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Protective properties of the arterial system against peripherally generated waves

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ABSTRACT

An anatomically detailed model consisting of a network of electric transmission lines is developed to simulate propagation of the pulse waves in humans. The simulations show that the real arterial tree geometry, together with the elastic and rheological parameters of particular segments, ensure an efficient protection of vital organs against pulse waves generated at peripheral locations. Because locomotive movements are the most obvious source of such disturbances, additional cyclic perturbations are applied to the model femoral arteries. It is shown that the impact of such peripherally generated pulse waves onto the pressure profiles at the ascending aorta and at other vital locations of the system is surprisingly weak independently of synchronization/desynchronization with the heart action period. This may witness to an intrinsically protective nature of the arterial tree anatomy in addition to its known functionality of the optimal blood supply at possibly low lumen volume. The extent of the protection is also studied in the presence of a complete arterial embolism at the left common carotid artery.

1. Introduction

Since its introduction some decades ago, one-dimensional (1-D) modelling of the blood flow in deformable vessels has proven to be a simple and effective approach to simulations of the hemodynamics in the larger arteries of the body [1,2]. Numerous useful results consistent with medical records [3–6] have been obtained. Advanced modelling is needed to assist surgical procedures like radial harvesting for coronary revascularization [7] and reconstructive surgery [8,9]. In its simplest realization the model involves lengths and topology of the arterial segments as well as the propagation parameters: the phase velocity and the characteristic impedance of each segment. This allows the reflection and transmission coefficients to be calculated at each bifurcation [5,6].

In the present work we focus on the response of the whole arterial system to the pulse waves produced by the heart and to other waves generated at different arterial locations. Muscles contraction in limbs and other organs must also propagate through the system. As a first attempt, we use 55 main arteries as depicted in Fig. 1. The radii, curvatures and elastic parameters of the vessels are available in literature [1–6]. However, these parameters are usually not constant along the segments. A practical method to obtain the electric parameters from the detailed anatomical data is sketched in Section 2. The 1-D model can predict blood pressure during exercise using data collected at rest [10]. The measurement results from 11 healthy volunteers at rest and during steady exercise are available in literature [11,12].

The purpose of the present study is to give a quantitative account of the effect of physical exercise on the temporal pressure and flow profiles in the arterial system, i.e. to find out how the corresponding disturbances of pressure and flow propagate through the entire system. The problem is of practical importance because physical exercise is a significant prophylactic factor against ischemic diseases. The results may also help to define indications to the participants of spaceflights to efficiently minimize inflight overload and other disturbances. The problem is also interesting from the fundamental research point of view, namely to what extent passive models, i.e. the ones without voltage and/or current sources are capable to reproduce the physiology of the arterial system (for models of neural heart rate control via baroreceptors and reflex arc see e.g. [13–15]). Finally, the present study tests the assumption that allows one to reduce the arterial circulatory system to a network of 1-D transmission lines.

2. Propagation of pulse waves

The elasticity of vessel walls is at the origin of the wave-like character of blood flow in arteries. In principle the number of

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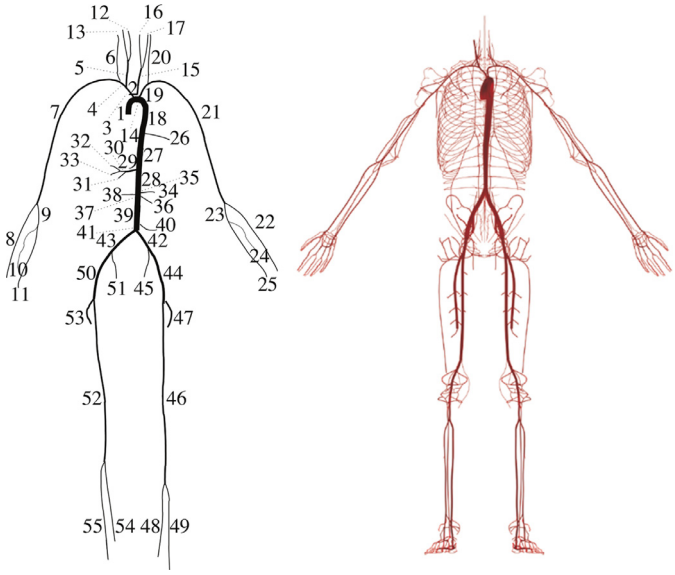

Fig. 1. The arterial tree of the human body. Left - topology of the models of the arterial network available in [10], right - real topology.

propagation modes is infinite [16,17], but the most significant effect belongs to the Young mode easily palpable at wrist. Restricting the attention to this mode allows one to treat the vessels as monomode 1-D waveguides.

