Investigating the Role of Neck Muscle Activation and Damping Characteristics in Brain Injury Mechanism

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Abstract

Purpose: This study aimed to investigate the role of neck muscle activity and neck damping characteristics in traumatic brain injury mechanisms.

Methods: We used a previously validated head-neck finite element model, incorporating various components such as scalp, skull, cerebrospinal fluid, brain, neck muscles, ligaments, cervical vertebrae, and intervertebral discs. Loading conditions included a golf ball impact NBDL linear acceleration and Zhang's linear/rotational accelerations. Three muscle activation levels and variations in neck damping (viscoelastic intervertebral discs) were examined. Injury estimation employed Head Injury Criterion, Brain Injury Criterion, and maximum principal strain.

Results: In the NBDL acceleration scenario, increasing neck muscle activation consistently reduced HIC and BrIC values, with the most significant decrease occurring from no activation to medium-low activation. Neck damping had a moderate mitigating effect on HIC. The mean MPS decreased modestly with muscle activation, more substantially in the occipital region, and neck damping reduced MPS. In the Zhang scenario, HIC, BrIC, and MPS exhibited similar trends. Damping consistently decreased HIC and BrIC values. Golf ball impact scenarios showed increased risk of TBI, but higher muscle activation and neck damping reduced this risk. MPS analysis revealed nuanced impacts across brain regions, with neck damping showing substantial influence in the occipital region.

Conclusion: Our study underscores the pivotal role of neck muscle activation and damping in mitigating TBIs by countering impact forces. Our approach holds promise for advancing effective TBI prevention and protection strategies across diverse contexts.

Keywords: Simulation and modeling, Finite element method, Traumatic brain injury, Computational biomechanics, Neck damping, Active muscles

Statements and Declarations:

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Introduction

Traumatic brain injury (TBI) occurs when a sudden mechanical impact to the head or body leads to structural and functional changes in the brain [31]. It is a major public health concern worldwide, often referred to as the "silent epidemic" [37]. According to the Global Burden of Diseases, Injuries, and Risk Factors (GBD) report, the prevalence of TBI increased by 24.4% from 1990 to 2019, with an estimated 49 million cases globally in 2019. Among these cases, approximately 7.1 million represented years lived with disability (YLD) [18]. In the United States alone, the Centers for Disease Control and Prevention (CDC) Wonder database reported 69,473 TBI-related deaths in 2021 and 223,135 TBI-related hospitalizations in 2019 [34]. The leading causes of TBI are falls, followed by motor vehicle accidents, assault, and firearm-related injuries [9, 33, 35]. Sports (e.g., football, hockey, etc.) and recreational activities (e.g., bicycling, horse riding, and playground activities) are also leading causes of TBIs [20, 41]. According to a 2019 CDC report approximately 283,000 children (under the age of 18) visited the emergency department due to TBIs associated with sports and recreational activities [39]. Another study reports that more than 54% of concussions in Canada are sports related with sport having a role in more than 85% of concussions in the 16-34 age group [20]. The increasing prevalence of TBI underscores the need for comprehensive preventive measures and improved understanding of the mechanisms involved in brain injuries.

Based on injury characteristics, TBIs can be classified into two main categories: (1) focal and (2) diffuse injuries [46]. Focal injuries such as focal cortical contusion or intracerebral hemorrhage are localized brain injuries that predominantly caused by blunt trauma forces or sudden accelerations to the head [2, 29, 54]. Such blunt forces and/or accelerations cause the brain to undergo a high acceleration relative to the skull [2]. In general, this relative motion between skull and brain is delayed due to the presence of cerebrospinal fluid (CSF) around the brain during normal activity [21]. Furthermore, the brain gets pressed against the skull at the impact site, causing a positive pressure (coup), while generating a negative pressure (countercoup) on the opposite site of the impact. This coup-contrecoup mechanism primarily contributes to focal brain injuries, which are characterized by localized damage to a specific area of the brain [5]. On the other hand, diffuse injuries such as traumatic axonal injury and vasogenic edema predominantly occur as a result of acceleration forces rather than blunt impact forces [54]. When the head undergoes a rapid acceleration or deceleration, as seen in car accidents or falls, the brain experiences widespread damage throughout its structure. Diffuse injuries involve more extensive disruption of brain tissue and can affect multiple areas simultaneously [2, 29, 54]. Understanding the distinction between focal and diffuse injuries is crucial in comprehending the diverse mechanisms and manifestations of TBIs. It's worth noting that many TBI cases (e.g., motor vehicle accidents [1], falls [19],) involve both focal and diffuse injuries, for instance it has been reported in a study on the moderate and severe TBI that both focal lesions and DAI occur in 50% of the

patients [2, 42]. This highlights the complex nature of TBIs and the need for comprehensive diagnostic and treatment approaches to address their multifaceted effects on the brain.

Throughout history, researchers from both biomechanics and neuroscience domains have employed a range of experimental (both in-vivo and in-vitro studies) and computational methods to study the TBI mechanisms. In-vivo experimental studies primarily utilized quantitative video analysis [4] and wearable sensors [48] to understand the injury characteristics and TBI mechanisms based on head kinematics. However, these approaches are limited in their ability to measure internal parameters of the brain and other head components. On the other hand, in-vitro experimental studies used anthropometric test device [16] and/or cadavers [25], to explore the mechanical response of internal head components. Nevertheless, both experimental approaches may not fully capture the complexities of the living human body. Recognizing the importance of approaching TBI from a multi-faceted perspective, researchers have also investigated TBI mechanisms from a neuroscience standpoint [29, 53]. This complementary approach helps to better understand the underlying neural processes involved in brain injury, complementing the biomechanical insights. Clinical settings often rely on blood biomarkers or Glasgow coma scale for diagnosing TBI [27], while neuroscientists employ animal trials to study the biochemical effects of head impact. Various animal models, such as fluid percussion injury, controlled cortical impact injury, weight drop impact acceleration injury, and blast injury, have been used to explore TBI mechanisms from a biochemical perspective [53]. However, it is important to recognize that animal trials may not fully replicate TBI development in humans, which can explain the failure of promising drugs in clinical studies [29, 53]. Furthermore, these methods primarily focus on the biochemical reactions of the brain to standard impacts and do not provide sufficient insight into the mechanical response of the brain to real-life scenarios. To overcome the limitations of experimental methods, researchers have turned to computational approaches in TBI research. Notably, finite element (FE) models have gained popularity as powerful tools for studying TBI. These models can offer a highly detailed representations of the biomechanical behavior of head and neck structures, enabling the study of both external kinematics and internal responses of the brain and other head components during various impact scenarios [23]. Consequently, the FE method has become a very popular approach in gaining a comprehensive understanding of the biomechanics involved in TBI. By leveraging computational models, researchers can overcome the limitations of experimental methods and gain a more comprehensive understanding of the biomechanics involved in traumatic brain injury.

