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

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27 **Abstract**

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

45

46 **Keywords:** vascular biomechanics; fluid structure interaction; numerical simulation; stenosis;  
47 blood flow

48

49

50

51 **Global variables:**  
52  
53  $\rho$  the density  
54  $\eta$  the viscosity  
55  $R_0$  the healthy arterial radius  
56  $h_0$  the thickness of the healthy arterial wall  
57  $e$  the fibrous cap thickness  
58  $L$  the stenosis length  
59  $R_m$  the external stenosis remodelling radius  
60  $S$  the stenosis severity in terms of diameter  
61  $S_{area}$  the stenosis severity in terms of area  
62  $(\alpha_k)_k$  the series of Womersley number  
63  $\mathbf{e}_z$  the longitudinal axis in the cylindrical frame  
64  $\mathbf{e}_r$  the radial axis in the cylindrical frame  
65  $\mathbf{n}$  exterior normal  
66  $\mathbf{u}$  the displacement vector  
67  $\mathbf{v}$  the velocity  
68  $p$  the pressure  
69  $\boldsymbol{\sigma}$  the stress tensor  
70  $\mathbf{F}$  the deformation gradient tensor  
71  $\mathbf{C}$  the Green-Cauchy right strain tensor  
72  $\varphi$  the strain energy density  
73  $J$  the volume change invariant  
74  $I_1, I_4$  and  $I_6$  the first, fourth and sixth coordinate invariants  
75  $c, k_1$  and  $k_2$  the parameters of the Holzapfel model  
76  $\beta$  the angle between these two fibres family  
77  $\mathbf{M}$  and  $\mathbf{M}'$  the direction's vectors of the two fiber's family in the cylindrical coordinate  
78  $\mathbf{w}$  the mesh velocity in the Arbitrary Lagrangian Eulerian scheme  
79  $\tau$  the wall shear stress  
80  $\varepsilon_R$  the average radial strain of the plaque  
81  $\gamma$  the average shear strain of the plaque  
82  $U_r$  the radial displacement component of the fibrous cap at the middle of the stenosis  
83  $U_z$  the longitudinal displacement of the fibrous cap at the middle of the stenosis  
84  $P_1$  the linear type of finite elements  
85  $P_2$  the quadratic type of finite elements  
86  $\Omega$  a domain  
87  $\partial\Omega$  a boundary  
88  
89 **Indexes:**  
90  
91  $s$  the solid part  
92  $f$  the fluid part  
93  $fs$  fluid-structure  
94  $i$  the material number ( $1 \leq i \leq 4$ )  
95  $in$  inlet

96 *out* outlet  
97 *ext* exterior  
98 *sym* symmetric  
99 *max* maximum  
100  
101 **Operators:**  
102  
103  $\det(\cdot)$  the determinant of a tensor  
104  $\frac{\partial \cdot}{\partial t}$  the first derivative with respect to time  
105  $\frac{\partial^2 \cdot}{\partial t^2}$  the second derivative with respect to time  
106  $\nabla \cdot$  the divergence operator  
107  $\nabla$  the gradient operator  
108 **I** the identity matrix  
109  ${}^t \mathbf{A}$  transpose of the matrix **A**  
110  $Re(\cdot)$  the real part of an imaginary number  
111  $J_0(\cdot)$  the zero order Bessel function of first kind  
112  
113

## 114 **1. Introduction**

115

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

120

121 In current clinical practice, carotid endarterectomy is the most frequently used treatment for  
122 pathological plaques. The decision leading to surgery is based on the degree of endoluminal  
123 stenosis [5,6]. But this criterion alone is insufficient to predict the plaque fracture and the  
124 necessity to identify other criteria is a major issue for public health [1]. Therefore, there is a  
125 strong medical and economical interest in developing new tools for a better understanding of  
126 this situation.

127

128 Histological studies have related plaque vulnerability with thin fibrous cap, large necrotic core  
129 [2] and inflammation after macrophages or T-cells infiltration in the lipid core [3] or foam  
130 cells infiltration in the fibrous cap [4]. Advanced magnetic resonance imaging (MRI) allows  
131 *in vivo* virtual histology of plaques [7,8].

132

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

141

142 2-D and 3-D patient-specific finite-element (FE) models of diseased vessels are probably the  
143 best way to obtain stress distributions for specific plaques. The models are based on histology  
144 or pre-fracture medical imaging [18]. Their purpose is to associate the mechanical descriptors  
145 to the mechanical process of plaque rupture.

146

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

- 149 • fluid models [19-21] with rigid plaques
- 150 • 2D solid models under pressure loads [14,22-26] considering cross sections of  
151 idealized plaques

152 • or fluid structure interaction (FSI) models taking into account both the combination of  
153 shear and pressure loads [9,10,18,27-33].

154  
155 FSI models are probably the most realistic models but their models are complex. The  
156 interaction between the blood flow and the plaque is not yet fully understood, especially due  
157 to the large variability of plaques [8]. Using idealized models allows investigating how the  
158 mechanical action of the blood onto the plaque is affected by the geometry and the  
159 mechanical properties. For instance, Li Z.Y. et al. [15] studied the influence of stenosis  
160 severity and fibrous cap thickness on stresses in plaques with 2D plane-strain models. Li M.X.  
161 et al. [16] investigated the stress distribution for different degrees of stenoses also with 2D-  
162 axisymmetric models. Valencia et al. [34] investigated the influence of the severity of  
163 stenoses on stresses with 3D-axisymmetric models. Tang et al. performed many 3D FSI  
164 studies on idealized plaque models. They analysed wall stress and strain in symmetric and  
165 asymmetric plaque models with two different stenosis severities: 50% and 78% [17,35]. They  
166 analysed also the influence of pressure loads and stenosis severity on the cyclic compression  
167 of plaques [12,36].

168  
169 The effect of plaque length has never been investigated in the literature. The present paper  
170 aims at addressing this lack. A FSI 2D axisymmetric model of the blood flow in a smooth  
171 pipe is considered, with a thick, deformable, heterogeneous and axisymmetric stenosis,  
172 mimicking an atherosclerotic plaque in a straight segment of artery. The model allows rapid  
173 modifications of the geometrical and constitutive parameters of the plaque for evaluating the  
174 influence of all these parameters.

175

176

177 **2. Methods**

178

179 **2.1 Geometrical model**

180

181 A schematic of the geometry in the reference configuration (i.e. in unloaded pressure-free  
182 conditions [32,37]) is provided in Fig. 1. The model of the plaque is axisymmetric (Fig. 1-D).

183 The initial stenosis shape starts from a sinus shape function along the longitudinal direction. It  
184 is composed of the healthy arterial wall outside and of a fibrous cap containing the lipid core  
185 inside (Fig. 1-B).

186

187 The healthy arterial lumen has an inner radius of  $R_0 = 3$  mm and a wall thickness of  
188  $h_0 = 0.5$  mm (Fig. 1-C), which corresponds to average values for the wall of the carotid artery  
189 [38].

190

191 Most of vulnerable carotid plaques present a positive remodelling [39] which is modelled as  
192 an external remodelling radius of the vessel. It is set here to  $R_m = 1$  mm [39].

