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Parametric Stochastic Modelling of Uncertainties in the Mechanical Study of the Abdominal Aneurysm Aorta

Anissa Eddhahak[1], Faîza Mohand Kaci[2] and Mustapha Zidi[2]
[1]*Ecole Spéciale des Travaux Publics, du Bâtiment et de l'Industrie (ESTP), Institut de recherche en constructibilité (IRC),*
[2]*CNRS EAC 4396, Université Paris-Est Créteil Val de Marne, Faculté de médecine, Centre de Recherches Chirurgicales, France*

1. Introduction

1.1 Abdominal Aortic Aneurysm (AAA)

Human body is subjected to many cardiovascular diseases (CVDs) claiming 17.1 million lives a year. Abdominal aortic aneurysms (AAAs), for instance, are the 3th leading cardiovascular cause of death (Sakalihasan et al., 2005; Allaire et al., 2009). The AAA represents a widening of the abdominal aorta generally caused by the hardening of the arteries known as the atherosclerosis. The accumulation of the plaques on the arterial wall leads to its weakness. The blood flow pressure can therefore cause the expansion of the weak arterial part leading thereby to abdominal aneurysm rupture. The AAA can affect either men and women, however statistics have shown that male are five times more likely than female to get AAA. Apart from atherosclerosis, there are many factors which may contribute to the AAA development such as smoking, high blood pressure (hypertension), diabetes, aging, etc.

1.2 Actual pathology solutions

The surgical treatment has been developed in order to avoid aneurysm rupture. Until now, the management of AAAs is instrumental, with intervention decided once the risk of aortic rupture exceeds the risk of elective repair. Traditionally, there are currently two modes of repair available for AAAs, open aneurysm repair, and endovascular stent grafting repair. The first repair consists of opening the abdomen in order to remove the aneurismal aorta part and replace it by a synthetic Dacron tube sewn in place. The second method is less invasive than the open surgey and consists on guiding a graft (stent) within the blood vessel by a catheter just above the damaged aorta section and fastening it to the vessel wall. This technique aims to strengthen the aorta wall and therefore to prevent aneurysm bursting.

It must be noted that the surgical treatment of AAAs carries a high mortality rate of 6 to 14% (Teufelsbauer et al., 2002). No proved cellular or gene therapy exists to inhibit growth or

promote healing human AAA. For this reason, fundamental studies were developed *in-vivo* in animal models to recapitulate features of human aneurysms in the hope of finding treatments which could stop AAA expansion (Dobrin et al., 1984) or promote repair. Thus, different experimental approaches have been used like elastase perfusion (Anidjar et al., 1990) or xenograft implantation (Allaire et al., 1994). Elastase perfusion targetting elastin provides vessels which have some characteristics similar to those observed in human AAAs and has been used to study *in-vitro* the "pressure – diameter" response in canine carotids and human iliacs (Dobrin et al., 1984). O'Connell et al. (2003) employed a rat elastase AAA model to investigate the correlation between arterial mechanical properties and tissue microstructure of AAA. Nevertheless, the clinical relevance of the elastase model is not sufficient since it doesn't create the thrombus as observed in the human AAA.

1.3 AAA Cell therapy (endovascular gene) and biomechanical approach

Apart from surgey and endoprothesis treatments, clinical solutions based on cell therapy have been developed. These approaches are based on the finding that AAAs develop because of extracellular matrix destruction and wall atrophy. The xenograft model, for example, consists in decellularizing an abdominal aorta of a particular animal species (i.e. guinea pig), and to graft it orthotopically into a different species (i.e. rat). This process was used by Allaire et al. (2004) in order to evaluate the impact of the injection of smooth muscle cells into formed AAAs and to determine the proportions of elastin, collagen, and nuclear density in the three layers of the graft wall by morphometric methods after diameter stabilization. The authors investigated the efficiency of endovascular smooth muscle cell seeding in promoting endovascular healing and stability in already-developed AAA by matrix metalloprotease-driven injury. In the same experimental model Dai et al. (2005) have developed an endovascular gene therapy approach and showed that a time-limited expression of TGF-ß1 is sufficient for diameter stabilization.

So far no mechanical approach has been developed to evaluate the impact of gene therapy on AAA stabilization. In fact, one of the challenges is to investigate this experimental approach to estimate the variation of stress distributions in the AAA during its expansion and stabilization.

Thus, much attention has been focused over the years on the biomechanics of aneurysms especially to wall stress assessment and constitutive models (Raghavan et al., 1996; Li and Kleinstreuer, 2005; Di Martino et al., 2006; Watton and Hill, 2009). Accordingly, numerous analytical and numerical models have been developed for this objective (Humphrey, 2002; Vorp, 2007) but none in endovascular biotherapies of expanding AAAs using the Xenograft model.

