QUEST FOR THE LINK BETWEEN THE DYNAMIC BEHAVIOUR OF AN ABDOMINAL AORTIC ANEURYSM AND BLOOD HAEMATOCRIT: FLUID-STRUCTURE INTERACTION STUDY

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ABSTRACT

The Abdominal Aortic Aneurysm (AAA) is a local dilation of the abdominal aorta and it is a cause for serious concern because of the high mortality associated with its rupture. The understanding of the phenomena related to the creation and the progression of an AAA is of crucial importance. In this work the complicated interaction between the blood flow and the AAA wall is numerically examined using a fully coupled Fluid-Structure Interaction method. The study investigates the possible link between the dynamic behaviour of an AAA and the blood viscosity variations attributed to the haematocrit value, while it also incorporates the pulsatile blood flow, the non-Newtonian behaviour of blood and the hyperelasticity of the arterial wall. It was found that blood viscosity has no significant effect on von Mises stress magnitude and distribution, whereas there is a close relation between the haematocrit value and the Wall Shear Stress (*WSS*) magnitude in AAAs. This *WSS* variation can possibly alter the mechanical properties of the arterial wall and affect its growth rate or even its rupture possibility.

INTRODUCTION

An Abdominal Aortic Aneurysm (AAA) is a cardiovascular abnormality that causes serious concern in a worldwide scale. An AAA is a local dilation of the abdominal aorta, usually over 3 cm in diameter and is mostly reported in men, aged 65 or older that are smokers. Little is known about the mechanism of AAA creation, so, understanding the phenomena related to the creation, progression and behaviour of an AAA is of crucial importance, since mortality in AAA patients appears significantly high. Determining AAA's rupture risk factors, or any factors that cause its appearance or its evolution, may lead to its prevention or possible cure. Risk factors for the development or rupture of AAAs may include multiple biochemical or mechanical processes occurring in parts of the aortic wall. As a matter of fact, the current study investigates the possible link between the changes in blood viscosity due to haematocrit (the volumetric percentage of red blood cells in blood, H_t) variations and the dynamic behaviour of an AAA.

The complicated interaction between the blood flow and the AAA wall is numerically examined by using a fully coupled Fluid-Structure Interaction (*FSI*) method. It is common place that coupling the fluid dynamics component of the simulation with the solid domain simulation is essential to reach more representative results of the overall AAA behaviour [1].

Wall Shear Stress (*WSS*) should also be considered, when studying AAAs, as a significant parameter that can alter the arterial wall properties [2]. These alterations may greatly affect the AAA wall as they can reduce the wall's resistance and accelerate its rupture [3]. When simulating blood flows, the importance of a pulsatile boundary condition for the blood flow is proved [4] to play key part in subsequent results. Previous research reveals that the non-Newtonian nature of blood shall not be ignored as it plays a great role in various characteristics of the flow, predominantly *WSS* [5]. The present study uses a non-Newtonian model for blood, i.e. the Casson model [6] that integrates simultaneously, through the viscosity modelling, the haematocrit dependency. This study incorporates all the above issues to investigate the possible link between the dynamic behaviour of an abdominal aortic aneurysm (AAA) and the blood viscosity variations due to different haematocrit values. This

is accomplished by using simulation tools that implement an *FSI* method. Specifically, for a typical range of haematocrit values we will numerically estimate the values of wall displacement, the von Mises stress of the AAA wall and the *WSS*.

METHODOLOGY

Multiphysics simulation is a crucial tool, which attempts to accurately predict complex phenomena that will occur where multiple types of coupled physics interact. It is a well-established method in product engineering, as it drives development processes and can influence engineering simulation efforts. One type of multiphysics problems is the *FSI* which occurs when a fluid interacts with a solid structure causing its deformation and, as a result, altering the fluid flow itself [7]. As a matter of fact the study of an AAA is a pure *FSI* problem.

