ISSN 2176-5480

22nd International Congress of Mechanical Engineering
November 3-7, 2013, Ribeirão Preto, SP, Brazil

HEMODYNAMICS FEATURES AS RUPTURE RISK PREDICTORS OF ABDOMINAL AORTIC ANEURYSMS

Guillermo Vilalta
Federal University of São João del-Rei (UFSJ), Thermal Science and Fluid Department. 36307-352. Brazil
e-mail: gvilalta@ufsj.edu.br

Jayme P. Ortiz
São Paulo University, Mechanical Engineering Department, 05508-010, São Paulo, Brazil.
e-mail: jportiz@usp.br

José Alberto Vilalta
Polytechnical University of Havana,Industrial Engineering Department. 19340. Havana-Cuba
e-mail: jvilalta@ind.cujae.edu.cu

Carlos Vaquero
University and Clinical Hospital of Valladolid, 47011. Spain.
e-mail: cvaquero@med.uva.es

Abstract. *Nowadays, it is recognized that current clinical criteria to AAA rupture risk prediction are not entirely reliable. Hence the need to find some biomechanical predictors to improve this prediction. The physical principle of aneurysm rupture is a manifestation of the balance between the arterial wall strength and the forces generated by blood flow inside the aneurysm. Therefore the hemodynamics may predispose aneurysms to rupture. Aim of this study was to assess the hemodynamic features associated with aneurysm real lumen geometry and its influence on AAA rupture risk. Three patient-specific AAAs were selected and 3D reconstruction and geometric characterization (maximum diameter, length and asymmetry) were performed. The AAA was modeled using volume element technique which allows to solve fluid conservation equations. Blood was modeled as laminar, Newtonian with properties as typically used in the literature. The wall was assumed rigid. The results provide quantitative and qualitative predictions of the flow patterns influence on the hemodynamic stress-HS (pressure + wall shear stress) distribution related to geometric parameters of real lumen. The flow patterns are dominated by recirculating regions inside the aneurysmatic sac whose structure and intensity depends on the cardiac cycle phase. Due to this behavior, most of the AAA lumen wall surface is exposed to low flow-induced HS. Locally elevated HS are located close to the distal neck and posterior wall. The HS increases with diameter and asymmetry and decreases with length. One of the AAA model presents high rupture risk because its HS is lightly lower than the peak wall stress (PWS) value recognized in the literature for the rupture. Up to end of the study, no AAA rupture occurred. Hemodynamic stresses may play an important role in determining a more realistic and accurate biomechanical determinants for improving the AAA rupture risk prediction.*

Keywords: AAA, Rupture Risk, Prediction, Pearson´s correlation

1. INTRODUCTION

The abdominal aorta is the largest artery in the abdominal cavity and carries blood from heart and abdomen to all vital organs. A common disease of the abdominal aorta, is the abdominal aortic aneurysm (AAA), which involves a focal dilatation of this artery with an increase of the vessel diameter exceeding the non-dilated adjacent aorta by more than 50%. The AAA has increasingly been recognized as an important health problem in the last decade because of its statistic are of great concern.

The lack of an accurate AAA rupture risk index remains an important problem in the clinical management of the disease. Nowadays, it is recognized that current clinical criteria to AAA rupture risk prediction are not entirely reliable. The main clinical criteria in deciding on the treatment of AAA patients are: a) the peak transverse diameter and b) the growth rate. If the peak diameter reaches the upper threshold (5-5.5 cm) or the maximum diameter expansion rate is > 0.5 cm/yr for smaller AAAs the patient may be submitted for surgical intervention, also depending on the state of health and willingness of the patients. The main limitation of this practice is that these criteria, although have a significant empirical basis, can be considered insufficient because they have not a physically sound theoretical basis. Hence the need to find some biomechanical predictors to improve this prediction.

The physical principle of aneurysm rupture is a manifestation of the balance between the arterial wall strength and the forces generated by blood flow inside the aneurysm. Therefore the hemodynamics may predispose aneurysms to rupture playing an important role.

ISSN 2176-5480

Aim of this study was to assess the hemodynamics features associated with of aneurysm real lumen geometry and its influence on AAA rupture risk.

2. MATERIALS AND METHODS

2.1 AAA geometry

Three patient-specific AAAs were selected and 3D reconstruction and geometric characterization (maximum diameter, length and asymmetry) were performed.

