CFD MODEL OF FLOW HEMODYNAMICS IN ABDOMINAL AORTIC ANEURYSM

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Abstract: The paper deals with analysis of the hemodynamic factors (static pressure, wall shear stress (WSS)) that affect the growth of aortic aneurysm. A CFD model developed for turbulent blood flow and transient pressure at the outlet of the aorta was solved in real geometry reconstructed on the basis of CT images. USG analysis was applied to determine transient blood flow velocity, fix boundary and initial conditions and verify the model. Analysis of the results shows that WSS distribution changes significantly both along and around a cross-section of aorta wall, while static pressure remains constant. Comparing CT images with WSS profiles around a cross-section of aorta wall, a correlation was found between the thickness of the intraluminal thrombus (ILT) and the value of WSS. Analysis of the calculations proves that despite a substantial difference between absolute values of static pressure and WSS, the effect of WSS on hemodynamic forces affecting the aorta wall cannot be omitted due to WSS contribution in thrombus development which changes both flow hemodynamics and mechanical properties of the wall.

Introduction

Recently, a growing number of patients with abdominal aortic aneurysm (AAA) and dramatic complications that accompany its rupture, require quick development of hemodynamic models of this vascular segment to identify places endangered with the rupture, aneurysm growth rate and predicted time of rupture.

Resistance of the aorta against forces related to pulsating blood flow depends on the composition and structure of the aorta wall. Elastic and collagen fibres are responsible for mechanical properties of the wall [1, 2]. In the medium layer of the aorta, elastin forms lamellae located concentrically around the vessel lumen, alternately with a layer of smooth muscle cells. Collagen fibres form, in the outer layer mainly, a mesh protecting against excessive stretching [3, 4]. A loss of elastic fibres in the aorta wall leads to its excessive stretching, weakening of collagen fibres and aneurysm formation [1, 2].

It is assumed that ca. 80% aneurysms grow steadily from 0.2 to 0.5 cm a year (about 10% per year), and in 20% above 0.5 cm annually [5]. The most frequently used prognostic factor is AAA diameter. Patients with aneurysms of diameter smaller than 4 cm are observed making control USG every 6 months. When the AAA diameter ranges between 5 and 6 cm, the risk increases greatly, so operation is recommended in most patients with aneurysm of diameter equal to 6 cm or bigger.

In natural evolution, the abdominal aortic aneurysm increases both crosswise and lengthwise, which finally results in its rupture. Most often the rupture is to retro-peritoneal space, rarely to free peritoneal cavity, inferior caval vein, duodenum or renal vein. In the moment when pressure between the ruptured aneurysm and extravasated blood is equalised, a thrombus is formed which hampers further extravasation.

Computational fluid dynamics (CFD) plays a growing role in studies on the effect of flow hemodynamics on processes that take place in blood vessels. The CFD models can be used, among the others, to calculate distributions of stresses that cause stretching of vascular wall segments and hydraulic resistance. Information on the distribution of stresses and pressures, i.e. hemodynamic forces that accompany blood flow is crucial to identify places endangered with vascular wall rupture.

Analysis of hemodynamic phenomena that occur in vascular segments require the application of models which reflect predominant hemodynamic features of vessel shape (aneurysm) and show compatibility of flow characteristic of the model with real flow characteristic in the segment.

Basing on a computer simulation it was shown that in the process of aneurysm rupture more important is its asymmetry (geometry) than size [5]. Both crosswise and lengthwise growth of aneurysm observed in clinical examinations proves that from the point of view of flow hemodynamics, shear stresses and pressure distribution contribute equally to aneurysm growth.

The paper is dedicated to analysis of hemodynamic factors (pressure and wall shear stress WSS) that have the most significant effect on the growth rate of aortic aneurysm.

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Medical data

Data necessary to develop a CFD model of flow hemodynamics in AAA were collected during standard medical monitoring procedures of patients suffering from aneurysm. Patients with a fast growth rate of aneurysm diameter were included into the program of additional clinical examinations (CT, USG-Doppler, angiography). The examinations enabled determination of hemodynamic parameters of blood vessels (linear dimensions, aneurysm volume increment, the amplitude of vascular wall oscillations, etc.) which are crucial for development of kinetic equations that control the aneurysm growth rate.

