Validation of Local Brain Kinematics of a Novel Rat Brain Finite Element Model under Rotational Acceleration
- Clarification of Diffuse Axonal Injury Mechanisms -

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ABSTRACT: Relative brain-skull displacement under head rotational acceleration in rats was evaluated experimentally. For this, a thin pin was entered the cortex and rigidly attached to the skull prior to impact. For peak rotational accelerations of 1.7 Mrad/s^2, the pin scarred the brain cortex: 1.2 mm superficially and less centrally. These measurements were used to validate the brain kinematics of a new anatomically detailed FE model of the head-neck complex of a rat. This model is intended to be used to clarify brain loading mechanisms and to develop brain tissue injury threshold for Diffuse Axonal Injuries as detected through immune-histology.

KEY WORDS: safety, finite element method (FEM), injury mechanism, Diffuse Axonal Injuries [C1]

1. Introduction

Traumatic brain injuries (TBI) of diffuse type (DBI) such as concussions and diffuse axonal injuries, which commonly occur from rotational accelerations of the head in sports and traffic accidents, account for a large proportion of the disabling and lethal injuries. Reduction of these requires the introduction of improved protection which should be developed using appropriate test and computational methods, and injury criteria.

Numerous research programs, of which the majority use animal experimental data have suggested rotational injury criteria and associated limits (1). In the past head trauma experiments using non-human primates (NHP) were conducted (2)-(4). The global injury thresholds obtained from the experiments, were scaled to humans but the scaling methods utilized (5)(6) have been criticized and scarcely validated (7). In addition to the experimental work, efforts to develop NHP Finite Element (FE) models of the head of the primate specimens have been suggested (8)(9) in order to better understand the kinematics of the brain inside the skull and to establish tissue strain based thresholds usable for humans.

Recently, efforts to study DBI experimentally and to establish strain based tissue level injury criterion based on computer simulations with non-primate animal species such as swine (10), sheep (11), and rats (12)(14) are being conducted. Although many of these models have proven useful to improve the understanding of brain biomechanics, for the quantification of injury limits, the motion of the brain inside the accelerated skull demands validation.

One critical aspect to achieve further validation level of the brain-skull interaction in brain FE models is the generation of experimental methods that allow the quantification of the amount of brain-skull slip under different loading conditions. This aspect has been qualitatively studied by performing craniotomies before impacts or high speed X-rays (15)(17) to living primate specimens. In addition, quantification has been done with Post mortem human subjects (18). However, to our best knowledge, the brain-skull relative displacement under rotational injurious loading conditions has not been quantified in small animals and no FE-model of small animals includes a validated brain-skull interface.

The aim of this study is to obtain new local brain-slip experimental measurements and utilise them to validate a detailed rat head FE model useful to study TBI caused by head rotational accelerations. Thereafter, the FE model will be used to simulate extensive series of rotational acceleration experiments for the development of strain based injury thresholds. These will, when properly applied, allow the development of protective strategies that will enable a reduction of the amount of rotation induced TBI in humans.

2. Methodology

The methodology of the work presented here consists of:

- Experimental quantification of brain-skull relative displacement under head rotational acceleration
- Development of a rat brain FE model from medical images
- Validation of local brain-skull relative motion of the rat brain FE model against the generated experimental data
- Analysis of brain kinematics under rotational acceleration and preliminary comparison between brain tissue strain

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31
patterns and DBI locations as detected through immune-histology

2.1 Experimental quantification of brain-skull relative displacement under head rotational acceleration

Rats were exposed to rotational acceleration using a test bench developed and extensively used in the past for rotational acceleration induced mTBI research \(^{(19)-(22)}\). Figure 1 illustrates the trauma producing equipment, the positioning of the specimen and the center of rotation during trauma.

![Fig. 1 Illustration of the trauma producing equipment (left) and the positioning of the specimen (right). Reproduced from \(^{20}\) with permission.]

The experiments were performed at the Experimental trauma Unit, Neuroscience Department, Karolinska Institute in accordance with the Swedish National Guidelines for Animal Experiments, under the approval of the Animal Care and Use Ethics Committee in Stockholm, Sweden [doc. N143/09].

2.1.1 Specimen preparation protocol and rotational trauma delivery

Three male Sprague-Dawley rats, with an average body mass of 0.501 kg, were anesthetized with 2.4 ml/kg intra-abdominal injections.

