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Nonlinear Dynamical Behavior of the Deep White Matter During Head Impact

Javid Abderezaei,¹ Wei Zhao,² Carissa L. Grijalva,³ Gloria Fabris,¹ Songbai Ji,² Kaveh Laksari,³ and Mehmet Kurt^{1,4,*}

¹*Department of Mechanical Engineering, Stevens Institute of Technology, Hoboken, NJ, USA*

²*Department of Biomedical Engineering, Worcester Polytechnic Institute, Worcester, MA, USA*

³*Department of Biomedical Engineering, University of Arizona, Tucson, AZ, USA*

⁴*Translational and Molecular Imaging Institute, Icahn School of Medicine at Mount Sinai, New York, NY, USA*

Traumatic brain injury (TBI) is a major public health concern, affecting as many as 3 million people each year in the US. Despite substantial research efforts in recent years, our physical understanding of the cause of injury remains rather limited. In this paper, we characterized the nonlinear dynamical behavior of the brain-skull system through modal analysis and advanced finite element (FE) simulations. We observed nonlinear behavior in the deep white matter (WM) structures near dural folds with an energy redistribution of around 30% between the dominant modes. We found evidence of shear wave redirection near falx and tentorium (approximately 15° in the axial and 8° in the coronal plane) as a result of geometric nonlinearities. The shift in the falx modeshape which was perpendicular to the brain's deformation caused geometrical nonlinear effects at the falx-brain tissue boundary. This was accompanied by a lateral sliding of tentorium below the brain tissue which induced higher local strains at its interface with deep regions of the brain. We observed that deep regions of the brain with high principal strains coincided with the identified nonlinear regions. The onset of nonlinear behavior in brain tissue is closely tied with the previously reported concussion thresholds, suggesting a possible link between the damage mechanism and the underlying nonlinear brain biomechanics.

I. INTRODUCTION

Traumatic brain injury (TBI) is a major cause of death in the US, with at least 2.8 million people diagnosed annually [1] and about twice as many unreported cases, especially among athletes [2–4]. In order to develop diagnostic and preventive strategies against this growing epidemic, it is imperative to develop a deep physical understanding of the mechanisms of brain injury. Evaluating the patterns of brain movement and deformation during a head impact has long been one of the most powerful strategies for attempting to predict brain damage. The first efforts to understand the underlying mechanics of the brain date back to 1943, when Holbourn proposed to model the brain as a mechanical system, and studied the relation between different head kinematic inputs and deformation metrics. He hypothesized that rotational rather than translational acceleration is the dominant cause of larger strains in the brain [5, 6]. Kornhauser proposed a second-order mass-spring system to analyze the brain mechanically, and suggested that as the brain deformation surpasses a specific threshold, it can result in injury [7].

One direct benefit of better understanding the dynamics of brain-skull system would be to come up with useful clinical injury metrics for defining the severity of TBI. Many scientists have focused on developing such criteria [8], either based on kinematics (*e.g.* head injury criterion (HIC) [9, 10]) or on brain FE models, (*e.g.* cumulative strain damage measure (CSDM) [11]). However, these

scalar measures treat the whole-brain as a single unit, and lack the sufficient mechanical and dynamical understanding of the characteristics of the brain-skull interface. Hence, the question remains as to why specific anatomical structures, notably brainstem and corpus callosum (CC), reveal a higher susceptibility to strain [12–14].

A crucial point to consider is that the brain is a complex biomechanical system with an intricate geometry, non-uniform inter-facial boundary conditions, and significantly inhomogeneous and nonlinear material properties [15, 16]. This complexity can result in nonlinear effects, which are a common feature in dynamical systems. Previous studies especially in the structural dynamics community have shown that such nonlinearities can lead to many interesting phenomena, such as energy localization [17, 18], targeted energy transfer [19–21] and nonlinear modal interactions [22, 23]. To address the lack of physical understanding of the brain, a recent study from Laksari *et al.* analyzed the sensitivity of the human brain to deformation and found the localized modes and multimodal behavior - a common characteristics of a nonlinear system - in deep regions of this tissue [15]. Others also hinted at nonlinear behavior around the gray-white matter junction and CC. Sabet *et al.* reported disrupted strain fields at those regions through tagged MRI imaging, when they evaluated the deformation of the brain during a rotational acceleration [13]. Further analysis of the brain showed that localized strain concentrations occur at different regions of a linear and nonlinear viscoelastic medium during impact to the head [24, 25]. Gurdjian *et al.* found that the concentration of shear strains are in the vicinity of the CC and brainstem, which interface with stiff ventricular or membranous structures [14] such as the falx cerebri and tentorium cerebelli; these

* Corresponding author.
mkurt@stevens.edu

membranes also play a role in mechanical response of the brain, by reflecting or redirecting shear waves [26].

