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Computational Simulation of the Mechanical Response of Brain Tissue under Blast Loading

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Abstract

In the present study, numerical simulations of nonlinear wave propagation and shock formation in brain tissue have been presented and a new mechanism of injury for Blast-Induced Neurotrauma (BINT) is proposed. A quasilinear viscoelastic (QLV) constitutive material model was used that encompasses the nonlinearity as well as the rate dependence of the tissue relevant to BINT modeling. A one-dimensional model was implemented using the discontinuous Galerkin -finite element method and studied with displacement-input and pressure-input boundary conditions. The model was validated against LS-DYNA finite element code and theoretical results for specific conditions that resulted in shock wave formation. It was shown that a continuous wave can become a shock wave as it propagates in the QLV brain tissue when the initial changes in acceleration are beyond a certain limit. The high spatial gradient of stress and strain at the shock front cause large relative motions at the cellular scale at high temporal rates even when the maximum stresses and strains are relatively low. This gradient-induced local deformation may occur away from the boundary and is proposed as a contributing factor to the diffuse nature of BINT.

Keywords

Brain Tissue; Quasilinear Viscoelasticity; hyperelasticity; Blast Loading; Discontinuous Galerkin Method; Injury Biomechanics

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1 Introduction

Traumatic brain injury (TBI) is continuously reported as one of the leading causes of death and disability in the world [1]. In the United States alone 1.4 million people sustain a form of TBI each year, of whom 52,000 die and over 200,000 are hospitalized [2]. In addition to the TBI cases occurring in motor vehicle accidents and sports related injuries, which are quoted as the major causes of TBI, a relatively more recent aspect of TBI, known as blast induced neurotrauma (BINT), has also become of increasing concern. BINT is referred to as the signature wound of war in Iraq and Afghanistan wars affecting almost 20% of the soldiers [3,4]. The mechanisms of BINT are not yet completely understood and the available experimental and computational models to simulate blast injuries are currently limited, signifying the need for improvement in such models [5].

A blast wave is a pressure wave with finite amplitude that in a short time period releases a significant amount of energy. A sharp increase in pressure and air density starts propagating away from the source of explosion. As the blast over-pressure (BOP) enters a biological tissue, high-rate stress waves, which have longitudinal and shear components, develop that can have devastating effects on the tissue depending on the nature of the waves and the type of tissue [5]. The duration of a blast wave, depending on the explosion's intensity, varies in the range of 100 μ s to a few milliseconds, and it can induce pressure rises of up to 1 MPa [5,6]. Such high rates of loading may lead to propagation of acceleration waves and shock waves, due to the nonlinear behavior of the tissue, that are not present at low rates of loading. The nonlinear viscoelastic wave propagation in brain tissue has been mostly overlooked in the BINT literature.

Since the pioneering works in the field of nonlinear viscoelasticity in 1960s and 1970s [7–11], little attention has been given to the possibility of propagation of acceleration waves and shock waves in nonlinear viscoelastic soft tissues including brain tissue. Based on theoretical results, in a previously undisturbed semi-infinite nonlinear viscoelastic medium, when certain conditions are met, discontinuities in acceleration, i.e., acceleration waves, and/or in velocity or strain, i.e., shock waves, can develop and propagate. This is counter-intuitive to the expected dissipative character of viscoelastic media and indeed in the case of linear viscoelasticity acceleration waves are always dissipative, i.e., their amplitudes decay in time and space, and do not lead to shock waves.

While an analytical solution for the propagation of one-dimensional acceleration waves in nonlinear viscoelastic materials exists, the solutions to general displacement or pressure inputs require a numerical solution. For BINT applications, it is necessary to use a numerical method that can handle the complex constitutive model of brain tissue derived based on latest reported results and also the propagation of acceleration and shock waves in such medium.

The Discontinuous Galerkin (DG) method was used in this study as it has been shown in the past to be an efficient method in studying the propagation of discontinuities in a continuum [12,13]. The DG method is an extension of the conventional finite element (FE) method so it retains the capabilities of FE methods in handling irregular meshes and a wide range of

material models. In addition, due to locality of this method (each element only communicates with neighboring elements), it has much better potential for parallelization and lower computational cost. The main advantage of the DG method for this study compared to the FE method is its ability to capture shock wave motion and evolution with minimal numerical artifact. In most commercial FE software, a shock wave is handled by using artificial numerical diffusion in the numerical solution and reducing a shock wave to a wave with steep gradient. This is performed by using the bulk viscosity effect explained in [14]. To the best of the authors' knowledge, DG has not been performed as a computational approach to study wave propagation in nonlinear viscoelastic tissues.

The objective of this study was to put forth a novel DG computational method to capture and study the formation and propagation of shock waves in brain tissue. The effects of tissue characteristics such as nonlinearity and viscoelasticity on the stress waves, in particular steepening of the wave front in contrast with dispersion and damping, are studied. The theoretical rationale together with the numerical results are then utilized in order to propose an injury mechanism for BINT based on shock wave formation in brain tissues.

