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An In-silico Simulation of Pressure Wave Excursions after Impact to the Frontal Lobe of a Homogenous Model of the Brain

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Abstract: Traumatic impacts to the crania are known to have chronic effects on cerebral tissue and cognitive function. However, the inaccessibility of healthy brain tissue has limited studies of the mechanical behavior of the brain during impact. Consequently, it is uncertain how specific impacts lead to injury. The well-known Head Injury Criterion (HIC) metric is stochastic in nature. Therefore, it cannot provide deterministic indications of the severity of certain impacts and is ambivalent to trauma location.

This research investigated an impact to the anterior region of an isomorphic model of intracranial tissue. The impact was 18 mm lateral of the sagittal plane. The impact propagation through the brain model was observed with a focus on the depth of wave penetration. The brain model was simplified by removing material property variations across white and grey matter and in the ventricles that contain cerebrospinal fluid regions. Ultimately, homogeneity of the simplified model was assumed to show more conservative results than what may be observed in practice.

The simulation showed the significance of the initial impact magnitude and location with respect to any propagated wave. It was observed that despite minimizing the effect of damping in the model, pressure waves were not significant at the anterior of the brain. Thus, it was concluded that it is unlikely that secondary impact during TBI has a significant correlation to the location and severity of injury sustained.

Keywords: Brain tissue modelling, Traumatic brain injury, Solid modelling

1. INTRODUCTION

There has been a worldwide increase in the rate of traumatic brain injury (TBI) (Maas et al., 2008). This increase in TBI rate may be due to greater incidence of transport related accidents and increased participation in contact sports (Maas et al., 2008, D. M. Sosin, 1996). TBI has been established as a leading cause of morality and disability in the youth of the first world (Finfer and Cohen, 2001, Jennett and MacMillan, 1981). Severe TBI leads to mortality and morbidity that places a significant burden on healthcare providers worldwide (Te Ao et al., 2014). Mild TBI is detrimental on the lifestyle and economic output of those affected (Bombardier et al., 2010, Jorge et al., 2004). The compounded effects of TBI mean it is important to better understand its causes, and how to better reduce the rate and intensity of potentially harmful head impacts.

While extensive research has been performed to understand the mechanics of TBI (Post et al., 2018, Goriely et al., 2015, Ghajari et al., 2017), there is lack of consensus as to how to model cranial tissue for simulation. In particular, injury severity has a mild correlation with injury impact and site (Bailes et al., 2013). Rotational acceleration causes increased TBI severity and can cause damage to the deeper regions of the cerebral tissue than equivalent linear acceleration (Post et al., 2018, Mark W. Greve, 2009). Another commonly held belief is that repeated impacts compound TBI severity (D. M. Sosin, 1996, McAllister and McCrea, 2017). However, the compounding impact theory has also been scientifically contradicted (Bailes et al., 2013). The gold-standard of classification for TBI, the Head Injury Criteria (HIC), does not distinguish a difference between rotational and linear acceleration. In fact, the HIC is somewhat stochastic in nature (Goriely et al., 2015) leading to an intrinsic ambiguity.

Hence, current research provides contradictory, ungeneralizable, and imprecise relationships between impacts and TBI outcomes. Hence, further investigation is required to understand the TBI phenomenon and enable the optimisation of mitigating measures. In particular, novel methodologies are required to discover more deterministic links between impact mechanics and TBI severity.

This describes a method for modelling the propagation of pressure waves through the cerebral tissue following point impact on the frontal lobe. The initial impact can be compared to the propagated pressure and subsequent reflection of waves throughout the intracranial tissue. The wave reflection off the irregular surface of the skull interior was hypothesised to cause potentially harmful constructive interference in pressure waves.

2. METHOD

The Partial Differential Equation Toolbox™ features of MATLAB were used as a basis of modelling brain tissue

mechanics. The base equation for PDE's in the tool box can be seen in Equation 1.

$$\mathbf{M\ddot{u} + D\dot{u} - \nabla \cdot (C \otimes \nabla u) + Au = f} \quad (1)$$

