Available online at www.sciencedirect.com
ScienceDirect
Comput. Methods Appl. Mech. Engrg. 196 (2007) 3070–3078
Computer methods in applied mechanics and engineering
www.elsevier.com/locate/cma

Theory of small on large: Potential utility in computations of fluid–solid interactions in arteries

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Received 22 August 2005; received in revised form 28 April 2006; accepted 7 June 2006

Abstract

Recent advances in medical imaging, computational methods, and biomechanics promise to enable significant improvements in engineering-based decision making in vascular medicine, surgery, and training. To realize the potential of this approach, however, we must better synthesize the separate advances, particularly those in biofluid mechanics and arterial wall mechanics. In this paper, we describe a method for exploiting the typically small deformations experienced by arteries during the cardiac cycle while retaining essential features of the complex nonlinear, anisotropic behavior of the wall relative to unloaded configurations. In particular, we show that the well-known theory of small deformations superimposed on large can facilitate computations of fluid–solid interactions by exploiting methods familiar in linearized elasticity without compromising the description of the nonlinear wall mechanics. Indeed, the theory reveals potential errors when one simply tries to employ standard linearized results straight away. For purposes of illustration, small on large results are provided for the rabbit basilar artery and a constitutive relation for arteries recently proposed by Holzapfel and colleagues. It now remains for future studies to implement this approach in coupled fluid–solid problems.

PACS: 46.15.−x; 87.19.Rr

Keywords: Hemodynamics; Vascular mechanobiology; Wall stress; Vascular stiffness

1. Introduction

It is well known that hemodynamic loads on the vasculature play key roles in modulating normal geometry and properties as well as in contributing to the genesis and progression of vascular disease [1–3]. Indeed, blood flow induced wall shear stresses are key regulators of endothelial cell biology [4] just as blood pressure induced intramural stresses are key regulators of vascular smooth muscle cell and fibroblast biology [5]. Whereas advances in computational hemodynamics (e.g., [6]) and arterial wall mechanics (e.g., [7]) have been spectacular, there remains a need for computationally efficient and robust approaches that model the fluid–solid interactions. Indeed, as revealed in recent reviews [8–10], most computational biosolid mechanics studies ignore the dynamic loading imposed on the arterial wall by the blood whereas most computational biofluid mechanics studies either assume that the arterial wall is rigid or that it exhibits a linear elastic behavior. Because deformations of arterial walls are small during the cardiac cycle, the use of appropriately linearized elasticity in fluid–structure interaction problems can be justified, and in fact significant progress has been made in recent years in solving blood flow problems by assuming linear elastic behaviors of the wall (e.g., [11,12]). Nevertheless, there has not been much attempt to connect the linearized

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doi:10.1016/j.cma.2006.06.018

elastic response with complexities of the wall such as residual stress, anisotropy, and nonlinear behaviors that change with smooth muscle activation or growth and remodeling. There is, therefore, a pressing need for solutions for the inherent fluid–solid interactions that admit the dynamical loading and do not compromise the description of the actual wall mechanics. Only in this way will we be able to understand better, and thus address, the complexities of vascular biology, physiology, and pathophysiology.

In this paper, we suggest that the theory of small deformations superimposed on large can be exploited when solving coupled fluid–solid interaction problems. In particular, this approach allows one to include the effects of residual stress, nonlinear material behavior, anisotropy, smooth muscle contractility, and growth and remodeling of the arterial wall while recovering equations relevant throughout the cardiac cycle that can be solved using methods common to linearized elasticity. To illustrate this approach, we use a constitutive relation proposed by Holzapfel et al. [13] to fit passive data on the mechanical behavior of the rabbit basilar artery and a constitutive relation proposed by Rachev and Hayashi [14] to simulate the effects of changing smooth muscle tone. Using these nonlinear constitutive relations, we compute the linearized constitutive relation and study changes in the structural stiffness over the cardiac cycle. It is, of course, only the structural stiffness of the wall that affects the hemodynamic solutions.

2. General theory of small on large deformation

2.1. Preliminaries

Let the motion of a solid-like body $\mathscr{B}$ be represented by mappings χ of a particle from a reference configuration $\kappa_R(\mathscr{B})$ to a current configuration $\kappa(\mathscr{B})$ at time t, namely:

$$\mathbf{x} = \chi(\mathbf{X}, t), \tag{1}$$

where $\mathbf{X}$ and $\mathbf{x}$ are position vectors relative to reference and current configurations, respectively. Moreover, let the body occupy a configuration $\kappa_o(\mathscr{B})$ at an intermediate time t_o characterized by a large strain measured from the reference configuration. Then, let the position in the intermediate (stressed) configuration be denoted by $\mathbf{x}_o = \chi(\mathbf{X}, t_o)$. Hence, we can consider that a small displacement $\mathbf{u} = \mathbf{u}(\mathbf{x}_o, t)$, superimposed upon the large deformation, yields the "current" position $\mathbf{x}$ at time t. The current position can thus be written as

