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A Biomechanical Model of Artery Buckling

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Abstract

The stability of arteries under blood pressure load is essential to the maintenance of normal arterial function and the loss of stability can lead to tortuosity and kinking that are associated with significant clinical complications. However, mechanical analysis of arterial bent buckling is lacking. To address this issue, this paper presents a biomechanical model of arterial buckling. Using a linear elastic cylindrical arterial model, the mechanical equations for arterial buckling were developed and the critical buckling pressure was found to be a function of the wall stiffness (Young's modulus), arterial radius, length, wall thickness, and the axial strain. Both the model equations and experimental results demonstrated that the critical pressure is related to the axial strain. Arteries may buckle and become tortuous due to reduced (sub-physiological) axial strain, hypertensive pressure, and a weakened wall. These results are in accordance with, and provide a possible explanation to the clinical observations that these changes are the risk factors for arterial tortuosity and kinking. The current model is also applicable to veins and ureters.

Keywords

Artery buckling; Bent buckling; Stability; Critical pressure; Axial strain; Tortuosity; porcine

INTRODUCTION

Arteries are subjected to significant mechanical loads from internal blood flow and contiguous tissue tethering (Han and Fung 1995; Nichols and O'Rourke 1998). Both mechanical strength and stability are essential to normal arterial function. The study of arterial mechanics has established a rich body of knowledge concerning arterial wall constitutive equations, wall stress, strength, and the adaptation of the arterial wall in response to hemodynamic (pressure and flow) changes (Fung 1993; Langille 1996; Ku 1997; Nichols and O'Rourke 1998; Fisher et al. 2001). However, little research has been done to address the mechanical stability of the arteries. Although the cross-sectional collapse of arteries and veins due to low blood pressure has been examined and collapsible tube models of arteries and veins have been developed (Aoki and Ku 1993; Drzewiecki et al. 1997; Fung 1997; Tang et al. 2001), the biomechanical model for arterial bent buckling is lacking.

On the other hand, arterial tortuosity or kinking often occur in human internal carotid arteries or iliac arteries with significant clinical complications (Metz et al. 1961; Weibel and Fields 1965; Pancera et al. 2000; Dawson et al. 2002; Aleksic et al. 2004). For example, kinking of the internal carotid artery can lead to stroke, vertigo, syncope, blackout, persistent tinnitus,

and other cerebrovascular deficiencies (Weibel and Fields 1965; Pancera et al. 2000; Aleksic et al. 2004). Recent experimental results showed that reduced axial tension leads to artery tortuosity suggesting that the tortuosity may be due to mechanical buckling (Jackson et al. 2005). Therefore, it is important to develop the theory and applicable equations to predict the critical load for artery buckling.

Though arteries and engineering vessels, such as water pipes and gas tanks are all under internal pressure, their axial loads are different: arteries are under significant axial tension while engineering pressurized vessels are often under axial compression. While the mechanical buckling of pressurized engineering vessels has been studied extensively (Timoshenko and Gere 1961; Flugge 1973; Jones 1994), little has been done on the buckling of arteries.

The objectives of this study were to establish a biomechanical model for arterial bent buckling, to determine the critical buckling loads, and to determine the effect of axial stretch on the critical load.

METHODS

Mechanical model

Let's consider a simple case of an open-ended arterial segment with pinned support at both ends (both ends are free to rotate but restricted from lateral movement while one end is allowed to move axially). The artery is modeled as linear-elastic, thin-walled, circular cylinders under internal pressure p and axial (longitudinal) tension N with an axial elongation of stretch ratio λ_z . Arterial radius, wall thickness, and length under the pressure are designated as a , t , and L , respectively. All these parameters are assumed to be constants along the artery segment. Instead of solving the differential equations for shell buckling (Timoshenko and Gere 1961; Flugge 1973; Kollar and Dulacska 1984), we used a semi-inverse approach to establish the arterial buckling equation. This approach uses assumed deformation patterns to fit the boundary conditions and the equilibrium equations to find the solution (Ugural and Fenster 2003). Based on the Euler column buckling theory (Gere 2004) and our experimental observations of arterial bent buckling patterns, we assumed that the artery buckles into a sine shape. The central axis of the buckled artery deforms:

