Vortex dynamics and transport phenomena in stenotic aortic models using Echo-PIV

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Keywords: lagrangian coherent structures, ultrasound, blood flow dynamics, atherosclerosis

Abstract
Atherosclerosis is the most fatal cardiovascular disease. As disease progresses, stenoses grow inside the arteries blocking their lumen and altering blood flow. Analysing flow dynamics can provide a deeper insight on the stenosis evolution. In this work we combined Eulerian and Lagrangian descriptors to analyze blood flow dynamics and fluid transport in stenotic aortic models with morphology, mechanical and optical properties close to those of real arteries. To this end, vorticity, particle residence time (PRT), particle’s final position (FP) and finite time Lyapunov’s exponents (FTLE) were computed from the experimental fluid velocity fields acquired using ultrasonic particle imaging velocimetry (Echo-PIV). For the experiments, CT-images were used to create morphological realistic models of the descending aorta with 0%, 35% and 50% occlusion degree with same mechanical properties as real arteries. Each model was connected to a circuit with a pulsatile programmable pump which mimics physiological flow and pressure conditions. The pulsatile frequency was set to 𝑅𝑒 ≈ 0.9 Hz (55 bpm) and the upstream peak Reynolds number (Re) was changed from 1100 to 2000. Flow in the post-stenotic region was composed of two main structures: a high velocity jet over the stenosis throat and a recirculation region behind the stenosis where vortex form and shed. We characterized vortex kinematics showing that vortex propagation velocity increases with Re. Moreover, from the FTLE field we identified Lagrangian coherent structures (i.e. material barriers) that dictate transport behind the stenosis. The size and strength of those barriers increased with Re and the occlusion degree. Finally, from the PRT and FP maps, we showed that independently of Re, the same amount of fluid remains on the stenosis over more than a pulsatile period.

1. Introduction
Cardiovascular diseases represent one of the major causes of death in the world (Mendis et al 2011). Blood flow dynamics has shown to be crucial in understanding the different causes of gradual or acute changes during diseases affecting the cardiovascular system. For example, flow pattern and vortex dynamics in the left ventricle are correlated with altered cardiac function (Hong et al 2008, Faludi et al 2010, Bermejo et al 2014). In arteries, regions of disrupted flow or with large recirculation are likely to favor atherosclerosis (Zarins et al 1983, Martorell et al 2014), one of the most prevalent and dangerous cardiovascular disease (Mendis et al 2011). In atherosclerosis fat, cholesterol and other substances accumulate inside the artery creating a plaque or stenosis which narrows the arterial lumen. During growth or plaque rupture, atherosclerosis may lead to heart failure, stroke or even death. Several factors have been associated with plaque growth and rupture such as the local influx of inflammatory cell, extracellular matrix synthesis, local modulation of coagulating factors and genetic predisposition. Moreover, disturbance of blood flow patterns has been shown in vivo and in vitro to be proatherogenic by promoting oxidative and inflammatory response of the arterial wall. Meanwhile, laminar and
steady flow has been shown to be atheroprotective by inhibiting inflammation and oxidative stress (Nigro et al 2011). However, despite all the research allowing the detection of systemic markers and the development of treatment for this pathology, prediction of plaque rupture continues to be misunderstood (Lutgens et al 2003).

Eulerian approaches have shown to be a powerful tool in analysing blood flow dynamics in stenotic vessels (Varghese et al 2007, Katritsis et al 2010, Pielhop et al 2012, Geoghegan et al 2013, Usmani and Muralidhar 2016, Choi et al 2018). In these works, streamlines, vorticity, turbulent kinetic energy and wall shear stresses were computed from the instantaneous velocity data obtained either via numerical studies (Varghese et al 2007, Katritsis et al 2010), in vitro experiments (Pielhop et al 2012, Geoghegan et al 2013, Choi et al 2018) or both (Usmani and Muralidhar 2016). Alternatively, Lagrangian descriptors can provide direct information on the transport topology in large vessels (e.g. flow mixing, stirring, recirculation, stagnation and separation) (Shadden and Taylor 2008). Analysing the transport topology in stenotic vessels may provide valuable information on how stenosis grows and eventually ruptures (Xu et al 2009, Rayz et al 2010, Shadden and Hendabadi 2013).

Lagrangian coherent structures (LCS) are the most common Lagrangian descriptors and are usually defined as locally strongest attracting or repelling material barrier (Shadden 2012, Haller 2015). They reveal structures that govern fluid transport and mixing in complex flows and have been applied to oceanography, geophysics and atmospheric flows (please refer to references within the reviews Shadden 2012 and Haller 2015). Another Lagrangian descriptor is the fluid particle residence time (PRT), which measures the time that a fluid parcel spends in a given region. Recently LCS and PRT have been applied to study hemodynamics in the left ventricle (Espa et al 2012, Töger et al 2012, Charonko et al 2013, Hendabadi et al 2013, Rossi et al 2016, Badal et al 2017, Di Labbio et al 2018) and large vessels (Shadden and Taylor 2008, Vétel et al 2009, Rayz et al 2010, Azizani and Shadden 2012, Jeronimo et al 2019, Jeronimo and Rival 2020). Moreover, most of these studies use only one Lagrangian descriptor (either LCS or PRT) to describe flow. Although PRT and LCS can both provide a Lagrangian analysis on their own, they give different information about the flow. LCS reflect the material barriers that organize flow and which may be responsible for high or low PRT values in a given region. To our knowledge, only a few works combined LCS and PRT to study material transport and flow topology in the left ventricle (Hendabadi et al 2013, Di Labbio et al 2018) and downstream of a dysfunctional bileaflet mechanical aortic valve (Darwish et al 2020).