The reconstruction of blood flow domain geometry, the first and difficult step towards development of the CFD model, was performed on the basis of CT examination. Data necessary for determination of initial and boundary conditions as well as for verification of developed CFD model were collected from USG-Doppler examinations at several areas of abdominal aorta: below renal aorta (inlet), aneurysm neck, distal part, iliac arteries.

Flow geometry

In order to reconstruct the real geometry on the basis of computer tomograms (CT) we used our own software for 2D image processing, extraction of 3D geometry based on the series of cross sections of the reconstructed organ (aorta) and numerical mesh generation.

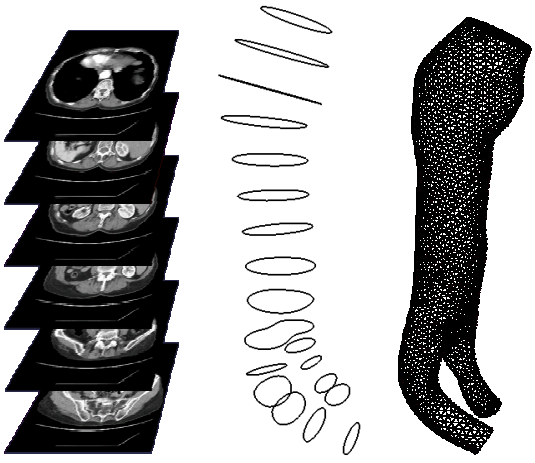

Figure 1: Flow geometry segmentation: a) CT images, b) abdominal aorta contours (flow area), c) finite elements mesh.

The segmentation method used in this paper has been presented in [6]. This approach allowed us to determine the geometry of the vessel using approximation of the vessel shape by a series of ellipses located in planes perpendicular to the vessel axis. A detailed description of the segmentation method was presented in [7]. The main advantage of the segmentation method applied is immunity to image artefacts resulting in a smooth surface of the vessel.

Flow geometry obtained in this way was recorded in a STL format (Stereo Lithography) and imported into a Gambit 2 numerical preprocessor (Fluent INC). A three-dimensional, tetrahedral volume mesh of flow domain was generated on the basis of STL geometry. Preliminary calculations performed to get the mesh independent solution show that 50,000 cells mesh size provides good accuracy of the calculations.

CFD Model

In numerical simulations a commercial CFD package Fluent 6.2 was used.

Blood flow in the cardiovascular system caused by cardiac contractions has pulsating character and for the aorta is usually in laminar and transient range.

To analyse the effect of pressure and shear stress on the aorta wall, a CFD model was developed for turbulent flow and transient pressure at the aorta outlet.

In the calculations Large Eddy Simulation (LES) model was applied [7].

A transient profile of outlet pressure was generated on the basis of systolic an diastolic pressures of a selected patient. To describe blood viscosity, Quemada's rheological (non-Newtonian) model was applied [8, 9]. Blood density was assumed constant and equal to 1040 kg/m^3 [10]. Calculations were performed in unsteady-state conditions.

Boundary conditions

Blood flow velocities in the areas of abdominal aorta described above, were taken from a USG-Doppler examination. After USG analysis, real transient blood flow velocity profiles were extracted and described by the Fourier series (Fig. 2). Twelve Fourier terms were considered to obtain satisfactory approximation of the velocity profile.

Data taken about 1 cm below the renal aorta were used to determine transient velocity profile at the inlet to the flow domain (inlet boundary condition). Data from aneurysm neck, distal part, iliac arteries of the aorta were used to verify CDF calculation results [7].

