

631. Pressure dynamics of blood vessels when modeling pathological processes

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Abstract. This paper studies variations of blood pressure acting onto blood vessel wall as well as dynamics of the blood-flow velocity in the pathological blood vessels by evaluating blood-flow turbulence and taking into account that blood is a non-Newtonian fluid. Finite element method was used to construct models of healthy blood vessels, aneurysm, atherosclerosis and deflections. It was determined that local pressure exerted onto blood vessels walls and blood flow velocity variation depend on size and type of the pathology. Local blood pressure in the pathology location could be up to 7 times higher in comparison to systolic blood pressure. It is established that the most dangerous pathology type is atherosclerosis. Obtained research results could be applied in evaluation of human efficiency.

Keywords: local pressure, elastic blood vessels, blood flow, pathology, turbulence, velocity, non-Newtonian fluid, pathology type.

Introduction

In scientific literature and among practicing surgeons there is a widely established opinion that in the places of pathology the blood-flow creates conditions itself to bypass them, however there is still a shortage of research performed in this field and the available results do not prove the fact unambiguously [1-15].

Atherosclerosis is the main reason for stroke. The plaque that forms inside the carotid artery obstructs blood flow to the parts [3, 6-10]. Blood pressure and wall stress have considerable effects on artery remodeling [5, 8, 11-14]. High shear stress can damage normal endothelium, may also activate platelets and cause platelet aggregation and thrombus formation [4, 7, 14, 15]. Given the well-recognized predisposition to atherosclerotic plaque formation to specific arterial regions with curvature, such as carotid bifurcation, bifurcation, hemodynamics and arterial geometry may both offer useful information for carotid artery stenosis evaluation. Local hemodynamics and consequently the wall shear stress are strongly influenced by the morphologic characteristics of the stenosis and the flow waveforms. Furthermore, to date there are no more reliable methods to determine wall shear stress in the recirculation zones downstream in the stenosis in clinical evaluation. Thus patient-specific information on hemodynamics generated from computational fluid dynamics (CFD) could clarify the relationship between artery stenosis and symptoms and ultimately, the risk of stroke [6, 7].

A lot of research work has been performed on this topic, but most of it considered the blood vessel as the solid body during calculation of velocities, pressures. There is still a lack of those studies where the blood vessel model allows elastic deformation [6, 12]. Furthermore,

insufficient research is devoted to the turbulent blood-flow, where blood is treated as a non-Newtonian fluid.

Therefore the aim of this research is to analyze blood pressure acting onto blood vessels walls and evaluate dynamics of blood flow velocity in pathological blood vessels. For analysis of local blood pressure in the place of pathology and flow velocity dynamics in the pathological blood vessels we account for pathology type, flow turbulence and non-Newtonian fluid properties.

Mathematical model

Blood vessels with different pathology types were chosen for the research (Fig. 1). Finite element method was applied to develop the following models: healthy blood vessel, aneurysm, atherosclerosis with one and two atherosclerosis discs (blood vessel stenosis 25 %, 50 % and 75 %), single and double deflections. The following input parameter are used for the analysis: blood vessel diameter $d=0,02$ m ($d=2R$, R – blood vessel radius), blood vessels wall thickness $h=0,001$ m, vessel length $L=0,11$ m, blood flow velocity $v=0,5$ m/s, turbulent blood flow, blood – non-Newtonian fluid, tension model $E=4,66$ MPa, Poisson's ratio $\nu=0,49$, blood vessel wall density $\rho=1060$ kg/m³. Blood flow was analyzed by means of Navier - Stokes equation in vector form [16]. In Cartesian coordinate system with coordinates (x, y, z) , the operator is defined in terms of partial derivative operators:

$$\rho \left(\frac{\partial v}{\partial t} + v \nabla v \right) = -\nabla p + \mu \nabla^2 v + f ; \quad \nabla = i \frac{\partial}{\partial x} + j \frac{\partial}{\partial y} + k \frac{\partial}{\partial z} ; \quad (1)$$

where ρ – is the fluid density; v – is the flow velocity; ∇ – is the del operator; ∇p – is gradient of pressure of surfaces; μ – is the (constant) dynamic viscosity; f – stands for other body forces; p – is pressure.