Despite an extensive body of literature on FE-based TBI studies, the specific roles played by cervical intervertebral discs, neck muscles, and ligaments in TBI mechanism remain unclear. Intervertebral discs provide damping for the head-neck system [36]. In a mechanical system, the purpose of a damper is to absorb the impact energy. Similarly, in the head-neck system, neck damping should be capable of absorbing the impact

energy, thereby reducing the load on other components, particularly the brain. Moreover, the neck muscles exert forces on the head-neck system, with the magnitude of these forces depending on the strength of the individual muscle and its level of activation [51]. Additionally, the direction of these forces relies on the muscle's role and position, whether it acts as a flexor or extensor. In a given impact scenario, the neck muscles are activated to counteract the external forces applied to the head-neck system, thereby mitigating the severity of the impact [11]. However, it is crucial to ensure correct muscle activation because incorrect activation may not only fail to protect against the impact but potentially worsen the situation. Accordingly, the potential of neck strength as a preventive measure for reducing head injury severity has been explored [24], but conflicting findings have been reported across various experimental studies [7, 30, 32, 38]. To address this discrepancy, Jin et al. [24] employed a computational head-neck model to investigate the impact of neck muscle activation levels and strategies on brain injury during an impact. Their findings revealed that while higher muscle activation aided in reducing the risk of brain injury, the strategy of muscle activation had a more significant effect. In another study by Bruneau et al. [6], the impact of muscle activation during an impact was examined, showing that neck muscle activation only moderately affected head-neck kinematics, resulting in an 11-16% increase in reaction moment at T1. However, these studies did not account for the influence of neck damping characteristics. Conversely, Dirisila et al. [10] incorporated a spring-damper system in their head-neck model to investigate the impact of neck damping during an impact. Their observations indicated that neck damping, characterized by viscoelastic properties, had minimal effects on the brain response in the initial stages of impact but subsequently reduced the brain's stress response in later stages of the simulation. Furthermore, Fice et al. [15] focused on studying the effect of muscle activation on capsular ligament strain during rear impact and demonstrated that muscle activation significantly reduced capsular ligament strain. This collection of studies sheds light on the multifaceted aspects of the neck's contribution to TBI protection. While muscle activation levels and strategies play a crucial role, the influence of neck damping characteristics should also be considered. Integrating these factors can lead to a more comprehensive understanding of TBI mechanisms and facilitate the development of effective preventive strategies.

Therefore, the goal of this study was to explore the role of neck muscle activity and neck damping characteristics in TBI mechanism. Specifically, we aimed to assess the effects of different muscle activation levels (no activation, low/medium activation, and high activation) and neck damping properties (hyper-viscoelastic/hyper-elastic intervertebral disks) on the mechanical response of the head-neck system under various impact scenarios, including golf ball impacts, linear acceleration, and combined linear and rotational acceleration. By conducting these simulations, we seek to gain a comprehensive understanding of the mechanisms underlying TBI protection and provide insights for the development of effective preventive strategies. This research has the potential to enhance our knowledge of the neck's role in mitigating brain injuries and contribute to improved safety measures in impact-related activities.

Methods

Head-neck model

We used our previously validated head-neck FE model to investigate our study objective [3]. The model incorporates various components, including the scalp, skull, cerebrospinal fluid (CSF), brain, dura mater, pia mater, neck muscles, neck ligaments, cervical vertebrae (C1-C7), and intervertebral discs [3]. This model was created using head and neck MRI data derived from a male participant (age: 42 years, height: 176 cm, and weight: 106 kg), resulting in a comprehensive structure comprising 1.36 million elements. Within this framework, the scalp, skull, and neck segments contributed 0.27 million, 0.23 million, and 0.072 million tetrahedral elements, respectively. Additionally, the model featured 0.1 million quad shell elements, with the dura and pia maters accounting for 0.05 million and 0.04 million elements, respectively. We modeled the skull, pia mater, dura mater, and vertebrae as linear elastic materials. The scalp was represented using a linear viscoelastic material, the CSF as a hyperelastic material, the intervertebral discs as hyper-viscoelastic materials, and the brain's gray and white matter as a hyper-viscoelastic material. One notable feature of this model is its incorporation of the hill type muscle model [52], allowing us to simulate active muscle behavior and investigate the impacts of muscle activation on our study objectives. Moreover, the viscoelastic properties of the intervertebral discs contribute to the damping characteristics of the neck [8]. By incorporating a hyper-viscoelastic material model, we were able to examine the effects of neck damping characteristics.

Loading conditions

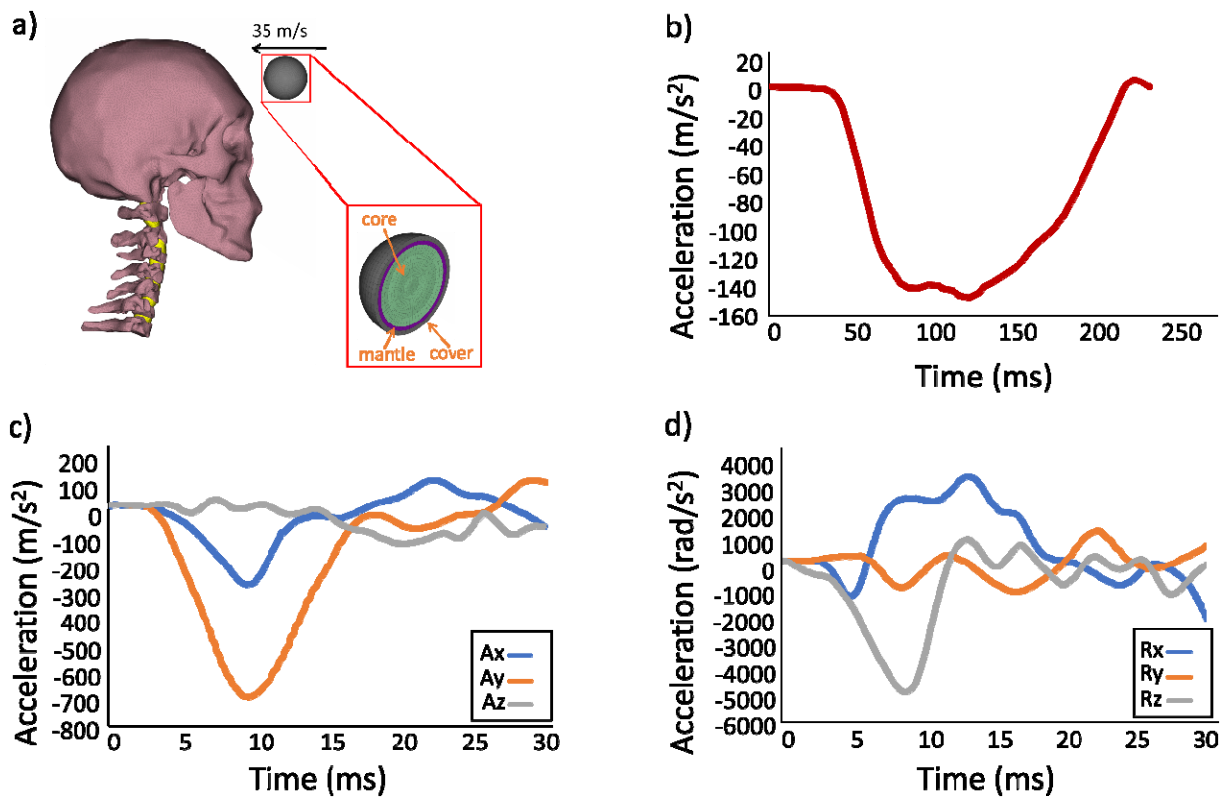

Fig. 1 loading conditions in this study: (a) golf ball impact simulation (muscles, ligaments, and scalp are excluded from the figure for better visualization) [28]. (b) Linear acceleration profile during NBDL study [13]. (c) Linear acceleration (Ax, Ay, Az) profiles from helmet-to-helmet collisions in American football [56]. (d) Rotational acceleration (Rx, Ry, Rz) profiles from the same collisions [56].

