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Effects of Geometric Variations on the Buckling of Arteries

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Abstract

Arteries often demonstrate geometric variations such as elliptic and eccentric cross sections, stenosis, and tapering along the longitudinal axis. Effects of these variations on the mechanical stability of the arterial wall have not been investigated. The objective of this study was to determine the buckling behavior of arteries with elliptic, eccentric, stenotic, and tapered cross sections. The arterial wall was modeled as a homogenous anisotropic nonlinear material. Finite element analysis was used to simulate the buckling process of these arteries under lumen pressure and axial stretch. Our results demonstrated that arteries with an oval cross section buckled in the short axis direction at lower critical pressures compared to circular arteries. Eccentric cross-sections, stenosis, and tapering also decreased the critical pressure. Stenosis led to dramatic pressure variations along the vessel and reduced the buckling pressure. In addition, tapering shifted the buckling deformation profile of the artery towards the distal end. We conclude that geometric variations reduce the critical pressure of arteries and thus make the arteries more prone to mechanical instability than circular cylindrical arteries. These results improve our understanding of the mechanical behavior of arteries.

Keywords

Artery; stability; buckling; tortuosity; tapering; elliptic cross section; eccentric cross section; stenosis; plaque; critical pressure; mode shape; wall stress; finite element analysis; anisotropic; strain energy function

INTRODUCTION

Arteries are under lumen pressure and axial tension *in vivo* (Han and Fung 1995; Nichols and O'Rourke 1998). Lumen pressure and axial tension make arteries mechanically stable by preventing collapse of the arterial lumen and bending or curling along the arterial axis (Aoki and Ku 1993; Fung 1993; Fung 1997; Pedley and Luo 1998; Han 2007; Wong et al. 2007). While collapse of the vessel lumen has been well documented, the bent buckling of tubular arteries was only reported very recently (Han 2007; Han 2008; Han 2009a; Rachev 2009; Cui and Zhang 2010). Artery buckling generates additional local wall stress and affects blood flow and normal functioning of arteries. Thus full understanding of the buckling behavior of arteries is very important for vascular studies.

Many arteries are not ideal circular cylinders, but instead have geometric variations such as oval or eccentric cross sections, initial curvature or tapering along the axial direction, or

stenosis due to plaque buildup in the lumen (Fung and Liu 1989; Han and Fung 1991; Aoki and Ku 1993; Han and Fung 1996; MacLean and Roach 1998; Fleischmann et al. 2001; Zeina et al. 2007; Han 2009b). While there is a fair understanding of how these variations affect the wall stress, it is unclear how they will affect the stability of arteries. For example, there have been extensive studies regarding plaque development, rupture, and stress analysis (Lee et al. 1993; Zheng et al. 2005; Li et al. 2007; Gao et al. 2009; Tan et al. 2009; Cui et al. 2010), but how stenotic plaques affects the stability of arteries and how mechanical buckling affects the stress in the plaque remain unclear. Therefore, it is necessary to understand the effect of geometric variations on the bent buckling of arteries.

It has been shown that atherosclerotic arteries with an eccentric cross section are more prone to lumen collapse than circular arteries (Aoki and Ku 1993), and our recent pilot study using an isotropic Mooney-Rivlin material model suggested that tapered arteries have lower critical pressures while those with oval cross sections have higher critical pressures compared to straight circular arteries (Northcutt et al. 2009). However, it is well known that the arterial wall is nonlinear and anisotropic. Hence, it is necessary to further study effects of geometric variations on artery buckling taking into account the anisotropy of the arterial wall.

The objective of this study was to investigate the effect of geometric variations on the critical pressure and deformation pattern of arteries with an anisotropic Fung strain energy function using finite element analysis.

METHODS

The effects of varying cross-section shapes, tapering along the vessel axis, and stenotic lumen plaque on the buckling behavior of the artery were examined through finite element analysis of model arteries.

Artery models

Models of tubular arteries with circular, oval, eccentric cross sections, tapering along vessel axis, and plaques in the lumen were generated using the commercial software SolidWorks®. Normal cylindrical arteries (controls) were generated based on a typical rabbit thoracic aorta which had a lumen diameter and wall thickness of 2.79 mm and 0.59 mm, respectively (Chuong and Fung 1984; Humphrey 2002). A length of 50 mm was used. Normal artery models with these dimensions were used as the baseline for creating all other geometrically altered artery models.