2. Xenograft model

In order to validate concepts of an endovascular gene therapy developed in surgical research laboratory (Allaire et al., 2004; Dai et al., 2005; Allaire et al., 2009), the experimental xenograft model of AAA was used. First, the abdominal aorta of male guinea pigs was removed and decellularized with a detergent, 0.1% sodium dodecyl sulfate (SDS). Then, the aorta was grafted orthotopically into male Lewis rats (200g). Two weeks after

xenotransplantation, the aneurysm was formed. The rats were reoperated in order to exclude the xenograft from the blood flow by clamps. An aortectomy performed in the healthy aorta so that a PE10 catheter was introduced into the lumen of AAA. A suspension of viruses representing the gene of interest, TGF-ß1 (Ad-sTGF-ß1), or a control gene, *Escherichia coli* ß-galactosidase (Ad-LacZ) was injected. Finally, the aortotomy was sutured and the blood flow reestablished (Figure 1). Both length and diameter of AAA were measured using an operative microscope under beating heart, before artery treatment or harvest. The measured length corresponded to the distance between the two suture lines of the xenograft. The diameter indicated the maximum dilatation level. Note that no tortuosity of the AAA was observed during measurement.

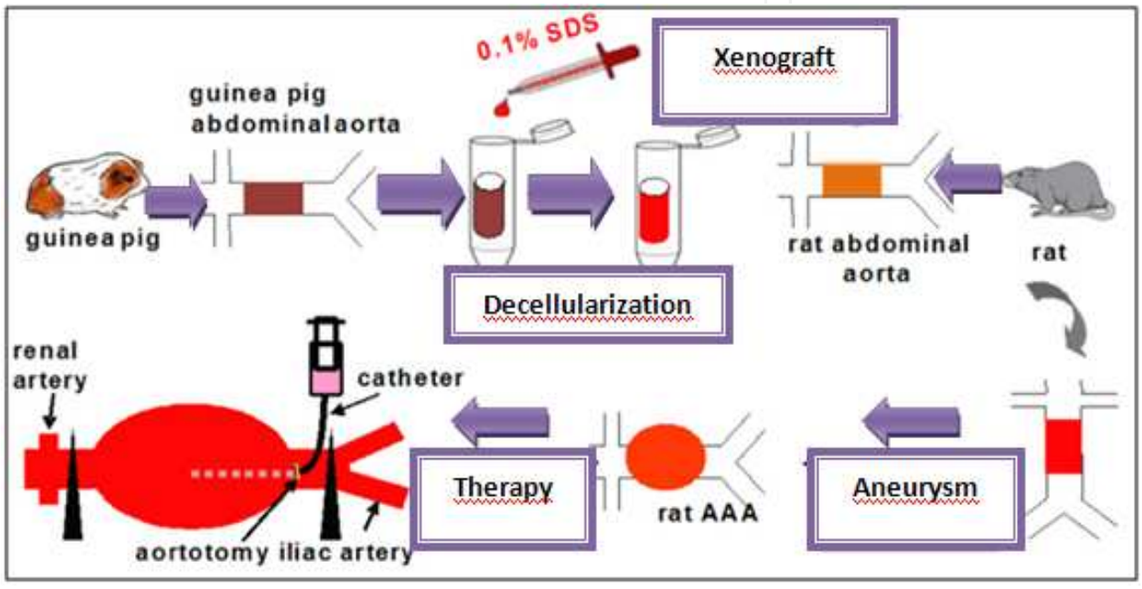

Fig. 1. Different stages of a Xenograft process

In order to study the mechanical behaviour of AAAs in rats during their expansion, , we have used here a membrane model (Humphrey, 2002) based on our experience in gene therapy as well as the xenograft model. Accordingly, it was assumed that the shape of aneurysms is a "parabolic-exponential" function (Elger et al., 1996; Rodriguez et al., 2008, Mohand-Kaci et al., 2011) depending on diameter and length measurements so that the mechanical problem can be solved analytically. Then, in order to investigate the influence of parametric random uncertainty on the growth of AAAs, experimental measurements performed in laboratory were used. A stochastic approach (Jaynes, 1957; Eddhahak et al., 2009) using the principle of maximum entropy is described here to investigate the effect of experimental uncertainties on the evaluation of aorta wall stresses.

3. Mechanical model

Note that the shape of AAAs represents a critical factor influencing the stress distributions in the aorta wall and the aorta rupture mechanism. It was revealed by imaging techniques (Sacks et al., 1999; Lu et al., 2007) that aneurysm can occur in a large variety of complex shapes and

sizes. Nevertheless, for simplification reasons, we considered in the present study that the AAA of rats can be described by an axisymmetric "perfect" membrane (Figure 2). Thus, the AAA is defined with a « parabolic-exponential » shape (Elger et al., 1996; Rodriguez et al., 2008) with the parameters R_0, R_a and L_a which denote respectively the initial radius of the abdominal aorta, the radius and the length of the AAA measured during its expansion.

