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I. INTRODUCTION

Active musculature in the neck is an important contributor to head and neck response, and the presence of active musculature has been associated with reduced injury in acceleration events [1-6]. Due to the lack of active musculature in anthropomorphic test devices (ATDs), and the limited exposures allowed for human volunteers, a computational neck and head model was used to evaluate the effect of varying muscle activation and strength parameters on head kinematics, and the potential for injury at the tissue-level (CIIs, crash-induced injuries). Current muscle activation schemes include active and passive tissues [7], which require several parameters, including the muscle activation onset time from the moment of impact, muscle activation levels, and the muscle physiological cross sectional area (PCSA). Activation onset times from the literature are consistent, at 72(6) ms for flexors and 75(6) ms for extensors from [8], however there is limited data regarding PCSA for the neck muscles. The aim of this study was to evaluate the sensitivity of head kinematics and tissue-level response to variations in activation time, PCSA and activation level.

II. METHODS

A validated head and neck finite element (FE) model (Global Human Body Models Consortium (GHBMC) 50th percentile male HBM, M50 V4.3)[9], incorporating active musculature, was used to conduct the current study. The model included the seven cervical vertebrae (C1-C7) and first thoracic vertebra (T1), intervertebral discs (IVD), upper and lower cervical spine ligaments, passive and active musculature, and a simplified model of the head with equivalent mass and geometric properties (Fig. 1(a) and (b)). Three-dimensional solid elements were used to model the passive behaviour of the musculature, and embedded one-dimensional Hill-type elements were used to model the active behaviour (Fig. 1(c)), resulting in 27 pairs of cervical flexor and extensor muscles. A motion boundary condition applied to T1 simulated a neutral positioned 8g frontal impact from previous experiments conducted by the Naval Biodynamics Laboratory (NBDL) [10].

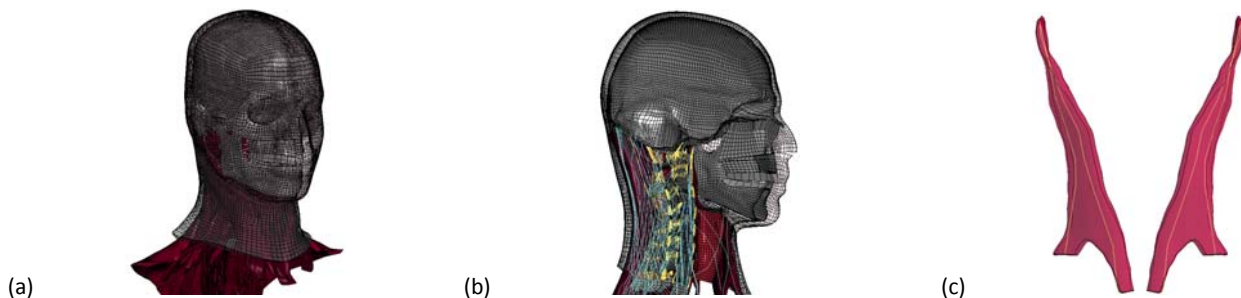


Fig. 1. GHBMC M50 head and neck: (a) oblique view; (b) sagittal cross-section; (c) sternocleidomastoid.

Baseline active muscle parameters included muscle activation onset time of 74 ms [8], muscle PCSA values reported by [11], peak isometric stress of 0.5 MPa [12], and muscle activation levels of 100% for both extensor and flexor muscles. Four cases were investigated (Table I). Neck muscle activation time (60–80 ms) and PCSA (baseline +/-30%) were identified from the literature.

TABLE I
ACTIVATION SCHEMES

Parameters / Cases	Superior	Baseline	Inferior	No Activation
Activation onset time	60	74	80	-
Muscle PCSA	+30%	Reference [11]	-30%	Reference [11]
Activation	100%	100%	100%	0%

III. INITIAL FINDINGS

Head centre of gravity (CG) kinematic responses, including X-Acceleration (AX), Z-Acceleration (AZ), Y-Rotational Acceleration (RAY) and Y-Rotation Displacement (RDY) (Fig. 2), were evaluated for each test case. The maximum strains of the lower cervical spine ligaments - capsular ligament (CL) (Fig. 3), anterior longitudinal ligament (ALL), posterior longitudinal ligament (PLL), ligamentum flavum (LF) and interspinous ligament (ISL) - were calculated using physiological neutral ligament lengths, following Fice [13].