2 Governing Equations

Since the focus of this study was to understand the effects of brain material properties on shock wave formation and propagation, a one dimensional (1D) domain for simplicity is considered. Using the conventional continuum mechanics approach, the position of a material point denoted by $\mathbf{X}$ in the undeformed (reference) configuration is described by $\mathbf{x} = \mathbf{x}(\mathbf{X}; t)$ in the deformed configuration. The deformation gradient tensor ($\mathbf{F}$), the right Cauchy-Green strain tensor ($\mathbf{C}$) and the Lagrangian strain tensor ($\mathbf{E}$), defined below, were used as measures of deformation:

$$\mathbf{F} = \frac{\partial \mathbf{x}}{\partial \mathbf{X}}, \quad \mathbf{C} = \mathbf{F}^T \mathbf{F}, \quad \mathbf{E} = \frac{1}{2} (\mathbf{C} - \mathbf{I}) \quad (1)$$

where $\mathbf{I}$ is the identity tensor. For a one-dimensional deformation, $\mathbf{F}$ reduces to a single stretch ratio $\lambda = x/X$ while $u = x - X$ denotes displacement, and $\epsilon = \lambda - 1$ is used as strain. The Cauchy equation of motion in the Lagrangian description simplifies to a single equation:

$$\frac{\partial P}{\partial X} = \rho_0 \frac{\partial v}{\partial t} \quad (2)$$

where P is the first Piola-Kirchhoff (engineering) stress, ρ_0 is the density of the material in the undeformed configuration, and $v = u/t$ is the velocity field. For regions where velocity and strain are continuous, a compatibility equation can be written between velocity and strain in the following form:

$$\frac{\partial v}{\partial X} = \frac{\partial \epsilon}{\partial t} \quad (3)$$

Equation (2), together with Equation (3), can be written as a system of two first-order partial differential equations (PDE):

$$\frac{\partial}{\partial t} \begin{bmatrix} \epsilon \\ v \end{bmatrix} = \frac{\partial}{\partial X} \begin{bmatrix} v \\ P/\rho_0 \end{bmatrix} \quad (4)$$

Stress (P) is related to strain (ϵ) through a constitutive model. In the subsequent sections, a hyperelastic and a nonlinear viscoelastic constitutive model suitable for brain tissue will be considered and their effects on the development of shock waves based on the governing Equation (4) will be studied, but before that the general conditions for the development of shock waves is discussed in the next section.

3 Shock Formation Criteria

To study shock formation, the governing Equation (4) is written in the following form:

$$\frac{\partial}{\partial t} \begin{bmatrix} \epsilon \\ v \end{bmatrix} + \mathbf{A} \frac{\partial}{\partial X} \begin{bmatrix} \epsilon \\ v \end{bmatrix} = 0 \quad (5)$$

where

$$\mathbf{A} = \begin{bmatrix} 0 & -1 \\ -c^2 & 0 \end{bmatrix} \quad \text{and} \quad c(\epsilon) = \sqrt{\frac{1}{\rho_0} \frac{\partial P}{\partial \epsilon}}$$

are the coefficients' matrix (consisting of the material parameters) and the stress wave velocity in the medium, respectively. P/ϵ is the local tangent modulus that in general can be a function of strain ϵ . In a system of first-order PDEs, as in Equation (5), characteristic lines are two sets of curves in the $X-t$ plane aligned with the eigenvectors of the coefficient matrix $\mathbf{A}$, along which the system of PDEs simplifies to two ordinary differential equations (ODEs) [15]. The eigenvalues of $\mathbf{A}$ give the speed of propagation of waves along the characteristic lines. The above system has the following eigenvalues and eigenvectors:

$$\Lambda = \pm c, \quad \mathbf{Q} = \begin{bmatrix} -1/c & 1/c \\ 1 & 1 \end{bmatrix} \quad (6)$$

the values of which depend on c and therefore on P/ϵ . In a linear material, where the tangent modulus is a constant, the characteristic lines have a constant slope and therefore the solution of the ODE along each line remains unique. However, in a nonlinear material where the tangent modulus is a function of strain level, the slope of the characteristic curves changes accordingly and may result in colliding characteristic lines at a particular point in space and time. When the characteristic lines collide, the solution to the PDE is no longer continuous, and is instead defined in the weak sense [16]. This situation is known as a shock wave discontinuity, in which the solutions (ϵ and v) have an abrupt jump with large spatial and temporal gradients (infinite in theory). In a stiffening material when the tangent modulus has a positive curvature in tension and a negative curvature in compression, shock wave discontinuity can evolve from a smooth initial condition. In contrast, in a softening material, a stress wave disperses. The high stress gradients and rates that are reached in a stiffening material as the stress wave propagates in the medium, are proposed to be a

mechanism of injury in the case of brain tissue undergoing BOP based on the constitutive material model presented in the next section.

4 Constitutive Model for Brain Tissue

The constitutive model presented in this section follows the model reported previously in [17]. The Lagrangian strain tensor ($\mathbf{E}$) and the second Piola-Kirchhoff stress tensor ($\mathbf{S} = \mathbf{F}^{-1}\mathbf{P}$) are chosen as the measures of strain and stress that satisfy the principle of objectivity. Viscoelasticity is implemented using a quasi-linear viscoelastic (QLV) model consisting of a convolution integral between a strain dependent instantaneous nonlinear hyperelastic function $\mathbf{S}^e(\mathbf{E})$, and a time-dependent decaying reduced relaxation function $G(t)$:

$$\mathbf{S}(t) = \int_0^t G(t - \xi) \frac{\partial \mathbf{S}^e}{\partial \mathbf{E}} \frac{\partial \mathbf{E}}{\partial \xi} d\xi \quad (7)$$

For simplicity, brain tissue is assumed to be isotropic and $\mathbf{S}^e$ is determined from a compressible generalized Rivlin strain energy density function W :

$$W = C_{10}(J_1 - 3) + C_{01}(J_2 - 3) + C_{11}(J_1 - 3)(J_2 - 3) + \frac{1}{2}K(J - 1)^2 \quad (8)$$