For all three cases in the TAC screening was used contrast agent to obtain a good definition of lumen. Using the VMTK(Vascular Modeling Toolkit) an open source software it was possible to realize the segmentation of the 3 packs of images, using automatic segmentation based in Level Set techniques. Then the surfaces were processed by a Fast Marching Cubes algorithm to generate the volume, necessary for the flow field computations. To realize the geometric characterization it was necessary to generate the centerline of the 3 images, then using some module of own development was possible to export in external files all the data which correspond to each point of centerline. The result obtained representing the aneurysmatic sac is shown in Fig. 1.

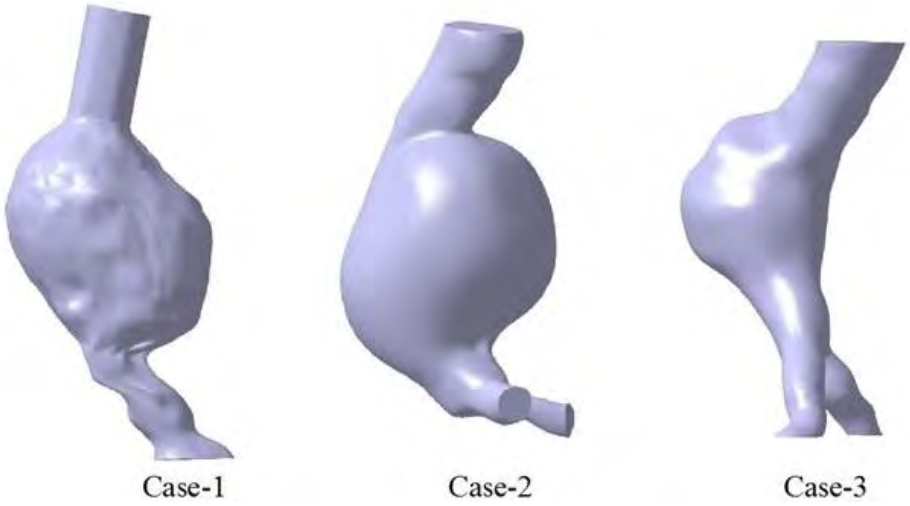

Figure 1. 3D reconstructed aneurysmatic sac geometry extracted from individual set of CT images.

The AAAs geometric characterization is summarized in Table 1, where D is the peak diameter, d is the non-deformed aorta diameter, L is the aneurysm length which is measured from the proximal neck to the distal neck and β is the asymmetry coefficient.

Table 1. Geometric characterization of AAAs.

Case	D (mm)	d (mm)	L (mm)	β (-)
1	45.3	15.9	55.1	0.57
2	33.1	19.4	52.5	0.39
3	39.6	25.4	62.5	0.02

2.2 Governing equation and Boundary conditions

AAAs were modeled using volume finite technique (Fluent v.14) which allow to solve the typical fluid conservation equations: the Navier-Stokes and Continuity. The assumptions of incompressible, laminar, homogeneous and Newtonian fluid with average properties (density of 1050 kg/m^3 and dynamic viscosity coefficient of 4x10^{-3} Pa.s) were used in this work. The external surface of the lumen, that represents the inner layer of the arterial wall, was modeled as rigid and impermeable. For an incompressible flow in the absence of body force the conservation are expressed in compact form as follows:

$$\nabla \cdot \boldsymbol{V} = 0 \tag{1}$$

$$\rho \frac{d\boldsymbol{V}}{dt} = -\nabla \boldsymbol{p} + \mu \nabla^2 \mathbf{V} \tag{2}$$

ISSN 2176-5480

where $\boldsymbol{V}$ is the velocity vector whose rectangular components are represented by (u;v;w), μ is the dynamic viscosity coefficient and $\boldsymbol{p}$ is the pressure.

The boundary conditions for the velocity $\boldsymbol{V}$ are given by equations 3.1 and 3.2. These are: at the inlet boundary a fully developed parabolic profile (3.1) with no-slip at the walls (3.2). Time dependent normal traction due to luminal pressure at the outlet (3.3) is the boundary condition . These conditions are expressed, in mathematical form, as follows:

$$u = 2\big(u(t)\big)\left(1 - \left(\frac{2r}{dr}\right)^2\right);\ v = 0|_{z=0} \tag{3.1}$$

$$u = 0|_{wall} \tag{3.2}$$

$$\tau_{nn} = \hat{n} \cdot p(t) I \cdot \hat{n} \tag{3.3}$$

Figure 2 shows the volumetric flow rate and pressure pulsatile waveforms used in the present study, which are based on in vivo measurements of the human aorta in the aortic segment.

