Medical Engineering and Physics 43 (2017) 39–47

Contents lists available at ScienceDirect

Medical Engineering and Physics

journal homepage: www.elsevier.com/locate/medengphy

A one-dimensional arterial network model for bypass graft assessment

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ARTICLE INFO

Article history:
Received 15 March 2016
Revised 5 January 2017
Accepted 5 February 2017

Keywords:
Arterial network
1D model
Stenosis
Bypass graft

ABSTRACT

We propose an arterial network model based on one-dimensional hemodynamic equations to study the behavior of different vascular surgical bypass grafts in the case of an arterial occlusive pathology: a stenosis of the Right Iliac artery. We investigate the performances of three different bypass grafts (Aorto-Femoral, Axillo-Femoral and cross-over Femoral) depending on the degree of obstruction of the stenosis. Numerical simulations show that all bypass grafts are efficient since we retrieve in each case the healthy hemodynamics downstream of the stenosed region while ensuring at the same time a global healthy circulation. We analyze in detail the behavior of the Axillo-Femoral bypass graft by performing hundreds of simulations where we vary the values of its Young's modulus [0.1–50 MPa] and radius [0.01–5 cm]. Our analysis shows that Young's modulus and radius of commercial bypass grafts are optimal in terms of hemodynamic considerations. Our numerical findings prove that this model approach can be used to optimize or plan patient-specific surgeries, to numerically assess the viability of bypass grafts and to perform parametric analysis and error propagation evaluations by running extensive simulations.

1. Introduction

Arterial diseases such as stenoses are frequent clinical pathologies, and their prevalence is evaluated from 3% to 10% in the global population with a significant growth from 15% to 20% in persons over 70 years old [1]. Stenoses correspond to the partial or total obstruction of an artery and can cause symptoms going from intermittent claudication to severe ischemia. These symptoms result from a decrease in blood supply as the diseased vessel providing vascularization is narrowed or occluded. When untreated, stenoses can have severe consequences and can lead to the amputation of the stenosed member, especially when they occur in the arteries of the lower members, such as in the Iliac arteries.

When the symptoms are too severe or when medical treatment fails, surgery is necessary to restore blood flow downstream of the stenosed member. This can be done by angioplasty stenting, where the obstructed segment is replaced by a prosthesis (stent) during an endovascular substitution surgery. An alternative solution consists in inserting a bypass graft to redirect the flow of blood from a healthy artery to bypass the obstructed vessel and restore blood flow downstream of the stenosis. In both cases, the mechanical role of these grafts or conduits is to replace or bypass vessels that have become occluded or severely obstructed by a disease process [2].

Numerical studies following local endovascular graft replacements have been reported previously (e.g., [3,4]). We propose to study instead extracorporeal bypass graft procedures. To do so, we consider a detailed model of the systemic network which presents a stenosis of the Right Iliac artery. In this pathological case, the most common bypass graft configurations are: Aorto-Femoral, Axillo-Femoral and cross-over Femoral, defined by the combination of the name of the healthy or donor artery (Aorto for Aorta, Axillo for Axillary and cross-over for the opposite artery, the Left Femoral Artery) and the name of the receptor artery, in our case the Right Femoral artery which follows distally the narrowed site.

The aim of this communication is to use a one-dimensional (1D) model to compute blood flow in each segment of the considered model network before and after extracorporeal bypass graft surgery. To help clinicians optimize surgical repair, we evaluate the viability of each bypass graft by computing the flow rate and pressure downstream of the stenosed member, which is an a posteriori evaluation of the quality of the surgery. Clinicians often prefer the Aorto-Femoral bypass graft. However, for weak patients who cannot tolerate the aortic clamping required to insert the Aorto-Femoral bypass graft, the preferred solution is an extra-anatomic Axillo-Femoral bypass graft [5]. Furthermore, it has the shortest graft survival time among the three previously named bypass grafts [6,7]. We therefore study in detail the optimization of the

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http://dx.doi.org/10.1016/j.medengphy.2017.02.002
1350-4533/

geometrical and mechanical characteristics of the Axillo-Femoral bypass graft. We hope that this numerical approach will be used in the future to define the optimal parameters of new prosthesis and help clinicians plan surgeries.

Here, we present a numerical model and model arterial network, as applied to the study of flow through three different arterial bypass graft configurations, along with the results of a parametric study of the Axillo-Femoral bypass graft. We propose only hemodynamic predictions based on fluid mechanics equations, regardless of biological phenomena and their consequences. Nevertheless, we are aware that short term graft failures can be caused by infections or hemorrhages, while long-term failures are the result of intimal hyperplasia of the graft site, with a proliferation and a migration of vascular smooth muscle cells near the arterial wall [6].

2. Numerical model

To compute the hemodynamics in an artery, we use a set of one-dimensional (1D) equations expressed in terms of the flow rate Q, the cross-sectional area A and the internal average pressure P in the artery. This 1D system of equations results from the integration of the Navier–Stokes equations for an incompressible Newtonian fluid over the cross-sectional area of the artery, leading to the following mass and momentum 1D conservation equations

$$\frac{\partial A}{\partial t} + \frac{\partial Q}{\partial x} = 0, \tag{1}$$

$$\frac{\partial Q}{\partial t} + \frac{\partial}{\partial x}\left(\frac{Q^2}{A}\right) + \frac{A}{\rho}\frac{\partial P}{\partial x} = 2\pi\nu\left[r\frac{\partial u_x}{\partial r}\right]_{r=R}, \tag{2}$$

