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Buckling instability in arteries

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HIGHLIGHTS

- Incremental theory is applied to a residually stressed, two-layered anisotropic model
- The critical buckling load is found for an artery subject to axial stretch and internal pressure.
- Reduced opening angle and strain softening increase the likelihood of buckling.
- Competition between geometry and mechanics maygive rise to counter-intuitive results.

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ABSTRACT

Arteries can become tortuous in response to abnormal growth stimuli, genetic defects and aging. It is suggested that a buckling instability is a mechanism that might lead to artery tortuosity. Here, the buckling instability in arteries is studied by examining asymmetric modes of bifurcation of two-layer cylindrical structures that are residually stressed. These structures are loaded by an axial force, internal pressure and have nonlinear, anisotropic, hyperelastic responses to stresses. Strain-softening and reduced opening angle are shown to lower the critical internal pressure leading to buckling. In addition, the ratio of the media thickness to the adventitia thickness is shown to have a dramatic impact on arterial instability.

1. Introduction

The arterial system is a highly complicated and regulated mechanical system that must sustain variations in internal pressure due to blood flow and variations in axial stretch due to tethering to other tissues. The stability of arteries is essential to maintaining normal arterial function. Instability can occur in the form of vessel tortuosity (abnormal twists and turns) observed in vascular diseases. Tortuous arteries are seen in elderly patients with atherosclerosis and hypertension as well as in children suffering from connective tissue disorders such as Ehlers Donlos syndrome, Marfans syndrome and arterial tortuosity syndrome. Instability can lead to serious complications including stroke, vertigo, blackout, fainting, and persistent tinnitus (Weibel and Fields, 1965; Pancera et al., 2000; Aleksic et al., 2004). Some implications, including dissection and ischaemia (Zegers et al., 2007), can be life-threatening, yet the biomechanical basis of why arterial buckling occurs is not fully understood.

Axial strain has been shown to play a role in the pathogenesis of arterial tortuosity. During regular homeostatic functions, arteries are tethered to other tissues and they operate under a large stretch. Jackson et al. (2005) showed that partial unloading of axial tension can induce arterial tortuosity. More explicitly, they studied the relationship between the development of tortuosity and mechanical forces imposed on arterial tissue. The axial strain in rabbit carotid arteries was reduced from $62 \pm 2\%$ and maintained at $33 \pm 2\%$ for 12 weeks. After this time period, all vessels became tortuous due to tissue growth and remodeling, and they concluded that a substantial axial strain is necessary to sustain arterial stability.

In addition to axial strain, wall stiffness is also known to play a role in the mechanical stability of arteries (Fung, 1997). Collagen and elastin are important extracellular matrix components that determine the elasticity and strength of blood vessels. Studies have suggested that arterial tortuosity observed in connective tissue disorders is due to genetic defects in elastogenesis or collagen synthesis (Curran et al., 1993; Dietz et al., 1991; Loeys et al., 2004, 2005; Mátyás et al., 2006; Ewart et al., 1993; Cheng et al., 2009). In aging arteries, collagen degradation, deposition, and structural alteration can occur (Kratky et al., 1999). Elastin and collagen are the major load bearers in the arterial wall. Consequently elastin and collagen degradation weakens the mechanical strength of the arterial wall (Dobrin and Canfield, 1984; Kitoh et al., 1993). Experimental studies have shown that elastin and collagen degradation reduces the critical pressure (Lee et al., 2012; Martinez and Han, 2012). However, the composition of elastin and collagen affects the strain stiffening properties of the arterial wall,

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and the explicit role of strain-softening on buckling has not been explored previously. It is suggested that localized strain softening can occur in the arterial tissue of patients suffering from Marfan's syndrome (Haughton and Merodio, 2009). The role of material properties, including mechanical strength and strain stiffening properties, on arterial stability needs to be explored further.

The role of residual stress (stresses that persist in the absence of external tractions or body forces) in arterial stability is even less understood. Even in their rest length and in the absence of pressure, Bergel noted that arteries are still stressed (see Cardamone et al., 2009). The importance and existence of these residual stresses in arteries were revealed by the work of Fung and Vaishnav and Vossoughi in the 1980s (Fung, 1991, 1983; Vaishnav and Vossoughi, 1983, 1987). Residual stresses in arteries are demonstrated by slicing a ring from an artery and observing how this ring opens when a radial cut is made. The opening that results from the cut is the so-called "opening angle" measurement, a kinematic measure of residual strains (Fung, 2010). The stresses associated with the opening angle are known to play a fundamental role in regulating transmural stress gradient and lowering circumferential stress at the inner walls (Chaudhry et al., 1997; Humphrey, 2002). However, the role in mechanical stability is not understood. Hardening of the arterial wall in aged arteries can decrease the opening angle and may affect the stability of the vessel. Liu et al. (2014) considered the effect of residual stress through imposing an opening angle. However, a thorough sensitivity analysis was not performed to help us understand the mechanical effect of size of opening angle. In addition, the arteries were shown to have a higher likelihood of buckling when residual stress was incorporated in the model, which is contradictory to previous work on residually stressed cylindrical structures (Vandiver and Goriely, 2008; Goriely and Vandiver, 2010).

