Numerical Cerebrospinal System Modeling in Fluid-Structure Interaction

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Abstract

Objective Cerebrospinal fluid (CSF) stroke volume in the aqueduct is widely used to evaluate CSF dynamics disorders. In healthy population, aqueduct stroke volume represents around 10% of the spinal stroke volume while intracranial subarachnoid space stroke volume represents 90%. Amplitude of the CSF oscillations through the different compartments of the cerebrospinal system is function of the geometry and the compliances of each compartments but we suspect that it could also be impacted by the cardiac cycle frequency. To study this CSF distribution, we have developed a numerical model of the cerebrospinal system taking into account cerebral ventricles, intracranial subarachnoid spaces, spinal canal and brain tissue in fluid-structure interaction.

Materials and Methods A numerical fluid-structure interaction model is implemented using a finite element method library to model the cerebrospinal system and its interaction with the brain based on the fluid mechanics equations and the linear elasticity equations coupled in a monolithic formulation. The model geometry, simplified in a first approach, is designed in accordance to realistic volumes ratio of the different compartments: a thin tube is used to mimics the high flow resistance of the aqueduct. CSF velocity and pressure, and brain displacements are obtained as simulation results, whence CSF flow and stroke volume are calculated from this results.

Results Simulation results show significant variability of aqueduct stroke volume and intracranial subarachnoid spaces stroke volume in the physiological range of cardiac frequencies.

Conclusions Fluid-structure interactions are numerous in the cerebrospinal system and difficult to understand in the rigid skull. The presented model highlights significant variations of the stroke volumes under cardiac frequency variations only.

Keywords: Cerebrospinal fluid, Intracranial pressure, Stroke volume, Fluid-structure interaction, Finite element method

Introduction

Cerebrospinal fluid (CSF) stroke volume in the aqueduct (SV_{aq}) is widely used to evaluate CSF dynamics disorders. In healthy population, SV_{aq} represents around 10% of the spinal stroke volume (SV_{spi}) while intracranial subarachnoid spaces (SAS) stroke volume (SV_{sas}) represents around 90% of SV_{spi} [1]. Amplitude of the CSF oscillations through the different compartments of the cerebrospinal system is function of the geometry and the compliances of each compartments but we suspect that it could also be impacted by the cardiac frequency. To study the CSF distribution, a numerical model of the cerebrospinal system has been developed taking into account cerebral ventricles, intracranial subarachnoid spaces, spinal canal and brain tissue in fluid-structure interaction.

Previous numerical models of the cerebrospinal system have already been studied, using fluid equations only [2] or fluid and porous media equations [3] but they are not relevant in this study, so a new numerical framework is introduced. Model geometry and parameters are detailed and numerical simulations are run. A first step of validation is done before results presenting and discussion.

Materials and Methods

Numerical framework

A numerical fluid-structure interaction model is implemented using the finite element method library FreeFem++ [4] to model the cerebrospinal system and its interactions with the brain. This model is based on the fluid mechanics equations, the Navier-Stokes equations, and the solid mechanics equations, the linear elasticity equations, coupled in a monolithic formulation [5].

The Navier-Stokes equations, that describe the fluid behavior in a moving domain, read as follow:

$$\rho^F \frac{\partial \mathbf{u}}{\partial t} + \rho^F ((\mathbf{u} - \mathbf{U}) \cdot \nabla) \mathbf{u} - 2\mu^F \nabla \cdot \boldsymbol{\epsilon}(\mathbf{u}) + \nabla p = \mathbf{f}^F$$

$$\nabla \cdot \mathbf{u} = 0$$

Where $\mathbf{u}$ is the velocity, p the pressure, $\mathbf{U}$ the domain velocity, $\mathbf{f}^F$ the volumetric external force (taken here to zero, gravity is neglected), ρ^F the density and μ^F the dynamic viscosity for the fluid; and where:

$$\boldsymbol{\epsilon}(\mathbf{u}) = \frac{1}{2} (\nabla \mathbf{u} + (\nabla \mathbf{u})^T)$$

The linear elasticity equations, that describe the solid deformation, read as follow:

$$\rho^S \frac{\partial^2 \mathbf{d}}{\partial t^2} - \nabla \cdot \boldsymbol{\sigma}^S(\mathbf{d}) = \mathbf{f}^S$$

Where $\mathbf{d}$ is the displacement, $\mathbf{f}^S$ the volumetric external force (taken here to zero, gravity is neglected), ρ^S the density for the solid; and where:

$$\boldsymbol{\sigma}^S(\mathbf{d}) = \lambda (\nabla \cdot \mathbf{d}) \mathbf{I} + 2\mu^S \boldsymbol{\epsilon}(\mathbf{d})$$

Where λ and μ^S are the Lamé's coefficients and $\mathbf{I}$ is the identity matrix.

Lamé's coefficients are defined using the Young's modulus and the Poisson's ratio as follow:

$$\lambda = \frac{\nu E}{(1 - 2\nu)(1 + \nu)}$$

$$\mu^S = \frac{E}{2(1 + \nu)}$$

Where E is the Young's modulus and ν the Poisson's ratio.