As it was specified, these are a triphasic pulses appropriated for normal hemodynamic conditions in the abdominal segment of the human aorta as early reported by Mill et al. (1970) (*apud* Finol et al. 2003). In both waveforms, the cycle period are 1 s, with peak flow occurring at 0,304 s and peak pressure occurring at 0,5 s. The time-average Reynolds number Rem≈300 is acceptable for the conditions of the simulation, according to the results reported by Pedersen et al. (1993). The Womersley number which characterizes the flow frequency, the geometry and the viscous fluid properties is 12,2 what is a typical value for the aortic segment. These numbers are defined using the following characteristic magnitudes: mean velocity and inlet boundary diameter, corresponding to the non-deformed aorta.

Because the inlet boundary condition is applied over the proximal neck of the aneurysm, a non-dilated segment of the aorta, the use of an input transient pulse based on normal physiology conditions is justified.

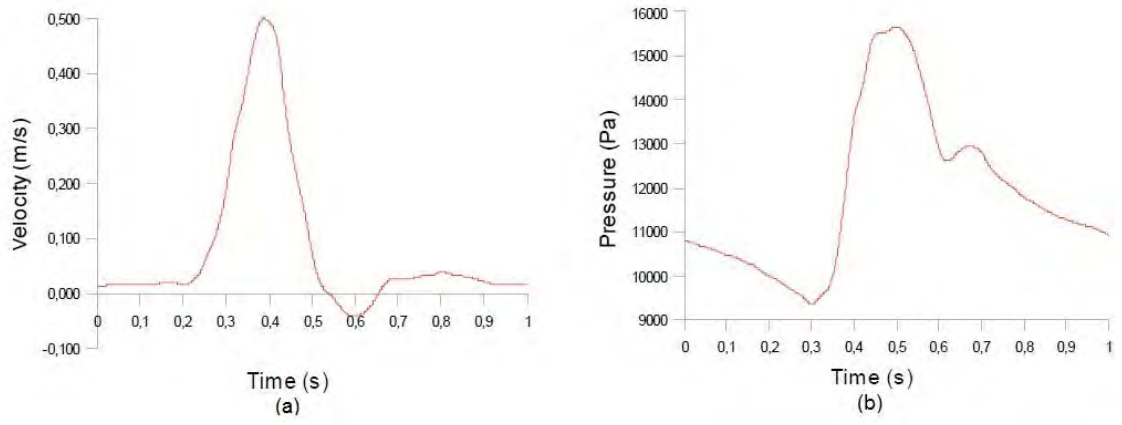

Figure 2. In vivo luminal pulsatile (a) velocity waveform and (b) pressure waveform reproduced from Mills et al. (1970). Inlet peak systolic flow occurs at t = 0,4 s and outlet peak systolic pressure at t = 0.5 s.

3. RESULTS AND DISCUSSION

The prevailing aetiology hypothesis is that AAA results from the coupling between structural changes in inner layers of the arterial wall and disturbed patterns of hemodynamics stresses acting on the vessel wall. As presented in previous works (Salsac et al, 2006; Scotti and Finol, 2007), it has been shown that the so-called disturbed flow conditions, that develop within the AAA, such as rapid decrease in the velocity and regions of very high (or low) hemodynamic stresses gradients, may all contribute in various ways to the vascular disease, primarily via their effects on the endothelium.

According to velocity and pressure waveform prescribed as boundary conditions, at peak systolic pressure the velocity profile is in its late systolic phase and thus the blood at the inlet has begun to decelerate. Consequently, recirculating regions develop establishing local and temporal hemodynamic stresses distribution.

The results here obtained provide quantitative and qualitative predictions of the flow patterns influence on the hemodynamic stress-HS (pressure + wall shear stress) distribution related with main geometric parameters of real lumen (Vilalta et al, 2012a).

An important feature observed in the vortical structure is that the blood field is dominated by the presence of vortices in the region close to the aneurysmatic sac wall. From a complete analysis of cardiac cycle (6 cycles), it is observed that the residual vortices left from the previous cycle (t=0s) are present in the aneurysm up to t=0.2s,

ISSN 2176-5480

approximately. At this interval of time, there are two low velocity vortices in the distal end of the aneurysm, both anterior and posterior wall. This results in one toroid-shaped vortex that loop around the core flow with a shape like a ring.

