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A novel non-invasive ultrasonic method to assess total axial stress of the common carotid artery wall in healthy and atherosclerotic men

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ABSTRACT

In the present study, developing a new non-invasive method independent from blood flow, we estimated and compared the total axial stress of the common carotid artery wall in healthy and atherosclerotic subjects. Consecutive ultrasonic images of the common carotid artery of 48 male subjects including healthy, with less and more than 50% stenosis in carotid artery were recorded. Longitudinal displacement and acceleration was extracted from ultrasonic image processing using a block matching algorithm. Furthermore, images were examined using a maximum gradient algorithm and time rate changes of the internal diameter and intima-media thickness were extracted. Finally, axial stress was estimated using an appropriate constitutive equation. Statistical analysis results showed that with stenosis initiation and its progression, axial acceleration and stress increase significantly. According to the results of the present study, maximum axial stress of the arterial wall is 1.713 ± 0.546, 1.993 ± 0.731 and 2.610 ± 0.603 (kPa) in normal, with less and more than 50% stenosis in carotid artery respectively. Whereas minimum axial stress is -1.714 ± 0.676, -1.982 ± 0.663 and -2.593 ± 0.661 (kPa) in normal, with less and more than 50% stenosis in carotid artery respectively. Moreover, internal diameter and intima-media thickness of the artery also increase significantly with stenosis initiation and its progression. In this study, the feasibility of axial wall stress computation for human common carotid arteries based on non-invasive in vivo clinical data is concluded. We found a strong and graded association between axial stress and severity of carotid stenosis, which might be used to discriminate healthy from atherosclerotic arteries.

1. Introduction

The mechanical properties of the arterial wall are of great importance and have the potential to provide significant contributions for clinical cardiovascular issues such as the assessment of plaque vulnerability and the mechanical optimization of balloon angioplasty and stent deployment. The clinical significance of arterial wall mechanics, however, is limited due to the lack of appropriate constitutive equations of human arteries, which are fundamental prerequisites for analytical and computational stress–strain analyses (Schulze-Bauer and Holzapfel, 2003). Since atherosclerosis, the major cause of human mortality in the world, is a complex trait closely associated with multiple risk factors including hypertension, hyperlipidemia, and diabetes mellitus, mechanical forces could change in response to these risk factors (Jiang et al., 2000). Mechanical stresses of the arterial wall have been shown to participate in the pathogenesis of atherosclerosis as local factors (Cunningham and Gotlieb, 2005). Axial stress and stretch are the most important hemodynamic forces which exist in addition to the radial compressive pressure.

Numerous studies have formulated constitutive relations to describe the mechanical behavior of arteries and to compute the associated wall stresses that influence the mechanobiology (Masson et al., 2008). Three stresses—flow induced wall shear, intramural circumferential and axial—dominate the mechanical behavior (Fig. 1) and therefore regulate the three primary geometric variables of importance: luminal radius, wall thickness, and axial length (Holzampfel and Gasser, 2000). Arteries in the human body are constantly moving due to the mechanical stresses they are subjected to. In essence, blood pressure, blood flow and tethering to the surrounding tissue cause stresses on the arterial wall resulting in its motion. Stresses can result in movement and strains in three directions (Golemati et al., 2003). This motion may be responsible for tissue rupture and cerebrovascular symptoms.

Nevertheless, most attention in the literature has been focused on roles of wall shear stress and circumferential wall stress and numerous studies have been carried out to understand the

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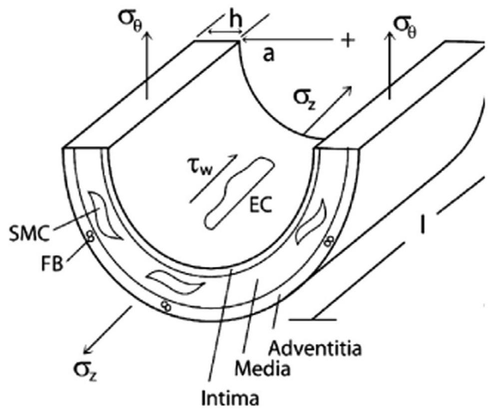

Fig. 1. Schema of the three primary stresses that act on the arterial wall (radius *a*, thickness *h*, and length *l*). These stresses are wall shear (τ_w) and intramural circumferential ($\sigma_{\theta\theta}$) and axial (σ_{zz}) stresses. Arterial wall consists of three layers (from the inner to outer wall—intima, media, and adventitia, respectively) occupied by three cell types respectively (endothelial—EC, smooth muscle—SMC, and fibroblast—FB) (Humphrey et al., 2009).

changes of wall shear stress and circumferential wall stress with age, atherosclerosis, hypertension and along the arterial tree (Stroev et al., 2007; Oshinski et al., 2006; Katritsis et al., 2007; Samijo et al., 1998; Bussy et al., 2000; Girerd et al., 1998). Whereas it has been known for more than a century that arteries also experience significant axial stretches (and stresses) in vivo (Humphrey et al., 2009). However, less attention has been paid to this aspect of the biomechanics, which is perhaps due to the lack of a direct noninvasive method of in vivo measurement. Patel and Fry (1966) measured small longitudinal movement of arterial wall and emphasized the importance of longitudinal tethering of arteries. Tozzi et al. (2001) reported the length reduction in the common carotid artery of pigs. Sunagawa et al. (2000) measured the longitudinal movement of the arterial wall using the Doppler method. Persson et al. (2002) and Cinthio et al. (2006, 2005), using a non-invasive ultrasonic method based on block matching algorithm, demonstrated that during cardiac cycle there is a distinct longitudinal displacement of the human arterial wall of the same magnitude as the diameter changes. It should be noted that using ultrasound, motion could be estimated with block matching algorithm by comparing the patterns in a small region with a searching block in the consecutive frames (Golemati et al., 2003).

