# Analysis of Blast Induced Intracranial Pressure Dynamics in Cerebrospinal Fluid Leading to Traumatic Brain Injury

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**ABSTRACT**

Neurologists while treating soldiers who had survived explosions in the warfront came across a medical paradox. Evidences of memory deficits, speech problems and difficulties with decision-making were seen in these patients. The paradox was that most of these patients had not suffered a direct head injury even though their scan reports suggested enlargement of brain ventricles with minor bleeding in certain cases. Such medical conditions are called Blast Induced Traumatic Brain Injury (TBI). There have been studies in the past showing the development of stress in various parts of the brain due to explosions, though none of them directly correlate the expansion of Cerebrospinal fluid (CSF) pathways and brain ventricles, believed to be the indication of TBI. Our work primarily explores the role of intracranial CSF pressure dynamics in the genesis of TBI, emphasizing the evolution of the unsteady pressure field within CSF pathways, ventricular and the subarachnoid regions of the brain. The pressure gradient between the ventricles and the porous parenchyma is found to be considerably high and occurs within few milliseconds of blast application, which is indicative of the ventricular expansion. Our work also qualitatively evaluates the CSF pressure instabilities at various instants of time.

## 1. INTRODUCTION

Neurologists during the Balkan war of the 1990s [1] observed that soldiers who had encountered blasts in the battlefield suffered from memory deficits, dizziness, speech problems and difficulties with decision-making although had no obvious brain injuries. The computed tomography and magnetic resonance imaging scans of these patients indicate signs of internal damages. To be specific, these include the enlargement of brain ventricles with evidences of minor bleeding in specific cases. Such kinds of trauma due to blast exposure as already mentioned in the abstract are referred to as TBI. There are various studies that show the development of stress within the brain structures due to a nearby explosion, which may be a possible indication of TBI. However, this area of research needs a lot more explorations to enrich our understandings correlating the interactions of the blast wave and brain tissues with the enlargement of ventricles by excitation of CSF.

The annual victims of TBIs in the United States have been estimated to be 1.4 million [2, 3] which include military personnel and civilians. There has been a dramatic increase in these numbers in the recent past which can be attributed to the tactics of asymmetric warfare. Statistics from the conflict in Iraq show that several thousand United States soldiers have sustained TBI, 69% as a result of blasts [4, 5]. The injuries from blast exposure have been categorized into three major components: primary, secondary, and tertiary [6]. Primary blast injury is associated with the direct exposure of the organs to the blast wave, typically lungs, ears and gastrointestinal track. In secondary blast injury, flying debris due to blast cause impact in the individual, while in tertiary injury the victim is thrown into stationary objects by the blast. Both the secondary and tertiary injuries are due to mechanical trauma that has been the subject of decades of research. However,

the role of primary blast exposure in the development of TBI remains less understood [7]. Primary blast may induce linear and rotational cranial accelerations, but the sharp wave front of blast overpressure will also trigger complex pressure wave motions, transmitted by the skull into the high water content subarchnoid spaces and brain ventricles.

There are various consequences of TBI including easily visualized damage and intra-parenchymal or extra-axial hemorrhages. It's seen that one-third of the deaths of TBI are the result of traumatic axonal injury occurring at the microscopic level [8] that is difficult to be clinically visualized. This pathological condition is referred to as diffuse axonal injury or DAI and defines a multifocal process that can be widespread throughout the white matter tracks and other areas of the brain.

The CSF surrounds the brain as well as the spinal cord [9]. It suspends the brain through its buoyant force and acts as a damper by protecting the brain from impacts on the cranial vault wall. It is produced in the choroid plexuses of the brain and is drained mainly through the superior sagittal sinus. This steady flow of the CSF is influenced by a periodic pulse which is governed by the cardiac cycle. The CSF is contained in the ventricles and the subarchnoid space within the brain. The ventricles are four cavities which are interconnected by pathways. Figure 1 illustrates the anatomy of brain.