Oval arteries were created by reducing minor diameter D_2 while keeping the major diameter D_1 the same as normal cylindrical arteries (Figure 1A). The minor lumen diameters used were in the range of 1.40 mm to 2.79 mm that corresponded to minor to major diameter ratios in the range of $\frac{1}{2}$ to 1 (MacLean and Roach 1998).

Eccentric arteries were created by offsetting the center of the lumen and the outer wall by an offset distance δ (Figure 1B). The lumen and outer diameters of the artery were kept the same as the normal cylindrical arteries (2.79 mm and 3.97 mm, respectively). Two offset distances were designated to generate offset ratios, $\delta/r_i = 0.1$ and 0.23 (r_i is lumen radius) which represented the dimensions of a typical human artery (Aoki and Ku 1993).

Tapered arteries were created by reducing the lumen and outer diameters of the vessel linearly along its axis. The tapering level was described by

$$\alpha = \frac{D_{proximal} - D_{distal}}{2L} \quad (1)$$

where L , $D_{proximal}$ and D_{distal} are the axial length and the lumen diameters at the proximal and distal ends, respectively. Lumen diameters of 2.5 mm, 2.03 mm, and 0.81 mm were used for the distal end of arteries with an overall length of 50 mm, to generate a tapered angle of 0.16° , 0.43° , and 1.13° respectively, which simulated the tapering level in human left and right coronary arteries (Timmins et al. 2008). Two scenarios for the wall thickness were simulated: a constant wall thickness along the vessel and a linearly reduced wall thickness that kept the wall thickness to radius ratio ($t/r_i=0.43$) the same at the distal and proximal ends.

Stenotic arteries were created by addition of a local atherosclerotic plaque in the lumen of the normal cylindrical artery model. Various sizes and shapes of plaques were created including asymmetric and axis-symmetric plaques of various levels of stenosis and lengths (Figures 1D, 1E). Four levels of stenosis (20%, 50%, 75% or 90% reduction of lumen cross sectional areas) were created with plaque lengths of 16 mm and 20 mm, which were less than half of the total length of the model vessels.

Material Models

The arterial wall was modeled as a homogenous, incompressible, anisotropic nonlinear material with a Fung strain energy function in the form of (Fung 1993; Humphrey 2002):

$$w = \frac{c}{2} e^Q \quad (2)$$

with

$$Q = b_1 E_\theta^2 + b_2 E_z^2 + b_3 E_r^2 + 2b_4 E_\theta E_z + 2b_5 E_z E_r + 2b_6 E_\theta E_r \quad (3)$$

where b_1 , b_2 , b_3 , b_4 , b_5 , b_6 , and C are material constants. Values of material constants used were $b_1 = 1.0672$, $b_2 = 0.4775$, $b_3 = 0.0499$, $b_4 = 0.0903$, $b_5 = 0.0585$, $b_6 = 0.0042$, $C = 0.0224$ kPa (Chuong and Fung 1986).

The mechanical properties of atherosclerotic plaques are generally nonlinear and could be stiffer or softer than the arterial wall depending on the concentration of lipid and calcium contents (Lee et al. 1996). Accordingly, plaques were assumed to be homogenous and have a similar Fung strain energy function. While the material constants b_1 , b_2 , b_3 , b_4 , b_5 , and b_6 of the plaque were chosen to be the same as that of the arterial wall, the constant C was chosen as 0.00224, 0.0224, and 0.224 kPa to simulate plaques that range from 10 times softer, the same as, and 10 times stiffer than the arterial wall, respectively, since changing C directly changes the stress at given strains (Datir 2010).

To compare with our previous results and to illustrate the effect of wall material anisotropy, we also simulated the buckling behavior of normal cylindrical arteries with the same dimensions but with an isotropic nonlinear material of the Mooney-Rivlin strain energy function given in the form of:

$$w = C_1(I_1 - 3) + C_2(I_1 - 3)^2 + C_3(I_1 - 3)^3 \quad (4)$$

where I_1 is the first invariant of the wall stress, and C_1 , C_2 , and C_3 are material constants. We used the material constants $C_1 = 3.757$ kPa, $C_2 = -0.9931$ kPa, and $C_3 = 1.409$ kPa that

described the average of axial and circumferential behaviors for the same rabbit thoracic artery given in equation (3) for Fung strain energy function (Raghavan et al. 2004).

