European Journal of Mechanics B/Fluids 35 (2012) 111–116

Contents lists available at SciVerse ScienceDirect

European Journal of Mechanics B/Fluids

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

A lumped hydrodynamic model to assess ageing and hypertension effects on the aortic stiffness

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ARTICLE INFO

Article history:
Available online 21 January 2012

Keywords:
Zero-dimensional fluid dynamic modelling
Hypertension
Ageing
Windkessel model
Aortic impedance
Aortic stiffness

ABSTRACT

A Windkessel model is used to evaluate and separate the effects of ageing from those of hypertension on the aortic flow and pressure waveforms. The ageing- and hypertension-induced changes in the blood flow and pressure behaviour are simulated by using clinical data concerning the dependence of the Young modulus and aortic input impedance on age. The simulated pressure and flow waveforms show a typical steepening due to the ageing process. Using the model outcomes, we separate the effect of arterial tissue ageing from the effect of hypertension (here intended as a generic increase in mean blood pressure). The age-dependent component results to be more important than the pressure-dependent one especially in young and middle-aged individuals. However, the relative importance of the hypertension-driven term increases for the elderly, and reaching a value of the order of 20% becomes relevant. All the obtained results agree well with the qualitative expected behaviour.

1. Introduction

High blood pressure, termed *hypertension*, is a condition that afflicts almost 1 billion people throughout the world and is a leading cause of morbidity and mortality [1]. Persistent hypertension can lead to different life-risk events, such as strokes (also know as cerebrovascular accidents), myocardial infarction, heart failure, arterial aneurysm, renal dysfunction, and visual problems [2,3]. It has clearly been demonstrated that a moderate elevation of arterial blood pressure determines a decrease in life expectancy [3,4]. This is due to a modification of the physiological equilibrium, which entails augmented pressure wave speeds, different shapes of the velocity profile, and several changes in the composition, structure and mechanical properties of the arterial walls. These hypertension-driven changes, which usually increase with age, summed with the physiological degeneration process of the tissues entail a substantial variation in blood flow and pressure field throughout the arterial tree.

An application of a Windkessel model is here presented with the aim of evaluating and separating the effects of ageing from those of hypertension on the aortic pressure and flow. Starting from *in vivo* values of the growth in aortic wall stiffness with age, we relate this stiffening with the two main factors of influence [5]: hypertension and physiological tissue degeneration.

The adopted lumped model domain includes the first portion of the aorta, where some real geometrical features (e.g., aortic arch and bifurcation) and some mechanical characteristics (e.g., permeability and orthotropic viscoelastic behaviour) are neglected. In this simplified version, we assume that the heart pumps blood into a straight, short and uniform elastic tube.

The model is governed by a classical one-dimensional ordinary differential equation system, where, by integration on the longitudinal dimension, the above mentioned simplifications are introduced. The variables are the mean pressure and flow, and a time-dependent Young modulus and aortic input impedance are imposed. In this way, the Windkessel model is able to simulate how the flow and pressure are influenced by age-related changes in the whole arterial tree.

From the analysis of different simulations, a simple relation is introduced to describe the trend of the Young modulus as the sum of contributions caused by hypertension and ageing. We quantify how these two processes are individually responsible for the well-known Young modulus growth during ageing. The ageing component is found to be predominant in the stiffening process, especially in young and middle-aged subjects. However, the pressure dependent component reaches a relative contribution of almost 20% in the elderly.

2. Model

Let us consider a short cylindrical portion of the aorta of constant length (l), delimited by the inlet (Ω_1) and the outlet (Ω_2) sections and by the deformable lateral surface (Ω_s), as illustrated in Fig. 1. The local section is described by the area $\Omega(x, t)$ and the radius $R(x, t)$, where x is the longitudinal coordinate and t is the

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doi:10.1016/j.euromechflu.2012.01.004

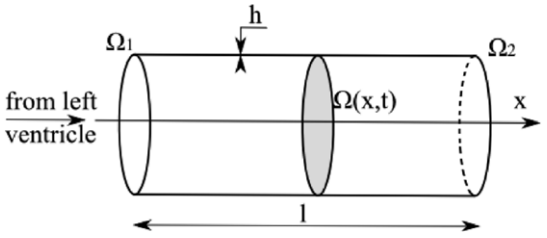

Fig. 1. Sketch of the considered domain.

time. Blood is assumed to be homogeneous and Newtonian, as usually assumed for large arteries [6]. Small variations from an initial undeformed state and laminar flow are assumed. The dependent variables are the blood flow $Q(x, t)$ and pressure $p(x, t)$.

The governing equations are

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

$$\frac{\partial Q}{\partial t} + \alpha \frac{\partial}{\partial x}\left(\frac{Q^2}{\Omega}\right) + \frac{\Omega}{\rho}\frac{\partial p}{\partial x} + \frac{2\pi R \tau_0}{\rho} = 0, \tag{2}$$

where τ_0 is the mean wall shear stress in the x direction, ρ is the blood density, and α is a correction factor that takes into account the differences between the momentum associated with a flat velocity profile and the real one. This coefficient is assumed to be unitary in the first part of the descending aorta, as suggested in [7], where the flow is similar to an undeveloped entrance-type flow [6]. For pulsating flow in slightly tapered vessels, the velocity profile is almost flat with the exception of a thin boundary layer close to the wall of width $\delta \ll R$, where velocity decays to zero for the no slip condition. According to [8], the boundary layer thickness can be estimated as $\delta = (\nu T/(2\pi))^{1/2}$ where ν is the kinematic viscosity and T is the heart period. The resistive term in (2) can be therefore written as

$$\frac{2\pi R \tau_0}{\rho} \simeq \frac{2\pi \nu R}{\delta}\frac{Q}{\Omega}, \tag{3}$$

where the viscous stress is assumed to perfectly in-phase with the mean velocity [9].