The mass matrix (**M**) a diagonal matrix with density values (**ρ**) and is shown in Equation 2. The definition of Rayleigh Damping (Petrov et al., 2014b) allows a damping matrix (**D**) and is shown in Equation 3. The matrix (**C**) represents the tensor element which is a function of Poisson's Ratio (v) and Young's Modulus (E) (Equation 4)

$$\mathbf{M} = diag(\rho) \quad (2)$$

$$\mathbf{D} = \alpha\mathbf{M} + \beta\mathbf{A} \quad (3)$$

$$\mathbf{D} = \alpha\mathbf{M} \quad (3a)$$

$$\mathbf{C} = \begin{bmatrix} 2\mu+\lambda & 0 & 0 & 0 & \lambda & 0 & 0 & 0 & \lambda \\ 0 & \mu & 0 & \mu & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \mu & 0 & 0 & 0 & \mu & 0 & 0 \\ 0 & \mu & 0 & \mu & 0 & 0 & 0 & 0 & 0 \\ \lambda & 0 & 0 & 0 & 2\mu+\lambda & 0 & 0 & 0 & \lambda \\ 0 & 0 & 0 & 0 & 0 & \mu & 0 & \mu & 0 \\ 0 & 0 & \mu & 0 & 0 & 0 & \mu & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \mu & 0 & \mu & 0 \\ \lambda & 0 & 0 & 0 & \lambda & 0 & 0 & 0 & 2\mu+\lambda \end{bmatrix} \quad (4)$$

Where: $\mu = \frac{E}{2(1+v)}$, and: $\lambda = \frac{Ev}{(1+v)(1-2v)}$

The stiffness component of the damping model (**A**) can be attributed to returning the structure to its neutral position. However since this model is for an elastic solid, the presence of boundary conditions and relative displacement within the perturbed domain is what returns the solid to its neutral position ($\nabla\cdot(\mathbf{C}\otimes\nabla\mathbf{u})$). Consequently, the stiffness component can be disregarded in equations 1 and 3 for the purposes of this study. Equation 1a shows the final form of the PDE used.

$$\mathbf{M(\ddot{u} + \alpha\dot{u}) - \nabla \cdot (C \otimes \nabla u) = f} \quad (1a)$$

MATLAB 2019a's createpde.m was used to produce a transient structural solid model, this was configured using the 'structural' and 'transient-solid' settings. The brain geometry file (.stl filetype generally associated with Solidworks™ software) obtained from an MRI of a healthy brain [15] was modified to replace ventricle structure with homogenous brain tissue. This modification allowed isomorphic modelling of brain tissue and effectively removed the need to model fluid-solid boundaries. The resulting model was then imported as a geometry using importGeometry.m. Mesh generation was achieved via generateMesh.m and was limited to have maximum allowable length of 6.3 mm and, minimum allowable length of 3.5 mm; with an imposed graduation of 1.5. In order to reduce the computational load, the mesh structure was reduced from quadratic to a linear order.

Table 1 outlines the structural properties of the geometry. The functions structuralProperties.m, and structuralDamping.m were used to apply these properties. The geometry faces had the default fixed boundary conditions applied. The default displacement of 0 m and velocity of 0 ms^{-1} were applied to the model.

Table 1. Model nomenclature and values

Symbol	Meaning	Value
ρ	Mass Density	1130 kgm^{-3} *
E	Young's Modulus	31.5 MPa *
v	Poisson's Ratio	0.45 *
α	Rayleigh Mass Coefficient	0.25 s^{-1} **
(* Jennett et al. 1981** Sosin et al. 1996)		

To mimic an impulse on the cranium, a boundary load was used to apply the input force. A 10 kN force was simulated for 1 ns in the posterior direction on the anterior surface of the brain using the function structuralBoundaryLoad.m. The high impact force over a short period of time was made to model instantaneous impact and limit the effect of force transmittance on the simulation outcomes. The impact was located 18 mm transverse from medial line of the model.

The solve.m function ran for 15 seconds at 0.025 second time increments to achieve the simulation. Interpolation of Von Mises stress across the common horizontal plane for each impact was achieved with the function interpolateVonMisesStress.m. The simulation ran on a desktop computer with four core i7-4790 CPU running at 3.6 GHz and 16 GB ram.

