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# A One-Dimensional Arterial Network Model for Bypass Graft Assessment

A. R. Ghigo<sup>a</sup>, S. Abou Taam<sup>a,b</sup>, X. Wang<sup>a</sup>, P-Y Lagrée<sup>a</sup>, J-M Fullana<sup>a</sup>  
<sup>a</sup> Sorbonne Universités, UPMC Univ Paris 06  
CNRS, UMR 7190, Institut Jean Le Rond d'Alembert, F-75005 Paris  
<sup>b</sup> Hôpital Privé Claude Galien, 20 route de Boussy

91480 Quincy-Sous-Sénart

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## 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 the 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.

*Keywords:* arterial network, 1D model, stenosis, bypass graft

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## Corresponding Author

Jose-Maria Fullana

Institut Jean le Rond d'Alembert, UMR CNRS 7190, Tour 55-65 5<sup>e</sup> étage - Bureau 524

Université Pierre et Marie Curie, 4 Place Jussieu, 75005, Paris

Tel (33) (0) 1 44 27 71 41, Fax (33) (0) 1 44 27 52 59

## 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 of 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 can not 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 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.

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

## 64 2. Numerical Model

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

$$\frac{\partial A}{\partial t} + \frac{\partial Q}{\partial x} = 0, \quad (1)$$

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

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

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

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

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

and the parameter  $\nu_s$  its viscoelastic behavior, that we describe using a Kelvin-Voigt model [8]

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

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

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

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

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

85 The network used in the numerical simulations is constructed by connecting different arterial segments together.  
 86 These connections take place at branching points. As an example, we considered a simple branching problem: a  
 87 single parent vessel connected to two daughter arteries. In this configuration, there are six unknowns at the iteration  
 88  $n + 1$  (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  
 89 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  
 90 the iteration  $n$ . To determine these unknowns, we impose the basic laws of conservation at the branching point, that  
 91 is the conservation of mass flux

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

92 and the continuity of total pressure

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

93 The pressures  $P$  are expressed as a function of the cross-sectional area  $A$  using the constitutive relation (3). By matching  
 94 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

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

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

$$EF = \frac{EDV - ESV}{EDV} \times 100, \quad (5)$$

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

$$V_e = Q_{max} \frac{T}{\pi}. \quad (6)$$

Finally we have

$$Q_{max} = EF \pi \frac{EDV}{T}, \quad (7)$$

and we can now define  $Q_{max}$ , for a given period  $T$  and a given  $EDV$  as a function of  $EF$ . With this approach we propose a simple heart model that allows us to define a pathological heart by reducing  $EF$ . This behavior is physiologically meaningful if in the case of a pathological heart, the system reacts by either increasing  $EDV$  by expanding the muscle fibers or by reducing the period  $T$  by increasing the cardiac rhythm.

### 3. Numerical methods and results

In this section we present the numerical protocol and detail the numerical results obtained with the healthy network, the pathological network presenting an obstruction of the Right Iliac artery and the pathological network treated with three different bypass grafts (Axillo-Femoral, Femoral-Femoral and cross-over Femoral bypass grafts).

The numerical protocol is the following:

1. we first simulate blood flow in a healthy network (Figure 1 (a)). We use the computed numerical data as the target blood flow we compare the other numerical results to;
2. we then build the pathological network by narrowing the cross-sectional area of the Right Iliac artery (Figure 1 (b)). As we record all hemodynamic variables everywhere in the network for different degrees of obstruction of the stenosis, we are able to observe the global changes depending on the degree of obstruction;
3. we finally build three repaired networks by introducing in the pathological network the Axillo-Femoral, Femoral-Femoral and cross-over Femoral bypass grafts using elastic tubes inserted between the donor and the receptor arteries of each bypass graft (Figure 1 (c) for the Axillo-Femoral, the other two are presented in Figure 3). We then compare the numerical results obtained with the three repaired networks to those obtained with pathological and healthy networks.

The key points of clinical repair are first the ability of the bypass graft to restore blood flow in the previously non-perfused region (here the network downstream of Right Femoral artery, number 52 in Figure 1), and second, ensuring that the repair does not ill-balance the rest of the hemodynamic circulation. In the following, both key points are systematically for each repaired network.

#### 3.1. Healthy state

The healthy network we consider represents the principal arteries of the great circulation (55 segments). It is used in the literature as the basic model of the systemic network. Its topology is presented in Figure 1 (a), where every artery is given a number (ID) useful to understand the numerical results. Each artery of the healthy network is described by geometrical and mechanical parameters adapted from [12] and presented in Table A.1. Compared to [12], we have added a viscoelastic term to the wall model. This viscoelastic term exists in physiological conditions and is very important from the hemodynamic point of view [8, 9]. Without it, high frequency components would be present in the pulse wave signal [9]. In the literature on 1D network models, this viscoelastic term is usually not included as its coefficients are hard to evaluate experimentally. Here, we use the work of [13], where the viscosity of the aortic walls

142 of dogs was modeled by a Kelvin-Voigt model and where the values of  $\phi$  range between  $3.8 \pm 1.3 \times 10^3 \text{ Pa} \cdot \text{s}$  and  
 143  $7.8 \pm 1.1 \times 10^3 \text{ Pa} \cdot \text{s}$ . Hence, in all numerical simulations we assume  $\phi = 5 \times 10^3 \text{ Pa} \cdot \text{s}$  to calculate the coefficient  $C_v$ .