In this paper, we used a brain FE model to simulate brain's response under various rotational accelerations. By using a modal decomposition technique, we characterized the dynamical response of the brain and identified its patterns of regional and overall nonlinearity. We then identified potential anatomical features which contributed to the observed nonlinear behavior. Finally, we investigated how these distinct responses correlated with a brain injury metric, *i.e.* CSDM and its implications for mild TBI (mTBI).

II. METHODS

Brain responses were simulated using the Worcester Head Injury Model (WHIM; [16, 27]). WHIM is an FE brain injury model, where the brain tissue is modeled as an isotropic, homogeneous material using a second-order Ogden hyperelastic model (Figure 1(a); see Supplemental Material [28]). We analyzed brain's deformation under a range of coronal rotational accelerations that contain kinematics shown to cause concussive injury [29, 30]. We chose coronal plane, since it has been shown that rotations in this direction can cause large strains in CC [12, 31], which is one of the structures often used to predict mTBI [8, 32, 33]. We applied a half-sine acceleration impulse (Figure 1(b)) to the center of gravity of the head by varying the amplitude α between $1-10 \text{ krad/s}^2$ and duration Δt between $5-25 \text{ ms}$ (with increments of 1 krad/s^2 and 5 ms , respectively).

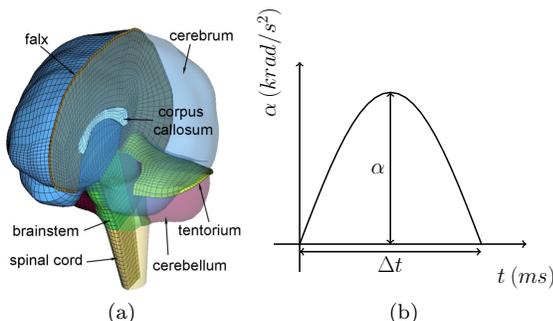


FIG. 1. Finite element simulation of the brain using WHIM. a) Brain anatomical features in WHIM model [27]. b) Half-sine rotational acceleration pulse with varying amplitude ($\alpha = 1 - 10 \text{ krad/s}^2$) and duration ($\Delta t = 5 - 25 \text{ ms}$), imposed to the center of gravity of the head.

Having simulated the brain responses for various accelerations, we implemented modal analysis decomposition to understand the important features of the brain dynamics. Modal analysis is a widely used method in structural dynamics with applications in damage detection [34–36], dynamic characterization [37, 38] and model-order reduction [39, 40]. In this study we used proper orthogonal decomposition (POD), a modal analysis technique that

extracts the modes from a system by performing an order reduction to transform a high-dimensional data-set into a lower degree of freedom one to obtain its relevant but unexpected hidden behaviors [41–43]. In order to apply POD, we consider a temporal sequence of the brain's nodal relative displacement fields $u_{brain}(x, y, z, t)$, where x, y, z are the spatial location of each node considered at time t . For any real u_{brain} in the form of an $m \times n$ matrix, there exists a factorization called singular value decomposition (SVD) that can be written [41, 44]:

$$u_{brain} = USV^T \quad (1)$$

where $U = [v_1, v_2, \dots, v_m]$ is an $m \times m$ orthonormal matrix containing the left singular vectors v_i which correspond to proper orthogonal modes (POMs); V is an $n \times n$ orthonormal matrix containing the right singular vectors. S is an $m \times n$ diagonal matrix of real and non-negative diagonal entries, containing the singular values σ_i which correspond to the portions of the energy of POD modes (see Supplemental Material [28]).

In order to analyze how the modeshape of various structures of the brain tissue changes with increasing acceleration, the first non-dimensional modeshape corresponding to $\alpha = 1 \text{ krad/s}^2$ and $\Delta t = 5 \text{ ms}$ (minimum rotational acceleration of the simulations) was defined as the linear baseline of the system. To quantify the percentage change of the i -th POM at various α and Δt compared to the i -th POM of the linear baseline, the following formulation was introduced:

$$\Delta v_i = \left| \frac{v_i(x, y, z, \alpha, \Delta t) - v_i(x, y, z, \alpha_{ref}, \Delta t_{ref})}{v_i(x, y, z, \alpha_{ref}, \Delta t_{ref})} \right| \times 100 \quad (2)$$

where $v_i(x, y, z, \alpha, \Delta t)$ is the POM of the system at $\alpha = 1 - 10 \text{ krad/s}^2$ and $\Delta t = 5 - 25 \text{ ms}$, and $v_i(x, y, z, \alpha_{ref}, \Delta t_{ref})$ corresponds to the linear baseline POM of the system.