The AAA studied is positioned just below the renal bifurcation. A simplified geometry was selected as a benchmark to emulate a real-life AAA for this current parametric and methodological study. The non-affected part of the model was assumed to have a diameter of 20 mm [8]. The aortic sac was modelled asymmetrically to match representative abnormalities. The maximum aneurismal diameter was set to 55 mm, which falls in the lower threshold zone for high rupture risk and subsequently is a candidate for surgical treatment [9]. The total length of the geometry designed is *L*= 90 mm. ANSYS DesignModeler® was used to create a simplified AAA model (Figure 1). The dimensions of the simplified AAA model are shown in Table 1.

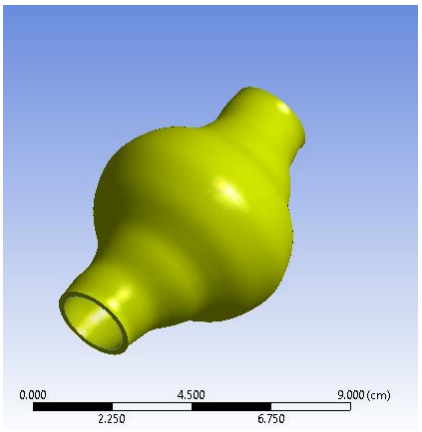

Figure 1. *View of the AAA wall.*

Table 1. *Geometrical parameters of the AAA model.*

Parameter	Value
Total length of the AAA, *L*	90 mm
Internal inlet diameter of the AAA, D_i	20 mm
Maximum aneurismal diameter , D_{max}	55 mm
Arterial wall thickness, *k*	2 mm

The governing equations for the fluid flow are the Navier-Stokes equations for incompressible non-Newtonian flow. The momentum equations (Equation 1) are expressed in the Arbitrary Lagrangian-Eulerian form (ALE) for the fluid domain [10]:

$$\rho_b \frac{\partial u}{\partial t} + \rho_b \left((u - \dot{d}_b) \cdot \nabla \right) u - \nabla \cdot \tau_b = f_b \quad (1)$$

where $\dot{d}_b$ is the velocity vector of the moving mesh interface, ρ_b is the blood density, 1050 kg/m^3, and f_b the body forces per unit volume. The fluid stress tensor (τ_b) is defined in Equation 2:

$$\tau_b = -P\boldsymbol{I} + 2\mu \boldsymbol{D}(\boldsymbol{u}) \quad (2)$$

where $D(u)$ is the strain rate tensor and μ the dynamic viscosity.

Equation 2 can be rewritten as follows:

$$\tau_b = -P\boldsymbol{I} + 2\mu(\dot{\gamma}) \boldsymbol{D}(\boldsymbol{u}) \quad (3)$$

where $\dot{\gamma}$ is the shear rate. Amongst the various proposed models for relating blood viscosity and shear rate [6] the Casson model was preferred since its constants can be expressed as a function of haematocrit, H_t. The viscosity of blood, $\mu(\dot{\gamma})$, is modelled via Equation 4 [11]:

$$\mu = \left(\sqrt{\frac{\tau_y}{\gamma}} + \sqrt{\mu_\infty} \right)^2 \quad (4)$$

where τ_y is the yield stress and μ_∞ is the asymptotic viscosity, characteristic for high $\dot{\gamma}$ values. The yield stress is a measure of the blood resistance to the flow at very low $\dot{\gamma}$ values [6]. A proposed model, which relates the terms of Equation 4 with the H_t value [6], is used in this study.

For the solid domain, the governing equation follows the movement of the solid material on a moving coordinate system. The solid elastodynamics are described by Equation 5:

$$\nabla \cdot \tau_W + f_W = \rho_W \ddot{d_W} \quad (5)$$

where τ_W is the stress tensor on the arterial wall, f_W , the arterial wall force per unit volume, ρ_W, the arterial wall density, 2000 $\frac{kg}{m^3}$, and $\ddot{d_W}$, the arterial wall local acceleration. The model used to describe the arterial wall properties was the 2nd order Mooney-Rivlin model [12] that perceives the arterial wall as a nonlinear, isotropic and hyperelastic with constant values: C_1=17.4 N/cm^2, C_2=188.1 N/cm^2 were obtained from Raghavan & Vorp [13].

