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Pressure wave propagation in full-body arterial models: A gateway to exploring aging and hypertension

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Abstract

It is now widely recognized that changes in arterial wall properties have a significant impact on hemodynamic indices such as pressure pulse amplification and pulse wave velocity. It is also becoming increasingly evident that changes in wall mechanics may progress both spatially and temporally (e.g., in age-related arterial stiffening and hypertension). Modeling studies can help delineate how local changes in stiffness affect global hemodynamics. Previously, several modeling studies have investigated blood and pressure in full-body scale arterial trees using one-dimensional formulations. In this paper, we work towards the goal of deepening our understanding of arterial pulse propagation phenomena while incorporating detailed information on localized hemodynamics. To this end, we present the first multi-scale simulation of unsteady blood flow and pressure within a three-dimensional deformable full-body arterial network. This simulation framework builds upon previous advances in fluid-structure interaction, multi-scale outflow boundary conditions, and perivascular tissue support modeling. We consider application examples featuring realistic distributions of spatially and temporally varying mechanical properties. Simulations successfully demonstrate realistic pressure and flow waveforms, regional blood flow distribution, pressure pulse amplification and pulse wave velocity.

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1. Introduction

Because of the complex geometry and material properties of the central arteries, and the

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hemodynamics therein that also depend on effects arising from distal conduit and resistance vessels, large scale computational models of vascular mechanics are needed to improve our ability to interpret current clinical findings and to advance our fundamental understanding of the mechanisms of disease progression in arterial hypertension. There have been several seminal computational studies of full-body scale hemodynamics within the arterial system [1–3]. These studies have described the physics of flow and pressure wave propagation in a one-dimensional setting. In this paper, we present the first multi-scale computational model of fully three-dimensional and unsteady hemodynamics within the primary large arteries in the human, literally from head to foot, while including important effects arising from the distal resistance vessels. This model is built upon previous key advances in multi-scale inflow and outflow boundary condition formulations [4], computationally efficient methods for fluid-structure interaction [5] and perivascular tissue support [6], and methods for anisotropic field-driven mesh adaptation [7]. Resulting computational findings on local pressure waves promise to relate common clinical measurements such as radial pressures to the more important but less easily measured *cPP*, which has greater prognostic value [8]. Computer simulations may establish these relationships in a more rigorous fashion than through the use of transfer functions whose accuracy has been disputed. Similarly, computational findings on the effects of local changes in wall properties on global metrics such as carotid-femoral pulse wave velocity (CF-PWV) promise to provide increased insight into relationships between evolving wall properties and the temporal progression of hypertension and aging-related changes in hemodynamics. Finally, detailed information on local pressure and velocity fields promise to enable new mechanobiological hypotheses regarding large artery disease to be formulated and tested. Whereas examination of these and other important areas of vascular therapeutics and biology will be pursued subsequently, the primary goal of this work is to set the stage by developing an underlying theoretical and computational framework.

The structure of this paper is as follows: After reviewing the medical image data and geometric modeling techniques, we describe the multi-scale blood flow modeling framework, specifically the formulations for inflow/outflow boundary conditions, fluid-structure interaction and perivascular tissue support. We then present the methodology adopted for vessel wall material parameter specification and outflow boundary conditions. Then, in the results section, we report detailed hemodynamics in a full-body scale model representing the large arteries of the human body, expanding the trunk model to include the main arteries in the legs, arms, and head, for a total of 82 outflow faces. We highlight the accuracy of simulation results such as regional blood flow, pressure and flow waveforms, and pressure pulse propagation down the aorta. We demonstrate the capabilities of the proposed framework to produce head-to-toe three-dimensional subject-specific hemodynamics that may significantly enhance our current understanding of wave propagation phenomena and its relationship with arterial stiffening within the human vasculature. We conclude with discussion remarks on the progress made thus-far in the field of multi-scale computational modeling of arterial hemodynamics, on the pressing need to develop experimental techniques to measure *in vivo* tissue mechanical parameters required by the ever more sophisticated computational models, and on the potential of computational modeling to identify improved indicators of stiffening that may allow earlier therapeutic interventions.

2. Methods

2.1. Image data and anatomical reconstruction

2.1.1. Medical image data

We combined computed tomographic angiography (CTA) image data from two different patients to reconstruct a nearly complete network of the major arteries from the head to the lower legs. Two datasets

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