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Simulation of abdominal aortic aneurysm growth with updating hemodynamic loads using a realistic geometry

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ABSTRACT

Advances in modeling vascular tissue growth and remodeling (G&R) as well as medical imaging usher in a great potential for integrative computational mechanics to revolutionize the clinical treatment of cardiovascular diseases. A computational model of abdominal aortic aneurysm (AAA) enlargement has been previously developed based on realistic geometric models. In this work, we couple the computational simulation of AAA growth with the hemodynamics simulation in a stepwise, iterative manner and study the interrelation between the changes in wall shear stress (WSS) and arterial wall evolution. The G&R simulation computes a long-term vascular adaptation with constant hemodynamic loads, derived from the previous hemodynamics simulation, while the subsequent hemodynamics simulation computes hemodynamic loads on the vessel wall during the cardiac cycle using the evolved geometry. We hypothesize that low WSS promotes degradation of elastin during the progression of an AAA. It is shown that shear stress-induced degradation of elastin elevates wall stress and accelerates AAA enlargement. Regions of higher expansion correlate with regions of low WSS. Our results show that despite the crucial role of stress-mediated collagen turnover in compensating the loss of elastin, AAA enlargement can be accelerated through the effect of WSS. The present study is able to account for computational models of image-based AAA growth as well as important hemodynamic parameters with relatively low computational expense. We suggest that the present computational framework, in spite of its limitations, provides a useful foundation for future studies which may yield new insight into how aneurysms grow and rupture.

1. Introduction

Understanding of the underlying processes that lead to the growth and structural weakening of an abdominal aortic aneurysm (AAA) is of critical importance in both diagnosis of the lesion progression and design of the patient-specific intervention. AAAs have been associated with local and systemic alterations of the aorta, influenced by age as well as genetic factors [1–3]. Marked reduction of the elastin content in AAA tissues has been reported in several studies [4–7]. It has been suggested that elastin degradation is attributed to the elevated activation of proteolytic matrix metalloproteinases (MMPs) that can be induced by various factors such as the abnormal distribution of wall shear stress (WSS) [8–11], inflammatory responses [12–14], and intraluminal thrombus formation [15,16]. Although it has been suggested that aneurysm growth is likely to occur in regions where the vessel wall is exposed to abnormally high/low WSS, the effect of WSS on the expansion rate of aneurysms is poorly understood. High WSS has been related to the initiation of cerebral aneurysms [17,18], whereas low shear has been associated with aneurysm progression [19], thrombus formation [20] and its rupture [18,21]. In this study, we test the hypothesis that an adverse decrease in WSS promotes elastin degeneration, and use computational simulations to track the possible time course of changes in the mechanical state of the aortic wall and its effects on processes governing the aneurysm expansion.

Although an AAA is often characterized by a thinning media with marked reduction of elastin, increasing evidence suggests that AAA formation is predominantly due to the growth and remodeling (G&R) of the aortic wall by collagen turnover [12,22,3]. Based on understandings of the ubiquitous role of mechano-regulated G&R of collagen in vascular adaptations, a number of computational models of (cerebral/aortic) aneurysm expansion have been developed where the stress/strain-mediated collagen turnover governs the expansion rate [23–29]. The previous computational models have been promising in improving our understanding of the underlying mechanisms involved in aneurysm enlargement. In the cerebral aneurysm model proposed in [24], collagen was assumed

