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HEMODYNAMIC NUMERICAL STUDY OF AN ANEURYSM IN THE VICINITY OF A THREE-DIMENSIONAL ARTERIAL BIFURCATION

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Abstract. *In the last decades, there has been a growing concern to measure the dynamic parameters of blood, given the immense losses of lives due to cardiovascular diseases in the history of humanity, among them, the aneurysms. The formation and development of an aneurysm is predominantly degenerative and results from a complex interaction between the biological effects of the arterial wall and the flow and stress due to the hemodynamics. This work intends to analyze the blood only as a Newtonian fluid and to collaborate for a better understanding of the properties of the blood flows inside Abdominal Aortic Aneurysms (AAA) such as velocity, pressure and wall shear stress, considering only turbulent effects. The governing equations were solved using Ansys-Fluent® software, considering the Spallart-Allmaras and k-ε RNG models to describe the turbulence. In this way it was possible to compare the turbulence models and the results obtained were close because both models behave well in situations of adverse flows.*

Keywords: *Abdominal aortic aneurysm, Hemodynamics, Turbulence models, Bioengineering.*

1. INTRODUCTION

Abdominal Aortic Aneurysm (AAA) is an abnormal dilation of the walls of a blood vessel, either an artery or a vein (Azevedo, 2010). Abnormal dilation is understood when it has diameters greater than 50% of normal, which occurs most often in the infrarenal region (Johnston *et al*., 1991). Since the disease is silent, about 70% of cases are diagnosed late (Law, 1998). This is the cause of many sudden deaths due to rupture of AAA, which occurs immediately or a few hours after the onset of abdominal or lumbar pain (Puech-Leão and Kauffman, 1998).

According to Pio (2013), about 6.5 thousand people die annually in Brazil for aneurysm. Data from the Brazilian Ministry of Health indicate that in 2010 there were 6,680 deaths, of which 4,016 in the Southeast, 1,063 in the South, 977 in the Northeast, 456 in the Midwest and 168 in the North regions. In previous years, 2009 and 2008, Brazil recorded, respectively, 6,419 and 6,413 deaths due to aneurysms.

The behavior of the aneurysms differs on a case-by-case basis. This can be explained by structural factors of the arterial wall that can be acquired at birth, undetermined causes and specific diseases, as well as hemodynamic factors that alter the flow in certain regions of the artery, besides factors such as age, sex, cigarette use or Drugs, which favor aneurysms with different characteristics due to arterial wall injury (Azevedo, 2010).

When velocities of blood flow inside the vessels increase, for example, when passing through an obstruction, there is transition from laminar to turbulent flow. The predominantly parabolic profile, characteristic of the laminar flow, is altered and the cellular components can adhere to each other and to the subendothelial layer. Turbulence delays the entry of coagulation inhibition factors, allowing plaque growth, hardening and, consequently, losing the flexibility of the arterial wall, promoting an increase in flow resistance, leading to a significant increase in blood pressure (Cotran, Kumar and Collins, 1999).

This effect of reverse flow in the infrarenal abdominal aorta and in the iliac arteries has been simulated using Computational Fluid Dynamics (CFD) methods. Such simulations are gaining importance in the study of flow in

aneurysms and arteries as they provide detailed information on the velocity and tension distributions in the wall of the aneurysm, which may be used in the future to predict aneurysm rupture (Taylor and Draney, 2004).

Órfão (2014) quoting Azevedo (2010) presents two theories related to the causes of aneurysm rupture. In the case in one theory, it is stated that the wall of the aneurysm is submitted to a low wall shear stress (WSS), processes such as remodeling of the arterial wall would be triggered, giving rise to weak spots on the wall of the aneurysm, resulting in its rupture. The second theory considers that the processes associated with high levels of WSS are responsible for the damages caused in the wall of the vessel, resulting in its rupture. Thus, the study of blood flow is important to characterize the details of the shear stress fields.

Geraldes (2015) also relates the rupture of the aneurysm with the hemodynamic parameters of the flow, such as WSS. According to the author, regions subject to high WSS can cause damage to the wall of the blood vessel. While in the regions of low WSS atherosclerosis (hardening of the artery wall) occurs due to the low mass diffusion of lipids, and in this way allows a longer contact time between the lipid molecules and the artery wall. In addition, in regions of low velocity flow, where WSS is generally low, a predisposition to the formation of thrombi (blood clotting point near the wall) is observed.

