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A numerical parametric study of the mechanical action of pulsatile blood flow onto axisymmetric stenosed arteries

Tristan Belzacq*†, Stéphane Avril† Emmanuel Leriche‡ and Alexandre Delache‡

*Corresponding author
Email: belzacq@emse.fr

†Ecole Nationale Supérieure des Mines, Centre Ingénierie et Santé, CNRS UMR 5146, 158 cours Fauriel, 42023 Saint-Etienne cedex 2, France

‡ Laboratoire de Mécanique de Lille, Université de Lille, Avenue Paul Langevin, Cité Scientifique, F-59655 Villeneuve d'Ascq cedex, France

‡ Laboratoire de Mécanique des fluides et d'acoustique LMFA, site de St-Etienne, Université de Lyon, F-42023 St-Etienne, France, Université Jean Monnet de St-Etienne, Faculté des Sciences et Techniques, 23 rue du Docteur Paul Michelon, 42023 St-Etienne cedex 2, France
Abstract

In the present paper, a fluid structure interaction model is developed, questioning how the mechanical action of the blood onto an atheromatous plaque is affected by the length and the severity of the stenosis. An axisymmetric model is considered. The fluid is assumed Newtonian. The plaque is modelled as a heterogeneous hyperelastic anisotropic solid composed of the arterial wall, the lipid core and the fibrous cap. Transient velocity and pressure conditions of actual pulsatile blood flow are prescribed. The simulation is achieved using the Arbitrary Lagrangian Eulerian scheme in the COMSOL commercial Finite Element package. The results reveal different types of behaviour in function of the length (denoted \(L\)) and severity (denoted \(S\)) of the stenosis. Whereas large plaques \((L>10\text{mm})\) are mostly deformed under the action of the blood pressure, it appears that shorter plaques \((L<10\text{mm})\) are significantly affected by the shear stresses. The shear stresses tend to deform the plaque by pinching it. This effect is called: “the pinching effect”. It has an essential influence on the mechanical response of the plaque. For two plaques having the same radius severity \(S=45\%\), the maximum stress in the fibrous cap is 50\% larger for the short plaque \((L=5\text{mm})\) than for a larger plaque \((L=10\text{mm})\), and the maximum wall shear stress is increased of 100\%. Provided that they are confirmed by experimental investigations, these results may offer some new perspectives for understanding the vulnerability of short plaques.

Keywords: vascular biomechanics; fluid structure interaction; numerical simulation; stenosis; blood flow
Global variables:

- $\rho$ the density
- $\eta$ the viscosity
- $R_0$ the healthy arterial radius
- $h_0$ the thickness of the healthy arterial wall
- $e$ the fibrous cap thickness
- $L$ the stenosis length
- $R_m$ the external stenosis remodelling radius
- $S$ the stenosis severity in terms of diameter
- $S_{area}$ the stenosis severity in terms of area
- $(\alpha_k)_k$ the series of Womersley number
- $e_z$ the longitudinal axis in the cylindrical frame
- $e_r$ the radial axis in the cylindrical frame
- $n$ exterior normal
- $u$ the displacement vector
- $v$ the velocity
- $p$ the pressure
- $\sigma$ the stress tensor
- $F$ the deformation gradient tensor
- $C$ the Green-Cauchy right strain tensor
- $\varphi$ the strain energy density
- $J$ the volume change invariant
- $I_1, I_4$ and $I_6$ the first, fourth and sixth coordinate invariants
- $c, k_1$ and $k_2$ the parameters of the Holzapfel model
- $\beta$ the angle between these two fibres family
- $M$ and $M'$ the direction’s vectors of the two fiber’s family in the cylindrical coordinate
- $w$ the mesh velocity in the Arbitrary Lagrangian Eulerian scheme
- $\tau$ the wall shear stress
- $\varepsilon_r$ the average radial strain of the plaque
- $\gamma$ the average shear strain of the plaque
- $U_r$ the radial displacement component of the fibrous cap at the middle of the stenosis
- $U_z$ the longitudinal displacement of the fibrous cap at the middle of the stenosis
- $P_1$ the linear type of finite elements
- $P_2$ the quadratic type of finite elements
- $\Omega$ a domain
- $\partial \Omega$ a boundary

Indexes:

- $s$ the solid part
- $f$ the fluid part
- $fs$ fluid-structure
- $i$ the material number ($1 \leq i \leq 4$)
- $in$ inlet
Operators:

- det(.) the determinant of a tensor
- $\frac{\partial}{\partial t}$ the first derivative with respect to time
- $\frac{\partial^2}{\partial t^2}$ the second derivative with respect to time
- $\nabla$, the divergence operator
- $\nabla$, the gradient operator
- $I$, the identity matrix
- $A^T$, transpose of the matrix $A$
- $\text{Re}(.)$, the real part of an imaginary number
- $J_0(.)$, the zero order Bessel function of first kind

1. Introduction

The fracture of vulnerable carotid atherosclerotic plaques is the major cause of cerebrovascular thromboembolic events such as strokes and ischemic attacks [1]. The vulnerability is believed to be related to mechanical forces, vessel surface condition, cell activities and chemical environment [2-4].

In current clinical practice, carotid endarterectomy is the most frequently used treatment for pathological plaques. The decision leading to surgery is based on the degree of endoluminal stenosis [5,6]. But this criterion alone is insufficient to predict the plaque fracture and the necessity to identify other criteria is a major issue for public health [1]. Therefore, there is a strong medical and economical interest in developing new tools for a better understanding of this situation.
Histological studies have related plaque vulnerability with thin fibrous cap, large necrotic core [2] and inflammation after macrophages or T-cells infiltration in the lipid core [3] or foam cells infiltration in the fibrous cap [4]. Advanced magnetic resonance imaging (MRI) allows \textit{in vivo} virtual histology of plaques [7,8].