$$x_c = C \sin\left(\frac{\pi z}{L}\right), \quad (1)$$

where, C is a constant and z is the coordinate in the axial direction (Fig. 1). Accordingly, the displacement of the cylindrical arteries in the radial, circumferential, and axial (longitudinal) directions are given by:

$$\begin{aligned} u &= C \cos \phi \sin\left(\frac{\pi z}{L}\right) \\ v &= -C \sin \phi \sin\left(\frac{\pi z}{L}\right) \\ w &= -\frac{\pi a}{L} C \cos \phi \cos\left(\frac{\pi z}{L}\right) + (\lambda_z - 1)z \end{aligned} \quad (2)$$

wherein ϕ is the polar angle of the point from the x axis. The second term in the third equation for w represents the axial elongation of the artery due to the longitudinal tension that generated the stretch ratio λ_z . Thus the axial strain in the arterial wall generated by the buckling (bending) is given by:

$$\varepsilon_z = \frac{\partial w}{\partial z} = \frac{\pi^2 a}{L^2} C \cos \phi \sin\left(\frac{\pi z}{L}\right) + (\lambda_z - 1) \quad (3)$$

In the buckled arteries, the internal pressure generates an uneven lateral load that can be calculated based on the free-body diagram shown in Fig. 2. While the horizontal resultant of pressure load is zero due to symmetry, the vertical resultant equals to the integral of the vertical projection of the pressure load along the circumference. Therefore, the lateral load per unit length, $q(z)$ produced by the internal pressure p is

$$q(z) = pa \int_0^{2\pi} (1 + \varepsilon_z) \cos \phi d\phi \quad (4)$$

By taking Eq. (3) into (4), integrating to $d\phi$, and re-arranging, we have

$$q(z) = \frac{p\pi^3 a^2}{L^2} C \sin\left(\frac{\pi z}{L}\right) \quad (5)$$

The buckled arteries are under this distributed lateral load $q(z)$, axial tension N , and a restriction force Q_0 at the ends as shown in Fig. 3. All the loads applied to the artery are in equilibrium when the artery is buckled. Therefore, the bending moment $M(z)$ can be computed using the simple beam theory through the following two approaches that should give the same results.

First, the bending moment $M(z)$ at axial location z can be determined using the equilibrium equations for all the loads.

$$M(z) = Q_0 z - N \cdot C \cdot \sin\left(\frac{\pi z}{L}\right) - \int_0^z q(\xi) d\xi (z - \xi) \quad (6)$$

Wherein the lateral reaction force Q_0 caused by distributed load $q(z)$ is given by

$$Q_0 = \frac{1}{2} \int_0^L q(z) dz = \frac{p\pi^2 a^2}{L} C \quad (7)$$

Therefore, by taking equations (5) and (7) into equation (6) and integration, we have

$$M(z) = (-N + p\pi a^2) C \cdot \sin\left(\frac{\pi z}{L}\right) \quad (8)$$

On the other hand, the bending moment $M(z)$ and the normal force N in the axial direction can be obtained by integrating the stress over the cross-sectional area A .

$$M(z) = \int_A (E \varepsilon_z dA) (a \cos \phi) = E \frac{\pi^3 a^3 t}{L^2} C \cdot \sin\left(\frac{\pi z}{L}\right) \quad (9)$$

$$N = \int_A (E \varepsilon_z) dA = E(\lambda_z - 1) 2\pi a t \quad (10)$$

where E represents the Young's modulus of the wall.

By combining the two approaches above and comparing Eqs. (8)–(10), we obtain the critical pressure for artery buckling as

$$p_{cr} = E \frac{\pi^2 a t}{L^2} + 2E(\lambda_z - 1) \frac{t}{a} \quad (11)$$

Therefore, arteries buckle when internal pressure exceeds this critical pressure.

