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Introduction: Cervical myelopathy occurs when the cervical spinal cord is chronically compressed due to bony or soft tissue impingement. It commonly affects adults over the age of 50 years and causes upper extremity numbness, loss of hand dexterity, gait disturbances, and decreased proprioception. Historically, neutral cervical spine images were the standard to determine if spinal cord impingement was present. However recent studies including flexion and extension MRI images of cervical spine have shown this disease is highly depended on the dynamic motion of the spine[1]. Little is understood regarding the spinal cord stress during cervical spinal motion which causes cervical myelopathy. Since measuring in vivo cord stress on human subjects is unfeasible several groups have used finite element (FE) modeling to investigate cord stress distributions[2,3]. While these models have given insight into stress distributions they are primitive given they either model only the cord, ignore important anatomic structures, or use basic material property assumptions. The goal of our study was to create a FE model which incorporates all major anatomic features of the C2-T1 spine and spinal cord to predict cord stress during flexion and extension.

Methods: A 3D FE model of the cervical spine (C2-T1) was previously developed and validated[4]. A CAD model of the cervical spinal cord was created based on histologic sections from levels C3, C5, C8 and scaled to fit the geometry of the aforementioned C2-T1 model. IA-FEMesh was used to create a hexahedral mesh of the cord, from which quadrilateral elements were extracted to define the pia. In a similar manner, a quadrilateral mesh of the dura was generated. A convergence study was performed to ensure solution stability. The resulting cord, including the pia and dura, is defined by 42,239 elements. Once introduced into the C2-T1, the entire model consisted of 229,649 elements (Figure 1). Dentate and mengiovertebral ligaments were modeled as truss elements with no compression forces allowed. Placement of the dentate and mengiovertebral ligaments was based on anatomical reports[5]. The dura, pia, and cord were modeled as hyperplastic Mooney-Rivlen and material constants were calculated based on published in vitro tissue testing[6]. The dentate and mengiovertebral ligaments were modeled as linear elastic. The cerebral spinal fluid (CSF) was modeled as a fluid cavity enclosed by the dura and pia, while the pressure in the extradural and intradural space was based on published data[7]. General contact was used to define contact between all surfaces. The inferior endplate of the T1 vertebra was fixed in all directions as were the inferior most nodes of the spinal cord, pia, and dura. Using Abaqus 6.13-1/Standard a quasi-static rotation of 44.5° in flexion and 48.9° in extension was applied to the most superior portion of C2. This rotation corresponded to a reaction torque of -2 Nm and +2Nm, respectively. The cord, pia, and dura were allowed to move freely within the spinal canal. Axial stretch between cord root entries (cord stretch) was obtained as was the von Mises stress throughout the cord.
**Results:** In extension, cord stretch between the root entries in the axial direction ranged from -12.49mm at C2-C3 to 0.38mm at C6-C7, average -4.72±5.47mm. At full extension, a von Mises stress of 0.113 MPa was seen on the anterior spinal cord at the C6 level due to bony impingement (Figure 2). Additional stresses ranging from 0.012-0.036 MPa were present on the anterior and posterior cord at levels C4 and C5. In flexion, axial cord stretch ranged from -2.01mm at C4-C5 to 0.58mm at C2-C3, average -0.90±0.97mm. At full flexion, increased von Mises stress on the cord was observed along the anterior surface with a localized stress of 0.022MPa between levels C4 and C7.

**Discussion:** In this study we developed a model of the cervical spinal cord from C2 to T1 including the pia, dura, CSF, dentate and mengiovertebral ligaments and incorporated into our pre-existing cervical spine model. We utilized this model to determine both the cord stretch and amount of stress occurring in the cord during flexion and extension. One limitation of our model is the cord is not fixed at the caudal end as it would be in vivo, in the future we plan to drive caudal cord displacement with measurements taken from MRI measurements. Additionally as with all FE models, our model has certain intrinsic limitations such as using a single anatomic model as a global representation and simplified material properties for biologic tissues. However we have aimed to minimize these limitations by incorporating all major spinal anatomy and hyper-elastic material properties into the cord and our cord displacement and stress values are comparable to published data. Viljoen et al reported in healthy patients the thoracic spinal cord stretches -2.0mm to 6.7mm in the axial direction during flexion which corresponds to our data[8]. Additionally previously published models of cervical myelopathy, which only incorporated the cord, have shown maximum von Mises stresses to be 0.03-0.06MPa.[2,3] Our stress results also fall within this range, except for the bony impingement seen in extension which is twice as large. However as this is due to a bony impingement over a smaller area the stress values are reasonable. Finally, and most clinically important, is the locations of higher stress are primarily seen at levels C4-C6. This aligns with clinical evaluation as patients most often have cord compression at C5-C6[9]. Overall we have developed a model of the cervical spine and spinal cord which allows us to predict cord stresses during dynamic motion of the neck. This will, in the future, help us to better understand the progression of cervical myelopathy specifically by incorporating anatomic abnormalities such as osteophytes which lead to stenosis. Finally we aim to incorporate electrophysiology to determine how mechanical stress on the cord affects signaling pathways during dynamic motion of the spine.

**Significance:** Cervical myelopathy is a debilitating disease causing loss of dexterity and gait disturbances and is caused by dynamic impingement of the spinal cord. Little is understood regarding the stresses experienced by the cord.
Figure 1: Model of the cervical (A) spinal cord, (B) dura, and (C) spinal cord integrated with the C2-T1 model.
Figure 2: Spinal cord in extension. The arrow highlights the area of bony impingement on the anterior surface.