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COMPUTATIONAL FLUID STRUCTURE INTERACTION OF A TWO-LAYER MODEL AORTA WITH AN ABDOMINAL AORTIC ANEURYSM. THE XXV COBEM

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Abstract: *The aneurysm is a cardiovascular disease that consists of a dilatation of a particular blood vessel. One of the major problems is to try to prevent this vessel from suffering a rupture due to dilation and in turn the failure of the organ or body region that is nourished by it. In this context, the current work aims to obtain a structural mapping of the region affected by the aneurysm and to evaluate the biomechanical characteristics of the arterial wall and its interaction with the fluid flow of the blood. In this way, a computational simulation of the bilayer model of an aortic artery conditioned to an abdominal aneurysm (AAA) using the fluid-structure interaction (FSI) method was performed to achieve the proposed goals. The FSI method is a joint analysis of the blood flow characteristics with the arterial wall structure, in which the interaction occurs between the pressure and velocity fields obtained from the blood flow to the arterial wall structure. The results presented are comparative fluid-structural maps of the artery with and without aneurysm and an assessment of which layer of the artery (adventitia or media) will be more prone to failure.*

Keywords: *Aneurysm, Fluid-Structure-Interaction, Two-Layer-Model, Computational Simulation, Biomechanical.*

1. INTRODUCTION

Aortic Abdominal Aneurysm (AAA) is an asymptomatic cardiovascular disease that causes irreversible diametral dilatation in the thoracic aorta region, affecting the structure of the arterial wall whose rupture can lead to death. Due to this, studies carried out in the field of mechanical characteristics of the aortic artery and its functioning are fundamental to prevent, predict and avoid the risk of rupture besides contribute as information for a better diagnosis. These studies can be used as a source for surgical solutions, such as insertion of an endoprosthesis or complete removal of the affected region.

The understanding of the mechanical characteristics of the structural health of a vessel conditioned to an AAA is complex since biological tissues are materials that exhibit anisotropic, viscoelastic and non-linear behavior (Holzapfel *et al.*, 2016). These characteristics are due to the constitution of arterial tissue, whose composition is basically by collagen and elastin, important protein substances in the structural formation of biological tissues. Another factor that contributes to the complexity of the problem is that the arteries are formed by three layers: *intima*, *media* and *adventitia*, which mechanical contribution depends on the location of the artery in the human body.

The present work aims to perform a computational simulation of the aortic artery conditioned to an abdominal aneurysm (AAA), considering the geometric two-layer model and using the Fluid-Structure Interaction (FSI) methodology, which provides more numerical results believable to the actual operation of the vessel in the human body.

2. MATERIALS AND METHODS

The two-way FSI method is characterized by the interactive response between the fluid and the structure, it being necessary the knowledge of the fluid dynamics characteristics of the flow as well as the mechanical characteristics of *intima*, *media* e *adventitia* the structure.

2.1 Geometry

Firstly, the geometric model was developed from Digital Imaging and Communications in Medicine (DICOM) obtained by computed tomography, using a free medical image processing program (*InVesalius*). The model was edited, using *Meshmixer* (free 3D CAD-sculpting software distributed by *Autodesk*), in order to obtain a healthy two-layered artery and a similar artery with aneurysm. The geometry was created to represent two layers of the artery: media and adventitia. The thickness of each layers are 1 mm in the body of the artery and 0.5 mm in the carotid arteries. The models, in STL files, were exported and converted in solids in *SpaceClaim* (*Ansys19.0*®). Fig. 1 shows the geometries generated, respectively as described above.

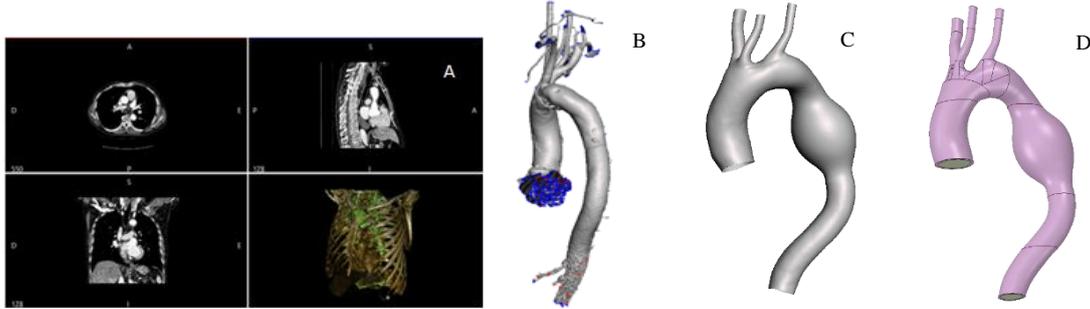


Figure 1: A- Computed tomography used in InVesalius; B- Image of the model to be worked on Meshmixer; C- Meshmixer final image; D- Ansys final image

