Numerical Investigation of Rotational Head Impacts in Atrophic Alzheimer's Brains Using Finite Element Analysis

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ABSTRACT

Traumatic brain injury (TBI) is a critical concern in elderly populations, particularly in individuals with Alzheimer's disease (AD), where brain atrophy may influence injury outcomes. This study employed finite element analysis to investigate the effects of rotational head impacts on two brain models: one representing healthy aging with a 0.5% annual atrophy and another representing AD with a 4% annual atrophy.

The peak normal stress in the frontal region was slightly lower in the AD model (0.24803 MPa) compared to the healthy aging model (0.25025 MPa), likely due to increased cerebrospinal fluid thickness. However, the first principal strain at the end of the simulation was higher in the AD model (0.1222) than in the healthy aging model (0.1171), indicating greater tissue deformation. These findings highlight that while atrophy-related CSF increases can reduce stress, they may also enhance relative motion between brain and skull, potentially increasing the risk of injury.

Future studies should incorporate patient-specific atrophy patterns and various impact scenarios to better understand brain vulnerability in neurodegenerative conditions.

Keywords: *Alzheimer's disease, Brain atrophy, Traumatic brain injury (TBI), Fluid-Structure Interaction (FSI), Computational modeling, Subdural hematoma.*

1. *INTRODUCTION*

Alzheimer's disease (AD) is a debilitating neurodegenerative condition characterized by a progressive decline in cognitive function and significant structural alterations within the brain [1-3]. One of the hallmark features of AD is the pronounced loss of brain tissue commonly referred to as brain atrophy which results from the accumulation of amyloid-beta plaques, tau-related neurofibrillary tangles, and subsequent neuronal degeneration [1-5]. This gradual loss of brain volume contributes to the deterioration of cognitive and motor functions, ultimately impairing the quality of life of affected individuals. Reports in the literature indicate that annual rates of brain atrophy in AD patients typically range between 1% and 4% [6-8], which is substantially higher than the relatively modest decline of approximately 0.2% to 0.5% per year observed in healthy aging populations [9-14].

As populations age, the prevalence of both AD and traumatic brain injury (TBI) increases, with older adults exhibiting heightened vulnerability to external mechanical forces due to age-related structural changes in the brain [15]. Importantly, brain atrophy may not only affect cognitive abilities but may also influence the mechanical response of the brain to external impacts. The reduction in brain volume associated with AD leads to an enlargement of the subdural space between the brain and the skull, potentially modifying the way mechanical loads are transmitted during traumatic events such as falls or vehicular collisions [15-16]. Consequently, understanding how progressive atrophy alters the brain's mechanical environment under impact conditions is essential for improving injury prevention strategies and informing protective measures for at-risk populations.

Finite element (FE) modeling has emerged as a valuable computational tool for analyzing the mechanical behavior of biological tissues under different loading scenarios [17]. By incorporating detailed anatomical structures and realistic material properties, FE models can simulate the brain's response to dynamic loads with high fidelity. Recent advancements in medical imaging, image segmentation, and computational techniques have facilitated the development of subject-specific and disease-specific FE brain models that account for individual variations and pathological conditions [17-20]. Such models provide unique opportunities to investigate how diseases like AD may influence injury risk and to explore the underlying biomechanical mechanisms that could exacerbate or mitigate brain injuries.

The primary objective of this study was to employ finite element modeling to investigate the biomechanical effects of progressive brain atrophy in Alzheimer's disease on the brain's response to rotational head impacts.

2. MATERIALS & METHODS

2.1. Brain Geometry and Atrophy Modeling

In this study, two finite element brain models were developed based on different neurological conditions: normal aging and Alzheimer's disease (AD). The healthy aging model incorporated a 0.5% annual brain volume reduction, while the AD model included a 4% annual volume reduction to simulate accelerated atrophy.

Table 1 summarizes the scale factors applied to each model, along with the corresponding brain volumes and volume changes relative to the initial baseline. The scale factors were implemented uniformly across the brain geometry using COMSOL Multiphysics to simulate atrophy-induced shrinkage.

Table 1. *Summary of brain volume scaling applied to simulate atrophy in normal aging and Alzheimer's disease models after one year, showing scale factors and resulting brain volumes.*

Model No.	Neurological Condition	Time	Scale Factor	Brain volume cm^3	Volume Change Compared to Initial (%)
0	Healthy Young Brain	0	1	1453.2	0
1	Healthy Aging	1 Year	0.998330	1445.9	0.50
2	Severe AD	1 Year	0.986484	1395	4.00

2.2. Finite Element Model and Material Properties

The finite element model included the brain parenchyma, CSF, and skull. The mechanical properties assigned to each component are summarized in Table 2.