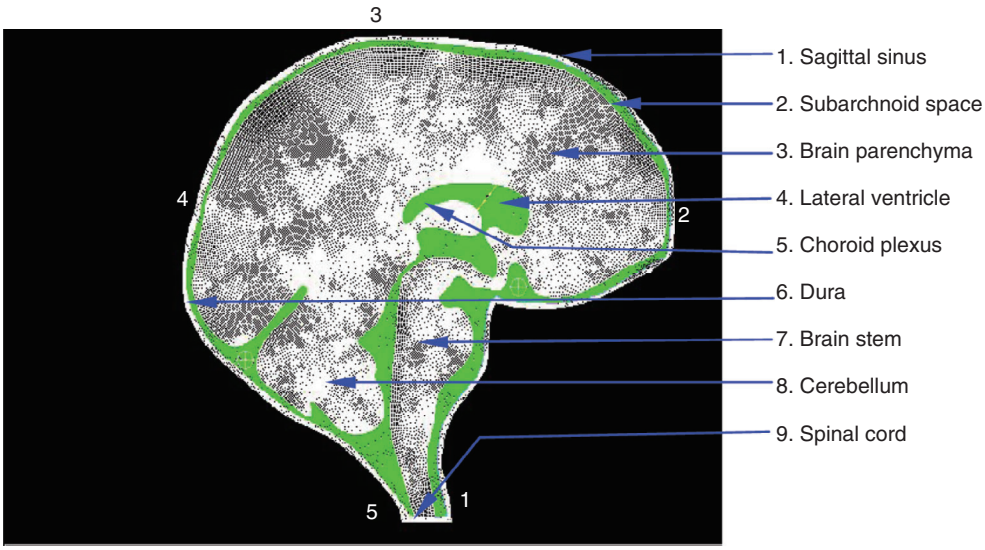



Figure 1. Computational domain, meshed geometry and boundaries.

Recent studies on the subject modeled the brain as a viscoelastic media simulating [2] intracranial wave physics. In this, the reason for TBI was indicated to be the stretching of the axonal fibers (DAI) caused by shear forces over a short span of time (2 ms). In another similar study it was stated that a significant translational and rotational acceleration over a span of 5-20 ms was the cause [10]. However none of the above studies directly correlates the reason behind the expansion of CSF pathways and brain ventricles, which is believed to be the indication of TBI according to Ref. 1. No work has been done so far to illustrate how exactly the pressure wave propagates in CSF, its paramount function being its action as a cushion against shocks and hence the protection of the brain from trauma. An analysis is hence extremely desired illustrating the pressure dynamics of CSF under influence of shock wave that may suggest the reason for expansion of the ventricles and subarchnoid space because of pressure augmentation within the CSF pathways, which in a way correlates directly the pathological findings in TBI.

Hence the primary focus of our work is to explore the role of the intracranial CSF pressure dynamics in the genesis of traumatic brain injury from blast. Here, emphasis is to evaluate qualitatively the unsteady pressure field within CSF pathways, ventricular and the subarchnoid regions with the brain parenchyma, which may draw an insight on this complex intracranial phenomena and the cause for the sudden ventricular expansion due to the advent of the blast wave.

## 2. METHOD

We have used the physics of fluid flow to quantify the intracranial CSF dynamics. The fundamental conservation laws of mass and momentum i.e. the continuity and Navier-Stokes equations have been used to solve the CFS flow under the influence of blast. The governing equations with appropriate boundary conditions in the present case are solved by a commercial computational fluid dynamics (CFD) solver Fluent 6.3 to visualize flow physics within the CSF pathways. The solution methodology invoked here uses the unsteady SIMPLE algorithm on unstructured grid, which is second order accurate in space and time. The anatomy of the brain including CSF spaces was extracted from digitalization of a human brain MRI [11] to define the computational domain and boundaries. Our work is limited to two-dimensional modeling. A very careful mesh generation has been done in Gambit of Fluent taking into account the intricacy of CSF flow path, ventricles and other parts of the brain anatomy. A total of 67458 nodes with 57527 mixed quadrilateral and triangular cells and 27880 triangular cells were used (Fig. 1 for details). Different assumptions have been made to replicate CSF fluid and the brain structure that have been discussed later.