2.2 Mesh and Time Step evaluation

The mesh test was performed from an initial choice of the length of the mesh element h defined by the ASME V & V 20 standards, presented by Eq (1). Both the flow and the structure, the mesh was refined until the convergence values of pressure and velocity (for flow), stress and strain (for structure) .The characteristics of mesh are shown in Tab.1 and Fig. 2.

$$h = \left[\frac{1}{N} \sum_{i=1}^n (\Delta V_i) \right]^{1/3} \quad (1)$$

Table 1: Flow and Structural Mesh

Mesh	Elements	Nodes	$h(m)$
Flow	1067435	250661	0,00020
Structural	246826	485268	0,00015

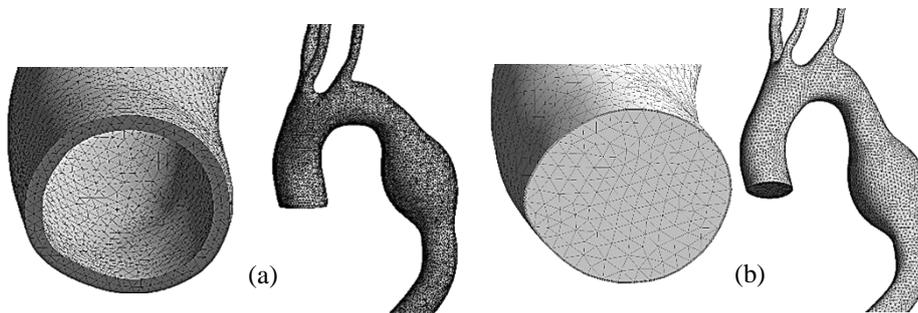


Figure 2: (a)Structural Mesh (b) Flow Mesh

The timestep evaluation was defined according with Courant-Friedrichs-Lewis condition, presented by the Eq.(2). The value calculate for this problem was 0.005s, considering a cardiac cycle of 0.8s, therefore, totalizing with 160 intervals.

$$C = \frac{u\Delta t}{\Delta x} \quad (2)$$

2.3 Fluid Dynamics Modeling

The equations of conservation of momentum and mass were used in the software to solve the arterial flow.

$$\frac{\partial u}{\partial x} + \frac{\partial v}{\partial y} + \frac{\partial w}{\partial z} = \nabla \cdot \vec{V} = 0 \quad \text{and} \quad \rho \frac{Du}{Dt} = -\frac{\partial p}{\partial x} + \text{div}(\mu \text{ grad } u) + S_{M_x} \quad (3)$$

The blood was considered a non-Newtonian fluid, using relationship between the rate of deformation and the shear stress defined by the model of Carreau-Yasuda presented in Eq (4). The values of the parameters used are presents in the Tab. 2.

$$\frac{\eta - \eta_\infty}{\eta_0 - \eta_\infty} = [1 + (\lambda \dot{\gamma}_{xy})^a]^{\frac{n-1}{a}} \quad (4)$$

Table 2: Numeric parameters for the Carreau-Yasuda model (Lin *et al.*, 2017)

Parameters	η_0	η_∞	λ	n	a
Value	0,056 Pa.s	0,0035 Pa.s	3,313 s	0,3568	2

In addition, a turbulence model k- ω SST (Shear Stress Transport) was used to satisfy the turbulent effect condition for low Reynold values and in order to restrict the turbulent viscosity value. Equation (5), Eq. (6) and Tab. 3 are the k- ω SST model and the parameter used in this work.

$$\mu_t = \rho \frac{\kappa}{\omega} e^{-\frac{\partial(\rho\kappa)}{\partial t}} + \text{div}(\rho\kappa U) = \text{div} \left[\left(\mu + \frac{\mu_t}{\sigma_\kappa} \right) \text{grad}(\kappa) \right] + P_\kappa - \beta' \rho \kappa \omega \quad (5)$$

$$\frac{\partial(\rho\omega)}{\partial t} + \text{div}(\rho\omega U) = \text{div} \left[\left(\mu + \frac{\mu_t}{\sigma_\omega} \right) \text{grad}(\omega) \right] + \alpha \frac{\omega}{\kappa} P_\kappa - \beta \rho \omega^2 \quad (6)$$

Table 3: Numeric parameters for the turbulence model k- ω SST .(Silveira, Matheus Rodrigues, 2017)

Parameters	α	β	β'	σ_κ	σ_ω	$\rho(\text{kg/m}^3)$
Value	0,52	0,075	0,0828	8	6	1060

Finally, the boundary conditions used are presented in Fig. 3 and Fig. 4, referring to the pressure curve (Olufsen *et al.*, 2000) used as outlet and velocity of the blood flow (Silveira, 2017) used as inlet