To address the focal injury mechanism commonly associated with impact scenarios, we utilized a golf ball impact scenario, where a golf ball hits the frontal area of the head model with an initial velocity of 35 m/s. The golf ball was represented as a three-layered structure consisting of an inner core, a mantle, and a cover. The core and mantle were modeled using hyper-viscoelastic materials, while the cover was modeled using a hyper-viscoelastic material (Table 1) [9]. Equation 1 describes the hyperelastic behavior of golf ball layers, whereas equation 2 describes their viscoelastic response.

(1)

(2)

In equations 1 and 2, I is invariant of the right Cauchy-Green deformation tensor, W is the strain energy density function, is the decay constant, G_j is the shear relaxation modulus, G is shear modulus for frequency independent damping and SIGF is the stress limit for frequency independent damping. We connected the golf ball's three layers using a tied boundary condition.

Table 1 Mechanical characteristics of the golf ball [9].

	Outer diameter (mm)	No. of elements	ρ (kg/m^3)	ν	C_{01} (MPa)	C_{10} (MPa)	G_1 (MPa)	β_1 (S^{-1})	G (MPa)	SIGF (kPa)
Core	35.4	8580	1125	0.49	1.50	6.00	10.50	37000	4.50	9.00
Mantle	38.8	1800	1274	0.49	3.67	14.70	18.30	18000	18.30	36.70
Cover	42.8	2040	950	0.49	10.50	42.00	-	-	-	-

In contrast, diffuse injuries are primarily associated with acceleration scenarios [2]. To account for this injury mechanism, we incorporated acceleration scenarios into our study. Specifically, we utilized the linear acceleration scenario from the Naval Biodynamics Laboratory (NBDL) study [47] and the combined linear and rotational acceleration scenario proposed by Zhang et al [56]. The NBDL acceleration profile was applied to the entire head-neck model, while the Zhang acceleration profile specifically targeted the center of mass of the head. The Zhang's acceleration scenario was derived from the recreation of American football accidents. Considering the ongoing debate surrounding the main cause of injuries, whether it is linear or rotational acceleration, it was imperative for us to include both scenarios in our study. By integrating these various loading conditions, we aim to gain comprehensive insights into the effects of different loading mechanisms on traumatic brain injuries.

Study design

In this study, our objective was to examine the effects of neck muscle activations and neck damping characteristics on mechanical responses of the brain and the head-neck system. We considered three activation strategies across all neck muscles: 1) no activation level, representing unprepared muscles experiencing an unanticipated impact scenario, 2) a low-to-medium activation level, indicating weak or inadequate muscle activation, and 3) high activation level, simulating tensed muscle conditions under anticipated impact scenarios. Throughout the impact duration, these muscle activation levels remained constant. To accurately represent the no activation level, we applied a 5% activation level to all neck muscles, based on previous research indicating that muscles exhibit approximately 5% activity even when they are not actively contracting [43]. For the second and third activation strategies, we classified the neck muscles into two groups: flexor muscles and extensor muscles.

In the golf ball's frontal impact, the flexor muscles play a crucial role in counteracting the impact force. Therefore, for the low-to-medium activation level, we applied a 25% activation level to the flexor muscles and 10% to the extensor muscles. Similarly, for the high activation level, we simulated tensed muscle conditions by using 80% flexor muscle activation and 10% extensor muscle activation.

Conversely, in the simulation of NBDL acceleration, where neck hyperflexion needs to be attenuated, the extensor neck muscles should be activated. Thus, we applied a 25% activation level to the extensor muscles and 10% to the flexor muscles for the low-to-

medium activation level. For the high activation level, we increased the extensor muscle activation to 80% while maintaining the flexor muscle activation at 10%.

In scenarios involving Zhang's combined linear and rotational acceleration, which do not strictly fall under flexion or extension, we applied the same activation level to all neck muscles. Specifically, we used a 25% neck muscle activation level for the low-to-medium activation level and 80% for the high activation level. These muscle activation levels were kept constant throughout the impact duration.

Furthermore, we examined the influence of neck damping characteristics, focusing specifically on the viscoelastic properties of intervertebral discs. To manipulate neck damping, we conducted impact scenarios with intervertebral discs that either included or excluded their viscoelastic properties.

Injury estimation

Researchers have made significant efforts to establish a connection between brain injury and specific variables to predict the occurrence of brain injury. Over time, various head injury criteria have been developed for this purpose. Initially, linear kinematics of the head were utilized as predictors in popular head injury criteria like HIC (Head Injury Criterion) [22] and GSI (Gad Severity Index) [17]. However, it was later suggested that rotational kinematics might be the primary cause of brain injury [44]. Consequently, alternative head injury criteria based on rotational kinematics were developed. For example, Takhounts et al. [44] devised brain injury criteria (BrIC) by correlating head rotational kinematics with brain strain. While the ease of measuring head kinematics made them a practical and popular option for use in head injury criteria, more accurate estimations of brain injury can be obtained by measuring brain tissue characteristics such as maximal principal strain or intracranial pressure. However, these measures are challenging to assess experimentally and are predominantly obtained through computational models [48]. Among these, the Head Injury Criterion (HIC) [22] has been widely used as a predictive metric for brain injuries (Equation 3).

$$HIC = max\left(\left[\frac{1}{t_2 - t_1}\int_{t_1}^{t_2} a(t)dt\right]^{2.5}(t_2 - t_1)\right) \quad (3)$$

The variable t represents time in milliseconds (ms), while a(t) denotes the linear acceleration profile measured in g's. The Head Injury Criterion (HIC) quantifies the maximum value of the average acceleration over a specific time duration, raised to the power of 2.5 and multiplied by the duration itself. It is recommended to impose a limit on the time duration to enhance the relevance of HIC. In this study, we adopted HIC_{15}, which restricts the time difference between t_2 and t_1 to a maximum of 15 ms. HIC_{15} is a widely implemented variant of HIC, known for its popularity and acceptance in the field. The HIC serves as a valuable tool for anticipating the likelihood of skull fracture (AIS ≥ 2), as indicated by Equation 4.

$$p(fracture) = N\left(\frac{\ln(HIC) - \mu}{\sigma}\right) \quad (4)$$

In this equation, N represents the cumulative normal distribution, while μ and σ values are 6.96352 and 0.84664, respectively [12].

