Flow stagnation volume and abdominal aortic aneurysm growth: Insights from patient-specific computational flow dynamics of Lagrangian-coherent structures

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ABSTRACT

Abdominal aortic aneurysms (AAA) are localized, commonly-occurring dilations of the aorta. When equilibrium between blood pressure (loading) and wall mechanical resistance is lost, rupture ensues, and patient death follows, if not treated immediately.

Experimental and numerical analyses of flow patterns in arteries show direct correlations between wall shear stress and wall mechano-adaptation with the development of zones prone to thrombus formation. For further insights into AAA flow topology/growth interaction, a workout of patient-specific computational flow dynamics (CFD) is proposed to compute finite-time Lyapunov exponents and extract Lagrangian-coherent structures (LCS).

This computational model was first compared with 4-D phase-contrast magnetic resonance imaging (MRI) in 5 patients.

To better understand the impact of flow topology and transport on AAA growth, hyperbolic, repelling LCS were computed in 1 patient during 8-year follow-up, including 9 volumetric morphologic AAA measures by computed tomography-angiography (CTA). LCS defined barriers to Lagrangian jet cores entering AAA. Domains enclosed between LCS and the aortic wall were considered to be stagnation zones. Their evolution was studied during AAA growth.

Good correlation – 2-D cross-correlation coefficients of 0.65, 0.86 and 0.082 (min, max, SD) – was obtained between numerical simulations and 4-D MRI acquisitions in 6 specific cross-sections from 4 patients. In follow-up study, LCS divided AAA lumens into 3 dynamically-isolated zones: 2 stagnation volumes lying in dilated portions of the AAA, and circulating volume connecting the inlet to the outlet. The volume of each zone was tracked over time. Although circulating volume remained unchanged during 8-year follow-up, the AAA lumen and main stagnation zones grew significantly (8 cm³/year and 6 cm³/year, respectively).

This study reveals that transient transport topology can be quantified in patient-specific AAA during disease progression by CTA, in parallel with lumen morphology. It is anticipated that analysis of the main AAA stagnation zones by patient-specific CFD on a yearly basis could help to predict AAA growth and rupture.

1. Introduction

Abdominal aortic aneurysms (AAA) are localized dilations of the abdominal aorta which can rupture if equilibrium between artery resistance and blood pressure is lost. AAA prevalence reaches 8.9% among men and 2.2% among women: it is the 14th leading cause of death in the USA [29]. AAA risk factors are typically the same as for atherosclerosis, i.e., gender (male), smoking, age, hypertension and hyperlipidemia [29], but genetic factors are also believed to contribute to AAA development, growth and rupture.

AAA are usually asymptomatic and are therefore often detected through unrelated examinations or dedicated screenings, such as echography or X-ray computed tomography (CT-scan). Once detected, AAA risk assessment is generally based on maximal diameter [38] (Dmax criterion). Patients will undergo elective surgery if Dmax is over a statistically-based threshold: 55 mm for men and less for women [10]. If Dmax is below these values, AAA are examined yearly by ultrasound or CT-scan, until the surgical threshold is reached or if AAA Dmax increases
more than 1 cm per year [12]. Because of poor diagnostic performance of $D_{\text{max}}$ [67], new metrics have been introduced for better AAA-rupture risk assessment [10]. Efforts were first made to provide a better standard for maximum diameter [38]. The increasing availability of patient-specific 3-D AAA models from computed tomography angiography (CTA) now makes individualized hemodynamic analyses possible, with blood flow simulation, fluid-solid interaction and multiphysics modeling. The latter couple mechanics with biology, enabling, for example, simplified simulations of AAA growth [19,63,70] by replicating the evolution of wall composition and rheology.

The key factors that differentiate one AAA from another are geometric shape, mechanical tissue properties and flow topology [57]. Some morphological features with known linkage to AAA rupture risk are, indeed, volume, surface, bulge height, tortuosity and local surface curvature [54,57]. Besides the above geometric attributes, mechanical wall stresses in AAs depend on tissue properties, which are essentially heterogeneous and nonlinear [44] and patient specific [46]. As it is impossible to fully characterize such properties as well as complex micro-to macro-scale interconnections, handling generalized numerical models for rupture risk prediction becomes difficult. Blood flow and its altered topology are known to play a key role in both wall-fluid shearing action and transport perturbations in AAA evolution [4,53].