### 2.1. Governing equations for CSF flow

CSF motion in the ventricles and the subarchnoid space can be described by the mass and momentum equations for an incompressible Newtonian fluid [13] as,

$$\text{Conservation of mass: } \vec{\nabla} \cdot \vec{V} = 0 \tag{1}$$

Conservation of momentum:

$$\rho\left(\frac{\partial \vec{V}}{\partial t} + \vec{V} \cdot \vec{\nabla}\vec{V}\right) = -\vec{\nabla}p + \mu\vec{\nabla}^2\vec{V} \tag{2}$$

where, $\vec{V}$ is the velocity vector, $\rho$ is the density of CSF and $\mu$ is its viscosity. The properties of CSF are close to water and are tabulated in Table I.

Table I. Properties used for CSF fluid and brain tissue.

| **Material properties** | |
|---|---|
| 1. Porosity of tissue and sagittal sinus ($\varphi$): | 0.3 |
| 2. Permeability of the tissue ($\kappa$): | $7 \times 10^{-16}$ m$^2$ |
| 3. Source term at choroid plexus: | $2.2 \times 10^{-10}$ kg/m$^3$sec |
| 4. Density of CSF ($\rho$): | 998.2 kg/m$^3$ |
| 5. Viscosity of CSF ($\mu$): | 0.0001003 kg/m-s |
| 6. Inertia resistance of tissue ($\beta$): | $10^4$ m$^{-1}$ |

### 2.2. CSF flow through the brain tissues

In this paper, the brain tissues (parenchyma, brain stem and cerebellum) have been treated as homogeneous isotropic porous media. An advanced representation of the brain parenchyma could have been made by considering the white matter to be viscoelastic with its directional dependence on the permeability, porosity and diffusivity. This would aid us to incorporate flexural changes in the skull and hence the brain tissue due to sudden advent of blast wave. However, such detailed representation of the brain tissue has not been considered for sake of simplicity. The CSF is primarily produced in the choroid plexus from the blood and also from the brain tissue from which it seeps through the porous extracellular space towards the ventricles. Two thirds of the CSF production enters the ventricles from the choroid arteries [14, 15] and the rest is generated diffusely throughout the brain parenchyma. Due to modeling complexity we have neglected the generation of CSF in brain parenchyma and considered only the production in choroid plexus. The continuity equation of CSF flow in the choroid plexus has a source term ($S$) to account for new CSF production as in Eq. 3.

The CSF flow though the extracellular space of the brain parenchyma is modeled by the continuity and momentum equations for flow though porous media. The momentum balance of CSF seepage is the Navier-Stokes equation augmented by an additional term quantifying frictional interaction of CSF with the brain tissue. Equation (4), a generalization of the simpler Darcy's law of flow through porous media [16, 17] is used here. The material properties of the brain tissues are listed in Table I.

Thus, CSF generation in the choroid plexus:

$$\vec{\nabla} \cdot \vec{V} = S \tag{3}$$

CSF fluid seepage through the porous medium:

$$\rho\left(\frac{\partial \vec{V}}{\partial t} + \vec{V} \cdot \vec{\nabla}\vec{V}\right) = -\vec{\nabla}p + \mu\vec{\nabla}^2\vec{V} - \left(\frac{\mu}{k}\vec{V} + \frac{\beta\rho}{2}\vec{V}^2\right) \tag{4}$$

For realistic prediction, proper boundary conditions are imposed with suitable assumptions. The pressure wave generated by an explosive blast needs to be formulated keeping in mind that the wave formed is a function of time and distance from the blast location apart from the mass of the detonation.

### 2.3. Formulation of the blast wave

The formulation of the blast wave follows from Ref. 12. A mathematical description of the pressure waveforms that represent the ranges and sizes of explosions associated with TBI can be accomplished by taking into account a reference explosion and distance from the explosion. The scaling equations are a system of empirically developed equations, accounting for accuracy within specific ranges from the blast, as well as the type of blast (nuclear or conventional) and are described in G.F. Kinney and K.J. Graham's book, *Explosive Shocks in Air*. The equations that are critical to this research are summarized.

The first step in determining the appropriate waveform is to determine a scaled distance ($Z$) that relates the modeled (actual) distance to a reference explosion:

$$Z = \frac{f_d \times (\text{actual distance})}{W^{1/3}} \tag{5}$$

In this calculation, $W$ is the explosive yield in kilograms of charge of the modeled explosive. The parameter, $f_d$, is considered the transmission factor. Most explosions leading to TBI occur in near proximity to the victim, and therefore a transmission factor of $f_d = 1$ is assumed for this study.