3. RESULTS

After processing, the mesh geometry consisted of 14105 Nodes, 70307 Elements, 1298 Vertices, and 879 Edges. A rendering of this Mesh is shown in Figure 1. The pressure wave was successfully simulated until pressure wave dissipation. Pressure wave distributions across the horizontal plane upon which the impact occurred are shown in Figure 2. Figure 3 shows the Von Mises stress propagation through the direct line on which the impact occurred on over time

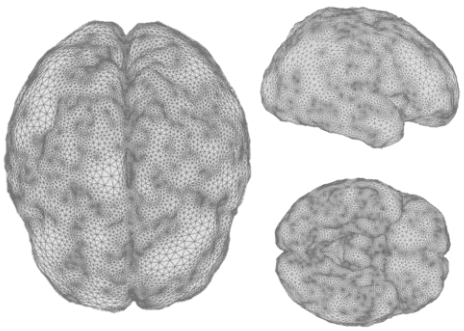

Fig 1. Superior (left), transverse (top right) and inferior (bottom right) views of the brain mesh geometry.

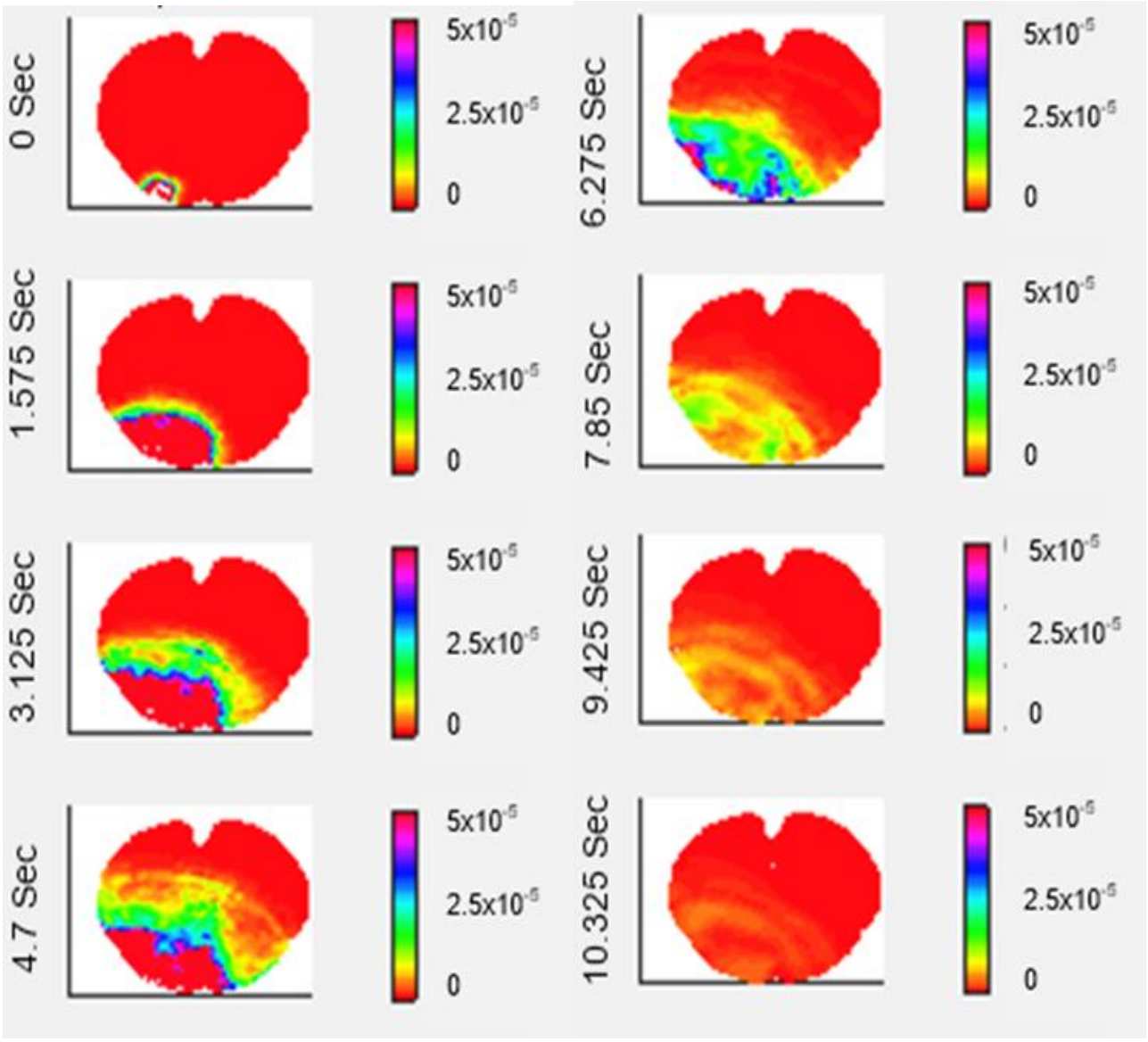

Fig 2. Simulated impact to the front of the brain, displaying the normalised Von Mises stress over time.