When systolic acceleration occurs (*t* between 0,2 and 0,38s), an increase of the recirculating region is verified occupying almost all the aneurysmatic sac. This vortices distribution is defined by the fluid jet entrance to expansion aneurysmatic. At the middle stage of systole, a strong pressure gradient (15,6 kPa) provokes the ejection of the vortices downstream, as the flow accelerates over time. In these conditions, the flow reattaches to the wall. Typical symmetric flow patterns and streamlined profile absent of vortices occurs in the aneurysmatic sac during the peak flow phase, where also occur the maximum velocities and velocities gradients.

At the end stage of the systole (*t*=0,40-0,50s) a high velocity jet is submitted to significant hemodynamics disturbance, which begins in the proximal end at t=0,42s. At t=0,5s, the symmetric ring vortex is shed off the wall at the point of flow separation near to proximal end while at the distal end and the aneurism center there is a forward flow pattern. This description has also been reported in Finol et al, 2003.

In the period of flow reversal (t=0,5-0,55s), a translation of the vortex center towards the centerline occurs, because of the decreasing of the intensity of recirculating flow, which result in an extension of the wake of the vortex into the inlet section of the aneurysm.

Finally, at the beginning of the diastole, occurs the translation of this vortex towards the distal end an intensive and axisymmetric recirculation is present close to the aneurysm distal end and also a negative velocity appears along anterior wall.

The time dependent flow field computation showed that most of the AAA lumen wall surface is exposed to low flow-induced HS. This behavior is reflected in the HS distribution. Figure 3 and Figure 4 show the regional distribution of wall shear stress (normalized with Poiseuille flow for control model) and pressure, respectively. Locally elevated HS were located close to the distal neck and posterior wall.

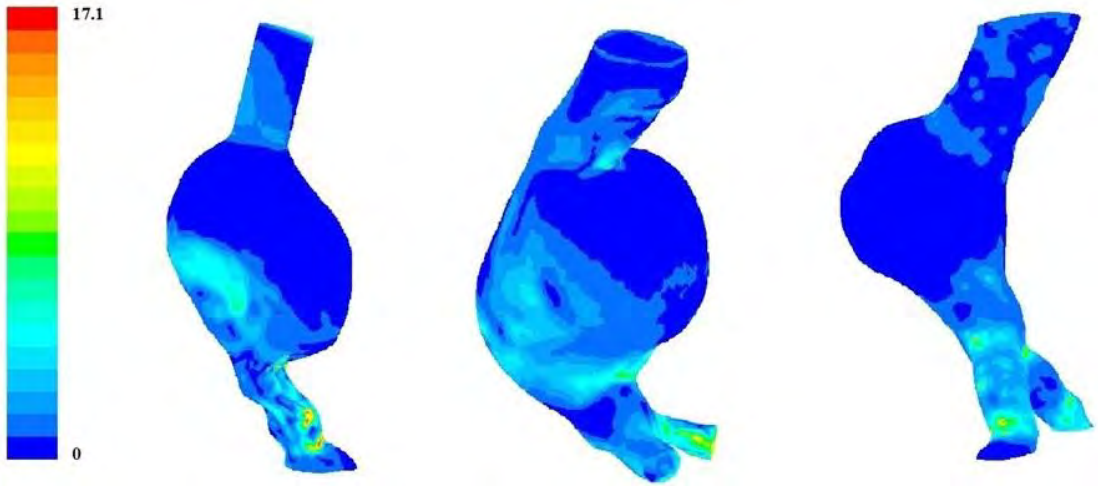

Figure 3. Normalized wall shear distribution during peak systolic.

The hemodynmics stress increases with diameter and asymmetry and decreases with length. None of the AAA models are in risk of rupture, because the peak HS values are lower than the peak wall stress value recognized in the literature for the rupture.

The hemodynamics also plays a significant role in both the biological and mechanical factors that exist within AAAs. For example, fluid flow has been correlated with changes at the cellular level (Vilalta et al, 2012b), and increase in wall stress, reduced nutrient supply to the wall, and the development or stabilization of intra-luminal thrombus.

In general, this behavior may cause degenerative lesions of aneurysmal wall, altering the wall thickness and could eventually cause rupture.