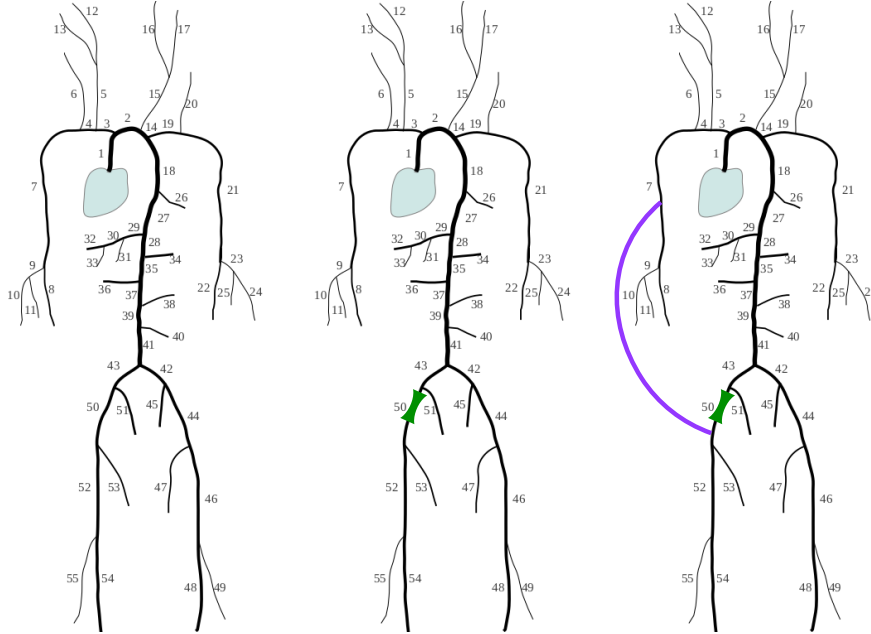


Figure 1. Arterial tree. (a) “Healthy” network. (b) Pathological network. (c) “Repaired” network. The pathological network (b) is modeled by narrowing the cross-sectional area of the Right Iliac artery (number 50, green) and the extracorporeal bypass graft by an elastic tube (purple). In each segment, a 1D model of fluid flow with viscoelastic wall is solved numerically. The flow is imposed by given heart pulses, with a realistic reflection coefficient at the end of each terminal arteries. Table A.1 presents the geometrical and mechanical data used in numerical computations.

144 The flow in each arterial segment is computed using the 1D numerical model presented in the previous section. The  
 145 simulations are performed over 10 heart periods. Any data we present is taken from the final period to ensure that a  
 146 permanent state is reached, where each heart period is identical to the next. The recorded data for the healthy network  
 147 contains the values of the blood flow rate  $Q_{healthy}$ , the cross-sectional area  $A_{healthy}$  and the blood pressure  $P_{healthy}$  in  
 148 every artery and at every recorded time of the final period. These numerical results are the target values we use from  
 149 now on to evaluate the severity of the pathological situation and to assess the restorative properties of the bypass grafts.

### 150 3.2. Pathological case

#### 151 3.2.1. Numerical protocol

152 We model the stenosis by narrowing the cross-sectional area of a portion of the Right Iliac artery (number 50 in  
 153 Figure 1 (b)). The length of the occlusion is 5 cm and the degree of obstruction is directly related to the ratio of the  
 154 cross-sectional area of the stenosed artery  $A_{\%}$  over the cross-sectional area of the healthy artery  $A_{healthy}$ . We define this  
 155 ratio as  $I_s = \frac{A_{healthy} - A_{\%}}{A_{healthy}} \times 100$ . Four control sites are chosen to evaluate the hemodynamical influence of the stenosis on  
 156 the flow rate and pressure waveforms. Two are located in the lower legs, in the Right Femoral artery (number 52) and



157 in Left Femoral artery (number 46). The other two are located in the arms, in the Right Subclavian artery (number 7)  
 158 and in the Left Subclavian artery (number 21). The Right Femoral artery (number 52) is the principal assessment point  
 159 of our numerical study as the flow rate passing through it characterizes the leg's perfusion and therefore the degree of  
 160 ischemia. The other control sites (Left Femoral, Left and Right Subclavian) are used in clinical routines to evaluate if  
 161 a bypass graft surgery is successful.

### 162 3.2.2. Results

163 Figure 2 (a) shows the variation with the degree of obstruction  $I_s$  of the blood flow rate  $Q$  averaged over the final  
 164 period at the four previously defined control sites. The first observation is that under 60% to 70% of obstruction there  
 165 is no significant variation of flow rate with respect to the healthy state ( $I_s = 0\%$ ). This behavior is well known in the  
 166 medical community (i.e. renal arteries in pigs and human carotid arteries [14, 15]). Above 70% of obstruction, the  
 167 flow rate drastically decreases in the Right Femoral artery (number 52) due to the obstruction of its proximal artery,  
 168 the Right Iliac artery (number 50). We note that for an occlusion of 90% there is almost no blood flow in the Right  
 169 Femoral artery (number 52). Conversely, the flow rate moderately increases in the other control sites to compensate  
 170 for the reduction of flow rate in the network distal to the stenosis. This as a clear example of how we can monitor  
 171 global variations in the network caused by a local perturbation.