Loads and boundary conditions

Model arteries were subjected to lumen pressure and axial elongation. A uniform static lumen pressure was applied to all model arteries and a non-uniform pressure distribution along the lumen, obtained from the computational fluid dynamics analysis, was also applied in plaqued arteries. The artery was allowed to expand radially and circumferentially under lumen pressure with no side restraints. Both end surfaces of the artery were restrained from rotation but were allowed to expand radially (and circumferentially). Since arteries are under significant longitudinal strain *in vivo* (Han and Fung 1995; Nichols and O'Rourke 1998), axial stretch ratios of 1.0, 1.3, 1.5, and 1.7 were achieved by applying designated axial displacements to the distal end of the artery while the other end of the artery was fixed in the longitudinal direction. Specifically, the nodes at the end surfaces were allowed to move in the radial and circumferential directions, but only moved a given value in the axial direction to achieve the desired axial stretch ratio. This selection of boundary conditions best simulated the *in vivo* end restriction of the arteries, fully matched *in vitro* experimental conditions (Han 2007; Han 2009c; Martinez et al. 2010), and minimized the end effects on the stress and deformation of the arteries in FEA analysis (Datir 2010).

Since severe lumen stenosis affects the pressure distribution along the axis of the vessel (Ku et al. 1990), 3D computation fluid dynamics analysis was done using FLUENT to determine the pressure variation along the plaque surface for plaque arteries with 20%, 50% and 90% stenosis level. For this analysis, an inlet mass flow of 1.6 ml/sec and inlet pressure of 14 kPa was used as the boundary condition (Zeina et al. 2007). A density of 1025kg/m³ and a viscosity of 4 centipoise were used for the fluid and a convergence criterion of 0.0001 was used in the analysis. The Reynolds number was estimated to be approximately 1400. The resulting pressure distributions were then used in the buckling analysis of these stenotic arteries (Datir 2010).

Finite element analysis

All model arteries were meshed in ABAQUS using hybrid, three-dimensional tetrahedral elements (C3D4H). Fine meshes with 5 elements for every 1mm were used. Static General Analysis was carried out to determine the buckling pressure and mode shape at given axial stretch ratios. In order to facilitate the buckling analysis of arteries with normal cylindrical, tapered, and with eccentric cross section, a small imperfection in the form of an initial bend of 2 degrees was given to the central axis of the arteries. Our previous analysis demonstrated that a small initial curvature did not affect the critical pressure of arteries (Han 2009b). The lumen pressure was gradually increased to determine the critical pressure of buckling. The critical pressure was determined as the pressure when the deflection of the vessel axis reached a value of ~0.1 to 0.2 mm and the deflection starts to increase quickly with increasing pressure (i.e. the turning point of the deflection-pressure curve where a sudden increase in slope occurred). This approach also gave the deflection of the arteries post-buckling (Datir 2010).

For normal cylindrical arteries, the buckling pressures were obtained using both the isotropic Mooney-Rivlin strain energy function and the anisotropic Fung strain energy function. These results were compared with the critical pressure obtained using the theoretical buckling equations established by Han (Han 2008).

Experimental observation of the buckling pattern of tapered arteries—To experimentally validate the buckling mode shape of tapered arteries, we collected two

specimens of tapered porcine carotid arteries from a local slaughter house. The arteries (immediately distal to the sinus, ~60 mm in length) were attached to cannulae at both ends, stretched axially to a sub-physiological axial stretch ratio of 1.3, and gradually pressurized for buckling tests as previously described (Han 2007; Martinez et al. 2010). The lumen pressure was slowly increased to generate bent buckling and continued until a large deflection was reached.

RESULTS

We simulated the buckling behavior of arteries with various geometric variations including circular, eccentric, and oval cross sections, tapering, and stenotic plaque in the lumen using finite element analysis. The critical pressures, distributions of the von Mises stress in the arteries, as well as the deformed shapes were obtained.

Effect of arterial wall material on buckling of circular cylindrical arteries

The buckling behavior of circular cylindrical arteries was analyzed using both Mooney Rivlin and Fung strain energy functions. While both strain energy functions yielded similar deformation history and shape, the isotropic Mooney-Rivlin strain energy function overestimated the critical pressure compared to the Fung strain energy function (Figure 2). The numerical simulation results obtained using the Fung strain energy function matched well with the results obtained using the theoretical equations established by Han (Han 2008).