III. RESULTS

Having simulated the relative displacement of the global brain-skull system for each coronal rotational acceleration, we performed POD on this data to find the corresponding POMs of each acceleration as well as their contribution to the energy of the system (Figure 2). With changing duration and amplitude, we found a minor change of 2.7% in the energy contribution of the dominant POM of the whole brain (Figure 2(a)), which indicates a weak global nonlinear dynamical behavior. This was also confirmed by the high Pearson's correlation coefficient (PCC = 0.93) between the dominant modeshapes of the brain corresponding to $\alpha = 1 \text{ krad/s}^2$, $\Delta t = 5 \text{ ms}$ and $\alpha = 10 \text{ krad/s}^2$, $\Delta t = 25 \text{ ms}$ (Figure 2(b)). The negligible variation in the energy contribution and the high PCC between modeshapes of the brain at various

accelerations indicate that, in spite of the brain's intricate geometry and material nonlinearity, this system *as a whole* behaves linearly in the aforementioned acceleration levels.

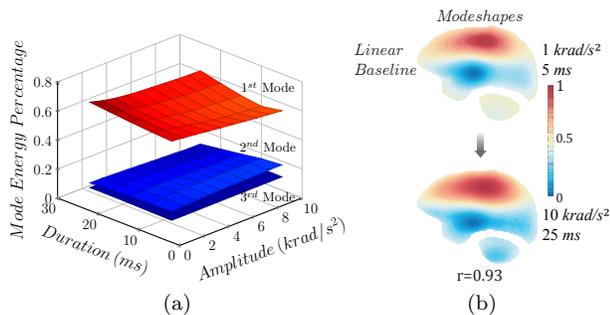


FIG. 2. Brain tissue does not exhibit global nonlinear behavior: (a) Energy contribution of the first three POMs corresponding to varying amplitude and duration in coronal plane. Negligible change of 2.7% in the energy redistribution of the first POM is an indication of weak global nonlinearity. (b) High correlation between the brain's dominant modeshape at minimum and maximum acceleration level. A mid-sagittal section is shown as a reference.

One possible explanation behind this globally linear dynamical behavior despite many sources of nonlinearities could be the localization of nonlinear effects in certain brain regions. In order to test this hypothesis, we conducted POD on various brain substructures. We compared the change of the dominant modeshape (v_1) at different α and Δt with the linear baseline to find the local nonlinear dynamics in the brain. We observed that as the acceleration level increased, the dominant modeshape of the regions surrounded by deep white matter (WM), near the falx cerebri and tentorium cerebelli varied more than 50% (Figure 3(a)). To further analyze these particular regions, we also evaluated the change of energy contribution of the first three modeshapes (Figure 3(b)). We found that as the acceleration increased to $\alpha = 10 \text{ krad/s}^2$ and $\Delta t = 25 \text{ ms}$, the energy level of the dominant POM of local nonlinear regions in the deep WM decreased by approximately 30%. These results hint at the dependence of modeshape and its energy contribution on the input excitation level, which is a common indicator of the nonlinear effects in a system [41]. We also compared the volume of the identified nonlinear region at various acceleration levels with the volume of the WM and observed that at the maximum simulated acceleration, the nonlinear region approximately covers 25% of the WM (Figure 3(c)). Furthermore, we analyzed this nonlinearity ratio with respect to the concussion threshold suggested by Margulies and Thibault [45] (a criterion developed based on Kelvin-voigt model which was later confirmed as a tolerance curve for mTBI [46]) and by Ommaya [47] (who extrapolated this threshold from primate experiments). These concussion thresholds, as adapted from their original form in velocity-acceleration curves, seem to lie within the transition range from the linear to

the nonlinear regime. Intriguingly, this could suggest a possible correlation of brain nonlinearity with the onset of mTBI.

