INTRODUCTION
Cervical spine injuries result in significant financial cost to the injured and society. Spinal cord injuries can lead to severe disability and death. Understanding of the biomechanics of these injuries is based on decades of study that consider in vivo and ex vivo axial compression. Early biomechanical studies used whole cadavers but lacked measurement of cervical kinetics and kinematics because the neck was surrounded by musculature. More recent studies use isolated cervical spine-head complexes that allow measurement of cervical kinetics and kinematics because musculature is removed. Both whole cadaver and spine-head studies suggest that, during head impact, the cervical spine buckles as the head impact moves it toward the relatively more massive torso and further that the type of buckling and resulting injuries may be correlated [1]. Some impact studies describe cables tethered to cervical anatomy to simulate the role of neck musculature in controlling neck posture and have successfully recreated clinically-observed injuries. However, these cables do not recreate lines of action of compressive follower loads that neck musculature supplies in vivo and that has been shown through recent modeling to significantly increase neck injury risk [2]. For quasi-static loading, ex vivo experimental work has shown that compressive follower loads (guided at each level) increase the compressive loads at which buckling occurs but without follower loads the isolated cervical spine could not even support the weight of the head[3]. Due to this evidence, we hypothesize that it is important to simulate in vivo compressive follower load in ex vivo experiments for head-first impact. Moreover, we hypothesize that buckling in past ex vivo impact experiments [1] may be due to absence of any approach to simulate musculature that is present in vivo. The objective of this work was to compare the kinematics/kinetics of buckling, during head-first impact, of isolated cervical head/neck complexes instrumented with follower load to those without preload.

METHODS
We simulated head-first impact of cervical spine-head complexes (n=12) using a drop-tower. Six complexes (Occ.-T1) were instrumented with follower load (FL group), while six were not (NFL group). Occiputs of all specimens were fixed to a biofidelic surrogate head and T1 was encased in a mounting cup. The metrics relevant to buckling that are reported in this abstract are: impact speed; time to neck buckle; the vertebral levels at which the neck buckles; and the total angular divergence at the levels at which the neck buckled. The criterion to determine buckling and vertebral level of buckling was 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 rotation direction that was greater than 2 degrees.

RESULTS
The mean impact speeds were 2.8 m/s (NFL) and 2.9 m/s (FL). There was a significant difference (p=0.009) in time to buckle between the NFL and FL group, which had means of 4.9 msec and 2.5 msec, respectively. For the FL group, 3/6 of specimens buckled across C4/5 (Fig. 1), 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 (sign convention in Fig. 1), 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 hyperextension across the spine (Fig. 1).

Figure 1: (left) sagittal plane view showing initial inclination of vertebra (red lines) and rotated inclination (blue) at the time of buckle; (right) vertebral rotations during head-first impact.

DISCUSSION & CONCLUSIONS
Follower loads did not prevent buckling in these ex vivo head-first impacts. Significantly lower buckle time in the FL group indicates increased mechanical coupling between the head and T1 in specimens with follower preloads. 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. Significantly lower rotation divergence in the FL group compared to the NFL group seems to be consistent with increased spine stiffness in the sagittal plane. It may be important to simulate these effects in computational models of head first impact. Furthermore, 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.

REFERENCES