The rst three terms describe the material behavior in distortion (no volume change) and the last term is for dilatation. Parameters C_{10} , C_{01} and C_{11} are the Rivlin material parameters and K is the bulk modulus. J_1 and J_2 are the rst and second invariants of the isochoric part of the right Cauchy-Green strain tensor, and $J = \det(\mathbf{F})$ is the determinant of the deformation gradient tensor, which represents change of volume. The 1D instantaneous stress-strain relationship (P^e vs. ϵ) can be simplified as a polynomial expansion that vanishes at $\epsilon = 0$:

$$P^e = p_1^e \epsilon + \frac{1}{2} p_2^e \epsilon^2 + \dots \quad (9)$$

where

$$\begin{aligned} p_1^e &= \left(\frac{8}{3} (C_{10} + C_{01}) + K \right), \\ p_2^e &= - \left(\frac{56}{9} C_{10} + \frac{152}{9} C_{01} \right) \end{aligned}$$

are the rst-order (linear) and second-order moduli, respectively. It is clear from Equation (9) that stiffening or softening of the material behavior near $\epsilon = 0$ is dependent on the sign of p_2^e . Also for the material to be physically accurate, the elastic modulus in this region should be positive ($p_1^e > 0$) to give positive energy near $\epsilon = 0$. As for the rate-dependence of brain tissue, the following relaxation function in the form of a Prony series was employed:

$$G(t) = \sum_{i=1}^6 G_i e^{-\beta_i t} + G_\infty \quad (10)$$

Characterization of the material properties given in Equations (9) and (10), which is suitable for BINT modeling, is described in detail in Laksari *et al.* [18]. A summary of the results is given here. The elastic response P^e (Equation 9) was derived as:

$$P^e(\epsilon) = 2749.41\epsilon - 39271.6\epsilon^2 + \dots \quad (kPa) \quad (11)$$

Equation (11) shows that near $\epsilon = 0$ the response of brain tissue is stiffening in compression but is softening in tension. Therefore it is expected that shock waves could form for compressive waves.

The reduced relaxation function $G(t)$ in Equation (10) was determined as:

$$G(t) = 0.5894e^{-1702t} + 0.3929e^{-719t} + 0.009e^{-100t} + 0.0027e^{-10t} + 0.0016e^{-t} + 0.0016e^{-0.1t} + 0.0028 \quad (12)$$

The first two terms that decay after about 4 milliseconds are the dominant terms in Equation (12) indicating that significant reduction of stress occurs in the first few milliseconds after an abrupt loading of brain tissue. How this QLV model would affect the formation of shock waves in brain tissue is discussed in the next section.

5 Theory of Nonlinear Wave Propagation in Brain Tissue

Based on the nonlinear theory of wave propagation in materials with memory developed by Coleman and his colleagues [7] in a one-dimensional medium that is initially at rest, an acceleration wave (a wave with a jump in the acceleration at the wave front but continuous in velocity and strain) either disperses or evolves into a shock wave (discontinuity in velocity and strain) depending on the initial value of the jump and the stress-strain relationship of the material. The equation that describes the evolution of the jump in acceleration (α) can be written as:

$$\alpha(t) = \frac{\alpha_c}{\left(\frac{\alpha_c}{\alpha_0} - 1\right)e^{t/\tau} + 1} \quad (13)$$

where α_c is the critical amplitude, beyond which the acceleration wave grows and becomes a shock wave (when $t \rightarrow \infty$), α_0 is the initial value of $\alpha(t)$, and T is the time-constant for the change in $\alpha(t)$. The parameters α_c and T are dependent on the material parameters and can be written as:

$$\alpha_c = \frac{p_1^e}{p_2^e} G'(0) U_0, \quad \tau = -\frac{2}{G'(0)} \quad (14)$$

where U_0 is the wave velocity at zero strain, i.e., $U_0 = c(\epsilon=0) = \sqrt{p_1^2/\rho_0}$ and $G'(0) = dG/dt(t=0)$ is the initial slope of the relaxation curve that is negative for real materials. For brain material properties presented in Section (4) the acceleration wave parameters, as reported in [18], become $U_0 = 52.43 \text{ m/s}$, $\alpha_c = 481.59 \text{ g}$ ($1 \text{ g} = 9.81 \text{ m/s}^2$), and $T = 1.55 \text{ ms}$. These values are used to verify the numerical algorithm that is developed in this study for determining shock wave formation in brain tissue, which is described in the next section.

6 Discontinuous Galerkin (DG) Finite Element Model

A Discontinuous Galerkin (DG) FE algorithm was implemented in this study to investigate the propagation of acceleration waves and formation of shock waves in brain tissue. The formulations used are presented in this section briefly following the works by Cockburn and colleagues [19]. The governing Equation (4) for a 1D medium can be written in the following vector form:

$$\frac{\partial \mathbf{q}}{\partial t} + \frac{\partial \mathbf{f}(\mathbf{q})}{\partial x} = 0 \quad (15)$$

where

$$\mathbf{q} = \begin{bmatrix} \epsilon \\ v \end{bmatrix} \quad \text{and} \quad \mathbf{f} = \begin{bmatrix} -v \\ -P/\rho_0 \end{bmatrix}$$