Despite the numerous works involving hemodynamics in pathological conditions, only a few have focused on studying flow dynamics in stenotic vessels. In the experimental works of Jeronimo et al PRT was measured using two dimensional ultrasonic (Jeronimo et al 2020) and optical (Jeronimo et al 2019, Jeronimo and Rival 2020) particle tracking velocimetry in steady and unsteady flow conditions for different Reynolds and Strouhal numbers. As vessel model, they used a rigid acrylic pipe with a smooth axisymmetric constriction followed by an unrealistic sudden expansion. Other works used instantaneous velocity data obtained either via numerical studies (Varghese et al 2007, Katritsis et al 2010, Usmani and Muralidhar 2016) or in vitro experiments (Pielhop et al 2012, Geoghegan et al 2013, Usmani and Muralidhar 2016, Choi et al 2018). Numerical studies can account for patient specific morphology (Katritsis et al 2010), providing highly resolved three-dimensional velocity fields. However, they are unable to simulate the mechanical properties of the vessel wall, which is always assumed to be rigid. This is also the case for the experiments (Pielhop et al 2012, Geoghegan et al 2013, Usmani and Muralidhar 2016, Choi et al 2018) where transparent models made of acrylic or silicone are required because optical particle velocimetry is used to measure the fluid velocity field. For example, in the work of Usmani and Muralidhar (2016) they used a compliant stenotic model made of silicone where its distensibility was changed by modifying the wall thickness of the model. This type of material does not mimic the anisotropic, nonlinear, elastic behavior of the vessel wall. Finally, these works focus on studying flow from an Eulerian perspective. To our knowledge, no experimental work reported the use of LCS to study flow in stenotic vessels. Moreover, a combined approach that uses LCS and PRT is advisable to provide a broad description of the transport process in stenotic vessels.

Consequently, in this work we combined vorticity, LCS, PRT and final position (FP) maps to study vortex dynamics and fluid transport in stenotic aortic models with morphology, mechanical and optical properties close to those of real arteries. The flow dynamics behind the stenosis was studied as a function of Reynolds number and the degree of occlusion from the particle velocity fields acquired using ultrasonic particle imaging velocimetry (Echo-PIV). Finally, results were discussed and compared to those reported in the literature.

2. Materials and methods

2.1. Model manufacturing

The aortic models for this study were developed following the same procedure described in Bernal et al (2019a). By segmentation of anonymous CT-images of a healthy patient, the lumen and the adventitia layer of the descending aorta were reconstructed (lumen volume and inflated volume in figure 1(a)). For each geometry
(lumen and inflated), two molds were fabricated using computer numerically controlled machine. Then, the lumen volume was inserted into the mold created from the inflated geometry (figure 1(b)). The gap between these two elements allowed the injection of a 10% b/w Polyvinyl Alcohol (PVA) solution. Prior to the injection of the polymer, the core was wrapped with a reinforcing fabric to allow the models to withstand physiological pressure levels and to have mechanical properties similar to that of healthy arteries (Bernal et al. 2019a, 2019b). Finally, the whole setting was subject to 7 cycles of freezing and thawing to polymerize PVA, after which the mold was opened and the core was removed. The models dimensions were 17 cm in length, 2.4 cm of external diameter and ≈0.3 cm of wall thickness. For healthy physiological pressures (diastolic/systolic) of 80/120 mmHg, the models exhibit a nonlinear change in their shear modulus from ≈133 kPa to ≈209 kPa with 10% of error. These values were obtained by fitting a Lamb wave model to the phase velocity dispersion curve obtained from the shear wave propagation along the wall of the model. Precise details on the mechanical evaluation of the models are given in the works of Bernal et al. (2019a, 2019b). Moreover, as demonstrated in Bernal et al. (2019a, 2019b) the mechanical behavior of the models is very similar to those of swine aortas.

Additional models with different degree of occlusion were made by modifying the lumen geometry resulting in a stenosis made of PVA (figure 1(b)). The profile of the stenosis was chosen to be Gaussian following $z_s = A_o \exp(-x^2/(2\sigma^2))$ where $z_s$ is the height of the stenosis and $\sigma = 1.85$ cm, which gives a full width at half maximum of ≈3.35 cm. The value of $A_o$ was chosen to achieve an occlusion maximum height of ≈0.8 cm and ≈1.2 cm which resulted in two different models with 35% and 50% of occlusion in diameter, respectively. The Young’s modulus of the stenosis was $55 \pm 5$ kPa (mean value ± standard deviation) and was evaluated using the general elastography preset of an Aixplorer system (Supersonic Imagine, France). As reported by previous studies, the Young’s modulus of the stenosis may be underestimated by the Aixplorer system due to the dispersion of the shear wave inside the stenosis (Widman et al. 2015, Marlevi et al. 2018). Assuming a 300–400Hz central frequency for the shear wave, the resulting shear wavelength (≈1 cm) in our experiments is smaller than the stenosis thickness (≈1.5 cm, more than twice the thickness used in Widman et al. 2015 and Marlevi et al. 2018). Therefore, we believe dispersive effects to be weak when compared to Marlevi et al. (2018) and Widman et al. (2015). Nevertheless, we do not discard that the Young’s modulus may be affected by a small underestimation of no more than 10%. Figures 1(c) and (d) show a cross sectional B-mode image of each of the stenotic models with the Gaussian profile highlighted by a full red line.