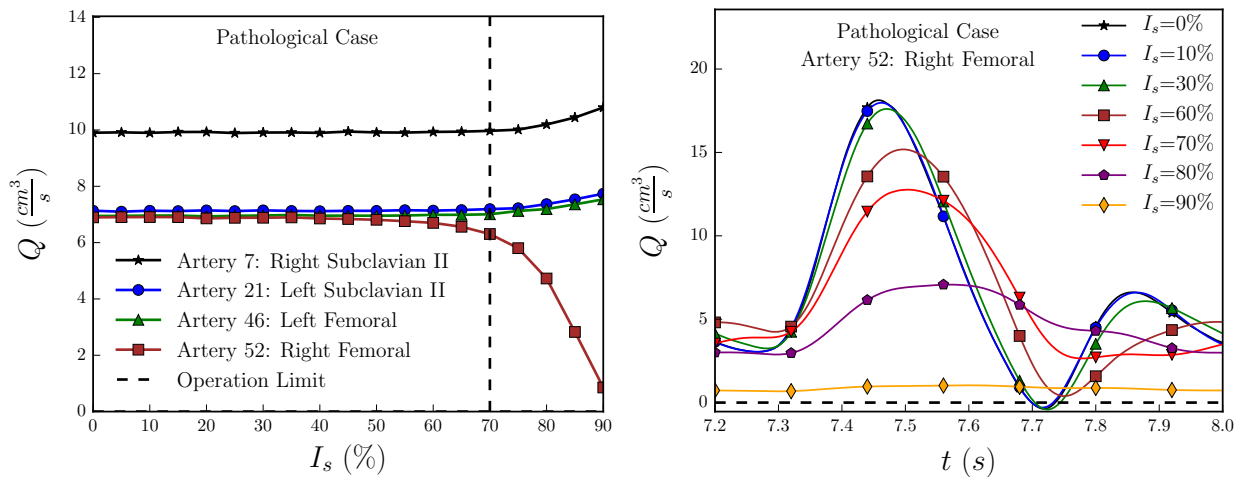


Figure 2. (a) Flow rate averaged over a cycle as function of the obstruction degree  $I_s$  for the following arteries: Right Femoral, (number 52), Left Femoral, (number 46), Right Subclavian (number 7) and Left Subclavian, (number 21). As the ratio  $I_s$  increases, the flow rate drops in the Right Femoral artery, distal to the stenosis, whereas the flow rate increases in all other segments to compensate this drop. (b) Instantaneous flow rate as function of time over a cycle in the Right Femoral artery (number 52) for different degree of obstruction. As the ratio  $I_s$  increases, the waveform loses its pulsatility and flattens and the average flow rate drops.

172 Figure 2 (b) presents the time evolution of the blood flow rate  $Q$  over the final period in the Right Femoral artery  
 173 (artery 52). These results are correlated to those of Figure 2 (a) but provide additional information: first, as expected,

174 the flow rate decreases in average as the ratio  $I_s$  increases; second the positions of the maximum and minimum peaks  
 175 are shifted, due to a time shift in the traveling waves; third the maximum amplitude decreases significantly as the ration  
 176  $I_s$  increases and we observe that for  $I_s = 70\%$  the amplitude drops by 30% and for  $I_s = 80\%$  it drops by 60%. For  
 177  $I_s = 90\%$ , the amount of blood perfusion in the leg is minimal and the waveform is a flat line. This last point indicates  
 178 that as the degree of obstruction  $I_s$  increases, the signal loses its pulsatility and flattens.

### 179 3.3. Bypass grafts

#### 180 3.3.1. Numerical protocol

181 We study here the three most commonly used bypass grafts to treat a stenosis of the Right Iliac arteries: the Axillo-  
 182 Femoral (AxF) where the donor artery is the Axillary artery (artery 7), the cross-over Femoral (FF) where the donor  
 183 artery is the opposite Common Femoral artery (artery 44) and the Aorto-Femoral (ArF) where the donor artery is the  
 184 Abdominal Aorta (artery 39). Each bypass graft is connected to the pathological network using two connection points:  
 185 the proximal anastomosis, connecting the bypass graft to the donor artery, and the distal anastomosis, linking the  
 186 bypass graft to the receptor artery. For each of these three bypass grafts, the distal anastomosis is located downstream  
 187 of the stenosis, in the distal part of the Right Iliac artery (artery 50). In Figure 3 we represent the topology of the three  
 188 different pathological network treated with a bypass graft.

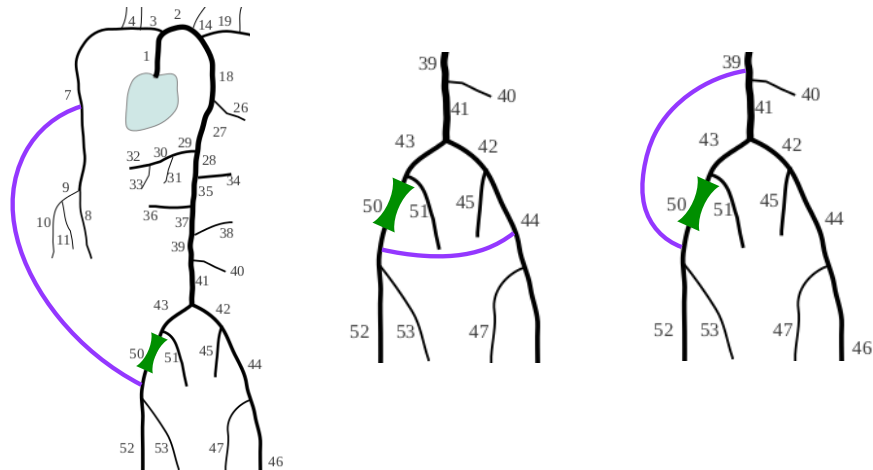


Figure 3. Sketch of three bypasses with the donor artery : (left) Axillo-Femoral (AxF) and donor artery, Right Axillary artery (number 7), (center) cross-over-Femoral (FF) and donor artery, Left Femoral artery (number 44) and (right) Aorto-Femoral (ArF) and donor artery, Abdominal Aorta (numer 39).

189 Each bypass graft we study is made of the same composite material which is constituted principally of polyethylene  
 190 terephthalate (Dacron). From the literature [16], we obtain their mechanical and geometrical characteristics, that is a  
 191 Young's modulus equal to  $9 \times 10^6 Pa$ , an internal diameter of  $0.8 cm$  and a thickness of  $0.05 cm$ . The length  $L$  of each

192 bypass graft depends on the geometric distance between the proximal and distal anastomoses: Axillo-Femoral, 40 cm,  
193 cross-over-Femoral, 20 cm and Aorto-Femoral, 20 cm.