Experimental measurement

The relationship between the critical pressure and axial stretch ratio was tested using porcine carotid arteries. Carotid arteries were harvested from farm pigs (~6 months old and ~250 lb in body weight) at a local abattoir and transported to our lab in ice-cold phosphate-buffered saline solution (PBS). After the loose connective tissues were trimmed off, the arteries were checked for leaks and then tied at both ends onto cannulae inside a tissue chamber designed in our lab as previously described (Han et al. 2003; Han et al. 2006). The cannulae were connected to a pressure meter and a syringe pump. The arteries were stretched axially to achieve designated axial stretch ratios and were gradually pressurized with PBS using the syringe pump until significant buckling occurred. The loading procedures were recorded with a digital video camera. The critical pressure was determined as the pressure when the arterial deflection started to increase from the initial baseline and reached 1 mm.

To evaluate the accuracy of the proposed model, we used equation (11) to estimate the critical pressures for the tested arteries. The first estimation was made using a constant Young modulus of $E = 200$ kPa based on our previous measurements (Davis et al. 2005). The second estimation was made using the Young modulus estimated for the arteries based on the Laplace's law using the following equations:

$$\sigma = \frac{pa}{t} = E \left(\frac{\Delta a}{a} \right) \quad (12)$$

RESULTS

Theoretical studies were performed to determine the effects of dimensional parameters and axial stretch ratio on the critical buckling pressure. Three arteries were tested to measure the buckling pressure and validate the model predictions.

The effect of end restrictions

When the arteries are restrained differently, the critical load may change. The critical pressure of arteries with various end supports can be determined following the concept of “equivalent length” for Euler columns (Gere 2004). For example, when both ends are fixed against rotation, the buckled shape of an artery would become a full sine wave. Note that the deflection curve is symmetrical and has a zero slope at the midpoint and at both ends, the equivalent length for the arteries with two fixed ends is $L_e = 0.5L$. Therefore, the critical buckling pressure for arteries in general can be written as

$$p_{cr} = E \left[\pi^2 \left(\frac{a}{L_e} \right)^2 + 2(\lambda_z - 1) \right] \left(\frac{t}{a} \right) \quad (13)$$

For arteries *in vivo*, the ends are neither free to rotate nor locked in place but instead have a certain level of elastic restraint. So L_e would be in the range between $0.5L$ to L . Thus the critical pressures obtained with pin-supported ends and fixed ends provide estimations for the upper and lower limits of the critical pressure for different boundary conditions at the ends.

The effect of arterial length, radius, and wall thickness

The critical pressure is related to arterial dimensions including the arterial length, radius, and wall thickness. The effects can be evaluated by two non-dimensional parameters, slenderness ratio (L/a) and wall thickness to radius ratio (t/a), as given in equation (13). The relationship between the slenderness ratio, wall thickness to radius ratio, and the critical pressure are illustrated in Figure 4. It is seen that higher slenderness ratio reduces critical pressure while higher wall thickness to radius ratio increases the critical pressure.

The relationship between critical pressure and axial stretch ratio

The model equation (13) suggests a linear relation between the critical pressure and the axial stretch ratio (Figure 5). It is seen that for a given artery with given slenderness ratio and wall thickness to radius ratio, the critical pressure decreases when the axial stretch ratio decreases.

While it is evident that high pressure can cause arteries to buckle, it is important to note that arteries may buckle or become tortuous at normal physiological pressure due to reduced axial stretch ratio even when the arteries are under axial elongation ($\lambda_z > 1$). While reducing the axial stretch ratio in pressurized arteries to subphysiological levels lead to arterial buckling, increasing the axial stretch ratio will increase the stability of the artery. The critical axial stretch ratio of arteries, i.e. the minimum axial stretch ratio needed to maintain arterial stability can be determined from equation (13) as

$$(\lambda_z)_{cr} = 1 + \frac{p}{2E} \left(\frac{t}{a} \right) - \frac{\pi^2}{2} \left(\frac{L_e}{a} \right)^2 \quad (14)$$

This equation indicates that higher pressure, low material stiffness, larger slenderness ratio (longer segment), or smaller thickness to radius ratio (thinner wall) will increase the critical axial stretch ratio needed to maintain the stability of the artery (see Figure 6).

