Fluid-structure interaction in the cerebral venous sinus

Fluid-structure Interaction in the Cerebral Venous Transverse Sinus

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***Abstract*— The biomechanics of the cerebral venous system plays an important role in determining blood flow to the brain. Computational approaches to help elucidate the role of the cerebral venous system in health and disease have largely focused on lumped-parameter models and one-dimensional computational fluid dynamics simulations. To expand upon the prior work, and to investigate the possible role of cerebral venous collapse in normal physiology and pathological conditions, we developed a fluid-structure interaction (FSI) model of the cerebral venous transverse sinus (TS), coupled to a lumped-parameter representation of the upstream cerebral circulation to provide boundary conditions for the FSI simulation. We simulated the effects of local venous hemodynamics on the TS distention and investigated TS vascular collapse under increased intracranial pressure, as has been hypothesized in the pathogenesis of idiopathic intracranial hypertension. Our baseline simulations reproduced pressures and flows in the cerebral venous system that compared favorably with what has been reported in the literature. The FSI simulations under increased intracranial pressure showed a decreased venous flow through and progressive collapse of the TS veins. Our simulations captured the dynamic behavior of the vascular collapse and may help shed light on the interactions between the cerebrovascular and cerebrospinal fluid spaces in health and disease.**

I. Introduction

The cerebral venous circulation exhibits the interesting biomechanical property of vascular collapse that is thought to be important in normal cerebrovascular physiology and may play a role in pathological conditions such as hydrocephalus [1] or Idiopathic Intracranial Hypertension (IIH) [2]. In normal cerebral physiology, the cerebral perfusion pressure (CPP) is defined as arterial blood pressure (ABP) minus intracranial pressure (ICP), CPP = ABP−ICP, rather than ABP minus venous pressure. This relation implies flow limitation in the cerebral venous bed. Such flow limitation is a highly nonlinear biomechanical and fluid dynamic phenomenon [3]. In IIH, collapse or stenosis of the transverse sinus (TS) is thought to set up a vicious cycle that leads to an increase in superior sagittal sinus pressure, decreased reabsorption of cerebrospinal fluid, increased intracranial pressure and further collapse of the TS [2]. Yet despite the importance of cerebral venous collapse in health and disease, the phenomenon remains poorly understood.

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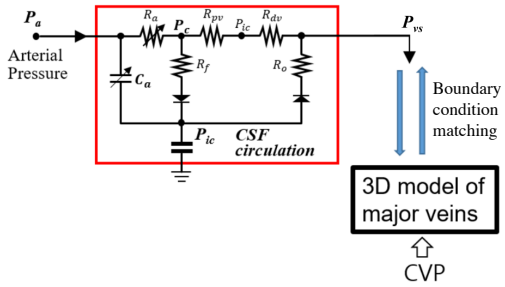

Fig. 1. Lumped parameter model of the cerebral circulation coupled to three-dimensional cerebral venous model. *R*, *C* and *P* stand for resistance, capacitance and compartment pressure, respectively. Subscripts are as follows: *a* – arterial; *ic* – intracranial; *c* – capillary; *f* – formation; *o* – outflow; *pv* – proximal veins; *dv* – distal veins; *vs* – venous sinus.

Several prior simulation studies have focused on the cerebral venous system. Gadda *et al.* [4] and Gisolf *et al.* [5], for example, simulated the effect of posture change on venous hemodynamics using lumped-parameter models (LPM). Recent one-dimensional fluid simulations [6] and computational fluid dynamic (CFD) studies [7] also focused on elucidating the cerebral venous hemodynamics. During vascular collapse, changes in venous fluid dynamics inherently affect the behavior of the vascular wall. To capture the dynamics of vascular collapse in detail, and its effect on physiology and pathology, we developed a detailed mechanistic, three-dimensional (3D) model of the large superficial cerebral venous vasculature, coupled to a LPM of the upstream vascular system. In particular, we solve the fluid-structure interaction (FSI) problem between the fluid domain and the vascular wall domain for the TS portion of the cerebral venous system. An increase of intracranial pressure was simulated to reveal the role of local venous hemodynamic effects on the dynamics and progression of TS vascular collapse.