Effect of oval cross-section

Arteries with oval cross sections tended to buckle in the direction of the minor diameter axis and in general, buckling lead to an increase in von Mises stress on the convex side of the arteries (Figure 3A). Ovalness reduced the critical pressure of the arteries, however, when the minor to major diameter ratio was below 0.8, the critical pressure did not drop further. Instead, the critical pressure recovered with further reduction of the minor to major diameter ratio. The critical pressure was even higher than the control when the minor to major diameter ratio is below 0.5 (Figure 3B). To determine the possible underlying mechanism, we simulated the critical pressure of a series of cylindrical vessel of equal wall thickness but various lumen diameters. The results demonstrated that the critical pressure depended on the lumen diameter of the vessel in a non-monotonic fashion. While a decrease in diameter from the control value reduced the critical pressure, further reduction in lumen diameter increased the critical pressure (Figure 4). Simulations using a nonlinear theoretical model (Han 2008) confirmed the same trend. These results suggested that reduction in the mean diameter of the oval arteries may account for the initial decrease and subsequence increase in critical pressure when the minor diameter decreases.

Effect of eccentric cross-section

Arteries with eccentric cross sections buckled, either in the direction of the offset or in the opposite direction, at a critical pressure lower than that of normal concentric arteries (Figure 5A). The buckling pressure decreased with increased levels of the offset ratio (δ/r_i) at a stretch ratio of 1.3 and 1.5 (Figure 5B).

Effect of vessel tapering

Tapering made the buckling mode shape deviate from the sinusoidal shape with the peak deflection point shifted towards the distal end (Figure 6A). Buckling also led to an increase of von Mises stress on the convex side of the artery.

For tapered arteries with equal wall thickness to radius ratio (t/r_i) at both ends, tapering reduced the critical pressure of the artery compared to the control artery (Figure 6B). For the

tapered arteries with constant wall thickness from proximal end to distal end, the critical pressure was lower at the small tapering angle (0.16°), but increased at larger tapering angles (Figure 6C). Further simulations of cylindrical vessels using the cross-sectional dimension (radius and wall thickness) of the distal end of the tapered vessels demonstrated a similar trend (see Figure 4). For instance, a cylindrical vessel with a diameter equal to the diameter of the distal end of tapered arteries with a 0.16° tapering angle had a lower critical pressure, but cylindrical vessels with a diameter equal to the diameter of the distal end of tapered arteries with 0.43° and 1.13° angles had a higher critical pressure. These results suggested that the changes in critical pressure were due to the variations in average diameter.

In addition, experimental testing of two tapered porcine carotid arteries demonstrated a consistent pattern of shifting of buckled shape towards the distal end (Figure 7). A more pronounced deflection was observed post-buckling when the pressure was increased beyond the critical pressure. The observed buckling mode shape was consistent with the simulation results given above.

Effect of lumen plaques under uniform lumen pressure

Under uniform lumen pressure, stenotic plaques affect the critical pressure in a complex pattern depending on the level of stenosis, shape and stiffness of the plaques, and the axial stretch ratio in the artery. Arteries with asymmetric plaques tend to deflect to the opposite side of the vessel which resulted in a reduction in axial stress in the plaque region (Figure 8).

For arteries with symmetric plaque of the same stiffness as the arterial wall, the symmetric plaque increased the critical pressure compared to control arteries. Higher levels of stenosis led to higher critical pressures (Figure 9A). Buckling led to a higher stress in the plaque on the convex side of the artery. In contrast, asymmetric plaques reduced the critical pressure and a higher level of stenosis led to a lower critical pressure (Figure 9B).

At a given stenosis level, the critical pressure depended on the shape of the plaque and the axial stretch ratio. For example, at a 50% stenosis level, arteries with asymmetric plaques buckled at a lower pressure compared to arteries with symmetric plaques. A reversed trend is seen at a higher stretch ratio of 1.6 (Table 1).

Furthermore, our simulations showed that arteries with shorter plaque lengths had a lower critical pressure than arteries with longer plaque lengths. For arteries with asymmetric plaques of the same stiffness as the artery wall, the critical pressure was not affected by the axial length of the plaque at an axial stretch ratio of 1.3 (Figure 10). However, the critical pressure was lower in arteries with a 16 mm long plaque than in arteries with a 20 mm long plaque at a higher stretch ratio of 1.5.