There has also been considerable effort using computational models to perform mechanical analysis for atherosclerotic plaques and identify critical mechanical descriptors as stresses or strains related to plaque rupture. There is no universal technique that can measure the strain/stress field in the plaque but it is widely believed that stress concentration in the fibrous cap of vulnerable plaques can cause the rupture [9,10]. Large cyclic variations of strain/stress may also lead to artery fatigue [11-13]. Several studies, for example, indicate that the critical strain/stress conditions are affected by the stenosis severity, the lipid pool size, the fibrous cap thickness and the stenosis eccentricity [14-17].

2-D and 3-D patient-specific finite-element (FE) models of diseased vessels are probably the best way to obtain stress distributions for specific plaques. The models are based on histology or pre-fracture medical imaging [18]. Their purpose is to associate the mechanical descriptors to the mechanical process of plaque rupture.

Nevertheless the sparsity of data regarding plaque rupture reveals the limitations of the predictive models. The models may be:

- fluid models [19-21] with rigid plaques
- 2D solid models under pressure loads [14,22-26] considering cross sections of idealized plaques
or fluid structure interaction (FSI) models taking into account both the combination of shear and pressure loads [9,10,18,27-33].

FSI models are probably the most realistic models but their models are complex. The interaction between the blood flow and the plaque is not yet fully understood, especially due to the large variability of plaques [8]. Using idealized models allows investigating how the mechanical action of the blood onto the plaque is affected by the geometry and the mechanical properties. For instance, Li Z.Y. et al. [15] studied the influence of stenosis severity and fibrous cap thickness on stresses in plaques with 2D plane-strain models. Li M.X. et al. [16] investigated the stress distribution for different degrees of stenoses also with 2D-axisymmetric models. Valencia et al. [34] investigated the influence of the severity of stenoses on stresses with 3D-axisymmetric models. Tang et al. performed many 3D FSI studies on idealized plaque models. They analysed wall stress and strain in symmetric and asymmetric plaque models with two different stenosis severities: 50% and 78% [17,35]. They analysed also the influence of pressure loads and stenosis severity on the cyclic compression of plaques [12,36].

The effect of plaque length has never been investigated in the literature. The present paper aims at addressing this lack. A FSI 2D axisymmetric model of the blood flow in a smooth pipe is considered, with a thick, deformable, heterogeneous and axisymmetric stenosis, mimicking an atherosclerotic plaque in a straight segment of artery. The model allows rapid modifications of the geometrical and constitutive parameters of the plaque for evaluating the influence of all these parameters.
2. Methods

2.1 Geometrical model

A schematic of the geometry in the reference configuration (i.e. in unloaded pressure-free conditions [32,37]) is provided in Fig. 1. The model of the plaque is axisymmetric (Fig. 1-D). The initial stenosis shape starts from a sinus shape function along the longitudinal direction. It is composed of the healthy arterial wall outside and of a fibrous cap containing the lipid core inside (Fig. 1-B).

The healthy arterial lumen has an inner radius of \( R_0 = 3 \text{ mm} \) and a wall thickness of \( h_0 = 0.5 \text{ mm} \) (Fig. 1-C), which corresponds to average values for the wall of the carotid artery [38].

Most of vulnerable carotid plaques present a positive remodelling [39] which is modelled as an external remodelling radius of the vessel. It is set here to \( R_m = 1 \text{ mm} \) [39].

The reference for the plaque length is set to \( L = 10 \text{ mm} \). This value corresponds to the average length of carotid plaques [40]. The stenosis height \( H_0 \) is related to the stenosis severity, denoted \( S \). Eq. 1 gives the expression of the stenosis severity as the ratio between the stenosis height and the healthy radius [5,6]. In the present study as in [5,6], the radius severity is considered. Clinicians sometimes consider the stenosis severity in terms of cross-sectional area reduction (denoted \( S_{area} \)). Eq. 2 gives the conversion formula between the radius severity and the area severity.
\[ S = 1 - \frac{R_0 - H_0 \cdot e}{R_0} \]  
\[ S_{area} = 1 - (1 - S)^2 \]  

According to NASCET or ECST studies [5,6], a plaque is vulnerable if the stenosis radius severity is above 70%. However it is widely believed that this criterion alone is not sufficient to characterize the vulnerability of plaques [1]. The current study considers moderately severe plaques (reference value \( S = 45\% \)) and investigate the effect of other parameters on the vulnerability.

The fibrous cap thickness is a critical geometrical characteristic for plaque vulnerability because thrombo-embolic events result from the fibrous cap rupture. To place the study in a case of vulnerable plaque, a thin fibrous cap is considered (\( e = 0.1 \text{mm} \)) [15]. To simplify the idealized plaque model, the fibrous cap thickness is homogeneous along the stenosis.

The arterial length upstream stenosis is set to 20 mm. This is necessary for establishing the flow without having the influence of inflow boundary conditions prescribed at the inlet. For the same reason, the arterial length downstream the stenosis is set to 50 mm ensuring the establishment of the eventual flow recirculation.

### 2.2 Fluid and structural equations

A pulsatile flow of a viscous Newtonian and incompressible fluid is considered in an axisymmetric pipe (Fig. 1-A), with a pulsation \( \omega = 2\pi \) [41]. This flow behaviour is suitable for simulating the flow in large arteries because Non-newtonian effects are believed to have a
minor influence (see discussion in section 4.3). Its dynamic viscosity is denoted $\eta_f$ and the
density of the fluid is denoted $\rho_f$. The fluid velocity field $\mathbf{v}_f$ and pressure field $p$ are
governed by the unsteady incompressible Navier-Stokes equations written in the Arbitrary
Lagrangian-Eulerian (ALE) formulation \cite{42}:

$$
\begin{align*}
\rho_f \frac{\partial \mathbf{v}_f}{\partial t} + \rho_f (\mathbf{v}_f - \mathbf{w}) \cdot \nabla \mathbf{v}_f - \nabla \mathbf{f} = 0 \\
\nabla \cdot \mathbf{v}_f = 0
\end{align*}
$$
in $\Omega_f$, \hspace{1cm} (3)

where $\mathbf{w}$ is the mesh velocity related to the ALE formulation, $\mathbf{f}$ the Cauchy stress tensor in
the fluid and $\Omega_f$ is the fluid domain depicted in Fig.1-A.

