vivo head first impact models do not incorporate musculature. We hypothesize that the simulation of the stabilizing mechanisms of musculature during head first osteoligamentous cervical spine experiments will have considerable influence on the resulting kinematics and injury mechanisms. We simulated head first impact using cadaveric cervical spines with surrogate heads (n=12). Six spines were instrumented with a follower load to simulate in vivo compressive muscle forces, while six were not. The principal finding was that mechanical coupling between the head and the base of the cervical spine (T1) was increased in specimens with follower load. Increased coupling was indicated by reduced time between head impact and peak neck reaction force (and spine buckling) and reduced vertebral rotations, during buckling, in specimens with follower load. These preliminary results suggest that simulating follower load that may be similar to in vivo muscle forces results in significantly different buckling behaviour, and therefore potentially different injury mechanics occur in vivo than in many biomechanical tests where musculature is not simulated.

INTRODUCTION

Cervical spine and spinal cord injuries occur as a result of axial impact and spine compression in motor vehicle rollovers [1-6], falls [7, 8], and several sports. Worldwide, the incidence of spinal cord injuries is estimated to be 10.4 to 83 per million [9]. These injuries result in significant financial cost to the injured and to society. The effects on quality of life are catastrophic and profound for the injured person. Previous epidemiology indicates the majority of survivors of spinal cord injury have associated injury in the osteoligamentous structures of the cervical spine, often typical of compression of the cervical spine, such as fractures and dislocation of vertebra and facet joints, respectively, in the region C4-C6 [2, 10-12].

Current understanding of the biomechanics of these injuries is based on decades of epidemiology, mathematical models and on *ex vivo* experimental studies and on combinations of these approaches. Loading rates in axial impact experiments are considered to more accurately recreate both *in vivo* loading during head first impact and associated injury mechanisms relative to quasi-static experiments. Recent biomechanical studies use isolated head and

osteoligamentous cervical spine models, or cervical spines affixed to anthropomorphic heads [10, 13-16]. In these studies, neck musculature is removed allowing visual access to the ligamentous and bony structures of the cervical spine and, therefore, vertebra-level kinematics to be resolved throughout impact. The biomechanical "cost" associated with visual access to the osteoligamentous spine is loss of neck musculature and, therefore, any important biomechanical contributions that this musculature may have on the mechanics of injury. Mathematical models have also been applied to study the mechanics of the cervical spine and head [17] and show that both spine posture and degree of muscle activation considerably influence cervical spine injury risk. *In vivo*, neck musculature is primarily responsible for controlling neck posture, motion and stability. Despite these central roles that muscles play, very few *ex vivo* biomechanical investigations have simulated neck musculature.

Ex vivo, postural control of cadaveric head and neck complexes is achieved using externally applied forces on the head or vertebra [10, 18, 19]. For example, researchers at the Medical College of Wisconsin aligned cervical vertebra into a straight column, away from *in vivo* lordotic posture, prior to head first impact and documented clinically relevant spine fractures in the region C4-6 in several specimens [10]. While this technique is noteworthy because it created injuries consistent with real-world trauma [2, 12], it is limited because it did not simulate the lines of action or magnitudes of *in vivo* muscle force that are present *in vivo* to achieve the vulnerable spine posture that was tested.

The osteoligamentous cervical spine, without real or simulated musculature, has been shown to buckle at less than 10 N of quasi-static load [20]. As this is less than the weight of the head under static conditions, it is clear that neck musculature stabilizes the cervical column and increases the load at which buckling occurs. Patwardhan and colleagues have shown that the load-carrying capacity of the cervical spine increases with the application of follower load [21] (compressive load applied to the spine, with the line of action guided at each vertebral level to follow the curvature of the spine). In axial impact experiments with isolated head and osteoligamentous neck complexes, without musculature, Nightingale and colleagues have shown that the spine fails by both first order and higher order dynamic buckling modes [13].

We hypothesize that it is important to simulate the stabilizing mechanisms of musculature during head first osteoligamentous cervical spine experiments as this could both prevent the buckling cervical spine behavior documented by Nightingale and colleagues and increase the bio-fidelity of the model with respect to *in vivo* head-first impact. Our specific objectives were to document differences in the dynamic behavior, caused by head first impact, between cervical spines with and without simulated muscle forces. Differences in forces/moments (kinetic metrics), and vertebral motions throughout the impact (kinematic metrics) were documented for the *ex vivo* cervical spines.