193

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



201 
$$S = 1 - \frac{R_0 - H_0 - e}{R_0} \quad (1)$$

202 
$$S_{area} = 1 - (1 - S)^2 \quad (2)$$

203

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

209

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

214

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

219

## 220 **2.2 Fluid and structural equations**

221

222 A pulsatile flow of a viscous Newtonian and incompressible fluid is considered in an  
223 axisymmetric pipe (Fig. 1-A), with a pulsation  $\omega = 2\pi$  [41]. This flow behaviour is suitable  
224 for simulating the flow in large arteries because Non-newtonian effects are believed to have a

225 minor influence (see discussion in section 4.3). Its dynamic viscosity is denoted  $\eta_f$  and the  
 226 density of the fluid is denoted  $\rho_f$ . The fluid velocity field  $\mathbf{v}_f$  and pressure field  $p$  are  
 227 governed by the unsteady incompressible Navier-Stokes equations written in the Arbitrary  
 228 Lagrangian-Eulerian (ALE) formulation [42]:

$$229 \quad \begin{cases} \rho_f \frac{\partial \mathbf{v}_f}{\partial t} + \rho_f (\mathbf{v}_f - \mathbf{w}) \cdot \nabla \mathbf{v}_f - \nabla \cdot \boldsymbol{\sigma}_f = \mathbf{0} \\ \nabla \cdot \mathbf{v}_f = 0 \end{cases} \quad \text{in } \Omega_f, \quad (3)$$

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

232

233 The displacement vector, denoted  $\mathbf{u}_s$ , and the Cauchy stress tensor, denoted  $\boldsymbol{\sigma}_s$ , of the solid  
 234 part, with respect to the reference configuration, satisfy the following equation [43]:

$$235 \quad \rho_s \frac{\partial^2 \mathbf{u}_s}{\partial t^2} - \nabla \cdot \boldsymbol{\sigma}_s = \mathbf{0} \quad \text{in } \Omega_s, \quad (4)$$

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

238

### 239 **2.3 Fluid and structural boundary conditions**

240

241 At the inlet of the fluid domain, a Womersley velocity profile [44] is applied and at the outlet,  
 242 a pressure is imposed implying no normal viscous stress (see Eq. 5). The time variations of  
 243 the pressure and the velocity profiles are written using Fourier decomposition, with 18 and 6  
 244 terms respectively for ensuring agreement with experimental data. Fourier decomposition is  
 245 applied to the temporal signals shown in Fig. 2.

$$\left\{ \begin{array}{l}
\mathbf{v}_f(r,t) = -\frac{c_0}{4\eta_f} R^2 \left(1 - \left(\frac{r}{R}\right)^2\right) - \frac{R^2}{\eta_f} \sum_{k=0}^6 \operatorname{Re} \left( \frac{ic_k}{\alpha_k^2} \left(1 - \frac{J_0(\alpha_k \frac{r}{R} i^{\frac{3}{2}})}{J_0(\alpha_k i^{\frac{3}{2}})}\right) e^{ik\omega t} \right) \mathbf{e}_z \quad \text{on } \partial^{in}\Omega_f \\
p(t) = \sum_{k=0}^{18} \operatorname{Re}(p_k e^{ik\omega t}) \quad \text{on } \partial^{out}\Omega_f \\
\boldsymbol{\sigma}_f^{viscous} \mathbf{e}_z = \mathbf{0} \quad \text{on } \partial^{out}\Omega_f
\end{array} \right. , \quad (5)$$

247 where  $\partial^{in}\Omega_f$  and  $\partial^{out}\Omega_f$  are respectively the inlet and the outlet boundaries of the fluid

248 depicted in Fig. 1-A,  $\mathbf{e}_z$  is the longitudinal vector and  $J_0$  is the zero order Bessel function of

249 first kind,  $R$  is the radius of the pipe at the inlet and  $(\alpha_k = R \sqrt{\frac{k\omega\rho_f}{\eta_f}})_{0 \leq k \leq 6}$  is the series of

250 Womersley numbers.

251

252  $\boldsymbol{\sigma}_f^{viscous}$  represents the viscous stress part of the Cauchy stress tensor in the fluid  $\sigma_f$  (see Eq. 8

253 in the section 2.4).

254

255 The axial velocity at the inlet is represented in Fig. 2-A and the pressure at the outlet is

256 represented in Fig. 2-B [46].

257

258 The Fourier coefficients  $c_k$  (Tab. 3) of the Womersley profile are computed from the axial

259 velocity data measured non-invasively using PC-MRI in the common carotid artery of a

260 volunteer [46]. Moreover the pressure Fourier coefficients  $p_k$  (Tab. 3) are deduced from the

261 variations over cardiac cycles of the pressure measured non-invasively using the applanation

262 tonometry technique on the same volunteer [47].

263

264 At the boundaries of the elastic solid, the displacement vector  $\mathbf{u}_s$  and the Cauchy stress tensor  
 265  $\boldsymbol{\sigma}_s$ , with respect to the reference configuration, satisfy the following equations [43]:

266

$$267 \quad \begin{cases} \boldsymbol{\sigma}_s \mathbf{n}_{s,ext} = \mathbf{0} & \text{on } \partial^{ext} \Omega_s \\ \mathbf{u}_s \cdot \mathbf{e}_z = 0 & \text{on } \partial^{in} \Omega_s \text{ and } \partial^{out} \Omega_s \end{cases}, \quad (6)$$

268 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.

269

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

$$272 \quad \begin{cases} \mathbf{v}_f = \frac{\partial \mathbf{u}_s}{\partial t} = \mathbf{w} \\ \boldsymbol{\sigma}_s \mathbf{n}_s + \boldsymbol{\sigma}_f \mathbf{n}_f = \mathbf{0} \end{cases} \text{ on } \partial \Omega_{fs}, \quad (7)$$

273 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,  
 274 mesh velocity  $\mathbf{w}$  is derived following a Laplace smoothing method from the interface  
 275 conditions.

276

## 277 **2.4 Fluid and structural properties**

278

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

$$281 \quad \boldsymbol{\sigma}_f = -p\mathbf{I} + \eta_f (\nabla \mathbf{v}_f + {}^t \nabla \mathbf{v}_f), \quad (8)$$

282 with  $\eta_f = 0.005 \text{ Pa}\cdot\text{s}$  [42]. The first and second terms are respectively the hydrostatic  
 283 component  $\boldsymbol{\sigma}_f^{\text{hydrostatic}}$  and the viscous stress component  $\boldsymbol{\sigma}_f^{\text{viscous}}$ . The density of the fluid is set  
 284 to  $\rho_f = 1050 \text{ kg/m}^3$  [41].

285  
 286 Holzapfel et al. established a hyperelastic anisotropic constitutive equation for artery  
 287 components [48]. This model is used here for each component of the plaque. Hyperelasticity  
 288 implies the existence of a strain energy density function  $\varphi$  depending upon the Green-Cauchy  
 289 right strain tensor  $\mathbf{C} = {}^t\mathbf{F}\mathbf{F}$ , where  $\mathbf{F}$  is the deformation gradient tensor [43]. Thus  $\varphi = \varphi(\mathbf{C})$   
 290 and the associated Cauchy stress tensor is given by:

$$291 \quad \boldsymbol{\sigma}_s = 2J^{-1}\mathbf{F}\frac{\partial\varphi}{\partial\mathbf{C}}\mathbf{F}, \quad (9)$$

292 where  $J = \det(\mathbf{F})$  is the volume change invariant. This formulation is valid for a solid without  
 293 residual stresses. It was proposed in [48] to separate the isotropic and the anisotropic part of  
 294  $\varphi$ . The anisotropy is defined by two preferred directions corresponding to two families of

295 collagen fibres. The angle between these two families of fibres is denoted  $\beta$ .  $\mathbf{M} = \begin{pmatrix} 0 \\ \cos(\beta) \\ \sin(\beta) \end{pmatrix}$

296 and  $\mathbf{M}' = \begin{pmatrix} 0 \\ \cos(\beta) \\ -\sin(\beta) \end{pmatrix}$  represent the directions of fibres in the local coordinate system.