A sequence of preparations of the animal was carried out prior to trauma. A midline incision was made through the skin and periorbit on the skull vault. Then the tissue adhered to the nose and the frontal and parietal bones was removed. The exposed bone was then gently sanded. A metal plate, referred to as skull cap, was then shaped to match the contour of the exposed skull and firmly glued to it with a dental glue (Super-Bond Cand B; Sun Medical Co., Shiga, Japan).

An attachment plate was fastened to the skull cap and finally the animal head was inserted and secured to a rotating bar that rotates freely around a horizontal axis (Figure 1). During trauma, a weight denominated the striker (weight 0.010 kg) was accelerated in a specially designed air driven accelerator (CNC-Process AB, Hova, Sweden) and was made to hit a rubber block that was glued onto a striker target. The impulse produced subjected the rotating bar and the animal head to a short sagittal plane rearward rotational acceleration. This acceleration phase was followed by a rearward rotation at nearly constant velocity. Finally, the striker target makes contact with a rigid cross beam which is covered with high density foam. The rotational acceleration magnitude was selected by modifying the striker speed which was varied by means of modifying the air pressure in the accelerator. An Endeveco Isotron 2255B-01 accelerometer was mounted on the rotating bar at a radius of 36.5 mm from the center of rotation.

2.1.2 Evaluation of boundary conditions

In order to confirm that the skull, the head cap, and other attachments did not spring back during trauma, high speed video recordings were captured and processed to monitor head and neck kinematics. Prior to trauma, two rectangular carbon fiber plates were glued (Loctite 401) to the foremost part of the skull cap and to the spinous process of the second cervical vertebra (C2), respectively. Photo markers were attached to the plates to enable film analysis. In addition, the test rig stand that was fabricated in aluminum was replaced, for enhanced visibility, by a unit fabricated in transparent plastic. The motion of the head-neck complex was captured with a Photron SAL 1 high speed video at 25000 fps with a resolution of 448x448 pixels. The positions of the photo markers and the tip of the upper incisor tooth were digitized (Tema v. 3.5.012 by Image Systems AB). By processing the high speed videos, the center of rotation of the skull and C2 were deducted and used to apply the boundary conditions in subsequent FE-simulations of these experiments. Figure 2 shows an illustration of the skull and the neck of a rat with the carbon fiber plates (black rectangle with white circles). Sequential positions of the skull markers (dark blue lines), the C2 markers (black lines) and the incisive teeth (pink triangles) as measured from high speed video are superimposed to the illustration.

![Fig 2. Illustration of rat skull and neck with markers for high speed video tracking and evaluate displacements.]

2.1.3. Enhancement of rotational trauma test rig to evaluate local brain-skull relative displacement

For this specific task, the skull cap was redesigned and the specimen preparation protocol slightly modified. Following the gluing of the skull cap, a 0.6 mm diameter hole was drilled through the skull at a location of 3.5 mm to the rear and 2.2 mm to the right of Bregma. A 0.5 mm diameter steel needle was...
inserted through a guide mounted to the skull cap, the hole previously drilled through the skull and the dura until the tip reached 3 mm into the brain cortex and firmly fixed with an Allen screw. Figure 3 shows an illustration and a picture of the redesigned skull cap and inserted needle.

Fig. 3 Illustration of the location of the skull cap on the specimens’s head (left) sketch of the unmodified skull cap (middle) and picture of the redesigned skull cap including the penetration pin taken on a 5x5 mm grid paper sheet (right)

By introducing this modification, when the skull was subjected to rotational acceleration, a scar was produced in the brain cortex when the brains lagged behind or continued their rotational motions when the skull was subjected to rotational decelerations. Directly after trauma, the needle was carefully removed and the animals were formaldehyde perfused. The brains were frozen, 14 µm cryostat sections were cut at a distance of 0.5, 1.0, 1.5 and 2.0 mm from the surface, thawed onto chromealum–gelatine treated slides. Cresyl violet stain (Certistain, 0.25mg/ml), which is a basic dye, was applied to the sections to indicate absence neurons in the scar created by the needle. The slides were examined using a 40× lens in a Nikon E600 microscope. Images were captured with a Nikon Digital Sight DS-U1 (5 megapixel) camera; controlled with Nikon EclipseNet software. The length of the scars, and the blood clot that was formed, were evaluated on the captured images.