The displacement vector, denoted $\mathbf{u}_s$, and the Cauchy stress tensor, denoted $\mathbf{f}_s$, of the solid
part, with respect to the reference configuration, satisfy the following equation \cite{43}:

$$
\rho_s \frac{\partial^2 \mathbf{u}_s}{\partial t^2} - \nabla \mathbf{f}_s = 0 \text{ in } \Omega_s ,
$$

where $\Omega_s$ is depicted in Fig.1-A and $\rho_s$ is the density of the constituents in the solid domain,
which is assumed homogeneous.

### 2.3 Fluid and structural boundary conditions

At the inlet of the fluid domain, a Womersley velocity profile \cite{44} is applied and at the outlet,
a pressure is imposed implying no normal viscous stress (see Eq. 5). The time variations of
the pressure and the velocity profiles are written using Fourier decomposition, with 18 and 6
terms respectively for ensuring agreement with experimental data. Fourier decomposition is
applied to the temporal signals shown in Fig. 2.
\[
\begin{align*}
v_f(r,t) &= \frac{c_0}{4\eta_f} R^2 (1 - \frac{r^2}{R^2}) - \frac{R^2}{\eta_f} \sum_{k=0}^{6} \text{Re} \left( \frac{ic_k}{\alpha_k^2} \left( 1 - \frac{J_0 \left( \frac{\alpha_k}{3} \frac{r^3}{R} \right)}{J_0 (\alpha_k i^2)} \right) e^{ik\omega t} \right) e_z \quad \text{on} \quad \partial^\text{in} \Omega_f \\
p(t) &= \sum_{k=0}^{16} \text{Re} \left( p_k e^{ik\omega t} \right) \quad \text{on} \quad \partial^\text{out} \Omega_f \\
\sigma_{\text{viscous}} \cdot e_z &= 0 \quad \text{on} \quad \partial^\text{out} \Omega_f
\end{align*}
\]

where \( \partial^\text{in} \Omega_f \) and \( \partial^\text{out} \Omega_f \) are respectively the inlet and the outlet boundaries of the fluid depicted in Fig. 1-A, \( e_z \) is the longitudinal vector and \( J_0 \) is the zero order Bessel function of first kind, \( R \) is the radius of the pipe at the inlet and \( (\alpha_k = R \sqrt{\frac{k^2 \rho_f}{\eta_f}})_{0 \leq k \leq 6} \) is the series of Womersley numbers.

\( \sigma_{\text{viscous}} \) represents the viscous stress part of the Cauchy stress tensor in the fluid \( \sigma_f \) (see Eq. 8 in the section 2.4).

The axial velocity at the inlet is represented in Fig. 2-A and the pressure at the outlet is represented in Fig. 2-B [46].

The Fourier coefficients \( c_k \) (Tab. 3) of the Womersley profile are computed from the axial velocity data measured non-invasively using PC-MRI in the common carotid artery of a volunteer [46]. Moreover the pressure Fourier coefficients \( p_k \) (Tab. 3) are deduced from the variations over cardiac cycles of the pressure measured non-invasively using the applanation tonometry technique on the same volunteer [47].
At the boundaries of the elastic solid, the displacement vector $\mathbf{u}$, and the Cauchy stress tensor $\sigma$, with respect to the reference configuration, satisfy the following equations [43]:

$$
\begin{align*}
\sigma_s \mathbf{n}_{s,ext} &= 0 \quad \text{on} \quad \partial^{ext} \Omega_s \\
\mathbf{u}_s \cdot \mathbf{e}_z &= 0 \quad \text{on} \quad \partial^{in} \Omega_s \quad \text{and} \quad \partial^{out} \Omega_s
\end{align*}
$$

(6)

where $\partial^{ext} \Omega_s$, $\partial^{in} \Omega_s$, $\partial^{out} \Omega_s$ and the normal vector $\mathbf{n}_{s,ext}$ are depicted in Fig.1-A.

At the fluid-structure interface $\partial \Omega_{fs}$ (see Fig. 1-A), the kinematic and dynamic conditions apply, ensuring continuity of velocity fields and normal stresses:

$$
\begin{align*}
\mathbf{v}_f &= \frac{\partial \mathbf{u}_s}{\partial t} = \mathbf{w} \\
\sigma_f \mathbf{n}_s + \mathbf{J}_f \mathbf{n}_f &= 0 \quad \text{on} \quad \partial \Omega_{fs}
\end{align*}
$$

(7)

where $\partial \Omega_{fs}$ and the normal vectors $\mathbf{n}_f$ and $\mathbf{n}_s$ are depicted in Fig. 1-A. In the fluid domain, mesh velocity $\mathbf{w}$ is derived following a Laplace smoothing method from the interface conditions.

2.4 Fluid and structural properties

In a viscous Newtonian incompressible fluid, the expression of the Cauchy stress tensor depends linearly on the strain rate:

$$
\sigma_f = -\rho \mathbf{I} + \eta_f (\nabla \mathbf{v}_f + \nabla^T \mathbf{v}_f) ,
$$

(8)
with \( \eta_f = 0.005 \text{Pa.s} \) [42]. The first and second terms are respectively the hydrostatic component \( \sigma_f^{\text{hydrostatic}} \) and the viscous stress component \( \sigma_f^{\text{viscous}} \). The density of the fluid is set to \( \rho_f = 1050 \text{kg/m}^3 \) [41].