In order to couple this two equations, the continuity of velocity and stress is imposed at the fluid-structure interface as follow:

$$\mathbf{u} = \frac{\partial \mathbf{d}}{\partial t}$$

$$\sigma^F(\mathbf{u}, p)\mathbf{n} = -\sigma^S(\mathbf{d})\mathbf{n}$$

Where $\mathbf{n}$ is the normal vector and:

$$\sigma^F(\mathbf{u}, p) = -p\mathbf{I} + 2\mu^F\epsilon(\mathbf{u})$$

The monolithic formulation of this problem leads to the following matrix form, where fluid and structure problems are strongly coupled and solved at the same time:

$$\begin{pmatrix} A & B^T & 0 \\ B & \epsilon & 0 \\ 0 & 0 & \epsilon \end{pmatrix} \begin{pmatrix} U \\ P^F \\ P^S \end{pmatrix} = \begin{pmatrix} L \\ 0 \\ 0 \end{pmatrix}$$

Where ϵ is a stabilization term, A and B are the representatives matrix of the problem, U is the fluid velocity, P^F is the fluid pressure, P^S is the solid pressure (equal to zero) and L represents external and coupling conditions.

Geometry

The model geometry, simplified in a first approach, is deigned in accordance to realistic volumes ratio of the different compartments of the cerebrospinal system. Brain, intracranial subarachnoid spaces and ventricles are designed using two dimensions' disks and a thin tube is used to mimics the high flow resistance of the aqueduct, Fig. 1.

Figure 1. Geometry of the model

The cranium has a radius of 10 cm, intracranial SAS have a thickness of 0.75 cm, ventricles have a radius of 3 cm, aqueduct has a radius of 0.2 cm and spinal canal has a radius of 1 cm.

Parameters

Fluid and structure parameters, provided from the literature, are taken in accordance to the CSF and brain mechanical physiological parameters, Tab. 1.

Table 1. Mechanical parameters

Parameter	Symbol	Value	Unity
Fluid density	ρ^F	1	g.cm^{-3}
Fluid viscosity	μ^F	0.001	$\text{g.cm}^{-1}.\text{s}^{-1}$
Solid density	ρ^S	1.1	g.cm^{-3}
Solid Young's modulus	E	4.10^3	$\text{g.cm}^{-1}.\text{s}^{-2}$
Fluid Poisson's ratio	ν	0.35	-

The model input is located on the spinal canal end where a velocity profile, close to the physiological one, is imposed:

$$v = A \sin\left(\frac{2\pi t}{p}\right)$$

Where A is the velocity amplitude and p the period of the cardiac cycle.

Remark 1. In physiological conditions, CSF oscillations come from brain expansion at each cardiac cycle. In our model, this is the inverse phenomena: CSF oscillations cause the brain deformations. This behavior has been chosen, for future use of physiological measures, because CSF flow is easily measurable by phase contrast MRI; contrariwise, brain deformation is hard to obtain.

Simulations are done with different values of the heart rate period (p) keeping the same SV_{spi} , Fig. 2. Twenty cardiac cycles are simulated and the results are extracted from the last cardiac cycle. CSF velocity and pressure, and brain displacements are obtained as simulation results, whence CSF flow and stroke volume are calculated from this results.

Figure 2. Input of the model

Remark 2. A lot of cardiac cycles are simulated to ensure the stabilization of the system.

Results

Validation

Model validity is successfully performed on numerical benchmarks (specific cases) to ensure the correct agreement of the algorithm. In addition, the flow conservation is calculated in the used model geometry during a cardiac cycle: the net flow error is $1.4\% \pm 1.3SD$.

Study case

As a second validation, geometry and flow are measured in a healthy subject by MRI to design the model and impose an input velocity. Simulation results, Fig. 3 and Fig. 4, show the good agreement with the PC-MRI measured data and with the physiological ICP curve shape and amplitude.

Figure 3. Simulation results and PC-MRI measurements of the flow.

Figure 4. Simulation results of pressure.

Cardiac cycle impact

Simulation results, Fig. 5 and Fig. 6, show significant variability of SV_{aq} and SV_{sas} in the physiological range of cardiac frequencies; an inversion of the distribution is even observed for slow cardiac frequencies. Pressure gradient

(maximum pressure minus minimum pressure during a cardiac cycle) is equally modified by the cardiac frequency variations; it is exponentially increased as the cardiac frequency growth.

Figure 5. Stroke volume function of cardiac frequency

Figure 6. Pressure gradient function of cardiac frequency

Discussion

The linear elasticity is justified in this application since small deformations are obtained in the simulation results. The proposed model is validated in numerical (benchmark) and physiological (flow conservation) points of view.

Numerical results in the study case are in accordance to PC-MRI measured data. In addition, pressure curve presents a physiological amplitude and shape (peaks and valleys).

Cardiac frequency has an impact on the CSF distribution between the two main intracranial compartments: ventricles and intracranial SAS; stroke volume distribution is in a physiological range for normal (around 60 - 100 bpm) and high cardiac frequencies, contrariwise, the stroke volume distribution tends to be equal in the two main compartments as cardiac frequency decrease.

Cardiac frequency has equally an impact on the pressure gradient in the two compartments, pressure gradient grown as cardiac frequency increase. As the SV_{spi} is conserved, high cardiac frequency causes a rapid inflow of CSF into the intracranial compartment that can explain this phenomenon.

As the absolute pressure term does not appear in the Navier-Stokes formulation (only his gradient), it is impossible to obtain an information about the absolute pressure of the CSF: only the gradient of the intracranial pressure is calculated.

Conclusions

A numerical model is presented taking into account the numerous fluid-structure interaction in the cerebrospinal system closed in the rigid skull. This model highlights significant variations of the stroke volume distribution under cardiac frequencies variations only.

As perspectives, spatial distribution of the intracranial pressure gradient will be studied. An improvement of the model including arterial and venous flow will be designed.

Another perspective could be the determination of the absolute intracranial pressure regarding Marmarou's studies and the pressure volume curve [7, 8].

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