METHODS

Twelve human cadaveric cervical spines (occiput to T2, Figure 1a and Figure 2), were obtained from research tissue banks and kept frozen prior to these experiments. Donor medical record information was examined and specimens with more than age-appropriate degeneration or with other spinal pathology were excluded. Each specimen was dissected free of muscle tissue while carefully preserving the osteoligamentous structures. The T1 and T2 vertebrae were embedded in dental stone such that the C4-C5 disc was horizontal in the spine's neutral posture and such that the C7-T1 intervertebral joint was unrestricted. Six of these specimens were instrumented with a

follower load [16, 21, 22] (FL group), as described below, while the remaining six specimens were not (NFL group).

In the FL group (2 male, 4 female; mean age = 72 years; range 65 to 84 years), a follower load consisting of tensioned bilateral cables (Figure 2) (22.7 lb test rating, Berkley Gorilla Tough, Spirit Lake, Iowa) passing through self-tapping guides screwed through the anterior cortex of the lateral masses of each cervical vertebrae (after removing the arch of bone protecting the vertebral artery) was added to simulate an overall resultant muscle force for the spine. The guides were located such that the tensioned cables passed through the approximate flexion-extension centre of rotation on each vertebra [23-25]. Cable guides were also embedded in the dental stone encasing T1-T2 (Figure 2) and these guides were used as anchors for the cables. A physiologically-reasonable follower load of 125 N (62.5 N each side) was applied by tensioning the bilateral cables against the anchors in the dental stone [16, 22, 26]. Tension was created by compression springs (Figure 2, compression rate of 9.5 N/mm) which were mounted to the surrogate head and that were attached to the cables [22]. The NFL group (3 male, 2 female; mean age = 79 years; range 73 to 87 years; age and sex of one specimen unknown) had neither guides nor cables. Four photo-reflective spherical markers mounted on pins were inserted into each vertebra (Figure 2). The markers allowed kinematic reconstruction of vertebral motions. All cervical spines were fixed to a biofidelic surrogate head (Figure 1a and Figure 2) which was mounted to the bony occiput [16].

Unconstrained head-first axial impact was simulated using a purpose built drop tower [16] (Figure 1a). The carriage weight approximates the upper torso weight of a 50^{th} percentile human male (~15 kg). The cervical spine specimens were mounted to the carriage in an inverted orientation such that when the carriage was released head-first impact occurred between a steel impact platen and the surrogate head. Once mounted, the spine-head complexes were oriented to nominally recreate resting posture (i.e. resting lordosis and no anteroposterior head rotation) using cables that break away upon impact and, therefore, do not influence the mechanics of the head-first impact. A drop height of 0.6 m was used to obtain an impact velocity of approximately 3 m/s, which has previously been established as the approximate tolerance for cervical spine injuries in head-first impacts [10, 11, 13]. A 6-axis load cell (MC3A, AMTI, Watertown, MA) was mounted caudal to T1/2 to measure reaction forces and moments. A 20kN uni-axial load cell (LC, Omega Engineering Inc., Stamford, CT, USA) was mounted between the steel impact platen and drop tower support structure and measured the axial force at the head.

Two high speed digital cameras (Phantom V9.0, Vision Research Inc.) were used to record the head first impact and, in particular, the motions of the photo-reflective markers at 1000 frames per second. Load cell voltage acquisition (146 kHz) and digital camera image recording were triggered and synchronized using built in facilities of the high speed cameras and camera control software.

Analog voltage data recorded from the load cells during the head first impact was digitally filtered in compliance with the SAE standard for impact testing: J211-1 [27].

The photo-reflective marker positions and position of the mount-cup, as recorded by the two high speed cameras, were post-processed to give three-dimensional marker and mount-cup positions relative the coordinate system shown in Figure 1a. The pre-impact position and inclination of the surrogate head were also calculated. Post-processing comprised two main stages: first, marker motions were calculated using commercially available software (TEMA, Image Systems AB, Sweden); and second, a series of calculations collectively referred to as direct linear transformation (DLT) [28] were implemented to obtain three-dimensional marker

motions and specific pre-impact head-to-neck alignment measurements including the head inclination relative to the Z-axis (Figure 1b) and the distance from the mount-cup center and head-mount center, $D_{c\text{-}c}$ (Figure 1b). To allow calculation of vertebral rotations in the sagittal plane, vectors were defined (in post processing) between vertebral markers at each vertebral level (shown in Figure 3). For each vertebra, vectors were defined between the markers that exhibited the least relative motion with respect to one another.

