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Dedicated to Professor Iulian Coroian on the occasion of his 70^{th} *anniversary*

A finite element approximation for the complex geometry of the carotid artery

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ABSTRACT. Given a parametric model of a tubular object-a generalized cylinder with elliptical or cylindrical cross-section combined with a deformable model, an alternative finite element method is used-the so called web-method in order to provide an approximate solution over all elements. This is obtained by assuming a weighted function to relate internal values on the constructed grid.

1. INTRODUCTION

The main suppliers of blood to the middle cerebral and the anterior cerebral region are known to be the carotid arteries.The common carotid artery divides asymmetrically into two daughter branches:

- internal carotid;
- external carotid;

forming the carotid bifurcation.

This arteries can be thought of as thick-walled, multi-layered tubes whose innermost surface is exposed to flowing blood and with the outermost surface bounded by the surrounding tissue. The three distinct layers the walls of the arteries consist of, can be described as follows. The innermost layer is the *intima*, or epithelial lining of the artery. The middle layer is the *media*, or muscular layer, which gives the artery its stiffness, elasticity and strength. The outer layer is the *adventitia*, which is composed of loose connective tissue. The radius of the lumen is roughly one-half of the radius of the artery itself.

Current medical studies state that atherosclerosis is a response to injury that is mediated (or directed) by the endothelial cells that line the arteries. Usually this disease develops near areas of increased curvature and tortuousness; in the course of plaque formation three stages can be differentiated.

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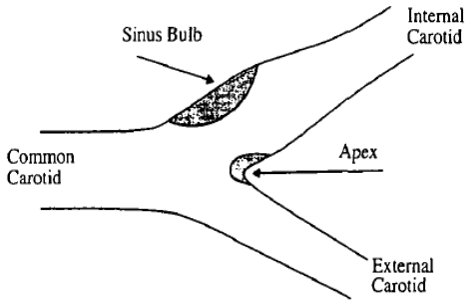

Figure 1. Distinct accumulation of the atherosclerotic plaque in the carotid artery

First lipids from blood accumulate in the subendothelium. Second, the lipid material is ingested in macrophages, forming foam cells. Finally smooth muscle cells migrate from the muscular layer and become transformed into fibroblasts. These form a collagenous(fibrous) matrix within the plaque and also form a fibrous cap on the lumenal side of the plaque, below the intimal layer. Up to this point, the plaque structure is stable. There is increasing evidence that inflammation plays an important role in the evolution of plaque. Beginning with the foam stage, an inflammatory process is apparent. Ongoing inflammation causes the breakdown of foam cells and other components of plaque and the accumulation of inflammatory debris. The inflammatory process disrupts the structure of the plaque, weakens the fibrous cap and extends to the intima.

There is generally little or no change in hemodynamics when an artery is first narrowed by disease, but a relatively rapid decrease in pressure and flow occurs with greater degrees of narrowing. Large plaques cause stenosis, while small ones are less likely to do so. This fact has been verified histologically and sonographically, with direct observations of the plaque complications, including fibrous cap disruption and ulceration. The concept of critical stenosis is a gross simplification of a very complex interplay of numerous factors, such as the length and diameter of the narrowed segment, the roughness of the endothelial surface, the degree of irregularity of the narrowing and its shape-whether the narrowing is abrupt or gradual-the ratio of the narrowed segment to that of the normal vessel, the rate of flow, the arteriovenous pressure gradient and the peripheral resistance beyond stenosis.

Several approaches were made using a classic finite element approximation methods (i.e. Galerkin method)(see Defino [2], Ibragimov et al [4] and Passerini [8]) in order to capture certain features observed in the medical studies of the particular development of atherosclerosis i.e. the localization of immune cells, the build-up of lipids and debris and the isolation of lesion by smooth muscle cells. In this paper we present the application of an alternative finite element method-the so called web-method on a deformable model depicting the carotid arteries. This is obtained by assuming a weighted function to relate internal values on the constructed grid.

2. MODEL DESCRIPTION

In medical imaging, segmentation (see Kaazemput-Mofrad [5]) is a process of classifying and separating different tissues. It is a prerequisite for qualification of morphological disease manifestation, for volume visualization and modeling of individual objects, for chirurgical operation planning and simulation. Deformable models appear to be one of the most promising segmentation techniques. Fully three dimensional models are based on the incompressible Navier-Stokes equations possibly coupled to describe the blood viscosity and the mechanical deformation of the vascular tissue. This approach is well suited for investigating the effects of the geometry on the blood flow and the possible physiopathological impact of hemodynamics.

Let us refer to the simple model of a cylindrical pipe which was been split at the section Γ into two halves. The left one is described using the so called Euler Equations:

$$\frac{\partial S}{\partial t} + \frac{\partial Q}{\partial x} = 0 \tag{2.1}$$

$$\frac{\partial Q}{\partial t} + \frac{\partial(\alpha\frac{Q^2}{S})}{\partial x} + \frac{S}{\rho}(\frac{\partial P}{\partial x}) + K_R\frac{Q}{S} = 0 \tag{2.2}$$

Here $S = S(t,x)$ represents the area of the vascular section at the abscissa x along the vessel axis and the time t, $Q = Q(t,x)$ is the corresponding flow rate, $P = P(t,x)$ is the pressure, ρ is the blood density, α the so called momentum correction and K_R a coefficient related to the viscosity of blood.

Area and pressure are related by the following relation:

$$P = P_{ext} + \frac{\sqrt{\pi}h_0E}{(1-\nu^2)S_0}(\sqrt{S} - \sqrt{S_0}) \tag{2.3}$$

where E is the Young modulus of the wall, h_0 is the wall thickness and ν the Poisson ratio.