However, HIC does not consider the rotational kinematics of the head. To account for this, we used the Brain Injury Criterion (BrIC) [44] as an additional parameter for estimating brain injuries (Equation 5).

$$BrIC = \sqrt{\left(\frac{\omega_x}{\omega_{xC}}\right)^2 + \left(\frac{\omega_y}{\omega_{yC}}\right)^2 + \left(\frac{\omega_z}{\omega_{zC}}\right)^2} \quad (5)$$

In equation 5, ω_x, ω_y, and ω_z denotes angular velocity in x, y, and z directions respectively. Conversely, ω_{xC}, ω_{yC}, and ω_{zC} stands for the critical value of these parameters. We adopted these critical values from the literature with values of 66.25, 56.45, and 42.87 rad/s for ω_{xC}, ω_{yC}, and ω_{zC} respectively [44]. Unlike the HIC, BrIC allows for the prediction of TBIs spanning AIS 1 to 5. This prediction is accomplished by correlating BrIC with brain strain, (Equations 6-10) [44].

$$p(AIS\ 1) = 1 - e^{-\left(\frac{BrIC - 0.523}{0.065}\right)^{1.8}} \quad (6)$$

$$p(AIS\ 1) = 1 - e^{-\left(\frac{BrIC - 0.523}{0.324}\right)^{1.8}} \quad (7)$$

$$p(AIS\ 1) = 1 - e^{-\left(\frac{BrIC - 0.523}{0.531}\right)^{1.8}} \quad (8)$$

$$p(AIS\ 1) = 1 - e^{-\left(\frac{BrIC - 0.523}{0.647}\right)^{1.8}} \quad (9)$$

$$p(AIS\ 1) = 1 - e^{-\left(\frac{BrIC - 0.523}{0.673}\right)^{1.8}} \quad (10)$$

Furthermore, by leveraging Finite Element (FE) modeling, we can access internal brain mechanical behavior, allowing us to go beyond kinematic-based injury metrics. We computed the brain's maximum principal strain (MPS) as estimation of brain injury in frontal, parietal, and occipital regions of the brain. Notably, brain MPS has been suggested as a reliable measure for predicting diffuse brain injuries.

We utilized the LS-DYNA (Livermore Software Technology Corporation, USA) explicit solver for conducting all impact simulations. These simulations were executed on the high-performance computing center located at our university. The computing center boasts cutting-edge infrastructure, including two AMD EPYC 7702 CPUs and 500 GB of memory per node, which provided ample computational resources to meet the demands of our simulations. Following the completion of the simulations, we performed the postprocessing of the results using META (BETA CAE Systems SA, Greece) and MATLAB R2021b (MathWorks, USA). This combination of software tools allowed us to effectively analyze and interpret the obtained data.

Results

Linear Acceleration (NBDL study)

Our simulation of the NBDL revealed a consistent trend, where an increase in muscle activation led to a reduction in both HIC and BrIC values, irrespective of neck damping. Notably, the most significant decrease in HIC occurred during the transition from no activation to medium-low activation, resulting in a 64% reduction with damping and a 68% reduction without damping (Figure 2a). Additionally, there was a smaller decline in HIC when progressing from medium-low to high activation, although the effect was relatively modest, with a 5% reduction under damping and a 3% reduction without damping conditions. Moreover, our findings indicated that the incorporation of damping into intervertebral discs had a moderate mitigating effect on HIC, with a reduction of less than 10%.

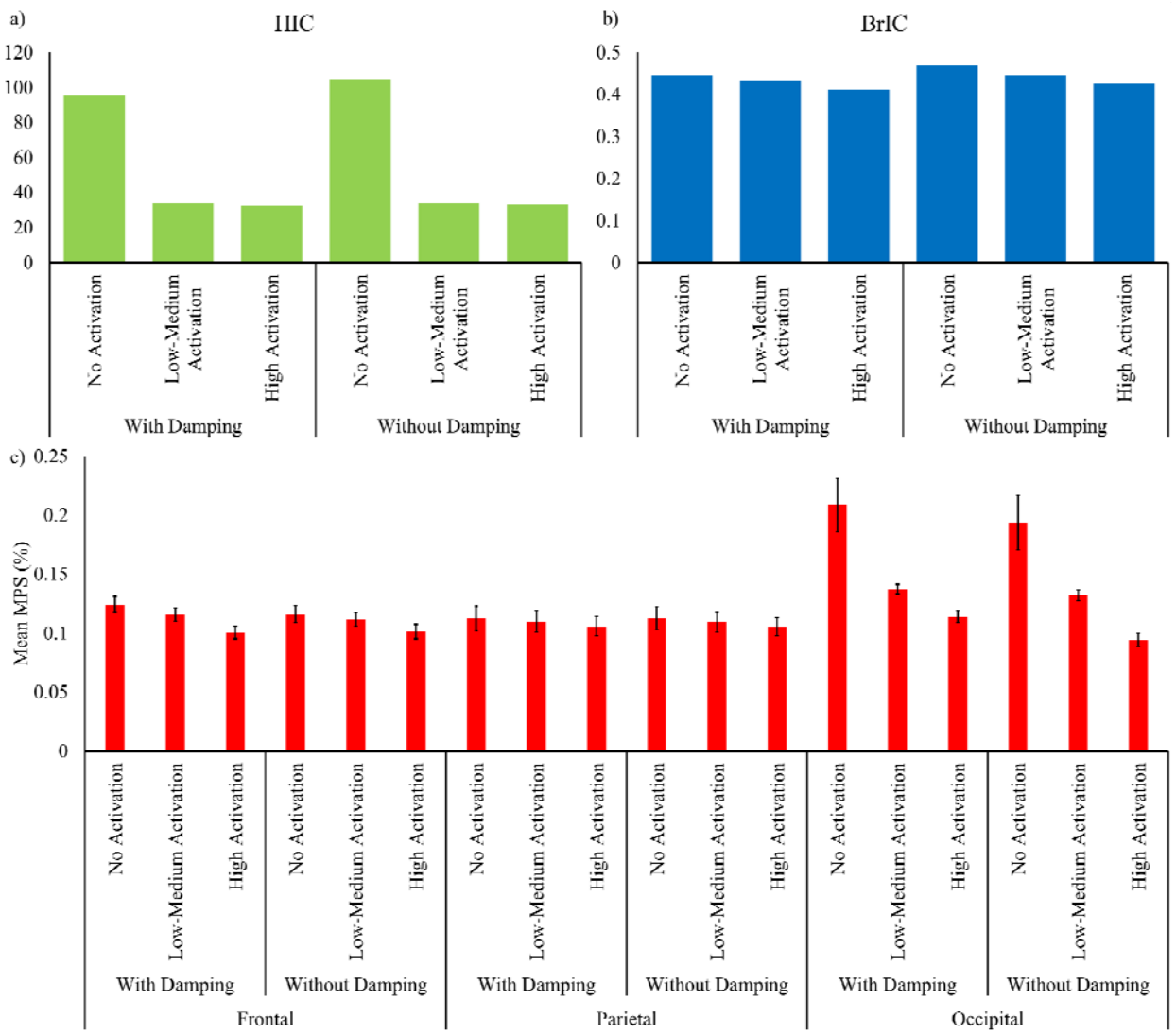

Fig. 2 Comparative analysis of a) HIC, b) BrIC, and c) MPS (mean±standard error) across Varied Simulated Conditions in NBDL simulated scenario.