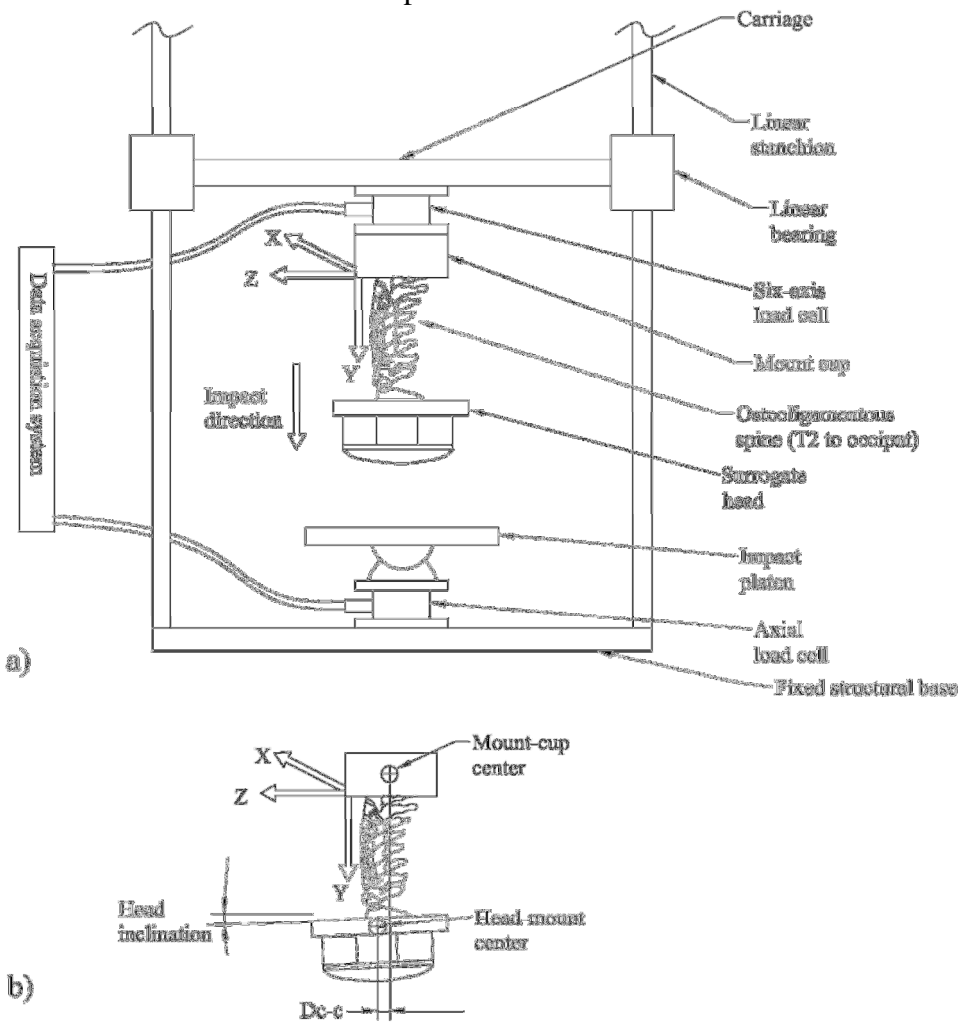

Figure 1: a) schematic of vertical drop-tower used to simulate axial head-first impact. The carriage translates along the Y-axis and is constrained in the X- and Z-axes by linear stanchions/bearings. Direction of translation is the Y-direction and impact occurs between the surrogate head and impact platen; b) schematic of cervical spine and surrogate head complex showing mount-cup center to head-mount center distance and head inclination.

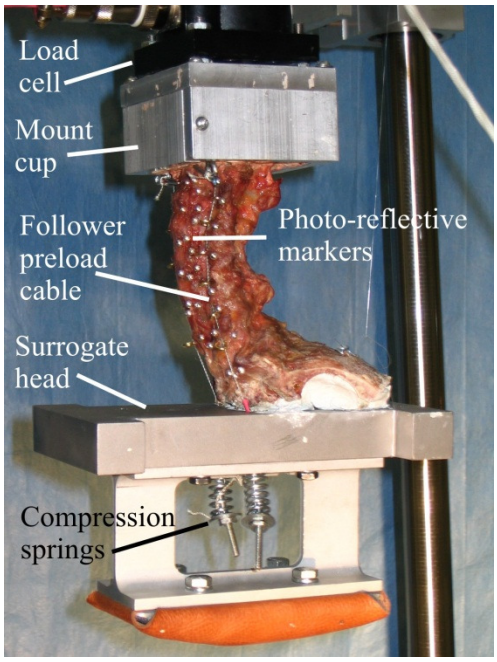

Figure 2: photograph of osteo-ligamentous cervical spine and surrogate head complex showing follower load and compression springs. The lordotic posture shown was typical of all specimens.