Several metrics can efficiently account for the wall-shearing action [25,41], but evaluation of transport changes in complex AAA flow remains challenging.

The presence of thrombus deposits over the AAA wall modifies its composition and mechanical behavior [69]. Thrombus formation [7] has been linked to flow separation in the dilated portion of the aorta. The present study was motivated by the need for new tools to understand the contribution of fluid transport changes to AAA and thrombus growth.

The concept of Lagrangian-coherent structures (LCS) is an efficient way of characterizing transport in complex fluid flow. To cite Peacock and Haller [39], the LCS approach is a means of identifying key material lines that organize fluid-flow transport. LCS form separatrices surfaces, which divide the domain into dynamically-isolated regions and reveal the hidden flow skeleton. LCS defining Lagrangian jet cores [21] can be extracted from maxima ridges in the so-called finite-time Lyapunov exponent (FTLE) scalar field. FTLE quantifies the rate of stretching between flow trajectories integrated over time. Previous studies [2,3,52] have shown that extraction of LCS from the FTLE field allows the observation of blood flow transport over a complete cardiac cycle from an Eulerian point of view.

The purpose of this article is to provide an efficient numerical workflow to compute dynamically isolated zones from LCS inside the AAA lumen geometry extracted from patient-specific CTA. We hypothesize the identification of these stagnation zones could be useful to predict thrombus formation and its impact on AAA growth and vulnerability. The following section presents a numerical simulation model of 3-D blood flow, with special emphasis on boundary conditions. Numerical flow was validated in 4 patients undergoing 4-D phase contrast MRI. The model then computed flow transport and stagnation zones in 9 CTA-based AAA geometries acquired over 8 years in specific patient follow-up. The chronologic evolution of altered flow topology in the lumen was computed and compared to morphological changes of AAA.

2. Materials and methods

This section describes the 3-D computational flow dynamics (CFD) model and, more specifically, defines the parameters involved in boundary conditions. Patient-specific conditions were unavailable in the context of a retrospective study. Simulated velocity magnitudes were compared with those obtained by 4-D MRI velocimetry in 4 patients.

2.1. Blood flow simulation

2.1.1. Domain simulation

Four patients with diagnosed AAA were enrolled: they were regularly followed by CT-scan to assess AAA growth and eligibility criteria for open or endovascular repair. The study was approved by the local Institutional review board, and all patients gave signed informed consent form. First, blood flow was computed in the last follow-up CTA in 4 patients with AAA (aged 70 ± 11.2 years) and compared to MRI velocimetry for validation. Then, blood stagnation zones on 9 follow-up CTAs acquired between 2006 and 2013 ($\Delta_{\text{tavg}}$ = 1 year, 14 days) were studied in another patient (age 79 years).

CTA voxel size ranged from $(0.75 \times 0.75 \times 0.75)$ mm$^3$ to $(0.85 \times 0.85 \times 1)$ mm$^3$ in the 3 orthogonal axes: anterior-posterior, right-left and cranio-caudally, respectively. The lumen was segmented using the region growing tool of MITK [36] according to the following procedure: 1) placing the seed point in the lumen 2) Setting the upper and lower threshold values, varying on each scan. Particular attention was paid to the upper limit to avoid the segmentation of the bones. 3) Evolution of the contour until the whole domain is segmented. After segmentation, segmented lumen were smoothed using a Taubin [60] filter, avoiding the shrinkage of the geometry. Outlets were extruded (1 diameter length) to provide circular boundary. Final surfaces were overlaid on the CTA and validated by a radiologist.

2.1.2. Boundary conditions

2.1.2.1. Inlet. A time-varying flow rate was imposed at the inlet [31], located approximately 5 cm upstream of the upper renal artery (Fig. 1). Though actual inlet flow patterns may present either single or double swirl structures [34], no generic spatial velocity profile can currently describe flow entering AAA. Considering the lack of available information, a time-dependent flow rate from Mills et al. [31] was mapped on a Womersley profile.