194 To assess the performances of each bypass graft, we define three control sites where we compare the healthy,  
195 pathological and repaired data. The first is located in the Right Femoral artery (number 52), downstream of the stenosis  
196 and the distal anastomosis, and is identical to the control site used previously to analyze the pathological network. The  
197 second and third control sites are respectively situated in the upstream and downstream segments of the proximal  
198 anastomosis.

### 199 3.3.2. Results

200 For each bypass graft we first study the predicted perfusion hemodynamics in the first control site located down-  
201 stream of the stenosis, in the Right Femoral artery (number 52). Figure 4 (a) presents the evolution with the degree of  
202 obstruction  $I_s$  of the time-averaged blood flow rate in the pathological network (same as Figure 2 (a) for the artery 52)  
203 and in the three repaired networks obtained using the AxF, ArF and FF bypass grafts. Figure 4 (b) shows the temporal  
204 evolution of the blood flow rate over the final heart cycle for  $I_s = 90\%$ . These figures should be compared to Figures  
205 2 (a) and 2 (b). We observe in Figure 4 (a) that for all three bypass grafts configurations, we retrieve in average the  
206 blood flow rate of the healthy case for every value of  $I_s$  considered. Figure 4 (b) indicates that the repaired waveforms  
207 are similar to the target healthy one although the amplitudes of the peaks are a slightly underestimated. The delay in  
208 the position of the maximum and minimum flow rate peaks is caused by a change in the length of vessel traveled by  
209 the wave starting from the heart. Overall we retrieve for all three bypass graft configurations the target average blood  
210 flow rate as well as the approximate shape of the waveform. From the analysis of Figure 4 we conclude that all three  
211 bypass graft are successful in retrieving the healthy flow rate in the first control site distal to the obstructed segment  
212 (Right Femoral artery 52).

213 We complete our study by analyzing the time-averaged blood flow rate in each donor artery. In the subsequent  
214 numerical results we focus on the remaining two control sites: the upstream and downstream segments of the proximal  
215 anastomosis, which differ from one bypass graft configuration to the next. We expect that each bypass graft will supply  
216 the missing blood flow rate to the diseased lower leg (Right Femoral artery 52) whilst maintaining a healthy perfusion  
217 in the donor site (downstream segments of the proximal anastomosis).

218 For the FF bypass graft the donor artery is the opposite Femoral artery (Left Femoral artery, number 44 in Figure  
219 1). Figure 5 presents the evolution of the time-averaged blood flow rate with the degree of obstruction  $I_s$  in the two  
220 control sites, upstream and downstream of the proximal anastomosis. We observe that upstream of the donor site the  
221 flow rate increases proportionally to the degree of obstruction. Indeed the donor artery must know supply blood to  
222 both its downstream segment and the stenosed member and therefore increases its flow rate, in comparison with the

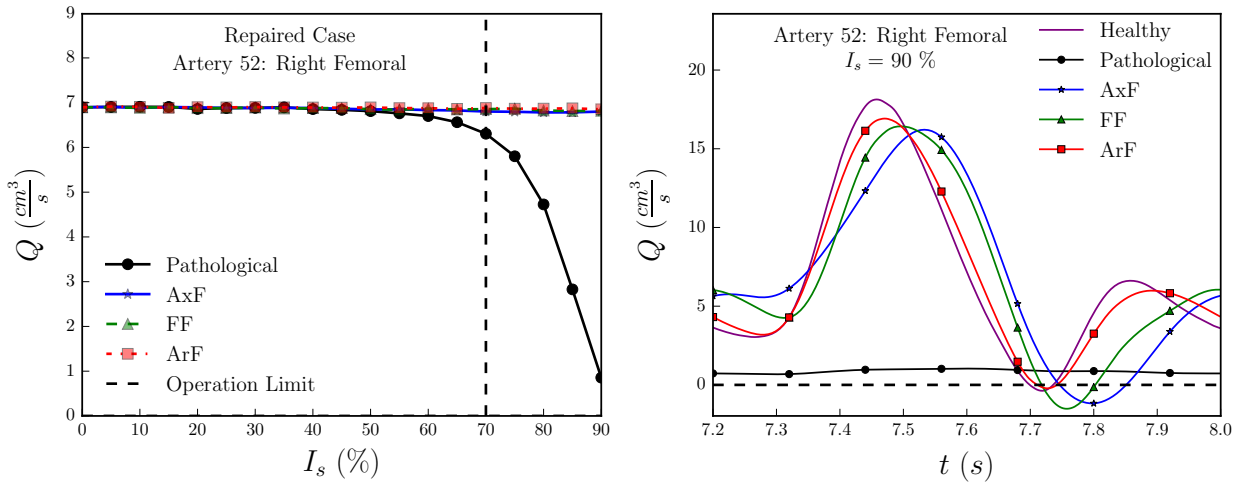


Figure 4. (a) Averaged flow rate over a cycle as function of the degree of obstruction  $I_s$  (Artery 52 : Right Femoral) (b) Instantaneous flow rate as function of time over a cycle (Artery 52 : Right Femoral) for healthy, pathological with  $I_s = 90\%$  and for the three bypasses. We observe that for all three bypass graft configurations, we are able to recover the target healthy flow rate (average values and waveform) distal to the stenosis.

