

# Three Band Analysis in a FSI model of aorta

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## Introduction

The role of hemodynamics in development of cardiovascular pathologies such as atherosclerosis and aneurysm has been recognized both in vitro and in vivo<sup>4</sup>. Mathematical models, simulating the behaviour of the cardiovascular system in healthy and unhealthy conditions, are becoming the gold standard for advanced speculation. In particular it has been shown that hemodynamical instabilities, such as “disturbed” flow patterns and low or high oscillating values of Wall Shear Stress (WSS), correlate with plaques formation and subsequent growth<sup>4</sup>. In this work, a fluid-structure interaction (FSI) model of thoracic aorta is presented and its behaviour both in healthy and unhealthy states is discussed. For the unhealthy case, three virtual aneurysm models are created with different budge size in order to study how it effects the WSS patterns. Moreover, in order to evaluate the importance of taking into account FSI in a blood vessel, the WSS profiles are analyzed both for rigid and compliant cases. Finally, the recent risk indicator TBD<sup>3</sup> (Three Band Diagram) is applied to better investigate the underlying differences between the results obtained.

## Material and Method

The numerical models used in the present work are based on an axisymmetric geometry consisting of three subdomains (Fig 1):

1. the fluid domain (blue) consists of a cylinder with radius of 1.25 cm;
2. the arterial wall (red) consists of a cylinder with an inner radius of 1.25 cm and an outer radius of 1.45 cm;
3. a third fictitious solid domain (green), introduced to avoid to fix the external surface of the arterial wall and to mimic tissue around it, consisting of a cylinder with an inner radius of 1.45 cm and an outer radius of 4.45 cm.

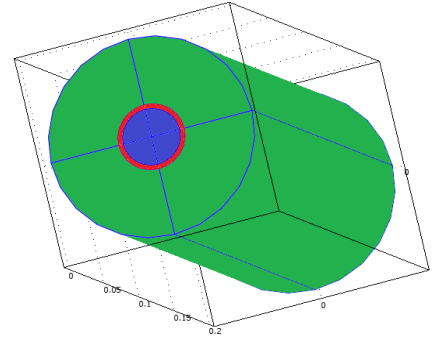


Figure 1: CAD geometry of the healthy blood vessel

In the unhealthy case, three axisymmetric aneurysm models are created by inserting a budge with different lengths and diameters. In particular, the results presented in this paper are related to an aneurysmatic model with a 3 cm long dilated segment and a maximum transverse diameter of 5 cm.

The aorta is a large vessel, therefore the fluid structure interaction between arterial walls and blood cannot be neglected. For such reason, in this study an ALE<sup>6</sup> (Arbitrary Lagrangian Eulerian) approach is employed in order to take into account the change in the computational fluid domain caused by the displacements induced by the propagation of the pulse wave in the vessel. The blood flow is assumed to be incompressible and Newtonian, thus governed by the Navier-Stokes equations which are formulated in the ALE framework<sup>6</sup>:

$$\rho_f \left( \frac{\partial \underline{v}_f}{\partial t} \right)_{\tilde{A}} + \rho_f (\underline{v}_f - \underline{w}) \cdot \nabla_x \underline{v}_f + \nabla p - \mu \nabla_x^2 \underline{v}_f = 0$$

$$\nabla_x \cdot \underline{v}_f = 0$$

(1)

Here  $\nabla$  and  $\nabla^2$  are the gradient and the laplacian operator calculated with respect to the spatial coordinate  $x$  in the current frame,  $\underline{v}_f$  is the fluid velocity field,  $\left( \frac{\partial \underline{v}_f}{\partial t} \right)_{\tilde{A}}$  is the ALE time-derivative of  $\underline{v}_f$ ,  $p$  is the fluid pressure,  $\underline{w}$  is the velocity of the fluid computational domain which is computed via opportune operator prescribed by ALE method<sup>6</sup>,  $\rho_f$  is the blood density and  $\mu$  is the dynamic viscosity.

The total Lagrangian formulation of the elastodynamics balance equations are used to model the two solid domains, readily:

$$\hat{J}_s \hat{\rho}_s \frac{\partial^2 \hat{u}_s}{\partial t^2} - \nabla_{\hat{x}} \cdot (\hat{F}_s \hat{S}_s) = 0 \quad (2)$$

Here  $\nabla$  is the gradient operator calculated with respect to the spatial coordinate  $\hat{x}$  in the reference frame,  $\hat{u}_s$  is the displacement field of the solid wall,  $\hat{\rho}_s$  is the density of the arterial vessel,  $\hat{S}_s$  is the second Piola-Kirchhoff stress tensor,  $\hat{F}_s$  is the deformation gradient tensor and  $\hat{J}_s$  its determinant. The two solid domains are assumed to be constituted by a non linear isotropic and homogeneous hyperelastic material. In particular a Mooney-Rivlin material model describes the behaviour of the arterial wall while a St.Venant-Kirchhoff strain energy density function is adopted for the fictitious solid domain. Parameters are reported in Table 1.

