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In silico characterization of the effects of size, distribution, and modulus contrast of aortic focal softening on pulse wave propagations

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Abstract Examining the change in regional stiffness of the arterial wall may prove as a reliable method for detecting various cardiovascular diseases. As suggested by Moens–Korteweg relationship, the pulse wave velocity (*PWV*) along the arteries has been shown to correlate to the stiffness of the arterial wall; the higher the stiffness, the higher the *PWV*. The current primary clinical practice of obtaining an average *PWV* between remote sites such as femoral and carotid arteries is not as clinically effective, since various cardiovascular diseases are shown to be accompanied by focal changes in stiffness. Therefore, methods to examine the *PWV*s focally are warranted. Extending on the findings of previous studies, pulse wave propagations along aortas with wall focal *softening* were addressed in this study using two-way coupled fluid–structure interaction (FSI) simulations of arterial pulsatile motions. Spatio-temporal maps of the wall displacement were used to evaluate the regional pulse wave propagations and velocities. In particular, soft wall inclusions of different number, size, and modulus were examined. The findings showed that the qualitative markers on the pattern of the wave propagations such as the existence of forward, reflected, and standing waves, as well as the quantitative markers such as *PWV*, linear coefficient of the propagating waves, and the width of the standing waves, provide a reliable tool to distinguish between the natures of the wall focal softening. Future studies are needed to include physiologically-relevant wall inhomogeneity in order to further implicate on the clinical potentials of the inverse problem for noninvasive diagnosis.

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Introduction

The majority of cardiovascular diseases (CVDs) have been shown to be accompanied by alterations in mechanical properties of the arterial wall.[1] Changes in aortic wall stiffness has been widely agreed as an independent indicator of CVDs such as aortic calcification and aneurysm.[2–10] Determining arterial stiffness has been known to be a part of clinical diagnosis, therapy, and follow up procedures.[11,12] The established concept based on the Moens–Korteweg relationship has proven that the velocity of the aortic pulse wave is correlated to the arterial stiffness; the stiffer the wall, the higher the pulse wave velocity (*PWV*),[13–15] and therefore can be used to estimate the wall stiffness noninvasively based on *PWV* measurements. Current clinical practice for measuring the *PWV* includes acquiring the temporal pulse pressure profiles at carotid and femoral artery sites[11,16] and then obtaining the average *PWV* via dividing the over-the-skin distance by the time delay between the two pulse profiles.[17,18] There are various inaccuracies associated with this method mostly due to the incorrect arterial geometry and/or assuming a single longitude flow direction between the carotid and femoral arteries which leads to an underestimation or overestimation of the true traveled distance and the *PWV* therein.[11,19] Furthermore, the wall stiffness has been shown to vary locally along the artery[20] which makes an average *PWV* not properly representing or detecting the associated stiffness variations. Therefore, this current clinical practice may prove not efficient in diagnosing a majority of CVDs initiation and progression that have been shown to entail focal tissue degradation.[21–23]

Several approaches have been taken to overcome the aforementioned issues. The first approach is to use mathematical models for providing a local approximation of the PWV using measurable parameters such as blood pressure, velocity, arterial diameter, flow rate, and luminal area.[24] The second approach includes an ultrasound based method of Pulse Wave Imaging (PWI), which also provides a local estimate of the *PWV*,[25,26] with the feasibility studies shown on different frontiers such as animal studies in vivo and in vitro, phantom studies, and clinical studies.[27–32] The efficacy and reliability of both methods to pathological conditions involving high focal variations has been found compromised since such wall variations create complex wave propagation dynamics consisting of multiple forward and reflected waves that make the wave quantification and *PWV* measurement challenging.[27,32–34] In order to shed light on the pulsatile wave dynamics in aortas with focal inhomogeneities, Fluid Structure Interaction (FSI) simulations could be very helpful. The feasibility of using Coupled Eulerian Lagrangian (CEL) solver of Abaqus (*Simulia*, *RI*, *USA*) in modeling fluid-induced aortic pulse waves has been shown with findings being validated against phantom and in vitro studies.[30,31] Particularly, recent studies have shown the efficacy of CEL-based simulations in reliably detecting the existence of focal stiffening and softening of the aortic walls.[35] In addition, a further study confirms the establishment of qualitative and quantitative markers that are present when stiffening heterogeneities of the wall is created in terms of number, size, and modulus of focal hard inclusions,[36] which can potentially be used for PWI-based diagnosis purposes. Building on the infrastructure of the previous studies, this study further extends the quantified findings on the pulse wave propagations to the aortas with wall focal *softening*, aiming at identifying the relevant diagnostic markers.

Methods

Computational modeling

A Dell Precision™ with Intel Core i7-3840QM and 32 GB Ram was used to perform dynamic two-way Fluid Structure Interaction (FSI) simulations of pulse wave propagations along the walls of a 3D aortic geometry. The Coupled Eulerian-Lagrangian (CEL) explicit solver of Abaqus 6.11–1 (*Simulia*, *RI*, *USA*) was used to describe the fluid dynamics and to capture the fluid–solid interactions. Defining the initial position of a moving particle in the material at the reference time, the new position of the same particle at the current time and the resulted velocity and acceleration can be obtained either in Lagrangian coordinate system (*e.g.* such as for the motion of the wall material), or the Eulerian coordinate system (*e.g.* such as for the motion of the fluid material), and based on the principle of coordinate-invariance, the displacement, velocity and acceleration fields obtained from either coordinate system are equal. In the CEL solver for the finite element method, the motions of the particles in fluid (*i.e.* flow) are formulated in Eulerian coordinate system, in which the mesh topology consists of elements that are affixed in the space while material is allowed to cross in/out of the element boundaries. However, the motions for the solid (*i.e.* aortic wall) domains are formulated using Lagrangian coordinate system, where the elements are affixed to and move with the particles during the material deformation. In the present model, the Lagrangian part was constructed as a straight cylindrical geometry ($L = 250$ mm; $d_i = 24$ mm; $h = 2.2$ mm), with a Young's modulus of $E_w = 5.12$ MPa, density of $\rho_w = 1050$ kg/m^3, and Poisson's ratio of $\nu_w = 0.48$.[37–39] The Eulerian domain was established to encompass the entire Lagrangian domain in order to accommodate the potential presence of fluid and the FSI thereof on the deformed geometries at all times during the simulation. Without the loss of generality in generating relevant wave dynamics, fluid was assumed to be Newtonian, with material properties as density $\rho_f = 1000$ kg/m^3, reference sound speed $c_f = 1483$ m/s and viscosity $\eta_f = 0.0001$ N/m^2.[40,41] Boundary conditions on the inlet and outlet were applied as full constraint in all 6 degrees of freedom. A pulse profile with magnitude $V_0 = 5$ m/s was applied as the inlet flow, acting as the drive in generating the FSI-induced pulsatile motions in the wall. The pulse profile is a smooth step function over time. The flow parameters were chosen in consistency with similar numerical and experimental studies,[30,31,35,36] primarily in order to induce strong enough displacement in the aortic walls, so the resulted wave propagations would be fully detectable. Step increases in amplitude from 0 to 1 every 0.001 *s*. A frictionless FSI interface was defined between the wall and