Effect of lumen plaque under non-uniform pressure

Our computational fluid dynamic simulations demonstrated that blood flow in arteries generated a small gradual pressure drop along the axial direction and the presence of plaque made it non-uniform in the plaque region (Figure 11). Since the pressure distribution profiles along the axis are defined by the entrance pressure value, we used the entrance pressure value to represent the pressure pattern. When these non-uniform pressure distributions along the axis were applied, model vessels buckled at lower pressures indicated by entrance pressures that were lower than the critical values determined previously under uniform static pressure. Arteries with symmetric plaques of the same stiffness as the arterial wall buckled at lower pressures compared to arteries without plaque (cylindrical vessel under the linear decreasing axial pressure distribution), though the decrease was less at a higher level of stenosis (Figure 12A). Similarly, when a non-uniform pressure is applied to

arteries with asymmetric plaque of the same stiffness as that of the arterial wall, the arteries buckled at lower pressures compared to arteries without plaque (Figure 12B). Higher levels of stenosis led to lower critical pressures as well.

In addition, the stiffness of the plaque also affected the critical pressure. At a given symmetric stenosis level of 50%, arteries with a plaque 10 times softer than the arterial wall buckled at lower critical pressures while arteries with a plaque 10 times stiffer buckled at higher critical pressures compared to arteries with plaque of equal stiffness (Figure 13). Simulations done under uniform pressure also showed the same trend.

DISCUSSION

We studied the buckling of arteries with circular, oval, eccentric cross sections, and tapering, as well as plaque buildup using finite element analysis. Our results demonstrated that oval, eccentric, and tapered cross sections reduced the critical pressure of the artery compared to normal cylindrical arteries.

Our results showed that cylindrical arteries with the wall material modeled by a Mooney-Rivlin isotropic strain energy function predicted a higher critical pressure compared to the wall material modeled by a Fung anisotropic strain energy function. The difference was most likely due to the fact that material constants used in the Mooney-Rivlin strain energy function represented the average of the circumferential and axial stress strain curves thus overestimated the axial stiffness of the wall (Raghavan et al. 2004), while the Fung anisotropic strain energy function accurately reflected the fact that the wall is less stiff axially than circumferentially.

Another interesting finding was that tapering shifted the peak deflection point towards the distal end of the artery and decreased the critical pressure of the artery. In addition, our results also showed that arteries with asymmetric plaques were more prone to buckling than arteries with symmetric plaques. Though plaques may either increase or decrease the critical pressure depending on their dimensions and stiffness under the uniform pressure, plaques consistently reduced the critical pressure when the pressure variations due to stenosis were considered. Thus, we concluded that stenotic plaques will reduce the critical pressure of arteries.

Model parameters used in the simulations were within physical range. The data from (Han and Fung 1991) showed that the ovalness (minor to major diameter ratio) of pig aortas varied from 0.4 to 0.9. In our study, we modeled oval arteries with ovalness varying from 0.4 to 1. Angiographic data showed that human right coronary arteries tapered by 9% with a tapering angle of 0.43° (Timmins et al. 2008), both of these were included in our modeled tapered arteries. To model eccentric artery we used the same dimensions which were used by (Aoki and Ku 1993) representing dimensions of typical human arteries. From the photograph of the pig aorta (Han and Fung 1991) we calculated that the pig aorta was eccentric with $\delta/r_i=0.2$ which was within the range of simulations in our study. These previous studies demonstrated that the model parameters used in this study were reasonable. Additionally, the close match between results of cylindrical vessels from finite element simulations and previous theoretical models validated our FEA simulation results.

While the linear model predicted structure failure due to the undefined deflection at the critical pressure (Han 2007; Han 2009c), our analysis showed limited deflection in buckled arteries. This is due to the fact that the undefined deflection is true only within the linear small deformation domain. With geometric nonlinearity in the large deformation domain, the deflection is limited and the deflection only increases with continued increase of the

pressure load, similar to the buckling of beam-columns (Timoshenko and Gere 1963; Han 2009c).

