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Blast induced mild traumatic brain injury/concussion: A physical analysis

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Currently, a consensus exists that low intensity non-impact blast wave exposure leads to mild traumatic brain injury (mTBI). Considerable interest in this "invisible injury" has developed in the past few years but a disconnect remains between the biomedical outcomes and possible physical mechanisms causing mTBI. Here, we show that a shock wave travelling through the brain excites a phonon continuum that decays into specific acoustic waves with intensity exceeding brain tissue strength. Damage may occur within the period of the phonon wave, measured in tens to hundreds of nanometers, which makes the damage difficult to detect using conventional modalities.
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INTRODUCTION

Mechanisms promoting the increasingly recognized and prevalent mild brain injury/concussion affecting soldiers exposed to blast during conflicts in Iraq and Afghanistan remain unclear.[1,2] As the blast wave encounters the body, part of the wave may be reflected or, in some part, propagated through the body generating high frequency stress waves and lower frequency sheer waves which cause injury. Air containing organs or viscera were, in the past, considered to be most vulnerable to the effects of the shock wave front and the barometric overpressure of the blast.[3] Recent experience demonstrates occurrence of a subtle brain injury, mild traumatic brain injury (mTBI)/concussion, resulting from non impact primary blast injury seen in current conflicts,[3,4] and documented experimentally in low level primary blast exposure.[5] Conventional imaging with computerized tomography and magnetic resonance scanning, MRI, fail to reveal structural damage. Recently, diffusion tensor imaging MRI techniques showing changes in water diffusion, particularly in the white matter tracts, have attracted increasing attention[6] in soldiers exposed to blast.

A shock wave front passing through the human brain causes injury but the fundamental pathogenesis of mTBI/concussion remains obscure. The mode of transmission of the blast energy through skull is also incompletely understood, and the possibility of transmission of pressure from the thoracic or abdominal cavities also has been considered.[7,8] Irrespective of the mode of shock wave front or blast wave pressure injury, forces causing these injuries may occur at low shock wave intensities in the range of 10-11.5 kPa.[5,9] Physical explanation for these low level effects likely relates to the physical effects upon water content in brain tissue: 80% in gray matter and 70% in white matter.[10] In addition, the brain within the skull is bathed in watery cerebro-spinal fluid externally and internally within its ventricular system.

While lower shock wave intensities seem to cause mTBI, the mechanism of this effect remains unclear. The local strain and stress caused by such low shock wave intensity appears insufficient to cause direct tissue damage. Animal studies suggest that about ~10% brain tissue strain is needed.[11,12] Immediate anatomic and observable histological tissue changes can be minimal and inconsistent in primary blast shock models.[5,9] The expected damage length scale associated with a shock wave can be estimated. Using a 100 kPa pressure wave and a typical 1–10 microsecond rise time for the shock, the relevant length scale is of the order of 1–10 mm at 1 km/s propagation velocity. A force of this magnitude should have effects that are evident using conventional anatomic and imaging methods. In most cases of mTBI/concussion, tissue damage related to mTBI remains invisible to conventional brain imaging methods.

The fact that mTBI fails to provide easily observable traces suggests a physical mechanism likely related to the length scale of the shock wave. Mild brain injury may in the future, as a result of evolving research on spinal fluid or serum biomarkers,[13] demonstrate post injury markers including metabolic changes on MRI and blood brain diffusion changes occurring in the absence of anatomic pathologic change.[14,15] The possibility of concentrating or amplifying wave effects include the reflection of a shock wave from soft/hard interfaces, or shock wave interactions at anatomic boundaries between gray and white matter. In either case, reflection and focusing effects appear to be insufficient to change the linear scale to smaller dimensions. If the concentration effects increase the shock intensity by 10 times (the maximum observed numbers are ~20 times shock intensity increase), this only lowers the expected characteristic length scale of the damage to 0.1–1 mm.