Holzapfel et al. established a hyperelastic anisotropic constitutive equation for artery components [48]. This model is used here for each component of the plaque. Hyperelasticity implies the existence of a strain energy density function \( \varphi \) depending upon the Green-Cauchy right strain tensor \( \mathbf{C} = \mathbf{F} \mathbf{F}^\top \), where \( \mathbf{F} \) is the deformation gradient tensor [43]. Thus \( \varphi = \varphi(\mathbf{C}) \) and the associated Cauchy stress tensor is given by:

\[
\mathbf{\sigma}_s = 2J^{-1}\mathbf{F} \frac{\partial \varphi}{\partial \mathbf{C}} \mathbf{F}^\top,
\]

where \( J = \det(\mathbf{F}) \) is the volume change invariant. This formulation is valid for a solid without residual stresses. It was proposed in [48] to separate the isotropic and the anisotropic part of \( \varphi \). The anisotropy is defined by two preferred directions corresponding to two families of collagen fibres. The angle between these two families of fibres is denoted \( \beta \). \( \mathbf{M} = \begin{pmatrix} 0 \\ \cos(\beta) \\ \sin(\beta) \end{pmatrix} \) and \( \mathbf{M}' = \begin{pmatrix} 0 \\ \cos(\beta) \\ -\sin(\beta) \end{pmatrix} \) represent the directions of fibres in the local coordinate system.

Then, the strain energy density \( \varphi \) is written such as:

\[
\varphi(I_1, I_4, I_6, J) = \frac{c}{2} (I_1 - 3) + \frac{k_1}{2k_2} \sum_{i=4,6} (e^{k_2(I_i - 1)^2} - 1) + \frac{\kappa}{2} (J - 1)^2,
\]

where \( I_1 = tr(\mathbf{C}) \), \( I_4 = \mathbf{M}.(\mathbf{C}\mathbf{M}) \) and \( I_6 = \mathbf{M}'.(\mathbf{C}\mathbf{M}') \), \( c \), \( \kappa \), \( k_1 \) and \( k_2 \) are material parameters. Arterial tissue is often assumed as nearly incompressible [49]. The modulus of
compressibility $\kappa$ is set to 500,000 kPa here in order to ensure this hypothesis. The density is set to $\rho_s = 900 \text{kg/m}^3$. Reference values of parameters $c$, $k_1$ and $k_2$ are given in Tab. 1. They were taken from experimental data [50].

To simplify the model, average properties of the media and of the adventitia reported in [50] are taken. For deducing the properties of the healthy artery reported in Tab. 1, a weighted average is derived, with a weight of $\frac{2}{3}$ for the media and a weight of $\frac{1}{3}$ for the adventitia [38]. The equation used to derive parameter $c$ is given Eq. 11. A similar equation is used for the other mechanical parameters ($k_1$, $k_2$ and $\beta$).

$$c_{\text{wall}} = \frac{2}{3} c_{\text{media}} + \frac{1}{3} c_{\text{adventitia}} \quad (11)$$

2.5 Numerical computation

FSI simulations are performed using commercial FE solver COMSOL Multiphysics [51]. The compatible finite-element types are $P_2$ for the fluid velocity $v_f$, $P_1$ for the fluid pressure $p$ and $P_2$ for the solid displacement $u_s$ [52]. The model geometry is meshed using triangular mesh generation in COMSOL Multiphysics, consisting in $N_f$ elements for the fluid domain ($2680 \leq N_f \leq 3316$) and $N_s$ for the solid domain ($3476 \leq N_s \leq 4325$), with $N_{\text{fibrous cap}}$ elements in the thickness of the fibrous cap ($1025 \leq N_{\text{fibrous cap}} \leq 1650$), depending on the different model geometries considered in the parametric study. The coupled fluid-structure problem is discretized using a Galerkin-Least-Square method (GLS) and an implicit temporal discretization of order 5 using a Backward Differentiation Formula (BDF) with an adaptive time step. The non-linear problems are solved using a Newton-Raphson algorithm.
The mesh is refined close to the wall in order to take into account the viscous boundary layer: the mesh size is prescribed 3 times finer near the wall than at the centre.

The minimum and the maximum Reynolds number (Eq. 12) are respectively $Re_{\text{min}} = 300$ and $Re_{\text{max}} = 2000$. The value of $Re_{\text{max}}$ justifies the use of a turbulence model [41]. The flow is modelled as being turbulent with the $k-\omega$ model [51].

$$Re = \frac{\rho V D}{\mu}, \quad (12)$$

where $D$ is the inner diameter of the artery and $V$ is the axial velocity: for $Re_{\text{min}}$, $D$ and $V$ are taken at diastole in the healthy artery upstream the plaque and for $Re_{\text{max}}$, $D$ and $V$ are taken at systole at the top of the stenosis.

The geometry described in section 2.1 is the unloaded pressure-free geometry corresponding to the initial condition of the numerical computation. The simulation is performed over four cardiac cycles. During the first cardiac cycle, the pressurization and the average blood flow are applied before considering pulsatile effects. During this stage, the fluid viscosity, the pressure at the outlet and the velocity at the inlet are set gradually in order to ensure numerical convergence:

- the axial velocity at the inlet is increased linearly from 0m/s to 0.3m/s (see $v_z(t=0)$ Fig. 2-A)
- the pressure at the outlet is increased linearly from 0mmHg to 91mmHg (see $p(t=0)$ Fig. 2-B)
- the fluid viscosity is decreased linearly from 0.05Pa.s to 0.005Pa.s
Afterwards, three cycles of the actual pulsatile flow (see Fig. 2) are computed. The flow is fully established and periodic over the last cycle. Hence, the last cycle is used for the analysis.