2.1.2.2. Wall. In AAA, wall geometry is characterized by the aorta and thrombus deposits. Aortic wall mechanical properties vary with vessel size and location as well as patient age. Wall rheology becomes highly patient-specific and heterogeneous with the progression of atherosclerotic disease [44]. The thrombus is also a complex, layer-structured and heterogeneous material [69]. In practice, it is difficult, if not impossible, to characterize 3-D wall rheology by non-invasive techniques for fluid-structure simulations. Wall stiffness increases with disease progression and patient age [61]. In addition, thrombus alters wall stress distribution and augments apparent stiffness [66]. The present study considered the wall and thrombus as rigid materials, and applied no-slip conditions. This simplification turned out to be acceptable according to the velocity measurements performed by MRI (see following text and Fig. 2). Also, the potential impact on FTLE computation proved to be negligible [15].

2.1.2.3. Outlets. Blood flow can present complex recirculation patterns in diseased aorta that can stretch down to outlet planes. Reverse flow is also a natural phenomenon occurring in large arteries during diastole [17,24]. Artificial extension of outlets, leading to unrealistic geometries and increased number of mesh elements, is common practice in arterial flow simulations addressing these issues. Neumann boundary conditions on velocities were rather adopted with gradient to control blood backflow in the domain derived from the solution proposed [17]. A 3-element Windkessel model was applied on each outlet (Fig. 1) [35,65]. Model parameters were chosen according to Nan Xiao et al. [72] study.

2.1.3. Flow regime

Reynold numbers in our simulations were within the physiological range [18], varying from 1634 to 1954 at the proximal inlet. Womersley...
numbers at the proximal inlet varied from 10.00 to 14.97, which was within the physiological range [32]. Blood was modelled as an homogeneous non-Newtonian fluid following the Quemada model [5,28]. Model parameters were chosen according to the study of Buchanan & al. [11], based on the rheological data from Merril & al. [16]. Expressed a modified Casson model, the viscosity can be written as:

$$\eta = \left( \sqrt{\eta_\infty} + \sqrt{\frac{\tau_0}{\lambda + \sqrt{\gamma}}} \right)^2$$

With $\eta$ the apparent viscosity, $\gamma$ the shear rate, $\eta_\infty$ the viscosity when $\gamma \to \infty$, $\tau_0$ the shear stress when $\gamma \to 0$ and $\lambda$ the characteristic time. Numerical values are $\eta_\infty = 0.002654 \text{ Pa s}$; $\tau_0 = 0.004360 \text{ Pa s}$ and $\lambda = 0.02181 \text{s}^{-1}$ [11].

2.1.4. Numerical methods
The domain was discretized using a polyhedral mesh with an average of 463000 elements (SD 51470; range: 408570–568026). Navier-Stokes equations were discretized with finite volume methods (FVM) implemented in the OpenFOAM toolbox [22,23]. The solver used is a large time-step transient solver for incompressible for solving pressure–velocity coupling, using the PIMPLE (merged PISO-SIMPLE) algorithm. Simulations were initialized using first order schemes in time (Euler) and space (Gauss upwind) up to temporal convergence. Second order schemes in time (Crank Nicolson) and space (Gauss linear upwind) were then used up to temporal convergence. The time step of the simulation
Table 1. Comparison between the simulated blood flow and the MRI measurements performed on 4 patients. Comparisons are done at the systolic peak and numerical values of the maximum cross-correlation are given.

<table>
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<tr>
<th>Slices locations</th>
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<td><img src="image17" alt="Image of normalized velocity 6" /></td>
<td><img src="image18" alt="Image of MRI 6" /></td>
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Fig. 2. Comparison between the simulated blood flow and the MRI measurements performed on 4 patients. Comparisons are done at the systolic peak and numerical values of the maximum cross-correlation are given.
was automatically adapted to keep the CFL [14] condition below 1, leading to approximately mean time step size of $3.9 \times 10^{-4}$ s (SD: $2.23 \times 10^{-4}$; range: $1.15 \times 10^{-4} - 9.25 \times 10^{-4}$). Temporal convergence verification was performed on the velocity at a control location in the AAA between consecutive cardiac cycles. It was achieved after 5–7 cardiac cycles. We accessed resources from Calcul Québec and Compute Canada¹ for FVM computations.