297

298 Then, the strain energy density  $\varphi$  is written such as:

$$299 \quad \varphi(I_1, I_4, I_6, J) = \frac{c}{2}(I_1 - 3) + \frac{k_1}{2k_2} \sum_{i=4,6} (e^{k_2(I_i-1)^2} - 1) + \frac{\kappa}{2}(J - 1)^2, \quad (10)$$

300 where  $I_1 = \text{tr}(\mathbf{C})$ ,  $I_4 = \mathbf{M} \cdot (\mathbf{C}\mathbf{M})$  and  $I_6 = \mathbf{M}' \cdot (\mathbf{C}\mathbf{M}')$ ,  $c$ ,  $\kappa$ ,  $k_1$  and  $k_2$  are material  
 301 parameters. Arterial tissue is often assumed as nearly incompressible [49]. The modulus of

302 compressibility  $\kappa$  is set to 500,000 kPa here in order to ensure this hypothesis. The density is  
 303 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  
 304 were taken from experimental data [50].

305  
 306 To simplify the model, average properties of the media and of the adventitia reported in [50]  
 307 are taken. For deducing the properties of the healthy artery reported in Tab. 1, a weighted  
 308 average is derived, with a weight of  $\frac{2}{3}$  for the media and a weight of  $\frac{1}{3}$  for the adventitia  
 309 [38]. The equation used to derive parameter  $c$  is given Eq. 11. A similar equation is used for  
 310 the other mechanical parameters ( $k_1$ ,  $k_2$  and  $\beta$ ).

$$311 \quad c^{wall} = \frac{2}{3}c^{media} + \frac{1}{3}c^{adventitia} \quad (11)$$

312

## 313 **2.5 Numerical computation**

314

315 FSI simulations are performed using commercial FE solver COMSOL Multiphysics [51]. The  
 316 compatible finite-element types are  $P_2$  for the fluid velocity  $\mathbf{v}_f$ ,  $P_1$  for the fluid pressure  $p$   
 317 and  $P_2$  for the solid displacement  $\mathbf{u}_s$  [52]. The model geometry is meshed using triangular  
 318 mesh generation in COMSOL Multiphysics, consisting in  $N_f$  elements for the fluid domain  
 319 ( $2680 \leq N_f \leq 3316$ ) and  $N_s$  for the solid domain ( $3476 \leq N_s \leq 4325$ ), with  $N_{fibrous\ cap}$

320 elements in the thickness of the fibrous cap ( $1025 \leq N_{fibrous\ cap} \leq 1650$ ), depending on the

321 different model geometries considered in the parametric study. The coupled fluid-structure  
 322 problem is discretized using a Galerkin-Least-Square method (GLS) and an implicit temporal  
 323 discretization of order 5 using a Backward Differentiation Formula (BDF) with an adaptative  
 324 time step. The non-linear problems are solved using a Newton-Raphson algorithm.

325

326 The mesh is refined close to the wall in order to take into account the viscous boundary layer:  
327 the mesh size is prescribed 3 times finer near the wall than at the centre.

328

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

332 
$$Re = \frac{\rho VD}{\mu}, \quad (12)$$

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

336

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

- 343
- the axial velocity at the inlet is increased linearly from 0m/s to 0.3m/s (see  $v_z(t=0)$   
344 Fig. 2-A)
  - the pressure at the outlet is increased linearly from 0mmHg to 91mmHg (see  $p(t=0)$   
345 Fig. 2-B)
  - the fluid viscosity is decreased linearly from 0.05Pa.s to 0.005Pa.s  
347

348 Afterwards, three cycles of the actual pulsatile flow (see Fig. 2) are computed. The flow is  
349 fully established and periodic over the last cycle. Hence, the last cycle is used for the analysis.

350

## 351 **2.6 Parametric study**

352

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

- 355 • the fibrous cap thickness denoted  $e$ ,
- 356 • material parameter  $k_1$  in the arterial wall and in the fibrous cap, respectively denoted  
357  $k_1^{wall}$  and  $k_1^{cap}$ ,
- 358 • stenosis severity  $S$  defined in Eq. 1
- 359 • stenosis length  $L$ .

360

361 Each parameter is set independently of the other parameters (unidirectional parametric study).

362 The geometrical properties of the reference model are:  $e = 0.1$  mm,  $S = 45\%$ ,  $L = 10$  mm. The  
363 reference values for  $k_1$  are reported in Tab. 1. Simulations and analysis are performed for a  
364 wide range of these parameters using an interface between the COMSOL software and the  
365 MATLAB software [51,53].

366

367 The current study considers moderately severe plaques, between 20% and 70%. The current  
368 study is more focused on the effect of the stenosis length, as the effect of this parameter is not  
369 clearly understood and there exists a large range of plaque lengths. Plaques in the carotid  
370 artery may be short ( $L < 10$  mm) [18,40]. Therefore, plaque lengths ranging between 5 mm and  
371 20 mm are considered in this study (20 mm corresponds to a very long plaque [40]).

372



373 The fibrous cap thickness and the material parameters are taken into account for investigating  
374 the effect of the plaque stiffness.

375

## 376 **2.7 Analysed criteria**

377

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

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

$$381 \quad \sigma_{\max}^{VM} = \text{Max}(\sigma^{VM}) = \text{Max}(\sqrt{\sigma_1^2 + \sigma_2^2 - \sigma_1\sigma_2}), \quad (13)$$

382 where  $\sigma_1$  and  $\sigma_2$  are the principal stresses in the radial and longitudinal directions

- 383 • the maximum wall shear stress (WSS),

$$384 \quad \tau_{\max} = \text{Max}(\|\boldsymbol{\sigma}_f^{\text{viscous}} \mathbf{n}_f\|) \quad (14)$$

- 385 • the average radial strain of the plaque,

$$386 \quad \varepsilon_R = \frac{U_R}{H_0 + h_0 + e + R_m}, \quad (14)$$

387 where  $U_R$  is the radial displacement of the fibrous cap at the middle of the stenosis,

- 388 • the global shear strain of the plaque,

$$389 \quad \gamma = \frac{U_z}{H_0 + h_0 + e + R_m}, \quad (16)$$

390 where  $U_z$  is the longitudinal displacement of the fibrous cap at the middle of the  
391 stenosis.

392

393 The maximum von Mises equivalent stress,  $\sigma_{\max}^{VM}$ , defined in Eq. 13, is chosen to reflect the  
394 vulnerability of the plaque. The maximum WSS,  $\tau_{\max}$ , is mostly associated with the  
395 formation, growth and remodelling of the plaque [19-21,54].

396

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

401

## 402 **2.8 Convergence study**

403

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

406

407 Temporal convergence is obtained using an adaptative time step, with a maximal value of  
408 0.001s. It was checked that the four criteria remain unchanged by decreasing the maximum  
409 value of the time step.