Larsson et al. (2011, 2015) proposed a speckle tracking (ST) algorithm for estimating wall strains (radial, longitudinal and circumferential) in a phantom mimicking carotid artery. They used the sonomicrometry results as reference for ST and found high correlation between ST based mean peak strains and those of reference measurements.

Larsson et al. (2010) estimated the longitudinal strain in common carotid artery of five sheep via ST algorithm and compared to corresponding values resulted from sonomicrometry. They found a high correlation (r=0.95) between the results of the two methods.

Idzenga et al. (2012) extracted the adventitia shear strain induced by the pulsatile blood pressure in 16 asymptomatic subjects using RF ultrasound. They found a significant correlation between time occurrence of maximum shear strain and the time of dicrotic notch in the distension waveform.

Svedlund and Ming evaluated the feasibility of the velocity vector imaging (VVI) method in extracting 2D motion of the common carotid artery wall in healthy subjects and subjects with coronary artery disease. Results of their study showed that there is no significant difference between the longitudinal movement of the right and left carotid arteries, or between that of the near and far walls. But the total longitudinal displacement (sum of absolute values of maximum systolic plus the maximal diastolic displacements) averaged over a 1 cm segment of artery wall is significantly different between the groups (Svedlund and Ming, 2011).

Based on the knowledge of the longitudinal movement of the arterial wall, it seems that new information on the mechanical properties, particularly mechanical forces, their type and pattern acting on the vessel wall, might be achieved. Besides, recent studies showed that the longitudinal motion waveform of arterial wall could be obtained from consecutive ultrasonic images. Therefore, given that longitudinal motion equation is achievable by curve fitting on the motion waveform, the axial force (hence, the axial stress) could be assessed by differentiating the motion equation. In this study, we took a step forward, by using the derivatives of the longitudinal motion, proposed, and implemented a novel method for estimating the mean axial stress acting on the artery wall. The proposed approach is designed to calculate the axial stress of the common carotid artery wall in healthy and atherosclerotic subjects.

2. Methods

2.1. Basic equations

Considering the artery as an incompressible cylindrical tube subjected to various loads, with end effects ignored, and in the absence of inertial and body forces, the equilibrium equations are

$$\operatorname{div}(\sigma) = 0 \tag{1}$$

where div(●) denotes the spatial divergence of the spatial tensor field (●). Note that in cylindrical polar coordinates (r, θ, z), because of the geometrical and constitutive symmetry, Cauchy stress components must be independent of θ and z and are expected to be functions of r alone. The only non-trivial component of (1) is

$$\frac{d\sigma_{rr}}{dr} + \frac{\sigma_{rr} - \sigma_{\theta\theta}}{r} = 0 \tag{2}$$

The inner wall of the artery is subjected to an internal pressure P, the outer wall is free of loads, and tethering results in an axial force N. From this equation and the boundary condition $\sigma_{rr}|_{r_0} = 0$ on the outer surface of the tube, the radial Cauchy stress σ_{rr} may be calculated as

$$\sigma_{rr} = \int_{\xi}^{r_o} (\sigma_{rr} - \sigma_{\theta\theta}) \frac{dr}{r}, \quad r_i \le \xi \le r_o \tag{3}$$

The internal pressure $P = -\sigma_{rr}|_{r=r_i}$ is then obtained in the form

$$P = \int_{r_i}^{r_o} (\sigma_{\theta\theta} - \sigma_{rr}) \frac{dr}{r} \tag{4}$$

When the state of deformation is known, expressions for the axial force N can be calculated via the definition (Holzampfel and Gasser, 2000; Humphrey et al., 2009)

$$N = 2\pi \int_{r_i}^{r_o} \sigma_{zz} r dr \tag{5}$$

For a thick-walled cylindrical tube, the reduced equations for the mean circumferential stress ($\sigma_{\theta\theta}$) and mean axial stress (σ_{zz}) could be given simply as (Humphrey, 2008)

$$\sigma_{\theta\theta} = \frac{pd}{2h} \tag{6}$$

$$\sigma_{zz} = \frac{N}{\pi h(h + d)} \tag{7}$$

where p, d, h and N denote internal pressure, internal diameter, wall thickness and axial force of the deformed tube, respectively. By substituting $N = \rho Aha$ in Eq. (7)—where ρ, A, h and a are wall density, a unit surface element, the intima-media thickness and the axial acceleration of the arterial wall, respectively—the axial stress of the arterial wall can be determined as

$$\sigma_{zz} = \frac{\rho Aa}{\pi(h + d)} \tag{8}$$

It has been reported that there is a shear strain (and thus shear stress) between IMT and adventitia. Therefore, the longitudinal movement of the adventitia is smaller than that of IMT (Cinthio and Ahlgreny, 2010). In this study, IMT is used because it can be considered as an integral layer moving uniformly and stress