DISSECTING THE ROLE OF ELASTIN BIOMECHANICS USING THE FINITE ELEMENT METHOD

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Introduction

Connective tissue diseases, such as Marfan Syndrome (MFS), can lead to cardiovascular failure due to changes in the microstructure and -organization of soft tissues. It is characterized by aortic dilation and aneurysms, sometimes resulting in dissections, in addition to observable localized disruptions in the elastin lamellae—which play the primary load-bearing role in arteries at physiological pressures [1]. In previous ex-vivo studies, we have assessed the straightening of elastin lamellae in mouse carotid arteries at different intraluminal quasi-static pressures using propagation-based synchrotron imaging [2]. This study outlines a computational approach with the goal of mirroring the biomechanical behavior of the imaged mouse arteries at their corresponding intraluminal pressures using the Finite Element Method (FEM). We report the Diameter-Pressure curve from 0-240 mmHg and compare to [2].

Methods

Segmented synchrotron images of the carotid artery are discretized into a mesh. Elastin lamellae are modelled as T2D2 truss elements (light green in Fig.1 b-c) with thickness corresponding to lamellae in [2] and the fiber-dependent matrix (dark green in Fig.1 b-c) is discretized and modelled as C2D3 plane-stress elements in 2D. A static analysis using Abaqus/Standard is used, and the elements of the lamellae and matrix meshes are coupled using cubic distributing continuum coupling [3]. The elastin lamellae are defined as neo-Hookean materials while the underlying matrix was defined as an anisotropic Gasser-Ogden-Holzapfel (GOH) material model [4]. With the goal of replicating the Diameter/Pressure curve, the material properties are adjusted iteratively while making three key assumptions:

1. Out-of-plane (axial) components of fibers are not accounted for, and the radial direction corresponds to the local coordinate component which was assumed to be distributed along a linear curvature gradient between lamellae, where the circumferential directions of the fibers are parallel to the closest lamella.
2. We assumed the neo-Hookean component of the GOH model to be zero or negligible. As such, we assume the artery's isotropic response to arise from the interaction between lamellae and the underlying matrix.
3. Residual stresses are not taken into account in our model, and the compression of elastin, along with the tension in the adventitia is not accounted for.

Results and Discussion

The Diameter/Pressure curve, shown in Fig. 1 d shows that we can replicate the overall compliant characteristic of the artery using our method, with the dashed line representing data from our simulation falling between the variance of experimental diameters measured. We aim to integrate Residual Stress to our approach by applying loads that homogenize transmural circumferential stresses. Furthermore, added verification of this method will be done by comparing 3D segmented meshes to [2]. We hope to then use this method to gain a deeper understanding in the mechanical role of extent of elastin fragmentation/fenestration in aortic dissection.

References

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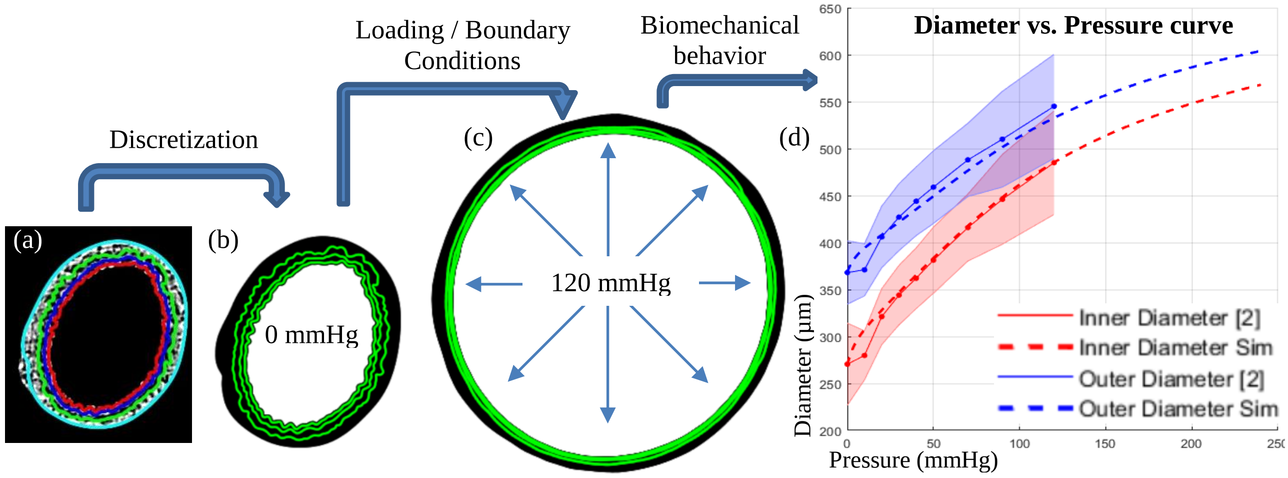

Figure 1 Illustration of our method in which we discretize segmentations of the arterial cross-sections , and apply the FEM on corresponding meshes