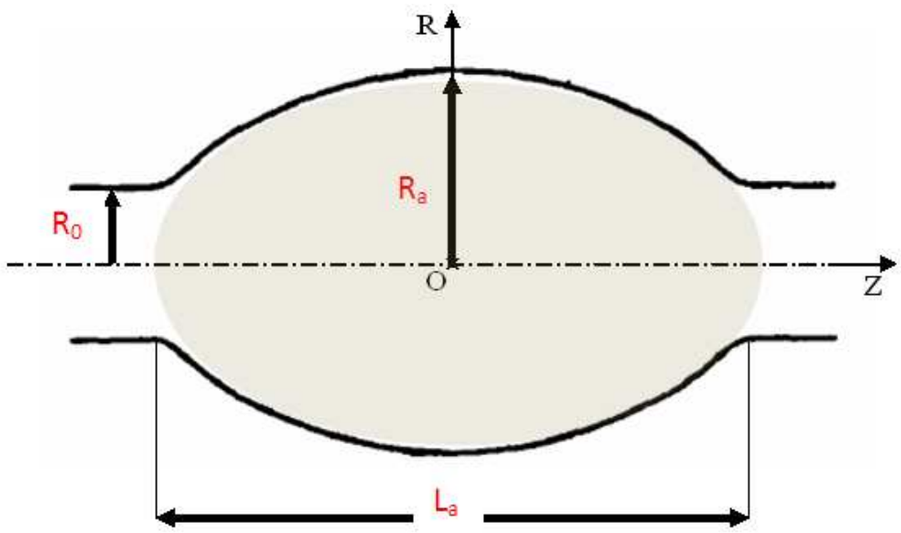

Fig. 2. AAA simplified shape

In the following, given the experimental uncertainties of aorta geometric variables, both radius R_a and length L_a of the aneurysm will be modelled respectively by the random variables $\mathbf{R_a}$ and $\mathbf{L_a}$ (in bold letters). Accordingly, the considered shape was defined by the following function

$$\mathbf{R}(Z) = R_0 + [(\mathbf{R_a} - R_0) - \boldsymbol{\alpha_3}\frac{Z^2}{R_0}].\exp(-\boldsymbol{\alpha_1}\left|\frac{Z}{R_0}\right|^{\boldsymbol{\alpha_2}}) \tag{1}$$

where p_1 is a constant whereas $\boldsymbol{\alpha_2}$ and $\boldsymbol{\alpha_3}$ are random functions since they depends on random variables. They are linked to $\mathbf{R_a}$ and $\mathbf{L_a}$ by

$$\boldsymbol{\alpha_2} = \frac{4.605}{(0.5\times\mathbf{L_a}/\mathbf{R_a})^{\alpha_1}},\ \boldsymbol{\alpha_3} = \frac{R_0(\mathbf{R_a}-R_0)}{(0.5\times\mathbf{L_a})^2} \tag{2}$$

Moreover, a mechanical study of AAAs has been suggested based on data derived from the xenograft rat protocol. For that, we considered the static membrane theory (Humphrey, 2002) which is independent of the AAA wall material properties. By assuming that the AAA is in equilibrium under a uniform intraluminal pressure, one can write the equilibrium equations for the membrane as

$$\begin{cases} \frac{d}{d\mathbf{R}}(\mathbf{R}\boldsymbol{\sigma_1}) = \boldsymbol{\sigma_2} \\ \mathbf{K_1}\boldsymbol{\sigma_1} + \mathbf{K_2}\boldsymbol{\sigma_2} = 1 \end{cases}, \tag{3}$$

where σ_1 and σ_2 represent respectively the longitudinal and circumferential stresses. These stresses, normalized by the pressure, are given by

$$\sigma_1(Z) = \frac{1}{2.K_2} \qquad \sigma_2 = (1 - \frac{K_1}{2.K_2}) \tag{4}$$

K_1 and K_2 represent the local curvatures functions obtained from Eq. (1) and expressed as

$$K_1 = \frac{-d^2R/dZ^2}{[1+(dR/dZ)^2]^{\frac{3}{2}}}, \quad K_2 = \frac{1}{R.[1+(dR/dZ)^2]^{\frac{1}{2}}}. \tag{5}$$

In order to investigate the wall stresses variations, Von-Mises characteristic equivalent stress was also computed

$$\sigma_{vm} = \sqrt{\sigma_1^2 + \sigma_2^2 - \sigma_1\sigma_2}. \tag{6}$$

Note that the normalized stresses given by Eq. (4) and Eq. (6) depend on geometric random variables characterizing the expansion of AAAs.

In Table 1, we present the measurements corresponding to 4 control and treated groups of rats sacrified at 3 or 28 days. All data are expressed as the average ± the standard error/deviation (SE). Statistical analysis was carried out by one-way ANOVA followed by the Mann Whithney U test. The information $p < 0.05$ was considered statistically significant. In addition, it must be also highlighted that the intraluminal pressure was taken constant and equal to the mean value during a cardiac cycle in AAA of rats. The days D0 and D14 correspond respectively to the Xenograft implantation and the endovascular gene therapy in the artificially formed AAAs.

	Radius (mm)		Length (mm)	
3 days after treatment	Control (n=4)	Treated (n=6)	Control (n=4)	Treated (n=6)
D0	0,75 ± 0,02	0,78 ± 0,02	10,33 ± 0,45	11,38 ± 0,39
D14	1,16 ± 0,12	1,22 ± 0,07	11,88 ± 0,75	13,67 ± 0,40
D14+3	1,18 ± 0,11	1,22 ± 0,07	11,95 ± 0,79	13,83 ± 0,33
Relative variation* (%)	1,32 ± 1,32	0,00 ± 0,00	0,85 ± 4,43	1,31 ± 0,83
p	0,24		0,90	
28 days after treatment	Control (n=5)	Treated (n=5)	Control (n=5)	Treated (n=5)
D0	0,77 ± 0,03	0,76 ± 0,02	11,36 ± 0,42	10,84 ± 0,26
D14	1,15 ± 0,05	1,56 ± 0,16	14,44 ± 0,76	15,70 ± 1,74
D14+28	1,82 ± 0,11	1,71 ± 0,24	16,50 ± 1,48	16,30 ± 1,58
Relative variation* (%)	59, 13 ± 11,24	8, 69 ± 6,80	13,62 ± 4,99	4,50 ± 2,98
p	0,02**		0,17	

