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Journal of Biomechanics 40 (2007) 3672–3678

JOURNAL OF BIOMECHANICS

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A biomechanical model of artery buckling

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Accepted 18 June 2007

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 an 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 (subphysiological) axial strain, hypertensive pressure, and a weakened wall. These results are in accordance with, and provide a possible explanation to the clinical observations that hypertension and aging 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; Buckling equation

1. 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, syncopes, 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

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0021-9290/$ - see front matter © 2007 Elsevier Ltd. All rights reserved.
doi:10.1016/j.jbiomech.2007.06.018

(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.

2. Methods

2.1. Mechanical model

Let us 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:

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

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

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

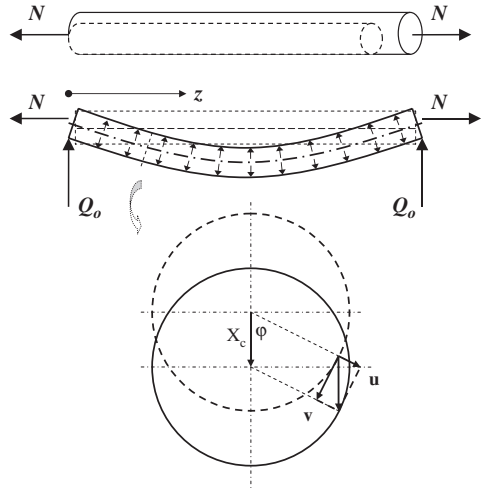

Fig. 1. Schematics illustrating the deformations of a cylindrical artery (top) buckled under internal pressure and axial tension (middle). The radial and circumferential displacement of the wall, u and v, respectively, are the corresponding projections of the lateral deflection x_c of the longitudinal central axis (bottom panel). The longitudinal axis is denoted by the z-axis. The solid lines represent the deformed shapes and the dotted lines represent the initial shapes.

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

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

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

$$q(z) = pa\int_0^{2\pi}(1+\varepsilon_z)\cos\varphi\,\mathrm{d}\varphi. \tag{4}$$

By substituting Eq. (3) into (4), integrating for $\mathrm{d}\varphi$, and re-arranging, we have

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

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:

$$M(z) = Q_0 z - NC\sin\left(\frac{\pi z}{L}\right) - \int_0^z q(\xi)\,\mathrm{d}\xi(z-\xi). \tag{6}$$

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

$$Q_0 = \frac{1}{2}\int_0^L q(z)\mathrm{d}z = \frac{p\pi^2 a^2}{L}C. \tag{7}$$

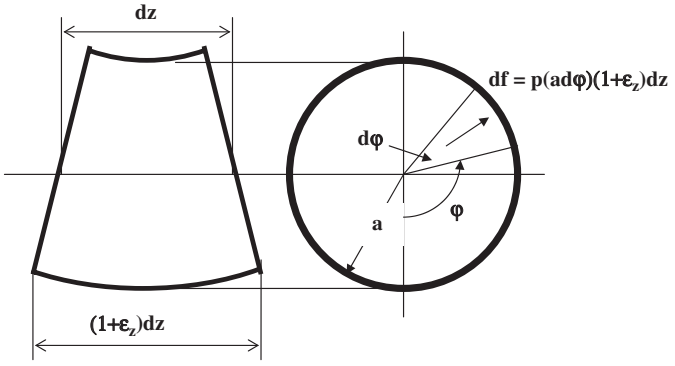

Fig. 2. Schematics showing a deformed segment of a buckled artery in the lateral view (left) and the cross-sectional view (right).

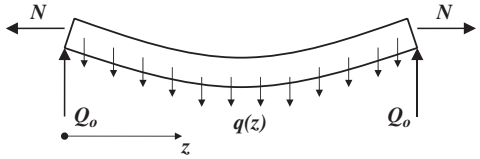

Fig. 3. The free-body diagram of a buckled artery with pin-supported ends. Q_0 represents the lateral reaction forces.