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Artery buckling analysis using a four-fiber wall model

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Abstract

Artery bent buckling has been suggested as a possible mechanism that leads to artery tortuosity, which is associated with aging, hypertension, atherosclerosis, and other pathological conditions. It is necessary to understand the relationship between microscopic wall structural changes and macroscopic artery buckling behavior. To this end, the objectives of this study were to develop arterial buckling equations using a microstructure-based 4-fiber reinforced wall model, and to simulate the effects of vessel wall microstructural changes on artery buckling. Our results showed that the critical pressure increased nonlinearly with the axial stretch ratio, and the 4-fiber model predicted higher critical buckling pressures than what the Fung model predicted. The buckling equation using the 4-fiber model captures the experimentally observed reduction of critical pressure induced by elastin degradation and collagen fiber orientation changes in the arterial wall. These results improve our understanding of arterial stability and its relationship to microscopic wall remodeling, and the model provides a useful tool for further studies.

Keywords

Mechanical instability; critical buckling pressure; artery buckling; fiber model; artery wall; extracellular matrix; elastin; collagen

Introduction

Arterial tortuosity often occurs in internal carotid artery, iliac artery, coronary artery, retinal artery, and small arterioles (Weibel and Fields 1965; Jakob et al. 1996; Amemiya and Bhutto 2001; Pries et al. 2009; Han 2012). It is associated with high blood pressure, aging,

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Conflict of interest

The authors have no conflict of interest.

atherosclerosis, and deficiency of extracellular matrix (ECM) components of the arterial wall (Dobrin and Canfield 1984; Del Corso et al. 1998; Pancera et al. 2000; Coucke et al. 2006; Thore et al. 2007). Understanding the mechanism of artery tortuosity is of clinical importance.

Recently, we have shown that arteries may lose stability and buckle into tortuous shapes (Han 2007; Han 2008; Han 2009a), and artery buckling has been suggested as a possible mechanism that leads to tortuosity (Han 2012; Han et al. 2013). Arterial buckling equations have been developed by modeling the artery as a thick-walled nonlinearly elastic cylinder using the phenomenology-based Fung exponential energy function (Han 2008; Han 2009a; Lee et al. 2012; Liu and Han 2012). Experimental results also demonstrated that artery buckling was affected by cellular functions and ECM changes (Lee et al. 2012; Hayman et al. 2013). Therefore, it is necessary to develop buckling equations that link the ECM microstructure to the critical buckling pressure.

Based on the microstructure of the arterial wall, Holzapfel and colleagues developed a constitutive equation that models the artery wall as a mixture of isotropic non-collagen matrix (elastin) reinforced by two families of anisotropic collagen fibers aligned helically (Holzapfel et al. 2000). This model was extended to a four-fiber model by adding two fiber families oriented in the axial and circumferential directions (Baek et al. 2007; Hu et al. 2007). Using this microstructure-based wall model in buckling analysis will enable us to link the critical buckling load to the microscopic extracellular matrix remodeling, which has been shown to modulate the mechanical stiffness and critical buckling pressure (Lee et al. 2012; Martinez and Han 2012).

Therefore, the objectives of this study were to develop and validate arterial buckling equations using 4-fiber model of the vessel wall and apply the model to illustrate the effects of wall structure on artery buckling.

Methods

Model equations

Artery wall is modeled as incompressible materials consisting of isotropic non-collagenous matrix (elastin) and four families of anisotropic collagen fibers aligning axially, circumferentially, and helically. The strain-energy function is given by (Baek et al. 2007; Hu et al. 2007)

$$W = \frac{b_0}{2}(I_1 - 3) + \sum_{k=1,2,3,4} \frac{b_{k1}}{4b_{k2}} \left\{ \exp \left[b_{k2} (\lambda_k^2 - 1)^2 \right] - 1 \right\} \quad (1)$$