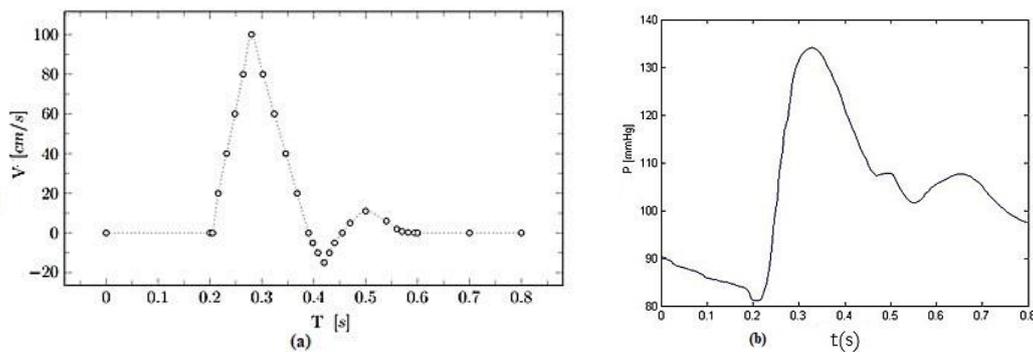


Figure 3: (a) Inlet Velocity pulse graph of the cardiac cycle (b) Outlet Blood pressure in a cardiac cycle.

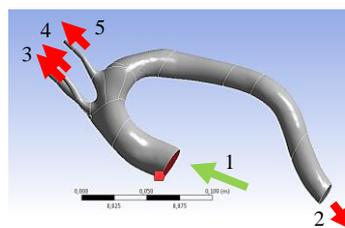


Figure 4: 1- Inlet Velocity; 2-5 Outlet blood pressure.

2.4 Structural Modeling

The aorta is a thick blood vessel composed of three layers of tissue: intima, media, adventitia. However, the intima layer has low mechanical contribution compared to the media layer and adventitia (Schulze-Bauer and Holzapfel, 2003). The adopted model considered only these two layers. The arterial wall was considered orthotropic, hyperelastic, using the Mooney-Rivlin hyperelastic model, showed in Eq.(7) and Tab. 4. This model uses Strain Energy Function (SEF) described by Fung (1993).

$$W = C_{10}(I_1 - 3) + C_{20}(I_1 - 3) + \frac{(J-1)^2}{d} \quad (7)$$

Table 4: Constitutive Mooney Rivlin parameters for arterial wall (Simsek and Kwon, 2015).

Layer	$C_{10}(MPa)$	$C_{20}(MPa)$	$d(MPa^{-1})$
Media	0.227	2.453	0.881
Adventitia	0.151	1.636	1.322

The kinematic modeling of the arterial wall is based on the deformations due to the pressure field of the flow and is solved in the software using the Eq.(8).

$$\dot{\chi} = \mathbf{V} \quad \text{and} \quad \text{Div}(\mathbf{FS}) + \mathbf{b}_0 = \rho_0 \dot{\mathbf{V}} \quad (8)$$

Where $S = \mathbf{S}(\mathbf{X}, t)$ is the second stress tensor of Piola-Kirchoff, $\mathbf{b}_0 = \mathbf{b}_0(\mathbf{X}, t)$ is the body force (defined per unit volume relative to Ω_0) which is referred to the reference position \mathbf{X} , and ρ_0 is the density of wall equal to 1300 kg/m³. The term $\rho_0 \dot{\mathbf{V}}$ characterizes the inertia force per unit volume. The structural contour conditions are presented by Eq. (9) and Eq. (10).

$$\mathbf{u}(\mathbf{X}, t) = \bar{\mathbf{u}} \quad \text{in} \quad \partial\Omega_{0u}; \quad [\mathbf{F}(\mathbf{X}, t)\mathbf{S}(\mathbf{X}, t)]\mathbf{N} = \bar{\mathbf{T}} \quad \text{in} \quad \partial\Omega_{0\sigma} \quad \text{for} \quad t \in [0, T] \quad (9)$$

$$\mathbf{u}(\mathbf{X}, t)|_{t=0} = \mathbf{u}_0 \quad \text{in} \quad \tilde{\Omega}_0; \quad \mathbf{V}(\mathbf{X}, t) = \mathbf{V}_0 \quad \text{ein} \quad \tilde{\Omega}_0 \quad (10)$$

3. RESULTS AND DISCUSSIONS

The characteristics of the FSI simulation performed in this study are presented in Tab.5, Tab. 6 and Tab .7. Two cases were simulated: Abdominal Aortic Aneurism(AAA) and Healthy Aorta (H.A.). Ansys® 19.2 licensed for UFMG Labbio was used in all simulation.