2.2. Hemodynamic simulator

Each model was connected to a hemodynamic work bench simulator, which has already been used for the elasticity assessment under physiological conditions of vascular grafts, vessel models and femoral ovine arteries (Suarez Bagnasco et al. 2014, Bernal et al. 2019a). The simulator consists of a programmable piston pump that mimics flow and pressure wave from the heart (Balay et al. 2010), two pressure sensors (one at the inlet of the model and one at the outlet), a reservoir that could be pressurized using a manual sphygmomanometer and a water bath where the model is placed.

2.3. Echo-PIV

The fluid velocity field was measured through ultrasonic particle imaging velocimetry (Echo-PIV), which is based on 2D cross-correlation of consecutive speckle images (Kim et al. 2004, Zheng et al. 2006, Jensen et al. 2016a). To this end, the circuit was filled with degassed water seeded with neutrally buoyant polyamide particles (Dantec Dynamics, Denmark) with a mean diameter of 50 µm. These particles have a Stokes number of approximately $1 \times 10^{-4}$ and can be assumed to closely follow the flow. A particles density of 0.2 g l$^{-1}$ was used in all the experiments.

**Figure 1.** Manufacturing process of the stenotic aortic models. (a) Reconstructed volumes from CT-images: lumen volume in gray and inflated volume in red shadow. (b) Lumen volume wrapped in the reinforcing fabric inside the acrylic mold created from the inflated volume. Cross-sectional B-mode images of the models presenting a (c) 50% and (d) 35% occlusion in diameter. The red full line corresponds to the Gaussian profile used to create the stenosis.
For Echo-PIV, ultrasonic B-mode images were acquired using plane wave insonification (Montaldo et al 2009) at 200 Hz frame rate during 2.5 s. A custom-made linear probe (Vermon, Tours, France) with 256 elements (0.2 mm pitch) working at 15 MHz driven by a Verasonics Research Ultrasound System was used in the experiments. The axial resolution (i.e. along depth) of the B-modes images was 0.1 mm. The cross sectional center plane of the model was imaged by positioning the probe parallel to the aortic model with its first element facing towards the direction of the flow (figure 2(a)). To avoid constraint the movement of the wall, the ultrasound probe was not in direct contact with the model using the water bath as coupling medium between probe and model. As illustrated in figure 2(a) by different colored rectangles, three regions of interest (ROI) were imaged in the experiments: upstream (black dashed–dotted line), post-stenotic (red full line) and on the stenosis throat (blue dashed line). To image these three ROIs the probe was moved along the x-direction by a step by step motor. For each experiment, a time lapse of ten periods was waited before the ultrasound acquisition to avoid start-up effects.

Finally, PIVlab software was used to compute the velocity fields from the B-mode images using a direct correlation approach with windows of $0.64 \times 0.32$ cm$^2$ and 75% overlap (Thielicke and Stamhuis 2014). This resulted in a lateral and axial resolution for the velocity fields of 1.6 mm and 0.8 mm, respectively.

### 2.4. Flow conditions

The pulsatile frequency was set to $\approx 0.9$ Hz (55 bpm) for all the experiments, close to that of a normal human heart. Moreover, the profile of the pressure wave form was set to be close to the physiological pressure wave profile for the descending aorta (O’Rourke et al 1968). Figure 2(b) shows representative pressures at the inlet and outlet of the 50% occluded model.

Given a fixed flow rate, flow dynamics is essentially controlled by the Reynolds number ($Re$), which relates the flow velocity ($v$) with lumen size ($D$) as $Re = Dv/\nu$, where $\nu$ is the kinematic viscosity of water ($1.0 \times 10^{-6}$ m$^2$ s$^{-1}$). Taking into account the internal diameter of our models and the kinematic viscosity of water, the pulsatile pump was programmed to achieve three different peak velocities inside the models: 9.5, 7 and 5 cm s$^{-1}$. This corresponds to peak Reynolds numbers of approximately 2000, 1500 and 1100, respectively, which lie within the physiological range of the aortic artery (Ha et al 2018). Nevertheless, small deviations from these values are expected in the experiments because of the small differences in the mechanical response of the models. Therefore, the exact Reynolds number for each experiment was computed by measuring the internal diameter and the velocity obtained through Echo-PIV. As an example, figures 2(c)–(e) show the lumen, velocity and $Re$ as a function of time for the $Re = 2000$ experiment in the 50% occluded model. Velocity and diameter are affected by a 5% error which results in an uncertainty of 10% for $Re$ (gray shadow in figure 2(e)). The internal diameter of the model was estimated by tracking the movement of the wall from the B-mode images using an intensity threshold of $-6$ dB. A summary of the precise experimental conditions is given in table 1. Repeatability of the experiments, models’ mechanical properties and flow patterns was checked from experiments carried out over a five-month period.
In table 1, the values of Re set on the programmable pump and $Re_{max}$ agree within the margins of error for all the experiments. Therefore, in this work we will use the set value to label each experiment. Finally, it is important to mention that the pressure wave-form and the shape of the velocity traces presented in figure 2 were preserved for all experiments defining a time reference indicated by the numbered red circles in figure 2(d). Times $t_1$ to $t_6$ correspond to 0.25T, 0.40T, 0.55T, 0.70T, 0.85T and T, respectively, being T the pulsatile period.