4. CONCLUSIONS

The influence of the hemodynamic features on the rupture risk of fusiform AAA was investigated. The main conclusions drawn from present work are:

1. The flow patterns are dominated by recirculating regions inside the aneurysmatic sac whose structure and intensity depend of the cardiac cycle phase. The time dependent flow field computation showed that most of the AAA lumen wall surface is exposed to low flow-induced HS.

2. The HS increases with diameter and asymmetry and decreases with length.

ISSN 2176-5480

3. Hemodynamic stresses may play an important role in determining a more realistic and accurate biomechanical determinants for improving the AAA rupture risk prediction.

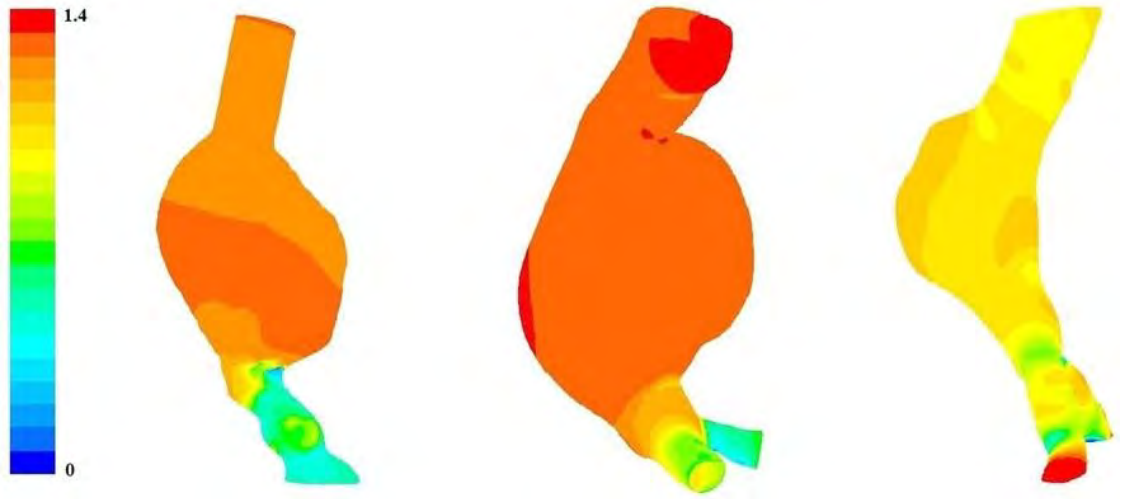

Figure 4. Pressure distribution on AAA wall during peak systolic.

5. ACKNOWLEDGEMENTS

This The authors gratefully acknowledge the financial support of the Ministerio de Ciencia e Innovación-Spain, grant PTQ06-2-0218, and the Junta de Castilla y León-Spain through ADE Investments and Services, grant Advanced Simulation of Deformable Systems II.

.

6. REFERENCES

Finol, E.A., Keyhani, K., Amon, C.H. (2003) "The effect of Asymmetry in Abdominal Aortic Aneurysm Under Physiologically Realistic Pulsatile Flow Conditions". J. Biomechanical Engineering. 125(2), pp.207-217.

Pedersen, E., Sung, H., Burlson, A., Yoganathan, A. (1993) "Two-dimensional Velocity Measurement in a Pulsatile Flow Model of the Normal Abdominal Aneurysm Simulating Different Hemodynamics Conditions". J. Biomech., 26: 1237-1247.

Salsac, A.V., Sparks, S.R., Chomaz, J.M., Lasheras, J.C. (2006;) "Evolution of the wall shear stresses during the progressive enlargement of symmetric abdominal aortic aneurysms" J. Fluid. Mech. 560, pp.19-51.

Scotti CM, Fino EA. (2007) "Compliant biomechanics of abdominal aortic aneurysm: A fluid-structural interaction study. Computer and Structures.; 85: 1097-1113.

Vilalta, G., Soudah, E., Vilalta, J.A., Nieto, F., (2012a) " Hemodynamic features associated with abdominal aortic aneurysm (AAA) geometry". Proceedings 18th Congress of the European Society of Biomechanics. July 1-4; Lisbon Portugal.

Vilalta, G., Nieto, F., San Norberto, E., Pérez, M.Á., Vilalta. J.A., Vaquero, C. (2012b) "Biomechanical approach to improve the abdominal aortic aneurysm rupture risk". http://dx.doi.org/10.5772/46030. In Yasuo Murai editor. Aneurysm. Croatia; pp.77-108.

7. RESPONSIBILITY NOTICE

The authors are the only responsible for the printed material included in this paper.