The understanding of the flow on the endothelial cells of the blood vessels is necessary, since shear stress and wall pressure are important characteristics to be analyzed, because these cells are permanently exposed to shear stresses. For the range of normal values, from a physiological standard, the shear stresses are between 1 and 7 *Pa*. However, when these values of shear stresses increase to values greater than 10 *Pa*, there is a provision for thrombosis. When tensions increase to values greater than 35 *Pa*, injury and deterioration of the endothelium occurs. For low shear stresses with values ranging from -0.4 to +0.4 *Pa*, the appearance of atherogenesis is associated. Aneurysm rupture is also associated with elevated mean blood pressure. This can be explained qualitatively due to the effect of hemodynamics, with a higher average arterial pressure on the wall of the aneurysm, with its consequent weakening (Geraldes, 2015).

In general, see Figure 1, the main factors that are associated with the formation and growth of an abdominal aortic aneurysm are:

- Minimum and maximum shear stress in the wall of the aneurysm;
- Efective stress on artery wall (Von Mises stress);
- Transmural pressure;
- Hidrostatic pressure;
- Maximum diameter of the artery during systole;
- Mechanical properties of the artery wall (Young's modulus, Poisson's coefficient, rupture stress).

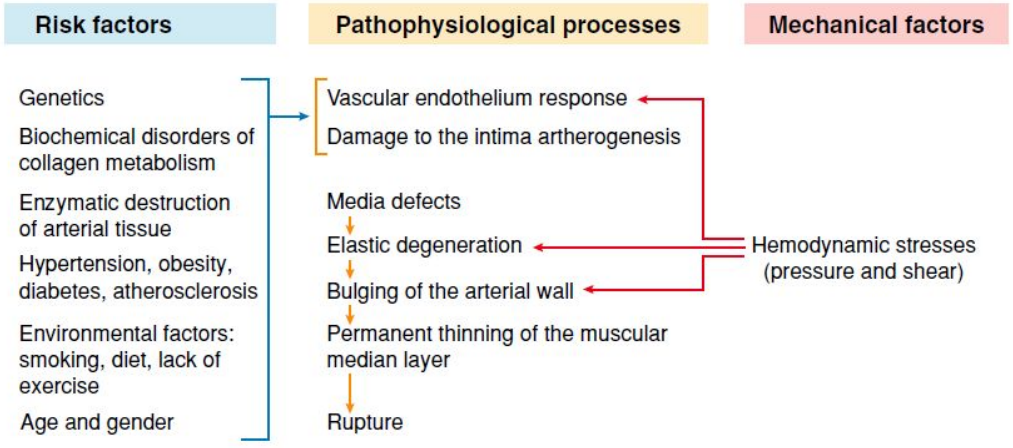

Figure 1. Pathogenesis of arterial aneurysms. Interaction between mechanical stimuli and physiological processes. (Lasheras, 2007)

In order to have parameters values associated with aneurysm rupture from CFD simulations, suitable models for flow and blood (fluid) should be used. As there is no consensus on the best model of turbulence to study flow in aneurysms, this study aims to compare models of turbulence in the flow of abdominal aneurysms, by comparing parameters such as velocity and pressure fields and the wall stresses for an aneurysm that is located in the arterial bifurcation.

2. COMPUTATIONAL PROCEDURE

The methodology adopted for the blood flow behavior study in the bifurcation along the cardiac cycle is formed by some main steps:

- Construction of the 3D biomodel (Figure 3) generated by means of DICOM images using Invesalius software;
- Generation of the mesh using Ansys software;
- Simulation of blood flow using Fluent software for a rheological model and a turbulent model.

In the present work, all simulations were performed using the computational resources available in Computational Simulation Laboratory in Thermal Sciences (M4) at the UNESP - Ilha Solteira, which are presented in Table 1.

Table 1 – Hardware Settings Used.