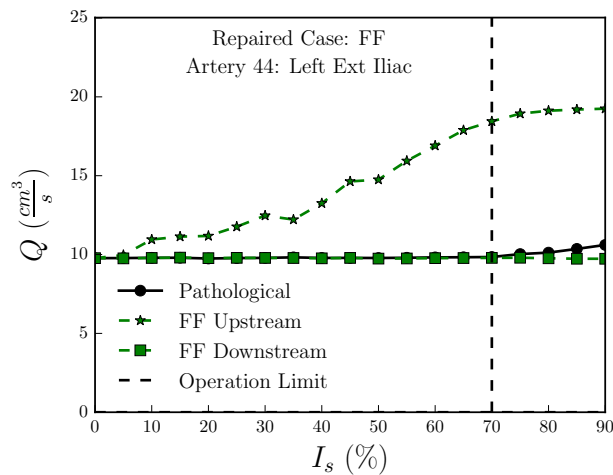


Figure 5. Cross-over Femoral bypass graft: average flow rate over a cycle in the opposite Femoral artery (Artery 44). Upstream of the proximal anastomosis, the flow rate increases to properly vascularize the bypass graft, depending on the degree of obstruction  $I_s$ . Downstream of the proximal anastomosis, we recover the healthy ( $I_s = 0\%$ ) flow rate.

223 healthy case ( $I_s = 0\%$ ). The downstream blood flow rate does not change compared to the healthy case ( $I_s = 0\%$ )  
 224 indicating that the opposite lower leg, downstream of the proximal anastomosis, is correctly supplied. We note that for  
 225 a severe stenosis ( obstruction of 90 %) the upstream blood flow rate is twice the basal one.

226 For the ArF bypass graft the donor artery is the Abdominal Aorta (artery 39), the principal path carrying blood to  
 227 both lower legs. Figure 6 presents the evolution of the time-averaged blood flow rate with the degree of obstruction  
 228  $I_s$  in the two control sites, upstream and downstream of the proximal anastomosis. We observe that upstream of the  
 229 proximal anastomosis, the blood flow rate does not change with the degree of obstruction  $I_s$ , contrary to the FF bypass

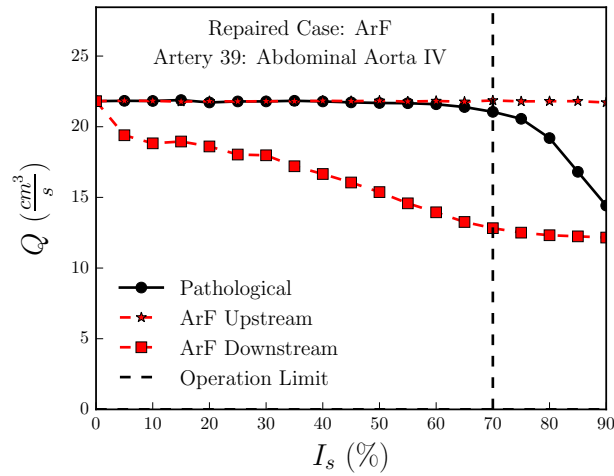


Figure 6. Aorto-Femoral bypass graft: average flow rate over a cycle at the donor artery (Artery 39). Upstream of the proximal anastomosis, the flow rate remains unchanged since blood flow passing through the bypass graft to vascularize the right leg was already supplied by the Aorta in the healthy case. Downstream of the proximal anastomosis, the flow rate decreases with the degree of obstruction  $I_s$  since now only the blood supplying the left leg is passing downstream of the proximal anastomosis.

230 graft configuration. Indeed, in the healthy configuration the Abdominal Aorta already carries blood the the Right  
 231 Femoral artery, therefore no compensation mechanism is required upstream of the donor site. Conversely we observe  
 232 that the downstream of the proximal anastomosis, the blood flow rate decreases as the degree of obstruction increases,  
 233 in comparison to the healthy configuration ( $I_s = 0\%$ ). Indeed, since the blood that supplies the stenosed member (Right  
 234 Femoral artery 52) now flows through the bypass graft, only the blood supply for the left leg remains downstream of  
 235 the donor site. This behavior shows that the bypass graft is indeed carrying blood the stenosed member. Finally, we  
 236 note that in absence of stenosis ( $I_s = 0\%$ ) the downstream blood flow is symmetrically shared between the two legs  
 237 and that for a severe stenosis ( $I_s = 90\%$ ) the downstream blood flow rate is half the basal one.

238 Figure 7 presents the evolution for the AxF bypass graft of the time-averaged blood flow rate with the degree of  
 239 obstruction  $I_s$  in the two control sites, upstream and downstream of the proximal anastomosis. The results are identical  
 240 to those obtained with the FF bypass graft (Figure 5). The same analysis can be performed and we conclude that this  
 241 bypass graft configuration correctly supplies the stenosed member while maintaining the healthy flow rate downstream  
 242 of the donor site. We also note that for an obstruction of 90% the upstream blood flow rate is twice the basal one.

243 These results show that for all three bypass graft configurations, the target behaviors are obtained and the bypass  
 244 graft surgery is successful.

### 245 3.4. Optimization

246 Of the three bypass grafts considered here, the AxF has the highest chance of graft failure. Indeed, the AxF is the  
 247 longest bypass graft. Moreover, the AxF bypass graft surgery is performed on patients who are not healthy enough to

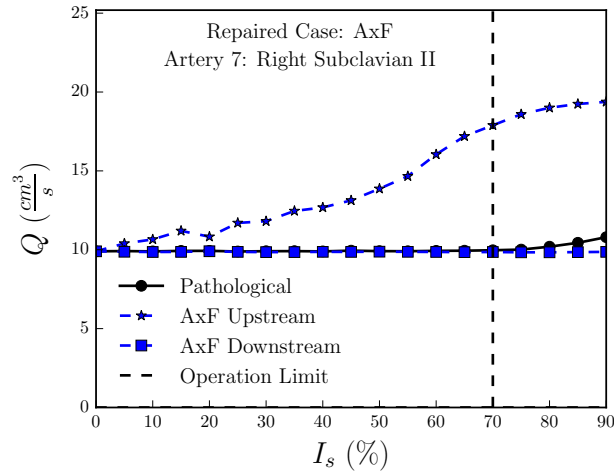


Figure 7. Axillo-Femoral bypass graft: averaged flow rate over a cycle at the donor artery (number 7). Upstream of the proximal anastomosis, the flow rate increases to properly vascularize the bypass graft, depending on the degree of obstruction  $I_s$ . Downstream of the proximal anastomosis, we recover the healthy ( $I_s = 0\%$ ) flow rate.