410

411 The spatial convergence is obtained using over 2680  $P_2P_1$  elements for the fluid domain and  
412 over 3476  $P_2$  elements for the solid domain. It has been checked that increasing the degree of  
413 the shape functions to  $P_3$  for the fluid velocity,  $P_2$  for the fluid pressure and  $P_3$  for the solid  
414 displacement has only a marginal influence on the analysed criteria (Tab. 2).

415

416

## 417 **3. Results**

418

### 419 **3.1. Response of the stenosed artery**

420

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

426

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

431

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

436 In the next sections, the results of the parametric study are presented. In Fig. 4 and 5, the  
437 influence of parameters  $e$ ,  $k_1^{wall}$  and  $k_1^{cap}$ ,  $S$ ,  $L$ , onto  $\sigma_{max}^{VM}$ ,  $\tau_{max}$ ,  $\varepsilon_R$  and  $\gamma$  is displayed.

438

### 439 **3.2. Influence of the fibrous cap thickness.**

440

441 The influence of the fibrous cap thickness  $e$  onto the mechanical criteria is studied. For that,  
442 parameters  $(S,L)$  are set to  $(45\%,10\text{mm})$  and  $k_1^{wall}$  and  $k_1^{cap}$  are set to the values reported in  
443 Tab. 1. The increase of  $\sigma_{\max}^{VM}$  with respect to the decrease of the fibrous cap thickness  $e$ ,  
444 shown in Fig. 4-A1, is in agreement with other studies stating that a thin fibrous cap is the  
445 parameter mostly associated with the plaque vulnerability [15,23]. The increase of  $\sigma_{\max}^{VM}$  is  
446 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  
447 al. [15]. They showed that  $e \leq 0.1\text{mm}$  could result in plaque rupture, even for a small stenosis  
448 severity.

449

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

454

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

457

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

460

### 461 **3.3 Influence of the stiffness of the constituents.**

462

463 The influence of the material parameters  $k_1^{wall}$  and  $k_1^{cap}$  onto the mechanical criteria are  
464 studied considering that the other parameters are set to  $(e,S,L)=(0.1\text{mm},45\%,10\text{mm})$ . These

465 parameters are increased in the following range of values (in kPa):  $24.53 \leq k_1^{wall} \leq 300$  and  
466  $23.7 \leq k_1^{cap} \leq 300$ .

467

468 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  
469 more stable when each of these parameters increases. This result is in agreement with other  
470 studies reporting that a calcified plaque (stiffer) is more stable. Moreover Imoto et al [56]  
471 showed that a calcified inclusion in the fibrous cap can stabilize the plaque. However,  
472 Vengrenyuk et al. [57] showed that a fibrous cap with micro-calcification inclusions is related  
473 with high stress concentration and plaque fracture. This means that local and small inclusions  
474 may have the opposite effect of large calcifications.

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

483

### 484 **3.4 Influence of the stenosis severity**

485

486 The effects of the stenosis severity is investigated through the following range of values:  
487  $25\% < S < 70\%$  with a constant plaque length  $L=10\text{mm}$ . The fibrous cap thickness is set to  
488  $e=0.1\text{mm}$  and the values of  $k_1^{wall}$  and  $k_1^{cap}$  are reported in Tab. 1. In current clinical practice,

489 when a vulnerable plaque is subjected to triggering events, the degree of severity of  
 490 endoluminal stenosis (Eq. 1) is evaluated and the plaque is diagnosed as vulnerable if this  
 491 criterion is beyond 70% [5,6]. With our model, the mechanical response is analyzed within a  
 492 range of stenosis severities which are below the vulnerability threshold. Fig. 5-A (left) shows  
 493 that  $\sigma_{\max}^{VM}$  is around 400kPa for  $20\% \leq S \leq 45\%$  ( $\sigma_{\max}^{VM}(20\%) = 404.53$  kPa,  
 494  $\sigma_{\max}^{VM}(35\%) = 413.19$  kPa and  $\sigma_{\max}^{VM}(45\%) = 384.46$  kPa) and increases to around 500kPa for  
 495  $S=55\%$  ( $\sigma_{\max}^{VM}(55\%) = 540.31$ kPa) and to around 600kPa for  $S=70\%$  ( $\sigma_{\max}^{VM}(70\%) = 627.80$ kPa)  
 496 which confirms that the degree of endoluminal stenosis affects the plaque vulnerability.

497

### 498 **3.5 Influence of the stenosis length**

499

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

507

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

512

513 **4. Discussion**

514

515 **4.1 Compression or shear effects**

516

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

523

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

529

530 This transition is illustrated in Fig. 6-A which shows the stress distribution in the fibrous cap  
531 ( $\sigma^{VM}$ ) for different severities and different lengths. It is noticeable that for  
532  $(S, L) = (35\%, 10\text{mm})$  or for  $(S, L) = (45\%, 15\text{mm})$  the stress distribution is controlled by the  
533 blood pressure. In this case, high stresses are localized on the top of the stenosis. On the other  
534 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  
535 distribution is controlled shear which induces a localization of high stresses upstream stenosis.

536

537 If shear overwhelms compression, the plaque length has a significant effect onto the plaque  
538 vulnerability. The shorter the plaque, the more vulnerable.

539

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

544

#### 545 **4.2 Plaque pinching**

546

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

- 551 • the shear coming from two flows:
  - 552 • the global flow upstream stenosis
  - 553 • the flow recirculation downstream stenosis
- 554 • the depression coming from:
  - 555 • the Venturi effect on the top of the stenosis
  - 556 • the recirculation downstream stenosis

557

558 Each flow compresses the stenosis on both sides, upstream and downstream. This  
559 phenomenon is schematized in Fig. 7-A. Due to the Venturi effect and flow recirculation, the  
560 pressure decreases [58], inducing outward tractions onto the plaque. These phenomena may  
561 also induce buckling in the fibrous cap.



562

563 In Fig. 7-B, the response of the plaque has been plotted by considering only the action of the  
564 blood pressure, without the action of blood flow. The results show that the mechanical  
565 response is completely different, without pinching effect.

566

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

571

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

576

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

580

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

585

586 **4.3 Main limitations**

587

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

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

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

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

612 which show that varying the parameters of the model results mainly in variations of the  
613 compliance of the artery itself.

614

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

621

622 **The blood viscosity.** Stenoses have an influence on the blood flow but can also have an  
623 impact on its viscosity. The blood is not a Newtonian fluid. The blood, composed of 80%  
624 plasma, may be assumed as a Newtonian fluid in healthy arteries with diameter larger than  
625 5 mm. But the presence of red cells influences the blood viscosity when the hematocrite (ratio  
626 between the volume of red cells and the volume of plasma) increases. It is the case when the  
627 arterial lumen decreases or when the red cells aggregates [41,61]. This aggregation occurs in  
628 an area with many red cells and where the shear stress is less than 1 Pa [62]. Such a zone can  
629 be localized in a recirculation like downstream the stenosis. The red cells are trapped in a  
630 zone where they can aggregate. This case should be taken into account if one would like to  
631 refine the local shear stress just downstream the stenosis.

