# Quantification of Upper Cervical Spine Motion Sensitivity to Ligament Laxity Using a Finite Element Human Body Model for Occupant Safety

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## I. INTRODUCTION

There is a need to reposition Finite Element (FE) Human Body Models (HBMs) from a neutral to a non-neutral driving posture to evaluate the risk of injuries in out-of-position impacts, including repositioning of the craniocervical complex. Changing craniocervical postures in the physiologic regime can be achieved by applying an external load to the head until the desired posture is obtained. This method is preferred over morphing methods since tissue stresses resulting from the change in posture are included in the model, which is an important factor for predicting crash-induced injuries [1]. The upper cervical spine (UCS), comprising C0-C2, contributes to the large range of craniocervical physiologic motion, therefore the ligaments in UCS are required to predict biofidelic strains as a result of posture change. An existing UCS FE model, previously validated for dynamic bending motions [2], was re-evaluated in physiologic loading and found to be stiffer at the intersegmental level compared to experimental data. The physiologic rotational motion of the UCS is characterised by neutral and linear zones. The neutral zone is the region where the rotation of the spine increases with a negligible amount of external moment, which is governed by laxity in the ligaments [3]. The linear zone is the region where the spine resists rotation due to engagement of the ligaments. Measuring the laxity of ligaments in a laboratory setting is challenging as the laxity is lost once the ligaments are dissected for testing. Numerical optimisation methods can be used to determine the laxity in UCS ligaments, to obtain biofidelic response in the UCS. However, due to the complicated connectivity of the UCS, involving 14 ligaments, arriving at a converged laxity value using optimisation methods is challenging. The current study presents a new quantification of ligament laxity contributions to different loading modes required to undertake ligament laxity optimisation of the UCS and to accurately reposition HBM.

## II. METHODS

The UCS model comprising the skull (C0), C1 and C2 was extracted from the GHBMC M50 v.5.0 HBM, representing an average stature male (Fig. 1). The force-displacement curves reported by Mattucci [4] for the UCS ligaments were assigned to the UCS ligaments in the model. The model was constrained at C2 and a moment of 1.5 Nm was applied to C0 in flexion, extension and axial rotation, as reported in experimental studies [5-6]. To identify the UCS ligament sensitivity, it was necessary to systematically vary the laxity values and analyse the variations in response to different modes of loading. Laxity variation using one-at-a-time manual method is prone to errors due to the large design-space as a result of multiple parameters (14 UCS ligaments) and three loading modes. In flexion, the 10 ligaments were varied for laxity, including: the posterior atlanto-occipital membrane (PA-OM), posterior atlanto-axial membrane (PA-AM), interspinous ligament (ISL), upper- and lower- crux, transverse, apical, tectorial membrane (TM), C0-C1 capsular (CL01) and C1-C2 capsular (CL12). These ligaments were investigated based on their biomechanical function of restricting the UCS in flexion. For extension, four ligaments were varied, including: anterior atlanto-occipital membrane (AA-OM), anterior atlanto-axial membrane (AA-AM), CL01 and CL12. For axial rotation, eight ligaments were varied, including alar, atlanto-axial alar, TM, CL01, CL12, AA-AM, PA-AM and ISL. Ligament laxities were varied between 0 mm (representing no laxity) to 8 mm (determined from preliminary studies as a large value encompassing the range of motion for the UCS). A Latin Hypercube Sampling (LHS)-based Design of Experiments (DOE) approach was used to generate the simulations for each loading mode while minimising the design-space. The models were solved using a commercial FE software (LS-DYNA R9.2). The rotation responses at 1.5 Nm for C0-C1 and C1-C2 in all three modes of loading were extracted and Radial Basis Functions (RBF) were used to develop metamodels for the responses. Based on the metamodels, sensitivity (expressed as a percentage of total motion) of the variables on the response were calculated using Global Sensitivity Analysis (GSA). The DOE, metamodelling and GSA were automated using an optimisation software (LS-OPT v.5.1, LST) to determine the dominant ligaments and ligament interactions, not previously investigated.