The maximum overpressure $p_0$, against the atmospheric pressure $pa$, can then be calculated by:

$$p_o = p_a \times \frac{808\left[1+\left(\frac{Z}{4.5}\right)^2\right]}{\sqrt{1+\left(\frac{Z}{0.0048}\right)^2}\sqrt{1+\left(\frac{Z}{0.32}\right)^2}\sqrt{1+\left(\frac{Z}{1.35}\right)^2}} \tag{6}$$

The two critical components of the pressure wave profile that describe its potential to cause injury to humans or damage to structures are the maximum overpressure and the duration of the positive phase. Together, these can be used to estimate the energy impulse associated with the blast. The impulse is defined as the integral of the pressure-time curve (Fig. 2). The duration of the positive phase ($t_d$) of the pressure profile is estimated by:

$$t_d = W^{1/3} \times \frac{980\left[1+\left(\frac{Z}{0.54}\right)^{10}\right]}{\left[1+\left(\frac{Z}{0.02}\right)^3\right]\left[1+\left(\frac{Z}{0.74}\right)^6\right]\sqrt{1+\left(\frac{Z}{6.9}\right)^2}} \tag{7}$$

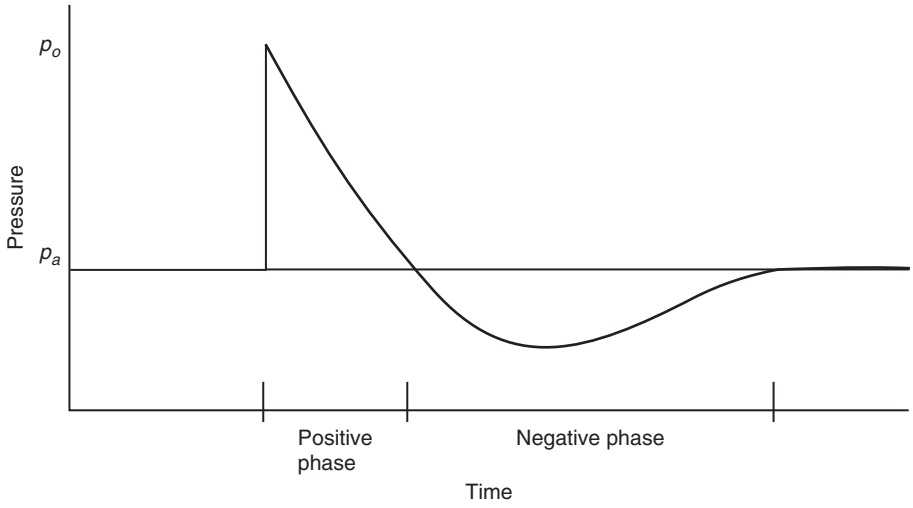



Figure 2. Typical pressure profile for an explosive blast in air.

Thus, the actual waveform is approximated by the Friedlander equation [12] as:

$$p = p_o\left(1 - \frac{t}{t_d}\right)e^{-\alpha\frac{t}{t_d}} \tag{8}$$

A typical pressure profile of a wave generated by an explosive blast following Eqs. 5-8 is illustrated in Fig. 2. It illustrates an initial jump discontinuity from ambient pressure to a maximum overpressure $p_0$. This is followed by a rapid decay of the positive overpressure back to the ambient level within a few milliseconds. The inertia of motion of expanded and displaced air results in a negative pressure phase that lasts approximately three times as long as the positive phase, before returning to ambient pressure. Further pressure oscillations are possible, but are not typically observed for blasts in air.

A blast intensity of around $10^6$ Pa assuming 20% reduction in the skull [2] was imposed on the anterior side of the modeled geometry and the blast conditions were equivalent to those predicted for a location 2 to 3 m distant from a detonated explosive device constructed from a 3 kg charge according to the above equations.