The method of obtaining 1-D equation of motion in a distensible vessel of varying cross section has been developed in [10,18,19]. The governing equations involve conservation of mass and the momentum balance in a control volume of the 1-D vessel [18,19]. The volumetric flow, as a function of space x and time t, $Q(x,t) = AU$ relates the cross section area A and the average axial velocity U tethered in longitudinal direction. The pressure P is assumed constant across the section, whereas the radial and azimuthal components of velocity are neglected. Gravitational effects are ignored because all the simulations performed in this work refer to a supine subject. The system of equations is:

$$\begin{cases} \dfrac{\partial A}{\partial t} + \dfrac{\partial Q}{\partial x} = 0 \\ \dfrac{\partial Q}{\partial t} + \dfrac{\partial}{\partial x}\left(\alpha \dfrac{Q^2}{A}\right) + \dfrac{A}{\rho}\dfrac{\partial P}{\partial x} = \dfrac{f}{\rho}, \end{cases} \tag{1}$$

where ρ is the blood density, $f(x,\ t)$ is the frictional force per unit length and $\alpha(x,t) = \frac{1}{AU^2}\int_A u^2 \mathrm{d}\sigma$ is a non-dimensional profile shape factor (also called the Coriolis coefficient) that accounts for the non-linearity of the sectional integration of the velocity $\mathbf{u} = \mathbf{u}(\mathbf{x}, t)$. Here we will use the approximate value $\alpha \approx 1$ in the convective acceleration term of Eq. (1). Blood density ρ and viscosity μ of the blood are assumed to be constant at 37 °C so that $\rho = 1050$ kg m^{-3} and $\mu = 4.0$ mPa s [10]. Eq. (1) can be derived by integrating the incompressible Navier-Stokes equations over a generic cross section of a cylindrical domain [1,20–24].

In 1-D modelling the velocity profile is commonly assumed to be constant in shape and axisymmetric. A typical profile satisfying the no-slip condition $(u|_{r=R} = 0)$ is [1,20]:

$$u(x,r,t) = U\frac{\xi+2}{\xi}\left[1 - \left(\frac{r}{R}\right)^{\xi}\right], \tag{2}$$

where r is the radial coordinate, $R(x,\ t)$ is the radius of the lumen (assumed to be circular) and $\xi = \frac{2-\alpha}{\alpha-1}$ is a constant.

An explicit algebraic relationship between P and A (the tube law) is also required to close Eq. (1). The tube law is determined by the viscoelastic properties of the vessel walls [25]. Voigt-type visco-elastic laws reproduce, in the first approximation, the main effects of the walls' properties on the blood flow in large arteries, including hysteresis and creep [26–31]. An example of this type of law that neglects the effects of wall inertia and longitudinal prestress [32] is given by [31]:

$$P = P_e(A;x) + \frac{\Gamma(x)}{A_0(x)\sqrt{A}}\frac{\partial A}{\partial t} \tag{3}$$

$$\begin{aligned} P_e(A;x) &= P_{ext} + \frac{\beta(x)}{A_0(x)}(\sqrt{A} - \sqrt{A_0(x)}), \\ \beta(x) &= \frac{4}{3}\sqrt{\pi}E(x)h(x), \qquad \Gamma(x) = \frac{2}{3}\sqrt{\pi}\varphi h(x), \end{aligned} \tag{4}$$

where P_e is the elastic component of pressure, $h(x)$ is the wall thickness, $\beta(x)$ is related to the wall elasticity whereas $E(x)$ and $\Gamma(x)$ to the wall viscosity $\varphi(x)$; both independent of the transmural pressure. The reference area $A_0(x)$ is the vessel area when $P = P_{ext}$ and $\frac{\partial A}{\partial t} = 0$, which are typical initial conditions for numerical analysis. Therefore, the local cross-sectional area $A(x,\ t)$ will depend on the shape of the artery given by $A_0(x)$ and the mechanical properties of the wall, which may change with x. For example, the arterial wall becomes stiffer as the distance from the heart increases.

In the 1-D formulation the nodes connecting the arterial segments are treated as discontinuities, which is consistent with the long-wavelength approximation. Detailed 3-D calculation of flow at arterial bifurcations show that the flow is generally very complex with the possibility of transient separation and the development of secondary flows [10]. Most of these flow features are confined to the region near the bifurcation and the long wave approximation allows one to neglect their effects on the pulse wave in the 1-D formulation.

The linearization of the governing equations yields an analogy with a transmission line: where resistance R, inductance L, and capacitance C are calculated per unit length of vessel as follows:

$$R = \frac{2(\xi+2)\pi\mu}{A_0^2}, \qquad L = \frac{\rho}{A_0}, \qquad C = \frac{2A_0^{3/2}}{\beta}, \tag{5}$$

Eq. (5) allows us to determine the wave speed $c = \sqrt{\frac{1}{CL}}$ and the characteristic impedance $Z = \frac{\rho c}{A_0}$ for every segment of a given artery [10].

3. Model of the arterial tree

We solve the nonlinear 1-D Eqs. (1) and (3) using finite element methods, such as Galerkin [32] and Taylor-Galerkin (combined with operator splitting techniques) [10] schemes. The linearized system of governing equations yields an analytical solution for wave reflection and transmission where the physical properties of the arteries change. At a splitting and merging junction, the reflection coefficients R_f^a, R_f^b, R_f^c for wave propagating in the parent a and two daughter, vessels b and c respectively can be defined as the ratio of the pressure amplitude in the reflected wave to the pressure amplitude in the incident wave. They can be expressed as a function of the characteristic impedance of each segment:

$$R_f^a = \frac{(Z_0^a)^{-1} - (Z_0^b)^{-1} - (Z_0^c)^{-1}}{(Z_0^a)^{-1} + (Z_0^b)^{-1} + (Z_0^c)^{-1}}. \tag{6}$$

The transmission coefficients T_a, T_b, T_c for waves propagating in the parent, a, and two daughter, b and c, vessels can be defined as the ratio of the pressure perturbation transmitted to the other two vessels to the pressure perturbation in the vessel where the initial wave is propagated:

$$T^j = 1 + R_f^j, \qquad j = a, b, c. \tag{7}$$