

**POST-ANGIOPLASTIC CONTACT MECHANICS WITH DIFFERENT LEVELS OF  
ARTHEROSCLEROTIC PLAQUE**

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**ABSTRACT**

The main goal of balloon angioplasty is to extend the active cross-section of a partly blocked artery. In the last stage of the medical operation, a metallic frame (stent) is introduced in the open space of the artery (lumen) and expanded to the desired diameter. It is generally accepted that some of the main causes of post-angioplasty restenosis are the global stresses induced in the artery by the expanding stent and the local interaction between the stent and the arterial wall. In a blocked artery a thick layer of hard plaque deposition usually covers a significant section of the wall. Therefore, to choose an appropriate stent and improve upon the angioplasty success rate, a fundamental understanding of the local interaction between the stent and the plaque, as well as between the stent and the healthy wall is vital.

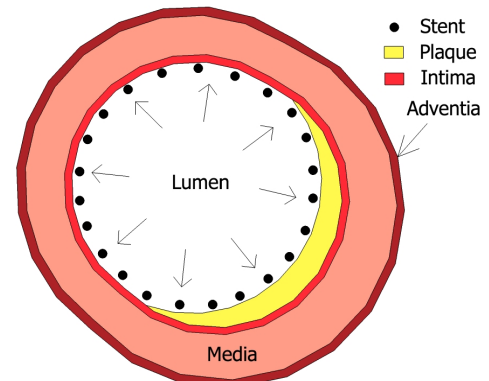
The goal of the present study is to find a correlation between the local thickness of the plaque layer, contact geometry and the stent/artery radial mismatch.

**INTRODUCTION**

Currently, heart related problems are the most significant cause of death in the western world. It is estimated that in 2010, over 785,000 Americans will have a new coronary attack, 470,000 a recurrent attack and 195,000 silent first myocardial infarctions [1]. The cause of many of these incidents is untreated (or recurring) coronary atherosclerosis, which is the result of an accumulation of arteriosclerotic plaque on the inner side of the arterial wall. This is the conglomerate of cholesterol, fatty acids and calcium, interconnected in a fibrous connective tissue. The resulting deposit is quite hard. It also reduces the inner diameter of the artery and affects the blood flow.

There are two traditional solutions: a pathological therapy or a by-pass surgery. Often the first one is contemplated too

late, while the latter is regarded as overly invasive. The third alternative; the solution of choice, is to implant a metallic mesh (stent) at a precise location along the artery to support the arterial wall, thus dilate the lumen (see figure 1). However, besides lumen dilation, the radial mismatch between the stent and the artery also induces stresses in the arterial wall.



**Figure 1: Schematic representation of an artery after angioplasty**

The artery consists of successive bonded concentric layers, each of hyperelastic behavior. The stent is a dense mesh. To fully understand the ensuing interactions, any global deformation must be taken into account, together with the contact mechanics of the stent/artery or stent/plaque interfaces. However, such an analysis can be rather lengthy, and therefore, often prohibitive in practice [2].

The current paper predicts the local deformation of the arterial wall, as well as the induced subsurface stress field during the stent-plaque interaction, whilst presuming different plaque thickness. This study is a first step towards a consistent model, which would consider the stent interaction with the arterial wall.

## NOMENCLATURE

$D$	$0.5 \div 1\text{mm}$ : diametral mismatch
$R$	$2.125\text{mm}$ – radius of the stent
$r$	$55\mu\text{m}$ – significant dimension of the beam cross section
$l_j$	distance between adjacent stent beams
$E_i$	Elasticity of successive layers of the artery
$d_i$	thickness of successive layers of the artery
$P_0$	average constant pressure
$P_k$	amplitude of the $k^{\text{th}}$ harmonic of the applied pressure
$l_{\text{max}}$	maximum depth
$x$	coordinate along the contact surface
$y$	coordinate into the depth of the contacting solid
${}_k u_y$	subsurface deflection due to $k^{\text{th}}$ harmonic
$\nu_i$	Poisson ratio of successive layers of the artery
$\xi$	$\in \{\text{artery, plaque}\}$

## MATEMATICAL MODEL

The arterial wall consists of successive soft-tissue layers with fairly well-defined mechanical properties [2]. The stent is an interconnected frame of thin beams. To achieve a permanent bond, the stent diameter is necessarily larger than the arterial internal diameter. Teodorescu *et al.* [3] proposed a model for the contact between the stent and the healthy artery tissue. In such a case the softer innermost layer of the artery (intima) protects the contact and leads to lower contact pressures. The prediction was considered for diametral mismatch of up to  $1\text{mm}$ . However, after the angioplasty, the main goal of the stent is to compress the plaque layer. Therefore, the current paper extends the previous model for the contact of the stent with a layer of plaque.

