ELASTIN ANISOTROPY IN VASCULAR STRAIN ENERGY FUNCTIONS

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ABSTRACT

Vascular wall shows nonlinear anisotropic mechanical properties. The identification of a strain energy function (SEF) is the preferred method to describe its complex nonlinear elastic properties. None of the currently proposed constituent-based models succeeded in describing accurately results of multi-dimensional mechanical tests. We hypothesized that shortcomings of current models are partly due to unaccounted anisotropic properties of elastin. We performed inflation-extension tests on common carotid of rabbits before and after enzymatic degradation of elastin and applied constituent-based SEFs, with an isotropic and an anisotropic elastin part, on the experimental data. We used transmission electron microscopy (TEM) and serial block-face scanning electron microscopy (SBFSEM) to examine the arterial elastin structure. Our results show that constituent-based models with an anisotropic elastin part, characterize more accurately arterial biomechanics than models with an isotropic elastin. Furthermore, there is structural evidence behind the elastin anisotropy in arteries based on electron microscopy techniques.

KEY WORDS: elastin, anisotropy, rabbit, structural strain energy functions, electron microscopy.

1. INTRODUCTION

The vessel wall exhibits relatively strong nonlinear properties and undergoes wide range of deformations. These characteristics make the identification of a strain energy function (SEF), the preferred method to describe the complex nonlinear elastic properties of the vascular tissue. None of the currently proposed structural models succeeded in describing accurately and simultaneously both the pressure-radius (P-r_o) and pressure-longitudinal force (P-F_z) curves. We hypothesized that the shortcomings of current models are partly due to unaccounted anisotropic properties of elastin.

2. METHODS

2.1. Experimental Setup

A set of experimental data was obtained from inflation-extension tests on two groups of carotid arteries of rabbits in the totally passive state: a control group and an elastase treated group, as described by Fonck et al. [1]. This data (pressure-diameter, longitudinal force-pressure and zero-stress-state geometry) provided for a complete biaxial mechanical characterization of rabbit carotid artery in the presence and absence of elastin. The data was served as the basis for validating the applicability of the new biomechanical model of the vascular wall.

2.2. Transmission Electron Microscopy (TEM)

In order to look at the ultra-structure of the arterial elastin, an intact artery has been fixed at its in vivo longitudinal stretch (λ_z=1.4) and its mean physiological pressure (70 mmHg). The artery has been prepared for TEM and stained with palladium to enhance the elastin contrast. Circumferential and longitudinal sections have been acquired to give more detailed information on the elastin structure.

2.3. Serial Block-faced Scanning Electron Microscopy (SBF-SEM)

We used the same blocks prepared for the TEM. Image stacks were obtained using Serial block-face scanning electron microscopy SBFSEM developed by Denk and Horstmann [2] (Denk and Horstmann 2004). Images were done on three different sites in media at the accelerating voltage of 3.5keV and in the low vacuum mode (0.35 Torr). The section thickness was 50 nm and the obtained images were 2048x2048 pixels with 8.9 nm per pixel.

2.4. Theoretical Considerations

Our proposed SEF is based on the Zulliger et al.'s model [3]. The Zulliger et al.'s model consists of an isotopic part accounting for elastin fibres ψ_{elast} and an anisotropic part representing collagen fibers ψ_{coll}:

$$\psi = f_{elast}\ \psi_{elast} + f_{coll}\ \psi_{coll} \quad (1)$$

where f_{elast} and f_{coll} are fractions of elastin and collagen in the tissue.
We introduce an anisotropic SEF for elastin by considering a family of fibers in the circumferential direction embedded in a neo-Hookean matrix:

$$\Psi_{elast} = c^{i}_{elast}\,(I_1 - 3) + c^{a}_{elast}\,(I_4^{"} + \frac{2}{\sqrt{I_4^{"}}} - 3) \quad (2)$$

Where

$$I_4^{"} = e_\theta . C . e_\theta = \lambda_\theta^2 \quad (3)$$

λ_f is the stretch in the main elastin fiber direction, C is the right Cauchy-Green deformation tensor and e_f indicate the unit vector in the direction of fibers .C^{i}_{elast} represents the modulus for the isotropic elastin component and C^{a}_{elast} is an elastic constant for the anisotropic part of elastin. To test the model, experimental P-r_o and P-F_z curves of both artery groups were fitted simultaneously and the quality of fit was assessed.

3. RESULTS

When only P-r_o curves are fitted, both the anisotropic and the isotropic models gave excellent results. However, the original Zulliger et al.'s model with an isotropic part was not able to fit well both the P-r_o and P-F_z experimental data based on control arteries simultaneously (Figure 1). Further, when we applied the original model on elastase treated arteries, considering only the collagen part of the original model and in the absence of elastin, we obtained a good fit on both P-r_o and P-F_z sets of data, suggesting that the collagen part of the SEF is well enough defined to account for the role of collagen fibers in the absence of elastin (Figure 2). Finally, when the anisotropic model was applied to the entire set of data (control and elastase-treated), we obtained a much better fit as compared to the original isotropic mode.