248 survive the more invasive surgical procedures required to implement the FF or ArF bypass grafts.

249 For these reasons we choose to perform a detailed analysis to determine the optimal mechanical (the Young modulus  
 250  $E$ ) and geometrical (the radius  $R$ ) parameters of the AxF bypass graft. In order to give arguments for discussion  
 251 we perform hundreds of simulations where we vary the values of the Young modulus [ $0.1 - 50 MPa$ ] and radius  $R$   
 252 [ $0.01 - 5 cm$ ] of the AxF bypass graft. As before, we use as a target the healthy data in the Right Femoral artery  
 253 (number 52 in Figure 1).

254 Figures 8 presents a log-log scale contour plot of the normalized flow rate  $\frac{Q}{Q_{healthy}}$  in the AxF bypass graft obtained  
 255 for different values of the Young modulus  $E$  and the radius  $R$  in a pathological network with  $I_s = 90\%$ . The red  
 256 circle in the middle of the Figure 8 indicates the actual values of the bypass graft's Young's modulus  $E$  and radius  $R$ .  
 257 For these values the normalized flow rate  $\frac{Q}{Q_{healthy}} \approx 100\%$  indicating that the healthy flow rate is restored in average  
 258 downstream of the stenosis. Starting from this point we analyze the results by moving along the horizontal and vertical  
 259 directions, that is for  $E$  constant and varying  $R$  (horizontal) and for  $R$  constant and varying  $E$  (vertical).

260 For a constant Young's modulus  $E$ , we analyze the effect of changing the radius  $R$  of the bypass graft. Moving  
 261 along the horizontal direction towards the left starting from the red circle, the radius  $R$  decreases. Consequently the  
 262 hydraulic resistance of the bypass graft increases leading to a decrease of the the normalized flow rate  $\frac{Q}{Q_{healthy}}$ . Moving  
 263 now towards the right, the radius  $R$  increases. Even though for a large range of values of the Young's modulus  $E$   
 264 the value of  $\frac{Q}{Q_{healthy}}$  is close to  $100\%$ , it is clear that as the radius increase the blood volume inside of the bypass also  
 265 increases. This results in a decrease of the flow rate distal to the proximal anastomosis and could lead to the ischemia  
 266 of the right hand. Increasing the radius  $R$  also implies decreasing blood flow velocity in the bypass graft which results

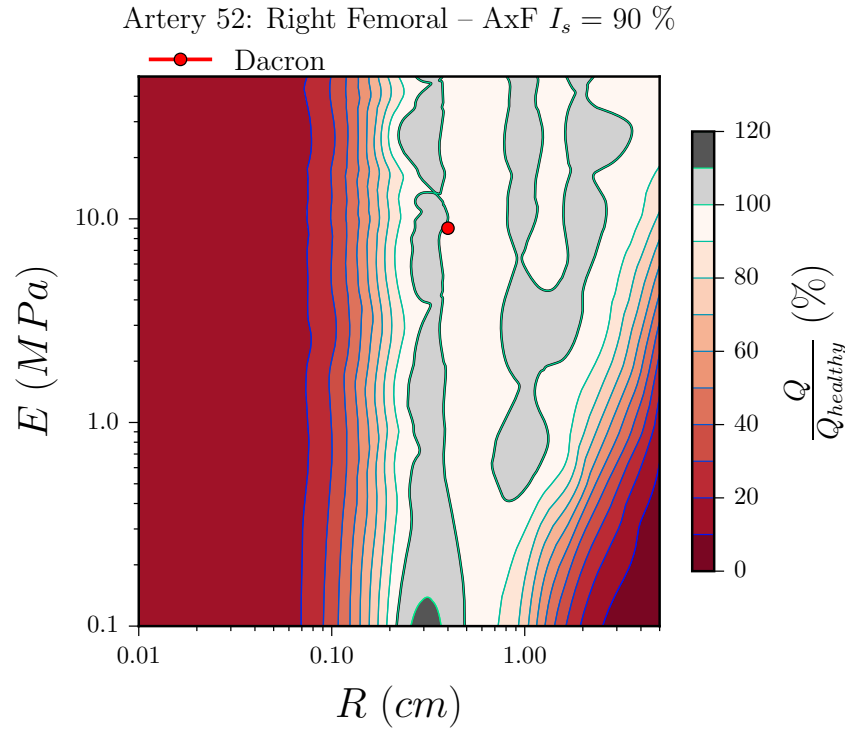


Figure 8. Contour plot for the normalized time-averaged flow rate  $\frac{Q}{Q_{healthy}}$  as function of the Young modulus  $E$  and the radius  $R$  for a stenosis of  $I_s = 90\%$ . The red circle corresponds to the actual values of the Young modulus  $E$  and the radius  $R$  used in numerical simulations, which are situated in an optimal zone (100%). When the radius decreases, the resistance of the tube increases and therefore less flow is bypassing through the bypass graft. When the Young's modulus decreases, the tube becomes more compliant and stores more flow. Both behaviors reduce the quality of the bypass graft.

267 in a smaller shear rate along the bypass. This increases aggregation and coagulation processes which are key factors in  
 268 the onset of graft failure. From a physiological and mechanical point of view for a given value of the Young's modulus  
 269  $E$ , the optimal radius  $R$  should be taken from a  $\frac{Q}{Q_{healthy}} \approx 100\%$  region and be as small as possible to ensure an optimal  
 270 distal and proximal blood perfusion.