where b_0 , b_{k1} , b_{k2} ($k=1,2,3,4$) are material constants; $I_1 = \text{tr}(\mathbf{F}^T \mathbf{F})$, $\mathbf{F} = \text{diag}(\lambda_r, \lambda_\theta, \lambda_z)$ is the deformation gradient, with λ_r , λ_θ , and λ_z being the stretch ratios in the radial, circumferential, and axial (longitudinal) directions, respectively; note that λ_z is uniform in the vessel and can be designated as a constant λ_{z0} ; λ_k represents the stretch ratio of the k th fiber family, $\lambda_k^2 = \lambda_\theta^2 \sin^2 \alpha_k + \lambda_z^2 \cos^2 \alpha_k$; α_k is the angle between the fiber and the axial direction of the artery, $\alpha_1 = 0^\circ$, $\alpha_2 = 90^\circ$, $\alpha_3 = -\alpha_4 = \alpha_0$. The 3rd and 4th fiber families are

assumed to have the same material properties due to axi-symmetry of arteries, i.e. $b_{31} = b_{41}$, $b_{32} = b_{42}$. Then, in terms of the radial, circumferential, and axial (longitudinal) Green strains E_r , E_θ , and E_z , equation (1) becomes:

$$W = b_0(E_\theta + E_z + E_r) + \frac{b_{11}}{4b_{12}} \{\exp(4b_{12}E_z^2) - 1\} + \frac{b_{21}}{4b_{22}} \{\exp(4b_{22}E_\theta^2) - 1\} + 2\frac{b_{31}}{4b_{32}} \left\{ \exp \left[4b_{32} \left(E_\theta \sin^2 \alpha_0 + E_z \cos^2 \alpha_0 \right)^2 \right] \right\} \quad (2)$$

Based on the equilibrium equation for cylindrical artery under lumen pressure and axial tension, the lumen pressure p and axial tension N can be expressed as (Fung 1993; Humphrey 2002; Han 2009a):

$$p = \int_{r_i}^{r_e} \left(\lambda^2 \frac{\partial W}{\partial E} - \lambda_r^2 \frac{\partial W}{\partial E_r} \right) \frac{d\xi}{\xi} \quad (3)$$

$$N = \pi \int_{r_i}^{r_e} \left(2\lambda_z^2 \frac{\partial W}{\partial E_z} - \lambda_r^2 \frac{\partial W}{\partial E_r} - \lambda^2 \frac{\partial W}{\partial E} \right) r dr + \pi r_i^2 p \quad (4)$$

where r_i and r_e are artery inner and outer radius, respectively.

Using the approach previously employed in developing the buckling equation (Han 2008; Han 2009a; Lee et al. 2012), we obtained the equation for determining the critical buckling pressure p_{cr} as (see Appendix for detailed derivation),

$$p_{cr} = \left[N + \left(\frac{\lambda_{z0}\pi}{l_e} \right)^2 H \right] / (\pi r_i^2) \quad (5)$$

where l_e is the equivalent length which equals half of the vessel length for an artery with fixed ends, and H is given by

$$H = \pi \int_{r_i}^{r_e} \left(J_z - \frac{1}{3} J_\theta \right) r^3 dr + \frac{\pi r_i^3}{3} \int_{r_i}^{r_e} J_\theta dr \quad (6a)$$

with

$$\begin{aligned} J_z &= 2b_0 + \left[4b_{11}E_z \exp(4b_{12}E_z^2) + 8b_{31}E_\alpha \exp(4b_{32}E_\alpha^2) \cos^2 \alpha_0 \right] + (1+2E_z) \left[16b_{11}b_{12}E_z^2 \exp(4b_{12}E_z^2) + 2b_{11} \exp(4b_{12}E_z^2) \right] \\ J_\theta &= (1+2E_\theta) \left[32b_{31}b_{32}E_\alpha^2 \exp(4b_{32}E_\alpha^2) \cos^2 \alpha_0 \sin^2 \alpha_0 + 4b_{31} \exp(4b_{32}E_\alpha^2) \cos^2 \alpha_0 \sin^2 \alpha_0 \right] \end{aligned} \quad (6b)$$

Inflation test and buckling test

Four porcine carotid arteries, harvested from farm pigs (B.W. 100–150kg) at a local slaughterhouse, were tested to validate model predictions. Pressurized inflation tests were conducted following a protocol previously described (Lee et al. 2012). Briefly, arteries were cannulated at both ends, one closed with a stopper and allowed to move freely, and the other connected to a pressure meter and a syringe pump filled with PBS. After preconditioning,

the arteries were gradually pressurized by the syringe pump, and the outer diameters and lengths of the arteries were recorded simultaneously and were measured offline using ImagePro. These data were fitted with Eqs. (3) and (4) to determine the material constants.