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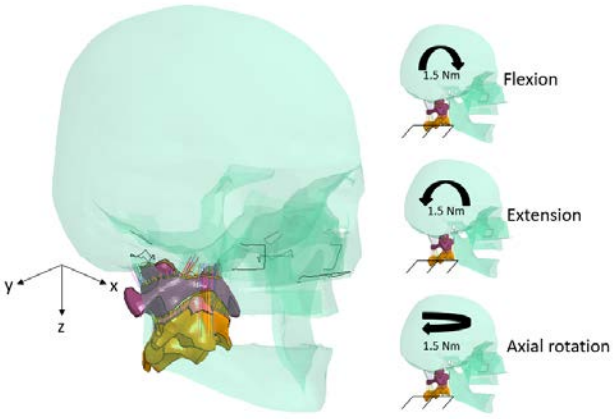


Fig. 1. Detailed FE model of UCS with boundary conditions for three load cases, 1.5 Nm moment applied following [5-6].

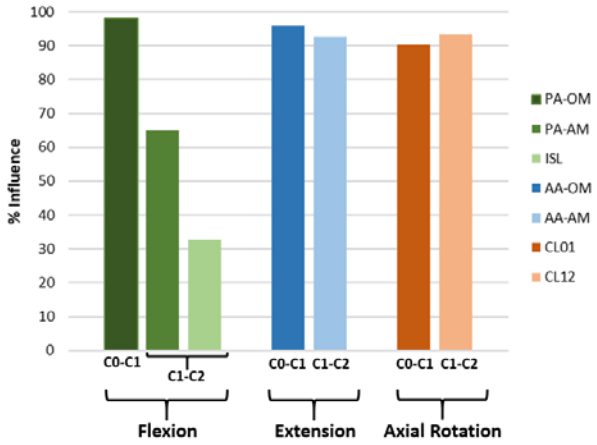


Fig. 2. Major contribution (% of response) of UCS ligaments in flexion, extension and axial rotation.

## III. INITIAL FINDINGS

The metamodels describing the responses for each mode of loading demonstrated an average $R^2$ value of 0.98 for predicted vs. computed responses, demonstrating an excellent fit and applicability of the GSA method to identify ligament contributions to response. Responses in each loading mode were a result of interaction between multiple ligaments. In most cases, the response was a result of high dominance from one particular ligament (>90%), while other ligaments cumulatively contributed ≤10% of the response. In general, the major contributions of ligament laxity to the range of motion were from PA-OM and PA-AM in flexion, AA-OM and AA-AM in extension, and CL01 and CL12 in axial rotation (Fig. 2).

**Flexion:** the laxity of PA-OM contributed 98.1% of the flexion response in the C0-C1 joint. In C1-C2, the PA-AM contributed 64.9% of the response in flexion, followed by ISL contributing 32.8%.

**Extension:** the AA-OM had a contribution of 95.6% at C0-C1 for extension response. AA-AM, CL01 and CL12 laxities contributed only 1.5%, 2.6% and 0.4%, respectively, to C0-C1 extension. At the C1-C2 level, the laxity in AA-AM contributed 92.5% of the response, followed by laxities of CL12 (5.1%), CL01 (1.5%) and AA-OM (0.9%).

**Axial Rotation:** in axial rotation, CL01 and alar laxities contributed 90.3% and 2.6% of C0-C1 rotation response, respectively. For C1-C2 axial rotation, CL12 and ISL laxities contributed 93.3% and 4.3% of the response.

## IV. DISCUSSION

The complex interconnectivity in the UCS makes it challenging to quantify the contributions of ligaments using experimental methodologies, and interactions between ligaments further complicate this process. The current study investigated the effect of laxity at the intersegmental level, whereas all previous studies have assessed response of the entire C0-C1-C2 complex, which can mask some of the sensitivities. The use of the LHS method for DOE was effective in creating a design-space that had 100, 23 and 68 cases in flexion, extension and axial rotation, respectively. This method of DOE was rigorous when compared to the conventional method of varying parameters one-at-a-time, which may miss important contributions from ligaments. The existing UCS model demonstrated a stiffer response in flexion, which can be attributed to the lower number of ligaments investigated for laxity; and this has been improved in the present study. For example, the ISL was identified as important in flexion, but has not been included in previous studies. The reported percentage values are inclusive of interaction from other variables, which would have been challenging to identify using manual methods as described in the previous study [2]. Biofidelic response of the UCS in the physiologic regime can incorporate strains at the tissue level when the model is repositioned by the application of external loads. Ultimately, improved biofidelity is needed for the UCS to predict the initial conditions of a model for out-of-position scenarios, and to predict the potential for crash-induced injuries in the neck for non-neutral postures.

## V. REFERENCES

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