### 2.5. LCS identification

A common method to identify LCS is by computing the finite-time lyapunov exponents (FTLE). As demonstrated in Shadden et al (2005) the surfaces that maximize the FTLE field (i.e. ridges) correspond to the LCSs. FTLE measure the rate of separation between initially adjacent particles that are advected by the flow over a finite time interval ($\tau$). Therefore, LCS will represent surfaces of large particle separation, which act as a material barrier identifying regions with different flow dynamics.

To compute the FTLE field the first step consists in seeding the fluid domain with a grid of particles. These particles will be advected from $t_0$ to $t_0 + \tau$ by integrating the particle velocity field over time. This gives the flow map $\phi^{t_0+\tau}_{t_0}$: $r(t_0) \rightarrow r(t_0 + \tau)$, where $r$ denotes the particle position. The amount of stretching about a trajectory can be defined in terms of the Cauchy–Green tensor

$$C(t_0, t_0, \tau) = \nabla \phi^{t_0+\tau}_{t_0}(t_0), \nabla \phi^{t_0+\tau}_{t_0}(t_0)$$  \hspace{1cm} (1)

evaluated at the initial position $t_0 = r(t_0)$. The maximum stretching $||\delta r||_{max}$ is aligned with the eigenvector of maximum eigenvalue $\lambda_{max}$ of C. Consequently, the FTLE $\Lambda$ may be computed as:

$$\Lambda(t_0, t_0, \tau) = |\tau^{-1}| \ln \sqrt[3]{\lambda_{max}(t_0, t_0, \tau)}. $$  \hspace{1cm} (2)

For unsteady flows, equation (2) is computed for a range of times $t_0$ to provide a time series of the FTLE allowing to follow the dynamics of the LCSs.

Given $\tau$, trajectories can be integrated forward ($\tau > 0$) or backward ($\tau < 0$) in time. For a positive $\tau$, the FTLEs measure the rate of separation, thus identifying repelling structures ($\Lambda^+$ field). On the contrary, if $\tau$ is negative, the FTLEs measure the rate of convergence, thus identifying attracting structures ($\Lambda^-$ field). The choice of $\tau$ is usually related to the characteristic flow time scales. In this work a characteristic time scale is imposed by the period $T$ of the pulsatile flow. Another characteristic time scale is given by the advective time in the post-stenotic region ($D/v \sim 0.3–0.5$ s). In light of both time scales, $\tau = T/3 = 0.35 s$ was used in this work.

To obtain sharper fields, FTLEs are usually computed in a grid finer than the original grid used for the particle velocity field. In this work the grid of the particle velocity data was subdivided to contain $3 \times 3$ fluid particles. Further refinement of the particle grid (i.e. $4 \times 4$ and $5 \times 5$ fluid particles) resulted in negligible change in the results. Particles were advected using a 4th order Runge–Kutta method with a cubic interpolation. Lastly, as LCSs (i.e. ridges of the FTLE field) we considered all the FTLEs whose value exceeded a threshold of 50% of the maximum value of the FTLE field (this threshold value is larger than the field mean value plus three times its standard deviation).

### Table 1. Summary of the experimental flow conditions.

| Occlusion | $Re$ | $v_{max}$ (cm s$^{-1}$) | $v_{min}$ (cm s$^{-1}$) | $D_{max}$ (cm) | $D_{min}$ (cm) | $Re_{max}$ | $Re_{min}$ | $P_{max}$ (mmHg)$^a$ | $P_{min}$ (mmHg)$^b$ |
|-----------|-----|-----------------|-----------------|--------------|--------------|----------|----------|----------------|----------------|
| 50%       | 2000 Inlet | 8.11 | 0.29 | 2.49 | 2.37 | 1950 | 100 | 128 | 37 |
|           | 1500 Inlet | 17.7 | 1.39 | 1.37 | 1.12 | 2290 | 160 | 135 | 30 |
|           | 1100 Inlet | 6.13 | 0.20 | 2.37 | 2.28 | 1422 | 47 | 64 | 24 |
|           | Throat | 12.1 | 0.49 | 1.22 | 1.06 | 1401 | 55 | 59 | 22 |
| 35%       | 2000 Inlet | 8.39 | 0.21 | 2.54 | 2.40 | 1983 | 50 | 95 | 18 |
|           | 1500 Inlet | 12.67 | 0.51 | 1.77 | 1.50 | 1963 | 81 | 92 | 18 |
|           | 1100 Inlet | 7.19 | 0.32 | 2.43 | 2.24 | 1694 | 71 | 45 | 11 |
|           | Throat | 9.12 | 0.28 | 1.64 | 1.39 | 1424 | 43 | 43 | 11 |
| 0%        | 2000 Inlet | 5.74 | 0.28 | 2.35 | 2.18 | 1288 | 65 | 27 | 8 |
|           | Throat | 7.58 | 0.22 | 1.55 | 1.31 | 1107 | 32 | 25 | 8 |

