Pressurization of Axially Prestretched Tube: Consequences for Arterial Mechanics

1 Introduction

Arteries in situ are significantly prestretched in an axial direction [1-4]. It is manifested as a retraction that is observed in autopsy or surgery when arteries are excised. The axial prestretch is expressed as the ratio of in situ to ex situ length of a cylindrical segment of an artery. Axial prestretch in arteries strongly depends on age [3-5]. It gradually decreases until totally relaxed state and even pre-compressed arteries can be observed [4-6]. The prestretch also depends on anatomical location [1, 4-7]. It increases with increasing distance from the heart and is well correlated with the cross-sectional area of the vascular wall [7].

Ex vivo mechanical tests with enzymatically treated arteries [8,9] and genetically modified animal models [10,11] suggest crucial role of elastin for bearing the prestretch. Fundamental role of elastic membranes in the axial prestretching is in accordance with distally increasing magnitude of the prestretch [7]. Caudally decreasing number of elastic lamellae, which sustain approximately constant axial force in the whole aorta [7], lead to increased force per lamella which results in increasing prestretch.

The axial prestretch of arteries has important physiological function. It was found that in vivo value of the prestretch enables arteries to sustain minimal changes in axial deformation and axial force during pressure cycle [12-14]. Originally, it was assumed that nearly zero axial deformation during pressure pulse transition is a consequence of significant tethering (attachment to surrounding tissue) [12]. Experimental studies, however, showed that longitudinal immobility of arteries is related to a biological tuning that couples constitutive properties, internal structure and physiological range of arterial loading.

To be more specific, it was found that a typical in vitro inflation behavior of an artery held at constant length is such that there is a value of the axial prestretch above which force–pressure relationship creates increasing curve and under prestretch smaller than this value the force–pressure relationship is decreasing [13-15]. Contrary, pressurization experiments with constant axial load have shown that for small values of the load arteries elongate during pressurization, whereas with large values of the axial load they shorten [16-18]. It is generally accepted that in vivo value of the prestretch (and axial force) is exactly the value under which an artery neither shortens nor elongates in pressure cycle. Some authors refer this value to as inversion stretch or the stretch at inversion point [16-18].

Previous studies of the axial prestretch have revealed that it is mechanically favorable for human arteries to operate in the prestretched state [12-18]. It minimizes mechanical work necessary to transmit pressure pulse because no axial displacements of the wall during pressure cycle means no work spent on these displacements. Since real arteries are rather viscoelastic than elastic, it also means, that there will be no energy dissipated in axial movement of artery wall. In the present paper we want to show another interesting property which can be observed when nonlinear elastic tube is inflated at constant axial load. It will be shown that at axial prestretching corresponding to the inversion point, when no axial movement is exhibited during inflation, also maximum internal volume of the pressurized tube is attained. Since large arteries serve as conduit in blood transport, it suggests that they operate in mechanically optimal regime which allows maximum of the blood to be transferred in one pressure cycle.

2 Methods

Our study will not deal with any specific artery, instead we will focus our attention only to general properties known from arterial elasticity, and by means of analytical model, it will be shown that maximal internal volume of the pressurized tube corresponds to loading at inversion axial stretch. To this end, the artery considered in our model is assumed to be made from Gent hyperelastic material [19,20]. The Gent hyperelastic model is well-known in elastomer mechanics and soft tissue elasticity and is frequently used in biomechanical studies describing physiological principles in arterial mechanics. It was used for example by Horný et al. [21] in their study of aging impact on the pretension sustained by
the human abdominal aorta, by Horgan and Saccomandi [22] and Sang et al. [23] to study the inflation-extension behavior of an artery and in [22] to study longitudinal oscillations of arterial strip. It is isotropic nonlinear material model exhibiting large strain stiffening which is a property common to all soft tissues.

**Constitutive model.** The specific form of the strain energy density function \( W \) corresponding to the Gent model is expressed in (1).

\[
W = -\frac{\mu J_n}{2} \ln \left( 1 - \frac{I_1 - 3}{J_n} \right)
\]

(1)

Here \( \mu \) is stress-like material parameter which at infinitesimal strains corresponds to the shear modulus and \( J_n \) is dimensionless parameter modulating nonlinear behavior of the material. \( I_1 \) is the first principal invariant of the right Cauchy-Green strain tensor \( C = F^T F \) where \( F \) denotes tensor of the deformation gradient. The material is assumed to be incompressible and its constitutive equation is written in the form of (2).

\[
\sigma = \frac{\partial W}{\partial F} F^T - \rho I
\]

(2)

In (2), \( \sigma \) denotes Cauchy stress tensor, \( I \) denotes second order unit tensor and \( \rho \) is Lagrangean multiplier enforced by incompressibility constraint which has to be determined from force boundary condition.