*: Between D14 and after treatment
p : Mann Whitney U test for % of variation
**: p< 0,05

Table 1. Experimental measurements of aorta radius and length

The plots of figure 3 depict the longitudinal and circumferential membrane stresses σ_1 and σ_2 computed with respect to the mean (or deterministic) model. The stresses are corresponding to the observation days D0=14days, D3=D0+3days and D28=D0+28days. The notation "Dx Ly" in the graph denotes the measurement recorded at the day x and corresponding to the rats group y. The comparison with the initial AAA at D0 reveals that the membrane aortic stresses change very slightly after 3 days in both control and treated cases. On the contrary, a significant increase is noticed at D28. Furthermore, as consequence of a stress gradient due to the axial lesion one can also remark that the stress pick appears always at the maximum radius of the AAA and the medium axial position (Z = 0).

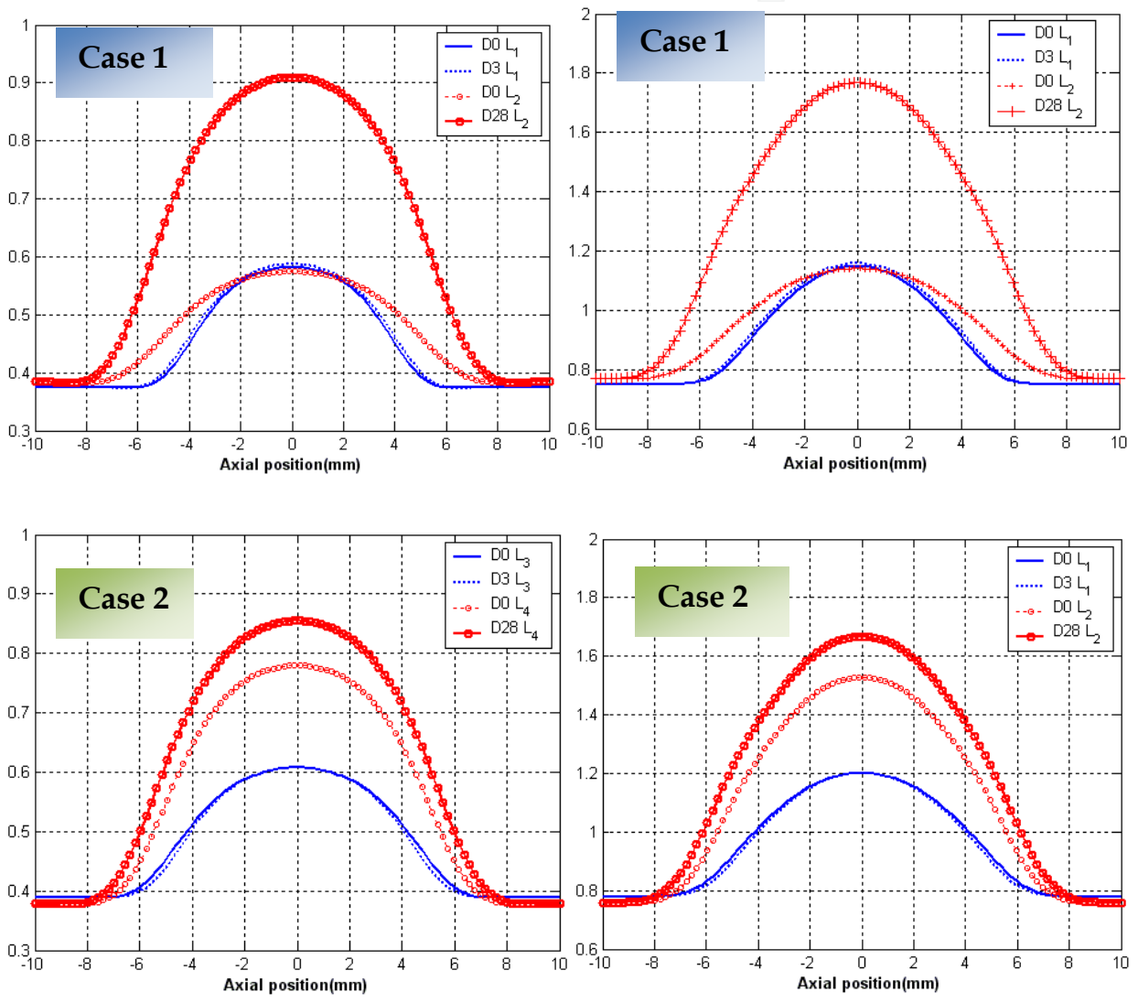

Fig. 3. Variation of normalized aortic stresses versus axial position (Right: σ_2, Left: σ_1).