We propose a mechanism that dramatically shortens the linear scale of the shock wave interaction within brain tissue. The example of a shock wave interacting with water is used, with the assumption that brain tissue physical properties on the whole are quantitatively similar to the properties of water. Cerebro-spinal fluid is even closer to water in its physical characteristics. This mechanism is based upon the

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dynamic behavior of phonons in water and predicts the length scale of damage to be ~200 nm. This phonon-based model has recently been shown to accurately describe failure waves in brittle solids.[16]

DISCUSSION OF MILD TBI MECHANISM: PHONON EXCITATION

A blast wave will subject a surface to a shock. A shock wave traveling through the brain is characterized by a shock front, which is a thermodynamic boundary between shocked and non-shocked states of water. The shock front thickness decreases in dimensions relative to the intensity of the shock or blast. For intense shocks, the shock wave front equals the interatomic spacing in the specific medium of propagation. The difference between the two states, the blast wave front and the blast wave pressure, is that some of the energy gets deposited behind the shock front, causing a change in thermodynamic parameters of pressure, volume (density), and temperature. In macroscopic terms, the energy deposited behind the shock front is responsible for acoustic attenuation of the shock energy, also called acoustic absorption. It is of interest to understand microscopically how this energy dissipates in the space behind the shock front. We approach this acoustic attenuation by considering the localized and delocalized phonon behavior at very short times (from 10^{-12} to 10^{-6} s) and in frequency space, energy space, and real space. It is easy to justify that short times, high frequency, and high energies are involved in the shock process by noting that the time is takes a shock caused by a blast to travel one atomic spacing (0.3 nm) in water is 2×10^{-12} s assuming a speed of sound in water of 1.48 km/s. Pressure applied to the material by the shock will excite phonons with frequencies up to $\sim 10^{12}$ Hz. Converting this frequency to energy by $E = h\nu$, where h is Planck's constant and ν is frequency yields $E = \sim 4.1 \times 10^{-3}$ eV.

Another way to estimate frequency, energy, and time of the phonon excitation is to use the usual macroscopic concept of particle velocity. From basic acoustics, the water particle velocity u is defined as $u = P/Z$, where P is the sound pressure (in Pascals) and Z is the acoustic impedance. At 37 °C, the water acoustic impedance $Z = 1.52 \times 10^6$ kg/(s·m^2). From the conservation of mass across the shock front (Ref. 17), the volume change for a planar shock is

$$V_0/V = c/(c - u). \quad (1)$$

Here, V and c are specific volume and speed of sound in shocked water where the subscript relates to unperturbed material and u is the particle velocity. Our inquiry concerns weak shocks where the speed of sound difference between shocked and non-shocked materials is negligible, so $c = c_0$ where c_0 is the non-shocked speed of sound in water. From Eq. (1) and for an example shock of 100 kPa overpressure the volume change is 4×10^{-5}. The corresponding maximum possible energy coupled into the water can be estimated from atomic displacement. Compressive and tensile water properties differ considerably such that the volume change is much larger for tensile loads. The dynamic strength of water increases with the strain rate, approaching 9.6 MPa at very high strain rates.[18] Assuming linear dependence of force on displacement, 100 kPa pressure corresponds then to 100 kPa/9.6 MPa or ~1% of dynamic binding energy.

Dynamic binding energy can be estimated from the evaporation energy of water (~0.47 eV per bond with understanding that this number is somewhat higher for a dynamic event[19]). One percent of the energy per bond provides an estimate of 4.7×10^{-3} eV energy deposition into the for a 100 kPa shock. This agrees well with the above estimate of 4.1×10^{-3} eV. This method of estimated energy in the shock front shows that this energy is dependent on the shock pressure.

Not all of the available energy is coupled to the water. However, liquid water is a disordered and non-compressible material. Typically, shock wave coupling is much higher for disordered materials than for crystalline materials. Attenuation of acoustic waves mainly depends on the wave frequency. Shock wave coupling is an evolving field, but insights have been gained from studies of vibrational mode coupling of absorbed molecules.[20] It is clear that translational modes do not couple to adjacent molecules while bending and rotational modes do couple, reinforcing the fact that energy does not couple in a solid material with an ideal defect-free cubic lattice.

PHONON SPECTRA OF WATER AND PHONON DECAY

We have established that phonons with frequencies of up to $\sim 10^{12}$ Hz can be readily excited by a shock wave with only 100 kPa overpressure. The phonon spectrum of water is well established.[21,22] There is a sharp drop in the water phonon density of states below $\sim 10^{11}$ Hz. After the passage of the shock front, the resulting excited phonon states decay into lower frequency states within $\sim 10^{-9}$ s.[23] Lower frequency phonons exhibit properties of both phonons (delocalized states) and acoustic waves (localized states) below a well-defined crossover frequency.[23] The region of interest in brain shock transmission is far below the molecular vibration (vibron) energies of water (0.2–1 eV), fitting into the lower end of the phonon spectrum continuum, which is below 1 meV or a $< \sim 0.3$ THz.