$^a$ This value corresponds to the Re number set on the programmable pump.
$^b$ For $P_{max}$ and $P_{min}$, the rows 'inlet' and 'throat' correspond to inlet and outlet of the model respectively.
2.6. Residence time and final position (FP) mapping
PRT maps have shown to be a powerful lagrangian tool when analysing transport phenomena. Each pixel of the map corresponds to the fluid particle’s initial position and the pixel value is given by the time spent by this particle within the ROI. To complete the information given by the PRT maps, in this work we introduced the FP maps. Analogous to PRT maps, each pixel of the FP map corresponds to the particle’s initial position. However, for the FP maps the ROI is subdivided in different color-coded subregions. Then, the pixel value is determined by the fluid particle’s FP within these subregions. In this work the post-stenotic ROI was subdivided into three different subregions (please refer to section 3.2.1 for the definition of subregions). For the PRT and FP maps the beginning of the systolic phase ($t = 0$ in figure 2) was considered as the initial time. Fluid particles were advected over one pulsatile period following the same procedure described in the preceding subsection.

3. Results

3.1. Eulerian description of the flow: particle velocity and vorticity fields
Flow in the unobstructed model and in the upstream region of the occluded models showed a laminar velocity profile without any flow instabilities (not shown). However, in the post-stenotic region of the occluded models, flow transitioned from laminar to vortex formation and shedding as $Re$ increased. Figure 3 shows five snapshots (times $t_2$ to $t_6$ in figure 2(d)) of the instantaneous velocity field superimposed over the vorticity $\omega = \nabla \times \nu$ for different experiments. The main features of the flow are highlighted by this figure: a high velocity jet over the stenosis throat and large recirculation region behind the stenosis where vortex form and shed. For a 35% occlusion, at $Re = 2000$ a small recirculation region without shedding appeared behind the stenosis (figure 3(d)). Below this $Re$ number flow was mostly laminar. Contrary, for the model with a 50% occlusion, vortex shedding happens for all the $Re$ (figures 3(a)–(c)). As expected, as $Re$ increased the vortices become larger and vorticity values higher. By comparing figures 3(a)–(c) vortices not only become larger and stronger but they also propagate faster as $Re$ increased. By tracking the center of the vortex as a function of time, vortex propagation velocities of 1.9 cm s$^{-1}$, 2.8 cm s$^{-1}$ and 6.2 cm s$^{-1}$ were found for $Re = 1100$, 1500 and 2000 respectively.

**Figure 3.** Five snapshots of the instantaneous particle velocity field superimposed over the vorticity field $\omega$ for the following experiments (a) 50% occlusion, $Re = 2000$, (b) 50% occlusion, $Re = 1500$, (c) 50% occlusion, $Re = 1100$ and (d) 35% occlusion, $Re = 2000$. Each snapshot corresponds to the times $t_2$ to $t_6$ indicated in figure 2(d) by the red dots. The color bar is the same for all panels.
3.2. Lagrangian description of the flow

As shown in figure 5 the main structures that govern flow behind the stenosis are the high velocity jet and the vortex. Consequently, those structures will be also responsible for dictating the transport phenomena in the post-stenotic region. Figure 4 shows six snapshots of the fluid particle’s advection in the 50% occluded model for Re = 1500. Fluid particles at the beginning of the systole (i.e. pre-existing fluid) are represented in blue dots, while fluid particles entering the domain during one pulsatile period are represented in red.

The main features of the fluid transport are highlighted by figure 4. As flow enters the domain through the jet, those fluid particles situated at the leading edge of the jet do not mix with the pre-existing fluid. Contrary, mixing occurs mainly in the recirculation zone behind the stenosis where pre-existing fluid is dragged and carried away by the vortex. Finally, there are fluid particles that are relatively stagnant and remain either on or behind the stenosis for the entire cycle. Representative trajectories of these three scenarios are presented in figure 4, where the fluid particle’s trajectory and its initial positions are represented by full black lines and a black full circle, respectively. The green star represents the particle’s position for each snapshot. Trajectories A and B correspond to fluid particles that were pushed downstream by the incoming flow. Trajectory C represents a fluid particle that was pushed downstream and then dragged by the vortex. Finally, trajectories D and E represent stagnant fluid particles on and behind the stenosis, respectively.

3.2.1. Residence time and FP maps

To evaluate these different flow behaviors we computed the PRT (figure 5) and the FP maps (figure 6). For the unobstructed model (not shown) no stagnant regions were observed. All particles traveled across the ROI following an approximately straight path oriented along x-axis. The time for a particle to travel across the ROI was \( T \) while the average PRT over the ROI was \( \approx \frac{T}{2} \).