For boundary conditions, a pulsatile inlet and a corresponding pulsatile pressure profile for the outlet (Figure 2) were reproduced from literature [14].

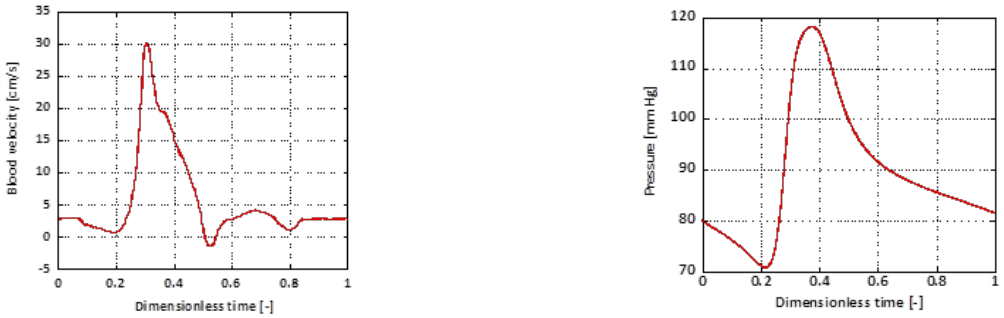

Figure 2. *Inlet boundary condition (Left) and Outlet boundary condition (Right).*

The heart rate period was T=1 s. The solid domain was fixed at the inlet and the outlet. On the outer surface of the arterial wall the relative pressure was set to zero. For the fluid part, the interface was designated as a no-slip wall. The interaction on the surface is described by Equations 6-8:

$$d_W = d_b \quad (6)$$

$$\boldsymbol{n} \cdot \sigma_W = \boldsymbol{n} \cdot \sigma_b \quad (7)$$

$$\dot{d_W} = \dot{d_b} \quad (8)$$

where d is the displacement for each domain and $\boldsymbol{n}$ is the normal vector of the corresponding surface, along with the corresponding subscripts *w*, for the arterial wall and *b* for the blood flow.

The coupled *FSI* simulations were executed by a commercial *CFD* code, the ANSYS Workbench® software (v. 19, ANSYS Inc.). The fluid domain was solved by ANSYS CFX® and the solid domain was solved by ANSYS Mechanical®. The coupling was performed by the ANSYS Workbench® coupling component. Hexahedral elements were used for the discretization of the fluid domain, while the solid domain was discretized using elements suitable for hyperelastic modelling in a one-layer layout. Optimum grid density for the fluid domain was selected by performing a grid dependency study, resulting in 402,800 cells for the fluid domain. The finite volume method and a fully coupled solver for the pressure and velocity, provided by ANSYS CFX®, were used for the fluid domain. The number of iterations ensure that the mass and momentum residual, as well as the data transfer residuals between the two FSI components reach adequate values. The *DNS* method for laminar flow was employed for the solution, as the flow is laminar. The physical time step (Δt) was set at Δt=0.5 ms. Several H_t values were tested, whereas results of two extreme values (30%, 50%) are presented and discussed.

RESULTS

The *FSI* simulations provide both qualitative and quantitative results that predict the blood flow patterns in the AAA (Figure 3). The temporal variation of blood velocity in the aneurysm follows the inlet boundary velocity profile. Reverse flow and recirculation zones are present during a full pulse.