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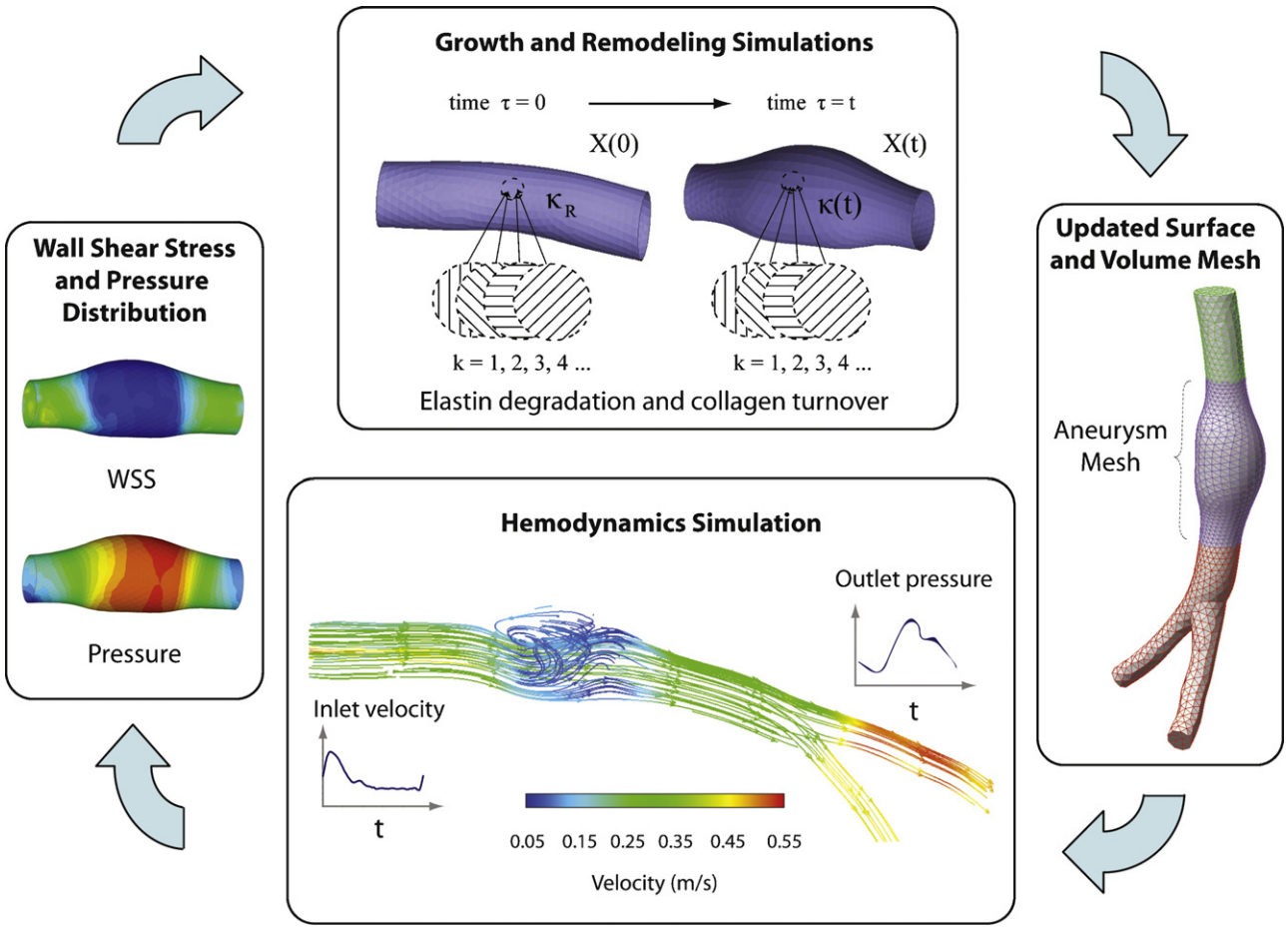

Fig. 1. Iterative loop and information transfer in the coupling between the hemodynamics and G&R simulations.

to be the only structural constituent responsible for the aneurysm enlargement [30,31]. However, elastin and smooth muscle (SM) cells are major components in abdominal aorta, and their continuous degradations and coupled interactions with stress-mediated collagen turnover are speculated to be the main cause for AAA enlargement. We employed a similar stress-mediated adaptation model and extended it to aortic aneurysms using an anatomically realistic geometry and studied the effect of spatial and temporal variations of elastin degradation on the intramural stress distribution and the subsequent aneurysm enlargement [32]. For clinical applications of these models, however, it is strongly desired to integrate the computational models of G&R with hemodynamics simulations to account for the role of hemodynamics variations. Recently, new computational frameworks that loosely couple vascular G&R simulation with hemodynamics simulation have been independently presented by Figueroa et al. [33] and Watton et al. [29], demonstrating their utility in modeling cerebral aneurysms using idealized geometries. In the present study, we employ the coupled framework by extending our previous AAA model [32] to simulate the evolution of an AAA while updating hemodynamic loads.

2. Methods

2.1. General computational framework

Following [33], we employ a fluid–solid-growth (FSG) simulation framework that utilizes loosely coupled iterations between short-term hemodynamics simulations and long-term G&R simulations, i.e., the hemodynamic loads on the vascular wall are updated in a stepwise manner as the AAA grows (Fig. 1). More specifically, in the iterative loop, the hemodynamics simulation computes blood flow during the cardiac cycle at a given time and the mechanical stimuli that affect vascular wall G&R (e.g., mean WSS and mean pressure) are extracted and transferred to the G&R simulations. The G&R simulation then simulates the evolution of the arterial wall over multiple time steps. When the shape of an AAA changes, the new shape is combined with extended (proximal and distal) regions and fed back to the hemodynamics simulation.

To simulate AAA enlargement, the central region of the abdominal aorta is used, and in order to obtain more accurate hemodynamic loads, the computational domain for the hemodynamics simulation is extended to the upper part of abdominal aorta (proximal side) and iliac branches (distal side). To characterize the hemodynamics within the blood vessel, unsteady blood flow is simulated within the reconstructed geometry using Fluent (Fluent Inc., Lebanon, NH, USA). A periodic velocity field corresponding to a prescribed inlet flow rate is used as an inlet boundary condition, and a periodic outlet back pressure is used as the outlet boundary condition. Lastly, the blood vessel is treated as having a rigid and impermeable wall.

Mean (time-averaged) WSS and the mean pressure are calculated for all nodes on the aneurysm wall over one cardiac cycle and transferred to the G&R simulation. The G&R part simulates the vessel wall adaptation accounting for elastin degradation and stress-mediated collagen turnover, both of which depend on the mechanical stimuli calculated from the hemodynamics simulation. For the G&R simulation, we use the finite element model of AAA enlargement developed by Zeinali-Davarani et al. [32], briefly described in the next section.