Note that the bending moment of inertia $I = \pi a^3 t$ for thin-walled cylinders, the first term in equation (11) represents the Euler critical buckling load of the cylinders $\frac{\pi^2 EI}{L^2}$ (Gere 2004).

Therefore, the first term $\frac{\pi^2 E}{(L_c/a)^2}(\frac{t}{a})$ in equation (13) is related to the “buckling strength” of the wall structure and the second term $2E(\lambda_2 - 1)(\frac{t}{a})$ is related to the “buckling strength” due to the axial tension. The relative contribution of the axial stretch to the critical pressure is

$$\% \text{Contribution of Axial Stretch to } p_{cr} = \frac{100\%}{\frac{\pi^2}{2}(\frac{a}{L_c})^2/(\lambda_z - 1) + 1} \quad (15)$$

It was found that the axial stretch in the physiological range of 1.3 to 1.6 contributes to 80–90% of the critical buckling load (Figure 7). Therefore, the axial stretch ratio is the dominating factor in preventing artery buckling.

Experimental Results and Comparison with Model Predictions

The critical pressures were experimentally measured from porcine carotid arteries at a series of axial stretch ratios. The results confirmed that the critical pressure is directly related to the axial stretch ratio, very close to a linear relationship in the tested range (Figure 8). The theoretical critical pressures were also computed with model equation (13) using the measured wall dimensions. Two theoretical estimations were obtained by using a constant Young’s modulus and modulus determined from equation (12), respectively. It seems that the modulus determined from equation (12) gave better estimations overall.

The slenderness ratio of these arteries was found to be in the range of 15–25 and the wall thickness to radius ratio was in the range of 0.1 to 0.2. These values support the selection of the parameters in the theoretical simulations (Figures 4–7).

DISCUSSION

This study established a biomechanical model of artery buckling and obtained a quantitative equation for determining the critical pressure and axial stretch ratio that lead to artery buckling. It was concluded that arteries may buckle and become tortuous due to high internal pressure even when the arteries are under axial elongation; the critical buckling pressure is proportional to the axial stretch ratio and wall stiffness (Young’s modulus). A certain level of axial stretch ratio is necessary to maintain the stability of the arteries and prevent them from buckling.

Clinical Relevance

Although there have been extensive studies on the clinical diagnosis and surgical treatment of artery tortuosity and kinking (Pancera et al. 2000; Dawson et al. 2002; Oliviero et al. 2003; Aleksic et al. 2004; Ballotta et al. 2005), the underlying biomechanical mechanism of artery kinking remains unclear. Our results demonstrated that arteries may buckle due to hypertensive pressure, reduced axial stretch, or changes in wall stiffness and dimensions. Buckled arteries could become tortuous or kinked, altering the wall stress distribution and disturbing or even disrupting the normal blood flow (Schep et al. 2002), thus leading to transitory ischemic attack to distal organs (Weibel and Fields 1965; Aleksic et al. 2004). It has been shown that cyclic bending of porcine femoral arteries affects the matrix and gene expression (Vorp et al. 1999) and is related to atherosclerosis (Stein et al. 1994). These changes would affect vascular function and lead to uneven wall remodeling that, in the long term, may lead to abnormalities in the arteries. In addition, our results are in agreement with, and provide a possible explanation to the clinical observations that artery tortuosity and kinking are associated with high blood

pressure, aging, atherosclerosis, and other pathological changes in the arteries (Del Corso et al. 1998; Pancera et al. 2000).

One of the new findings from our study is the significant effect of axial strain on the stability of arteries. A certain level of axial tension is essential in maintaining the stability of the arteries and preventing tortuosity, as experimental evidence has shown that tortuosity develops when the axial tension is reduced below the physiological level (Jackson et al. 2005). In addition, surgical observations showed that the redundant length of grafts could lead to kinking in vascular grafts which can be corrected by shortening the grafts to rebuild the axial tension (Han et al. 1998). Furthermore, surgical treatment to shorten the redundant arteries usually shows very positive outcomes in symptomatic patients with tortuous or kinked arteries (Zanetti et al. 1997; Fearn and McCollum 1998; Illuminati et al. 2003; Ballotta et al. 2005), indicating that increasing the axial stretch ratio increases arterial stability and prevents buckling.