2.2. Development of a rat brain FE model
2.2.1. Model geometry generation

An FE model of the head and neck of a rat was developed by combining a rat brain digital atlas in stereotactic coordinates (23), and an original set of Computed Tomography (CT) scans and Magnetic Resonance Images (MRI) from Sprague-Dawley rats. These images were taken at the Karolinska Experimental Research and Image Center (KERIC) in Stockholm (Sweden). A Mediso nanoScan® PET-MRI/CT multimodal small animal imaging system was used to take the CT-scans with 0.078 mm slice distance. For the MR images, former image sets taken by the MRI machine. The specimen from which MR images were provided had a body mass of 454 g. Several commercial software were used for the digitization of the brain atlas (GetData Graph Digitizer), segmentation of the medical images (Simpleware v4.2), and for the construction of a mesh (Hypermesh v11.0). For the simulation work, presented later, LS-Dyna (mpp971s R5.1.1) was used. Figure 4 illustrates the development process of the rat model described below.

Fig. 4 Rat FE model development process. ((*) from(23))

The geometry of the skull was obtained from the CT images and segmented prior to quad meshing. To develop the mesh of the brain a different technique was used. Here, the contours of the brain regions of interest in 58 coronal 1 mm equidistant sections of the brain, obtained from the Atlas (23), were identified and digitized. The digitized contours were imported as point data into the meshing software in a similar way as described by (14). The points were used to define lines, planes and volumes that allowed building a hexahedral mesh.

Since the dimensions of the Atlas' brain did not exactly correspond to the dimensions of the intracranial space of the skull from the CT, one more step was required to adjust the geometry of the brain mesh. To do this, the contours of the brain regions of interest and the cerebrospinal fluid space from the MR images were segmented and its contours digitized. The FE mesh based on the Atlas and the segmented brain geometry from the MR images were imported into the same software. Then, the FE mesh corresponding with each of the brain regions was morphed to fit the volume of the corresponding brain region in the MR images.

The final model, shown in Figure 5, consists of 147036 elements. It includes and differentiates between the skull, olfactory bulb, thalamus, brainstem, cerebrum, corpus callosum, hippocampus, cerebellum and neck. The neck includes the spinal cord and the first two cervical vertebrae. The skull mesh consists of 33490 quad elements of approximately 0.4 mm size. The brain is meshed with 79469 hexahedral elements of approximately average size of 0.35 mm. Olfactory bulb, thalamus and brainstem are meshed as a single longitudinal structure of shared elements aligned in the bottom part of the model. The cerebrum, corpus callosum, and hypothalamus are meshed as a second structure of shared elements which lower region is connected to the upper region of the thalamus by means of tied nodes. Finally, the cerebellum is meshed as a third structure which lower region is tied to the upper part of the brainstem in the vicinity of the pons.
2.2.2 Material modeling

The material properties of the anatomically heterogeneous brain regions were defined from rat brain tissue samples subjected to indentation tests\(^{(24)}\). These experiments were simulated with an FE model of cubic tissue samples of brain middle cortex compressed by a cylindrical rigid indentor, following the experimental descriptions of the original publication\(^{(24)}\). The nodes of the base were constrained in all directions, while the velocity of the indentor was prescribed to 750\(\mu\)m/s. Figure 6 shows a comparison between analytical and experimental results of the relaxation indentation tests with the results obtained from the simulation.

Finally, the Dura mater and the Pia-Arachnoid were modeled as elastic membranes (E= 40 MPa)\(^{(25)}\) and the skull bone was modeled as a rigid material.

2.2.3 Brain-skull interface modeling

The Pia and the Arachnoid complex (PAC) was modeled as a single membrane layer surrounding the different brain regions. To define the distance between the PAC and the Dura in the FE model, the thickness of the CSF layer as observed in the MR images was used.

The interaction between the PAC and the dura was simulated by defining a sliding contact that allows displacement in tangential direction to the skull while constrains the displacement in perpendicular direction in a similar way to that used for state-of-the-art human head FE models\(^{(25,26)}\). This was done by using a default ls-dyna contact type named *CONTACT_AUTOMATIC_ONE_WAY_SURFACE_TO_SURFACE_TIEBREAK with option 4 and a low friction coefficient of 0.05.