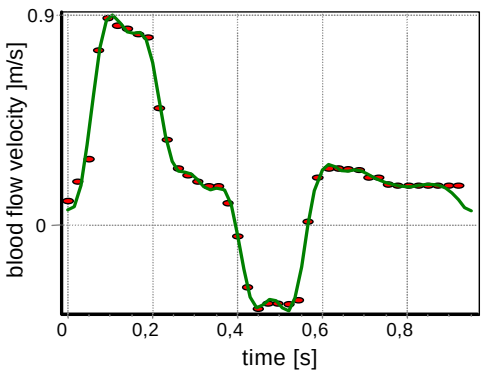

Figure 2: USG-Doppler data and Fourier series fit (12 terms).

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Results

Results of calculations are presented both in the form of 3D distributions and as 2D static pressure distributions and WSS in selected points along the aortic wall and around cross-sections of the aorta.

To analyse the effect of local unevenness of vascular wall caused by the presence of thrombus and pathological changes in aortic tissue on local values of static pressure and WSS. Fig. 3 shows a 3D distribution of static pressure (Fig. 3a) and wall shear stress (Fig. 3b) at systole. It follows from analysis of the Figure that in the regions where the highest pressures occur, we can observe the lowest WSS. The image of stress distribution observed in the Fig. 3 results from local wall unevenness and changes in the blood flow area.

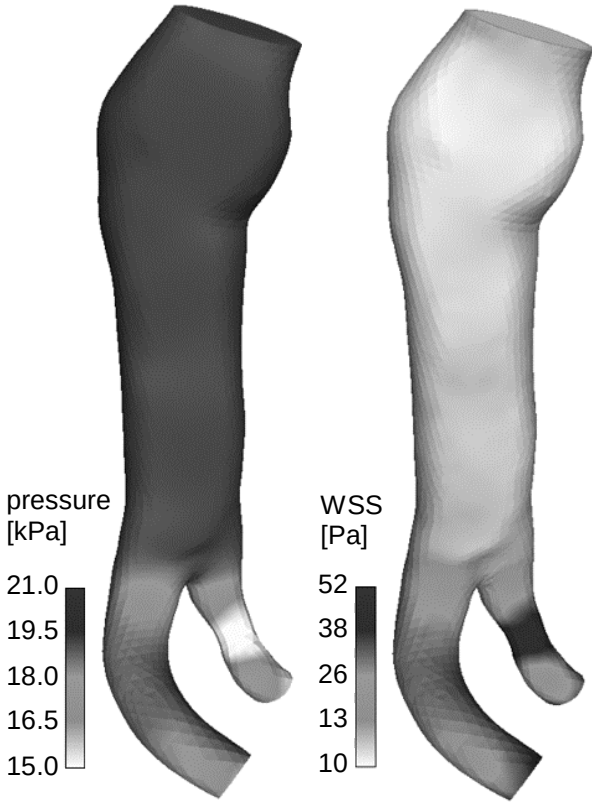

Figure 3: Pressure (a) and WSS (b) profiles at systole.

At the next stage, the distributions of pressure and WSS were analysed in six test points along the aorta and two points in iliac arteries (Fig. 4a) as well as in 4 cross-sections of the aorta (in the broadest aorta lumen I and stenosis II (see Fig. 4b)). For each cross-section the distribution of pressure and WSS transient profiles in six points along the aorta circumference were shown and compared.

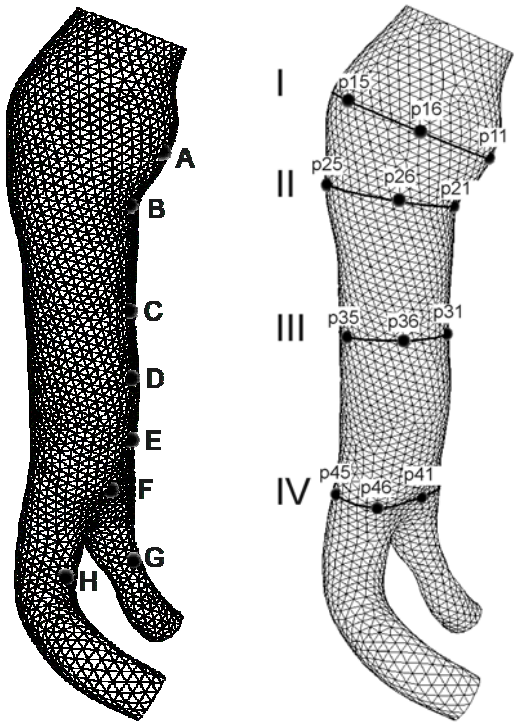

Figure 4: Probe points for simulation post-processing, a) pressure and WSS profiles along aorta wall, b) cross-sections for pressure and WSS distribution analysis.