For the occluded models flow behavior was quite different. Figure 5 shows the PRT maps for the different Re in the occluded models. From figure 5 it is possible to identify the two main flow structures: the high velocity jet with low PRT values (< \( T/2 \)) and the recirculation region (on and behind the stenosis) with high PRT values (> \( T/2 \)). For a given Re, the PRT of the fluid particles initially situated in the recirculation region increases with the degree of occlusion. For example, for Re = 2000, we observe an overall increase in the PRT by comparing the 50% (figure 5(a)) to the 35% (figure 5(d)) occluded model. Moreover, we observe that the area occupied by high PRT values (i.e. close to \( T \)) is larger for the 50% occluded model. This is consistently observed for the rest of the Re by comparing figures 5(b)–(c) and figures 5(c)–(f). Lastly, for a given degree of occlusion (figures 5(a)–(c) for a 50% occlusion and figures 5(d)–(f) for a 35% occlusion), the PRT over the ROI increases as Re decreases.

Since figure 5 only gives us information about the time a fluid particle spent within the ROI without any information on its position, we complete this temporal information by introducing the FP maps (figure 6). For the FP maps the ROI was subdivided into three different subregions representing: (i) the region occupied by the jet, (ii) the region on the stenosis and (iii) the region behind the stenosis. Those regions are indicated by white dashed lines in figure 6. More precisely, region i goes from the anterior wall of the model (i.e. proximal to the ultrasonic array) to the stenosis throat, occupying the whole post-stenotic region along the x-direction. Regions ii and iii both go from the stenosis throat to the posterior wall. However, region ii goes from \( X \approx 0.4 \) cm to...
$X \approx 3\, \text{cm}$, while region iii goes from $X \approx 3\, \text{cm}$ to $X \approx 4.8\, \text{cm}$, representing the regions on and behind the stenosis, respectively. The choice of these regions allowed us to evaluate the three different scenarios described above for figure 4: fluid particles pushed by the flow, fluid particles dragged by the vortex and stagnant particles that stayed on and behind the stenosis. In figure 6 the colors cyan, orange and red correspond to a fluid particle’s FP within regions i, ii and iii, respectively, which are indicated by a dashed white line. The color blue is for particles that left the region in one period.

In figure 6, we observe that part of the fluid particles with high PRT values, which initially were located on the stenosis (region ii), moved to region iii while the rest remained on the stenosis (i.e. region ii). The number of particles remaining on the stenosis does not strongly depend on $Re$. By comparing figures 6(a)–(c) and figures 6(d)–(f) we observe that the size of the orange surface remains approximately the same. However, we observe that the lower the $Re$ the more particles remain in region i and iii. Finally, we also observe some cyan regions within region ii for figures 6(a) and (b). These regions correspond to fluid particles that were dragged into region i by the vortex.
3.2.2. FTLE fields

Figure 7 shows five snapshots of the instantaneous ridges of the FTLE fields superimposed to the instantaneous particle velocity field for the following experiments: (a) 50% occlusion, $Re = 2000$, (b) 50% occlusion, $Re = 1500$, (c) 50% occlusion, $Re = 1100$, and (d) 35% occlusion, $Re = 2000$. Each snapshot corresponds to the times $t_2$ to $t_6$ indicated in figure 2(d). Repelling $(\Lambda^+)$ and attracting $(\Lambda^-)$ LCSs are represented in red and blue respectively. The color-scale is the same for all panels.

4. Discussion

In this work Eulerian and Lagrangian descriptors have been applied to study vortex dynamics and transport phenomena in stenotic aortic models with morphology, mechanical and optical properties close to those of real arteries. As described by previous numerical and experimental studies conducted in axisymmetric (Varghese et al 2007, Pielhop et al 2012, Geoghegan et al 2013) and non-axisymmetrical models (Usmani and Muralidhar 2016, Choi et al 2018), we observed that the flow in the post-stenotic region is composed of two main structures: a high velocity jet over the stenosis throat and a recirculation region behind the stenosis where vortex form and shed. As described by the early experimental works of Ojha et al (1989) in rigid stenotic models, flow in the poststenotic region may be divided in several zones characterized by different flow behaviors as a function of the distance to the stenosis throat. For a distance below three diameters there is a stable jet zone where vortex form and shed. Then, it follows a transition region, a turbulent region (between 4.5 and 7.5 diameters from stenosis) and relaminarization zone (after 7.5 diameters). This was later observed in the numerical works of Sherwin and
Blackburn (2005) and Varghese et al (2007). More recently, the work of Choi et al (2018) studied the turbulent kinetic energy generation in a deformable stenosis of 45%–48% severity in diameter for a peak $Re = 1803$. They found that the transition to turbulence begins at a distance of at least one diameter downstream of the stenosis throat. The downstream ROI used in our experiments goes up to approximately 1.5 diameter. Therefore, all experiments lie within the stable jet region. As shown in figure 2, pressure, lumen and velocity shown for $Re = 2000$ (highest $Re$ in our experiments) show negligible fluctuations and flow behavior in our experiments may be considered as periodical. Only, in the experiment for the 50% occlusion at $Re = 2000$ small velocity fluctuations were observed at the end of the ROI ($X > 4 \text{ cm}$ in figure 3(a)). This may indicate the beginning of the transition to turbulence region. However, a turbulence analysis is needed to fully verify this point, which is out of the scope of the current work.