To determine the critical pressure, both ends of the arteries were tied to fixed cannulae inside a tissue chamber and perfused under steady flow (Han 2007; Han 2008; Lee et al. 2012; Liu and Han 2012). The arteries were stretched axially to designated ratios (1.0 to 1.7), and then the lumen pressure was gradually elevated until larger deflections in the arteries were observed. The critical buckling pressure was determined to be the pressure when the deflection was ~0.5 mm compared with the baseline position at zero pressure.

In addition, four arteries with buckling test at axial stretch ratio 1.0 to 1.6 in a previous study (Lee et al. 2012) were also analyzed with the 4-fiber model.

Model prediction and simulation

The critical buckling pressures of all arteries were estimated by solving Eq. (3) – (6) simultaneously through iterations using the material constants determined above.

In addition, we performed parametric studies to simulate the effect of elastin matrix and collagen fiber on arterial critical buckling pressure. Since parameter b_0 is related to the isotropic matrix (elastin) and our initial results showed that b_{31} , b_{32} and α_0 of helical fibers played a more dominant role than the circumferential or axial fiber (see Table 1), simulations were conducted for various values of b_0 , b_{31} , b_{32} and α_0 , respectively, while all other parameters were kept unchanged.

Effect of residual stress

To examine the effect of residual stress on artery buckling, the opening angle ($2\Theta_0$) was taken into consideration during the simulation for a group of 3 additional arteries (Chuong and Fung 1986; Han and Fung 1996; Lee et al. 2012). The circumferential stretch ratio at deformed state λ_θ was then given by $(\pi/\Theta_0)*(r/R)$. For comparison purpose, we first did the curve-fitting and calculation of the critical buckling pressures with measured opening angle $2\Theta_0$, then repeated the curve-fitting and buckling pressure calculation but with $\Theta_0=180^\circ$ (i.e. no residual stress).

Results

The material constants of the 4-fiber model were obtained for the eight arteries (Table 1). It was notable that the material constants of fiber families 1 (axial) and 2 (circumferential) were much smaller than those of helical fibers, indicating the dominant role of helical fibers in determining the anisotropic properties of the arterial wall. Figure 1 illustrated the fitting results of one artery.

The critical buckling pressures of these 8 arteries at various axial stretch ratios were estimated using the 4-fiber model, then compared with experimental measurements and with model predictions using Fung strain-energy function (Fig. 2). It is seen that the critical

buckling pressures predicted using the 4-fiber model were higher than the value predicted by Fung model, especially in the higher stretch ratio range (>1.5).

Parametric studies showed that a decrease of b_0 reduced the critical buckling pressure. Previous study has shown that elastase treatment removed 50–60% of elastin content in arteries (Lee et al. 2012). We simulated the critical buckling pressure change after elastin degradation by reducing b_0 alone 40% to 60% while keeping other material constants unchanged. The buckling pressures estimated (Fig. 3) matched well with experimental results of elastin degradation at 40% to 60% after elastase treatment. In addition, an increase in fiber angle α_0 , i.e. an alignment of collagen fibers closer toward the circumferential direction, decreased the critical buckling pressure of the artery (Fig. 4, top). An increase in b_{31} and b_{32} increased the buckling pressure, and vice versa (Fig. 4, bottom).

Residual stress (opening angle) affected the critical buckling pressures for the 3 arteries examined (Fig. 5)., Before elastase treatment, the opening angles were big ($\Theta_0=150\text{--}160^\circ$) and the critical buckling pressures were close to those calculated without the consideration of residual stress (where $\Theta_0=180^\circ$), and were close to experimental measurements. After elastase treatment, the opening angles became smaller and the critical buckling pressures showed big differences with those calculated with $\Theta_0=180^\circ$, and with experimental results. These results indicated that higher residual stress had a bigger effect on the critical pressure values.