The pressure wave was successfully simulated until pressure wave dissipation. Pressure wave distributions across the horizontal plane upon which the impact occurred are shown in Figure 2. Figure 3 shows the Von Mises stress propagation through the direct line on which the impact occurred on over time

4. DISCUSSION

Figure 2 shows the model simulation of pressure in the intercranial tissue. There was initially high pressure proximal to the impact site. The pressure wave travelled through the brain tissue while dissipating. Figure 2 shows that the pressure wave did not reach the back of the brain. This implies that secondary impacts on the back of the brain are not likely to be a significant contributor to TBI for impact forces of the magnitude tested. The simulation used the minimal damping values from a range reported in (Petrov et al., 2014a). This minimized the effect of damping on the propagation of waves, increasing the ability of pressure waves to reach the posterior of the cranium. It is often assumed that pressure waves travel through the entirety of the intracranial medium. Therefore, it was deemed sensible to select tissue model mechanical parameters from the literature that enabled the simulation to match expected behaviour as close as possible. Higher damping would further decrease the penetration of waves and localise stress distributions closer to the impact site. Ultimately, reducing the ambiguity of model parameters would increase the confidence in the conclusions of this simulation.

In-vivo rheological measurement is difficult (Petrov et al., 2014a) and *ex-vivo* analysis returns a wide range of mechanical values for tissue parameters (Kaster et al., 2011, Nicolas et al., 2018, Feng, 2012). Consequently, experimentally determined mechanical properties of brain tissue reported in literature show greater variability than that which is typical of other biological tissues. This made the choice of properties to use in

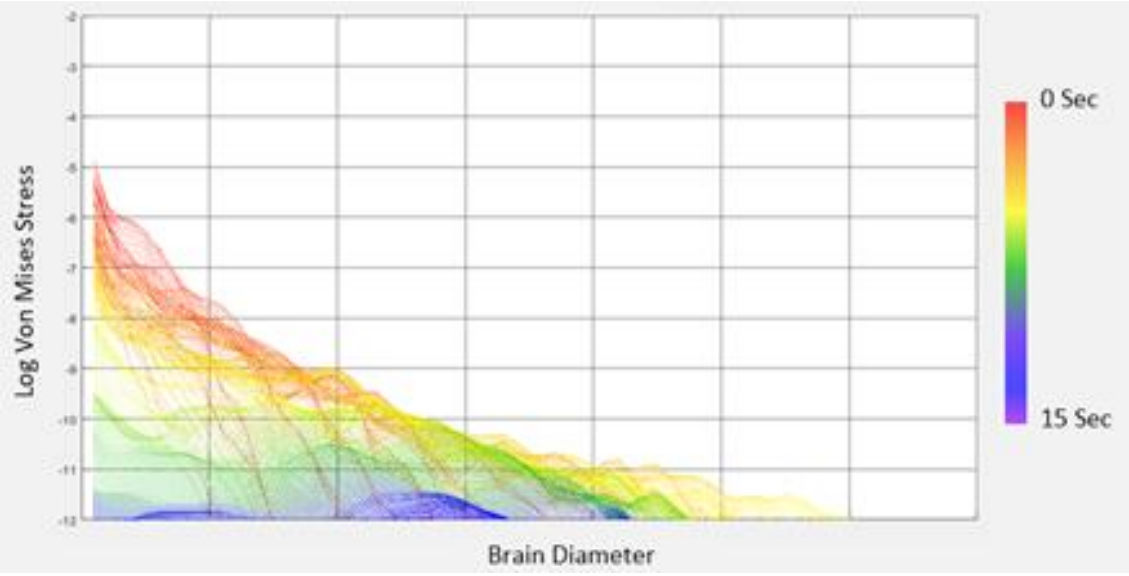

Fig 3. Simulated normalised Von-Mises stress propagation over time through the direct line of impact. Note that the line colour signifies time and thus, the stress wave energy is attenuated while travelling from left to right over time.

this study somewhat equivocal. Further rheological studies are required to refine the range of suitable model parameters for simulation.

