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## Effect of longitudinal anatomical mismatch of stenting on the mechanical environment in human carotid artery with atherosclerotic plaques

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### ABSTRACT

Longitudinal anatomic mismatch (LAM) of stenting (i.e., a stenotic artery segment is not fully covered by a deployed stent) worsens the mechanical environment in the treated artery, which most likely is the cause for the associated high risks of restenosis, myocardial infarction and stent thrombosis. To probe the possibility, we constructed a patient-specific carotid model with two components of plaques (lipid and calcified plaque) based on MRI images; we numerically compared three different stenting scenarios in terms of von Mises stress (VMS) distribution in the treated arteries, namely, the short stenting (LAM), the medium stenting and the long stenting. The results showed that the short stenting led to more areas with abnormally high VMS along the inner surface of the treated artery with a much higher surface-averaged VMS at the distal end of the stent than both the medium and long stenting. While the VMS distribution in the calcified plaques was similar for the three stenting models, it was quite different in the lipid plaques among the three stenting models. The lipid plaque of the short-stent model showed more volume of the lipid plaque subjected to high VMS than those of the other two models. Based on the obtained results, we may infer that the short stenting (i.e., LAM) may aggravate vascular injury due to high VMS on the artery-stent interaction surface and within the lipid plaque. Therefore, to obtain a better outcome, a longer stent, rather than a short one, might be needed for arterial stenting.

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### 1. Introduction

Stent implantation is an effective and preferential treatment for atherosclerosis [1,2]. However, this treatment faces the challenge of late failure resulting from in-stent restenosis and late stent thrombosis. According to clinical data, 15%–30% of patients who have had stents for longer than 6–12 months suffer from restenosis [3,4]. Among various risk factors for those adverse results, LAM widely presents in clinical studies [5,31]. LAM was defined as the situation when the injured or stenotic segment of artery was not fully covered by the stent. According to long-term outcomes of 1557 patients treated with stenting, LAM occurred in 910 patients (64.1%) [5]. Another clinical study showed that there was a 3-fold increase in myocardial infarction rates associated with LAM [6]. These clin-

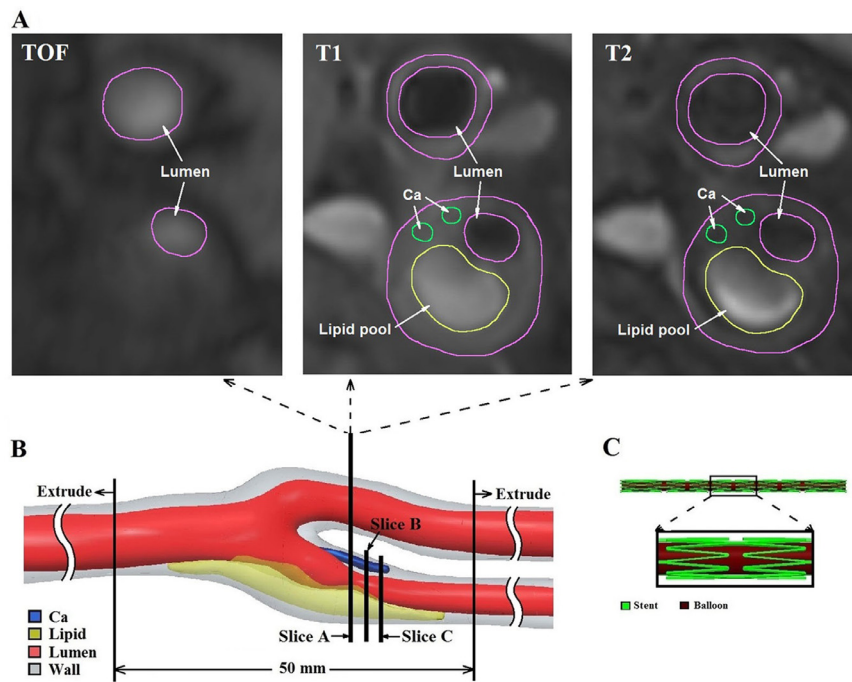
ical data indicated that LAM is a common occurrence during stent deployment and leads to a significantly higher risk for restenosis, revascularization, myocardial infarction and stent thrombosis [7].