$$\mathbf{x} = \mathbf{x}_o + \mathbf{u}(\mathbf{x}_o, t). \tag{2}$$

Deformation gradients associated with mappings from the reference to the intermediate and current configurations are thus given by

$$\mathbf{F}^o = \frac{\partial \chi(\mathbf{X}, t_o)}{\partial \mathbf{X}}, \quad \mathbf{F} = \frac{\partial \chi(\mathbf{X}, t)}{\partial \mathbf{X}}. \tag{3}$$

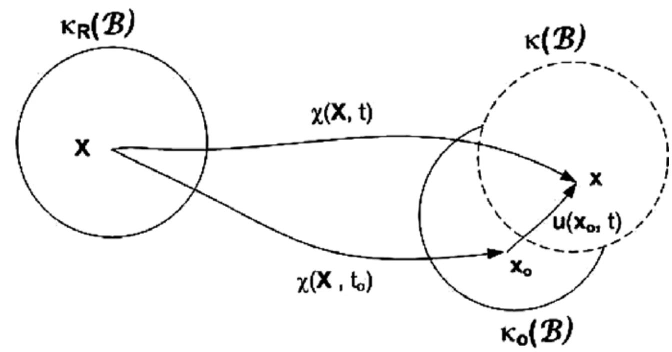

Fig. 1. Schematic view of the three primary configurations: $\kappa_R(\mathscr{B})$ is an unloaded reference, $\kappa_o(\mathscr{B})$ is a finitely deformed intermediate configuration, and $\kappa(\mathscr{B})$ is a "current" deformed configuration achieved via a small deformation from $\kappa_o(\mathscr{B})$.

The deformation gradient representing a mapping from the intermediate configuration to current configurations is similarly,

$$\mathbf{F}^* = \frac{\partial \mathbf{x}}{\partial \mathbf{x}_o} = \mathbf{I} + \mathbf{H}, \quad \text{where } \mathbf{H} = \frac{\partial \mathbf{u}}{\partial \mathbf{x}_o}. \tag{4}$$

The displacement gradient $\mathbf{H}$ can be divided into a symmetric part $\boldsymbol{\epsilon} = \frac{1}{2}(\mathbf{H} + \mathbf{H}^T)$ and a skew-symmetric part $\boldsymbol{\Omega} = \frac{1}{2}(\mathbf{H} - \mathbf{H}^T)$. If $\mathbf{H}$ is small, $\boldsymbol{\epsilon}$ and $\boldsymbol{\Omega}$ are identified as the infinitesimal strain and infinitesimal rotation, respectively. Regardless, gradients of the successive motions (Fig. 1) are

$$\mathbf{F} = \mathbf{F}^* \mathbf{F}^o. \tag{5}$$

For an isochoric motion, the material is subject to a kinematic constraint: $\det \mathbf{F} = 1$ in general, which reduces to $\mathrm{tr}(\boldsymbol{\epsilon}) = 0$ for an infinitesimal strain. The Cauchy stress $\mathbf{T}$ for an incompressible Green (hyper)elastic material can be written as

$$\mathbf{T} = -p\mathbf{I} + \hat{\mathbf{T}}, \quad \hat{\mathbf{T}} = \mathbf{F}\hat{\mathbf{S}}\mathbf{F}^T, \quad \hat{\mathbf{S}} = 2\frac{\partial \hat{W}}{\partial \mathbf{C}}, \tag{6}$$

where p is a Lagrange multiplier that enforces the isochoric motion, $\mathbf{C} = \mathbf{F}^T\mathbf{F}$ is the total right Cauchy–Green tensor, and $\hat{\mathbf{T}}$ is the deformation-dependent (or extra) part of the Cauchy stress. For purposes herein, it is convenient to relate $\hat{\mathbf{T}}$ to the extra part of the second Piola–Kirchhoff stress $\hat{\mathbf{S}}$, which in turn is computed directly from a stored energy function $W = \tilde{W}(\mathbf{F})$, or by material frame indifference, $W = \hat{W}(\mathbf{C})$.

At this juncture, we recognize that although complete analyses of arterial wall mechanics necessarily require $\mathbf{F}$ to be computed relative to a suitable reference configuration (e.g., a residual stress free sector obtained by introducing multiple cuts in an excised segment [5,15]), the focus of most computational biofluid mechanical analyses is on changes from diastole to systole. Because of the highly nonlinear material behavior of arteries, the deformation from a suitable reference configuration to an intact diastolic configuration is "large" whereas that from diastolic to