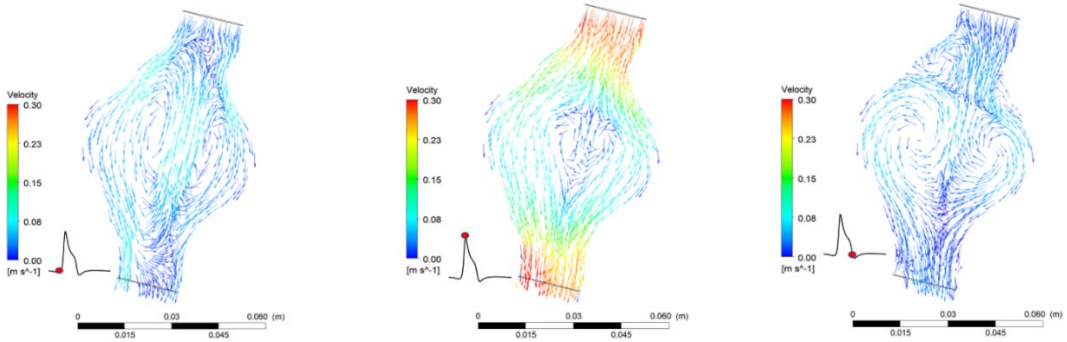

Figure 3. *Flow patterns in the AAA during one pulse, for H_t = 30%.*

Results revealed that total displacement of the aneurismal wall depends strongly on location. Namely, displacement is bigger near the areas where diameter variations are more pronounced. Maximum displacement during one pulse occurs when *t/T*= 0.373. No differences appear for the total wall displacement, when the results between the two H_t cases are being compared. The maximum displacement is 3.90 mm for both cases.

As expected, there is a strong correlation between the arterial wall displacement and the distribution of von Mises stress. Von Mises stress is a value used to predict yielding of materials under loading and it can be represented in an expression using different components of a stress tensor. Von Mises stress, σ_{vM}, can be calculated by Equation 9 [15]:

$$\sigma_{vM} = \sqrt{\frac{1}{2}[(\sigma_1 - \sigma_2)^2 + (\sigma_2 - \sigma_3)^2 + (\sigma_3 - \sigma_1)^2]} \quad (9)$$

where σ_1, σ_2 and σ_3 are the principal stresses in three-dimensional problems.

Peak σ_{vM} values appear at the area of maximum deformation of the arterial wall (Figure 4). Maximum values for both cases occur at *t/T* = 0.373. It was again revealed that, for the two H_t values tested, the σ_{vM} magnitude is not affected. For both H_t=30% and H_t= 50% the peak σ_{vM} value was 219.4 kPa.

WSS values vary significantly between the two cases tested and are influenced by both the location in the AAA and time. It is also found that *WSS* depends strongly on H_t, as an increase in H_t causes an increase in calculated peak *WSS* values. The results show that H_t greatly affects *WSS* magnitude. In non-affected areas (i.e. near the AAA endings), *WSS* attains normal values (>0.7 Pa) [2].

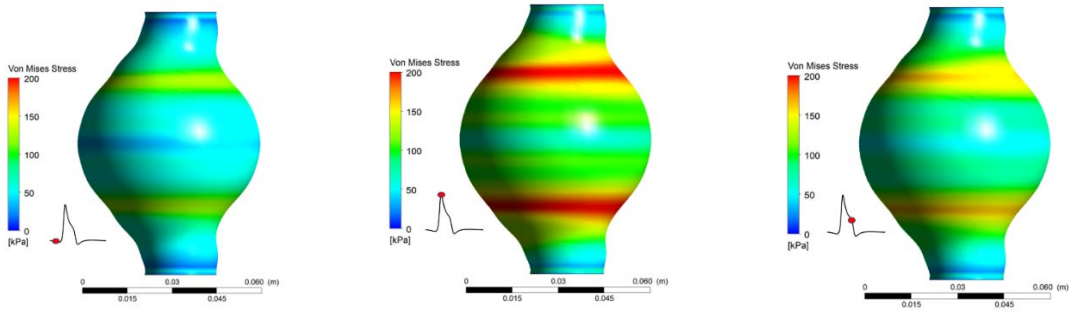

Figure 4*. von Mises Stress distribution during one pulse for H_t = 30%.*

It is reported [2] that *WSS* values less than 0.4 Pa (Figure 5) can result into plaque build-up in the arterial wall. So, assessing the *WSS* values on the AAA could provide valuable information.

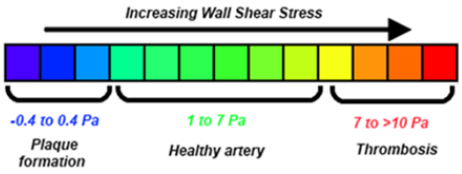

Figure 5. *Variations of WSS in human arteries* [2].