271 For a constant radius  $R$ , we analyze the effect of changing the Young's modulus  $E$  of the bypass graft. Moving  
 272 along the vertical direction towards the top or the bottom starting from the red circle,  $\frac{Q}{Q_{healthy}} \approx 100\%$  for every value  
 273 of the Young's modulus  $E$ . However, the bypass graft's Young's modulus  $E$  should be taken as close as possible to  
 274 the arteries' Young's modulus since elasticity jumps lead to impedance discontinuities and therefore higher reflected  
 275 pressure waves. Moreover, if the bypass graft's elasticity is too small, the bypass graft will become more compliant  
 276 and inflate, increasing the blood volume inside the bypass graft. Conversely, if the bypass graft's elasticity is too large,  
 277 high pressure peaks will be generated due to increased wave reflections.

278 Figures 9 presents a log-log scale contour plot of the normalized peak to peak flow rate  $\frac{Q_{max} - Q_{min}}{Q_{max} - Q_{min}^{healthy}} \frac{Q_{healthy}}{Q}$  in the  
 279 AxF bypass graft obtained for different values of the Young modulus  $E$  and the radius  $R$  in a pathological network

280 with an obstruction degree of 90 %. This quantity measures the pulsatility of the flow rate signal (and consequently  
 281 the pressure signal). We observe that both Figure 8 and Figure 9 are similar, and the previous analysis of Figure 8  
 282 can be applied. Nevertheless, Figure 9 provides additional information especially in the region of large radii. For a  
 283 fixed Young's modulus  $E$ , increasing the radius significantly decreases the value of  $\frac{Q_{max}-Q_{min}}{Q_{max}-Q_{min}^{healthy}} \frac{Q_{healthy}}{Q}$ . We previously  
 284 described this situation as a correlation between an increase of radius and an increase of the blood volume inside the  
 285 bypass graft. We prove here that this increase in blood volume in the bypass graft reduces its quality as the signal  
 286 loses its pulsatility.

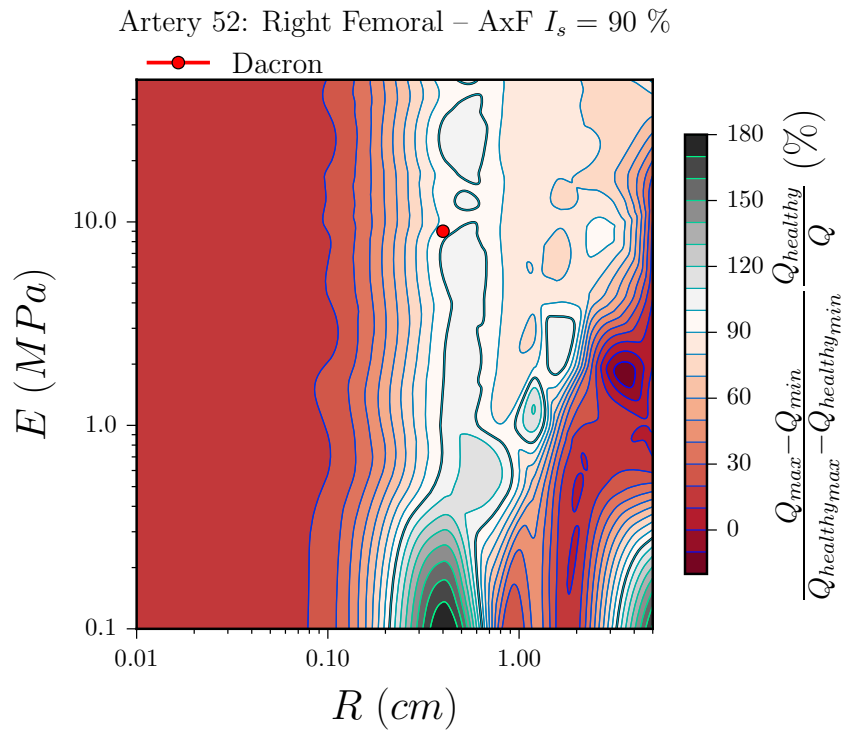


Figure 9. Contour plot for the normalized peak to peak flow rate  $\frac{Q_{max}-Q_{min}}{Q_{max}-Q_{min}^{healthy}} \frac{Q_{healthy}}{Q}$  as function of the Young modulus  $E$  and the radius  $R$  for a stenosis of 90 %. The red circle corresponds to the actual values of the Young modulus  $E$  and the radius  $R$  used in numerical simulations.

#### 287 4. Conclusion

288 We presented a model network comprising 55 viscoelastic arteries in which we modeled blood flow using a 1D  
 289 fluid-structure system of equations. We performed simulations of this complex nonlinear dissipative system in a  
 290 healthy and a pathological network presenting a stenosis of the Right Iliac artery. We then computed blood flow in a  
 291 repaired network where we considered the three classical bypass grafts used to treat a stenosis of the Iliac artery. Our  
 292 numerical results showed that all three bypass grafts are able to retrieved the healthy hemodynamics downstream of



293 the stenosed member whilst maintaining a global healthy circulation.

294 However, little is known about the evolution of the hemodynamics in a bypass graft when its geometrical and  
295 mechanical characteristics are changed. We therefore studied the optimization of the geometrical and mechanical  
296 characteristics of the Axillo-Femoral bypass. Indeed, this bypass graft is used on unhealthy weak patients who can  
297 not sustain other types of bypass graft surgeries and because it has the smallest graft survival time of the three studied  
298 bypass grafts. The optimization results (Figures 8 and 9) indicated that the mechanical characteristic of the bypass  
299 grafts used by clinicians are optimal and allow to retrieve the healthy circulation in the pathological network. Moreover,  
300 the numerical findings showed that choosing another set of parameters would lead to diminished performances of the  
301 bypass graft.