### 2.6 Parametric study

In our model, different plaque parameters are set for reproducing the variability of real plaques:

- the fibrous cap thickness denoted $e$,
- material parameter $k_1$ in the arterial wall and in the fibrous cap, respectively denoted $k_{1,\text{wall}}$ and $k_{1,\text{cap}}$,
- stenosis severity $S$ defined in Eq. 1
- stenosis length $L$.

Each parameter is set independently of the other parameters (unidirectional parametric study). The geometrical properties of the reference model are: $e = 0.1\,\text{mm}$, $S = 45\%$, $L = 10\,\text{mm}$. The reference values for $k_1$ are reported in Tab. 1. Simulations and analysis are performed for a wide range of these parameters using an interface between the COMSOL software and the MATLAB software [51,53].

The current study considers moderately severe plaques, between 20% and 70%. The current study is more focused on the effect of the stenosis length, as the effect of this parameter is not clearly understood and there exists a large range of plaque lengths. Plaques in the carotid artery may be short ($L < 10\,\text{mm}$) [18,40]. Therefore, plaque lengths ranging between 5mm and 20mm are considered in this study (20 mm corresponds to a very long plaque [40]).
The fibrous cap thickness and the material parameters are taken into account for investigating the effect of the plaque stiffness.

### 2.7 Analysed criteria

The analysis is focused on the response at the systole. A special attention is paid to the following criteria:

- the maximum von Mises equivalent stress in the fibrous cap defined as,

\[
\sigma_{\text{VM}}^{\text{max}} = \text{Max}(\sigma_{\text{VM}}) = \text{Max}(\sqrt{\sigma_1^2 + \sigma_2^2 - \sigma_1 \sigma_2}),
\]  

where \(\sigma_1\) and \(\sigma_2\) are the principal stresses in the radial and longitudinal directions.

- the maximum wall shear stress (WSS),

\[
\tau_{\text{max}} = \text{Max}(\|\sigma_{\text{viscous}} f\|)
\]

- the average radial strain of the plaque,

\[
\varepsilon_R = \frac{U_R}{H_0 + h_0 + e + R_m},
\]

where \(U_R\) is the radial displacement of the fibrous cap at the middle of the stenosis.

- the global shear strain of the plaque,

\[
\gamma = \frac{U_z}{H_0 + h_0 + e + R_m},
\]

where \(U_z\) is the longitudinal displacement of the fibrous cap at the middle of the stenosis.
The maximum von Mises equivalent stress, $\sigma_{\text{VM}}^{\text{max}}$, defined in Eq. 13, is chosen to reflect the vulnerability of the plaque. The maximum WSS, $\tau_{\text{max}}$, is mostly associated with the formation, growth and remodelling of the plaque [19-21,54].

Criteria $\gamma$ and $\varepsilon_R$ give an indication about the deformability of the plaque. The larger $\gamma$, the more deformable the plaque by shear. This mode of deformation is mostly induced by the drag force of the flow. The larger $\varepsilon_R$, the more deformable the plaque in compression. This mode of deformation is mostly induced by the pressure variations.

2.8 Convergence study

The four criteria presented in section 2.7 are used for assessing the convergence of the numerical resolution.

Temporal convergence is obtained using an adaptative time step, with a maximal value of 0.001s. It was checked that the four criteria remain unchanged by decreasing the maximum value of the time step.

The spatial convergence is obtained using over 2680 P$_2$P$_1$ elements for the fluid domain and over 3476 P$_2$ elements for the solid domain. It has been checked that increasing the degree of the shape functions to P$_3$ for the fluid velocity, P$_2$ for the fluid pressure and P$_3$ for the solid displacement has only a marginal influence on the analysed criteria (Tab. 2).
3. Results

3.1. Response of the stenosed artery

An example of results obtained from a FE analysis is shown in Fig. 3. Fig. 3-A shows the distribution of the von Mises equivalent stress in the plaque and in the healthy artery upstream and downstream the plaque, using a colour-coded representation plotted onto the deformed geometry at the systole. For visualizing the deformation between diastole and systole, the shape of the stenosed artery at diastole is represented in grey.

The percentage of diameter change between diastole and systole is about 5% in the healthy part of the artery (Fig. 3-A2), which corresponds to physiological conditions measured using MRI [46]. The percentage of diameter change is smaller in the stenosed region, due to the stiffening effect of the wall thickening (Fig. 3-A2).

The longitudinal component of the velocity \( v_z = \mathbf{v}_f \cdot \mathbf{e}_z \) is also represented at systole in Fig. 3-B using a colour-coded representation. Due to Venturi effect, the velocity increases from about 0.5 m/s upstream the stenosis to about 1.7 m/s downstream the stenosis. Recirculation occurs downstream the stenosis.

In the next sections, the results of the parametric study are presented. In Fig. 4 and 5, the influence of parameters \( e, k_{wall}^1 \) and \( k_{cap}^1 \), \( S, L \), onto \( \sigma_{\text{VM}}^{\text{max}}, \tau_{\text{max}}, e_R \) and \( \gamma \) is displayed.

3.2. Influence of the fibrous cap thickness.


The influence of the fibrous cap thickness $e$ onto the mechanical criteria is studied. For that, parameters $(S,L)$ are set to $(45\%,10\text{mm})$ and $k_1^{\text{wall}}$ and $k_1^{\text{cap}}$ are set to the values reported in Tab. 1. The increase of $\sigma_{\text{max}}^{\text{VM}}$ with respect to the decrease of the fibrous cap thickness $e$, shown in Fig. 4-A1, is in agreement with other studies stating that a thin fibrous cap is the parameter mostly associated with the plaque vulnerability [15,23]. The increase of $\sigma_{\text{max}}^{\text{VM}}$ is more important from $e=0.1\text{mm}$ to $e=0.05\text{mm}$. This result can be related to the result of Li et al. [15]. They showed that $e \leq 0.1\text{mm}$ could result in plaque rupture, even for a small stenosis severity.