632

633 **The boundary conditions.** The effect of pressure and flow variability is not analysed. Such  
634 analysis may be interesting with regard to the pinching effect and it will be achieved in the  
635 future. Moreover, the existence of an axial pretension in the artery (tethering effect) is also an

636 aspect that may be important, especially concerning the zero-stress state in real geometries  
637 [32,63]. Given that our geometries were ideal, this aspect was not considered.

638

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

649

#### 650 **The unloaded state of the artery**

651 The arterial radius for the unloaded (no pressure) condition was set at 3.00 mm and the  
652 average arterial radius for the pressurized condition (Fig. 3-A2) was 3.625 mm at diastole and  
653 3.825 mm at systole. This indicates an expansion of artery. This expansion was not calibrated  
654 upon physiological data. It was applied because the hyperelastic constitutive equations are a  
655 model of the mechanical behaviour with regard to the unloaded (no pressure) state of the  
656 artery. However, we did not study how the choice of the unloaded geometry affects the  
657 mechanical response of the plaque. Moreover, the effect of the axial prestretch is also an  
658 aspect that still has to be evaluated.

659

660

661

662 **5. Conclusion**

663

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

668

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

675

676

677 **6. Conflict of interest**

678

679 None.

680

681 **7. Acknowledgements**

682

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685

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872

873 **List of figures and table with caption:**

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904

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910

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917

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924

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926 model for different material properties tested in the parametric studies and comparison with  
927 the stress/stretch curves of the media and adventitia reported in [50].

928

929 Tab. 1

930

Components	$c$ (kPa)	$k_1$ (kPa)	$k_2$ (-)	$\beta$ ( $^\circ$ )
Fibrous cap	78.9	23.7	26.3	0
Healthy artery wall	10.58	24.53	22.13	21
Lipid pool	0.1	0.0	-	-

931

932

933 Tab. 2

934

Finite elements types	$\sigma_{\max}^{VM}$ (kPa)	$\tau_{\max}$ (Pa)	$\varepsilon_R$	$\gamma$
$P_2$ for $\mathbf{v}_f$ , $P_1$ for $p$ and $P_2$ for $\mathbf{u}_s$	384.46	43.254	0.141	0.119
$P_3$ for $\mathbf{v}_f$ , $P_2$ for $p$ and $P_3$ for $\mathbf{u}_s$	384.46	43.312	0.139	0.122

935

936

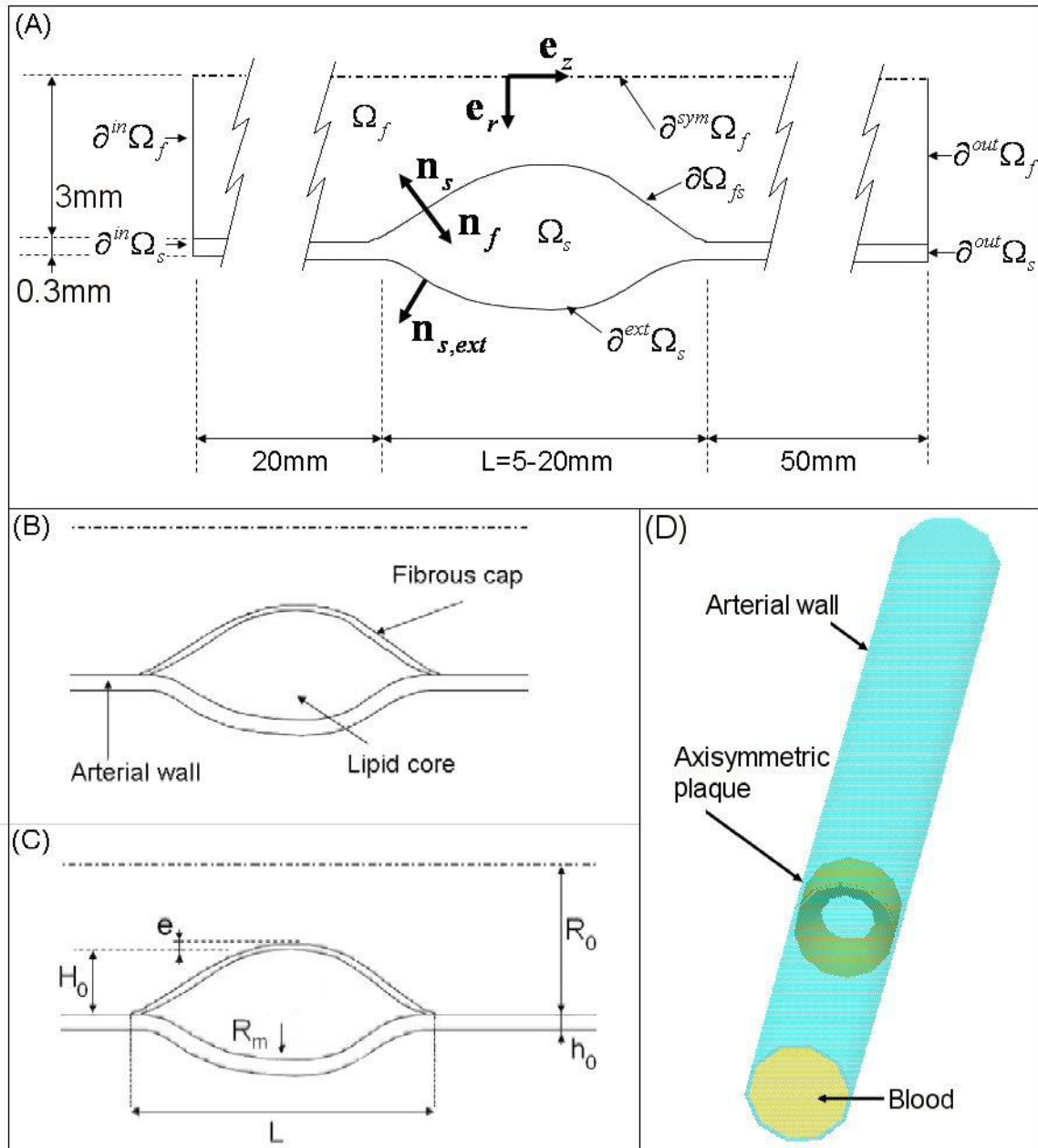
k	$v_k$ (cm/s)	$p_k$ (mmHg)
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1	-0,1229-11,0224i	-2.4929-12.407i
2	-2,3484+8,5141i	-4.5547-3.3544i
3	5,8539-2,0908i	-2.5546-0.32889i
4	-2,9313-1,1926i	-0.6181+0.45323i
5	1,3812+2,1390i	-0.19113-0.26335i
6	0,7306-0,9195i	-0.79729-0.68989i
7		-0.88357+0.1032i
8		-0.25735+0.22093i
9		-0.37814-0.015991i
10		-0.36849+0.32603i
11		0.015548+0.22243i
12		-0.070833+0.058148i
13		-0.068578+0.13458i
14		0.040223+0.12718i
15		0.030337+0.028246i
16		-0.0060027+0.02907i
17		0.0054199+0.046419i
18		0.0053679+0.0085823i