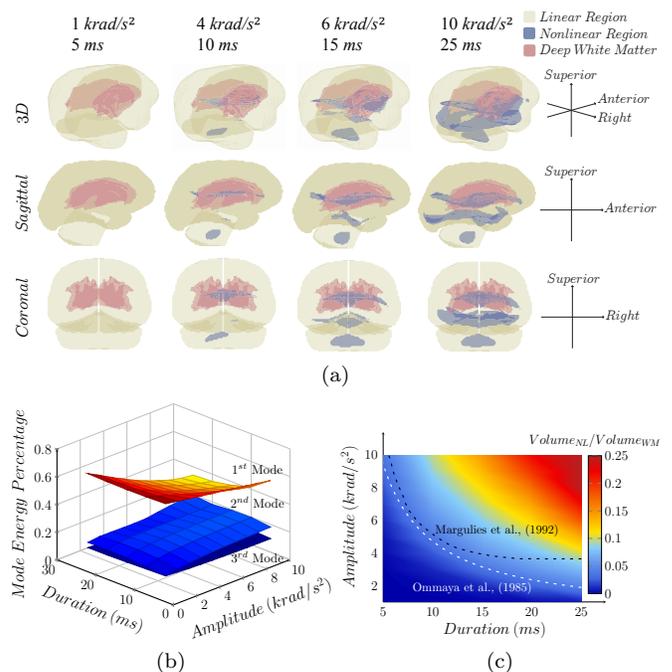


FIG. 3. Existence of local nonlinearity in the human brain: (a) By varying the acceleration amplitude and duration in the coronal plane, the normalized dominant modeshape ($v_1(x, y, z, \alpha, \Delta t)$) corresponding to each acceleration level is compared with the linear baseline ($v_1(x, y, z, \alpha_{ref}, \Delta t_{ref})$) and a nonlinear region is identified ($\Delta v_1 > 50\%$). (b) Energy redistribution of approximately 30% in the first three POMs of nonlinear regions in the deep WM is an indication of local nonlinearity. (c) Nonlinear region covers approximately 25% of the WM. Comparison of concussion thresholds with nonlinear region volume suggests a possible link with injury mechanism.

Having identified the structures exhibiting nonlinear dynamical effects in the brain tissue, in the next step, we applied POD to the dural folds in order to individually analyze the dominant modeshape of the falx cerebri and tentorium cerebelli (Figure 4). We observed that at the maximum acceleration level (compared to the linear baseline) the dominant modeshape of these structures changed its direction in axial and coronal plane, by $\Delta\theta \approx 15^\circ$ and $\Delta\varphi \approx 8^\circ$, respectively (see Supplemental Material [28], Table S1). Such a variance in the direction of modeshape of the dural folds could be a possible indication of their involvement in causing local nonlinear effects in the brain tissue.

Due to the idealized inputs used in our simulations, a natural follow-up question is whether such nonlinear effects are present in more complicated impact scenarios. To test this, we simulated a 6 degree-of-freedom (DOF) real-world head impact with a dominant rotational acceleration in the coronal direction [8]. We implemented

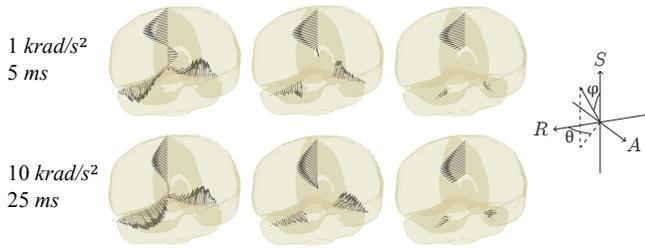


FIG. 4. Effect of falx and tentorium on shear wave redirection: The dominant modeshape of falx and tentorium for $\alpha = 1 \text{ krad/s}^2$, $\Delta t = 5 \text{ ms}$ and $\alpha = 10 \text{ krad/s}^2$, $\Delta t = 25 \text{ ms}$ shows that as the rotational acceleration increases, there is a change in the direction of these two structures of the brain, which in turn can cause shear wave reflection in its surrounding area. Three different sections of falx and tentorium are shown for reference.

previously published head kinematics (Figure 5(a)) of a football athlete who was equipped with an instrumented mouthguard [48] during a game in which he suffered from loss of consciousness (LOC). After applying POD, we found that the regions located near the CC and dural folds exhibit nonlinear effects, as in the case of idealized simulations (Figure 5(b)).