represent the primary variable and the ux (secondary variable) vectors, respectively. After the inner product of the above equation with N shape functions $\psi_j(x)$ and twice integrating by parts, the strong DG form of the partial differential equation can be written as:

$$\int_{D^k} \left(\frac{\partial \mathbf{q}}{\partial t} + \frac{\partial \mathbf{f}(\mathbf{q})}{\partial x} \right) \psi_i \, dx = [\mathbf{f}(\mathbf{q}) - \mathbf{f}^*(\mathbf{q})) \psi_i]_{x_l^k}^{x_r^k} \quad (16)$$

where D^k represents the k^{th} element of a series of non-overlapping elements that approximate the domain of the numerical solution. Subscriptions r and l represent the values at the two ends (right and left) of the 1D element. $\mathbf{f}^*$ is a numerical ux vector that compensates for the difference in nodal solutions obtained from adjacent elements. The choice of the numerical ux function is important for the stability of the DG scheme. In this study, the local Lax-Friedrichs ux was used that can be written as:

$$\mathbf{f}^*(\mathbf{q}^-, \mathbf{q}^+) = \frac{\mathbf{f}(\mathbf{q}^-) + \mathbf{f}(\mathbf{q}^+)}{2} + \frac{C}{2} \hat{\mathbf{n}} \cdot (\mathbf{q}^- - \mathbf{q}^+) \quad (17)$$

where superscripts⁻ and ⁺ represent the values in two adjacent elements and $\mathbf{n}$ is the outward pointing normal vector (+1 in the right side and -1 in the left), and C is the magnitude of the largest eigenvalue of the matrix $\mathbf{A} = \mathbf{f}'/\mathbf{q}$ (see Equation 5), representing the wave velocity in each element edge. The right-hand-side of Equation (16), for a continuous solution, as expected, will be zero.

Spatial discretization was accomplished using the same N shape functions for $\mathbf{q}$ and $\mathbf{f}$, considering that $\mathbf{f}(\mathbf{q})$ can generally be a nonlinear function of $\mathbf{q}$:

$$\mathbf{q}^k(x, t) = \sum_{i=1}^N \hat{\mathbf{q}}_i^k(t) \psi_i^k(x), \quad x \in D^k \quad (18)$$

$$\mathbf{f}^k(\mathbf{q}(x, t)) = \sum_{i=1}^N \hat{\mathbf{f}}_i^k(t) \psi_i^k(x) \quad (19)$$

where $\hat{\mathbf{f}}_i^k(t) = \mathbf{f}(\hat{\mathbf{q}}_i^k(t))$. In this manner, the combination of N equations similar to Equation (16) can be written in the following semi-discrete matrix form:

$$\mathcal{M}_{ij}^k \frac{d\hat{\mathbf{q}}_j}{dt} + \mathcal{S}_{ij}^k \hat{\mathbf{f}}_j^k = \left[(\mathbf{f}^k - \mathbf{f}^*) \psi_i(x) \right]_{x_i^k}^{x_r^k} \quad (20)$$

where

$$\mathcal{M}_{ij}^k = \int_{D^k} \psi_i \psi_j dx, \quad \mathcal{S}_{ij}^k = \int_{D^k} \psi_i \frac{\partial \psi_j}{\partial x} dx \quad (21)$$

are the mass and stiffness matrices, respectively. Equation (20) gives a nonlinear system of N equations for N unknowns $\hat{\mathbf{q}}_j$.

Normalized Legendre polynomials of order n were used as shape functions

$(\psi_n(x) = \hat{P}_{n-1}(x))$ with $N = 2$, i.e.,

$$\mathbf{q}^k(r, t) = \hat{\mathbf{q}}_1^k(t) \tilde{P}_0(r) + \hat{\mathbf{q}}_2^k(t) \tilde{P}_1(r) \quad (22)$$

where

$$\tilde{P}_0(r) = \frac{1}{\sqrt{2}}, \quad \tilde{P}_1(r) = \sqrt{\frac{3}{2}}r$$

and r is a normalized spatial variable with $r \in [-1, 1]$ along each element. These polynomials provide accurate results when integrated and give a diagonal mass matrix that can be easily inverted for numerical efficiency [13,20]. The integrations in Equation (21) were performed using the Gauss-Legendre-Lobatto quadrature. It is important to note again that the main feature of the DG method that is crucial for this application is that the solution can be discontinuous, i.e., for the same node i , $\hat{\mathbf{q}}_i$ in the left element and in the right element can be different. For the temporal integration of Equation (20) a third-order Runge-Kutta explicit scheme was used with adaptive time-stepping based on the maximum wave velocity in the entire domain. In addition, in order to avoid spurious oscillations near the discontinuities, a generalized slope limiter (Π^N) was utilized to ensure the conservation of physical properties as well as the formal accuracy of the numerical solution. The Π^N scheme makes use of the computational result as a piece-wise N^{th} -order polynomial and only applies limiting in the elements where oscillation is detected, therefore keeps the accuracy in regions of smooth solution [13].

For numerical simulations, a one-dimensional domain with brain material properties (a bar) with 130 mm length (average anatomical length for human brain in the anterior-posterior direction [21]) was considered as shown in Figure (1). The bar was considered to be fixed at

one end (no displacement) and free at the other to deform based on the loading input. At the free end, displacement and pressure boundary conditions were considered. The displacement profile is shown in Figure (1) on the left, where the maximum displacement was 1 mm (0.8% strain) and the ramp speeds resulted in average 400 and 800 s^{-1} strain rates. These ramp speeds were chosen based on the predication made from the nonlinear theory for the critical amplitude of acceleration wave to cover a range of changes in peak acceleration smaller or larger than the critical value. In the second set of simulations, based on the experimental pressure measurements in a rat BINT model with BOP generated from a shock tube and pressures measured in the third ventricle [22], two pressure profiles were scaled and applied to the model. The scaling factor of the two pressure profiles were determined to give initial peak pressures (p_{max}) according to the lower end (50 kPa) and mid-range (200 kPa) of the field measurements pertaining to blast induced brain injury as a result of blast explosions [23].