- *Brain Tissue was modeled as a viscoelastic material to capture its time-dependent mechanical behavior under dynamic loading. It was assigned a density of 1040 kg/m³, a Young's modulus of 0.0228 MPa, and a Poisson's ratio of 0.49. To represent its complex stress-relaxation behavior, a linear viscoelastic formulation with six relaxation moduli and corresponding relaxation rates was applied.*
- *CSF was modeled as an incompressible fluid-like material with a density of 1000 kg/m³.*
- *Skull Bone and Face Bone were treated as elastic structures with mechanical properties consistent with published data.*

Table 2. *Mechanical properties of the anatomical components included in the finite element brain model. Values are based on established biomechanical literature [20].*

Components	Behavior	Density (kg/m³)	Young's Modulus (MPa)	Poisson's Ratio	Viscoelastic Parameters (G, Pa)	Relaxation Rates (β, 1/s)
Skull Bone	Elastic	2120	7000	0.22	-	-
Face Bone	Elastic	1060	500	0.22	-	-
CSF	Fluid	1000	-	-	-	-
Brain	Viscoelastic	1040	0.0228	0.49	G1=3.1×10^5, G2=7.8×10^4, G3=6.2×10^3,	β1=1×10^6, β2=1×10^5, β3=1×10^4,

	G4=8×10^3, G5=1×10^3, G6=3×10^3	β4=1×10^3, β5=1×10^2, β6=1×10^1

2.3. Impact Loading Conditions

The initial finite element brain model used in this study was previously validated in multiple biomechanical investigations [20-21]. Building upon this validated baseline, rotational head impacts were simulated in the sagittal plane to replicate common injury mechanisms observed in frontal motor vehicle collisions. A peak translational acceleration of 450 g and a peak rotational acceleration of 26.2 krad/s² were applied, consistent with experimental kinematic data from Depreitere et al [22-23]. These accelerations were imposed at the head's center of gravity, combining clockwise rotation around the x-axis with translational movement along the z-axis to recreate a realistic impact scenario (see Figure 1 for the model geometry, finite element components, and loading conditions).

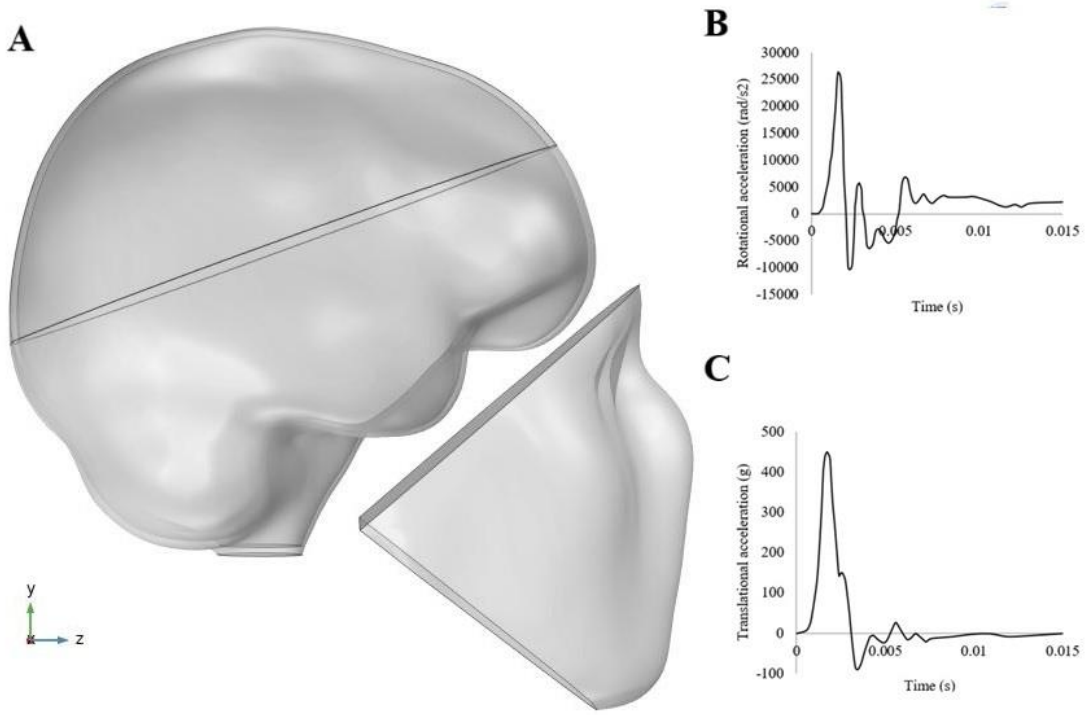

Fig. 1. *(A) Finite element brain model geometry illustrating major anatomical components, including the cerebrospinal fluid (CSF), and brain tissue. The applied impact loading conditions depict (B) rotational and (C) translational accelerations imposed at the head's center of gravity, simulating a sagittal plane rotational head impact scenario.*

