

Numerical simulation of rotational flow in hydrocephalic hydro-elastic models

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Abstract

Three-dimensional computational models of the cerebrospinal fluid (CSF) flow and brain tissue are presented for evaluation of their hydrodynamic conditions before and after shunting for seven patients with non-communicating hydrocephalus. We have developed a mathematical model of the fluid flow in cerebral ventricles during hydrocephalus is presented. The fluid-solid interaction simulation shows the CSF mean pressure is five times greater than normal subject. We present a brief overview of the clinical problems that are being addressed. This suggests that functional deficits observed in hydrocephalic patients could therefore be more related to the damage to periventricular white matter. we believe more accurately represent the anatomy of the brain and including time dependence.

Introduction

Many researchers have constructed computational models of hydrocephalus based on poroelasticity theory. Such models, it is thought, would provide greater comprehension of the problem and, as a result, better therapy. These existing poro elastic models only include hydrocephalus in its final sick form, not the transition from a healthy to a pathological state of the brain while the aqueduct is still open. Such models have also neglected to account for the intermittent effects of shunting, the most often utilized treatment for hydrocephalus. We use elasticity and fluid mechanics to create a mathematical model of the human brain and ventricular system. The model is used to research hydrocephalus, a pathological condition in which the normal flow of cerebrospinal fluid is disrupted, causing the brain to distort. Our model expands previous work in this area by considering flow across the aqueduct and including boundary constraints. This allows us to create a quantitative model of the start, improvement, and therapy of this disorder. We develop and solve the governing equations and boundary conditions for this model along with meaningful clinical findings.

CSF runs from production sites in the choroid plexuses of the lateral and third ventricles down a single thin cerebral aqueduct and into a fourth ventricle in a healthy brain. Our model expands earlier research on hydrocephalus by incorporating aqueduct flow with boundary constraints. A small quantity of fluid travels down the subarachnoid space around the spinal cord and then back up into the cranial subarachnoid space, however, the laws of physics make it difficult to explain how this flow could possibly be endured.

The dynamic interaction of pulsatile blood, brain, and CSF was studied using a mathematical framework that was based on in vivo inputs¹. The simulation proposed in this paper is obtained

for CSF physio-pathological disease hydrocephalus patients². An analysis of the lateral ventricular elasticity for congenital hydrocephalus in an asymmetric flow with species concentration³. The present study while using a simple geometric model provides a new direction in multi-physical transport phenomena in the hydrocephalus and furthermore a benchmark for more geometrically complex simulations⁴. The equations of motion for CSF flow in the ventricular and subarachnoidal pathways as well as CSF seepage inside the porous brain parenchyma were solved. The boundary conditions for the complex brain geometry were formulated⁵.

The experimental data was obtained from a normal subject and compared with a three-dimensional computational model of intracranial dynamics. Developed from subject-specific MR images, and using physiological boundary conditions as input, the model reproduces pulsatile CSF motion and predicts intracranial pressures and flow rates⁶. The numerical model was employed to investigate the effects of cross-sectional geometry and spinal cord motion on unsteady velocity, shear stress, and pressure gradient fields⁷. The system was divided into five submodels representing arterial blood, venous blood, ventricular CSF, cranial subarachnoid space, and spinal subarachnoid space. These submodels are connected by resistance and compliance. The model developed was used to reproduce certain functional characteristics observed in seven healthy volunteers, such as the distribution (amplitude and phase shift) of arterial, venous, and CSF flows⁸.

Time-resolved three-dimensional magnetic resonance velocity mapping has previously been used to investigate normal and pathologic blood flow patterns in the human vascular system. Here we used this technique to study the spatial and temporal dynamics of CSF flow in the ventricular system of 40 normal volunteers⁹.

CSF is absorbed in the arachnoid villi, which are small granulations located inside the arachnoid that provide into the dura mater. The barrier among CSF and blood in these granulations is minimal, allowing CSF to flow into the circulation and be absorbed. In contrast to CSF production, consumption is pressure-dependent, with the rate influenced by the differential between intraventricular and superior sagittal venous pressure. In the present study, these effects and ICC diagrams have been investigated using 3D FSI simulation up to 2.5 years after shunt surgery in a large number of NCH patients through a non-invasive method¹⁰.

A finite element parametric investigation on a head model under different scenarios of impact is conducted. In the study, the CSF material parameters are varied within the expected range of change, while other components of the head model are kept constant¹¹. There is in the literature the descriptors, connecting and non-connecting hydrocephalus to distinguish cases where the aqueduct is open or blocked but since we are interested in the whole range of states of the aqueduct we shall not use these here. The most frequent cause of congenital and infantile hydrocephalus is a malformation in one or more parts of the ventricular system, for example, stenosis of the aqueduct or membranous occlusion of the foramen of Monro.