Regarding the boundary conditions, the inlet and outlet surfaces of the fluid domain are supposed fixed in their axial position but free in the radial direction. The same condition is imposed on the inlet and outlet surfaces of the two solid domains, whereas the most external surface is kept fixed. At the fluid-wall interface, continuity of velocity and stress is imposed. In addition, on the fluid domain inlet an experimental time dependent pulsed velocity profile<sup>7</sup> with a peak value of 0.6 m/s is imposed, whereas a zero pressure with no viscous stress condition is applied as outlet. The outlet boundary is posed at 6 m far from the inlet to avoid that the formation of reflection waves effects the hemodynamics of the fluid. This implies that an interruption of the simulation after few pulses is mandatory.

In this study the TBD<sup>3</sup> risk indicator is applied. It consists in dividing a given wall shear stress signal in a triplet of “daughter” functions of a control threshold:

$$S^+ \equiv S(t)H^+(\sigma) \quad S^- \equiv S(t)H^-(\sigma) \quad S^0 \equiv S(t)H^0(\sigma) \quad (3)$$

Here  $H^+(\sigma) = 1$  if  $S(t) > \sigma$  and 0 otherwise,  $H^-(\sigma) = 1$  if  $S(t) < -\sigma$  and 0 otherwise and finally  $H^0(\sigma) = 1$  if  $-\sigma < S(t) < \sigma$  and 0 elsewhere. The main idea of such TBD analysis is to inspect the number of intervals visited by the signal and their individual extent as a function of the running threshold. Numerical simulations are performed by using Comsol Multiphysics engine running on a multiprocessor workstation. A triangular mesh spatial discretization is adopted for all three domains: quadratic Lagrangian elements are used for the solid domains displacement  $\hat{u}_s$ , the fluid domain velocity  $\underline{w}$  and the fluid velocity  $\underline{v}_f$  and linear Lagrangian elements are adopted, as usual, for the fluid pressure  $p$ . The time integration method proceeds through a generalized-alpha algorithm linked to the direct solver PARDISO with a threshold of 0.75 and absolute and relative tolerances of  $10^{-5}$ . For each numerical model, three simulated heartbeats are performed with an adaptive time step calculated on the basis of the mesh size and the velocity field of the fluid in order to achieve numerical convergence. The computational time is on the order of 7 hours per simulation.

## Discussion and Results

In this work the behaviour of the thoracic aorta in healthy and unhealthy aneurysmatic conditions is simulated. The pressure gap is physiological as well as the velocity profile, the displacements of the arterial wall and the propagation velocity of the pressure wave<sup>6, 7, 8</sup>. Streamlines patterns are analyzed to evaluate vortex formation and translation in the budge of the aneurysm during every cardiac cycle (Fig. 2A). The wall shear stress profiles are calculated both in the deformable and rigid case verifying that in the rigid case the WSS is overestimated in agreement with the results obtained by Bazilevs et al<sup>1</sup>. In the healthy case the calculated WSS is very similar to the experimental measurements performed in vivo<sup>5</sup> while the magnitude of the mean value of WSS calculated in the unhealthy case agrees with the results reported in other numerical studies<sup>2</sup>. Finally the TBD analysis is applied to the simulated WSS signals in order to verify if the behaviour of the blood vessel is correctly identified. As compared to standard mean value of WSS, TBD provides an immediate risk information contained in the dynamic structure of the signal. This allows a quick visual assessment of the risk sensitivity for individual fluctuations of the physiological risk thresholds (Fig. 2B and Fig. 2C).

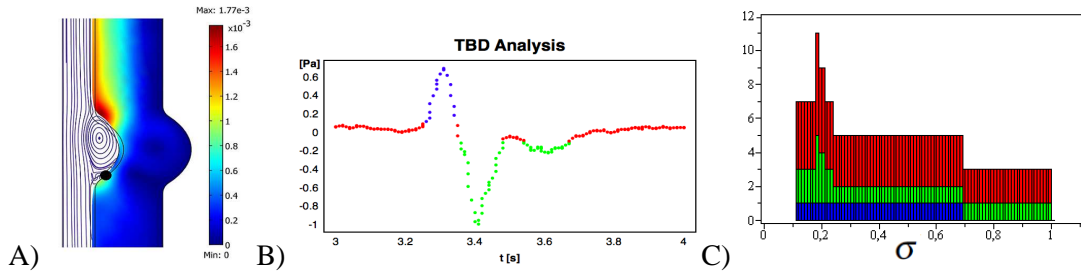


Figure 2: A) Streamlines pattern and displacement field in aneurysmatic blood vessel. B) WSS signal and TBD threshold analysis. C) TBD histogram analysis.

## Conclusion

In this work a numerical analysis of a healthy and unhealthy fluid-structure model of thoracic aorta are performed through existing risk indicators. Future applications include the use of more realistic 3D models with anisotropic materials to model arterial wall. The future main target is a risk assessment for patient specific data.

	Density [kg/m <sup>3</sup> ]	Dynamic viscosity [cP]	Mooney-Rivlin constants [Pa] $C_1=C_2$	Poisson coefficient	Young modulus [Pa]
Fluid domain	1050	4	-	-	-
Arterial wall domain	1200	-	$50 \cdot 10^3$	-	-
Fictitious elastic domain	900	-	-	0.45	500

Table 1: Parameters adopted in the simulations

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