It is suggested that mechanical instability in arteries is a mechanism that may lead to tortuosity (Goriely and Vandiver, 2010; Han, 2012; Han et al., 2013). Buckling in particular is a possible type of instability. Han (2009) and Liu et al. (2014) developed an arterial buckling equation by modeling the artery as a homogeneous nonlinearly elastic cylinder using an anisotropic model. However, the critical pressures predicted were consistently higher than the experimental measurements, especially at high stretch ratios. One limitation of their model is the homogeneous-wall assumption. Arterial walls are composed of layers with different mechanical properties for the intima–media and adventitia layers (Fung, 2010). In addition, their buckling equation was derived from Bernoulli beam theory, which assumes that there is no shear deformation. Instead, using the theory of incremental deformations superposed on a known large deformation, the buckling instability can be recovered by a bifurcation argument, usually referred to, in the nonlinear elasticity theory, as incremental theory (Green et al., 1952). Although the computation is rather cumbersome, it could provide much information about the instability and the unstable modes selected in the bifurcation process.

The incremental theory has been used to study cylinders subjected to various types of loads. Haughton and Ogden (1979a,b) examined the bifurcations of both membrane tubes and thick-walled tubes subject to inflation and axial loading. More recently, Zhu et al. (2010) investigated the problem of the finite axisymmetric deformation of a thick-walled cylindrical elastic tube subject to pressure on its external lateral boundaries for an incompressible isotropic neo-Hookean material. Vandiver and Goriely (2008, 2009) and Goriely and Vandiver (2010) performed a bifurcation analysis on a two-layer model to study the role of differential growth and residual stress in the stability of plant stems and arteries.

In this paper we investigate asymmetric bifurcations from a circular cylindrical configuration of an artery subjected to combined axial stretch and internal pressure. We use a full three-dimensional nonlinear bifurcation analysis of inhomogeneous deformations to help us understand the role of opening angle, strain-stiffening, and geometry in arterial stability.

2. Model and methods

The arterial wall consists of three layers, each containing specific histological features: the intima (the innermost layer), the media, and the adventitia (the outer layer). The intima is known to have negligible (solid) mechanical contributions in healthy young arteries (Holzapfel et al., 2004) and therefore the arterial wall is approximated as a two-layer structure containing the media and adventitia. The artery is assumed to be a cylindrical tube of anisotropic material (see Section 2.2). To take into account residual stress (Chuong and Fung, 1986; Han and Fung, 1996), the artery is assumed to have an opening angle of $2\varphi_0$ (see Fig. 1). It is assumed that the open sector of the tube in $\mathcal{B}_0$ is stress-free, with an inner layer of radii A and B, an outer layer of radii B and C and length L. Before bifurcation, we consider a finite deformation from $\mathcal{B}_0$ to $\mathcal{B}_f$ in which the configuration in $\mathcal{B}_f$ is assumed to be cylindrical. Then, we will consider incremental solutions around the finite cylindrical deformation that do not respect the cylindrical symmetry.

2.1. Finite deformation

The cylindrical polar coordinates are given by (R,Θ,Z) in $\mathcal{B}_0$ and (r,θ,z) in $\mathcal{B}_f$. The deformation is described by

$$r=r(R),\quad \theta=\frac{\pi}{\pi-\varphi_0}\Theta,\quad z=\lambda_z Z, \tag{1}$$

where $A\le R\le C$, $-(\pi-\phi_0)\le\Theta\le\pi-\phi_0$ and $0\le Z\le L$. The deformation gradient tensor is $\mathbf{F}=\mathrm{diag}(\lambda_r,\lambda_\theta,\lambda_z)$, where λ_r, λ_θ and λ_z are the principal stretches given by

$$\lambda_r=\frac{\partial r}{\partial R},\quad \lambda_\theta=\frac{\pi r}{(\pi-\varphi_0)R},\quad \lambda_z=\lambda_z, \tag{2}$$

and the incompressibility condition is given by $\lambda_r\lambda_\theta\lambda_z=1$. Substituting the expressions from (2) into the incompressibility condition yields

$$\frac{\pi r\lambda_z}{(\pi-\varphi_0)R}\frac{\partial r}{\partial R}=1, \tag{3}$$

which can be integrated to obtain

$$r(R)=\left(a^2+\frac{(\pi-\varphi_0)}{\pi\lambda_z}(R^2-A^2)\right)^{1/2}. \tag{4}$$

The equilibrium equation in the straight axisymmetric artery is

$$\frac{dT_{rr}}{dr}+\frac{T_{rr}-T_{\theta\theta}}{r}=0, \tag{5}$$

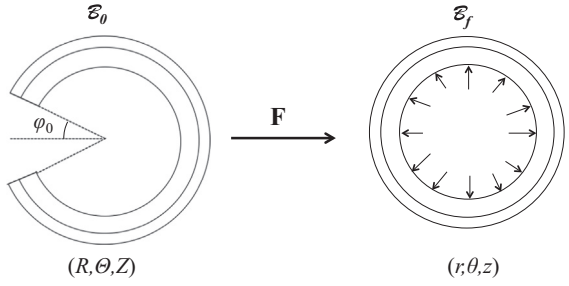

Fig. 1. Schematic of the geometry and deformation. An opening angle $2\varphi_0$ is assumed in the unstressed configuration. The tensor **F** describes the deformation from the unstressed artery to an artery with residual stress and subjected to an internal pressure.