INTRODUCTION: Rheumatoid arthritis (RA) involvement of the occipito-atlanto-axial (C0-C1-C2) complex is commonly seen, however the biomechanical role during disease progression is not well understood. Investigation of progressive disease states does not lend itself to traditional in vitro methods such as cadaver experimentation. Thus, the authors implemented the finite element (FE) method to study the biomechanical factors, if any, that contribute to the development and advancement of RA and its associated clinically-observed lesions.

METHODS: A ligamentous three-dimensional finite element model of the C0-C1-C2 complex (Fig. 1) was generated from 0.5 mm thick serial computed tomography scans. Validation of the model was performed by comparison of baseline kinematic predictions to published cadaveric and reported range of motion data. Transverse, alar, and capsular ligament stiffnesses were sequentially reduced by 50%, 75%, and 100% (removal) and the model subjected to full flexion loading (1.5 Nm). Stress profiles at the transverse ligament-odontoid process junction were monitored. Changes in loading profiles through the C0-C1 and C1-C2 lateral articulations and their associated capsular ligaments were computed. Posterior atlanto-dental interval (PADI) values were calculated to correlate ligamentous destruction to advancement of atlanto-axial subluxation (AAS).

RESULTS: Model rotation predictions (at 0.3Nm) fell within one standard deviation of reported to in vivo and in vitro published values. The model
predicted that the average contact stress at the posterior base of the odontoid process, 1,281 kPa in the baseline model, is reduced to 880 kPa and 780 kPa with transverse ligament stiffness reductions of 50% and 75%, respectively. Decreases in loads, associated with transverse ligament compromise, through the lateral C0-C1 and C1-C2 articulations were also predicted by the model Fig. 2). These loading reductions were compensated by their posterior capsular ligaments, with approximately 50% increases in the ligaments seen after transverse ligament removal. PADI values indicated that the transverse ligament had the greatest effect on AAS during the early stages of the disease (no alar and capsular ligament damage). Subsequent coincident involvement of the alar and capsular ligaments produced advanced AAS (PADI<11mm).

CONCLUSIONS: To the best of our knowledge, this is the first report of a validated, three-dimensional model of the C0-C1-C2 complex with application to RA. The data indicate that there may be a mechanical component (in addition to enzymatic degradation) associated with the osseous resorption seen during RA. Specifically, erosion of the base of the odontoid may involve Wolff's Law unloading considerations. Changes through the lateral aspects of the atlas suggest that this same mechanism may be partially responsible for the osseous loss seen during progressed RA. PADI values indicate that significant changes in craniovertebral junction loading occurs after 75% transverse ligament disruption. Complete transverse ligament disruption (removal) coupled with alar and/or capsular ligament compromise is requisite if advanced AAS is to be clinically observed.