The ratio between the volume of the lipid core and the fibrous cap thickness is sometimes used to characterise the plaque vulnerability: the greater this ratio, the more vulnerable the plaque [55] but Gao and Long [27] showed that the stress level in the fibrous cap is more sensitive to the fibrous cap thickness than to the lipid core volume.

Moreover as shown in Fig. 4, when $e$ decreases, the average compression strain $\varepsilon_R$ decreases, whereas the average shear strain $\gamma$ and the maximal WSS $\tau_{\text{max}}$ increases.

The stress criteria $\sigma_{\text{max}}^{\text{VM}}$ and $\tau_{\text{max}}$ and the deformation criteria $\varepsilon_R$ and $\gamma$ will be analysed in the discussion considering a thin and homogenous fibrous cap ($e=0.1\text{mm}$).

### 3.3 Influence of the stiffness of the constituents.

The influence of the material parameters $k_1^{\text{wall}}$ and $k_1^{\text{cap}}$ onto the mechanical criteria are studied considering that the other parameters are set to $(e,S,L)=(0.1\text{mm},45\%,10\text{mm})$. These
parameters are increased in the following range of values (in kPa): $24.53 \leq k_1^{wall} \leq 300$ and $23.7 \leq k_1^{cap} \leq 300$. 

The variations of $\sigma_{\max}^{VM}$ with respect to $k_1^{wall}$ and $k_1^{cap}$ (Fig. 4-A2,A3) shows that the plaque is more stable when each of these parameters increases. This result is in agreement with other studies reporting that a calcified plaque (stiffer) is more stable. Moreover Imoto et al [56] showed that a calcified inclusion in the fibrous cap can stabilize the plaque. However, Vengrenyuk et al. [57] showed that a fibrous cap with micro-calcification inclusions is related with high stress concentration and plaque fracture. This means that local and small inclusions may have the opposite effect of large calcifications.

The effect of $k_1^{wall}$ and $k_1^{cap}$ on the other parameters (compression strain $\varepsilon_R$ and average shear strain $\gamma$) is marginal within the range of tested values. Moreover, the mechanical properties of the healthy part of the artery affect only slightly the maximal WSS $\tau_{\max}$, whereas the mechanical properties of the fibre cap on the WSS is more pronounced. The decrease of $\tau_{\max}$ with regard to an increase of $k_1^{cap}$ is interesting; it shows that a compliant plaque is more prone to local erosion by wall shear stress. The behaviour of a compliant plaque, using the material parameters given in Tab. 1 [50], onto the stress and deformation criteria will be detailed in the discussion.

### 3.4 Influence of the stenosis severity

The effects of the stenosis severity is investigated through the following range of values: $25% < S < 70%$ with a constant plaque length $L=10\text{mm}$. The fibrous cap thickness is set to $e=0.1\text{mm}$ and the values of $k_1^{wall}$ and $k_1^{cap}$ are reported in Tab. 1. In current clinical practice,
when a vulnerable plaque is subjected to triggering events, the degree of severity of
endoluminal stenosis (Eq. 1) is evaluated and the plaque is diagnosed as vulnerable if this
criterion is beyond 70% [5,6]. With our model, the mechanical response is analyzed within a
range of stenosis severities which are below the vulnerability threshold. Fig. 5-A (left) shows
that $\sigma_{\text{max}}^{\text{VM}}$ is around 400kPa for $20% \leq S \leq 45\%$ ($\sigma_{\text{max}}^{\text{VM}} (20\%) = 404.53$ kPa,
$\sigma_{\text{max}}^{\text{VM}} (35\%) = 413.19$ kPa and $\sigma_{\text{max}}^{\text{VM}} (45\%) = 384.46$ kPa) and increases to around 500kPa for
$S=55\%$ ($\sigma_{\text{max}}^{\text{VM}} (55\%) = 540.31$kPa) and to around 600kPa for $S=70\%$ ($\sigma_{\text{max}}^{\text{VM}} (70\%) = 627.80$kPa)
which confirms that the degree of endoluminal stenosis affects the plaque vulnerability.

3.5 Influence of the stenosis length

Moreover, Fig. 5-A (right) shows that the plaque length is also strongly related to the plaque
vulnerability even though this criterion is not taken into account in clinical practice. For
instance, on one hand, the plaque with the parameters $(S,L)=(55\%,10\text{mm})$ has a similar $\sigma_{\text{max}}^{\text{VM}}$
value as the plaque with the parameters $(S,L)=(45\%,5\text{mm})$: $\sigma_{\text{max}}^{\text{VM}} (55\%,10\text{mm})=540.31$kPa and
$\sigma_{\text{max}}^{\text{VM}} (45\%,5\text{mm})=541.21$kPa. On the other hand, for two plaques having the same severity
$S=45\%$, the maximum stress $\sigma_{\text{max}}^{\text{VM}}$ is 50% larger in the short plaque $(L=5\text{mm})$ than in a larger
plaque $(L=10\text{mm})$, and the maximum WSS $\tau_{\text{max}}$ is increased of 100%.

In Fig. 5, it can be remarked that increasing the stenosis severity or decreasing the plaque
length has similar effects on the mechanical criteria. The influence of both the stenosis
severity and the plaque length on the fluid structure interaction and on the plaque
vulnerability will be discussed in the next section.
4. Discussion

4.1 Compression or shear effects

In Fig. 5-C and D, the deformability of the plaque is investigated through its average compression strain \( \varepsilon_R \) and its average shear strain \( \gamma \). Considering that the stenosis length is set to \( L = 10\text{mm} \), in Fig. 5-C and D (left), it can be noted that the mode of deformation changes drastically between \( S = 35\% \) and \( S = 45\% \), inducing a transition. On one hand, below the transition, the compression strains overwhelm the shear strains. On the other hand, beyond the transition severity, the opposite effect occurs.