### 2.4. Boundary conditions

The boundary conditions for the CSF flow in the cranium are summarized in Table II. The bulk CSF production is implemented as a source term at the choroid plexus. The pulsatile flow of CSF due to the heart pulse during systole is also accounted for and expressed as

$$v(t) = \alpha\left[\sin\left(\omega t - \frac{\pi}{2}\right) - \frac{1}{2}\cos\left(\omega t - \frac{\pi}{2}\right)\right] \tag{9}$$

Table II. Imposed boundary conditions (b.c).

| Locations | Information | Marked in Fig. 1 |
|---|---|---|
| Inlet pressure b.c | Induced blast pressure as per the Eq. 1-4. | Boundary 1-2-3 |
| Exit pressure b.c | Atmospheric pressure | Boundary 3-4-5 |
| Spinal cord b.c | Velocity induced by heart impulse as stated in Eq. 9. | Boundary 5-1 |
| Dura b.c | No slip, $u = v = w = 0$ | 6 |
| Choroid plexus b.c | CSF generation, imposed a source term | 5 |
| Sagittal sinus b.c | Distributed CSF sink | 1 |
| Porous Structures | Brain Parenchyma, Cerebellum, Brain Stem, Sagittal Sinus | 1, 3, 7, 8 |
| Fluid flow | CSF pathways | 2, 4 |

The frequency ($\omega$) of the pulsatile motion is set to 1 Hz approximating the normal cardiac cycle. Most scientists believe that the majority of reabsorption of the CSF occurs in the granulation of the sagittal sinuses. Accordingly, reabsorption of the fluid takes place at the top of the brain geometry. It is seen that in a hydrocephalus patient, a ventricular dilation due to increase in CSF pressure occurs indicating that the pressure increase in the subarchnoid space is not communicated to the spinal chord. This allows us to use a closed boundary condition with a pulsatile motion from spinal chord, which is actually the cause for CSF motion. Since we have no idea of what might be the velocities of the CSF after the blast impact, we use a pressure inlet boundary condition as a blast impulse with a certain reduction in blast intensity as we have no dynamic model to judge the skull deformation and the pressure outlet at the normal CSF pressure.

## 3. RESULTS AND DISCUSSION

A study is made to analyze the outcomes of CSF pressures inside the subarchnoid region and ventricular spaces and hence to indicate the cause behind their enlargement in TBI through CFD simulation. Here, the geometry is restricted to a 2D brain section as obtained in a human brain MRI. The meshed 2D geometry was imported to Fluent version 6.3 and a blast intensity of around $10^6$ Pa was imposed on the anterior side as stated. The unsteady pressure dynamics within CFS pathways after the onset of a blast are presented in Fig. 3 (a–h). These illustrate the cascading of events within a few milliseconds (ms), which are much thought provoking and indeed explain the pressure propagation through CSF fluid. The details are explained as follows.

Fig. 3a shows the peak rise of pressure in CSF in the frontal region of the brain due to the onset of blast wave (near the inlet pressure boundary) after a time interval of 0.5 milliseconds (ms). This abnormal increase in pressure would definitely perturb the flow of CSF and a sudden drop of CSF pressure is observed in the ventricular region. At about $t$ = 2 ms, there is not much change in the distribution of pressure gradient, however the magnitude of intense pressure in the frontal region is reduced and the pressure in the ventricular region is increased indicating that the pressure is being gradually transmitted to the ventricular spaces, Fig. 3b. It should be noted that the absolute pressure does not appear in the equations of motion of an incompressible fluid, but the pressure gradient $\nabla p$ appears. Hence, the prediction of absolute CSF pressure by CFD is not possible. The pressure of CSF in this paper means the pressure gradient.

At $t$ = 3 ms, a considerable change is observed in the pressure variation with the augmentation of pressure around the cerebellum depicting reflection of pressure wave from the boundary of the constricted passage between the brain and spinal chord, Fig. 3c. The pressure wave propagates along the subarchnoid spaces towards the rear part of the brain, where the magnitude is about $10^4$ Pa and the pressure diminution begins in the frontal region. At this instant, the pressure in the ventricles is also increased and a large pressure gradient of about $10^3$ Pa exists between the ventricles and the porous parenchyma. This pressure difference is much greater than what was observed by Hoff and Barber [18] during their studies with four patients suffering from pressure hydrocephalus, where they found that the mean ventricular pressure was 260 Pa higher than the mean pressure in the subdural space. This momentary rise in intense pressure may cause significant damage and deformations of the ventricular spaces.