2.2. CFD comparison with MRI velocimetry

MRI acquisitions were validated on a 3T magnet (Achieva X-Series, Philips Medical, Eindhoven, Netherlands) with 16-channel abdominal antenna and phase-contrast velocity sequence. Blood velocities in 4 volunteer patients were measured on AAA cross-sections orthogonal to maximal diameter ($D_{\text{max}} = 44.7 \pm 8.33$ mm) and proximal neck. Because of breathing artefacts, technically-successful acquisitions were obtained on only 6 slices for the 4 AAA patients investigated by MRI (Fig. 2, left column).

MRI spatial resolution was $1.6 \times 1.6 \times 6$ mm³, the temporal resolution 1/8th of the cardiac period and grid size 256 × 256. The chosen velocity encoding was 2 m/s to encompass the highest velocity likely to be met in the aorta and was not exceeded. Three velocity components were measured in each slice. Aortic flow was assumed to be periodic during MRI acquisition (ECG-gating). Since respiratory gating was not available, patients were asked, if possible, to hold their breath during acquisitions, i.e., for at least 20 s.

In addition, the lumen was segmented to evaluate its section variation over time (Fig. 3). Two healthy volunteers (35 ± 1.4 years old) were investigated as controls. Section surfaces were evaluated at maximal and proximal diameters of the 5 AAA and at mid-distance between the lowest renal artery and iliac bifurcation, approximately 2 cm under the lowest renal artery of the 2 healthy subjects.

2.3. Transport quantification

Flow in AAA is often characterized by the presence of jet cores [3,6,47,55], creating a coherent vortex in their wake [7]. Zones of low shearing and low stretching, isolated by the repelling surface, lie between the jet cores and dilated aortic wall. Repelling LCS can thus mark the boundaries of stagnant flow [50], the feature of interest in the present study.

LCS extraction has proven to be an efficient tool for the visualization of transport structures in biomedical [50], industrial [48] and environmental [43] contexts. LCS are not only a convenient way to observe transport barriers in flow, but they also help investigate the transport of unsteady flows where conventional techniques may fail [49]. FTLE field trenches bounded by repelling hyperbolic LCS have already been successful in viewing bioaccumulation in oceanic flows [37].

LCS were extracted from the FTLE field [1]. Given $\lambda_{\text{max}}$, maximal eigenvalue of the Cauchy-Green tensor of flow, the largest stretching at location $x_0$ was $\sqrt{\lambda_{\text{max}}(x_0)}$. Such exponentially-evolving stretching, FTLE at $x_0$, was defined as:

$$\sigma_{\lambda_{\text{max}}}^T(x_0) = \frac{1}{T} \ln \sqrt{\lambda_{\text{max}}(x_0)}$$

With $\sigma_{\lambda_{\text{max}}}^T(x_0)$ the FTLE field where particles where advected from the time $t_0$ to $t_0 + T$, starting at the location $x_0$.

To compute the Cauchy-Green deformation tensor, trajectories of particles seeded in each point of the grid were integrated over time with a Runge-Kutta integrator onto a subgrid with $0.25 \times 0.25 \times 0.25$ mm³ resolution. An auxiliary grid ($0.1 \times 0.1 \times 0.1$ mm³) was considered for the Cauchy-Green tensor computation. Particles were advected backward in time to obtain attractive LCS [52]. Integration time $T$ of 1.5 s allowed most particles to leave the domain [52]. FLTE computations were performed with VisIt [13] on a subdomain of the initial CFD domain (Fig. 4), in the AAA portion located between the lowest renal artery and the iliac bifurcation.

LCS ridges were computed as dot products of eigenvalues of the Hessian of FTLE [51].

Since flow topology varied highly over cardiac cycles, LCS did not necessarily close domain boundaries completely (Fig. 5). To close volumes of interest (stagnation volumes), as we assumed them to be smooth and continuous, a region-growing segmentation method [42] was chosen, with ITK-Snap [73]. This region-growing method works well on continuous fields. Yet, LCS are an ensemble of disconnected 3-D surfaces in AAA. Consequently, LCS barriers were added to FTLE field AAA to produce a new, continuous, smooth FTLE field, aFTLE. Initial seeding points were placed visually by the operator, and segmentation was allowed to grow until no change was seen.

3. Results

3.1. MRI velocimetry

3.1.1. CFD validation

Fig. 2 illustrates the localization of the slices used to compare experimental data to the simulation results. The velocity magnitude obtained from reconstruction of PC-MRI data at systolic peaks is compared to the simulated data for each slice and a cross-correlation value is computed. Experimental and simulated data are visually consistent, and cross correlation of velocity magnitude confirmed good representation of flow topology. 2-D maximum cross-correlation coefficients ranged between 0.65, 0.86, and 0.082 (min, max, SD).