Considering that the artery tissue is soft in comparison with that of the stent, as the first assumption the beams may be assumed to be parallel. Therefore, the problem can be simplified to a plane strain rigid punch indenting a soft layer [4]. The distance between any adjacent pair of beams is  $l_{1,2}$  (see figure 2), where for an 18 beam configuration  $l=l_1+l_2=2\pi R/9$ .

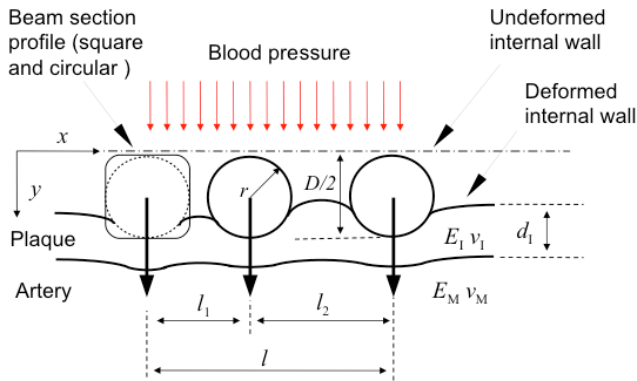


Figure 2: Equivalent model of the stent-plaque contact

As first approximation the blood pressure is assumed to be constant ( $p_{\text{blood}} \sim 100 \text{ mmHg}$ ). Unfortunately, this assumption loses its pulsatile nature, and consequently, its transient behavior. However, this improves the computation speed rather significantly. Thus:

$$p(x) = p_{\text{blood}} - p_{\text{atm}} + p_{\text{stent}}(x) \quad (1)$$

The contact mechanics model proposed by Teodorescu *et al.* [3,4] is carefully adopted here. This method decomposes the contact pressure distribution into a series of harmonic waves and predicts the resulting subsurface stress and strain fields for all given harmonic. Hence:

$$p(x) = p_{\text{blood}} - p_{\text{atm}} + \frac{P_0}{2} + \sum_{k=1}^{N \rightarrow \infty} P_k \cos(\alpha_k x - \phi_k) \quad (2)$$

$$\sigma_{ij}^{\xi} = \sigma_{ij}^{\xi} + \sum_{k=1}^{k \rightarrow \infty} {}_k \sigma_{ij}^{\xi}, \quad \varepsilon_{ij}^{\xi} = \varepsilon_{ij}^{\xi} + \sum_{k=1}^{k \rightarrow \infty} {}_k \varepsilon_{ij}^{\xi} \quad (3)$$

where  $i, j \in \{x, y\}$  and  $\xi \in \{\text{artery, plaque}\}$

The mechanical strength of a healthy artery comprises two of the successive layers (Adventitia and Media) [2]. Therefore, the hyperelastic behavior of the artery wall is calculated by averaging their mechanical properties ( $E \sim 1\text{MPa}$ ). The plaque is approximately twenty times harder and its thickness varies from section to section.

The  $k^{\text{th}}$  component of the subsurface deformation is computed as:

$$u_y = \int_y^{l_{\text{max}}} \varepsilon_y dy \quad (4)$$

## RESULTS AND DISCUSSIONS

Figure 3 predicts the deformed profile and the contact pressure for a stent with rectangular shaped beams indenting plaque layers of different thickness. The thinner layer leads to higher pressure peaks and higher local deformation.