Modeling was performed with ANSYS 12.0 software package.

During analysis of different pathology types (Fig. 1) various blood flow turbulences are considered therefore different velocity distribution is obtained in the pathological locations. Some typical cases are illustrated in the Figs. 2-3.

Given graphs demonstrate that for healthy blood vessel the blood velocity (v) could vary from 0 m/s till 0,5 m/s (Fig. 2, curve 2). When atherosclerosis is with one platelet, stenosis is 75 %. In that case blood velocity (v) differs from 0 m/s till 1,5 m/s (Fig. 3, curve 1 and Fig. 5). When atherosclerosis is with two platelet and stenosis reaches 75 %, blood velocity (v) in pathological location varies from 0 m/s till 2 m/s (Fig. 2, curve 1 and Fig. 4). We can observe that blood-flow velocity is determined by the pathological variations – contractions i.e. stenosis. Provided results indicate that turbulence influences blood flow-rate – in healthy blood vessel it varies from 0 m/s to 0,5 m/s, in blood vessel with pathology – to 2 m/s i.e. up to 4 times more with respect to healthy blood vessel (Fig. 2, curve 1). In the cross-section of the pathological vessel turbulence flow-rate differs from the laminar flow-rate in the healthy blood vessel. We can observe that when there is a double atherosclerosis platelet (Fig. 2, 1st curve), blood-flow velocity spread approximately according to symmetry law, meanwhile atherosclerosis with one platelet produces dissymmetry (Fig. 3, curve 1). Blood flow-rates spread alike in other pathological blood vessels.

In the case of different pathologies, we have different distribution of pressure exerted onto blood vessel walls over the entire blood vessel length. Blood vessels were investigated when systolic blood pressure was 120 mmHg ($1,5 \times 10^4$ Pa), 160 mmHg ($2,1 \times 10^4$ Pa) and 200 mmHg ($2,6 \times 10^4$ Pa). Some typical research cases are provided in Figs. 6-9.

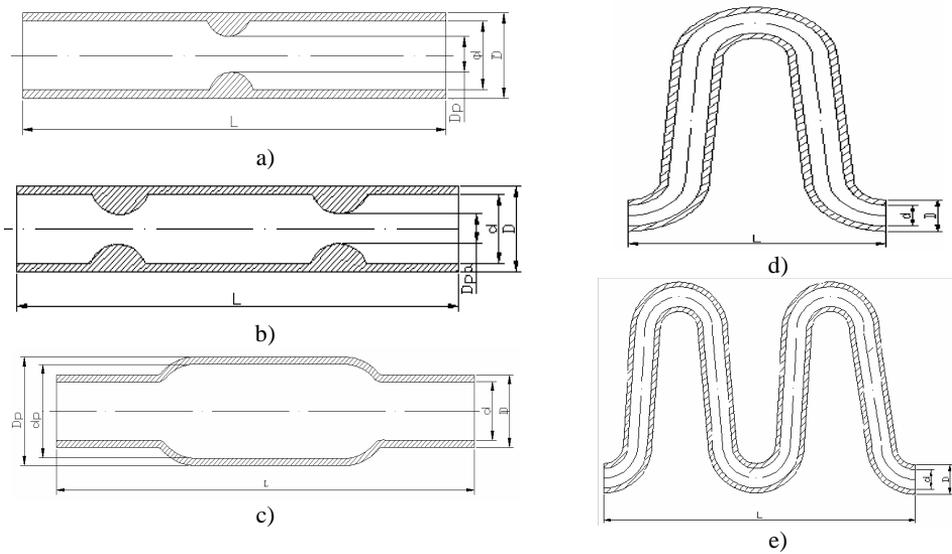


Fig. 1. Model of a blood vessel: a – atherosclerosis with one platelet; b – atherosclerosis with two platelets; c – aneurysm; d – one link; e – two links