3. RESULTS

The finite element simulations conducted in this study compared the biomechanical response of the healthy aging brain model (0.5% atrophy) with that of the atrophic Alzheimer's disease (AD) model (4% atrophy).

In the frontal region, the first principal strain—indicative of tissue deformation—was evaluated at the final time point of the analysis (0.015 s). The healthy aging model exhibited a maximum first principal strain of 0.1171, whereas the Alzheimer's disease model reached a slightly higher value of 0.1222. This difference suggests an increased susceptibility to deformation under impact loading in the atrophic brain.

The normal stress in the frontal region, representing the peak compressive stress experienced by the brain tissue, was recorded at 0.0017 s, which corresponds to the moment of maximum loading in the simulation. The healthy aging model exhibited a peak normal stress of 0.25025 MPa, while the Alzheimer's disease model displayed a marginally lower value of 0.24803 MPa.

In the occipital region, normal stress also peaked at 0.0017 s. The healthy aging model exhibited a compressive stress of -0.22769 MPa, whereas the Alzheimer's disease model showed a slightly less negative value of -0.22573 MPa.

Figure 2 illustrates the distribution of normal stress in the frontal region of the brain in both models at the time of peak stress, highlighting regional variations in tissue loading between the two atrophy scenarios. Figure 3 presents the temporal evolution of pressure in the frontal and occipital regions across the entire duration of the simulation, providing insight into the dynamic response of brain tissue under impact conditions.

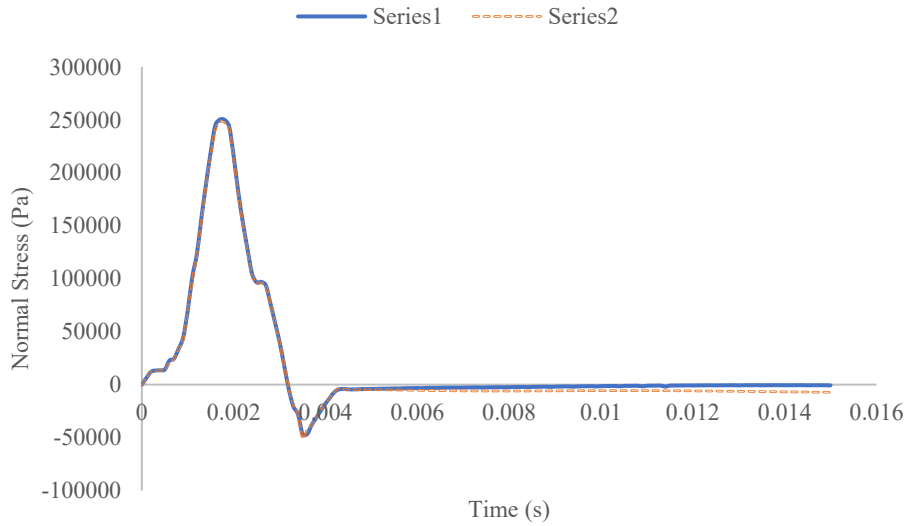

Fig. 2. *Distribution of normal stress in the frontal region of the brain for both the healthy aging and Alzheimer's disease models at the time of peak stress. This figure highlights regional variations in tissue loading between the two atrophy scenarios.*