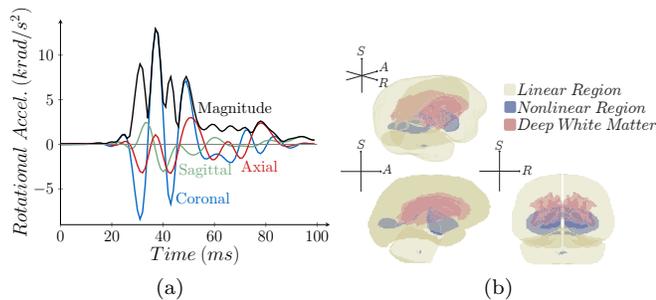


FIG. 5. Nonlinear regions of the human brain due to a real-world football head impact that led to LOC: a) Recorded 3D rotational acceleration of the football athlete who suffered LOC injury [8]. b) POD of the brain deformation showed nonlinear regions similar to the ones in an ideal coronal rotation.

Having observed such a phenomenon in the human brain's dynamical response, it is essential to examine the implications of these nonlinear effects in the existing injury metrics. To do so, we studied the correlation of the nonlinear regions with $\text{CSDM}_{0.35}$, a FE injury criteria which describes the total volume fraction of the brain that experiences strain levels larger than 35% [11, 49]. Interestingly, we observed that other than the expected high strain levels near the cortex, the brain tissue volumes which undergo such high strains are the regions connected to the nonlinear area (Figure 6(a)). This could indicate a physical correspondence between the nonlinear regions and high strain areas in this soft tissue. Following that, we analyzed whether the nonlinear regions correlate with the fiber tracts in the brain known to be the most

vulnerable to injury [50, 51]. We observed that the identified nonlinear regions overlap with the several commonly implicated WM tracts to TBI (Figure 6(b), [50]). These results could suggest a correlation between nonlinearity in the human brain tissue and TBI pathology.

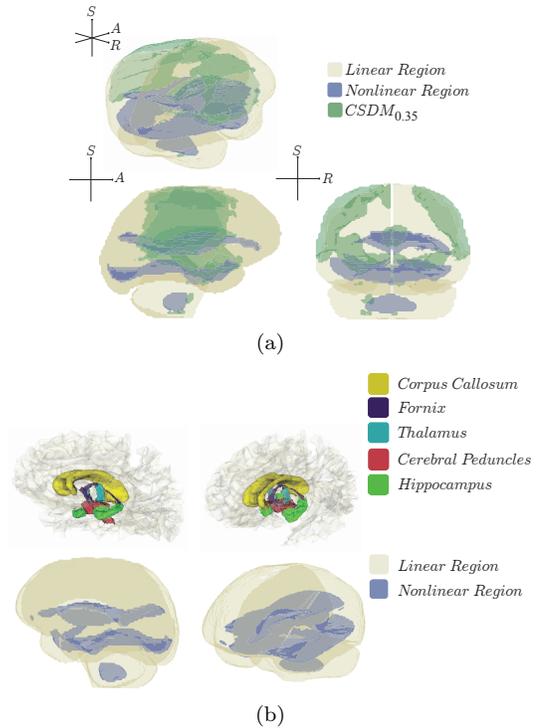


FIG. 6. Correspondence of nonlinear regions with tissue-level TBI metrics: a) We observed that high strain regions ($\text{CSDM}_{0.35}$) of the brain follows the nonlinear regions over tentorium, signaling a physical correspondence. b) The nonlinear regions entail several WM tracts of interest in TBI pathology.

IV. DISCUSSION

A comprehensive study of the mechanistic behavior of the brain is a necessity in order to better understand the underlying mechanisms of TBI. In this study, we found that with increasing excitation levels in the form of rotational accelerations to the center of the head, brain tissue structures within the deep WM as well as regions near the falx and tentorium, exhibit nonlinear dynamics. Such local nonlinearities take shape due to the brain's complex geometry, inhomogeneous material properties and especially its boundary condition with the skull and dural folds [15, 16, 52, 53]. Previous studies have shown how these stiff membranous structures (*i.e.* falx and tentorium) affect brain's deformation during head impacts by inducing large strains to the surrounding regions such as CC and brainstem by constraining the motion of the cerebellum [54, 55]. Zhang *et al.* hypothesized that in a coronal rotation, regions above the brainstem experience

