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Experimental and numerical studies of two arterial wall delamination modes

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ABSTRACT

Arterial wall dissection, which results from various pathophysiological processes, can lead to the occurrence of large area delamination in the aortic wall that can potentially block blood flow and lead to deleterious clinical conditions. Despite its critical clinical relevance, few studies have focused on investigating the failure mode of delamination in the arterial wall. In this study, we quantify the energy release rate of the medial layer of a porcine abdominal aorta via two delamination experiments: the mixed-mode delamination experiment and the "T"-shaped delamination experiment. A cohesive zone model (CZM) is applied to simulate the arterial wall delamination and Holzapfel-Gasser-Ogden (HGO) material model is used to capture the bulk arterial material behavior. A set of parameter values for the HGO and CZM models are identified through matching simulation predictions of the load vs. load-point displacement curve with experimental measurements. Then the parameter values and critical energy release rates obtained from experiments are used as input data for simulation predictions for two arterial wall delamination experiments. The simulation predictions show that the delamination front matches well with experimental measurements. Moreover, the mixed-mode delamination experiment reveals a shear mode-dominated failure event, whereas the "T"-shaped delamination experiment is an opening failure process. The integration of experimental data and numerical predictions of arterial delamination events provides a comprehensive description of distinct failure modes and aids in the prediction of aortic dissection.

1. Introduction

Aortic dissection, manifested as delamination and separation of the medial layer of the arterial wall, may result in significant blood flow directed into the newly created second lumen. The diverted blood flow further promotes the delamination process in the form of Mode I or Mixed-mode failure (Gasser and Holzapfel, 2006; Leng et al., 2015a). Moreover, with the inherent inhomogeneity of the arterial wall along the delamination path, the delaminated medial layer may be peeled from the other layers by a mixed-mode failure process (Leng et al., 2015a). The failure process within the media can trigger aortic dissection and may cause rupture of abdominal aortic aneurysms (Daugherty and Cassis, 2002; Golledge and Norman, 2010; Venkatasubramaniam et al., 2004) and false lumen patency of descending thoracic aorta (Bernard et al., 2001). Numerous studies have investigated the dissection behavior of arterial tissue under mode I (Ferrara and Pandolfi, 2010; Gasser and Holzapfel, 2006) or mixed-mode failure between atherosclerotic plaque and media (Leng et al., 2015a), yet little research has focused on comparing the contributions of these two failure modes to the delamination propagation process in the arterial wall.

It is well known that elastin is the major load bearing structural component of the arterial wall at low strain and that collagen fibers contribute to the stiffening of the arterial tissue at high strain as they are gradually recruited (Ferrara and Pandolfi, 2010; Zhou et al., 2015). Structurally motivated constitutive models of arterial tissue specifically account for the distinct mechanical behaviors of these two matrix proteins in determining the overall material response over a wide range of physiological loads. In particular, the Holzapfel-Gasser-Ogden (HGO) material model has been widely employed for modeling the mechanical properties of arterial walls and characterizing the local stress environment of mechano-sensitive vascular smooth muscle cells in the arterial wall under large deformations (Ferrara and Pandolfi, 2010; Leng et al., 2015a; Prim et al., 2016).

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The cohesive zone model (CZM) provides an effective method for modeling the interfacial damage within the arterial wall during the delamination processes. Numerous studies have employed the CZM approach to characterize interfacial debonding and delamination processes in fiber-reinforced composite materials (Roy and Dodds, 2001; Turon et al., 2006). This method has also been implemented to study the failure of arterial tissue in two- (Ferrara and Pandolfi, 2010; Gasser and Holzapfel, 2006) and three-dimensions (Gasser and Holzapfel, 2007; Leng et al., 2015a). Moreover, the CZM with an exponential cohesive law has been adopted for modeling the delamination behavior along the interface within the medial layer of porcine abdominal aorta (Camanho et al., 2003).

Existing studies in the literature have investigated the dissection of the arterial wall, but experimental investigations of arterial wall delamination events and the analyses and numerical simulations of such events have been limited. The objectives of this study are two-fold. First, an integrated experimental-computational approach is applied to study two forms of failures in porcine abdominal aorta specimens, quantifying the interfacial strength and critical energy release rate of the interface within the media via the CZM approach. The CZM method is validated by comparisons of the predicted loading-delamination-unloading curves and the predicted distances from the delamination front to the initial front with experimental measurements.

2. Materials and methods

2.1. Experimental procedure

The delamination experimental protocol in this study follows that used in our previous studies (Wang et al., 2013, 2014, 2011). One set of intact kidneys was obtained from the local slaughterhouse, rinsed in iced phosphate-buffered saline (PBS) solution and transported back to the laboratory. The abdominal aorta was isolated from the surrounding tissues, washed in PBS and dissected from the perivascular tissue. An approximately 30 mm long segment was cut, following a radial cut imposed onto the sample along the vessel axis, yielding a strip. Two groups of specimens oriented at the angle of 0° and 90°, respectively, with respect to the circumferential vessel axis were cut from porcine aortas (Fig. 1).

There are 12 specimens for each group. Six specimens oriented at the angle of 0° with respect to the circumferential vessel axis were used for the mixed-mode delamination experiment and the other six specimens were used for the “T” delamination experiment. These specimens are said to be oriented in the circumferential direction.

