

Lumped Parameter Outflow Models for 1-D Blood Flow Simulations: Effect on Pulse Waves and Parameter Estimation

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Received 19 October 2007; Accepted (in revised version) 10 December 2007

Available online 18 March 2008

Abstract. Several lumped parameter, or zero-dimensional (0-D), models of the micro-circulation are coupled in the time domain to the nonlinear, one-dimensional (1-D) equations of blood flow in large arteries. A linear analysis of the coupled system, together with *in vivo* observations, shows that: (i) an inflow resistance that matches the characteristic impedance of the terminal arteries is required to avoid non-physiological wave reflections; (ii) periodic mean pressures and flow distributions in large arteries depend on arterial and peripheral resistances, but not on the compliances and inertias of the system, which only affect instantaneous pressure and flow waveforms; (iii) peripheral inertias have a minor effect on pulse waveforms under normal conditions; and (iv) the time constant of the diastolic pressure decay is the same in any 1-D model artery, if viscous dissipation can be neglected in these arteries, and it depends on all the peripheral compliances and resistances of the system. Following this analysis, we propose an algorithm to accurately estimate peripheral resistances and compliances from *in vivo* data. This algorithm is verified against numerical data simulated using a 1-D model network of the 55 largest human arteries, in which the parameters of the peripheral windkessel outflow models are known *a priori*. Pressure and flow waveforms in the aorta and the first generation of bifurcations are reproduced with relative root-mean-square errors smaller than 3%.

AMS subject classifications: 92C35, 35L45

Key words: Pulse wave propagation, one-dimensional modelling, lumped parameter outflow models, time-domain coupling, arterial compliance, peripheral compliance, peripheral resistance, multiscale modelling.

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1 Introduction

Arterial pulse wavelengths are sufficiently long to mathematically justify the use of a one-dimensional (1-D) rather than a three-dimensional (3-D) approach when a global assessment of blood flow in the cardiovascular system is required. Several comparisons against *in vivo* [19, 26] and *in vitro* [2, 16] data have shown the ability of the nonlinear, 1-D equations of blood flow in compliant vessels [11, 14, 21, 23, 29] to capture the main features of pressure and flow waveforms in large arteries. These tests have increased our confidence in applying the 1-D formulation to clinically relevant problems [3–5, 13, 25, 26, 28] or to provide the boundary conditions for 3-D simulations [10]. However, the clinical relevance of 1-D modelling is subject to the availability of patient-specific data on the geometry, local pulse wave speeds, and boundary conditions of the arterial network to be simulated.

Recent progress in imaging technology has opened greater possibilities for the application of 1-D modelling. Imaging techniques such as computer tomography, magnetic resonance and ultrasound are now able to provide patient-specific information on vessel geometry as well as more limited information on local velocity profiles and pulse wave speeds. This information permits the use of the 1-D formulation to simulate patient-specific arterial networks provided that appropriate boundary conditions are prescribed. Although the inflow waveform at the root of the arterial model (typically at the ascending aorta) can be accurately measured at salient locations using medical imaging, determination of outflow boundary conditions based on measured data is more challenging.

Even if such data were available, it is computationally too expensive to model all vessels in the full systemic circulation using the 1-D formulation because of their large number, which increases exponentially as more generations of the arterial tree are introduced. Furthermore, the assumptions of the 1-D equations become less appropriate with the decreasing caliber of the vessels. For instance, blood flow in large arteries is pulsatile and dominated by inertia, whereas blood flow in smaller vessels is quasi-steady and dominated by viscosity [7]. Consequently, any 1-D model has to be truncated after a relatively small number of generations of bifurcations, and the haemodynamic effect of vessels beyond 1-D model arteries is typically simulated using lumped parameter or zero-dimensional (0-D) models governed by ordinary differential equations that relate pressure to the flow at the outflow of each 1-D terminal vessel [3–5, 13, 25, 26, 28]. Alternatively, the remainder of the arterial system can be simulated using structured tree models based on Womersley's elastic vessel theory under the assumption of periodic flow [18, 19].

The aim of this investigation is to provide appropriate outflow 0-D models for patient-specific simulations and to propose a strategy to estimate their parameters using data that can be measured *in vivo*. Several physiologically relevant 0-D models are coupled to the nonlinear, 1-D formulation using a time-domain algorithm that can accommodate periodic and transient phenomena. The resulting 1-D/0-D multiscale formulation is linearized to study the main effects of 0-D outflow parameters on pulse wave propagation in 1-D model arteries, and to devise a strategy to select the parameters of the outflow mod-