Table 5: Time step evaluation of cardiac cycle

Time Step	$T_1=0.20s$	$T_2=0.27s$	$T_3=0.42s$	$T_4=0.80s$
Cardiac Cycle	Systolic period	Systolic Peak	Diastolic Period	End of Diastole

Table 6: Parameters analyzed.

Flow	Pressure	Velocity	
Structural	Von mises Stress	Shear Stress	Wall Displacement

Table 7: Time and CPU Analysis

Cardiac Cycle duration	N° of Cycles Simulated	Time Step	N° Time Step	Simulation Time	CPU
0.80 s	2	0.005s	320	68 hours	Intel I7 16Gb NQuadro 2GB

3.1 Flow Analysis

The fluidynamics analisys of the flow is shown in Fig.5 and Fig. 6.

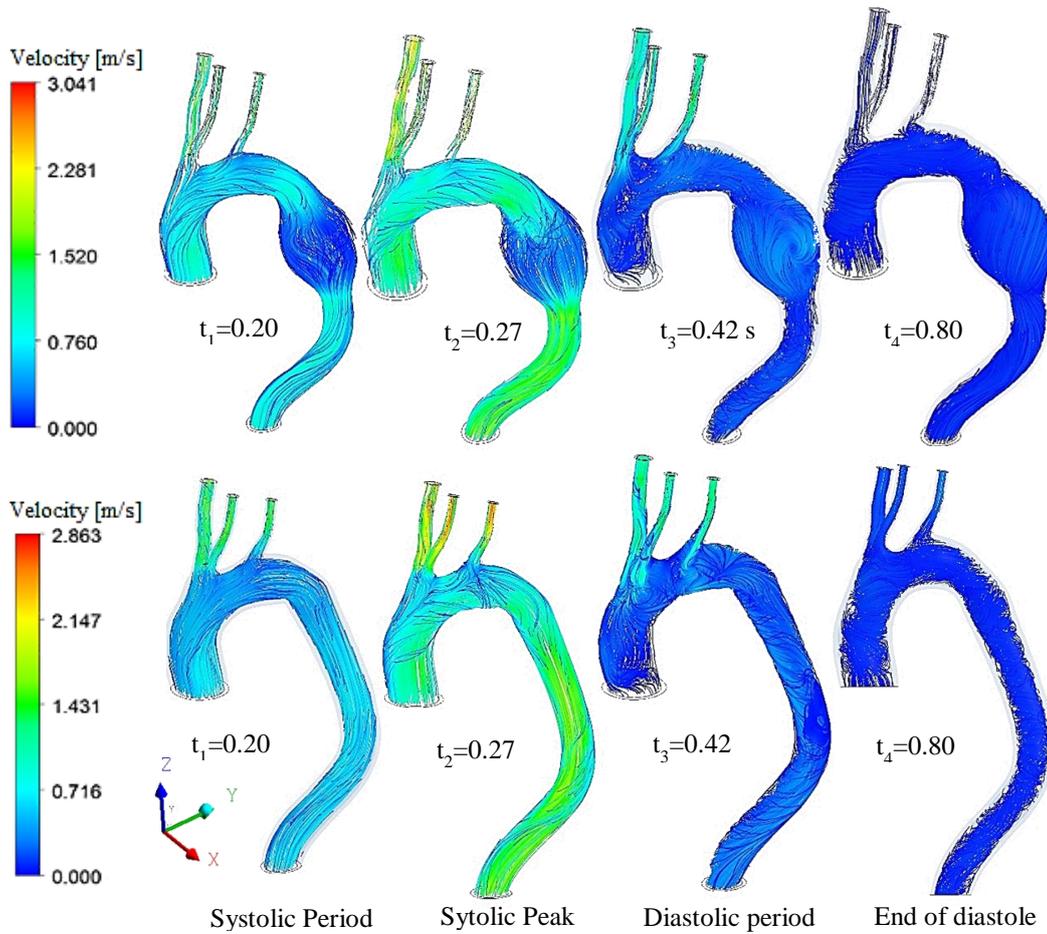


Figure 5: Flow Velocity

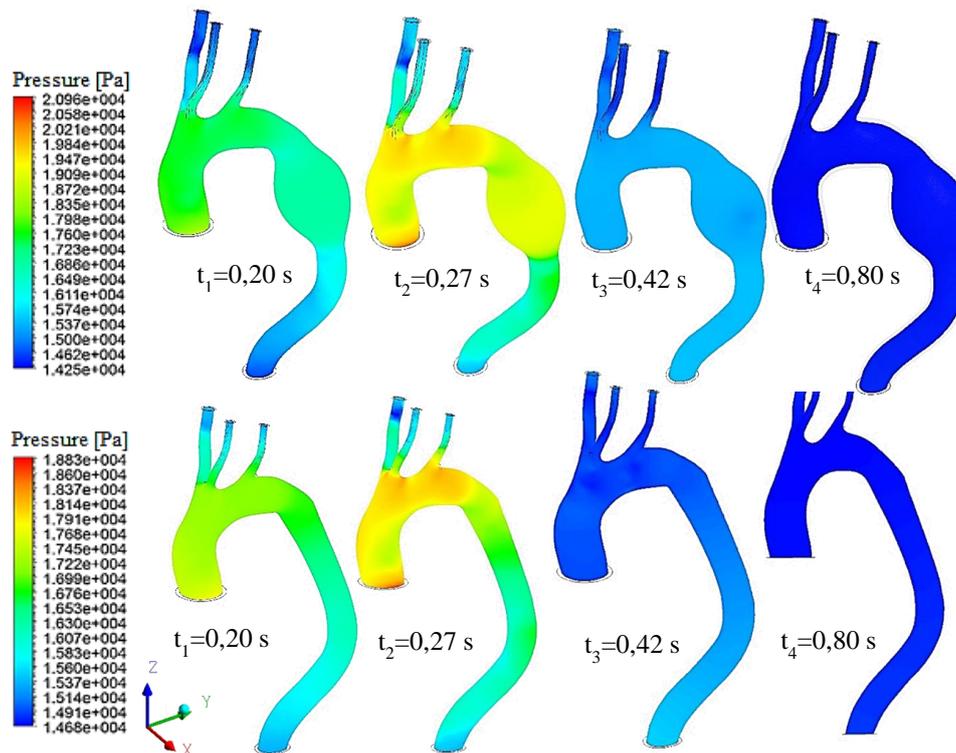


Figure 6: Flow Pressure field

The analysis of the flow evidences that the presence of the aneurysm generates an increase of pressure due to the volume of blood in the affected region being greater. In addition, the velocity of blood flow is reduced in the aneurysm caused by the diffusive effect present in the region. Turbulence is more present in the aneurysm than health aorta, since more vortices are generated due to the same diffusive effect. In this way, the blood flow in that region is compromised due to the loss of load caused by the disease, so the heart will make more effort to compensate for the loss of charge and boost the blood to the whole body.

3.2 Structural Analysis

The structural analysis performed for the *media* and *adventitia* layers is presented in Fig. 7 and Fig. 8.