where u_x is the axial velocity, ρ is the fluid density and ν is the kinematic viscosity of the fluid. We set $\rho = 1\,\mathrm{g/cm^3}$ and $\nu = 3.5\ 10^{-2}\,\mathrm{cm^2/s}$, which are typical values for blood. The internal pressure P is related to the cross-sectional area A through the following relationship

$$P = P_{ext} + \beta(\sqrt{A} - \sqrt{A_0}) + \nu_s\frac{\partial A}{\partial t}, \tag{3}$$

under the assumption that the arterial wall is thin, isotropic, homogeneous, incompressible and that it deforms axisymmetrically with each circular cross-section independently of the others. The parameter β describes the elastic behavior of the wall

$$\beta = \frac{\sqrt{\pi}Eh}{(1-\eta^2)A_0},$$

and the parameter ν_s its viscoelastic behavior, that we describe using a Kelvin–Voigt model [8]

$$\nu_s = \frac{\sqrt{\pi}\rho\phi h}{2\rho(1-\eta^2)\sqrt{A_0}A}.$$

Young's modulus E, the Poisson ratio η, the viscoelastic coefficient ϕ and the arterial thickness h are given in Table A.1 in Appendix A. More details can be found elsewhere [9]. By approximating the friction drag by $-C_f Q/A$ and using the expression (3) for the pressure P, we can re-write the momentum equation (2) as

$$\frac{\partial Q}{\partial t} + \frac{\partial}{\partial x}\left(\frac{Q^2}{A} + \frac{\beta}{3\rho}A^{\frac{3}{2}}\right) = -C_f\frac{Q}{A} + C_v\frac{\partial^2 Q}{\partial^2 x}. \tag{4}$$

We set $C_f = 22\pi\nu$ as was computed from coronary blood flow in [10] and we define $C_v = \frac{A\nu_s}{\rho}$.

From a mathematical point of view, the system of Eqs. (1)–(4) can be decomposed in a hyperbolic subproblem (transport equation) and a parabolic subproblem (viscoelastic source term). To obtain the numerical solution of both subproblems, we introduce a mesh in the axial direction by dividing each artery in a series of cells of size Δx. We then define the discrete time $t^n = n\Delta t$, where Δt is the time step. Using this decomposition of the space and time domains, we discretize the hyperbolic subproblem with a MUSCL (monotonic upwind scheme for conservation law) finite volume scheme and the parabolic subproblem with a Crank–Nicolson scheme. We compute the numerical solution using a code developed in our laboratory, written in C++ and parallelized with OpenMP. The numerical implementation of the full viscoelastic nonlinear system has been validated by comparing the computed solutions to analytic solutions of the linearized system and to experimental data [9,11].

The network used in the numerical simulations is constructed by connecting different arterial segments together. These connections take place at branching points. As an example, we considered a simple branching problem: a single parent vessel connected to two daughter arteries. In this configuration, there are six unknowns at the iteration $n+1$ (numerically speaking, n refers to time t^n and $n+1$ to time t^{n+1}): A_p^{n+1} and Q_p^{n+1} for the outlet of the parent artery and $A_{d_1}^{n+1}$, $Q_{d_1}^{n+1}$, $A_{d_2}^{n+1}$ and $Q_{d_2}^{n+1}$ for the inlets of the two daughter arteries. These quantities are function of the values at the iteration n. To determine these unknowns, we impose the basic laws of conservation at the branching point, that is the conservation of mass flux

$$Q_p^{n+1} - Q_{d_1}^{n+1} - Q_{d_2}^{n+1} = 0,$$

and the continuity of total pressure

$$\frac{1}{2}\rho\left(\frac{Q_p^{n+1}}{A_p^{n+1}}\right)^2 + P_p^{n+1} - \frac{1}{2}\rho\left(\frac{Q_{d_i}^{n+1}}{A_{d_i}^{n+1}}\right)^2 - P_{d_i}^{n+1} = 0.$$

The pressures P are expressed as a function of the cross-sectional area A using the constitutive relation (3). By matching at the branching point the incoming and outgoing characteristics of the hyperbolic subproblem, we obtain the last three equations we need to complete the resolution of the branching point problem. Energy losses should be taken into account due to the complex flow in the branching sites but, in practice, these losses have only secondary effects on the pulse waves [8], therefore we neglect them.

To drive the flow through the network, we prescribe inlet and outlet boundary conditions. These boundary conditions are: (i) an imposed physiological flow rate at the inlet of the ascending aorta and (ii) reflection coefficients imposed at the outlet of each terminal segment and characterizing the resistance of the vascular bed that is not taken into account in our model. These values are given in the last column of Table A.1 in Appendix A. The input flow rate signal we use in the numerical simulations is

$$Q(t) = \begin{cases} Q_{max}\sin(\frac{2\pi}{T}t) & \text{if } t \le T/2; \\ 0. & \text{if } t > T/2. \end{cases}$$

where T is the period of the heart cycle. To define the maximum flow rate Q_{max}, we introduce the ejection fraction EF, defined as

$$EF = \frac{EDV - ESV}{EDV} \times 100, \tag{5}$$

where EDV is the End Diastolic Volume and ESV is the End Systolic Volume. Healthy people typically have an EF between 50% and 65%. On the contrary, people with heart muscles damages (principally on the myocardium) have a low EF. The ejected volume $V_e = EDV - ESV$ during one period is computed by integrating $Q(t)$ over one period

$$V_e = Q_{max}\frac{T}{\pi}. \tag{6}$$

Finally we have

$$Q_{max} = EF\ \pi\ \frac{EDV}{T}, \tag{7}$$