Item	Description
Processor	Intel Core i7 3.4 *GHz*
RAM	16 *GB*
Hard Disk (HD)	1*TB*
Graphics	NVIDIA GeForce GT 640
Monitor	15”

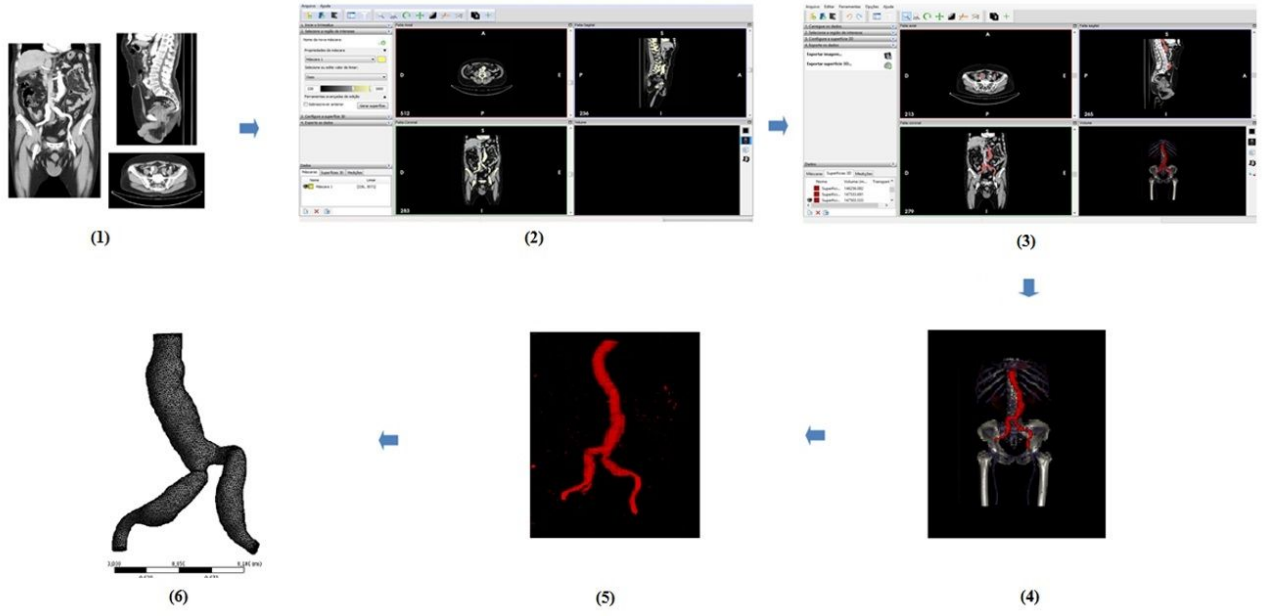

Figure 2. (1) Obtaining medical images, (2) Importing DICOM files, (3) Segmentation of the region of interest, (4) Obtaining the 3D biomodel, (5) Export of the 3D biomodel in STL format and (6) Reconstruction of 3D biomodel.

Because the blood flow is pulsatile and cyclic during the systolic phase, the velocity of the input is not defined to be a constant, but rather a time-varying periodic profile. For this feature, a User Defined Function (UDF) was used as boundary condition in the inlet, see Figure 3 and Figure 4. Sinnott, Cleary and Prakash (2006) proposed this model.

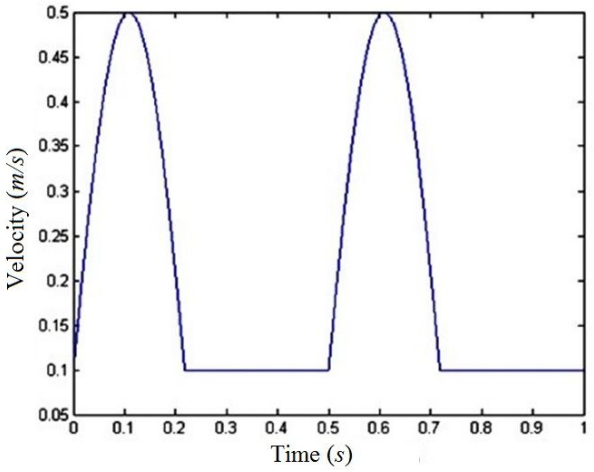

Figure 3. Velocity profile.

The boundary conditions considered at the walls of the artery is that of no-slip, that is, velocities equal zero are imposed on the walls. The outlet pressure is adopted as static pressure because the mean systolic and diastolic pressure are respectively 120 *mmHg* and 80 *mmHg* for a healthy human. Thus, the mean pressure of the two phases is 100 *mmHg* which is equivalent to about 13332 *Pa* as shown in Figure 4.