To reduce model complexity, the model used a linear, homogenous medium. The model did not differentiate between grey and white matter and did not include CSF fluid regions. The model also did not address the layer of CSF between the skull and brain. The nature of simulation meant these factors were deemed non-essential. It can be assumed that the addition of CSF in the ventricles would lead to a wave refraction and reflection. This would ultimately increase the randomness of the pressure distributions of the model. This may in turn result in more regions of constructive wave interference and increased range of wave propagation. Furthermore, addition of the CSF surrounding the brain is likely to provide a smoothing effect on the wave profiles observed across the overall domain. Ultimately, it is difficult to predict the effects of increasing the complexity of the model by incorporating the CSF and white/grey matter inhomogeneity. One may expect that incorporating ventricles may increase the variations and range of excursions of wave distribution. In contrast, the CSF between the brain and skull may lead to reduced randomness of wave distributions and reduced wave excursions. More detailed FEA analysis will be required to model the fluid structure interaction between the brain tissue and the CSF that surrounds and exists within the brain.

The accidental circumstances that cause TBI mean that collecting impact data is difficult. Some researchers measure kinematics of head motion in gridiron players or combats sports competitors (Crisco et al., 2011). However, it is difficult to determine the precise location, intensity and profile of the forces associated with this data. Simulation based on what is known about the mechanical properties of the brain allow further investigation into the factors that could affect the severity and nature of injury. The methods used in this investigation have the benefit of repeatability and controllability. While it can be assumed that these simulations will not perfectly emulate what occurs during specific impacts, they are a useful tool in further understanding and testing hypotheses about key factors relating impact to injury severity. Simulation enables multiple experiments without the need for costly *in-vivo* or *ex-vivo* animal studies.

The impacts simulated used entirely linear acceleration of the cranium. In reality there will almost certainly be some rotational contribution from the impact. It has been suggested that rotational acceleration may lead to further internal fissures within the brain (Post et al., 2018). Further investigation into how stress propagates as a result of rotational acceleration would be beneficial to further refine understanding of TBI mechanics.

It has been established that after a particular strain threshold is exceeded, brain tissue has a non-linear elasticity (Bilston, 2019). However, it was deemed sensible to use a linear elasticity for this study. It was assumed that increasing the model complexity would have a mild effect on the magnitude of wave distributions. However, it was assumed that this simplification of the model did not have a major effect on the overall outcomes. The use of a non-linear model would not affect the properties inherent to stress wave propagation through tissue. Hence, using a nonlinear model would alter the relative magnitude of pressure across the domain, but it would not alter the outcomes in a way that would alter the conclusions presented. In particular, the unpredictable nature of stress distributions observed throughout testing implies that a more accurate model would not necessarily lead to more accurate results.

This investigation simulated the effect of an impact on the frontal lobe on a brain geometry. The simulation showed the wave propagation did not reach the back of the brain with significant stress. If the outcomes of this research are repeated

with more detailed models of human brain structures, it will have connotations for brain protecting measures. In particular, this research implies that the primary impact is more critical to TBI severity than secondary impact from brain recoil. Furthermore, it shows that stress intensity remains highest proximal to the impact location.

5. CONCLUSIONS

This investigation used a model based approach to establish the behaviour of pressure wave propagation within intracranial tissue following an external impulse force. The dissipation of pressure wave energy during propagation was observed as it travelled from the local area of the impact. The pressure wave did not reach the posterior of the cranium with significant energy. This implies any injury sustained as a result of secondary impact during TBI is likely to be negligible with respect to any damage caused by the initial impact. The model used for this simulation was simplified to allow for more efficient computation. However, it may be assumed that the results of this study are more conservative than what might be observed in practice.

It was hypothesised that the irregular concavity of the skull interior might cause wave reflection. This could result in constructive interference, causing damage to localised areas deeper within the cranium. However, the simulation results implied that constructive interference from wave reflection would not be unlikely to have a significant affect due to the damping nature of the brain tissue mitigating the energy of the primary wave. Further analysis and rheological investigation is required to confirm the findings of this study.

REFERENCES

BAILES, J. E., PETRAGLIA, A. L., OMALU, B. I., NAUMAN, E. & TALAVAGE, T. 2013. Role of subconcussion in repetitive mild traumatic brain injury: a review. *Journal of neurosurgery,* 119**,** 1235-1245.

BILSTON, L. E. 2019. Brain tissue mechanical properties. *Biomechanics of the Brain.* Springer.

BOMBARDIER, C. H., FANN, J. R., TEMKIN, N. R., ESSELMAN, P. C., BARBER, J. & DIKMEN, S. S. 2010. Rates of major depressive disorder and clinical outcomes following traumatic brain injury. *Jama,* 303**,** 1938-1945.