Figures 5 and 6 show calculation of changes in pressure and WSS profiles along the aorta wall (see Fig. 4a).

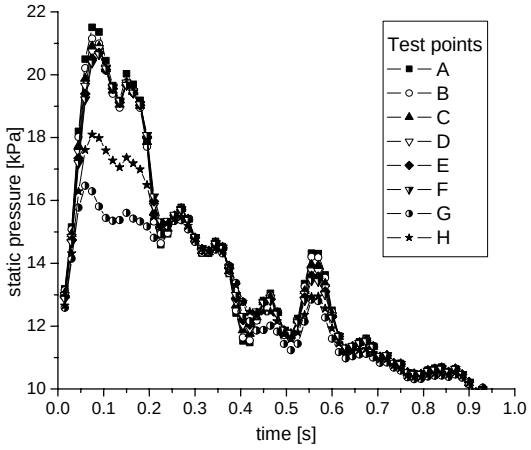

Figure 5: Transient pressure profiles at different points of abdominal aorta (see Fig. 4a)

Analysis of Fig. 5 shows that transient pressure profiles in the aorta are similar. Differences in pressure distribution observed in abdominal aorta result from small changes in cross-sections and resistance of flow and assume maximum values of about 1 kPa (e.g. at 0.1 s of heart cycle), i.e. ca. 5% of the maximum systolic pressure.

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A significant pressure decrease was observed in iliac arteries (points G and H – Fig. 4a) which was a result of a serious decrease of blood flow section.

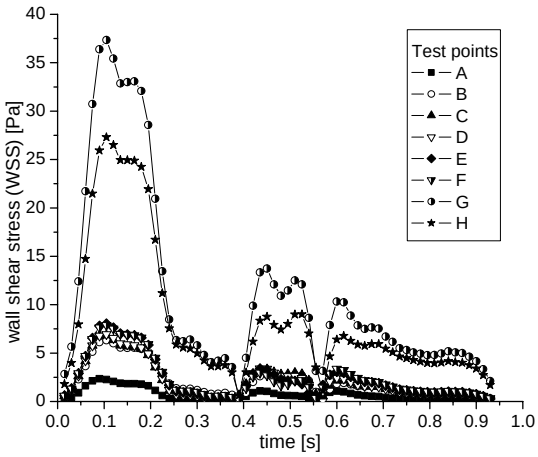

Figure 6: Transient wall shear stress (WSS) profiles at test points of aorta wall (see Fig. 4a)

Pressure changes from about 10 kPa to ca. 22 kPa (in iliac arteries from 18 to 16 kPa) in one cycle.

Shear stress distribution in the same heart cycle is shown in Fig. 6. Like in the case of pressure profiles, WSS distributions are similar except for the places of the broadest aorta lumen and in iliac arteries. Maximum values of WSS reach 10 Pa, and in iliac arteries 30 to 40 Pa, which is ca. 0.1% static pressure. This big difference in static pressure and WSS suggests that in the calculation of hemodynamic forces acting on the wall, the WSS can be neglected. However, the effect of WSS on the estimation of growth processes or changes of aneurysm size can be significant because of the contribution of WSS to the formation of thrombus which has an influence on mechanical properties of vascular walls [11].

It is worth noting that shear stress profiles correspond approximately to the absolute value of velocity curve (Fig. 2).

Pressure and WSS profiles along the aorta circumference

At the next stage, pressure and WSS profiles were analysed in selected points on the vessel wall around cross-sections of flow area (Figs. 7 to 11).