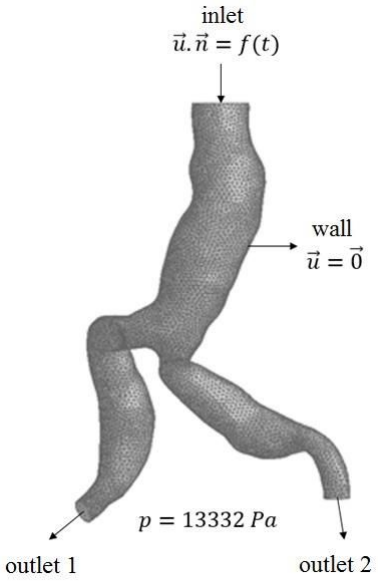

Figure 4. Boundary conditions on bifurcation.

3. RESULTS AND DISCUSSION

It is observed in both models at the outlets that in the pulse transition from 0.5 *s* to 0.6 *s* (velocity peak) the blood is forced through the aneurysm, according to the UDF, so recirculations are not so visible. When the pulse decelerates to 0.7 *s*, the adverse flow occurs and a non-linear behavior of the current lines becomes more visible at the end of the wrist, as shown in Figures 5 and 6.

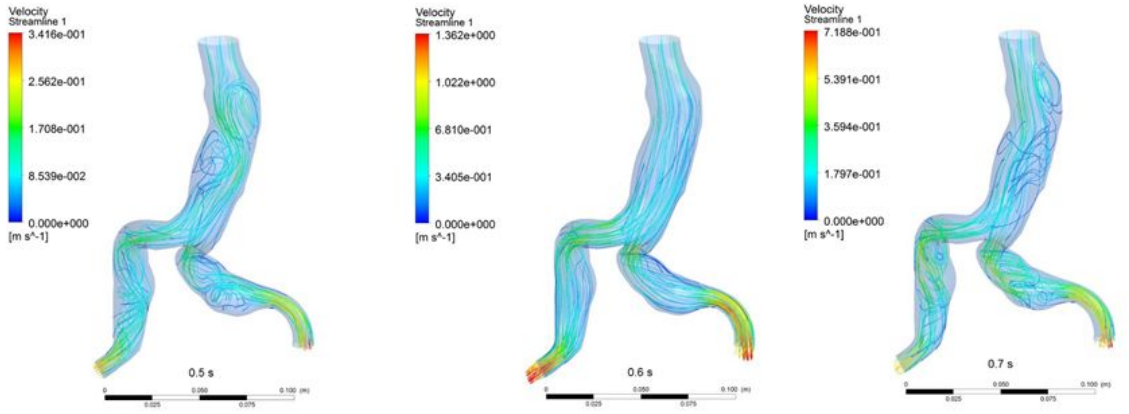

Figure 5. Velocities field in the domain using the Spalart-Allmaras model for the following instants: t = 0.5 *s*, 0.6 *s* (velocity peak) and 0.7 *s*, respectively.

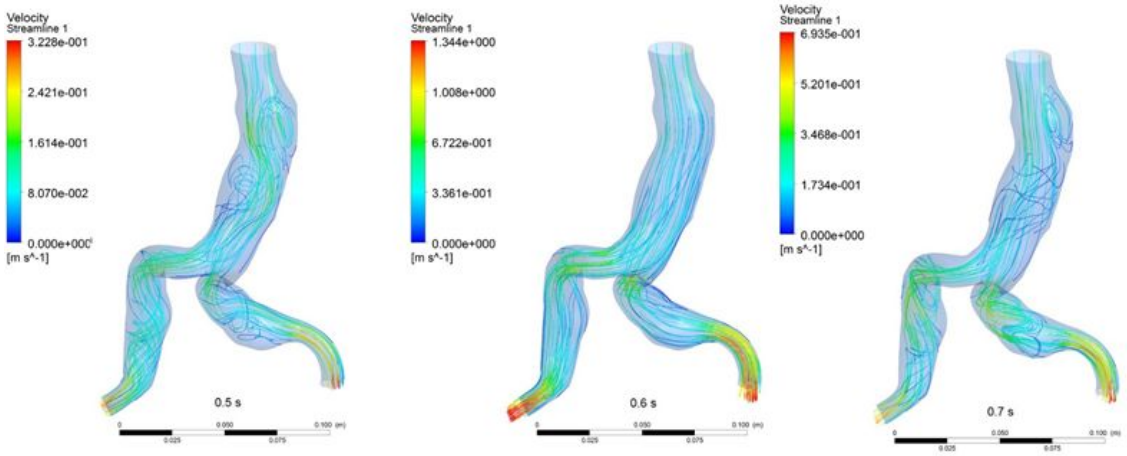

Figure 6. Velocities field in the domain using the *k*-ε RNG model for the following instants: *t* = 0.5 *s*, 0.6 *s* (velocity peak) and 0.7 *s*, respectively.