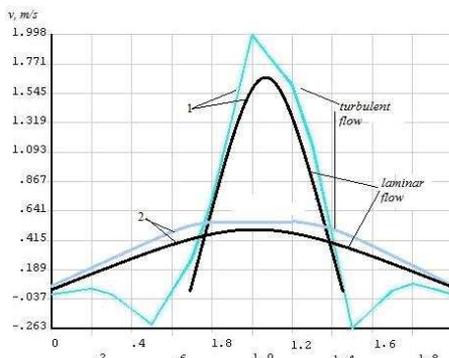


Fig. 2. Blood flow velocity variation in blood vessel cross-section, depending on the type of pathology and flow: 1 - healthy blood vessel; 2 - atherosclerosis with two platelet, stenosis 75 %

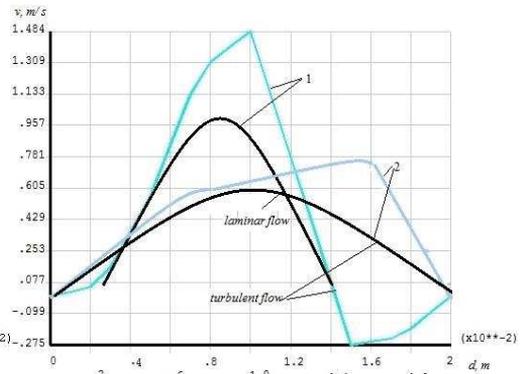


Fig. 3. Blood flow velocity changes in blood vessel cross-section, depending on the type of pathology and flow: 1 - atherosclerosis with one platelet, stenosis 75 %; 2 - double deflection

Performed computations (Figs. 6-10) indicate that when systolic blood pressure varies from 120 mmHg ($1,5 \times 10^4$ Pa) to 200 mmHg ($2,6 \times 10^4$ Pa), maximal local blood pressure varies from $0,5 \times 10^5$ Pa to 7×10^5 Pa. It could be up to 4,55 times higher with respect to systolic blood pressure (Fig. 10, 8 column).

Performed analysis demonstrates that when systolic blood pressure increases from 120 mmHg ($1,5 \times 10^4$ Pa) to 200 mmHg ($2,6 \times 10^4$ Pa), local blood pressure before first deflection changes from $0,7 \times 10^5$ Pa to 5×10^5 Pa, before second deflection – from $0,7 \times 10^5$ Pa to $3,9 \times 10^5$ Pa. It is up to 5,86 times higher near the first deflection and 4,88 times near the second when compared with systolic blood pressure (Fig. 10, 9 column).