The mechanism for the higher rate of clinical adverse events remains unclear. Nevertheless, the alteration of mechanical environment in a host artery after stent deployment has been widely recognized as being related to the late failure of stenting [1,8]. The altered local mechanical environment of the vessel is due to interactions between the stent and the artery [5,9]. The compressive force acting on the artery wall from the stent struts can trigger vascular injury and then stimulate the formation of intimal hyperplasia, and eventually leading to restenosis [8,10]. We hypothesize that LAM may induce an abnormal mechanical stress distribution in the host artery, hence leading to higher risks for restenosis, revascularization, myocardial infarction and stent thrombosis.

To probe this possibility, we constructed a patient-specific stenotic carotid artery model with two plaque components (lipid plaque and calcified plaque) based on MRI images of a patient

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**Fig. 1.** Models used in this simulation. (A) One representative segmented slice of the MRI images (TOF, T1, T2). (B) The re-constructed 3D artery model with different plaque components. (C) The stent-balloon model.

and numerically analyzed the von Mises stress (VMS) distribution in the model. The effect of different stenting scenarios (short, medium and long stenting) on the VMS distribution in the treated arteries was discussed.

## 2. Methods

### 2.1. Image-based carotid bifurcation models and stent-balloon model

Images of 25 transverse slices of the left carotid were acquired from a 62-year-old patient with atherosclerotic plaques. Each slice was captured by a 3-Tesla Imager (Siemens Medical Solutions, Forchheim, Germany) at a 2-mm thickness, a  $512 \times 512$  pixel image size, and a 0.26-mm pixel size. The patient provided written, informed consent to be in this study. This study was approved by the Ethical Committee of Beijing Anzhen Hospital and carried out in accordance with the regulations of the hospital.

The plaque, lumen and vessel were manually segmented using Mimics software (Materialise, Leuven, Belgium). The characterized plaque composition and lumen and vessel wall morphology were obtained by comparing the T1, T2 and TOF (time-of-flight) images of the carotid atherosclerosis. Fig. 1 displays the model used in this study. Because the signal intensity of calcified tissue in plaque is weak (black color in Fig. 1) in all MRI images. T1 images show the highest contrast in the calcification. So, to obtain more accurate contour plots of the calcification in this work, we segmented the calcification with T1 images referring to other series MRI images. The T2 images were suitable for characterizing lipid pools and outer boundaries, and the TOF images delineated the contour of the lumen (Fig. 2).

### 2.2. Stent

A three-dimensional design software, Pro/E (Parametric Technology Corporation), was used to construct carotid stents. These stents had an external diameter of 2.5 mm and a strut thickness of  $81 \mu\text{m}$  [11]. Additional geometrical features of these stents have

been described in previous works [12]. The stents were bent and distorted along the centerline of the lumen for proper positioning in complex, patient-specific geometries. Finally, the stents were assembled in the lumen along the lumen centerline.

To investigate the influence of LAM of stenting on the local mechanical environment after stenting, stenting procedure simulations were implemented for the following models:

**Model 1** used a stent shorter than the length of the lesion to investigate the influence of LAM on the local mechanical environment after stenting. The ratio of stent-to-lesion length was set at 0.75 [5,13].

**Model 2** and **Model 3** were constructed as controls with a stent longer than the length of the lesion and a medium stent, respectively. The ratio of stent-to-lesion length was set at 1.5 and 1, respectively [14–16].

### 2.3. Material properties

The carotid arterial wall and different plaque materials (e.g., lipid pools and calcifications) were assumed to be nonlinear, isotropic and incompressible. These materials have been widely defined by the Mooney–Rivlin hyperelastic constitutive equation [17,18]. The strain energy function is given by

$$W = C_{10}(I_1 - 3) + C_{01}(I_2 - 3) + C_{20}(I_1 - 3)^2 + C_{11}(I_1 - 3)(I_2 - 3) + C_{02}(I_2 - 3)^2 + \frac{1}{d}(J - 1)^2$$

where  $C_{10}$ ,  $C_{01}$ ,  $C_{20}$ ,  $C_{11}$ , and  $C_{02}$  represent material constants;  $I_1$  and  $I_2$  represent the first and second strain invariants, respectively;  $d$  represents the incompressible material parameter; and  $J$  represents the ratio of the deformed volume to the non-deformed volume. The arterial tissue and calcified portions of the wall material model were determined by curve-fitting data from uniaxial tensile tests of human carotid arterial tissue [19–21]. The values used in this study are provided in Table 1, and other material constants not present in the table are assumed to be 0, e.g.  $C_{30}$  for the artery is 0 Pa.

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