In this chapter, we build a model of the brain and ventricular system that is complicated enough to simulate the behavior of the hydrocephalic brain while residing simple sufficient for it to be mathematically accessible, and we use it to investigate the development and treatment of the disorder. The fluid dynamics of CSF circulation inside the skull system are oscillatory. The ventricular area equation of motion inside the porous medium with adequate pulsatile boundary conditions was simulated. The simulation proposed in this article is

obtained for patients with CSF physiopathological illness hydrocephalus. Using appropriate validity, we calculated the CSF flow velocity of patients.

Mathematical Formulation

The CSF is regarded as a Newtonian fluid in this investigation, with dynamic viscosity and density of 1.003×10^{-3} kg/ms and 998.2 kg/m³, respectively [1]. The brain tissue is classified as a linearly viscoelastic material, with storage and loss moduli of 2038 and 1356 Pa for healthy subjects, respectively, and 1594 and 1015 Pa for patients, and a density of 1040 kg/m³ [8, 9]. The lateral ventricles have a CSF flow rate of 0.35 cm³/min [1]. For numerical models, this number is employed as the value of the amplitude in the input fluids pulsatile flow rate function—the final segment of the ventricular system following the fourth ventricle is chosen as the flow output site. The usual baseline CSF pressure was established at 500 Pa, whereas the pathological baseline was set at 2700 Pa [6].

$$\frac{\partial u_c}{\partial x} + \frac{\partial w_c}{\partial y} = 0$$

$$\frac{\partial u_c}{\partial t} + Gw_0 \frac{\partial u_c}{\partial y} + 2\Omega' w_c = -\frac{1}{\rho} \frac{\partial p}{\partial x} + \nu \left(\frac{\partial^2 u_c}{\partial y^2} \right) - \frac{RN}{\rho} u_c$$

$$\frac{\partial u_c}{\partial t} + Gw_0 \frac{\partial w_c}{\partial y} - 2\Omega' u_c = -\frac{1}{\rho} \frac{\partial p}{\partial x} + \nu \left(\frac{\partial^2 w_c}{\partial y^2} \right) - \frac{RN}{\rho} w_c$$

$$u_c = \omega \left(\cos \omega t + \frac{1}{2} \sin \omega t \right), w = 0, \text{ at } y = 0 \text{ and } t \leq 0$$

$$u = u_0 \omega, w = 0 \text{ at } y = 1, t > 0$$

Let consider the pressure difference exponentially transverse with respect to angular velocity $\frac{\partial p}{\partial x} = \omega e^{-\omega t}$. We use pulsatile inlet boundary condition of [14] to specify the flow of CSF in the cranium layer. Non-dimensional quantities relevant to the problem and neglecting the dash symbol for our convenience

$$y' = \frac{y}{l}, t' = \frac{tw_0}{l}, w' = \frac{wl}{w_0}, u_c' = \frac{u_c}{u_0}, \omega' = \frac{\omega l^2}{\nu}$$

$$\theta' = \frac{T - T_c}{T_0 - T_c}, Re = \frac{w_0 l}{\nu}, G_{pm} = \frac{RNl^2}{\mu}, \Omega' = \frac{\Omega l^2}{\nu}$$

u and w refer the velocity of fluid in x and y direction respectively. w_0 is represented as characteristic velocity. Fluid density is referred as ρ . Da and Ω expressed as darcy number and

angular velocity, G_{pm} referred as particle mass parameter. ω represents Womersley number of CSF fluid.

The following were the above governing equations in dimensionless form

$$Re \left(\frac{\partial u_c}{\partial t} + G \frac{\partial u_c}{\partial y} \right) + 2\Omega w = -\alpha^2 e^{\varphi t} + \frac{\partial^2 u_c}{\partial y^2} - G_{pm} u_c \quad (1)$$

$$Re \left(\frac{\partial w_c}{\partial t} + G \frac{\partial w_c}{\partial y} \right) - 2\Omega w_c = -\alpha^2 e^{\varphi t} + \frac{\partial^2 w_c}{\partial y^2} - G_{pm} w_c \quad (2)$$

corresponding initial and boundary conditions are,

$$u_c = \alpha^2 \left(\cos \varphi t + \frac{1}{2} \sin \varphi t \right), w_c = 0 \text{ at } y = 0$$

$$u_c = \alpha^2 \cos n\varphi t, w = 0 \text{ at } y = 1. \text{ Were } n \text{ being an integer.}$$

u and w are functions of x and y , as we considered the fluid flows rotationally. To solve the above momentum equation, we introducing the complex velocity, $F_c = u + iw$