In the following sections, the results of the numerical simulations obtained for two different material models are discussed. First, only the hyperelastic part of the brain material model was considered. In this case, the input wave is expected to travel through the material with no decay; however, the steepening effect of the stiffening material is expected to be observed. Secondly, the complete QLV model was used to investigate the interaction between the nonlinear and damping effects of the material model.

The spatial resolution for reported results was 9 μm and an adaptive time-stepping was used for temporal integration. Comparison of the h -convergence (keeping the order of the polynomial constant and successively increasing the number of elements) and p -convergence (keeping the number of elements constant and increasing the order of the polynomial) with polynomials of up to order 5 ($\tilde{P}_5(r)$) showed a linear h -convergence and an exponential p -convergence.

7 Simulation Results

7.1 Hyperelastic Results

For simulations with hyperelastic material properties, as mentioned above, the material parameters were taken from the instantaneous hyperelastic parameters given in Section (4). In order to validate the DG results, a similar model was developed in LS-DYNA (LSTC, Livermore, CA), which is a commercially available explicit nonlinear finite element code. In the LSDYNA simulation, a three-dimensional bar was considered and constrained in two dimensions, allowing the deformation to take place only along one axis with the same spatial resolution as the DG model. The boundary conditions for the two ends of the bar were prescribed in the same fashion as described above. For the hyperelastic material model, *MAT-077-H* was used, which simulates a compressible generalized Rivlin material with an additional of Ogden energy function to account for the hydrostatic work [14]. The resulting strain energy function was identical to Equation (8).

The LS-DYNA code uses the method of bulk viscosity in order to treat shock waves. In this method, a viscous term is added to the hydrostatic pressure response that smears shock discontinuities into high-gradient but smooth solutions and keeps the solution unperturbed

far from the shock front [24,25]. This step is taken to ensure that the solution remains unique in the domain and the continuity assumptions of the finite element method are satisfied. However, as a result, the gradients of strain and stress at the wave front are reduced. In this study, since comparing strain and velocity gradients were of primary concern, care was taken so that the effect of bulk viscosity was reduced to a minimum as explained in the LS-DYNA user's manual ($Q_1 = 1.5$, $Q_2 = 0.06$). In order to give a measure of steepening of the stress waves, the value P'_{max} defined as the maximum stress gradient at the wave front normalized by the initial maximum stress gradient was calculated (Equation 23). Here, the gradient for a compressive wave in the shock front is reported as positive number.

$$P'_{max} = \frac{\left(\max \left(\frac{\partial P}{\partial x} \right) \right)_t}{\left(\max \left(\frac{\partial P}{\partial x} \right) \right)_{t=0+}} \quad (23)$$

While in theory P'_{max} becomes infinity when a shock wave is formed, in numerical solutions, due to spatial discretization, this value stays finite. Since in real brain tissue, shock fronts expectedly have finite thicknesses, P'_{max} gives a quantitative measure for comparing the steepness of the numerical shock front for models with equal spatial resolution.

7.1.1 Displacement Input—Figure (2) shows the results of DG and LS-DYNA simulations to the displacement input at the low and high strain rates (400 and 800 s^{-1}). As can be seen, there is generally a good agreement between the two simulations in terms of maximum amplitudes. In the slower rate (top row), the stress wave propagates through the medium with no significant shape change. In the case of the higher rate (bottom row), the stress wave steepens in the wave front, representing formation of a shock wave. The DG simulation predicts a higher P'_{max} in both cases. This difference becomes significant in the high rate case after about 1.3 ms, which means that, as the wave propagates deeper in the model, the DG simulation predicts a much steeper shock front compared to LS-DYNA. These simulations showed that the applied low strain rate was indeed subcritical and the high strain rate was supracritical.

The results of this section showed that the DG simulations showed a more rapid growth of P'_{max} . This effect was further studied for different spatial resolutions from 260 μm to 5 μm . This smaller length scale was chosen in order to compare the two methods at the cellular level of brain tissue and examine whether their difference in P'_{max} was dependent on the mesh resolution. For this purpose, the numerical shock thickness as a function of mesh resolution for the 800 s^{-1} strain rate was calculated as shown in Figure (3). The numerical shock thickness was defined as the distance between 95% and 5% of the maximum stress. As shown in the figure, if the effect of the shock wave on cellular features with 10 μm is to be studied, a mesh resolution of 5 μm is needed for the DG method. The shock thickness with the LS-DYNA model, for the same mesh resolution, will be more than 100 μm , which means that the cellular features effectively will not see any stress gradient. In other words, mesh refinement to model the geometry of brain cellular features in the LS-DYNA model is not sufficient to study the effect of shock waves on these features.

7.1.2 Pressure Input—In the pressure input simulations (Figure 4) also the DG and LS-DYNA simulations showed good agreement in the terms of the amplitude of the stress wave. The shape of the wave was unchanged in the low pressure (50kPa) input while for the higher pressure (200kPa) input the wave front became significantly steeper with time. In the case of pressure input, similar to displacement input, the stress gradient at the wave front was significantly higher in the DG model for the higher pressure input.