These results show that generic boundary conditions and blood rheology used in the computation of the CFD model are consistent for the analysis of AAA flow topology.

3.1.2. Rigid wall hypothesis validation

Lumen segmentation during different phases of the cardiac cycle gave an estimation of wall compliance and AAA size lumen variation. Relative surface section variation during 1 cardiac cycle was plotted in Fig. 3. As expected from the literature [64], patient age correlated to aorta wall stiffening led to small cross-sectional surface variation of 0.78 ± 0.54% and 1.82 ± 0.96% at $D_{\text{max}}$ location and at proximal neck respectively. As expected, for healthy patients, the aorta surface variation is greater: $7.95 \pm 4.41\%$ and $7.47 \pm 4.00\%$ at $D_{\text{max}}$ location at proximal neck respectively. The small change in wall deformation during cardiac pulse motivated the choice of a rigid wall model for AAA of elderly patients.

3.2. LCS

The morphology of the AAA case-study, followed by CTA during 8 years, presented 2 main bulges, a big one on the anterior side and a smaller one on the posterior (Fig. 4). The smaller one (posterior side), close to the spine, flattened slightly with time. The larger one, on the anterior side, grew over time in the direction of the abdominal cavity. These 2 bulges were the locations of recirculation zones secondary to jets created at the exit of the AAA proximal neck. These recirculation zones formed during peak-systole, when blood rapidly entered the dilated section of the AAA, forming Lagrangian jet cores. LCS from repelling FTLE (which characterizes stagnant flow) enclosed the AAA bulge areas, dividing the flow domain into 3 sub-domains (Fig. 6, top left): 2 isolated stagnation volumes and 1 stretched domain connecting the inlet to the outlet of the AAA (freely-circulating blood). Smaller barriers were visible inside these zones and denoted the presence of enclosed transport features (Fig. 7). Fig. 8 illustrates how the observation of instantaneous flow

¹ The operation of the supercomputer used is funded by the Canada Foundation for Innovation (CFI), NanoQuébec, RMGA and the Fonds de recherche du Québec - Nature et technologies (FRQ-NT).

contrasts with hyperbolic LCS.

3.3. Evolution of transport during patient follow-up

The 2 main stagnation zones were well discernible in all 9 simulations. Although their volumes increased (Fig. 9), their localization and overall shape remained unchanged. Even though LCS did not always provide perfectly-closed volumes, segmentation remained reproducible. On the first CT-scan, 3 segmentations were performed in each stagnation zone 3 times per 3 users: intra-user variability on volume was $/C_0$ 3.46, 4.03, 3.91 and inter-user variability was $/C_0$ 1.22, 1.08, and 1.16 (min, max, SD, in %). The smallest stagnation zone grew linearly from 1.81 to 4.11 cm$^3$ in 8 years (127% increase, Fig. 6), and the largest, from 16.17 to 63.66 cm$^3$ (293% increase, Fig. 6). Growth of both the large and small stagnation zones are correlated with lumen dilation (Pearson’s $r = 0.99$, $p = 0.0004$ and 0.93, $p = 0.002$, respectively). Average yearly growth rates were 8.14, 6.08 and 0.28 cm$^3$/year ($r^2 = 0.99$, 0.84 and 0.98) for the lumen, large and small stagnation zones, respectively (Fig. 6). The circulating zone, connecting the AAA inlet to the outlet, was only weakly correlated to lumen growth ($r [2] = 0.52$). Lumen growth was mostly composed of stagnation zones while the volume contributing to transport remains barely affected by it.

4. Discussion

The aorta’s role is to deliver oxygenated blood and nutrients as well as to remove waste products. Transport performance between blood and vessel wall is dictated by transit through the artery surface and fluid
mechanics [59]. With abdominal aortae losing their straight tubular shape with aging or aneurysm progression, blood flow topology is expected to change from its original state. The current study aims to provide an efficient methodology to locate and quantify transport topology alterations. The first step was to validate that our computation CFD model is consistent for the analysis of AAA flow topology using MRI velocimetry on four patient followed for AAA. Based on this flow model, dynamically-isolated zones were extracted from the flow simulation performed on 9 scans of 1 patient followed clinically with an unpaired AAA. Patient-specific geometric models were constructed from 3-D CTA of the aorta. Transport barriers were reconstructed inside the lumen from the transient flow field. Blood flow was solved using FVM with boundary conditions build from patient specific data by Nan Xiao et al. [72] and was confirmed by PC-MRI.