**Kinematics of inflation–extension response.** Studied artery is considered to be long thin-walled tube with closed ends that, in the reference configuration, has a middle radius \( R \), thickness \( H \), and length \( L \). Assume that during pressurization, the motion of a material particle located originally at \(( R, \Theta, Z )\), which is sufficiently distant from the ends, is described by the equations summarized in (3).

\[
r = \lambda_{rr} R, \quad h = \lambda_{hh} H, \quad z = \lambda_{zz} Z, \quad \Theta = \Theta
\]

(3)

Here \( r \) and \( h \) respectively denote deformed middle radius and thickness. Equations (3) express the fact that the tube uniformly inflates and extends and that it does not twist. Stretches \( \lambda_{kk} \) \((k = r, \Theta, z; K = R, \Theta, Z)\) are the components of the deformation gradient \( F \). Consequently, \( F = diag[\lambda_{rr}, \lambda_{\Theta \Theta}, \lambda_{zz}] \). Incompressibility constraint then reads \( det(F) = \lambda_{rr} \lambda_{\Theta \Theta} \lambda_{zz} = 1 \).

**Equilibrium of thin-walled tube.** Equilibrium equations of a thin-walled closed tube loaded by an internal pressure \( P \) can be written in the form (4). Here \( \sigma_r \), \( \sigma_\Theta \), and \( \sigma_z \) respectively, denote the radial, circumferential, and axial component of the Cauchy stress tensor. The radial component was considered to be zero due to the thin-wall assumption.

\[
\sigma_r = 0 \quad \sigma_\Theta = \frac{rP}{h} \quad \sigma_z = \frac{rP}{2h} + \frac{F_{red}}{2\pi rh}
\]

(4)

\( F_{red} \) in (4c) denotes additional axial force which ensures longitudinal prestretch of the tube. This loading remains constant during the pressurization of the tube. However, axial deformation may change according to mechanical state which results from a combination of equilibrium and constitutive equations and loading by internal pressure.

**Geometry and material parameters.** Constitutive parameters characterizing artery material were adopted from literature. In [21] Horný et al. conducted uniaxial tensile tests with cylindrical segments of the human abdominal aorta and approximated mechanical response by the model (1). For the purposes
of our calculations, parameters corresponding to 48 years old female individual were chosen. Specific values used in the study are in Table 1.

<table>
<thead>
<tr>
<th>age</th>
<th>gender</th>
<th>Jm</th>
<th>µ</th>
<th>R</th>
<th>H</th>
</tr>
</thead>
<tbody>
<tr>
<td>48</td>
<td>female</td>
<td>0.22</td>
<td>23</td>
<td>5.81</td>
<td>1.62</td>
</tr>
</tbody>
</table>

Simulation of inflation-extension behavior. Quasistaic inflation-extension behavior of the artery was simulated numerically in Maple 2015. The system of equations describing response to internal pressure and axial loading is obtained by substitution of (1) to (2) and subsequent substitution (2) into (4). Finally deformed radius and thickness in (4) are substituted from (3) and \( \lambda_{zR} = (\lambda_{zR}\lambda_{zZ})^{1/2} \) is used to eliminate radial stretch. One equation, (4a), is used to determine \( p \) coming from incompressibility constraint. The result is the system of two nonlinear equations which contain material parameters, reference dimensions and \( P, F_{red}, \lambda_{ab}, \) and \( \lambda_{zZ}. \) The final form of the governing equations is expressed in (5) and (6).

\[
\begin{align*}
\left[ \frac{\partial W}{\partial \lambda_{ab} \lambda_{zZ}^{\text{ini}}} \right]_{\lambda_{ab} = \lambda_{ab}^{\text{ini}}} &= \left[ \frac{\partial W}{\partial \lambda_{R} \lambda_{zZ}} \right]_{\lambda_{ab} = \lambda_{ab}^{\text{ini}}} = \frac{P}{2\pi RH} \lambda_{zZ} R \lambda_{zZ}^{\text{ini}} \lambda_{zZ}^{\text{ini}} \\
\left[ \frac{\partial W}{\partial \lambda_{zZ}} \right]_{\lambda_{ab} = \lambda_{ab}^{\text{ini}}} &= \left[ \frac{\partial W}{\partial \lambda_{R} \lambda_{zZ}} \right]_{\lambda_{ab} = \lambda_{ab}^{\text{ini}}} = \frac{P}{2\pi RH} \lambda_{zZ}^{\text{ini}} \lambda_{zZ}^{\text{ini}}
\end{align*}
\]

One may select two variables as independent and remaining quantities are computed from equations modeling the problem. Our solution is obtained in two steps. First, axial prestretch of the tube is induced by assigning \( \lambda_{zZ} = \lambda_{zZ}^{\text{ini}} \) at \( P = 0 \). In this step, the system (5-6) is used to compute \( F_{red} \) necessary to preelongated the tube. Simultaneously initial circimferential strech \( \lambda_{ab}^{\text{ini}} \) is obtained. In the second step, (5-6) is used to compute \( \lambda_{ab} \) and \( \lambda_{zZ} \) at a given \( P \) and \( F_{red} \). The sequence 0.5\((i - 1) \) kPa for \( i = 1,..,41 \) was used to simulate pressurization of the tube. The axial prestretch \( \lambda_{zZ}^{\text{ini}} \) considered in our study was 1 + 0.02\((i - 1) \) for \( i = 1,..,14 \) that includes typical values of the prestretch in the human abdominal aorta expected at 48 years old individual [3,4,21].

