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The influence of plaque composition on underlying arterial wall stress during stent expansion: The case for lesion-specific stents

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ABSTRACT

Intracoronary stent implantation is a mechanical procedure, the success of which depends to a large degree on the mechanical properties of each vessel component involved and the pressure applied to the balloon. Little is known about the influence of plaque composition on arterial overstretching and the subsequent injury to the vessel wall following stenting. An idealised finite element model was developed to investigate the influence of both plaque types (hypercellular, hypocellular and calcified) and stent inflation pressures (9, 12 and 15 atm) on vessel and plaque stresses during the implantation of a balloon expandable coronary stent into an idealised stenosed artery. The plaque type was found to have a significant influence on the stresses induced within the artery during stenting. Higher stresses were predicted in the artery wall for cellular plaques, while the stiffer calcified plaque appeared to play a protective role by reducing the levels of stress within the arterial tissue for a given inflation pressure. Higher pressures can be applied to calcified plaques with a lower risk of arterial vascular injury which may reduce the stimulus for in-stent restenosis. Results also suggest that the risk of plaque rupture, and any subsequent thrombosis due to platelet deposition at the fissure, is greater for calcified plaques with low fracture stresses.

1. Introduction

Atherosclerosis is one of the most serious and common forms of cardiovascular disease. An atherosclerotic plaque is an intimal lesion that typically consists of an accumulation of cells, lipids, calcium, collagen and inflammatory infiltrates [1]. These plaques can cause artery occlusion leading to a reduction in blood flow. Several procedures are available to revascularise an occluded artery, including balloon angioplasty and stenting, bypass surgery and atherectomy. In cases of coronary artery occlusion, most percutaneous revascularization procedures involve coronary stent implantation [2]. Placement of a coronary stent typically involves predilation of the atherosclerotic lesion with an angioplasty balloon and permanent deformation of the stent using balloon expansion, such that the stent remains expanded inside the vessel as a scaffold, to maintain patency. Overstretching of the artery during stenting can cause vascular injury, which is related to the degree of subsequent neointimal hyperplasia and restenosis [3].

Angioplasty and stenting are mechanical procedures, hence their outcome depends on the pressure applied by the cardiologist to the balloon, as well as the geometry (e.g. of the artery, plaque, stent and balloon) and the mechanical properties of each vessel component [2]. Balloon expansion pressures provided by manufacturers for their stent designs are typically less than 12 atm, however, clinicians frequently pressurise stents over a broad pressure range (10–17 atm) [4,5]. The expansion pressure is generally chosen based on clinical experience, the lesion type and observed vessel dilation under fluoroscopy.

It is also known that the composition and mechanical properties of plaques vary considerably as atherosclerosis progresses and that these properties can be determined using imaging techniques [6,7]. Plaques classified histologically as either cellular, hypocellular or calcified have been shown to have statistically different radial compressive stiffness [8] while non-significant differences have been reported in the tensile stiffness of histologically different groups of plaques [9]. While this latter study reported that hypocellular plaques were on average about twice as stiff as cellular plaques at physiological ranges of tensile stress, the large variability in tensile properties within the groups made the differences not statistically significant. The results of these and other studies [10] suggest that different plaques may respond differently to the same stenting procedure. Furthermore, they infer that the level of vascular injury during stenting may be dependent upon the plaque properties, implying that the long-term outcome of stenting is lesion dependant. Quantifying the injury or loading within different plaques under various different inflation pressures during stenting is

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therefore necessary to optimise both stenting procedures and the design of stents.

The finite element method is an established technique for investigating implant–vessel interactions during and following stent placement and has been used extensively in recent years to analyse different stent designs [11–14]. Finite element models of complex atherosclerotic plaques have been developed, initially in two-dimensions and/or without a stent present [15–17], and more recently in three-dimensions to investigate stent–artery interactions [11]. Little is known about the influence of changing plaque compositions during disease development on the load induced injury within a stented vessel. The objective of this study is to determine the influence of both plaque properties and balloon inflation pressures on vessel and plaque stresses due to the implantation of a balloon expandable coronary stent using the finite element method. We hypothesise that as a plaque develops and its mechanical properties change, the stresses developed within both the artery and plaque due to a stenting procedure will depend on the plaque composition. If this is indeed the case, it will provide compelling evidence for the development of lesion-specific stents to minimise injury to the vessel wall.

2. Materials and methods

A comparative three-dimensional numerical simulation of an idealised stent–plaque–artery interaction was developed to predict stress conditions within arterial tissue during stent deployment for various plaque compositions and stent deployment pressures. Three models were developed, each representing a plaque of different material composition, i.e. cellular, hypocellular or calcified. The models were built and meshed using the ANSYS 10.0 preprocessor (Ansys Inc., Pittsburgh, PA, USA) and solved using the explicit dynamics finite element code LS-DYNA 970 (LSTC, Livermore, CA, USA). The stent design used in the simulations was based on the 3.5 mm Driver stent (Medtronic, Minnesota, USA). The Driver stent has 10 crowns and 5 weld points in the circumference of each modular unit. The welds are offset by half a crown in subsequent modular units along the length of the stent. The geometry of the stent for the analyses was obtained from geometrical measurements of the commercially available stent design.