Indeed, the expansion of AAA influences the magnitude of the peak stress in the aorta wall. The stress amplification is then evaluated to 58% and 55 % for respectively σ_1 and σ_2 as shown in the upper curves. In addition, one can note that the difference between the upper row curves corresponding to the control case (case 1) is more important that the one of the

second row corresponding to the gene therapy case (case 2) notably at the axial position Z = 0. This first biological finding highlights the advantageous of the suggested gene therapy treatment which decreases the wall mechanical stresses in AAAs and improves therefore their mechanical behaviors. In Figure 4, we emphasize on this last finding by comparing the differences of membrane stresses in both treated and control cases.

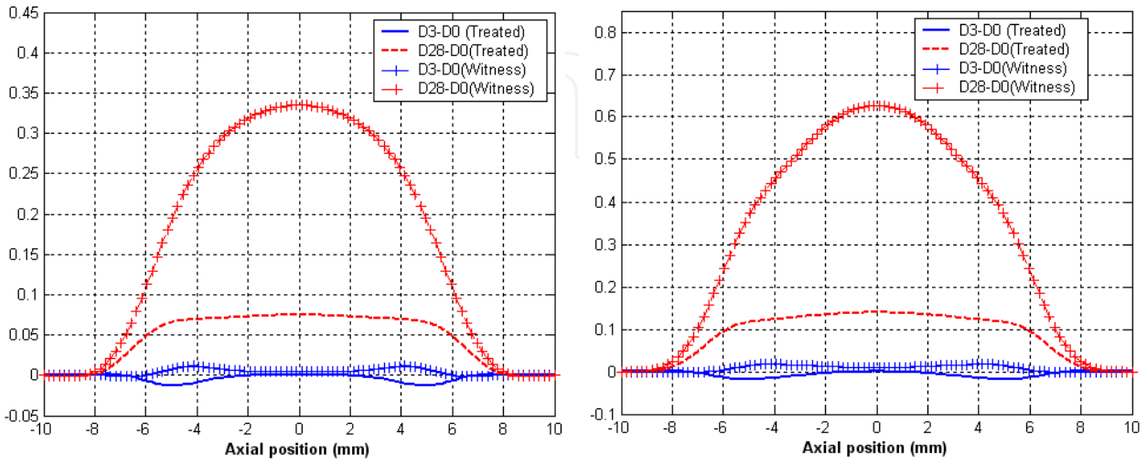

Fig. 4. Effect of gene therapy treatment on the membrane stresses (Right: σ_2, Left: σ_1).

4. Probabilistic parametric modelling of AAA uncertainties

In this section, a stochastic approach taking into account the random dispersion of the experimental measurements related to the growth of AAAs is presented. Dealing with *in-vivo* measurements of rat aortas, the experimental recorded values are often subjected to uncertainties due to the lack of accuracy. The probabilistic parametric approach is an efficient mechanical tool which allows the description of random uncertain parameters by adequate random variables. This description is performed by the attribution of suitable probability density functions (pdf) respective to the considered random variables. The construction of the pdf is not arbitrary and shall take into consideration the available information which may be, for instance, the mean of the random variable, the interval to which it belongs, the standard deviation, the higher order moment, etc.

More general, let consider a parameter x subjected to uncertainty, the random variable denoted X is the stochastic modelling associated to x. The dispersion of X is then measured by the entropy function defined by

$$S(X) = -\int_{-\infty}^{+\infty} p_X(x)\log(p_X(x))dx. \tag{7}$$

where p_X is the pdf associated to the random variable X. This function is determined according to the principle of maximum entropy (Shannon, 1948) which states that the determination of the pdf is obtained by the maximization of the uncertainty on the light of the available used information. The latter defines a set of constraints which govern the optimization problem. The mathematical resolution leads to express the pdf as

$$p_x(x) = \Pi_{[V^-,V^+]}(x)\exp\left(-\lambda_0 - \sum_{t=1}^{n}\lambda_t g_t(x)\right) \quad (8)$$

where λ_i are the Lagrange multipliers, $g_i(x)$ are the different constraints of the optimization problem and Π is the indicator function given by

$$\Pi_{[V^-,V^+]} = \begin{cases} 1 \; if \; x \in [V^-,V^+]] \\ 0 \; if \; not \end{cases} \quad (9)$$

Where $[V^-, V^+]$ represents the support of the pdf associated to the random variable X

The reader could consult (Soize, 2001; Kapur, 1992) for further information on the parametric probabilistic approach using the principle of maximum entropy.

The proposed stochastic approach is adapted to the biomechanical membrane model in order to analyze the influence of geometric parameters on the aorta stress distributions during the expansion of AAAs in the xenograft model.

In our case, the knowledge of the available information (average + support + standard deviation), the pdf of each random variable X can be expressed as

$$p_X(x) = \Pi_{[V^-,V^+]}(x)\exp(-\lambda_0(V^-,V^+,m_x,\sigma_x) - x\lambda_1(V^-,V^+,m_x,\sigma_x) - x^2\lambda_2(V^-,V^+,m_x,\sigma_x)), \quad (10)$$

where λ_0, λ_1 and λ_2 are the optimal values which minimize the convex function *H*

$$H(\lambda_0,\lambda_1,\lambda_2) = \lambda_0 + \lambda_1 m_x + \lambda_2 m_2 + \int_{V^-}^{V^+}\exp(-\lambda_0 - x\lambda_1 - x^2\lambda_2)dx\,. \quad (11)$$

Where m_2 denotes the second order moment linked to the average and the standard deviation of X by

$$m_2 = m_x^2 + \sigma_x^2\,. \quad (12)$$

For instance, the values of λ_0, λ_1 and λ_2 corresponding to $\mathbf{R_a}$ (control case, at D28) are respectively equal to 134.48, -150.41 and 41.32.