Vibration states below the phonon continuum have been measured by Brillouin spectroscopy.[24] The water condenses into ice for pressures above ~1 GPa with a pressure dependent phonon frequency of ~46 GHz. This pressure represents very intense blast and is, therefore, probably not related to mild brain injury. Liquid water has one main peak derived from non-equilibrium phonons excited by the shock wave. This peak position depends on many parameters but approximates an acoustic phonon excitation at 7.5 GHz.[25] At this point, multiple peaks in the density of states occur at frequencies below a few MHz, and these peaks overlap to form a ground level. A non-zero probability exists that some phonons will decay directly into the ground level, but this probability is much lower than for decay into the 7.5 GHz acoustic mode as decay probability exponentially depends on the energy difference between the levels. The corresponding

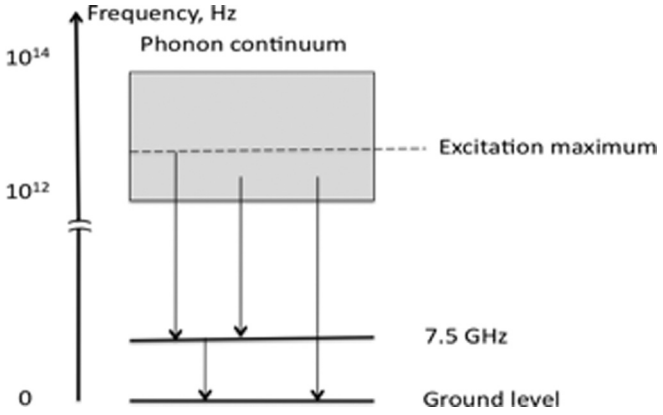

FIG. 1. Illustration of mild shock induced phonon excitation decay in water. The Y-axis gives an arbitrary frequency scale.

phonon excitation energy/frequency decay scheme is schematically illustrated in Figure 1.

CONSEQUENCE OF DECAY THROUGH 7.5 GHz ACOUSTIC MODE

The energy excitation maximum in water for a 100 kPa shock is at ~1.1 THz with orders-of-magnitude larger stored energy compared to the 7.5 GHz line. Rapid decay into this isolated line will produce a sine wave with increasing amplitude until the water ruptures at the sine wave maxima where the tensile force is highest. The corresponding wavelength of a sine wave at this frequency and for a 1.48 km/s speed of sound at 37 °C is 200 nm for 7.5 GHz line. This becomes the linear scale over which damage to brain tissue may be predicted from the rupture of water, which for the purpose of this consideration simulates a damaging process in brain tissue with high water content. Decay to the 7.5 GHz occurs in microseconds and possibly as short as ~100 nanoseconds. The brain damage caused by the rupture of water molecules happens within this short time frame as the shock wave passes though the brain.

The illustration of a phonon frequency causing rupture of water molecules is shown in Figure 2 for a specific point in the water volume. Pumping of a specific energy level, in this case corresponding to 7.5 GHz frequency, results in increasing amplitude of a sine wave. Water molecular bonds start to elongate. At some point, wave pressure exceeds dynamic tensile strength of water, which is approximately 9.6 MPa.[18] Then, the water ruptures with the period of a sine wave or 200 nm. The inset in Figure 2 schematically illustrates the rupture. The tensile strength of water may be a function of impurities and gas contents, therefore the actual value in brain tissue may be lower i*n vivo* than is illustrated by this model.

ANALOGY WITH BRITTLE MATERIALS

The model presented above has been validated for the brittle material soda-lime glass[16] that predicts the particle sizes of the broken glass from the phonon spectra of the glass. In Figure 3, we present a schematic picture of this process from Ref. 16 along side the same picture for water. For the glass, the shock wave front travels through the material and energy is stored behind the shock front due to coupling