For those experiments where vortex shedding was observed, we were able to track the vortex center from the vorticity snapshots and measure its axial velocity of propagation. Although we could not find any values reported in the literature for the vortex velocity of propagation in stenotic vessels, we were able to deduce them from the work of Geoghegan et al (2013). The values obtained in our work are smaller by factor $\sim 10$ when compared to the results deduced from (Geoghegan et al 2013). However, those experiments were conducted in an axisymmetrically occluded silicone model. Since the $Re$ used in our experiments and in Geoghegan et al (2013) are similar, the difference between the results is probably related to the elasticity difference between models, since silicone is far less compliant than PVA. Moreover, in their work they report a Kelvin–Helmholtz instability, which we do not observe in our experiments due to the asymmetry of our model. This is consistent with (Usmani and Muralidhar 2016, Choi et al 2018).

By computing the FTLE fields (figure 7), we were able to identify different material barriers associated to the vortex and the leading edge of the jet. Analogous LCSs were reported by previous studies but in significantly different hemodynamic situations. For example, in Hendabadi et al (2013) and Espa et al (2012) FTLE fields were computed to study transport in the left ventricle. In those studies, they found that during the left ventricle filling the backward FTLE field reveals a well-defined LCS that separates the injected blood from the pre-existing one. Other numerical studies in abdominal aortic aneurysm showed analogous behavior during diastole (Shadden and Taylor 2008, Arzani and Shadden 2012). The LCS described in these works is analogous to the attracting barrier I identified in figure 7. However, contrary to what is described in Shadden and Taylor (2008), Arzani and Shadden (2012), Espa et al (2012), Hendabadi et al (2013), due to the asymmetric nature of our models and flow, the LCS I does not roll originating a vortex ring whose trailing edge (i.e. facing upstream) is enclosed by a repelling barrier. This is evident from figure 7(a) for times $t_3$ and $t_4$: as vortex leave the post-stenotic region there are still ridges of the FTLE field separating the jet from the recirculation region.

In this work we also identified material barriers associated to the vortex (LCSs IIA and IIB in figure 7). Both LCSs surround the vortex at all times and are the main responsible for the mixing between the incoming and the pre-existing fluid as described in Shadden et al (2007). By comparing figures 4 and 7(b) we observed that during vortex build up, pre-existing fluid gets trapped between barriers IIA and IIB and travel along the vortex getting mixed and stirred with the incoming fluid. Similar dynamics were also observed for $Re = 2000$ and 1100, although it was not illustrated in figure 4. For the 35% occluded model at $Re = 2000$ those barriers were also visible around the vortex (specially for times $t_5$ and $t_6$ in figure 7(d)). Analogous results were reported by computing the FTLE fields from numerical studies in aortic aneurysm (Shadden and Taylor 2008, Arzani and Shadden 2012) and in the experimental work of Vétel et al (2009) in a model of a carotid bifurcation (Vétel et al 2009).

Lastly, in this work we showed that the strength of this barriers increases with $Re$ and consequently with vorticity.

As done previously (Di Labbio et al 2018, Hendabadi et al 2013, Darwish et al 2020), in this work the combination of the FTLE fields along with the PRT and FP maps helped to provide a more detailed description of the flow transport behind the stenosis. As observed in figures 4 and 7 the materials barriers identified in this work (i.e. LCSs I, IIA and IIB) are mainly associated to the fluid particles entering the domain (red dots in figure 4), while the PRT and FP maps provide detailed information on the transport of the pre-existing fluid (blue dots in figure 4). Although LCSs are responsible for high/low PRT and for the particles’ FP maps, the identification of stagnant regions from the FTLE field is not straightforward. Instead, the PRT and FP maps allowed direct visualization of those stagnant regions and study its dependence with $Re$ and the stenosis degree. By comparing figures 5 and 6, we observed that part of the fluid initially located on the stenosis (region ii), moved to region iii while the rest remained stagnant on the stenosis. In figure 6 we observed that the number of these stagnant particles does not strongly depend on $Re$ for the range of values explored in this work. Contrary, results reported by Jeronimo et al indicate that the number of stagnant particles increases as $Re$ increases (Jeronimo and Rival 2020), however, comparison between their results and the ones reported in this work is not straightforward. In Jeronimo and Rival (2020) they used peak $Re$ ranging from 7200 to 28 080 in a rigid smooth axisymmetric constriction followed by an unrealistic sudden expansion, which is quite different from the setup used in this work.
The arterial models used in this work have shown to exhibit nonlinear, anisotropic elastic properties close to those of real arteries (Bernal et al 2019a, 2019b). In this work different fluid structures (i.e. LCSs I, IIa and IIb) were identified. Moreover, the fluid transport induced by those structures was studied as a function of Re and stenosis degree using the PRT and FP maps. However, the impact of the anisotropic and nonlinear elastic properties on the fluid structures was not investigated. Future studies should focus on comparing rigid, linearly elastic and nonlinear elastic models to further understand how the arterial wall elasticity influences flow, particularly, the fluid structures described in this work.