Following Pedley's notation [7], we now introduce the wall distensibility, $D = \Omega^{-1}\partial\Omega/\partial p$, which, for a thin-walled isotropic arterial segment whose length is held constant, is related to the Young modulus E according to

$$D^{-1} = \frac{E}{1-\sigma^2}\frac{h}{d}, \tag{4}$$

being σ the Poisson ratio, equal to 1/2 for incompressibility, $d(x, t)$ the diameter and h the (constant) thickness of the aortic wall.

By assuming that the walls are subjected to only a small deformation from the initial state (which is marked with the subscript 0), the constitutive law can be written following the linear elasticity theory as

$$\frac{\partial \Omega}{\partial t} = \frac{\mathrm{d}\Omega}{\mathrm{d}P}\frac{\partial p}{\partial t} = \frac{(1-\sigma^2)\Omega_0 d}{Eh}\frac{\partial p}{\partial t} = \frac{3\Omega_0 d}{4Eh}\frac{\partial p}{\partial t}. \tag{5}$$

The main assumption of the elastic theory is that the Young modulus does not vary in a significant manner for the typical range of deformation. This is consistent to a one-dimensional approach [7,10], and it is in general accepted as reasonable [9,11]. Using Ω_0 in place of $\Omega(t)$ in (5) induces very small errors when physiological conditions are accounted for [7].

The integration of Eqs. (1)–(2) along the longitudinal coordinate leads to

$$\frac{3l\Omega_0 d}{4Eh}\frac{\mathrm{d}\bar{p}}{\mathrm{d}t} + Q_2 - Q_1 = 0, \tag{6}$$

$$l\frac{\mathrm{d}\bar{Q}}{\mathrm{d}t} + \alpha\left(\frac{Q_2^2}{\Omega_2^2} - \frac{Q_1^2}{\Omega_1^2}\right) + \int_0^l \left(\frac{\Omega}{\rho}\frac{\partial p}{\partial x} + \frac{2\pi\nu R}{\delta}\frac{Q}{\Omega}\right)\mathrm{d}x = 0, \tag{7}$$

where subscripts 1 and 2 refer to the inlet and outlet sections, respectively. The overbar marks the quantity averaged along the longitudinal coordinate.

Because of the small deformation, the instantaneous local area, Ω, can be approximated with its initial lumen, Ω_0. We also assume that the second term in the momentum equation (7) gives a negligible contribution, because of the small differences between the instantaneous inlet and outlet flows and areas. Eq. (7) is therefore rewritten as

$$\frac{\rho l}{\Omega_0}\frac{\mathrm{d}\bar{Q}}{\mathrm{d}t} + \frac{\rho 2\pi\nu R l}{\delta\Omega_0^2}\bar{Q} + (P_2 - P_1) = 0, \tag{8}$$

where P_1 and P_2 refer to the pressures averaged over the inlet and outlet sections, respectively. The coefficients of Eqs. (6) and (8) have the following meanings: the term $3l\Omega_0 d/(4Eh)$ quantifies the aorta compliance due to the wall elasticity, $\rho l/\Omega_0$ considers the inertia of the blood and $\rho 2\pi\nu R l/(\delta\Omega_0^2)$ assesses the resistance to the flow due the walls.

As we model the initial part of the aorta, the outflow and pressure characteristics of the pumping heart are set as inlet boundary conditions. With the aim of simulating the heart outflow cycle, its characteristic period is divided into the two standard physiological phases: diastole and systole. During the diastolic period, the flow is assumed to be null. The heart outflow in the systolic phase is simply described by a sine function with a frequency of 1.25 Hz, corresponding to a heart rate of 75 bpm [12]. The corresponding pressure is obtained from the inlet blood flow using the input impedance Z as:

$$P_1 = Q_1 Z(y), \tag{9}$$

where y is the age (in years). Using an age-dependent input impedance, it is possible to change the inlet pressure waveform to fit the different physiological conditions, following the natural variations that occur in the ageing process.

3. Young modulus and aortic input impedance modelling

Arterial ageing is characterized by a degenerative process, associated with remarkable quantitative and qualitative changes that mainly affect elastin, a major component of the extracellular matrix [3,13–15]. In elderly subjects, elastin fibres, which make up approximately 90% of the arterial elastic fibres, are more disorganized, sparser, thinner, and more fragmented than those of younger individuals [14–16]. Furthermore, the degraded elastin in arteries is gradually replaced by a much stiffer collagen [3,16]. At the same time, elastin may become calcified [16], while the collagen molecules acquire cross-links [3].

The main effects of this degenerative process consists of a growth in the arterial wall thickness and stiffness. These changes are mainly due to alterations in the structural and physical properties of the arterial wall [13] and are present during the normal ageing of population [17,18]. Age-related intima thickening also appears to be partly pressure-dependent, since it has been stopped or reduced by experimental blood pressure reductions in rats [19] and humans [20]. It is well-known that hypertension, which usually increases with age, has an important effect on the age-related increase in aortic pulse wave velocity [16,17],