The same two types of experiments were also performed on twelve specimens oriented at an angle of 90° with respect to the circumferential vessel axis, as shown in Fig. 1. These specimens are said to be oriented in the axial direction.

In each case, to initiate a delamination process in the medial layer of the arterial wall, a small initial delamination (5 mm long) with a straight front was carefully introduced at one end of the specimen inside the medial layer.

In the “mixed-mode” experiment (see Fig. 3a and c), the bottom surface of the lower portion of the specimen was glued to a glass plate in order to restrict its motion during loading, and the proximal end of the upper delaminated portion was peeled away by a micro-clamp. During the delamination process, the delaminated upper portion became almost parallel to the lower portion and to the not-yet delaminated interface (Fig. 3c).

In the “T”-shaped delamination experiment (see Fig. 3b and d), the proximal end of one of the two initially separated portions of the specimen was fixed by tissue glue to a glass plate and the proximal end of the other separated portion was pulled away by a micro-clamp. During the delamination process, the two delaminated portions stay parallel to each other but are approximately perpendicular to the not-yet delaminated interface (Fig. 3d).

During each experiment, the prescribed displacement and reaction load were recorded via the system actuator and load cell (Bose ELF 3200, Biodynamic Co, MN). The delamination process was recorded by a computer vision system in which two cameras were perpendicularly positioned to get both front and side views of the specimen.

For each experiment, the recorded reaction load and the prescribed displacement data are presented in the form of a load vs. displacement curve (e.g. see Fig. 4). Each curve consists of several cycles, and the curve for each cycle contains a loading phase, a delamination phase, and an unloading phase.

3. Theoretical framework

3.1. HGO model

The HGO model assumes that collagen fibers are oriented parallel to the arterial wall at a certain angle with respect to the vessel axis (Gasser et al., 2006; Holzapfel et al., 2000). The mechanical response of the arterial wall at low strain is governed by the amorphous matrix, while as strain increases, two families of collagen fibers are gradually recruited and begin to take up the load, contributing to the highly nonlinear mechanical behavior of the arterial tissue. The strain energy potential per unit reference volume in a decoupled form is given by:

$$\Psi(\boldsymbol{C}, \boldsymbol{H}_1, \boldsymbol{H}_2) = \Psi_{vol}(J) + \overline{\Psi}(\overline{\boldsymbol{C}}, \boldsymbol{H}_1, \boldsymbol{H}_2) \tag{1}$$

where $\boldsymbol{C}$ is the right Cauchy-Green strain tensor and $\overline{\boldsymbol{C}}$ denotes a modified counterpart, $\overline{\boldsymbol{C}} = \overline{\boldsymbol{F}}^T\overline{\boldsymbol{F}}$; $\overline{\boldsymbol{F}} = J^{-1/3}\boldsymbol{F}$, $\boldsymbol{F}$ is the deformation gradient tensor and $J = \det(\boldsymbol{F})$. The volumetric part, $\Psi_{vol}(J)$, is given by (ABAQUS, 2013),

$$\Psi_{vol}(J) = \frac{1}{D}\left(\frac{J^2-1}{2} - InJ\right) \tag{2}$$

where $\frac{1}{D}$ is analogous to the elastic modulus of the material.

The free-energy function $\overline{\Psi}$ (Gasser et al., 2006), is expressed as

$$\overline{\Psi}(\overline{\boldsymbol{C}}, \boldsymbol{H}_1, \boldsymbol{H}_2) = \frac{\mu}{2}(\overline{I}_1-3) + \frac{k_1}{2k_2}[e^{k_2[\kappa\overline{I}_1+(1-3\kappa)\overline{I}_{41}-1]^2}-1] + \frac{k_1}{2k_2}[e^{k_2[\kappa\overline{I}_1+(1-3\kappa)\overline{I}_{42}-1]^2}-1] \tag{3}$$

where μ is a parameter having the dimension of stress and representing the shear modulus of the amorphous matrix; $\overline{I}_1 = tr(\overline{\boldsymbol{C}})$ is the first invariant of $\overline{\boldsymbol{C}}$; k_1 denotes the relative stiffness of fibers; k_2 is a dimensionless parameter; $\overline{I}_{41}$ and $\overline{I}_{42}$ are tensor invariants equal to the square of the stretch in the direction of two families of fibers, respectively; $\boldsymbol{I}$ is the identity tensor; and κ is the dispersion parameter, describing the dispersion of the two families of fibers. $\kappa = 0$ when the two collagen fiber families are parallel to each other and $\kappa = 1/3$ when the collagen fibers distribute isotropically; γ denotes the angle between the mean fiber orientation of one family of fibers and the circumferential direction of the aorta.

3.2. Interface damage model

In this study, the CZM approach is employed to represent the interfacial behavior and to characterize interfacial damage in order to model arterial delamination failure. An exponential cohesive traction-separation law is used, which is implemented in the commercial finite element software ABAQUS 6.13 (Dassault Systèmes, France) through a user-defined UEL subroutine. As shown in Fig. 2b, δ_n and δ_s denote the displacement jumps (separation) normal and tangent to the cohesive surfaces, respectively. The sliding displacement δ_s across the cohesive surfaces can be calculated in the form (Ortiz and Pandolfi, 1999)

$$\delta_s = \sqrt{\delta_{s1}^2 + \delta_{s2}^2} \tag{4}$$

where δ_{s1} (the shearing displacement) and δ_{s2} (the tearing displacement)