In this study the profile of the stenosis was set to be of a smooth Gaussian shape. However, in vivo plaques are much more complex. Specifically, they are composed of multiple material entities and they present a much more corrugated non-smooth surface. Several studies have addressed the influence of the stenosis shape and roughness on the flow dynamics (Kefayati et al 2014, Choi et al 2018). For example, Choi et al (2018) demonstrated that jet deflection in a deformable stenosis model increased the rate of jet velocity and turbulent kinetic energy production when compared to a rigid model. Therefore, the shape and roughness of the stenosis will impact the fluid dynamics and transport phenomena. As demonstrated by Asnaghi et al, roughness will increase the disorder of flow reducing the vortex strength (Asnaghi et al 2020). Therefore, roughness on the stenosis will directly impact the material barriers associated to the vortex (i.e. LCSs IIa and IIb in figure 7) via turbulence generation. As a result, this will impact the FP and PRT maps which are essentially dictated by those structures. Specifically, the strength of these barriers will diminish with increasing roughness followed by an increase on the fluctuations on the particles trajectories. Consequently, we hypothesize that less particles will be caught by the vortex increasing their PRT and remaining stagnant behind the stenosis. However, future studies should be focused on varying the shape and roughness of the stenosis to verify this point. Lastly, it is important to mention that the methodology presented in the current work will be equally useful in studying fluid behavior in more realistic plaque models with disordered flow.

Our experiments were limited by the acquisition time of the ultrasound scanner. Consequently, particle’s advection was restricted to one pulsatile period as in Jeronimo and Rival (2020). Moreover, the velocity imaging through Echo-PIV was restricted to the center plane of the model. The current setup did not allow the study of the three dimensional nature of the flow. Future studies should aim in incorporating three dimensional information on vortical structures and transport phenomena. This can be implemented with the advent of new 3D ultrasound imaging technologies (Jensen et al 2016b) like matrix (Correia et al 2016) and row-columns (Sauvage et al 2018) arrays.

Lastly, combining LCS, PRT and FP maps provides an alternative approach to understand flow dynamics in stenotic models. As shown by Shadden and Hendabadi (2013) platelet activation potential is maximized along the LCSs. Moreover, from the PRT and FP maps we showed that stagnant particles remained on stenosis after one pulsatile period. These stagnant particles spend more time on the stenosis where platelet activation potential is large (i.e. large FTLE values as in figure 7) which may favor blood clot formation leading to stenosis growth. Further studies should focus on exploring the link between the fluid structures demonstrated in this work and their potential involvement in stenosis growth.

Furthermore, future studies should focus on the translation of the methodology and tools presented in this work into clinical applications. Echo-PIV with contrast agents has been widely used in the heart (Sengupta et al 2007, Kheradvar et al 2010, Gao et al 2015) and in vessels (Qian et al 2010, Zhang et al 2011, Poelma et al 2012). The main drawback of Echo-PIV is the intravenous administration of gas-filled microbubbles as contrast agent (Jensen et al 2016a). Moreover, other vector flow imaging modalities (e.g. vectorial Doppler, transverse oscillations or vectorial flow imaging using MRI) (Correia et al 2016, Jensen et al 2016a, 2016b, 2017, Yiu and Alfred 2016, Sauvage et al 2018, Madiena et al 2018, Nyrnes et al 2020) can be used to measure flow, while using a similar analysis (LCS, PRT and FP maps) to determine the fluid structures and transport phenomena in more complex models (Poepping et al 2004, Chee et al 2016, King et al 2011) or in particular clinical applications.

5. Conclusion

By combining Eulerian and Lagrangian descriptors, we analyzed the flow dynamics and transport phenomena in stenotic aortic models with morphology, mechanical and optical properties close to those of real arteries. To this end, vorticity, FTLEs, PRT and FP maps were computed from the particle velocity fields acquired using Echo-PIV. We characterized vortex kinematics showing that vortex propagation velocity increases with Re number. From the FTLE field we identified material barriers that dictate transport behind the stenosis. The size and strength of these barrier is also Re dependent. Moreover, from the PRT and FP maps, we showed that even for the highest Re, fluid parcels remain on the stenosis. Lastly, combining LCS, PRT and FP maps may provide an alternative method to study the flow dynamics in atherosclerotic models.
Acknowledgments

This work was supported by the CSIC–Uruguay R+D 2016 Project ‘Estudio de la dinámica de un flujo pulsátil y sus implicancias en hemodinámica vascular’, the CSIC–Uruguay grant ‘2018—FID 13—grupo ID 722—Física No Lineal’, ANII–Uruguay, PEDECIBA Física—Uruguay, the Administrative Department of Science, Technology and Innovation of the Colombian Government (Colciencias) and the Research and Development Center (CIDI) of the Universidad Pontificia Bolivariana through the program “Es tiempo de Volver” (Grant number 548B–01/16–04), Colombia. Nicasio Barrere acknowledges a doctoral scholarship (POSNAC–2015–1–109843) from ANII–Uruguay.

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