WALL DISTENSIBILITY MODERATELY AFFECTS WALL SHEAR STRESS TOPOLOGICAL SKELETON AT THE CAROTID BIFURCATION

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Introduction

A link between wall shear stress (WSS) topological skeleton (TS) features and the onset of atherosclerosis in arteries has recently emerged [1,2]. The WSS TS, which is composed of WSS contraction/expansion regions linking fixed points (where WSS vanishes), can be investigated using computational hemodynamics. However, most of the studies on arterial WSS TS were performed adopting computational fluid dynamics (CFD) simulations under the rigid wall assumption. The unexplored effect of wall distensibility is here determined by carrying out fully coupled two-way fluid-structure interaction (FSI) simulations.

Methods

Subject-specific diastolic fluid domains of five healthy carotid bifurcations and their respective wall thicknesses were reconstructed from magnetic resonance (MR) angiography [3], including 15 radii of the proximal common carotid artery (CCA). Subject-specific flow rates from MR measurements [3] were used for the CCA inflow boundary condition (BC) and for tuning three-element Windkessel models at the external and internal carotid artery (ECA and ICA) outlets. Arbitrary Lagrangian-Eulerian formulation-based FSI simulations were carried out modelling the vessel wall as a fibre-reinforced anisotropic nonlinear material implementing the Holzapfel-Gasser-Ogden model [3]. The initial loading state and the collagen fibres orientations were obtained through prestress of the vessel wall [3]. Viscoelastic external support was accounted for by imposing a Robin-type BC [4]. Rigid wall CFD simulations were carried out adopting the same fluid mesh and simulation settings as FSI. All simulations were carried out in Simvascular [4].

In addition to the canonical WSS-based indicators time-average WSS (TAWSS) and oscillatory shear index (OSI), WSS TS was analysed according to a Eulerian-based method [1]. WSS contraction/expansion regions were identified by negative/positive values of the divergence of the WSS unit vector field $\boldsymbol{\tau_u}$. WSS fixed points were identified and classified by computing the Poincaré index and the eigenvalues of the Jacobian matrix [1]. The variability of the WSS contraction/expansion action was quantified by the topological shear variation index (TSVI) [1]. Pooled 20th percentile (TAWSS) and 80th percentile (OSI and TSVI) values were used to quantify the relative surface area (SA) exposed to TAWSS below (OSI, TSVI above) the corresponding threshold value.

Results

Fixed points type (saddle points, stable/unstable nodes, and foci), location, and the extent and strength of contraction/expansion regions were comparable between rigid and distensible cases (as exemplified in Fig. 1). TAWSS, OSI, and TSVI presented similar distributions as well (TSVI distributions are presented in Fig. 1 for one case). Absolute differences in SAs extension between rigid and distensible simulations were 2.3±1.9% (TAWSS), 1.0±0.8% (OSI), and 3.0±3.1% (TSVI, as detailed in the rank plot of Fig. 1).
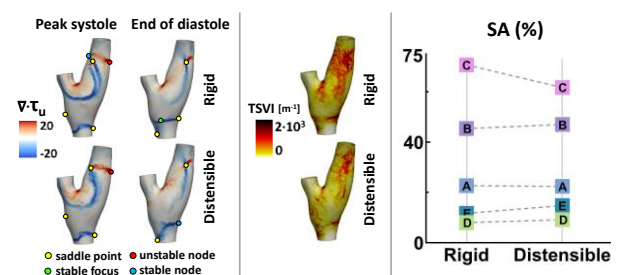

Figure 1: WSS TS (left panel) and TSVI distribution (mid panel) for a representative case (case C). Rank plot of SAs exposed to high TSVI (right panel).

Discussion

WSS TS features were only moderately affected by wall distensibility. Considering the increased complexity, cost, and inherent introduction of additional uncertainties to model wall distensibility, our findings suggest that rigid wall CFD simulations reasonably catch the WSS TS features of biomechanical interest. This is particularly relevant in view of a future clinical translation of CFD simulations [2], because of their reduced computational cost. However, the implemented FSI approach will allow the exploration of possible distinct or synergistic effects of hemodynamic and wall structural stimuli on atherosclerosis initiation.

References

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