938

939

940 Fig. 1

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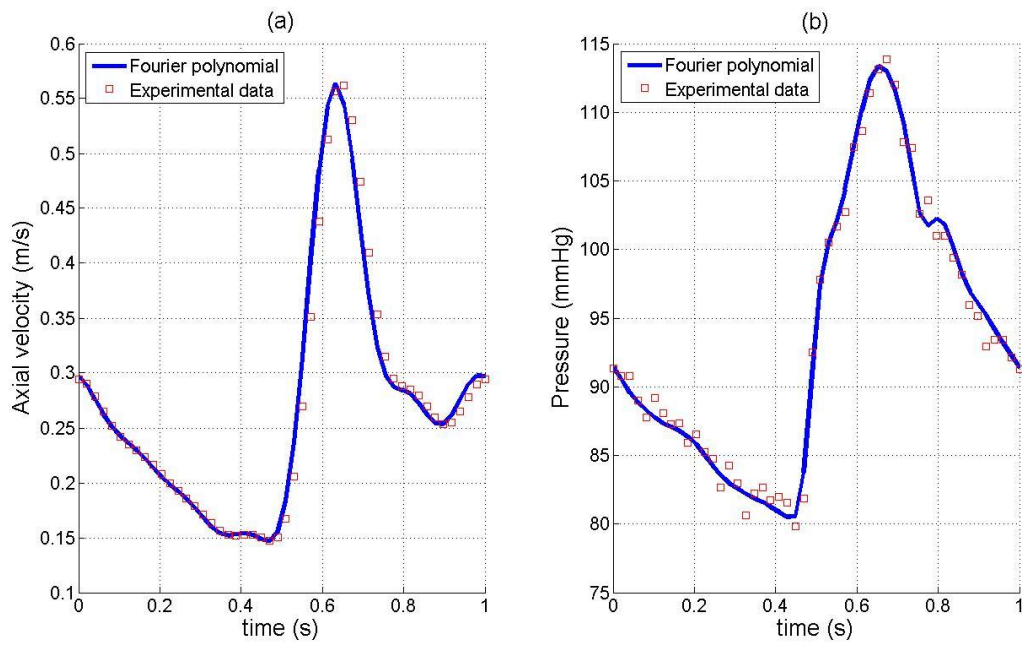


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944 Fig. 2

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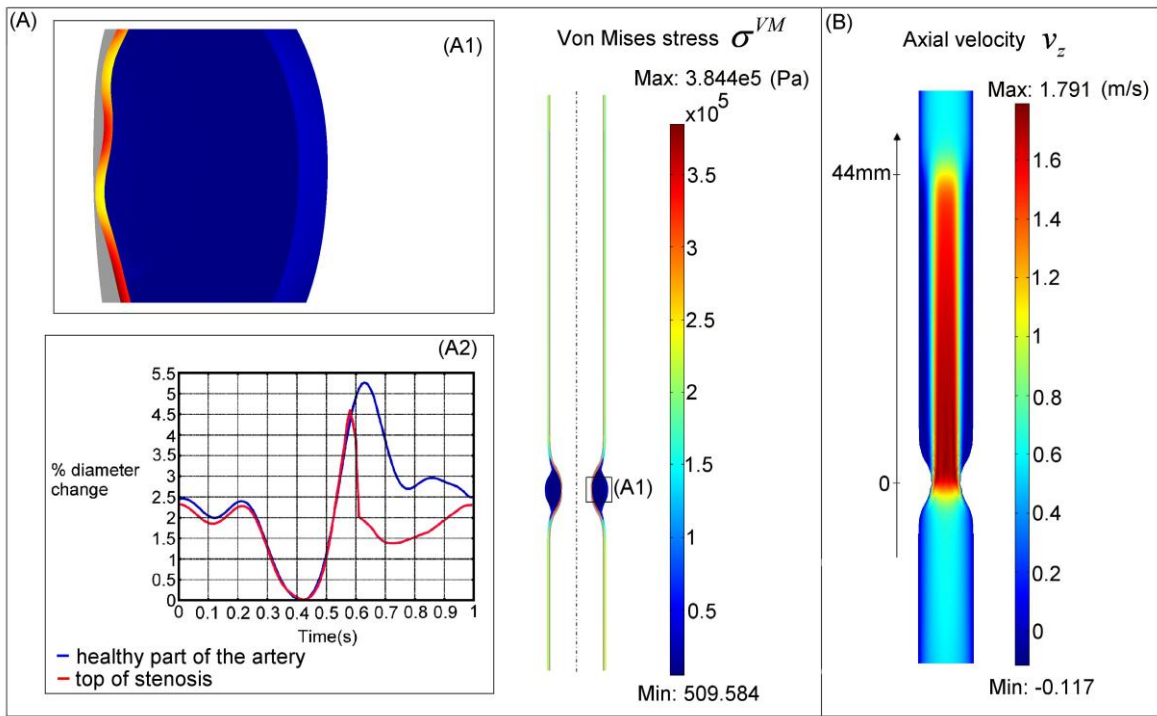


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948 Fig. 3

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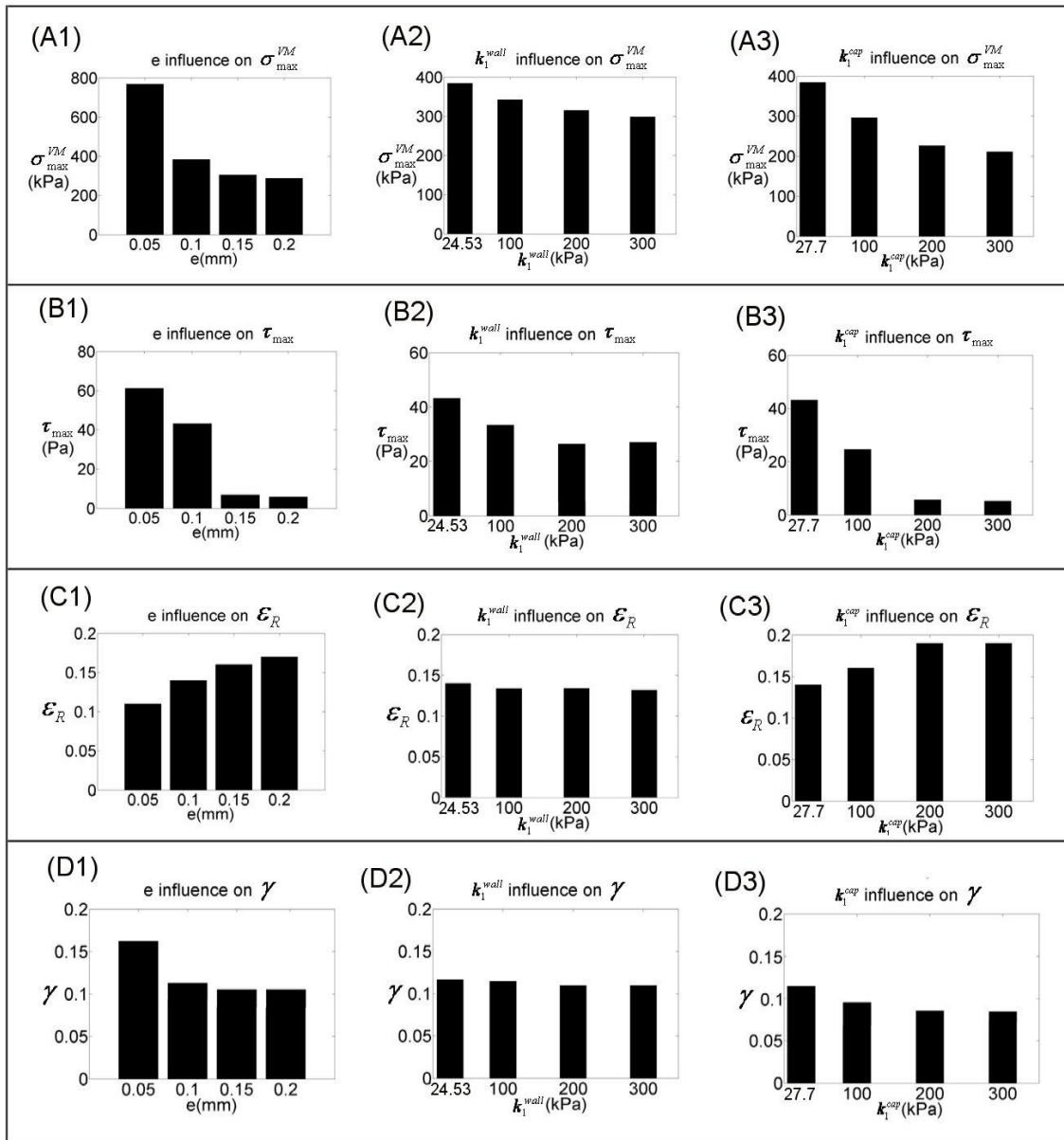