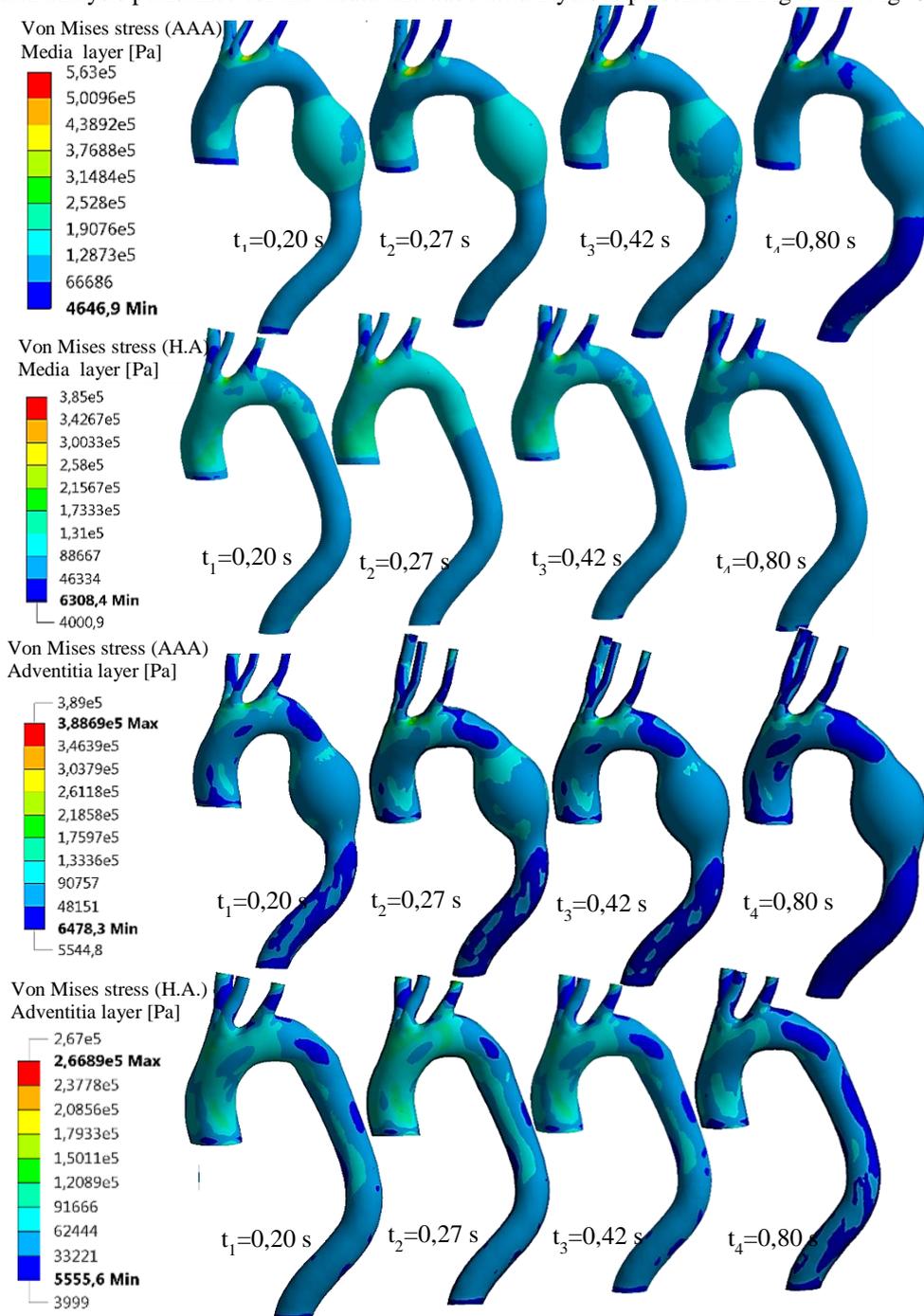


Figure 7: Von mises Stress Mapping for *adventitia* and *media* layer comparing AAA and H.A cases.