7.2 Viscoelastic Results

The DG model explained in Section (6) was used with the QLV material model presented in Section (4) to investigate shock wave formation in the viscoelastic brain tissue. Since LS-DYNA material library does not include a QLV model similar to the one described by Equation (7), the validation of the DG code in this case was performed against the theoretical results of acceleration waves in nonlinear viscoelastic materials explained in Section (5). For these validations a constant velocity input was considered that resulted in an initial jump in acceleration (acceleration wave) applied at the left boundary.

7.2.1 Jump in Acceleration—Acceleration waves with subcritical and supracritical initial conditions ($\alpha_0 = 100, 1000 \text{ g}$ respectively) were considered entering the free end of a long (1 m bar) 1D QLV model with brain material properties. The evolution of the acceleration wave, shown in Figure (5), agrees with the theoretical predictions given in Equation (13). For the subcritical initial condition, the acceleration wave is reduced over time and for the supra-critical initial condition, it is increased, which indicates formation of a shock wave. These results were considered as validation of the DG QLV model.

7.2.2 Displacement Input—The simulation results for displacement input with the QLV brain material (Figure 6) show that while the peak amplitude is decreasing for both rates as a result of viscoelastic damping, the stress gradient at the wave front initially increases in the case of the supracritical rate before it eventually decreases and the wave dissipates. This indicates that while the risk of injury due to peak stress is reduced as the stress wave is traveling inside the viscoelastic brain tissue, the risk of injury due to stress gradient may be increasing. This occurs when the initial loading rate surpasses the critical rate and while the wave has already penetrated deep into the tissue.

7.2.3 Pressure Input—The two pressure levels that resulted in significantly different steepening behavior of the wave front in the hyperelastic model were compared in the QLV model. In the case of pressure input, similar to the case of displacement input, the shape of the wave was changed in the case of supracritical input and was unchanged for subcritical input (Figure 7). It is clear from this figure that the stress gradient decreases at later times for both cases. The strain wave is also shown in the right graph. For a viscoelastic material, it is expected that the stress and strain waves have different trends due to the stress relaxation phenomena. The formation of shock discontinuity is more evident in the strain waves. However, even strain waves eventually become smooth as the wave dissipates. This means that high strain and stress gradients are reached in a limited time interval and therefore a limited spatial domain. Therefore, as in the case of displacement input, it can be concluded

that for a realistic pressure input to brain, a limited spatial domain in the tissue, away from the exposed boundary, will be at higher risk of injury due to high strain or stress gradients.

8 A Proposed Injury Mechanism of Nervous Tissue under Blast Loading

Based on the reported research over the past several decades on the mechanisms of TBI as a result of car accidents and sports injuries, three main mechanisms have been proposed namely, intracranial pressure, tissue stress and strain, and relative motion between brain and skull [26]. In recent years, due to improvements in body armor and protection against blast exposures, the number of soldiers who survive with various severity of BINT has increased [27]. Contrary to the brain tissue deformations induced in car accidents, where shear deformation plays an important role [28], the deformations created as a result of BOP are volumetric in nature and primarily result in changes in the hydrostatic pressure. Due to the incompressibility of brain tissue, the volumetric deformations are generally small in magnitude [29,30]. Also the peak pressure decreases as a pressure wave travels in the tissue due to viscoelastic damping (see Section 7.2.3). Therefore, it is yet puzzling how BOP results in diffuse brain injuries as in the case of shear-induced TBI. In this study, as introduced in [18], it is argued that the one of the characteristics of stress waves that may cause injury in the brain is high spatial gradient of stress and strain that may occur at the wave front. The high spatial gradients also occur at high temporal rates, which contributes to their injurious effect.

While it is not yet clear which elements of neural tissue microstructure are more vulnerable to biomechanical stresses and deformations, two mechanisms are conceivable for the mechanical failure of these elements. First, local stress and strain exceed certain threshold in a region encompassing multiple cellular structures, damaging several microstructural elements. Second, spatial gradients of stress and strain grow quickly, which cause neighboring microstructural elements or the two ends of a single element undergo large relative motions. The latter mechanism that is proposed here as a mechanism of injury, can occur even at stresses and strains that are below the injury threshold in the first mechanism.

To make this point clearer, a schematic of the nervous tissue including axons, neurons, astrocytes, oligodendrocytes, and small blood vessels are given in Figure (8). The length scale of these microstructural elements is in the range of $1 - 30 \mu m$ [31]. In such small spatial resolution, the stress gradients that can be induced from propagating dilatational and shear waves, where no shock is present, will be small. To affect this length scales, the waves need to have frequencies at several MHz to GHz, which would be highly dissipative. However, the waves can evolve into a shock wave, due to material nonlinearity, and create high stress and strain gradients relevant to the length scales observed in the nervous tissue microstructural elements. It is shown in the figure that the method of bulk viscosity that is used in the LS-DYNA FE code to handle shock waves, is not able to capture the wave front thicknesses as small as the microstructural elements when the mesh geometry is at the length scale of these elements. This observation regarding the length scale of the shock front thickness signifies the application of the DG method in handling shock waves in this study. The effect of strain rate in neuronal injury has been shown in previous studies [32–34]. Therefore, as mentioned earlier, occurrence of high strain rates due to shock waves can

therefore exacerbate the effect of the relative motion that is caused by the high stress and strain gradients at the shock front and increase the risk of injury.