2.1. Model geometry and mesh

Taking advantage of symmetry conditions, it was possible to utilize a one-eighth model of the problem (see Fig. 1). The artery was described as a semi-cylinder, 11 mm in length, with a 4 mm outer diameter and a wall thickness of 0.5 mm. The plaque covered a length of 7 mm and had a maximum wall thickness of 0.3 mm, corresponding to a maximum stenosis of 36% (the percentage stenosis is given by the percentage of the host vessel that is occluded by the area of the stenosis). Common nodes formed the boundary between the artery and plaque. The Driver stent geometry was described by eight segments (see Fig. 1), joined at periodic locations by a common node to represent the weld points. Each of the segments was 1 mm in length, with a diameter of 0.09 mm [18]. Thus, the stent is 1 mm longer than the stenosis with an overlap of 0.5 mm between the stent and the plaque on each side of the stenosis. The outer diameter of the stent in its initial unloaded crimped condition was 1 mm. The geometry of the stent was discretised into 8640 elements, while the artery and plaque geometries consisted of 19,800 and 14,000 elements, respectively. The mesh resolution was determined on the basis of a mesh density study. The artery mesh density was chosen to ensure minimum penetration during contact. All elements in the simulation were fully-integrated 8-node hexahedra, thus avoiding possible hourglassing effects.

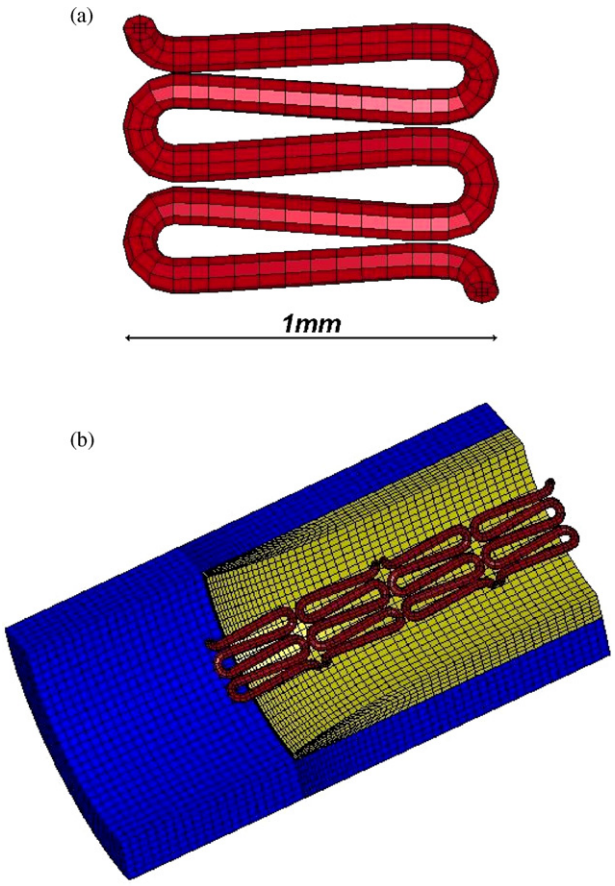

Fig. 1. (a) Driver stent geometry detail. (b) Discretised model geometry (stent in the unexpanded configuration).

2.2. Material properties

The arterial tissue and three types of plaque material (cellular, hypocellular and calcified) were defined by a Mooney–Rivlin hyperelastic constitutive equation. This has been found to adequately describe the non-linear stress–strain relationship of elastic arterial tissue [19].

The general polynomial form of the strain energy function in terms of the strain invariants, given by [20] for an isotropic hyperelastic material is:

$$W(I_1, I_2, I_3) = \sum_{p,q,r=0}^{n} C_{pqr}(I_1 - 3)^p (I_2 - 3)^q (I_3 - 3)^r \quad (1)$$

$C_{000} = 0.$

where W is the strain energy function of the hyperelastic material, I_1, I_2 and I_3 are the strain invariants and C_{pqr} are the hyperelastic constants. If the principal stretches of the material are denoted λ_1, λ_2 and λ_3, then the strain invariants for the material may be defined as:

$$I_1 = \lambda_1^2 + \lambda_2^2 + \lambda_3^2 \quad (2a)$$

$$I_2 = \lambda_1^2\lambda_2^2 + \lambda_1^2\lambda_3^2 + \lambda_2^2\lambda_3^2 \quad (2b)$$

$$I_3 = \lambda_1^2\lambda_2^2\lambda_3^2 \quad (2c)$$

Arterial tissue was assumed to be incompressible based on the results of previous studies [21,22]. For an incompressible material, the third invariant is given as $I_3 = 1$. The specific hyperelastic constitutive model used to model the arterial tissue in this study is a