Even though the location of 2 dynamically-isolated zones in the area of luminal dilatation could be guessed, their volume and boundaries could now be computed. Furthermore, stagnation in the bulbous part of the AAA was witnessed with particle residence times [56]. It was observed that the domain connecting the proximal to the distal neck in the lumen remained unperturbed and formed an effective circulation path in the entire lumen. Its approximately constant volume during entire follow-up went along with constant perfusion needed for the pelvic organs and lower limbs. The quasi-linear growth of the AAA agreed with the observations of Zambrano et al. [74] in 14 followed patients.

LCS-based methods have previously been applied to study flow in AAA [52] and various other domains [21,48]. In this AAA case-study, barriers between stagnation and circulating zones were highlighted in the lumen geometry, given the fact that the FTLE field was constructed from stretching between adjacent fluid particles. Distinct barriers to flow appeared as the arterial lumen dilated and became tortuous. Age could have been the initial insult, as buckling of the artery wall appears with loss of elastin and resulting lengthening [27]. Particles traveling along LCS may be subject to high shearing, the cause of platelet activation. If activated, i.e., subjected to a certain shear stress level for long enough [45,71], platelets entering stagnation zones are likely to adhere to the wall and create a substrate favourable to thrombus deposition. Low shear stress may also be a condition necessary for platelet adhesion [6] which happens in stagnation zones. Portions of the wall exposed to low shear can be visualized as time average wall shear stress and linked to thrombus deposition [74]. However, this only accounts for part of the adhesion mechanism. Looking at the aorta location exposed to stagnation zones could be an additional complete predictor of intra-luminal thrombus (ILT) deposition.

Thrombus covering the AAA wall will accelerate its degeneration. An important outcome is the increase of elastase and thus the rate of elastin destruction. ILT presence is potentially linked with local hypoxia and inflammation of the underlying wall [62]. In addition, ILT thickness is correlated to smooth muscle cell apoptosis, elastin degradation and MMP-2 concentration, relative to mechanical stability, accelerating AAA growth. During the cardiac cycle, ILT may partly withstand the mechanical load due to blood flow. In parallel, increased pressure load on the wall promotes the synthesis of collagen and proteoglycans, consequently approaching the AAA wall rupture limit [58]. However, wall degradation is mostly active in areas covered by thin ILT, barely quantifiable by CTA [68].

In the AAA studied, in the first CTA the ILT thickness was too low to be segmented. Thin ILT was nevertheless visible, facing both stagnation zones (Fig. 10) but did not cover a large part of the lumen, as seen in most AAA [74]. However, even in the early stage, this AAA presented well-defined dead circulation zones in bulging areas, as shown in Fig. 9. Even though platelet-endothelial cell adhesion is lower than platelet-fresh ILT adhesion, trapped and shear-activated platelets [4] slowly formed a thrombus layer over the wall. ILT will then grow from this location, because of platelet thrombus affinity, progressively filling existing concavity. Blood pressure and high flow rate will prevent lumen section reduction to preserve pelvis and lower limb perfusion. This minimal size could match the computed circulation zone, approximately constant with time (Fig. 6). Zambrano et al. [74] studied fast-growing AAA and observed constant diameter of the lumen section whereas $D_{\text{max}}$ progression was related to thrombus growth.

The most common way to study the role of blood circulation in AAA physiology is through WSS and WSS derivative. However, an AAA with or without ILT should not be considered the same way: in the absence of ILT, the flow exposed endothelium may be able to trigger vascular mecano-adaptation in response to flow modification. In contrast, in the presence of ILT, the WSS could be an indicator of platelet adhesion risk. Portions of the aorta covered by ILT and those without ILT presenting atherosclerotic plaques give totally different roughness data. This textural information is not visible on spatial resolution by CTA. Nevertheless, it is crucial for WSS numerical computation and constitutes a limitation of this approach. However, looking at the transport field limits the impact of the near wall lack of information.