high shear strains during lateral movement of the brain over tentorium [56]. Lu *et al.* also used tagged MRI to measure regional deformation of the brain *in vivo* as a result of head rotation. They observed that the propagated shear waves from exterior boundary (*i.e.* skull) is reflected as it reaches the falx and tentorium [57]. Given the potential influence of these anatomical features on the brain response, we further evaluated their effects on the initiation of the identified nonlinear regions. It became apparent that, as the input acceleration increases, there is a significant change in the direction of the dominant modeshape of falx and tentorium, which is due to their nonlinear geometry. Here we observed that the shift in falx modeshape is toward superior-inferior direction which is perpendicular to the right-left deformation of its surrounding tissue. This can cause geometric nonlinearity at the boundary of the falx and brain tissue. Tentorium modeshape on the other hand, has a change in its direction towards right-left, which can result in its lateral sliding below the soft tissue. This imposes higher strains at its interface with the brain compared to the surrounding regions of the tissue. Others have found similar results near the dural folds. Recently, Okamoto *et al.*, induced external harmonic vibrations to the human skull to obtain the dynamics of the brain *in vivo*. In this study they used magnetic resonance elastography (MRE) [58], a technique based on the imaging of shear wave propagation in tissue as a result of external actuation [59, 60]. They analyzed 15 human subjects and observed shear wave reflection/redirection near falx and tentorium. They also found approximately 9% difference between the cerebrum and cerebellum motion in AP direction which could induce higher strains around tentorium [58]. Such high strains at the boundary of these membranes have also been observed in other TBI studies, where bleeding as the result of subdural hematoma is frequently present along the falx and above tentorium [61].

This observed nonlinear behavior in the human brain might have important clinical relevance. The onset of nonlinear behavior (Figure 3(c)) seems to have coincided with the previously reported concussion thresholds by Margulies and Thibault [45] and by Ommaya [47], suggesting a possible mechanism of injury. Similar to their observations [45], we have found that in low duration regime the nonlinear region is less sensitive to impact amplitude and varies mostly with duration; whereas, in high duration this region is more sensitive to input amplitude and less dependent on the increase of duration. Another clinical implication of our results was demonstrated in a study by Hulkower *et al.*, who implemented diffusion tensor imaging (DTI) to identify fractional anisotropy (FA) abnormalities in the brains of patients who suffered TBI. They showed that WM tracts such as CC (including genu, splenium and body) as well as others like fornix, thalamus, cerebral peduncles and hippocampus are among the most common brain regions with abnormal FA in the TBI patients [50]. Hernandez *et al.*, also

identified peak principal strain in the CC as the best indicator of mTBI after a head impact [8].

Given the intricacies involved in the brain-skull system, our study inevitably comes with several limitations that should be addressed. Inclusion of more anatomical details such as cortical gyri could potentially affect the modal behavior of the tissue. This could change the energy contribution of the dominant POMs of the whole brain and make the global nonlinear behavior more pronounced. In addition, here we utilized an isotropic and homogeneous version of the WHIM, which could substantially limit the energy transfer between compression and shear waves, especially at the gray and white matter junctions. This approximation can also affect the shear wave redirection found at the interface with the dural folds and alter the patterns of nonlinearity. Nevertheless, an upgraded, anisotropic version of the WHIM [62] is already available to differentiate gray and white matter tissue properties, which could be applied to mitigate some of these limitations in the future. Another limitation of this study is lack of comparison with experimental data. In spite of the existence of promising imaging techniques to study brain motion, including magnetic resonance elastography (MRE) [59], amplified MRI (aMRI) [63], tagged MRI [64], and displacement encoded imaging with stimulated echoes (DENSE) MRI [65], measurement of brain deformation *in vivo* is still challenging. In a clinical setting, the range of acceptable actuation levels is rather narrow; therefore, only the linear response of the brain can be assessed, making a direct experimental comparison out of reach. An alternative approach is to study human cadaver head *in situ* under high excitation levels [66–68]. Such data sets provide valuable validation for the development of brain FE models (including WHIM [16]) which can therefore be used to simulate brain responses even at the high acceleration levels causing potential nonlinearity in the brain. Finally, it should be noted that although this study does not identify a conclusive link between brain tissue’s nonlinear behavior and the corresponding injury mechanism, we see our nonlinear analysis tool as an initial step in deciphering this link.

Considering the high number of TBI incidents [69, 70] and the importance of proper assessment of the severity of an injury, this study hints to the dire need of reevaluating how current brain injury criteria are developed. Existing deterministic tools such as BrIC, obtain injury risk curves based on the linear relationship that is present between BrIC and strain measures such as CSDM and maximum principal strain, respectively [71]. However, such criteria fail to take into account nonlinear effects present in the brain, which can cause higher strains in certain anatomical features. This is especially true in higher excitation levels, where regions within the deep WM and vicinity of dural folds exhibit characteristics common in a nonlinear system. Therefore, we hope this paper will encourage further studies on the contribution of certain brain substructures to the injury mechanism and their corresponding nonlinear dynamics.

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