Increasing the stenosis severity or decreasing the plaque length has similar effects on the mechanical criteria. Considering that the stenosis severity is set to \( S = 45\% \), in Fig. 5-C and D (right) it appears a transition zone between \( L = 10\text{mm} \) and \( L = 15\text{mm} \). Beyond the transition length, the compression strains overwhelm the shear strains, whereas the opposite effect occurs for short stenoses.

This transition is illustrated in Fig. 6-A which shows the stress distribution in the fibrous cap \( (\sigma^\text{VM}) \) for different severities and different lengths. It is noticeable that for \((S,L)=(35\%,10\text{mm}) \) or for \((S,L)=(45\%,15\text{mm}) \) the stress distribution is controlled by the blood pressure. In this case, high stresses are localized on the top of the stenosis. On the other hand, for \((S,L)=(45\%,10\text{mm}) \) or for \((S,L)=(55\%,10\text{mm}) \) or for \((S,L)=(45\%,5\text{mm}) \), the stress distribution is controlled shear which induces a localization of high stresses upstream stenosis.
If shear overwhelms compression, the plaque length has a significant effect onto the plaque vulnerability. The shorter the plaque, the more vulnerable.

If compression overwhelms shear, the study shows that a solid model without FSI, just considering pressure loads, is sufficient [23-26]. However, a model with FSI is necessary when the shear effects become significant [9,10,18,27-33]. This remark is interesting for choosing appropriate patient-specific models.

4.2 Plaque pinching

If shear overwhelms compression, results shown in Fig. 6-A give an interesting explanation for the deformed shape of the fibrous cap. Indeed, it can be observed that the plaque is compressed from both sides of the stenosis, upstream and downstream, resulting in a pinching effect of the plaque. The pinching effect comes from the coupled action of two phenomena:

- the shear coming from two flows:
  - the global flow upstream stenosis
  - the flow recirculation downstream stenosis
- the depression coming from:
  - the Venturi effect on the top of the stenosis
  - the recirculation downstream stenosis

Each flow compresses the stenosis on both sides, upstream and downstream. This phenomenon is schematized in Fig. 7-A. Due to the Venturi effect and flow recirculation, the pressure decreases [58], inducing outward tractions onto the plaque. These phenomena may also induce buckling in the fibrous cap.
In Fig. 7-B, the response of the plaque has been plotted by considering only the action of the blood pressure, without the action of blood flow. The results show that the mechanical response is completely different, without pinching effect.

The pinching effect appears for moderately thin fibrous cap ($e=0.2\text{mm}$) and increases when the fibrous cap thickness decreases. It is illustrated by the increase of the shear strain $\gamma$ and the decrease of the compression strain $\varepsilon_R$ with respect to the decrease of the fibrous cap thickness $e$, respectively shown in Fig. 4-C1 and D1.

As shown in Fig. 6-A, the plaque pinching does not present an upstream-downstream symmetry. For instance, for $L=5\text{mm}$ and for $S=55\%$ (Fig. 6-A), there is no symmetry because the global flow, the flow recirculation and the recirculation depression do not produce the same magnitude of force acting upstream and downstream the stenosis.

This puts in evidence that the action of blood flow is essential because it induces stress concentrations. Tang et al. evoked already that a local stress concentration is more closely related to plaque fracture [9] but the pinching effect has never been referenced.

Localization of the WSS ($\tau$) is also shown in Fig. 6-B, with high concentrations when the plaque is pinched. As WSS may be related with the plaque ulceration [19-21], the results show that when the pinching effect is strong, this may also induce plaque ulceration in real cases.

4.3 Main limitations
The mechanical properties of the artery. The Holzapfel material model was chosen because it is an appropriate model taking into account the nonlinear and anisotropic behaviour of arteries. Other material models could be used as Mooney-Rivlin models [28,59] and Ogden hyper-elastic models [13,15]. It has been verified that the pinching effect and the importance of plaque length are preserved even if these other material models are used. They are also preserved if different properties are considered for both layers of the healthy artery (media and adventitia, see section 2.4). Actually, as shown in Fig. 4-A2, B2, C2 and D2, the material properties of the healthy artery wall have a marginal influence on the pinching effect.

An important question is also the convexity of strain energy function defined in Eq. 10. As recommended by Holzapfel et al. [64], if $I_4$ and/or $I_6$ are less than 1, their contribution is cancelled from the strain energy function. This guarantees the convexity of the strain energy function whatever the material parameters.

Another important question is the impact of the choice of the material parameters of the artery on the mechanical response. The results displayed in Fig. 4 tend to show that the impact of the material properties of the artery onto the response of the plaque is less important than the impact of geometrical parameters like the severity, the thickness and the length.

However, the material properties of the artery used in the parametric study are defined as averages of the properties of the media and of the adventitia. It has been verified that this simplification does not alter the form of the mechanical response of the artery. For this verification, the longitudinal and circumferential stress/stretch curves of the different models of artery used in this paper have been plotted in Fig. 8 and compared to the stress/stretch curves of the original models taken from Gasser et al. [50]. It can be observed that the variety of parameters tested in our parametric study encompass the stress/stretch curves of the media and adventitia models reported in [50]. Moreover, the different curves have similar shapes,
which show that varying the parameters of the model results mainly in variations of the compliance of the artery itself.

**The perivascular tissues.** The vessel receives perivascular constraint from the surrounding tissues [60]. Considering the stiffness of the surrounding tissue may increase the rigidity of the structure, similarly as playing with the stiffness of the artery wall itself. It has been observed that the material properties of the healthy artery wall have a marginal influence on the analysed mechanical criteria (Fig. 4-A2, B2, C2 and D2). Then the effect of the surrounding tissue may not be considered as prominent.