Indeed, comparing the area averaged *WSS* values for the two H_t results in a major difference in *WSS* magnitude (Figure 6). For the lower H_t value the average *WSS* does not climb over 0.3 Pa during the whole pulse cycle, whereas for H_t=50%, *WSS* appears remarkably higher. Thus, a nearly 40% decrease in H_t can probably cause as much as 70% decrease in *WSS* values.

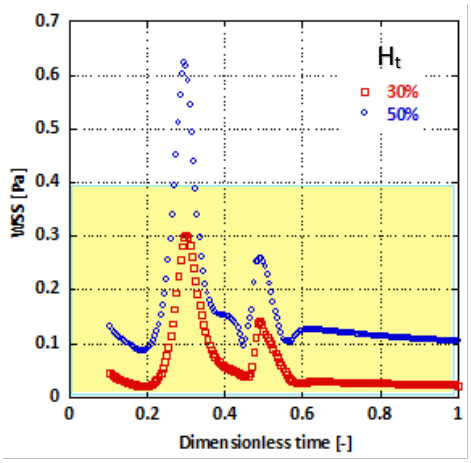

Figure 6. *Effect of H_t on the average WSS*

DISCUSSION

This study proposes a possible link between low H_t values with low *WSS* values in AAAs. This can cause serious abnormalities in the arterial wall's physical and mechanical properties [16], resulting in an increase of AAA's growth rate or rise in its rupture risk. The negative outcome of low *WSS* values on the arterial wall is a result of complex mechanical and biochemical phenomena, possibly linked to atherosclerosis [17]. O'Leary et al. [18] imply causal relation between plaque affected areas and high rupture risk in AAAs. This interaction might cause the adverse clinical situation of patients suffering from AAAs while having lower than normal H_t [19].

This hypothesis is widely accepted in literature, namely, the mechanism of generation and progression of AAAs and even its rupture risk can be linked with low-*WSS* induced alterations of the arterial wall [16,20,21].

Wang & Li [22] examined the influence of blood viscosity in AAAs and their conclusions agree qualitatively with the present study. Overall this study presents results that agree with previous works. Namely, the von Mises stress values, *WSS* in the aneurismal sac and displacement values, coincide qualitatively with previous computational research [1] as well as clinical observations [23].

CONCLUDING REMARKS

This work provides a qualitative insight on the way haematocrit could affect AAA's mechanics and haemodynamics, considering the pulsatile blood flow, the non-Newtonian behaviour of the blood and the hyperelasticity of the arterial wall. It was revealed that the variation in blood viscosity does not have a significant effect on AAA's wall solid dynamics as well as on σ_{vM} patterns, whereas a strong relation between H_t value and the shear stress acting on the arterial wall seems to exist. Lower H_t values result in lower viscosity values and consequently in lower *WSS* values, which in turn affect the aneurismal wall properties (Figure 7). This could be one of the causal paths describing the

effect of low haematocrit values on AAAs morbidity. Undoubtedly, further research is needed in the investigation of the link between H_t and the behaviour of AAAs.