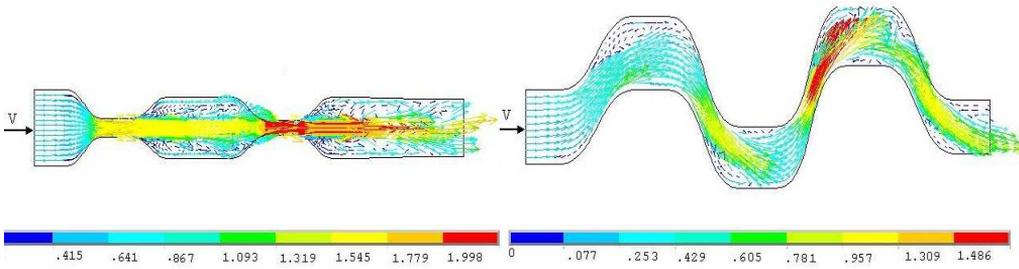


Fig. 4. Blood flow atherosclerosis with two platelet, stenosis 75 %

Fig. 5. Blood flow with double deflection

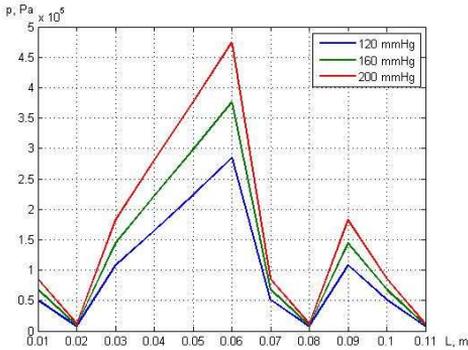


Fig. 6. Local pressure dependence on the length of the vessel pathology place: atherosclerosis with one platelet when stenosis 75 %

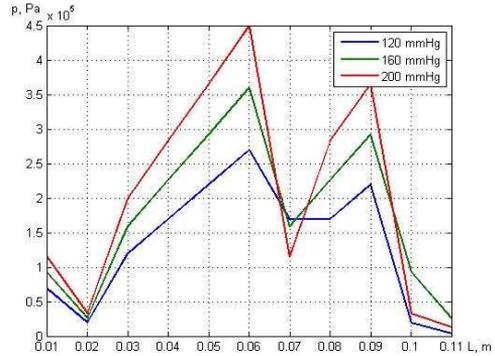


Fig. 7. Local pressure dependence on length of the vessel pathology place: atherosclerosis with double platelet when stenosis 75 %

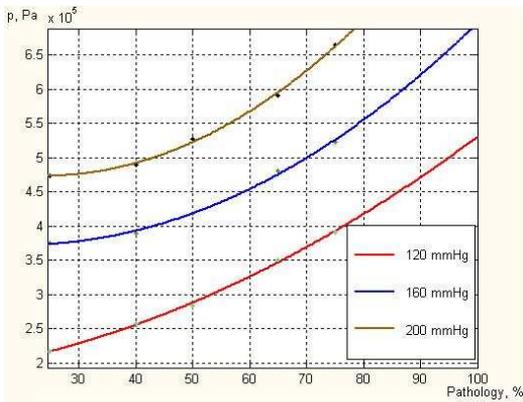


Fig. 8. Pathology size dependence on local pressure: atherosclerosis with one platelet when stenosis

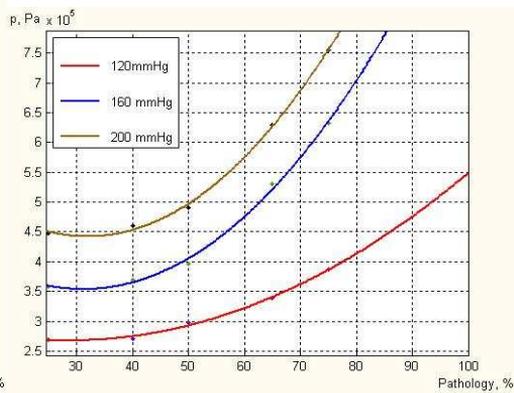


Fig. 9. Pathology size dependence on local pressure: atherosclerosis with double platelet when stenosis

The analysis reveals (Fig. 6, 8 and 10, 4 column) that when systolic blood pressure varies from 120 mmHg ($1,5 \times 10^4$ Pa) to 200 mmHg ($2,6 \times 10^4$ Pa), local blood pressure before atherosclerosis platelet vary from $0,5 \times 10^5$ Pa to $4,7 \times 10^5$ Pa. It could be up to 6,17 times larger than systolic blood pressure. When there is a 75 % atherosclerosis with double platelet, local blood pressure before first atherosclerosis platelet raises from $0,6 \times 10^5$ Pa to $4,5 \times 10^5$ Pa, it increased by a factor of 4,5. Before the second atherosclerosis platelet local blood pressure increases from $0,6 \times 10^5$ Pa to $3,7 \times 10^5$ Pa and the increase makes 3,83 times.