At $t$ = 4 ms, the pressure is high in the ventricular region and also in the porous parenchyma as compared to the subarchnoid spaces, Fig. 3d. The pressure gradient between the ventricle and the parenchyma as well as the subarchnoid spaces may cause a potential damage to the axons due to stretching. Here, the dark blue region in the frontal part of the brain indicates the advent of negative phase of the blast wave causing the pressure in CSF to reach a low value. The trend continues up to $t$ = 6.5 ms, Fig. 3e. At $t$ = 20 ms, the pressure is now travelling back from ventricular space towards the frontal region along the CSF pathways similar to a reflected wave motion, Fig. 3f. This unsteady phenomenon, indicating augmentation of pressure as a function of space and time appears very serious causing distortion in different parts of the brain. The pressure slowly eases to normal CSF pressure as time progresses, although even at $t$ = 30 ms, Fig. 3g, a pressure accumulation occurs behind the cerebellum. By 38 ms the pressure in the CSF pathways and other parts of the brain is almost stabilized, Fig. 3h.

The evolution of pressure history at certain points (marked P1, P2, P3 and P4) as shown in Fig. 4 is quite consequential. The pressure variation at P1 is similar to the blast wave since the point lies near the inlet boundary. At P2, which is behind the cerebellum, there is a sudden increase in pressure followed by a sharp drop within 1 ms and again the pressure increases to about $2 \times 10^5$ Pa 2 bars that decays as time progresses. It is interesting to note that the pressure at the rear part of the brain is