In the recirculation regions the velocities are lower, as can be seen in Figures 5 and 6, so that the shear stress in the wall adjacent to these regions are smaller.

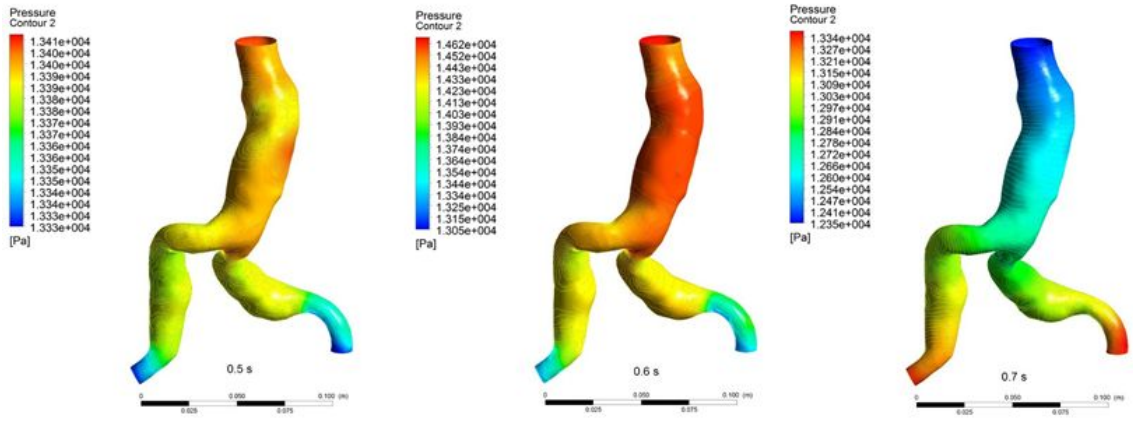

Figure 7. Pressure field on the surface of the domain using the Spalart-Allmaras model for the following instants: *t* = 0.5 *s*, 0.6 *s* (velocity peak) and 0.7 *s*, respectively.

It can be seen in Figures 7 and 8 regions with very high pressure, combined with regions with low speeds and recirculations, which causes regions of weakening of the aneurysm wall.

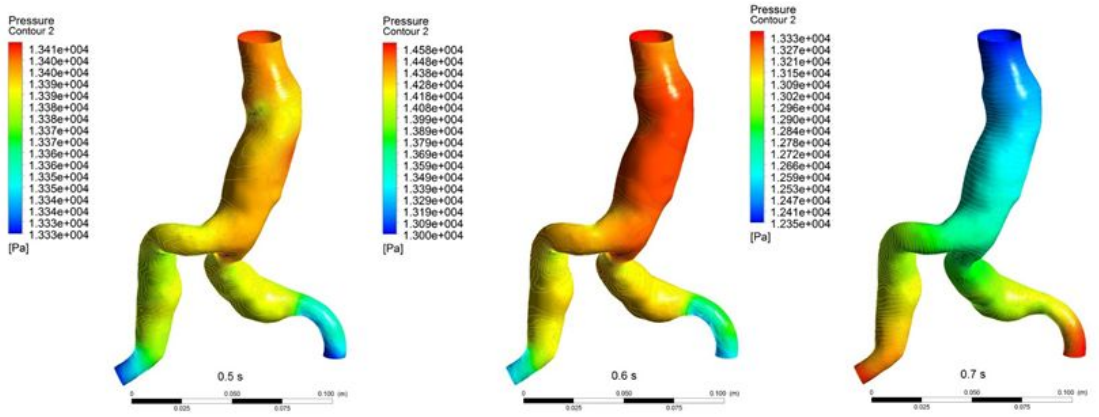

Figure 8. Pressure field in the surface of the domain using the *k-ε* RNG model for the following instants: $t = 0.5$ *s*, 0.6 *s* (velocity peak) and 0.7 *s*, respectively.