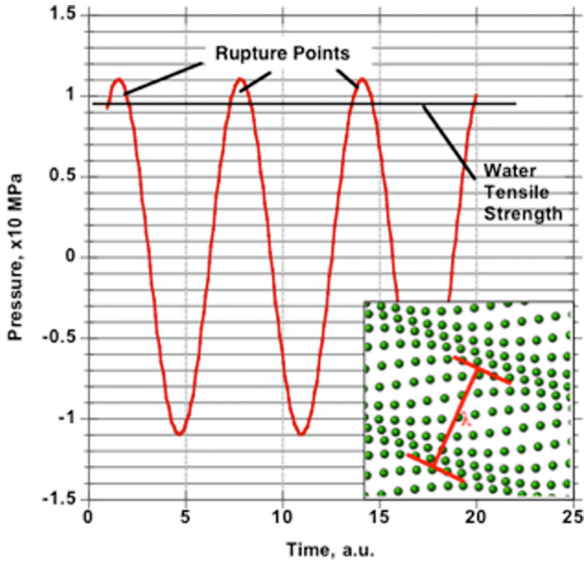

FIG. 2. Illustration of water rupture at a point in space in a pressure field that is induced by an acoustic wave formed by phonon decay. The sine wave period 133 ps for 7.5 GHz frequency. Time is given in arbitrary units. The dynamic tensile strength of water is shown as a horizontal solid line. When the sine wave amplitude exceeds the solid line, the water ruptures with the period of the wave. The inset schematically shows the development of water rupture in real space for a wavelength of ~50 nm (from Wikipedia: Brillouin scattering).

to the high frequency phonons. There is a ~microsecond delay before the glass breaks corresponding to the decay time of the high-energy phonons into the low frequency acoustic modes of the glass. Then, the glass breaks into 6 well-defined sizes from 120 to 7000 nm corresponding to 3 acoustic modes and two sound speeds. In the case of the water with one simple frequency, the water ruptures after a delay time while the high frequency phonons decay into this line. The water ruptures when the energy in the acoustic mode exceeds the tensile strength of the water, but then the water reforms, leaving no evidence behind that it has ruptured. However, subtle changes in cell structure, and composition may occur within approximate dimensions of 200 nm.

COMPARISON TO DATA

This model allows estimation of an absolute minimum pressure related to the rupture of water. We previously established that 0.1 MPa overpressure corresponds to 4.7×10^{-3} eV; 1 eV corresponds to a frequency of $2.4 \cdot 10^{14}$ Hz. Using linear scaling, and with the minimum energy phonon density peak of 7.5 GHz, the minimum overpressure to produce damage is approximately 9.5 kPa. This calculated value approximates experimental values ranging from 10–11.5 kPa in rat models, which produce brain cytoskeletal proteolysis, increased intracranial pressure, and chronic functional impairment.[5,9] In Ref. 9, pressure transducers implanted in the rat brains showed a 78%, 91%, and 140% increase in intercranial pressure 6, 10, and 10 h after blast exposure for 10, 30, and 60 kPa, respectively. Cognitive impairment, defined as the ratio of the time required to negotiate a maze after/before shock exposure, 2 days after exposure was 110% and 160% for 10 and 30 kPa, respectively. Cognitive impairment for the 30 kPa blast was still