II. Methods

A. Lumped parameter model of the cerebral circulation

The LPM representation of the cerebral circulation (Fig. 1) was adapted from the study by Ursino *et al.* [8]. The model is formulated as an electric-circuit analog, consisting of resistors and capacitors to model resistance to fluid flow and elastic soft-tissue properties, respectively. The arterial compliance C_a and microcirculatory resistance R_a are state-dependent to model cerebrovascular autoregulation. To simulate the model, we have largely adopted the parameter values chosen by Ursino [8]. The LPM provides mean compartmental pressures and flows over time. The pressure

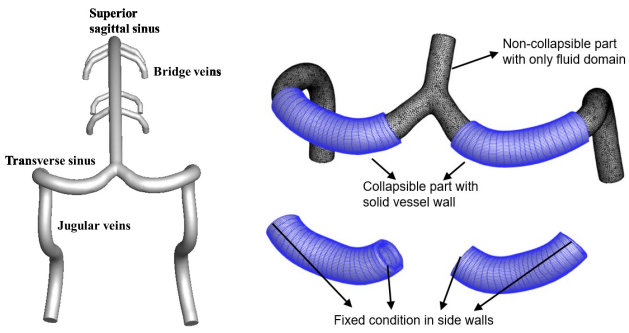

Fig. 2. Schematic geometry of the large superficial cerebral venous system (left) and computational mesh system of the fluid and solid domains used in the present study (right).

and flow trends at the distal venous compartment are used as boundary conditions for the 3D model of the large cerebral venous system.

B. Three-dimensional model of the transverse sinus

The geometry of the large superficial cerebral venous system was adapted from the idealized anatomical representations provided by Viewpoint, Inc. (http://www.viewpoint.com). Since our focus here is primarily on modeling the biomechanical behavior of the TS, the FSI portion of the model only covers the TS, as shown on the right of Fig. 2. Over these segments, we solve a 3D FSI problem in which the fluid and solid (vascular wall) domains and their interactions are updated in each time step. For the remainder of the 3D representation of the large superficial venous system, we solve the fluid domain with fixed wall conditions.

The TS FSI model is based on incompressible viscous flow and large-deformation nonlinear mechanics. We assume blood to be an incompressible Newtonian fluid and blood flow to be viscous dominated and laminar. Blood density, ρ, was taken to be 1,060 kg/m^3 and dynamic viscosity, μ, was set to 0.0035 $kg/(m \cdot s)$. We used a segregated finite-element method with a four-node tetrahedral mesh system to solve the 3D Navier-Stokes equation for venous flow. Since the inlet boundary of the fluid domain is coupled to the LPM, we did not specify an explicit boundary condition at the upstream end of the 3D model. Instead, as detailed in our previous work [12], [13], the LPM model was coupled to the 3D model by iteratively updating pressure and flow until consistency of the interfacial boundary condition was

TABLE I

COMPUTED VARIABLES AND REFERENCE VALUES.

Variable	Computed value	Reference values
Q_{jv}	10.5 ml/s	11.7 ml/s [9]
		12.0 ml/s [5]
P_{ic}	6.0 $mmHg$	5.0–15.0 $mmHg$ [10]
		11.0 $mmHg$ [11]
P_{vs}	6.5 $mmHg$	6.0 $mmHg$ [8]

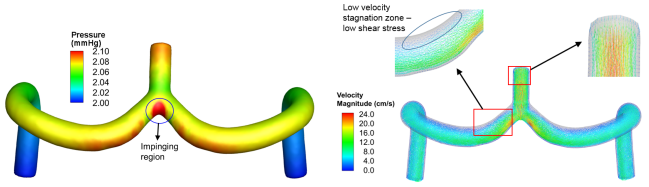

Fig. 3. Pressure distribution (left panel) and velocity vectors (right panel) of the flow domain.

achieved. For the outlet boundary, we impose a constant jugular venous pressure of 2 $mmHg$

The nonlinear finite-element method for the vessel wall is solved using a total Lagrangian approach for hyperelastic materials [14]. We implemented the Mooney-Livlin material model with an eight-node hexahedral mesh system. Since the material properties of the TS venous wall have not been described in detail, we used the bulk modulus and material properties of the jugular vein wall recently proposed by Kaul and Huang [15].

To solve the FSI problem, we applied Newton Raphson iterations to the solid domain coupled to our fluid solver. We specified the nodes of the side wall surface as fixed boundary condition (see Fig. 2). To treat the mesh deformation of the fluid domain induced by the interaction with the solid wall, we used the remeshing algorithm by Bazilevs *et al.* [16]. In our simulations, we avoided complete vessel collapse, which would imply contact between the opposing sides of the venous wall. Such contact is a complex mechanical interaction to represent in simulation, which we did not consider here.

III. RESULTS

A. Baseline simulation results

To validate the coupled LPM-3D cerebral venous simulation with FSI of the TS, we first evaluated the baseline case (no collapse) and compared the resultant volumetric flow rate, intracranial pressure, and venous sinus pressure (Table I). The realized values under baseline conditions compare favorably to values reported in the literature. Under these baseline conditions, no constriction of the TS lumen was occurred, though a low shear region was observed along the superior TS wall (Fig. 3). Fig. 4 shows the wall displacement and von Mises stress of the TS wall with only the left portion of TS vein walls shown. While regional differences in these variables exist, the magnitudes of these differences are quite small.