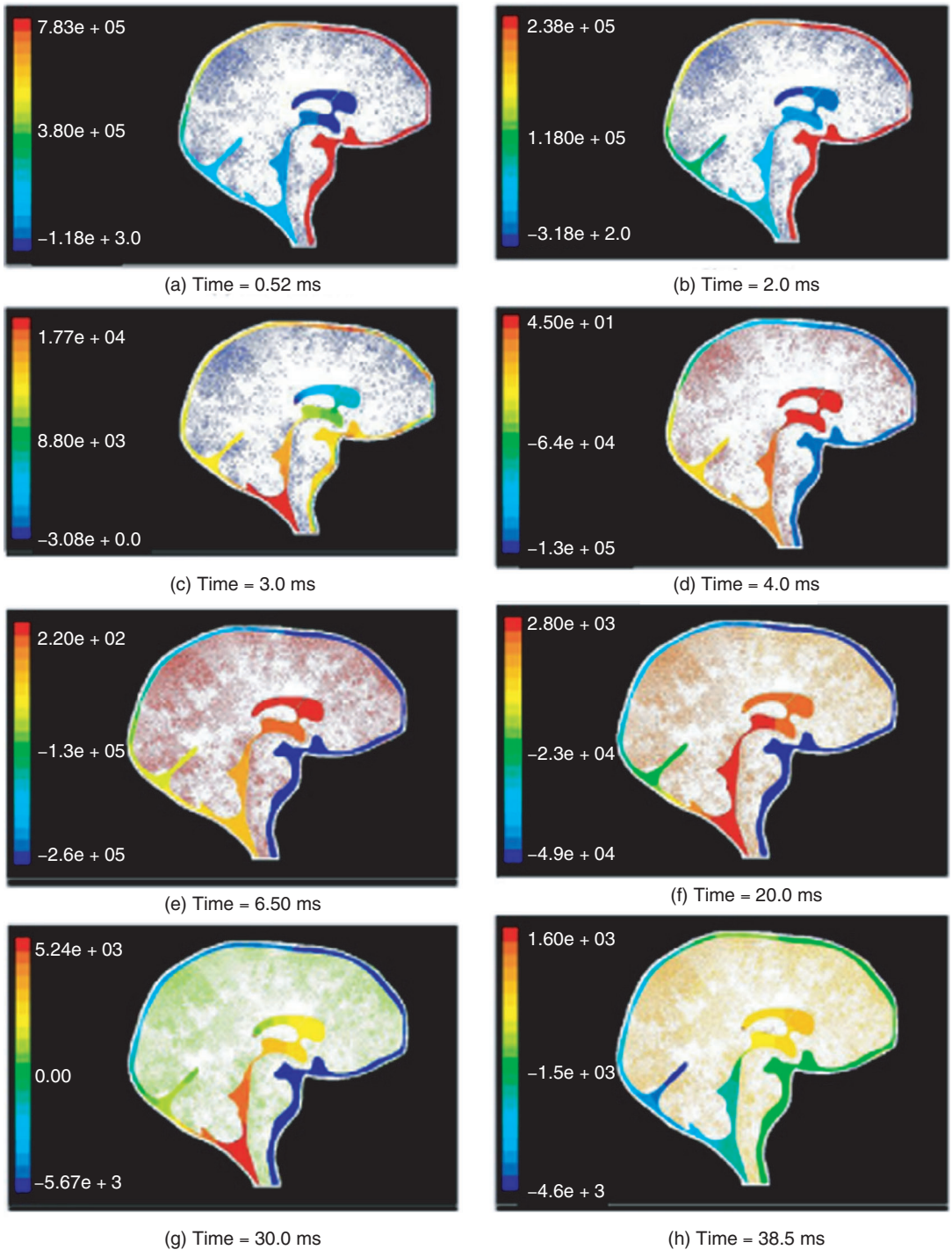



Figure 3. Pressure contours at different instant of time depicting unsteady pressure dynamics within CSF pathways due to exposure to a blast wave.

substantially high for a considerable time from 2.5 ms to 12 ms. This high pressure acting for a relatively longer time has a potential to cause serious material acceleration and associated injury, which has not been shown earlier.

Pressure excitation at P3 and P4, the ventricular space and the CSF passage below respectively, follow the trend of the blast wave but the interesting part is that there exists high pressure fluctuations during the time $t$ = 10 ms to 25 ms. The amplitude of oscillations in few instances shows very high values. A second phase of oscillations is seen at around 38 ms, although its amplitude is low compared

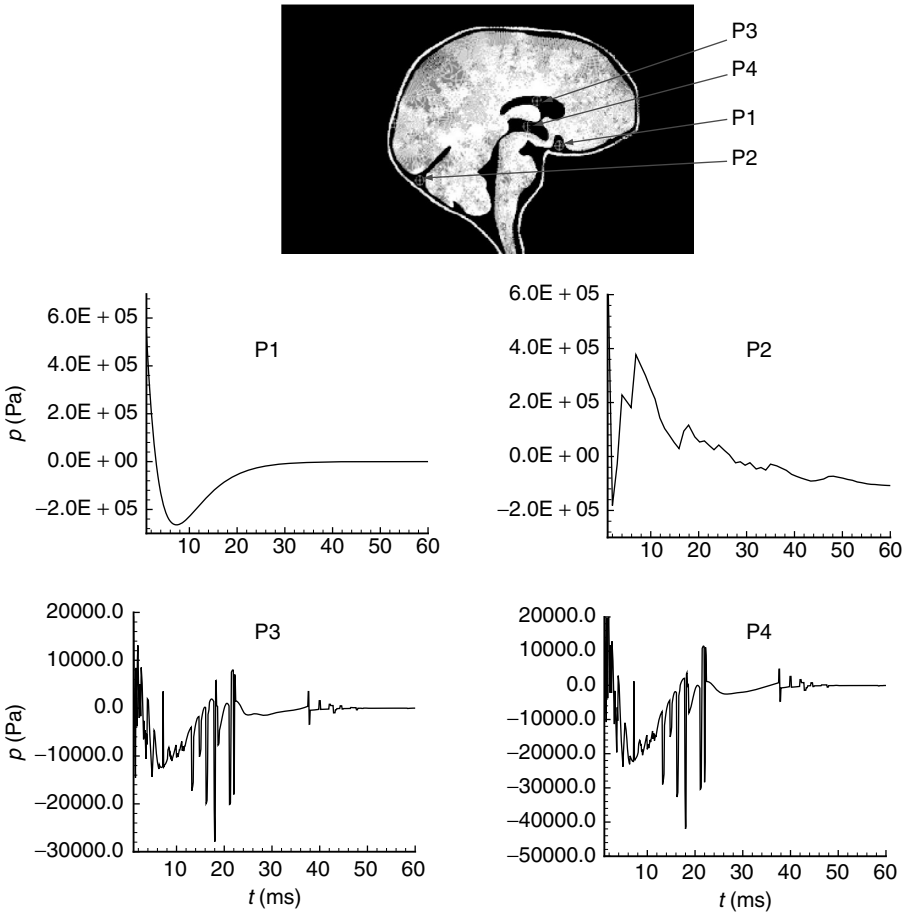
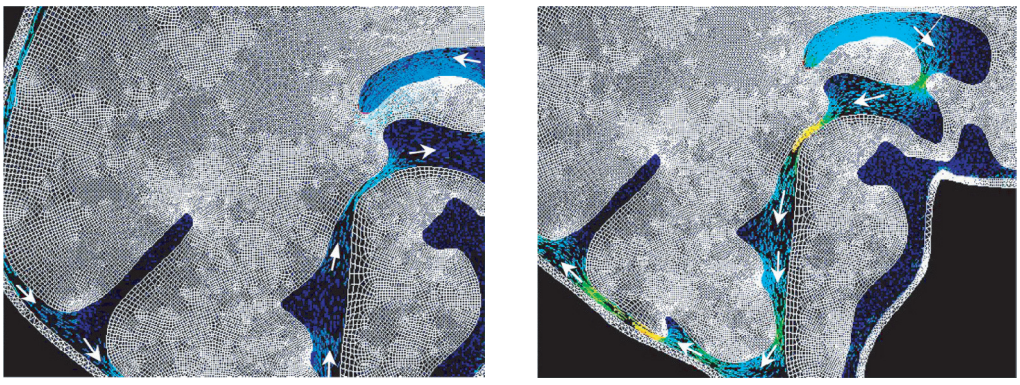