Thus, for each random variable, we performed 2000 random independent realizations according to the considered pdf. Figure 5 presents the different realizations of $\mathbf{R_a}$ and $\mathbf{L_a}$ for the control case.

The numerous trials performed for the random variables $\mathbf{R_a}$ and $\mathbf{L_a}$ allow the determination of different responses/realizations corresponding to the longitudinal and circumferential normalized membrane stresses $\boldsymbol{\sigma_1}$ and $\boldsymbol{\sigma_2}$. Accordingly, a confidence region with a high probability of 99% can be defined in order to predict the numerous potential aorta wall stresses responses. The upper and lower bounds of this confidence interval are plotted in figure 6 and compared with the mean model result for both treated and non treated cases.

As can be noticed, the deterministic model response lies inside the confidence interval. The stochastic membrane stresses results show a similar evolution than the deterministic model. Note that the estimation error recorded for $\boldsymbol{\sigma_1}$ and $\boldsymbol{\sigma_2}$, in cases 1 and 2, can reach

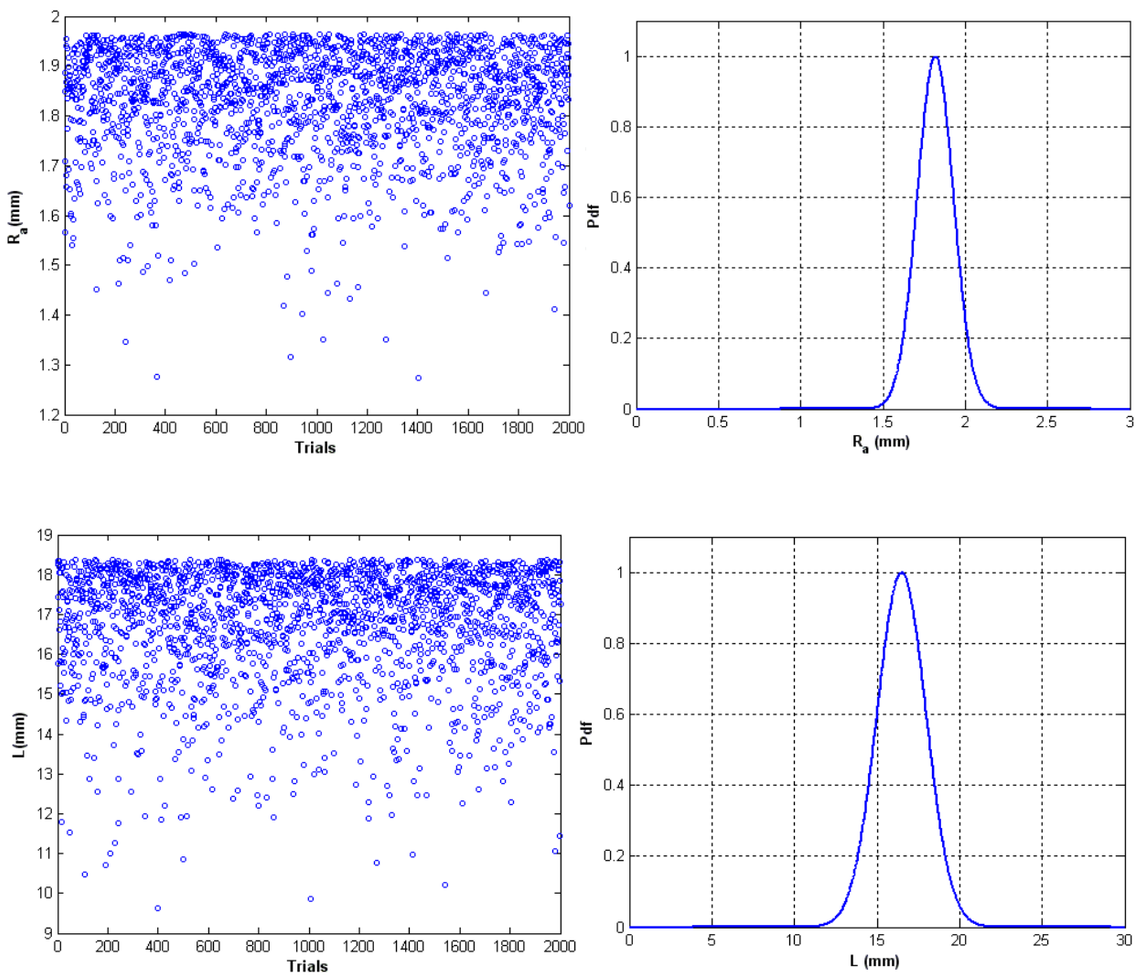

Fig. 5. Realizations and pdf of AAA radius and length

approximately 28%. The aortic mechanical stresses may be underestimated when a stochastic modelling is not considered. This last finding highlights the importance to take into account the parametric random uncertainties in order to obtain realistic estimations of the wall aorta membrane stresses.

