Development and validation of rat head model to predict impact acceleration induced traumatic axonal injury

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Abstract

Traumatic brain injury (TBI) is caused by local tissue deformation at the time of trauma leading to neurological dysfunction. Finite element (FE) modeling of TBI is an effective approach to compute local mechanical response and correlate to the injury location and severity. A few FE models of rodent TBI have been developed to simulate focal brain injury. No model is capable of simulating widespread traumatic axonal injury (TAI) resulting from impact-acceleration injury. The objective of this study is to develop and validate a high-resolution FE rat head/body model to correlate brain responses with head kinematics and axonal pathology induced by head impact.

The head geometry of an adult Sprague Dawley (395g) was acquired from the MicroCT images and T2-weighted MRI images (4.7 Tesla). The skull and brain structures were segmented, differentiated and meshed with 3D hexahedral elements. The FE rat head model consists of essential anatomical structures including scalp, sandwich skull, dura, arachnoid-pia, olfactory, cortex, corpus callosum (CC), hippocampus, cerebellum, ventricles, brainstem with pyramidal tract (Py), medial lemniscus (ml) and facial tissues. The rat head model was meshed with over 770,000 elements at resolution of 0.1-0.2mm. A FE rat body was also developed in order to mimic spinal-brainstem stretching during head impact. 13 different mechanical properties were used to represent the regional heterogeneities and the anisotropies of white matter tracts. The cortical displacement and head acceleration measured from in vivo dynamic cortex deformation (DCD) tests and Marmarou's impact-acceleration (IA) tests respectively, were used to validate and investigate the tissue responses in the brain. The model predicted tissue strain and pressure response was related to quantify TAI assessed by beta-APP and RMO14 immunochemistry.

The model predicted peak displacement of brain cortex surface fell well within the experimental results in DCD tests. The predicted peak head acceleration (980g) and head displacement matched average test results measured from in vivo IA tests of 1.25m and 2.25m. Simulation results showed that the CC strain was correlated to rotation velocity (R^2=0.68) and CC pressure rate was related to linear acceleration (R^2=0.56). The high strain was observed in brainstem dorsal (including CC), ventral cortex and hippocampus. Principal strains (0.35-0.42) were found in CC and brainstem with the highest strain in the Py (0.57). These outcomes agree with relative magnitude of axonal injury assessed on histological sections with DAI counts being higher in brainstem than in corpus callosum. The distribution of CC strain from median to lateral and Py strain from rostral to caudal agreed well with the TAI distribution.

An anatomically detailed FE model of an adult rat head with full body was validated against brain deformation and head kinematics measured directly from in vivo experiments. The validity in modeling of impact acceleration TBI enabled a thorough analysis of the tissue response

elicited by trauma in the entire brain, which has never been demonstrated. The preliminary correlation of high tissue strain with the region of histopathological damage in white matter shows promise that a validated computer model can improve understanding of pathogenesis of brain injury.