Figure 4. Variation of pressure from different points as marked in the CSF pathways.

to the previous ones. These oscillations could be associated with flow reversals and perturbations of the CSF flow. Furthermore the ventricular space is exposed to a large negative pressure for a long time $t$ = 4 ms to 12 ms (except for few instances). This figure also illustrates that although there are significant changes in pressure fields and hence induced stresses within a short interval of time of 6 ms after the advent of blast, yet there can be excitations that may appear over longer spans of time extending up to 30 ms.

The velocity fields of CSF are illustrated by velocity contours and magnified velocity vectors at instants of 3.5 ms and 95 ms after the onset of blast (Fig. 5). At 3.5 ms the flow is directed towards the



(a) Time = 3.5 ms (b) Time = 95 ms

Figure 5. Velocity vectors at two time intervals.

ventricles, which is attributed to the negative pressure in the region and this flow direction is opposite to the normal CSF flow. After a considerable long interval of time, almost normal fluid movements inside the subarchnoid and ventricular region are established, although the magnitudes in some portion are higher than the normal conditions.

The present study explains that the pressure wave can propagate through the CSF and may develop an intense pressure in CSF pathways, while the previous investigations did not consider CSF separately rather they modeled the whole brain as viscoelastic media [2] and showed how it responded under a shock wave. Thus, there is indeed a need to model both the CSF and the brain parenchyma separately and we believe that there exists a nonlinear interaction between the brain tissue and CSF which needs to be resolved.

## 4. CONCLUSIONS

Analysis through CFD has been made to indicate the pressure dynamics in CSF that may lead to blast induced TBI. Previous studies made in this direction concentrated on transmission of pressure pulse in the brain considering the parenchyma region as a viscoelastic medium, neglecting the role of CSF and did not discuss the reason behind the expansion of CSF space and brain ventricles. Our main analysis is centered on CSF examining its role in blast induced cases.

The momentum equations of fluid have been solved with realistic boundary conditions for the blast wave as well as the heart pulse and have been applied to the 2D brain geometry with necessary material properties of the brain. It has been observed that an unsteady pressure response is developed in the CSF pathways. The pressure gradient between the ventricles and the porous parenchyma is considerably high that indicates the reason of ventricular expansion. Instantaneous pressure also becomes high in the subarchnoid and backward region of the brain. A pressure oscillation is seen in the ventricular region that is noteworthy. The intense activity of this varying pressure gradient occurs within 6 ms of the blast application however it is noted that even after 30 ms the residual pressure in some portion of the CSF pathways is considerable, which then gradually reduces to normal CSF pressure as time progresses.

In our opinion, this work provides a qualitative evaluation of CSF pressure that may be related to the cause of expansion of CSF pathways and ventricular spaces as stated in Ref. 1 while observing patients suffering from TBI through CT scans. However, the present model can be improved by considering the grey matter to be a viscoelastic medium.

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