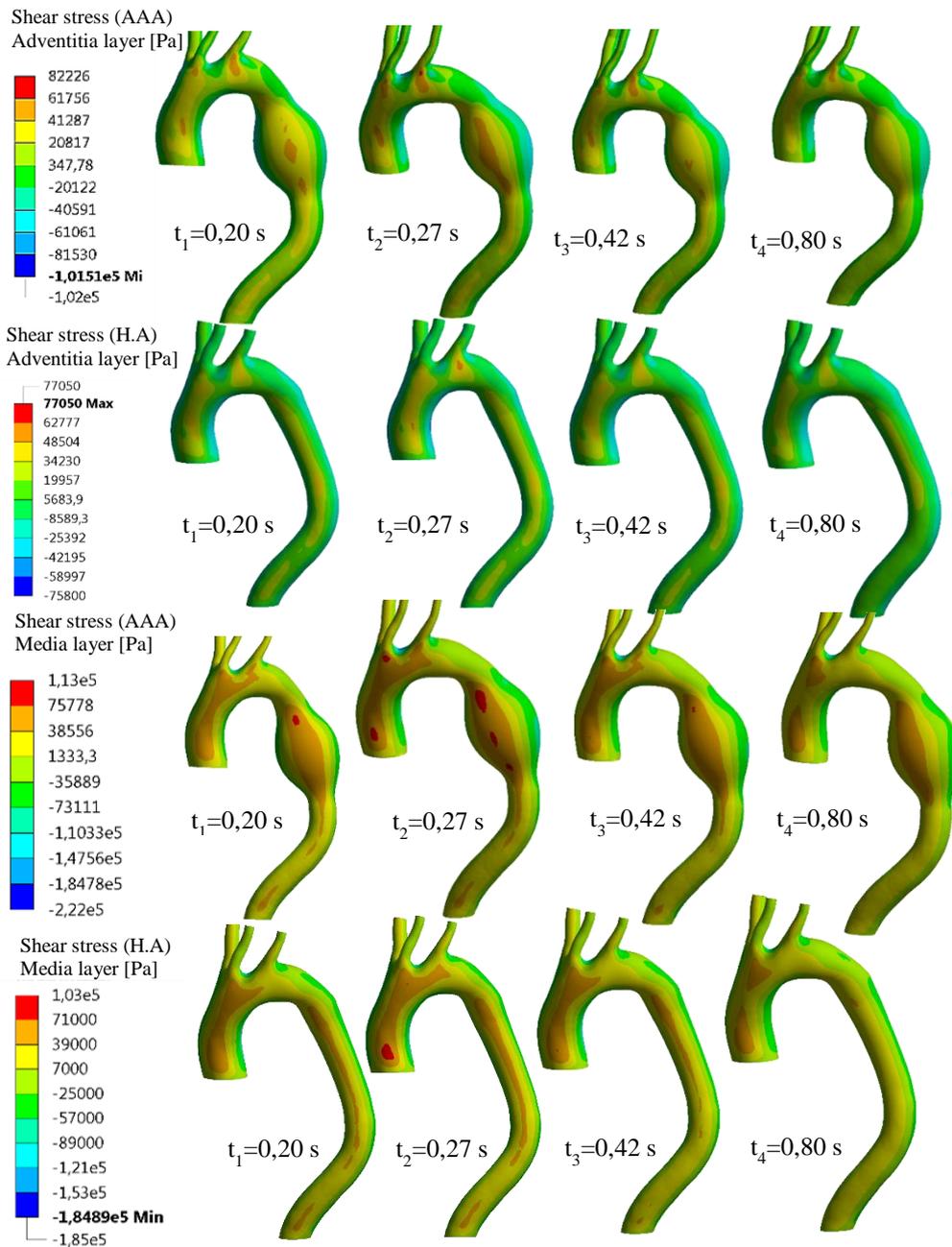


Figure 8: Shear stress mapping for *adventitia* and *media* layer comparing AAA and H.A. cases.

The structural analysis between AAA and H.A shows that the aneurysm elevates shear and total stress values in the affected region. Further, it is clear that the medial layer is subject to higher stress values than the adventitious layer. These amplified voltages generate a situation of abnormal structural behavior in the vessel, which will lead to rupture in the affected region. Figure 9 presents, respectively, the structural behavior of the layers for the von mises and shear stress criterion.

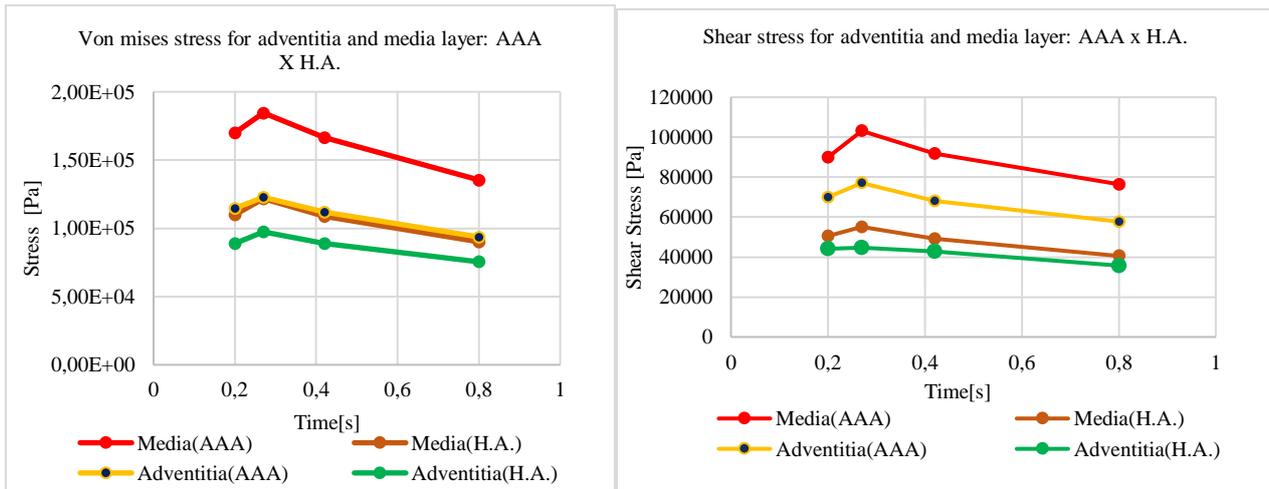


Figure 9: Von Mises and Shear stress analysis for *media* and *adventitia* layers: AAA X H.A

The arterial wall displacement is shown in Fig. 10.