Discussion

This study established the artery buckling equation using a 4-fiber reinforced arterial wall model. Our simulation results showed that the critical buckling pressure increased nonlinearly at higher stretch ratios. The changes in ECM structure and density affected the critical buckling pressure.

Noticed the dominant role of the helical fibers, we also determined the material constants and buckling pressures in these arteries using the 2-fiber model. The results were similar (data not shown), suggesting that the 4-fiber model could be simplified to the 2-fiber model for the simulations of porcine carotid arteries. The results of using the 4-fiber model are presented here without loss of generality.

Compared with previous studies using Fung's model (Han 2007; Han 2008; Lee et al. 2012; Liu and Han 2012), the advantage of using this 4-fiber model lies in its capability to link the microscopic elastin and collagen fiber changes (e.g. fraction of helical collagen fibers, and their orientation angle), due to various pathological conditions, to the overall mechanical instability of the arteries. Further studies are needed to quantify the predictive value of the 4-fiber model in ECM remodeling. The present study models elastin as an isotropic bulk material, while in a recently developed micro-structural model (Chen et al. 2011; Hollander et al. 2011) elastin was modeled as a network made of helical fibers in the media lamellae and thin inter-lamellae struts. This model was suggested to better relate the arterial wall structure to the material property, but further study is needed to investigate if this model would provide better estimations of critical buckling than the 4-fiber model.

The critical pressures predicted by using the 4-fiber model were consistently higher than those using Fung model and the experimental measurements, especially at high stretch ratios. This may be due to the drastic decrease of the helical fiber angle α_0 at high axial stretch ratios (>1.5). Our parametric study showed that a small variation of α_0 would lead to big changes in the material property constants and the critical pressure predicted (see Fig. 4a). At high axial stretch ratios (1.6 or 1.7), angle α_0 decreased drastically. That might cause the overestimation of critical pressure at high stretch ratios. However, this did not happen when using Fung's model. Further studies are needed to elucidate the underlying mechanism.

One limitation of this study is that our analysis simplified the arterial wall as a single homogenous layer. We did not use the two-layer wall model in our simulations due to lack of experimental data. The two-layer model needs to be investigated in the future. Another limitation of our study is that muscle contraction was ignored in the model analysis. It has been shown that smooth muscle contraction has significant effects on the critical buckling pressure (Hayman et al. 2013) and these effects need to be analyzed in future studies. Due to surrounding tissue tethering, the working length of arteries in vivo remains constant, as determined by the distance between bifurcation points and anatomical positions. The length normally changes very little when pressure changes. The length does change due to body movement (Han 2009a; Han 2012; Han et al. 2013), thus we simulated the buckling at various axial stretch ratios.

In conclusion, the present study develops a buckling equation using 4-fiber arterial wall modeling. This model improves our theoretical study and understanding of arterial stability under various conditions and its link to pathological changes in microstructure of the vessel wall.

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Appendix

Derivation of artery buckling equations using 4-fiber wall model

The Cauchy stress in arteries in the cylindrical coordinate (r, θ, z) can be expressed in terms of strain energy function W given in equation (2) as (Fung 1993; Humphrey 2002)

$$\sigma_i = \lambda_i^2 \frac{\partial W}{\partial E_i} + K, i=r, \theta, z \quad (A1)$$

where K is a Lagrangian multiplier for the incompressibility of the arterial wall.

The equilibrium equation for the axisymmetric cylindrical artery under lumen pressure is

$$\frac{\partial \sigma_r}{\partial r} + \frac{\sigma_r - \sigma_\theta}{r} = 0 \quad (\text{A2})$$

and boundary conditions are

$$\sigma_r(r_i) = -p; \sigma_r(r_e) = 0 \quad (\text{A3})$$

where p is the lumen pressure, r is the radial coordinate of a material point in the wall, and r_i and r_e are artery inner and outer radius, respectively. Integrating Eq. (A2) from r_e to r , and applying boundary conditions yield,

$$\sigma_r = \int_{r_e}^r (\sigma_\theta - \sigma_r) \frac{d\xi}{\xi} = \int_{r_e}^r (\lambda_\theta^2 \frac{\partial W}{\partial E_\theta} - \lambda_r^2 \frac{\partial W}{\partial E_r}) \frac{d\xi}{\xi} \quad (\text{A4a})$$