2.3 Validation of the rat brain FE model local brain-skull relative displacement

The average head rotational acceleration curve obtained from the experiments was utilized to prescribe the rotational acceleration to the skull FE model. The motion of C2 measured from the high speed videos was also prescribed to reproduce the neck kinematics. Under these conditions, a 2.5 ms duration rotational trauma was simulated. To validate the local brain-skull kinematics of the model, the trajectories followed by nodes from the brain cortex in the simulations were processed and compared to the maximum length of the scars measured at corresponding brain cortex depth levels in the experiments.

2.4. Analysis of brain kinematics and brain tissue strain at simulated trauma experiments and comparison of brain tissue strain patterns to experimental DAI maps

In order to improve the understanding of the kinematics of the cerebrum inside the rotating skull, an analysis of the brain cortex kinematics and tissue strain in comparison to the rotational acceleration applied to the skull in the simulated trauma. In addition, the brain tissue strain pattern from the simulated rotational experiment was qualitatively compared with a map of DAI locations\(^{(19)}\) as detected through beta Amyloid Precursor Protein (\(\beta\)-APP) immune-histology technique. The injuries were produced with the same experimental model and loading conditions utilized in the simulation study.

3. Results

Head rotational acceleration: The trauma induced an acceleration phase, a free rotation phase and a deceleration phase. Rotational acceleration peaks of 1.64, 1.73 and 1.65 Mrad/s\(^2\) were
measured in the three brain-slip experiments. A duration of 0.4 ms for the acceleration phase was obtained for all cases. Figure 7 shows the rotational acceleration recorded for each of the three experimental cases (thin color lines) and the calculated average curve (thick black line).

Fig. 7 Experimental rotational acceleration curves

Experimental and simulated brain-slip displacements: The penetrating pin caused a measurable brain-blood-barrier injury region in the brain cortex during rotation. The local relative displacement of the brain cortex with respect to the skull was evaluated by measuring the maximum length of the injured region on the microscope capture at 0.5 mm parallel slices of the brain. Figure 8 shows images of the brain sections of one of the specimens (RKI301). The images show the scar area identified surrounded by a black marker and the scale bar represents a 500 μm length. The sections were sliced in perpendicular direction to the insertion of the needle and at four levels from the brain surface. Scar lengths of 1.3, 1.4, 1.1 and 0.7 mm, respectively, were measured for this animal. Values obtained for this and the other experimental cases, as well as average values from the three experiments are shown in Table 2, below.

Table 2. Experimental and simulated brain-slip measurements

| Specimen   | Scratch length (mm) | 0.5 below surface | 1 below surface | 1.5 below surface | 2.0 below surface | Peak rotational acceleration (Mrad/s²) |
|------------|---------------------|-------------------|----------------|-------------------|-------------------|--------------------------------------|
| Experiment RKI300  | 1.1                | 1.1               | 0.9           | 0.6               | 1.64              |
| Experiment RKI301  | 1.3                | 1.4               | 1.1           | 0.7               | 1.73              |
| Experiment RKI302  | 1.1                | 1.2               | 1.1           | 0.6               | 1.65              |
| Experiment Average | 1.2                | 1.2               | 1.0           | 0.6               | 1.67              |
| Simulation      | 1.1                | 1.5               | 1.3           | 0.7               | 1.67              |

Brain cortex kinematics and tissue strain at simulated trauma: Figure 10 shows the skull rotational acceleration curve and the maximum principal strain of the element of the brain cortex which sustained the highest strains. Below, a sequence of images of the simulated rotational impact were also shown. Highest strains of about 0.3 occurred at 2.1 ms, after the skull is subjected to maximum rebound deceleration.

Fig. 8 Microscope images of slices at 0.5, 1, 1.5 and 2 mm taken in perpendicular direction to the needle, and the scar area identified.

The experimental average rotational acceleration curve (Figure 7) was used to simulate the rotational trauma. Nodal trajectories from corresponding areas at which the scratch length was measured were extracted from the simulations and compared to the experimental average measurements. Figure 9 shows brain displacements relative to the skull of nodes at four levels from the brain surface at the approximate location where the experimental measurements were taken. The origin of the coordinate system in the figure is placed on the skull inner surface at 3.5 mm to the rear and 2.5 mm to the right of Bregma.