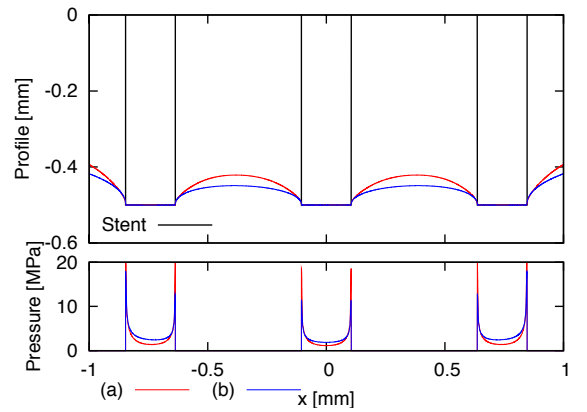
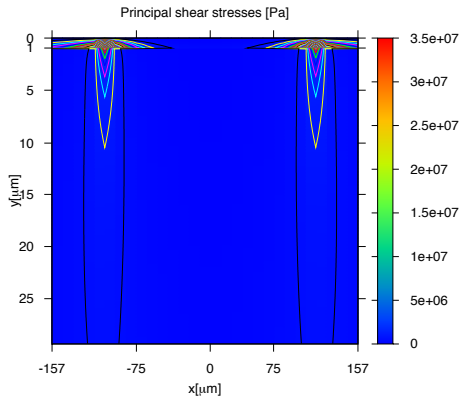
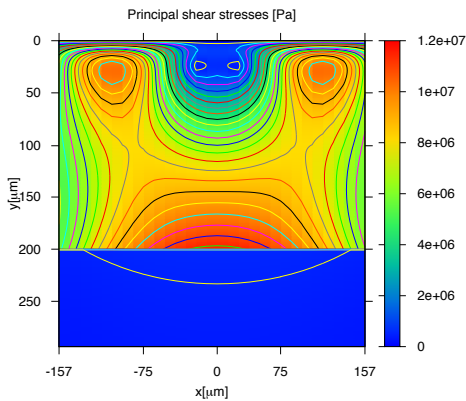


Figure 3: Contact profile and pressure distribution for a thin ( $1\mu\text{m}$  -a) and a thick ( $200\mu\text{m}$  -b) plaque for  $D=1\text{mm}$

Figure 4a) and 4b) show the subsurface stress field for a thin and for a thick arteriosclerotic plaque layers shown in figure 3. It should be noted that the higher contact pressures on the thin layer leads to very high subsurface stresses adjacent to the plaque-artery interface.



(a) Thin plaque layer:  $d=1\mu\text{m}$



(b) Thick plaque layer:  $d=200\mu\text{m}$

Figure 4: Principal stress field for  $D=1\text{mm}$  (see figure 5)

Figure 5 shows the maximum principal subsurface stresses for several plaque layers with different stent-artery diametral mismatch. It should be noted that in each case the thinner layer contains the higher stresses.

Figure 6 shows the maximum principal subsurface stresses for an equivalent stent with a circular beam profile. Although the maximum stresses are lower than for a square profile, the relatively thinner layers still promote higher stresses.

In reality, the plaque is very thick in the center of the arteriosclerotic deposition and becomes progressively thinner around the circumference. Therefore, the maximum subsurface stresses are much higher on the edges of the plaque region than in the middle. If there is a correlation between high subsurface stresses and plaque development, it is possible that excessively high stresses on the edges of the plaque would lead to plaque growth, therefore, contributing to restenosis.

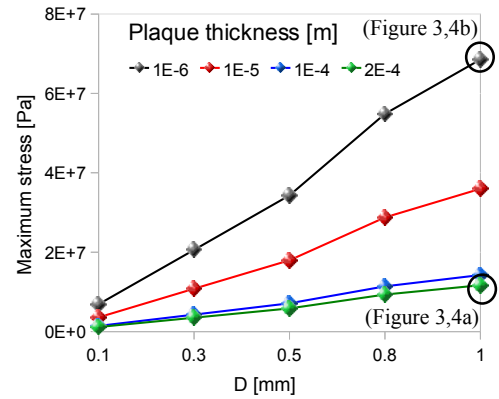


Figure 5: Maximum stresses for a square stent profile.

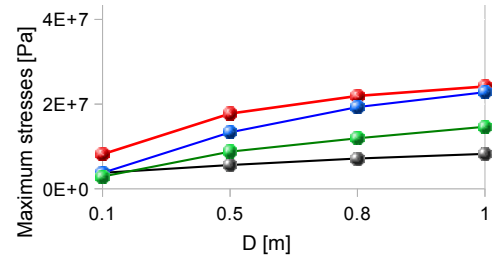


Figure 6: Maximum stresses for a circular stent profile.

## CONCLUSION

This paper proposes a new contact mechanics approach for the stent-plaque interaction. It is concluded that local subsurface stresses are higher for thinner layers of plaque, which corresponds to the edges of the plaque region. The excessively high sub-surface stresses could be one of the reasons contributing towards the extension of the plaque-covered area, and therefore, promoting restenosis.

## REFERENCES

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