302 Besides the numerical approach, our numerical findings over an "averaged patient" proved that numerical hemo-  
303 dynamic predictions could be used to optimize or plan surgeries for specific patients, under the conditions that the  
304 pathologies were well defined and the physiological parameters known. Indeed, the numerical tool is very fast in  
305 terms of computing time, and therefore is suited for computational intensive simulations such as parametric analyses  
306 and error propagation tasks, and for the evaluation of new bypass procedures.

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## 351 Appendix A. Wall Model and Geometrical and mechanical parameters of the network

352 The Table A.1 presents the name (ID), name, length, neutral cross-sectional area  $A_0$  and mechanical parameters  
353 used in the network.

Table A.1: Arterial network: Data adapted from [12] and [13]

ID	Name	$l$ (cm)	$A_0$ (cm <sup>2</sup> )	$\beta$ (10 <sup>6</sup> Pa/cm)	$C_v$ (10 <sup>4</sup> cm <sup>2</sup> /s)	$R_t$
1	Ascending aorta	4.0	6.789	0.023	0.352	–
2	Aortic arch I	2.0	5.011	0.024	0.317	–
3	Brachiocephalic	3.4	1.535	0.049	0.363	–
4	R.subclavian I	3.4	0.919	0.069	0.393	–

Table A.1: Arterial network: Data adapted from [12] and [13]

ID	Name	$l$ (cm)	$A_0$ (cm <sup>2</sup> )	$\beta$ (10 <sup>6</sup> Pa/cm)	$C_v$ (10 <sup>4</sup> cm <sup>2</sup> /s)	$R_t$
5	R.carotid	17.7	0.703	0.085	0.423	–
6	R.vertebral	14.8	0.181	0.470	0.595	0.906
7	R. subclavian II	42.2	0.833	0.076	0.413	–
8	R.radius	23.5	0.423	0.192	0.372	0.82
9	R.ulnar I	6.7	0.648	0.134	0.322	–
10	R.interosseous	7.9	0.118	0.895	0.458	0.956
11	R.ulnar II	17.1	0.589	0.148	0.337	0.893
12	R.int.carotid	17.6	0.458	0.186	0.374	0.784
13	R. ext. carotid	17.7	0.458	0.173	0.349	0.79
14	Aortic arch II	3.9	4.486	0.024	0.306	–
15	L. carotid	20.8	0.536	0.111	0.484	–
16	L. int. carotid	17.6	0.350	0.243	0.428	0.784
17	L. ext. carotid	17.7	0.350	0.227	0.399	0.791
18	Thoracic aorta I	5.2	3.941	0.026	0.312	–
19	L. subclavian I	3.4	0.706	0.088	0.442	–
20	L. vertebral	14.8	0.129	0.657	0.704	0.906
21	L. subclavian II	42.2	0.650	0.097	0.467	–
22	L. radius	23.5	0.330	0.247	0.421	0.821
23	L. ulnar I	6.7	0.505	0.172	0.364	–
24	L. interosseous	7.9	0.093	1.139	0.517	0.956
25	L. ulnar II	17.1	0.461	0.189	0.381	0.893
26	intercoastals	8.0	0.316	0.147	0.491	0.627
27	Thoracic aorta II	10.4	3.604	0.026	0.296	–
28	Abdominal aorta I	5.3	2.659	0.032	0.311	–
29	Celiac I	2.0	1.086	0.056	0.346	–
30	Celiac II	1.0	0.126	0.481	1.016	–
31	Hepatic	6.6	0.659	0.070	0.340	0.925

Table A.1: Arterial network: Data adapted from [12] and [13]

ID	Name	$l$ (cm)	$A_0$ (cm <sup>2</sup> )	$\beta$ (10 <sup>6</sup> Pa/cm)	$C_v$ (10 <sup>4</sup> cm <sup>2</sup> /s)	$R_t$
32	Gastric	7.1	0.442	0.096	0.381	0.921
33	Splenic	6.3	0.468	0.109	0.444	0.93
34	Sup. mesenteric	5.9	0.782	0.083	0.439	0.934
35	Abdominal aorta II	1.0	2.233	0.034	0.301	–
36	L. renal	3.2	0.385	0.130	0.481	0.861
37	Abdominal aorta III	1.0	1.981	0.038	0.320	–
38	R. renal	3.2	0.385	0.130	0.481	0.861
39	Abdominal aorta IV	10.6	1.389	0.051	0.358	–
40	Inf. mesenteric	5.0	0.118	0.344	0.704	0.918
41	Abdominal aorta V	1.0	1.251	0.049	0.327	–
42	R. com. iliac	5.9	0.694	0.082	0.405	–
43	L. com. iliac	5.8	0.694	0.082	0.405	–
44	L. ext. iliac	14.4	0.730	0.137	0.349	–
45	L. int. iliac	5.0	0.285	0.531	0.422	0.925
46	L. femoral	44.3	0.409	0.231	0.440	–
47	L. deep femoral	12.6	0.398	0.223	0.419	0.885
48	L. post. tibial	32.1	0.444	0.383	0.380	0.724
49	L. ant. tibial	34.3	0.123	1.197	0.625	0.716
50	L. ext. iliac	14.5	0.730	0.137	0.349	–
51	R. int. iliac	5.0	0.285	0.531	0.422	0.925
52	R. femoral	44.4	0.409	0.231	0.440	–
53	R. deep femoral	12.7	0.398	0.223	0.419	0.888
54	R. post. tibial	32.2	0.442	0.385	0.381	0.724
55	R. ant. tibial	34.4	0.122	1.210	0.628	0.716