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952 Fig. 4

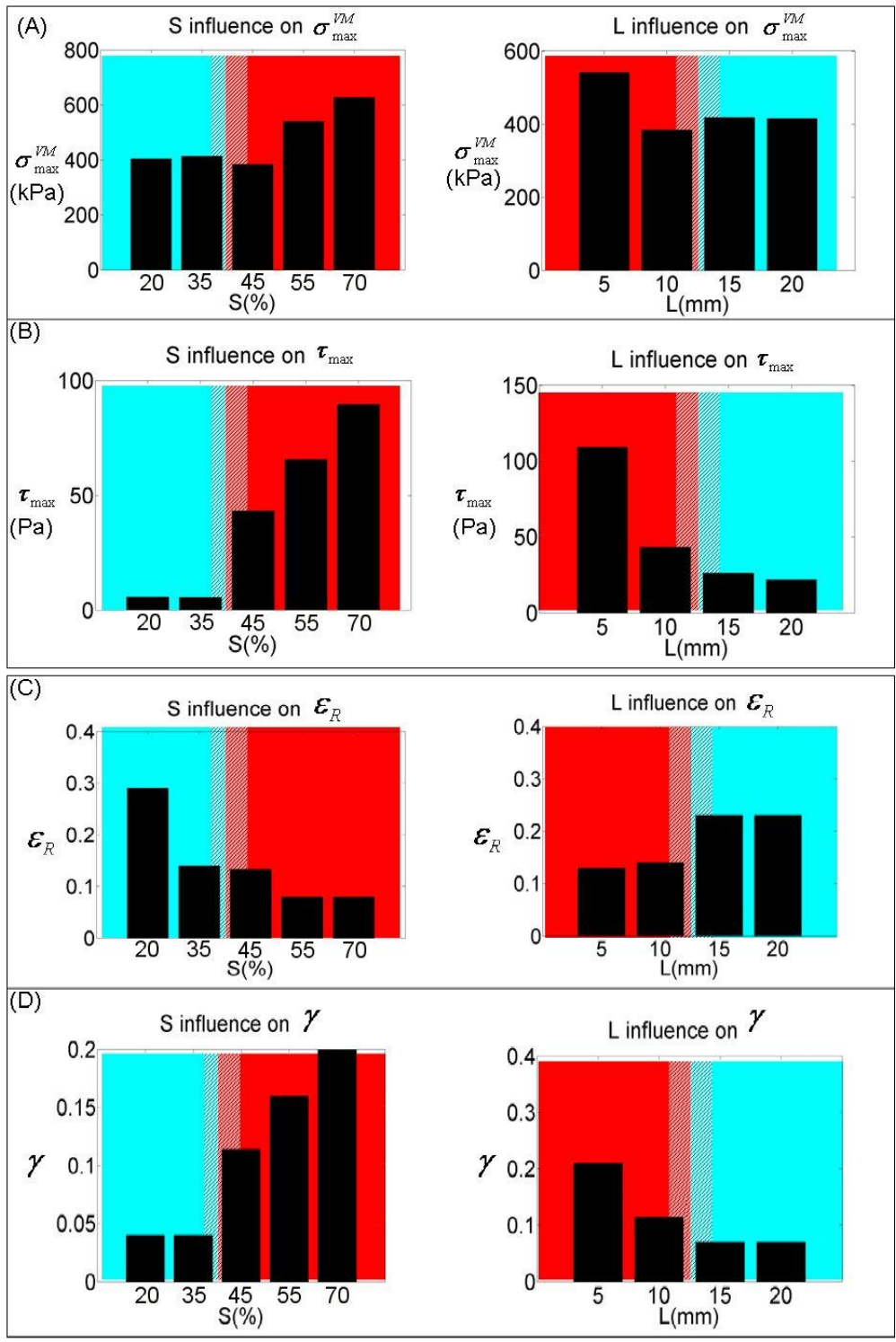
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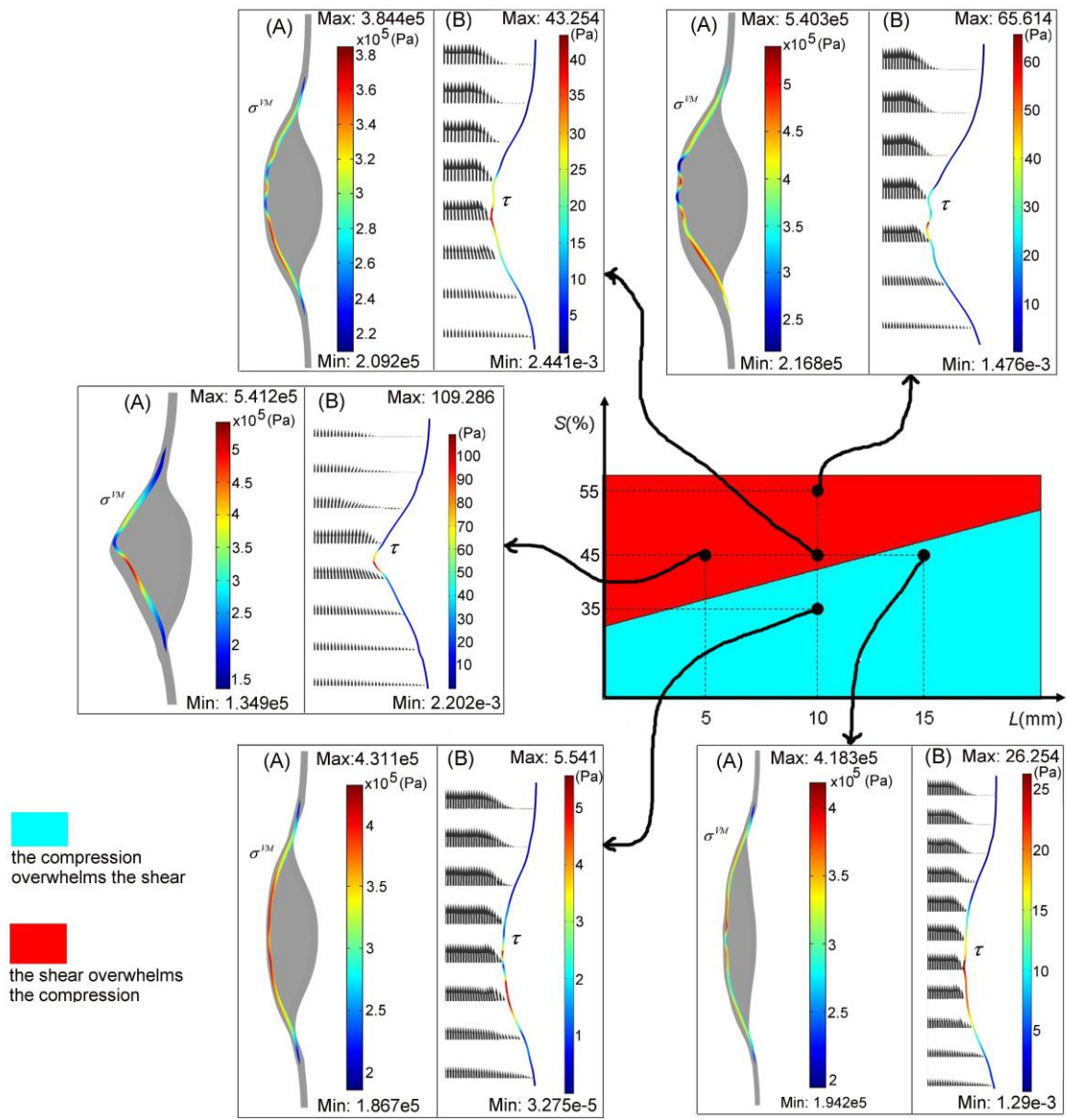