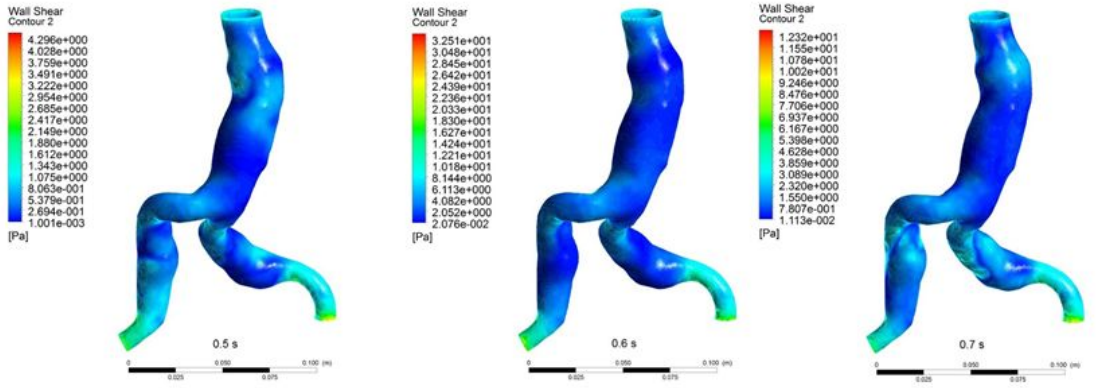

Figure 9. Shear stress distribution on the surface of the domain using the Spalart-Allmaras model for the following instants: $t = 0.5$ *s*, 0.6 *s* (velocity peak) and 0.7 *s*, respectively.

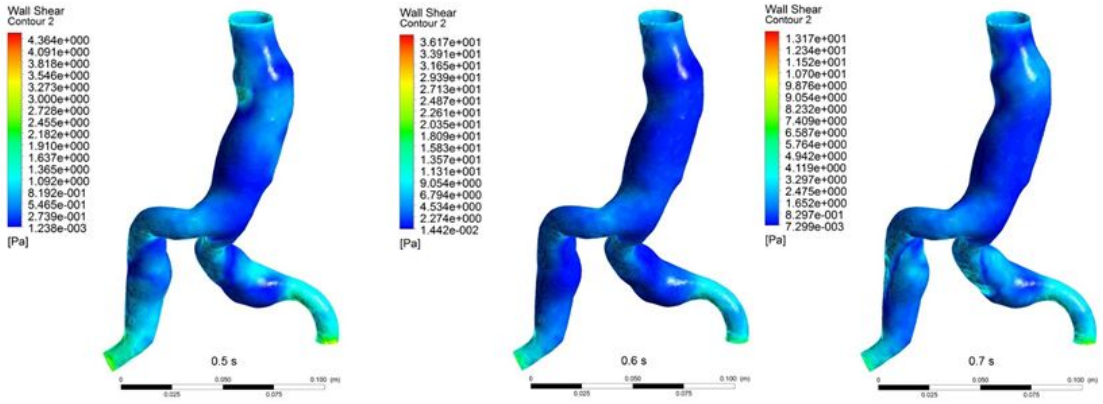

Figure 10. Shear stress distribution on the surface of the domain using the *k-ε* RNG model for the following instants: $t = 0.5$ *s*, 0.6 *s* (velocity peak) and 0.7 *s*, respectively.

It is observed in Figures 9 and 10, which show the WSS for the two models studied, that the maximum value of the shear stress for the Spalart-Allmaras and k-ε models occurs at the instant 0.6*s* that corresponds to the velocity peak, with

values of the order of 32 and 36 *Pa* respectively, which is outside the normal physiological range - 1 to 7 Pa. Due to the maximum WSS values are close to each other, from 32 *Pa* and 36 *Pa*, it can be observed that the Spalart-Allmaras model with its simplicity of 1 equation model offers a similar result to the *k-ε* model (2 equations).

In both models tested, the aneurysms were close to disruption due to the level of WSS, 35 *Pa*. (Geraldes, 2015). However, it is noteworthy that in none of these cases was the pathological effects such as fissures in the artery wall or even other diseases taken into account.

4. CONCLUSION

According to the study, the objectives were reached, as they showed comparisons between the Spalart-Allmaras models and k-ε RNG, evidencing that both models predict close values of hemodynamic parameters related to rupture of aneurysms. In addition, the time spent simulation for both models was close, due to the computational time being low. For a longer simulation time, the Spalart-Allmaras model may be adequate due to its simplicity, since it solves only one equation, which will decrease the computational cost.

5. ACKNOWLEDGEMENTS

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