The momentum equation in an explicit form of complex function which changes the equation of motion as follows

$$Re \left(\frac{\partial F_c}{\partial t} + G \frac{\partial F_c}{\partial y} \right) + 2\Omega F_c = -2I\alpha^2 e^{\varphi t} + \frac{\partial^2 F_c}{\partial y^2} - G_{pm} F_c \quad (3)$$

We assume a suitable boundary condition $F_c = \alpha^2 \left(\cos \varphi t + \frac{1}{2} \sin \varphi t \right), y = 0, t \leq 0$

$$F_c = \alpha^2 \cos n\varphi t, y = 1, t > 1$$

METHOD OF SOLUTION

With the aid of a model problem, we employ analytical techniques to comprehend the biologically advantageous nature of the brain's mechanisms and their physical impacts. The effectiveness of the numerical method might also benefit from the analytical method. As a result, we reduce those equations to an ordinary differential equation and solve the equation analytically. We solve the above governing equation using perturbation technique as the method is quite error less in nature, assuming the trail solution for velocity.

$$F_c(y, t) = F_0(y) + \frac{\epsilon}{2} e^{i\lambda t} F_1(y) + \frac{\epsilon}{2} e^{-i\lambda t} F_2(y) \quad (4)$$

λ refers to oscillation frequency and ϵ being an arbitrary constant parameter defined in such a way that $\epsilon \ll 1$. Let us consider u_0, u_1, u_2 refers zero order, first order and second orders of momentum equation respectively. I, i represents the complex variable of the above equations of F and u respectively

The above equation is solved by the analytical perturbation method which results as

$$F_0 = \alpha^2 e^{m_1 y} - A_2 (e^{m_1 y} - e^{m_2 y}) + \frac{2\alpha^2}{a} (e^{m_1 y} - 1) \quad (5)$$

$$F_1 = \alpha^2 \left(\cos \varphi t + \frac{1}{2} \sin \varphi t \right) e^{m_3 y} - \frac{(e^{m_3 y} - e^{m_4 y})}{(e^{m_3} - e^{m_4})} \left[\alpha^2 \left(\cos \varphi t + \frac{1}{2} \sin \varphi t \right) e^{m_3} - \alpha^2 \cos \varphi t + \frac{2\alpha^2 e^{\varphi t}}{b} (e^{m_3} - 1) \right] + \frac{2\alpha^2}{a} (e^{m_1 y} - 1) \quad (6)$$

$$F_2 = \alpha^2 \left(\cos \varphi t + \frac{1}{2} \sin \varphi t \right) e^{m_5 y} - \frac{(e^{m_5 y} - e^{m_6 y})}{(e^{m_5} - e^{m_6})} \left[\alpha^2 \left(\cos \varphi t + \frac{1}{2} \sin \varphi t \right) e^{m_5} - \alpha^2 \cos \varphi t + \frac{2\alpha^2 e^{\varphi t}}{b} (e^{m_5} - 1) \right] + \frac{2\alpha^2}{a} (e^{m_1 y} - 1) \quad (7)$$

RESULT AND DISCUSSION

To explore the various physical parameters such as dynamic viscosity μ , density, kinematic viscosity, Particle mass parameter, Elasticity, Reynolds number, Womersley number have been determined analytically and the results were plotted as follows. The intracranial pressure of normal subject human being was found to be 500Pa and 3000Pa for hydrocephalus patients [14]. The size of the lateral ventricle in hydrocephalus is 27.2 times larger than normal CSF. Furthermore, third and fourth ventricle elastance increases by about 60% and 40%, respectively, compared to normal CSF flow. As a result, there is a significant statistical difference in the expansion of the ventricular for hydrocephalus. To employ the behaviour of CSF for hydrocephalic patients we use the following parameters Porosity (0.25), Pressure gradient (2700), Elasticity(350Pa), Reynolds Number (468.3), Womersley Number (more than 8.9) and Resistance parameter (1.367).The graphs shows Velocity profile(cm/s) in y axis and Time 't' along x axis.

Fig 1 depicts the variation of Reynolds number with time variation. The value of Re varies from 130, 240,420 has been depicted in figure for hydrocephalus patients. Here it is showed that the velocity increases as the when Reynolds number escalates with time taken for cardiac cycle.

When the flow fluctuates during the early systole of the cardiac cycle, the velocity of CSF flow reaches a peak. When the frequency rises with time, the flow rate of hydrocephalus decreases with time. Hence increase on Womersley number for pulsatile flow with 6.2, 7.5 and 8.9 with varying velocity acquires the maximum flow volumetric flow rate. Fig: 4.