Similarly, the analysis of BrIC results revealed that both increasing muscle activation and the presence of neck damping contributed to a reduction in brain injury criteria (Figure 2b). However, it's important to note that the impact of these parameters on BrIC was less pronounced compared to their effects on HIC. BrIC decreased by 7% and 10% when transitioning from no activation to high activation conditions, with and without damping, respectively. Similarly, the effect of neck damping on BrIC amounted to a reduction of less than 6%. Crucially, our BrIC values consistently remained below the baseline threshold of 0.5. Furthermore, the relatively low HIC values indicated a negligible risk of TBI. As a result, the risk of TBI remained practically zero for NBDL simulated scenarios in this study.

This trend persisted, with the mean MPS decreasing as muscle activation increased in the frontal and parietal sites of the brain (Figure 2c). While this reduction was modest

(less than 20%) in the frontal and parietal regions, a more substantial effect was observed in the occipital region, where mean MPS decreased by more than 45% from no activation to high activation levels. Additionally, our results demonstrated that the introduction of intervertebral disc damping consistently led to a reduction in brain MPS across all conditions. The effect of neck damping was relatively low (less than 7%) in the frontal and parietal regions. However, this effect was more pronounced in the occipital region, where the addition of neck damping reduced the mean MPS by over 17% for the high activation condition.

Combined linear and rotational acceleration (Zhang study)

In the simulation scenario involving combined linear and rotational acceleration, HIC exhibited a substantial initial decrease from no activation to the medium-low activation condition (83% with damping and 74% without damping). Nevertheless, it displayed an increase with the escalation of muscle activity to high levels (507% with damping and 288% without damping). Regarding the influence of damping, it consistently decreased the HIC value across all conditions, albeit with a more modest impact (less than 10%) (Figure 3a).

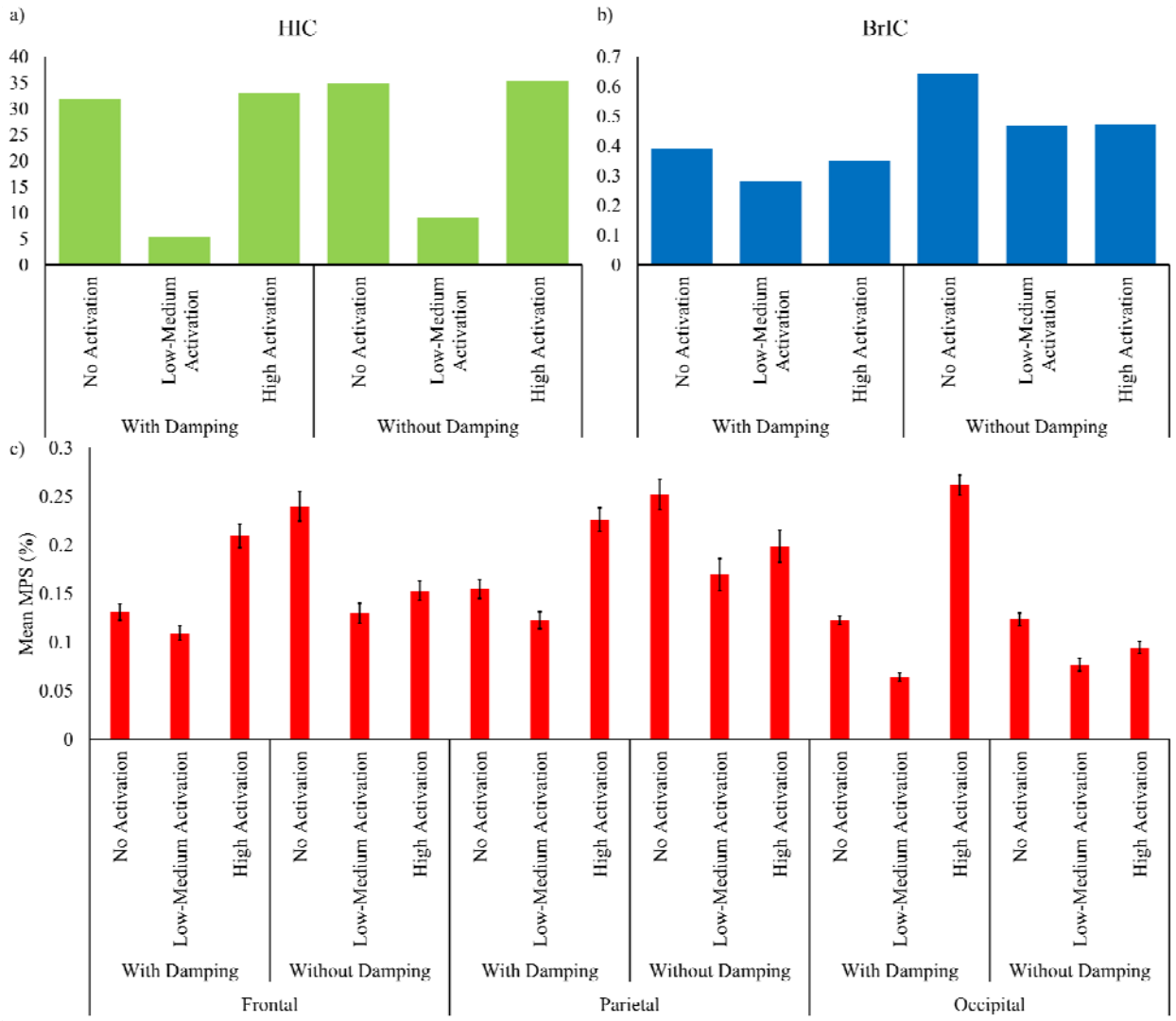

Fig. 3 Comparative analysis of a) HIC, b) BrIC, and c) MPS (mean±standard error) across Varied Simulated Conditions in Zhang's simulated Scenario.

Although the variability in BrIC values was comparatively lower than that in HIC, a similar trend was evident (Figure 3b). The BrIC values exhibited an initial decline from no activation to the low-medium activation level (28% with damping and 27% without damping), followed by an increase from low-medium activation to high activation levels (25% with damping and 1% without damping). In contrast, the impact of damping was more substantial, manifesting as a reduction in BrIC values across all conditions by over 34%. Analogous to the NBDL scenario, most of our HIC and BrIC values aligned with negligible risks of TBI. However, due to substantial increases in BrIC following the removal of damping from discs, BrIC values for the no activation and without damping condition rose to 0.64, corresponding to risks of AIS 1 to 5 injuries at 98.54%, 20.71%, 9.11%, 6.45%, and 6.04%, respectively.