**The blood viscosity.** Stenoses have an influence on the blood flow but can also have an impact on its viscosity. The blood is not a Newtonian fluid. The blood, composed of 80% plasma, may be assumed as a Newtonian fluid in healthy arteries with diameter larger than 5 mm. But the presence of red cells influences the blood viscosity when the hematocrite (ratio between the volume of red cells and the volume of plasma) increases. It is the case when the arterial lumen decreases or when the red cells aggregates [41,61]. This aggregation occurs in an area with many red cells and where the shear stress is less than 1 Pa [62]. Such a zone can be localized in a recirculation like downstream the stenosis. The red cells are trapped in a zone where they can aggregate. This case should be taken into account if one would like to refine the local shear stress just downstream the stenosis.

**The boundary conditions.** The effect of pressure and flow variability is not analysed. Such analysis may be interesting with regard to the pinching effect and it will be achieved in the future. Moreover, the existence of an axial pretension in the artery (tethering effect) is also an
aspect that may be important, especially concerning the zero-stress state in real geometries [32,63]. Given that our geometries were ideal, this aspect was not considered.

The geometry. This study offers an analysis of the influence of plaque shape on fluid structure interactions especially concerning the plaque length. Nevertheless there is no axisymmetric plaque in real case and the dimensions of the plaque used in our study are not representative of a given physiological scenario. The dimensions of the plaque were defined for encompassing a wide range of possible scenarios regarding plaque lengths, plaque severities, fibre cap thickness and material properties of the tissues. Other models are under development: asymmetric plaques, axisymmetric plaques with shape irregularities. The radius of the artery used in this study is based on the dimensions of the human common carotid artery [43,65]. It would also be interesting to evaluate the effect of the artery radius for mimicking for instance the plaque behaviour in the internal carotid artery which is smaller.

The unloaded state of the artery

The arterial radius for the unloaded (no pressure) condition was set at 3.00 mm and the average arterial radius for the pressurized condition (Fig. 3-A2) was 3.625 mm at diastole and 3.825 mm at systole. This indicates an expansion of artery. This expansion was not calibrated upon physiological data. It was applied because the hyperelastic constitutive equations are a model of the mechanical behaviour with regard to the unloaded (no pressure) state of the artery. However, we did not study how the choice of the unloaded geometry affects the mechanical response of the plaque. Moreover, the effect of the axial prestretch is also an aspect that still has to be evaluated.
5. Conclusion

This study shows that geometric and mechanical properties of atheromatous plaques affect significantly its mechanical response to the action of pulsatile blood flow. Notably for a short, severe and compliant stenosis, the blood pinches the plaque. In this case the stress localization and plaque vulnerability is emphasized.

These results may offer some new perspectives for understanding the vulnerability of short plaques. Unfortunately there are only few experimental papers available on this subject in the literature. Future work will consist in achieving such experimental investigations for characterizing the vulnerability of short plaques from clinical data. Moreover, more sophisticated models are under development in order to evaluate the effects of shape irregularities and asymmetry.
6. Conflict of interest

None.

7. Acknowledgements

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References


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Tab. 1: Material and structural parameters of the Holzapfel model describing the atheromatous plaque components.

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Tab. 3: Fourier coefficients for the velocity and pressure data.

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Fig. 5: Influence of the stenosis severity $S$ and the stenosis length $L$ onto: (A) the maximum von Mises equivalent stress in the fibrous cap $\sigma_{\text{VM}}^{\text{max}}$. (B) the wall shear stress (WSS) on the plaque $\tau_{\text{max}}$. (C) the average radial strain of the plaque $\varepsilon_R$. (D) the average shear strain of the plaque $\gamma$. On the left hand side, the stenosis length is set to $L=10\text{mm}$ and on the right hand side, the stenosis severity is set to $S=45\%$.

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Fig. 8: Circumferential (a) and longitudinal (b) stress/stretch curves of the healthy artery model for different material properties tested in the parametric studies and comparison with the stress/stretch curves of the media and adventitia reported in [50].
Tab. 1

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Tab. 2

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Fig. 1
Fig. 2

(a) Axial velocity (m/s) vs. time (s)

(b) Pressure (mmHg) vs. time (s)

Experimental data compared to Fourier polynomial approximation.
Fig. 3

(A)  (A1)  

(B)  

Von Mises stress $\sigma^{VM}$
Max: $3.844 \times 10^5$ (Pa)
Min: 509.584

Axial velocity $V_z$
Max: 1.791 (m/s)
Min: -0.117

% diameter change

- healthy part of the artery
- top of stenosis

44mm
Fig. 4

(A1) Influence on $\sigma_{\text{max}}^{\text{IV}}$

(B1) Influence on $\tau_{\text{max}}$

(C1) Influence on $\varepsilon_R$

(D1) Influence on $\gamma$

(A2) Influence on $\sigma_{\text{max}}^{\text{V}}$

(B2) Influence on $\tau_{\text{max}}$

(C2) Influence on $\varepsilon_R$

(D2) Influence on $\gamma$

(A3) Influence on $\sigma_{\text{max}}^{\text{VI}}$

(B3) Influence on $\tau_{\text{max}}$

(C3) Influence on $\varepsilon_R$

(D3) Influence on $\gamma$
Fig. 5

(A) S influence on $\sigma_{\max}^{\text{PM}}$ (kPa)

(B) S influence on $\tau_{\max}$ (Pa)

(C) S influence on $\varepsilon_R$

(D) S influence on $\gamma$

- the compression overwhelms the shear
- the shear overwhelms the compression
- transition zone
Fig. 6
Fig. 7

(A) With flow

(A1) Blood flow

(A2) Pressure

Min: 1.393e4
Max: 1.541e4 (Pa)

(A3) Stress

Min: 2.092e5
Max: 3.644e5 (Pa)

(B) Without flow

(B1) Blood pressure

(B2) Pressure

Min: 1.435e4 (Pa)
Max: 1.541e4 (Pa)

(B3) Stress

Min: 1.916e5
Max: 4.572e5 (Pa)