It was observed that in all points around the aorta (Fig. 3b) pressure changes in time were identical. An example of static pressure changes on the level of cross-section I is shown in Fig. 7.

Analysis of stress distributions on walls (Figs. 8 to 11) shows that the distribution of shear stresses is changing significantly along the aorta circumference.

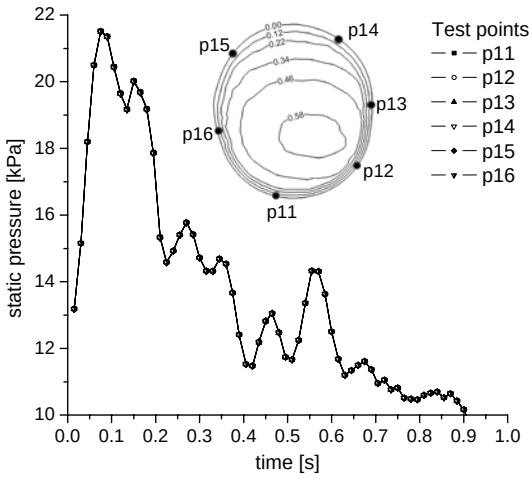

Figure 7: Pressure at artery wall and velocity contours [m/s] on the level of cross-section I (six test points see Fig. 4b)

The biggest differences in the profiles were observed in cross-sections I and II (see Fig. 4b), Figs. 8 and 9. In these places there are evident changes of artery lumen and flow trajectory that cause local disturbances of blood flow velocity. Figures 8 through 11 illustrate also blood flow velocity profiles in the form of contours which confirm the presence of disturbances of velocity profiles in each cross-section.

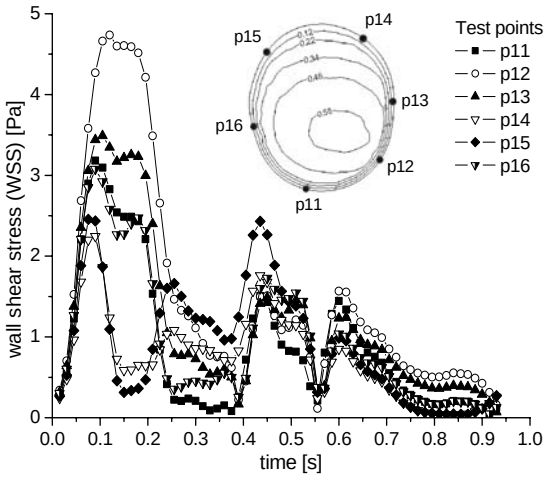

Figure 8: Wall shear stress (WSS) and velocity contours [m/s] in cross-section I (see Fig. 4b).

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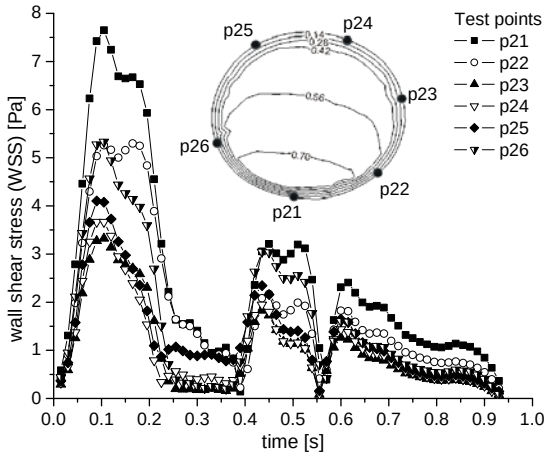

Figure 9: Wall shear stress (WSS) and velocity contours [m/s] in cross-section II (see Fig. 4b)

The next two Figures show WSS distributions at cross-sections III and IV, (Figs. 10 and 11), that are much more uniform.