Fig. 9 Brain cortex node trajectories relative to the skull at 0.5 (black), 1 (red), 1.5 (green) and 2 mm (blue) from the brain surface in the simulated experiments. (Lines corresponding with the skull inner surface and the brain cortex are added to the image as reference)

Results from the simulation show a total displacement of 1.1, 1.5, 1.3 and 0.7 mm, respectively. These results are also included in table 2 for comparison with the experimental values.
Simulated brain tissue strain pattern in comparison to experimental DAI injury locations: Figure 11 shows a comparison of the locations at which DAI were detected in the experiments with a pattern of the maximum principal strain of the brain FE model at 2.1 ms of the simulation, where tissue strains were the highest.

Fig. 11 Left: Schematics of the middle coronal section of the rat brain with stars indicating localization of β-APP-positive axons rotational acceleration induced DAI locations from (19).
Right: Maximum principal strain pattern at a middle coronal section of the rat brain FE model at the time at which strains were the highest.

4. Discussion

The simple and repeatable experimental method presented in this article allowed us to estimate how much total brain cortex displacement occurred due to sagittal rotation. Based on these data, the local brain-skin relative motion could be evaluated in the FE simulations. In the experiments, the pin scarred the brain cortex; 1.2 mm (in average) superficially (up to 1 mm from the surface) and less centrally. In two of these cases (RKi301 and RKi302), the length of the scurs at 0.5 mm from the surface was slightly smaller than that at 1 mm. This suggests that, although there was certainly brain-slip, some adherence effects may be occurring in the interaction between the brain and the skull. In the simulation, this effect was qualitatively reproduced, but quantitatively overrepresented, showing adequate brain displacements (1.1mm and 0.7mm) in the closest and furthest location to the brain surface (0.5 mm and 2 mm), but higher displacements (1.5mm and 1.3mm) more centrally (at 1 mm and 1.5mm). We hypothesize that this phenomenon may be affected by the lack of validation of the brain tissue material model against shear experiments, which may affect the ability of the model to simulate shear efforts.

The experimental method itself did not allow identifying if the higher amount of brain travelling occurs during the loading or during the unloading phase. However, the total brain displacement measured in the experiments in combination with the simulation work presented allowed us to analyze the brain cortex kinematics. According to our results, under the rotational loading conditions applied, during the rotation backwards of the head, the brain lagged behind the skull reaching a maximum relative displacement of up to 0.8mm at approximately 0.5ms. From then, the brain tends to travel back with respect to the skull, reaching the approximate initial position at about 1.5ms. At this stage, the skull is already being subjected to flexion downwards, while the brain keeps on travelling backwards with respect to the skull. Around 2.1ms after the initiation of trauma, the brain cortex reaches its maximum displacement with respect to the skull and suffers the highest displacement back.

Previous rotational trauma experimental series conducted showed DAI in the regions around Corpus callosum and Hippocampus. By simulating these experiments with the new FE model presented in this article, we could qualitatively verify the correspondence between the areas of highest strain concentrations in the FE model with the areas in which DAI were detected. This remarks the potential of the model to support experimental investigations towards the clarification of DAI mechanisms.

5. Limitations

In the experimental method presented, the needle penetrates the brain cortex to an approximate depth of 2.5 mm prior to trauma. During trauma, the brain travels against the sharped needle, provoking the scar. In this study, we worked on the hypothesis that the force induced by the stiff needle onto the soft brain tissue is not affecting the natural motion of the brain if compared to the same loading conditions when no needle is inserted. However, this requires further verification, since it may be affecting our results.

The brain tissue indentation experiments from literature (24) used to define the material properties of the brain FE model were conducted in compression at relatively low strain rates. In addition, the inability to record data during the ramp portion of the tests makes these data only usable for tissue strain rates above 100 ms, which does not necessarily capture the high-rate mechanical response of brain tissue at the rates used in the trauma experiments. To account for this limitation, further testing accompanied by simulation based sensitivity analysis need to be conducted.
6. Conclusions

An experimental method has been used to evaluate the relative displacement of the brain cortex with respect to the skull under injurious rotational loading with rats. The pattern observed in the three specimens showed consistency and repeatability. A maximum brain-skull relative displacement of 1.4 mm has been captured under head average rotational peak accelerations of 1.67 Mrad/s². In all cases, the maximum scratch length was found at 1 mm or less from the brain cortex upper surface.

The new experimental data have been used to validate the local brain cortex kinematics of a novel anatomically detailed rat brain FE model. Simulated rotational acceleration-deceleration trauma shows that the brain inside the skull is subjected to maximum strains in the late stage of the rebound phase. In addition, the model is able to reproduce concentration of strains in the areas where DAI was produced by rotational experiments.

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