In Fig 2 with various Resistance parameter 0.9, 1, 1.36 increases the in velocity of hydrocephalus attains the maximum level. Moreover, in Fig 3, the ventricular elasticity deforms as the fluid level increases the SAS. The porosity of brain parenchyma is estimates as 0.2 [4]. Hence the fluid amplitude increases as the permeability escalates proves the high-pressure CSF fluid flow.

The pressure of hydrocephalus is close to 3000Pa whereas for normal subjects is $\leq 500\text{Pa}$ [1]. We validate the increase in pressure graphically in Fig:5.

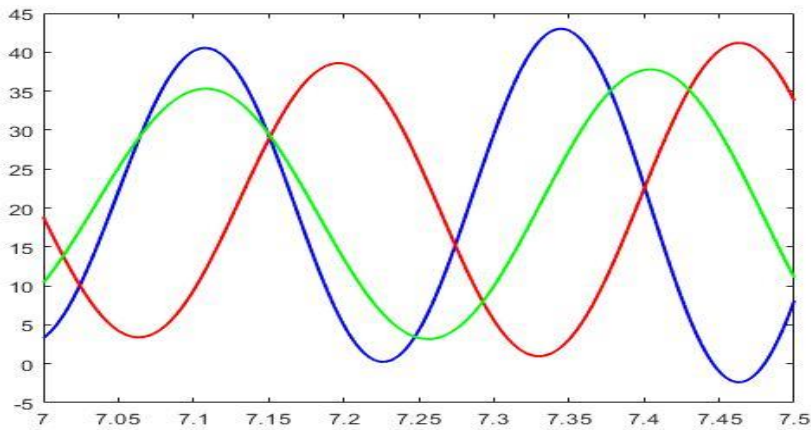


Fig: 1 Velocity profile with varying Reynolds number

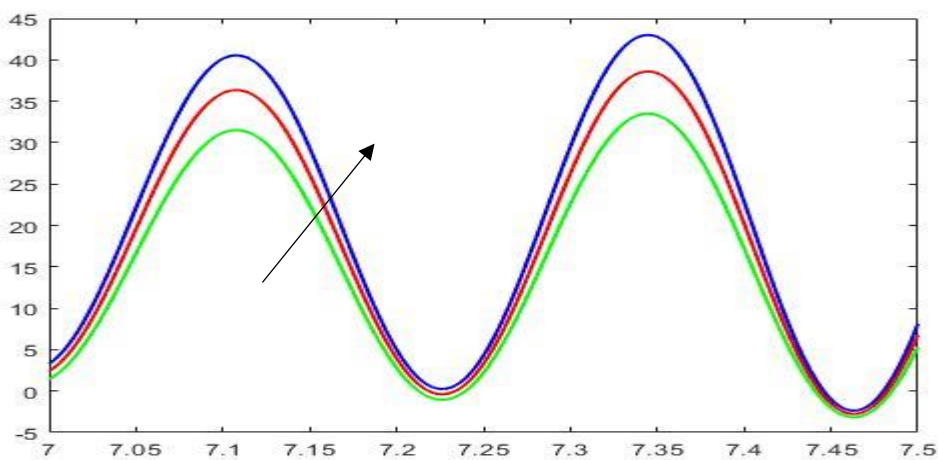


Fig: 2 Velocity profile with varying particle mass parameter

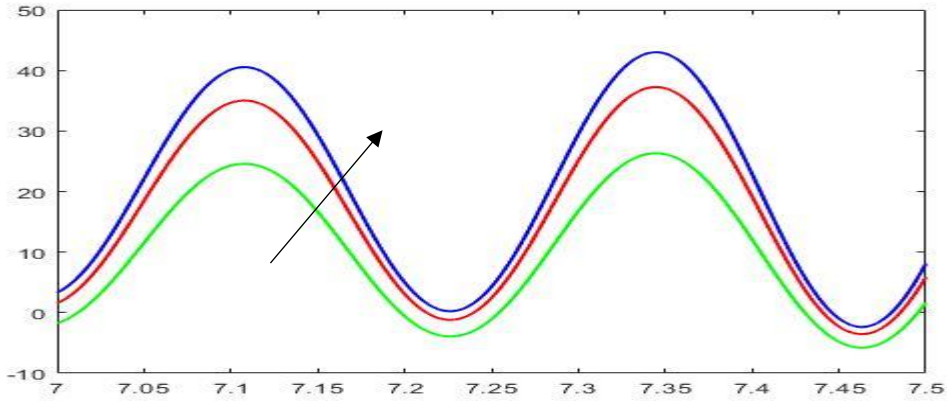


Fig: 3 Velocity profile with varying Elasticity

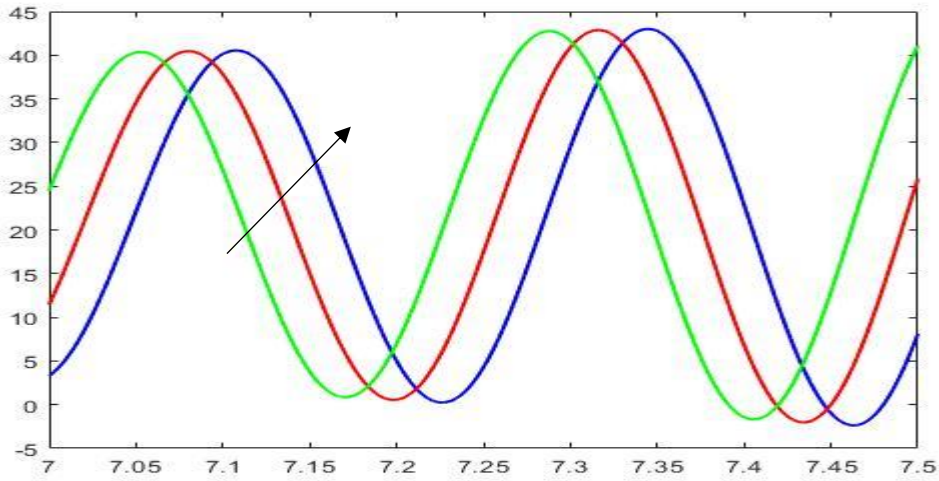


Fig: 4 Velocity profile with varying Womersley number

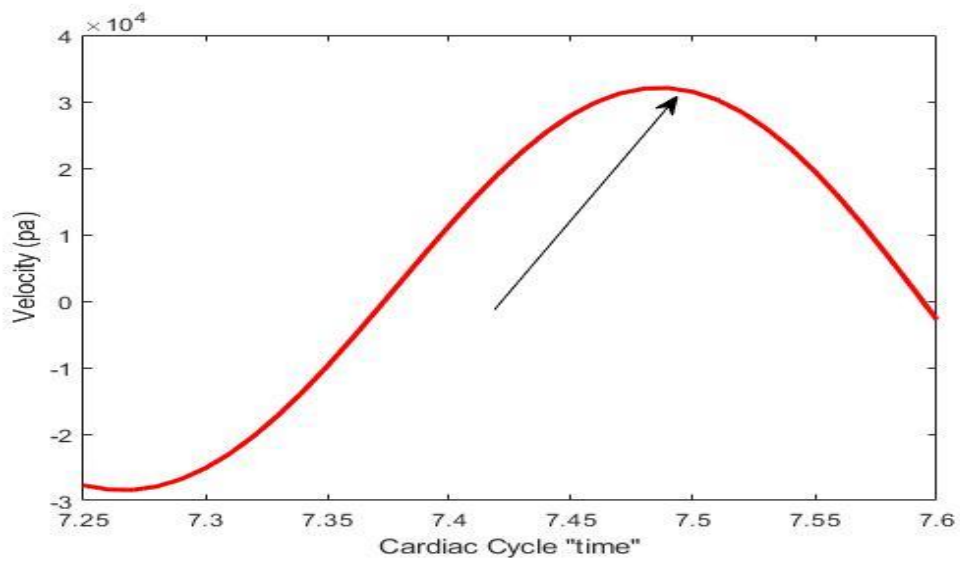


Fig: 5 Pressure of CSF from Velocity profile with varying with time 't'

CONCLUSION

In brain hydrocephalus, pressure difference varies significantly due to the influences of high CSF pulsatile velocity. This results in increase in ventricular enlargement of cranial system is clearly demonstrated elasticity graph. There agrees with the lesser compliance of ventricular when there is an increase in pressure. CSF circulation and production will affect the entire function of affected patients that leads to a brain damage.

The relevant parameters like Womersley number, elasticity, particle mass parameter, Darcy number predict the flow level in cardiac cycle. This study, in an idealized model of the hydrocephalus appropriate to neurological studies, has validated few interesting features of fluid behaviour in thermal transport. Following were the conclusions as identified from the present investigation.

The momentum of fluid flow enhances for increasing Darcy number, resistance parameter, Womersley number and Reynolds number. Increase in deformation (elasticity) with means of dimensional change results in increase in cerebrospinal fluid velocity.

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