B. Fluid-structure interaction results of TS vein

To exercise the model under presumed pathological conditions, we imposed a linear increase in ICP, which acts as the downstream pressure to cerebral perfusion and the outside pressure to the TS, thus promoting vascular collapse when increased. Here, we imposed an increase in ICP by 7 $mmHg$, which induced a reduction in cerebral venous flow by 20%.

Vascular constriction in both segments of the TS was observed as ICP was increased, with concomitant increase

in the gradients of the von Mises stress in the vascular wall (Fig. 5). Additionally, the constriction of the vascular lumen induces changes in the flow pattern and the luminal pressure distribution (Fig. 6), showing a stagnated flow region between the collapsed portion and the inlet of the TS. In this simulation, more flow passes from the inlet through the right side of the TS. The upstream luminal pressure in the left (more collapsed) branch of the TS is slightly higher than in the right (less collapsed) branch.

IV. Discussion

The local cerebral venous hemodynamics are important to understand mechanistically to elucidate disorders of the cerebrovascular and cerebrospinal fluid spaces, such as hydrocephalus and IIH. In this study, we aimed to simulate local hemodynamics of the TS veins, including the phenomenon of vascular collapse under realistic physiological conditions. For this purpose, we implemented a coupled LPM-3D model of the cerebral circulation, with special emphasis on the large superficial cerebral venous system and FSI representation of the TS veins (Figs. 1 and 2).

We see three main contributions from this study. First, we implemented a 3D FSI model (Fig. 2) of the collapsible TS veins coupled to a LPM of the upstream cerebral circulation and compared the computed baseline results with existing findings in the literature to validate the model (Table II). Up to now, most computational studies on cerebral venous hemodynamics have been based on lumped-parameter or 1D fluid dynamic models [4], [5], [6]. The recent study by Miraucourt *et al.* [7] extended the prior work to 3D simulations of cerebral venous hemodynamics, thought their study did not incorporate interactions between the fluid and solid domains. Thus, prior computational studies did not represent the complex biomechanical phenomena underlying venous vascular collapse and their dependence on local venous hemodynamics and cerebrospinal physiology. In general, FSI simulations still pose challenging problems regardless of the specific research field as they require significant computational time and resources, and convergence of the iterative solutions between solid and fluid domains is not guaranteed. Recently, 3D FSI simulations of cardiac valvular

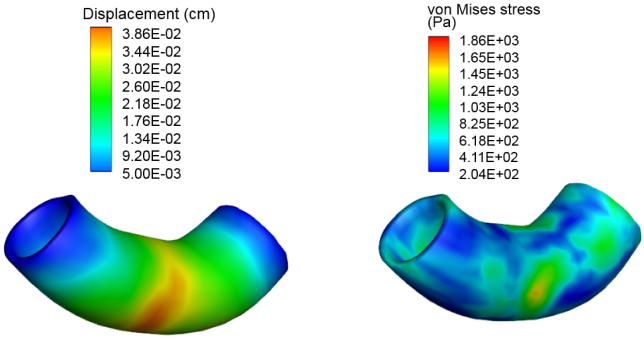

Fig. 4. Wall displacement from the reference configuration (left panel) and von Mises stress (right panel) of the solid domain.

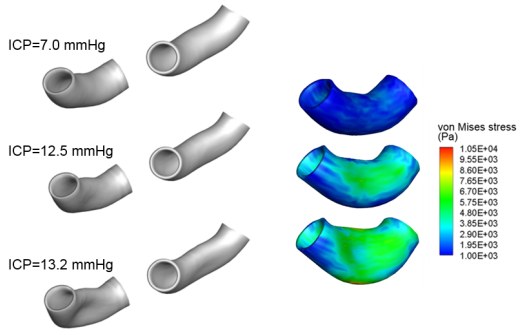

Fig. 5. Progressive collapse of the TS venous wall (left panel) and corresponding von Mises stress distribution (right panel).

mechanics were reported by Mao *et al.* [17], though to date such simulations have not been applied to the complex physiology of the cerebral veins. In this aspect, the present study provides a novel multiscale simulation method to analyze human cerebral venous hemodynamics.