Several buckling metrics were calculated from the data measured by the two load cells (i.e. the kinetics) and vertebral marker motions (kinematics) including: impact speed; time to neck buckle (T_b); the vertebral levels at which the neck buckles; the total angular divergence at the levels at which the neck buckled; pre-buckle peak compressive neck Y-force (F_n); post-buckle peak neck moment (M_n); the time from head impact to peak compressive neck Y-force ($T_{peak\text{-}neck}$); and the impulse delivered to the mount-cup from head impact to buckle (I_b). The criteria to determine buckling and vertebral level of buckling were based solely on mechanical factors (i.e. kinematics and kinetics and not osteoligamentous injury); specifically: failure of the neck to support compressive force while C7 continued to translate toward the head *and* divergence of angular rotation across any two or three vertebral levels as indicated by opposing vertebral rotation direction that was greater than 2 degrees. T_b were determined using similar methods to those presented by Nightingale and colleagues for determining time to injury [13]. T_b was defined as the earliest time at which the compressive neck Y-force dropped to 2/3 of the first peak magnitude (while all other buckling criteria were also met). The impulse was calculated by integrating compressive neck Y-force from the time of head impact to buckle.

Means were calculated for the FL and NFL groups for several measured and calculated results. For comparison between the FL and NFL groups, Wilcoxon-Rank-Sum tests were used.

RESULTS

A summary of subject data, pre-impact head-neck alignment measurements, axial impact and buckling results are presented in Table 1. The mean pre-impact surrogate head inclination was -0.3 degrees for the NFL group and -0.1 degrees for the FL group with no significant difference between the two groups ($p = 0.70$). The mean distance between the mount-cup center and

surrogate head center, D_{c-c}, was 26.9 mm for the NFL group and 9.3 mm for the FL group and there was a significant difference between the distances of the FL and NFL group ($p = 0.009$).

Table 1: Specimen information; pre-impact alignment measurements; Impact force, moment, buckle time, time to peak neck force, and impulse; and vertebral divergence data.

		Selected pre-impact alignment measurements		Impact force/moment and time measurements						Vertebral divergence and levels	
Specimen group	Specimen no. (Age/Sex)	Head inclination (deg.)[1]	Mount-cup center to Head center distance (mm)[2]	Impact speed (m/s)	F_n, (kN)[3]	M_n, (Nm)[3]	T_b, (msec)[4]	$T_{peak-neck}$, (msec)[4]	I_b (Nsec)	Vertebral divergence (deg.) [levels]	Total divergence (deg.)
No follower load	H1062 (87/M)	0.674	-18.204	2.8	1.884	138.8	5.15	4.63	3.6	11.107 [C3/4]	11.1
	H1092 (75/F)	1.252	-28.063	2.7	0.7497	76.96	3.66	3.25	0.7	4.84 [C3/4] , 5.641 [C4/5]	10.5
	H1094 (77/M)	0.539	-30.628	2.8	3.05	139.2	4.81	4.25	6.0	15.746 [C3/4], 5.924 [C4/5]	21.7
	H1095 (73/F)	0.923	-13.504	2.8	1.356	132.5	3.73	3.05	2.0	8.400 [C3/4], 11.192 [C4/5]	19.6
	H1099 (79/M)	-3.255	-41.328	2.8	2.04	129.2	6.31	5.25	4.6	10.529 [C3/4], 12.516 [C4/5]	23.0
	H1101 (--/--)	-2.133	-29.409	2.6	2.383	139.4	5.69	4.58	6.3	22.91 [C3/4], 12.68 [C4/5]	35.6
	mean	-0.333	-26.856	2.8	1.9	126.0	4.9	4.2	3.9		20
Follower load	H1091 (72/M)	4.721	-7.273	3.0	0.8041	75.16	3.15	2.21	1.3	2.355 [C4/5]	2.4
	H1116 (84/F)	0.221	-13.084	2.8	1.317	91.52	2.4	1.12+	2.5	6.397 [C3/4]	6.4
	H1177 (65/F)	-0.747	1.730	3.1	0.9327	74.58	1.49	1.38	0.3	8.237 [C4/5]	8.2
	H1184 (67/M)	-0.913	-3.666	2.9	1.166	94.77	2.55	1.49+	2.0	6.0791 [C4/5]	6.1
	H1096 (68/F)	-2.500	-10.353	2.9	2.105	90.29	3.88	2.63	4.5	5.492 [C3/4]	5.5
	H1183 (71/F)	-1.324	-23.117	2.8	1.812	25.17	1.65	1.03+	2.2	2.120 [C2/3]	2.1
	mean	-0.090	-9.294	2.9	1.4	75.2	2.5	1.6	2.1		5

