Estimation of Wall Shear Stress Using a Multi-Branched Model of the Human Arterial System

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Abstract

Background: Simulation of WSS is of interest in studies which attempt to identify the effect of alterations in the geometry and physical properties of the circulation on wall shear stress, and on the potential for disease development. The usual approach that has been taken in flow simulation is the use of computational fluid dynamics and finite element modelling. This can provide detailed 3D images showing the time varying wall shear stress patterns in particular arteries. However such simulations are computationally demanding, and only model a small section of the arterial system. An alternative approach is the use of a multi-branched model to provide estimates of WSS in different regions throughout the arterial tree.

Method: The arterial system was represented by a multi-branched model. Velocity profiles occurring in fully developed pulsatile flow were obtained using Womersley's theory. Mean and peak WSS were calculated in different arteries. Simulations for diabetes and atherosclerosis were also carried out. Stiffness of all the arteries was doubled to simulate diabetes. In this simulation we assumed tissue demand for oxygen and nutrients to be the same for a healthy person and a person having diabetes, so terminal resistance was adjusted so that average flow through the terminal vessels remained unchanged. In simulation of focal atherosclerosis the length of stenosis was somewhat arbitrarily taken as 0.7 cm in the middle of the femoral artery (12.7 cm long). The decrease of the vessel diameter was taken as 80% (severe stenosis). The plaque was considered to be 10 times stiffer than healthy arterial wall (calcified wall).

Results: Peak/mean WSS was found to be in the healthy arteries: (3.75/1.52 Pa) in brachial, (1.41/0.65 Pa) in common carotid, (4.01/2.02 Pa) in radial, (4.16/0.93 Pa) in femoral, (5.69/1.6 Pa) in popliteal, (6.56/3.87 Pa) in tibial, (1.74/0.22 Pa) in common iliac, (3.58/0.14 Pa) in thoracic aorta, and (4.69/0.38 Pa) in abdominal aorta. The magnitude of oscillations of WSS in the stiff arteries was found to be larger than in healthy arteries. Data for the stenosed arteries is presented in the following table:

WSS	Stenosed (P)	Stenosed (D)	Healthy (P)	Healthy (D)
Minimum	43.87	88.32	-1.869	-1.49
Maximum	295.7	199.3	4.779	4.243
Mean	114.6	114.7	0.9162	0.9165

Table 1: Peak and average WSS [Pa] in femoral artery just before (P) and after (D) the stenosis.

Conclusion: 1D multi-branched model is easy to implement and it is not computationally demanding. It may be used for detailed quantitative analysis of the velocity profiles obtained by assigning specific values to the various distal portions of the model of the human arterial tree. So, it may be a useful tool to estimate WSS in arteries, allowing the effect of alteration of model parameters on WSS to be investigated.