# A development of a highly efficient FSI simulation platform for diagnosis of cardiovascular diseases.

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# 1. Introduction

Numerical simulations of fluid-structure interaction (FSI) of blood vessels such as aortic bifurcation, carotid artery and aortic valve have received much attention in the last several decades. An accurate simulation of the FSI problems has an important in the diagnosis and treatment of cardiovascular diseases. In this work, we develop a highly efficient FSI simulation platform for diagnosis of cardiovascular diseases. The performance of the present method is confirmed by simulation of two blood flows interacting with a structure of cardiovascular system: a pulsatile blood flow interacting with a blood vessel in a carotid bifurcation of a rat and a pulsatile blood flow interacting with an aortic valve.

## 2. Numerical method

Blood flow is assumed to be a laminar, Newtonian and incompressible. Gravitational forces are neglected, and the governing equations of blood flow are the incompressible Navier-Stokes equations which can be written in the arbitrary Lagrangian-Eulerian (ALE) formulation as follows:

$$\nabla \cdot V = 0$$
  

$$\rho^{f} \left[ \frac{\partial V}{\partial t} + (V - W) \cdot \nabla V \right] = - \nabla p + \nabla \cdot \left[ \mu (\nabla V + (\nabla V)^{T}) \right]$$

where **v** and **w** denote the blood velocity and the velocity of grid nodes, respectively. Fluid density and dynamic viscosity are given  $\rho^{f}$ =1050 kg/m3 and  $\mu$  = 0.004 Pa.s. A non-slip condition is applied at the fluid-structure interface. Blood vessel is modeled as a

hyperelastic material for a non-linear behavior. The governing equation of the blood vessel is then written as follows in the Lagrangian form:

$$\rho^s \frac{\partial^2 u}{\partial t^2} = \nabla_X \cdot T$$

where  $\rho^s$  and  $\nabla_{X^*}$  denote the vessel density and divergent operator in reference configuration, and **T** is the first Piola-Kirchhoff stress tensor. Mooney-Rivlin model is employed to describe the non-linear behaviour of blood vessel.

The projection semi-implicit scheme for FSI problem proposed by Fernadez et al. [1] was able to reduce the simulation time with the numerical stability comparable to the fully implicit scheme. In this method, the pressure traction is treated implicitly whereas the (geometrical other components non-linearities, viscous and convective forces) are handled explicitly when the solid solver is combined with a projection scheme for the fluid solver. Based on the algorithm described in reference [1], we implement a semi-implicit coupling algorithm for FSI simulation by adopting based the fractional four-step method. We solve a separate pressure equation for fluid and a solid equation iteratively with the help of the pressure boundary condition used in this study [2].

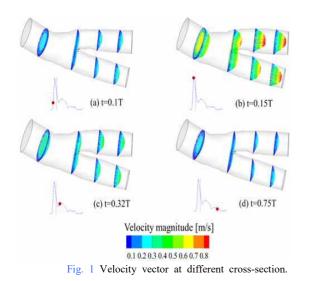
### 3. Results and discussions

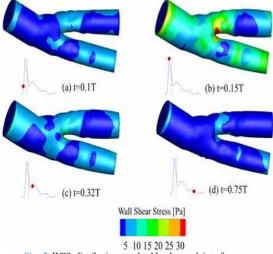
# 3.1 Blood flow interaction with a blood vessel in a carotid bifurcation of a rat

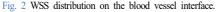
The first FSI simulation is blood flow interacting with a blood vessel in a carotid bifurcation of a rat, which includes a common carotid artery (CCA) branched into the internal and external carotid arteries (ICA and ECA, respectively) [3]. Fig. 1 shows the velocity

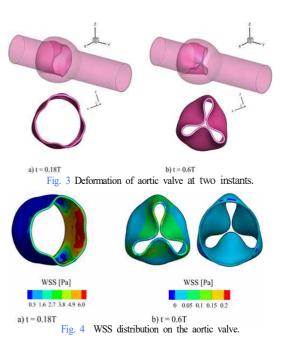
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profiles of blood in different cross-sections in the four instants Blood velocity reaches the maximum value (~0.8 m/s) at the peak flow-rate (t<sub>2</sub>=0.15T),and then decreased to~0.2 m/s at t<sub>4</sub>=0.75T. Accordingly, there is a rapid increase in wall shear stress (WSS) on the blood-vessel interface at the peak flow-rate as shown in Fig. 2. WSS is then decreased as the flow-rate decreases until t<sub>4</sub>=0.75T. It also shows that WSS of ICA is a little higher than that of ECA. Moreover, the maximum value of WSS is located around the branch of carotid artery in all instants.









### 3.2 Blood flow interaction with an aortic valve

The second FSI simulation is a pulsatile blood flow interacting with an aortic valve. The deformations of the valve at the two instants are illustrated in Fig. 3. The valve is fully opened at the systolic phase, where the flow rate reaches the maximum value ( $\sim$ 24.6 l/min). The valve is closed when the flow rate is zero at the diastolic phase. According to the pulsatile blood flow, there is a rapid increase in wall shear stress (WSS) on the inner wall of the valve at the peak flow-rate as shown in Fig. 4. WSS is then decreased as the flow-rate goes to zero when the valve is closed (t<sub>2</sub>=0.6T).

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#### References

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