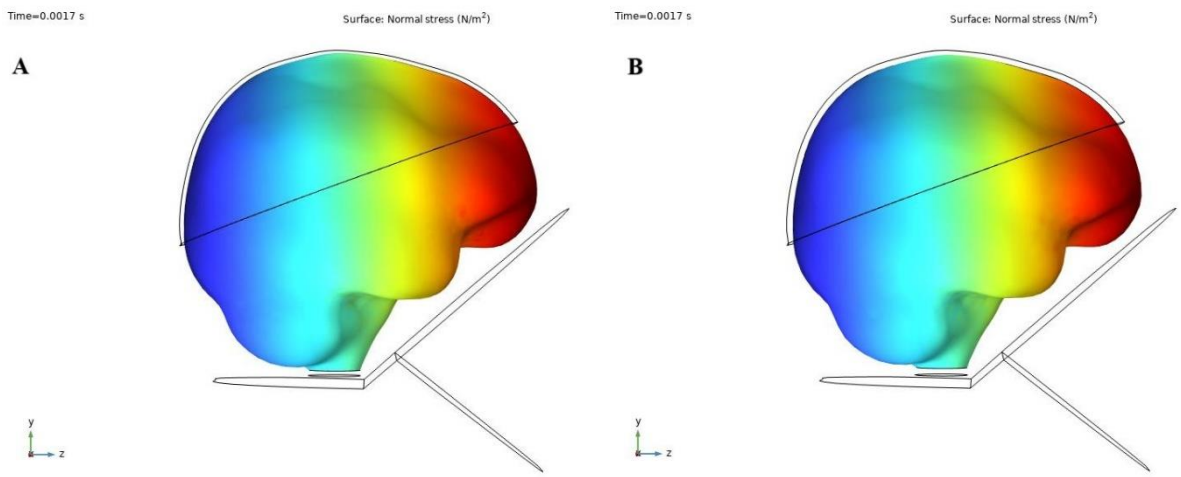

Fig. 3. *Temporal evolution of pressure in the frontal and occipital regions throughout the simulation, comparing (A) the healthy aging model and (B) the Alzheimer's disease model. The plots provide insight into the dynamic response of brain tissue under impact conditions.*

These results demonstrate the influence of progressive atrophy on the biomechanical response of the brain, with potential implications for the increased vulnerability of atrophic brains to traumatic injury.

4. DISCUSSION

The results of this study provide valuable insights into the biomechanical response of atrophic brain tissue under rotational impact loading. In the frontal region, the first principal strain increased from 0.1171 in the healthy aging model to 0.1222 in the Alzheimer's disease (AD) model, representing an approximate 4.35% increase in strain. This finding suggests that progressive atrophy in the AD model increases the tissue's susceptibility to deformation under dynamic loading.

Conversely, the normal stress in the frontal region decreased slightly, from 0.25025 MPa in the healthy aging model to 0.24803 MPa in the AD model a reduction of approximately 0.89%. This reduction can be attributed to the increased thickness of the CSF layer surrounding the atrophic brain, which acts as a protective cushion that dissipates impact forces.

However, previous studies, such as the work by Abdi et al. 2023 and Zhou al. 2019, have demonstrated that increased CSF thickness due to atrophy may also facilitate greater relative motion between the brain and skull, potentially leading to an elevated risk of traumatic brain injury. This finding aligns with our observation of increased strain in the atrophic model, underscoring the complex interplay between mechanical protection and vulnerability in atrophic brains.

In the occipital region, the normal stress decreased from -0.22769 MPa in the healthy aging model to -0.22573 MPa in the AD model a reduction of approximately 0.86%. While the magnitude of change was modest, it reflects the consistent trend of stress reduction due to the CSF buffering effect.

This study has several limitations. First, the finite element model focused primarily on the brain tissue, CSF, and skull; other important structures, such as blood vessels and meninges, were not explicitly modeled, which may influence the dynamic response. Second, the material properties assigned to the brain tissue, while based on established data, represent generalized values and may not capture all patient-specific variations. Third, the impact loading scenario, although consistent with experimental data, represents a simplified injury mechanism and may not encompass the diversity of real-world impact conditions.

Future studies should incorporate more detailed anatomical structures, including bridging veins and vascular networks, to better understand their role in strain and stress distribution. Additionally, patient-specific material properties derived from imaging data could improve model fidelity. Further investigation into the coupling of increased CSF thickness with relative brain-skull motion especially under various loading directions would help clarify the balance between mechanical protection and vulnerability in atrophic brains

5. CONCLUSION

This numerical study provided insights into the mechanical response of the brain under rotational head impacts by comparing healthy aging and Alzheimer's disease (AD) models featuring different atrophy rates. Our results demonstrate that despite a decrease in peak normal stress in the atrophic brain model attributable to the increased thickness of the cerebrospinal fluid layer the first principal strain was slightly higher in the AD model compared to the healthy aging scenario, indicating potential for increased vulnerability to tissue deformation.

These findings highlight the complex role of brain atrophy in modulating stress-strain relationships and injury mechanisms. Future studies should incorporate subject-specific atrophy patterns, different impact scenarios, and additional biological factors such as tissue heterogeneity and microstructural changes to enhance the accuracy and clinical relevance of finite element head models.

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