Currently, the axial strain is not specifically controlled in vascular surgery but is set based on a surgeon's guess and experience, thus tortuosity and kinking occur sometimes when the axial strain is low. The current model equations will be a very useful tool to determine the critical axial strain needed in arteries and veins in vascular reconstructive surgeries, vascular grafting, and surgical treatment of kinked arteries in order to prevent tortuosity or kinking.

Model limitations

The limitations of the proposed model include the linear elastic and thin wall assumptions. Arteries demonstrated large nonlinear deformation under the internal pressure. However, stress and deformation caused by the initiation of buckling are small deviations from the pre-buckling stress and deformation. Thus, the deformation generated by buckling can be treated as incremental deformation with the incremental wall stiffness. Therefore, the incremental modulus should be used when using equation (13). On the other hand, as we know, Laplace's law and Poiseuille's law are widely used to estimate the tensile stress and lumen shear stress in arteries under blood flow (Fung 1993; Ku 1997; Humphrey 2002), although more advanced theories exist. The reason is that these laws are simple to use and the estimations are usually within reasonable accuracy. Similarly, the current buckling equation gives a reasonable estimation of the critical buckling pressure. Another limitation of the current model is that the effect of contiguous tissue tethering was ignored. Tissue tethering may affect the stability of the arteries and needs to be investigated in future studies.

Significance

The stability of living tissues and organs is important in maintaining their normal function. Understanding the biomechanics of artery kinking has wide implications in vascular physiology and pathology, as well as vascular surgery. The current results broaden our understanding of vascular biomechanics and shed light on the stability of arteries. Although the current study was focused on arteries, the biomechanical model developed and the approach used in developing the model can be useful for veins, ureters, vascular grafts, and other biological organs and organelles.

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Figure 1.

Schematics illustrating the deformations of a cylindrical artery (top) buckled under internal pressure and axial tension (middle). The radial and circumferential displacement of the wall, u and v , respectively, are the corresponding projections of the lateral deflection x_c of the longitudinal central axis (bottom panel). The longitudinal axis is denoted by the z axis. Solid lines represent the deformed shapes and the dotted lines represent the initial shapes.

Figure 2. Schematics showing a deformed segment of a buckled artery in lateral view (left) and cross-sectional view (right).

Figure 3.

The free-body diagram of a buckled artery with pin-supported ends. Q_0 represents the lateral reaction forces.

Figure 4.

Model predicted relationship between the critical pressure and the axial stretch ratio. It is seen that the critical pressure increases linearly with the increase of axial stretch ratio. The wall thickness to radius ratio was assumed to be 0.1 and Young's modulus was assumed to be 200 kPa.

Figure 5.

Top: Model predicted critical pressure plotted against the slenderness ratio L/a of arterial segments at a given wall thickness to radius ratio of 0.1. Bottom: Model predicted critical pressure plotted against the wall thickness to radius ratio t/a of arterial segment at a slenderness ratio of 10. The Young's modulus was assumed to be 200 kPa in both graphs.

Figure 6.

Top: Model predicted critical axial stretch ratio plotted against the slenderness ratio for arteries under normotensive and hypertensive pressures. The wall thickness to radius ratio was assumed as 0.1. Bottom: Model predicted critical axial stretch ratio plotted against the wall thickness to radius ratio for arteries under normotensive and hypertensive pressures. The slenderness ratio was assumed as 20. The Young's modulus was assumed to be 200 kPa in both graphs.

Figure 7.

Relative contribution (%) of the axial stretch to the total critical pressure plotted with the stretch ratio (see equation 15).

Figure 8.

Experimentally measured critical pressure plotted with the axial stretch ratio for three porcine carotid arteries (solid circles). The hollow triangles with short dash lines (Model, given E) are the model predicted values using a constant Young's modulus of 200 kPa while the hollow diamonds with long dash lines (Model, hoop E) are model predicted values using the Young's modulus determined with equation 12. See text for details.