Similarly, our MPS results demonstrated a congruent pattern, with average MPS values experiencing an initial decline from no activation to the low-medium activation level across all three brain regions (more than 20%). Subsequently, an increase was observed from the low-medium activation to the high activation level, particularly evident with the use of high muscle activation (Figure 3c). Conversely, the introduction of damping reduced the mean MPS values for no activation and low-medium activation levels, whereas mean MPS increased upon the introduction of damping in the high activation condition.

Golf ball impact

The golf ball impact scenarios exhibited a heightened risk of brain injury compared to the pure acceleration scenarios. In these instances, the HIC values surpassed the established safe threshold of 700, signaling the presence of traumatic brain injury. Nevertheless, our findings indicate a noteworthy trend, where as muscle activation increased, the HIC values decreased (Figure 4a). Specifically, the HIC value dropped by more than 6% when transitioning from no muscle activation to high muscle activation. Additionally, the introduction of viscous damping to the intervertebral disc contributed to a reduction in the HIC value, albeit with an effect of less than 1%.

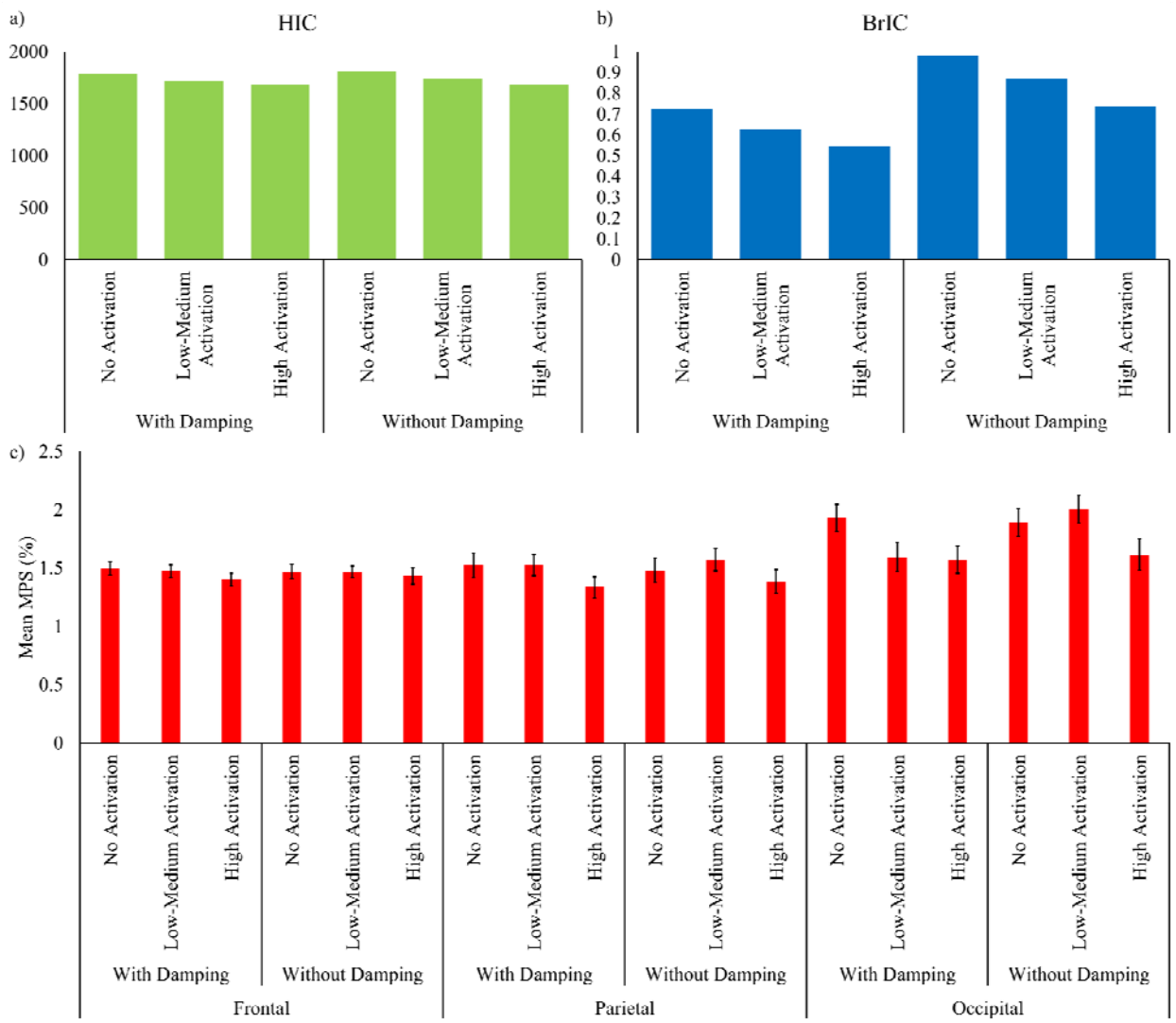

Fig. 4 Comparative analysis of a) HIC, b) BrIC, and c) MPS (mean±standard error) across Varied Simulated Conditions in golf ball impact simulated scenario.

The BrIC values exhibited similar trends, but the influence of muscle activation was notably more pronounced. BrIC saw a substantial drop of over 25% as muscle activation increased from none to high levels. Additionally, the incorporation of damping in the neck yielded a marked reduction of over 36% in BrIC values.

In contrast to the NBDL and Zhang simulated scenarios, the golf ball impact scenario resulted in a significant risk of traumatic brain injury (TBI) across all conditions (Figure 5). Given the non-linear relationship between BrIC and the Probability of TBI, even a small change in BrIC values could correspond to a substantial alteration in the probability of TBI. Our findings underscore that in all conditions, except for high activation with damping, an AIS 1 injury is nearly certain (Figure 5a). However, when examining AIS 2 to 5 injuries, we observe a steep decline in the risk of injury with both neck muscle activation and neck damping. Notably, the presence of disc damping

consistently reduced the probability of injury (abbreviated injury scale (AIS) 2 to 5) by at least half. Similarly, the transition from low muscle activation to high muscle activation consistently reduced the risk of injury (over 90% for the with damping condition and over 50% for the without damping condition) for AIS 2 to 5 injuries. However, HIC exhibited a subtle change in the risk of skull fracture with variations in neck muscle activation and neck damping (Figure 5b). The effect of neck damping amounted to less than a 1% reduction in the predicted probability of HIC-related skull fracture. Similarly, the effect of neck muscle activation resulted in a reduction of less than 4% in the predicted probability of HIC-related skull fracture.