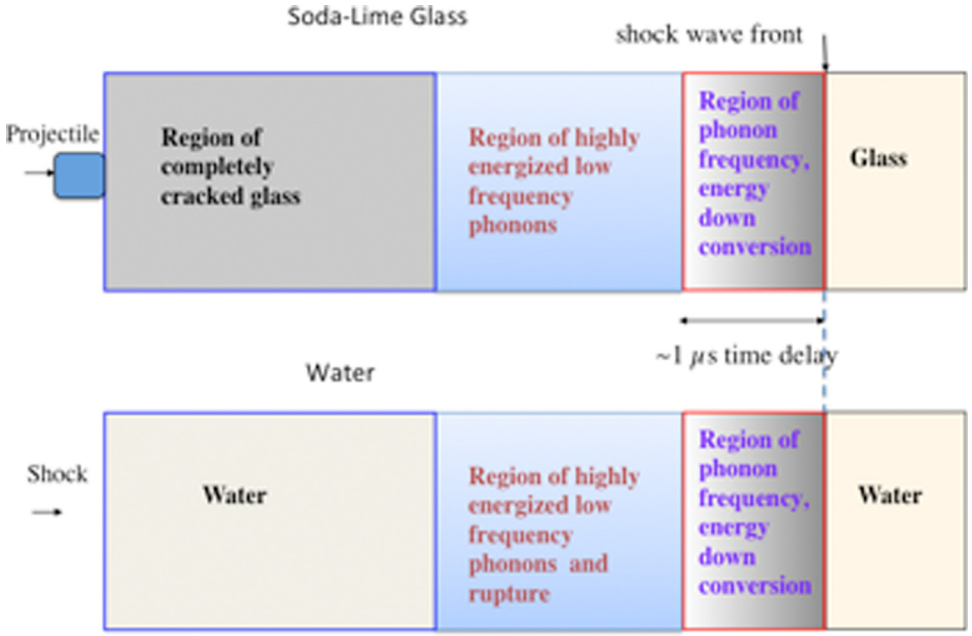

FIG. 3. Time snap shot of characteristic regions behind the propagating shock front in glass (top) and in water (bottom).

100% 9 days after exposure. While the human skull differs in thickness from the murine models thus affecting shock wave transmission, the values for brain tissue itself are likely similar for transmitted shock waves.

Mathematical models of the physics water dependent phonon interactions involved in non-impact blast induced mTBI could be verified in experimental models. One is to use light diffraction to catch the water in the ruptured state. In Figure 3, there will be a spatial region where the acoustic wave is of finite extent and that will form a momentary diffraction grating. By using a blue-green laser (for light transmission through water), appropriate diffraction angle, and pulse widths of nanoseconds gated to the shock time, it may be possible to observe short-lived water diffraction.

Another might employ water based biofidelic models in iterations more closely approximating brain tissue itself. Experimental models would be composed of an outer layer mimicking the diploic structure of the human skull particularly in its vulnerable frontal-orbital areas with an inner content of water linked gel-like structures such as sodium salts of cross-linked polyacrylate.[26]

A simple, single cell, well understood microorganism in suitable bio environment subjected to shock might also be used. Subsequent monitoring to look for impaired function followed by SEM microanalysis to observe cell damage would support the model if found.

CONCLUSIONS

We propose a new mechanism of damage in brain tissue subjected to a shock wave based on excitation of phonons with consequent decomposition into lower frequency oscillations. When the intensity of these oscillations exceeds the tensile strength of water, *water ruptures* within microseconds of the passage of the shock wave through the brain with the period of the phonon wave. This model also provides a rationale for the existence of a threshold blast intensity that causes non-impact primary blast TBI. A similar phenomenon has recently been shown to be operative in failure waves affecting brittle materials.[16] The corresponding linear scale of these effects is quite small—200 nanometers such that the dimensions, pressure, and timing of these forces that cause cellular and subcellular damage are difficult to detect by conventional means. However, the resulting damage is sufficient to cause cytoskeletal proteolysis, release of biomarkers and possible chronic functional impairment.[5,9] Anatomic characterization of changes in response to shock wave passage likely requires electron microscopy to define resulting subcellular and membrane changes. Current findings using MRI diffusion tensor imaging demonstrate abnormalities due to reduced anisotropy of water diffusion in the cerebellar peduncles, cingulum bundles, and in the orbito frontal white matter.[6] In this scenario, damage manifests itself on a very local scale at excitation levels well below the threshold for macro strain levels of 10% estimated for brain tissue damage.[12] While conventional imaging fails to exhibit tissue damage after primary non-impact blast exposure, cellular and subcellular effects appear to be sufficient to cause the clinical picture of mTBI/concussion. The phonon concept may be tested experimentally in short-pulse light diffraction and biofidelic surrogate models.

Forces ranging in magnitude of 10-100 kPa may enter brain tissue through the fronto-orbital area of skull directly exposed to blast, the spinal column in the neck and the brain stem from blast directed at the unprotected neck, and pressure waves reflected and magnified under the combat helmet.[27,28] Transmission of pressure through the neck vessels from the thoracic and body may also occur.[7] Shock effects due to phonon pumping differ greatly in the time scale of how the brain is damaged with respect to acceleration or direct impact. The phonon pumping mechanism described in this model takes place in ~microseconds, whereas acceleration from blunt impact or blast occurs over several milliseconds. Therefore, by the time the head begins to accelerate, sub cellular brain tissue damage is likely to have already occurred. In addition to accounting for blast effects which cause the clinical picture

of non impact mTBI/concussion, the "invisible injury," this model of injury from shock waves may be useful to develop methods to detect, prevent by selective shielding, and possibly attenuate the effects of non impact blast TBI.

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