The results from imaging show that elastin exists not only in the form of elastic lamellae (EL) but also elastic fibers are present in the interlaminal space (Figure 3). In circumferential sections, interlaminal elastin fibers (IEFs), normally seem to make a link between smooth muscle cells (SMCs) and the lamellae and are mostly in the same alignment with smooth muscle cells (Figure 3.b). In the longitudinal sections, we could mostly observe only the cross sections of the elastin fibers in the gap between SMCs and ELs. This suggests that elastin fibers are aligned with long axis of SMCs and therefore mostly oriented in circumferential direction. The results are in accordance with a recent work by O'Connell et al [4].

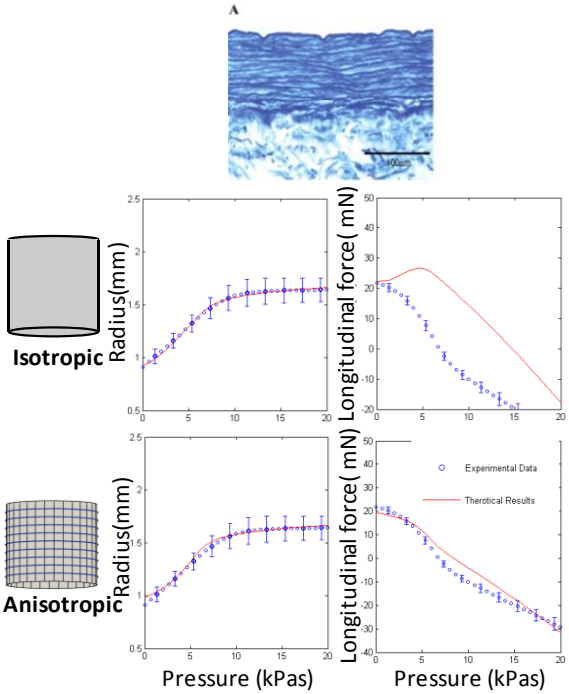


Figure 1. The isotropic and anisotropic models applied on the experimental data of intact arteries

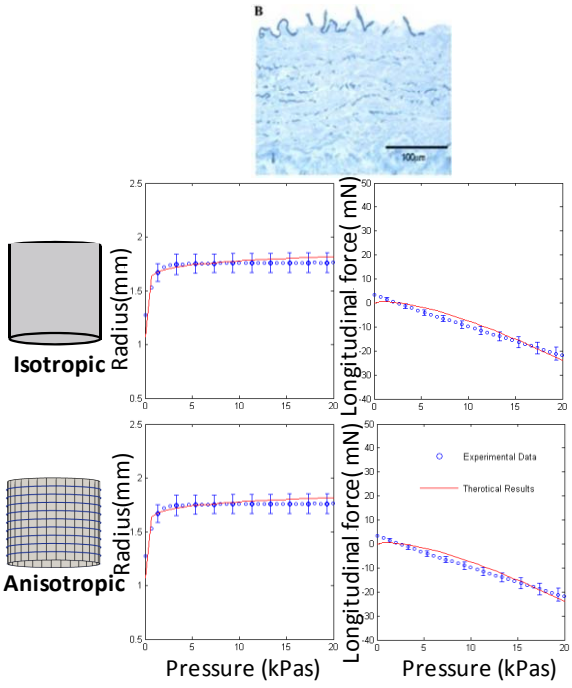
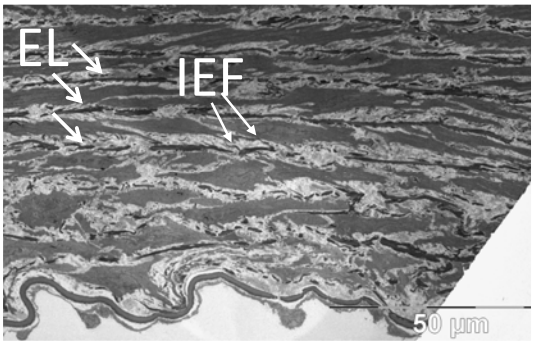
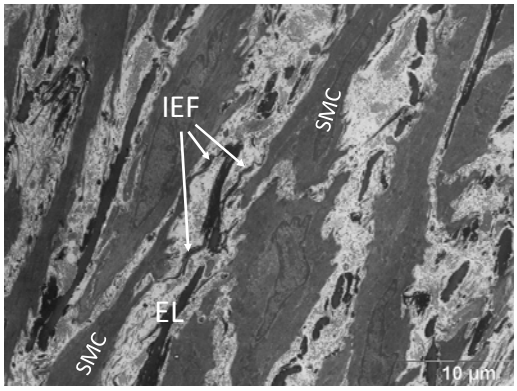


Figure 2. The isotropic and anisotropic models applied on the experimental data of elastase treated arterie



3.a



3.b

Figure 3. Cross section of a rabbit common carotid artery as seen by the transfer electron microscopy. IEF: interlaminal elastic fibers, El: elastic lamella , SMC: smooth muscle cell

4. CONCLUSION

We conclude that shortcomings of the isotropic model in fitting both the P-r_o and P-F_z experimental data lies in the identification of the SEF for elastin and that the SEF for the collagen component in Zulliger et al. is sufficient to give a 3D description of the arterial wall in the absence of elastin. The proposed anisotropic description of elastin seems to be in accordance with the ultrastructure of arterial elastin and provides a better characterization for the biomechanical response of the arterial wall, as compared to the isotropic model.

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