And combining this equation with equation (A1) gives,

$$\sigma_\theta = \lambda_\theta^2 \frac{\partial W}{\partial E_\theta} - \lambda_r^2 \frac{\partial W}{\partial E_r} + \int_{r_e}^r (\lambda_\theta^2 \frac{\partial W}{\partial E_\theta} - \lambda_r^2 \frac{\partial W}{\partial E_r}) \frac{d\xi}{\xi} \quad (\text{A4b})$$

$$\sigma_z = \lambda_z^2 \frac{\partial W}{\partial E_z} - \lambda_r^2 \frac{\partial W}{\partial E_r} + \int_{r_e}^r (\lambda_\theta^2 \frac{\partial W}{\partial E_\theta} - \lambda_r^2 \frac{\partial W}{\partial E_r}) \frac{d\xi}{\xi} \quad (\text{A4c})$$

Letting $r=r_i$ in equation (A4a) yields the lumen pressure p as:

$$p = \int_{r_i}^{r_e} (\lambda^2 \frac{\partial W}{\partial E} - \lambda_r^2 \frac{\partial W}{\partial E_r}) \frac{d\xi}{\xi} \quad (\text{A5})$$

And integrate the axial stress σ_z over the cross-sectional area yield the axial force N as (Fung 1993; Humphrey 2002; Han 2009a)

$$N = \int_A \sigma_z dA = \pi \int_{r_i}^{r_e} (2\lambda_z^2 \frac{\partial W}{\partial E_z} - \lambda_r^2 \frac{\partial W}{\partial E_r} - \lambda^2 \frac{\partial W}{\partial E}) r dr + \pi r_i^2 p \quad (\text{A6})$$

At buckling, the deflection curve of an artery depends on the boundary conditions, but the deflection u_c of a segment with an equivalent length l_e can be described by a half sine wave (Han 2009b)

$$u_c = C \sin\left(\frac{\pi z}{l_e}\right) \quad (\text{A7})$$

where C is a constant. The deflection yields an axial incremental strain (Han 2007; Han 2008; Han 2009a)

$$\Delta E_z = -\lambda_{z0}^2 \frac{\partial^2 u_c}{\partial z^2} r \cos \theta = \lambda_{z0}^2 \left(\frac{\pi}{l_e}\right)^2 \cdot C \cdot \sin\left(\frac{\pi z}{l_e}\right) r \cos \theta \quad (\text{A8})$$

The corresponding incremental stress σ_z can be obtained using equations (A4c):

$$\Delta \sigma_z = J_z \Delta E_z + \int_{re}^r J_\theta \Delta E_z \frac{d\xi}{\xi} \quad (\text{A9})$$

where λ_{z0} is the axial stretch ratio before buckling, (r, θ, z) denotes the cylindrical coordinates of a material point with reference to the center of the vessel lumen in the deformed state, and J_z and J_θ are given by

$$J_z = 2 \frac{\partial W}{\partial E_z} + (1+2E_z) \frac{\partial^2 W}{\partial E_z^2} - (1+2E_r) \frac{\partial^2 W}{\partial E_r \partial E_z} \quad J_\theta = (1+2E_\theta) \frac{\partial^2 W}{\partial E_\theta \partial E_z} - (1+2E_r) \frac{\partial^2 W}{\partial E_r \partial E_z} \quad (\text{A10})$$

Using Eq. (2), then J_z and J_θ for the 4-fiber model become

$$\begin{aligned} J_z &= 2b_0 + \left[4b_{11} E_z \exp(4b_{12} E_z^2) + 8b_{31} E_\alpha \exp(4b_{32} E_\alpha^2) \cos^2 \alpha_0 \right] + (1+2E_z) \left[16b_{11} b_{12} E_z^2 \exp(4b_{12} E_z^2) + 2b_{11} \exp(4b_{12} E_z^2) \right] \\ J_\theta &= (1+2E_\theta) \left[32b_{31} b_{32} E_\alpha^2 \exp(4b_{32} E_\alpha^2) \cos^2 \alpha_0 \sin^2 \alpha_0 + 4b_{31} \exp(4b_{32} E_\alpha^2) \cos^2 \alpha_0 \sin^2 \alpha_0 \right] \end{aligned} \quad (\text{A11})$$