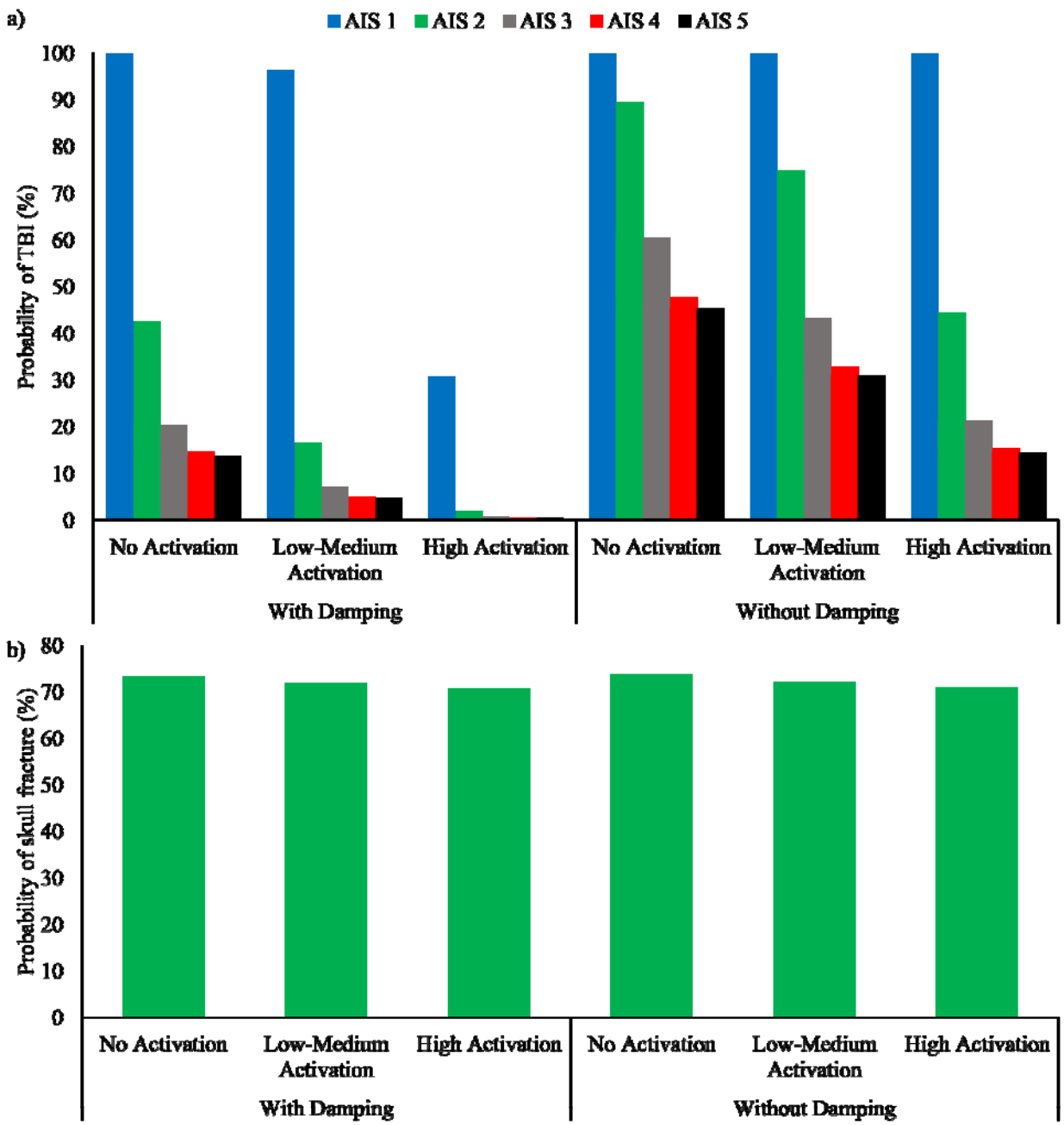

Fig. 5 a) Probability of TBI assessed through BrIC analysis, and b) Probability of skull fracture evaluated via HIC across diverse simulated scenarios in a golf ball impact simulation.

The MPS analysis revealed a nuanced set of results (Figure 4c). In the frontal region, both muscle activation and neck damping exhibited notably limited impacts, each contributing to less than 6% of the MPS variation when transitioning from no activation to high activation levels. However, a different pattern emerged in the parietal region. Here, neck muscle activation had a significant impact, more than doubling the MPS effect to over 12% in conditions with damping and 11% in conditions without damping.

In contrast, the influence of neck damping remained relatively subdued, accounting for less than 5% and 10% in the frontal and parietal regions, respectively. Notably, in the occipital region, the impact of neck damping demonstrated substantial increases, surpassing 25%.

Discussion

Our study aimed to address the inconsistency in previous research on the correlation between neck muscle activity and TBI [7, 30, 32, 38]. We aimed to quantify the impact of neck muscle activation and intervertebral disc damping on TBI outcomes across three scenarios: direct impact, pure linear acceleration, and combined linear and rotational acceleration. This research was motivated by the need for a more in-depth understanding of the complex relationship between neck characteristics and TBI. The inclusion of diverse TBI mechanisms will provide a holistic perspective, allowing for a more comprehensive assessment of the protective role played by neck muscle activation and intervertebral disc damping in distinct scenarios.

The impact of both neck muscle activation levels and neck damping on all three metrics is evident across our simulated scenarios. Neck muscles play a crucial role in countering impact forces, with the effectiveness of this response hinging on the magnitude, direction, and timing of muscle activation [14, 24]. Our investigation revealed an intriguing pattern: while high muscle activation minimized brain injury metrics in NBDL and golf ball impacts, the opposite holds true in Zhang's simulated scenario, where low to medium activation level performed better (Figures 2-4). This highlights the potential risks associated with inappropriately high muscle activation, potentially exacerbating TBI risks by contributing to the impact force. This pattern aligns with previous research, where Mihalik et al. [32] found that higher muscle strength can potentially lead to a higher risk of TBI, notably they found that athletes with stronger upper trapezius muscle experienced higher head impact telemetry severity profile values. Intriguingly, Schmidt et al. [40] discovered that athletes with stronger neck muscles faced an elevated risk of head injuries. Conversely, athletes exhibiting higher neck stiffness were associated with a reduced risk of injury. This suggests that while increased muscle strength alone may potentially worsen the impact of TBI, leveraging these muscles effectively to mitigate head-neck kinematics, as demonstrated in [24], and thereby enhancing neck stiffness, could contribute to protection against TBI. The ideal scenario for muscle activation strategy involves the brain accurately assessing situations and activating muscles accordingly. However, neurological disorders, such as amyotrophic lateral sclerosis [49] or motor neuron disease [45], may lead to suboptimal muscle activation, potentially worsening TBI impacts.