Notes:
1. inclination relative to Z axis, positive angle indicates flexion, negative indicates extension
2. negative indicates that head center is anterior to mount cup center
3. compressive force
4. positive CCW in figure 1
(+) Head force data not available, these times were estimated from high speed video and marker motions

The mean impact speed was 2.8 m/s in the NFL group and 2.9 m/s in the FL group (Table 1). There was no significant difference in the pre-buckle peak compressive neck forces, F_n, between the NFL and FL groups ($p = 0.24$) which had means of 1.9 kN and 1.4 kN, respectively. There was a significant difference in the post-buckle peak neck moment, M_n, between the NFL and FL groups ($p = 0.015$) which had means of 126.0 Nm and 75.2 Nm, respectively.

There were significant differences between the NFL and FL groups for both the time from head impact to peak pre-buckle compressive neck force, $T_{peak-neck}$ ($p = 0.004$), and the time from head impact to buckle, T_b ($p = 0.009$). The mean $T_{peak-neck}$ for the NFL group was 4.2 msec and for the FL group was 1.6 msec (Table 1). The mean T_b for the NFL group was 4.9 msec and for the FL group was 2.5 msec (Table 1). There was no significant difference in the impulse, I_b, from head impact to injury between the NFL group and FL group ($p = 0.200$), which had means of 3.9 Nsec and 2.1 Nsec, respectively.

In terms of vertebral divergence and buckling in the FL group, 3/6 of specimens buckled across C4/5 (example show in Figure 3), 2/6 across C3/4 and 1/6 across C2/3. For the NFL group, buckling in 5/6 specimens involved C4 rotation being nominally zero while C3 and C5 had negative and positive rotation (clockwise in Figure 1 is positive), respectively. The remaining NFL specimen buckled (opposing rotations) across C3/4. The FL specimens (avg. 5 degrees) had significantly smaller ($p=0.002$) rotations at buckle than the NFL (avg. 20 degrees). All specimens exhibited overall extension across the spine (Figure 3). We observed clinically relevant osteoligamentous injuries in all specimens and the reader is referred to our previous work for a partial injury dataset [16].

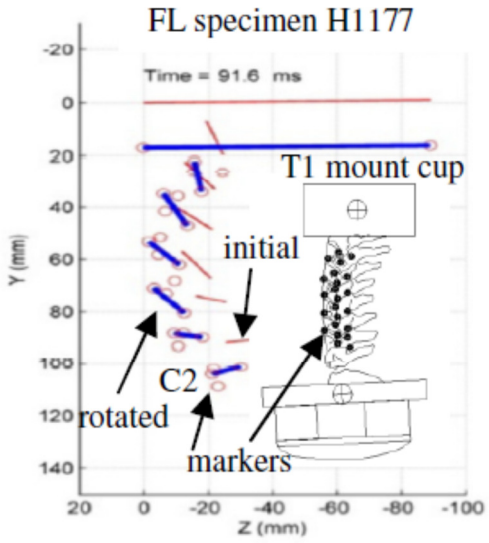

Figure 3: (inset) schematic showing pre-impact lordotic posture of cervical spine and surrogate head; red lines indicate initial pre-impact inclinations of vectors between vertebral markers while blue lines indicate rotated vertebra inclinations as impact progresses and show extension of cervical spine relative to initial posture; marker locations shown as red circles.

DISCUSSION

Follower load considerably altered the kinematics, kinetics and buckling of cadaveric cervical spines during head first axial impact. Overall the head was more closely coupled to the thorax and the buckling kinematics were constrained by follower load. Although there is a significant body of clinical and scientific literature for head first axial impact we are not aware of any studies that directly compare the dynamics and injuries of *ex vivo* cervical spines with and without follower loads.