Clinical relevance

Normal arteries are mechanically stable under physiological or even hypertensive pressure due to significant axial tension and surrounding tissue tethering. However, arteries may buckle under excessive pressure (Pancera et al. 2000; Han 2007). Arteries may also buckle under physiological pressure when the axial tension is reduced and/or the arterial wall is weakened due to elastin degradation because of aging or genetic defects (Nakamura et al. 2002; Jackson et al. 2005; Callewaert et al. 2008). Tortuous arteries can lead to stroke, cerebrovascular symptoms, and ischemic attacks (Del Corso et al. 1998; Pancera et al. 2000; Zegers et al. 2007; Illuminati et al. 2008). Mechanical buckling of the artery could be a possible reason for artery tortuosity (Han 2009a). Geometric variations reduce the critical pressure hence arteries with geometric variations are more vulnerable to instability (buckling) than cylindrical arteries.

It has been shown that human aortas are tapered from the thoracic to the abdominal (iliac bifurcation region), and tortuosity occurs mainly in the lower abdominal region (Nichols and O'Rourke 1998). Our model simulations, in combination with experiment results of porcine arteries, demonstrated the shift of the buckling shape/profile toward the distal end. The shift of the buckling shape to the distal end in tapered arteries provides an explanation for this phenomenon.

Atherosclerosis is often associated with tortuous arteries but the mechanism remains unclear (Del Corso et al. 1998). While previous computational fluid dynamics simulations demonstrated that vessel tortuosity may lead to local high shear and low shear regions along the tortuous vessel wall (Back et al. 1992; Smedby and Bergstrand 1996; Qiao et al. 2004; Wood et al. 2006; Liu et al. 2008), our current results suggest that development of stenotic plaques can also make the vessels prone to mechanical buckling and lead to tortuous shapes. While short exposure of mechanical buckling is recoverable once the pressure load is reduced or the factors that reduced the critical pressure of the arteries are removed, long-term exposure of buckling can lead to wall remodeling that may trigger atherosclerotic and other pathological changes due to sustained exposure to the altered flow and wall stresses. Combined, these lines of evidence suggest that the interactions between mechanical buckling and plaque build-up might be a mechanism for the association of artery tortuosity and atherosclerosis.

Limitations

A limitation of our current analysis is the assumption of the homogenous wall of the artery and homogenous material of plaque. The material properties should be considered as the average of the mechanical properties of the wall and plaques, respectively. Arterial walls have a layered structure with different mechanical properties for the intima, media, and adventitia layers (Fung 1993). The effect of these layered structures will be addressed in our future studies. Another limitation of this analysis is the negligence of the dynamic effect of pulsatile flow (Rachev 2009). While the effects of pulsatile flow need to be investigated in future studies, the current static model provided a reasonable estimation of the critical pressure (Han 2009a; Han 2009c) and was able to illustrate the effects of geometric variations. In fact, in small arteries and veins, the pressure is nearly steady and the static model can be applied directly (Lee and Han 2010; Martinez et al. 2010). The third limitation is the negligence of tissue tethering. The surrounding tissue support enhances the stability of the arterial wall and increases the critical buckling pressure. A detailed analysis of the

effects of tissue tethering on the mechanical stability of arteries can be found in one of our previous reports (Han 2009a).

While limitations exist, the current study of arteries under static pressure clearly demonstrated the effects of oval or eccentric cross sections, tapering, and lumen plaque on the buckling behavior of arteries. Inclusion of these variations better represents real arteries than the cylindrical models. These results help in understanding the mechanical behavior of blood vessels.

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Figure 1.

Schematic of models of arteries with an oval cross section (A), an eccentric cross section (B), tapered cross section (C), symmetric plaque (D), and asymmetric plaque (E).

Definitions of geometric parameters are illustrated. The plaque shape was assumed to be a spherical head with both cross sections and longitudinal counters as circular arcs. The dimensions are not proportional to scales.

Figure 2. Comparison of critical pressures of a rabbit artery obtained from FEA using a Mooney-Rivlin strain energy function, a Fung strain energy function, and from theoretical buckling equations (Han 2008) plotted as functions of the axial stretch ratio using the Fung strain energy function.

Figure 3.

Buckling of arteries with oval cross sections. (A) Top panel shows an artery with minor to major diameter ratios of 0.8 under a pressure of 0.9 kPa (before buckling) and 4.3 kPa (after buckling) at a stretch ratio of 1.3. Bottom panel shows an artery with minor to major diameter ratios of 0.4 under a pressure of 2.0 kPa (before buckling) and 7.5 kPa (after buckling) at a stretch ratio of 1.3. (B) Critical pressure plotted as a function of the level of ovalness (minor to major diameter ratio D_2/D_1).