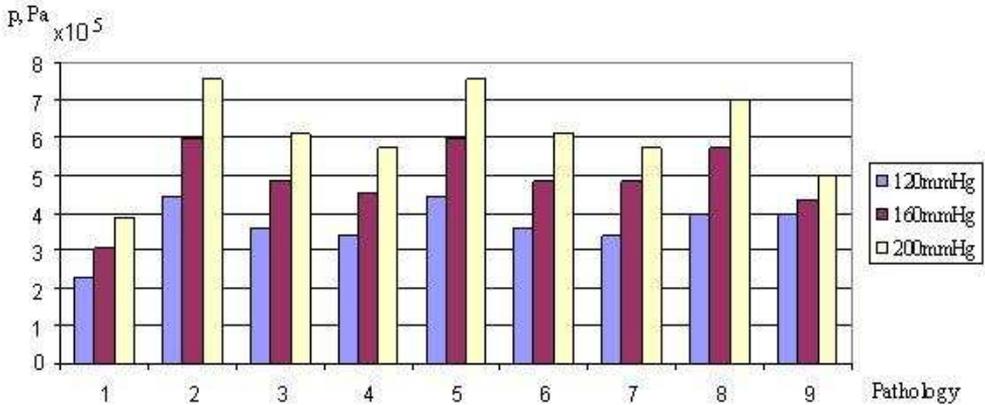


Fig. 10. Maximum pressure distribution according to pathology type 1 - aneurysm; 2 - atherosclerosis (1 plate) 25 %; 3 - atherosclerosis (1 plate) 50 %; 4 - atherosclerosis (1 plate) 75 %; 5 - atherosclerosis (2 plate) 25 %; 6 - atherosclerosis (2 plate) 50 %; 7 - atherosclerosis (2 plate) 75 %; 8 - one link; 9 - two links

Figure 10 provides distribution of maximal pressures acting onto the vessel walls. When 120 mmHg ($1,5 \times 10^4$ Pa) and 160 mmHg ($2,1 \times 10^4$ Pa) maximum is with atherosclerosis 25 % with one and two atherosclerosis platelet. When 200 mmHg ($2,6 \times 10^4$ Pa) the maximum is for atherosclerosis 25 % with one and two atherosclerosis platelet. Thus, these results suggest that atherosclerosis is the most dangerous.

Performed numerical study indicates that local pressure in the place of pathology of the blood vessel could increase from 3 to 7 times in comparison to systolic blood pressure. This is a hazardous situation, therefore it should be accounted for during evaluation of human efficiency. Atherosclerosis is the most critical pathology type. Fig. 8 and 9, local blood pressure in pathology location, indicate that pressure acting onto the wall, according its length, is different. Graphs reveal that maximal blood pressure places in the blood vessel cross-section are before and after pathology. Local pressure distribution is alike for other considered pathology types.

Actual local blood pressure value is obtained during estimation of turbulence in the non-Newtonian blood flow pathology location. This value is larger than laminar blood flow when Newtonian fluid is used.

Conclusions

The following conclusions may be formulated when summarizing blood-flow research results obtained for the blood vessel with pathology, where turbulence is taken into account.

In the case of different pathologies (aneurysms, atherosclerosis and deflections), different blood flow-rate law is obtained, which varies not according to parabolic law. Maximal blood-flow velocity could differ up to 4 times, depending on pathology type.

When there is different systolic blood pressure (from 120 mmHg (15996 Pa) to 200 mmHg (26660 Pa)), local pressure exerted onto vessel walls could increase up to 7 times near pathology location (atherosclerosis 75 %). This could lead to blood vessel crack conditions even when systolic blood pressure is within allowable limits.

Performed analytical study indicates that the most dangerous pathology type is atherosclerosis. Therefore it is necessary to consider and pay attention to pathology size and type.

Obtained research results are valuable in the process of assessment of human efficiency.

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