9 Discussion

For numerical simulation of nonlinear wave propagation in brain tissue a constitutive material model is needed that encompasses the nonlinearity as well as the rate dependence of the tissue relevant to BINT modeling. To predict the development of shock waves in brain tissue two parameters are particularly important in governing the behavior of an acceleration wave: the critical amplitude, beyond which an acceleration wave grows in time, and the decay rate, which determines how fast this growth occurs. These parameters, in turn, depend on the tissue material properties. Specifically, in addition to the slope of the instantaneous stress-strain curve (tangent modulus), the second-order modulus and the initial rate of reduction of the relaxation function determine the acceleration wave parameters. For brain material properties that are suitable for BINT applications, the results described in an earlier work by our group [18] were employed. Briefly, in that study a combination of medium rate finite compression test results and high rate finite torsion results were used to derive the material properties of brain tissue. Based on those material properties, it was confirmed that formation of shock waves in brain tissue is possible when the initial jump in compressive acceleration is beyond 482g. The growth time constant of the acceleration wave was determined to be 1.6ms, which indicates that significant growth can occur before the shock wave amplitude dissipates within the length scale of human brain.

The acceleration wave parameters clearly depend on the material properties of brain tissue and since determining the material properties directly for BINT applications is not a trivial task, it is important to do a parametric study to estimate the variations in the acceleration wave parameters. Since brain is an almost incompressible material, its most debatable material property is bulk modulus. In material characterization and numerical simulations it is common to assume a value of 0.49 for the linear Poisson's ratio of brain tissue [35, 36]. As described in [18], our group varied the Poisson's ratio between 0.485 and 0.495 and it was found that the values for the critical amplitude (319g to 996g) still fall within the range pertaining to blast loading conditions in the field.

In implementation of the discontinuous Galerkin (DG) finite element formulation, a one-dimensional model of brain tissue was studied. This simplification was necessary as the complexity of the problem requires a step by step approach and validation of the modeling work at each step. This model was nevertheless sufficient to argue the main point of this work which was the formation of shock waves in brain tissue in time scales and length scales relevant to BINT. It is expected that the possibility of transformation of compressive waves into shock waves is also valid for three-dimensions (3D). In 3D, there would be two types of waves propagating in brain tissue, namely longitudinal (dilatational) and shear (distortional) waves. The two wave types propagate at drastically different speeds due to brain incompressibility and therefore the time scale of their effects may be different. While a blast over pressure may result in primarily longitudinal waves in brain tissue, shear waves can also form upon reflections from boundaries (e.g., skull) and tissue interfaces (e.g., white/gray matter, or brain/vasculature). Each of these wave types can evolve into shock

waves due to material nonlinearity and result in gradient-induced injuries. A 3D model to study shock wave formation would therefore require validated representations of the brain tissue boundaries and interfaces at high loading rates.

While the model studied was finite with fixed boundary condition at one end, its length was long enough for shocks to form before the compressive wave reached the fixed boundary. At the fixed boundary, the stress wave would reflect. Reflection of waves in brain tissue was beyond the scope of this study and as explained above, a 3D model would be more suitable to study reflection. At the free boundary, since experimental data for the brain boundary conditions as a result of blast over pressures entering the head are lacking, two loading conditions that were considered to be relevant were chosen. In the displacement-input simulations (e.g., from compression of the skull), less than 1% nominal strain at different rates was applied with a smooth profile that resulted in peak accelerations below and above the critical acceleration. In the pressure-input simulations (e.g., pressure wave passing through the scalp and skull), pressure profiles reported in [22] for rat brain were selected as the basis and the peak pressure was scaled based on blast injuries reported in pigs [23]. Clearly for future studies directly measured boundary conditions that result in various degrees of BINT are needed.

The DG model was validated in two steps. First, it was validated for a hyperelastic material model. Although this is a special case of the nonlinear viscoelastic material model with no dissipation or constant reduced relaxation function, it gave the advantage that the DG model could be validated against commercial nonlinear finite element codes (LS-DYNA in this case). As a result, validation could be performed for the model with finite length and with arbitrary displacement-input or pressure-input boundary conditions. Additionally, comparison of the DG model with the LS-DYNA model showed that for capturing shock thicknesses at the length scale of neural tissue cellular features, the number of elements in the LS-DYNA model would become prohibitively large. This is due to the limitation of the method of bulk viscosity in smoothing a shock wave discontinuity [14]. To simulate a shock front thickness for neuronal injury, the mesh resolution of the DG model can be in the same order of the cells length scales while LS-DYNA, for the same mesh resolution, yielded a shock front thickness that was one order of magnitude larger. As a result, in the LS-DYNA simulations, individual neurons would not undergo significant stress and strain gradients as the shock wave passes through them. The second step of the validation of the DG code was with regard to the implementation of the nonlinear viscoelastic (QLV) material model. To this end, since the commercial codes do not have such material model, the only available theoretical prediction (propagation of 1D acceleration wave in a semi-infinite medium initially at rest) was utilized.

