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Research Paper

Mechanical response of brain tissue under blast loading



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ABSTRACT

In this study, a framework for understanding the propagation of stress waves in brain tissue under blast loading has been developed. It was shown that tissue nonlinearity and rate dependence are the key parameters in predicting the mechanical behavior under such loadings, as they determine whether traveling waves could become steeper and eventually evolve into shock discontinuities. To investigate this phenomenon, in the present study, brain tissue has been characterized as a quasi-linear viscoelastic (QLV) material and a nonlinear constitutive model has been developed for the tissue that spans from medium loading rates up to blast rates. It was shown that development of shock waves is possible inside the head in response to high rate compressive pressure waves. Finally, it was argued that injury to the nervous tissue at the microstructural level could be partly attributed to the high stress gradients with high rates generated at the shock front and this was proposed as a mechanism of injury in brain tissue.

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1. Introduction

While motor vehicle accidents and sports related injuries remain the major causes of traumatic brain injury (TBI) (Elkin and Morrison, 2007; CDC 2011), blast-induced neurotrauma (BINT) has recently become of increasing concern (Cernak and Noble-Haesslein, 2010; Harrigan et al., 2010). BINT is called the signature wound of Iraq and Afghanistan wars affecting almost 20% of the soldiers (Elder and Cristian, 2009). However, the mechanisms of such injuries are not yet completely understood and the available experimental and computational models to simulate blast injuries are currently limited (Nyein et al., 2010; Cernak and Noble-Haesslein, 2010), signifying the need for improvement in such models.

Based on what is known about the mechanics and pathology of BINT, it can be concluded that some form of stress

wave with high strain rate propagates deep into the brain tissue. When the head is subjected to blast over-pressure (BOP), assuming a linear approximation, dilatational and shear stress waves may propagate in brain tissue at two different time scales. The time scale of shear stress wave propagation would be in the order of 10 ms and would be similar to shear deformation rates that occur in automotive accidents (Zhang et al., 2004). The dilatational waves, however, due to low compressibility of brain tissue, would propagate at a much smaller time scale (around 10 μ s) and it is expected to be primarily significant in BINT due to the high amplitude of the incident pressure waves (50 to 500 kPa for mild to severe injuries) (Cernak et al., 2011). In addition to the amplitude of the intracranial pressure, its rate is expected to play a significant role in causing injury. For an almost incompressible brain material, only small dilatational

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deformations can be generated due to pressure, which at low rates are not expected to result in injury. Experimental results on cultured brain tissue show that small displacements applied at high rates may result in injury (Elkin and Morrison, 2007). Pathology of BINT shows that the injury is generally diffuse and affects regions deep at the center of the brain, e.g. hippocampus, implying that the stress waves travel throughout the tissue without significant damping (Levin and Wilde, 2010; Cernak and Noble-Haeusslein, 2010).

Brain tissue has been shown to exhibit nonlinear viscoelastic behavior in response to external loading (Miller and Chinzei, 1997; Laksari et al., 2012). Due to this nonlinearity, based on theoretical and experimental results, it is conceivable that dilatational waves steepen at the wave front and even develop into shock waves, resulting in high strain rates and gradients and therefore tissue injury. As the rate of loading increases the resulting stress waves attenuate at higher rates (Valdez and Balachandran, 2013). The maximum attenuation of the stress wave, which correlates with the amount of energy dissipation, coincides with the regions experiencing the shock front in a nonlinear viscoelastic medium, leading to localized large accumulations of energy. This highly concentrated and high-gradient loading may eventually result in tissue damage as the microstructural elements of the nervous tissue cannot bear such loads.

Coleman and colleagues developed the foundations of propagation of acceleration waves and shock waves in nonlinear viscoelastic media (Coleman and Noll, 1961; Coleman et al., 1964). Based on their theory, confirmed by experimental results in polymers (Barker, 1970; Nunziato and Schuler, 1973; Baer et al., 1996), when certain nonlinear conditions are met, acceleration waves (waves with discontinuous acceleration and rate of deformation fields) can develop into shock waves (waves with discontinuous velocity and deformation fields) in the medium. On the contrary, the discontinuity of acceleration waves in linear viscoelastic materials decays in time and space leading to continuous acceleration and reduced rate of deformation fields and therefore shock waves can never develop in such media. Therefore in order to accurately predict and model the propagation of blast-induced stress waves in brain tissue, the need for material characterization that includes both the nonlinearity of the tissue as well as its rate dependence for blast loading conditions is of critical importance. However, in recent studies, linear viscoelastic constitutive models have been primarily used for modeling blast-induced waves in brain tissue (Chandra et al., 2012; Zhu et al., 2011).