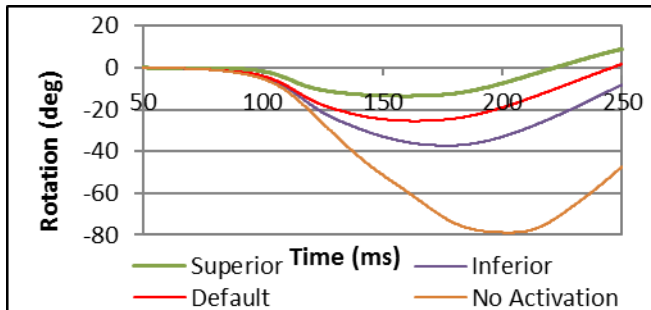


Fig. 2. Head CG RDY.

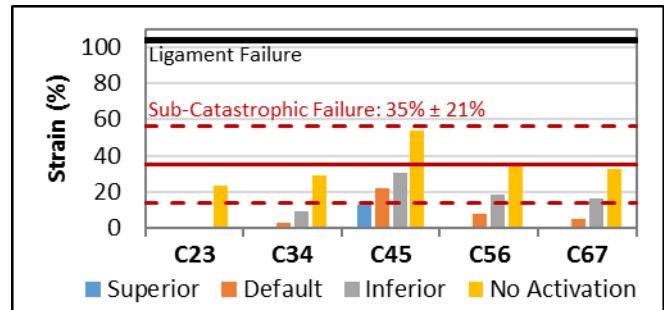


Fig. 3. Maximum CL strain during 8g frontal impact.

Transverse compression of the nerve root from movement of adjacent vertebral bodies during impact was investigated using the maximum height and width reduction of the intervertebral foramen (IVF) space [14]. IVD shear strain [15] and relative vertebral body rotation [16] were evaluated to assess IVD injury. No hard tissue failure was predicted from any of the simulations, although hard tissue failure was enabled in the model. All CIIs for the C4-C5 segment are presented in Table II, below.

TABLE II
CIIs FOR C4-C5 MOTION SEGMENT TISSUE-LEVEL RESPONSE FOR EACH IMPACT SCENARIO

CIIs	Superior	Baseline	Inferior	No Activation	CII Failure (SD)
CL Strain (%)	12.74	22.19	30.35	53.90	103.6 (80.9)
ALL Strain (%)	<0.01	<0.01	<0.01	0.67	30.8 (5)
PLL Strain (%)	0.01	0.01	0.01	7.42	18.2 (3.2)
LF Strain (%)	1.40	10.81	19.58	46.06	77.0 (12.9)
ISL Strain (%)	9.75	19.70	28.25	54.49	60.9 (11.2)
IVF Height Reduction (mm)	0.67	0.31	0.16	<0.01	5.7
IVD Shear Strain (rad)	0.54	0.62	0.65	0.79	0.40 (0.3)
Relative Rotation (deg)	5.20	7.10	8.70	13.80	13.1 (3.4)

IV. DISCUSSION

The AX, AZ and RAY of the head CG showed similar trends, with the superior activation case having the shortest onset of head acceleration and lowest magnitude followed by the baseline activation, inferior activation. The case with no activation resulted in the longest acceleration onset time and highest magnitude. The CG RDY showed an increased head rotation angle and shortened onset of head movement with decreasing PCSA, and increased muscle activation onset time. The no activation case showed a significant increase of 500% in head rotation angle when compared to the superior activation case. Reductions in IVF width and height for all activation schemes did not reach injurious levels, which were expected for a neutral positioned frontal impact. All ligament strains, IVD posterior shear strain and vertebral body relative rotation decreased with increasing muscle activation due to stiffening of the neck from muscle activation. Head CG accelerations showed low sensitivity to changes in the muscle activation scheme when compared to rotational displacement, and CIIs at the tissue level. Future studies will extend to different severity rear, lateral and frontal impact scenarios for a range of muscle activation schemes.

V. REFERENCES

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