Following the validations mentioned above, the DG code with QLV material model was used to investigate the nature of shock wave formation for displacement-input and pressure-input boundary conditions. An important result, which was not predicted by the theory, was that the stress and strain gradients at the shock front initially increased for supracritical conditions and then decreased. The increase in the gradients occurred while the wave amplitude was still significant. Over time, the viscoelastic damping caused the gradient to decrease and the wave to eventually dissipate. The implication of this finding is that while

the risk of injury due to high stress or strain continually decreases as the stress wave propagates in the brain tissue, the risk of injury due to high stress or strain gradient increases for some time period. Therefore, regions in the tissue that are away from the boundary may be at higher risk for gradient-induced injury. This result can partly explain the diffuse nature of injuries observed in BINT. It is not yet clear whether strain is a better measure for brain injury or stress. In terms of gradients, the simulations showed that at the shock front, strain gradients were higher, in a normalized sense, compared to stress gradients. This difference can be explained based on viscoelastic stress relaxation. It can be concluded that an injury model that is based on strain gradient, compared to stress gradient, may put the brain cells at a higher risk.

Variations in peak stress amplitude P'_{max} and normalized stress gradient P'_{max} at the shock front for the cases of subcritical and supracritical displacement inputs are demonstrated as a function of time in Figure (9). In this figure, the QLV simulation results are compared with simulation results with a linear viscoelastic material model for brain tissue (ignoring the second-order modulus). The figure shows that P'_{max} first increases as the displacement input reaches its peak. Due to viscoelasticity, the peak stress occurs after the peak strain and gradually dissipates. The QLV model, as expected (with a stiffening nonlinear stress-strain curve), predicts higher P_{max} than the linear model. The change in P'_{max} at the shock front in the QLV model is higher than the linear model and this difference, particularly for supracritical input, is drastic. The figure shows that a linear approximation would significantly underestimate gradient-induced injuries in brain tissue. In the QLV model subject to a supracritical input, P'_{max} increases significantly within 1.5ms, which indicates formation of a shock wave. Later, P'_{max} decreases as the damping effect of viscoelasticity takes over and dissipates the wave.

Experimental validation of the results presented in this study is currently difficult if not impossible since visualization of shock waves in a soft solid material is technically challenging. An alternative approach would be to induce blast brain injuries in an animal model in which the severity of injury can be varied in a controlled manner and compare the observed injury patterns with computational results. There would be challenges also in this approach. For example, not every detail in the animal brain can be modeled in the computational model. Additionally, the injuries would result from other mechanisms in addition to high gradient (e.g., high pressure and blood brain barrier breakage). Also the experimental pressure measurements will be limited to larger blood vessels and brain ventricles.

10 Conclusion

Simulations with the DG finite element code with nonlinear viscoelastic material model developed in this study showed that, for both displacement-input and pressure-input boundary conditions that represent blast loading conditions, compressive stress waves can develop into shock waves in brain tissue. Whether a shock wave will form depends on the intensity of the input and brain tissue material properties. A shock wave results in large gradients in stress and strain across brain tissue cellular features. These large gradients occur a few milliseconds after the peak stress and therefore may occur deep in the tissue away

from the incident surface. Local cellular high rate relative motions that are generated due to the passage of a shock wave may result in a type of injury that is termed high gradient-induced injury. Gradient-induced injuries would be distinct from injuries caused solely by high stress or strain amplitudes in terms of their location and mechanical deformation of the cells.

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Fig. 1.

On the left is a schematic of the 1D bar and the applied displacement input. The left boundary is compressed for 1 mm at $t_0 = 2.5$ and 1.25 ms corresponding to two strain rates (400 and 800 s^{-1}). On the right, a schematic of the 1D bar with the pressure boundary condition is given, where p_{max} is the peak over-pressure studied for two cases ($p_{max} = 50$ and 200 kPa).

Fig. 2. Stress (left column) and normalized maximum stress gradient P'_{max} (right column) as the wave propagates through the tissue for two strain rates of 400 s^{-1} (top row) and 800 s^{-1} (bottom row). The results in the left column are shown for three time snapshots $t \in \{2, 2.5, 3\} \text{ ms}$ from left to right respectively.

Fig. 3. Numerical shock front thickness as a function of element size (ΔX) for the strain rate of $800 s^{-1}$. The length scales relevant to brain tissue cellular features are shown.

Fig. 4. Stress response in the hyperelastic brain material as exposed to pressure inputs with 50 kPa (left) and 200 kPa (right) peak pressures at the boundary for three time snapshots $t \in \{2, 2.5, 3\}$ from left to right respectively.

Fig. 5.

Evolution of the acceleration wave for two cases of $\alpha_0 = 100$ g (subcritical) and $\alpha_0 = 1,000$ g (supracritical) initial jumps in acceleration.

Fig. 6. Viscoelastic stress wave results for 400 s^{-1} (left) and 800 s^{-1} (right) strain rate displacement inputs for time snapshots $t \in \{1, 1.5, 2, 2.5, 3\} \text{ ms}$ from left to right respectively.

Fig. 7.

The left two graphs show the stress response in the viscoelastic model as exposed to pressure inputs with 50kPa (top) and 200kPa (bottom) peak pressures at the boundary. The right graphs show the strain results for the same loading condition, where a shock discontinuity in strain is evident in the 200kPa case. In all graphs, four snapshots of $t \in \{1.5, 2, 2.5, 3\}$ are shown from left to right respectively.

Fig. 8. Schematic of nervous tissue microstructural elements interacting with a shock front. When the stress gradient is significant at the length scale of these elements, it will cause relative motion between the elements that may result in injury. The image was reproduced from [18].

Fig. 9.

Time histories of P_{max} (left columns) and P'_{max} (right column) for QLV and Linear viscoelastic models at 400 s^{-1} (top row) and 800 s^{-1} (bottom row) strain rates.