Recent progress in multiphysics coupling allows the simulation of thrombus deposition and growth as proven by Menichini et al. [30] and Biasetti et al. [8]. This gives direct information on the portion of the lumen exposed to it and the progressive alteration of the flow field. However, thrombus formation is the AAA is a long term process going with the deformation of the arterial wall, no considered in cited models. Alternatively, growth and remodelling of the lumen model exist [20] but do not include thrombus development. Extracting the dynamically isolated zones on the other hand do not require extensive hypothesis on the biological process of thrombus formation but gives information on the severity of the alteration of the flow and, and, as such, metrics for AAA classification.

A long-term challenge is to understand the mechanisms promoting AAA growth – to improve patient management, especially regarding eligibility and timing for open or endovascular repair. The measurement of AAA $D_{\text{max}}$, in multipplanar CT-scans, is the current way to assess AAA growth and growth rate over time in clinical studies. This 1-D measurement, however, cannot reflect the changes observed in complex AAA geometry.
5. Limitation

Extract of dynamically isolated zones requires a realistic flow field as support. This retrospective study relied on CT-scans acquired over a time span of 8 years and no flow information was recorded at the time. Generic simulation parameters were therefore used. This point has no influence on the presented workflow but the results, such as the shape and size of the dynamically isolated zones, can be altered. Numerical [26] and experimental [40] studies shows the presence of mild turbulence in AAA at patient rest, increasing with exercise. Turbulence in AAA will likely shrink recirculation zones [9] compared to our laminar simulation. At the inlet, the Wormersley velocity profile enforced with no rotational component. To the knowledge of the authors, not general model exists other than modeling the whole circulation upstream the aneurysm, including the aortic arch. Helicity was however measured [33, 34] and its role on the dynamically isolated zones should be investigated.

Recent work on transport proposes more robust methods for material surface extraction [21] not based on FTLE, allowing the visualization of hyperbolic but also parabolic and elliptic LCS [21]. Elliptic LCS may allow skipping the segmentation step because by definition when
Fig. 7. Coronal, axial and sagittal view of the aFTLE field in the lumen with dynamically isolated zones superimposed over (2006 follow-up). Lower left view shows the cut planes on the lumen geometry.

Fig. 8. Blood flow velocity magnitude and in plane vectors at two locations and three times. In overlay the two stagnation zones (red and green) and the circulation domain (white). The instantaneous velocity fields do not coincide with the extracted frontiers in the context of a pulsatile flow. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)
hyperbolic LCS are formed from Lagrangian jet cores it forms closed domains. As jet cores are interesting as a typical feature of AAA [3], both hyperbolic and elliptic LCS should be studied. Although not the most reliable mathematical tools to extract LCS, Shadden and Taylor [52] demonstrated the ability of the method to capture transport boundaries in the specific context of biological flow.

6. Conclusion

A computation flow dynamic model applicable to AAA was proposed and validated with MRI velocimetry. Transient flow transport topology evolution in this model was computed on an AAA over the span of 8 years and 9 CT-scans. Lumen geometries were reconstructed from injected CT images and transient flow simulated with FVM. The presented workflow allowed extraction of blood flow stagnation zones in an AAA and studied their evolution during its growth, with only clinically-available data. The steps to accomplish this goal are summarized below:

- Flow computation:
  - CT-scans with contrast agent are segmented by a region-growing approach. The geometry thus obtained is meshed with polyhedral elements and boundary conditions are set.
  - Navier-Stokes equations are solved over the domain until convergence with a transient flow inlet and 0-D outlets.
  - The resulting flow velocity fields are validated with MRI velocimetry.
- Stagnation zone extraction:
  - Stagnation zones are segmented from the FTLE field, by edge-based pre-processing. The FTLE field is convoluted with computed LCS.
  - Stagnation zones are segmented from the FTLE field, by edge-based pre-processing. The FTLE field is convoluted with computed LCS.

The proposed workflow provides a solution to not only visualize but also quantify the dynamically isolated zones of the blood flow in aneurysms. The identification of these stagnation zones can be performed with routine medical imaging in AAA patients. This opens the door for large scale comparative studies to improve our understanding on the role of flow topology and transport on ILT formation and their association with AAA growth and vulnerability.

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