Second, even in the baseline condition without vessel collapse, our simulations showed several interesting characteristics. A slight pressure gradient in the distal part of TS vein was observed while a larger pressure differential occurred at the bifurcation of the superior sagittal sinus into the two TS veins (right panel of Fig. 3). Additionally, we found that the superior portion of TS veins exhibited relatively low shear stress (right panel of Fig. 3). On the arterial side, low shear stress can induce inflammatory damage in vessel walls. Additionally, high pressures or high pressure gradients can also induce damage on the arterial wall. Compared to the arterial side, the pressures, pressure gradients, shear and wall stresses in our venous simulations are considerably smaller. Whether low shear stress and pressure gradients impart similar damage on venous walls, and may therefore contribute to vascular collapse even under baseline conditions, remains to be elucidated.

Third, we demonstrated that even comparatively modest increases in ICP, and concomitant changes in cerebral venous hemodynamics, can promote vascular collapse in our FSI simulations of the TS veins. It has been hypothesized that an uncompensated increase in ICP may set up a vicious cycle of reduced venous outflow, and increase in superior sagittal sinus pressure, and hence reduced reabsorption of cerebrospinal fluid, further increasing ICP [2]. Our simulations here did not explore the full range of such a vicious cycle as we did not represent the contact problem between opposite sides of the vascular wall and hence did not simulate to full vascular collapse. Nevertheless, we demonstrated that the large cerebral veins do show progressive collapse with increasing intracranial pressure.

Our initial exploration of cerebral venous vascular collapse has several limitations. First, the present simulations assumed a simplified venous geometry. Realistic geometries of the large cerebral veins are more complex than the present model. Second, the FSI model is currently limited to the TS portion of the large cerebral venous system and does

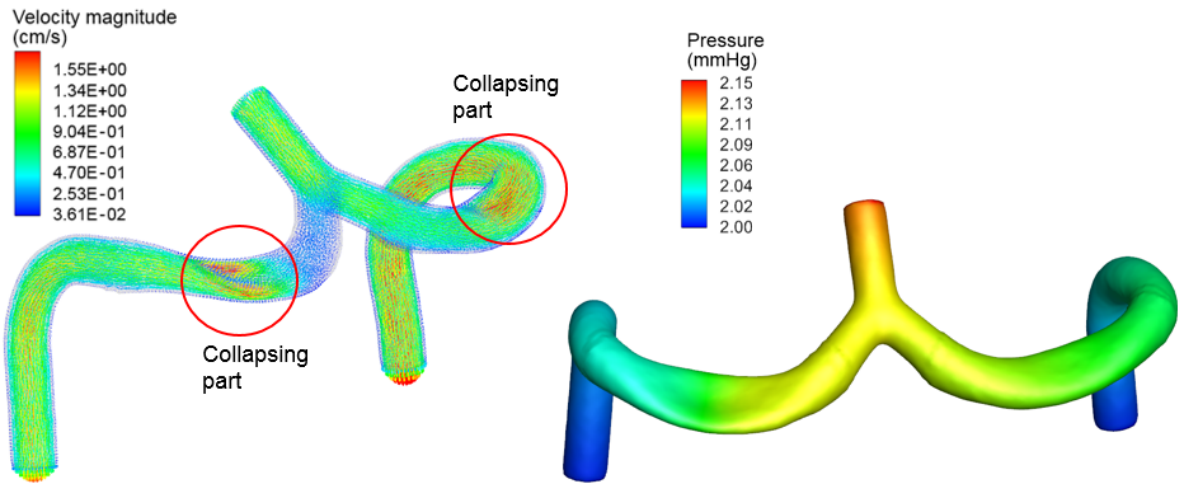

Fig. 6. Flow pattern (left) and pressure distribution (right) in the transverse sinus during vascular collapse due to increased intracranial pressure.

not extend to the the other large cerebral veins. Third, the material properties of large cerebral veins are not known. We adopted simple jugular venous wall properties, which surely oversimplifies the true biomechanical properties of the large cerebral veins. In fact, the large cerebral veins run between meningeal layers of stiff connective tissue that are known to have different biomechanical properties from thin-walled veins. Determination of realistic cerebral venous material properties is therefore an important area of further investigation and direction of refinement of our model here. Finally, we were only able to compare our computed results with very limited experimental data. In the future, more detailed validation is clearly desirable. Despite these limitations, we are optimistic that our approach will yield meaningful insights into the physiology and pathology of the cerebrovascular and cerebrospinal fluid systems through patient-specific modeling.

V. Conclusions

We developed a coupled LPM-3D model of the cerebral vasculature with FSI of the TS for modeling of venous collapse. The baseline simulation results of the model compared favorably with existing observations of pressures and flows in the cerebral venous system. We found that the 3D FSI model of the TS showed vascular collapse during progressive increase of intracranial pressure, affecting local hemodynamics through the TS. As our simulations become more refined, such detailed, mechanistic modeling of the large cerebral venous system can help our understanding of the pathogenesis of disorders of the cerebrospinal fluid system, including hydrocephalus and IIH.

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