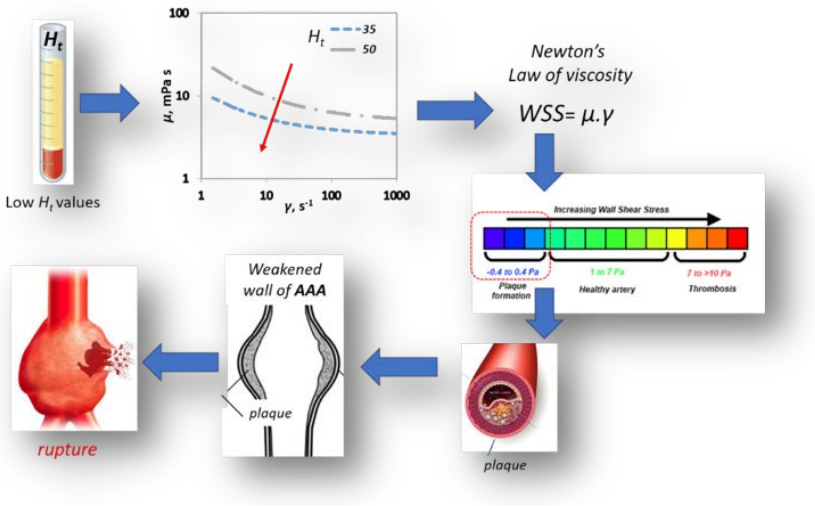

Figure 7. *Effect of H_t on AAA rupture.*

REFERENCES

[1] C.M. Scotti, J. Jimenez, S.C. Muluk, E.A. Finol, Comput. Methods Biomech. Biomed. Engin. 11 (2008) 301–322.
[2] A.M. Malek, S.L. Alper, S. Izumo, JAMA 282 (1999) 2035–2042.
[3] M. Xenos, S.H. Rambhia, Y. Alemu, S. Einav, N. Labropoulos, A. Tassiopoulos, J.J. Ricotta, D. Bluestein, Ann. Biomed. Eng. 38 (2010) 3323–3337.
[4] E.A. Finol, C.H. Amon, J. Biomech. Eng. 123 (2001) 474–484.
[5] A.G. Kanaris, A.D. Anastasiou, S.V. Paras, Chem. Eng. Sci. 71 (2012) 202–211.
[6] E.W. Errill, Physiol. Rev. 49 (1969) 863–888.
[7] Y. Bazilevs, K. Takizawa, T.E. Tezduyar, Computational Fluid-Structure Interaction: Methods and Applications, John Wiley & Sons, 2013.
[8] M.N. Syed, M.M. Ahmad, M.N. Ahmad, S. Hussaini, M.N. Muhammad, S.H.A. Pir, B.K. Khandheria, A.J. Tajik, K.A. Ammar, J. Am. Coll. Cardiol. 69 (2017) 2075.
[9] P.M. Brown, D.T. Zelt, B. Sobolev, J. Vasc. Surg. 37 (2003) 280–284.
[10] Y. Mesri, H. Niazmand, A. Deyranlou, M.R. Sadeghi, Int. J. Mod. Phys. C 26 (2014) 1550038.
[11] R.L. Fournier, Basic Transport Phenomena in Biomedical Engineering,Third Edition, CRC Press, 2011.
[12] M. Mooney, J. Appl. Phys. 11 (1940) 582–592.
[13] M.L. Raghavan, D.A. Vorp, J. Biomech. 33 (2000) 475–482.
[14] C.J. Mills, I.T. Gabe, J.H. Gault, D.T. Mason, J. Ross, E. Braunwald, J.P. Shillingford, Cardiovasc. Res. 4 (1970) 405–417.
[15] R. von Mises, Nachrichten Von Ges. Wiss. Zu Gött. Math.-Phys. Kl. 1913 (1913) 582–592.
[16] A.J. Boyd, D.C.S. Kuhn, R.J. Lozowy, G.P. Kulbisky, J. Vasc. Surg. 63 (2016) 1613–1619.
[17] A.M. Shaaban, A.J. Duerinckx, AJR Am. J. Roentgenol. 174 (2000) 1657–1665.
[18] S.A. O'Leary, J.J. Mulvihill, H.E. Barrett, E.G. Kavanagh, M.T. Walsh, T.M. McGloughlin, B.J. Doyle, J. Mech. Behav. Biomed. Mater. 42 (2015) 154–167.
[19] N. Diehm, J.F. Benenati, G.J. Becker, R. Quesada, A.I. Tsoukas, B.T. Katzen, M. Kovacs, J. Vasc. Surg. 46 (2007) 676–681.
[20] Reed D, Reed C, Stemmermann G, Hayashi T, Circulation 85 (1992) 205–211.
[21] C. Xu, C.K. Zarins, S. Glagov, J. Vasc. Surg. 33 (2001) 91–96.
[22] X. Wang, X. Li, Comput. Biol. Med. 41 (2011) 812–821.
[23] L.M. de Heer, R.P.J. Budde, W.P.T.M. Mali, A.M. de Vos, L.A. van Herwerden, J. Kluin, Int. J. Cardiovasc. Imaging 27 (2011) 1195–1204.