Both T_b and $T_{peak\text{-}neck}$ were significantly lower in the FL group which indicates increased coupling between the head and T1 in specimens that were instrumented with compressive follower loads. This increased coupling is believed to be caused by increased spine stiffness as a result of the addition of follower load and associated compression of intervertebral discs. This finding is also consistent with comparisons drawn in previous work. For example, in parallel work by Saari et al. (2011) [16], there was a reduction in the time-lag between head impact and neck loading in specimens with follower loads (approximately 1msec) relative to previous work by Nightingale et al. (1993) [29] which considered cervical spines without follower load in similar impact configurations (lags of 1.5 msec to 2.1 msec). In this current work, the metallic surrogate head may have also contributed to the increase in stiffness of the specimen. However, the potential for axial compression of an *ex vivo* skull is negligible in comparison to that of the uncompressed cervical spine. Thus, the influence of replacing the head on T_b and $T_{peak\text{-}neck}$ is believed to be minimal.

We performed a limited test of the sensitivity of our calculations of statistical significance to changes in the definition of our buckling criterion and found that our calculations were not altered by changes in the buckling criterion. More specifically, we re-calculated T_b based on 80% and 50% of the peak neck force (both above and below the 67% criteria we

describe in the results) and re-calculated vertebral divergence and the other buckling metrics. While small changes in the buckling metric magnitudes were found to result, the calculations of statistical significance between the buckling metrics for the FL and NFL groups did not change.

There were no significant differences between the FL and NFL groups for both F_n and mount-cup impulse. This suggests that the addition of follower load does not affect the transfer of torso momentum to the cervical spine.

The significant differences in M_n between the FL and NFL groups could contradict our earlier suggestion that the addition of follower load does not affect torso to neck momentum transfer. However, we hypothesize that the relatively high M_n of the NFL group are caused primarily by significant differences in head to mount-cup alignment. While head inclination was approximately constant across all specimens, the NFL group had significantly larger $D_{c\text{-}c}$ ($p = 0.009$), which would tend to increase the moment-arm ($D_{c\text{-}c}$ in Figure 1b) of head forces relative to the six-axis load cell.

Previous work considering head first axial impact has focused on injury mechanisms and the effect of pre-injury conditions such as posture and impact surface parameters. In several of these studies the vertebral marker kinematics indicate a serpentine neck deformation, sometimes referred to as second-order buckling, response. It is unknown whether second-order buckling occurs in real life accidents. Their occurrence in *ex vivo* studies is often associated with complex injury patterns. One of the common factors in all of these studies is that none accounted for the presence of musculature and the forces they exert on the cervical spine *in vivo*. However, neck muscles are thought to act as stabilizers of the otherwise unstable cervical spinal column.

The second order buckling mode seen in previous studies was not seen in this study for either the FL or NFL group. However, pre-impact alignment and the application of the follower load induced a lordotic curve in the specimens and this lordosis may predispose the specimens to extension, sometimes referred to as first order buckling, shown in Figure 3 which was typical of all specimens.

The primary limitation of *ex vivo* studies of head first impact is lack of realistic in vivo neck muscle forces. We partially addressed this limitation applying a representative "resultant" follower load [21, 22] which is a simplified model of neck musculature. The model could be improved by incorporating a greater number of simulated muscle forces that is more physiologically correct. The use of a greater number of simulated muscles could also afford greater control over initial posture and alignment relative to the simplified model applied here.

CONCLUSIONS

We hypothesized that it is important to simulate in vivo compressive follower load in ex vivo experiments for headfirst impact and further that buckling in past ex vivo impact experiments may be due to absence of any approach to simulate musculature that is present in vivo. Therefore, the objective of this work was to compare the kinematics and kinetics of buckling, during head-first impact, of isolated cervical head/neck complexes instrumented with follower load to those without follower load. We simulated head-first impact of cervical spine-head complexes (n=12) using a drop-tower. Six complexes were instrumented with follower load (FL group), while six were not (NFL group). Follower loads did not prevent buckling in these ex vivo headfirst impacts. Significantly lower buckle time in the FL group indicated increased mechanical coupling between the head and T1 in specimens with follower loads. Increased coupling is believed to be caused by increased axial spine stiffness as a result of the addition of

follower load and related compression of the intervertebral discs. These preliminary results suggest that simulating follower load that may be similar to in vivo muscle forces results in significantly different buckling behaviour, and therefore potentially different injury mechanics occur in vivo than in many biomechanical tests where musculature is not simulated.

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