and

$$E_\alpha = E_\theta \sin^2 \alpha_0 + E_z \cos^2 \alpha_0.$$

Accordingly, the bending moment is obtained by integrating the incremental axial stress σ_z over the cross section area A (Han 2008)

$$M = \int_A \Delta \sigma_z dA (r \cos \phi) = HC \left(\frac{\lambda_{z0} \pi}{l_e} \right)^2 \sin \left(\frac{\pi z}{l_e} \right) \quad (\text{A12})$$

with H given by

$$H = \pi \int_{ri}^{re} \left(J_z - \frac{1}{3} J_\theta \right) r^3 dr + \frac{\pi r_i^3}{3} \int_{ri}^{re} J_\theta dr \quad (\text{A13})$$

On the other hand, a lateral distributed load q generated by the internal pressure due to the buckling deflection u_c can be expressed as: (Han 2008; Han 2009b)

$$q = -p\pi r_i^2 \left(\frac{\partial^2 u_c}{\partial z^2} \right) = p\pi r_i^2 \left(\frac{\pi}{l_e} \right)^2 \cdot C \cdot \sin\left(\frac{\pi z}{l_e}\right) \quad (\text{A14})$$

Artery buckles when this distributed lateral load q and the axial tension N reach equilibrium. Based on the equation of equilibrium, the bending moment M can be expressed as:

$$M(z) = (-N + p\pi r_i^2)C \cdot \sin\left(\frac{\pi z}{l_e}\right) \quad (\text{A15})$$

Combining equations (A12) and (A15) yields the buckling equation (5) given in the text.

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Figure 1.

Deformation of a porcine carotid artery under lumen pressure. Axial stretch ratio (length change ratio) and circumferential stretch ratio (internal radius change ratio) plotted as functions of the luminal pressure. The markers are experimental data obtained from free-end inflation testing and solid curves are the fitting curves using the 4-fiber wall model.

Figure 2.

Comparison of critical buckling pressures of arteries from experimental measurement (○), 4-fiber model (solid line), and Fung's model (dash line) predictions. Critical pressures of top 4 arteries were from a previous study (Lee et al. 2012), and bottom 4 arteries were from the present study.

Figure 3.

The effect of elastin degradation on the critical buckling pressure in 2 representative arteries. The parameter b_0 was reduced 40% and 60% in the simulations for artery 1 (top) and artery 2 (bottom), respectively, while all other parameters were unchanged.

Figure 4.

The effect of collagen fiber angle α_0 (top) and parameter b_{31} (bottom) on the critical buckling pressure. All other parameters remained the same in all simulations.

Figure 5.

The effect of residual stress on the critical buckling pressure. Comparison of the critical buckling pressures of 3 arteries (from top to bottom) before (left) and after (right) elastase treatment, respectively. Solid curves and dotted curves are simulation results with and without considering opening angles (Θ_0) in the model, respectively. Square symbols are experimental data points.

Table 1

Material constants for 4-fiber model of porcine carotid arteries

Vessel	b_0 (kPa)	b_{11} (kPa)	b_{12}	b_{21} (kPa)	b_{22}	b_{31} (kPa)	b_{32}	α_0 (°)
1	1.00	2.5E - 08	0.044	2.37E - 08	1.0E - 10	6.13	0.21	50.38
2	4.33	1.0E - 10	1.0E - 10	1.0E - 10	1.0E - 10	3.45	0.50	48.09
3	2.00	1.0E - 10	0.078	1.0E - 10	1.0E - 10	4.69	0.31	54.39
4	2.37	1.0E - 10	1.0E - 10	2.8E - 09	0.027	10.02	0.28	52.12
5	1.85	4.8E - 07	1.0E - 07	1.0E - 07	1.0E - 07	9.30	1.38	56.30
6	9.47	1.0E - 10	0.98	2.91	0.78	2.38	1.08	52.48
7	7.89	1.5E - 09	1.0E - 11	3.85	1.0E - 11	2.51	3.12	58.43
8	1.0E - 10	13.38	2.6E - 04	36.98	1.0E - 10	6.25	3.53	65.47