Simulations of Monte Carlo (Kalos and Whitlock; 1992) are also carried out to show the convergence of the stochastic process by computing both the mean and the standard deviation (Std) of the Von-Mises stress for cases 1 and 2. Figure 7 illustrates this convergence reached at nearly the 1400th realization for cases 1 and 2. At convergence, the Von Mises equivalent stress is recorded. One can note that the averages of the normalized Von Mises stresses for cases 1 and 2 are respectively equal to 1.54 and 1.43.

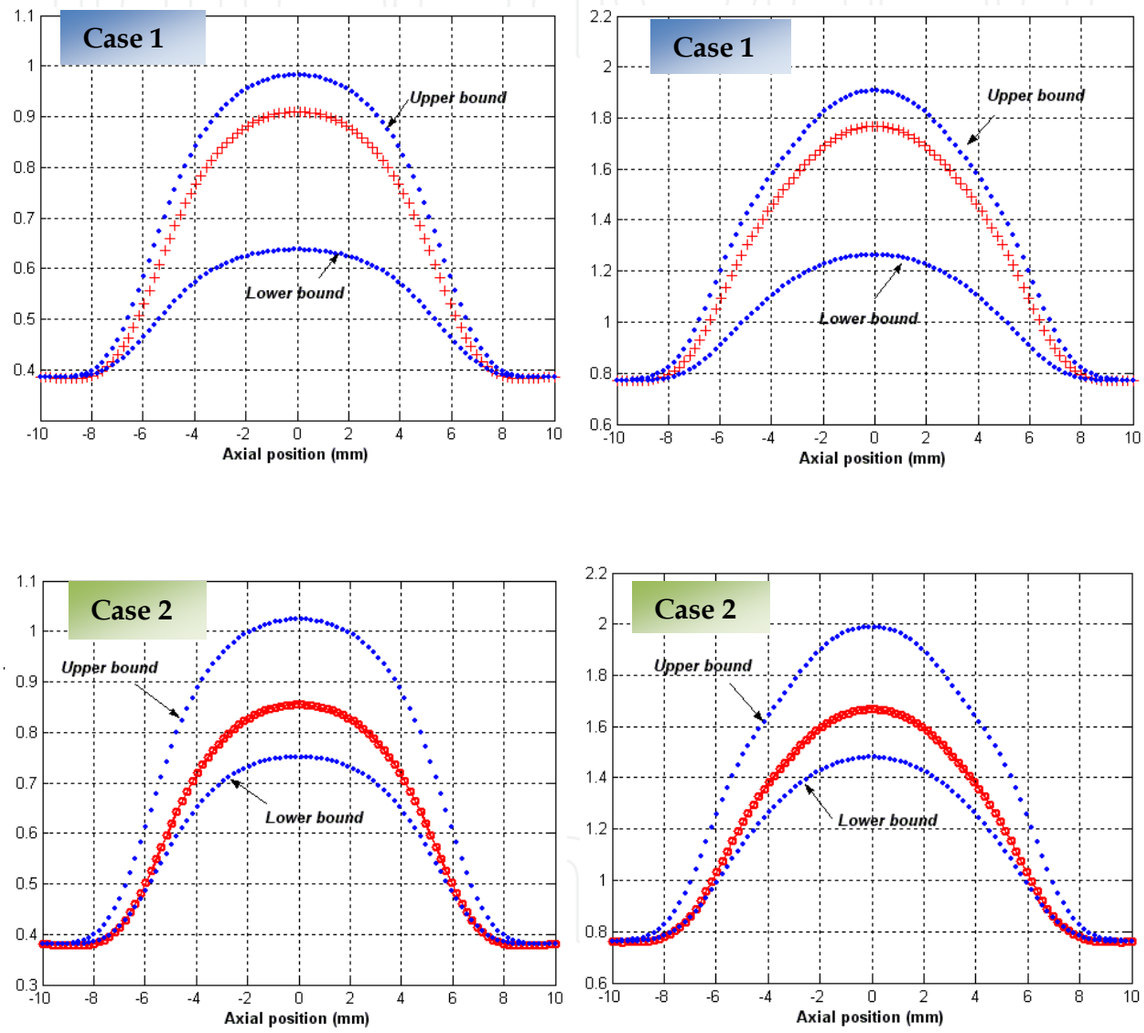

Fig. 6. Stochastic confidence intervals for the aorta membrane stresses (Right: σ_2, Left: σ_1).