Furthermore, our study delves into the nuanced effects of muscle activation in mitigating different TBI mechanisms. In the NBDL simulation, where the predominant injury mechanism is diffuse TBI due to pure linear acceleration and the brain acceleration magnitude is relatively low, we observed a more pronounced impact of muscle

activation on HIC (based on linear acceleration) compared to BrIC (based on rotational velocity) (Figure 2). A similar trend was noted in Zhang's simulated scenario (Figure 3), suggesting that neck muscles are particularly adept at controlling linear motion in impacts involving diffuse injuries. Conversely, in the golf ball impact, muscle forces proved more effective in reducing rotational motion of the brain (BrIC), illustrating the role of neck muscle activation in preventing focal injuries. Additionally, our results demonstrate that neck muscle activation moderately reduces brain strain in both coup (frontal) and countercoup (occipital) regions, thereby potentially mitigating TBI (Figure 4). Furthermore, our results highlight the limitations of muscle activation, as its effect on injury metrics was generally more pronounced in less severe impact scenarios like NBDL and Zhang simulations compared to a more severe impact scenario like the golf ball impact. Nevertheless, the modest decrease in injury metrics in more severe impacts, particularly BrIC, translates into a significant decrease in the risk of injury. This underscores the importance of muscle activation in protection against more severe impact scenarios, despite the limited force neck muscles can generate. The study emphasizes the critical role of precise muscle activation, extending beyond sheer strength, in mitigating TBI risks. Significantly, these findings underscore the potential vulnerability of individuals with neurological disorders, as compromised motor control may lead to suboptimal muscle activation. This raises concerns about the efficacy of traditional TBI prevention approaches in these populations, emphasizing the need for targeted interventions that consider individual variations in motor control.

In this study, we perturbed neck damping in our head-neck model to investigate the resulting changes in the brain’s mechanical response, specifically the impact of altering the viscoelastic properties of all intervertebral discs (Figures 2-4). Similar to any mechanical damper, the damping characteristics of the neck are expected to absorb impact energy and influence the phase difference between the impact force and tissue response. Our findings align with previous findings [10], demonstrating that neck damping effectively reduces brain injury metrics. The reduction in HIC was relatively modest across all three simulated scenarios. In contrast, the reduction in BrIC with the introduction of neck damping was more notable in the Zhang and golf ball simulated scenarios. This highlights the significance of intervertebral disc damping, especially in scenarios like golf ball impacts where the risk of TBI is high. It's noteworthy that the negligible risk of brain injury in NBDL and Zhang's simulated scenarios makes it challenging to relate the reduction in BrIC and HIC with a realistic decrease in TBI risk, as the risk remains effectively zero irrespective of muscle activation level or neck damping characteristics. A notable exception arose when we removed the damping properties of intervertebral discs in Zhang's acceleration scenario while keeping all neck muscles passive. Compared to the NBDL study, the effect of damping was more pronounced in Zhang's study. Even though BrIC values in Zhang's scenario were initially lower than NBDL's with damping conditions, removing disc damping caused BrIC values to surpass the NBDL's values, crossing the 0.5 threshold indicative of no injury. These results suggest that neck damping is particularly effective in mitigating adverse rotational motion of the brain compared to its linear motion. Moreover, although

neck damping reduced the mean MPS in the frontal and parietal regions of the brain, it was more effective in the occipital (countercoup) region.

To comprehensively study TBI, a reliable method for quantification is essential. In this research, we employed BrIC, HIC, and MPS as our TBI metrics. HIC, focusing solely on linear acceleration and overlooking rotational brain motion, fails to capture the brain's response to rotational loading, especially its tendency to deform in shear during impact [26]. In contrast, BrIC, derived from finite element head-neck models, correlates angular velocity induced by impact to brain strain, offering a more comprehensive understanding of TBI. While MPS directly reflects brain tissue response and the link between excessive strain and neuron failure/rupture, the practical challenges of incorporating individual neurons into the model persist. Consequently, widely used metrics like BrIC and HIC, despite potential limitations under varied impact scenarios [55], remain prevalent for TBI quantification based on head kinematics in engineering and medical fields. Our results indicated that both BrIC and HIC predicted no injury for the Zhang and NBDL scenarios, consistent with expectations due to relatively low acceleration values. However, a notable discrepancy emerged, as HIC consistently predicted lower values for Zhang's scenario, while BrIC suggested a higher TBI risk, closely mirroring NBDL's BrIC values. This aligns with our expectations, given that HIC does not account for rotational motion. Examination of mean MPS results revealed a similar trend to BrIC, with Zhang's values slightly higher. Importantly, BrIC, designed based on finite element simulations of football and car crashes [44], differs in origin from HIC, which originated from dummy tests for automobile crashworthiness [50], making it more suitable for a more diverse set of applications. In contrast, the golf ball direct impact scenario posed a high TBI risk for both BrIC and HIC. Interestingly, the two metrics presented divergent narratives, with HIC predicting a very high risk of skull fracture (>70%) and minimal variation based on muscle activation and neck damping, solely relying on linear brain motion. Conversely, BrIC provided nuanced results, displaying AIS ≥ 2 values ranging from 89% to less than 2% under different muscle activation and neck damping conditions. These findings align with previous studies [24], suggesting a greater variation in the risk of TBI. Moreover, as BrIC can estimate a range of TBI severities (AIS 1 to 5), it presents a more comprehensive approach to TBI estimation.

Several limitations were present in this study. Firstly, while we aimed to capture major TBI mechanisms by simulating three scenarios, the scope remains limited, and future investigations should consider exploring a broader range of impact and acceleration scenarios to enhance our understanding of the neck's role in TBI mechanisms. Additionally, we assessed TBI using only three injury metrics, albeit commonly used ones. The diverse parameters proposed for TBI quantification highlight the need for future studies to consider a more comprehensive array of metrics. Moreover, our focus on perturbing the damping characteristics of intervertebral discs exclusively, to study the effect of neck damping characteristics on TBI, may not fully capture the complexity of neck tissues. Muscles and ligaments, for instance, also contribute damping characteristics, and future research should account for these components in assessing

neck damping effects on TBI. While we attempted reasonable muscle activation strategies for each scenario, it's crucial to acknowledge that in reality, muscle activation strategies are governed by the motor control portion of the brain, involving intricate activation strategies to optimize motion. Future studies should strive to incorporate the complexity of neck muscle activation strategies to provide a more realistic simulated scenario. Furthermore, it's essential to recognize that muscle activation strategies and neck properties are influenced by various factors, including brain impairments, age-related changes, and sports-related injuries. These factors, which were not explicitly considered in our study, should be taken into account in future research to better reflect real-world conditions. Finally, our head-neck model simplified the representation of intervertebral discs as single solid parts, neglecting the inherent composition of annulus fibrosis and nucleus pulposus with distinct mechanical properties.

In summary, despite these limitations, our study emphasizes the crucial role of neck muscle activation and damping characteristics in mitigating TBIs by countering or absorbing impact forces. These insights inform practical applications, including tailored neck strength training protocols for improved TBI mitigation. Understanding the age-related decline in neck strength and damping provides valuable guidance for targeted TBI prevention strategies, especially for the elderly. These findings extend to populations prone to intervertebral disc injuries, like athletes, allowing the design of protective measures suited to their unique needs. Our approach holds promise for advancing effective TBI prevention and protection strategies across diverse contexts.

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