

VORTICITY TRANSPORT-BASED ANALYSIS OF THE ABDOMINAL AORTIC ANEURISM HEMODYNAMICS

Valentina Mazzi (1), Karol Calò (1), Maurizio Lodi Rizzini (1), Ludovica Saccaro (2,3), Diego Gallo (1), Angelo Iollo (2,3), Umberto Morbiducci (1)

1. *Polito^{BIO} Med Lab, Department of Mechanical and Aerospace Engineering, Politecnico di Torino, Italy*; 2. *IMB, UMR 5251, Université de Bordeaux, Talence, France*; 3. *Inria - Bordeaux Sud-Ouest, Talence, France*

Introduction

The Abdominal Aortic Aneurysm (AAA) is a vascular disease characterized by an unphysiological bulging of the abdominal aortic lumen. It has been recognized that local hemodynamics contribute to AAA progression because of its crucial role in transport of biochemicals and interaction with the wall [1]. In this context, a multitude of hemodynamic quantities has been proposed to provide biomechanical markers of disease evolution. However, these proposed quantities turned out to be only moderately associated with AAA progression, suggesting that a different perspective to decipher AAA flow complexity is needed. In this scenario, we propose a thorough characterization of the complex vortex structures produced and transported in the AAA, aiming at identifying vorticity-based quantities to be tested as potential markers and predictors of AAA evolution.

Methods

The geometry of eight AAAs was reconstructed from CT-scan data using PRAEVAorta (<https://www.nurea-soft.com>) [2]. The governing equations of fluid motion were numerically solved using the finite element open-source code SimVascular [3]. The AAA velocity data were used to compute the vorticity transport equation:

$$\frac{D \boldsymbol{\omega}}{D t} = (\boldsymbol{\omega} \cdot \nabla) \mathbf{u} + \nu \Delta \boldsymbol{\omega} \quad (1)$$

where \mathbf{u} is the velocity, $\boldsymbol{\omega}$ is the vorticity and ν is the kinematic viscosity. The material derivative of the vorticity is given by the contribution of a stretching term (first term on the right side), quantifying the vortex lengthening due to velocity gradients, and of a term quantifying vorticity diffusion due to viscosity (second term on the right side). Moreover, the local *swirling strength*, quantified by the absolute value of the imaginary part of the complex eigenvalue of the velocity gradient tensor, was here analyzed to reconstruct the vortex dynamics.

Results

Volumetric maps at mid-deceleration point and time-histories of the volume-average value of the swirling strength and of the stretching term in eq. (1) are presented in Figure 1A, B for three explanatory models (AAAI presents a quasi-physiological blood flow canalization, AAII is characterized by two consecutive expansions and AAIII presents a marked expansion). A pressure-gradient mechanism induces the formation of a well-defined vortex ring in AAII and AAIII, as the inflow jet enters the AAA expansion region.

Subsequently, it undergoes stretching and tilting, as confirmed by second peak of time-histories. The vortex ring dynamics along the systolic deceleration phase is analyzed in Figure 1C for AAII and AAIII by visualizing local swirling strength isosurfaces and vortex core lines. Contrarily to AAII and AAIII, the inflow jet does not roll up into a vortex ring in model AAAI, because of its quasi-physiological geometry.

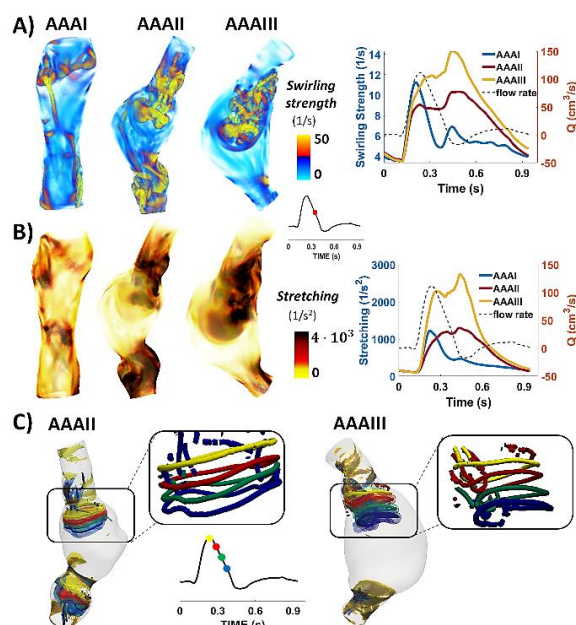


Figure 1: Volumetric maps and time-histories of volume-average value of A) swirling strength and B) stretching in three representative AAA models. C) Swirling strength isosurfaces and vortex core lines at different times along the cardiac cycle in AAII and AAIII.

Discussion

Here a vorticity transport-based analysis is proposed on computational hemodynamics models of AAA, aiming at describing complex vortex structures hidden in the AAA hemodynamic richness. These structures are expected to be involved in intraluminal thrombus formation, inflammatory mechanisms, and platelets dynamics [4]. The presented approach might represent a useful tool for elucidating the link between intravascular blood flow patterns and clinical observations.

References

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