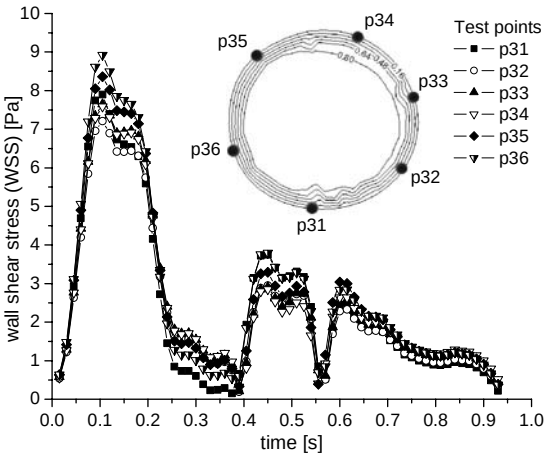

Figure 10: Wall shear stress (WSS) and velocity contours [m/s] in cross-section III (see Fig. 4b)

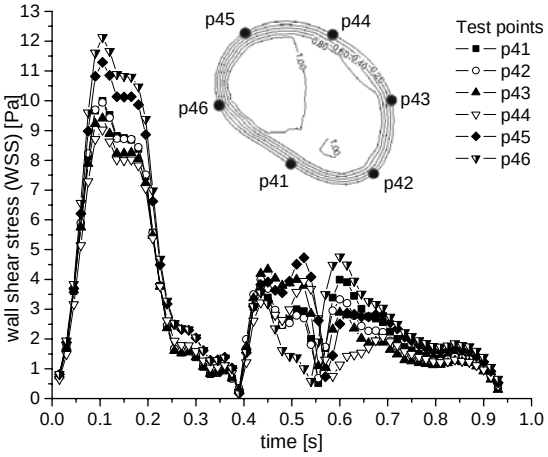

Figure 11: Wall shear stress (WSS) and velocity contours [m/s] in cross-section IV (see Fig. 4b)

To estimate the effect of WSS of flow hemodynamics in the aorta, and consequently, the system of forces acting on vascular walls, CT images were analysed in the cross-sections shown in Fig. 4b comparing thrombus thickness and WSS profiles around a cross-section of aorta wall. Figure 12 shows an example of thrombus thickness at cross-section II (Fig. 9) with points where changes in WSS were analysed.

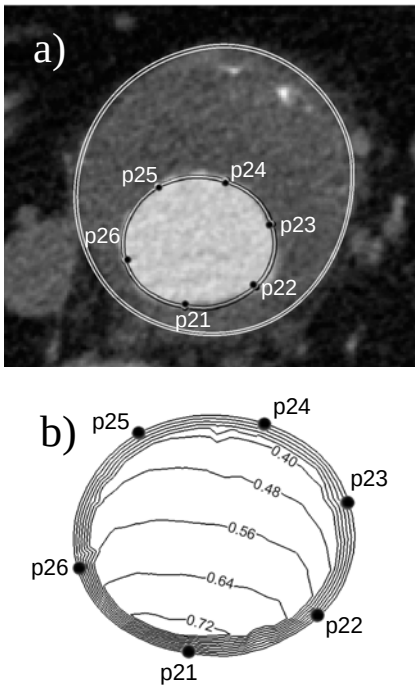

Figure 12: WSS and thrombus thickness analysis in cross-section II. a) CT image of abdominal aortic aneurysm with points of shear stress analysis. b) velocity profiles [m/s] at systole.

When comparing Figs. 9 and 12 it can be found that thrombus is the thickest in points where WSS values are the smallest (p23, p24, p25). Similar conclusions follow from the analysis of thrombus thickness in other cross-sections of the aorta. This confirms that statement that shear stresses should be taken into account in the calculation of forces that act on vascular walls.

Conclusions

Results of theoretical calculations and experimental CT data enabled estimation of the effect of WSS on flow hemodynamics in the aorta, and on the system of forces acting upon vascular walls. From analysis of CT images and WSS profiles along the aorta wall circumference it follows that the thickest thrombus is formed in places where WSS are the smallest [11].

This means that changes of WSS value in the aorta has an influence on thrombus formation and finally on flow conditions, mechanical parameters of the wall and consequently, on the forces acting on vascular walls.

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