CRISCO, J. J., WILCOX, B. J., BECKWITH, J. G., CHU, J. J., DUHAIME, A.-C., ROWSON, S., DUMA, S. M., MAERLENDER, A. C., MCALLISTER, T. W. & GREENWALD, R. M. 2011. Head impact exposure in collegiate football players. *Journal of Biomechanics,* 44**,** 2673-2678.

SOSIN D. M., SNIEZEK J. E., THURMAN D.J. 1996. Incidence of mild and moderate brain injury in the United States, 1991. *Brain Injury,* 10**,** 47-54.

FENG, Y. 2012. *Dynamic Deformation and Mechanical Properties of Brain Tissue.* Dissertation/Thesis, ProQuest Dissertations Publishing.

FINFER, S. R. & COHEN, J. 2001. Severe traumatic brain injury. *Resuscitation,* 48**,** 77-90.

GHAJARI, M., HELLYER, P. J. & SHARP, D. J. 2017. Computational modelling of traumatic brain injury predicts the location of chronic traumatic encephalopathy pathology. *Brain,* 140**,** 333-343.

GORIELY, A., GEERS, M. G., HOLZAPFEL, G. A., JAYAMOHAN, J., JÉRUSALEM, A., SIVALOGANATHAN, S., SQUIER, W., VAN DOMMELEN, J. A., WATERS, S. & KUHL, E. 2015. Mechanics of the brain: perspectives, challenges, and opportunities. *Biomechanics and modeling in mechanobiology,* 14**,** 931-965.

JENNETT, B. & MACMILLAN, R. 1981. Epidemiology of head injury. *Br Med J (Clin Res Ed),* 282**,** 101-104.

JORGE, R. E., ROBINSON, R. G., MOSER, D., TATENO, A., CRESPO-FACORRO, B. & ARNDT, S. 2004. Major depression following traumatic brain injury. *Archives of general psychiatry,* 61**,** 42-50.

KASTER, T., SACK, I. & SAMANI, A. 2011. Measurement of the hyperelastic properties of ex vivo brain tissue slices. *Journal of biomechanics,* 44**,** 1158-1163.

MAAS, A. I., STOCCHETTI, N. & BULLOCK, R. 2008. Moderate and severe traumatic brain injury in adults. *The Lancet Neurology,* 7**,** 728-741.

MARK W. GREVE, M. A. B. J. Z., MD 2009. Pathophysiology of Traumatic Brain Injury. *MOUNT SINAI JOURNAL OF MEDICINE,* 76**,** 8.

MCALLISTER, T. & MCCREA, M. 2017. Long-term cognitive and neuropsychiatric consequences of repetitive concussion and head-impact exposure. *Journal of athletic training,* 52**,** 309-317.

NICOLAS, E., CALLE, S., NICOLLE, S., MITTON, D. & REMENIERAS, J.-P. 2018. Biomechanical characterization of ex vivo human brain using ultrasound shear wave spectroscopy. *Ultrasonics,* 84**,** 119-125.

PETROV, A. Y., DOCHERTY, P. D., SELLIER, M. & CHASE, J. G. 2014a. Imaging of Rayleigh damping properties of the in vivo brain using parametric Magnetic Resonance Elastography. *IFAC Proceedings Volumes,* 47**,** 3881-3886.

PETROV, A. Y., DOCHERTY, P. D., SELLIER, M. & CHASE, J. G. 2014b. Multi-frequency inversion in Rayleigh damped Magnetic Resonance Elastography. *Biomedical Signal Processing and Control,* 13**,** 270-281.

POST, A., KENDALL, M., COURNOYER, J., TAYLOR, K., HOSHIZAKI, T. B., GILCHRIST, M. D., BRIEN, S., CUSIMANO, M. D. & MARSHALL, S. 2018. Falls resulting in mild traumatic brain injury and focal traumatic brain injury: a biomechanical analysis. *International Journal of Crashworthiness,* 23**,** 278-289.

TE AO, B., BROWN, P., TOBIAS, M., AMERATUNGA, S., BARKER-COLLO, S., THEADOM, A., MCPHERSON, K., STARKEY, N., DOWELL, A., JONES, K. & FEIGIN, V. L. 2014. Cost of traumatic brain injury in New Zealand. *Neurology,* 83**,** 1645.