The flow in the right hand side of the pipe is computed by the incompressible Navier-Stokes equations:

$$\rho\frac{\partial u}{\partial t} + \rho u\cdot(\nabla u) - \nabla\cdot(\mu\nabla u) + \nabla p = 0, \nabla\cdot u = 0 \tag{2.4}$$

where $u = u(x,y,z,t)$ is the velocity field, $p = p(x,y,z,t)$ the pressure, μ the blood viscosity.

Coupling the two models requires appropriate matching conditions. In the case of a rigid 3 D model, it is reasonable to prescribe the continuity of pressure and the flow rate:

$$P = \frac{1}{|\Gamma|}\int_\Gamma p d\gamma, Q = -\rho\int_\Gamma u\cdot n d\gamma \tag{2.5}$$

where $|\Gamma|$ is the area of the interface Γ.

Vascular compliance is often not relevant to the meaningfulness of 3 D results; simulation in this domains still demand computational costs significantly higher than in the rigid case.

3. THE WEB-METHOD AND RESULTS

For the analysis of stress and deformation through the wall thickness of the complex geometries like the carotid bifurcation, a numerical method is necessary. In conventional computational methods i.e. the standard finite element method, the domain of interest is partitioned into a set of regular polygons and piecewise continuous functions. Generating this mesh is however no easy task because of its poor reliance when treating areas of irregularity. In this direction a mesh-free alternative such as the web-method becomes appealing because of the freedom it provides in taking boundary conditions into account and thus generating a image more similar to that obtained in angiographic explorations (see Figure 2). We proceed by fitting the so called web-splines (weighted extended B-splines) to the specific geometric features of the carotids. At first also this way of approximation seems to be infeasible since web-splines do not conform to essential boundary conditions. This difficulty can be overcome through multiplication with a weight function ω which is always positive and vanishes outside the domain of interest. More explicitly this weighted function can be given as follows :

$$\omega(x) = 1 - \max(0, 1 - \frac{dist(x, \partial\Gamma)}{\delta})^{\upsilon} \tag{3.6}$$

where the parameter δ controls the width of the strip Γ/Γ_δ and υ the smoothness.

The starting point in applying the web-method is to lay a grid of width h over our given domain and to construct on it the finite element subspace, where the basis functions are B_i weighted extended B-splines :

$$B_i = \omega \sum_k e_{i,k} b_k \tag{3.7}$$

where $e_{i,k}$ is the extension coefficient needed to stabilize the basis b_k;
$k = (k_1, ..., k_m) \in Z^m$ a m-th variate tensor product of degree n and support $k + h + [0, n + 1]^m h$ normalized so that $\sum_k b_k = 1$

This weighted function will be constructed considering the following medical data and the fact that we assumed the vessel to be a cylindrical pipe. It is a known fact that the common proximal carotid has an internal diameter with an average of 6.2mm near the bifurcation. Right after the bifurcation the internal carotid has an average of 6.6mm . At the sinus bulb , the average diameter is also 6.6mm. Approximately 1.5cm from the apex, the internal carotid diameter is 4.4mm. The external carotid has an average diameter of 3.66mm.

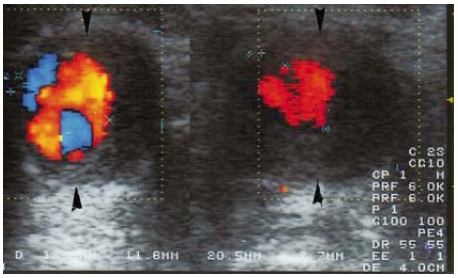

Figure 2. Concentric wall thickening in the proximal internal carotid artery

General the proximal branch of the common carotid displays a circular cross section and fairly uniform wall thickness of 0.9mm. In the region of the sinus bulb, the thickness is 0.55mm. From the sinus bulb the distal part of the internal carotid, the thickness ranges from 0.52mm to 0.4mm. The thickness of the external carotid varies from 0.6mm near the bifurcation to 0.4mm in the distal part of the bifurcation. The angle between the internal and external carotid is 50 degrees, but may be as low as 27 degrees. In the constructing of the model a longitudinal plane of symmetry was assumed through the axes of the three segments. The advantage of this plane of symmetry is that it allows a 50% reduction in the number of computations. The model shown in Figure 3 corresponds to the slice through the proximal internal carotid just upstream of the carotid bulb and downstream from stenosis.

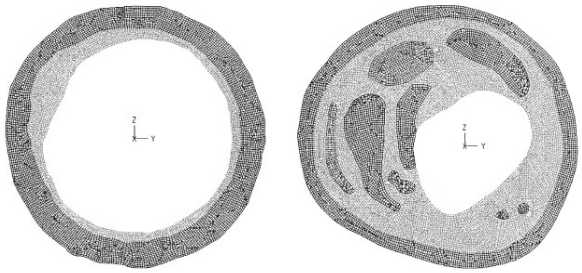

Figure 3. Finite element meshes in 2D histology derived models. Different shades of gray indicate different compositions of substance in the arterial wall-that is calcification and the lipid fibrous plaque

Each 2 D model was divided into regions of lipid, arterial wall and fibrous plaque and calcification. Fibrous plaque was assumed to be any region other than lipid, arterial wall and calcified areas.

4. Conclusions

In this paper we have opened the way to applying the web-method in order to provide an alternative to the standard finite element method applied on problems concerning blood flow on diseased carotid arteries.Clear advantages to standard finite element methods can be mentioned: no mesh generation is required, boundary conditions are represented exactly, regardless of degree, each grid point corresponds to one basis function. This can improve the study of the development, consistence, thickness and stability of the fatty deposits present in the case of atherosclerosis and can be of great relevance as a possible future non-invasive diagnosis technique.

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