Figure 4.

Variation of the critical pressure with the lumen diameter. The critical pressure of cylindrical vessels is plotted as a function of the lumen diameter at a constant wall thickness of 0.6 mm. Labels a, b, c, and d indicate the critical pressure of a cylindrical vessel of a lumen diameter of 1.01, 1.1, 1.393 and 2.786 mm, respectively.

Figure 5. Buckling shape and critical pressure of eccentric arteries. (A) Top panel shows an eccentric artery with $\delta/r_i = 0.1$ at lumen pressures of 1.2 kPa (before buckling) and 9.0 kPa (after buckling) at a stretch ratio 1.3. Bottom panel shows an eccentric artery with $\delta/r_i = 0.23$ at lumen pressures of 1.3 kPa (before buckling) and 6.0 kPa (after buckling) at a stretch ratio of 1.3. Arrows illustrate the direction of the deflection. (B) Critical pressure plotted as a function of the level of eccentricity (offset distance to inner radius ratio δ/r_i).

(A)

(B)

(C)

Figure 6.

Buckled shape and critical pressure of tapered arteries. (A). Top panel shows an artery with a 1.13° tapering angle at pressures of 1.9 kPa (pre buckling) and 8.4 kPa (post buckling). Bottom panel shows an artery with a 0.43° tapering angle at pressures of 3.5 kPa (pre buckling) and 7.2 kPa (post buckling). The axial stretch ratio is 1.3. (B) Critical pressure plotted as a function of the axial stretch ratio for tapered arteries of three different tapering angles. The wall thickness and lumen radius were assumed to decrease along the axis proportionally to maintain an equal wall thickness to radius ratio at the proximal and distal ends. (C) Critical pressure plotted as a function of the axial stretch ratio for tapered arteries of three different tapering angles. The wall thickness was assumed constant along the axis.

Figure 7.

Photographs of the buckled shape of a tapered porcine carotid artery. The artery has a tapering ratio of 0.78° (length of 65.3 mm, outer and inner diameter 7.6 mm, 5.2 mm and 5.8 mm, 3.4 mm at the proximal and distal ends, respectively) and was subjected to a lumen pressure and an axial stretch ratio of 1.3. The critical buckling pressure is 18.7kPa. Photographs were taken before buckling (top, at a pressure of 4 kPa,), at buckling (middle, at a pressure of 20 kPa), and post buckling (bottom, at a pressure of 24 kPa).

Figure 8. Pre- and post- buckling shapes and von Mises stress distributions (color coded) in stenotic arteries of different cross sectional plaque shapes. All plaques are at 50% stenosis level and the axial stretch ratio is 1.3. The pressures (pre- or post-buckling) are shown in kPa.

Figure 9.

Critical pressure plotted as a function of axial stretch ratio for arteries with symmetric stenotic plaques (A) and asymmetric stenotic plaques (B). The lumen pressure was assumed to be uniform and the plaque was assumed to have the same material properties as the artery wall.

Figure 10.

Comparison of the critical pressures of stenotic arteries of different plaque lengths (L_p). The lumen pressure was assumed to be uniform and the plaques were assumed to have the same material properties as the arterial wall.

Figure 11.

Comparison of lumen Pressure plotted as functions of the axial location for a control artery and for an artery with 50% asymmetric plaque at stretch ratio 1.3.

Figure 12.

Critical pressure plotted as a function of axial stretch ratio for arteries with symmetric stenotic plaques (A) and asymmetric stenotic plaques (B). The lumen pressure varies along the axis as determined from CFD simulation and the critical pressure is the entrance pressure at the proximal end (see text for details). The plaque was assumed to have the same material properties as the arterial wall.

Figure 13.

Critical pressure plotted as a function of axial stretch ratio for arteries with a 50% symmetric stenotic plaque that was 10 times softer or 10 times stiffer than the arterial wall as well as equally stiff as the arterial wall.

Table 1

Critical pressure of arteries with various shapes of stenotic plaque. The plaques were assumed to have the same material properties as the arterial wall. All plaques formed a 50% stenosis in the cross sectional area.

	Critical Pressure (kPa)	
Axial Stretch Ratio
1	0.12	0.42
1.3	3.05	3
1.5	10.32	12.45
1.6	12.04	22