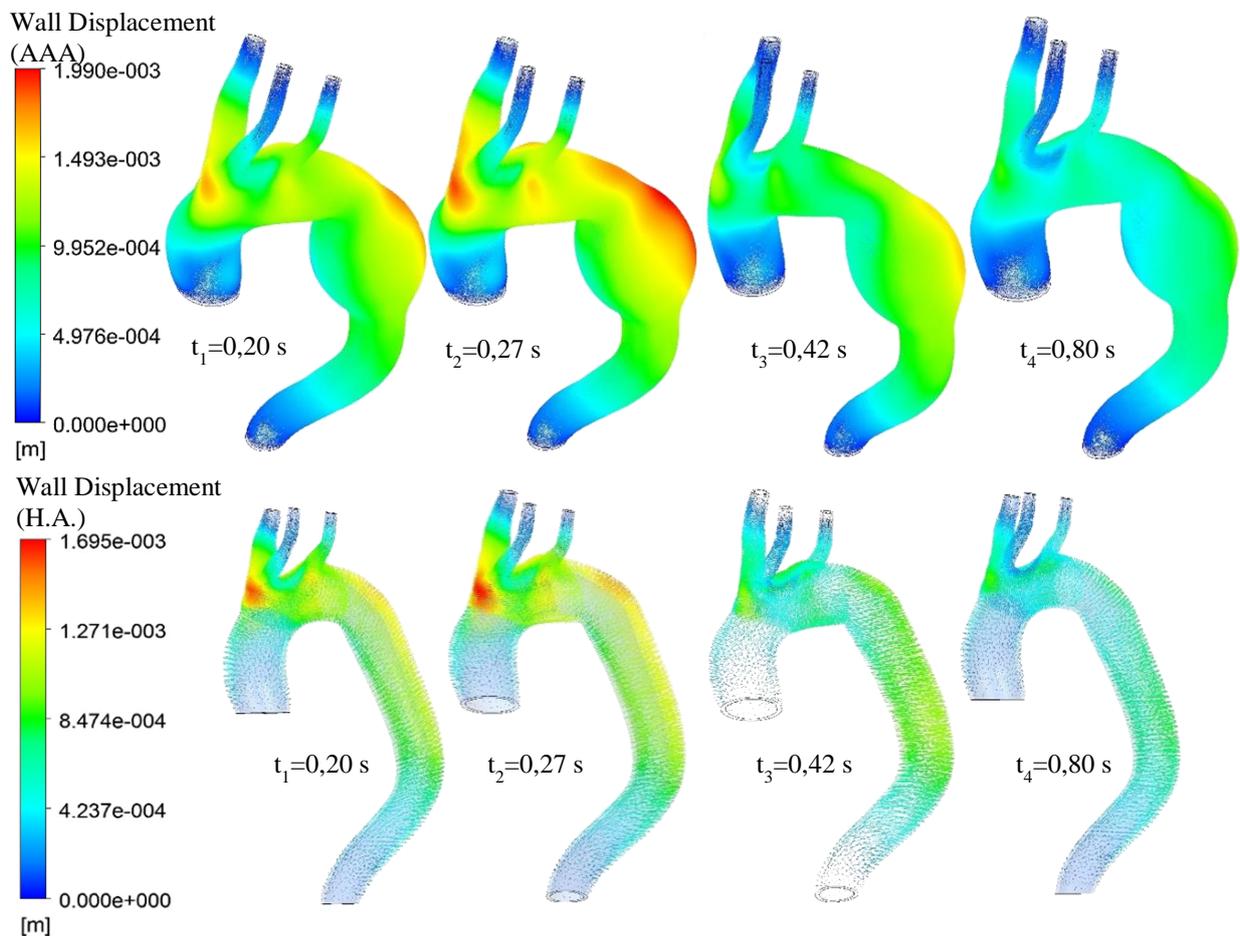


Figure 10: Wall displacement configuration AAA X H.A.

The arterial wall displacement configuration shows that the presence of AAA increases as much as it changes during the cardiac cycle, with higher values at the time of systolic peak.

4. CONCLUSION

The FSI simulation of the aortic artery conditioned to an abdominal aneurysm clearly reveals that the disease generates an abnormal cyclical state of tension in the affected region, which compromises the characteristic structural and dynamic fluid functioning of the arterial vessel.

The fluid-dynamic flow presents a change in the AAA region, where a decrease in velocity is evidenced by the diffusive effect of the diametrical increase of the vessel section. In addition, the local pressure increases, since the volume of fluid, where there is AAA, increases. It is also observed the generation of vortices due to the effect of the detachment of the limit layer of the flow, caused by the sudden change of the geometry. Consequently, this generates a state of increase of the cardiac effort that the heart realizes to pump the blood by the vessel, in order to compensate this loss of load caused by the presence of AAA.

The behavior of the layers regarding the conditions of AAA and Healthy Aorta reveals that the *media* layer presents a state of cyclic tension superior to the *adventitia* layer, which was expected, since this layer is in direct contact with the blood flow.

In the biological aspect, the structural simulations establish the arterial behavior was approximated to the data present in the literature. This leads to the conclusion that, as the aneurysm grows, the layers of the arterial wall undergo a molecular degeneration, caused by the increase of the stress, then the peptide bonds break down, and this generates a protein denaturation of collagen and elastin causing the eminent rupture of the vessel.

In summary, the FSI simulation highlights the possibility of a better understanding of the structural fluid behavior of the aorta conditioned to an abdominal aneurysm, which provides: information crucial for a more accurate diagnosis, increases the data richness for the construction of surgical implants, Stents and biocompatible prostheses.

5. ACKNOWLEDGEMENTS

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7. RESPONSIBILITY NOTICE

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