■ the compression overwhelms the shear   
■ the shear overwhelms the compression   
  transition zone

960 Fig. 6

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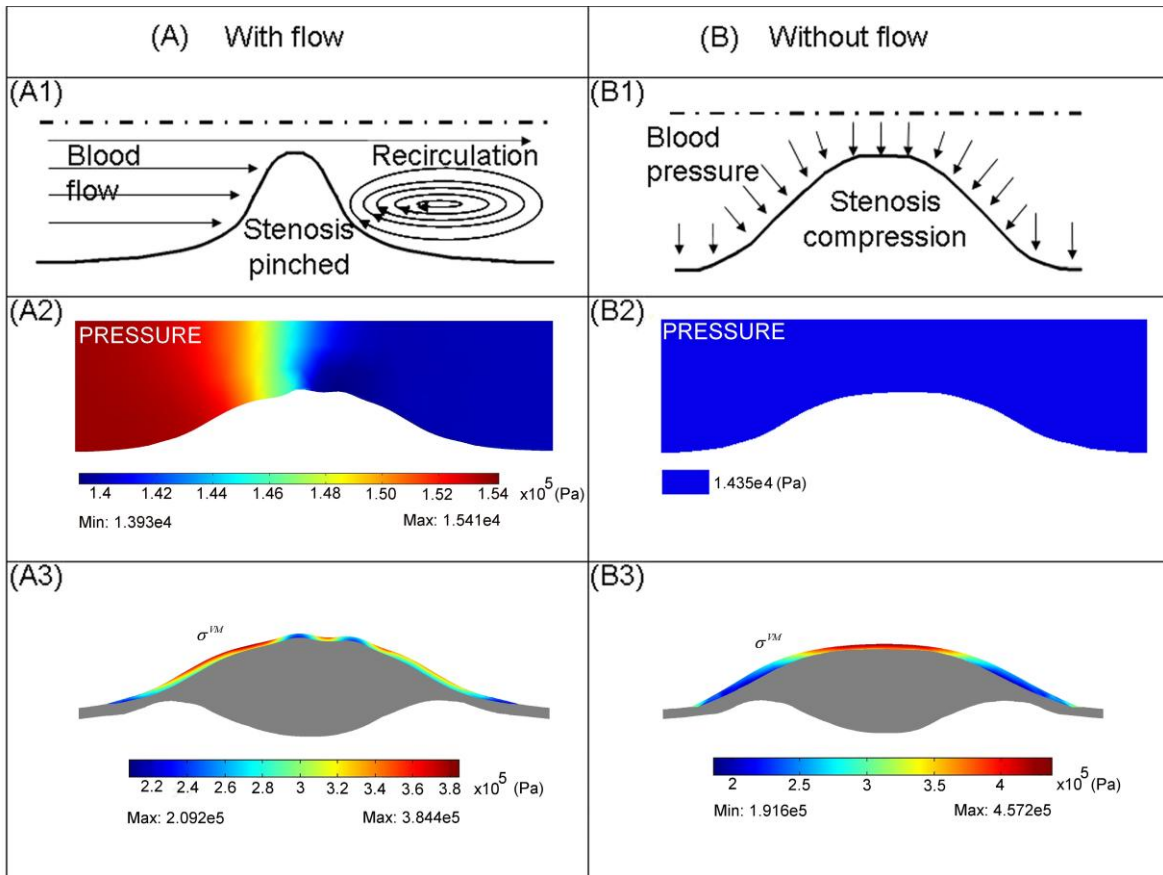


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964 Fig. 7

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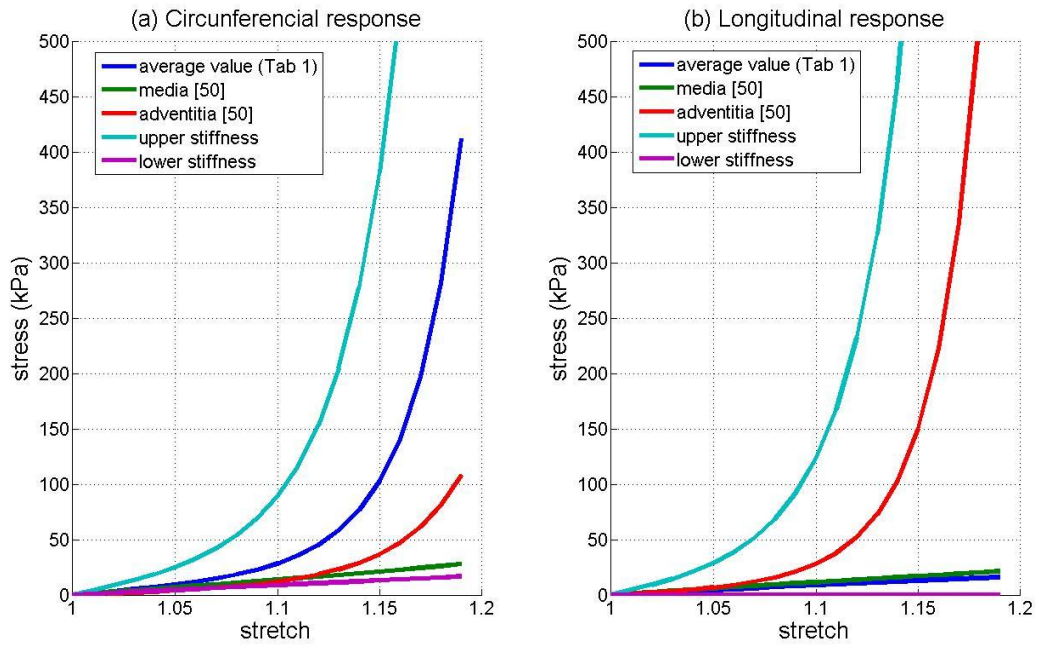
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968 Fig 8

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