3 Results and Discussion

The results obtained in our simulation are summarized in the Figure 1 – 3. The inflation-extension response expressed by means of \( P-\lambda_{ab} \) and \( P-\lambda_{zZ} \) relationships at constant axial load corresponding to \( \lambda_{zZ}^{\text{ini}} = 1, 1.02, 1.04,.., 1.26 \) is in the Figure 1. Colors of the curves uniquely correspond to selected values of \( \lambda_{zZ}^{\text{ini}} \) and specific value of the axial prestretch is evident from the right panel of the figure (\( P-\lambda_{zZ} \)). The figure clearly shows that there is a certain value of the prestretch under which the tube changes its response from inflation–extension to inflation–shortening. This is exactly inversion axial stretch and this mechanical response is highited by dotted curve and denoted by \( \lambda_{zZ}^{\text{inv}} \). Numerical computation showed that, in or specific case, it is approximately equal to 1.063.
Fig. 1. Inflation-extension response obtained from the model. $\lambda_{z\text{ini}} = 1, 1.02, 1.04,..., 1.26$ was used in the calculation. Inversion axial prestretch, under which the tube does not move axially, is highlighted by dotted line.

Figure 2 documents a relationship between inflating pressure and relative internal volume $v$, $v = \pi r^2 h/(\pi R^2 L) = \lambda_{\theta\Theta}^2 \lambda_{zZ}$. It is clear that maximal volume of the tube is attained when the tube is inflated at inversion axial stretch (dotted curve). If axes of the figure were changed (pressure as vertical axis and volume as horizontal axis), it would be evident that at the same time the inflation at inversion stretch also leads to minimal work performed during pressurization of the tube because it corresponds to minimal area under $P$–$v$ curve. To be exact, we should talk about work per unit volume instead of work because relative volume instead of volume as such is variable on the axis.

Finally Figure 3 shows traces of the kinematics of tube inflation in a phase space of the deformation. Left panel is for $\lambda_{zZ} - \lambda_{\theta\Theta}$ dependence and right panel shows dependence of relative volume $v$ on $\lambda_{zZ}$. Applied axial prestretch is indicated by color of the curve. Both $\lambda_{zZ} - \lambda_{\theta\Theta}$ and $v - \lambda_{zZ}$ also contains contours of constant pressure at $P = 0, 1, 2.5, 5, 10,$ and $20$ kPa. They are depicted with thin dotted curves. One observes that $\lambda_{zZ}^{\text{inv}}$ intersects contours of the constant pressure at their maximal volume which again documents that the inversion prestretch leads to optimal inflation conditions.

Fig. 2. Relative volume–pressure relationship at various $\lambda_{z\text{ini}}$. Dotted curve correspond to the inversion prestretch under which maximal volume is attained. Additionally to maximal volume, it is clear from the figure that also minimal work is spent when tube is inflated under inversion prestretch $\lambda_{zZ}^{\text{inv}}$. 
Although previous studies focused on the axial prestretch of arteries have pointed out that the prestretch is mechanically favorable, it was not, to the best of our knowledge, explicitly stated that the inversion stretch leads to maximal volume of the inflated tube. It suggests that tuning of the blood transport is highly sophisticated and optimized from biomechanical point of view.

Our present result is however only one cell to a jigsaw of mechanical principles acting in our cardiovascular system. One should not overestimate it because there are other questions which has to be considered and were not adressed in our study. Particullary, present result was obtained for one specific selection of material parameters but to reach physiological relevance it has to be generalized and proved for other, more accurate, models of arterial wall which will consider anisotropic behaviour of the wall. We also have to note that thin-wall assumption should be accepted with caution because it is known that arteries exhibit residual strains which cannot be modeled under this assumption. Results are also limited by the fact that our study was based on quasistatic formulation, but true blood pressure propagation is a wave problem. However, our present contribution is only preliminary study and in future we plan refine it in more accurate computational model.

Finally it is concluded that results obtained from simplified model suggest that axial prestretch in human arteries ensures not only constancy of axial force and axial deformation during pressure cycle but also maximum volume attained in the inflation of an artery.

References