In this study, a quasi-linear viscoelastic (QLV) material model was developed for brain tissue encompassing blast loading-rates. Subsequently, the propagation of dilatational waves at BOP time scales (a few milliseconds) was studied and it was shown that with large enough pressure amplitudes, acceleration waves can develop into shock waves. It is proposed that such shock waves can be a major contributor to blast related injuries.

2. Propagation of acceleration waves and shock waves

An acceleration wave is a mechanical wave whose velocity (v) and strain (E) fields are continuous functions of space (X) and

time (t), whereas their temporal and spatial derivatives ($\partial v/\partial t, \partial E/\partial t, \partial E/\partial X$) have a single jump discontinuity across the wave front. To demonstrate how acceleration wave parameters depend on the material properties in a concise manner, a one-dimensional medium will be considered in the following analysis. The amplitude of discontinuity in an acceleration wave, denoted by $\alpha(t)$, is defined as:

$$\alpha(t) = \left[\frac{\partial v}{\partial t} \right] \tag{1}$$

where $[\]$ represents the jump in the function at the wave front (difference between values immediately before and after the discontinuity). This is a particularly significant parameter when studying BOP traveling across brain tissue since, as will be discussed later, the magnitude of $\alpha(t)$ is related to the incoming pressure profile. An important aspect of the analysis of wave propagation is determining the evolution of $\alpha(t)$. Particularly, it can be shown that in nonlinear materials, $\alpha(t)$ can increase in time and lead to a discontinuity in velocity and strain. In materials with memory, i.e., viscoelastic materials, the evolution of $\alpha(t)$, when an acceleration wave enters a semi-infinite homogeneous region that is initially at rest, can be obtained by applying the Rankine–Hugoniot jump conditions (Appendix A) and written as:

$$\alpha(t) = \frac{\alpha_c}{((\alpha_c/\alpha_0) - 1)e^{t/\tau} + 1} \tag{2}$$

where τ and α_c are functions of the material properties and $\alpha_0 = \alpha(0)$. In Eq. (2), α_c acts as a critical amplitude for the acceleration wave discontinuity and $\tau > 0$ acts as the time constant for the change in $\alpha(t)$. For an acceleration wave there are three distinct possible cases. First, if $|\alpha_0| < |\alpha_c|$ or $\text{sgn}(\alpha_0) \neq \text{sgn}(\alpha_c)$, the discontinuity in acceleration monotonically decreases to zero ($\alpha(t) \rightarrow 0$). Second, if $|\alpha_0| = |\alpha_c|$ the acceleration wave is called self-preserving as its amplitude remains constant ($\alpha(t) = \alpha_0$). Finally, if $|\alpha_0| > |\alpha_c|$ and $\text{sgn}(\alpha_0) = \text{sgn}(\alpha_c)$, the acceleration wave will reach an infinite amplitude ($\alpha(t) \rightarrow \infty$) in a finite time (t_∞). In this case a shock wave, i.e., a discontinuity in velocity and strain, will form. This idea is further demonstrated in Fig. 1, where the jump in acceleration is represented by a kink in the velocity wave front. The angle of the kink depending on whether the initial jump in acceleration is smaller than, equal to, or larger than the critical amplitude, will increase, remain constant, or decrease toward 90°. The time of shock formation can be determined from the root of the denominator in Eq. (2) as:

$$t_\infty = - \ln \left(1 - \frac{\alpha_c}{\alpha_0} \right) \tau \tag{3}$$

In Fig. 1, the two cases where the acceleration amplitude is either decaying with t or increasing and blowing up at t_∞ are depicted. It should be noted that for compressive acceleration waves ($\alpha(t) > 0$) a shock wave can form when $\alpha_c > 0$ and conversely for tensile acceleration waves to become a shock wave $\alpha_c < 0$. The physical interpretation of the three cases mentioned above can be given based on the balance between the internal dissipation of viscoelastic materials and the nonlinearity of the instantaneous stress–strain response, which will be elaborated further in the subsequent section. The significance of the second and third cases in this study is

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