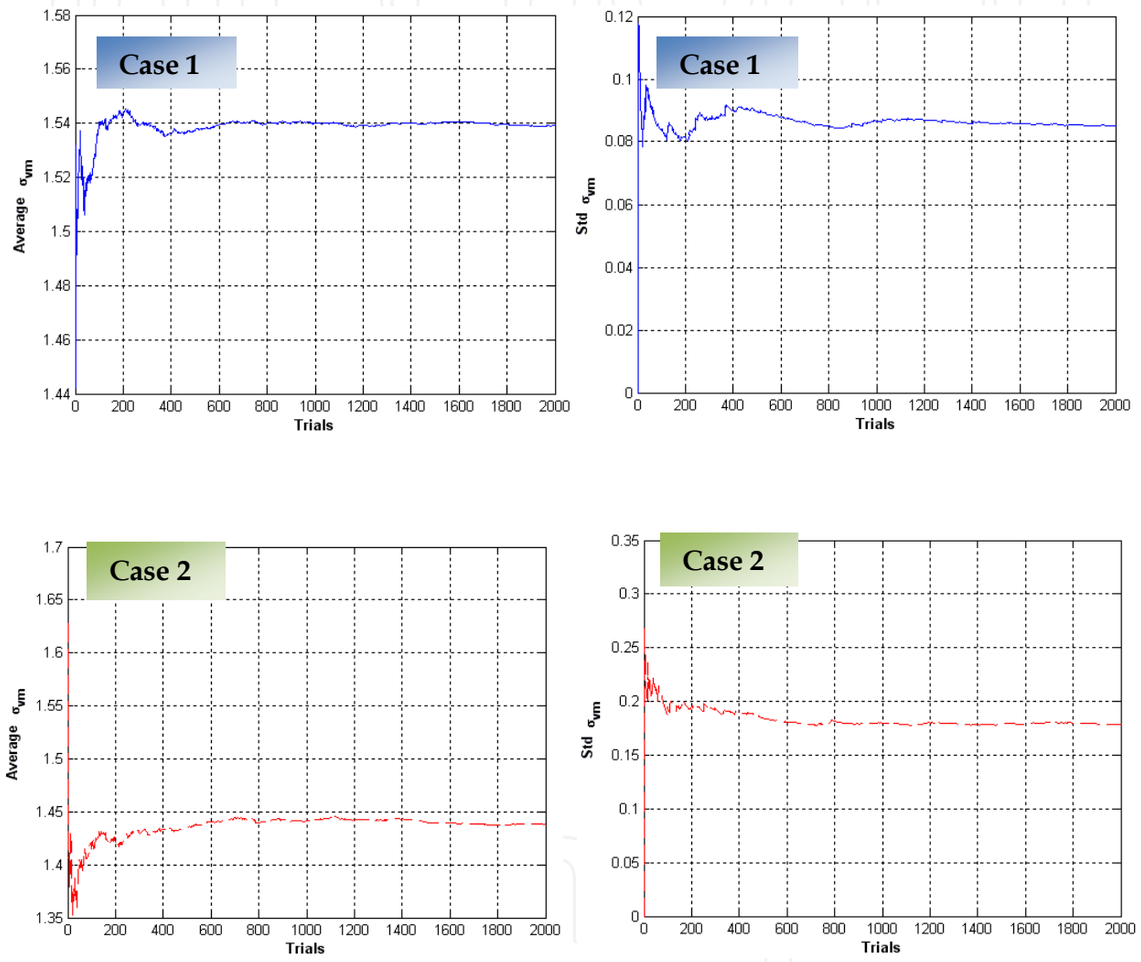

Fig. 7. Monte Carlo simulations

5. Conclusions

In this chapter, a stochastic biomechanical approach adapted to a xenograft model for AAA therapy is presented. The *in-vivo* geometric aorta characteristics (radius and length) were recorded at several days for both control and artificially damaged aortas. Thereby, experimental measurement uncertainties were considered and used for the assessment of parametric probabilistic model based on the principle of maximum entropy. It was shown that the presented endovascular gene therapy reduces significantly the stress variations while stabilizing AAA and likely prevented rupture probability of the artery. In addition, from a stochastic point of view the random experimental uncertainties were described by adequate probability density functions for a safe estimation of AAA wall stresses. Monte Carlo stochastic solver was used and it was noticed its reliability to reach convergence of the probabilistic simulations. This approach can also be generalized for other arterial diseases and can contribute to the improvement of our understanding of the arterial mechanical behavior.

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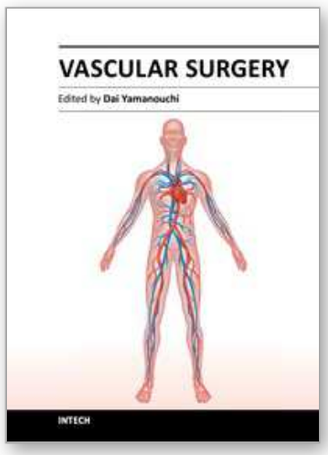

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This book aims to provide a brief overview of conventional open vascular surgery, endovascular surgery and pre- and post-operative management of vascular patients. The collections of contributions from outstanding vascular surgeons and scientists from around the world present detailed and precious information about the important topics of the current vascular surgery practice and research. I hope this book will be used worldwide by young vascular surgeons and medical students enhancing their knowledge and stimulating the advancement of this field.

How to reference

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InTech Europe
University Campus STeP Ri
Slavka Krautzeka 83/A
51000 Rijeka, Croatia
Phone: +385 (51) 770 447
Fax: +385 (51) 686 166
www.intechopen.com

InTech China
Unit 405, Office Block, Hotel Equatorial Shanghai
No.65, Yan An Road (West), Shanghai, 200040, China
中国上海市延安西路65号上海国际贵都大饭店办公楼405单元
Phone: +86-21-62489820
Fax: +86-21-62489821

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